Annex A. Atmospheric Science

A.1. Ambient Air Particle Monitoring

A.1.1. Measurements and Analytical Specifications

Table A-1. Summary of integrated and continuous samplers included in the field comparison.

Abbreviation	Instrument	Manufacturer / Research Institute
INTEGRATED PARTICLE OR GAS/PARTICLE INSTRUMENTS		
Dichot	Dichotomous Sampler with Virtual Impactor	Andersen Instruments (Smyrna, GA
AND-241 Dichot	Thermo Andersen Series 241 Dichotomous Sampler	Andersen Instruments
AND-246 Dichot	Thermo Andersen SA-246B Dichotomous Sampler	Andersen Instruments
AND-hIVOL10 FRM	Thermo Andersen GMW-1200 HiVol PM_{10} FRM Sampler	Andersen Instruments
ARA-PCM	ARA Particle Composition Monitor	Atmospheric Research and Analysis Inc. (Plano, TX)
CMU	CMU Speciation Sampler	Carnegie Mellon University (CMU), (Pittsburgh, PA)
DRI-SFS	DRI Sequential Filter Sampler	Desert Research Institute (Reno, NV)
HEADS (or HI)	Harvard EPA Annular Denuder System (or Harvard Impactor)	Harvard School of Public Health (Boston, MA)
IMPROVE_SS[b]	IMPROVE Speciation Sampler	URG Corp. (Chapel Hill, NC)
URG-3000N[b]	Modified IMPROVE Module C Sampler for Carbon	URG Corp.
MASS-400[b]	URG Mass Aerosol Speciation Sampler Model 400	URG Corp.
MASS-450[b]	URG MASS Model 450	URG Corp.
MiniVol	Battery-Powered Portable Low-Volume Sampler	Air Metrics Inc. (Eugene, OR)
PC-BOSS	Particle Concentrator-Brigham Young University Organic	Brigham Young University (Provo, UT)
SAMPLING SYSTEM		
PQ-200 FRM	BGI PQ-200 FRM Sampler	BGI Inc. (Waltham, MA)
PQ-200 FRMA	BGI PQ-200A FRM Audit Sampler	BGI Inc.
R&P-ACCU	R&P-Automated Cartridge Collector Unit Sampler	Rupprecht & Patashnick, Co. (Albany, NY
R&P-2000 FRM	R&P Partisol-2000 FRM Sampler	Rupprecht & Patashnick, Co.
R&P-2000 FRMA	R&P Partisol-2000 FRM Audit Sampler	Rupprecht & Patashnick, Co.
R&P-2025 Dichot[b]	R&P Partisol 2025 Dichotomous Sequential Air Sampler	Rupprecht & Patashnick, Co.
R&P-2025 FRM	R&P Partisol-Plus Model 2025 $PM_{2.5}$ Sequential Samplers	Rupprecht & Patashnick, Co.
R&P-2300[b]	R&P Partisol 2300 Chemical Speciation Sampler	Rupprecht & Patashnick, Co.

Note: Hyperlinks to the reference citations throughout this document will take you to the NCEA HERO database (Health and Environmental Research Online) at http://epa.gov/hero. HERO is a database of scientific literature used by U.S. EPA in the process of developing science assessments such as the Integrated Science Assessments (ISA) and the Integrated Risk Information System (IRIS).

Abbreviation	Instrument	Manufacturer / Research Institute
RAAS-100 FRM	Thermo Andersen Reference Ambient Air Sampler Model 100	Andersen Instruments
FRM SAMPLER		
RAAS-200 FRM	Thermo Andersen RAAS Model 200 FRM Audit Sampler	Andersen Instruments
RAAS-300 FRM	Thermo Andersen RAAS Model 300 FRM Sampler	Andersen Instruments
RAAS-400[b]	Thermo Andersen RAAS Model 400 Speciation Sampler	Andersen Instruments
SASSb	MetOne Spiral Ambient Speciation Sampler	Met One Instruments (Grants Pass, OR)
SCS	$PM_{2.5}$ Sequential Cyclone Sampler	New York University (New York, NY)
URG-PCM[b]	URG Particle Composition Monitor	URG Corp. (Chapel Hill, NC)
VAPS	URG Versatile Air Pollution Sampler	URG Corp.
CONTINUOUS MASS INSTRUMENTS		
BAM	B-Attenuation Monitor Model 1020	Met One Instruments
nano-BAM	Met One BAM Model 1020 with 150 nm impactor	Met One Instruments
CAMM	Continuous Ambient Mass Monitor	Developed by Harvard School of Public Health, comercialized by Thermo Andersen Instruments; now withdrawn from market
RAMS	Real-Time Ambient Mass Sampler (modi□ed Tapered Element Oscillation Microbalance with diffusion denuder and Na□on dryer)	Brigham Young University
TEOM	Tapered Element Oscillating Microbalance	Rupprecht & Patashnick, Co.
30 °C-TEOM	TEOM operated at 30 °C	Rupprecht & Patashnick, Co.
50 °C-TEOM	TEOM operated at 50 °C	Rupprecht & Patashnick, Co.
SES-TEOM	TEOM 1400a Series with Sample Equilibration System	Rupprecht & Patashnick, Co.
D-TEOM	Differential TEOM	Rupprecht & Patashnick, Co.
FDMS-TEOM	Filter Dynamics Measurement System TEOM	Rupprecht & Patashnick, Co.
ACCU-TEOM	TEOM 1400 Series with an automated cartridge collection unit	Rupprecht & Patashnick, Co.
CONTINUOUS PARTICLE LIGHT SCATTERING INSTRUMENTS		
Dust Trak	Dust Trak nephelometer	TSI Inc. (Shoreview, MN)
EcoTech	EcoTech Model M9003 nephelometer	EcoTech Pty Ltd., Australia (American EcoTech, Warren, RI)
NGN	NGN-2 nephelometer	Optec Inc. (Lowell, MI)
RR-M903	Radiance Research Nephelometer Model M903	Radiance Research Inc. (Seattle, WA)
CONTINUOUS ELEMENT INSTRUMENTS		
GFAAS	Graphite Furnace Atomic Absorption Spectrometry—aerosol collection as preconcentrate slurry	University of Maryland (College Park, MD)
SEAS	Semicontinuous Elements in Aerosol Sampler	University of Maryland
CONTINUOUS NITRATE INSTRUMENTS		
ADI-N	Aerosol Dynamics Inc. □ash volatilization analyzer	Aerosol Dynamics Inc. (Berkeley, CA)
ARA-N	Atmospheric Research and Analysis NO_3^- analyzer	Atmospheric Research and Analysis Inc.
R&P-8400N	R&P-8400N Flash Volatilization Continuous NO_3^- Analyzer	Rupprecht & Patashnick, Co.
CONTINUOUS SULFATE INSTRUMENTS		
ADI-S	Aerosol Dynamics Inc. Flash Volatilization Analyzer	Aerosol Dynamics Inc.
CASM	Continuous Ambient Sulfate Monitor (prototype of the TE-5020 by Thermo Electron [Franklin, MA])	Harvard School of Public Health

Abbreviation	Instrument	Manufacturer / Research Institute
R&P-8400S	R&P-8400S Flash Volatilization Continuous SO_4^{2-} Analyzer	Rupprecht & Patashnick, Co.
TE-5020	Thermo Electron Model 5020 SO_4^{2-} Particulate Analyzer	Thermo Electron Corp. (Franklin, MA)
CONTINUOUS MULTI-ION INSTRUMENTS		
AIM	Ambient Ion Monitor Model 9000 (Cl^-,NO_2^-,NO_3^-,PO_4^{3-}, SO_4^{2-}, NH_4^+,Na^+,Mg^{2+},K^+,Ca^{2+})	URG Corp.
Dionex-IC	Dionex Ion Chromatograph (F^-, Cl^-, NO_2^-, Br^-,NO_3^-, PO_4^{3-}, SO_4^{2-}, Li^+, NH_4^+ ,Na^+,Mg^{2+},K^+,Ca^{2+})	Dionex Corp.
ECN	Energy Research Center of the Netherlands IC-based sampler (Cl^-, NO_3^-, SO_4^{2+},NH_4^+ ,Na^+, Mg^{2+},K^+,Ca^{2+})	Energy Research Center of the Netherlands (Petten, the Netherlands
PILS-IC	Particle into Liquid Sampler, coupled with IC (Cl^-, NO_2^-, NO_3^-, PO_4^{3-}, SO_4^{2+},NH_4^+ ,Na^+,Mg^{2+},K^+,Ca^{2+})	Georgia Institute of Technology (Atlanta, GA)
TT	Texas Tech IC-based sampler (NO_3^-, SO_4^{2+})	Texas Tech University (Lubbock, TX)
CONTINUOUS CARBON INSTRUMENTS		
OC and EC		
ADI-C	ADI Flash Volatilization Carbon Analyzer	Aerosol Dynamics Inc.
RU-OGI	Rutgers University/Oregon Graduate Institute in-situ carbon analyzer (OC, EC)	Rutgers University (Camden, NJ)/Oregon Graduate Institute (Beaverton, OR)
R&P-5400	R&P-5400 continuous ambient carbon analyzer	Rupprecht & Patashnick, Co.
Sunset OCEC	Sunset Semi-Continuous Real-Time Carbon Aerosol Analysis Instrument	Sunset Laboratory, Inc. (Tigard, OR)
BC		
Aethalometer		Magee Scientiac Co. (Berkeley, CA)
AE-16	Magee AE-16 aethalometer (BC)	Magee Scientiac Co.
AE-20	Magee AE-20 dual wavelength aethalometer (BC)	Magee Scientiac Co.
AE-21	Magee AE-21 dual-wavelength aethalometer (BC)	Magee Scientiac Co.
AE-31	Magee AE-31 seven color aethalometer (BC)	Magee Scientiac Co.
DRI-PA	DRI Photoacoustic Analyzer (BC)	Droplet Measurement Technologies, Inc. (Boulder, CO)
MAAP	Multi-Angle Absorption Photometer, Model 5012 (BC)	Thermo Scientiac Corp. (Franklin, MA)
PSAP	Particle Soot Absorption Photometer (BC)	Radiance Research Inc. (Seattle, WA)
OTHER CARBON		
PAS-PAH	Photo-Ionization Monitor for PAHs (Model PAS 2000)	EcoChem Analytics (League City, TX)
PILS-WSOC	PILS-WSOC Analyzer, combination of PILS and total organic analyzer (TOA)	Georgia Institute of Technology
PARTICLE SIZING INSTRUMENTS FOR MASS AND CHEMICAL SPECIATION		
DRUM-3	Davis Rotating-Drum Uniform Size-Cut Monitor (0.1–2.5 μm in three stages)	University of California–Davis (Davis, CA)
DRUM-8	Davis Rotating-Drum Uniform Size-Cut Monitor (0.09- $>$ 5.0 μm in eight stages)	University of California–Davis
ELPI	Electrical Low Pressure Impactor (0.007–10 μm in 12 stages)	Dekati (Tampere, Finland)
LPI	Low Pressure Impactor (0.03–10 μm in 13 stages)	Aerosol Dynamics, Inc.
MOUDI	Micro Oriace Uniform Deposit Impactor	MSP Corp. (Minneapolis, MN)
MOUDI-100	MOUDI Model 100 (0.18–18 μm in eight stages)	MSP Corp.
MOUDI-110	MOUDI Model 110 (0.056–18 μm in 10 stages)	MSP Corp.

Abbreviation	Instrument	Manufacturer / Research Institute
Nano-MOUDI	Nano MOUDI (0.010–0.056 μm in three stages coupled to MOUDI Model 110)	MSP Corp.
PARTICLE NUMBER / VOLUME INSTRUMENTS		
APS	Aerodynamic Particle Sizer	TSI Inc.
APS-3320	TSI Model 3320 (0.5–20 μm)	TSI Inc.
APS-3321	TSI Model 3321 (0.5–20 μm; replaced TSI Model 3320)	TSI Inc.
DMA	Differential Mobility Analyzer	TSI Inc.
DMA-3081	TSI Model 3081 (0.01–1.0 m)	TSI Inc.
DMA-3085	TSI Model 3085 (0.002–0.15 μm)	TSI Inc.
EEPS	Engine Exhaust Particle Sizer (EEPS 0.056–0.56 μm)	TSI Inc.
FMPS	Fast Mobility Particle Sizer (FMPS 0.056–0.56 μm)	TSI Inc.
GRIMM-1108	Optical Particle Counter (OPC; 0.3–20 μm)	GRIMM Technologies, Inc. (Douglasville, GA)
SMPS	Scanning Mobility Particle Sizer	TSI Inc.
SMPS-3936	TSI Model 3936L (0.01–1.0 μm)	TSI Inc.
Nano-SMPS-3936	TSI Model 3936N (0.002–0.15 μm)	TSI Inc.
SMPS + C	SMPS and Condensation Nucleus Counter (0.005–0.35 or 0.01–0.875 μm)	GRIMM Technologies, Inc.
SMPS-custom	DMA Model 3071 and CPC Model 3010	TSI Inc.
WPS	Wide-Range Particle Spectrometer (0.01–10.0 μm)	MSP Corp.
SINGLE PARTICLE INSTRUMENTS		
AMS	Aerosol Mass Spectrometer (0.04–2 μm)	Aerodyne Research Inc. (Billerica, MA)
ATOFMS	Aerosol Time of Flight Mass Spectrometer (0.3–2.5 μm)	TSI Inc.
CNC, CPC	Condensation Nucleus Counters, Condensation Particle Counter	Various vendors
DAASS	Dry-Ambient Aerosol Size Spectrometer consisting of two SMPS and One APS (0.003–10 μm)	Carnegie Mellon University
LIBS	Laser-Induced Breakdown Spectroscopy	National Research Council, Industrial Materials Institute (Boucherville, Quebec, Canada)
PALMS	Particle Analysis by Laser Mass Spectrometer (0.22–2.5 μm)	NOAA (Boulder, CO)
RSMS-II	Rapid Single Particle Mass Spectrometer -II (0.035–1.1 μm)	University of Delaware (Newark, DE)
RSMS-III	Rapid Single Particle Mass Spectrometer RSMS-III (0.01–2.0 μm)	University of Delaware
LABORATORY INSTRUMENTS		
DRI Model 2001	DRI Model 2001 Thermal/Optical Carbon Analyzer (OC, EC, Eight Carbon Fractions with reflectance and transmittance laser correction)	Atmoslytic, Inc. (Calabasas, CA)
SEM	Scanning Electron Microscopy	Various vendors

[a]Now with Thermo Scientific, Franklin, MA.
[b]EPA-approved speciation sampler used in the Speciation Trends Network (STN).
[c]Now commercialized by Applikon Analytical, the Netherlands, and marketed under the name "MARGA" (Monitor for Aerosols and Gases in Ambient Air).
[d]Not available.

Source: Chow et al. (2008, 156355)

Table A-2. Summary of $PM_{2.5}$ and PM_{10} FRM and FEM samplers.

Manufacturer[a]	Sampler Name	Size Cut[b]	Description	FRM or FEM[c]	Designation #	FRN
BGI Inc.	PQ-100	PM_{10}	Louvered PM_{10} inlet; operates at flow rate of 16.7 L/min; 24-h integrated sampler; uses a mass flow meter to adjust to equivalent volumetric flow at ambient temperature and pressure.	FRM	RFPS-1298-124	Vol. 63, p. 69625, 12/17/98
BGI Inc.	PQ-200	PM_{10}		FRM	RFPS-1298-125	Vol. 63, p. 69625, 12/17/98
BGI Inc.	PQ-200	$PM_{2.5}$	Identical to PM_{10} sampler, but uses a WINS[e] impactor downstream of the PM_{10} inlet for $PM_{2.5}$ fractionation at 16.7 L/min; 24-h integrated sampler.	FRM	RFPS-0498-116	Vol. 63, p. 18911, 04/16/98
						Vol. 63, p. 31993, 06/11/98
BGI Inc.	PQ-200VSCC or PQ-200A-VSCC	$PM_{2.5}$	Same as BGI PQ200 $PM_{2.5}$ sampler, but with BGI VSCC instead of WINS impactor; PQ200A is a portable audit sampler, similar in design to PQ-200, but more compact in nature.	FEM (II)	EQPM-0202-142	Vol. 67, p. 15567, 04/02/02
R&P	R&P-2000	PM_{10}	R&P Partisol FRM Model 2000 PM_{10} sampler with louvered PM_{10} inlet; operates at flow rate of 16.7 L/min; 24-h integrated sampler; uses a mass flow meter to adjust to equivalent volumetric flow at ambient temperature and pressure; single-channel sampler.	FRM	RFPS-1298-126	Vol. 63, p. 69625, 12/17/98
R&P	R&P-2000	$PM_{2.5}$	R&P Partisol FRM Model 2000 $PM_{2.5}$ sampler, identical to PM_{10} sampler, but uses a WINS impactor downstream of the PM_{10} inlet for $PM_{2.5}$ fractionation at 16.7 L/min; 24-h integrated sampler; R&P2000A is a portable audit sampler.	FRM	RFPS-0498-117	Vol. 63, p. 18911, 04/16/98
R&P	R&P2000A	$PM_{2.5}$		FRM	RFPS-0499-129	Vol. 64, p. 19153, 04/19/99
R&P	R&P-2025	PM_{10}	R&P Partisol-Plus Model 2025 PM_{10} sequential sampler with louvered PM_{10} inlet; operates at 16.7 L/min; 24-h integrated sampler; uses a mass flow meter to adjust to equivalent volumetric flow at ambient temperature and pressure; sequential sampler with a capacity of 16 filter cassettes, allowing for two weeks of unattended daily sampling; filter exchange is performed pneumatically.	FRM	RFPS-1298-127	Vol. 63, p. 69625, 12/17/98
R&P	R&P-2025	$PM_{2.5}$	R&P Partisol-Plus Model 2025 $PM_{2.5}$ sequential sampler, identical to R&P-2025 PM_{10} sampler, but uses a WINS impactor downstream of the PM_{10} inlet for $PM_{2.5}$ fractionation at 16.7 L/min.	FRM	RFPS-0498-118	Vol. 63, p. 18911, 04/16/98
R&P	R&P2000-VSCC	$PM_{2.5}$	Same as R&P-2000 $PM_{2.5}$ sampler, but with BGI VSCC, instead of WINS impactor for $PM_{2.5}$ separation.	FEM (II)	EQPM-0202-143	Vol. 67, p. 15567, 04/02/02
R&P	R&P2000A-VSCC	$PM_{2.5}$	Same as R&P-2000A $PM_{2.5}$ sampler, but with BGI VSCC instead of WINS impactor for $PM_{2.5}$ separation.	FEM (II)	EQPM-0202-144	Vol. 67, p. 5567, 04/02/02
R&P	R&P-2025-VSCC	$PM_{2.5}$	Same as R&P-2025 $PM_{2.5}$ sampler, but with BGI VSCC instead of WINS impactor, for $PM_{2.5}$ separation.	FEM (II)	EQPM-0202-145	Vol. 67, p. 15567, 04/02/02
Andersen	RAAS-100	PM_{10}	Andersen Instruments, Inc. Model RAAS10-100 PM_{10} sampler with louvered PM_{10} inlet; operates at flow rate of 16.7 L/min; 24-h integrated sampler; volumetric flow measured by dry test meter at pump outlet modulates pump speed to maintain flow rate; single-channel.	FRM	RFPS-0699-130	Vol. 64, p. 33481, 06/23/99
Andersen	RAAS-100	$PM_{2.5}$	Graseby Andersen Model RAAS2.5-100 $PM_{2.5}$ sampler, similar to RAAS-100 PM_{10} with a WINS impactor for $PM_{2.5}$ separation.	FRM	RFPS-0598-119	Vol 63, p. 31991, 06/11/98
Andersen	RAAS200A	PM_{10}	Andersen Instruments, Inc. Model RAAS10-200 and RAAS2.5-100 Audit Samplers, portable compact version; similar to RAAS-100.	FRM	RFPS-0699-131	Vol. 64, p. 33481, 06/23/99
Andersen	RAAS-200A	$PM_{2.5}$		FRM	RFPS-0299-128	Vol. 64, p. 12167, 03/11/99
Andersen	RAAS-300	PM_{10}	Andersen Instruments, Inc. Model RAAS10-300, sequential sampler with louvered PM_{10} inlet, operates at 16.7 L/min; capacity to hold eight filter-holders for multiple day operation.	FRM	RFPS-0699-132	Vol. 64, p. 33481, 06/23/99

Manufacturer[a]	Sampler Name	Size Cut[b]	Description	FRM or FEM[c]	Designation #	FRN
Andersen	RAAS-300	$PM_{2.5}$	Graseby Andersen Model RAAS2.5-300 $PM_{2.5}$ sampler, similar to RAAS-300 PM_{10} sampler with a WINS impactor for $PM_{2.5}$ separation.	FRM	RFPS-0598-120	Vol. 63, p. 31991, 06/11/98
Thermo Scientific, Inc.	CAPS	$PM_{2.5}$	Model 605 Computer Assisted Particle Sampler (CAPS), 24-h integrated. Not available commercially.	FRM	RFPS-1098-123	Vol. 63, p. 8036, 10/29/98
Thermo Scientific, Inc.	RAAS 100-VSCC	$PM_{2.5}$	Same as RAAS-100 $PM_{2.5}$ sampler, but with BGI VSCC, instead of WINS impactor.	FEM (II)	EQPM-0804-153	Vol. 69, p. 47924, 08/06/04
Thermo Scientific, Inc.	RAAS 200-VSCC	$PM_{2.5}$	Same as RAAS-200 $PM_{2.5}$ sampler, but with BGI VSCC instead of WINS impactor.	FEM (II)	EQPM-0804-154	Vol. 69, p. 47924, 08/06/04
Thermo Scientific, Inc.	RAAS 300-VSCC	$PM_{2.5}$	Same as RAAS-300 $PM_{2.5}$ sampler, but with BGI VSCC instead of WINS impactor.	FEM (II)	EQPM-0804-155	Vol. 69, p. 47925, 08/06/04
URG Corp.	MASS-100	$PM_{2.5}$	Model MASS100 $PM_{2.5}$ sampler with louvered PM_{10} inlet followed by WINS impactor, operates at 16.7 L/min; 24-h integrated, volumetric flow measured by dry test meter at pump outlet modulates pump speed to maintain flow rate; single channel.	FRM	RFPS-0400-135	Vol. 65, p. 26603, 05/08/00
URG Corp.	MASS-300	$PM_{2.5}$	Model MASS300 $PM_{2.5}$ sampler with louvered PM_{10} inlet followed by WINS impactor, operates at 16.7 L/min; 24-h integrated, sequential sampler with circular tray holding six filters.	FRM	RFPS-0400-136	Vol. 65, p. 26603, 05/08/00
Tisch Environmental, Inc.	TE-6070 HiVol	PM_{10}	Model TE-6070 PM_{10} High-Volume Sampler, with TE-6001 PM_{10} size selective inlet; 8" x 10" filter holder.	FRM	RFPS-0202-141	Vol. 67, p. 15566, 04/02/02
Met One	BAM	PM_{10}	Models BAM 1020, GBAM 1020, BAM 1020-1, and GBAM 1020-1, with BX-802 inlet; glass-fiber filter tape with 1-h filter change frequency.	FEM	EQPM-0798-122	Vol. 63, p. 41253, 08/03/98

[a] BGI Inc.: BGI Incorporated, Waltham, MA. R&P: Rupprecht & Patashnick Company, Inc., Albany, NY, now Thermo Scientific, Inc., Franklin, MA. Andersen: Graseby Andersen, later Andersen Instruments, Inc., Smyrna, GA, now Thermo Scientific, Inc., Franklin, MA. Thermo Environmental Instruments, Inc., now Thermo Scientific, Inc., Franklin, MA. URG Corp.: URG Corporation, Chapel Hill, NC. Tisch Environmental, Inc., Cleves, OH. Met One Instruments, Inc., Grants Pass, OR

[b] The efficiency of an inlet3 is determined by its 50% cut-point (d50, the diameter at which half of the particles penetrate through the inlet, while the other half is retained by the inlet) and

[c] FRM: Federal Reference Method; FEM: Federal Equivalent Method. Roman numeral within parenthesis indicates FEM class.

[e] Particle separation in WINS is achieved by means of a single-jet round nozzle with flow directed into an impaction reservoir. The impaction surface consists of a Gelman Type A/E glass-fiber filter immersed in 1 mL of Dow Corning (Midland, MI) 704 diffusion pump oil housed in a reservoir.

Note: The geometric standard deviation (GSD, which is an indicator of the sharpness of the separation, and is derived by the square root of the ratio of particle diameters at penetrations of 16% and 84%, [d16/d84]0.5).

Source: Chow (1995, 077012); Watson and Chow (2001, 157123).

Table A-3. Measurement and analytical specifications for filter analysis of mass, elements, ions, and carbon.

Observable	Analytical Accuracy[a]	Precision[b]	Minimum Detectable Limit (MDL)	Interferences	Comparability	Data Completeness
$PM_{2.5}$ mass	± 5% [4]	± 10% [4]	0.04 $\mu g/m^3$ c to ~ 1 $\mu g/m^3$ [d 5,6]	Electrostatic charges need to be neutralized before measurement; positive (e.g., OC adsorption) and negative artifacts (e.g., nitrate volatilization)	Within 20% [4]	90 to 100% [6,7]

Observable	Analytical Accuracy[a]	Precision[b]	Minimum Detectable Limit (MDL)	Interferences	Comparability	Data Completeness
Elements	± 2 - 5% [4]	± 10% [4]	XRF: 0.4-30 ng/m^3 [g 8] PIXE: 6-360 ng/m^3 [d 9] ICP/MS: 0.004-25 ng/m^3 [10] 0.05-11.7 ng/m^3 [9,11] AAS: 0.02-7.15 ng/m^3 [12]	Volatile compounds may evaporate from filters due to vacuum in XRF and PIXE Potential contamination during extraction and incomplete extraction efficiency for ICP-MS and AAS Matrix interference and peak overlap may occur on heavily loaded samples.	10 to 30% depending on species [4]	90 to 100%h [6,7]
Nitrate	± 6% with spiked concentrations on Teflon[4] and ± 1-14% on nylon filters[13]	± 5 to 10% on replicate analysis [4,13,14] co-located precision ± 5-7%[14-16]	0.06 μg/m^3 [e] to 0.2 μg/m^3 [d 1,6,17]	Subject to volatilization from Teflon or quartz-fiber filters	Within 35% and probably greater [4]	85 to 100% [6,7]
Sulfate	± 5% [4]	± 6 to 10% [4,14,15]	0.06 μg/m^3 [e] to 0.2 μg/m^3 [d 1,6,13]	n/a	Typically within 10%; MOUDIs [13] to 20% lower than speciation samplers [4,17-19]	85 to 100% [6,7,20,21]
Ammonium	± 5% [4]	± 10% [4]	0.06 μg/m^3 [e] to 0.07 μg/m^3 [d 1,6]	Subject to volatilization from Teflon or quartz-fiber filters	Within 30% [4]	86 to 100% [6,7]
OC, EC, TC	± 5% for TC and OC. No standard exists to determine EC accuracy	OC: ± 20%	OC: 0.1 μg/m^3 [f] to 0.8 μg/m^3 [d]	Subject to adsorption (positive artifact) and volatilization (negative artifact) of organic gases to and from quartz-fiber filters	OC: Within 20 to 50%	86 to 100% [6,7]
		EC: ± 20%	EC: 0.03 μg/m^3 [d] to 0.1 μg/m^3 [f]		EC: Within 20 to 200%	
		TC: ± 10% [4]	TC: 0.8 μg/m^3 [d 1,6]		TC: Within 20% [4,17,22]	
Total mass of WSOC	DRI Model 2001 Carbon Analyzer: ± 5%[23] TOA: ± 3-7% [24,25]	DRI Model 2001 Carbon Analyzer: ± 10%[23] Sunset Carbon Analyzer: ± 3%[26] TOA: ± 5-10% [27]	DRI Model 2001 Carbon Analyzer: 0.1 - 0.23 μg C/m^3 [23] Sunset Carbon Analyzer: 0.05-0.22 μg C/m^3 [26,28] Elemental High TOC II: 0.05 μg C/m^3 [29] TOA: 0.12 μg C/m^3 [26]	Extraction efficiency and volume reduction steps	Within 17% [26]	n/a
Elements in water soluble matter: carbon, hydrogen, nitrogen, and sulfur	carbon: 1.5%; hydrogen: 3%; nitrogen: 3%; sulfur: 5% [30]	± 2%[30]	carbon: 0.3 μg/m^3 hydrogen:0.09 μg/m^3 nitrogen: 0.03 μg/m^3 sulfur: 0.10 μg/m^3 [30]	Contamination during sample drying step	n/a	n/a
Dissolved organic nitrogen	n/a	± 5-30%[31]	0.001μg N/m^3 while inorganic nitrogen is low; ≥ 0.071μg N/m^3 while inorganic nitrogen is high[31]	Concentration of inorganic nitrogen	Good correlation between UV and persulfate oxidation methods (R2 = 0.87) [31]	n/a
Neutral polyols and polyether	GC/MS: ± 4-8% [32]	GC/MS: ± 23% [33,34] Typically ± 20%, ranged from ± 10 to ± 30% [i 32,35,36,37,38] HPLC/MS: ± 5-26%[39]	GC/MS: Levoglucosan: 10 ng/m^3 [40] 2.08 ng/m^3 [j 31] 0.01-0.03 ng/m^3 [33,41] HPLC/MS: 9-648 pg/m^3 [39]	GCMS: Extraction recovery interfered by sample matrix Derivatization efficiency IC/PAD: Overlapping peaks in chromatogram	IC/PAD: Good correlation (R2 = 0.97) with HPLC/MS; and (R2 = 0.89) with GC/MS Method [42]	n/a
Mono- and Di-carboxylic acids	n/a	GC/MS: ± 5-11% on 3 replicates, ± 8 % in avg [43,44] IC: ± 10-15%[45]	GC/MS: 0.04-1.12 ng/m^3 [46] IC: 0.01-0.12 ng/m^3 [47]	GC/MS: Extraction recovery interfered by sample matrix Derivatization efficiency IC: Overlapping peaks in chromatogram	GC/MS: Within 50% for less volatile compounds [46]	n/a

Observable	Analytical Accuracy[a]	Precision[b]	Minimum Detectable Limit (MDL)	Interferences	Comparability	Data Completeness
Amino acids	n/a	± 9% [48]	1.65-23.6 pg/m^3 [k 48]	Derivatization efficiency Stability of derivatives Overlapping peaks in chromatogram	n/a	n/a
Mass of humic-like substances (HULIS)	n/a	n/a	0.083 ng/m^3 [l 49]	Separation efficiency	n/a	n/a

[a] Accuracy is the ability of analytical methods to quantify the observable of a standard reference material correctly; it does not refer to measurement accuracy if no standards available.[50]
[b] Refers to precision of co-located measurements, unless specified otherwise
[c] Based on 1 μg/filter limit of detection for 24-h samples, assuming a flow rate of 16.7 L/min
[d] Based on field blanks collected with FRM samplers; μg/filter converted to μg/m^3 basis assuming a flow rate of 16.7 L/min for 24-h
[e] Based on ½ of a 47-mm filter extracted in 15 mL deionized-distilled water (DDW) for 24-h samples, assuming a flow rate of 16.7 L/min
[f] Based on 0.2 μg/cm^2 detection limit and 13.8 cm2 deposit area for a 47-mm filter, assuming a flow rate of 16.7 L/min for 24-h
[g] Based on 24-h samples at a flow rate of 16.7 L/min and analyzed by XRF
[h] Except for samples from one FRM sampler at Atlanta Supersite, for which data recovery was 50%[7]; reason not reported.
[i] Reported as uncertainty in literature
[j] Based on 24-h samples at a flow rate of 16.7 L/min
[k] Based on 13.8 cm^2 deposit area for a 47-mm filter and extracted into a final volume of 200 μL, assuming a flow rate of 16.7 L/min for 24-h and molecular weight of amino acid = 150
[l] Based on 13.8 cm^2 deposit area for a 47-mm filter and extracted into a final volume of 200 μL, assuming a flow rate of 16.7 L/min for 24-h
n/a: Not available

[1]Chow (1995, 077012); [2]Watson and Chow (2001, 157123); [3] Watson et al. (1983, 045084); [4]Fehsenfeld et al. (2004, 156432); [5]Solomon et al. (2001, 156993); [6]Mikel (2001, 156762); [7]Mikel (2001, 156762); [8]Watson et al. (1999, 020949); [9]Solomon and Sioutas (2006, 156995); [10]Graney et al. (2004, 053756); [11]Tanaka et al. (1998, 157041); [12]Pancras et al. (2005, 098120); [13]John et al. (1988, 045903); [14]Hering and Cass (1999, 084958); [15]Fritz et al. (1989, 077387); [16]Hering et al. (1988, 036012); [17]Solomon et al. (2003, 156994); [18]Cabada et al. (2004, 148859); [19]Fine et al. (2003, 155775); [20]Hogrefe et al. (2004, 099003); [21]Drewnick et al. (2003, 099160); [22]Watson et al. (2005, 157125); [23]Ho et al. (2006, 156552); [24]Decesari et al. (2005, 144536); [25]Mayol-Bracero et al. (2002, 045010); [26]Yang et al. (2003, 156167); [27] Turšic et al. (2006, 157063); [28]Mader et al.(2004, 156724); [29]Xiao and Liu (2004, 056801); [30]Kiss et al. (2002, 156646); [31]Cornell and Jickells (1999, 156367); [32] Zheng et al. (2002, 026100); [33]Fraser et al. (2002, 140741); [34] Fraser et al. (2003, 042231) [35]Schauer er al. (1996, **051162**); [36]Fine et al. (2004); [37]Yue et al. (2004); [38]Rinehart et al. (2006, 115184); [39]Wan and Yu (2006, 157104); [40]Poore (2000, 012839); [41]Fraser et al. (2003a); [42]Engling et al. (2006, 156422); [43]Yu et al. (2005); [44]Tran et al. (2000); [45]Yao et al. (2004, 102213); [46]Li and Yu (2005, 156692); [47]Henning et al. (2003, 156539); [48]Zhang and Anastasio (2003, 157182); [49]Emmenegger et al. (2007, 156418); [50]Watson et al. (1989, 157119)

Source: Chow et al. (2008, 156355

Table A-4. Measurement and analytical specifications for filter analysis of organic species.

Organic Species	Analytical Accuracy		Precision		MDL		Interferences		Comparability
	TD	Solvent Extraction	TD	Solvent Extraction	TD	Solvent Extraction	TD	Solvent Extraction	
PAHs	± 2.8-24.1%[51] ± 4.4-29.4%[52] 13.8-26.5%[53] ± 0.5-12.9%[54] 0.05-4.83%[55]	Z-score values 0 to -1.9 [56] ± 4-8%[32] ± 6.5-22%[57]	Avg ± 3.2%, ranged from ± 0.05 to ± 11.5%55	Avg ± 8%, ranged from ± 3.8 to ± 15%[56] ± 23%[56] Avg ± 2.6%, ranged from ± 0.6 to ± 9.5%[57] typically ± 20%, ranged from ± 10 to ± 30%c [32,35-37]	0.016-0.48 ng/m^3 [a 58] 0.030-0.45 ng/m^3 [a 55]	0.83-1.66 ng/m^3 [b 38] 0.033-3.85 ng/m^3 [b 56] 0.01-0.03 ng/m^3 [33,34,37] 0.76-276 pg/m^3 [b 57]	Fragmentation of labile compounds	Possible contaminants from solvents and complicated extraction procedures Loss of volatile compounds during the extraction and pretreatment steps Possible carryover from injection port	R^2s for solvent extraction were 0.95 [58], 0.97 [55], and 0.98 [59]
n-Alkanes	n/a	± 4-8%[32]	Avg ± 3.2%, ranged from ± 0.05 to ± 11.5%[55]	± 23%[56] Typically ± 20%, from ± 10 to ± 30%c [32,35-37]	0.081-0.86 ng/m^3 [a 58] 0.061-0.97 ng/m^3 [a 55]	0.01-0.03 ng/m^3 [33,34,37]	Same as PAHs	Same as PAHs	R^2s for solvent extraction are 0.94 [58], and 0.98 [55,59]

	Analytical Accuracy		Precision		MDL		Interferences		
Hopanes	n/a	n/a	Avg ± 3.2%, ranged from ± 0.05 to ± 11.5%[55]	± 23%[56] Typically ± 20%, from ± 10 to ± 30%[c 32,35-37]	0.030-0.14 ng/m^3 [a 55]	0.83-1.66 ng/m^3 [b 38] 0.01-0.03 ng/m^3 [33,41] 0.01 ng/m^3 [37]	Same as PAHs	Same as PAHs	R^2s for solvent extraction are 0.99 [55] and 0.998 [59]
Steranes	n/a	n/a	Avg ± 3.2%, ranged from ± 0.05 to ± 11.5%[55]	n/a	0.018-0.063 ng/m^3 [a 55]	0.83-1.66 ng/m^3 [b 60]	Same as PAHs	Same as PAHs	R^2s for solvent extraction are 0.97 [55] and 0.998 [59]
Organic acids (including n-alkanoic acids, n-alkenoic acids, alkane dicarboxylic acids, aromatic carboxylic acids, resin acids)	n/a	± 4-8%[32]	± 10 to ± 29%[55]	± 24%[41] ± 23%[56] Typically ± 20%, from ± 10 to ± 30%[c 32,35-37]	Mono-carboxylic acids (C8, C12, and C16): 0.79, 2.0, and 3.2 ng/m^3 [a 54]	0.01-0.03 ng/m^3 [33,41]	Fragmentation of labile compounds Loss of polar species due to absorption onto the surface of the injector Improper station-ary phase column used during TD analysis Incomplete ther-mal desorption of analytes because of strong affinity with filter matrix	Possible conta-minants from sol-vents and com-plicated extraction procedures Loss of volatile compounds during the extraction and pretreatment steps Possible carryover from injection port Low derivatization efficiency	Correlation with solvent extraction method R2 = 0.731 [59]
Polyols and sugars, including guaiacol and substituted guaiacols, syringol and substituted syringols, anhydrosugars	n/a	± 4-8%[32]	n/a	± 23%56 Typically ± 20%, from ± 10 to ± 30%[c 32,35-37]	n/a	Levoglucosa: [10] ng/m^3 [61] 2.08 ng/m^3 [b 38] 0.01-0.03 ng/m^3 [33,41]	Same as organic acids	Same as organic acids	n/a

[a] Assumes 2.9 cm^2 filter used in analysis from a deposit area of 13.8 cm^2, and sample collection at a flow rate of 16.7 L/min for 24-h
[b] Assumes sample collection at a flow rate of 16.7 L/min for 24-h.
[c] Reported as uncertainty in literature.
[d] Assumes a final extract volume of 1 mL and sample collection at a flow rate of 16.7 L/min for 24-h. n/a: Not available

Source: [1]Chow (1995, 077012); [2]Watson and Chow (2001, 157123); [3] Watson et al. (1983, 045084); [4]Fehsenfeld et al. (2004, 156432); [5]Solomon et al. (2001, 156993); [6]Mikel (2001, 156762); [7]Mikel (2001, 156762); [8]Watson et al. (1999, 020949); [9]Solomon and Sioutas (2006, 156995); [10]Graney et al. (2004, 053756); [11]Tanaka et al. (1998, 157041); [12]Pancras et al. (2005, 098120); [13]John et al. (1988, 045903); [14]Hering and Cass (1999, 084958); [15]Fritz et al. (1989, 077387); [16]Hering et al. (1988, 036012); [17]Solomon et al. (2003, 156994); [18]Cabada et al. (2004, 148859); [19]Fine et al. (2003, 155775); [20]Hogrefe et al. (2004, 099003); [21]Drewnick et al. (2003, 099160); [22]Watson et al. (2005, 157125); [23]Ho et al. (2006, 156552); [24]Decesari et al. (2005, 144536); [25] Mayol-Bracero et al. (2002, 045010); [26]Yang et al. 2003; [27] Turšic et al. (2006, 157063); [28]Mader et al.(2004, 156724); [29]Xiao and Liu (2004, 056801); [30]Kiss et al. (2002, 156646); [31]Cornell and Jickells (1999, 156367); [32] Zheng et al. (2002, 026100); [33]Fraser et al. (2002, 140741); [34] Fraser et al. (2003, 042231) [35]Schauer er al. (1996, 051162); [36]Fine et al. (2004); [37]Yue et al. (2004); [38]Rinehart et al. (2006, 115184); [39]Wan and Yu (2006, 157104); [40]Poore (2000, 012839); [41]Fraser et al. (2003a); [42]Engling et al. (2006, 156422); [43]Yu et al. (2005); [44]Tran et al. (2000); [45]Yao et al. (2004, 102213); [46]Li and Yu (2005, 156692); [47]Henning et al. (2003, 156539); [48]Zhang and Anastasio (2003, 157182); [49]Emmenegger et al. (2007, 156418); [50](Watson et al., 1989, 157119); [51]Greaves et al. (1985, 156494); [52]Waterman et al. (2000, 157116); [53]Waterman et al. (2001, 157117); [54]Falkovich and Rudich (2001, 156427); [55]Chow et al. (2007, 156354); [56]Miguel et al. (2004, 123260); [57]Crimmins and Baker (2006, 097008); [58]Ho and Yu (2004, 156551); [59]Jeon et al. (2001, 016636); [60]Mazzoleni et al. (2007, 098038); [61]Poore (2000, 012839); [62]Butler et al. (2003); [63]Chow et al. (2006c); [64]Russell et al. (2004, 082453); [65]Grover et al. (2006); [66]Grover et al. (2005, 090044); [67]Schwab et al. (2006b); [68]Hauck et al. (2004); [69]Jaques et al. (2004, 155878); [70]Rupprecht and Patashnick (2003, 157207); [71]Pang et al (2002, 030353); [72]Eatough et al. (2001, 010303); [73]Lee et al. (2005, 128139); [74]Lee et al. (2005, 156680); [75]Babich et al. (2000, 156239);; [76]Lee et al. (2005c); [77]Lee et al. (2005, 128139); [78]Anderson and Ogren (1998, 156213); [79]Chung et al. (2001, 156357); [80]Kidwell and Ondov (2004, 155898); [81]Lithgow et al. (2004, 126616); [82]Weber et al. (2003, 157129); [83]Harrison et al. (2004); [84]Rattigan et al. (2006, 115897); [85]Wittig et al. (2004, 103413); [86]Vaughn et al. (2005, 157089); [87]Chow et al. (2005, 099030); [88]Weber et al (2001, 024640); [89]Schwab et al. (2006a); [90]Lim et al. (2003, 156697); [91]Watson and Chow (2002, 037873); [92]Venkatachari et al. (2006, 105918); [93]Bae et al. (2004a); [94]Arhami et al. (2006, 156224); [95]Park et al. (2005, 156843); [96]Bae et al. (2004b); [97]Chow et al. (2006a); [98]Arnott et al. (2005); [99]Bond et al. (1999); [100]Virkkula et al. (2005, 157097); [101]Petzold et al. (2002, 156863); [102]Park et al. (2006); [103]Arnott et al. (1999, 020650); [104]Peters et al. (2001); [105]Pitchford et al. (1997, 156872); [106]Rees et al. (2004, 097164); [107]Watson et al. (2000); [108]Lee et al. (2005, 156680); [109]Hering et al. (2004, 155837); [110]Watson et al. (1998); [111]Chakrabarti et al. (2004); [112]Mathai et al. (1990, 156741); [113]Kidwell and Ondov (2001, 017092); [114]Stanier et al. (2004); [115]Khlystov et al. (2005, 156635); [116]Takahama et al. (2004, 157038); [117]Chow et al. (2005, 156348); [118]Zhang et al. (2002); [119]Subramanian et al. (2004, 081203); [120]Chow et al. (2006b); [121]Birch and Cary (1996); [122]Birch (1998, 024953); [123]Birch and Cary (1996); [124]NIOSH (1996, 156810); [125]NIOSH (1999, 156811); [126]Chow et al. (1993); [127]Chow et al. (2007); [128]Ellis and Novakov (1982, 156416); [129]Peterson and Richards (2002, 156861); [130]Schauer et al. (2003); [131]Middlebrook et al. (2003, 042932); [132]Wenzel et al. (2003, 157139); [133]Jimenez et al. (2003); [134]Phares et al. (2003, 156866); [135]Qin and Prather (2006, 156895); [136]Zhang et al. (2005); [137]Bein et al. (2005, 156265); [138]Drewnick et al. (2004a); [139]Drewnick et al. (2004b); [140]Lake et al. (2003, 156669); [141]Lake et al. (2004, 088411)

Table A-5. Measurement and analytical specifications for continuous mass and mass surrogate instruments.

Instrument and Measurement Principle	Averaging Time	Analytical Accuracy[a]	Precision[b]	MDL	Interferences	Comparability	Data Completeness
INERTIA INSTRUMENTS							
TEOM Air is drawn through a size-selective inlet onto the filter mounted on an oscillating hollow tube. The oscillation frequency changes with mass loading on the filter, which is used to calculate mass concentration by calibrating measured frequency with standards.	10 min–24 h	± 0.75%[c]	± 5 μg/m^3 for 10-min avg[c,d] ± 1.5 μg/m^3 for 1-h avg[c,d]	0.01 μg, which is 0.06 μg/m^3 for 1-h avg[c]	Loses semi-volatile species at both 30°C and 50°C. SESTEOM, while less sensitive to relative humidity, does not completely eliminate loss of semi-volatile species	Underestimated FRM mass by 20 to 35% [62-64]	99% [65 87] to 92% [6]
FDMSTEOM. A self-referencing TEOM with a filter at 4 °C that accounts for volatile species. It is equipped with a diffusion Nafion dryer to remove particle-bound water. The Teflon (PTFE)-coated borosilicate glass-fiber filter that is maintained at 4 °C removes particles during the reference flow cycle. The flow alternates between a base and reference flow every 6 min. If a negative mass is measured during the reference flow, due to loss of volatiles from the filter, it is added to the mass made during the prior particle-laden samples to obtain total $PM_{2.5}$ concentration.	1-h - 24 h	± 0.75%[c]	< 10%[65]	0.01 μg, which is 0.06 μg/m^3 for 1-h avg[c]	n/a	9 to 30% higher than FRM mass Within 10% of mass by D- TEOM, PC- BOSS, RAMS and BAM [66,67]	95 to 99%[65,68] 57 to 65%[67]
Differential Tapered Element Oscillating Microbalance (D-TEOM) Similar to FDMS, but an electrostatic precipitator is used in place of the glass-fiber filter to remove particles during the 6 min reference flow cycle.	1-h - 24 h	± 0.75%[c]	< 10%[e 65,69,70]	0.01 μg, or 0.06 μg/m^3 for 1-h avg[c]	n/a	Within 10% of FDMS-TEOM [65,66]	86%[65]
RAMS A TEOM with a cyclone inlet, diffusion denuders, and Nafion dryer. Particles are collected on a "sandwich" filter (Teflon followed by carbon-impregnated glass-fiber filter) on the tapered oscillating element. The various denuders remove gas phase organic compounds, nitric acid, sulfur dioxide, nitrogen dioxide, ammonia, and ozone, which could otherwise be adsorbed by the TEOM filter.	10 min - 24 h	n/a	< 10%f [71]	± 1 to 2 μg/m^3 for 30-min avg [72]	n/a	10 to 20% higher than avg[g 72] FRM mass [73,74]	n/a

Instrument and Measurement Principle	Averaging Time	Analytical Accuracy[a]	Precision[b]	MDL	Interferences	Comparability	Data Completeness
PRESSURE DROP INSTRUMENT							
Continuous Ambient Mass Monitor (CAMM) Air is drawn through a Teflon-membrane filter tape and the pressure drop across the filter is monitored continuously. The proportion of pressure drop to aerosol loading is related to the PM concentration. The filter tape advances every 30–60 min to minimize volatilization and adsorption artifacts during sampling.	1-h – 24 h	n/a	28.1% for 1-h avg 15.9% for 24-h avg ($\sim$ 3.5 μg/m^3) [75]	$<$ 5 μg/m^3 for 1 h avg [75]	Needs effective sealing for good performance; even slight leaks may result in highly variable baseline. Probably less sensitive than D-TEOM or RAMS. [75,77]	Varied performance: within 2% of SES-TEOM and FRM at Houston, TX, while not correlated with D-TEOM or FRM at Rubidoux, CA.[76,77]	n/a
B-ATTENUATION INSTRUMENT							
B Attenuation Monitor (BAM) B rays electrons) are passed through a quartz-fiber filter tape on which particles are collected. The loss of electrons (B attenuation) caused by the particle loading on the filter is converted to mass concentration, after subtraction of blank filter attenuation.	1-h – 24 h	$\pm$ 3 μg for 24-h avg concentrations $<$ 100 μg/m^3 and 2% for 100 to 1,000 μg/m^3 $\pm$ 8 μg $<$ 100 μg/m^3 and 8% for 100 to 1000 μg/m^3 (1-h	$\pm$ 2 μg/m$^{3\,c,h}$	5 μg/m^3 for 1-h avg[1]	Water absorption by particles may result in higher mass measurements; maybe important at RH $>$85%	Up to 30% higher than FRM mass and within 2% of FDMS-TEOM [63,67]	93 to 99%[6,65,67]
LIGHT-SCATTERING INSTRUMENT							
Nephelometers (including DustTrak) A light source illuminates the sample air and the scattered light is detected at an angle (usually 90°) relative to the source. The signal is related to the concentration of the particles giving an estimate of the particle light scattering coefficient. Zero air calibrations can be performed using particle-free air.	5 min – 24 h	n/a	Nephelometers: $<$ 5% for TSI and NGNi nephelometers [78,79] DustTrak: Greater of 0.1% or 1 μg/m$^{3\,c,h}$	Nephelometer: $<$ 1.5 Mm-1 DustTrak: $\pm$ 1 μg/m^3 for 24-h avg[j]	Conversion factor to calculate mass concentration from bscat may vary depending on particle size, shape and composition. Light scattering by DustTrak proportional to dp ^{6} for dp $<$ 0.25 μm [79]	Typically good correlation with SES-TEOM and D-TEOM (R2 $>$0.80). Comparability depends on conversion factor used.	$>$80 to 98% for NGN2, RR-M903 and GreenTek Nephelometers [6] $>$80% for DustTrak [6 95] to 98% for GRIMM optical particle counter[65]

Instrument and Measurement Principle	Averaging Time	Analytical Accuracy[a]	Precision[b]	MDL	Interferences	Comparability	Data Completeness

a Accuracy is the ability of analytical methods to quantify the observable of a standard reference material correctly; does not refer to measurement accuracy, since no standards available.
b Refers to precision of co-located measurements, unless specified otherwise.
c Manufacturer-specified measurement parameter.
d Details not available on how the precision was obtained and whether it refers to co-located precision.
e Includes a combination of estimates: based on co-located precision and based on regression slopes.
f Co-located precision with respect to PC-BOSS reconstructed $PM_{2.5}$ mass.
g Using glass-fiber "sandwich" filter.
h Specified as "resolution" by the manufacturer.
i Co-located precision estimate based on regression slope for NGN nephelometer (slope = 1.01, intercept = -1.64 $\mu g/m^3$, R2 = 0.99).
j Specified as "Zero stability" by the manufacturer.
n/a: Not available.

Source: [1]Chow (1995, 077012); [2]Watson and Chow (2001, 157123); [3] Watson et al. (1983, 045084); [4]Fehsenfeld et al. (2004, 156432); [5]Solomon et al. (2001, 156993); [6]Mikel (2001, 156762); [7]Mikel (2001, 156762); [8]Watson et al. (1999, 020949); [9]Solomon and Sioutas (2006, 156995); [10]Graney et al. (2004, 053756); [11]Tanaka et al. (1998, 157041); [12]Pancras et al. (2005, 098120); [13]John et al. (1988, 045903); [14]Hering and Cass (1999, 084958); [15]Fritz et al. (1989, 077387); [16]Hering et al. (1988, 036012); [17]Solomon et al. (2003, 156994); [18]Cabada et al. (2004, 148859); [19]Fine et al. (2003, 155775); [20]Hogrefe et al. (2004, 099003); [21]Drewnick et al. (2003, 099160); [22]Watson et al. (2005, 157125); [23]Ho et al. (2006, 156552); [24]Decesari et al. (2005, 144536); [25] Mayol-Bracero et al. (2002, 045010); [26]Yang et al. 2003; [27] Turšic et al. (2006, 157063); [28]Mader et al.(2004, 156724); [29]Xiao and Liu (2004, 056801); [30]Kiss et al. (2002, 156646); [31]Cornell and Jickells (1999, 156367); [32] Zheng et al. (2002, 026100); [33]Fraser et al. (2002, 140741); [34] Fraser et al. (2003, 042231) [35]Schauer er al. (1996, 051162); [36]Fine et al. (2004); [37]Yue et al. (2004); [38]Rinehart et al. (2006, 115184); [39]Wan and Yu (2006, 157104); [40]Poore (2000, 012839); [41]Fraser et al. (2003a); [42]Engling et al. (2006, 156422); [43]Yu et al. (2005); [44]Tran et al. (2000); [45]Yao et al. (2004, 102213); [46]Li and Yu (2005, 156692); [47]Henning et al. (2003, 156539); [48]Zhang and Anastasio (2003, 157182); [49]Emmenegger et al. (2007, 156418); [50](Watson et al., 1989, 157119); [51]Greaves et al. (1985, 156494); [52]Waterman et al. (2000, 157116); [53]Waterman et al. (2001, 157117); [54]Falkovich and Rudich (2001, 156427); [55]Chow et al. (2007, 156354); [56]Miguel et al. (2004, 123260); [57]Crimmins and Baker (2006, 097008); [58]Ho and Yu (2004, 156551); [59]Jeon et al. (2001, 016636); [60]Mazzoleni et al. (2007, 098038); [61]Poore (2000, 012839); [62]Butler et al. (2003); [63]Chow et al. (2006c); [64]Russell et al. (2004, 082453); [65]Grover et al. (2006); [66]Grover et al. (2005, 090044); [67]Schwab et al. (2006b); [68]Hauck et al. (2004); [69]Jaques et al. (2004, 155878); [70]Rupprecht and Patashnick (2003, 157207); [71]Pang et al (2002, 030353); [72]Eatough et al. (2001, 010303); [73]Lee et al. (2005, 128139); [74]Lee et al. (2005, 156680); [75]Babich et al. (2000, 156239); [76]Lee et al. (2005c); [77]Lee et al. (2005, 128139); [78]Anderson and Ogren (1998, 156213); [79]Chung et al. (2001, 156357); [80]Kidwell and Ondov (2004, 155898); [81]Lithgow et al. (2004, 126616); [82]Weber et al. (2003, 157129); [83]Harrison et al. (2004); [84]Rattigan et al. (2006, 115897); [85]Wittig et al. (2004, 103413); [86]Vaughn et al. (2005, 157089); [87]Chow et al. (2005, 099030); [88]Weber et al (2001, 024640) ; [89]Schwab et al. (2006a); [90]Lim et al. (2003, 156697); [91]Watson and Chow (2002, 037873); [92]Venkatachari et al. (2006, 105918); [93]Bae et al. (2004a); [94]Arhami et al. (2006, 156224); [95]Park et al. (2005, 156843).

Table A-6. Measurement and analytical specifications for continuous elements.

Instrument and Measurement Principle	Averaging Time	Analytical Accuracy[a]	Precision	MDL	Interferences	Comparability	Data Completeness
Semi-continuous Elements in Aerosol System (SEAS) Particles are collected at 30-min interval for subsequent laboratory atomic absorption analysis for elements. Aerosol collection is through condensational growth by direct steam injection. The grown particles are separated from the airstream using virtual impactor. The droplets accumulate in a slurry that is pumped to a separate sample vial for each time period.	15-30 min	± 10%b for Mn, Fe, Ni, Cu, Zn, Se, Cd, and Sb ± 20%[b] for Cr, As, and Pb [80]	20 to 43%[c 80]	Al: 440 pg Cr: 6.7 pg Mn: 9.9 pg Fe: 85 pg Ni: 42 pg Cu: 26 pg Zn: 43 pg As: 27 pg Se: 33 pg Cd: 3.2 pg Sb 160 pg Pb: 31 pg[80]	Spectral interferences limit the number of elements detected simultaneously	n/a	n/a

Instrument and Measurement Principle	Averaging Time	Analytical Accuracy[a]	Precision	MDL	Interferences	Comparability	Data Completeness
Laser-Induced Breakdown Spectroscopy (LIBS) Used for in-situ single particle analysis. A high-power pulsed laser is projected into particles producing high-temperature plasma. Photons emission from relaxing atoms in the excited states provides characteristics of individual elements.	A few seconds	n/a	n/a	Na: 143 fg Mg: 53 fg Al: 184 fg Ca: 50 fg Cr: 166 fg Mn: 176 fg Cu: 15 fg[81]	n/a	n/a	n/a

[a] Accuracy is the ability of analytical methods to quantify the observable of a standard reference material correctly; does not refer to measurement accuracy, since no standards are available.
[b] Based on analysis of standard reference material (SRM) 1643d from National Institute of Standards and Technology (NIST).
[c] Based on error propagation.
n/a: Not available

Source: [80](Kidwell and Ondov, 2004, 155898); [81](Lithgow et al., 2004, 126616).

Table A-7. Measurement and analytical specifications for continuous NO_3^-.

Instrument and Measurement Principle	Averaging Time	Analytical Accuracy	Precision	MDL	Interferences	Comparability	Data Completion
FLASH VOLATIZATION INSTRUMENTS							
Aerosol Dynamics Inc. continuous nitrate analyzer (ADIN) Particle collection by humidification and impaction followed by flash volatilization and detection of the evolved gases in a chemiluminescent NO_x analyzer.	10 min	n/a	n/a	0.1 $\mu g/m^3$ for 10-min avg [82]	n/a	Within 30% of filter and continuous NO_3^-. See Weber et al. [82] for details.	93%[7]
Rupprecht and Patashnick continuous nitrate analyzer (R&P-8400N) Particle collection by impaction followed by flash volatilization and detection of the evolved gases in a chemiluminescent NO_x analyzer. A carbon honeycomb denuder, installed at the inlet to the Nafion humidifier removes nitric acid and ammonia vapor.	10 min	n/a	6.3%-23%[b] [83]	0.17 to 0.3 $\mu g/m^3$ for 24-h avg [83,84] 0.24 $\mu g/m^3$ to 0.45 $\mu g/m^3$ for 10-min avg [83,85]	Conversion and volatilization efficiency appears to depend on ambient composition; extent of underestimation increases with higher concentrations. [84,86]	20 to 45% lower than filter NO_3^- [20,82-85,87]	$>$80 to $>$94%[6,20,83-85]

Instrument and Measurement Principle	Averaging Time	Analytical Accuracy	Precision	MDL	Interferences	Comparability	Data Completion
DENUDER-DIFFERENCE INSTRUMENT							
Atmospheric Research and Analysis nitrate analyzer (ARAN) Sampled air passes through a 350ºC molybdenum (Mo) mesh that converts particulate nitrate into NO. A pre-split stream with a Teflon filter installed upstream of an identical converter (i.e., particle-free air) is used as a reference. NO in both streams is quantified by chemiluminescence and their difference determines the particulate nitrate concentration. The instrument inlet contains a potassium iodide- coated denuder to remove HNO_3 and NO_2.	30 sec	n/a	n/a	0.5 $\mu g/m^3$ for 30-sec avg [82]	n/a	Within 30% of filter and continuous NO_3^-. See Weber et al. [82] for details.	76%[7]
SAMPLE DISSOLUTION FOLLOWED BY IC ANALYSIS INSTRUMENTS							
Energy Research Center of the Netherlands (ECN) IC-based ion analyzer Collects particles into water drops using a steam jet aerosol collector, via cyclone. The combined flow from collected droplets containing dissolved aerosol components and wall steam condensate is directed to an anion IC for analysis of nitrate. Interfering gases are pre-removed by a rotating wet annular denuder system.	1-h	n/a	n/a	0.1 $\mu g/m^3$ [82]	n/a	Within 30% of filter and continuous NO_3^-. See Weber et al. [82] for details.	100%[7]
Texas Tech University (TT) ion analyzer Particles in the sample stream are processed through a cyclone and a parallel plate wet denuder, then collected alternatively on one of two 2.5 cm pre-washed glass fiber filters for a period of 15 min. The particles on the freshly sampled filter are automatically extracted for 6.5 min with water and analyzed for nitrate by IC.	15-30 min	n/a	n/a	0.010 $\mu g/m^3$ [82]	n/a	Within 30% of filter and continuous NO_3^-. See Weber et al. [82] for details.	97%[7]
Particle into Liquid Sampler-Ion Chromatography (PILS-IC) Ambient particles are mixed with saturated water vapor to produce droplets collected by impaction. The resulting liquid stream is analyzed with an IC to quantify aerosol ionic components.	1 h	n/a	10%-15%[c] [7,82,88]	0.05 to 0.1 $\mu g/m^3$ [20,82,88]	Consistent water quality is essential for good precision.	Within 10% of nylon-filter NO_3^- and 37% higher than R&P-8400N [20]	65 to 70%[20]
Dionex-IC The gas-denuded air stream enters the annular channel of a concentric nozzle, where deionized water generates a spray that entrains the particles. The flow is then drawn through a 0.5 μm pore size PTFE filter. The remaining solution is aspirated by a peristaltic pump and sent to IC for ion analysis.	1-h	n/a	14%[d] [65]	n/a	Consistent water quality is essential for good precision.	Bias of $<$ 10% relative to filter NO_3^-.[65]	n/a
Ambient Ion Monitor (AIM; Model 9000) Air is drawn through a size-selective inlet into a liquid diffusion denuder where interfering gases are removed. The stream enters a super-saturation chamber where the resulting droplets are collected through impaction. The collected particles and a fraction of the condensed water are accumulated until the particles can be injected into IC for hourly analysis.	1-h	n/a	n/a	0.1 $\mu g/m^3$ for 1-h avg[e]	n/a	n/a	n/a

Instrument and Measurement Principle	Averaging Time	Analytical Accuracy	Precision	MDL	Interferences	Comparability	Data Completion
PARTICLE MASS SPECTROMETER INSTRUMENT							
Aerosol Mass Spectrometer (AMS) Air stream is drawn through an aerodynamic lens and focused into a beam in a vacuum chamber. This aerosol beam is chopped by a mechanical chopper and the flight time of the particles through a particle-sizing chamber is determined by the time-resolved mass spectrometer measurement. The particle impacts onto a 600 °C heated plate where it decomposes and is analyzed by a quadruple mass spectrometer. The nitrate ion, along with other ions, is detected by the mass spectrometer.	A few seconds	n/a	n/a	0.03 $\mu g/m^3$ [20]	Subject to interferences from fragments of other species with mass to charge ratio in the same range as fragments of nitrate. Highly refractory materials are not detected.	Within 10% of nylon-filter NO_3 -, and within 15% of PILS-IC and 30% of R&P-8400N [20]	94 to 98%[20]

[a] Accuracy is the ability of analytical methods to quantify the observable of a standard reference material correctly; does not refer to measurement accuracy, since no standards are available.
[b] Overall uncertainty estimated by error propagation.
[c] Uncertainty estimated from uncertainties in flow rates and calibrations; does not refer to co-located precision.
[d] Co-located precision with respect to PC-BOSS $PM_{2.5}$ total particulate NO_3 (the sum of the denuded front filter [non-volatilized NO_3-] and HNO_3-absorbing backup filter [volatilized NO_3]).
[e] Manufacturer specified measurement parameter
n/a: Not available.

Source: [1]Chow (1995, 077012); [2]Watson and Chow (2001, 157123); [3] Watson et al. (1983, 045084); [4]Fehsenfeld et al. (2004, 156432); [5]Solomon et al. (2001, 156993); [6]Mikel (2001, 156762); [7]Mikel (2001, 156762); [8]Watson et al. (1999, 020949); [9]Solomon and Sioutas (2006, 156995); [10]Graney et al. (2004, 053756); [11]Tanaka et al. (1998, 157041); [12]Pancras et al. (2005, 098120); [13]John et al. (1988, 045903); [14]Hering and Cass (1999, 084958); [15]Fritz et al. (1989, 077387); [16]Hering et al. (1988, 036012); [17]Solomon et al. (2003, 156994); [18]Cabada et al. (2004, 148859); [19]Fine et al. (2003, 155775); [20]Hogrefe et al. (2004, 099003); [21]Drewnick et al. (2003, 099160); [22]Watson et al. (2005, 157125); [23]Ho et al. (2006, 156552); [24]Decesari et al. (2005, 144536); [25] Mayol-Bracero et al. (2002, 045010); [26]Yang et al. 2003; [27] Turšic et al. (2006, 157063); [28]Mader et al.(2004, 156724); [29]Xiao and Liu (2004, 056801); [30]Kiss et al. (2002, 156646); [31]Cornell and Jickells (1999, 156367); [32] Zheng et al. (2002, 026100); [33]Fraser et al. (2002, 140741); [34] Fraser et al. (2003, 042231) [35]Schauer er al. (1996, 051162); [36]Fine et al. (2004); [37]Yue et al. (2004); [38]Rinehart et al. (2006, 115184); [39]Wan and Yu (2006, 157104); [40]Poore (2000, 012839); [41]Fraser et al. (2003a); [42]Engling et al. (2006, 156422); [43]Yu et al. (2005); [44]Tran et al. (2000); [45]Yao et al. (2004, 102213); [46]Li and Yu (2005, 156692); [47]Henning et al. (2003, 156539); [48]Zhang and Anastasio (2003, 157182); [49]Emmenegger et al. (2007, 156418); [50](Watson et al., 1989, 157119); [51]Greaves et al. (1985, 156494); [52]Waterman et al. (2000, 157116); [53]Waterman et al. (2001, 157117); [54]Falkovich and Rudich (2001, 156427); [55]Chow et al. (2007, 156354); [56]Miguel et al. (2004, 123260); [57]Crimmins and Baker (2006, 097008); [58]Ho and Yu (2004, 156551); [59]Jeon et al. (2001, 016636); [60]Mazzoleni et al. (2007, 098038); [61]Poore (2000, 012839); [62]Butler et al. (2003); [63]Chow et al. (2006c); [64]Russell et al. (2004, 082453); [65]Grover et al. (2006); [66]Grover et al. (2005, 090044); [67]Schwab et al. (2006b); [68]Hauck et al. (2004); [69]Jaques et al. (2004, 155878); [70]Rupprecht and Patashnick (2003, 157207); [71]Pang et al (2002, 030353); [72]Eatough et al. (2001, 010303); [73]Lee et al. (2005, 128139); [74]Lee et al. (2005, 156680); [75]Babich et al. (2000, 156239); [76]Lee et al. (2005c); [77]Lee et al. (2005, 128139); [78]Anderson and Ogren (1998, 156213); [79]Chung et al. (2001, 156357); [80]Kidwell and Ondov (2004, 155898); [81]Lithgow et al. (2004, 126616); [82]Weber et al. (2003, 157129); [83]Harrison et al. (2004); [84]Rattigan et al. (2006, 115897); [85]Wittig et al. (2004, 103413); [86]Vaughn et al. (2005, 157089); [87]Chow et al. (2005, 099030); [88]Weber et al (2001, 024640) ; [89]Schwab et al. (2006a); [90]Lim et al. (2003, 156697); [91]Watson and Chow (2002, 037873); [92]Venkatachari et al. (2006, 105918); [93]Bae et al. (2004a); [94]Arhami et al. (2006, 156224); [95]Park et al. (2005, 156843); [96]Bae et al. (2004b); [97]Chow et al. (2006a); [98]Arnott et al. (2005); [99]Bond et al. (1999); [100]Virkkula et al. (2005, 157097); [101]Petzold et al. (2002, 156863); [102]Park et al. (2006); [103]Arnott et al. (1999, 020650); [104]Peters et al. (2001); [105]Pitchford et al. (1997, 156872); [106]Rees et al. (2004, 097164); [107]Watson et al. (2000); [108]Lee et al. (2005, 156680); [109]Hering et al. (2004, 155837); [110]Watson et al. (1998); [111]Chakrabarti et al. (2004); [112]Mathai et al. (1990, 156741); [113]Kidwell and Ondov (2001, 017092); [114]Stanier et al. (2004); [115]Khlystov et al. (2005, 156635); [116]Takahama et al. (2004, 157038); [117]Chow et al. (2005, 156348); [118]Zhang et al. (2002); [119]Subramanian et al. (2004, 081203); [120]Chow et al. (2006b); [121]Birch and Cary (1996); [122]Birch (1998, 024953); [123]Birch and Cary (1996); [124]NIOSH (1996, 156810); [125]NIOSH (1999, 156811); [126]Chow et al. (1993); [127]Chow et al. (2007); [128]Ellis and Novakov (1982, 156416); [129]Peterson and Richards (2002, 156861); [130]Schauer et al. (2003); [131]Middlebrook et al. (2003, 042932); [132]Wenzel et al. (2003, 157139); [133]Jimenez et al. (2003); [134]Phares et al. (2003, 156866); [135]Qin and Prather (2006, 156895); [136]Zhang et al. (2005); [137]Bein et al. (2005, 156265); [138]Drewnick et al. (2004a); [139]Drewnick et al. (2004b); [140]Lake et al. (2003, 156669); [141]Lake et al. (2004, 088411).

Table A-8. Measurement and analytical specifications for continuous SO_4^{2-}.

Instrument and Measurement Principle	Averaging Time	Analytical Accuracy[a]	Precision	MDL	Interferences	Comparability	Data Completeness
FLASH VOLATILIZATION INSTRUMENTS							
Aerosol Dynamics, Inc. continuous sulfate analyzer (ADI-S) Particle collection by impaction followed by flash volatilization and detection of the evolved gases by a UV-fluorescence SO_2 analyzer.	10 min	n/a	n/a	0.4 $\mu g/m^3$ [82]	n/a	Within 15% of filter and continuous SO_4^{2-} See Weber et al. [82] for details	100%[7]

Instrument and Measurement Principle	Averaging Time	Analytical Accuracy[a]	Precision	MDL	Interferences	Comparability	Data Completeness
Rupprecht and Patashnick continuous sulfate analyzer (R&P-8400S) Particle collection by impaction followed by flash volatilization and detection of the evolved gases by a UV-fluorescence SO_2 analyzer. An activated carbon denuder at the inlet to the Nafion humidifier removes SO_2.	10 min	n/a	25% on avg $<$ 15% at conc. >9 $\mu g/m^3$ and >30% at conc. <2 $\mu g/m^3$ [b 84]	0.48 $\mu g/m^3$ [85]	SO_4^{2-} to SO_2 conversion and volatilization efficiency appears to depend on ambient composition [84]	10 to 30% lower than filter SO_4^{2-} [20,21,84]	84 to 95%[6,20,21,84,85]
THERMAL REDUCTION INSTRUMENTS							
Continuous Ambient Sulfate Monitor (CASM) Sampled air passes through a Na_2CO_3 coated annular denuder to remove ambient SO_2 and is subsequently split into independent sample and filter flows. The sample flow passes through a quartz tube containing a stainless steel rod maintained at 1000 °C that reduces sulfate to SO_2. The flow then passes through a PTFE filter and into a trace-level SO_2 fluorescence analyzer.	15 min	n/a	n/a	n/a	n/a	Up to 25% lower than filter SO_4^{2-} and within 6% of R&P8400S, PILS-IC and AMS [20,21]	80 to 98%[20,21]
Thermo Electron Model 5020 sulfate particulate analyzer (TE-5020) The commercial version of CASM, with slight changes in the sample flow path.	15 min	n/a	$<$ 10%c [89]	0.3 $\mu g/m^3$ for 24-h avg [89] 0.5 $\mu g/m^3$ for 15-min avgd	SO_4^{2-} to SO_2 conversion efficiency depends on ambient composition [89]	$\sim$20% lower than filter SO_4^{2-} [89]	88 to 90%[89]
SAMPLE DISSOLUTION FOLLOWED BY IC ANALYSIS INSTRUMENTS							
Energy Research Center of the Netherlands (ECN) IC-based ion analyzer Entrains particles into water drops using the steam jet aerosol collector. The drops are collected using a cyclone and the combined flow from collected droplets containing dissolved aerosol components and wall steam condensate is directed to an anion IC for analysis of sulfate. Interfering gases are pre-removed by a rotating wet annular denuder system.	1-h	n/a	n/a	n/a	n/a	Within 15% of filter and continuous SO_4^{2-} See Weber et al. [82] for details.	100%[7]
Texas Tech University (TT) ion analyzer Particles in the sample stream, after being processed through a cyclone and a parallel plate wet denuder, are collected alternatively on one of two 2.5 cm pre-washed glass fiber filters for a period of 15 min. The particles on the freshly sampled filter are automatically extracted for 6.5 min with water and analyzed for sulfate by IC.	30 min	n/a	n/a	n/a	n/a	Within 15% of filter and continuous SO_4^{2-} See Weber et al. [82] for details.	100%[7]
Particle into Liquid Sampler-Ion Chromatography (PILS-IC) Ambient particles are mixed with saturated water vapor to produce droplets collected by impaction. The resulting liquid stream is analyzed with an IC to quantify aerosol ionic components.	1-h	n/a	10%-15%[e] [7,82,88]	0.1 to 0.[18] $\mu g/m^3$ [82,88]	Consistent water quality is essential for good precision.	Within 30% of filter and other continuous SO_4^{2-} [20,21]	65 to 70%[20,21]
Dionex-IC The gas-denuded air stream enters the annular channel of a concentric nozzle, where deionized water generates a spray that entrains the particles. The flow is then drawn through a 0.5 μm pore size PTFE filter. The remaining solution is aspirated by a peristaltic pump and sent to IC for ion analysis.	1-h	n/a	11%f [65]	n/a	Consistent water quality is essential for good precision.	Within 10% of filter SO_4^{2-} [65]	n/a

Instrument and Measurement Principle	Averaging Time	Analytical Accuracy[a]	Precision	MDL	Interferences	Comparability	Data Completeness
Ambient Ion Monitor (AIM; Model 9000) Air is drawn through a size-selective inlet into a liquid diffusion denuder where interfering gases are removed. The stream enters a super saturation chamber where the resulting droplets are collected through impaction. The collected particles and a fraction of the condensed water are accumulated until the particles can be injected into IC for hourly analysis.	1-h	n/a	n/a	0.1 $\mu g/m^3$ for 1-h avgd	n/a	n/a	n/a
PARTICLE MASS SPECTROMETER							
Aerosol Mass Spectrometer (AMS) Airstream is drawn through an aerodynamic lens and focused into a beam in a vacuum chamber. This aerosol beam is chopped by a mechanical chopper and the flight time of the particles through a particle-sizing chamber is determined by the time-resolved mass spectrometer measurement. The particle impacts onto a 600 °C heated plate where it decomposes and is analyzed by a quadruple mass spectrometer. The sulfate ion, along with other ions, is detected by the mass spectrometer.	A few seconds	n/a	n/a	n/a	Subject to interferences from fragments of other species with mass to charge ratio in the same range as fragments of sulfate. Highly refractory materials are not detected.	Up to 30% lower than filter SO_4^{2-} and within 5% of R&P8400S, PILS-IC and CASM [20,21]	93 to 98%[20,21]

[a] Accuracy is the ability of analytical methods to quantify the observable of a standard reference material correctly; does not refer to measurement accuracy, since no standards available.
[b] Overall uncertainty estimated by error propagation.
[c] Co-located precision estimate based on regression slope (slope = 0.95, intercept = 0.01 to 0.2, R2 > 0.98).
[d] Manufacturer specified measurement parameter.
[e] Uncertainty estimated from uncertainties in flow rates and calibrations; does not refer to co-located precision.
f Co-located precision with respect to PC-BOSS $PM_{2.5}$ SO_4^{2-}
n/a: Not available

Source: [1]Chow (1995, 077012); [2]Watson and Chow (2001, 157123); [3] Watson et al. (1983, 045084); [4]Fehsenfeld et al. (2004, 156432); [5]Solomon et al. (2001, 156993); [6]Mikel (2001, 156762); [7]Mikel (2001, 156762); [8]Watson et al. (1999, 020949); [8]Solomon and Sioutas (2006, 156995); [10]Graney et al. (2004, 053756); [11]Tanaka et al. (1998, 157041); [12]Pancras et al. (2005, 098120); [13]John et al. (1988, 045903); [14]Hering and Cass (1999, 084958); [15]Fritz et al. (1989, 077387); [16]Hering et al. (1988, 036012); [17]Solomon et al. (2003, 156994); [18]Cabada et al. (2004, 148859); [19]Fine et al. (2003, 155775); [20]Hogrefe et al. (2004, 099003); [21]Drewnick et al. (2003, 099160); [22]Watson et al. (2005, 157125); [23]Ho et al. (2006, 156552); [24]Decesari et al. (2005, 144536); [25] Mayol-Bracero et al. (2002, 045010); [26]Yang et al. 2003; [27] Turšic et al. (2006, 157063); [28]Mader et al.(2004, 156724); [29]Xiao and Liu (2004, 056801); [30]Kiss et al. (2002, 156646); [31]Cornell and Jickells (1999, 156367); [32] Zheng et al. (2002, 026100); [33]Fraser et al. (2002, 140741); [34] Fraser et al. (2003, 042231) [35]Schauer er al. (1996, 051162); [36]Fine et al. (2004); [37]Yue et al. (2004); [38]Rinehart et al. (2006, 115184); [39]Wan and Yu (2006, 157104); [40]Poore (2000, 012839); [41]Fraser et al. (2003a); [42]Engling et al. (2006, 156422); [43]Yu et al. (2005); [44]Tran et al. (2000); [45]Yao et al. (2004, 102213); [46]Li and Yu (2005, 156692); [47]Henning et al. (2003, 156539); [48]Zhang and Anastasio (2003, 157182); [49]Emmenegger et al. (2007, 156418); [50](Watson et al., 1989, 157119); [51]Greaves et al. (1985, 156494); [52]Waterman et al. (2000, 157116); [53]Waterman et al. (2001, 157117); [54]Falkovich and Rudich (2001, 156427); [55]Chow et al. (2007, 156354); [56]Miguel et al. (2004, 123260); [57]Crimmins and Baker (2006, 097008); [58]Ho and Yu (2004, 156551); [59]Jeon et al. (2001, 016636); [60]Mazzoleni et al. (2007, 098038); [61]Poore (2000, 012839); [62]Butler et al. (2003); [63]Chow et al. (2006c); [64]Russell et al. (2004, 082453); [65]Grover et al. (2006); [66]Grover et al. (2005, 090044); [67]Schwab et al. (2006b); [68]Hauck et al. (2004); [69]Jaques et al. (2004, 155878); [70]Rupprecht and Patashnick (2003, 157207); [71]Pang et al (2002, 030353); [72]Eatough et al. (2001, 010303); [73]Lee et al. (2005, 128139); [74]Lee et al. (2005, 156680); [75]Babich et al. (2000, 156239); [76]Lee et al. (2005c); [77]Lee et al. (2005, 128139); [78]Anderson and Ogren(1998, 156213); [79]Chung et al. (2001, 156357); [80]Kidwell and Ondov (2004, 155898); [81]Lithgow et al. (2004, 126616); [82]Weber et al. (2003, 157129); [83]Harrison et al. (2004); [84]Rattigan et al. (2006, 115897); [85]Wittig et al. (2004, 103413); [86]Vaughn et al. (2005, 157089); [87]Chow et al. (2005, 099030); [88]Weber et al (2001, 024640) ; [89]Schwab et al. (2006a); [90]Lim et al. (2003, 156697); [91]Watson and Chow (2002, 037873); [92]Venkatachari et al. (2006, 105918); [93]Bae et al. (2004a); [94]Arhami et al. (2006, 156224); [95]Park et al. (2005, 156843); [96]Bae et al. (2004b); [97]Chow et al. (2006a); [98]Arnott et al. (2005); [99]Bond et al. (1999); [100]Virkkula et al. (2005, 157097); [101]Petzold et al. (2002, 156863); [102]Park et al. (2006); [103]Arnott et al. (1999, 020650); [104]Peters et al. (2001); [105]Pitchford et al. (1997, 156872); [106]Rees et al. (2004, 097164); [107]Watson et al. (2000); [108]Lee et al. (2005, 156680); [109]Hering et al. (2004, 155837); [110]Watson et al. (1998); [111]Chakrabarti et al. (2004); [112]Mathai et al. (1990, 156741); [113]Kidwell and Ondov (2001, 017092); [114]Stanier et al. (2004); [115]Khlystov et al. (2005, 156635); [116]Takahama et al. (2004, 157038); [117]Chow et al. (2005, 156348); [118]Zhang et al. (2002); [119]Subramanian et al. (2004, 081203); [120]Chow et al. (2006b); [121]Birch and Cary (1996); [122]Birch (1998, 024953); [123]Birch and Cary (1996); [124]NIOSH (1996, 156810); [125]NIOSH (1999, 156811); [126]Chow et al. (1993); [127]Chow et al. (2007); [128]Ellis and Novakov (1982, 156416); [129]Peterson and Richards (2002, 156861); [130]Schauer et al. (2003); [131]Middlebrook et al. (2003, 042932); [132]Wenzel et al. (2003, 157139); [133]Jimenez et al. (2003); [134]Phares et al. (2003, 156866); [135]Qin and Prather (2006, 156895); [136]Zhang et al. (2005); [137]Bein et al. (2005, 156265); [138]Drewnick et al. (2004a); [139]Drewnick et al. (2004b); [140]Lake et al. (2003, 156669); [141]Lake et al. (2004, 088411)

Table A-9. Measurement and analytical specifications for ions other than NO_3^- and SO_4^{2-}.

Instrument & Measurement Principle	Averaging Time	Analytical Accuracy[a]	Precision	Minimum Detectable Limit (Mdl)	Interferences	Comparability	Data Completeness
SAMPLE DISSOLUTION FOLLOWED BY IC ANALYSIS INSTRUMENTS							
NO_2^- by Particle into Liquid Sampler-Ion Chromatography (PILS-IC) Ambient particles are mixed with saturated water vapor to produce droplets collected by impaction. The resulting liquid stream is analyzed with an IC to quantify aerosol ionic components.	1-h	n/a	10%[b 88]	0.14 $\mu g/m^3$ [20]	Consistent water quality is essential for good precision	n/a	n/a
NH_4^+ by Particle into Liquid Sampler-Ion Chromatography (PILS-IC) Ambient particles are mixed with saturated water vapor to produce droplets collected by impaction. The resulting liquid stream is analyzed with an IC to quantify aerosol ionic components.	1-h	n/a	10%[b 88]	0.05 $\mu g/m^3$ [88]	Consistent water quality is essential for good precision	~5% lower than all-sampler avgc at Atlanta [7]	n/a
Cl^-, Na^+, K^+, Ca^{++} by Particle into Liquid Sampler-Ion Chromatography (PILS-IC) Ambient particles are mixed with saturated water vapor to produce droplets collected by impaction. The resulting liquid stream is analyzed with an IC to quantify aerosol ionic components.	1-h	n/a	10%[b 88]	0.1 $\mu g/m^3$ [88]	Consistent water quality is essential for good precision	n/a	n/a
Cl^-, NO_2^-, NO_3^-, PO_4^{3-}, SO_4^{2-}, NH_4^+, Na^+, Mg^{++}, K^+, Ca^{++} by Ambient Ion Monitor (AIM; Model 9000) Air is drawn through a size-selective inlet into a liquid diffusion denuder where interfering gases are removed. The stream enters a super saturation chamber where the resulting droplets are collected through impaction. The collected particles and a fraction of the condensed water are accumulated until the particles can be injected into IC for hourly analysis.	1-h	n/a	n/a	0.1 $\mu g/m^3$ for 1-h avgd	n/a	n/a	n/a

Instrument & Measurement Principle	Averaging Time	Analytical Accuracy[a]	Precision	Minimum Detectable Limit (Mdl)	Interferences	Comparability	Data Completeness

[a] Accuracy is the ability of analytical methods to quantify the observable of a standard reference material correctly; does not refer to measurement accuracy, since no standards are available.
[b] Uncertainty estimated from uncertainties in flow rates and calibrations; does not refer to co-located precision.
[c] All-sampler avg appears to include a combination of 10 integrated and 3 continuous samplers, although specific details are missing 7. Performance evaluations at sites dominated by semi-volatile ammonium nitrate are needed.
[d] Manufacturer specified measurement parameter

Source: [1]Chow (1995, 077012); [2]Watson and Chow (2001, 157123); [3] Watson et al. (1983, 045084); [4]Fehsenfeld et al. (2004, 156432); [5]Solomon et al. (2001, 156993); [6]Mikel (2001, 156762); [7]Mikel (2001, 156762); [8]Watson et al. (1999, 020949); [9]Solomon and Sioutas (2006, 156995); [10]Graney et al. (2004, 053756); [11]Tanaka et al. (1998, 157041); [12]Pancras et al. (2005, 098120); [13]John et al. (1988, 045903); [14]Hering and Cass (1999, 084958); [15]Fritz et al. (1989, 077387); [16]Hering et al. (1988, 036012); [17]Solomon et al. (2003, 156994); [18]Cabada et al. (2004, 148859); [19]Fine et al. (2003, 155775); [20]Hogrefe et al. (2004, 099003); [21]Drewnick et al. (2003, 099160); [22]Watson et al. (2005, 157125); [23]Ho et al. (2006, 156552); [24]Decesari et al. (2005, 144536); [25] Mayol-Bracero et al. (2002, 045010); [26]Yang et al. 2003; [27] Turšic et al. (2006, 157063); [28]Mader et al.(2004, 156724); [29]Xiao and Liu (2004, 056801); [30]Kiss et al. (2002, 156646); [31]Cornell and Jickells (1999, 156367); [32] Zheng et al. (2002, 026100); [33]Fraser et al. (2002, 140741); [34] Fraser et al. (2003, 042231) [35]Schauer er al. (1996, 051162); [36]Fine et al. (2004); [37]Yue et al. (2004); [38]Rinehart et al. (2006, 115184); [39]Wan and Yu (2006, 157104); [40]Poore (2000, 012839); [41]Fraser et al. (2003a); [42]Engling et al. (2006, 156422); [43]Yu et al. (2005); [44]Tran et al. (2000); [45]Yao et al. (2004, 102213); [46]Li and Yu (2005, 156692); [47]Henning et al. (2003, 156539); [48]Zhang and Anastasio (2003, 157182); [49]Emmenegger et al. (2007, 156418); [50](Watson et al., 1989, 157119); [51]Greaves et al. (1985, 156494); [52]Waterman et al. (2000, 157116); [53]Waterman et al. (2001, 157117); [54]Falkovich and Rudich (2001, 156427); [55]Chow et al. (2007, 156354); [56]Miguel et al. (2004, 123260); [57]Crimmins and Baker (2006, 097008); [58]Ho and Yu (2004, 156551); [59]Jeon et al. (2001, 016636); [60]Mazzoleni et al. (2007, 098038); [61]Poore (2000, 012839); [62]Butler et al. (2003); [63]Chow et al. (2006c); [64]Russell et al. (2004, 082453); [65]Grover et al. (2006); [66]Grover et al. (2005, 090044); [67]Schwab et al. (2006b); [68]Hauck et al. (2004); [69]Jaques et al. (2004, 155878); [70]Rupprecht and Patashnick (2003, 157207); [71]Pang et al (2002, 030353); [72]Eatough et al. (2001, 010303); [73]Lee et al. (2005, 128139); [74]Lee et al. (2005, 156680); [75]Babich et al. (2000, 156239); [76]Lee et al. (2005c); [77]Lee et al. (2005, 128139); [78]Anderson and Ogren (1998, 156213); [79]Chung et al. (2001, 156357); [80]Kidwell and Ondov (2004, 155898); [81]Lithgow et al. (2004, 126616); [82]Weber et al. (2003, 157129); [83]Harrison et al. (2004); [84]Rattigan et al. (2006, 115897); [85]Wittig et al. (2004, 103413); [86]Vaughn et al. (2005, 157089); [87]Chow et al. (2005, 099030); [88]Weber et al (2001, 024640) ; [89]Schwab et al. (2006a); [90]Lim et al. (2003, 156697); [91]Watson and Chow (2002, 037873); [92]Venkatachari et al. (2006, 105918); [93]Bae et al. (2004a); [94]Arhami et al. (2006, 156224); [95]Park et al. (2005, 156843); [96]Bae et al. (2004b); [97]Chow et al. (2006a); [98]Arnott et al. (2005); [99]Bond et al. (1999); [100]Virkkula et al. (2005, 157097); [101]Petzold et al. (2002, 156863); [102]Park et al. (2006); [103]Arnott et al. (1999, 020650); [104]Peters et al. (2001); [105]Pitchford et al. (1997, 156872); [106]Rees et al. (2004, 097164); [107]Watson et al. (2000); [108]Lee et al. (2005, 156680); [109]Hering et al. (2004, 155837); [110]Watson et al. (1998); [111]Chakrabarti et al. (2004); [112]Mathai et al. (1990, 156741); [113]Kidwell and Ondov (2001, 017092); [114]Stanier et al. (2004); [115]Khlystov et al. (2005, 156635); [116]Takahama et al. (2004, 157038); [117]Chow et al. (2005, 156348); [118]Zhang et al. (2002); [119]Subramanian et al. (2004, 081203); [120]Chow et al. (2006b); [121]Birch and Cary (1996); [122]Birch (1998, 024953); [123]Birch and Cary (1996); [124]NIOSH (1996, 156810); [125]NIOSH (1999, 156811); [126]Chow et al. (1993); [127]Chow et al. (2007); [128]Ellis and Novakov (1982, 156416); [129]Peterson and Richards (2002, 156861); [130]Schauer et al. (2003); [131]Middlebrook et al. (2003, 042932); [132]Wenzel et al. (2003, 157139); [133]Jimenez et al. (2003); [134]Phares et al. (2003, 156866); [135]Qin and Prather (2006, 156895); [136]Zhang et al. (2005); [137]Bein et al. (2005, 156265); [138]Drewnick et al. (2004a); [139]Drewnick et al. (2004b); [140]Lake et al. (2003, 156669); [141]Lake et al. (2004, 088411)

Table A-10. Measurement and analytical specifications for continuous carbon.

Instrument and Measurement Principle	Averaging Time	Analytical Accuracy[a]	Precision	Minimum Detectable Limit	Interferences	Comparability	Data Completeness
PARTICLE COLLECTION ON IMPACTOR FOLLOWED BY FLASH VOLATILIZATION INSTRUMENT							
Aerosol Dynamic Inc. continuous carbon analyzer (ADI-C) Particle collection by impaction followed by flash oxidation and detection of the evolved gases by a non-dispersive infrared CO_2 analyzer. OC is estimated as twice the oxidizable carbon. EC is not quantified.	10 min	n/a	n/a	OC: 2 $\mu g/m^3$ EC, TC: not applicable, since it measures only OC [90]	n/a	15 to 22% lower OC than that by R&P-5400 and RU-OGI	83%[7]
PARTICLE COLLECTION ON FILTER / IMPACTOR FOLLOWED BY HEATING/ANALYSIS INSTRUMENTS							
Rupprecht and Patashnick 5400 continuous ambient carbon analyzer (R&P-5400) Particles collected on an impactor, which is heated to 275 °C to 350 °C, then to 700 °C after sample collection is complete. Evolved CO_2 is measured by an infrared detector. OC is defined as the carbon measured at the lower temperature, and EC is the remaining carbon measured at the higher temperature.	1-h	n/a	n/a	OC: 0.5 $\mu g/m^3$ EC: 0.5 $\mu g/m^3$ TC: 0.5 $\mu g/m^3$ [90]	n/a	20 to 60% lower TC than filter TC by TOR or TOT.[91,92]	56 to 60%[8,91]

Instrument and Measurement Principle	Averaging Time	Analytical Accuracy[a]	Precision	Minimum Detectable Limit	Interferences	Comparability	Data Completeness
Rutgers University-Oregon Graduate Institute (RU-OGI) in-situ thermal/optical transmittance carbon analyzer. Air is sampled through a quartz-fiber filter for 1-h and then analyzed by heating through different temperature steps to determine OC and EC. Sample flow is pre-split into two identical systems that alternate every hour between sampling and analysis mode to achieve continuous measurements.	30 min	n/a	3%[b,7]	OC: 0.3 $\mu g/m^3$ EC: 0.5 $\mu g/m^3$ TC: 0.4 $\mu g/m^3$ [90]	n/a	8% higher OC and 20% lower EC than R&P5400 [90]	86%7
Sunset semi-continuous realtime carbon aerosol analysis instrument (Sunset OCEC) Particles collected on a quartz-fiber filter are subject to heating temperature ramps following the NIOSH 5040 TOT protocol and the resulting CO_2 is analyzed by nondispersive infrared (NDIR) detector to quantify OC and EC. Instrument is alternated between sampling and analytical mode.	1-h	n/a	OC: 10%[c] EC: 20%[c] TC: 10%[c] [93,94]	OC: n/a EC: n/a TC: 0.4 $\mu g/m^3$ (1-h avg)[95]	n/a	Within 7 to 25% of filter OC and EC and within 15% for TC. Wide variation due to differentces in temperature and analysis protocols. [92,95,96]	80 to 89%6,95
LIGHT ABSORPTION INSTRUMENTS							
Aethalometer (AE-16, AE-21, AE-31) Attenuation of light transmitted through a quartz-fiber filter tape that continuously samples aerosol is measured and converted to a BC mass concentration using σ_{abs} of 14625/λ (m^2/g).	5 min	n/a	5 to 10%[d,7,97]	BC e: 0.1 $\mu g/m^3$ [90]	Subject to multiple scattering effects by particle and filter matrix resulting in absorption enhancement. Empirical corrections have been proposed 98 that can correct for such effects.	Within ± 25% of RU-OGI, Sunset and filter EC by TOR/TOT.[90-92]	75 to 90%6
Particle Soot Absorption Photometer (PSAP) Attenuation of light transmitted through a glass-fiber filter that continuously samples aerosol is measured to quantify light absorption (b_{abs}).	1 min	n/a	6 to 8%[99,100]	BC f: 0.1 $\mu g/m^3$ [90]	Instrument includes an empirical correction for scattering and loading effects 99 and adjustments have been proposed for the three wavelength model [100]	~50% lower than AE-16, RU-OGI and R&P-5400 EC.[90]	n/a
Multi-Angle Absorption Photometer (MAAP) Light transmittance at 0° and reflectance from a glass-fiber filter at 130° and 165° from the illumination direction are used in a radiative transfer model to estimate b_{abs} and is converted to BC using σ_{abs} of 6.6 m^2/g.	1 min	n/a	12%[g,101]	BC h: 0.05 $\mu g/m^3$ (or b_{abs} = 0.33 Mm-1 for 10-min avg) 0.02 $\mu g/m^3$ (or b_{abs} = 0.13 Mm-1 for 30-min avg)[101]	The instrument is designed to minimize multiple scattering and loading effects by measuring both transmittance and reflectance and using a two-stream approximation radiative transfer model to calculate b_{abs}.	Within 18% of filter EC by IMPROVE_TOR (R2 = 0.96) and up to 40% higher than Sunset EC. [102]	n/a

Instrument and Measurement Principle	Averaging Time	Analytical Accuracy[a]	Precision	Minimum Detectable Limit	Interferences	Comparability	Data Completeness
DRI Photoacoustic Analyzer (DRI-PA) Light absorption by particles in air results in a heating of the surrounding air. The expansion of the heated air produces an acoustic (sound wave) signal which is detected by a microphone to determine b_{abs}, which is converted to BC using σ_{abs}s = 5 m^2/g for the 1047 nm instrument and σ_{abs} = 10 m^2/g for the 532 nm instrument.	5 sec	n/a	n/a	BC i: 0.04 $\mu g/m^3$ (or b_{abs} = 0.4 Mm-1 for 10-min avg) at 532 nm[103]	At 532 nm, absorbance by NO_2 interferes with that by particles. Accounted by either removing NO_2 from sample line using denuders or by doing a periodic background (particle-free air) subtraction.	Good correlation (R2 > 0.80), but more than 40% lower than aethalometer, MAAP and filter IMPROVE_TOR EC. Suggests need for a different σ_{abs}.[102]	n/a
PHOTO-IONIZATION INSTRUMENTS							
Photoionization monitor for 91%[6T] polycyclic aromatic hydrocarbons (PAS-PAH) The air stream is exposed to UV radiation, which ionizes the particle-bound PAH molecules. The charged particles are collected on a filter element and the piezoelectric current is proportional to the particle-bound PAH.	5 min	n/a	n/a	~ 3 ng/m^3 [j,k]	n/a	n/a	> 91%6t

[a] Accuracy is the ability of analytical methods to quantify the observable of a standard reference material correctly; does not refer to measurement accuracy, since no standards are available.
[b] No specific details on how the precision was estimated; appears to be based on replicate analysis, may not represent overall co-located measurement precision
[c] Co-located precision estimates based on variation in avg ratios of replicate analysis using laboratory instrument and regression slopes (Slopes for OC = 1.01, EC = 0.82, TC = 0.94; R2 = 0.97 - 0.99) of co-located field measurements.
[d] Estimated using co-located AE-21 and AE-31 BC measurements at Fresno, CA.97
[e] While the default manufacturer recommended conversion factor (or mass absorption efficiency, σabs) is 16.6 m^2/g at 880 nm, Lim et al. (2003) assumed a value of 12.6 m^2/g.
[f] Assuming a σabs of 10 m^2/g.
[g] Co-located precision estimate based on the variability of the avg ratio (0.99 ± 0.12).
[h] Assuming a σabs of 6.5 m^2/g.
[i] Assuming a σabs of 10 m^2/g at 532 nm and 5 m^2/g at 1047 nm.
[j] Specified by manufacturer as "lower threshold"; needs to be calibrated with site-specific PAH. Typically used as a relative measure in terms of electrical output in femtoamps.
[k] Manufacturer specified measurement parameter
n/a: Not available.

Source: [1]Chow (1995, 077012); [2]Watson and Chow (2001, 157123); [3] Watson et al. (1983, 045084); [4]Fehsenfeld et al. (2004, 156432); [5]Solomon et al. (2001, 156993); [6]Mikel (2001, 156762); [7]Mikel (2001, 156762); [8]Watson et al. (1999, 020949); [9]Solomon and Sioutas (2006, 156995); [10]Graney et al. (2004, 053756); [11]Tanaka et al. (1998, 157041); [12]Pancras et al. (2005, 098120); [13]John et al. (1988, 045903); [14]Hering and Cass (1999, 084958); [15]Fritz et al. (1989, 077387); [16]Hering et al. (1988, 036012); [17]Solomon et al. (2003, 156994); [18]Cabada et al. (2004, 148859); [19]Fine et al. (2003, 155775); [20]Hogrefe et al. (2004, 099003); [21]Drewnick et al. (2003, 099160); [22]Watson et al. (2005, 157125); [23]Ho et al. (2006, 156552); [24]Decesari et al. (2005, 144536); [25] Mayol-Bracero et al. (2002, 045010); [26]Yang et al. 2003; [27] Turšic et al. (2006, 157063); [28]Mader et al.(2004, 156724); [29]Xiao and Liu (2004, 056801); [30]Kiss et al. (2002, 156646); [31]Cornell and Jickells (1999, 156367); [32] Zheng et al. (2002, 026100); [33]Fraser et al. (2002, 140741); [34] Fraser et al. (2003, 042231) [35]Schauer er al. (1998, 051162); [36]Fine et al. (2004); [37]Yue et al. (2004); [38]Rinehart et al. (2006, 115184); [39]Wan and Yu (2006, 157104); [40]Poore (2000, 012839); [41]Fraser et al. (2003a); [42]Engling et al. (2006, 156422); [43]Yu et al. (2005); [44]Tran et al. (2000); [45]Yao et al. (2004, 102213); [46]Li and Yu (2005, 156692); [47]Henning et al. (2003, 156539); [48]Zhang and Anastasio (2003, 157182); [49]Emmenegger et al. (2007, 156418); [50](Watson et al., 1989, 157119); [51]Greaves et al. (1985, 156494); [52]Waterman et al. (2000, 157116); [53]Waterman et al. (2001, 157117); [54]Falkovich and Rudich (2001, 156427); [55]Chow et al. (2007, 156354); [56]Miguel et al. (2004, 123260); [57]Crimmins and Baker (2006, 097008); [58]Ho and Yu (2004, 156551); [59]Jeon et al. (2001, 016636); [60]Mazzoleni et al. (2007, 098038); [61]Poore (2000, 012839); [62]Butler et al. (2003); [63]Chow et al. (2006c); [64]Russell et al. (2004, 082453); [65]Grover et al. (2006); [66]Grover et al. (2005, 090044); [67]Schwab et al. (2006b); [68]Hauck et al. (2004); [69]Jaques et al. (2004, 155878); [70]Rupprecht and Patashnick (2003, 157207); [71]Pang et al (2002, 030353); [72]Eatough et al. (2001, 010303); [73]Lee et al. (2005, 128139); [74]Lee et al. (2005, 156680); [75]Babich et al. (2000, 156239); [76]Lee et al. (2005c); [77]Lee et al. (2005, 128139); [78]Anderson and Ogren (1998, 156213); [79]Chung et al. (2001, 156357); [80]Kidwell and Ondov (2004, 155898); [81]Lithgow et al. (2004, 126616); [82]Weber et al. (2003, 157129); [83]Harrison et al. (2004); [84]Rattigan et al. (2006, 115897); [85]Wittig et al. (2004, 103413); [86]Vaughn et al. (2005, 157089); [87]Chow et al. (2005, 099030); [88]Weber et al (2001, 024640) ; [89]Schwab et al. (2006a); [90]Lim et al. (2003, 156697); [91]Watson and Chow (2002, 037873); [92]Venkatachari et al. (2006, 105918); [93]Bae et al. (2004a); [94]Arhami et al. (2006, 156224); [95]Park et al. (2005, 156843); [96]Bae et al. (2004b); [97]Chow et al. (2006a); [98]Arnott et al. (2005); [99]Bond et al. (1999); [100]Virkkula et al. (2005, 157097); [101]Petzold et al. (2002, 156863); [102]Park et al. (2006); [103]Arnott et al. (1999, 020650); [104]Peters et al. (2001); [105]Pitchford et al. (1997, 156872); [106]Rees et al. (2004, 097164); [107]Watson et al. (2000); [108]Lee et al.(2005, 156680); [109]Hering et al. (2004, 155837); [110]Watson et al. (1998); [111]Chakrabarti et al. (2004); [112]Mathai et al. (1990, 156741); [113]Kidwell and Ondov (2001, 017092); [114]Stanier et al. (2004); [115]Khlystov et al. (2005, 156635); [116]Takahama et al. (2004, 157038); [117]Chow et al. (2005, 156348); [118]Zhang et al. (2002); [119]Subramanian et al. (2004, 081203); [120]Chow et al. (2006b); [121]Birch and Cary (1996); [122]Birch (1998, 024953); [123]Birch and Cary (1996); [124]NIOSH (1996, 156810); [125]NIOSH (1999, 156811); [126]Chow et al. (1993); [127]Chow et al. (2007); [128]Ellis and Novakov (1982, 156416); [129]Peterson and Richards (2002, 156861); [130]Schauer et al. (2003); [131]Middlebrook et al. (2003, 042932); [132]Wenzel et al. (2003, 157139); [133]Jimenez et al. (2003); [134]Phares et al. (2003, 156866); [135]Qin and Prather (2006, 156895); [136]Zhang et al. (2005); [137]Bein et al. (2005, 156265); [138]Drewnick et al. (2004a); [139]Drewnick et al. (2004b); [140]Lake et al. (2003, 156669); [141]Lake et al. (2004, 088411)

Table A-11. Summary of mass measurement comparisons.

Site / Period / Sampler / Configuration	Summary of Findings
1. Birmingham, AL (11/04/96 To 11/23/96) 2. Denver-Adams City, CO (12/11/96 To 1/7/97) 3. Bakersfield, CA (1/21/97 To 3/19/97) 4. Denver-Welby, Co (12/12/96 To 12/21/96) 5. Phoenix, AZ (12/06/96 To 12/21/96) 6. Azusa, CA (3/25/97 To 5/19/97) 7. Research Triangle Park (RTP), NC (1/17/97 To 8/14/97) 8. Rubidoux, CA (1/6/99 To 2/26/99) 9. Atlanta, GA (8/3/99 To 8/31/99)	**Peters et al., (2001, 017108)[104]: Pitchford et al.,** (1997, 156872)[105] **dataset** Co-located precision (CV) for the RAAS2.5-100 samplers ranged from 1.5% at Bakersfield to 6.2% at Birmingham. In Birmingham, CV for two co-located Harvard Impactor was 1% and for three Dichots was 6.2%. The IMPROVE samplers had greater variability, with a CV of 11.3% (Denver-Adam City) and 10.8% (Bakersfield). Partisol and RAAS showed the strongest pairwise comparison (slope = 1.0 ± 0.06, intercept = 0.26 ± 1.81, and correlation = 1.0), within the EPA equivalency criteria. Strong relationships (correlation > 0.96; slope = 0.9 – 1.12, intercept < 3σ) were observed for other samplers in reference to the RAAS. At Denver-Welby, 6 RAAS samplers were deployed (3 with and 3 without temperature compensation for flow control). The units with temperature compensation had a positive bias relative to the non-temperature compensated units. Non-FRM samplers did not meet the EPA equivalency criteria, despite strong linear relationships with the FRM sampler. **Peters et al.[104]: RTP 97 dataset** CV was 1.7%, 2.3%, 3.4%, 6.4% for the PQ200, Partisol 2000, RAAS2.5-100, and Dichot, respectively. Dichot flows were valve controlled and set visually by the operator using rotameters. Good one-to-one correspondence was observed for FRM comparisons. The FRM averages were within -1.2% to 3.2%, within the acceptable ± 10% range Peters et al. 104: Rubidoux 99 and Atlanta 99 dataset In Rubidoux, the precision for PQ200 was 6.1%, higher than at RTP 97. In Atlanta, the grouped data from PQ200, RAAS2.5-300, and Partisol yielded a precision of 1.7%. Linear regression results met the EPA equivalency criteria for all FRMs.

SAMPLER	FLOW RATE (L/MIN)	FILTER TYPE[A]	DENUDER[B]
RAAS2.5-100 $PM_{2.5}$ FRM	16.7	Teflon (n/a)	None
RAAS2.5-300 $PM_{2.5}$ FRM	16.7	Teflon (n/a)	None
RAAS2.5-200 $PM_{2.5}$ FRM	16.7	Teflon (n/a)	None
R&P Partisol 2000 $PM_{2.5}$ FRM	16.7	Teflon (n/a)	None
R&P Partisol-plus 2025 $PM_{2.5}$ FRM	16.7	Teflon (n/a)	None
BGI PQ200 $PM_{2.5}$ FRM	16.7	Teflon (n/a)	None
Sierra Instruments SA-244 Dichot	16.7	Teflon (n/a)	None
IMPROVE $PM_{2.5}$	22.8	Teflon (n/a)	None
Harvard $PM_{2.5}$ Impactor	10	Teflon (n/a)	None
Airmetrics battery powered $PM_{2.5}$ MiniVol	5	Teflon (n/a)	None

ATLANTA SUPERSITE, GA: 8/3/99 TO 9/1/99 4 km NW of downtown, within 200 m of a bus maintenance yard and several warehouse facilities, representative of a mixed commercial-residential neighborhood.				**Solomon et al.** [17] $PM_{2.5}$ mass from individual samplers was compared to all-sampler avgs, called the filter relative reference (filter RR) value. Overall agreements were within ± 20% of filter RR. FRM samplers were within 3.5% of filter RR. Avg mass measured by RAAS-400, SASS and URG-PCM were within ± 10% of filter RR. Avg mass measured by MASS-400, R&P-2300 and R&P-2025 dichot were greater than filter RR but within ± 20%. Avg mass measured by PC-BOSS (BYU) and ARA-PCM were lower than filter RR within ± 10%. All samplers except PC-BOSS (TVA) had R2 > 0.80, relative to filter RR. While avg mass for each sampler was within 20%, daily variability was > 50% of filter RR. Glycerol in the Na_2CO_3 denuder may have contaminated the filter in the MASS-400 sampler resulting in higher $PM_{2.5}$ values. PC-BOSS samplers removed particles < 0.1 μm aerodynamic diameter from $PM_{2.5}$ measurements. Corrections were made using sulfate (SO_4^{2-}) concentrations in the major flow or immediately after the $PM_{2.5}$ inlet, but before the flow split-up. This was insufficient to bring PC-BOSS mass close to filter RR. PC-BOSS was also equipped with upstream denuders ahead of
Sampler	**Flow Rate (L/Min)**	**Filter Type[a]**	**Denuder[b]**	
R&P-2000 FRM	16.7	Teflon (P)	None	
RAAS-100 FRM	16.7	Teflon (P)	None	
RAAS-400	24	Teflon (P)	None	
SASS	6.7	Teflon (P)	None	
MASS-400	16.7	Teflon (P)	Na_2CO_3	
R&P-2300	10	Teflon (P)	None	
R&P-2025 Dichot:				
$PM_{2.5}$	15	Teflon (P)	None	
$PM_{10-2.5}$	1.67	Polycarbonate	None Na_2CO_3/Citric	

Site / Period / Sampler / Configuration					Summary of Findings
URG-PCM	16.7	Teflon (P)		Acid	the filters, which may have enhanced loss of semi-volatile components, resulting in a lower mass on the filter.
ARA-PCM	16.7	Teflon (n/a)		Na_2CO_3/Citric acid	**Butler et al.** [62]
PC-BOSS (operated by TVA)	105	Teflon (W)		CIF	The sum of individual species accounted for ~78% of the RAAS-100 FRM $PM_{2.5}$ mass concentration.
PC-BOSS (operated by BYU)	150	Teflon (W)		CIF	TEOM explained ~82 to 92% of the species sum of RAAS with R2 = 0.86.
$PM_{2.5}$ CONTINUOUS SAMPLER	**FLOW RATE (L/MIN)**	**INLET TEMPERATURE**	**DRYER**	**OTHER**	
TEOM	16.7	30 °C	Nafion	$PM_{2.5}$	
ATLANTA SUPERSITE, GA: 11/21/01 TO 12/23/01					**Lee et al.** [73]
$PM_{2.5}$ SAMPLER	**FLOW RATE (L/MIN)**	**FILTER TYPE[a]**		**DENUDER[b]**	RAMS $PM_{2.5}$ adjusted using particle concentrator efficiency of 0.5.
R&P-2025 FRM	16.7	Teflon (n/a)		None	Good correlation between SES-TEOM and Radiance Research M903s (R2 = 0.80), while medium correlation was found between CAMM and Radiance Research M903 (R2 = 0.64) or RAMS and Radiance Research M903 (R2 = 0.63).
					CAMM = (0.75 ± 0.03) SES-TEOM + (2.51 ± 0.51); R2 = 0.78; N = 196
$PM_{2.5}$ CONTINUOUS SAMPLER	**FLOW RATE (L/MIN)**	**INLET TEMPERATURE**	**DRYER**	**OTHER**	RAMS = (0.85 ± 0.06) SES-TEOM + (5.34 ± 1.04); R2 = 0.52; N = 96
TEOM	16.7	30 °C	Nafion	$PM_{2.5}$	RAMS = (0.91 ± 0.07) CAMM + (5.71 ± 1.20); R2 = 0.43; N = 196
SES-TEOM	16.7	30 °C	Nafion	$PM_{2.5}$	Semi-volatile material explains the difference between RAMS and SES TEOM.
CAMM	0.3	n/a	Nafion	$PM_{2.5}$	CAMM = (0.75 ± 0.08) R&P-2025 FRM + (2.47 ± 1.02); R2 = 0.76; N = 31
RAMS	16.7	30 °C	Nafion	$PM_{2.5}$ TEA & CIF denuders With particle concentrator	RAMS = (0.97 ± 0.22) R&P-2025 FRM + (2.39 ± 3.42); R2 = 0.64; N = 13
Radiance Research M903	n/a	n/a	Nafion	bscat	SES-TEOM = (1.07 ± 0.05) R&P-2025 FRM + (-1.34 ± 0.71); R2 = 0.95; N = 26
Radiance Research M903	n/a	n/a	None	bscat	CAMM vs. FRM yielded lower slopes (0.75) with high intercepts.
PITTSBURGH SUPERSITE, PA: 7/1/01 to 6/1/02 6 km east of downtown in a park on the top of a hill					**Cabada et al; [18]: Rees et al.** [106]
SAMPLER	**FLOW RATE (L/MIN)**	**FILTER TYPEa**		**DENUDER**	MOUDI PM_{10} = 0.80 Dichot PM_{10}, R2 = 0.85
MOUDI-110	30	Teflon (P,d)		None	MOUDI $PM_{2.5}$ = 1.03 Dichot $PM_{2.5}$, R2 = 0.78
And-241 Dichot	16.7	Teflon (P		None	MOUDI $PM_{2.5}$ = 1.01 FRM $PM_{2.5}$, R2 = 0.78
R&P-2000 $PM_{2.5}$ FRM	16.7	Teflon (W)		None	Dichot $PM_{2.5}$ = 0.97 FRM $PM_{2.5}$ + 0.02; R2 = 0.94
					Good agreement for $PM_{2.5}$ FRM, Dichot, and MOUDI. Lower slope for PM_{10} suggests loss of coarse particles in the MOUDI sampler.
$PM_{2.5}$ CONTINUOUS SAMPLER	**FLOW RATE (L/MIN)**	**INLET TEMPERATURE**	**DRYER**	**OTHER**	Ultrafine (< 100 nm) mass ($PM_{0.10}$) measurements had high uncertainties (~30%)
SES-TEOM	16.7	30 °C	Nafion	$PM_{2.5}$	Ultrafine mass by MOUDI showed no correlation with ultrafine volume (V0.10) by DAASS. Ratio of $PM_{0.10}/PM_{2.5}$ mass ratio showed reasonable agreement with volume ratio (V0.10/V2.5, R2 = 0.55, slope = 0.76). Bounce of large particles to smaller stages in MOUDI was small, since mass ratio ($PM_{0.10}/PM_{2.5}$) did not exceed volume ratio (V0.10/V2.5). Low correlation between ultrafine mass and volume could be due to the ultrafine mass measurement uncertainty or due to fundamental differences in the measurement methods employed by MOUDI and DAASS. Ambient conditions and characteristics of the aerosols (such as non-spherical shapes of fresh particles) could also influence these estimates.
DAASS	n/a	30 °C	Nafion or None	$PM_{2.5}$	

Site / Period / Sampler / Configuration	Summary of Findings
	Rees et al. [106] SES-TEOM $PM_{2.5}$ = 1.02 FRM $PM_{2.5}$ + 0.65; R2 = 0.95 Volatilization did not affect SES-TEOM performance when $PM_{2.5}$ mass > 20-30 $\mu g/m^3$. When ambient temperature was < -6 °C, and when mass was low, SES-TEOM was lower (up to 50%) than FRM or Dichot.

FRESNO SUPERSITE, CA and other CRPAQS sites; 12/2/99 to 2/3/01. Some comparisons included data till 12/29/03 . Fresno Supersite was located 5.5 km northeast of downtown in a mixed residential-commercial neighborhood. 107

SAMPLER	FLOW RATE (L/MIN)	FILTER TYPEa	DENUDER
RAAS-100 $PM_{2.5}$ FRM	16.7	Teflon (P)	None
RAAS-300 $PM_{2.5}$ FRM	16.7	Teflon (P)	None
R&P-2000 $PM_{2.5}$ FRM	16.7	Teflon (P)	None
R&P-2025 $PM_{2.5}$ FRM	16.7	Teflon (P)	None
RAAS-400 $PM_{2.5}$	24	Teflon (P)	None
SASS $PM_{2.5}$	6.7	Teflon (P)	None
And-246 Dichot			
$PM_{2.5}$	15	Teflon (P)	None
$PM_{10-2.5}$	1.67	Teflon (P)	None
DRI-SFS $PM_{2.5}$	113	Teflon (P)	None
MiniVol $PM_{2.5}$	5	Teflon (P)	None
MOUDI-100	30	FEPb Teflon (P)	None
And-hIVOL PM_{10} FRM	1130	Teflon (P)	None

Chow et al. [63]

$PM_{2.5}$ measurements from the 11 filter samplers were within ~20% of each other, except for MiniVols, which were 20 to 30% lower than RAAS-300 FRM.

All the FRM samplers were within ± 10% of each other.

All the filter samplers were well correlated with each other (R2 > 0.90).e

DRI-SFS (with HNO_3 denuder) and And-246 Dichot $PM_{2.5}$ were lower (~5% and 7%, respectively, on avg) than FRM, possibly due to nitrate (NO_{3-}) volatilization.

Poor correlation (R2) found between TEOM $PM_{2.5}$ concentrations and RAAS-100 FRM. TEOM $PM_{2.5}$ was lower than RAAS-100 FRM by 22%. Heating of TEOM inlet to 50 °C resulted in loss of semi-volatile components such as ammonium nitrate (NH_4NO3) and possibly some semi-volatile organic compounds.

TEOM PM_{10} concentrations were 28% lower than the And-hIVOL10 FRM on avg, ranging from 13% in summer to 43% in winter.

TEOM was neither equivalente nor comparablee to the FRM sampler for $PM_{2.5}$ or PM_{10}.

BAM $PM_{2.5}$ concentrations showed high correlation (R2 > 0.90) with the RAAS-100 and RAAS-300 FRM samplers, with slopes ranging from 0.92 to 0.97. BAM $PM_{2.5}$ was typically higher than FRM (17 to 30%) except at Bakersfield, CA, where it was 21% lower, suggesting a BAM calibration difference between Bakersfield and other sites.

BAM PM_{10} concentrations were 26% higher than And-hIVOL PM_{10} FRM concentration on avg (R2 > 0.92).

Higher BAM measurements were attributed to water absorption by hygroscopic particles. BAM $PM_{2.5}$ and PM_{10} deviations were larger for concentrations < 25 $\mu g/m^3$.

Grover et al. [65]

PC-BOSS $PM_{2.5}$ = (0.88 ± 0.04) FDMS-TEOM + (6.7 ± 4.3); R2 = 95; n = 29

PC-BOSS $PM_{2.5}$ = (1.11 ± 0.07) D-TEOM + (7.5 ± 6.1); R2 = 0.90; n = 29

TEOM50C $PM_{2.5}$ = (0.80 ± 0.01) TEOm30C + (1.1 ± 3.1); R2 = 0.91; n = 507

TEOm30C $PM_{2.5}$ = (0.50 ± 0.01) FDMS-TEOM -(1.7 ± 6.9); R2 = 0.68; n = 516

Heated GRIMM PM concentrations were lower than FDMS-TEOM and ambient temperature GRIMM, suggesting loss of semi-volatile matter.

Data recovery was greater than 95% for all continuous instruments, except for D-TEOM, which had 86% recovery.

Reasonable agreement was seen between FDMS-TEOM, D-TEOM, BAM, and GRIMM $PM_{2.5}$ when semi-volatile matter was dominated by NH_4NO3. However, the FDMS-TEOM was higher than the other instruments during high concentration periods, associated with days with a high fraction of semi-volatile organic compounds (SVOCs). Possible differences in SVOCs may have contributed to the differences between FDMS and other instruments.

CONTINUOUS SAMPLER	FLOW RATE (L/MIN)	INLET TEMPERATURE	DRYER	OTHER
TEOM	16.7	50 °C	None	$PM_{2.5}$ and PM_{10}
BAM	16.7	Ambient	None	$PM_{2.5}$ and PM_{10}

Site / Period / Sampler / Configuration					Summary of Findings
SAMPLER	**FLOW RATE (L/MIN)**	**FILTER TYPEa**		**DENUDERb**	
PC-BOSS $PM_{2.5}$	150	Teflon (W)		CIF	
CONTINUOUS SAMPLER	**FLOW RATE (L/MIN)**	**INLET TEMPERATURE**	**DRYER**	**OTHER**	
TEOM	16.7	50 °C	None	$PM_{2.5}$	
TEOM	16.7	30 °C	None	$PM_{2.5}$	
FDMSTEOM	16.7	30 °C	Nafion	$PM_{2.5}$	
D-TEOM	16.7	30 °C	Nafion	$PM_{2.5}$	
GRIMM1100	1.2	Ambient	None	bscat	
GRIMM1100	1.2	80 °C heater, resulting in aerosol temperature	Heater	bscat	
BAM	16.7	Ambient	None	$PM_{2.5}$	
HOUSTON SUPERSITE, TX; 1/1/00 to 2/28/02 The Houston Supersite included three sites located in southeast Texas including one on the grounds of a municipal airport at the edge of a small community, one adjacent to the highly industrial ship channel and one on the grounds of a middle school in a suburban community.					**Russell et al. [64]; Lee et al. [108]** Good correlations between 24-h SES-TEOM $PM_{2.5}$ and R&P-2025 FRM mass. CAMM = (0.93 ± 0.03) RAMS + (3.14 ± 0.74); R2 = 0.81 SES-TEOM = (0.92 ± 0.03) RAMS + (1.52 ± 0.77); R2 = 0.80 SES-TEOM = (1.01 ± 0.03) CAMM + (-1.91 ± 0.79); R2 = 0.83 Correlation of Radiance Research M903 and SES-TEOM was good (R2 = 0.95), while that of Radiance Research M903 with CAMM or RAMS was poor (R2 ~ 0.4). RAMS > SES-TEOM at high temperature and low RH (< 60%), suggesting loss of water and particulate NO_3^- from SES-TEOM. CAMM = (1.02 ± 0.08) R&P-2025 + (1.62 ± 1.35); R2 = 0.89 RAMS = (1.10 ± 0.08) R&P-2025 + (0.68 ± 1.28); R2 = 0.89 SES-TEOM = (1.09 ± 0.07) R&P-2025 + (0.21 ± 1.27); R2 = 0.94 Integrated mass < Continuous $PM_{2.5}$ mass. Difference possibly related to loss of SVOCs and NO_3^- from integrated sampler
$PM_{2.5}$ SAMPLER	**FLOW RATE (L/MIN)**	**FILTER TYPE[a]**		**DENUDER**	
R&P-2025 FRM	16.7	Teflon (n/a)		None	
CONTINUOUS SAMPLER	**FLOW RATE (L/MIN)**	**INLET TEMPERATURE**	**DRYER**	**OTHER[b]**	
TEOM	16.7	50 °C	None	$PM_{2.5}$	
SES-TEOM	16.7	30 °C	Nafion	$PM_{2.5}$ Aug-Sep '00	
CAMM	0.3	Ambient	Nafion	$PM_{2.5}$ Aug-Sep '00	
RAMS	16.7	30 °C	Nafion	$PM_{2.5}$ TEA & CIF denuders; Aug-Sep '00	
Radiance Research M903	n/a	n/a	Nafion	Bscat Aug-Sep '00	
LOS ANGELES SUPERSITE, CA; 9/01 to 8/02 The Los Angeles Supersite consisted of multiple sampling locations in the South Coast Air Basin to provide wide geographical and seasonal coverage, including urban "source" sites and downwind "receptor" sites.					**Jaques et al. [69]; Hering et al. [109]** Dichot $PM_{2.5}$ = 0.83 MOUDI + 1.23; R2 = 0.83 (n = 37) Dichot $PM_{2.5}$ showed higher NO_3^- loss than MOUDI, consistent with anodized aluminum surfaces serving as efficient denuders that remove volatilized NO_3^-.2,110. D-TEOM $PM_{2.5}$ = 1.18 MOUDI – 1.28; R2 = 0.86 (n = 20) Over-estimation of D-TEOM may be due to particle losses in the MOUDI. $PM_{2.5}$ by D-TEOM during ESP-off phase (net artifact effect) tracked well with the NO_3^- concentrations. NO_3^- vaporization from the TEOM was caused by the temperature of the TEOM filter (~30 – 50 °C) rather
SAMPLER	**FLOW RATE (L/MIN)**	**FILTER TYPE[a]**		**DENUDER[b]**	
R&P-2025 Dichot					
$PM_{2.5}$	15	Teflon (P)		None	
$PM_{10-2.5}$	16.7	n/a		None	
MOUDI-110	30	Teflon (P		None	
HEADS $PM_{2.5}$	10	Teflon (n/a)		$NaHCO_3$	

Site / Period / Sampler / Configuration					Summary of Findings
CONTINUOUS SAMPLER	**FLOW RATE (L/MIN)**	**INLET TEMPERATURE**	**DRYER**	**OTHER**	than the pressure drop across the filter. Vaporization from the TEOM had a time constant between 10 to 100 min, depending on ambient and TEOM filter temperatures; the vapor pressure, and the extent of vapor saturation upstream and downstream of the TEOM filter. The mass measured during 5 min periods (ESP-on and off cycle in D-TEOM) provides an estimate of the dynamic vaporization losses.
D-TEOM	16.7	30 °C	Nafion	$PM_{2.5}$	
Nano-BAM (BAM-1020 with d50 148 ± 10 nm inlet)	16.7	Ambient	None	~150 nm cut-point at 16.7 L/min	
SMPS-3936	0.3	Ambient	None	Number to mass assuming spherical particles of 1.6 g/cc density	**Chakrabarti et al.** [111] Good agreement between MOUDI PM0.15 and Nano-BAM PM0.15 (MOUDI PM0.15 = 0.97 Nano-BAM PM0.15 + 0.60; R2 = 0.92; n = 24) Nano-BAM captured peak PM0.15 concentrations not quantified by SMPS. Potential particle agglomeration (with resulting high surface areas) caused SMPS to include particles in the accumulation- rather than ultrafine-mode, since mobility diameter is a function of surface area.
RUBIDOUX, CA; 08/15/01 to 09/07/01, 07/01/03 to 07/31/03. Rubidoux is located in the eastern section of the South Coast Air Basin (SoCAB) in the north-west corner of Riverside County, 78 km downwind of the central Los Angeles metropolitan area and in the middle of the remaining agricultural production area in SoCAB.					**Grover et al.** [66] **(2003 measurements):** D-TEOM = (0.98 ± 0.02) FDMS-TEOM + (-0.6 ± 5.3); R2=0.85; n = 426; excludes 38 data points when FDMS-TEOM $PM_{2.5}$ was higher than DTEOM $PM_{2.5}$ by ~21 $\mu g/m^3$. RAMS = (0.93 ± 0.02) FDMS-TEOM + (2.4 ± 8.2); R2 = 0.81; n = 337 FDMS-TEOM = (0.96 ± 0.06) PC-BOSSconstructed mass + (-0.3 ± 3.9); R2 = 0.90; n = 33 R&P-2025 FRM = (0.96 ± 0.06) FDMS-TEOM + (-9.3 ± 3.9); R2 = 0.90; n = 29 The R&P-2025 FRM $PM_{2.5}$ was, on avg, ~32% lower than FDMSTEOM. Losses of NH_4NO3 and organics can account for the difference. TEOM @ 50 °C $PM_{2.5}$ was consistently lower than FDMS-TEOM, DTEOM or RAMS and was, on avg, ~ 50% lower than FDMS-TEOM. This difference is due to loss of semi-volatile NO_3- and organics from the heated TEOM. FDMS-TEOM and D-TEOM needed little attention from site operators. **Lee et al.** [76] **(2001 measurements)** D-TEOM $PM_{2.5}$ and Radiance Research M903s light
SAMPLER	**FLOW RATE (L/MIN)**	**FILTER TYPE**[a]		**DENUDER**[b]	
PC-BOSS $PM_{2.5}$	150	Teflon (W)		CIF	
R&P-2025 $PM_{2.5}$ FRM	16.7	Teflon (n/a)		None	
CONTINUOUS SAMPLER	**FLOW RATE (L/MIN)**	**INLET TEMPERATURE**	**DRYER**	**OTHER**	
TEOM	16.7	50 °C	None	$PM_{2.5}$	
FDMS-TEOM	16.7	30 °C	Nafion	$PM_{2.5}$	
D-TEOM	16.7	30 °C	Nafion	$PM_{2.5}$	
RAMS	16.7	30 °C	Nafion	$PM_{2.5}$ Denuders used	
CAMM	0.3	n/a	None	$PM_{2.5}$	
Radiance Research M903	n/a	n/a	Nafion	bscat	
Radiance Research M903	n/a	n/a	None	bscat	

Site / Period / Sampler / Configuration	Summary of Findings
	scattering (with and without dryers) showed good correlation. D-TEOM = (3.69 ± 0.09) Radiance Research M903no-dryer + (2.74 ± 0.89); R2 = 0.84; n = 299 D-TEOM = (3.79 ± 0.10) Radiance Research M903dryed + (4.08 ± 0.84); R2 = 0.83; n = 312 Radiance Research M903no-dryer = (1.03 ± 0.01) Radiance Research M903dryed + (0.34 ± 0.05); R2 = 0.98; n = 513; absorbed water did not affect relationship to $PM_{2.5}$. CAMM and RAMS compared poorly (R2 = 0 to 0.25) with D-TEOM, Radiance Research M903s and among themselves. RAMS correlated well with D-TEOM for $PM_{2.5}$ $> 30\ \mu g/m^3$ due to RAMS's efficient particle collection of larger particle sizes (historically associated with high mass loadings at this site) in the $PM_{2.5}$ size range. D-TEOM $PM_{2.5}$ correlated well with ADI-N sized NO_3 (R2 = 0.62) and OC by Sunset OCEC (R2 = 0.61) suggesting that D-TEOM measured $PM_{2.5}$ mass with minimum loss of SVOCs. RAMS showed R2 of 0.20 (NO_3-) to 0.30 (OC), while CAMM showed no correlation.

LINDON, UT; 01/29/03 to 02/12/03

SAMPLER	FLOW RATE (L/MIN)	FILTER TYPE[a]	DENUDER[b]
PC-BOSS $PM_{2.5}$	150	Teflon (W)	CIF

CONTINUOUS SAMPLER	FLOW RATE (L/MIN)	INLET TEMPERATURE	DRYER	OTHER
TEOM	16.7	30 °C	None	$PM_{2.5}$
FDMS-TEOM	16.7	30 °C	Nafion	$PM_{2.5}$
RAMS	16.7	30 °C	Nafion	$PM_{2.5}$ Denuder used

Grover et al. [66]

RAMS required regular maintenance.

RAMS = (0.92 ± 0.03) FDMS-TEOM + (1.3 ± 3.9); R2 = 0.69; n = 332

PC-BOSS constructed mass = (0.89 ± 0.21) FDMS-TEOM + (1.8 ± 2.8); R2 = 0.66; n = 11

TEOM @ 30 °C $PM_{2.5}$ was consistently lower than FDMS-TEOM and the difference was consistent with concentrations SVOCs and NH_4NO3 measured by PC-BOSS.

PHILADELPHIA, PA; 07/02/01 to 08/01/01 At water treatment center in a grassy field surrounded by mixed deciduous and pine trees on three sides and a river on the other. Within 0.5 km of Interstate I-95 and within 30 km from downtown Philadelphia.

SAMPLER	FLOW RATE (L/MIN)	FILTER TYPE[a]	DENUDER[b]
Harvard Impactor $PM_{2.5}$	10	Teflon (n/a)	n/a

CONTINUOUS SAMPLER	FLOW RATE (L/MIN)	INLET TEMPERATURE	DRYER	OTHER
SES-TEOM	16.7	35 °C	Nafion	$PM_{2.5}$
CAMM	0.3	n/a	Nafion	$PM_{2.5}$
RAMS	16.7	30 °C	Nafion	$PM_{2.5}$ TEA & CIF denuders With particle concentrator
Radiance Research M903	n/a	n/a	Nafion	bscat
Radiance Research M903	n/a	n/a	None	bscat

Lee et al. [73]

Radiance Research M903dryer = (0.78 ± 0.01) Radiance Research M903no dryer + (0.30 ± 0.03); R2 = 0.95

Radiance Research M903s vs. CAMM, R2 = 0.78

Radiance Research M903s vs. RAMS, R2 = 0.63

Radiance Research M903s vs. SES-TEOM, R2 = 0.72

CAMM = (0.60 ± 0.03) SES-TEOM + (2.0 ± 0.42); R2 = 0.71; N = 185

RAMS = (0.71 ± 0.04) SES-TEOM + (2.51 ± 0.59); R2 = 0.63; N = 185

RAMS = (0.93 ± 0.06) CAMM + (2.44 ± 0.68); R2 = 0.55; N = 185

Both RAMS and CAMM under-measured ambient $PM_{2.5}$.

CAMM = (0.70 ± 0.06) HI + (0.16 ± 0.96); R2 = 0.87; N = 22

SES-TEOM = (1.0 ± 0.10) HI + (-0.68 ± 1.74); R2 = 0.89; N = 15

BALTIMORE SUPERSITE, MD; 05/17/01 to 06/11/01. Located near a freeway and bus yard.

SAMPLER	FLOW RATE (L/MIN)	FILTER TYPE	DENUDER

Lee et al. [73]

Radiance Research M903dryed = (0.65 ± 0.02) Radiance

Site / Period / Sampler / Configuration					Summary of Findings
RAAS-100 $PM_{2.5}$ FRM	16.7	Teflon		None	Research M903no dryer + (1.80 ± 0.20); R2 = 0.75, suggesting influence from particle-bound water.
					High correlation (R2 = 0.75) between Radiance Research M903s.
CONTINUOUS SAMPLER	**FLOW RATE (L/MIN)**	**INLET TEMPERATURE**	**DRYER**	**OTHER**	Poor correlation among the continuous instruments.
SES-TEOM	16.7	35 °C	Nafion	$PM_{2.5}$	Radiance Research M903s did not follow $PM_{2.5}$ concentrations measured by other continuous instruments.
CAMM	0.3	n/a	Nafion	$PM_{2.5}$	
RAMS	16.7	30 °C	Nafion	$PM_{2.5}$ TEA & CIF denuders; No particle	CAMM = (0.32 ± 0.07) SES-TEOM + (9.45 ± 1.61); R2 = 0.14; N = 120 RAMS = (0.82 ± 0.10) SES-TEOM + (6.41 ± 2.09); R2 = 0.38; N = 120
Radiance Research M903	n/a	n/a	Nafion	bscat	RAMS = (0.71 ± 0.12) CAMM + (11.3 ± 2.23); R2 = 0.21; N = 120
Radiance Research M903	n/a	n/a	None	bscat	CAMM = (0.80 ± 0.29) RAAS-100 FRM + (-0.83 ± 5.85); R2 = 0.60; N = 7
					RAMS = (1.05 ± 0.12) RAAS-100 FRM + (4.80 ± 2.60); R2 = 0.90; N = 11
					SES-TEOM = (0.86 ± 0.10) RAAS-100 FRM + (2.96 ± 1.99); R2 = 0.90; N = 10
SEATTLE, WA; 01/28/01 to 02/21/01 Urban area near major highway and interstate, 8 km southeast of downtown.					**Lee et al.** [108]
					Radiance Research M903dryed = 0.94 ± 0.00 Radiance Research M903no dryer; R2 = 1.0.
SAMPLER	**FLOW RATE (L/MIN)**	**FILTER TYPE**[a]		**DENUDER**[b]	Correlation of Radiance Research M903 vs. SES-TEOM, R2 = 0.80, while that of Radiance Research M903 with CAMM was R2 = 0.84 and with RAMS was R2 = 0.72.
MASS $PM_{2.5}$	16.7	Teflon (n/a)		Na_2CO_3 denuder	CAMM = (1.07 ± 0.05) RAMS + (1.03 ± 0.55); R2 = 0.61
					SES-TEOM = (0.95 ± 0.03) RAMS + (1.24 ± 0.38); R2 = 0.72
CONTINUOUS SAMPLER	**FLOW RATE (L/MIN)**	**INLET TEMPERATURE**	**DRYER**	**OTHER**	SES-TEOM = (0.87 ± 0.03) CAMM + (0.55 ± 0.37); R2 = 0.74
SES-TEOM	16.7	30 °C	Nafion	$PM_{2.5}$	SES-TEOM likely lost semi-volatile organic matter.
CAMM	0.3	Ambient	Nafion	$PM_{2.5}$	Continuous $PM_{2.5}$ samplers were similar to filter $PM_{2.5}$ sampler. Number of samples was small (~ 7).
RAMS	16.7	30 °C	Nafion	$PM_{2.5}$ TEA & CIF denuders	Some SES-TEOM mass values were less than MASS filter values suggesting that loss of mass is likely for a SES-TEOM at 30 °C, particularly during the cold season.
Radiance Research M903	n/a	n/a	Nafion	bscat	
Radiance Research M903	n/a	n/a	None	bscat	
NEW YORK SUPERSITE, NY; 01/01/03 to 12/31/04 Urban site located at Queens College, NY, about 14 km west of Manhattan, within 2 km of freeways, and within 12 km of international airports. A rural site was located at Pinnacle State Park surrounded by golf course, picnic areas, undeveloped forest lands, and no major cities within 15 km.					**Schwab et al.** [67]
					FDMS-TEOM had operational difficulties resulting in low data capture (65% at urban site and 57% at rural site).
					BAM had data captures greater than 95% at both sites.
SAMPLER	**FLOW RATE (L/MIN)**	**FILTER TYPE**[a]		**DENUDER**[b]	Urban site:
R&P-2025 $PM_{2.5}$ FRM	16.7	Teflon (n/a)		None	BAM = (1.02 ± 0.02) FDMS-TEOM + 1.72; R2 = 0.93; n = 244
R&P-2300 $PM_{2.5}$	16.7	Teflon (n/a)		None	FDMS-TEOM = (1.25 ± 0.02) FRM – (0.63 ± 0.26); R2 = 0.95; n = 238
					BAM = (1.28 ± 0.03) FRM + (1.27 ± 0.38); R2 = 0.88; n = 320
CONTINUOUS SAMPLER	**FLOW RATE (L/MIN)**	**INLET TEMPERATURE**	**DRYER**	**OTHER**	Rural site:
TEOM	16.7	50 °C	None	$PM_{2.5}$	FDMS-TEOM = (1.09 ± 0.02) FRM – (0.004 ± 0.18); R2 = 0.95; n = 349
FDMS-TEOM	16.7	30 °C	Nafion	$PM_{2.5}$	
BAM	16.7	"smart" heater on @ RH > 44%		$PM_{2.5}$	$PM_{2.5}$ FDMS-TEOM > FRM > TEOM50°C, suggesting that FRM captured a fraction, but not all, of the volatile components. TEOM50°C volatilizes $PM_{2.5}$, particularly during winter.

Site / Period / Sampler / Configuration	Summary of Findings

[a]Filter Manufacturer in parentheses - W: Whatman, Clifton, NJ; P: Pall-Gelman, Ann Arbor, MI; S: Schleicher & Schnell. Keene, NH; n/a: not available or not reported.
[b]Na_2CO_3: Sodium carbonate; NaHCO3: Sodium bicarbonate CIF: Charcoal Impregnated Filter; FEP: Fluorinated Ethylene Propylene copolymer; TEA: Triethanolamine; TSP: Total Suspended PM.
[c]37 mm filter.
[d]37-mm after-filter for stages smaller than 0.16 μm and 47-mm for higher stages.
[e]Equivalence requires correlation coefficient (r) ≥ 0.97, linear regression slope 1.0 ± 0.05 and an intercept 0 ± 1 $\mu g/m^3$; Comparability requires r > 0.9 and linear regression slope equal 1 within 3 standard errors and intercept equal zero within 3 standard errors; Predictability requires r > 0.9. 91, 112

Source: [1]Chow (1995, 077012); [2]Watson and Chow (2001, 157123); [3] Watson et al. (1983, 045084); [4]Fehsenfeld et al. (2004, 156432); [5]Solomon et al. (2001, 156993); [6]Mikel (2001, 156762); [7]Mikel (2001, 156762); [8]Watson et al. (1999, 020949); [9]Solomon and Sioutas (2006, 156995); [10]Graney et al. (2004, 053756); [11]Tanaka et al. (1998, 157041); [12]Pancras et al. (2005, 098120); [13]John et al. (1988, 045903); [14]Hering and Cass (1999, 084958); [15]Fritz et al. (1989, 077387); [16]Hering et al. (1988, 036012); [17]Solomon et al. (2003, 156994); [18]Cabada et al. (2004, 148859); [19]Fine et al. (2003, 155775); [20]Hogrefe et al. (2004, 099003); [21]Drewnick et al. (2003, 099160); [22]Watson et al. (2005, 157125); [23]Ho et al. (2006, 156552); [24]Decesari et al. (2005, 144536); [25] Mayol-Bracero et al. (2002, 045010); [26]Yang et al. 2003; [27] Turšic et al. (2006, 157063); [28]Mader et al.(2004, 156724); [29]Xiao and Liu (2004, 056801); [30]Kiss et al. (2002, 156646); [31]Cornell and Jickells (1999, 156367); [32] Zheng et al. (2002, 026100); [33]Fraser et al. (2002, 140741); [34] Fraser et al. (2003, 042231) [35]Schauer er al. (1996, 051162); [36]Fine et al. (2004); [37]Yue et al. (2004); [38]Rinehart et al. (2006, 115184); [39]Wan and Yu (2006, 157104); [40]Poore (2000, 012839); [41]Fraser et al. (2003a); [42]Engling et al. (2006, 156422); [43]Yu et al. (2005); [44]Tran et al. (2000); [45]Yao et al. (2004, 102213); [46]Li and Yu (2005, 156692); [47]Henning et al. (2003, 156539); [48]Zhang and Anastasio (2003, 157182); [49]Emmenegger et al. (2007, 156418); [50](Watson et al., 1989, 157119); [51]Greaves et al. (1985, 156494); [52]Waterman et al. (2000, 157116); [53]Waterman et al. (2001, 157117); [54]Falkovich and Rudich (2001, 156427); [55]Chow et al. (2007, 156354); [56]Miguel et al. (2004, 123260); [57]Crimmins and Baker (2006, 097008); [58]Ho and Yu (2004, 156551); [59]Jeon et al. (2001, 016636); [60]Mazzoleni et al. (2007, 098038); [61]Poore (2000, 012839); [62]Butler et al. (2003); [63]Chow et al. (2006c); [64]Russell et al. (2004, 082453); [65]Grover et al. (2006); [66]Grover et al. (2005, 090044); [67]Schwab et al. (2006b); [68]Hauck et al. (2004); [69]Jaques et al. (2004, 155878); [70]Rupprecht and Patashnick (2003, 157207); [71]Pang et al (2002, 030353); [72]Eatough et al. (2001, 010303); [73]Lee et al. (2005b); [74]Lee et al. (2005, 156680); [75]Babich et al. (2000, 156239); [76]Lee et al. (2005c); [77]Lee et al. (2005b); [78]Anderson and Ogren (1998, 156213); [79]Chung et al. (2001, 156357); [80]Kidwell and Ondov (2004, 155898); [81]Lithgow et al. (2004, 126616); [82]Weber et al. (2003, 157129); [83]Harrison et al. (2004); [84]Rattigan et al. (2006, 115897); [85]Wittig et al. (2004, 103413); [86]Vaughn et al. (2005, 157089); [87]Chow et al. (2005b); [88]Weber et al (2001, 024640) ; [89]Schwab et al. (2006a); [90]Lim et al. (2003, 156697); [91]Watson and Chow (2002, 037873); [92]Venkatachari et al. (2006, 105918); [93]Bae et al. (2004a); [94]Arhami et al. (2006, 156224); [95]Park et al. (2005, 156843); [96]Bae et al. (2004b); [97]Chow et al. (2006a); [98]Arnott et al. (2005); [99]Bond et al. (1999); [100]Virkkula et al. (2005, 157097); [101]Petzold et al. (2002, 156863); [102]Park et al. (2006); [103]Arnott et al. (1999, 020650); [104]Peters et al. (2001, 017108); [105]Pitchford et al. (1997, 156872); [106]Rees et al. (2004, 097164); [107]Watson et al. (2000); [108]Lee et al.(2005, 156680); [109]Hering et al. (2004, 155837); [110]Watson et al. (1998); [111]Chakrabarti et al. (2004); [112]Mathai et al. (1990, 156741); [113]Kidwell and Ondov (2001, 017092); [114]Stanier et al. (2004); [115]Khlystov et al. (2005, 156635); [116]Takahama et al. (2004, 157038); [117]Chow et al. (2005, 156348); [118]Zhang et al. (2002); [119]Subramanian et al. (2004, 081203); [120]Chow et al. (2006b); [121]Birch and Cary (1996); [122]Birch (1998, 024953); [123]Birch and Cary (1996); [124]NIOSH (1996, 156810); [125]NIOSH (1999, 156811); [126]Chow et al. (1993); [127]Chow et al. (2007); [128]Ellis and Novakov (1982, 156416); [129]Peterson and Richards (2002, 156861); [130]Schauer et al. (2003); [131]Middlebrook et al. (2003, 042932); [132]Wenzel et al. (2003, 157139); [133]Jimenez et al. (2003); [134]Phares et al. (2003, 156866); [135]Qin and Prather (2006, 156895); [136]Zhang et al. (2005); [137]Bein et al. (2005, 156265); [138]Drewnick et al. (2004a); [139]Drewnick et al. (2004b); [140]Lake et al. (2003, 156669); [141]Lake et al. (2004, 088411)

Table A-12. Summary of element and liquid water content measurement comparisons.

SITE / PERIOD / SAMPLER	SUMMARY OF FINDINGS
College Park, MD; 11/18/1999 to 11/19/1999, 11/22/1999 Adjacent to a parking lot in the University of Maryland campus, influenced by motor vehicles, coal-fired power plants and incinerators ~21 km southwest of site and regionally transported material. **Concentrated Slurry/Graphite Furnace Atomic Absorption Spectrometry (GFAAS) (collectively known as Semi-Continuous Elements in Aerosol Sampler, SEAS)** Ambient air is pulled in at a flow rate of 170 L/min. Particles are grown using steam injection to about 3 to 4 μm in diameter, which are then concentrated and separated from the air stream in the form of a slurry using impactors. The slurry is collected in glass sample vials, which are subsequently analyzed by GFAAS in the laboratory.	**Kidwell and Ondov (2004, 155898; 2001, 017092)** Overall collection efficiency (of the entire system) measured using latex particles was 40% for particles initially 0.1 to 0.5 μm in diameter, increasing with size to 68% for particles 3 μm in diameter. Major losses were in the virtual impactor major flow channel and in the condensers. Six elements were detected *simultaneously*, limited by spectral interference and the minimum detectable limit (MDL). Twelve elements (Al, Cr, Mn, Fe, Ni, Cu, Zn, As, Se, Cd, Sb, and Pb) were measured. MDLs ranged from 3.2 picogram (pg = 10^{-12} gram) to 440 pg. Comparison with NIST standards showed good agreement, except for Al, Cr and Fe, due to poor atomization. The method was valid for dissolved solutions, but not for large particles (> 10 μm). Overall avg relative standard deviation (RSD) was 20 to 43% by error propagation, mainly due to the collection and analytical efficiencies. There were possible memory effects due to particle adhesion to impactor collection surfaces. Lower MDLs may be possible through redesign and introduction of a wash cycle between samples. A 2.5 μm inlet might improve analytical efficiency by removing coarse particles.
Pittsburgh Supersite, PA; 08/26/2002 to 09/02/2002 6 km east of downtown in a park on the top of a hill. **Laser Induced Breakdown Spectroscopy (LIBS)** Ambient air was concentrated using a $PM_{2.5}$ inlet and a virtual impactor. The concentrated stream was transported through a Teflon tube to the sample cell of the LIBS system. The sample cell was excited using a Nd: YAG laser. The resulting plasma was collected and focused into a spectrometer, generating spectra characteristic of different elements.	**Lithgow et al. (2004, 126616)** Calibration was done by sampling particle-laden streams with known metal concentrations. Good linear fits with correlation coefficients 0.97 to 0.99 Seven metals (Na, Mg, Al, Ca, Cr, Mn, and Cu) were analyzed. The MDLs were in the order of femtograms (fg = 10^{-15} gram) per sample. This system has the capability of identifying the components, quantifying them and also giving a particle size distribution. Mass was underestimated because of missing small particles.

SITE / PERIOD / SAMPLER	SUMMARY OF FINDINGS
Pittsburgh Supersite, PA; 07/01/2001 to 08/31/2001, 01/01/2002 to 07/01/2002. 6 km east of downtown in a park on the top of a hill. **Dry Ambient Aerosol Size Spectrometer (DAASS)** Measures the aerosol size distribution (using nano-SMPS, SMPS and APS) alternatively, at ambient relative humidity (RH) (ambient channel) and at low RH (18 ± 6%) (dry channel). A comparison of the two size distributions provides information on the water absorption and change in size due to RH.	**Stanier et al. (2004, 095955); Khlystov et al. (2005, 156635)** Measured water content ranging from less than 1 $\mu g/m^3$ to 30 $\mu g/m^3$, constituting < 5% to 100% of the dry aerosol mass. Small differences between dry and ambient channels of the DAASS. Number concentrations were within 5% of each other. Additional sources of error are associated with temperature differences between measured outdoor ambient temperature and the temperature at which the ambient measurement channel was maintained. Although the measurement system was placed in a ventilated enclosure, it was ~4 °C higher than ambient temperature during July 2001. During winter, the system was maintained at a minimum temperature of 9 °C, while the outdoor temperature dropped to -5 °C. This caused differences in RH sensed by the system in the ambient channel versus the actual outdoor RH. RH differences cause underestimation of the particle number at sizes < 200 nm and an overestimation at sizes >200 nm. This causes the volume growth factor to be higher by 2 to 14%, with the highest bias occurring at high RH and low temperature (92% outside RH and -5 °C). The difference in temperature might also lead to evaporation of semi-volatile components such as NH_4NO3. For the winter period, it was estimated that, for the worst case, the volume growth factor would be underestimated by about 10% for 60 - 90% RH. Insufficient purging of dry air between the dry and ambient cycles (implying the need for supplemental vacuum power during the vent stages) causes uncertainties in estimated growth factors. Correction factors were between 0.97 and 1.03. Water content estimated by DAASS can be used to evaluate the thermodynamic models. For the Pittsburgh study, the models underestimated the water content by 37%. Data from DAASS showed that the aerosol was wet even at ambient RH less than 30%.

Table A-13. Summary of $PM_{2.5}$ NO_3^- measurement comparisons.

SITE / PERIOD / SAMPLER / CONFIGURATION				SUMMARY OF FINDINGS
ATLANTA SUPERSITE, GA: 8/3/99 to 9/1/99 4 km NW of downtown, within 200 m of a bus maintenance yard and several warehouse facilities, representative of a mixed commercial-residential neighborhood.				**Solomon et al[17]** $PM_{2.5}$ NO_3^- from each sampler was compared to the all-sampler avgs, called the filter relative reference (filter RR) value. Overall agreements were within 30-35% of filter RR. Wide scatter from paired comparisons, possibly due to volatilized NO_3^-, differences in denuder design and filter types, and low concentrations (close to analytical uncertainty). A small positive artifact (few tenths of $\mu g/m^3$) might be present when using Na_2 CO_3 impregnated filters, due to possible collection (and subsequent oxidation) of HONO and NO_2 on carbonate-impregnated filters. In addition, glycerol in Na_2CO_3 coated denuders may contaminate the filters downstream. $PM_{2.5}$ NO_3^- R&P-2000 FRM and MOUDI-100 samplers are consistently lower than other samplers. **Weber et al. [82]** Hourly $PM_{2.5}$ NO_3^-were compared to all-sampler averages (continuous RR), similar to the approach used for integrated filter samplers. Overall agreements were within ± 20-30% (or ± 0.2 $\mu g/m^3$) except for ARA-N. Except for ARA-N, good correlations (R2 = 0.70 to 0.90) were found during the
SAMPLER	**FLOW RATE (L/MIN)**	**FILTER TYPE[a]**	**DENUDER[b]**	
R&P-2000 FRM	16.7	Quartz (P)	None	
RAAS-400	24	Nylon (P)	MgO	
SASS	6.7	Nylon (P)	MgO	
MASS-400	16.7	Teflon (P)-Nylon (P	Na_2CO_3	
MASS-450	16.7	Quartz (P)	None	
R&P-2300	10	Nylon (P)	Na_2CO_3	
VAPS	15	Polycarbonatec (front & back-up)	Na_2CO_3	
URG-PCM	16.7	Teflon (P)-Cellulose-fiber (W	Na_2CO_3	
ARA-PCM	16.7	Teflon (n/a)-Nylon (n/a)	Na_2CO_3/Citric acid	
PC-BOSS (TVA)	105	Teflon (W)- Nylon (P)	CIF	
PC-BOSS (BYU)	150	Teflon (W)- Nylon (P)	CIF	
PC-BOSS (BYU)	150	Quartz (P)- CIF (S)	CIF	
MOUDI-100	30	Teflon (n/a- Quartz (n/a	None	

SITE / PERIOD / SAMPLER / CONFIGURATION				SUMMARY OF FINDINGS
				second half of the study. The poor performance of ARA-N was probably due to an inefficient denuder (25-60% efficient) resulting in high background. Large discrepancies between continuous and filter RR, probably due to low ambient concentrations (study avg = 0.5 $\mu g/m^3$) near the detection limit ($\sim$ 0.1 $\mu g/m^3$, except for ARA-N, which had 0.5 $\mu g/m^3$). The ARA-N was within 13%, ADI-N, ECN and PILS-IC within 18% and TT within 26% of filter RR (all $<$ 0.2 $\mu g/m^3$ difference). Filter samples showed more variability (Relative Standard Deviation, RSD = 22%) than continuous measurements (RSD = 13%). This is probably due to sampling artifacts in filter samples; NO_3^-volatilization in continuous monitors is expected to be minimal due to shorter averaging times and rapid stabilization in solutions.
CONTINUOUS SAMPLER	**FLOW RATE (L/MIN)**	**DENUDER**	**ANALYSIS METHOD[b]**	
ADI-N	1	Activated Carbon	NOx Chemiluminescence	
ARA-N	3	Potassium iodide (KI) and dual sodium chlorite ($NaClO_2$)	NOx Chemiluminescence	
PILS-IC	5	Two URG annular glass denuders in series containing citric acid and CaCO3	IC	
ECN	16.7	Rotating annular wet denuder system	IC	
TT	5	Wet parallel plate denuder	IC	
PITTSBURGH SUPERSITE, PA; 7/1/01 to 8/1/02 6km east of downtown in a park on the top of a hill				**Cabada et al. [18]; Takahama et al. [116]** More than 70% ($\sim$ 0.5 $\mu g/m^3$) of NO_3 mass was lost from MOUDI samplers during summer. MOUDI NO_3 = 0.27 CMU; R2 = 0.40; Summer MOUDI NO_3 = 0.99 CMU; R2 = 0.49; winter **Wittig et al. [85]** Avg conversion efficiency to NOx (tested using NH_4NO3 solution) was 0.85 ± 0.08. Gas analyzer efficiency was stable at 0.99 ± 0.04. Corrections were made for instrument offset, software calculation error, conversion efficiency, gas analyzer efficiency, vacuum drift, and sample flow drift. The overall avg correction was 8%, ranging from -62% to 93%. Data Recovery $>$80%. Data loss was associated with vacuum pump failures and excessive flash strip breakage. R&P-8400N = 0.83 CMU + 0.20 $\mu g/m^3$; R2 = 0.84 Under-estimation in the R&P-8400N could be due to incomplete particle collection or incomplete conversion of various forms of NO_3^-. Used co-located filter measurements for final calibration.
SAMPLER	**FLOW RATE (L/MIN)**	**FILTER TYPE[a]**	**DENUDER[b]**	
MOUDI-110	30	Teflon (W Teflon (W)-	None	
CMU	16.7	Nylon (W)	MgO/Citric acid	
R&P-2000 FRM	16.7	Teflon (W)	None	
FRESNO SUPERSITE, CA and other CRPAQS sites; 12/2/99 to 2/3/01 Located 5.5 km northeast of downtown in a mixed residential-commercial neighborhood. [107]				**Chow et al. [87]** Maximum NO_3^- volatilization was observed during summer (Jun - Aug), while the lowest volatilization was observed during winter (Dec-Feb). Seasonal avg volatilized NO_3^- in particulate NO_3^- (PNO_3^-, the sum of non-volatilized and volatilized NO_3^-) ranged from less than 10% during winter to more than 80% during summer. Volatilized NH_4NO_3 accounted for 44% of actual $PM_{2.5}$ mass (i.e., measured mass plus volatilized NH_4NO_3) in Fresno during summer. Front-quartz non-volatilized NO_3^- concentrations were similar for DRISFS (0.52 ± 0.26 $\mu g/m^3$)
SAMPLER	**FLOW RATE (L/MIN)**	**FILTER TYPE[a]**	**DENUDER**	
DRI-SFS	113	Quartz (Pellulose	Al_2O3	
RAAS-400	24	Quartz (P)-Nylon (P)	Na_2CO_3	
RAAS-400	24	Quartz (P)-Quartz (P)	None	
RAAS-100 FRM	16.7	Quartz (P)	None	
CONTINUOUS SAMPLER	**FLOW RATE (L/MIN)**	**DENUDER**	**ANALYSIS METHOD[b]**	
R&P-8400N	5	Activated Carbon	NOx Chemiluminescence	

SITE / PERIOD / SAMPLER / CONFIGURATION				SUMMARY OF FINDINGS
				and RAAS-100 FRM (0.81 ± 0.33 $\mu g/m^3$) for warm months (May-Sep). With preceding denuders, the DRI-SFS
SAMPLER	**FLOW RATE (L/MIN)**	**FILTER TYPE[a]**	**DENUDER**	
PC-BOSS	150	Teflon (W)- Nylon (P)	CIF	PNO_3 concentration (3 ± 1.9 $\mu g/m^3$) was much higher than the RAAS100 FRM NO_3^-, suggesting that the FRM sampler removed gaseous nitric acid (HNO_3) resulting in NO_3^- volatilization. FRM Teflon-membrane filters are subject to similar NO_3^-losses.
CONTINUOUS SAMPLER	**FLOW RATE (L/MIN)**	**DENUDER**	**ANALYSIS METHOD[b]**	
R&P-8400N	5	Activated Carbon	NO_x Chemiluminescence	
Dionex-IC	5	Parallel plate wet denuder	IC	**Chow et al. [117]**
				High correlation (R2 > 0.90) between 24-h avg R&P-8400N NO_3 and SFS filter NO_3^- concentrations, but R&P-8400N NO_3^- was 7 to 25% lower than filter NO_3^-.
				Limited comparison (n < 15) with filter samples at Bakersfield showed that the slopes were close to unity during early morning hours, while they decreased during the afternoon hours, indicating possible loss of $NO_3^-$by the R&P-8400N instrument.
				The R&P-8400N required substantial maintenance and careful operation.
				Grover et al. [65]
				Dionex-IC NO_3 = (0.71 ± 0.04) PC-BOSS NO_3 + (3.2 ± 1.1); R2 = 0.91; n = 29
				R&P-8400N = (1.10 ± 0.06) PC-BOSS NO_3 - (0.8 ± 1.8); R2 = 0.93; n = 29
				R&P-8400N = (0.55 ± 0.01) Dionex-IC + (1.4 ± 1.8); R2 = 0.75; n = 493
				R&P-8400N measured less than DIONEX IC, particularly at high RH. R&P-8400N may suffer incomplete flash vaporization under conditions of high RH.
BALTIMORE SUPERSITE, MD; 2/14/02 to 11/30/02 Adjacent to a parking lot in the University of Maryland campus, influenced by motor vehicles, coal-fired power plants and incinerators ~21 km southwest of site and regionally transported material.				**Harrison et al. [83]**
				Corrections were made to R&P-8400N data for software calculation error, conversion efficiency, gas analyzer efficiency, vacuum drift and sample flow drift.
SAMPLER	**FLOW RATE (L/MIN)**	**FILTER TYPE[a]**	**DENUDER**	
SASS	6.7	Nylon (n/a)	MgO	The relative uncertainty of R&P-8400N measurements averaged 8.7%, ranging from 6.3% to 23%.
CONTINUOUS SAMPLER	**FLOW RATE (L/MIN)**	**DENUDER**	**ANALYSIS METHOD[b]**	Data capture > 95%.
R&P-8400N	5	Activated Carbon	NO_x Chemiluminescence	R&P-8400N underestimated SASS filter NO_3^- by

SITE / PERIOD / SAMPLER / CONFIGURATION	SUMMARY OF FINDINGS
	~33%, attributed to variations in conversion efficiency, matrix effects, and impaction efficiency. This suggested a true conversion efficiency of 68% as compared to an avg conversion efficiency of R&P-8400N to NO_x (tested using potassium nitrate solution) of 0.90 ± 0.04. Large errors occurred when the concentrations were near the detection limit, when the temperature difference (between instrument and ambient) was large, and when the ambient relative humidity (RH) was < 40%. Ridged flash strips produced lower dissociation losses than flat strips. Reliable measurements were obtained when the instrument-outdoor temperature differences were minimal and when grooved/ridged flash strips were used. A co-located filter measurement was used for final corrections.
NEW YORK SUPERSITE, NY; 06/29/01 to 08/05/01 and 07/09/02 to 08/07/02 Urban site located at Queens College, NY, about 14 km west of Manhattan, within 2 km of freeways, and within 12 km of international airports. Rural site located at Whiteface mountain, 600 m above sea level, in a clearing surrounded by deciduous and evergreen trees and no major cities within 20 km of the site. **SAMPLER** / **FLOW RATE (L/MIN)** / **FILTER TYPE[a]** / **DENUDER** R&P-2300 / 10 / Nylon (n/a) / Na_2CO_3 **CONTINUOUS SAMPLER** / **FLOW RATE (L/MIN)** / **DENUDER** / **ANALYSIS METHOD[b]** R&P-8400N / 5 / Activated Carbon / NO_x Chemiluminescence PILS-IC / 5 / Na_2CO_3 and citric acid / IC AMS / 0.1 / None / Mass Spectrometry	**Hogrefe et al. [20]** Data completeness: 86 - 88% for R&P-8400N, 94 -98% for AMS, and 65 - 70% for PILS-IC. Some PILS measurements were invalidated owing to larger aqueous flow caused by bigger tubing. Larger aqueous flow and inconsistent water quality affected NO_3^- concentrations. R&P-8400N NO_3^- was lower than R&P-2300 filter NO_3^-. PILS-IC was within 5% of R&P-2300 filter NO_3^- concentrations. At the urban site, AMS was within 10% of the filter NO3concentration. At the rural site, AMS had a slope of 0.51 and R2 of 0.46, compared with filter NO_3^-.
NEW YORK SUPERSITE, NY; 10/01 to 07/05 (urban), 07/02 to 07/05 (rural) Urban site located at a school in South Bronx, NY in a residential area, within a few kilometers away from major highways and a freight yard (experiencing significant truck traffic). Rural site located at Whiteface mountain, 600 m above sea level, in a clearing surrounded by deciduous and evergreen trees and no major cities within 20 km of the site. **SAMPLER** / **FLOW RATE (L/MIN)** / **FILTER TYPEA** / **DENUDER[b]** R&P-2300 / 10 / Nylon (n/a) / Na_2CO_3 TEOM-ACCU / 16.7 / Zefluor / None **CONTINUOUS SAMPLER** / **FLOW RATE (L/MIN)** / **DENUDER** / **ANALYSIS METHOD[b]** R&P-8400N / 5 / Activated Carbon / NO_x Chemiluminescence	**Rattigan et al. [84]** Data capture was more than 94%. Data were adjusted for span and zero drifts, conversion efficiency, flow drift, and blanks. R&P-8400N NO_3^- was systematically lower than R&P-2300 filter NO3over all concentration ranges, except at < 1 $\mu g/m^3$. Urban: R&P-8400N = 0.59 R&P-2300 NO_3 + 0.28; R2 = 0.88; n = 305 Rural: R&P-8400N = 0.73 R&P-2300 NO_3 + 0.01; R2 = 0.90; n ~ 161; however concentrations were low with 95% of data < 1 $\mu g/m^3$. Required weekly or biweekly maintenance by trained personnel.
LOS ANGELES SUPERSITE, CA; 7/13/01 to 9/15/01 (Rubidoux) and 9/15/01 to 2/10/02 (Claremont) Multiple sampling locations in the South Coast Air Basin (SoCAB), including urban "source" sites and downwind "receptor" sites. **SAMPLER** / **FLOW RATE (L/MIN)** / **FILTER TYPEa** / **DENUDER[b]** MOUDI / 30 / Teflon (P / None HEADS / 10 / Teflon (n/a) - GF-GF / Carbonate	**Fine et al. [19]** MOUDI = 0.68 HEADS; R2 = 0.88 ADI-N Sized = 0.80 HEADS; R2 = 0.79 ADI-N Sized = 1.12 MOUDI; R2 = 0.53 ADI-N NO_3^- showed better agreement with HEADS at lower concentrations, the ADI-N deviated (biased low) from the HEADS concentrations at higher NO_3^-concentrations.

SITE / PERIOD / SAMPLER / CONFIGURATION				SUMMARY OF FINDINGS
CONTINUOUS SAMPLER	**FLOW RATE (L/MIN)**	**DENUDER**	**ANALYSIS METHOD[b]**	This deviation was attributed to NO_3^- vaporization, loss of NO_3^- associated with particles less than 0.1 μm not collected by the ADI-N sampler, or loss of particles in the ADI-N inlet tubing.
ADI-N Sized	0.9	Activated Carbon	NO_X Chemiluminescence	The underestimation of NO_3^- by MOUDI compared to HEADS may be due to NO_3^- volatilization from MOUDI stages, since SO_4^{2-} comparisons showed MOUDI to explain 85% of HEADS SO_4^{2-}. ADI-N and MOUDI showed better correlation (R2 = 0.67) for the 1 to 2 μm size range NO_3 relative to other size ranges (R2 < = 0.56). This is possibly due to NO_3^- in the form of non-volatilized sodium nitrate (NaNO3) than volatilized NH_4NO3 in the 1-2 μm size range. Single particle analysis also indicated this possibility of NaNO3 in the 1 to 2 μm range.
RUBIDOUX, CA; 07/01/03 to 07/31/03 Located in the eastern section of SoCAB in the north-west corner of Riverside County, 78 km downwind of the central Los Angeles metropolitan area and in the middle of the remaining agricultural production area in SoCAB.				**Grover et al. [66]** R&P-8400N = (0.65 ± 0.07) PC-BOSS + (3.3 ± 2.4); R2 = 0.73; n = 31 At higher concentrations (No numerical value reported), R&P-8400N NO_3^- was lower than PC-BOSS NO_3^-, possibly due to incomplete volatilization of NH_4NO3 in R&P-8400N at higher concentrations (and higher relative humidity). At the urban site, the continuous instruments correlated well with filter NO_3^- measurements and among themselves (R2 ≥ 0.89). At the rural site, R2 ranged from 0.61 to 0.83, except for the AMS versus R&P2300 comparison, with an R2 of 0.46.

SAMPLER	**FLOW RATE (L/MIN)**	**FILTER TYPE[a]**	**DENUDER[b]**
PC-BOSS	150	Teflon (W)-Nylon (P)	CIF

CONTINUOUS SAMPLER	**FLOW RATE (L/MIN)**	**DENUDER**	**ANALYSIS METHOD[b]**
R&P-8400N	5	Activated Carbon	NO_X Chemiluminescence
R&P-8400N	5	Activated Carbon	NOx Chemiluminescence
PILS-IC	5	Na_2CO_3 and Citric acid	IC
AMS	0.1	None	Mass Spectrometry

SAMPLER	FLOW RATE (L/MIN)	FILTER TYPE[a]	DENUDER[b]

[a]Filter Manufacturer in parenthesis - W: Whatman, Clifton, NJ; P: Pall-Gelman, Ann Arbor, MI; S: Schleicher & Schnell. Keene, NH; n/a: not available or not reported.
[b]Al_2O3: Aluminum oxide; GF: Na_2CO_3 impregnated Glass Fiber Filters; IC: Ion chromatography; MgO: Magnesium oxide; Na_2CO_3: Sodium carbonate; NaHCO3: Sodium bicarbonate NO_X: Oxides of nitrogen; CIF: Charcoal Impregnated Filter; FEP: Fluorinated Ethylene Propylene copolymer; TEA: Triethanolamine; TSP: Total Suspended PM.
[c]Na_2CO_3 impregnated.
[d]37-mm filter.

Source: [1]Chow (1995, 077012); [2]Watson and Chow (2001, 157123); [3] Watson et al. (1983, 045084); [4]Fehsenfeld et al. (2004, 156432); [5]Solomon et al. (2001, 156993); [6]Mikel (2001, 156762); [7]Mikel (2001, 156762); [8]Watson et al. (1999, 020949); [9]Solomon and Sioutas (2006, 156995); [10]Graney et al. (2004, 053756); [11]Tanaka et al. (1998, 157041); [12]Pancras et al. (2005, 098120); [13]John et al. (1988, 045903); [14]Hering and Cass (1999, 084958); [15]Fritz et al. (1989, 077387); [16]Hering et al. (1988, 036012); [17]Solomon et al. (2003, 156994); [18]Cabada et al. (2004, 148859); [19]Fine et al. (2003, 155775); [20]Hogrefe et al. (2004, 099003); [21]Drewnick et al. (2003, 099160); [22]Watson et al. (2005, 157125); [23]Ho et al. (2006, 156552); [24]Decesari et al. (2005, 144536); [25] Mayol-Bracero et al. (2002, 045010); [26]Yang et al. 2003; [27] Turšic et al. (2006, 157063); [28]Mader et al.(2004, 156724); [29]Xiao and Liu (2004, 056801); [30]Kiss et al. (2002, 156646); [31]Cornell and Jickells (1999, 156367); [32] Zheng et al. (2002, 026100); [33]Fraser et al. (2002, 140741); [34] Fraser et al. (2003, 042231) [35]Schauer er al. (1996, 051162); [36]Fine et al. (2004); [37]Yue et al. (2004); [38]Rinehart et al. (2006, 115184); [39]Wan and Yu (2006, 157104); [40]Poore (2000, 012839); [41]Fraser et al. (2003a); [42]Engling et al. (2006, 156422); [43]Yu et al. (2005); [44]Tran et al. (2000); [45]Yao et al. (2004, 102213); [46]Li and Yu (2005, 156692); [47]Henning et al. (2003, 156539); [48]Zhang and Anastasio (2003, 157182); [49]Emmenegger et al. (2007, 156418); [50](Watson et al., 1989, 157119); [51]Greaves et al. (1985, 156494); [52]Waterman et al. (2000, 157116); [53]Waterman et al. (2001, 157117); [54]Falkovich and Rudich (2001, 156427); [55]Chow et al. (2007, 156354); [56]Miguel et al. (2004, 123260); [57]Crimmins and Baker (2006, 097008); [58]Ho and Yu (2004, 156551); [59]Jeon et al. (2001, 016636); [60]Mazzoleni et al. (2007, 098038); [61]Poore (2000, 012839); [62]Butler et al. (2003); [63]Chow et al. (2006c); [64]Russell et al. (2004, 082453); [65]Grover et al. (2006); [66]Grover et al. (2005, 090044); [67]Schwab et al. (2006b); [68]Hauck et al. (2004); [69]Jaques et al. (2004, 155878); [70]Rupprecht and Patashnick (2003, 157207); [71]Pang et al (2002, 030353); [72]Eatough et al. (2001, 010303); [73]Lee et al. (2005b); [74]Lee et al.(2005, 156680); [75]Babich et al. (2000, 156239); [76]Lee et al. (2005c); [77]Lee et al. (2005b); [78]Anderson and Ogren (1998, 156213); [79]Chung et al. (2001, 156357); [80]Kidwell and Ondov (2004, 155898); [81]Lithgow et al. (2004, 126616); [82]Weber et al. (2003, 157129); [83]Harrison et al. (2004); [84]Rattigan et al. (2006, 115897); [85]Wittig et al. (2004, 103413); [86]Vaughn et al. (2005, 157089); [87]Chow et al. (2005b); [88]Weber et al (2001, 024640) ; [89]Schwab et al. (2006a); [90]Lim et al. (2003, 156697); [91]Watson and Chow (2002, 037873); [92]Venkatachari et al. (2006, 105918); [93]Bae et al. (2004a); [94]Arhami et al. (2006, 156224); [95]Park et al. (2005, 156843); [96]Bae et al. (2004b); [97]Chow et al. (2006a); [98]Arnott et al. (2005); [99]Bond et al. (1999); [100]Virkkula et al. (2005, 157097); [101]Petzold et al. (2002, 156863); [102]Park et al. (2006); [103]Arnott et al. (1999, 020650); [104]Peters et al. (2001); [105]Pitchford et al. (1997, 156872); [106]Rees et al. (2004, 097164); [107]Watson et al. (2000); [108]Lee et al.(2005, 156680); [109]Hering et al. (2004, 155837); [110]Watson et al. (1998); [111]Chakrabarti et al. (2004); [112]Mathai et al. (1990, 156741); [113]Kidwell and Ondov (2001, 017092); [114]Stanier et al. (2004); [115]Khlystov et al. (2005, 156635); [116]Takahama et al. (2004, 157038); [117]Chow et al. (2005, 156348); [118]Zhang et al. (2002); [119]Subramanian et al. (2004, 081203); [120]Chow et al. (2006b); [121]Birch and Cary (1996); [122]Birch (1998, 024953); [123]Birch and Cary (1996); [124]NIOSH (1996, 156810); [125]NIOSH (1999, 156811); [126]Chow et al. (1993); [127]Chow et al. (2007); [128]Ellis and Novakov (1982, 156416); [129]Peterson and Richards (2002, 156861); [130]Schauer et al. (2003); [131]Middlebrook et al. (2003, 042932); [132]Wenzel et al. (2003, 157139); [133]Jimenez et al. (2003); [134]Phares et al. (2003, 156866); [135]Qin and Prather (2006, 156895); [136]Zhang et al. (2005); [137]Bein et al. (2005, 156265); [138]Drewnick et al. (2004a); [139]Drewnick et al. (2004b); [140]Lake et al. (2003, 156669); [141]Lake et al. (2004, 088411)

Table A-14. Summary of $PM_{2.5}$ SO_4^{2-} measurement comparisons

SITE/PERIOD/SAMPLER/ CONFIGURATION				SUMMARY OF FINDINGS
ATLANTA SUPERSITE, GA: 08/03/99 to 09/01/99 4 km NW of downtown, within 200 m of a bus maintenance yard and several warehouse facilities, representative of a mixed commercial-residential neighborhood.				**Solomon et al. [17]** $PM_{2.5}$ SO_4^{2-} from each sampler was compared to all-sampler averages, called the filter relative reference (filter RR) value. The samplers agreed to within 10% of filter RR, except for the PC-BOSS (TVA) and MOUDI-100. While avg mass was within 10%, daily variability was > 50% of filter RR. All samplers, except for the PC-BOSS (TVA), correlated well (R2 > 0.90) with daily filter RR. PC-BOSS (TVA) had instrument leaks. The R&P-2000 FRM, on avg, agreed within 1% of filter RR. MOUDI-100 was ~13% low compared to filter RR. **Weber et al. [82]; Zhang et al. [118]** Hourly $PM_{2.5}$ SO_4^{2-} were compared to all-sampler averages (continuous RR), similar to the approach used for filter samplers. Overall agreement was within 16% or 2 $\mu g/m^3$. Good correlations (R2 = 0.76 to 0.94) were found during the second half of the study, except for TT versus ADI. Good correlation (R2 = 0.84) was found between continuous and filter-based SO_4^{2-}. Continuous RR = (1.15 ± 0.15), Filter RR + (0.41 ± 1.73) Variability among continuous SO_4^{2-} instruments
SAMPLER	**FLOW RATE (L/MIN)**	**FILTER TYPE[a]**	**DENUDER[b]**	
R&P-2000 FRM	16.7	Quartz (P)	None	
RAAS-400	24	Teflon (P)	None	
SASS	6.7	Teflon (P)	None	
MASS-450	16.7	Quartz (P)	None	
R&P-2300	10	Quartz (P)	NOne	
VAPS	15	Quartz (P)	XAD-4	
URG-PCM	16.7	Teflon (P)-Cellulose-fiber (W		
ARA-PCM	16.7	Teflon (n/a)	Na_2CO_3/Citric acid	
ARA-PCM	16.7	Nylon (n/a)	Na_2CO_3/Citric acid	
PC-BOSS (TVA)	105	Teflon (W)	CIF	
PC-BOSS (TVA)	105	Quartz (P)	CIF	
PC-BOSS (BYU)	150	Teflon (W)	CIF	
PC-BOSS (BYU)	150	Quartz (P)	CIF	
MOUDI-100	30	Teflon (n/a- Quartz (n/a	None	

SITE/PERIOD/SAMPLER/ CONFIGURATION	SUMMARY OF FINDINGS

(RSD = 13%) was similar to that for NO_3 instruments. Filter sample variability was low (RSD = 8%) indicating more uniformity among samplers.

The ECN and TT instruments were within 15%, PILS-IC was within 20% and ADI-S was within 26% of filter RR.

CONTINUOUS SAMPLER	FLOW RATE (L/min)	DENUDER	ANALYSIS METHOD[b]
ADI-S	2.7	Activated Carbon	SO_2, UV Fluorescence
PILS-IC	5	Two URG annular glass denuders in series containing citric acid & CaCO3	IC
ECN	16.7	Rotating annular wet denuder system	IC
TT	5	Wet parallel plate denuder	IC

PITTSBURGH SUPERSITE, PA; 070/1/01 to 08/01/02
6km east of downtown in a park on the top of a hill

SAMPLER	FLOW RATE (L/MIN)	FILTER TYPE[a]	DENUDER[b]
MOUDI-110	30	Teflon (W	None
CMU	16.7	Teflon (W)	MgO/Citric acid
R&P-2000 FRM	16.7	Teflon (W)	None

CONTINUOUS SAMPLER	FLOW RATE (L/min)	DENUDER	ANALYSIS METHOD[b]
R&P-8400S	5	Activated Carbon	SO_2 UV Fluorescence

Cabada et al., [18]; Takahama et al., [116]

MOUDI SO_4^{2-} 0.80 CMU; R2 = 0.95; Summer

MOUDI SO_4^{2-} 0.97 CMU; R2 = 0.48; winter

Wittig et al. [85]

Avg conversion efficiency to SO_2 (tested using ammonium sulfate [$(NH_4)_2SO_4$] solution) was 0.65 ± 0.07. Gas analyzer efficiency was stable at 0.99 ± 0.06.

Corrections were made for instrument offset, software calculation error, conversion efficiency, gas analyzer efficiency, vacuum drift, and sample flow drift. The overall correction was, on avg, -1% and ranged from -90% to 100% for individual samples.

Data Recovery > 90%. Data loss was associated with vacuum pump failures or excessive flash strip breakage.

R&P-8400S (SO_4^{2-}) = 0.71 CMU + 0.42 $\mu g/m^3$; R2 = 0.83

Underestimation is attributed to incomplete particle collection or incomplete conversion of various forms of SO_4^{2-}.

Used co-located filter measurements for final calibration.

LOS ANGELES SUPERSITE, CA; 07/13/01 to 09/15/01 (Rubidoux) and 09/15/01 to 02/10/02 (Claremont)
Multiple sampling locations in the South Coast Air Basin (SoCAB), including urban "source" sites and downwind "receptor" sites.

SAMPLER	FLOW RATE (L/MIN)	FILTER TYPE[a]	DENUDER
MOUDI	30	Teflon (P	None
HEADS	10	Teflon (n/a) -GF[c]-GF[c]	Carbonate

Fine et al. [19]

MOUDI explained 85% of HEADS SO_4^{2-} (R2 = 0.89; n = 40)

NEW YORK SUPERSITE, NY; 06/29/01 to 08/05/01 and 07/09/02 to 08/07/02
Urban site located at Queens College, NY, about 14 km west of Manhattan, within 2 km of freeways, and within 12 km of international airports. Rural site located at Whiteface mountain, 600m above sea level, in a clearing surrounded by deciduous and evergreen trees and no major cities within 20 km of the site.

SAMPLER	FLOW RATE (L/MIN)	FILTER TYPE[a]	DENUDER[b]
R&P-2300	10	Nylon (n/a)	Na_2CO_3
SCS	42	Zefluor (n/a)	None

Drewnick et al. [21]; Hogrefe et al. [20]

Data completeness: 89 - 93% for R&P-8400S, 94 - 98% for AMS, 81-98% for CASM, and 65-70% for PILS-IC.

The urban site data showed good correlations (R2 = 0.87 to 0.94) with slopes ranging from 0.97 to 1.01. At the rural site, the variability was large (R2 = 0.73 to 0.91) with slopes ranging from 0.76 to 1.32. SO_4 from PILS-IC was overestimated by

SITE/PERIOD/SAMPLER/ CONFIGURATION				SUMMARY OF FINDINGS
TEOM-ACCU	16.7	Zefluor (n/a)	None	~25% when compared to the AMS at the rural site.
CONTINUOUS SAMPLER	**FLOW RATE (L/min)**	**DENUDER**	**ANALYSIS METHOD[b]**	Filter samples were within 5% of each other, except for comparison of ACCU with R&P-2300 at the rural site, with high correlations (R2 = 0.97 to 1.0). ACCU underestimated SO_4^{2-} by ~15%.
R&P-8400S	5	Activated Carbon	SO_2 UV Fluorescence	Continuous versus six-h SCS filter comparisons showed high R2 (0.91 to 0.95) at the urban site. Continuous instruments consistently measured lower SO_4^{2-} concentrations compared to the SCS filter measurements (slopes 0.68 to 0.73)
PILS-IC	5	Na_2CO_3 and Citric acid	IC	
AMS	0.1	None	Mass Spectrometry	On avg, 85% of the filter-based SO_4^{2-} was measured by the continuous instruments with consistent relationships. At the rural site, PILS-IC overestimated SO_4^{2-} concentrations (slopes 1.11 to 1.15), AMS and R&P-8400S showed slopes of 0.71-0.74 against SCS and ACCU, while it ranged from 0.53- 0.68 against R&P-2300.
CASM	5	Na_2CO_3 and Carbon and a Nafion dryer	SO_2 UV Fluorescence	Error estimates:
				Sampling losses: 2-3% for AMS and PILS-IC, 5-10% for R&P-8400S and none for CASM.
				Continuous instruments probably experienced more inlet transport losses (~ 25%) than filter samplers due to longer inlet lines.
				Small (< 2%) positive artifact was found in filters.
NEWYORK SUPERSITE, NY; 10/01 to 07/05 (urban), 07/02 to 07/05 (rural) Urban site located at a school in South Bronx, NY in a residential area, within a few kilometers from major highways and a freight yard (experiencing significant truck traffic). Rural site located at Whiteface mountain, 600m above sea level, in a clearing surrounded by deciduous and evergreen trees and no major cities within 20 km of the site. The study by Schwab et al.[89] was based at a rural site located at Pinnacle State Park surrounded by golf course, picnic areas and undeveloped forest lands and no major cities within 15 km.				**Rattigan et al. [84]**
INTEGRATED SAMPLER	**FLOW RATE (L/MIN)**	**FILTER TYPE[a]**	**DENUDER[b]**	Data capture was above 85%. Data loss was primarily due to frequent flash strip failures, every 2 weeks and without warning.
R&P-2300	10	Nylon (n/a)	Na_2CO_3	Data were adjusted for span and zero drifts, measured conversion efficiency, flow drift, and blanks.
TEOM-ACCU	16.7	Zefluor	None	Calibrations used aqueous standards of $(NH_4)_2SO_4$ and oxalic acid solution in 1: 4 ratio. Lower fractions of oxalic acid showed lower conversion efficiencies.
CONTINUOUS SAMPLER	**FLOW RATE (L/min)**	**DENUDER**	**ANALYSIS METHOD[b]**	Urban South Bronx site:
R&P-8400S	5	Activated Carbon	SO_2 pulsed fluorescence	R&P-8400S = 0.82 TEOM-ACCU + 1.15; R2 = 0.84; n = 513
TE-5020 (07/14/04 to 11/01/04)	5	Na_2CO_3	SO_2 pulsed fluorescence	R&P-8400S = 0.74 R&P-2300 + 1.14; R2 = 0.81; n = 322
				Rural Whiteface mountain:
				R&P-8400S = 0.75 TEOM-ACCU + 0.22; R2 = 0.95; n = 207
				R&P-8400S = 0.78 R&P-2300 + 0.17; R2 = 0.85; n = 198
				Required weekly or biweekly maintenance by trained personnel
				Schwab et al. [89]
				TE-5020 = 0.78 ACCU – 0.2; R2 = 0.94
				Similar studies at St. Louis, MO, show slopes near unity. This suggests that the instrument is sensitive to aerosol composition.
				Low maintenance and calibration requirements for TE-5020 compared to PILS-IC and R&P-8400S.

SITE/PERIOD/SAMPLER/ CONFIGURATION	SUMMARY OF FINDINGS
FRESNO SUPERSITE,CA; 12/01/03 to 12/23/03 Located 5.5 km northeast of downtown in a mixed residential-commercial neighborhood. Flow Sampler (L/min) Filter Type[a] Denuder[b]	**Grover et al. [65]** Dionex-IC SO_4^{2-} (1.03 ± 0.03) PC-BOSS SO_4 + (0.2 ± 0.3); R2 = 0.98; n = 27 R&P-8400S SO_4^{2-} (0.95 ± 0.05) Dionex-IC SO_4 + (0.3 ± 0.6); R2 = 0.68; n = 195

SAMPLER	FLOW RATE (L/MIN)	FILTER TYPE[a]	DENUDER[b]
PC-BOSS	150	Teflon (W)- Nylon (P)	CIF

CONTINUOUS SAMPLER	FLOW RATE (L/min)	DENUDER	ANALYSIS METHOD[b]
R&P-8400S	5	Activated Carbon	SO_2 pulsed fluorescence
Dionex-IC	5	Parallel plate wet denuder	IC

[a]Filter Manufacturer in parentheses - W: Whatman, Clifton, NJ; P: Pall-Gelman, Ann Arbor, MI; S: Schleicher & Schnell. Keene, NH; n/a: not available.

[b]Al_2O3: Aluminum oxide; IC: Ion chromatography; CIF: Charcoal Impregnated Filter; FEP: Fluorinated Ethylene Propylene copolymer; MgO: Magnesium oxide; Na_2CO_3: Sodium carbonate; $NaHCO3$: Sodium bicarbonate NO_x: Oxides of nitrogen; SO_2: Sulfur dioxide; TEA: Triethanolamine; TSP: Total Suspended PM; UV: Ultraviolet; XAD-4: Hydrophobic, non-polar polyaromatic resin.

[c]Na_2CO_3 impregnated.

[d]37-mm filter.

Source: [1]Chow (1995, 077012); [2]Watson and Chow (2001, 157123); [3] Watson et al. (1983, 045084); [4]Fehsenfeld et al. (2004, 156432); [5]Solomon et al. (2001, 156993); [6]Mikel (2001, 156762); [7]Mikel (2001, 156762); [8]Watson et al. (1999, 020949); [9]Solomon and Sioutas (2006, 156995); [10]Graney et al. (2004, 053756); [11]Tanaka et al. (1998, 157041); [12]Pancras et al. (2005, 098120); [13]John et al. (1988, 045903); [14]Hering and Cass (1999, 084958); [15]Fritz et al. (1989, 077387); [16]Hering et al. (1988, 036012); [17]Solomon et al. (2003, 156994); [18]Cabada et al. (2004, 148859); [19]Fine et al. (2003, 155775); [20]Hogrefe et al. (2004, 099003); [21]Drewnick et al. (2003, 099160); [22]Watson et al. (2005, 157125); [23]Ho et al. (2006, 156552); [24]Decesari et al. (2005, 144536); [25] Mayol-Bracero et al. (2002, 045010); [26]Yang et al. 2003; [27] Turšic et al. (2006, 157063); [28]Mader et al.(2004, 156724); [29]Xiao and Liu (2004, 056801); [30]Kiss et al. (2002, 156646); [31]Cornell and Jickells (1999, 156367); [32] Zheng et al. (2002, 026100); [33]Fraser et al. (2002, 140741); [34] Fraser et al. (2003, 042231) [35]Schauer er al. (1996, 051162); [36]Fine et al. (2004); [37]Yue et al. (2004); [38]Rinehart et al. (2006, 115184); [39]Wan and Yu (2006, 157104); [40]Poore (2000, 012839); [41]Fraser et al. (2003a); [42]Engling et al. (2006, 156422); [43]Yu et al. (2005); [44]Tran et al. (2000); [45]Yao et al. (2004, 102213); [46]Li and Yu (2005, 156692); [47]Henning et al. (2003, 156539); [48]Zhang and Anastasio (2003, 157182); [49]Emmenegger et al. (2007, 156418); [50](Watson et al., 1989, 157119); [51]Greaves et al. (1985, 156494); [52]Waterman et al. (2000, 157116); [53]Waterman et al. (2001, 157117); [54]Falkovich and Rudich (2001, 156427); [55]Chow et al. (2007, 156354); [56]Miguel et al. (2004, 123260); [57]Crimmins and Baker (2006, 097008); [58]Ho and Yu (2004, 156551); [59]Jeon et al. (2001, 016636); [60]Mazzoleni et al. (2007, 098038); [61]Poore (2000, 012839); [62]Butler et al. (2003); [63]Chow et al. (2006c); [64]Russell et al. (2004, 082453); [65]Grover et al. (2006); [66]Grover et al. (2005, 090044); [67]Schwab et al. (2006b); [68]Hauck et al. (2004); [69]Jaques et al. (2004, 155878); [70]Rupprecht and Patashnick (2003, 157207); [71]Pang et al (2002, 030353); [72]Eatough et al. (2001, 010303); [73]Lee et al. (2005b); [74]Lee et al. (2005, 156680); [75]Babich et al. (2000, 156239); [76]Lee et al. (2005c); [77]Lee et al. (2005b); [78]Anderson and Ogren (1998, 156213); [79]Chung et al. (2001, 156357); [80]Kidwell and Ondov (2004, 155898); [81]Lithgow et al. (2004, 126616); [82]Weber et al. (2003, 157129); [83]Harrison et al. (2004); [84]Rattigan et al. (2006, 115897); [85]Wittig et al. (2004, 103413); [86]Vaughn et al. (2005, 157089); [87]Chow et al. (2005b); [88]Weber et al (2001, 024640) ; [89]Schwab et al. (2006a); [90]Lim et al. (2003, 156697); [91]Watson and Chow (2002, 037873); [92]Venkatachari et al. (2006, 105918); [93]Bae et al. (2004a); [94]Arhami et al. (2006, 156224); [95]Park et al. (2005, 156843); [96]Bae et al. (2004b); [97]Chow et al. (2006a); [98]Arnott et al. (2005); [99]Bond et al. (1999); [100]Virkkula et al. (2005, 157097); [101]Petzold et al. (2002, 156863); [102]Park et al. (2006); [103]Arnott et al. (1999, 020650); [104]Peters et al. (2001); [105]Pitchford et al. (1997, 156872); [106]Rees et al. (2004, 097164); [107]Watson et al. (2000); [108]Lee et al. (2005, 156680); [109]Hering et al. (2004, 155837); [110]Watson et al. (1998); [111]Chakrabarti et al. (2004); [112]Mathai et al. (1990, 156741); [113]Kidwell and Ondov (2001, 017092); [114]Stanier et al. (2004); [115]Khlystov et al. (2005, 156635); [116]Takahama et al. (2004, 157038); [117]Chow et al. (2005, 156348); [118]Zhang et al. (2002); [119]Subramanian et al. (2004, 081203); [120]Chow et al. (2006b); [121]Birch and Cary (1996); [122]Birch (1998, 024953); [123]Birch and Cary (1996); [124]NIOSH (1996, 156810); [125]NIOSH (1999, 156811); [126]Chow et al. (1993); [127]Chow et al. (2007); [128]Ellis and Novakov (1982, 156416); [129]Peterson and Richards (2002, 156861); [130]Schauer et al. (2003); [131]Middlebrook et al. (2003, 042932); [132]Wenzel et al. (2003, 157139); [133]Jimenez et al. (2003); [134]Phares et al. (2003, 156866); [135]Qin and Prather (2006, 156895); [136]Zhang et al. (2005); [137]Bein et al. (2005, 156265); [138]Drewnick et al. (2004a); [139]Drewnick et al. (2004b); [140]Lake et al. (2003, 156669); [141]Lake et al. (2004, 088411)

Table A-15. Summary of $PM_{2.5}$ carbon measurement comparisons.

SITE/PERIOD/SAMPLER/ CONFIGURATION	SUMMARY OF FINDINGS
ATLANTA SUPERSITE, GA: 08/03/99 to 09/01/99 4 km NW of downtown, within 200 m of a bus maintenance yard and several warehouse facilities, representative of a mixed commercial-residential neighborhood.	**Solomon et al. [17]** **Organic Carbon (OC);** $PM_{2.5}$ OC from each sampler was compared to the all-sampler avg, called the relative reference (RR) value. The samplers agreed to within 20 to 50% of RR. Only front filter OC is reported without artifact correction. Denuded samplers showed lower OC (20 to 35%) than RR, while non-denuded sampler OC was higher (5 to 35%). Among non-denuded samplers, as filter face velocity decreased, OC increased, with the exception of R&P-2300.

SAMPLER	FLOW RATE (L/MIN)	FILTER TYPE[a]	DENUDER[b]	ANALYSIS METHOD[c]
R&P-2000 FRM	16.7	Quartz (P)	None	NIOSH 5040-TOT
RAAS-400	24	Quartz (P)	None	NIOSH 5040-TOT
SASS	6.7	Quartz (P)-Quartz (P)	None	NIOSH 5040-TOT
MASS-450	16.7	Quartz (P)	None	NIOSH 5040-TOT

SITE/PERIOD/SAMPLER/ CONFIGURATION					SUMMARY OF FINDINGS
R&P-2300	10	Quartz (P)-Quartz (P)	None	NIOSH 5040-TOT	OC positive artifacts ranged from 2 to 4 $\mu g/m^3$
VAPS	15	Quartz (P)	XAD-4	NIOSH 5040-TOT	**EC:**
URG-PCM	16.7	Quartz (P)-Quartz (P)	XAD-4	Front: NIOSH 5040-TOT; Backup: custom-TOT[d]	$PM_{2.5}$ EC from each sampler was compared to the all-sampler avg, called the relative reference (RR) value. The samplers agreed to within 20 to 200% of RR.
ARA-PCM	16.7	Quartz (n/a)-Quartz (n/a)	CIF	IMPROVE_TOR	TOT samples showed less EC than RR by 15 to 30%, while TOR samples showed more EC than RR by 40 to 90%. PCBOSS (BYU) > RR value by 140%. EC by TOR is ~ twice EC by TOT.
PC-BOSS (TVA)	150	Quartz (P)-CIF (n/a)	CIF	Front: IMPROVE_TOR; Backup: TPV	Major difference in EC is due to the carbon analysis protocol and optical monitoring correction (i.e., transmittance, reflectance).
PC-BOSS (BYU)	150	Quartz (P)-CIF (S)	CIF	TPB	**Lim et al.** (2003)
MOUDI-100	30	Al Foilf-Quartz (n/a	None	Custom-TOR to suit Al[c]	TC concentrations measured by the RU-OGI and R&P-5400 correlated reasonably well (R2 = 0.83), with a slope of 0.96. The ratio of the mean RU-OGI to mean R&P-5400 TC was 1.02.
CONTINUOUS SAMPLER	**FLOW RATE (L/min)**	**DENUDER**	**OC**	**EC**	**COMMENTS**
ADI-C	2.7	Activated Carbon	Not known	n/a	Part of SO_4^{2-} instrument w/CO_2 non-dispersive infrared (NDIR) analyzer; data corrected for avg field blank; OC = 2 oxidized OC
RU-OGI	16.1	None	700 in He	850 in 2% O_2	TOT; Dynamic blank for adsorption correction
R&P-5400	16.7	None	275 in air	750 in air	No pyrolysis correction
PSAP	1.26	None		b_{abs}@ 565 nm	10m^2/g factor
AE-16	4	None		b_{abs}@ 880 nm	12.6 m^2/g factor
R&P-5400 OC was 8% lower than the RU-OGI (R2 = 0.73), while the R&P-5400 EC was 20% higher than RU-OGI (R2 = 0.74).					
OC measured by ADI-C was lower than R&P-5400 and RUOGI by 15% and 22%, respectively.					
EC from PSAP and AE-16 correlated well (R2 = 0.97). PSAP was lower by ~50%, compared with AE-16, R&P-5400 and RU-OGI.					
EC measured by AE-16 was ~12% higher than RU-OGI. Calibration factors for the light absorption instruments need to be adjusted for better correlation.					
Calibration factor might be non-linear over the range of absorbance measured.					
The mean OC from R&P-5400 and RU-OGI were within 10% of filter RR values. Mean ADI-C OC was 14% lower than filter RR OC.					
EC from continuous instruments was 2-2.5 times filter RR EC; continuous TC was also greater than filter RR TC by 17% (R&P-400) to 27% (RU-OGI).					
PITTSBURGH SUPERSITE, PA; 06/01/01 to 07/31/02 Six km east of downtown in a park on the top of a hill.					**Subramanian et al.** [119]
SAMPLER	**FLOW**	**FILTER TYPE / PACK[a]**		**DENUDER**	**ANALYSIS METHOD[c]**
CMU Custom-1	16.7	*Non-denuded sample*	Teflon (P/W)-Quartz (P) (QBT)	None	NIOSH 5040-TOT
	16.7	*Non-denuded sample*	Quartz (P)-Quartz (P) (QBQ)	None	NIOSH 5040-TOT
CMU Custom-2	16.7	*Denuded sample*	Denuder-Quartz (P)-CIG (S)	Activated Carbon	NIOSH 5040-TOT
	16.7	*Dynamic blank (DYN)*	Teflon (P/W)-Denuder-Quartz (P)-CIG (S)	Activated Carbon	NIOSH 5040-TOT
Particulate OC (POC) was estimated from denuded sample (Quartz OC + CIG OC) after subtracting DYN POC.					
Denuder efficiency (1-DYN POC/UDB POC) was 94 ± 3%. No seasonal variability or deterioration in denuder performance was observed.					
Positive artifact due to denuder breakthrough was 18.3 ± 12.5% of the denuded sample POC.					
Negative artifact (CIGsample-CIGDYN) was, on avg, 6.3 ± 6.2% of POC.					
Positive artifact was 34 ± 10% from QBT, and was 13 ± 5% from QBQ. QBT > > QBQ.					

SITE/PERIOD/SAMPLER/ CONFIGURATION						SUMMARY OF FINDINGS
	16.7	*Non-denuded blank (UDB)*	Teflon (P/W)-Quartz (P)-CIG (S)	None	NIOSH 5040-TOT	QBT over-corrected the positive artifact by 20%. OC volatilization from the front Teflon filter that subsequently-adsorbed on the back-up quartz filter, resulted in an overestimation of the positive artifact. Non-denuded QBQ provided a more representative estimate of the positive artifact on the non-denuded front quartz filter for 24-h samples. However, it was not suitable for 4-6 h samples, because the filters were not in equilibrium with the air stream. Positive artifact dominated when sampling with a non-denuded quartz filter. Comparison of 24-h avg non-denuded front quartz OC versus denuded POC over the year showed an intercept of 0.53 $\mu g/m^3$, indicative of a positive artifact on quartz filter samples. The artifacts were higher in summer on an absolute basis; however, they showed no seasonal variation when expressed as a fraction of POC.

ST. LOUIS SUPERSITE, IL, MO; 01/01/02 to 12/31/02
Three km east of St. Louis, MO City center, also impacted by industrial sources, and located in a mixed residential light commercial neighborhood.

SAMPLER	FLOW RATE (Lmin)	FILTER TYPE/PACK[a]	DENUDER[b]	ANALYSIS METHOD[c]
University of Wisconsin Custom-1	24	Quartz (P)	None	ACE Asia TOT
		Denuder-Quartz (P)	CIF	ACE Asia TOT
University of Wisconsin Custom-2	24	Denuder-Quartz (P)	CIF	ACE Asia TOT
		Teflon (n/a)-Denuder-Quartz (P)	CIF	ACE Asia TOT

CONTINUOUS SAMPLER	FLOW RATE (L/min)	DENUDER	OC	EC	COMMENTS
Sunset OCEC	8	CIF	340, 500, 615, 870 °C in 100% He	550, 625, 700, 775, 850, 900 °C in 2% O_2, 98% He	ACE Asia TOT; CH_4 FID detector

Bae et al. [93, 96]

Denuder breakthrough was 0.17 ± 0.15 $\mu g/m^3$, and constituted less than 5% of annual avg OC concentration.

Non-denuded OC = (1.06 ± 0.02) × denuded OC + (0.34 ± 0.10)

Equivalence of OC intercept and denuder breakthrough implies that the low-level artifact is caused by denuder breakthrough.

Non-denuded EC = (1.04 ± 0.03) × denuded EC + (0.07 ± 0.03), indicating negligible EC artifact.

Results suggested higher summer-time OC artifact, on an absolute basis.

Comparison of continuous Sunset TC and OC with 24-h filter samples showed good correlations (R2) of 0.89 and 0.90, respectively.

Continuous Sunset TC in $\mu g/m^3$ = (0.97 ± 0.02) × filter TC + (0.83 ± 0.11), indicating comparability with the filter measurements.

Continuous Sunset OC = (0.93 ± 0.02) × filter OC + (0.94 ± 0.09)

Positive intercept was interpreted to be a blank correction for the continuous measurements.

EC comparison was poor with large scatter in data (R2 = 0.60), probably due to low EC concentrations (avg = 0.70 $\mu g/m^3$), close to the detection limit (0.5 $\mu g/m^3$).

FRESNO SUPERSITE, CA and other CRPAQS sites; 12/02/99 to 02/03/01, 12/1/03 to 11/30/04
Fresno Supersite was located 5.5 km northeast of downtown in a mixed residential-commercial neighborhood.

SAMPLER	FLOW RATE (Lmin)	FILTER TYPE/PACK[a]	DENUDER[b]	ANALYSIS METHOD[c]
DRI-SFS	113	Quartz (P)	None	IMPROVE_TOR
		Teflon (P)-Quartz	None	IMPROVE_TOR
RAAS-400	24	(P) (QBT) Quartz (P)-Quartz (P) (QBQ)	None	IMPROVE_TOR
RAAS-400	24	Quartz (P)-Quartz (P) (QBQ)	XAD-4 / CIF	IMPROVE_TOR

Watson and Chow [91]; Chow et al. [117]; Chow et al. [120]; Watson et al. [6]; Park et al. [102]

Non-denuded RAAS-400 and RAAS-100 FRM measured equivalent TC. DRI-SFS, RAAS-400 and RAAS-100 FRM samplers showed comparability for front filter TC, OC and EC measurements.

Positive OC artifact was 1.62 ± 0.58 $\mu g/m^3$ (~24% of non-denuded front quartz OC) from QBT, and 1.12 ± 0.91 $\mu g/m^3$ (~17% of non-denuded front quartz OC) from QBQ. QBT >> QBQ

Results from CRPAQS showed, on avg, a positive OC artifact of 34% (of the non-denuded front quartz OC) from QBT and 17.5% (of the non-denuded front quartz

SITE/PERIOD/SAMPLER/ CONFIGURATION						SUMMARY OF FINDINGS
RAAS-100 FRM	16.7	Quartz (P)	None		IMPROVE_TOR	OC) from QBQ. Positive artifact was higher during summer than winter. Negative artifact was, on avg, 0.61 ± 0.58 $\mu g/m^3$ (~10% of POC) at Fresno. Over all the CRPAQS sites, it ranged from 2.3% in winter to 11% in summer, with an avg of 4.9%. Positive artifact is estimated to be 0.5 $\mu g/m^3$. No difference in denuded quartz backup OC was found between using XAD and CIF denuders. Comparison of R&P-5400 TC, OC, and EC against filter samples showed poor correlation (R2 < 0.55). TC from R&P-5400 was 40–60% higher than filter TC by TOR. None of the R&P-5400 versus TOR filter comparisons were comparable or predictable, due to several frequent instrument malfunctions during the experiment and the small data set (~35 data points). IMPROVE_TOR EC was consistently 20–25% higher than aethalometer BC. IMPROVE_TOR EC was comparable to MAAP BC. Comparison of light absorption (b_{abs}) from DRI-PA (1047 nm), MAAP (670 nm), and AE (880 nm) analyzers with the filter IMPROVE_TOR EC, gave a σabs of 2.3, 5.5 and 10 m^2/g, differing from the default conversion factors of 5, 6.5, and 16.6 m^2/g used for each instrument at the specified wavelength. **Grover et al. [65]** R&P-5400 TC = (0.50 ± 0.01) Sunset TC + (3.6 ± 1.5); R2 = 0.73; n = 480 Sunset TC = (0.63 ± 0.05) PC-BOSS TC + (4.1 ± 3.2); R2 = 0.86; n = 29 R&P-5400 TC = (0.41 ± 0.02) PC-BOSS TC + (6.7 ± 1.6); R2 = 0.91; n = 29

CONTINUOUS SAMPLER	FLOW RATE (L/min)	DENUDER	OC	EC	COMMENTS
R&P-5400	16.7	None	275 °C in air	750 °C in air	No pyrolysis correction
Sunset OCEC	8.5	CIG	250, 500, 650, 850 °C in He	650, 750, 850, 940 °C in 2% O_2 in He	Transmittance
MAAP	16.7	None		b_{abs} @ 670 nm	Transmittance 6.5 m^2/g factor
AE-16	6.8	None		b_{abs} @ 880 nm	Transmittance 14625/λ m^2/g factor, where λ is in nm
AE-21	6.8	None		b_{abs} @ 370, 880 nm	
AE-31	6.8	None		b_{abs} @ 370, 470, 520, 590, 660, 880 and 950 nm	
DRI-PA	3	None		b_{abs} @ 1047 nm	Absorption, 5 m^2/g factor

SAMPLER	FLOW RATE (L/min)	FILTER TYPE/PACK[a]	DENUDER[b]	ANALYSIS METHOD[c]
PC-BOSS	150	Quartz (P)-CIG (S)[†]	CIF	TPV

CONTINUOUS SAMPLER	FLOW RATE (L/min)	DENUDER[b]	OC	EC	COMMENTS
R&P-5400	16.7	None	375 °C in air	750 °C in air	No pyrolysis
Sunset OCEC	8.0	CIG	250, 500, 650, 850 °C in He	650, 750, 850 °C in 2% O_2 & 98% He	NIOSH 5040_TOT NDIR CO_2 detector

BALTIMORE SUPERSITE, MD; 02/15/2002 to 11/30/2002
East of downtown in an urban residential area. Within 91 m of bus maintenance facility.

Park et al. [95]

Data capture 93.8%

Compared to SASS, Sunset underestimated OC and EC by 22% and ~11.5%, respectively.

Higher OC in SASS was attributed to the absence of a denuder (i.e., positive artifact by gaseous adsorption) and to temperature differences between the STN_TOT and Sunset_TOT carbon analysis temperature protocols.

EC discrepancy was probably related to the differences

SAMPLER	FLOW RATE (L/min)	FILTER TYPE/PACK[a]	DENUDER[b]	ANALYSIS METHOD[c]
SASS	6.7	Quartz (P)-Quartz (P)	None	STN_TOT

CONTINUOUS SAMPLER	FLOW RATE (L/min)	DENUDER[b]	OC	EC	COMMENTS

SITE/PERIOD/SAMPLER/ CONFIGURATION							SUMMARY OF FINDINGS
Sunset OCEC	8	Carbon	600 °C, then 870 °C in He	870 °C in 2% O_2 in He	TOT; CH4 FID detector; Denuder breakthrough ~ 0.5 – 1 μg C/m^3; Used 0.5 to correct OC concentrations		in temperature protocol.

RUBIDOUX, CA; 07/13/03 to 07/26/03

Rubidoux is located in the eastern section of the South Coast Air Basin (SoCAB) in the north-west corner of Riverside County, 78 km downwind of the central Los Angeles metropolitan area and in the middle of the remaining agricultural production area in SoCAB.

SAMPLER	FLOW RATE (L/min)	FILTER TYPE/PACK[a]	DENUDER[b]	ANALYSIS METHOD[c]
PC-BOSS	150	Quartz (P)-CIG (S)	CIF	TPB (CIG heated to 450 °C in N_2)

CONTINUOUS SAMPLER	FLOW RATE (L/min)	DENUDER[b]	OC	EC	COMMENTS
Sunset OCEC	8	CIF	n/a	n/a	TOT; NDIR detector; NIOSH 5040 protocol
Sunset OCEC	8	CIF	n/a	Not measured	TOT; has blank quartz filter before entering analyzer. Used as "blank" stream for quantifying OC artifacts; 3-step analysis only in He.

Grover et al. [66]

Sunset OCEC TC = (0.90 ± 0.06) PC-BOSS + (2.0 ± 2.1); R2 = 0.93; n = 21

Sunset TC was adjusted for carbon artifacts measured by second (blank) instrument.

NEW YORK SUPERSITE, NY; 01/12/04 to 02/05/04

Urban site located at Queens College, NY, about 14 km west of Manhattan, within 2 km of freeways, and within 12 km of international airports.

INTEGRATED SAMPLER	FLOW RATE (L/min)	FILTER TYPE/PACK[a]	DENUDER[b]	ANALYSIS METHOD[c]
R&P-2300	10	Quartz	None	STN_TOT

CONTINUOUS SAMPLER	FLOW RATE (L/min)	DENUDER[b]	OC	EC	COMMENTS
R&P-5400	16.7	None	340 °C in air	750 °C in air	No pyrolysis correction
Sunset OCEC	n/a	CIF	600, 870 °C in He	870 °C at 10% O_2 in He	Transmittance
AE-20	n/a	None		b_{abs} @ 370, 880 nm	Transmittance, 14625/ m^2/g factor, where is in nm
AMS	n/a	None	n/a	n/a	~ 1 µm cut-point

Venkatachari et al. [92]

Regression of OC from Sunset OCEC against $PM_{2.5}$ mass concentration yielded an intercept of 1.14 µg/m^3, which was used as a measure of the positive artifact on the Sunset data. The Sunset OC data was corrected for this artifact.

AE-20 BC concentrations were ~86% of Sunset EC and R&P2300 filter EC concentrations.

AE-20 versus R&P-5400 showed high scatter.

Sunset Optical EC = 0.58 ± 0.05 Sunset Thermal EC; R2 = 0.86; n = 506

Sunset Optical EC = 0.62 ± 0.05 AE-20 BC; R2 = 0.96; n = 539

R&P-5400 TC tracked filter TC closely, but differed widely for OC and EC.

Sunset OC = (0.75 ± 0.76) R&P-2300 OC +

(0.08 ± 0.36); R2 = 0.67; n = 16

Sunset OC = (0.98 ± 0.11) R&P-5400 OC - (0.47 ± 0.17); R2 = 0.44; n = 327

R&P-5400 OC = (0.60 ± 0.47) R&P-2300 OC + (0.58 ± 0.82); R2 = 0.58; n = 17

RUBIDOUX, CA; 07/13/03 to 07/26/03

Rubidoux is located in the eastern section of the South Coast Air Basin (SoCAB) in the north-west corner of Riverside County, 78 km downwind of the central Los Angeles metropolitan area and in the middle of the remaining agricultural production area in SoCAB.

Organic matter measurements by AMS showed reasonable correlation (R2 = 0.76) with filter (R&P-2300) OC, while being poorly correlated with continuous OC by Sunset (R2 = 0.32) and R&P-5400 (R2 = 0.36)

Sunset EC = (1.21 ± 0.44) R&P-2300 EC – (0.03 ± 0.13); R2 = 0.94; n = 16
Sunset EC = (1.35 ± 0.12) R&P-5400 EC + (0.06 ± 0.04); R2 = 0.61; n = 327
R&P-5400 EC = (0.49 ± 0.46) R&P-2300 EC + (0.09 ± 0.26); R2 = 0.77; n = 15
Sunset TC = (0.86 ± 0.39) R&P-2300 TC – (0.06 ± 0.69); R2 = 0.77; n = 16
Sunset TC = (1.31 ± 0.10) R&P-5400 TC – (1.15 ± 0.15); R2 = 0.59; n = 327
R&P-5400 TC = (0.77 ± 0.58) R&P-2300 TC + (0.35 ± 1.37); R2 = 0.83; n = 16

[a]Filter Manufacturer in parentheses - W: Whatman, Clifton, NJ; P: Pall-Gelman, Ann Arbor, MI; S: Schleicher & Schnell. Keene, NH; n/a: not available. QBT: quartz backup filter behind Teflon front filter. QBQ: quartz backup filter behind Quartz front filter.

[b]Al_2O_3: Aluminum oxide; IC: Ion chromatography; CIF: Charcoal Impregnated Filter; CIG: Charcoal Impregnated Glass-Fiber Filter; FEP: Fluorinated Ethylene Propylene copolymer; MgO: Magnesium oxide; Na_2CO_3: Sodium carbonate; $NaHCO_3$: Sodium bicarbonate NO_x: Oxides of nitrogen; SO_2: Sulfur dioxide; TEA: Triethanolamine; TSP: Total Suspended PM; UV: Ultraviolet; XAD-4: (hydrophobic, non-polar polyaromatic resin.

[c]NIOSH 5040_TOT: National Institute of Occupational Safety and Health Method 5040 Thermal Optical Transmittance Protocol. [121, 122, 123, 124, 125] OC: 250, 500, 650, 850 °C for OC1, OC2, OC3, and OC4 fractions, respectively, for 60, 60, 60, 90 sec respectively, in 100% He atmosphere. EC: 650, 750, 850, 940 °C for EC1, EC2, EC3, and EC4 fractions, respectively, 30, 30, 30, > 120 sec respectively, in 98% He and 2% O_2 atmosphere. OPT: Pyrolysis correction by transmittance. IMPROVE_TOR: Interagency Monitoring of Protected Visual Environments Thermal Optical Reflectance Protocol. [126] OC fractions: 120, 250, 450, 550 °C for OC1, OC2, OC3, and OC4 fractions, respectively, until a well defined peak has evolved at each step, with a time limit of min 80 sec and max of 580 sec, in 100% He atmosphere. EC fractions: 550, 700, 800 °C for EC1, EC2, and EC3 fractions, respectively, until a well defined peak has evolved at each step, with a time limit of min 80 sec and max of 580 sec, in 2% O_2 and 98% He atmosphere. OPR: Pyrolysis correction for pyrolyzed organic carbon (OP) by reflectance. OC = OC1+OC2+OC3+OC4+OP EC = EC1+EC2+EC3-OP TC = OC+EC. IMPROVE_A TOR: [127] Note that as of May, 2007, the U.S. EPA is switching samples from the Speciation Trends Network thermal optical transmittance protocol to the IMPROVE_A protocol. OC: 140, 280, 480, 580 °C for OC1, OC2, OC3, and OC4, fractions, respectively, until a well defined peak has evolved at each step, with a time limit of 80 sec and max of 580 sec, in 100% He atmosphere EC: 580, 740, 840 °C for EC1, EC2, and EC3 fractions, respectively, until a well defined peak has evolved at each step, with a time limit of min 80 sec and max of 580 sec, in 2% O_2 and 98% He atmosphere. OPR: Pyrolysis correction for pyrolyzed organic carbon (OP) by reflectance. OPT: Pyrolysis correction by transmittance. TPV: Temperature Programmed Volatilization. [17, 81, 128] For CIF Filters: Heated from 50 °C to 300 °C at a ramp rate of 10 °C/min in N_2. For Quartz filters: Heated from 50 °C to 800 °C at a ramp rate of 28 °C/min in 70% N_2 and 30% O_2; EC estimated from high temperature peak (> 450 °C) on thermogram obtained from quartz-fiber filter analysis; No pyrolysis correction. STN_TOT: Speciation Trends Network Thermal Optical Transmittance Protocol.[129] OC: 310, 480, 615, 920 °C for 60, 60, 60, 90 sec respectively, in 100% He atmosphere. EC: 600, 675, 750, 825, 920 °C for 45, 45, 45, 45, 120 sec respectively, in 98% He and 2% O_2 atmosphere. ACE Asia TOT: Aerosol Characterization Experiments in Asia Thermal Optical Transmittance Protocol. [130] OC: 340, 500, 615, 870 °C for 60, 60, 60, 90 sec respectively, in 100% He atmosphere. EC: 550, 625, 700, 775, 850, 900 °C for45, 45, 45, 45, 45, 120 sec respectively, in 98% He, 2% O_2. Pyrolysis correction by transmittance.

[d]Custom TOT: XAD-4 impregnated quartz, analyzed in He-only atmosphere with a maximum temperature 176 °C; EC is not measured.

[e]Custom TOR to suit Al substrate; details not reported.

[f]37-mm filter

Source: [1]Chow (1995, 077012); [2]Watson and Chow (2001, 157123); [3] Watson et al. (1983, 045084); [4]Fehsenfeld et al. (2004, 156432); [5]Solomon et al. (2001, 156993); [6]Mikel (2001, 156762); [7]Mikel (2001, 156762); [8]Watson et al. (1999, 020949); [9]Solomon and Sioutas (2006, 156995); [10]Graney et al. (2004, 053756); [11]Tanaka et al. (1998, 157041); [12]Pancras et al. (2005, 098120); [13]John et al. (1988, 045903); [14]Hering and Cass (1999, 084958); [15]Fritz et al. (1989, 077387); [16]Hering et al. (1988, 036012); [17]Solomon et al. (2003, 156994); [18]Cabada et al. (2004, 148859); [19]Fine et al. (2003, 155775); [20]Hogrefe et al. (2004, 099003); [21]Drewnick et al. (2003, 099160); [22]Watson et al. (2005, 157125); [23]Ho et al. (2006, 156552); [24]Decesari et al. (2005, 144536); [25] Mayol-Bracero et al. (2002, 045010); [26]Yang et al. 2003; [27] Turšic et al. (2006, 157063); [28]Mader et al.(2004, 156724); [29]Xiao and Liu (2004, 056801); [30]Kiss et al. (2002, 156646); [31]Cornell and Jickells (1999, 156367); [32] Zheng et al. (2002, 026100); [33]Fraser et al. (2002, 140741); [34] Fraser et al. (2003, 042231) [35]Schauer er al. (1996, 051162); [36]Fine et al. (2004); [37]Yue et al. (2004); [38]Rinehart et al. (2006, 115184); [39]Wan and Yu (2006, 157104); [40]Poore (2000, 012839); [41]Fraser et al. (2003a); [42]Engling et al. (2006, 156422); [43]Yu et al. (2005); [44]Tran et al. (2000); [45]Yao et al. (2004, 102213); [46]Li and Yu (2005, 156692); [47]Henning et al. (2003, 156539); [48]Zhang and Anastasio (2003, 157182); [49]Emmenegger et al. (2007, 156418); [50](Watson et al., 1989, 157119); [51]Greaves et al. (1985, 156494); [52]Waterman et al. (2000, 157116); [53]Waterman et al. (2001, 157117); [54]Falkovich and Rudich (2001, 156427); [55]Chow et al. (2007, 156354); [56]Miguel et al. (2004, 123260); [57]Crimmins and Baker (2006, 097008); [58]Ho and Yu (2004, 156551); [59]Jeon et al. (2001, 016636); [60]Mazzoleni et al. (2007, 098038); [61]Poore (2000, 012839); [62]Butler et al. (2003); [63]Chow et al. (2006c); [64]Russell et al. (2004, 082453); [65]Grover et al. (2006); [66]Grover et al. (2005, 090044); [67]Schwab et al. (2006b); [68]Hauck et al. (2004); [69]Jaques et al. (2004, 155878); [70]Rupprecht and Patashnick (2003, 157207); [71]Pang et al (2002, 030353); [72]Eatough et al. (2001, 010303); [73]Lee et al. (2005b); [74]Lee et al. (2005, 156680); [75]Babich et al. (2000, 156239); [76]Lee et al. (2005c); [77]Lee et al. (2005b); [78]Anderson and Ogren (1998, 156213); [79]Chung et al. (2001, 156357); [80]Kidwell and Ondov (2004, 155898); [81]Lithgow et al. (2004, 126616); [82]Weber et al. (2003, 157129); [83]Harrison et al. (2004); [84]Rattigan et al. (2006, 115897); [85]Wittig et al. (2004, 103413); [86]Vaughn et al. (2005, 157089); [87]Chow et al. (2005b); [88]Weber et al (2001, 024640) ; [89]Schwab et al. (2006a); [90]Lim et al. (2003, 156697); [91]Watson and Chow (2002, 037873); [92]Venkatachari et al. (2006, 105918); [93]Bae et al. (2004a); [94]Arhami et al. (2006, 156224); [95]Park et al. (2005, 156843); [96]Bae et al. (2004b); [97]Chow et al. (2006a); [98]Arnott et al. (2005); [99]Bond et al. (1999); [100]Virkkula et al. (2005, 157097); [101]Petzold et al. (2002, 156863); [102]Park et al. (2006); [103]Arnott et al. (1999, 020650); [104]Peters et al. (2001); [105]Pitchford et al. (1997, 156872); [106]Rees et al. (2004, 097164); [107]Watson et al. (2000); [108]Lee et al. (2005, 156680); [109]Hering et al. (2004, 155837); [110]Watson et al. (1998); [111]Chakrabarti et al. (2004); [112]Mathai et al. (1990, 156741); [113]Kidwell and Ondov (2001, 017092); [114]Stanier et al. (2004); [115]Khlystov et al. (2005, 156635); [116]Takahama et al. (2004, 157038); [117]Chow et al. (2005, 156348); [118]Zhang et al. (2002); [119]Subramanian et al. (2004, 081203); [120]Chow et al. (2006b); [121]Birch and Cary (1996); [122]Birch (1998, 024953); [123]Birch and Cary (1996); [124]NIOSH (1996, 156810); [125]NIOSH (1999, 156811); [126]Chow et al. (1993); [127]Chow et al. (2007); [128]Ellis and Novakov (1982, 156416); [129]Peterson and Richards (2002, 156861); [130]Schauer et al. (2003); [131]Middlebrook et al. (2003, 042932); [132]Wenzel et al. (2003, 157139); [133]Jimenez et al. (2003); [134]Phares et al. (2003, 156866); [135]Qin and Prather (2006, 156895); [136]Zhang et al. (2005); [137]Bein et al. (2005, 156265); [138]Drewnick et al. (2004a); [139]Drewnick et al. (2004b); [140]Lake et al. (2003, 156669); [141]Lake et al. (2004, 088411)

Table A-16. Summary of particle mass spectrometer measurement comparisons.

Spectrometer	Inlet Characteristics[a] (Flow Rate [L/Min] Size Inlet Dryer Aerodynamic Diameter, μm Particle Sizing Method)	Volatilization/ Ionization Method[a]	Hit Rates[b]	Mass Spectrometer[c]	Particle Analysis/ Classification	Other
PALMS	n/a $PM_{2.5}$ cyclone Nafion (17 days) / None (4 days) 0.35 - 2.5 Light scattering	LDI, ArF 193 nm 2x109 to 5x109 W/cm2	14 to 100%, overall 87%	Single TOF reflectron; Ion polarity needs to be pre-selected	Peak ID/regression tree analysis	Pure sulfuric acid ($H2SO_4$), $(NH_4)_2SO_4$, and water (H_2O) have relatively high ionization thresholds (i.e. difficult to ionize). Fraction of molecules ionized in the particles is on the order of 10^{-5} to 10^{-6}.
ATOFMS	1 None None 02 – 2.5 Aerosol TOF	LDI, Nd: YAG 266 nm laser ~ 1x108 W/cm2	25-30%, occasionally as low as 5%	Dual TOF reflectron; Detects both positive and negative ions	Aerosol TOF	
RSMS-II	n/a None Nafion 0.015 - 1.3 Aerodynamic focusing Need to pre-select sizes to be analyzed	LDI, Arf laser, 193 nm 1x108 to 2x108 W/cm2	n/a	Single linear TOF; Ion polarity needs to be pre-selected	Peak ID/artificial neural network	
AMS	n/a $PM_{2.5}$ cyclone None 0.05 - 2.5 Aerosol TOF	T ~ 550 °C/ EI	n/a	Quadrupole; Mass weighted size distributions on pre-selected positive ions only.	ID using standard EI ionization databases	Does not detect/ analyze highly refractory materials such as metals, sea salt, soot etc. Fraction of molecules ionized in the particles is on the order of 10^{-6} to 10^{-7}

Middlebrook et al. [131]; Wenzel et al. [132]; Jimenez et al.[133]

Particle sizing is approximate in PALMS, while ATOFMS, RSMS-II and AMS provide relatively accurate particle sizing.

Particle transmission in AMS is ~100% (i.e., it uses all particles in the sampled air) between 60 and 600 nm, while that for PALMS, ATOFMS and RSMS-II range from 10^{-6} for submicron particles to 2% for supermicron (>0.8 μm) particles.

AMS has fewer matrix effects (due to separate volatilization and ionization steps) compared to single-step LDI instruments.

While four major particle classifications (organic/ SO_4^{2-}, sodium/potassium sulfate, soot/hydrocarbon and mineral) were observed by all three laser instruments, they differed in the classification frequencies. Differences in frequencies that are detected and grouped are related to the differences in the laser ionization conditions (e.g., wavelength), particle transmission, sizing method and the way the spectra were classified.

Shorter ionization wavelengths are able to produce ions more easily than longer ones.

Low hit rates in ATOFMS corresponded to periods of high SO_4^{2-} concentrations. Low hit rates in PALMS were related to a variety of factors including high SO_4^{2-} concentrations, differing laser fluence and laser position relative to particle beam. Use of a dryer in PALMS enhanced ionization of particles that were difficult to ionize at high ambient RH.

The RSMS-II and ATOFMS were less sensitive to SO_4^{2-} and hence may have fewer organic/SO_4^{2-} particles (i.e., underestimate SO_4^{2-}, pure sulfuric acid etc.).

The PALMS, ATOFMS and RSMS (laser based instruments) are qualitative, while the AMS can be quantitative. The relative ratio of ion intensities from the laser instruments, however, may be indicative of relative concentrations, thus giving semi-quantitative information.

Comparison of the ratio of NO_3 to SO_4 peaks with the results from the semi continuous instruments showed better correlation with the AMS (R2 = 0.93) than PALMS (R2 = 0.65 for non-dry particles to 0.70 for dry particles). While reasonable correlations between the PALMS and the composite semi-continuous data indicate the possibility for calibration of laser-based data for certain ions, the calibration factors may vary depending on the particle matrix, water content and laser ionization parameters, and averaging the spectra according to these factors may minimize these effects.

Comparison of AMS SO_4 with PILS SO_4 showed good correlation (R2 = 0.79), and the data uniformly scattered around a 1:1 line. NO_3 comparison was poor (R2 = 0.49) because of the low signal to noise ratio at low concentrations

The continuum between particle classifications indicates that the particles were not adequately represented by non-overlapping classifications.

Spectrometer	Inlet Characteristics[a] (Flow Rate [L/Min] Size Inlet Dryer Aerodynamic Diameter, μm Particle Sizing Method)	Volatilization/ Ionization Method[a]	Hit Rates[b]	Mass Spectrometer[c]	Particle Analysis/ Classification	Other
HOUSTON SUPERSITE, TX; 08/23/00 to 09/18/00 Houston Regional Monitoring Site was located < 1.0 km north of the Houston ship channel, where chemical and other industries are present. The site was located between a railway to the south and a chemical plant to the north. Major freeways were located just to the north and east of the sampling site.						
SPECTROMETER	INLET CHARACTERISTICS (FLOW RATE [L/MIN] SIZE INLET DRYER AERODYNAMIC DIAMETER, μM PARTICLE SIZING METHOD)	VOLATILIZATION/ IONIZATION METHOD[a]	HIT RATES[b]	MASS SPECTROMETER[c]	PARTICLE ANALYSIS/ CLASSIFICATION	OTHER
RSMS-II	n/a None Nafion 0.035 – 1.14 Aerodynamic focusing; Need to pre-select sizes to be analyzed	LDI, ArF laser, 193 nm	n/a	Single linear TOF; Ion polarity needs to be pre-selected	Peak ID/artificial neural network	At each size point, aerosol was sampled in each cycle for either 10 min or until mass spectra for 30 particles per major class were collected, whichever came first.
Phares et al. [134] 27,000 spectra were classified using a neural network into 15 particle types Fifteen particle type mass spectra were presented along with their size distribution, avg time of day occurrence, and wind direction dependence Major classes were a K^+ dominant, Si/Silicon Oxide, Carbon, Sea Salt, Fe, Zn, Amines, Lime, Vanadium, Organic Mineral, Pb and K, Al, and a Pb salt particle type.						
FRESNO SUPERSITE, CA: 11/30/00 to 2/4/01 Urban location in a residential neighborhood.						
SPECTROMETER	INLET CHARACTERISTICS (FLOW RATE [L/MIN] SIZE INLET DRYER AERODYNAMIC DIAMETER, μM PARTICLE SIZING METHOD)	VOLATILIZATION/ IONIZATION METHOD[a]	HIT RATES[b]	MASS SPECTROMETER[c]	PARTICLE ANALYSIS/ CLASSIFICATION	OTHER
ATOFMS	1 None None 0.3 – 2.5 Aerodynamic	LDI, ND: YAG 266 nm	n/a	Dual reflectron TOF	Peak ID/artificial neural network	ATOFMS unscaled detected particles tracked B attenuation monitor $PM_{2.5}$ mass concentration
Qin and Prather [135] Biomass burning particles reached a maximum at night and a minimum during the day. These particles were less than 1 μm in diameter and accounted for more than 60% of the particles detected at night. Another particle class characterized by high mass carbon fragments had a similar diurnal pattern. These particles were larger than 1 μm and were interpreted as biomass particles that have undergone gas to particle conversion of semi-volatile species followed by dissolution in a water droplet.						
PITTSBURGH SUPERSITE, PA; 09/07/02 TO 09/22/02 FOR AMS; 09/20/01 to 09/26/02 for RSMS-III 6 km east of downtown in a park on the top of a hill						

Spectrometer	Inlet Characteristics[a] (Flow Rate [L/Min] Size Inlet Dryer Aerodynamic Diameter, μm Particle Sizing Method)	Volatilization/ Ionization Method[a]	Hit Rates[b]	Mass Spectrometer[c]	Particle Analysis/ Classification	Other
SPECTROMETER	INLET CHARACTERISTICS (FLOW RATE [L/MIN] SIZE INLET DRYER AERODYNAMIC DIAMETER, μM PARTICLE SIZING METHOD)	VOLATILIZATION/ IONIZATION METHOD[a]		MASS SPECTROMETER[c]	OTHER	
AMS	1.4 cc/s $PM_{2.5}$ cyclone None 0.05 – 1.0 Aerosol TOF	T - 600 °C/ EI		Quadrupole; Mass weighted size distributions on pre-selected positive ions only.	Particle size-cut of ~ 1 μm	
RSMS-III	n/a None Nafion 0.03 – 1.1 Aerodynamic focusing; Need to pre-select sizes to be analyzed.	LDI, ArF laser, 193 nm		Dual TOF feflectron; Detects both positive and negative ions	At each size point, aerosol was sampled in each cycle for either 10 min or until mass spectra for 30 particles per major class were collected, whichever came first	

Zhang et al.[136]; Bein et al.[137]

The AMS observed 75% of the SO_4^{2-} measured by R&P-8400S (R2 = 0.69).

Collection efficiency (CE) of 0.5 used for SO_4^{2-}, NO_3 and NH_4^+ and 0.7 for organics to correct mass concentrations for incomplete detection. Use of a constant CE irrespective of size and shape may overestimate accumulation mode (mostly, oxygenated) organics (true CE ~ 0.5) and underestimate smaller mode (primary) organics (true CE ~ 1.0).

Comparison of AMS organics (organic matter, OM) with OC measured by a continuous Sunset OCEC instrument showed good correlation (R2 = 0.88) with a slope of 1.69. A 24-h avg comparison, showed a slope of 1.45. These values are in the typical range of 1.2 to 2.0 for OM/OC ratios.

AMS could be used along with the SMPS to estimate particle density. The AMS did not always agree with SMPS, probably due to non-spherical particles (irregular) such as soot from fresh traffic emissions, whose mass may be overestimated by the SMPS.

Comparison of AMS mass with the MOUDI, showed differences for aerodynamic diameters > 600 nm, probably due to the AMS transmission being less than unity for particles larger than 600 nm.

For RSMS-III, 54% of the detected particles were assigned to one class (carbonaceous ammonium nitrate). This class was preferentially detected during the colder months and was detected from many different wind directions.

The next largest RSMS-III class was EC/OC/K class at 11%, and is believed to be from biomass burning.

An unidentified organic carbon RSMS-III class (3.3% of all detected particles) was seen to be highly dependent on wind direction dependence and was primarily detected during August and September of 2002. These particles likely originated from a landfill.

NEW YORK SUPERSITE; 06/30/01 to 08/05/01 (urban); 07/09/02 to 08/07/02: (rural)

Urban Site: Queens College, Queens, New York, located at the edge of a parking lot and within 1 km from expressways and highways in New York City Metropolitan area.

Rural Site: Whiteface Mountain, New York, located in a cleared area surrounded by mix of deciduous and evergreen trees, ~2 km away from the closest highway with no major cities within 20 km.

SPECTROMETER	INLET CHARACTERISTICS (FLOW RATE [L/MIN] SIZE INLET DRYER AERODYNAMIC DIAMETER, μM PARTICLE SIZING METHOD)	VOLATILIZATION/ IONIZATION METHOD[a]	MASS SPECTROMETER[c]	OTHER
AMS	0.1 $PM_{2.5}$ cyclone None 0.02 – 2.5 Aerosol TOF	T – 700 °C/ EI	Quadrupole; Mass weighted size distributions on pre-selected positive ions only.	Data are 10-min averages

Spectrometer	Inlet Characteristics[a] (Flow Rate [L/Min] Size Inlet Dryer Aerodynamic Diameter, μm Particle Sizing Method)	Volatilization/ Ionization Method[a]	Hit Rates[b]	Mass Spectrometer[c]	Particle Analysis/ Classification	Other

Drewnick et al. [138, 139]; Hogrefe et al. [20]

Transport losses were 1.3% on avg.

Inlet losses (at the inlet of AMS) were 1.9%, on avg, ranging from 11% for a 20 nm particle to 9% for a 2.5 μm particle, with a minimum of 0.7% for a 350 nm particle

Overall measurement uncertainty of particle diameter was ~11%.

The AMS was reliable with proper calibration, care, and maintenance. Valid 10 min averages were obtained for all components more than 93% of the time.

The mass to charge ratios (m/z) of fragments from different components may overlap (e.g., NH+, a fragment of NH_4^+ and CH_3^+, a fragment of organic species, have m/z = 15) resulting in an interference (called as isobaric interference) Interfering signals were not used to calculate concentrations. This loss in concentration was adjusted by applying a correction factor determined from laboratory studies.

Typical interferences were from fragments of organic species, water and oxygen.

With adjustments, the SO_4^{2-}, NO_3^-, and ammonium concentrations measured by the AMS were consistently lower than that measured by other co-located instruments, probably due to incomplete focusing of the $(NH_4)_2SO_4$ and NH_4NO3 particles by the aerodynamic lens.

At the urban site, AMS NO_3 was within 10% of the filter NO_3 concentration. At the rural site, it had a slope of 0.51 and R2 of 0.46.

AMS SO_4 showed good agreement with R&P-8400S at both the rural and urban locations (R2 = 0.89 to 0.92, slope = 0.99, n = 407 to 695) and was within 70 to 85% of filter SO_4^{2-} concentration.

Comparison of the total non-refractory mass measured by the AMS with the $PM_{2.5}$ TEOM mass (operated at 50 °C or with dryer) at the urban location, showed good correlation (R2 = 0.91) with near zero intercept (0.22 $\mu g/m^3$). On avg, the AMS observed 64% of the mass measured by the TEOM.

The unexplained mass (36%) was attributed to transport losses, transmission and optical losses, and refractory components in the aerosol sample (e.g., metals, EC). The mass closure was within the estimated uncertainty of the AMS mass measurements (5 to 10%).

BALTIMORE SUPERSITE, MD; 04/01/02 to 11/30/02

East of downtown in an urban residential area. Within 91 m of a bus maintenance facility.

SPECTROMETER	INLET CHARACTERISTICS (FLOW RATE [L/MIN] SIZE INLET DRYER AERODYNAMIC DIAMETER, μM PARTICLE SIZING METHOD)	VOLATILIZATION/ IONIZATION METHOD[a]	MASS SPECTROMETER[c]	OTHER
RSMS-III	0.2 – 18, based on particle size chosen None Nafion 0.045 – 1.3 Aerodynamic focusing; Need to pre-select sizes to be analyzed	LDI, ArF laser, 193 nm	TOF with dual ion polarity	At each size set point, aerosol was sampled in each cycle for either 10 min or until mass spectra from 30 particles were collected, whichever came first.

Lake et al. [140, 141]

Utilizing both positive and negative ion detection enables detection of more species. However, detection efficiencies of negative ions decreased for smaller particles.

SO_4^+ concentration (number or mass) was not accurately quantified.

RSMS-III was most efficient in 0.050 to 0.77 μm range.

Particle compositions could be related to specific source categaories.

Table A-17. Summary of particle mass spectrometer measurement comparisons.

Spectrometer	Inlet Characteristics[a] (Flow Rate [L/Min] Size Inlet Dryer Aerodynamic Diameter, μm Particle Sizing Method)	Volatilization/ Ionization Method[a]	Hit Rates[b]	Mass Spectrometer[c]	Particle Analysis/ Classification	Other
PALMS	n/a $PM_{2.5}$ cyclone Nafion (17 days) / None (4 days) 0.35 - 2.5 Light scattering	LDI, ArF 193 nm 2x109 to 5x109 W/cm2	14 to 100%, overall 87%	Single TOF reflectron; Ion polarity needs to be pre-selected	Peak ID/regression tree analysis	Pure sulfuric acid (H_2SO_4), $(NH_4)_2SO_4$, and water (H_2O) have relatively high ionization thresholds (i.e. difficult to ionize). Fraction of molecules ionized in the particles is on the order of 10^{-5} to 10^{-6}.
ATOFMS	1 None None 02 – 2.5 Aerosol TOF	LDI, Nd: YAG 266 nm laser $\sim$ 1x108 W/cm2	25-30%, occasionally as low as 5%	Dual TOF reflectron; Detects both positive and negative ions	Aerosol TOF	
RSMS-II	n/a None Nafion 0.015 - 1.3 Aerodynamic focusing Need to pre-select sizes to be analyzed	LDI, Arf laser, 193 nm 1x108 to 2x108 W/cm2	n/a	Single linear TOF; Ion polarity needs to be pre-selected	Peak ID/artificial neural network	
AMS	n/a $PM_{2.5}$ cyclone None 0.05 - 2.5 Aerosol TOF	T $\sim$ 550 °C/ EI	n/a	Quadrupole; Mass weighted size distributions on pre-selected positive ions only.	ID using standard EI ionization databases	Does not detect/ analyze highly refractory materials such as metals, sea salt, soot etc. Fraction of molecules ionized in the particles is on the order of 10^{-6} to 10^{-7}

Middlebrook et al. [131]; Wenzel et al. [132]; Jimenez et al.[133]

Particle sizing is approximate in PALMS, while ATOFMS, RSMS-II and AMS provide relatively accurate particle sizing.

Particle transmission in AMS is $\sim$100% (i.e., it uses all particles in the sampled air) between 60 and 600 nm, while that for PALMS, ATOFMS and RSMS-II range from 10^{-6} for submicron particles to 2% for supermicron (>0.8 μm) particles.

AMS has fewer matrix effects (due to separate volatilization and ionization steps) compared to single-step LDI instruments.

While four major particle classifications (organic/ SO_4^{2-}, sodium/potassium sulfate, soot/hydrocarbon and mineral) were observed by all three laser instruments, they differed in the classification frequencies. Differences in frequencies that are detected and grouped are related to the differences in the laser ionization conditions (e.g., wavelength), particle transmission, sizing method and the way the spectra were classified.

Shorter ionization wavelengths are able to produce ions more easily than longer ones.

Low hit rates in ATOFMS corresponded to periods of high SO_4^{2-} concentrations. Low hit rates in PALMS were related to a variety of factors including high SO_4^{2-} concentrations, differing laser fluence and laser position relative to particle beam. Use of a dryer in PALMS enhanced ionization of particles that were difficult to ionize at high ambient RH.

The RSMS-II and ATOFMS were less sensitive to SO_4^{2-} and hence may have fewer organic/SO_4^{2-} particles (i.e., underestimate SO_4^{2-}, pure sulfuric acid etc.).

The PALMS, ATOFMS and RSMS (laser based instruments) are qualitative, while the AMS can be quantitative. The relative ratio of ion intensities from the laser instruments, however, may be indicative of relative concentrations, thus giving semi-quantitative information.

Comparison of the ratio of NO_3 to SO_4 peaks with the results from the semi continuous instruments showed better correlation with the AMS (R2 = 0.93) than PALMS (R2 = 0.65 for non-dry particles to 0.70 for dry particles). While reasonable correlations between the PALMS and the composite semi-continuous data indicate the possibility for calibration of laser-based data for certain ions, the calibration factors may vary depending on the particle matrix, water content and laser ionization parameters, and averaging the spectra according to these factors may minimize these effects.

Comparison of AMS SO_4 with PILS SO_4 showed good correlation (R2 = 0.79), and the data uniformly scattered around a 1: 1 line. NO_3 comparison was poor (R2 = 0.49) because of the low signal to noise ratio at low concentrations

The continuum between particle classifications indicates that the particles were not adequately represented by non-overlapping classifications.

HOUSTON SUPERSITE, TX; 08/23/00 to 09/18/00

Houston Regional Monitoring Site was located $<$ 1.0 km north of the Houston ship channel, where chemical and other industries are present. The site was located between a railway to the south and a chemical plant to the north. Major freeways were located just to the north and east of the sampling site.

Spectrometer	Inlet Characteristics[a] (Flow Rate [L/Min] Size Inlet Dryer Aerodynamic Diameter, μm Particle Sizing Method)	Volatilization/ Ionization Method[a]	Hit Rates[b]	Mass Spectrometer[c]	Particle Analysis/ Classification	Other
SPECTROMETER	INLET CHARACTERISTICS (FLOW RATE [L/MIN] SIZE INLET DRYER AERODYNAMIC DIAMETER, μM PARTICLE SIZING METHOD)	VOLATILIZATION/ IONIZATION METHOD[a]	HIT RATES[b]	MASS SPECTROMETER[c]	PARTICLE ANALYSIS/ CLASSIFICATION	OTHER
RSMS-II	n/a None Nafion 0.035 – 1.14 Aerodynamic focusing; Need to pre-select sizes to be analyzed	LDI, ArF laser, 193 nm	n/a	Single linear TOF; Ion polarity needs to be pre-selected	Peak ID/artificial neural network	At each size point, aerosol was sampled in each cycle for either 10 min or until mass spectra for 30 particles per major class were collected, whichever came first.
Phares et al. [134]						
27,000 spectra were classified using a neural network into 15 particle types						
Fifteen particle type mass spectra were presented along with their size distribution, avg time of day occurrence, and wind direction dependence						
Major classes were a K^+ dominant, Si/Silicon Oxide, Carbon, Sea Salt, Fe, Zn, Amines, Lime, Vanadium, Organic Mineral, Pb and K, Al, and a Pb salt particle type.						
FRESNO SUPERSITE, CA: 11/30/00 to 2/4/01 Urban location in a residential neighborhood.						
SPECTROMETER	INLET CHARACTERISTICS (FLOW RATE [L/MIN] SIZE INLET DRYER AERODYNAMIC DIAMETER, μM PARTICLE SIZING METHOD)	VOLATILIZATION/ IONIZATION METHOD[a]	HIT RATES[b]	MASS SPECTROMETER[c]	PARTICLE ANALYSIS/ CLASSIFICATION	OTHER
ATOFMS	1 None None 0.3 – 2.5 Aerodynamic	LDI, ND: YAG 266 nm	n/a	Dual reflectron TOF	Peak ID/artificial neural network	ATOFMS unscaled detected particles tracked B attenuation monitor $PM_{2.5}$ mass concentration
Qin and Prather [135]						
Biomass burning particles reached a maximum at night and a minimum during the day. These particles were less than 1 μm in diameter and accounted for more than 60% of the particles detected at night.						
Another particle class characterized by high mass carbon fragments had a similar diurnal pattern. These particles were larger than 1 μm and were interpreted as biomass particles that have undergone gas to particle conversion of semi-volatile species followed by dissolution in a water droplet.						
PITTSBURGH SUPERSITE, PA; 09/07/02 TO 09/22/02 FOR AMS; 09/20/01 to 09/26/02 for RSMS-III 6 km east of downtown in a park on the top of a hill						
SPECTROMETER	INLET CHARACTERISTICS (FLOW RATE [L/MIN] SIZE INLET DRYER AERODYNAMIC DIAMETER, μM PARTICLE SIZING METHOD)	VOLATILIZATION/ IONIZATION METHOD[a]	MASS SPECTROMETER[c]	OTHER		
AMS	1.4 cc/s $PM_{2.5}$ cyclone None 0.05 – 1.0 Aerosol TOF	T - 600 °C/ EI	Quadrupole; Mass weighted size distributions on pre-selected positive ions only.	Particle size-cut of ~ 1 μm		

Spectrometer	Inlet Characteristics[a] (Flow Rate [L/Min] Size Inlet Dryer Aerodynamic Diameter, μm Particle Sizing Method)	Volatilization/ Ionization Method[a]	Hit Rates[b]	Mass Spectrometer[c]	Particle Analysis/ Classification	Other
RSMS-III	n/a None Nafion 0.03 – 1.1 Aerodynamic focusing; Need to pre-select sizes to be analyzed.	LDI, ArF laser, 193 nm		Dual TOF feflectron; Detects both positive and negative ions	At each size point, aerosol was sampled in each cycle for either 10 min or until mass spectra for 30 particles per major class were collected, whichever came first	

Zhang et al.[136]; Bein et al.[137]

The AMS observed 75% of the SO_4^{2-} measured by R&P-8400S (R2 = 0.69).

Collection efficiency (CE) of 0.5 used for SO_4^{2-}, NO_3 and NH_4^+ and 0.7 for organics to correct mass concentrations for incomplete detection. Use of a constant CE irrespective of size and shape may overestimate accumulation mode (mostly, oxygenated) organics (true CE $\sim$ 0.5) and underestimate smaller mode (primary) organics (true CE $\sim$ 1.0).

Comparison of AMS organics (organic matter, OM) with OC measured by a continuous Sunset OCEC instrument showed good correlation (R2 = 0.88) with a slope of 1.69. A 24-h avg comparison, showed a slope of 1.45. These values are in the typical range of 1.2 to 2.0 for OM/OC ratios.

AMS could be used along with the SMPS to estimate particle density. The AMS did not always agree with SMPS, probably due to non-spherical particles (irregular) such as soot from fresh traffic emissions, whose mass may be overestimated by the SMPS.

Comparison of AMS mass with the MOUDI, showed differences for aerodynamic diameters $>$ 600 nm, probably due to the AMS transmission being less than unity for particles larger than 600 nm.

For RSMS-III, 54% of the detected particles were assigned to one class (carbonaceous ammonium nitrate). This class was preferentially detected during the colder months and was detected from many different wind directions.

The next largest RSMS-III class was EC/OC/K class at 11%, and is believed to be from biomass burning.

An unidentified organic carbon RSMS-III class (3.3% of all detected particles) was seen to be highly dependent on wind direction dependence and was primarily detected during August and September of 2002. These particles likely originated from a landfill.

NEW YORK SUPERSITE; 06/30/01 to 08/05/01 (urban); 07/09/02 to 08/07/02: (rural)

Urban Site: Queens College, Queens, New York, located at the edge of a parking lot and within 1 km from expressways and highways in New York City Metropolitan area.

Rural Site: Whiteface Mountain, New York, located in a cleared area surrounded by mix of deciduous and evergreen trees, $\sim$2 km away from the closest highway with no major cities within 20 km.

SPECTROMETER	INLET CHARACTERISTICS (FLOW RATE [L/MIN] SIZE INLET DRYER AERODYNAMIC DIAMETER, μM PARTICLE SIZING METHOD)	VOLATILIZATION/ IONIZATION METHOD[a]	MASS SPECTROMETER[c]	OTHER
AMS	0.1 $PM_{2.5}$ cyclone None 0.02 – 2.5 Aerosol TOF	T – 700 °C/ EI	Quadrupole; Mass weighted size distributions on pre-selected positive ions only.	Data are 10-min averages

Spectrometer	Inlet Characteristics[a] (Flow Rate [L/Min] Size Inlet Dryer Aerodynamic Diameter, μm Particle Sizing Method)	Volatilization/ Ionization Method[a]	Hit Rates[b]	Mass Spectrometer[c]	Particle Analysis/ Classification	Other

Drewnick et al. [138, 139]; Hogrefe et al. [20]

Transport losses were 1.3% on avg.

Inlet losses (at the inlet of AMS) were 1.9%, on avg, ranging from 11% for a 20 nm particle to 9% for a 2.5 μm particle, with a minimum of 0.7% for a 350 nm particle

Overall measurement uncertainty of particle diameter was ~11%.

The AMS was reliable with proper calibration, care, and maintenance. Valid 10 min averages were obtained for all components more than 93% of the time.

The mass to charge ratios (m/z) of fragments from different components may overlap (e.g., NH+, a fragment of NH_4^+ and CH_3^+, a fragment of organic species, have m/z = 15) resulting in an interference (called as isobaric interference) Interfering signals were not used to calculate concentrations. This loss in concentration was adjusted by applying a correction factor determined from laboratory studies.

Typical interferences were from fragments of organic species, water and oxygen.

With adjustments, the SO_4^{2-}, NO_3^-, and ammonium concentrations measured by the AMS were consistently lower than that measured by other co-located instruments, probably due to incomplete focusing of the $(NH_4)_2SO_4$ and NH_4NO3 particles by the aerodynamic lens.

At the urban site, AMS NO_3 was within 10% of the filter NO_3 concentration. At the rural site, it had a slope of 0.51 and R2 of 0.46.

AMS SO_4 showed good agreement with R&P-8400S at both the rural and urban locations (R2 = 0.89 to 0.92, slope = 0.99, n = 407 to 695) and was within 70 to 85% of filter SO_4^{2-} concentration.

Comparison of the total non-refractory mass measured by the AMS with the $PM_{2.5}$ TEOM mass (operated at 50 °C or with dryer) at the urban location, showed good correlation (R2 = 0.91) with near zero intercept (0.22 $\mu g/m^3$). On avg, the AMS observed 64% of the mass measured by the TEOM.

The unexplained mass (36%) was attributed to transport losses, transmission and optical losses, and refractory components in the aerosol sample (e.g., metals, EC). The mass closure was within the estimated uncertainty of the AMS mass measurements (5 to 10%).

BALTIMORE SUPERSITE, MD; 04/01/02 to 11/30/02
East of downtown in an urban residential area. Within 91 m of a bus maintenance facility.

SPECTROMETER	INLET CHARACTERISTICS (FLOW RATE [L/MIN] SIZE INLET DRYER AERODYNAMIC DIAMETER, μM PARTICLE SIZING METHOD)	VOLATILIZATION/ IONIZATION METHOD[a]	MASS SPECTROMETER[c]	OTHER
RSMS-III	0.2 – 18, based on particle size chosen None Nafion 0.045 – 1.3 Aerodynamic focusing; Need to pre-select sizes to be analyzed	LDI, ArF laser, 193 nm	TOF with dual ion polarity	At each size set point, aerosol was sampled in each cycle for either 10 min or until mass spectra from 30 particles were collected, whichever came first.

Lake et al. [140, 141]

Utilizing both positive and negative ion detection enables detection of more species. However, detection efficiencies of negative ions decreased for smaller particles.

SO_4^+ concentration (number or mass) was not accurately quantified.

RSMS-III was most efficient in 0.050 to 0.77 μm range.

Particle compositions could be related to specific source categaories.

Spectrometer	Inlet Characteristics[a] (Flow Rate [L/Min] Size Inlet Dryer Aerodynamic Diameter, μm Particle Sizing Method)	Volatilization/ Ionization Method[a]	Hit Rates[b]	Mass Spectrometer[c]	Particle Analysis/ Classification	Other

[a]EI: Electon Impact; LDI: Laser Desorption / Ionization
[b]Hit rate refers to the number of particles with a mass spectrum as a fraction of the number of particles detected. It does not apply to RSMS and AMS because there is no separate detection
[c]TOF: Time fo Flight
ATLANTA SUPERSITE, GA; 08/03/99 to 09/01/99
4 km NW of downtown, within 200 m of a bus maintenance yard and several warehouse facilities, representative of a mixed commercial-residential neighborhood.

Source: [1]Chow (1995, 077012); [2]Watson and Chow (2001, 157123); [3] Watson et al. (1983, 045084); [4]Fehsenfeld et al. (2004, 156432); [5]Solomon et al. (2001, 156993); [6]Mikel (2001, 156762); [7]Mikel (2001, 156762); [8]Watson et al. (1999, 020949); [9]Solomon and Sioutas (2006, 156995); [10]Graney et al. (2004, 053756); [11]Tanaka et al. (1998, 157041); [12]Pancras et al. (2005, 098120); [13]John et al. (1988, 045903); [14]Hering and Cass (1999, 084958); [15]Fritz et al. (1989, 077387); [16]Hering et al. (1988, 036012); [17]Solomon et al. (2003, 156994); [18]Cabada et al. (2004, 148859); [19]Fine et al. (2003, 155775); [20]Hogrefe et al. (2004, 099003); [21]Drewnick et al. (2003, 099160); [22]Watson et al. (2005, 157125); [23]Ho et al. (2006, 156552); [24]Decesari et al. (2005, 144536); [25] Mayol-Bracero et al. (2002, 045010); [26]Yang et al. 2003; [27] Turšic et al. (2006, 157063); [28]Mader et al.(2004, 156724); [29]Xiao and Liu (2004, 056801); [30]Kiss et al. (2002, 156646); [31]Cornell and Jickells (1999, 156367); [32] Zheng et al. (2002, 026100); [33]Fraser et al. (2002, 140741); [34] Fraser et al. (2003, 042231) [35]Schauer er al. (1996, 051162); [36]Fine et al. (2004); [37]Yue et al. (2004); [38]Rinehart et al. (2006, 115184); [39]Wan and Yu (2006, 157104); [40]Poore (2000, 012839); [41]Fraser et al. (2003a); [42]Engling et al. (2006, 156422); [43]Yu et al. (2005); [44]Tran et al. (2000); [45]Yao et al. (2004, 102213); [46]Li and Yu (2005, 156692); [47]Henning et al. (2003, 156539); [48]Zhang and Anastasio (2003, 157182); [49]Emmenegger et al. (2007, 156418); [50](Watson et al., 1989, 157119); [51]Greaves et al. (1985, 156494); [52]Waterman et al. (2000, 157116); [53]Waterman et al. (2001, 157117); [54]Falkovich and Rudich (2001, 156427); [55]Chow et al. (2007, 156354); [56]Miguel et al. (2004, 123260); [57]Crimmins and Baker (2006, 097008); [58]Ho and Yu (2004, 156551); [59]Jeon et al. (2001, 016636); [60]Mazzoleni et al. (2007, 098038); [61]Poore (2000, 012839); [62]Butler et al. (2003); [63]Chow et al. (2006c); [64]Russell et al. (2004, 082453); [65]Grover et al. (2006); [66]Grover et al. (2005, 090044); [67]Schwab et al. (2006b); [68]Hauck et al. (2004); [69]Jaques et al. (2004, 155878); [70]Rupprecht and Patashnick (2003, 157207) ; [71]Pang et al (2002, 030353); [72]Eatough et al. (2001, 010303); [73]Lee et al. (2005b); [74]Lee et al. (2005, 156680); [75]Babich et al. (2000, 156239); [76]Lee et al. (2005c); [77]Lee et al. (2005b); [78]Anderson and Ogren (1998, 156213); [79]Chung et al. (2001, 156357); [80]Kidwell and Ondov (2004, 155898); [81]Lithgow et al. (2004, 126616); [82]Weber et al. (2003, 157129); [83]Harrison et al. (2004); [84]Rattigan et al. (2006, 115897); [85]Wittig et al. (2004, 103413); [86]Vaughn et al. (2005, 157089); [87]Chow et al. (2005b); [88]Weber et al (2001, 024640) ; [89]Schwab et al. (2006a); [90]Lim et al. (2003, 156697); [91]Watson and Chow (2002, 037873); [92]Venkatachari et al. (2006, 105918); [93]Bae et al. (2004a); [94]Arhami et al. (2006, 156224); [95]Park et al. (2005, 156843); [96]Bae et al. (2004b); [97]Chow et al. (2006a); [98]Arnott et al. (2005); [99]Bond et al. (1999); [100]Virkkula et al. (2005, 157097); [101]Petzold et al. (2002, 156863); [102]Park et al. (2006); [103]Arnott et al. (1999, 020650); [104]Peters et al. (2001); [105]Pitchford et al. (1997, 156872); [106]Rees et al. (2004, 097164); [107]Watson et al. (2000); [108]Lee et al. (2005, 156680); [109]Hering et al. (2004, 155837); [110]Watson et al. (1998); [111]Chakrabarti et al. (2004); [112]Mathai et al. (1990, 156741); [113]Kidwell and Ondov (2001, 017092); [114]Stanier et al. (2004); [115]Khlystov et al. (2005, 156635); [116]Takahama et al. (2004, 157038); [117]Chow et al. (2005, 156348); [118]Zhang et al. (2002); [119]Subramanian et al. (2004, 081203); [120]Chow et al. (2006b); [121]Birch and Cary (1996); [122]Birch (1998, 024953); [123]Birch and Cary (1996); [124]NIOSH (1996, 156810); [125]NIOSH (1999, 156811); [126]Chow et al. (1993); [127]Chow et al. (2007); [128]Ellis and Novakov (1982, 156416); [129]Peterson and Richards (2002, 156861); [130]Schauer et al. (2003); [131]Middlebrook et al. (2003, 042932); [132]Wenzel et al. (2003, 157139); [133]Jimenez et al. (2003); [134]Phares et al. (2003, 156866); [135]Qin and Prather (2006, 156895); [138]Zhang et al. (2005); [137]Bein et al. (2005, 156265); [138]Drewnick et al. (2004a); [139]Drewnick et al. (2004b); [140]Lake et al. (2003, 156669); [141]Lake et al. (2004, 088411)

Source: Chow et al. (2008, 156355

Table A-18. Summary of key parameters for TD-GC/MS and pyrolysis-GC/MS.

Reference	Sample Type	TD Unit	Analytical Instrument	Total Analysis Time
TD-GC/MS WITH RESISTIVELY HEATED EXTERNAL OVEN				
Greaves et al. (1985, 156494; 1987, 156495); Veltkamp et al. (1996, 081594)	Aerosol sample and NIST SRM 1649	A cylindrical aluminum block containing a heating cartridge connected to a thermocouple	HP 5892A GC/MS in EI mode	ambient sample: 55.5 min NIST standard: 45.5 min
Waterman et al. (2000, 157116)	NIST SRM 1640a	External oven mounted on the top of the GC/MS system	HP 5890 GC/Fisons MD 800 MS, scan range: 40–520 amu	90 min
Waterman et al. (2001, 157117)	NIST SRM 1649a	Same as above	HP 5890 GC/Fisons MD 800 MS, scan range: m/z 40 to 520	90 mins
Sidhu et al. (2001, 155202)	Aerosol collected on glass ◻ber ◻lters from combustion of alternative diesel fuel.	A stainless steel tube (0.635 cm O.D.laced in a GC oven	Two GCs and one MS. The ◻rst GC is used as the TE unit. The second GC separates the desorbent.	Ua
Hays et al. (2003, 156529; 2004, 156530); Dong et al. (2004, 156409)	Aerosol collected from residential wood combustion, residential oil furnace and ◻replace appliance	A glass tube placed in an external oven (TDS2 Gerstel Inc.)	Aglient 6890 GC/5793 MSD, scan range: 50 to 500 amu	99 min

Reference	Sample Type	TD Unit	Analytical Instrument	Total Analysis Time
CURIE POINT TD-GC/MS				
Jeon et al. (2001, 016636)	High-volume PM_{10} ambient samples collected along the U.S./Mexico border	Curie point pyrolyzer	HP 5890 GC/5792 MSD	Ua
Neususs et al. (2000, 156804)	Ambient aerosol collected during the 2nd Aerosol Characterization Experiment	Curie point pyrolyzer	Fisons Trio 1000	35 min
IN-INJECTION PORT TED-GC/MS				
Helmig et al. (1990, 156536)	Aerosol samples collected on glass-fiber filters at a forest site	GC injector port, with modified septum cap	Carlo Erba Mega 5160 GC/VG 250/70 SE MS, scan range: 45-400 amu	47 min
Hall et al. (1999, 156512)	NIST SRM 1649	Micro-scale sealed vessel placed inside the injector port	HP 5890 GC/Fisons MD 800 MS, scan range: 40-500 amu	82.5 min
Blanchard and Hopper (1997) (1997, 157195); Blanchard et al. (2002, 189737)	Aerosol samples collected on quartz-and-glass filters in Ontario	A GC injection port was added with three minor components, including a small T-connector, 3-way valve, and needle valve	HP 5892A GC/5972A MS in EI mode	71 min
Falkovich and Rudich (2001, 156427) ; Falkovich et al. (2004, 156428); Graham et al. (2004, 156490)	NIST SRM 1649a; urban aerosols collected with an 8-stage impactor in Tel-Aviv, Israel	Direct Sample Introduction (DSI) device (ChromatoProbe, Varian Co.)	Varian Saturn 3400 GC/MS	64.2 min
Ho and Yu (2004, 156551); Yang et al. (2005, 102388)	Ambient aerosol samples collected on Teflon-impregnated glass-fiber filters in Hong Kong and on quartz filters at Nanjing, China	Conventional GC injection port. No modification of GC injector and liner	HP 5890 GC/5791 MSD, scan range: 50-650 amu	41.5 min
TD-GC X GC-MS				
Welthagen et al. (2003, 104056); Schnelle-Kreis et al. (2005, 112944)	Ambient samples in Augsburg, Germany	Injection port Optic III with autoloader (ATAS-GL, Veldhoven, NL)	Agilent 6890 GC/LECO Pegasus III TOF/MS with a LECO Pegasus 4D GCxGC modulator	175 min
Hamilton et al. (2004, 156516)	$PM_{2.5}$ aerosol collected in London	Conventional GC injection port	The same as above, scan range: 20-350 amu	93.7 min
Hamilton et al. (2005, 088173)	Secondary organic aerosol formed during the photo-oxidation of toluene with OH radicals	The same as above	The same above	102.5 min
IN SITU SEMI-CONTINUOUS AND CONTINUOUS TD SYSTEMS				
Williams et al. (2006, 156157)	In situ aerosol samples collected in Berkley, CA	Collection-TE cell with conventional GC injection port	Agilent 6890 GC/5793 MSD, scan range: 29-550 amu	59 min
PYROLYSIS TD-GC/MS				
Voorhees et al. (1991, 157101)	$PM_{0.6}$ and $PM_{>0.45}$ collected on quartz fiber in pristine regions of Colorado	A tube furnace directly interfaced to an GC/MS	Extrel Simulscan GC/MS, scan range: 35-450 amu	31.7 min
Subbalakshmi et al. (2000, 157023)	Ambient aerosol collected on glass-fiber filters in Jakarta, Indonesia	A pyroinjector	Agilent 6890 GC/5973 MS, scan range: 50-550 amu	63.5 min
Fabbri et al. (2002, 156426)	PM_{10} collected on glass-fiber filters in an industrial are of Italy	A pyrolyzer directly connected to the GC injector port through an interface heated at 250° C	Varian 3400 GC/Saturn II ion trap MS, scan range: 45-400 amu	57 min
Blazso et al. (2003, 156278)	$PM_{2.6}$ collected on quartz-fiber filters and size-segregated aerosol sampled collected on A1 foils in Brazil	A pyrolyzer	Agilent 6890 GC/5973 MS	30.3 min
Labban et al. (2006, 156665)	PM_{10} of re-suspended soil collected on quartz-fiber filters	Curie point pyrolyzer	HP 5890 GC/5972 MS	25.5. min

Reference	Sample Type	TD Unit	Analytical Instrument	Total Analysis Time

[a]Total analysis time could not be determined because of insufficient experimental details

Source: Chow et al. (2007, 157209)

A.1.2. Networks

Table A-19. Relevant Spatial Scales for PM_{10}, $PM_{2.5}$, and $PM_{10\text{-}2.5}$ Measurement

Spatial Scales	PM_{10}	$PM_{2.5}$	$PM_{10\text{-}2.5}$
Microscale (~ 5 – 100 m)	This scale would typify areas such as downtown street canyons, traffic corridors, and fence line stationary source monitoring locations where the general public could be exposed to maximum PM_{10} concentrations. Microscale particulate matter sites should be located near inhabited buildings or locations where the general public can be expected to be exposed to the concentration measured. Emissions from stationary sources such as primary and secondary smelters, power plants, and other large industrial processes may, under certain plume conditions, likewise result in high ground level concentrations at the microscale. In the latter case, the microscale would represent an area impacted by the plume with dimensions extending up to approximately 100 meters. Data collected at microscale sites provide information for evaluating and developing hot spot control measures.	This scale would typify areas such as downtown street canyons and traffic corridors where the general public would be exposed to maximum concentrations from mobile sources. In some circumstances, the microscale is appropriate for particulate sites; community-oriented SLAMS sites measured at the microscale level should, however, be limited to urban sites that are representative of long-term human exposure and of many such microenvironments in the area. In general, microscale particulate matter sites should be located near inhabited buildings or locations where the general public can be expected to be exposed to the concentration measured. Emissions from stationary sources such as primary and secondary smelters, power plants, and other large industrial processes may, under certain plume conditions, likewise result in high ground level concentrations at the microscale. In the latter case, the microscale would represent an area impacted by the plume with dimensions extending up to approximately 100 meters. Data collected at microscale sites provide information for evaluating and developing hot spot control measures. Unless these sites are indicative of population-oriented monitoring, they may be more appropriately classified as SPM.	This scale would typify relatively small areas immediately adjacent to: Industrial sources; locations experiencing ongoing construction, redevelopment, and soil disturbance; and heavily traveled roadways. Data collected at microscale stations would characterize exposure over areas of limited spatial extent and population exposure, and may provide information useful for evaluating and developing source-oriented control measures.
Middle Scale (~ 100 – 500 m)	Much of the short-term public exposure to coarse fraction particles (PM_{10}) is on this scale and on the neighborhood scale. People moving through downtown areas or living near major roadways or stationary sources, may encounter particulate pollution that would be adequately characterized by measurements of this spatial scale. Middle scale PM_{10} measurements can be appropriate for the evaluation of possible short-term exposure public health effects. In many situations, monitoring sites that are representative of micro-scale or middle-scale impacts are not unique and are representative of many similar situations. This can occur along traffic corridors or other locations in a residential district. In this case, one location is representative of a neighborhood of small scale sites and is appropriate for evaluation of long-term or chronic effects. This scale also includes the characteristic concentrations for other areas with dimensions of a few hundred meters such as the parking lot and feeder streets associated with shopping centers, stadia, and office buildings. In the case of PM_{10}, unpaved or seldomly swept parking lots associated with these sources could be an important source in addition to the vehicular emissions themselves.	People moving through downtown areas, or living near major roadways, encounter particle concentrations that would be adequately characterized by this spatial scale. Thus, measurements of this type would be appropriate for the evaluation of possible short-term exposure public health effects of particulate matter pollution. In many situations, monitoring sites that are representative of microscale or middle-scale impacts are not unique and are representative of many similar situations. This can occur along traffic corridors or other locations in a residential district. In this case, one location is representative of a number of small scale sites and is appropriate for evaluation of long-term or chronic effects. This scale also includes the characteristic concentrations for other areas with dimensions of a few hundred meters such as the parking lot and feeder streets associated with shopping centers, stadia, and office buildings.	People living or working near major roadways or industrial districts encounter particle concentrations that would be adequately characterized by this spatial scale. Thus, measurements of this type would be appropriate for the evaluation of public health effects of coarse particle exposure. Monitors located in populated areas that are nearly adjacent to large industrial point sources of coarse particles provide suitable locations for assessing maximum population exposure levels and identifying areas of potentially poor air quality. Similarly, monitors located in populated areas that border dense networks of heavily-traveled traffic are appropriate for assessing the impacts of resuspended road dust. This scale also includes the characteristic concentrations for other areas with dimensions of a few hundred meters such as school grounds and parks that are nearly adjacentto major roadways and industrial point sources, locations exhibiting mixed residential and commercial development, and downtown areas featuring office buildings, shopping centers, and stadiums.

Spatial Scales	PM_{10}	$PM_{2.5}$	$PM_{10-2.5}$
Neighborhood Scale (~500 m – 4 km)	Measurements in this category represent conditions throughout some reasonably homogeneous urban sub-region with dimensions of a few kilometers and of generally more regular shape than the middle scale. Homogeneity refers to the particulate matter concentrations, as well as the land use and land surface characteristics. In some cases, a location carefully chosen to provide neighborhood scale data would represent not only the immediate neighborhood but also neighborhoods of the same type in other parts of the city. Neighborhood scale PM_{10} sites provide information about trends and compliance with standards because they often represent conditions in areas where people commonly live and work for extended periods. Neighborhood scale data could provide valuable information for developing, testing, and revising models that describe the larger-scale concentration patterns, especially those models relying on spatially smoothed emission fields for inputs. The neighborhood scale measurements could also be used for neighborhood comparisons within or between cities.	Measurements in this category would represent conditions throughout some reasonably homogeneous urban sub- region with dimensions of a few kilometers and of generally more regular shape than the middle scale. Homogeneity refers to the particulate matter concentrations, as well as the land use and land surface characteristics. Much of the $PM_{2.5}$ exposures are expected to be associated with this scale of measurement. In some cases, a location carefully chosen to provide neighborhood scale data would represent the immediate neighborhood as well as neighborhoods of the same type in other parts of the city. $PM_{2.5}$ sites of this kind provide good information about trends and compliance with standards because they often represent conditions in areas where people commonly live and work for periods comparable to those specified in the NAAQS. In general, most $PM_{2.5}$ monitoring in urban areas should have this scale.	Measurements in this category would represent conditions throughout some reasonably homogeneous urban sub-region with dimensions of a few kilometers and of generally more regular shape than the middle scale. Homogeneity refers to the particulate matter concentrations, as well as the land use and land surface characteristics. This category includes suburban neighborhoods dominated by residences that are somewhat distant from major roadways and industrial districts but still impacted by urban sources, and areas of diverse land use where residences are interspersed with commercial and industrial neighborhoods. In some cases, a location carefully chosen to provide neighborhood scale data would represent the immediate neighborhood as well as neighborhoods of the same type in other parts of the city. The comparison of data from middle scale and neighborhood scale sites would provide valuable information for determining the variation of $PM_{10-2.5}$ levels across urban areas and assessing the spatial extent of elevated concentrations caused by major industrial point sources and heavily traveled roadways. Neighborhood scale sites would provide concentration data that are relevant to informing a large segment of the population of their exposure levels on a given day.
Urban Scale (~4 – 50 km)		This class of measurement would be used to characterize the particulate matter concentration over an entire metropolitan or rural area ranging in size from 4 to 50 kilometers. Such measurements would be useful for assessing trends in area-wide air quality, and hence, the effectiveness of large scale air pollution control strategies. Community-oriented $PM_{2.5}$ sites may have this scale.	
Regional Scale (~50 – 100s km)		These measurements would characterize conditions over areas with dimensions of as much as hundreds of kilometers. As noted earlier, using representative conditions for an area implies some degree of homogeneity in that area. For this reason, regional scale measurements would be most applicable to sparsely populated areas. Data characteristics of this scale would provide information about larger scale processes of particulate matter emissions, losses and transport. $PM_{2.5}$ transport contributes to elevated particulate concentrations and may affect multiple urban and State entities with large populations such as in the eastern United States. Development of effective pollution control strategies requires an understanding at regional geographical scales of the emission sources and atmospheric processes that are responsible for elevated $PM_{2.5}$ levels and may also be associated with elevated O_3 and regional haze.	

Table A-20. Major routine operating air monitoring networks[d]

Network	Lead Agency	Number of Sites	Initiated	Measurement Parameters	Location of Information and/or Data
STATE / LOCAL / FEDERAL NETWORKS					
NCore[a] – National Core Monitoring Network	EPA	75	2008	O_3, $NO/NO_2/NO_Y$, SO_2, CO, $PM_{2.5}/PM_{10-2.5}$, $PM_{2.5}$ speciation, NH_3, HNO_3, surface meteorology[b]	http://www.epa.gov/ttn/amtic/monstratdoc.html

Network	Lead Agency	Number of Sites	Initiated	Measurement Parameters	Location of Information and/or Data
SLAMS1 – State and Local Ambient Monitoring Stations	EPA	~3000	1978	O_3, NO_X/NO_2, SO_2, $PM_{2.5}/PM_{10}$, CO, Pb	http://www.epa.gov/air/oaqps/qa/monprog.html
STN—$PM_{2.5}$ Speciation Trends Network	EPA	300	1999	$PM_{2.5}$, $PM_{2.5}$ speciation, major Ions, metals	http://www.epa.gov/ttnamti1/specgen.html
PAMS—Photochemical Assessment Monitoring Network	EPA	75	1994	O_3, NO_X/NO_Y, CO, speciated VOCs, carbonyls, surface meteorology & Upper Air	http://www.epa.gov/air/oaqps/pams/
IMPROVE—Interagency Monitoring of Protected Visual Environments	NPS	110 plus 67 protocol sites	1988	$PM_{2.5}/PM_{10}$, major ions, metals, light extinction, scattering coefficient	http://vista.cira.colostate.edu/IMPROVE/
CASTNet – Clean Air Status and Trends Network	EPA	80+	1987	O_3, SO_2, major ions, calculated dry deposition, wet deposition, total deposition for sulfur/nitrogen, surface meteorology	http://www.epa.gov/castnet/
GPMN—Gaseous Pollutant Monitoring Network	NPS	33	1987	O_3, $NO_X/NO/NO_2$, SO_2, CO, surface meteorology, (plus enhanced monitoring of CO, NO, NO_X, NO_Y, and SO_2 plus canister samples for VOC at 3 sites)	http://www2.nature.nps.gov/air/Monitoring/network.cfm#data
POMS—Portable Ozone Monitoring Stations	NPS	14	2002	O_3, surface meteorology, with CASTNet-protocol filter pack (optional) sulfate, nitrate, ammonium, nitric acid, sulfur dioxide	http://www2.nature.nps.gov/air/studies/portO_3.cfm
Passive Ozone Sampler Monitoring Program	NPS	43	1995	O_3 dose (weekly)	http://www2.nature.nps.gov/air/Studies/Passives.cfm
NADP/NTN—National Atmospheric Deposition Program / National Trends Network	USGS	200+	1978	Major Ions from precipitation chemistry	http://nadp.sws.uiuc.edu/
NADP/MDN—National Atmospheric Deposition Program / Mercury Deposition Network	None	90+	1996	Mercury from precipitation chemistry	http://nadp.sws.uiuc.edu/mdn/
AIRMoN—National Atmospheric Deposition Program / Atmospheric Integrated Research Monitoring Network	NOAA	8	1992	Major Ions from precipitation chemistry Note: some sites began in 1976 as part of the DOE MAP3S program; early data are archived on NADP and ARL servers.	http://nadp.sws.uiuc.edu/AIRMoN/
IADN—Integrated Atmospheric Deposition Network	EPA	20	1990	PAHs, PCBs, and organochlorine compounds are measured in air and precipitation samples	http://www.epa.gov/glnpo/monitoring/air/
NAPS—National Air Pollution Surveillance Network	Canada	152+	1969	SO_2, CO, O_3, NO, NO_2, NO_X, VOCs, SVOCs, PM_{10}, $PM_{2.5}$, TSP, metals	http://www.etc-cte.ec.gc.ca/NAPS/index_e.html

Network	Lead Agency	Number of Sites	Initiated	Measurement Parameters	Location of Information and/or Data
CAPMoN—Canadian Air and Precipitation Monitoring Network	Canada	29	2002	O_3, NO, NO_2, NO_Y, PAN, NH_3, $PM_{2.5}$, PM_{10} and coarse fraction mass, $PM_{2.5}$ speciation, major ions for particles and trace gases, precipitation chemistry for major ions	http://www.msc.ec.gc.ca/capmon/index_e.cfm
Mexican Air Quality Network	Mexico	52-62	Late 1960's	O_3, NO_X, CO, SO_2, PM_{10}, TSP,VOC	http://www.ine.gob.mx/dgicur/calaire/indicadores.html
Mexican City Ambient Air Quality Monitoring Network	Mexico	49	Late 1960's	O_3, NO_X, CO, SO_2, PM_{10}, TSP,VOC	http://www.ine.gob.mx/dgicur/calaire/indicadores.html
AIR TOXICS MONITORING NETWORKS					
NATTS—National Air Toxics Trends Stations	EPA	23	2005	VOCs, Carbonyls, PM_{10} metals[c], Hg	http://www.epa.gov/ttn/amtic/airtoxpg.html
State/Local Air Toxics Monitoring	EPA	250+	1987	VOCs, Carbonyls, PM_{10} metals[c], Hg	http://www.epa.gov/ttn/amtic/airtoxpg.html
NDAMN—National Dioxin Air Monitoring Network	EPA	34	1998 - 2005	CDDs, CDFs, dioxin-like PCBs	http://cfpub.epa.gov/ncea/CFM/recordisplay.cfm?deid=54811
TRIBAL MONITORING NETWORKS					
Tribal Monitoring[e]	EPA	120+	1995	O_3, NO_X/NO_2, SO_2, $PM_{2.5}$/PM_{10}, CO, Pb	http://www.epa.gov/air/tribal/airprogs.html#ambmon
Industry / Research Networks					
New Source Permit Monitoring	None	variable	variable	O_3, NO_X/NO_2, SO_2, $PM_{2.5}$/PM_{10}, CO, Pb	Contact specific industrial facilities
HRM Network—Houston Regional Monitoring Network	None	9	1980	O_3, NO_X, $PM_{2.5}$/PM_{10}, CO, SO_2, Pb, VOCs, surface meteorology	http://hrm.radian.com/houston/how/index.htm
ARIES / SEARCH—Aerosol Research Inhalation Epidemiology Study / SouthEastern Aerosol Research and Characterization Study experiment	None	8	1992	O_3, NO/NO_2/NO_Y, SO_2, CO, $PM_{2.5}$/PM_{10}, $PM_{2.5}$ speciation, major Ions, NH_3, HNO_3, scattering coefficient, surface meteorology	http://www.atmospheric-research.com/studies/SEARCH/index.html
SOS – SERON—Southern Oxidant Study - Southeastern Regional Oxidant Networks	EPA	~40	1990	O_3, NO, NO_Y, VOCs, CO, surface meteorology	http://www.ncsu.edu/sos/pubs/sos3/State_of_SOS_3.pdf
NATIONAL/GLOBAL RADIATION NETWORKS					
RadNet—formerly Environmental Radiation Ambient Monitoring System (ERAMS)	EPA	200+	1973	Radionuclides and radiation	http://www.epa.gov/enviro/html/erams/
SASP -- Surface Air Sampling Program	DHS	41	1963	89Sr, 90Sr, naturally occurring radionuclides, 7Be, 210Pb	http://www.eml.st.dhs.gov/databases/sasp/
NEWNET—Neighborhood Environmental Watch Network	DOE	26	1993	Ionizing gamma radiation, surface meteorology	http://newnet.lanl.gov/
Solar Radiation Networks					

Network	Lead Agency	Number of Sites	Initiated	Measurement Parameters	Location of Information and/or Data
UV Index – EPA Sunrise Program[f]	EPA	~ 50 U.S. cities	2002	Calculated UV radiation index	http://www.epa.gov/sunwise/uvindex.html
UV Net -- Ultraviolet Monitoring Program	EPA	21	1995/2004	Ultraviolet solar radiation (UV-B and UV-A bands), irradiance, ozone, NO_2	http://www.epa.gov/uvnet/access.html
NEUBrew (NOAA-EPA Brewer Spectrophotometer UV and Ozone Network	NOAA	6	2005	Ultraviolet solar radiation (UV-B and UV-A bands), irradiance, ozone, SO_2	http://www.esrl.noaa.gov/gmd/grad/neubrew/
UV-B Monitoring and Research Program	USDA	35	1992	Ultraviolet-B radiation	http://uvb.nrel.colostate.edu/UVB/index.jsf
SURFRAD – Surface Radiation Budget Network	NOAA	7	1993	Solar and infrared radiation, direct and diffuse solar radiation, photosynthetically active radiation, UVB, spectral solar, and meteorological parameters	http://www.srrb.noaa.gov/surfrad/index.html
AERONET – Aerosol RObotic NETwork MPLNET – Micro-pulse Lidar Network	NASA co-located networks	22 + other participants 8	1998 2000	Aerosol spectral optical depths, aerosol size distributions, and precipitable water Aerosols and cloud layer heights	http://aeronet.gsfc.nasa.gov/index.html http://mplnet.gsfc.nasa.gov/
PRIMENet -- Park Research & Intensive Monitoring of Ecosystems NETwork[g]	NPS	14	1997	ozone, wet and dry deposition, visibility, surface meteorology, and ultraviolet radiation	http://www.cfc.umt.edu/primenet/Assets/Announcements/99PReport.pdf

[a]NCore is a network proposed to replace NAMS, as a component of SLAMS; NAMS are currently designated as national trends sites.
[b]surface meteorology includes wind direction and speed, temperature, precipitation, relative humidity, solar radiation (PAMS only).
[c]PM_{10} metals may include arsenic, beryllium, cadmium, chromium, lead, manganese, nickel, and others.
[d]Some networks listed separately may also serve as subcomponents of other larger listed networks; as a result, some double counting of the number of individual monitors is likely.
[e]The number of sites indicated for tribal monitoring is actually the number of monitors, rather than sites. The number of sites with multiple monitors is probably < 80.
[f]Sunrise program estimates UV exposure levels through modeling - does not include measurements.
[g]NEUBREW is a subset Original UV brewer network (UV Net); PRIMENET participated in UV Net program.

A.1.1. Monitor Distribution with Respect to Population Density

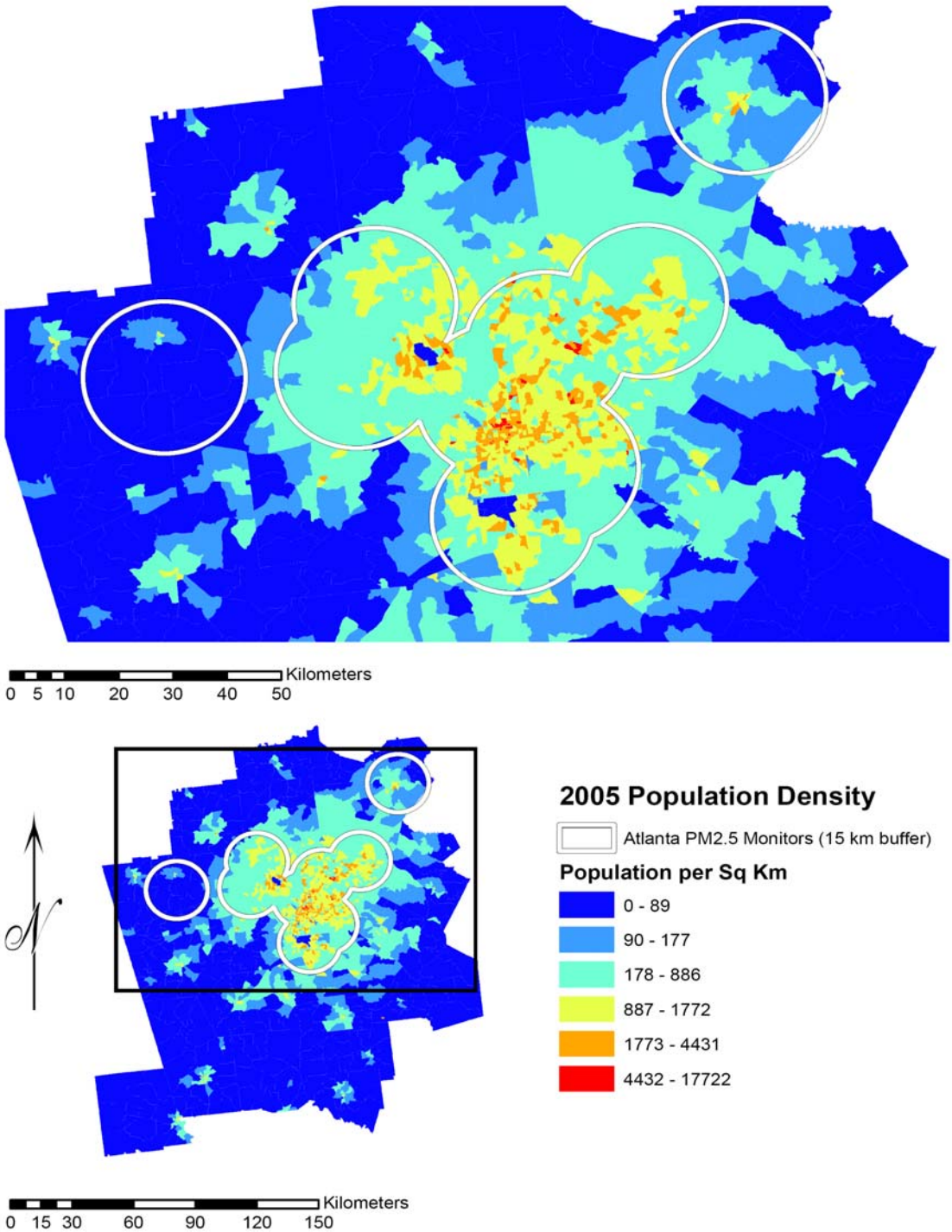


Figure A-1. $PM_{2.5}$ monitor distribution in comparison with population density, Atlanta, GA.

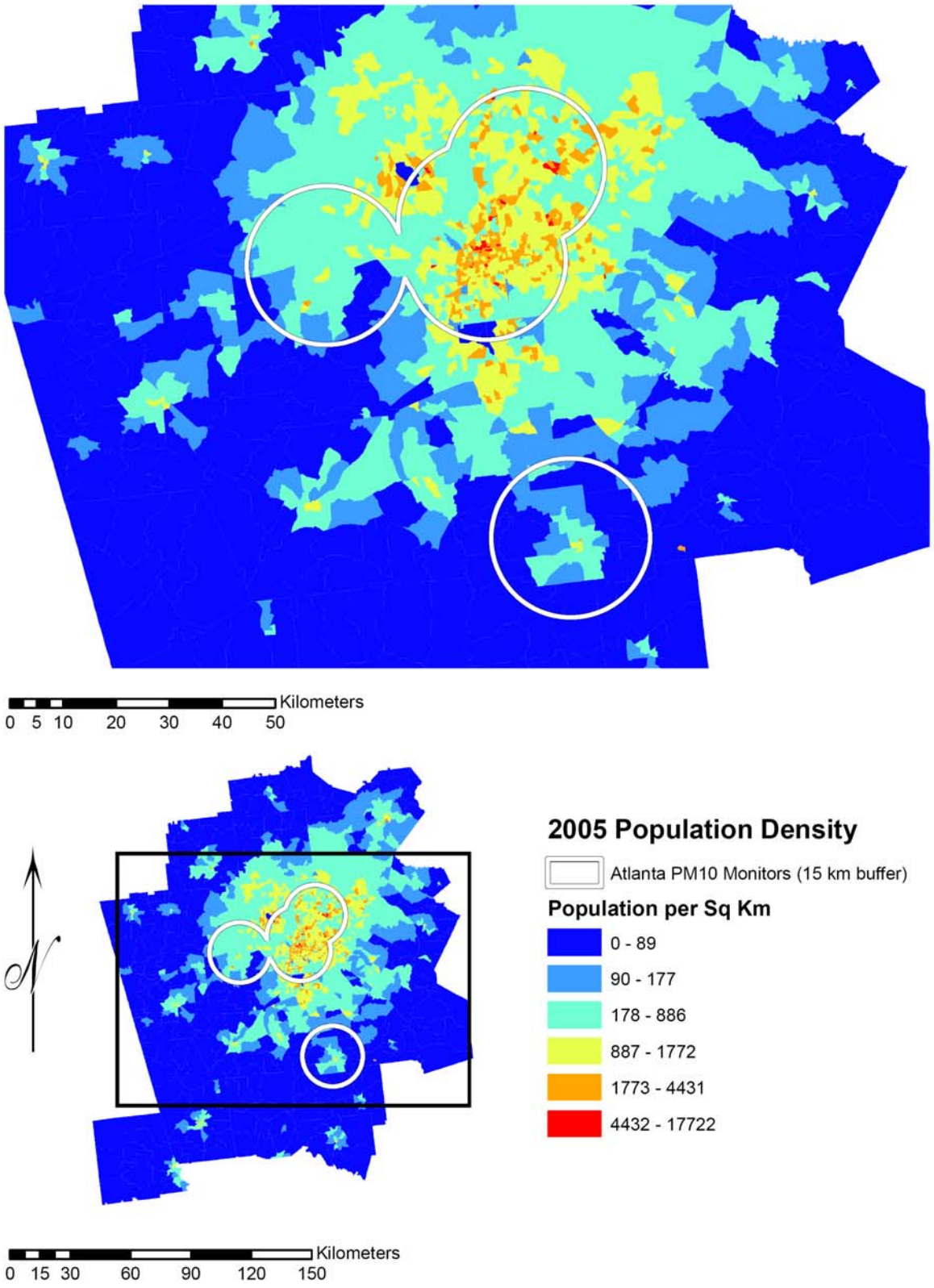


Figure A-2. PM_{10} monitor distribution in comparison with population density, Atlanta, GA.

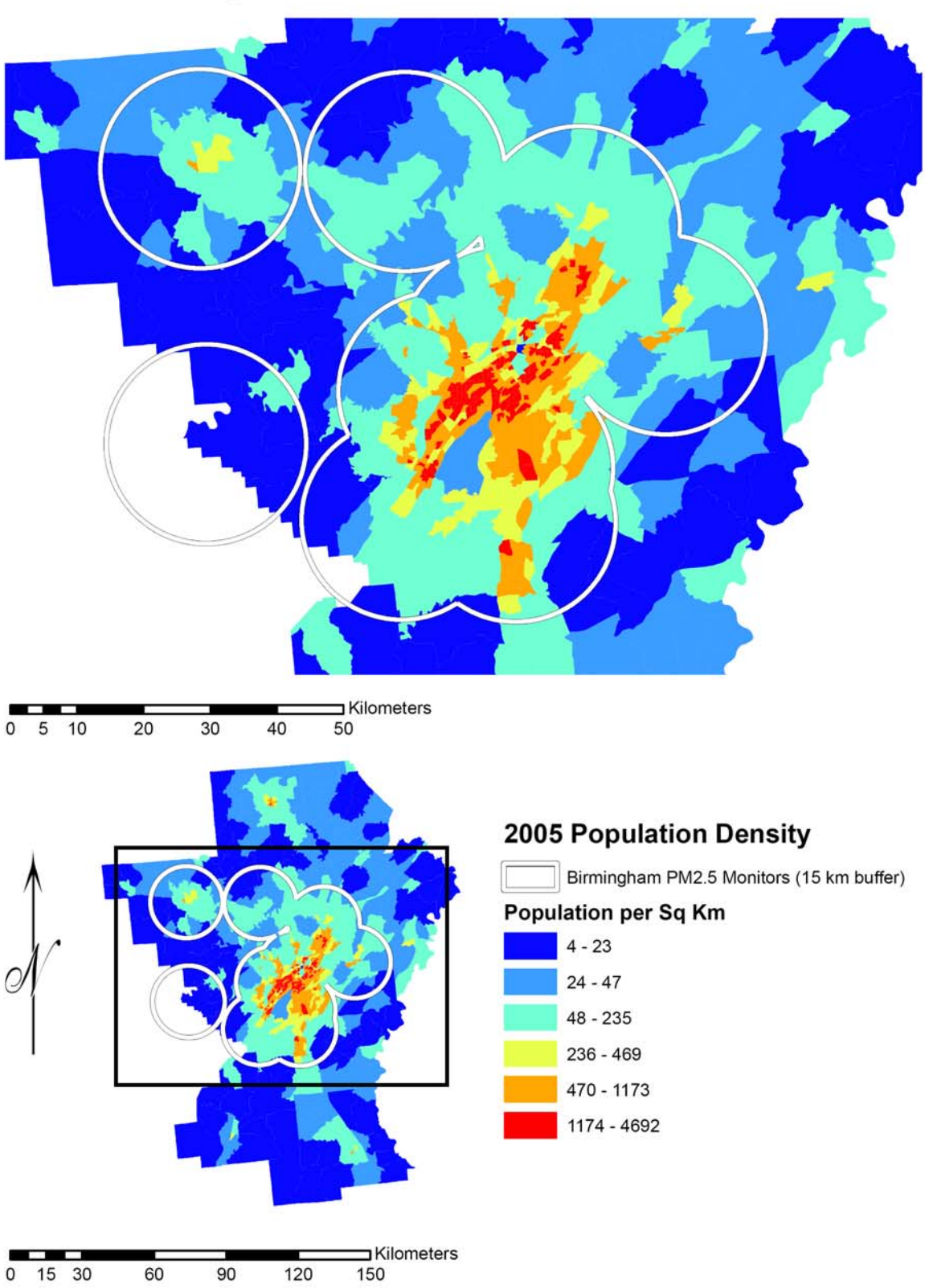


Figure A-3. $PM_{2.5}$ monitor distribution in comparison with population density, Birmingham, AL.

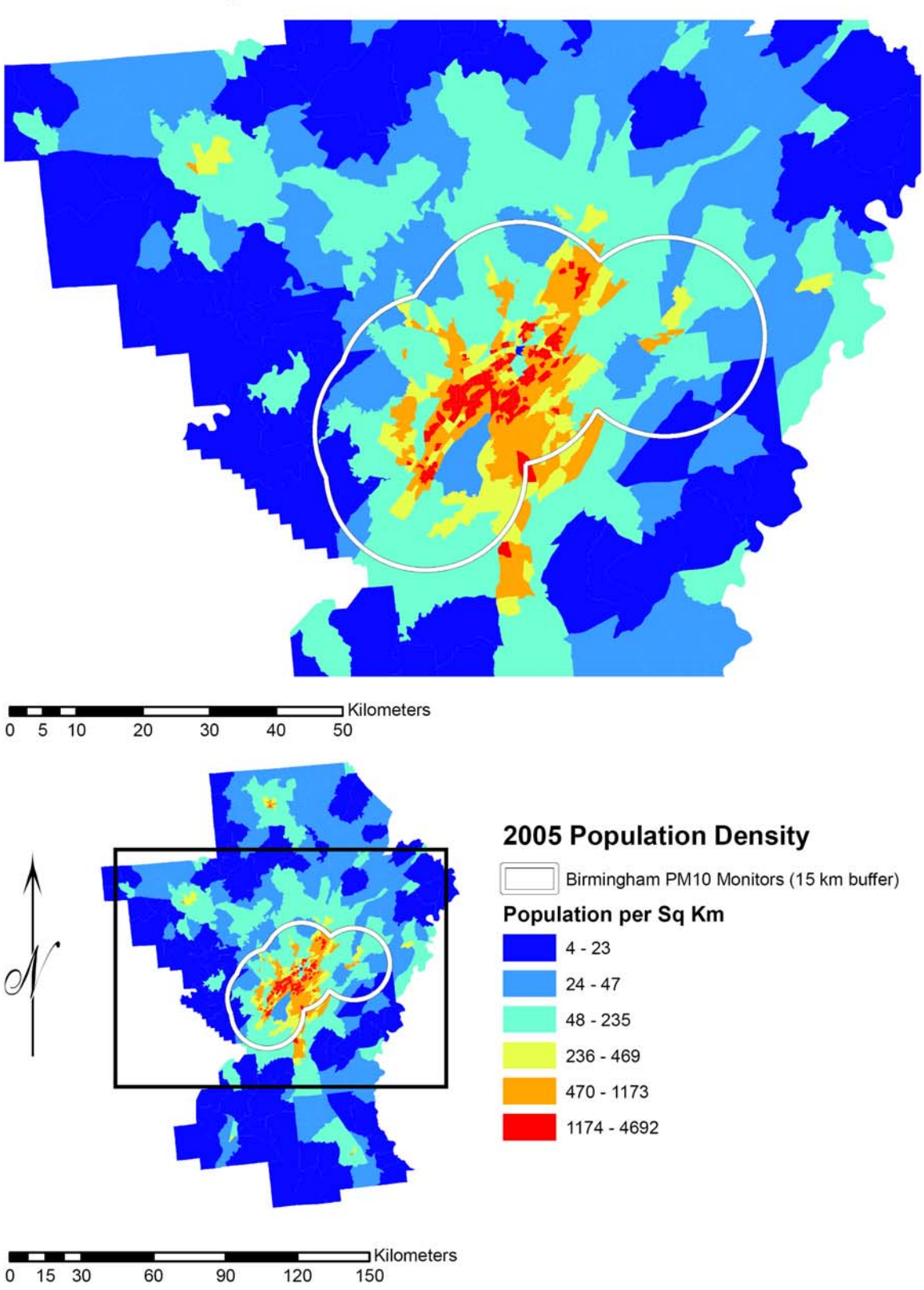


Figure A-4. PM_{10} monitor distribution in comparison with population density, Birmingham, AL.

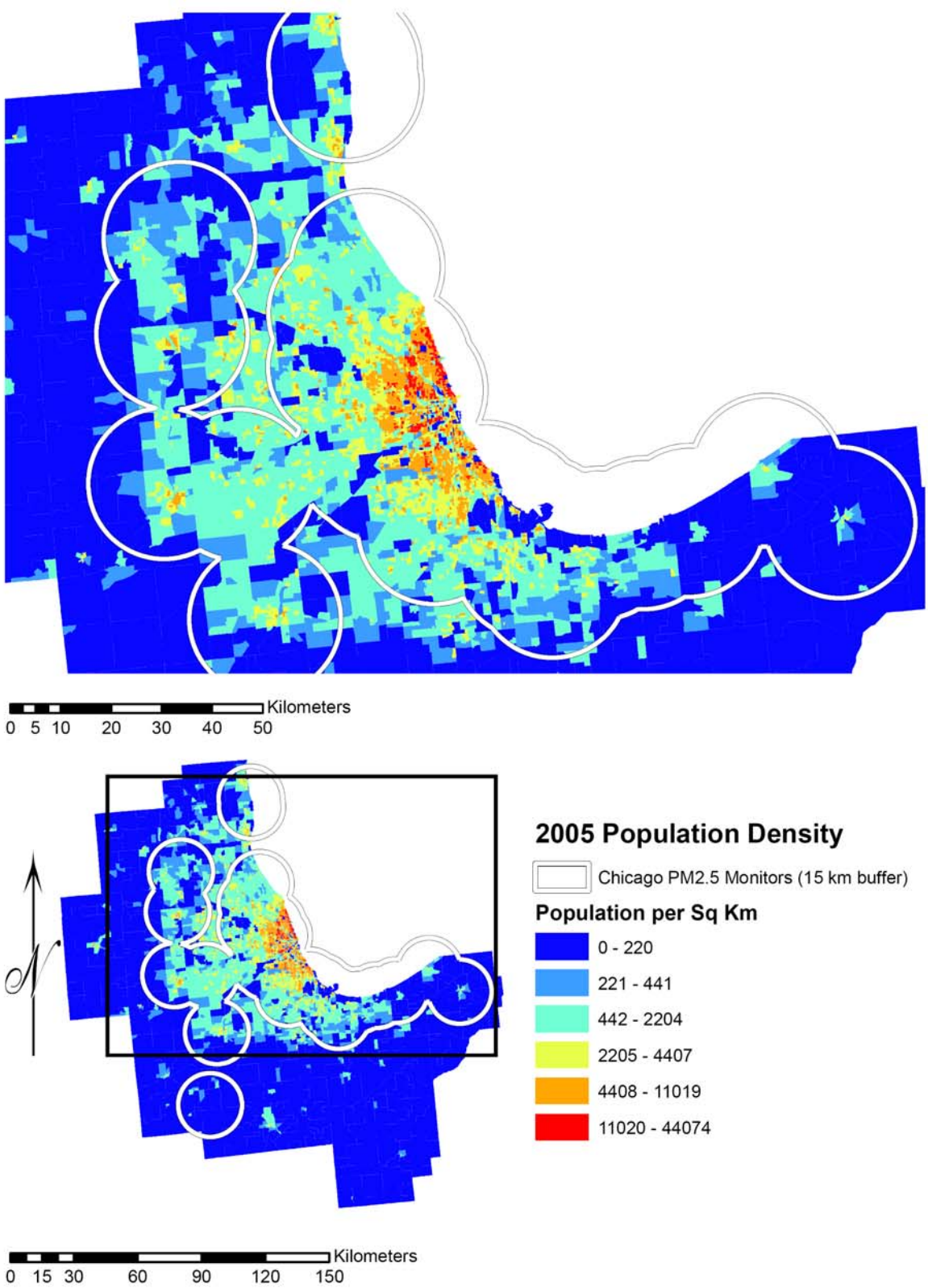


Figure A-5. $PM_{2.5}$ monitor distribution in comparison with population density, Chicago, IL.

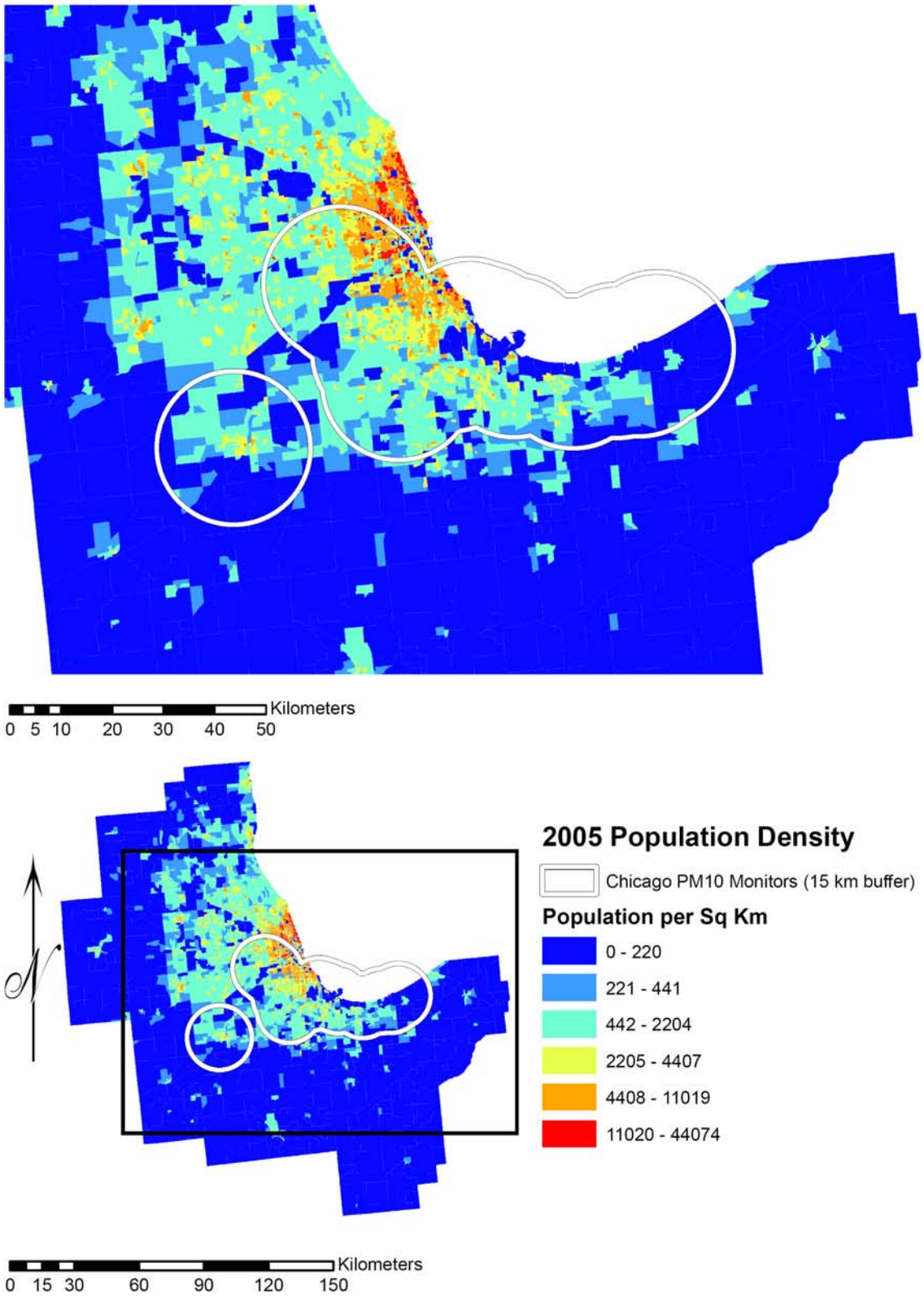


Figure A-6. PM_{10} monitor distribution in comparison with population density, Chicago, IL.

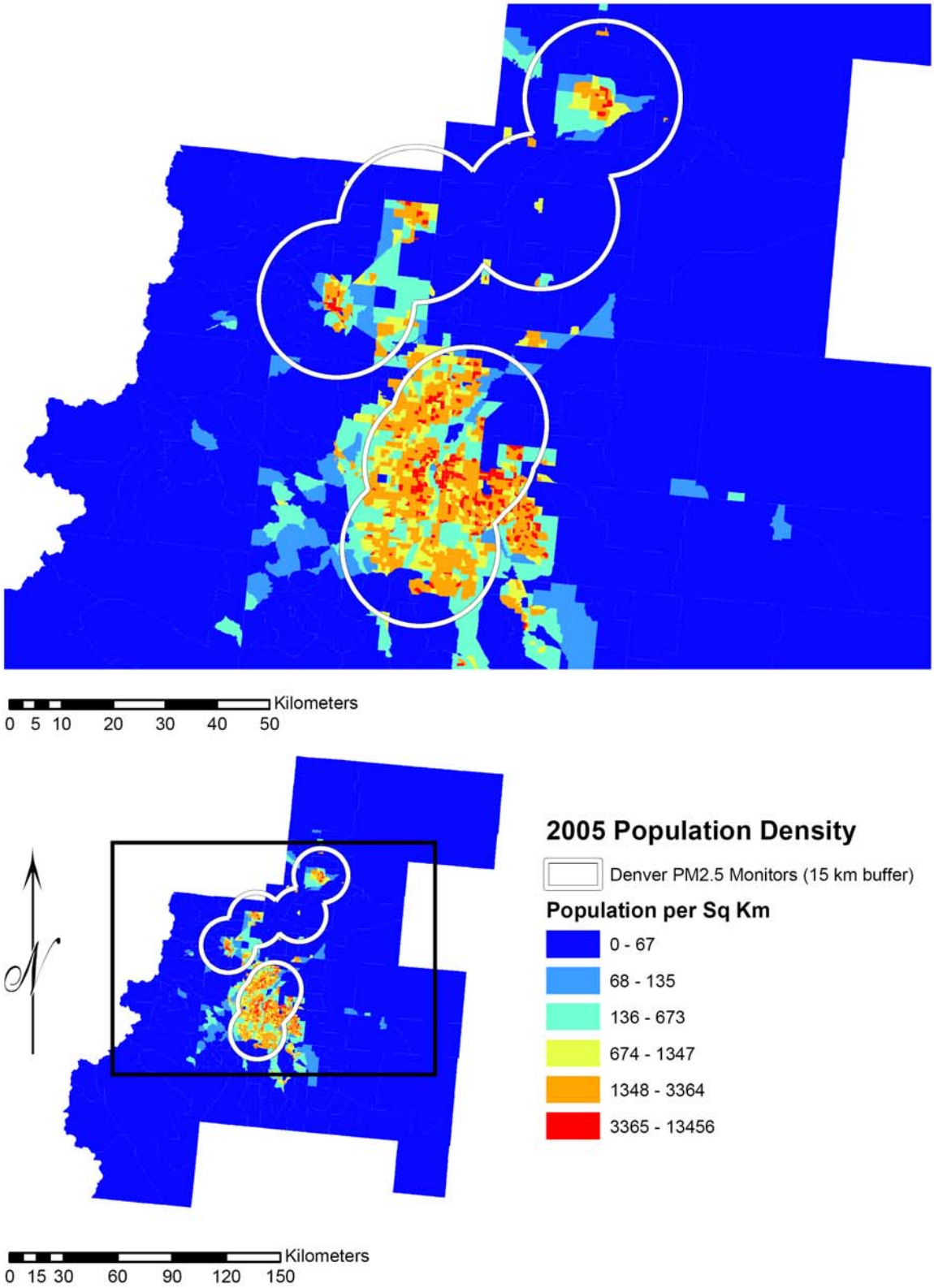


Figure A-7. $PM_{2.5}$ monitor distribution in comparison with population density, Denver, CO.

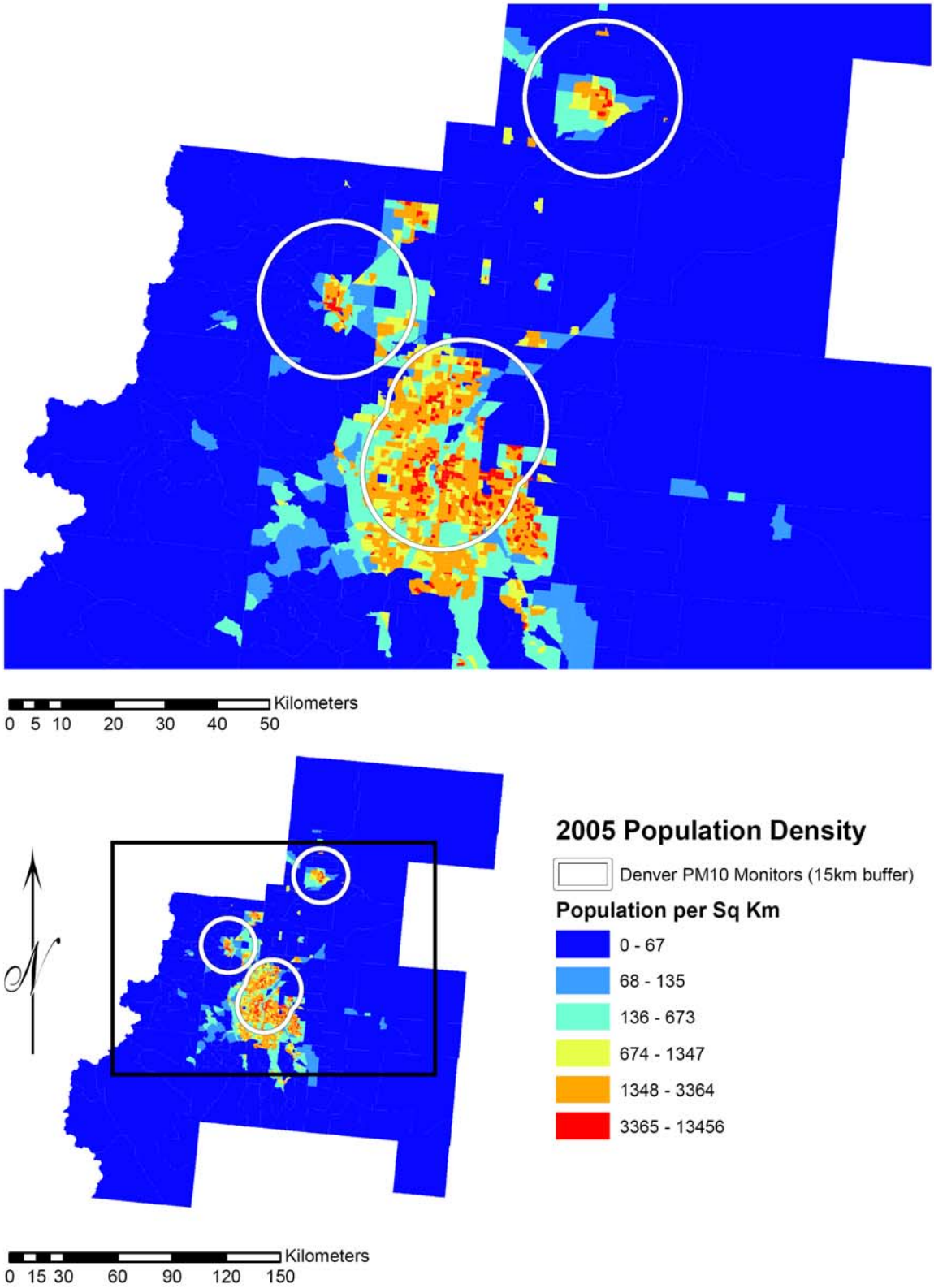


Figure A-8. PM_{10} monitor distribution in comparison with population density, Denver, CO.

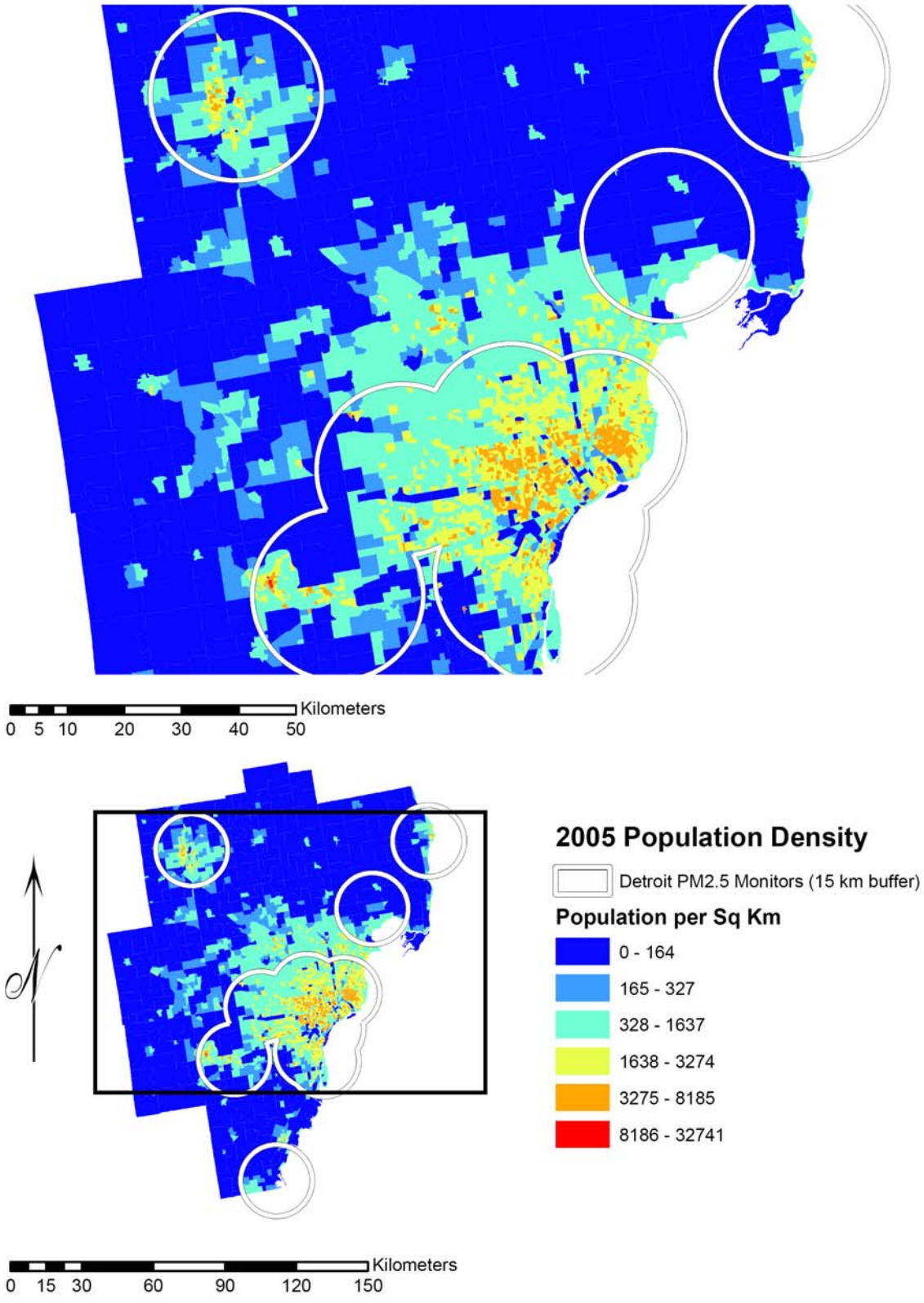


Figure A-9. $PM_{2.5}$ monitor distribution in comparison with population density, Detroit, MI.

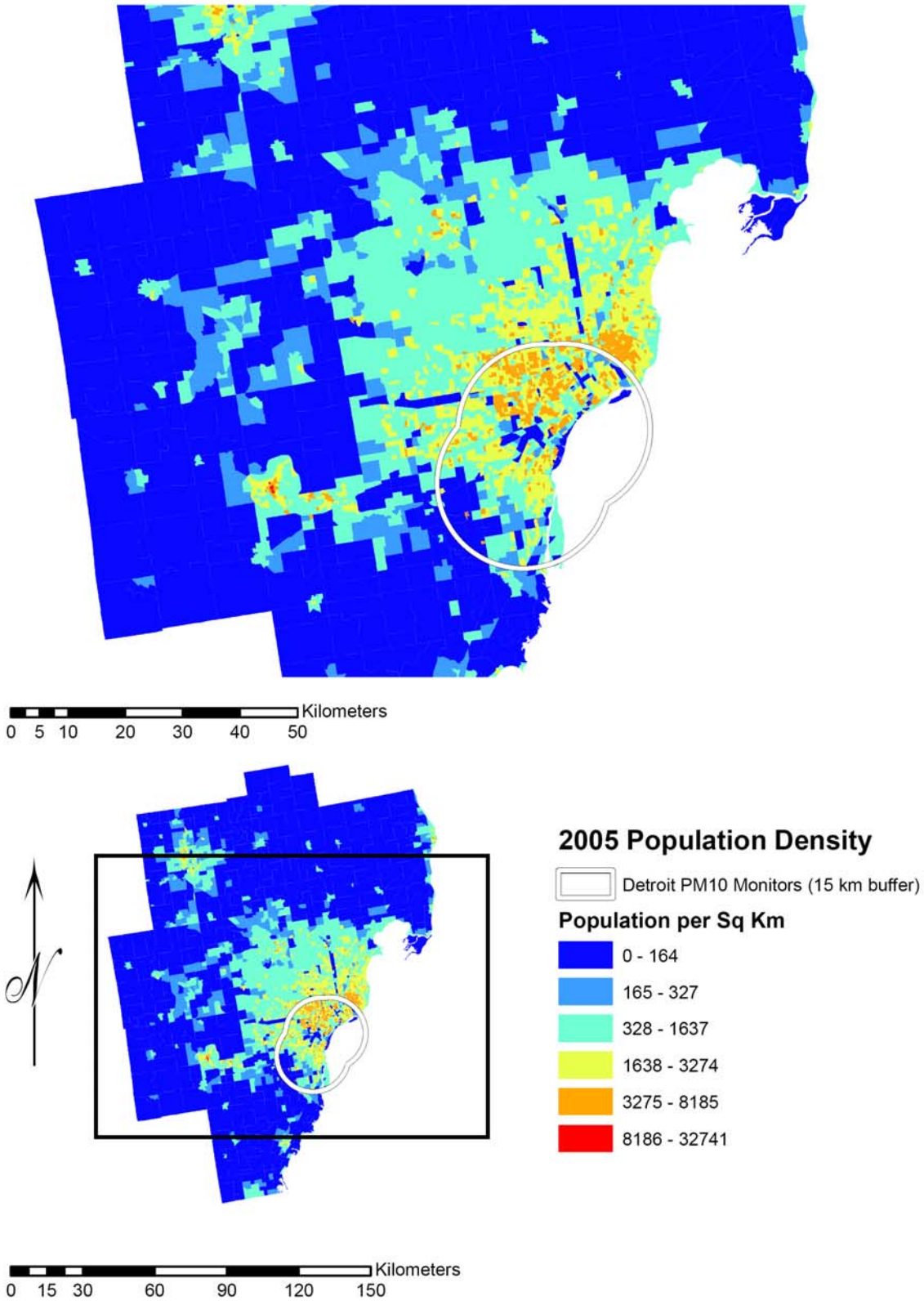


Figure A-10. PM_{10} monitor distribution in comparison with population density, Detroit, MI.

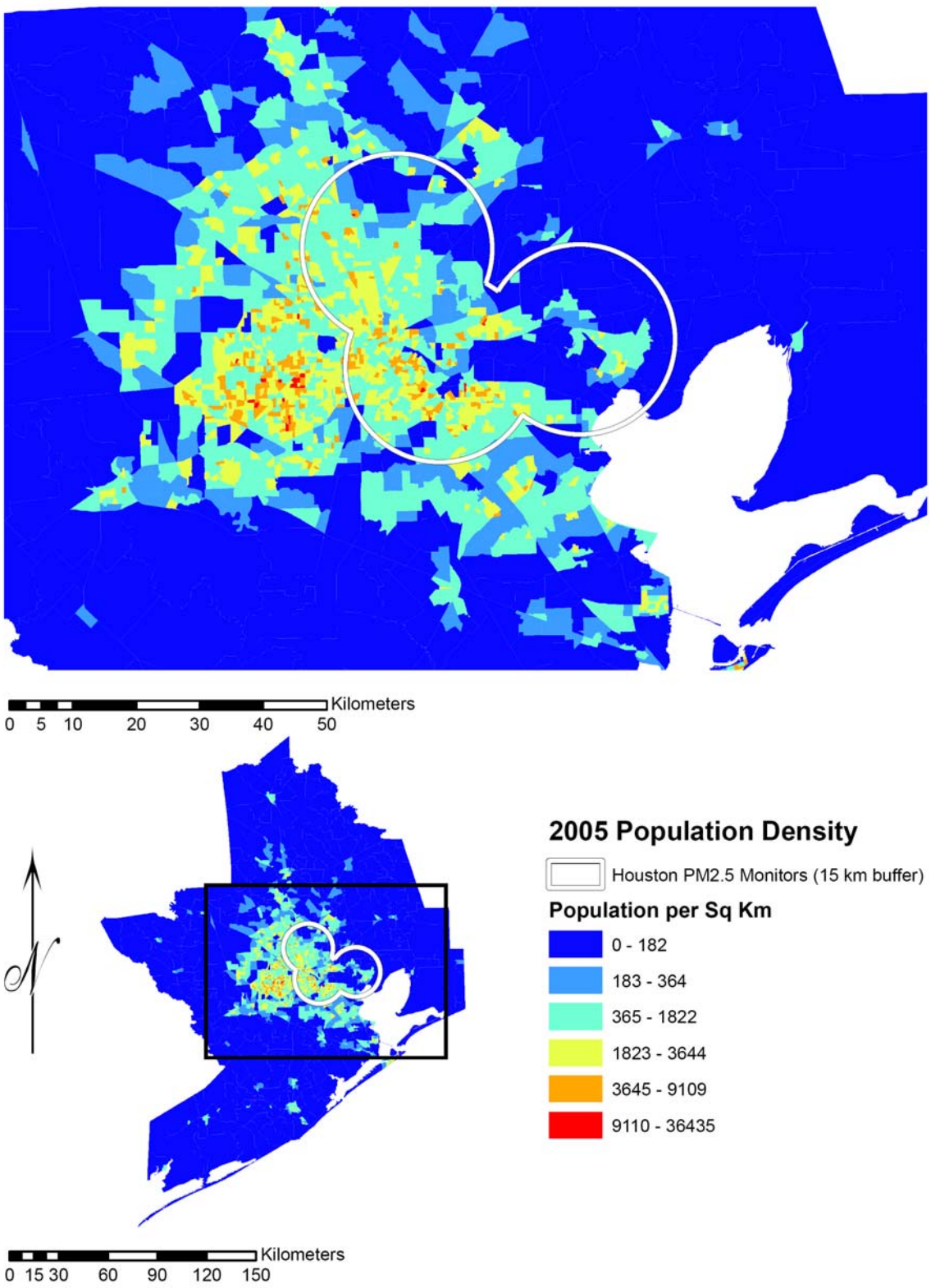


Figure A-11. PM_{10} monitor distribution in comparison with population density, Detroit, MI.

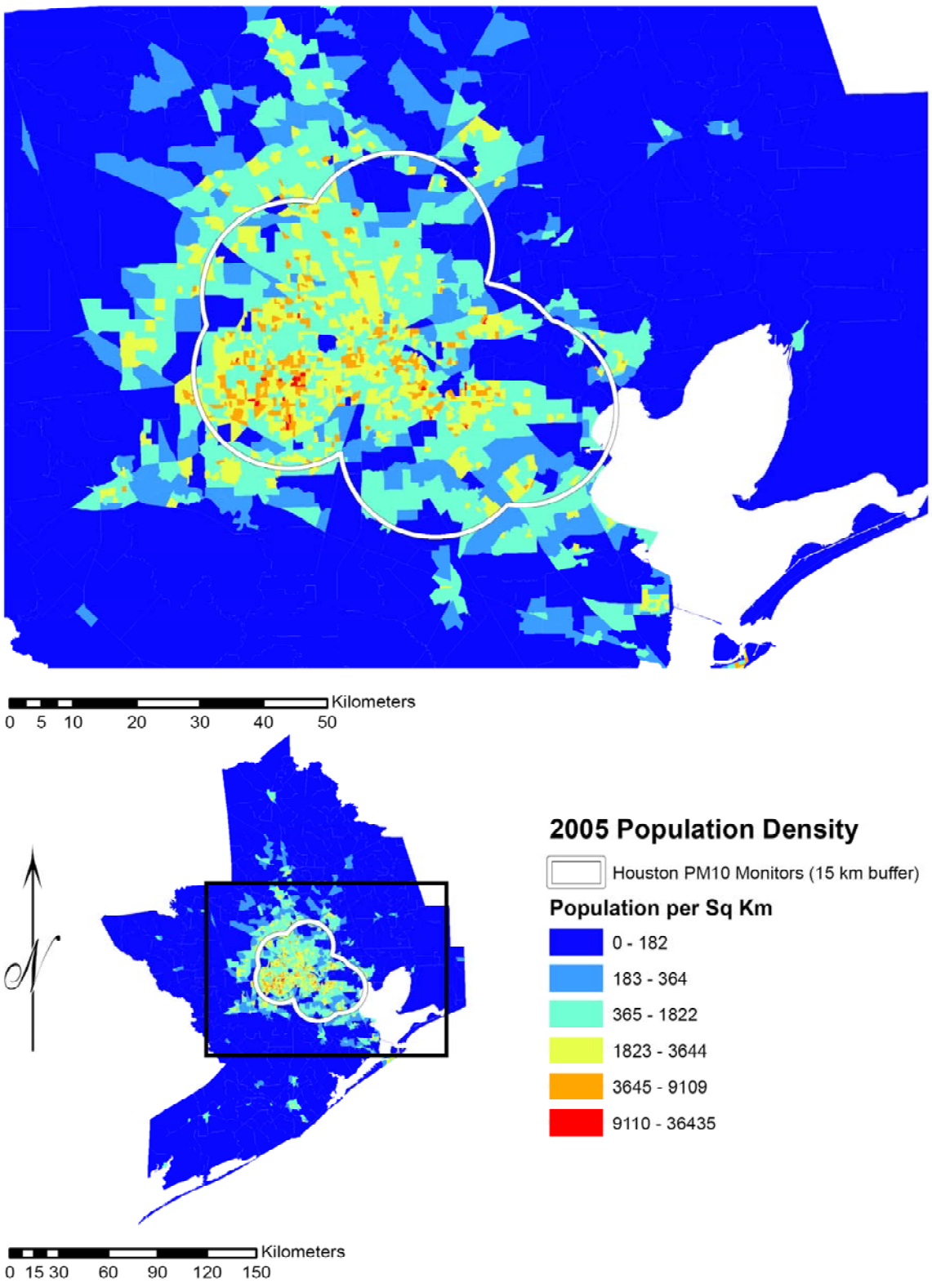


Figure A-12. PM_{10} monitor distribution in comparison with population density, Houston, TX.

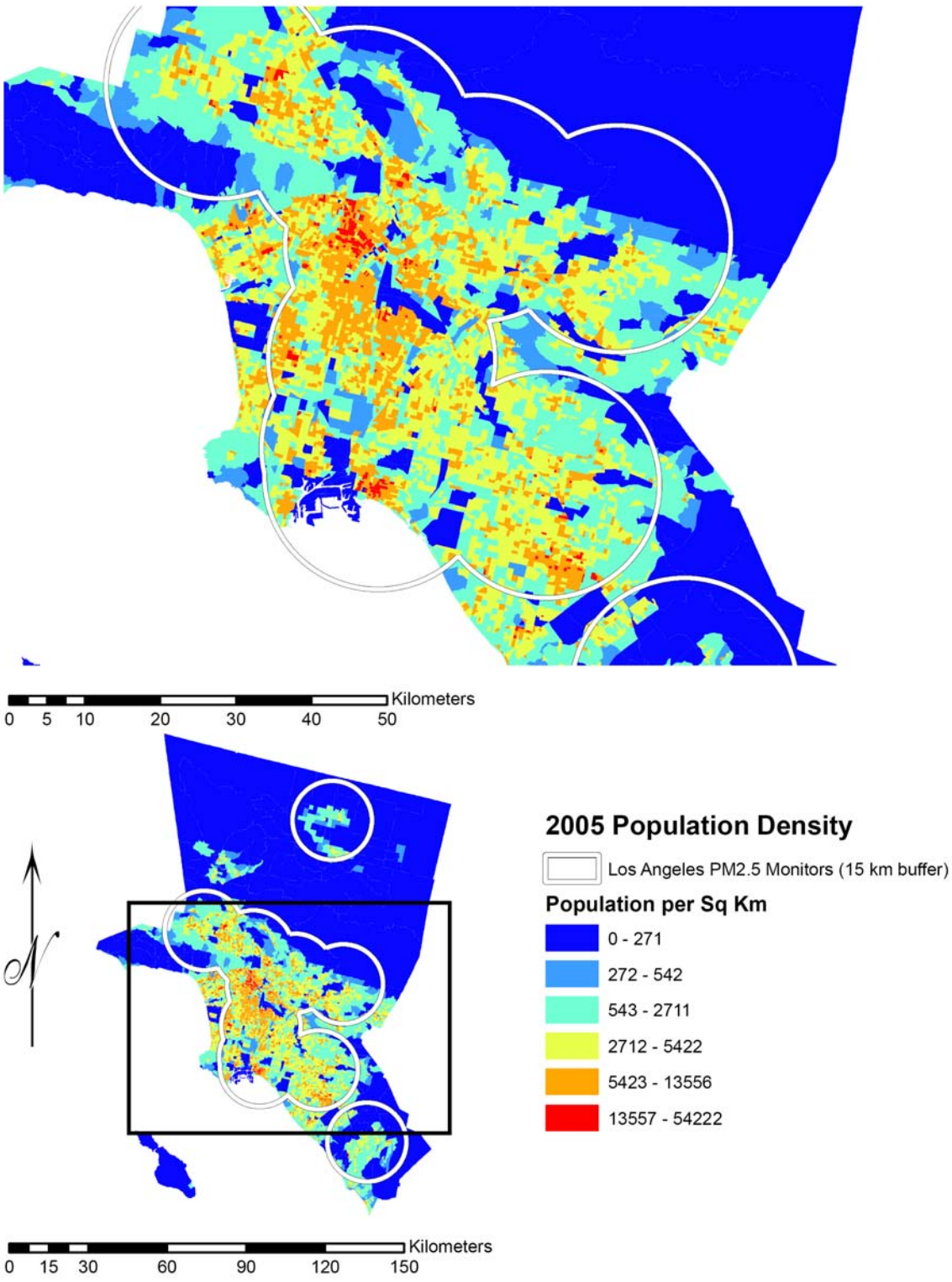


Figure A-13. $PM_{2.5}$ monitor distribution in comparison with population density, Los Angeles, CA.

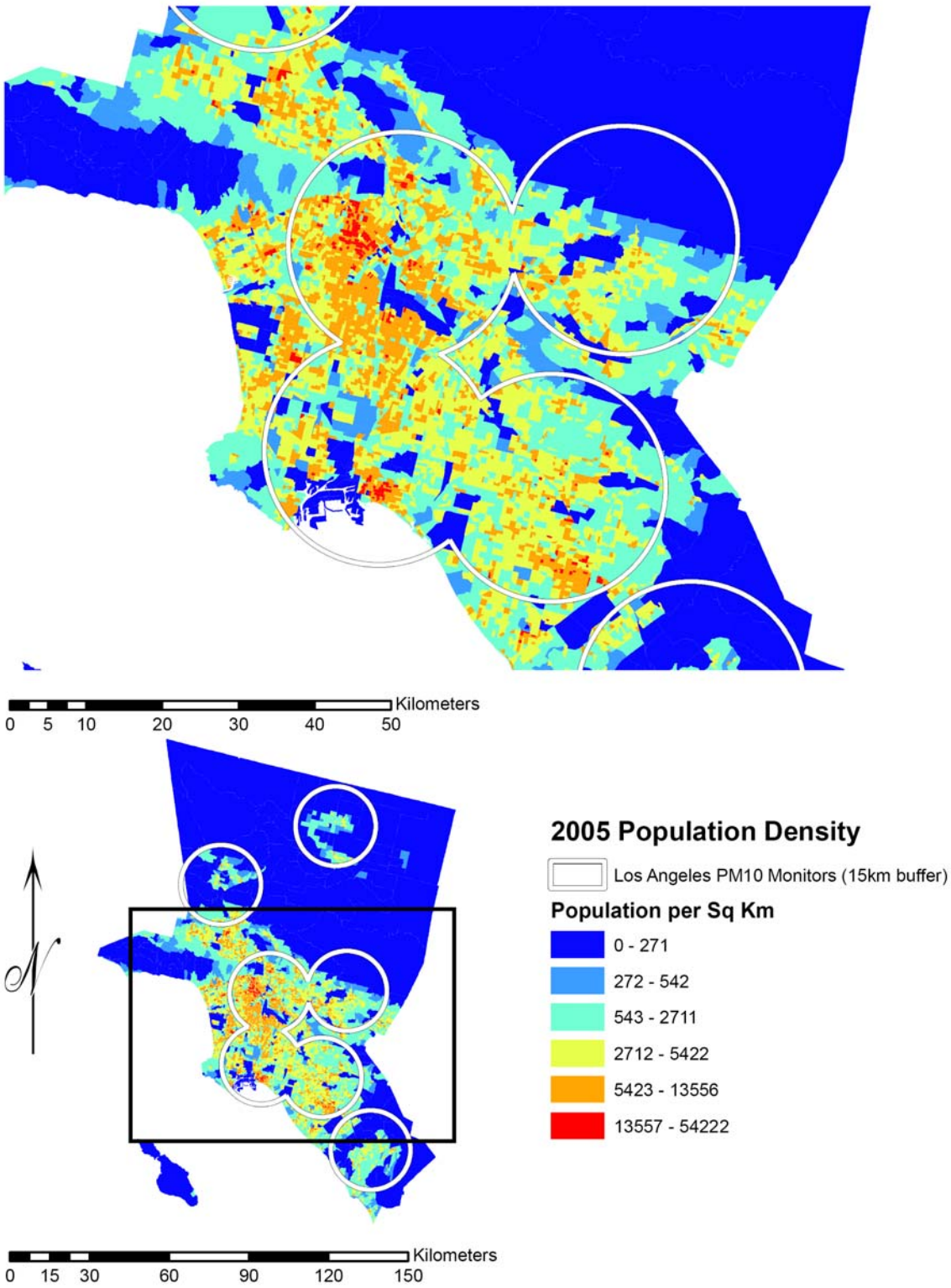


Figure A-14. PM_{10} monitor distribution in comparison with population density, Los Angeles, CA.

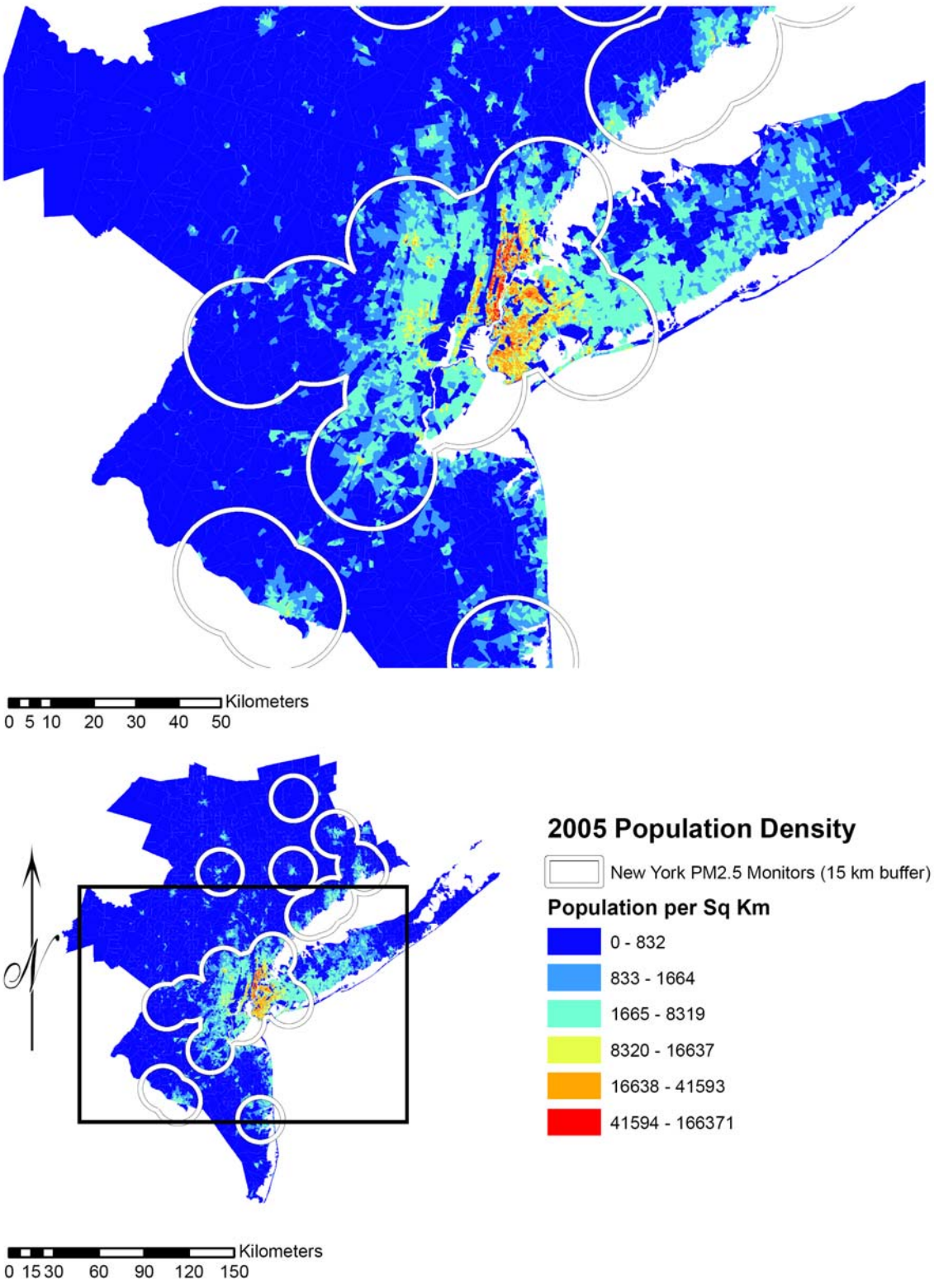


Figure A-15. $PM_{2.5}$ monitor distribution in comparison with population density, New York City, NY.

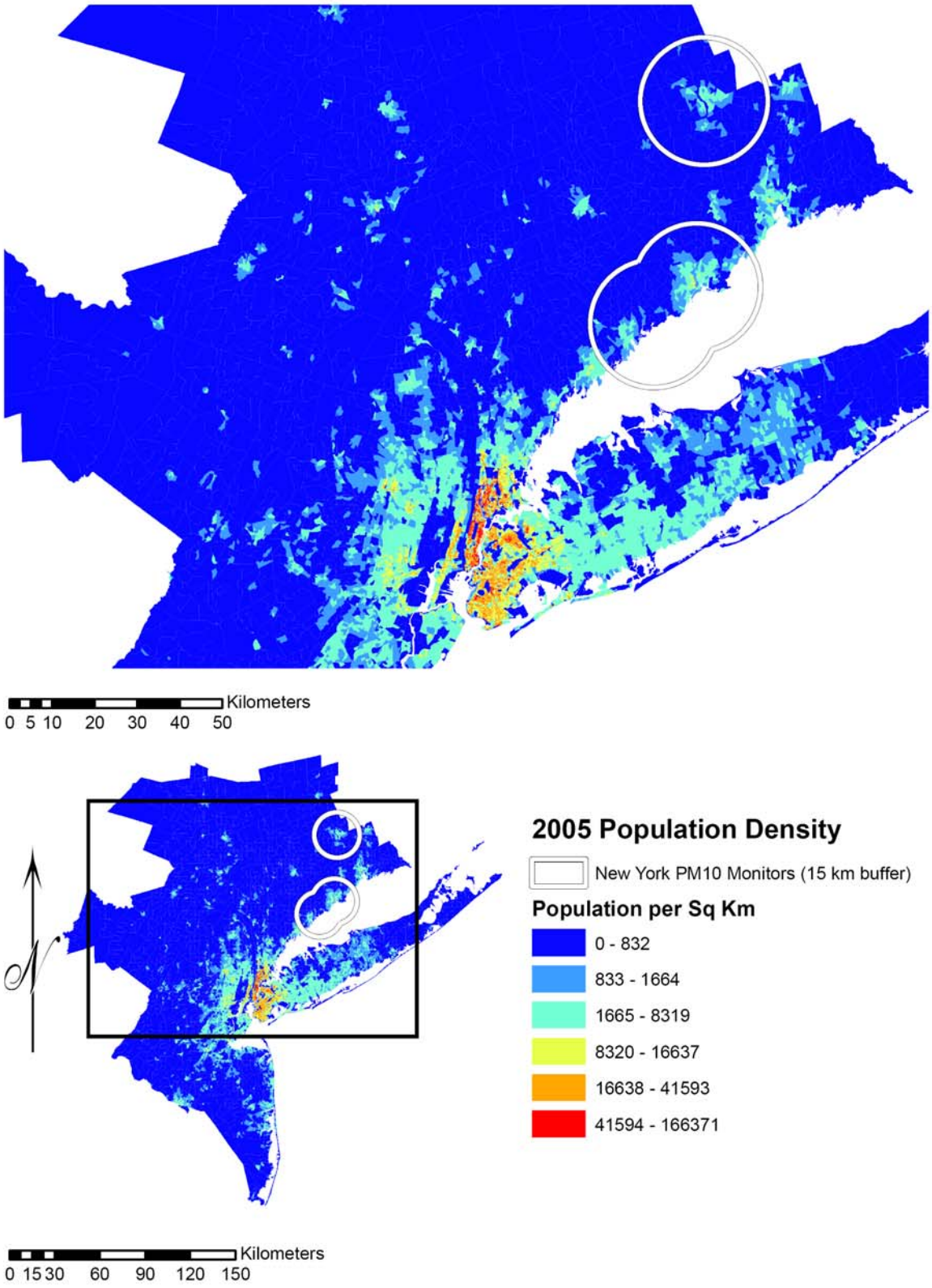


Figure A-16. PM_{10} monitor distribution in comparison with population density, New York City, NY.

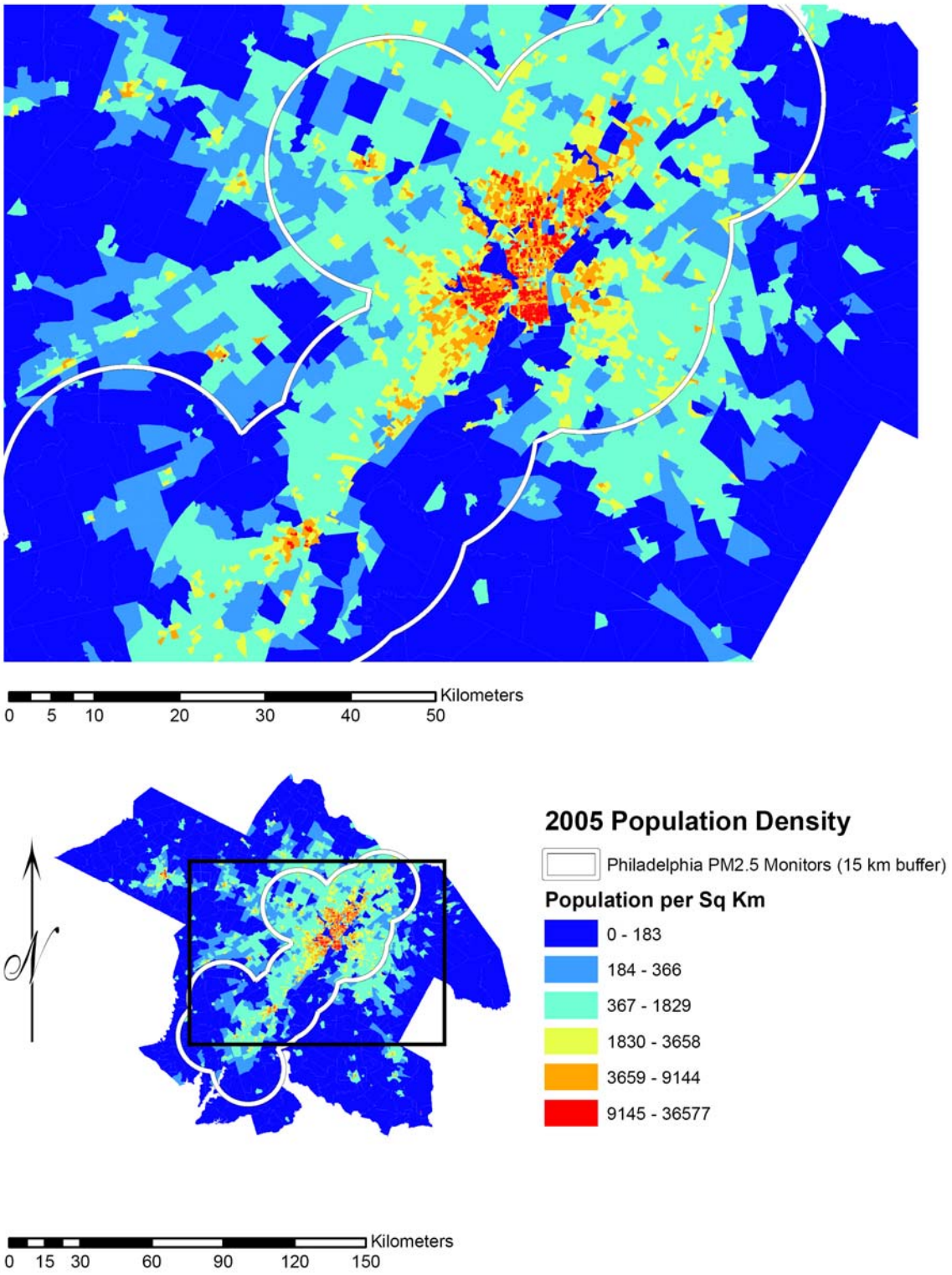


Figure A-17. $PM_{2.5}$ monitor distribution in comparison with population density, Philadelphia, PA.

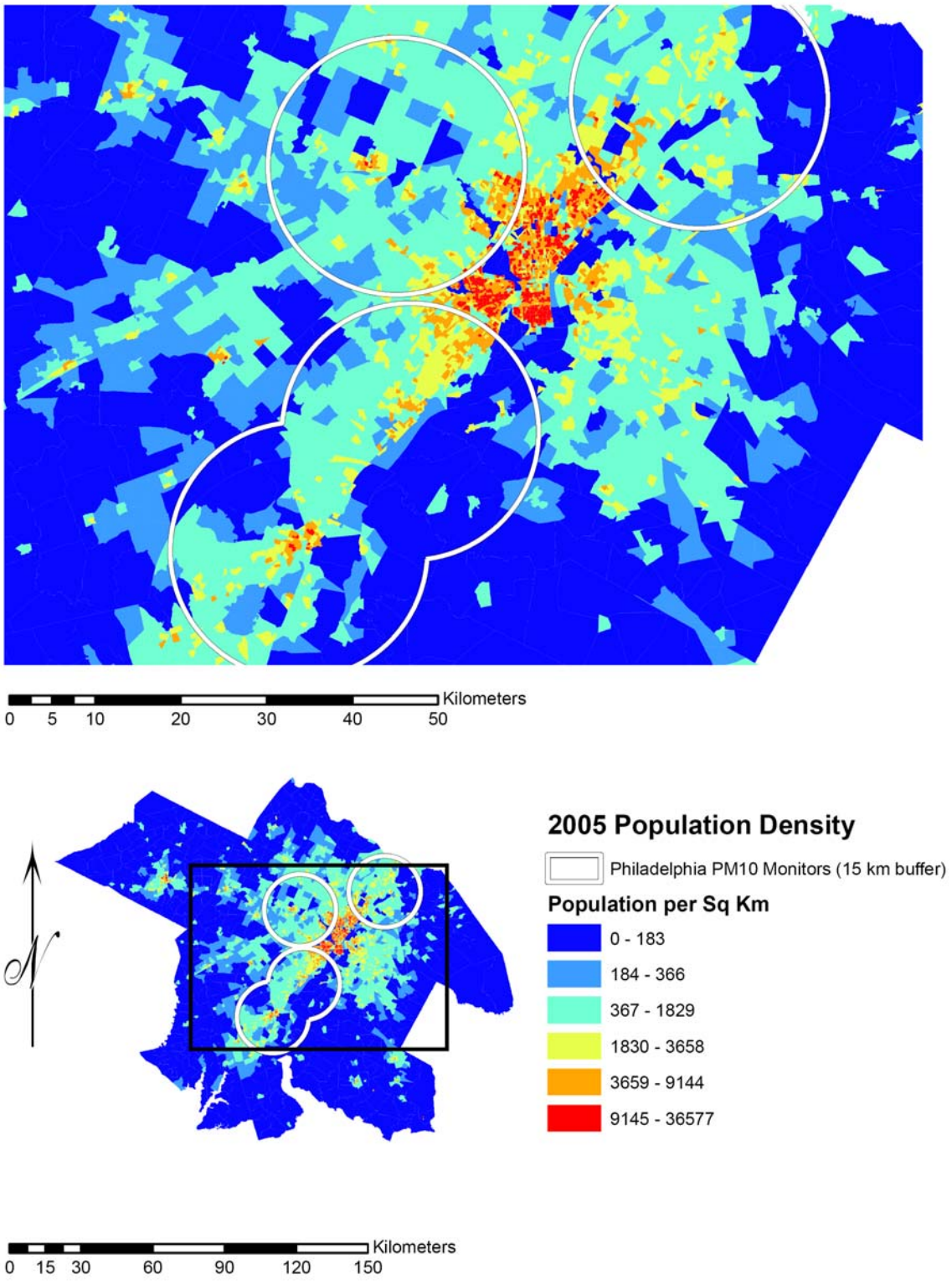


Figure A-18. PM_{10} monitor distribution in comparison with population density, Philadelphia, PA.

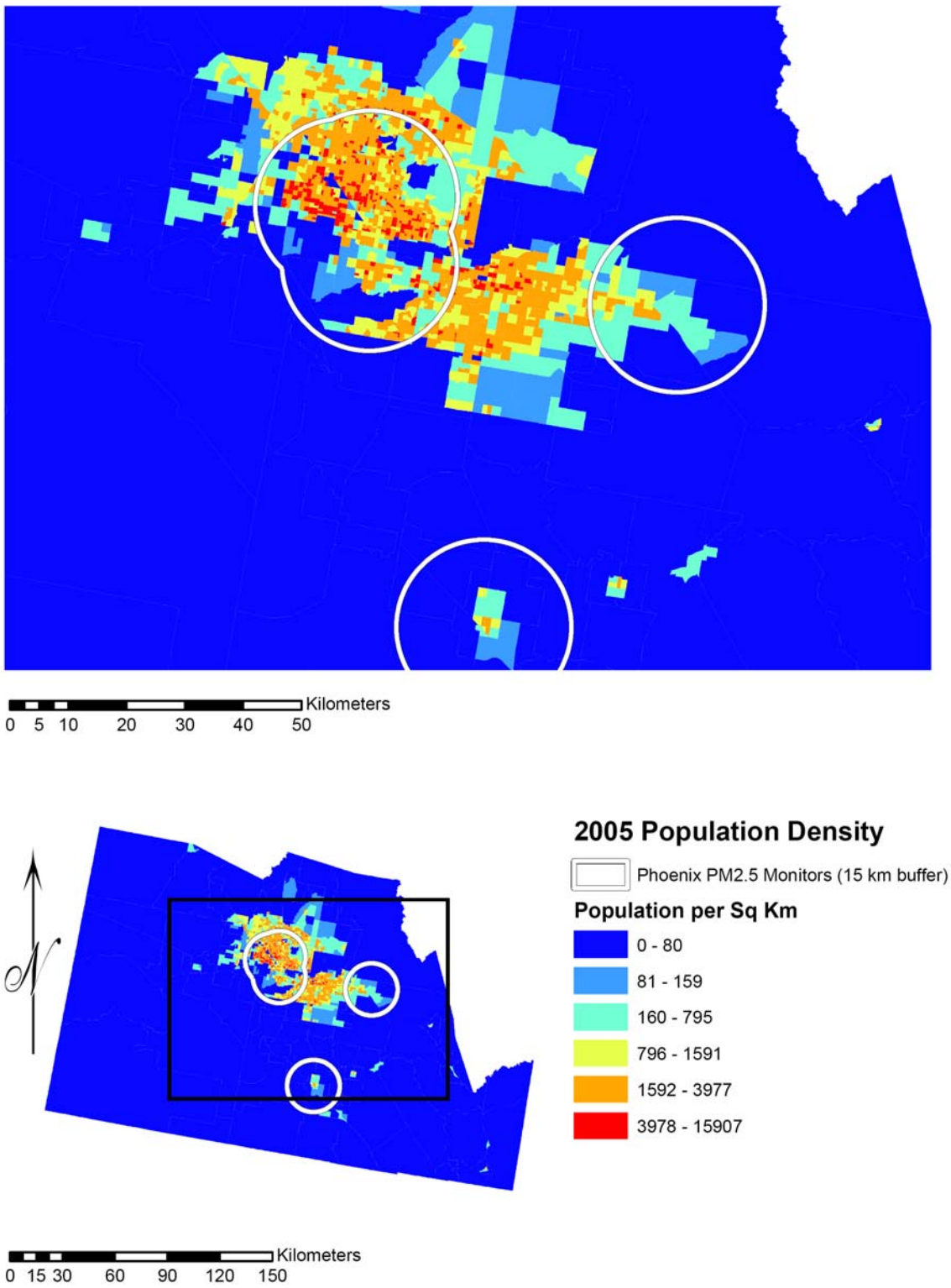


Figure A-19. $PM_{2.5}$ monitor distribution in comparison with population density, Phoenix, AZ.

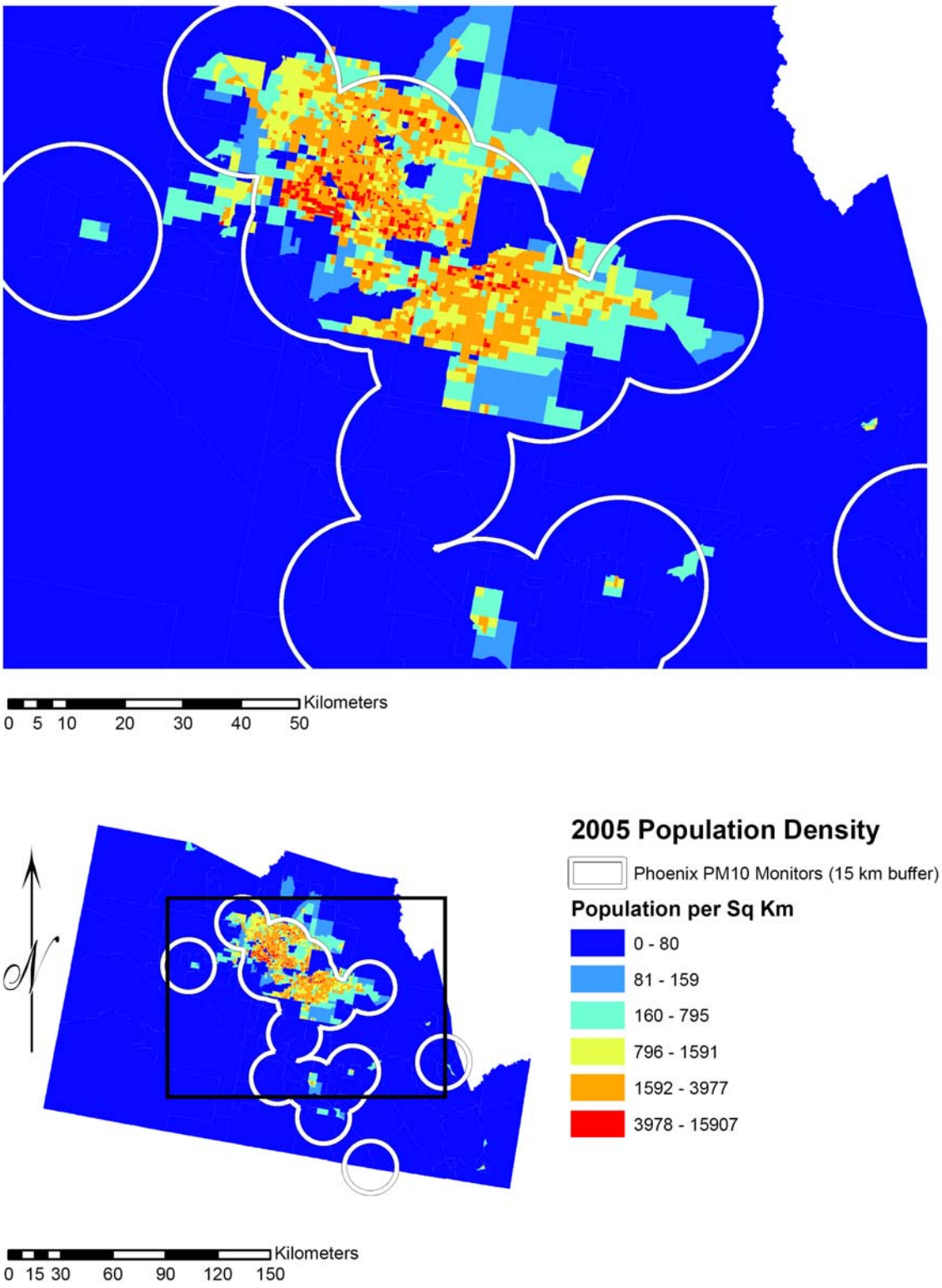


Figure A-20. PM_{10} monitor distribution in comparison with population density, Phoenix, AZ.

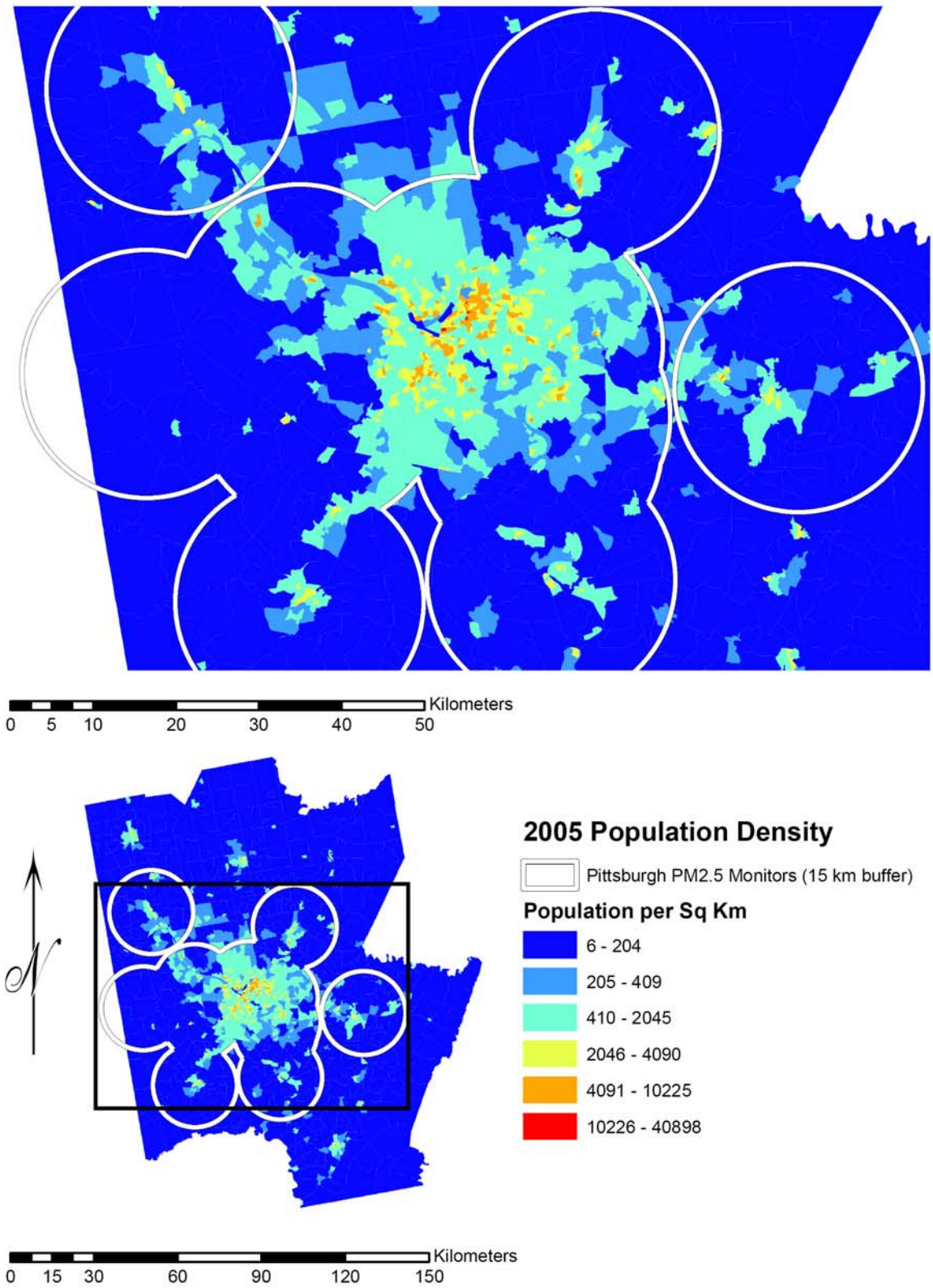


Figure A-21. $PM_{2.5}$ monitor distribution in comparison with population density, Pittsburgh, PA.

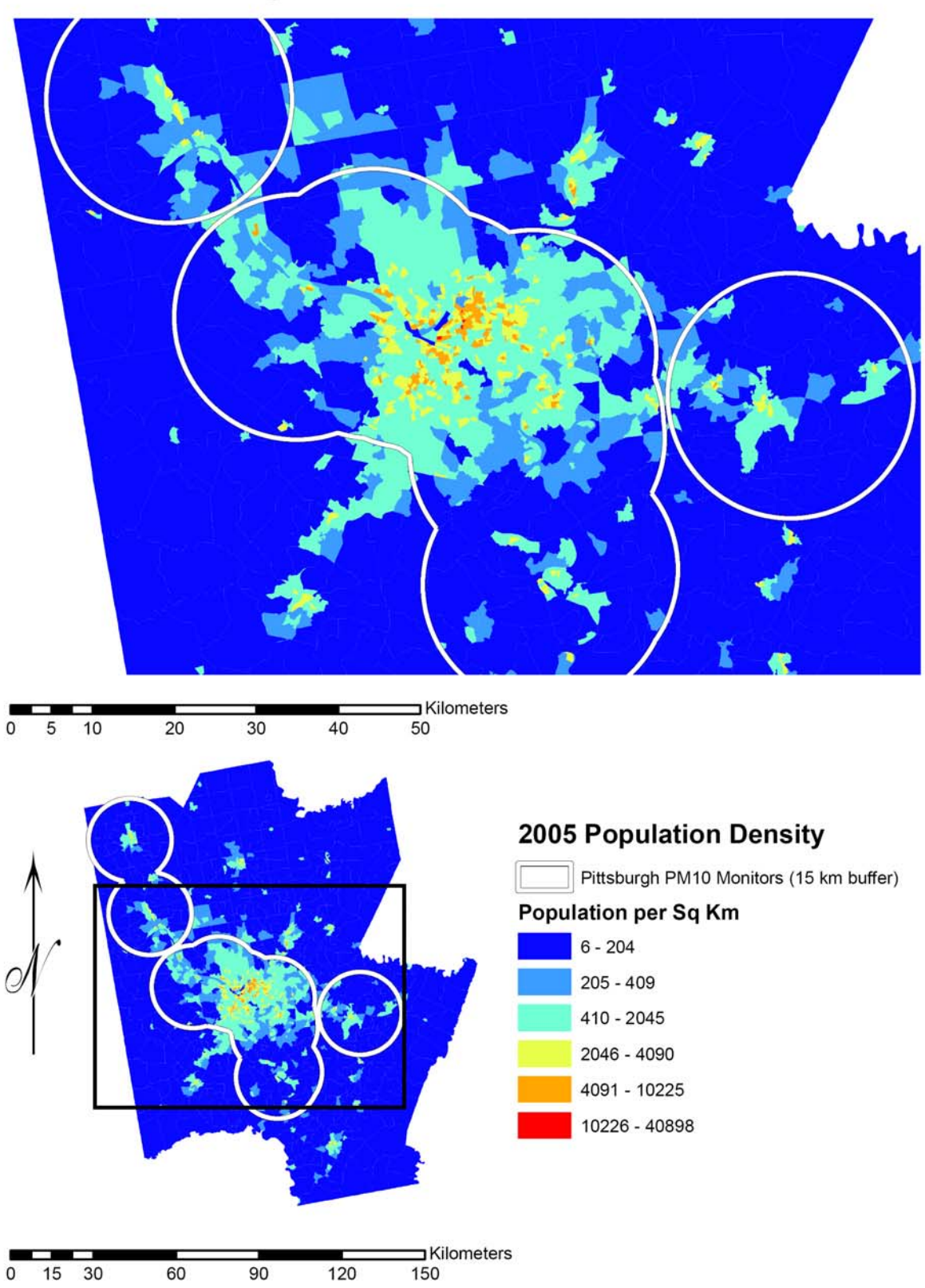


Figure A-22. PM_{10} monitor distribution in comparison with population density, Pittsburgh, PA.

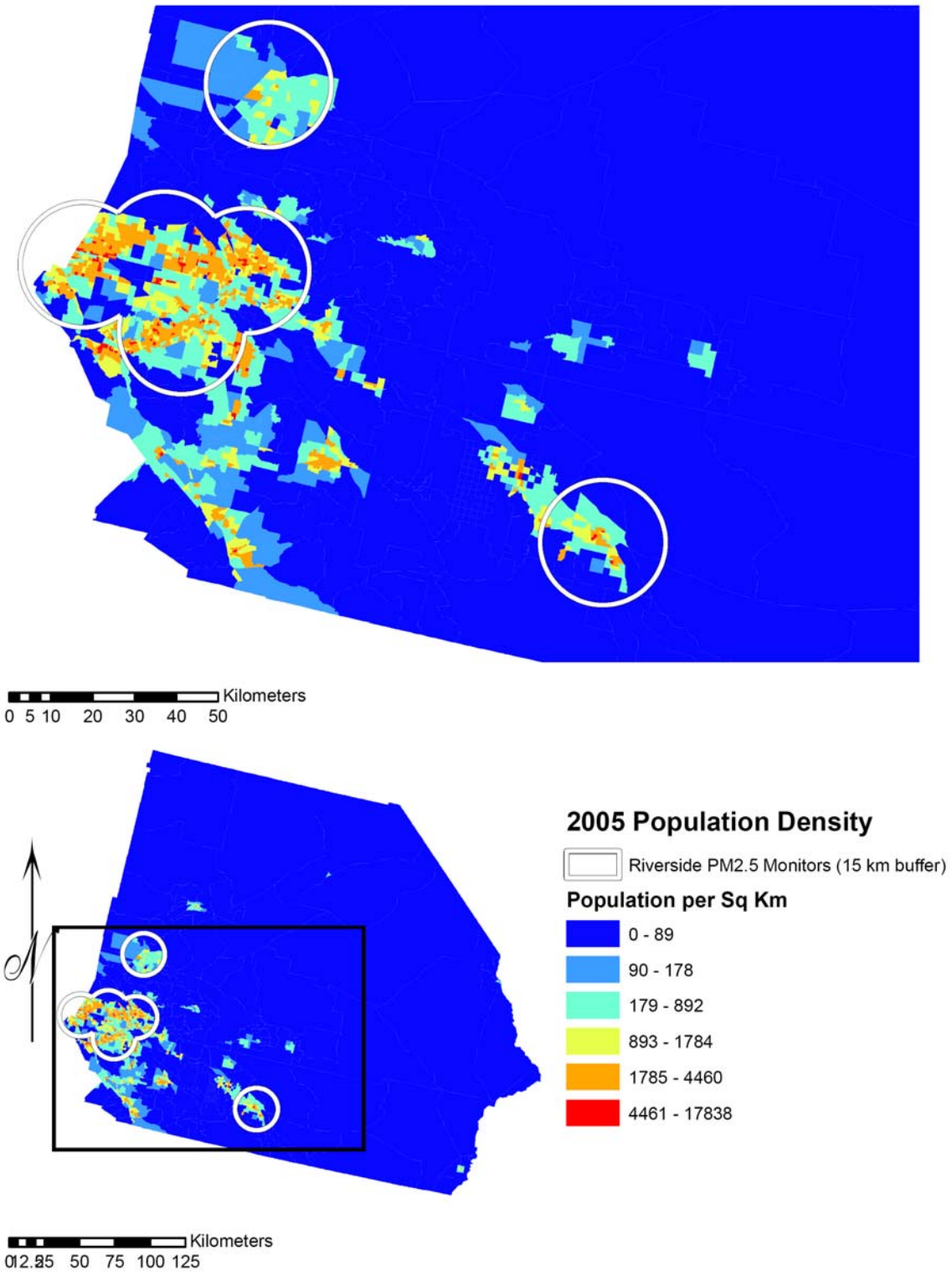


Figure A-23. $PM_{2.5}$ monitor distribution in comparison with population density, Riverside, CA.

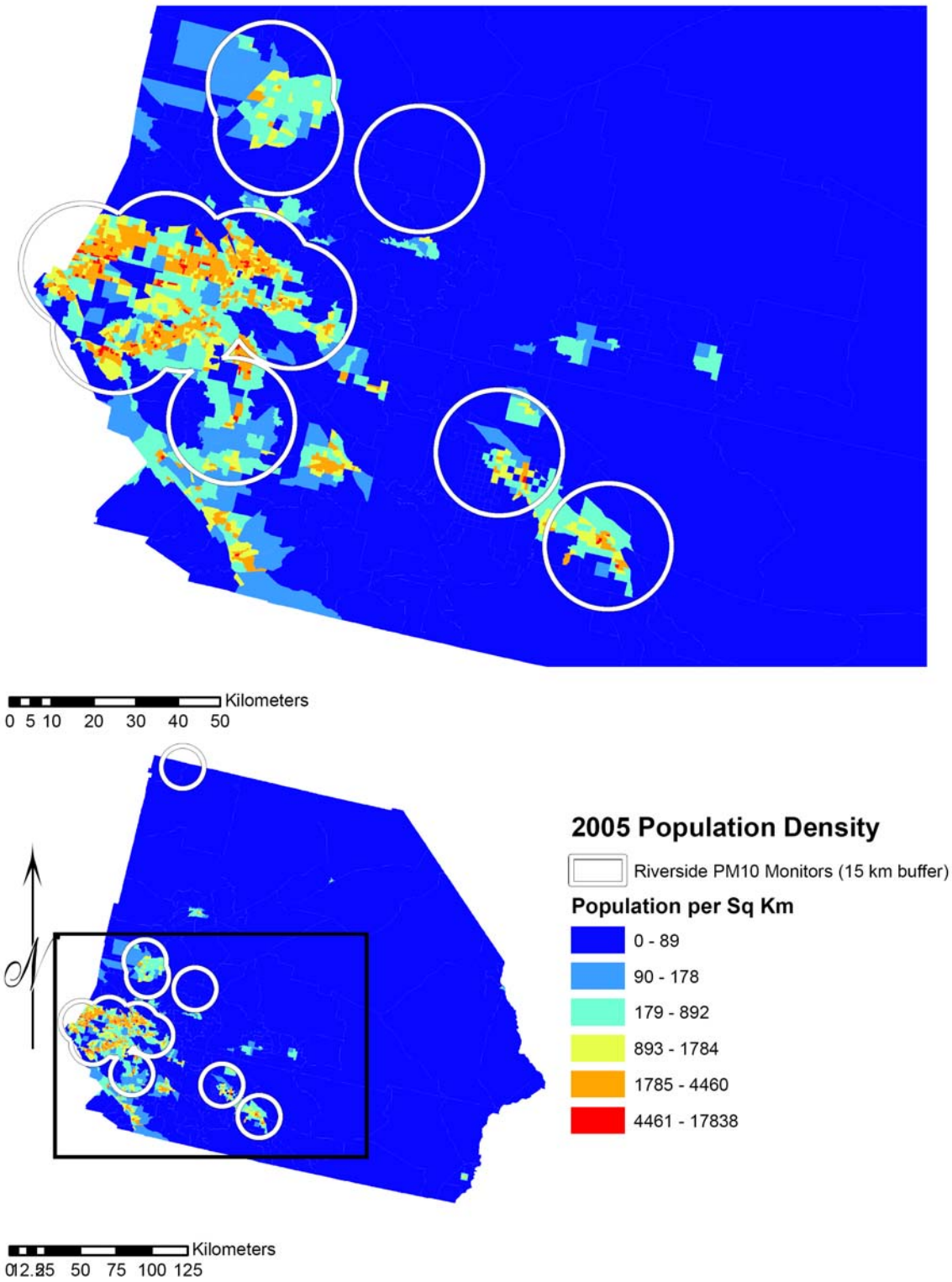


Figure A-24. PM_{10} monitor distribution in comparison with population density, Riverside, CA.

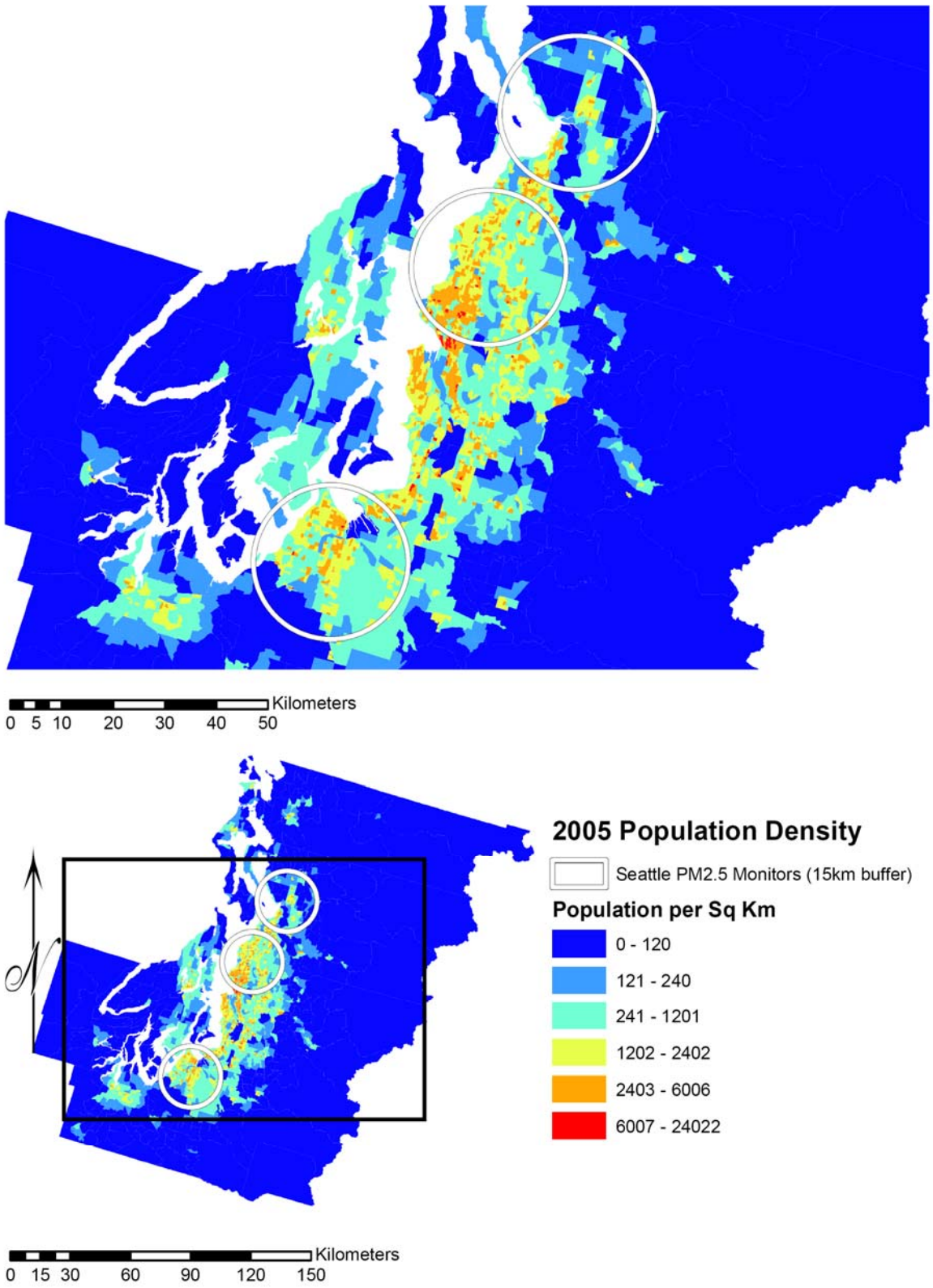


Figure A-25. $PM_{2.5}$ monitor distribution in comparison with population density, Seattle, WA.

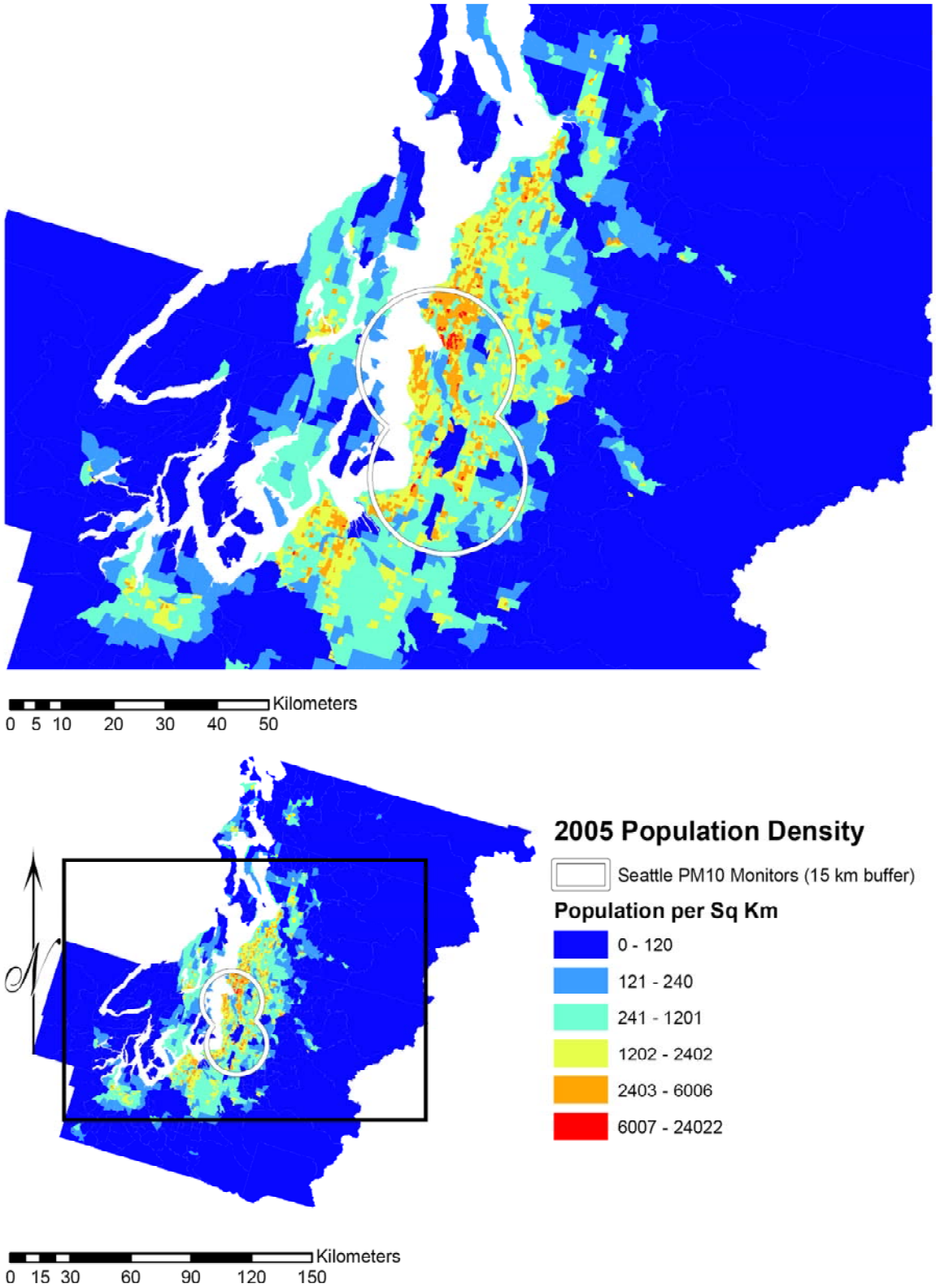


Figure A-26. $PM_{2.5}$ monitor distribution in comparison with population density, Seattle, WA.

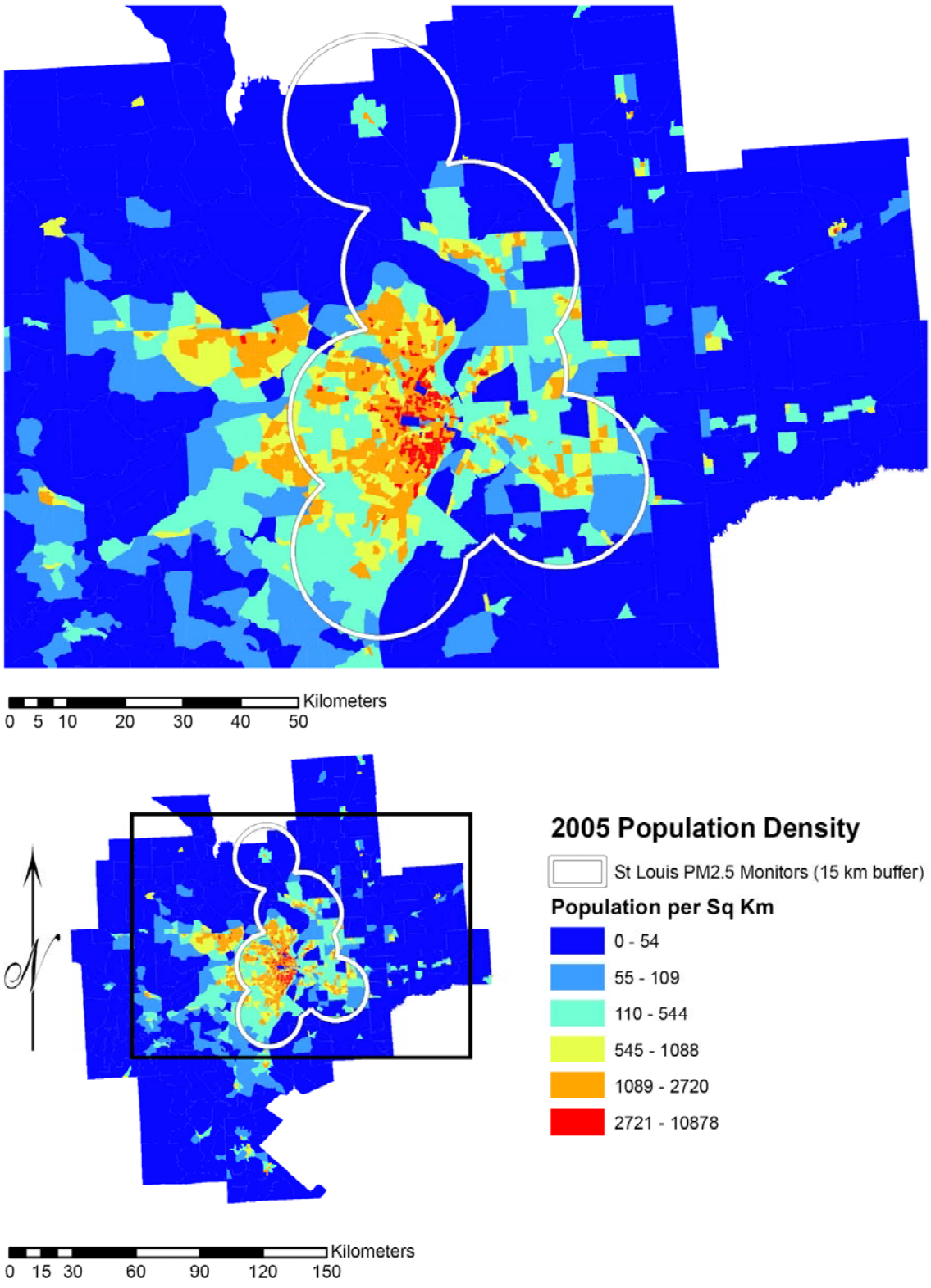


Figure A-27. $PM_{2.5}$ monitor distribution in comparison with population density, St. Louis, MO.

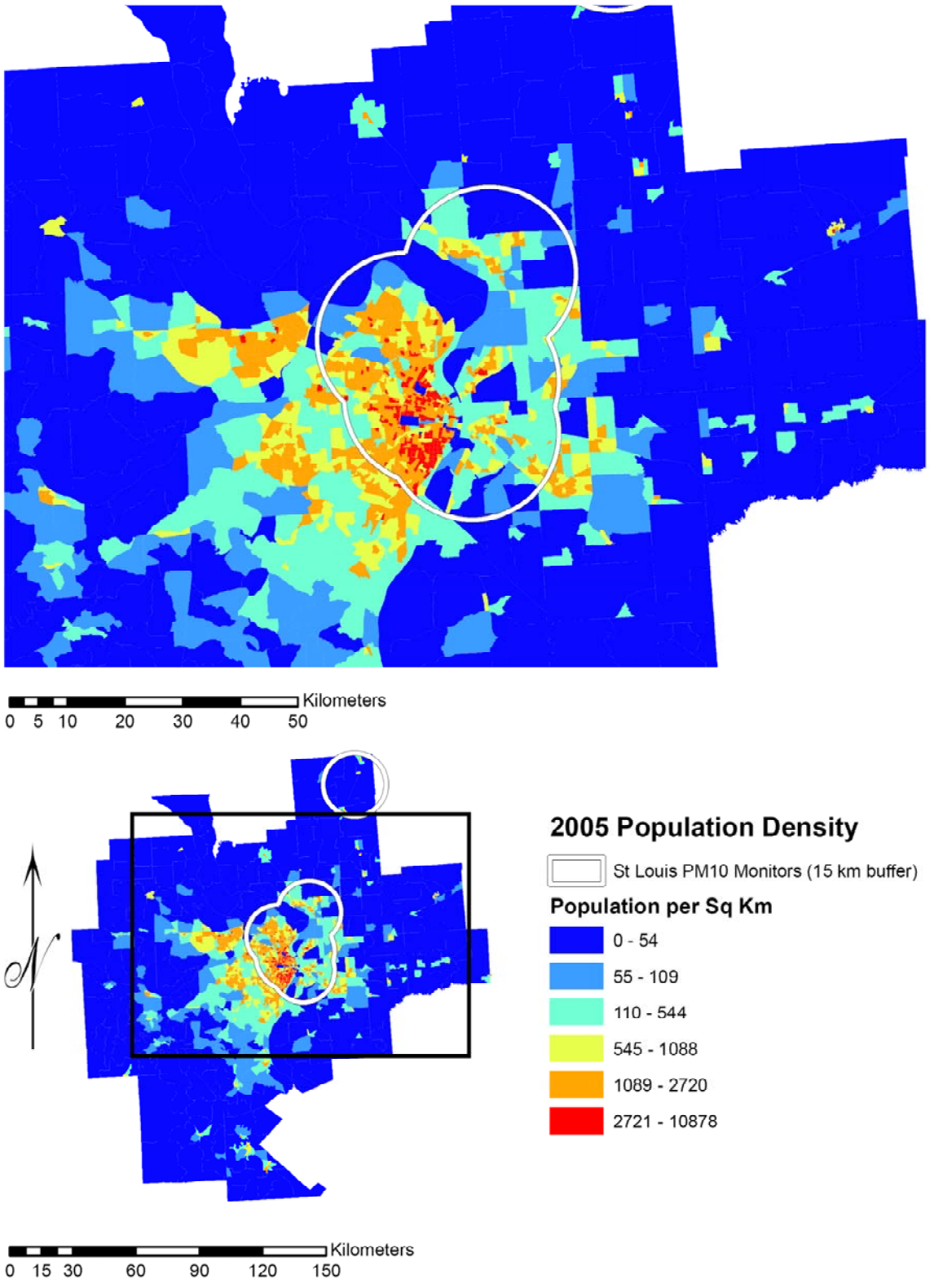


Figure A-28. PM_{10} monitor distribution in comparison with population density, St. Louis, MO.

A.2. Ambient PM Concentration

A.2.1. Speciation Trends Network Site Data

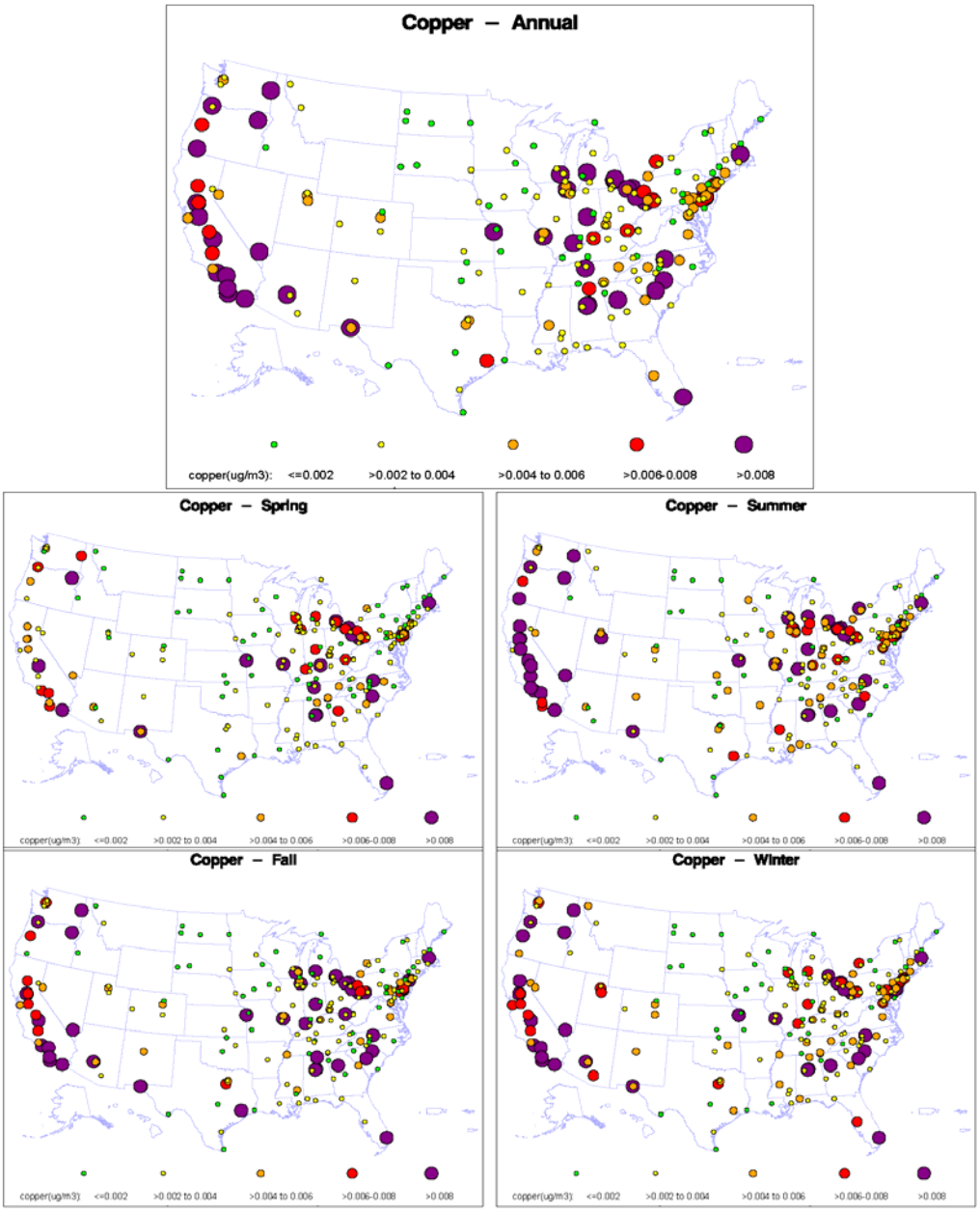


Figure A-29. Three-yr avg of 24-h $PM_{2.5}$ Cu concentrations measured at CSN sites across the U.S., 2005-2007.

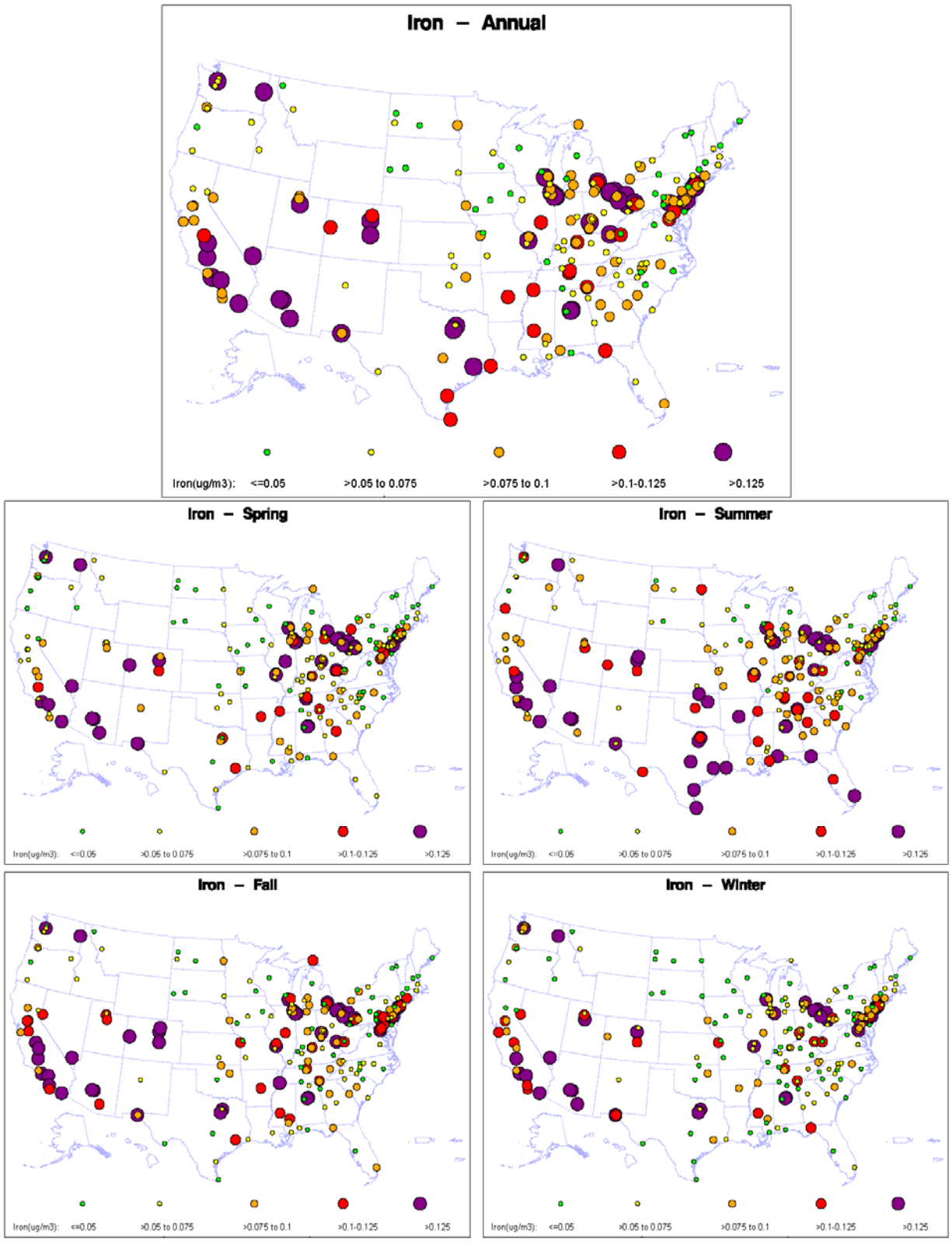


Figure A-30. Three-yr avg of 24-h $PM_{2.5}$ iron concentrations measured at CSN sites across the U.S., 2005-2007

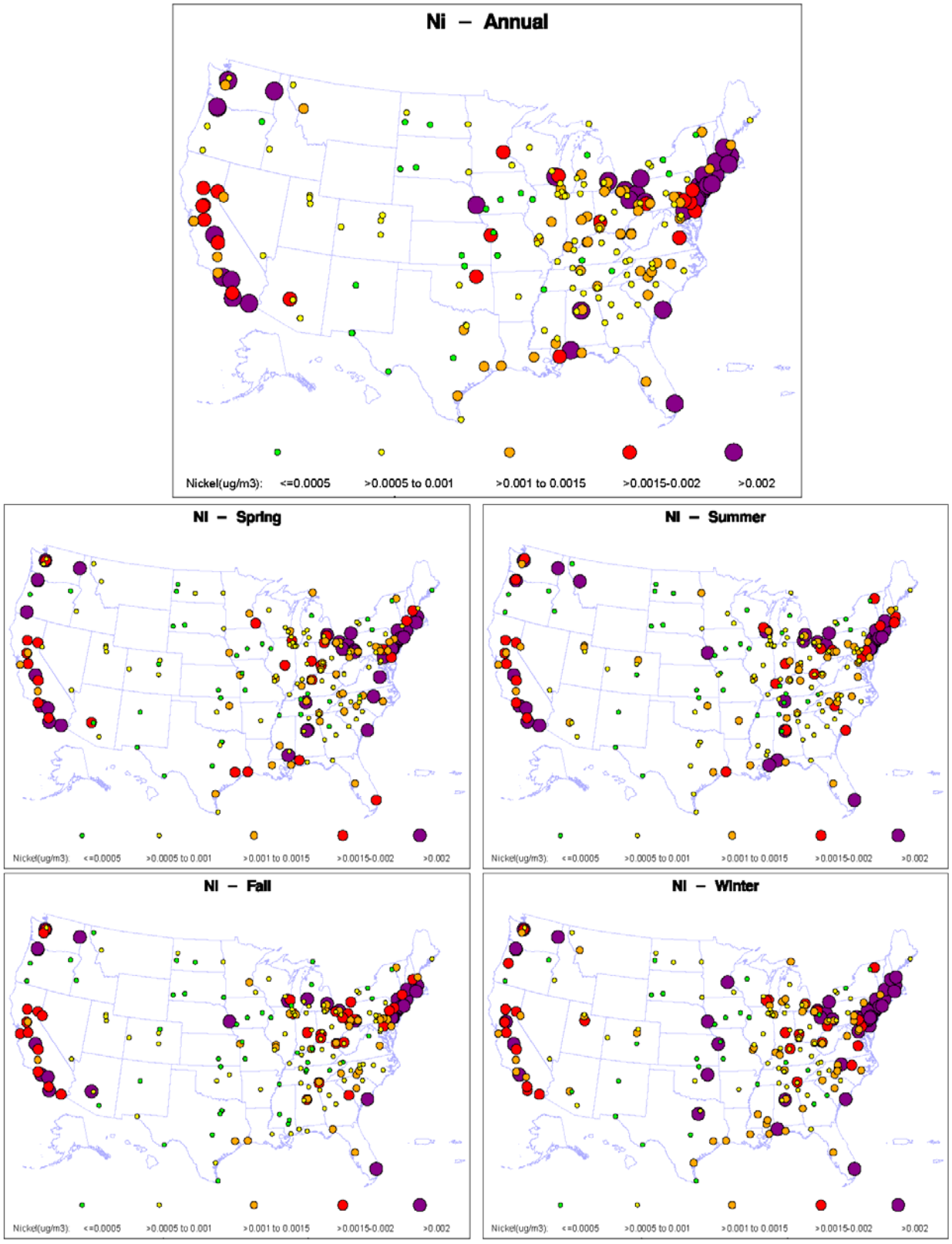


Figure A-31. Three-yr avg of 24-h $PM_{2.5}$ nickel concentrations measured at CSN sites across the U.S., 2005-2007

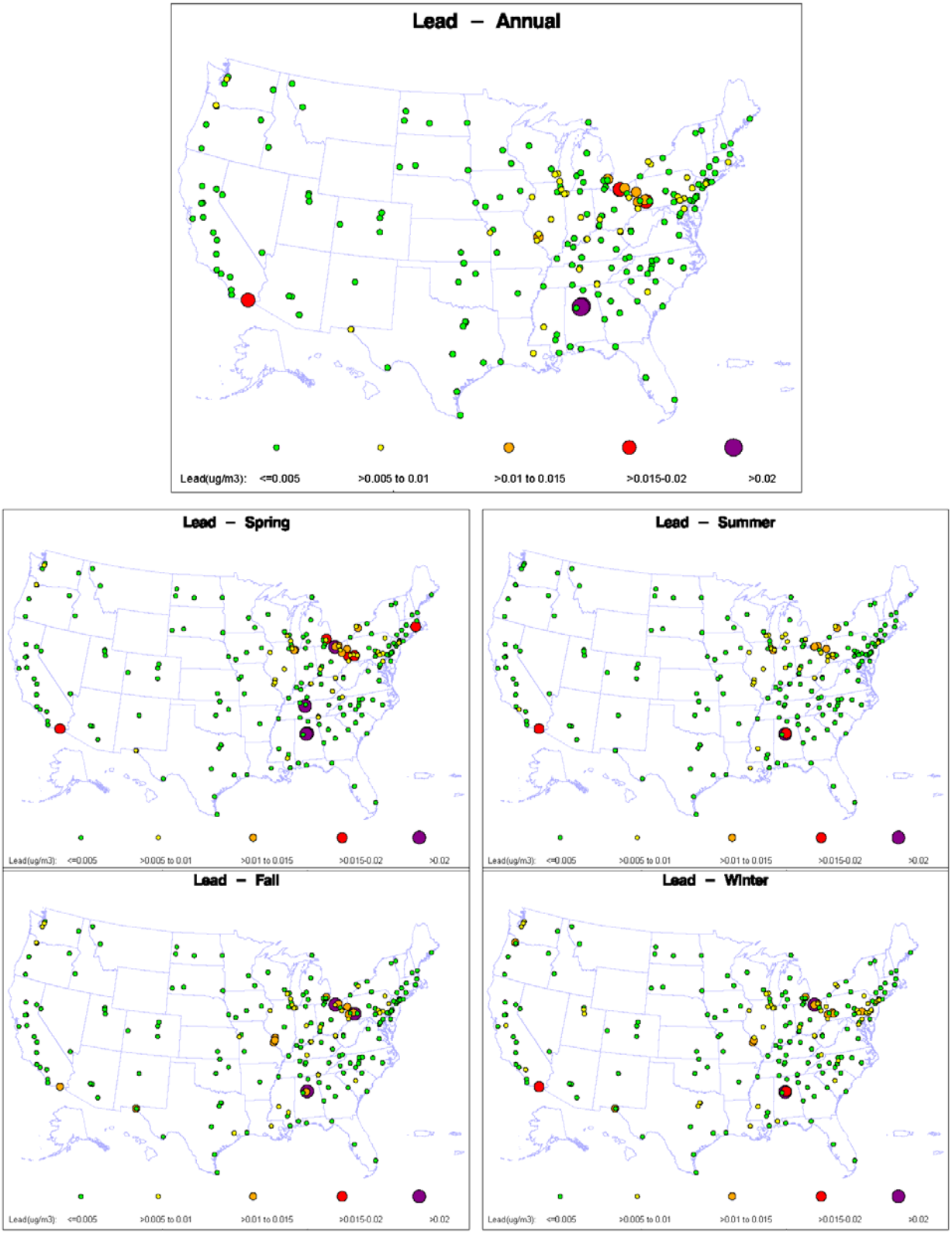


Figure A-32. Three-yr avg of 24-h $PM_{2.5}$ lead concentrations measured at CSN sites across the U.S., 2005-2007

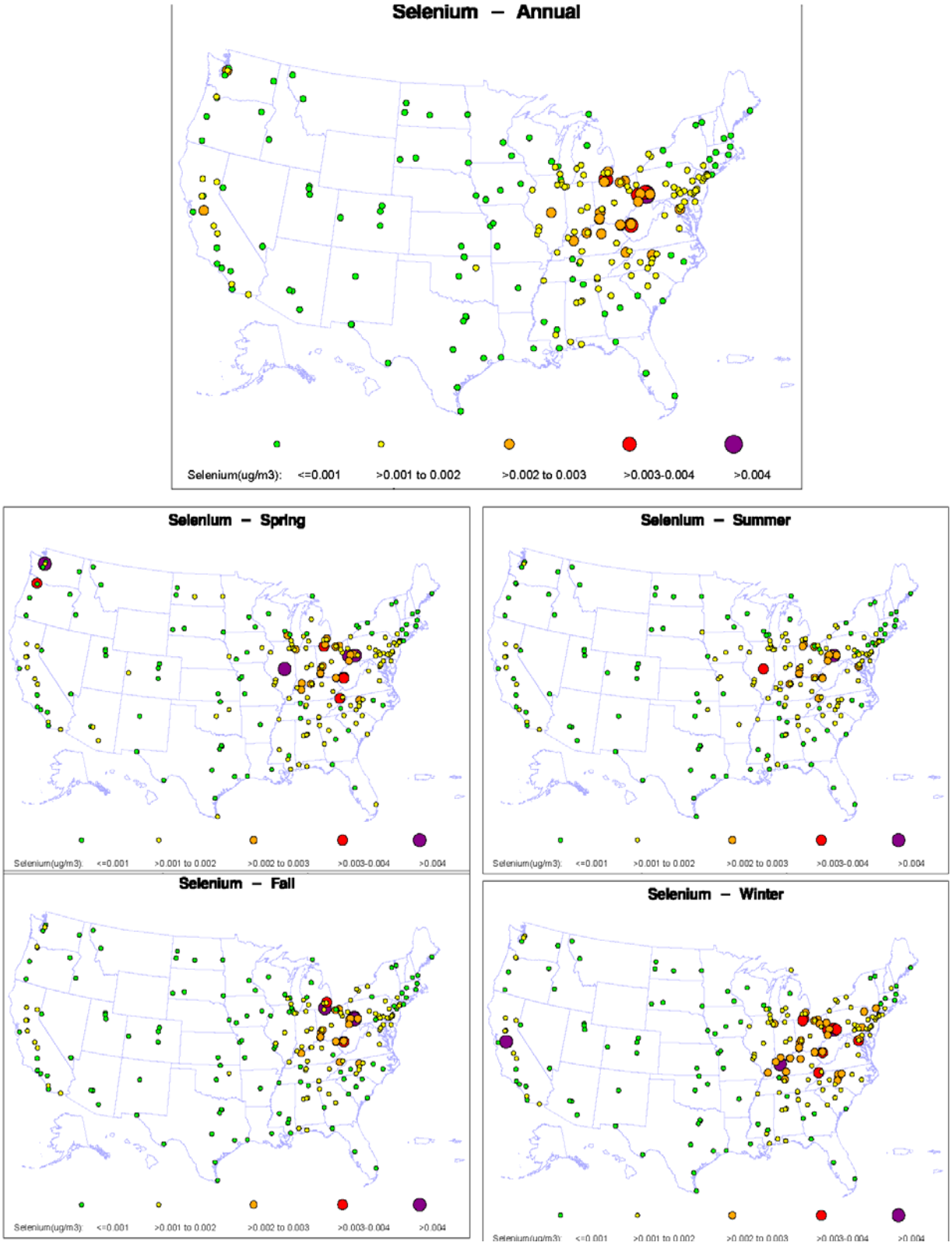


Figure A-33. Three-yr avg of 24-h $PM_{2.5}$ selenium concentrations measured at CSN sites across the U.S., 2005-2007

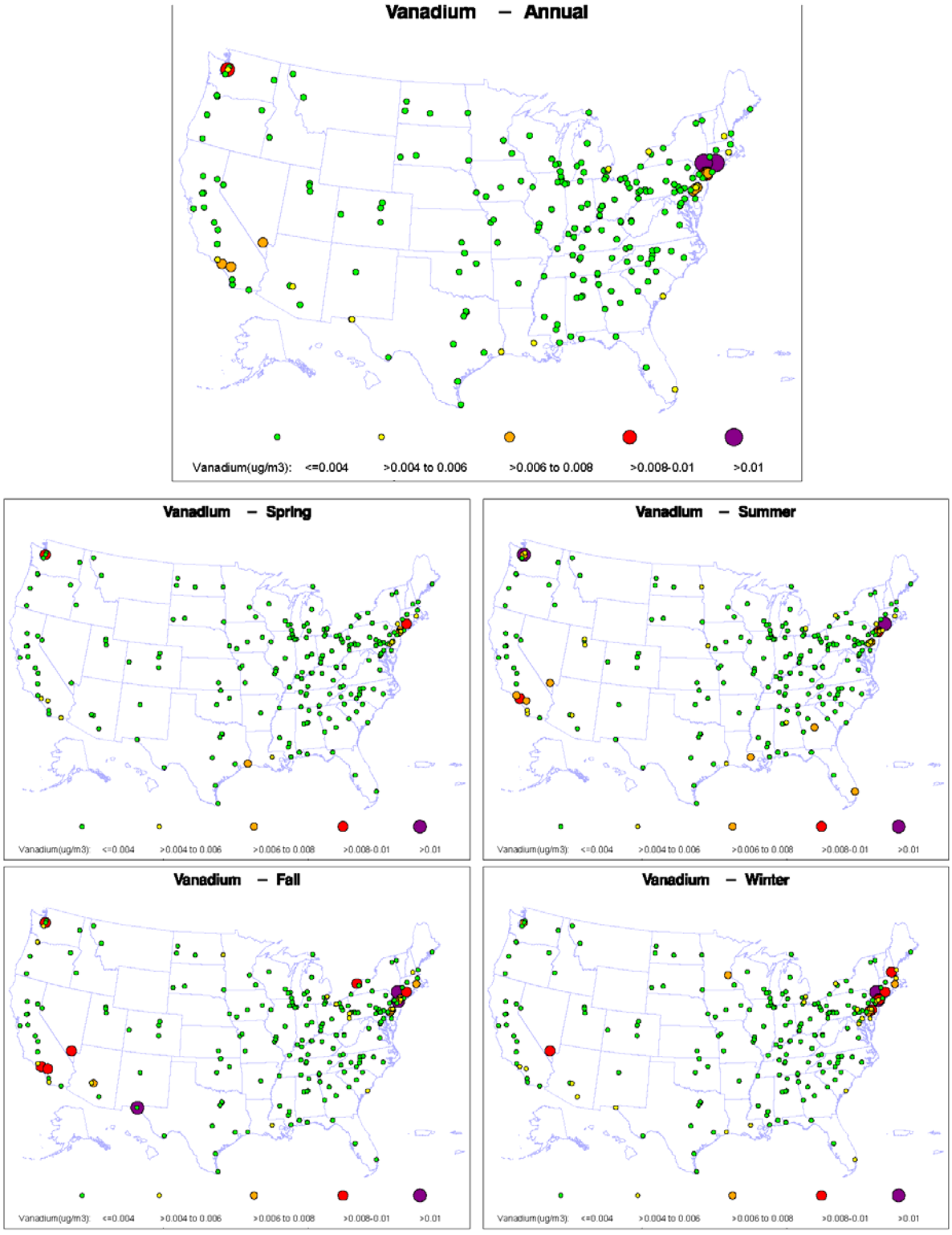


Figure A-34. Three-yr avg of 24-h $PM_{2.5}$ vanadium concentrations measured at CSN sites across the U.S., 2005-2007

A.2.2. Intraurban Variability

The following figures and tables exemplify the intraurban variability among $PM_{2.5}$, $PM_{10\text{-}2.5}$ and PM_{10} measurements for select CSAs/CBSAs (2005-2007) including Atlanta, Birmingham, Chicago, Denver, Detroit, Houston, New York City, Philadelphia, Phoenix, Riverside, Seattle and St. Louis. Maps are included to show monitor locations relative to major roadways. Box plots show the median and interquartile range of concentrations with whiskers extending to the 5th and 95th percentiles at each site during (1) winter (December-February); (2) spring (March-May); (3) summer (June-August); and (4) fall (September-November). Tables of inter-sampler comparison statistics and scatter plots of inter-sampler correlation vs. distance illustrate variability present in each area.

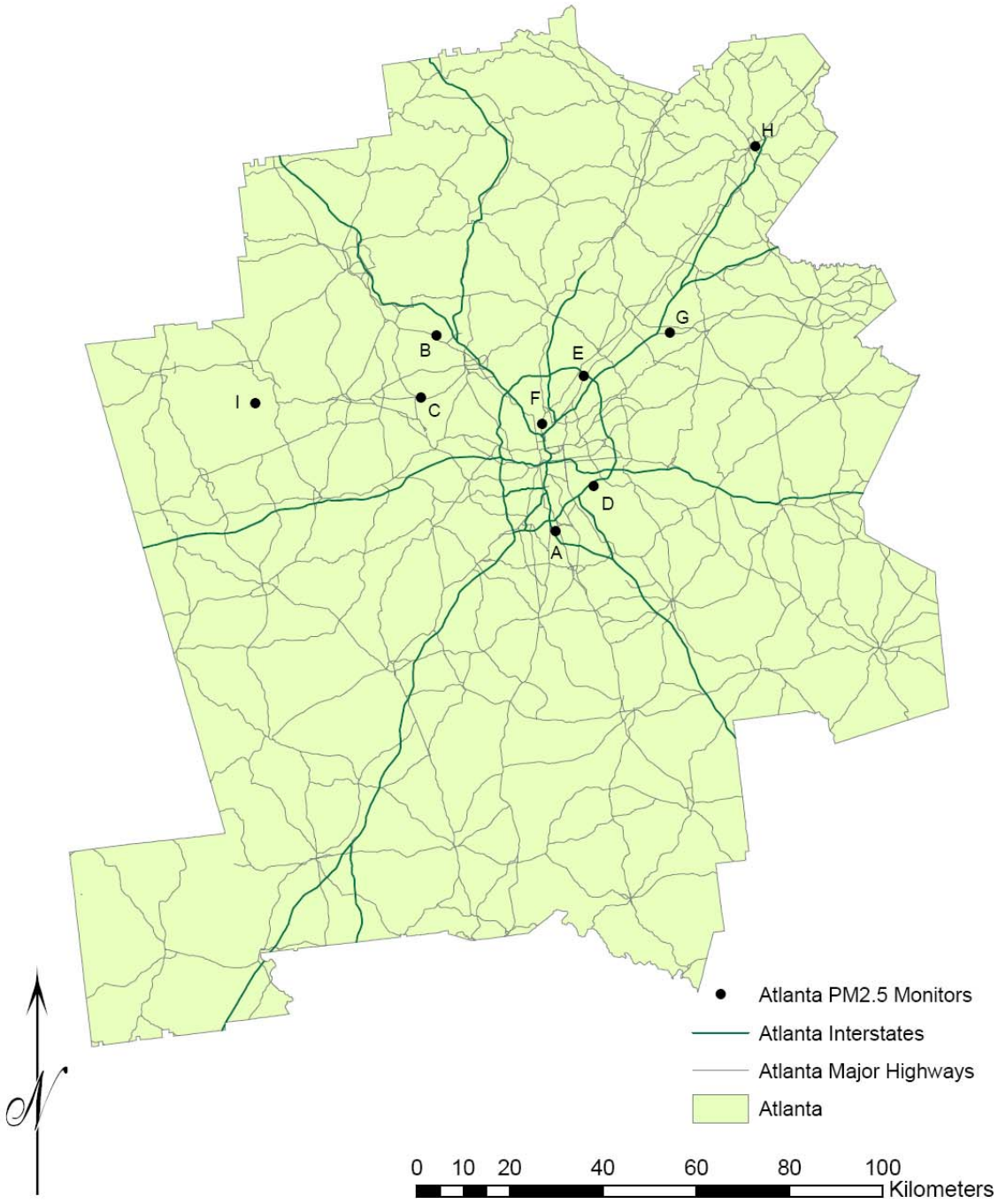


Figure A-35. $PM_{2.5}$ monitor distribution and major highways, Atlanta, GA.

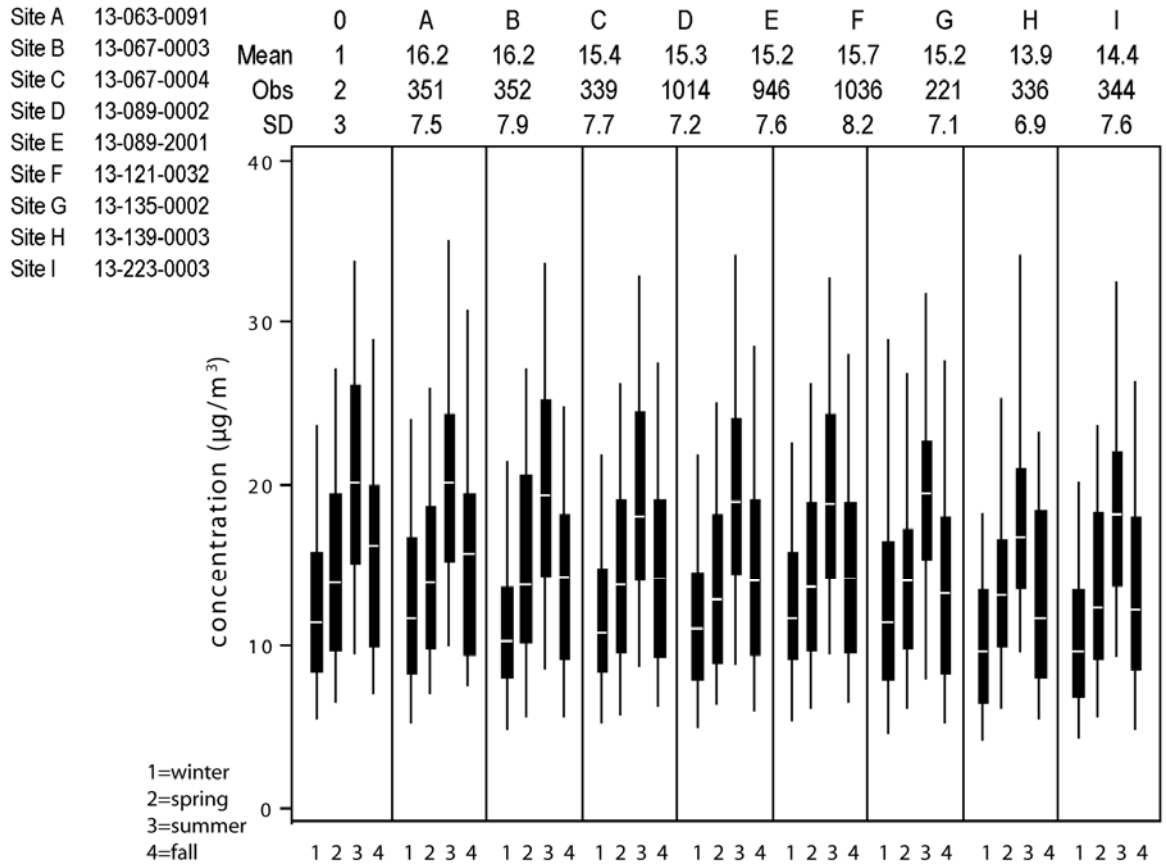


Figure A-36. Box plots illustrating the seasonal distribution of 24-h avg $PM_{2.5}$ concentrations for Atlanta, GA.

Table A-1. Inter-sampler correlation statistics for each pair of $PM_{2.5}$ monitors reporting to AQS for Atlanta, GA.

	A	B	C	D	E	F	G	H	I
A	1.00	0.88	0.87	0.93	0.89	0.91	0.85	0.72	0.85
	(0.0, 0.00)	(5.2, 0.11)	(6.2, 0.12)	(3.9, 0.11)	(5.3, 0.12)	(4.6, 0.11)	(6.9, 0.15)	(8.7, 0.19)	(7.2, 0.15)
	351	330	310	330	315	334	207	319	326
B		1.00	0.96	0.89	0.88	0.91	0.88	0.78	0.88
		(0.0, 0.00)	(4.1, 0.08)	(5.7, 0.12)	(4.6, 0.10)	(3.6, 0.08)	(5.6, 0.13)	(9.0, 0.17)	(6.5, 0.13)
		352	309	327	314	333	205	313	321
C			1.00	0.87	0.86	0.88	0.85	0.79	0.90
			(0.0, 0.00)	(5.2, 0.12)	(5.6, 0.11)	(4.4, 0.10)	(5.8, 0.13)	(7.9, 0.17)	(4.5, 0.11)
			339	315	304	324	193	298	303
D			CT	1.00	0.89	0.80	0.87	0.74	0.82
				(0.0, 0.00)	(4.8, 0.12)	(3.7, 0.11)	(5.8, 0.13)	(8.3, 0.18)	(7.3, 0.15)
				1014	883	978	208	314	322
E					1.00	0.79	0.88	0.74	0.83
					(0.0, 0.00)	(3.8, 0.11)	(5.3, 0.12)	(7.8, 0.17)	(6.4, 0.14)
					946	904	208	305	309
F		R				1.00	0.88	0.70	0.84
		(P90, COD)				(0.0, 0.00)	(5.3, 0.12)	(8.5, 0.19)	(6.3, 0.14)
		N				1036	213	321	327
G							1.00	0.73	0.79
							(0.0, 0.00)	(8.8, 0.17)	(7.4, 0.15)
							221	195	198
H								1.00	0.76
								(0.0, 0.00)	(8.7, 0.17)
								336	309
I									1.00
									(0.0, 0.00)
									344

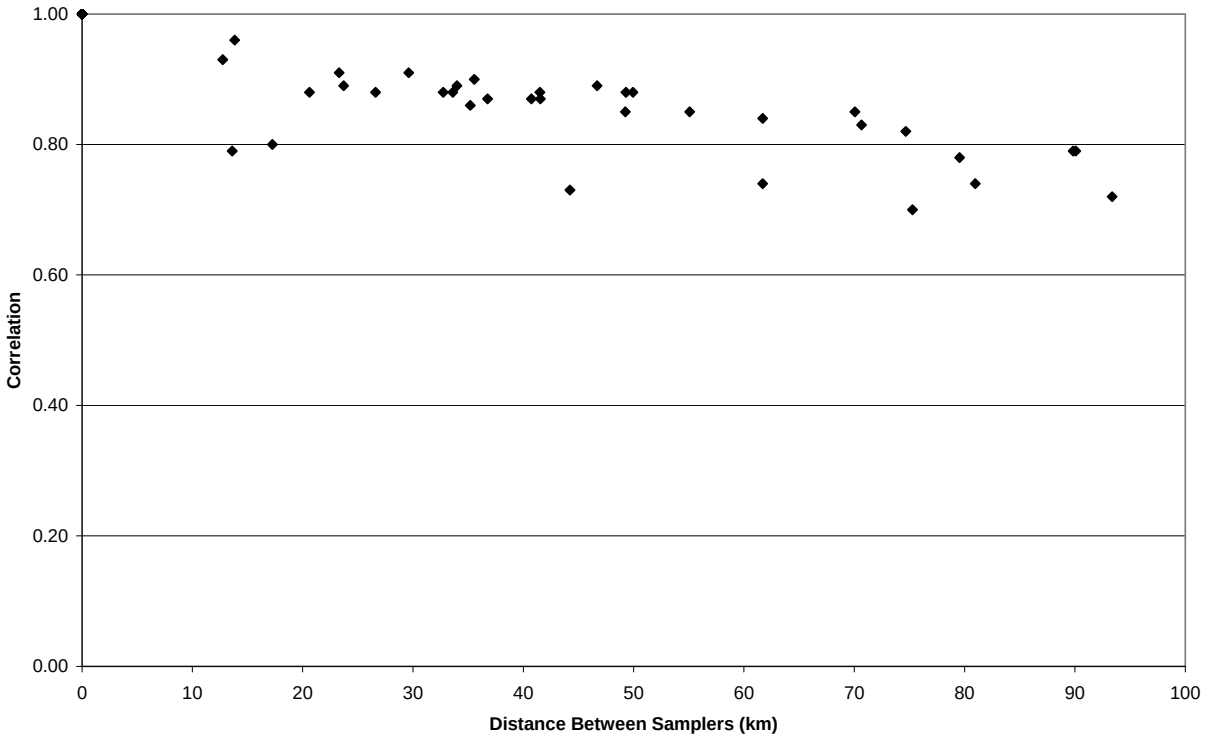


Figure A-37. $PM_{2.5}$ inter-sampler correlations as a function of distance between monitors for Atlanta, GA.

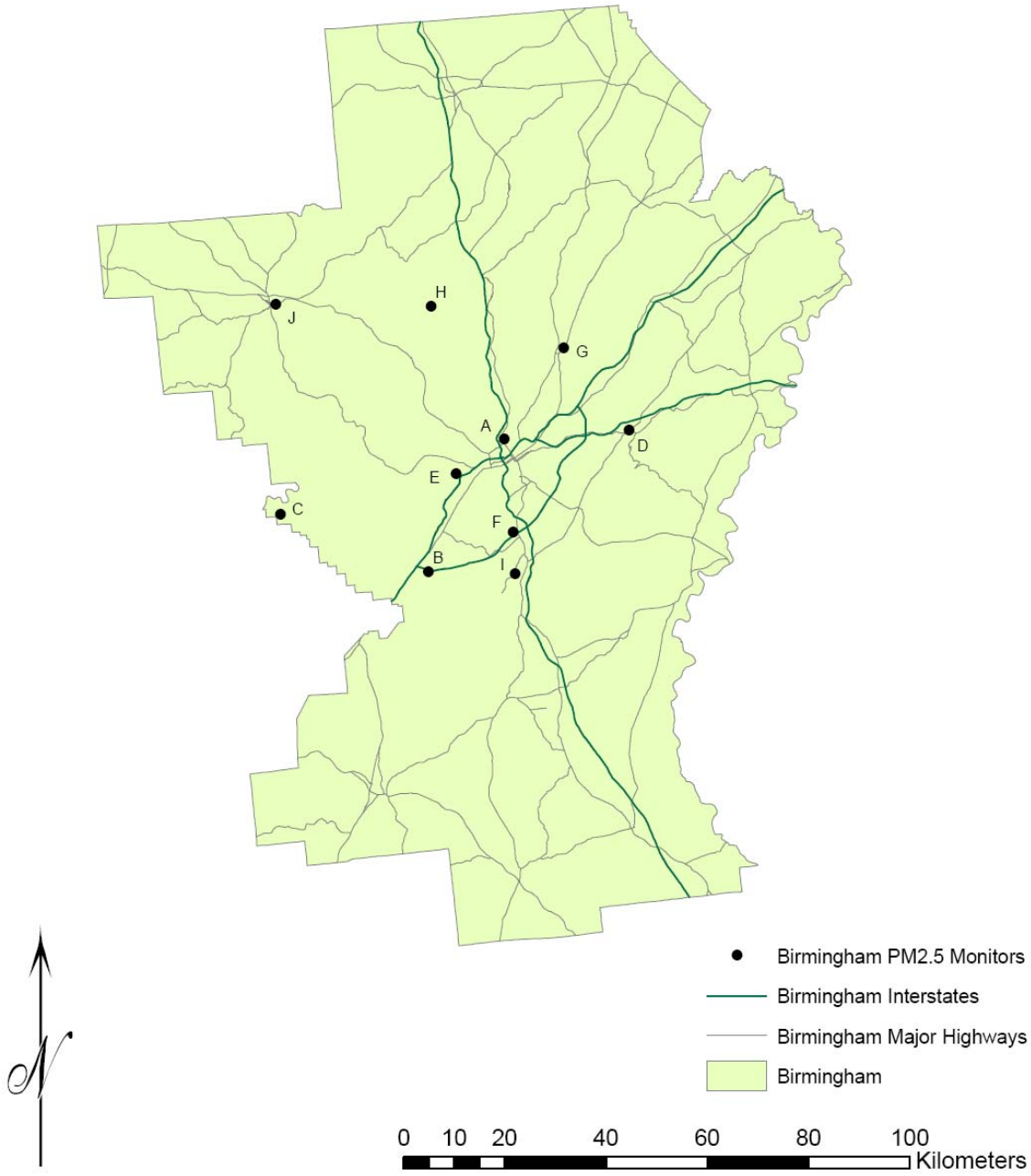


Figure A-38. $PM_{2.5}$ monitor distribution and major highways, Birmingham, AL.

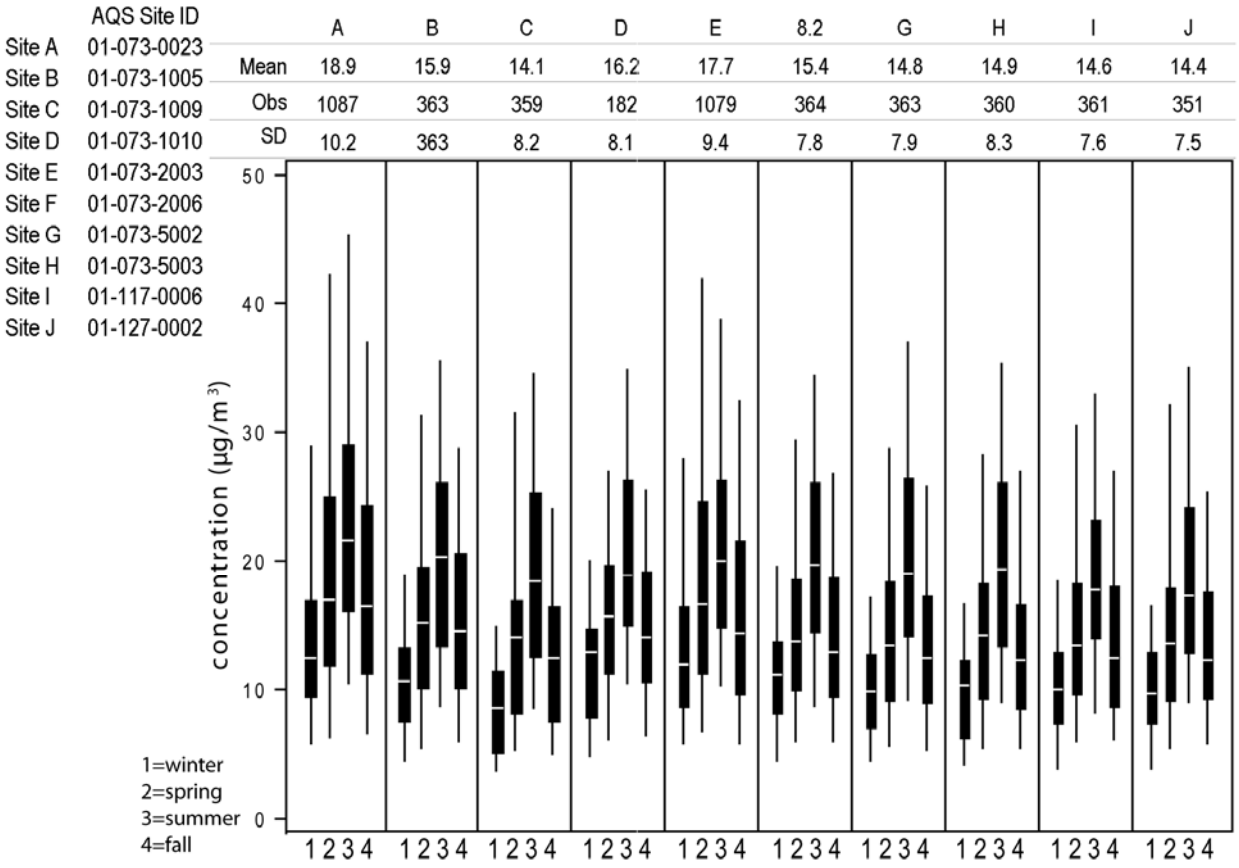


Figure A-39. Box plots illustrating the seasonal distribution of 24-h avg $PM_{2.5}$ concentrations for Birmingham, AL.

Table A-2. Inter-sampler correlation statistics for each pair of $PM_{2.5}$ monitors reporting to AQS for Birmingham, AL.

	A	B	C	D	E	F	G	H	I	J
A	1.00	0.91	0.86	0.91	0.88	0.91	0.87	0.88	0.88	0.84
	(0.0, 0.00)	(10.4, 0.15)	(13.7, 0.21)	(9.7, 0.13)	(8.1, 0.13)	(10.8, 0.15)	(12.6, 0.18)	(11.7, 0.18)	(12.3, 0.18)	(12.5, 0.19)
	1087	360	356	182	1072	361	360	357	358	348
B		1.00	0.93	0.93	0.85	0.96	0.91	0.93	0.93	0.89
		(0.0, 0.00)	(5.3, 0.12)	(4.7, 0.09)	(8.3, 0.15)	(3.6, 0.08)	(5.4, 0.11)	(5.1, 0.11)	(4.9, 0.10)	(6.1, 0.12)
		363	356	181	359	358	360	355	358	348
C			1.00	0.93	0.81	0.93	0.91	0.94	0.90	0.90
			(0.0, 0.00)	(5.9, 0.13)	(10.1, 0.20)	(4.6, 0.12)	(4.3, 0.12)	(4.0, 0.10)	(4.9, 0.12)	(4.9, 0.11)
			359	180	355	354	355	350	353	343
D				1.00	0.88	0.96	0.95	0.95	0.93	0.89
				(0.0, 0.00)	(7.9, 0.12)	(3.6, 0.08)	(3.8, 0.09)	(4.7, 0.10)	(4.7, 0.10)	(6.1, 0.12)
				182	179	179	181	179	180	174
E					1.00	0.87	0.85	0.85	0.86	0.81
					(0.0, 0.00)	(8.1, 0.15)	(8.7, 0.16)	(8.8, 0.17)	(9.2, 0.16)	(10.6, 0.18)
					1079	360	359	356	357	347
F		R				1.00	0.95	0.95	0.95	0.90
		(P90, COD)				(0.0, 0.00)	(3.9, 0.09)	(4.1, 0.10)	(3.4, 0.09)	(5.6, 0.11)
		N				364	359	354	357	348
G							1.00	0.96	0.92	0.89
							(0.0, 0.00)	(3.3, 0.08)	(4.5, 0.10)	(4.9, 0.11)
							363	356	359	350
H								1.00	0.91	0.93
								(0.0, 0.00)	(5.0, 0.11)	(4.3, 0.09)
								360	354	344
I									1.00	0.87
									(0.0, 0.00)	(5.8, 0.12)
									361	349
J										1.00
										(0.0, 0.00)
										351

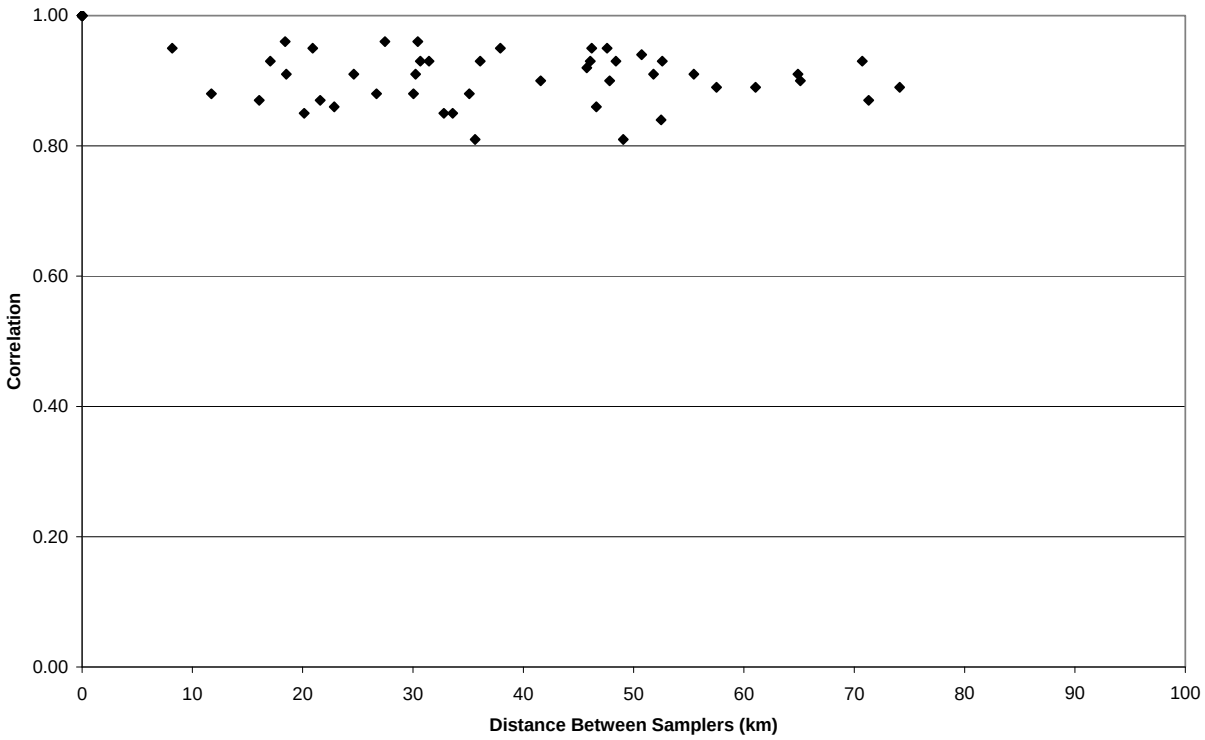


Figure A-40. $PM_{2.5}$ inter-sampler correlations as a function of distance between monitors for Birmingham, AL.

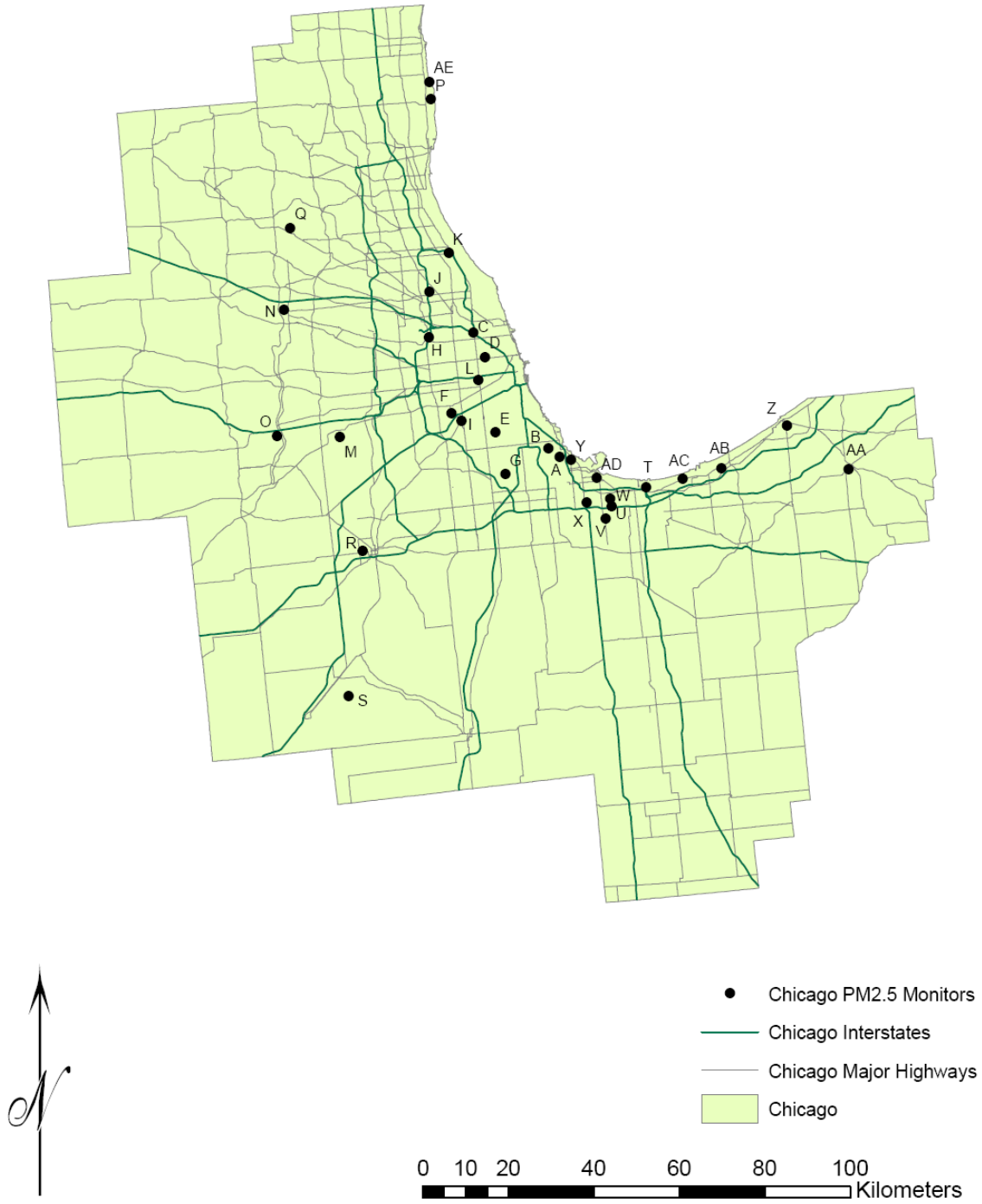
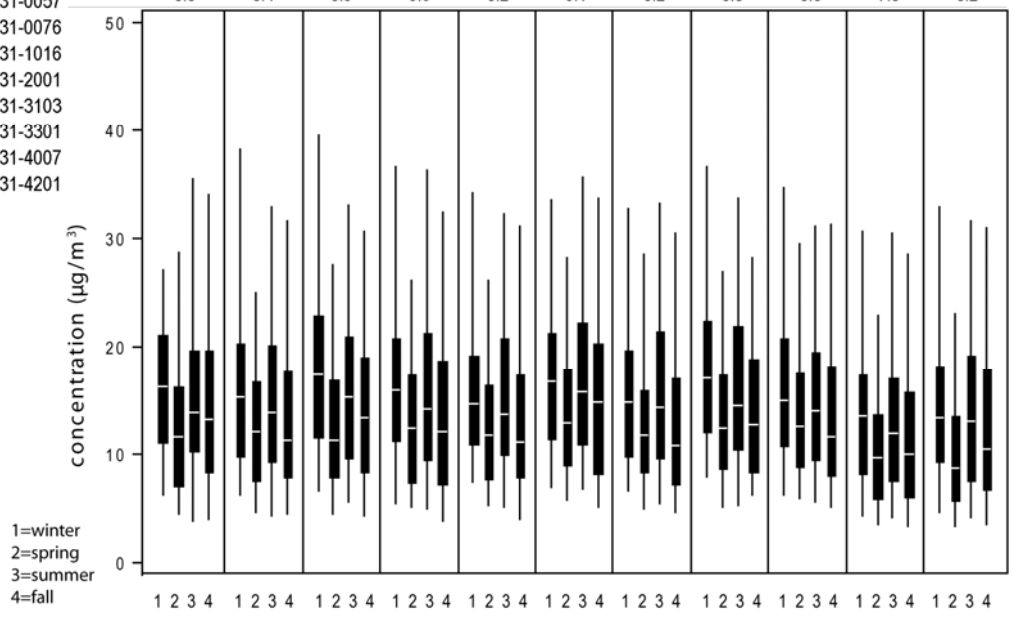
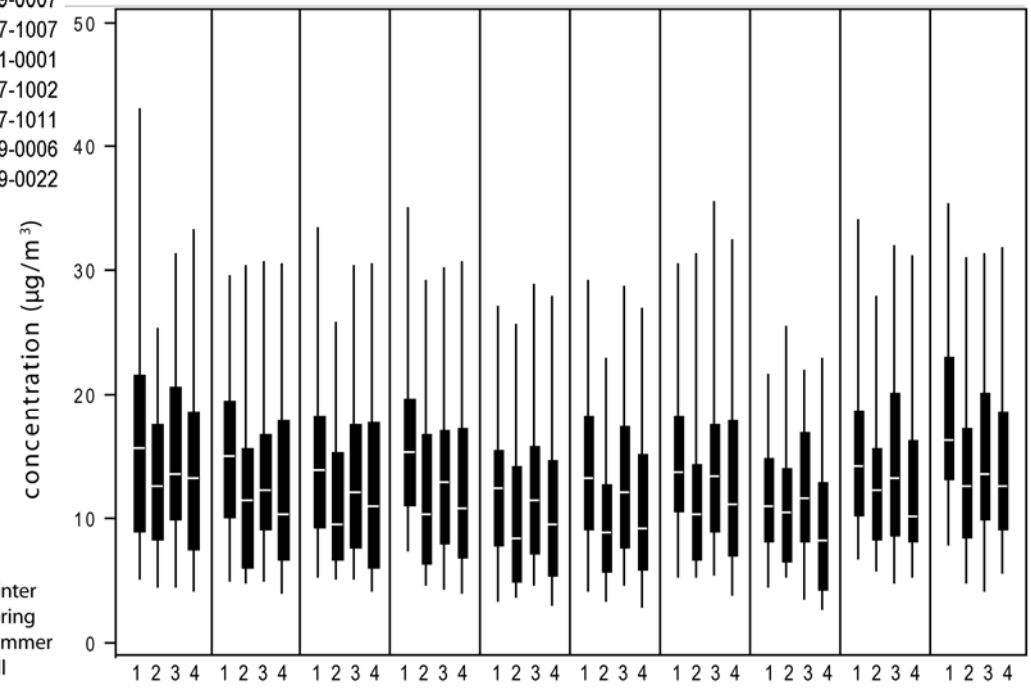


Figure A-41. $PM_{2.5}$ monitor distribution and major highways, Chicago, IL.

	AQS Site ID	A	B	C	D	E	F	G	H	I	J	K
Site A	17-031-0022	Mean	15.3	14.7	15.6	15.1	14.8	16.4	14.6	15.9	15.2	12.6	13.2
Site B	17-031-0050	Obs	178	343	984	333	351	345	350	335	361	356	361
Site C	17-031-0052	SD	8.8	8.4	8.9	9.0	8.2	9.1	8.2	8.5	8.3	7.8	8.2
Site D	17-031-0057												
Site E	17-031-0076												
Site F	17-031-1016												
Site G	17-031-2001												
Site H	17-031-3103												
Site I	17-031-3301												
Site J	17-031-4007												
Site K	17-031-4201												



	AQS Site ID	L	M	N	O	P	Q	R	S	T	U
Site L	17-031-6005	Mean	15.1	14.0	13.5	14.3	12.1	12.3	14.0	11.7	14.4	15.6
Site M	17-043-4002	Obs	331	179	176	174	181	347	175	164	330	351
Site N	17-089-0003	SD	8.7	8.5	8.5	8.9	8.2	7.5	8.5	7.2	7.7	8.2
Site O	17-089-0007											
Site P	17-097-1007											
Site Q	17-111-0001											
Site R	17-197-1002											
Site S	17-197-1011											
Site T	18-089-0006											
Site U	18-089-0022											



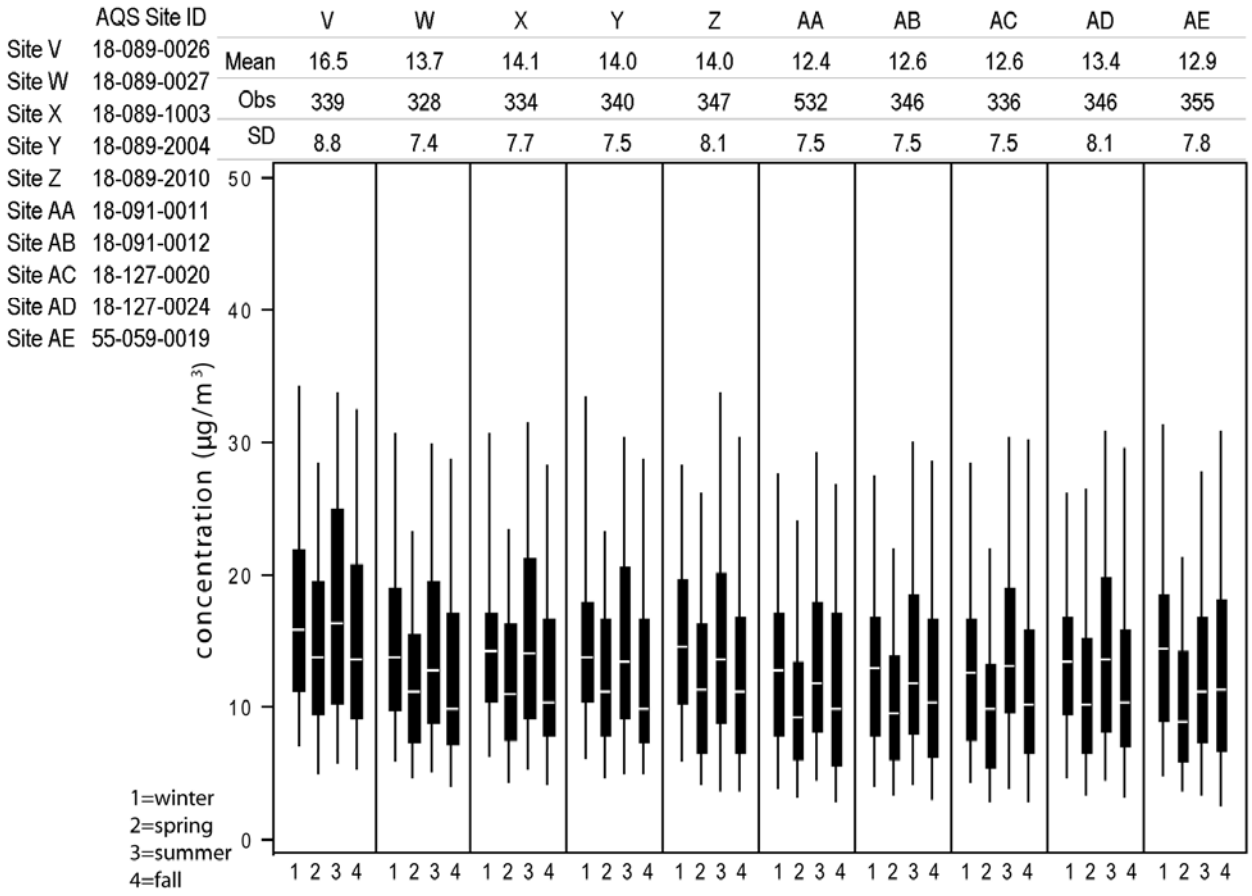


Figure A-42. Box plots illustrating the seasonal distribution of 24-h avg $PM_{2.5}$ concentrations for Chicago, IL.

Table A-3. Inter-sampler correlation statistics for each pair of $PM_{2.5}$ monitors reporting to AQS for Chicago, IL.

	A	B	C	D	E	F	G	H	I	J	K	L	M	N	O
A	1.00	0.98	0.93	0.94	0.97	0.95	0.97	0.94	0.96	0.91	0.95	0.95	0.91	0.92	0.89
	(0.0, 0.00)	(3.1, 0.08)	(5.5, 0.12)	(4.7, 0.11)	(3.9, 0.09)	(5.7, 0.13)	(3.9, 0.09)	(4.6, 0.12)	(4.2, 0.11)	(6.8, 0.16)	(5.8, 0.14)	(4.6, 0.12)	(5.7, 0.15)	(6.6, 0.15)	(6.0, 0.16)
	178	156	176	149	154	154	151	156	164	163	166	141	165	152	156
B		1.00	0.94	0.95	0.97	0.95	0.97	0.95	0.96	0.93	0.93	0.95	0.92	0.93	0.90
		(0.0, 0.00)	(4.6, 0.11)	(3.6, 0.10)	(3.3, 0.08)	(5.2, 0.13)	(2.7, 0.07)	(4.3, 0.11)	(3.4, 0.09)	(6.3, 0.16)	(6.5, 0.15)	(4.0, 0.10)	(5.1, 0.15)	(5.8, 0.14)	(5.2, 0.15)
		343	320	276	300	296	296	289	312	315	306	288	157	152	150
C			1.00	0.96	0.92	0.91	0.90	0.94	0.92	0.90	0.91	0.92	0.88	0.92	0.86
			(0.0, 0.00)	(4.4, 0.11)	(5.7, 0.11)	(4.8, 0.11)	(6.0, 0.12)	(4.3, 0.11)	(5.5, 0.11)	(8.8, 0.18)	(7.2, 0.17)	(4.5, 0.12)	(7.5, 0.16)	(7.9, 0.16)	(7.5, 0.17)
			984	313	325	318	324	312	336	332	337	311	178	175	173
D				1.00	0.94	0.93	0.94	0.95	0.94	0.93	0.93	0.92	0.89	0.96	0.88
				(0.0, 0.00)	(3.8, 0.10)	(4.2, 0.12)	(3.8, 0.10)	(4.1, 0.13)	(3.3, 0.10)	(6.2, 0.15)	(5.2, 0.14)	(3.6, 0.10)	(5.3, 0.14)	(5.1, 0.13)	(4.5, 0.15)
				333	286	280	283	270	299	296	289	273	151	146	145
E					1.00	0.95	0.98	0.95	0.98	0.92	0.92	0.95	0.95	0.94	0.92
					(0.0, 0.00)	(5.0, 0.11)	(2.4, 0.06)	(4.5, 0.11)	(2.6, 0.07)	(5.8, 0.16)	(5.7, 0.15)	(4.4, 0.10)	(4.8, 0.11)	(5.0, 0.11)	(4.6, 0.13)
					351	306	304	292	320	321	313	286	159	154	152
F						1.00	0.95	0.95	0.96	0.89	0.91	0.94	0.94	0.94	0.94
						(0.0, 0.00)	(5.1, 0.12)	(4.5, 0.12)	(4.5, 0.10)	(8.5, 0.20)	(7.9, 0.19)	(5.7, 0.12)	(7.0, 0.15)	(7.9, 0.17)	(7.9, 0.16)
						345	301	294	322	323	311	285	161	157	154
G							1.00	0.95	0.97	0.90	0.91	0.94	0.95	0.95	0.95
							(0.0, 0.00)	(4.9, 0.12)	(3.0, 0.07)	(6.3, 0.15)	(5.8, 0.14)	(4.7, 0.10)	(4.2, 0.11)	(5.0, 0.12)	(4.4, 0.12)
							350	284	315	318	309	287	154	149	148
H								1.00	0.95	0.91	0.92	0.94	0.93	0.94	0.91
								(0.0, 0.00)	(4.3, 0.11)	(7.4, 0.19)	(6.4, 0.18)	(4.4, 0.13)	(6.4, 0.16)	(7.1, 0.16)	(5.9, 0.17)
								335	311	309	302	275	164	157	156
I									1.00	0.90	0.92	0.96	0.96	0.95	0.93
									(0.0, 0.00)	(6.7, 0.17)	(5.9, 0.16)	(3.9, 0.10)	(4.6, 0.12)	(5.3, 0.13)	(4.6, 0.14)
									361	341	328	304	173	169	166
J										1.00	0.92	0.90	0.91	0.94	0.89

	A	B	C	D	E	F	G	H	I	J	K	L	M	N	O
										(0.0, 0.00)	(4.7, 0.13)	(7.0, 0.17)	(5.7, 0.14)	(4.4, 0.12)	(5.4, 0.16)
										356	330	304	171	165	164
K											1.00	0.93	0.94	0.96	0.92
					R						(0.0, 0.00)	(5.9, 0.15)	(5.2, 0.13)	(4.0, 0.10)	(4.9, 0.15)
					(P90, COD)						361	292	173	166	167
L					N							1.00	0.94	0.95	0.92
												(0.0, 0.00)	(6.4, 0.13)	(5.9, 0.13)	(6.0, 0.14)
												331	147	142	142
M													1.00	0.97	0.95
													(0.0, 0.00)	(3.9, 0.09)	(2.7, 0.11)
													179	160	165
N														1.00	0.95
														(0.0, 0.00)	(3.8, 0.11)
														176	152
O															1.00
															(0.0, 0.00)
															174

1.

	P	Q	R	S	T	U	V	W	X	Y	Z	AA	AB	AC	AD	AE
A	0.90	0.89	0.91	0.83	0.96	0.83	0.93	0.95	0.96	0.95	0.98	0.94	0.93	0.95	0.94	0.88
	(8.0, 0.19)	(7.5, 0.19)	(5.6, 0.16)	(8.0, 0.24)	(4.4, 0.11)	(7.2, 0.16)	(5.9, 0.13)	(4.7, 0.12)	(4.4, 0.10)	(4.5, 0.12)	(3.4, 0.10)	(5.8, 0.17)	(6.8, 0.17)	(6.0, 0.16)	(5.4, 0.15)	(7.1, 0.17)
	166	151	157	145	154	162	159	149	156	160	160	154	159	158	159	162
B	0.90	0.90	0.91	0.83	0.95	0.81	0.94	0.95	0.96	0.96	0.98	0.94	0.92	0.93	0.87	0.87
	(8.0, 0.20)	(6.7, 0.17)	(5.5, 0.16)	(8.0, 0.24)	(3.9, 0.10)	(6.7, 0.15)	(5.2, 0.11)	(5.0, 0.11)	(4.0, 0.10)	(4.2, 0.11)	(2.9, 0.09)	(5.9, 0.17)	(6.8, 0.17)	(6.5, 0.18)	(5.3, 0.16)	(7.2, 0.17)
	159	290	153	143	292	310	300	289	292	300	309	288	308	299	305	311
C	0.89	0.91	0.86	0.78	0.90	0.76	0.89	0.90	0.90	0.90	0.93	0.87	0.88	0.88	0.84	0.79
	(10.2, 0.22)	(8.3, 0.19)	(7.1, 0.17)	(10.4, 0.25)	(6.9, 0.13)	(8.5, 0.18)	(6.4, 0.13)	(7.7, 0.15)	(7.1, 0.14)	(7.9, 0.15)	(6.7, 0.15)	(8.6, 0.20)	(8.9, 0.20)	(9.6, 0.21)	(8.7, 0.18)	(8.5, 0.20)
	180	324	172	164	309	327	315	305	311	317	323	491	323	313	323	333
D	0.90	0.94	0.89	0.80	0.92	0.74	0.91	0.94	0.93	0.93	0.95	0.92	0.90	0.90	0.85	0.87
	(7.6, 0.19)	(6.5, 0.16)	(5.5, 0.14)	(8.6, 0.22)	(5.0, 0.12)	(7.8, 0.19)	(5.9, 0.13)	(5.4, 0.13)	(5.4, 0.13)	(5.8, 0.13)	(4.8, 0.13)	(6.4, 0.17)	(6.9, 0.18)	(7.0, 0.19)	(6.2, 0.17)	(6.9, 0.17)
	153	278	147	135	280	294	283	273	282	284	292	274	291	287	290	297
E	0.91	0.92	0.94	0.87	0.94	0.77	0.94	0.96	0.95	0.96	0.96	0.93	0.91	0.90	0.86	0.87
	(8.3, 0.18)	(5.6, 0.16)	(4.9, 0.12)	(7.1, 0.20)	(4.1, 0.10)	(7.5, 0.17)	(5.6, 0.12)	(4.3, 0.10)	(4.4, 0.11)	(3.9, 0.09)	(3.9, 0.11)	(5.8, 0.17)	(6.9, 0.17)	(6.8, 0.18)	(6.3, 0.16)	(7.3, 0.17)
	160	294	155	142	300	320	310	299	303	310	317	292	314	304	313	318
F	0.91	0.92	0.92	0.87	0.90	0.74	0.93	0.92	0.92	0.92	0.93	0.91	0.90	0.87	0.83	0.84
	(10.5, 0.23)	(8.6, 0.20)	(8.5, 0.17)	(10.0, 0.25)	(6.9, 0.14)	(9.2, 0.19)	(5.4, 0.11)	(8.2, 0.16)	(7.2, 0.15)	(7.6, 0.15)	(6.3, 0.16)	(8.5, 0.22)	(9.4, 0.21)	(9.1, 0.23)	(8.3, 0.20)	(9.3, 0.21)
	163	295	159	144	302	320	308	297	305	311	316	292	317	306	317	322
G	0.91	0.91	0.95	0.88	0.94	0.76	0.95	0.97	0.96	0.97	0.96	0.92	0.92	0.90	0.87	0.85
	(7.9, 0.19)	(5.9, 0.16)	(4.1, 0.12)	(7.1, 0.21)	(3.9, 0.10)	(7.5, 0.17)	(4.7, 0.10)	(3.7, 0.09)	(3.6, 0.09)	(3.5, 0.08)	(3.4, 0.11)	(5.7, 0.17)	(6.8, 0.16)	(6.8, 0.18)	(5.9, 0.15)	(7.5, 0.17)
	156	292	154	140	293	315	303	293	296	303	312	288	311	300	308	314
H	0.91	0.93	0.92	0.82	0.92	0.78	0.92	0.91	0.92	0.92	0.93	0.90	0.89	0.88	0.83	0.88
	(9.3, 0.23)	(7.1, 0.20)	(6.6, 0.17)	(9.6, 0.26)	(5.7, 0.13)	(7.5, 0.17)	(6.1, 0.13)	(6.8, 0.15)	(5.9, 0.15)	(6.7, 0.14)	(5.9, 0.15)	(7.7, 0.22)	(8.1, 0.22)	(8.3, 0.22)	(8.1, 0.20)	(7.6, 0.20)
	165	284	158	145	287	307	297	288	292	299	303	281	301	293	301	307
I	0.91	0.92	0.95	0.87	0.93	0.78	0.94	0.95	0.94	0.95	0.95	0.92	0.91	0.89	0.85	0.86
	(8.2, 0.21)	(6.1, 0.17)	(4.7, 0.12)	(7.7, 0.22)	(4.2, 0.10)	(7.1, 0.17)	(5.0, 0.10)	(4.6, 0.11)	(4.8, 0.11)	(4.7, 0.10)	(4.2, 0.13)	(6.5, 0.18)	(6.8, 0.18)	(7.4, 0.19)	(6.6, 0.17)	(7.0, 0.18)
	175	314	168	154	318	338	327	316	322	328	335	306	334	323	334	339
J	0.92	0.91	0.90	0.85	0.89	0.73	0.89	0.89	0.89	0.89	0.91	0.89	0.88	0.87	0.82	0.87
	(5.6, 0.14)	(5.1, 0.14)	(6.2, 0.16)	(6.7, 0.21)	(6.1, 0.17)	(8.6, 0.22)	(8.7, 0.20)	(6.0, 0.16)	(6.3, 0.16)	(6.2, 0.16)	(5.9, 0.16)	(5.6, 0.16)	(5.8, 0.16)	(6.3, 0.17)	(6.3, 0.17)	(6.4, 0.15)
	173	313	167	153	319	341	329	317	327	329	337	307	335	327	336	340
K	0.94	0.93	0.93	0.86	0.91	0.78	0.89	0.91	0.90	0.91	0.92	0.92	0.91	0.90	0.85	0.91
	(5.2, 0.12)	(4.2, 0.12)	(6.1, 0.16)	(7.2, 0.20)	(5.1, 0.16)	(8.0, 0.21)	(8.4, 0.19)	(5.2, 0.15)	(5.5, 0.15)	(5.2, 0.15)	(5.4, 0.14)	(5.2, 0.15)	(4.9, 0.15)	(5.3, 0.16)	(5.3, 0.16)	(5.1, 0.13)
	176	298	169	155	310	327	319	304	313	319	325	301	325	315	323	328
L	0.90	0.92	0.93	0.86	0.93	0.75	0.92	0.93	0.93	0.92	0.95	0.92	0.92	0.90	0.85	0.89
	(9.3, 0.20)	(6.7, 0.17)	(6.7, 0.14)	(8.9, 0.21)	(5.5, 0.12)	(8.2, 0.19)	(6.2, 0.13)	(5.7, 0.13)	(5.8, 0.13)	(6.2, 0.13)	(5.0, 0.13)	(7.2, 0.17)	(7.6, 0.17)	(7.4, 0.18)	(7.3, 0.16)	(7.1, 0.17)
	151	285	144	132	285	301	290	282	286	293	299	277	299	286	294	301
M	0.92	0.95	0.96	0.88	0.91	0.74	0.90	0.94	0.92	0.93	0.92	0.91	0.91	0.88	0.89	0.89
	(6.2, 0.16)	(4.5, 0.14)	(3.4, 0.09)	(6.3, 0.19)	(5.9, 0.14)	(9.0, 0.22)	(8.0, 0.17)	(4.7, 0.12)	(5.2, 0.14)	(5.0, 0.13)	(5.8, 0.16)	(6.4, 0.17)	(5.5, 0.15)	(6.9, 0.19)	(6.2, 0.17)	(6.9, 0.17)
	175	157	165	152	162	171	166	159	164	168	169	158	168	165	168	171
N	0.92	0.98	0.94	0.89	0.92	0.81	0.91	0.92	0.91	0.93	0.93	0.91	0.91	0.88	0.89	0.90
	(5.4, 0.13)	(2.8, 0.08)	(4.1, 0.12)	(5.8, 0.17)	(6.2, 0.13)	(7.9, 0.20)	(8.3, 0.17)	(4.9, 0.12)	(5.6, 0.14)	(4.6, 0.13)	(4.9, 0.15)	(5.4, 0.15)	(4.9, 0.14)	(6.5, 0.17)	(5.4, 0.16)	(6.0, 0.14)
	162	151	153	140	156	165	160	157	158	162	165	153	162	158	161	165
O	0.88	0.92	0.93	0.86	0.89	0.75	0.89	0.92	0.90	0.91	0.90	0.89	0.90	0.87	0.87	0.87
	(7.5, 0.18)	(4.9, 0.15)	(3.8, 0.13)	(6.5, 0.20)	(5.9, 0.15)	(8.8, 0.22)	(7.6, 0.17)	(5.1, 0.13)	(5.8, 0.15)	(5.5, 0.14)	(5.6, 0.16)	(6.1, 0.17)	(5.7, 0.16)	(7.0, 0.19)	(6.2, 0.17)	(7.0, 0.18)
	166	152	157	145	155	166	161	154	158	161	162	151	161	159	162	166
P	1.00	0.92	0.90	0.84	0.88	0.73	0.87	0.89	0.89	0.89	0.90	0.89	0.90	0.89	0.89	0.92
	(0.0, 0.00)	(5.2, 0.13)	(7.1, 0.17)	(7.2, 0.20)	(8.5, 0.20)	(12.0, 0.26)	(10.9, 0.24)	(6.7, 0.18)	(7.4, 0.18)	(6.9, 0.18)	(7.6, 0.19)	(6.1, 0.16)	(5.7, 0.15)	(6.3, 0.17)	(6.4, 0.17)	(5.7, 0.13)
	181	159	166	152	164	174	168	160	166	169	171	158	170	167	170	173
Q		1.00	0.92	0.85	0.88	0.71	0.89	0.90	0.88	0.90	0.90	0.89	0.88	0.86	0.82	0.91
		(0.0, 0.00)	(5.4, 0.16)	(7.2, 0.19)	(6.1, 0.18)	(9.3, 0.24)	(9.1, 0.21)	(5.5, 0.16)	(6.5, 0.16)	(5.5, 0.16)	(6.3, 0.16)	(5.3, 0.16)	(5.3, 0.16)	(6.3, 0.18)	(5.9, 0.17)	(5.5, 0.14)
		347	154	139	290	309	296	289	294	302	306	292	303	293	303	310
R			1.00	0.91	0.93	0.76	0.90	0.94	0.93	0.94	0.91	0.92	0.92	0.88	0.89	0.89
			(0.0, 0.00)	(5.8, 0.18)	(5.1, 0.13)	(8.6, 0.22)	(7.5, 0.17)	(4.4, 0.11)	(5.0, 0.13)	(4.0, 0.12)	(5.8, 0.17)	(6.2, 0.17)	(5.6, 0.16)	(7.1, 0.19)	(6.4, 0.17)	(7.1, 0.17)
			175	143	157	167	161	153	160	161	164	153	164	160	163	166
S				1.00	0.83	0.66	0.81	0.86	0.83	0.87	0.83	0.84	0.84	0.81	0.82	0.80
				(0.0, 0.00)	(8.5, 0.22)	(11.3, 0.28)	(11.6, 0.26)	(6.7, 0.20)	(8.0, 0.21)	(7.2, 0.19)	(7.3, 0.22)	(6.1, 0.21)	(7.4, 0.20)	(7.8, 0.23)	(7.1, 0.22)	(9.0, 0.22)
				164	144	153	148	143	146	148	151	141	151	149	148	153
T					1.00	0.81	0.93	0.95	0.97	0.96	0.97	0.92	0.91	0.92	0.87	0.85
					(0.0, 0.00)	(5.9, 0.15)	(6.2, 0.12)	(3.4, 0.10)	(3.2, 0.09)	(2.9, 0.08)	(3.2, 0.12)	(5.2, 0.17)	(5.5, 0.16)	(5.4, 0.18)	(4.9, 0.15)	(6.6, 0.18)
					330	318	307	297	302	305	315	284	312	311	313	319
U						1.00	0.77	0.79	0.81	0.81	0.81	0.78	0.76	0.79	0.74	0.69
						(0.0, 0.00)	(7.6, 0.17)	(6.6, 0.17)	(6.0, 0.15)	(6.3, 0.16)	(6.4, 0.17)	(8.1, 0.22)	(8.4, 0.22)	(7.2, 0.21)	(7.0, 0.19)	(10.0, 0.23)
						351	327	319	322	326	336	305	334	324	333	338
V							1.00	0.96	0.97	0.95	0.95	0.93	0.91	0.90	0.88	0.83
							(0.0, 0.00)	(5.9, 0.11)	(4.8, 0.10)	(5.8, 0.12)	(5.7, 0.14)	(7.7, 0.20)	(8.6, 0.20)	(8.3, 0.21)	(6.9, 0.17)	(9.3, 0.22)
							339	306	314	316	325	292	323	314	321	325
W								1.00	0.98	0.98	0.96	0.95	0.93	0.92	0.89	0.85
								(0.0, 0.00)	(2.8, 0.06)	(2.5, 0.07)	(3.6, 0.11)	(4.5, 0.15)	(4.8, 0.15)	(5.4, 0.16)	(3.9, 0.13)	(6.9, 0.17)

	P	Q	R	S	T	U	V	W	X	Y	Z	AA	AB	AC	AD	AE
								328	299	306	312	281	310	306	311	316
X									1.00	0.98	0.97	0.95	0.93	0.94	0.91	0.85
									(0.0, 0.00)	(2.3, 0.07)	(3.3, 0.10)	(4.6, 0.14)	(4.9, 0.14)	(4.9, 0.15)	(3.6, 0.11)	(6.8, 0.17)
									334	311	318	286	319	305	316	321
Y										1.00	0.97	0.95	0.93	0.92	0.89	0.85
										(0.0, 0.00)	(3.6, 0.11)	(4.7, 0.16)	(5.0, 0.15)	(5.3, 0.17)	(4.4, 0.14)	(6.7, 0.18)
										340	322	296	322	311	321	326
Z											1.00	0.95	0.93	0.94	0.89	0.86
											(0.0, 0.00)	(4.6, 0.15)	(5.3, 0.15)	(4.9, 0.15)	(4.1, 0.14)	(6.8, 0.17)
											347	305	331	321	328	335
AA												1.00	0.98	0.97	0.89	0.88
												(0.0, 0.00)	(2.4, 0.07)	(2.9, 0.08)	(3.2, 0.11)	(5.9, 0.17)
												532	305	287	300	304
AB													1.00	0.96	0.89	0.86
													(0.0, 0.00)	(3.1, 0.09)	(3.7, 0.11)	(6.5, 0.17)
													346	317	328	333
AC														1.00	0.91	0.85
														(0.0, 0.00)	(2.8, 0.10)	(6.7, 0.17)
														336	320	322
AD															1.00	0.79
															(0.0, 0.00)	(7.2, 0.18)
															346	332
AE																1.00
																(0.0, 0.00)
																355

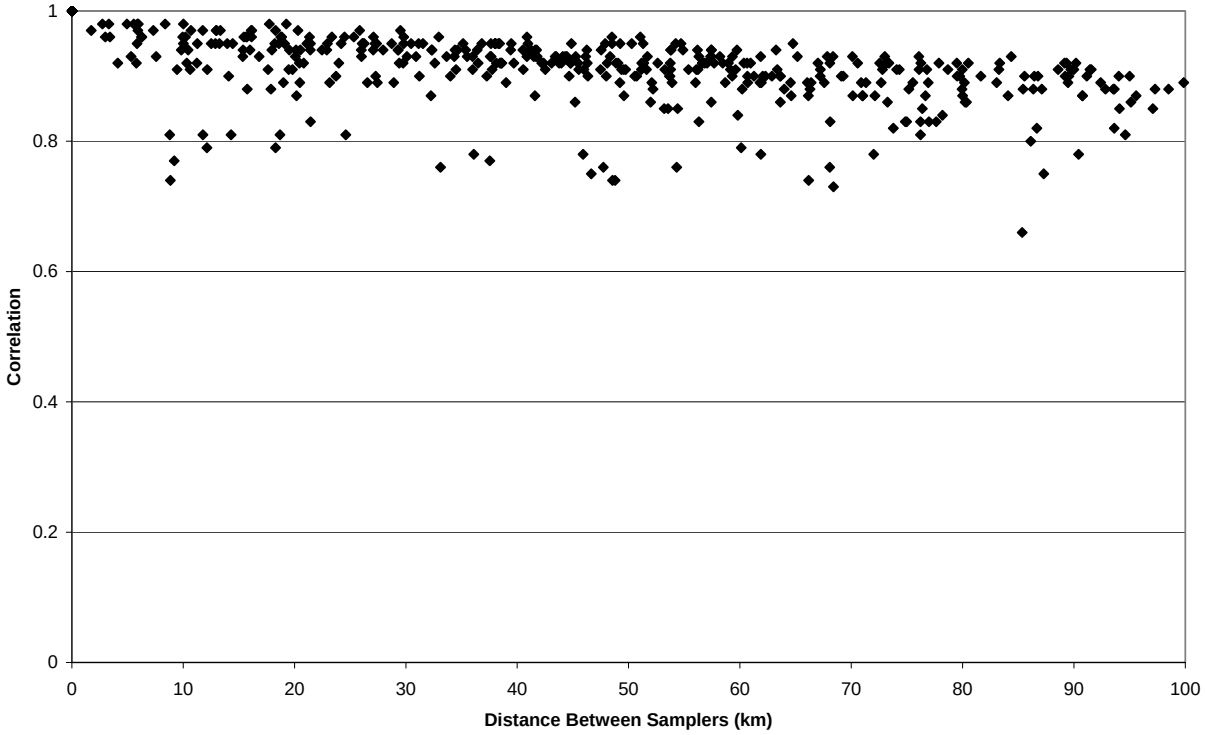


Figure A-43. $PM_{2.5}$ inter-sampler correlations as a function of distance between monitors for Chicago, IL.

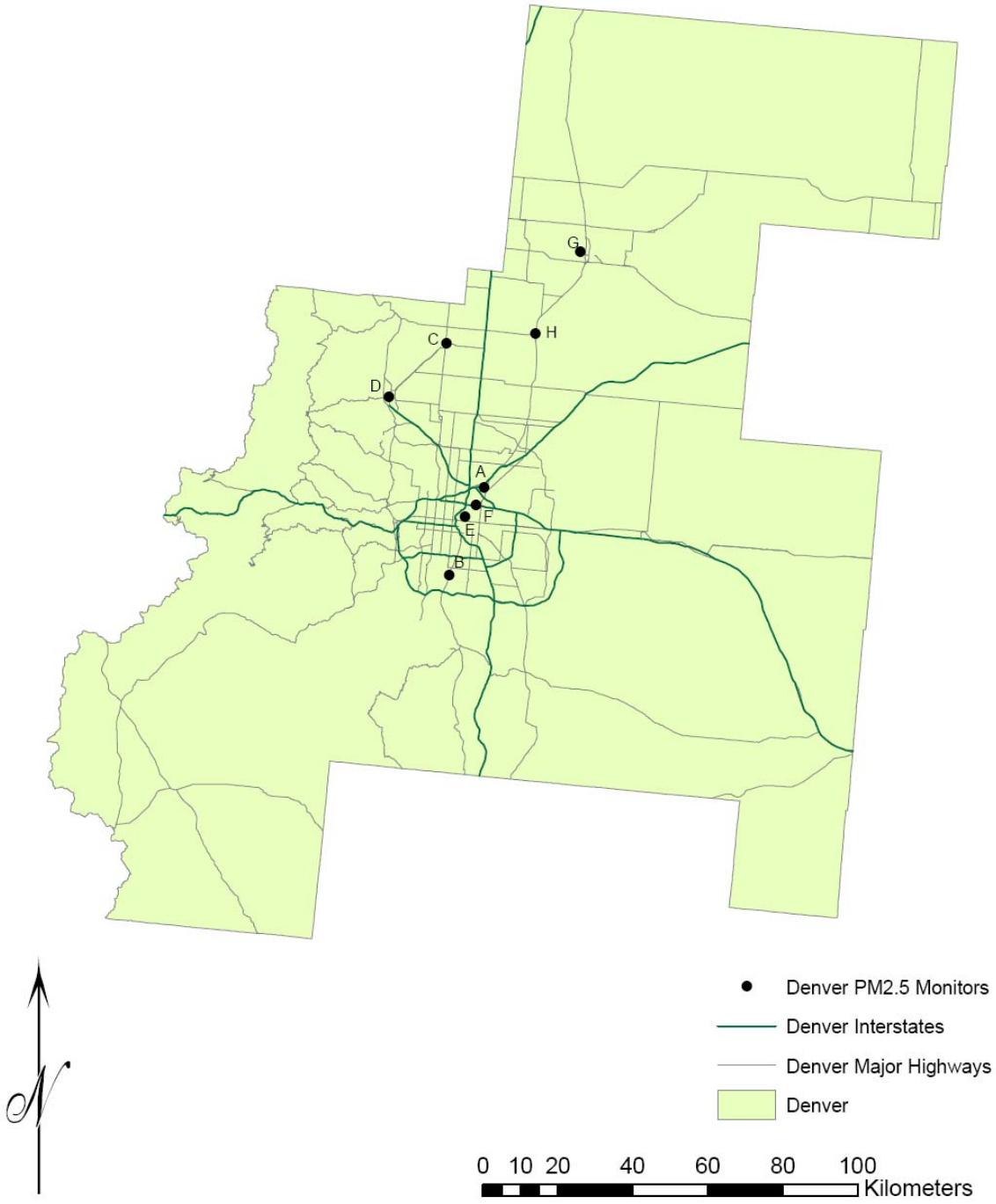


Figure A-44. $PM_{2.5}$ monitor distribution and major highways, Denver, CO.

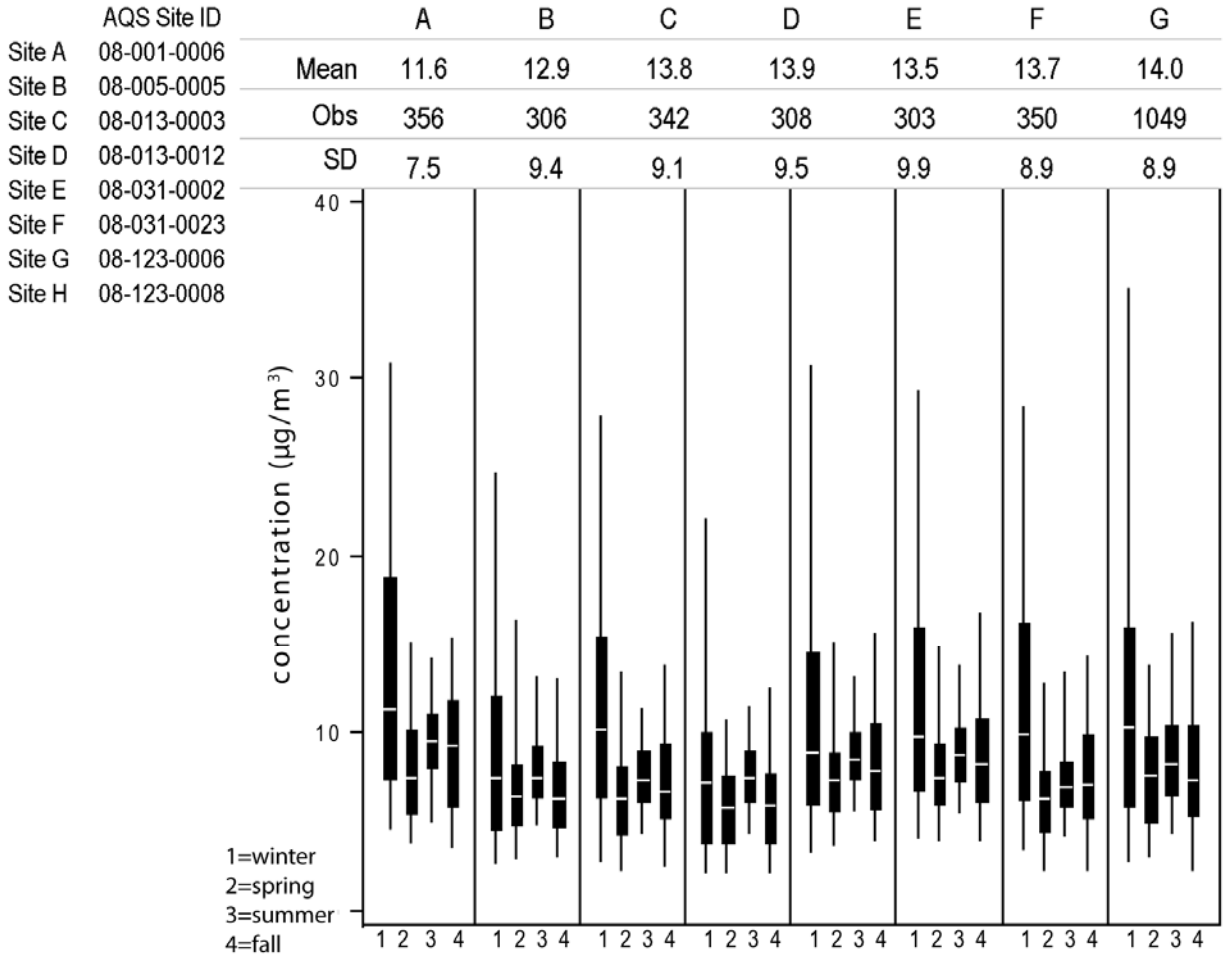


Figure A-45. Box plots illustrating the seasonal distribution of 24-h avg $PM_{2.5}$ concentrations for Denver, CO.

Table A-4. Inter-sampler correlation statistics for each pair of $PM_{2.5}$ monitors reporting to AQS for Denver, CO.

	A	B	C	D	E	F	G	H
A	1.00	0.74	0.84	0.68	0.86	0.91	0.76	0.83
	(0.0, 0.00)	(6.0, 0.21)	(5.4, 0.17)	(7.9, 0.26)	(4.1, 0.14)	(3.0, 0.11)	(5.9, 0.19)	(4.6, 0.14)
	369	353	347	332	362	339	341	325
B		1.00	0.58	0.76	0.92	0.84	0.50	0.49
		(0.0, 0.00)	(5.7, 0.19)	(3.9, 0.17)	(3.2, 0.13)	(4.4, 0.17)	(7.8, 0.23)	(6.6, 0.21)
		363	344	328	356	336	337	323
C			1.00	0.74	0.71	0.75	0.83	0.88
			(0.0, 0.00)	(4.4, 0.19)	(4.5, 0.17)	(5.4, 0.18)	(3.5, 0.14)	(3.7, 0.13)
			361	326	354	336	333	320
D				1.00	0.82	0.77	0.54	0.57
				(0.0, 0.00)	(5.6, 0.21)	(6.0, 0.24)	(7.2, 0.24)	(6.4, 0.24)
				354	347	332	318	305
E					1.00	0.94	0.64	0.60
					(0.0, 0.00)	(2.3, 0.09)	(7.1, 0.21)	(5.6, 0.18)
		R			1046	969	353	330
F		(P90, COD)				1.00	0.68	0.69
		N				(0.0, 0.00)	(6.6, 0.21)	(5.9, 0.17)
						1006	333	317
G							1.00	0.88
							(0.0, 0.00)	(3.4, 0.13)
							359	313
H								1.00

A	B	C	D	E	F	G	H
							(0.0, 0.00)
							334

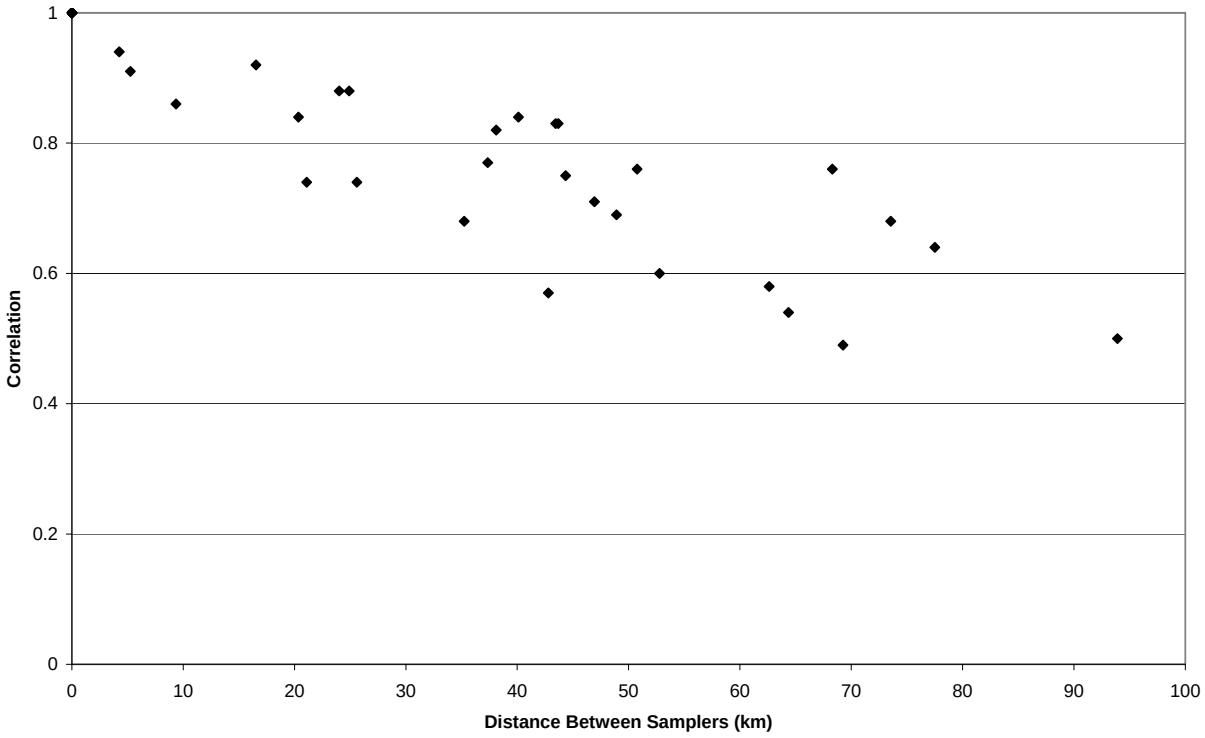


Figure A-46. $PM_{2.5}$ inter-sampler correlations as a function of distance between monitors for Denver, CO.

Figure A-47. $PM_{2.5}$ monitor distribution and major highways, Detroit, MI.

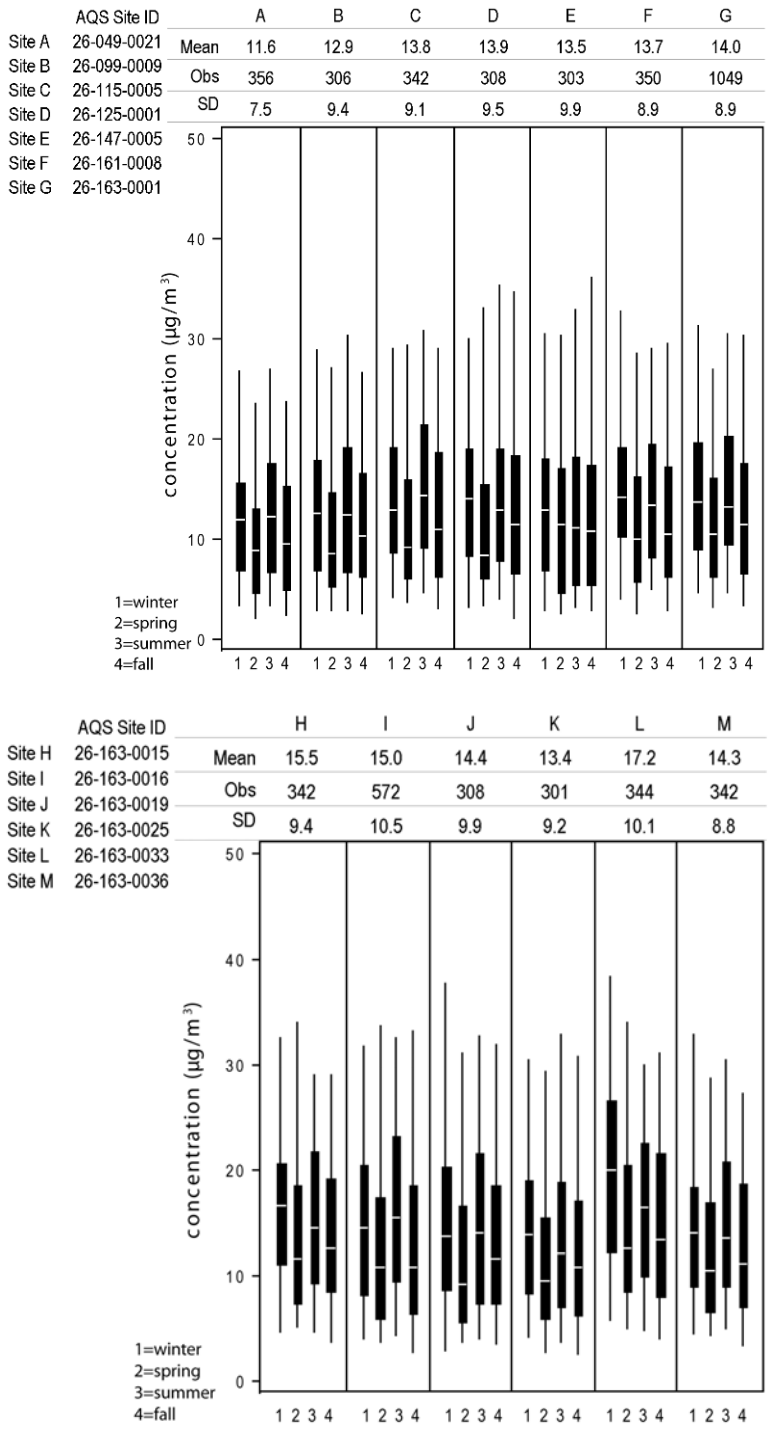


Figure A-48. Box plots illustrating the seasonal distribution of 24-h avg $PM_{2.5}$ concentrations for Detroit, MI.

Table A-5. Inter-sampler correlation statistics for each pair of $PM_{2.5}$ monitors reporting to AQS for Detroit, MI.

	A	B	C	D	E	F	G	H	I	J	K	L	M
A	1.00	0.91	0.86	0.91	0.89	0.90	0.89	0.88	0.89	0.91	0.92	0.87	0.88
	(0.0, 0.00)	(5.9, 0.17)	(7.8, 0.19)	(6.7, 0.17)	(7.6, 0.18)	(5.9, 0.18)	(8.1, 0.20)	(8.3, 0.22)	(8.0, 0.19)	(7.3, 0.17)	(5.5, 0.16)	(11.0, 0.26)	(7.8, 0.21)
	356	299	333	301	296	341	349	334	284	301	293	336	333
B		1.00	0.90	0.94	0.92	0.92	0.93	0.90	0.92	0.91	0.92	0.89	0.91
		(0.0, 0.00)	(6.8, 0.17)	(5.3, 0.14)	(5.9, 0.16)	(5.8, 0.17)	(6.2, 0.18)	(7.5, 0.21)	(5.8, 0.18)	(4.9, 0.16)	(5.4, 0.17)	(10.2, 0.24)	(6.1, 0.19)
		306	286	296	290	294	300	288	277	297	286	292	288
C			1.00	0.90	0.87	0.91	0.93	0.90	0.91	0.90	0.89	0.87	0.93
			(0.0, 0.00)	(7.0, 0.16)	(8.8, 0.20)	(5.5, 0.15)	(5.9, 0.14)	(7.2, 0.17)	(6.3, 0.16)	(6.2, 0.14)	(6.2, 0.16)	(10.4, 0.20)	(4.9, 0.13)
			342	289	284	326	335	320	273	286	279	321	319
D				1.00	0.93	0.94	0.96	0.92	0.94	0.94	0.94	0.91	0.92
				(0.0, 0.00)	(6.3, 0.15)	(4.5, 0.14)	(4.3, 0.13)	(5.8, 0.16)	(4.5, 0.12)	(3.8, 0.11)	(3.6, 0.13)	(8.2, 0.18)	(6.2, 0.15)
				308	292	296	303	291	281	297	291	290	290
E					1.00	0.90	0.90	0.89	0.90	0.90	0.90	0.87	0.87
					(0.0, 0.00)	(7.5, 0.18)	(7.3, 0.20)	(8.2, 0.22)	(7.0, 0.19)	(6.4, 0.18)	(6.9, 0.18)	(10.7, 0.25)	(7.7, 0.21)
					303	291	297	286	276	292	284	288	288
F						1.00	0.95	0.90	0.92	0.92	0.95	0.89	0.93
						(0.0, 0.00)	(4.5, 0.13)	(6.2, 0.17)	(5.7, 0.15)	(5.2, 0.14)	(3.9, 0.12)	(9.8, 0.21)	(5.7, 0.15)
						350	343	326	280	297	288	329	326
G							1.00	0.94	0.95	0.92	0.93	0.90	0.95
							(0.0, 0.00)	(5.1, 0.14)	(4.9, 0.12)	(4.5, 0.14)	(5.6, 0.16)	(8.2, 0.18)	(4.7, 0.12)
							1049	336	549	302	295	337	335
H								1.00	0.93	0.91	0.89	0.91	0.91
								(0.0, 0.00)	(4.8, 0.15)	(5.4, 0.15)	(6.9, 0.18)	(7.6, 0.16)	(6.1, 0.15)
								342	273	290	288	321	319
I									1.00	0.92	0.90	0.92	0.93
			R						(0.0, 0.00)	(4.4, 0.13)	(6.1, 0.14)	(7.9, 0.18)	(5.8, 0.14)
			(P90, COD)						572	279	271	274	274
J			N							1.00	0.91	0.90	0.91
										(0.0, 0.00)	(5.3, 0.15)	(8.1, 0.17)	(5.6, 0.13)
										308	288	291	291
K											1.00	0.88	0.91
											(0.0, 0.00)	(9.5, 0.21)	(6.3, 0.16)
											301	281	283
L												1.00	0.91
												(0.0, 0.00)	(8.5, 0.17)
												344	322
M													1.00
													(0.0, 0.00)
													342

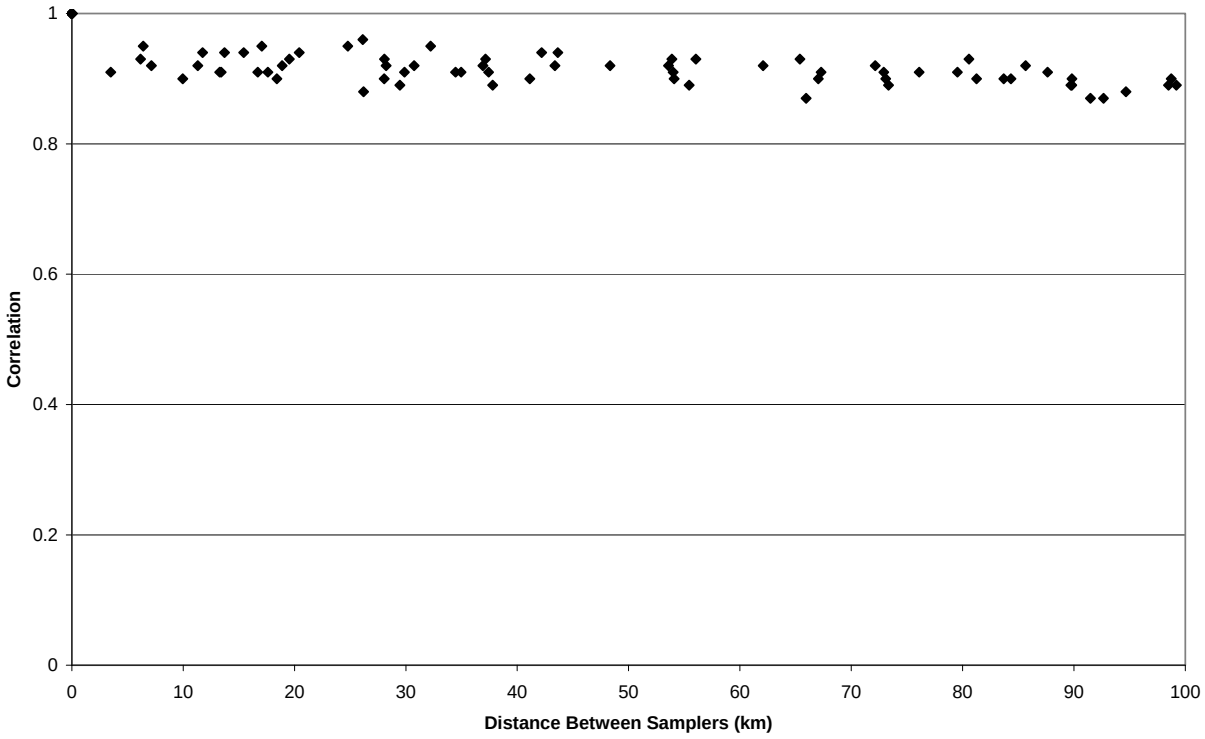


Figure A-49. $PM_{2.5}$ inter-sampler correlations as a function of distance between monitors for Detroit, MI.

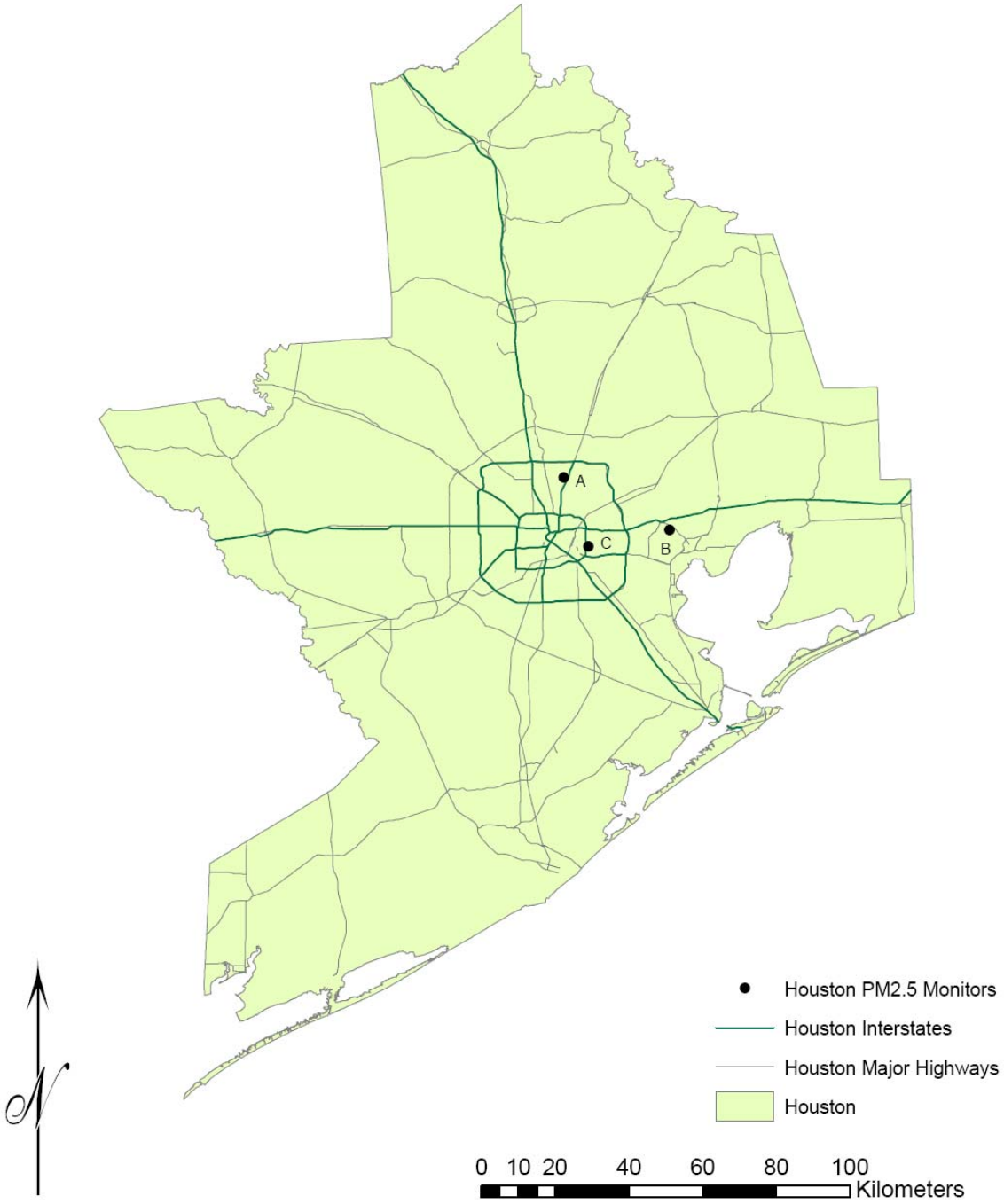


Figure A-50. $PM_{2.5}$ monitor distribution and major highways, Houston, TX.

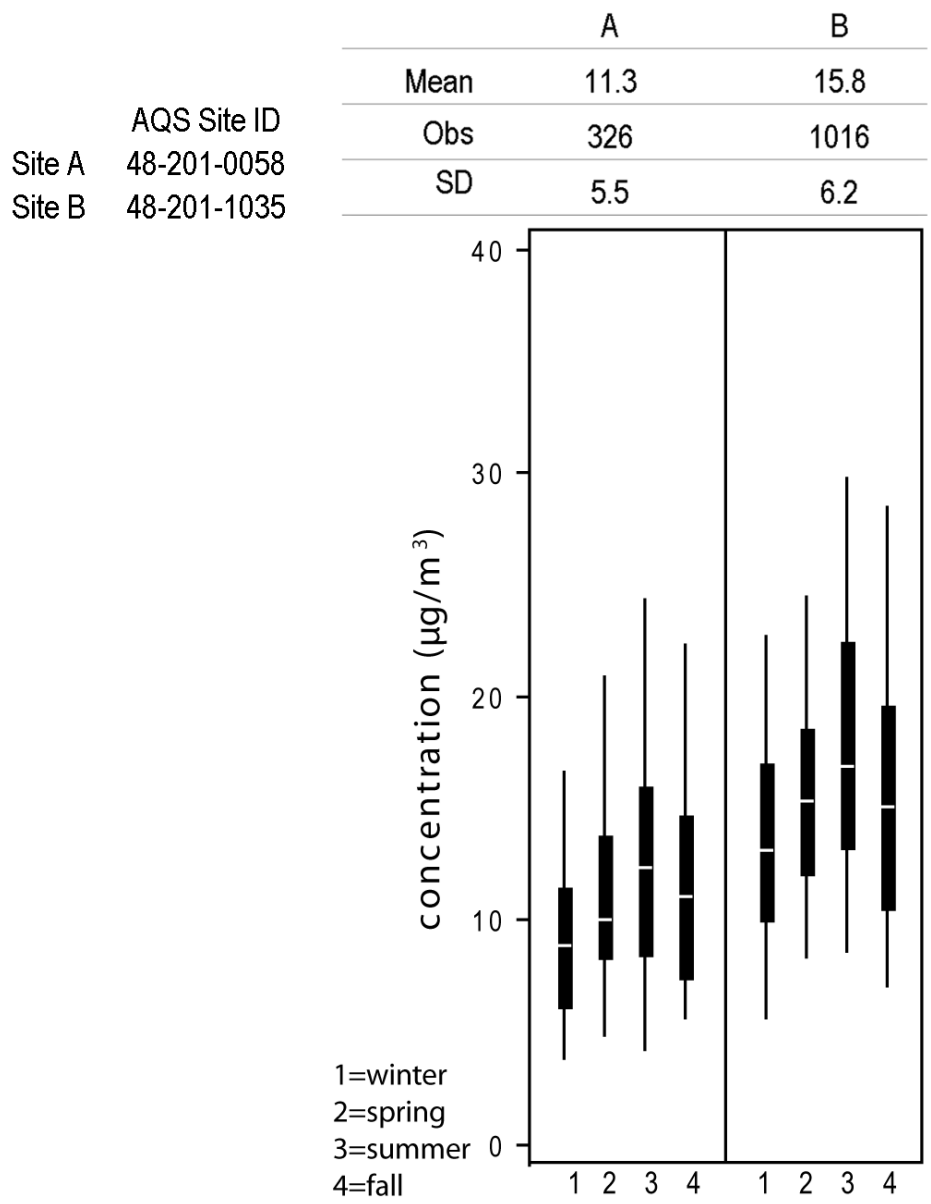


Figure A-51. Box plots illustrating the seasonal distribution of 24-h avg $PM_{2.5}$ concentrations for Houston, TX.

Table A-6. Inter-sampler correlation statistics for each pair of $PM_{2.5}$ monitors reporting to AQS for Houston, TX.

		A	B
A		1.00	0.66
		(0.0, 0.00)	(10.0, 0.24)
		326	310
B			1.00
			(0.0, 0.00)
			1016

R (P90, COD) N	

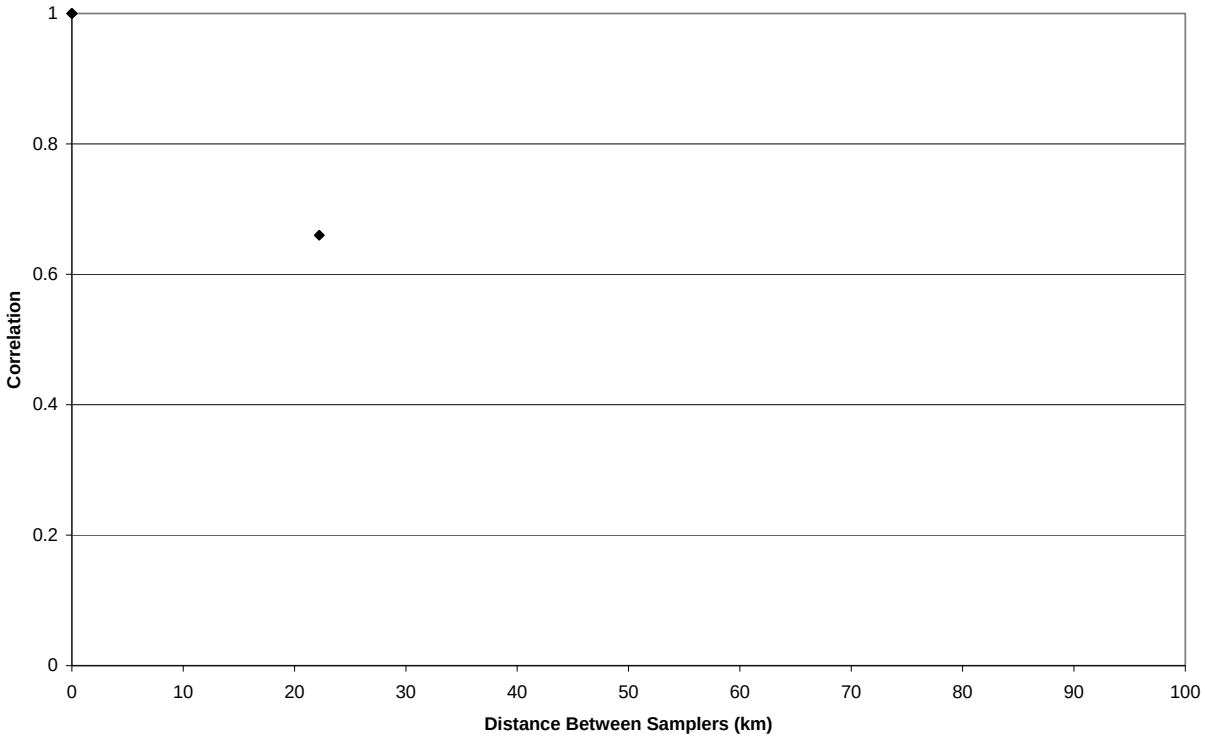


Figure A-52. $PM_{2.5}$ inter-sampler correlations as a function of distance between monitors for Houston, TX.

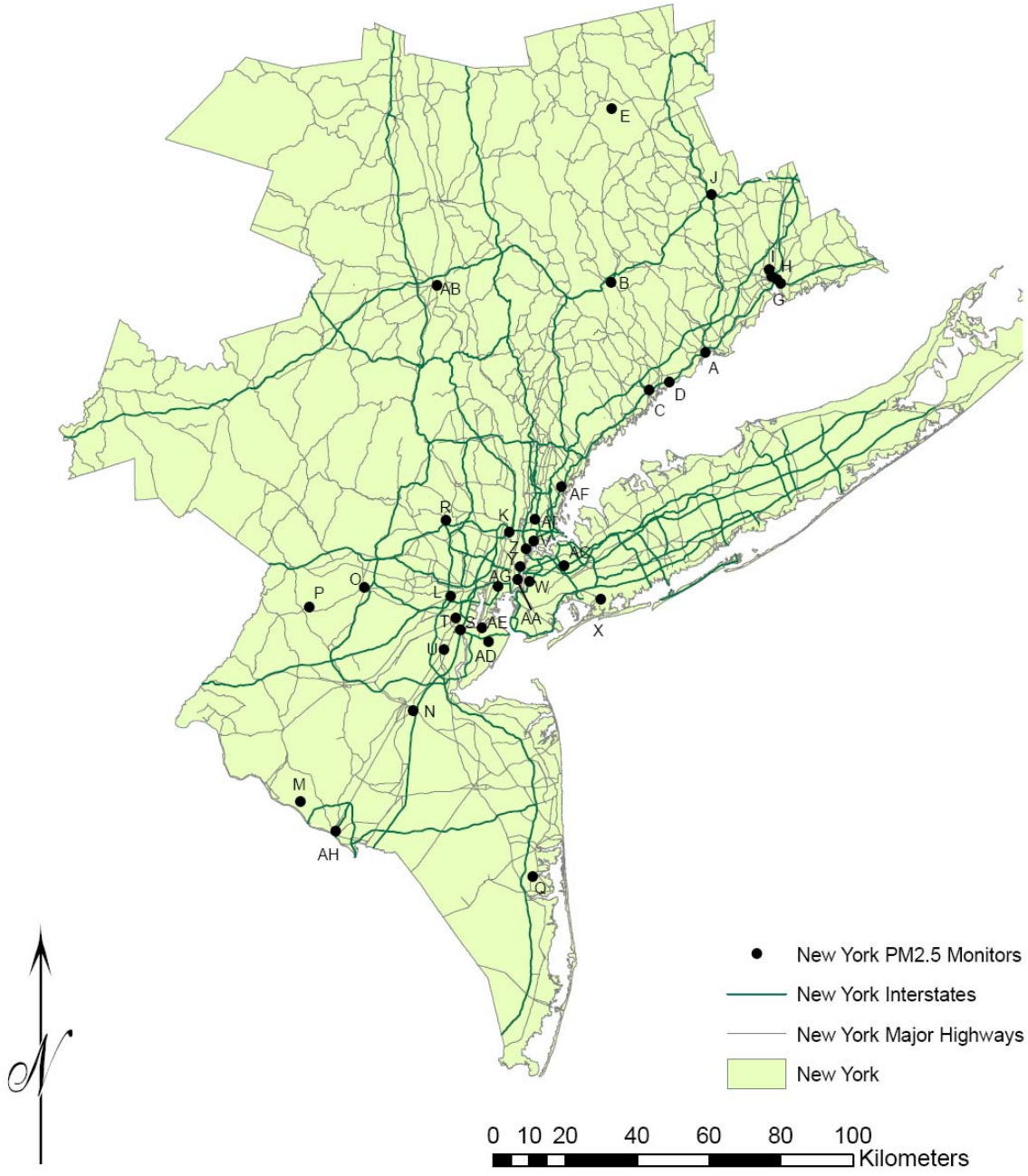
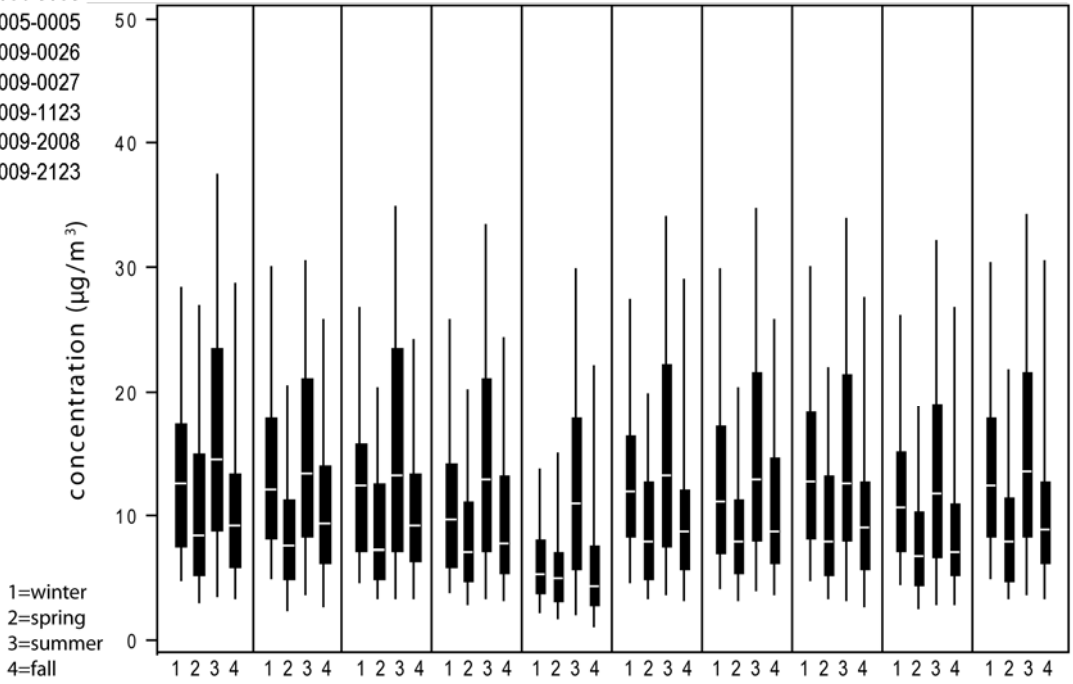
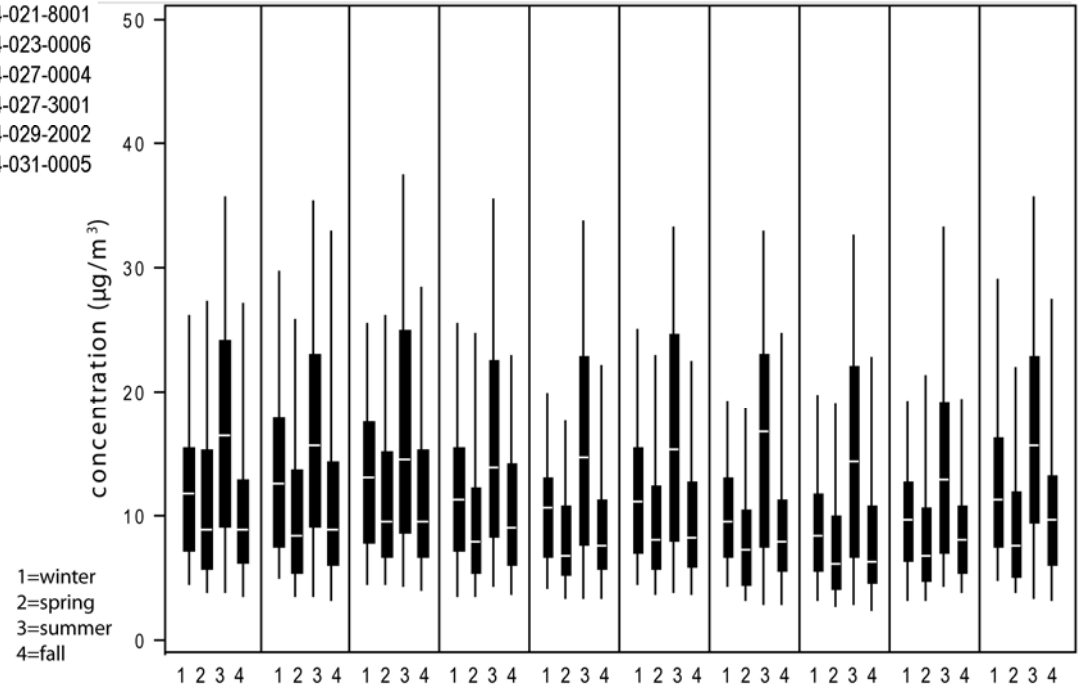


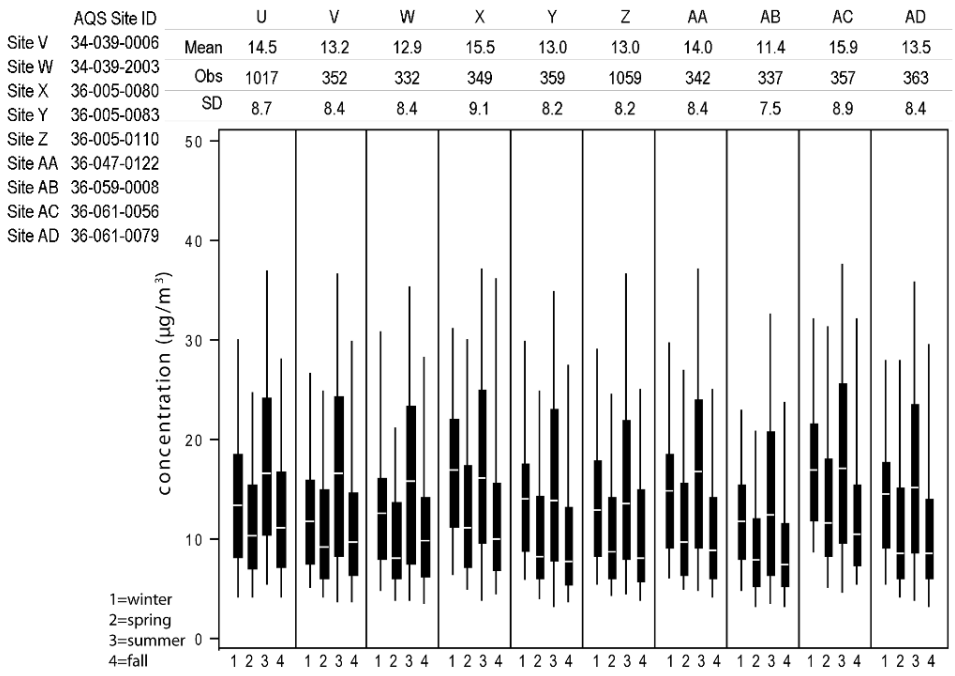
Figure A-53. $PM_{2.5}$ monitor distribution and major highways, New York City, NY.

	AQS Site ID	A	B	C	D	E	F	G	H	I	J
Site A	09-001-0010	Mean	13.2	12.5	12.3	11.2	8.0	12.1	12.4	12.8	11.1	12.6
Site B	09-001-1123	Obs	349	339	332	565	341	341	992	338	344	352
Site C	09-001-3005	SD	8.7	8.3	8.1	7.6	6.8	8.0	8.1	8.6	7.5	8.2
Site D	09-001-9003											
Site E	09-005-0005											
Site F	09-009-0026											
Site G	09-009-0027											
Site H	09-009-1123											
Site I	09-009-2008											
Site J	09-009-2123											



	AQS Site ID	K	L	M	N	O	P	Q	R	S	T
Site K	34-003-0003	Mean	13.2	13.3	13.7	12.3	10.8	12.1	11.3	10.0	10.6	12.9
Site L	34-013-0015	Obs	345	334	559	545	313	336	336	357	550	330
Site M	34-017-1003	SD	8.9	8.8	8.5	7.7	7.0	7.7	7.7	7.4	6.8	8.7
Site N	34-021-0008											
Site O	34-021-8001											
Site P	34-023-0006											
Site Q	34-027-0004											
Site R	34-027-3001											
Site S	34-029-2002											
Site T	34-031-0005											





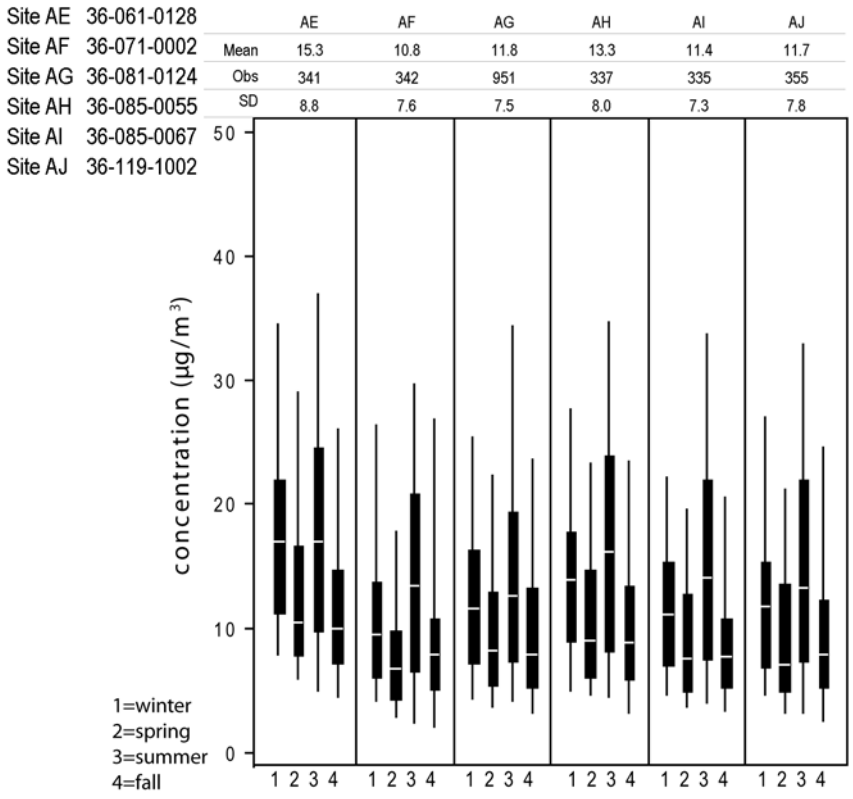


Figure A-54. Box plots illustrating the seasonal distribution of 24-h avg $PM_{2.5}$ concentrations for New York City, NY.

Table A-7. Inter-sampler correlation statistics for each pair of $PM_{2.5}$ monitors reporting to AQS for New York City, NY.

Site	A	B	C	D	E	F	G	H	I	J	K	L	M	N	O	P	Q	R
A	1.00	0.89	0.97	0.97	0.82	0.96	0.96	0.96	0.96	0.93	0.91	0.91	0.92	0.88	0.84	0.87	0.89	0.84
	(0.0, 0.00)	(5.3, 0.15)	(3.6, 0.09)	(4.8, 0.11)	(11.8, 0.33)	(3.8, 0.11)	(4.0, 0.11)	(3.4, 0.10)	(4.6, 0.12)	(5.1, 0.12)	(5.8, 0.12)	(5.7, 0.12)	(5.5, 0.13)	(6.6, 0.16)	(9.1, 0.19)	(8.3, 0.16)	(7.6, 0.16)	(9.3, 0.21)
	349	322	316	322	325	328	321	324	326	335	329	316	331	301	296	321	318	316
B		1.00	0.93	0.91	0.78	0.91	0.92	0.91	0.91	0.92	0.83	0.84	0.85	0.82	0.79	0.82	0.82	0.78
		(0.0, 0.00)	(4.5, 0.13)	(5.3, 0.14)	(10.4, 0.32)	(4.7, 0.13)	(4.6, 0.13)	(4.6, 0.14)	(5.0, 0.14)	(4.5, 0.13)	(7.3, 0.17)	(7.1, 0.17)	(7.8, 0.19)	(7.2, 0.19)	(7.7, 0.20)	(7.6, 0.18)	(6.6, 0.18)	(8.4, 0.22)
		339	312	315	319	316	313	313	315	330	319	305	321	291	292	310	307	305
C			1.00	0.98	0.82	0.96	0.95	0.96	0.97	0.94	0.91	0.91	0.91	0.89	0.84	0.88	0.89	0.84
			(0.0, 0.00)	(3.4, 0.08)	(10.8, 0.32)	(3.9, 0.10)	(4.1, 0.11)	(3.6, 0.10)	(4.0, 0.11)	(4.8, 0.11)	(5.7, 0.13)	(5.8, 0.14)	(6.5, 0.15)	(5.4, 0.15)	(6.9, 0.17)	(6.3, 0.14)	(6.2, 0.15)	(8.2, 0.20)
			332	314	309	310	308	307	310	319	314	299	316	287	289	307	305	297
D				1.00	0.85	0.96	0.96	0.94	0.96	0.92	0.90	0.89	0.91	0.88	0.87	0.89	0.90	0.86
				(0.0, 0.00)	(8.4, 0.29)	(3.4, 0.11)	(3.8, 0.11)	(5.0, 0.13)	(3.0, 0.10)	(5.5, 0.13)	(7.1, 0.15)	(6.9, 0.15)	(6.7, 0.18)	(6.3, 0.17)	(6.5, 0.16)	(6.0, 0.15)	(5.5, 0.14)	(6.6, 0.18)
				565	314	316	532	315	313	325	319	308	517	506	288	311	309	330
E					1.00	0.82	0.82	0.79	0.83	0.81	0.80	0.77	0.76	0.76	0.79	0.78	0.87	0.87
					(0.0, 0.00)	(10.0, 0.31)	(10.7, 0.33)	(11.4, 0.33)	(8.8, 0.28)	(10.3, 0.32)	(12.5, 0.34)	(13.0, 0.34)	(13.8, 0.39)	(11.6, 0.35)	(9.1, 0.30)	(10.4, 0.32)	(7.9, 0.28)	(7.3, 0.24)
					341	321	313	317	319	330	322	305	323	294	291	316	311	305
F						1.00	0.99	0.98	0.98	0.94	0.88	0.89	0.89	0.86	0.85	0.88	0.87	0.83
						(0.0, 0.00)	(2.1, 0.07)	(2.9, 0.09)	(2.8, 0.09)	(4.7, 0.11)	(6.7, 0.14)	(6.8, 0.15)	(6.8, 0.16)	(6.4, 0.17)	(6.8, 0.18)	(6.1, 0.15)	(7.3, 0.16)	(7.5, 0.21)
						341	314	319	321	328	321	308	323	293	295	312	310	308
G							1.00	0.96	0.98	0.93	0.88	0.89	0.89	0.84	0.84	0.86	0.87	0.82
							(0.0, 0.00)	(2.9, 0.10)	(3.6, 0.11)	(5.2, 0.12)	(7.1, 0.15)	(6.7, 0.15)	(6.9, 0.16)	(6.9, 0.18)	(8.0, 0.19)	(7.6, 0.16)	(8.1, 0.17)	(8.4, 0.23)
							992	315	319	326	319	309	526	513	286	310	306	327
H								1.00	0.98	0.94	0.88	0.89	0.89	0.84	0.82	0.85	0.85	0.79
								(0.0, 0.00)	(3.7, 0.10)	(3.7, 0.10)	(7.1, 0.14)	(7.1, 0.14)	(6.6, 0.16)	(6.7, 0.18)	(8.1, 0.20)	(7.8, 0.17)	(7.5, 0.17)	(9.2, 0.23)
								338	320	324	318	303	321	292	285	310	307	304
I									1.00	0.95	0.89	0.90	0.89	0.87	0.85	0.87	0.88	0.83
									(0.0, 0.00)	(4.1, 0.11)	(7.0, 0.16)	(7.0, 0.16)	(7.7, 0.20)	(6.4, 0.18)	(6.6, 0.17)	(6.5, 0.16)	(6.5, 0.15)	(7.6, 0.19)
									344	327	324	307	323	296	291	313	313	310
J										1.00	0.87	0.87	0.87	0.84	0.79	0.82	0.84	0.79
										(0.0, 0.00)	(7.0, 0.16)	(7.2, 0.16)	(8.5, 0.17)	(6.9, 0.18)	(7.9, 0.20)	(8.1, 0.18)	(7.5, 0.17)	(9.0, 0.22)
										352	332	316	334	303	299	321	322	316
K											1.00	0.95	0.93	0.88	0.86	0.90	0.92	0.86
			R (P90, COD)								(0.0, 0.00)	(3.4, 0.09)	(4.5, 0.12)	(6.4, 0.15)	(7.5, 0.17)	(5.7, 0.13)	(5.8, 0.14)	(8.7, 0.20)
											345	314	330	301	296	317	319	312
L			N									1.00	0.97	0.91	0.86	0.94	0.93	0.87
												(0.0, 0.00)	(4.1, 0.10)	(6.4, 0.14)	(8.0, 0.18)	(5.2, 0.12)	(5.9, 0.13)	(8.3, 0.20)
												334	321	289	288	309	303	301
M													1.00	0.91	0.86	0.93	0.92	0.85
													(0.0, 0.00)	(5.5, 0.14)	(8.4, 0.21)	(6.7, 0.15)	(7.5, 0.18)	(9.7, 0.25)
													559	499	300	326	318	337
N														1.00	0.93	0.95	0.91	0.88
														(0.0, 0.00)	(4.7, 0.14)	(4.1, 0.11)	(5.8, 0.15)	(7.2, 0.20)
														545	270	293	292	316
O															1.00	0.93	0.91	0.94
															(0.0, 0.00)	(4.3, 0.12)	(4.9, 0.14)	(4.3, 0.14)
															313	294	287	279
P																1.00	0.94	0.91
																(0.0, 0.00)	(4.9, 0.12)	(5.5, 0.16)
																336	308	303
Q																	1.00	0.95
																	(0.0, 0.00)	(3.8, 0.13)
																	336	307
R																		1.00
																		(0.0, 0.00)
																		357

	S	T	U	V	W	X	Y	Z	AA	AB	AC	AD	AE	AF	AG	AH	AI	AJ
A	0.75	0.89	0.90	0.90	0.88	0.92	0.94	0.93	0.93	0.88	0.89	0.94	0.88	0.89	0.93	0.90	0.89	0.96
	(10.4, 0.21)	(6.1, 0.13)	(7.1, 0.15)	(6.0, 0.13)	(7.2, 0.15)	(7.2, 0.16)	(4.0, 0.11)	(4.7, 0.12)	(5.5, 0.13)	(7.6, 0.18)	(7.3, 0.18)	(4.4, 0.11)	(7.2, 0.19)	(6.7, 0.16)	(5.1, 0.12)	(6.2, 0.15)	(7.5, 0.16)	(4.4, 0.12)
	323	315	337	299	316	332	342	348	325	320	340	346	326	323	299	317	318	338
B	0.68	0.84	0.83	0.83	0.84	0.84	0.88	0.85	0.84	0.81	0.81	0.86	0.81	0.92	0.84	0.87	0.86	0.88

	S	T	U	V	W	X	Y	Z	AA	AB	AC	AD	AE	AF	AG	AH	AI	AJ
	(10.8,	(5.9,	(8.6,	(6.5,	(6.8,	(9.0,	(5.9,	(6.8,	(7.3,	(7.9,	(8.4,	(7.0,	(8.8,	(5.8,	(6.6,	(6.8,	(7.1,	(5.5,
	0.23)	0.16)	0.20)	0.18)	0.18)	0.21)	0.16)	0.17)	0.18)	0.20)	0.22)	0.17)	0.23)	0.16)	0.17)	0.18)	0.18)	0.15)
	314	307	328	290	305	325	334	338	317	313	331	336	315	316	292	311	309	329
C	0.76	0.89	0.88	0.89	0.89	0.92	0.95	0.93	0.92	0.89	0.88	0.93	0.88	0.89	0.93	0.91	0.89	0.96
	(8.5,	(6.1,	(7.8,	(6.4,	(6.0,	(7.8,	(4.4,	(5.4,	(5.6,	(6.1,	(7.5,	(5.3,	(7.4,	(6.7,	(4.7,	(5.7,	(6.0,	(3.5,
	0.20)	0.14)	0.18)	0.15)	0.15)	0.18)	0.11)	0.13)	0.15)	0.16)	0.20)	0.12)	0.20)	0.15)	0.11)	0.14)	0.15)	0.10)
	307	304	321	283	297	317	326	331	311	306	325	330	308	312	282	305	302	322
D	0.80	0.88	0.88	0.87	0.88	0.89	0.94	0.92	0.91	0.89	0.87	0.92	0.85	0.90	0.92	0.90	0.91	0.96
	(7.7,	(7.3,	(8.1,	(7.1,	(6.9,	(9.7,	(5.6,	(6.2,	(7.0,	(5.9,	(9.6,	(6.6,	(9.2,	(5.4,	(4.8,	(6.5,	(5.3,	(3.7,
	0.19)	0.16)	0.20)	0.17)	0.17)	0.21)	0.14)	0.15)	0.17)	0.16)	0.23)	0.15)	0.23)	0.14)	0.12)	0.16)	0.14)	0.10)
	509	306	537	326	304	324	332	548	315	313	330	336	315	313	496	308	310	328
E	0.67	0.79	0.74	0.76	0.75	0.75	0.80	0.78	0.76	0.73	0.74	0.79	0.70	0.88	0.77	0.75	0.80	0.84
	(9.8,	(11.3,	(14.9,	(11.7,	(12.1,	(15.2,	(11.5,	(13.1,	(13.9,	(10.1,	(15.7,	(13.1,	(15.0,	(7.6,	(11.3,	(12.5,	(9.4,	(9.8,
	0.32)	0.34)	0.40)	0.36)	0.36)	0.41)	0.34)	0.36)	0.38)	0.33)	0.43)	0.35)	0.42)	0.26)	0.32)	0.36)	0.31)	0.29)
	315	306	329	290	307	324	334	340	319	314	332	338	316	316	294	310	309	331
F	0.79	0.87	0.86	0.86	0.87	0.89	0.93	0.91	0.90	0.90	0.87	0.91	0.86	0.89	0.93	0.89	0.89	0.94
	(7.9,	(6.7,	(8.5,	(6.8,	(6.6,	(8.2,	(5.0,	(6.4,	(6.7,	(5.6,	(8.4,	(6.3,	(8.0,	(6.4,	(4.6,	(5.7,	(5.3,	(4.1,
	0.19)	0.15)	0.19)	0.16)	0.16)	0.19)	0.12)	0.14)	0.15)	0.16)	0.20)	0.14)	0.21)	0.16)	0.12)	0.15)	0.15)	0.12)
	316	306	329	293	309	325	335	340	320	317	334	339	319	317	290	312	310	332
G	0.77	0.87	0.87	0.86	0.88	0.89	0.92	0.90	0.90	0.88	0.86	0.91	0.85	0.88	0.90	0.89	0.88	0.93
	(8.7,	(6.3,	(7.8,	(7.0,	(6.3,	(8.3,	(5.4,	(5.7,	(7.1,	(7.5,	(8.1,	(6.4,	(8.2,	(6.7,	(5.2,	(6.1,	(6.1,	(5.0,
	0.21)	0.15)	0.18)	0.16)	0.15)	0.17)	0.13)	0.14)	0.15)	0.17)	0.19)	0.14)	0.20)	0.17)	0.13)	0.15)	0.16)	0.14)
	513	304	928	327	303	319	329	958	315	308	327	333	314	311	856	312	309	325
H	0.74	0.86	0.86	0.87	0.87	0.90	0.92	0.89	0.90	0.88	0.87	0.91	0.86	0.88	0.90	0.88	0.86	0.93
	(9.6,	(6.6,	(8.4,	(7.1,	(6.9,	(7.5,	(5.2,	(5.6,	(6.4,	(7.3,	(7.9,	(5.7,	(7.3,	(6.9,	(5.6,	(6.8,	(6.4,	(5.0,
	0.22)	0.15)	0.18)	0.16)	0.16)	0.17)	0.13)	0.14)	0.15)	0.19)	0.19)	0.13)	0.20)	0.17)	0.14)	0.16)	0.17)	0.13)
	314	304	326	289	306	322	331	337	315	310	329	335	313	313	290	308	308	327
I	0.76	0.88	0.87	0.88	0.88	0.90	0.93	0.92	0.91	0.87	0.87	0.92	0.86	0.90	0.93	0.89	0.89	0.94
	(8.1,	(7.1,	(8.7,	(7.4,	(6.9,	(9.4,	(5.7,	(6.5,	(7.2,	(6.2,	(9.6,	(6.3,	(9.2,	(5.5,	(5.1,	(6.7,	(5.8,	(4.1,
	0.20)	0.17)	0.21)	0.17)	0.17)	0.22)	0.15)	0.16)	0.18)	0.17)	0.24)	0.16)	0.24)	0.13)	0.14)	0.18)	0.16)	0.12)
	315	308	332	293	309	326	334	343	323	313	332	338	318	319	296	310	311	330
J	0.67	0.84	0.85	0.86	0.86	0.88	0.90	0.88	0.86	0.81	0.85	0.89	0.84	0.90	0.88	0.87	0.84	0.91
	(11.1,	(6.6,	(9.0,	(6.7,	(6.8,	(8.8,	(6.1,	(7.1,	(7.3,	(8.2,	(9.0,	(6.9,	(8.9,	(6.4,	(6.4,	(7.5,	(7.7,	(5.6,
	0.22)	0.16)	0.19)	0.16)	0.17)	0.19)	0.14)	0.16)	0.17)	0.19)	0.21)	0.15)	0.22)	0.16)	0.15)	0.16)	0.18)	0.14)
	327	316	343	301	318	337	345	351	330	324	343	349	327	329	301	321	320	341
K	0.74	0.94	0.92	0.95	0.92	0.92	0.93	0.92	0.93	0.84	0.90	0.93	0.88	0.89	0.90	0.91	0.89	0.92
	(10.9,	(3.9,	(5.7,	(3.4,	(4.3,	(6.0,	(3.8,	(4.2,	(4.3,	(8.5,	(6.2,	(3.8,	(6.2,	(7.4,	(5.9,	(5.0,	(6.5,	(4.8,
	0.21)	0.11)	0.14)	0.10)	0.12)	0.15)	0.12)	0.12)	0.12)	0.19)	0.17)	0.11)	0.18)	0.17)	0.13)	0.13)	0.15)	0.13)
	320	317	336	302	317	330	339	344	324	318	338	343	321	321	294	318	314	335
L	0.78	0.94	0.97	0.98	0.95	0.94	0.93	0.94	0.95	0.86	0.91	0.95	0.91	0.89	0.92	0.96	0.92	0.92
	(9.8,	(3.9,	(4.5,	(2.9,	(4.0,	(6.3,	(4.5,	(4.2,	(4.1,	(8.1,	(6.3,	(4.2,	(5.9,	(6.8,	(5.4,	(4.0,	(6.4,	(5.4,
	0.20)	0.11)	0.12)	0.08)	0.10)	0.15)	0.12)	0.11)	0.11)	0.18)	0.17)	0.10)	0.17)	0.17)	0.12)	0.11)	0.14)	0.13)
	313	303	325	292	303	314	323	333	306	305	322	327	309	306	283	305	299	319
M	0.80	0.93	0.97	0.97	0.96	0.95	0.95	0.96	0.97	0.88	0.94	0.96	0.93	0.88	0.95	0.96	0.93	0.93
	(9.9,	(5.4,	(3.8,	(3.5,	(4.7,	(4.9,	(4.7,	(3.5,	(3.4,	(8.3,	(4.5,	(3.5,	(4.5,	(8.5,	(5.0,	(4.3,	(6.8,	(5.7,
	0.22)	0.13)	0.09)	0.09)	0.11)	0.11)	0.12)	0.10)	0.09)	0.20)	0.12)	0.10)	0.13)	0.21)	0.14)	0.10)	0.16)	0.17)
	504	318	534	341	319	331	342	545	326	320	339	345	326	323	484	319	318	338
N	0.88	0.86	0.90	0.91	0.91	0.86	0.90	0.89	0.91	0.89	0.85	0.90	0.85	0.85	0.91	0.92	0.92	0.90
	(6.4,	(6.5,	(6.5,	(5.7,	(4.5,	(8.2,	(5.9,	(5.4,	(5.3,	(5.6,	(8.1,	(5.3,	(7.7,	(7.5,	(4.9,	(4.6,	(5.3,	(5.4,
	0.17)	0.16)	0.15)	0.13)	0.13)	0.18)	0.14)	0.15)	0.14)	0.17)	0.18)	0.14)	0.18)	0.20)	0.14)	0.13)	0.14)	0.16)
	492	287	519	313	290	301	309	529	297	289	308	313	294	292	477	292	293	306
O	0.87	0.82	0.86	0.87	0.88	0.84	0.87	0.86	0.88	0.89	0.82	0.88	0.81	0.84	0.88	0.89	0.92	0.88
	(5.6,	(7.2,	(9.9,	(7.3,	(6.4,	(11.1,	(6.7,	(8.6,	(8.2,	(5.2,	(10.3,	(8.4,	(10.6,	(7.0,	(6.1,	(6.1,	(4.7,	(5.4,
	0.16)	0.18)	0.22)	0.18)	0.16)	0.24)	0.18)	0.19)	0.20)	0.15)	0.25)	0.18)	0.25)	0.18)	0.16)	0.18)	0.14)	0.16)
	295	289	302	280	284	299	308	312	292	295	307	311	290	290	269	290	283	304
P	0.86	0.89	0.92	0.94	0.95	0.88	0.90	0.89	0.92	0.89	0.87	0.91	0.87	0.87	0.91	0.94	0.95	0.91
	(6.2,	(6.2,	(7.4,	(5.0,	(4.0,	(8.9,	(5.9,	(6.8,	(6.4,	(5.3,	(8.9,	(6.4,	(8.3,	(6.7,	(5.5,	(4.7,	(3.5,	(5.0,
	0.15)	0.14)	0.17)	0.12)	0.11)	0.19)	0.14)	0.15)	0.14)	0.14)	0.21)	0.14)	0.20)	0.16)	0.13)	0.12)	0.10)	0.14)
	312	307	325	296	305	319	329	335	312	309	327	333	313	311	285	307	306	326
Q	0.79	0.92	0.91	0.93	0.92	0.89	0.90	0.90	0.90	0.83	0.90	0.91	0.87	0.91	0.89	0.90	0.91	0.92
	(8.1,	(5.0,	(8.2,	(6.2,	(5.4,	(9.7,	(6.3,	(7.3,	(7.3,	(6.9,	(9.9,	(6.9,	(9.5,	(4.8,	(6.0,	(6.3,	(5.3,	(5.5,
	0.19)	0.14)	0.20)	0.14)	0.15)	0.22)	0.16)	0.17)	0.18)	0.19)	0.24)	0.16)	0.24)	0.14)	0.15)	0.17)	0.14)	0.13)
	313	303	327	287	304	321	328	335	314	306	329	332	311	312	287	303	302	324
R	0.82	0.86	0.84	0.85	0.87	0.82	0.87	0.84	0.86	0.83	0.81	0.86	0.78	0.88	0.86	0.87	0.90	0.88
	(6.5,	(7.6,	(10.9,	(8.2,	(7.0,	(11.6,	(8.6,	(10.0,	(9.1,	(7.0,	(11.2,	(9.5,	(11.0,	(6.2,	(7.6,	(7.8,	(5.7,	(6.3,
	0.20)	0.21)	0.26)	0.21)	0.20)	0.28)	0.21)	0.23)	0.24)	0.21)	0.30)	0.22)	0.30)	0.17)	0.20)	0.22)	0.17)	0.17)
	330	296	347	291	304	314	323	355	309	301	324	327	309	308	304	298	302	320
S	1.00	0.69	0.77	0.75	0.78	0.72	0.78	0.79	0.82	0.88	0.74	0.78	0.73	0.69	0.85	0.82	0.88	0.79
	(0.0,	(10.4,	(10.5,	(10.5,	(9.2,	(12.5,	(8.6,	(9.3,	(9.4,	(5.0,	(11.6,	(9.9,	(11.5,	(10.3,	(7.2,	(8.5,	(5.5,	(8.1,
	0.00)	0.22)	0.24)	0.21)	0.19)	0.25)	0.19)	0.21)	0.20)	0.16)	0.26)	0.20)	0.25)	0.22)	0.17)	0.18)	0.14)	0.19)
	550	306	525	324	306	325	336	536	319	314	333	339	322	316	478	310	312	331
T		1.00	0.92	0.93	0.93	0.92	0.91	0.91	0.90	0.80	0.87	0.93	0.85	0.89	0.88	0.90	0.86	0.91
		(0.0,	(6.0,	(4.5,	(4.8,	(6.7,	(5.2,	(4.9,	(5.6,	(8.4,	(6.9,	(4.9,	(7.2,	(6.3,	(6.2,	(5.8,	(7.3,	(5.9,
		0.00)	0.15)	0.12)	0.13)	0.16)	0.14)	0.13)	0.14)	0.20)	0.18)	0.12)	0.20)	0.17)	0.14)	0.14)	0.17)	0.14)
		330	319	293	301	313	323	329	308	303	321	327	306	308	281	306	298	319
U			1.00	0.98	0.97	0.92	0.91	0.93	0.94	0.84	0.91	0.94	0.90	0.86	0.90	0.96	0.91	0.90
			(0.0,	(3.9,	(5.0,	(5.4,	(6.9,	(5.2,	(4.9,	(9.9,	(5.0,	(5.2,	(5.9,	(9.6,	(6.9,	(5.8,	(8.4,	(7.5,
			0.00)	0.10)	0.12)	0.12)	0.15)	0.13)	0.12)	0.22)	0.12)	0.12)	0.14)	0.23)	0.17)	0.12)	0.18)	0.19)
			1017	341	325	337	347	987	332	326	346	351	330	330	878	325	323	343
V				1.00	0.97	0.93	0.92	0.92	0.94	0.83	0.90	0.94	0.91	0.88	0.89	0.96	0.91	0.91
				(0.0,	(2.8,	(6.1,	(5.0,	(4.4,	(4.2,	(8.2,	(6.6,	(4.3,	(6.1,	(7.0,	(5.8,	(4.0,	(6.4,	(5.3,
				0.00)	0.09)	0.14)	0.13)	0.12)	0.12)	0.19)	0.16)	0.11)	0.16)	0.18)	0.15)	0.10)	0.15)	0.15)
				352	288	300	307	351	294	290	305	311	294	290	301	291	287	304
W					1.00	0.90	0.91	0.91	0.92	0.85	0.89	0.93	0.88	0.87	0.89	0.96	0.92	0.90

S	T	U	V	W	X	Y	Z	AA	AB	AC	AD	AE	AF	AG	AH	AI	AJ
				(0.0, 0.00)	(7.0, 0.16)	(5.5, 0.13)	(5.3, 0.13)	(4.8, 0.13)	(7.0, 0.18)	(6.8, 0.17)	(5.0, 0.12)	(6.9, 0.18)	(6.8, 0.18)	(5.1, 0.14)	(3.7, 0.10)	(4.9, 0.13)	(5.0, 0.15)
				332	316	325	331	310	309	323	328	308	310	281	304	303	320
X					1.00	0.96	0.97	0.95	0.86	0.94	0.97	0.93	0.88	0.93	0.92	0.88	0.93
					(0.0, 0.00)	(5.8, 0.13)	(4.4, 0.11)	(5.0, 0.11)	(10.0, 0.23)	(3.3, 0.09)	(4.5, 0.11)	(4.1, 0.11)	(9.8, 0.24)	(6.9, 0.17)	(6.5, 0.14)	(9.2, 0.20)	(8.2, 0.19)
					349	344	349	328	324	342	348	326	326	301	319	319	340
Y						1.00	0.97	0.96	0.90	0.93	0.98	0.93	0.89	0.97	0.93	0.92	0.97
						(0.0, 0.00)	(3.2, 0.08)	(3.9, 0.09)	(6.5, 0.16)	(5.4, 0.15)	(2.8, 0.08)	(5.4, 0.15)	(6.5, 0.18)	(3.3, 0.09)	(4.9, 0.12)	(5.3, 0.13)	(3.5, 0.11)
						359	359	338	333	352	358	337	335	308	328	329	350
Z							1.00	0.97	0.90	0.94	0.98	0.92	0.88	0.95	0.93	0.91	0.95
							(0.0, 0.00)	(2.9, 0.09)	(7.2, 0.17)	(4.4, 0.13)	(1.8, 0.07)	(4.6, 0.14)	(7.8, 0.19)	(4.0, 0.10)	(4.7, 0.11)	(6.1, 0.14)	(4.9, 0.14)
							1059	342	337	357	363	341	342	919	337	335	355
AA								1.00	0.92	0.94	0.98	0.93	0.87	0.97	0.95	0.94	0.95
								(0.0, 0.00)	(7.1, 0.18)	(3.8, 0.11)	(2.9, 0.07)	(4.1, 0.11)	(8.1, 0.20)	(4.0, 0.11)	(4.3, 0.10)	(6.6, 0.15)	(5.1, 0.15)
								342	317	336	341	319	319	292	313	312	335
AB									1.00	0.85	0.89	0.86	0.79	0.95	0.90	0.93	0.90
									(0.0, 0.00)	(9.2, 0.24)	(7.2, 0.17)	(9.0, 0.23)	(8.1, 0.20)	(4.1, 0.13)	(6.1, 0.16)	(3.8, 0.12)	(5.5, 0.16)
									337	330	337	316	313	291	310	310	329
AC										1.00	0.95	0.98	0.84	0.96	0.91	0.89	0.91
										(0.0, 0.00)	(4.4, 0.13)	(3.0, 0.08)	(10.4, 0.26)	(6.6, 0.18)	(6.6, 0.15)	(9.3, 0.21)	(7.4, 0.22)
										357	356	334	336	304	326	326	348
AD											1.00	0.93	0.89	0.97	0.95	0.93	0.96
											(0.0, 0.00)	(4.6, 0.14)	(7.1, 0.18)	(4.0, 0.10)	(4.4, 0.10)	(6.0, 0.13)	(4.6, 0.13)
											363	341	339	311	333	333	354
AE												1.00	0.82	0.94	0.92	0.89	0.89
												(0.0, 0.00)	(10.0, 0.26)	(6.2, 0.18)	(5.6, 0.15)	(8.4, 0.20)	(8.0, 0.22)
												341	319	290	313	314	332
AF													1.00	0.86	0.87	0.87	0.91
													(0.0, 0.00)	(7.0, 0.16)	(7.1, 0.18)	(6.4, 0.16)	(5.5, 0.14)
													342	289	310	313	331
AG														1.00	0.93	0.94	0.96
														(0.0, 0.00)	(4.8, 0.12)	(4.5, 0.11)	(3.7, 0.11)
														951	289	283	304
AH															1.00	0.97	0.92
															(0.0, 0.00)	(4.1, 0.10)	(4.9, 0.15)
															337	307	327
AI																1.00	0.92
																(0.0, 0.00)	(4.8, 0.14)
																335	324
AJ																	1.00
																	(0.0, 0.00)
																	355

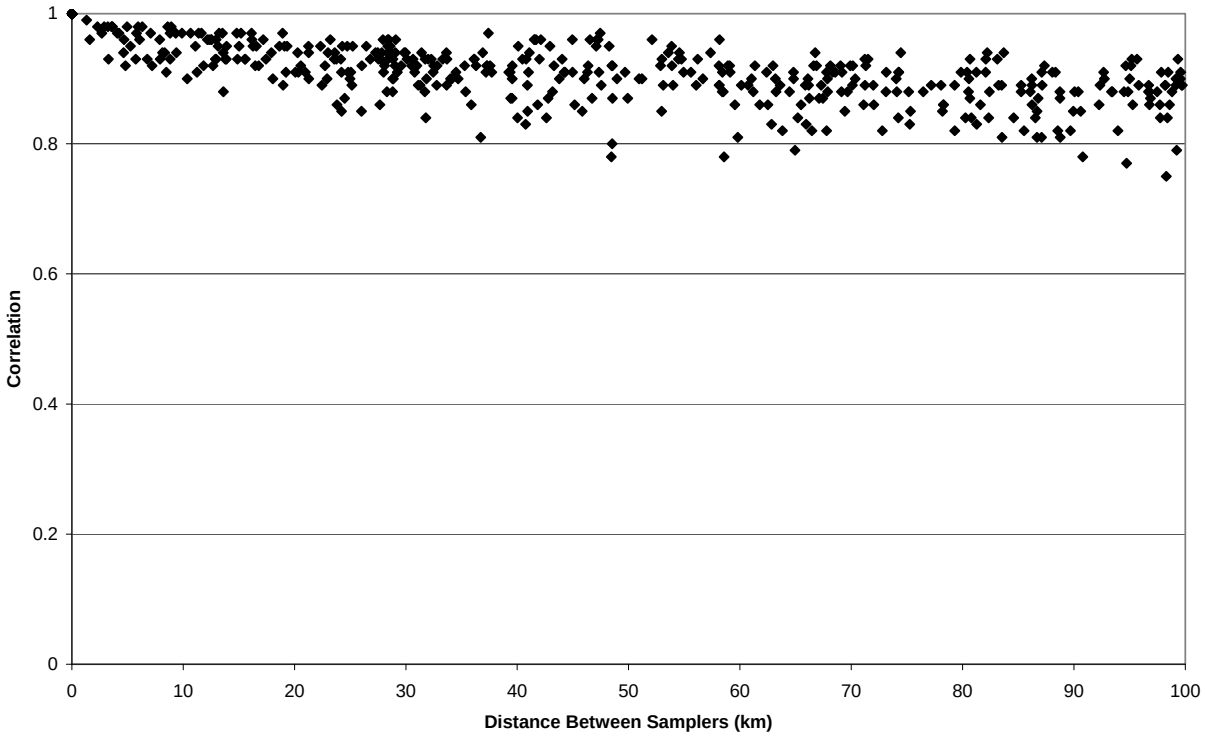


Figure A-55 $PM_{2.5}$ inter-sampler correlations as a function of distance between monitors for New York City, NY.

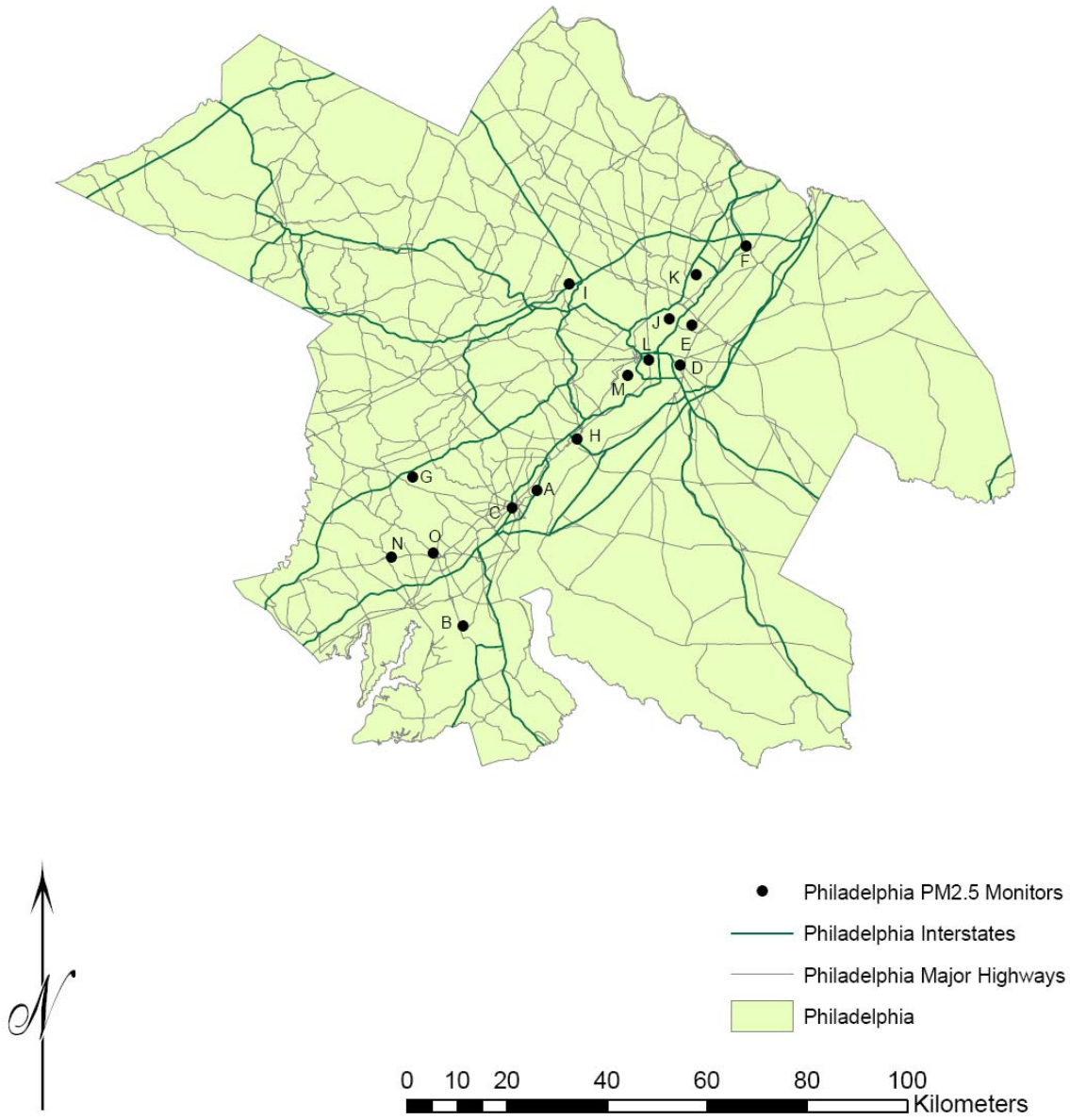


Figure A-56. $PM_{2.5}$ monitor distribution and major highways, Philadelphia, PA.

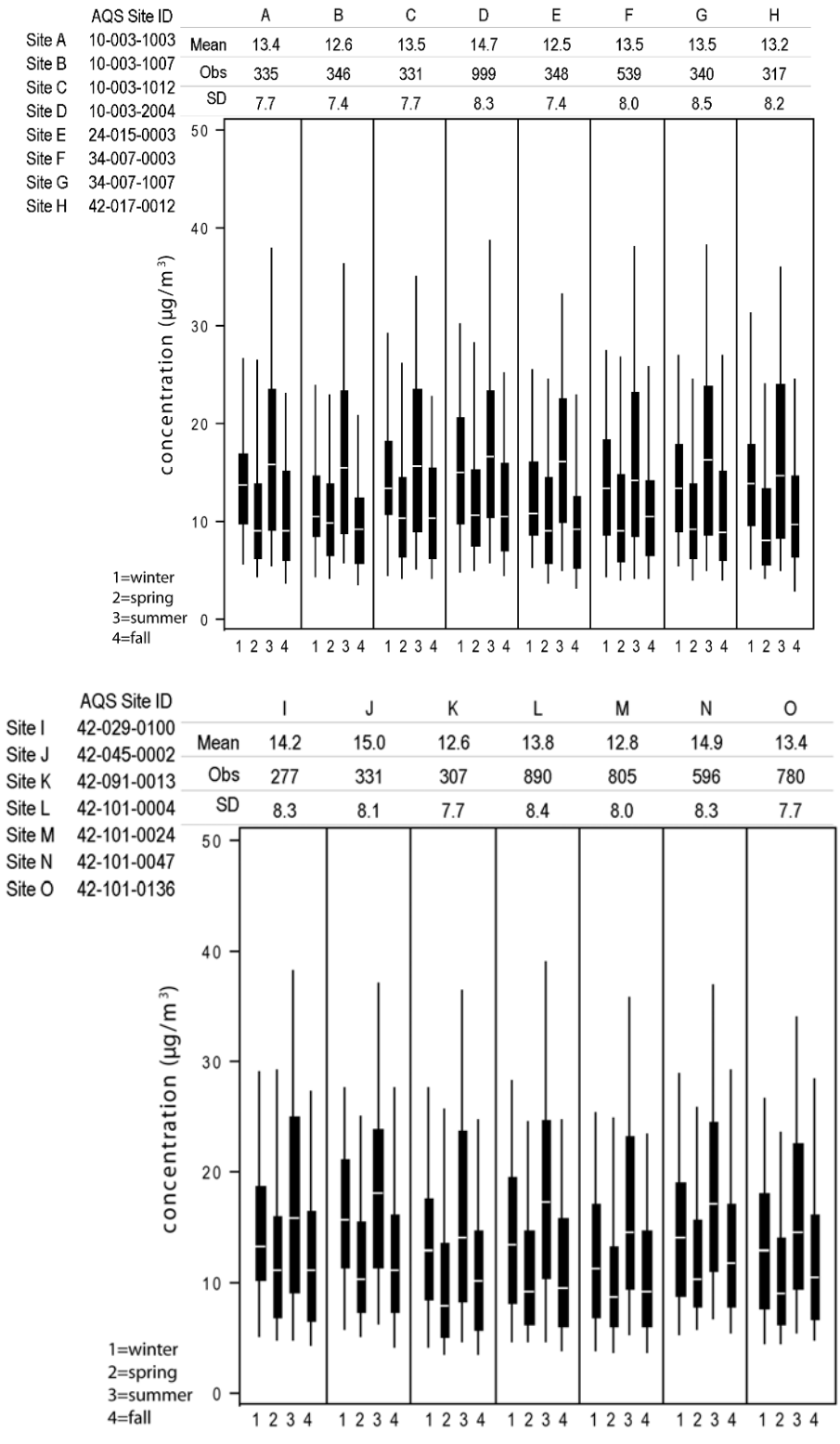


Figure A-57. Box plots illustrating the seasonal distribution of 24-h avg $PM_{2.5}$ concentrations for Philadelphia, PA.

Table A-8. Inter-sampler correlation statistics for each pair of $PM_{2.5}$ monitors reporting to AQS for Philadelphia, PA.

	A	B	C	D	E	F	G	H	I	J	K	L	M	N	O
A	1.00	0.94	0.96	0.98	0.92	0.96	0.93	0.89	0.95	0.92	0.86	0.96	0.96	0.95	0.97
	(0.0, 0.00)	(4.7, 0.12)	(3.1, 0.08)	(3.2, 0.08)	(4.8, 0.12)	(3.5, 0.10)	(4.2, 0.11)	(5.3, 0.13)	(4.2, 0.12)	(4.6, 0.14)	(4.7, 0.15)	(3.5, 0.08)	(3.7, 0.10)	(4.5, 0.12)	(3.2, 0.08)
	335	305	282	318	311	312	308	289	247	298	277	283	243	236	236
B		1.00	0.95	0.93	0.94	0.92	0.88	0.83	0.90	0.87	0.81	0.91	0.92	0.88	0.89
		(0.0, 0.00)	(4.3, 0.12)	(6.4, 0.15)	(3.4, 0.11)	(5.2, 0.14)	(6.0, 0.15)	(6.8, 0.17)	(6.7, 0.17)	(6.5, 0.18)	(5.9, 0.18)	(6.5, 0.14)	(5.0, 0.14)	(7.3, 0.17)	(5.9, 0.13)
		346	288	329	318	313	315	293	253	302	285	293	253	238	246
C			1.00	0.96	0.95	0.94	0.88	0.88	0.93	0.88	0.84	0.93	0.93	0.91	0.93
			(0.0, 0.00)	(4.3, 0.09)	(3.5, 0.11)	(4.7, 0.12)	(5.3, 0.14)	(6.0, 0.14)	(3.5, 0.12)	(6.6, 0.16)	(5.5, 0.17)	(5.0, 0.12)	(4.8, 0.13)	(6.0, 0.14)	(4.6, 0.11)
			331	312	289	292	286	270	242	278	261	281	245	225	237
D				1.00	0.91	0.94	0.92	0.88	0.94	0.90	0.85	0.95	0.93	0.93	0.95
				(0.0, 0.00)	(6.5, 0.15)	(4.9, 0.12)	(5.0, 0.14)	(6.3, 0.15)	(4.1, 0.12)	(5.3, 0.14)	(5.8, 0.18)	(4.3, 0.11)	(5.6, 0.14)	(4.2, 0.10)	(4.5, 0.11)
				999	325	490	317	297	257	312	287	801	732	540	704
E					1.00	0.91	0.87	0.83	0.90	0.86	0.86	0.88	0.90	0.87	0.89
					(0.0, 0.00)	(5.6, 0.14)	(6.1, 0.15)	(6.7, 0.16)	(6.6, 0.16)	(7.1, 0.19)	(5.7, 0.15)	(6.8, 0.15)	(5.3, 0.13)	(7.0, 0.18)	(5.7, 0.13)
					348	320	321	301	255	310	287	296	255	242	254
F						1.00	0.95	0.90	0.92	0.89	0.87	0.96	0.96	0.95	0.96
						(0.0, 0.00)	(3.4, 0.09)	(5.3, 0.13)	(5.4, 0.14)	(5.9, 0.16)	(4.4, 0.15)	(3.7, 0.10)	(3.6, 0.10)	(4.5, 0.13)	(3.4, 0.09)
						539	317	296	261	309	284	466	437	414	396
G							1.00	0.90	0.90	0.87	0.85	0.93	0.97	0.92	0.96
							(0.0, 0.00)	(4.8, 0.14)	(5.9, 0.16)	(6.2, 0.17)	(4.7, 0.16)	(3.7, 0.09)	(3.1, 0.09)	(5.7, 0.13)	(3.5, 0.08)
							340	295	258	305	289	288	251	235	240
H								1.00	0.84	0.83	0.89	0.90	0.94	0.87	0.89
								(0.0, 0.00)	(5.7, 0.16)	(8.0, 0.19)	(4.4, 0.13)	(5.0, 0.13)	(4.0, 0.12)	(5.9, 0.17)	(4.8, 0.13)
								317	240	288	275	273	234	215	227
I									1.00	0.87	0.81	0.91	0.92	0.90	0.92
									(0.0, 0.00)	(5.5, 0.17)	(5.7, 0.17)	(4.9, 0.14)	(5.4, 0.15)	(5.2, 0.16)	(5.1, 0.14)
			R						277	248	228	235	215	196	195
J			(P90, COD)							1.00	0.79	0.89	0.89	0.89	0.91
			N							(0.0, 0.00)	(7.4, 0.21)	(5.8, 0.15)	(6.4, 0.17)	(5.7, 0.13)	(5.0, 0.14)
										331	278	282	246	237	231
K											1.00	0.87	0.95	0.84	0.86
											(0.0, 0.00)	(4.7, 0.15)	(3.7, 0.13)	(6.8, 0.20)	(4.3, 0.13)
											307	268	230	211	212
L												1.00	0.98	0.95	0.97
												(0.0, 0.00)	(3.1, 0.09)	(3.7, 0.11)	(3.4, 0.07)
												890	672	512	630
M													1.00	0.95	0.96
													(0.0, 0.00)	(4.7, 0.14)	(3.2, 0.09)
													805	495	563
N														1.00	0.97
														(0.0, 0.00)	(3.5, 0.10)
														596	447
O															1.00
															(0.0, 0.00)
															780

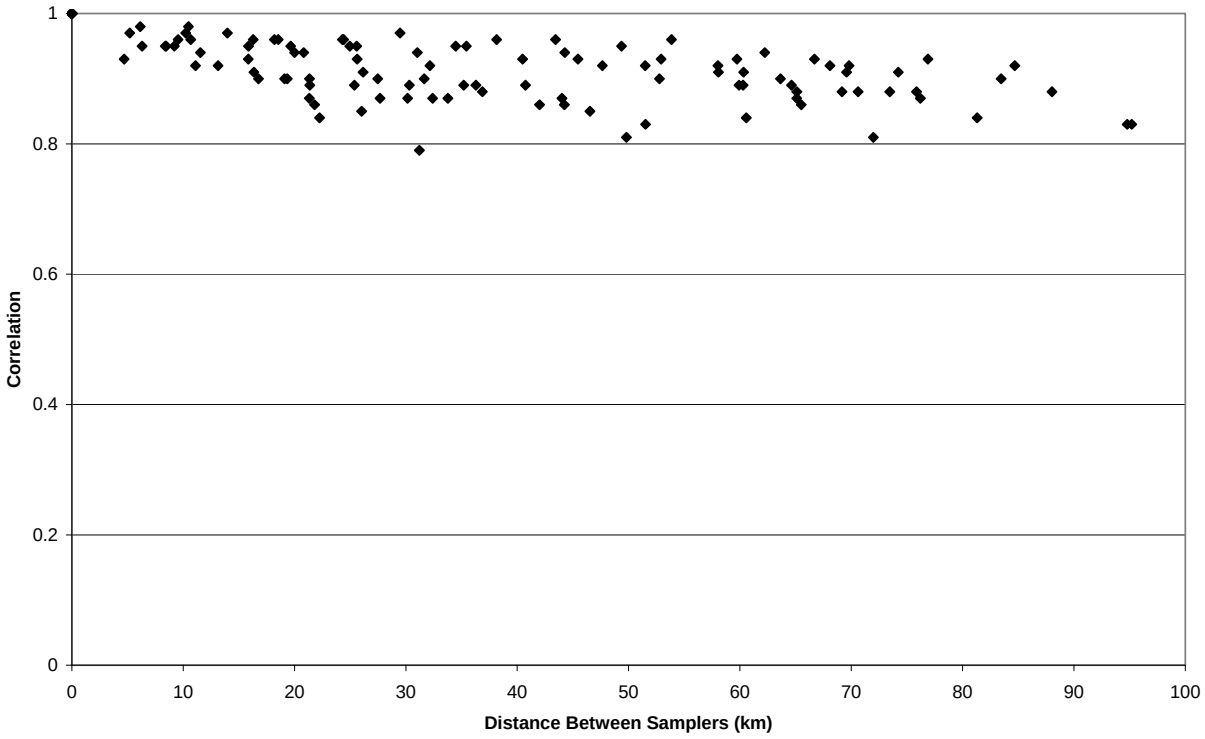


Figure A-58. $PM_{2.5}$ inter-sampler correlations as a function of distance between monitors for Philadelphia, PA.

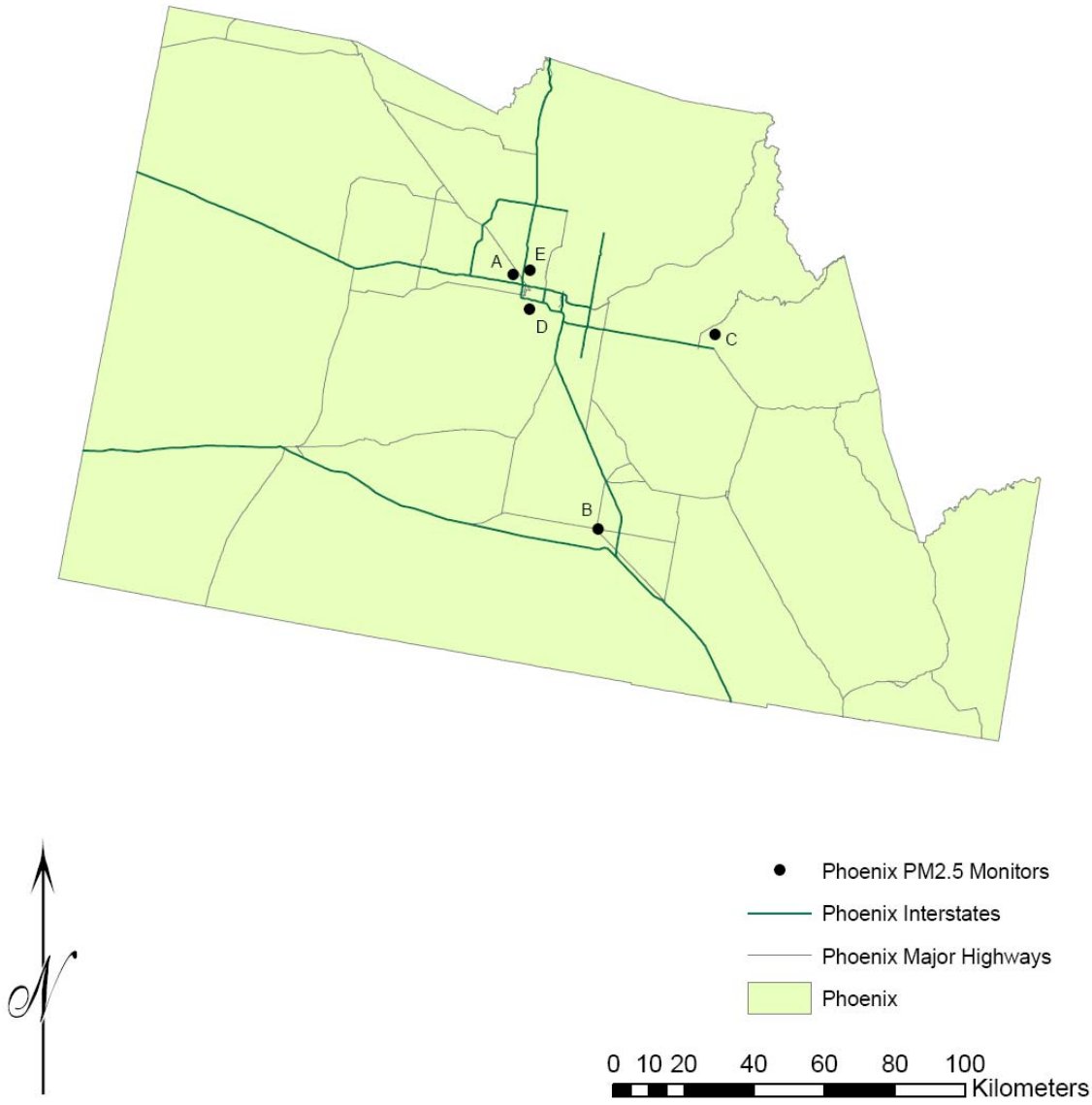


Figure A-59. $PM_{2.5}$ monitor distribution and major highways, Phoenix, AZ.

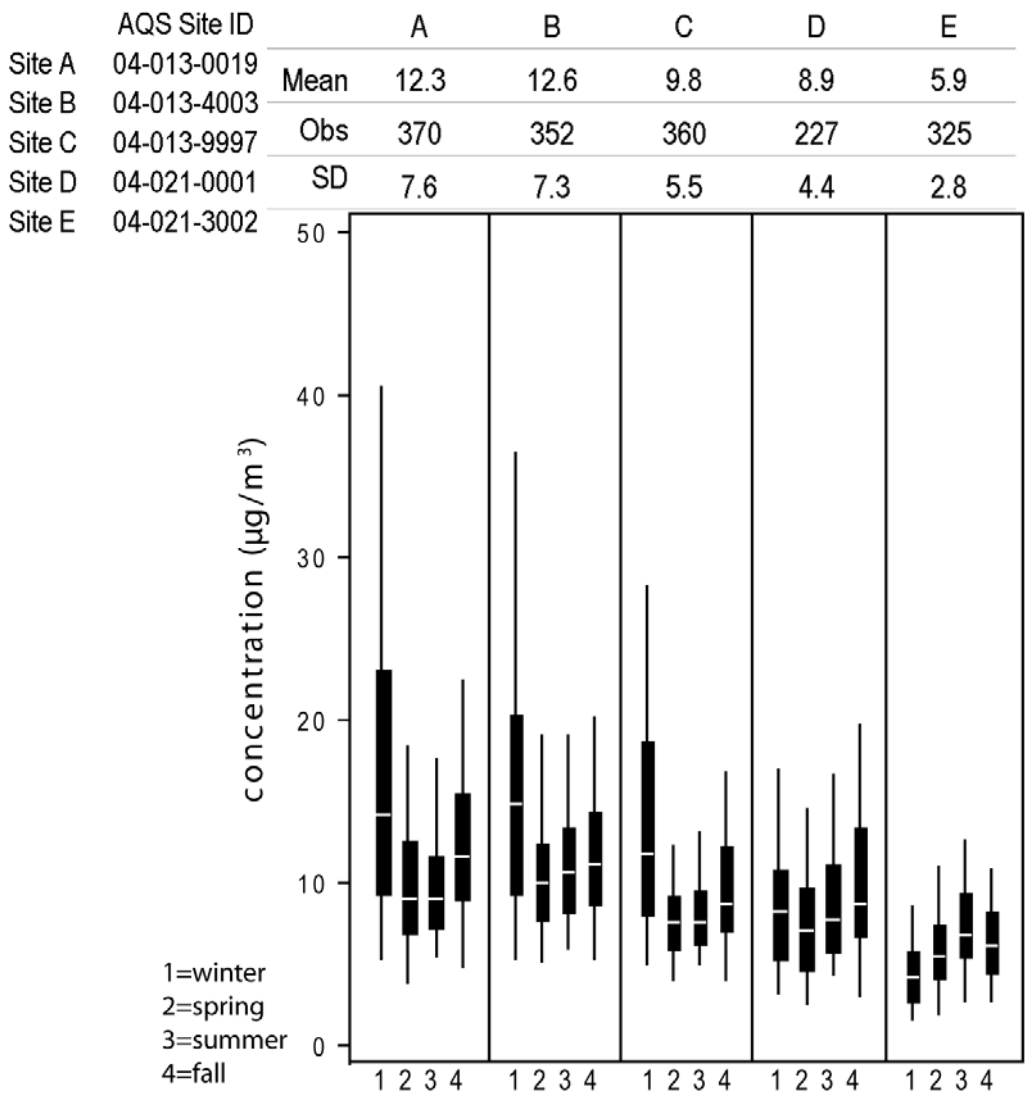


Figure A-60. Box plots illustrating the seasonal distribution of 24-h avg $PM_{2.5}$ concentrations for Phoenix, AZ.

Table A-9. Inter-sampler correlation statistics for each pair of $PM_{2.5}$ monitors reporting to AQS for Phoenix, AZ.

	A	B	C	D	E
A	1.00	0.87	0.92	0.50	0.12
	(0.0, 0.00)	(6.4, 0.15)	(6.5, 0.16)	(10.4, 0.25)	(14.4, 0.40)
	370	345	355	222	321
B		1.00	0.89	0.54	0.23
		(0.0, 0.00)	(6.8, 0.17)	(9.6, 0.25)	(13.2, 0.40)
		352	338	212	307
C			1.00	0.54	0.18
			(0.0, 0.00)	(7.2, 0.20)	(9.3, 0.33)

	A	B	C	D	E
			360	216	315
D				1.00	0.51
	R			(0.0, 0.00)	(7.8, 0.27)
	(P90, COD)			227	200
	N				
E					1.00
					(0.0, 0.00)
					325

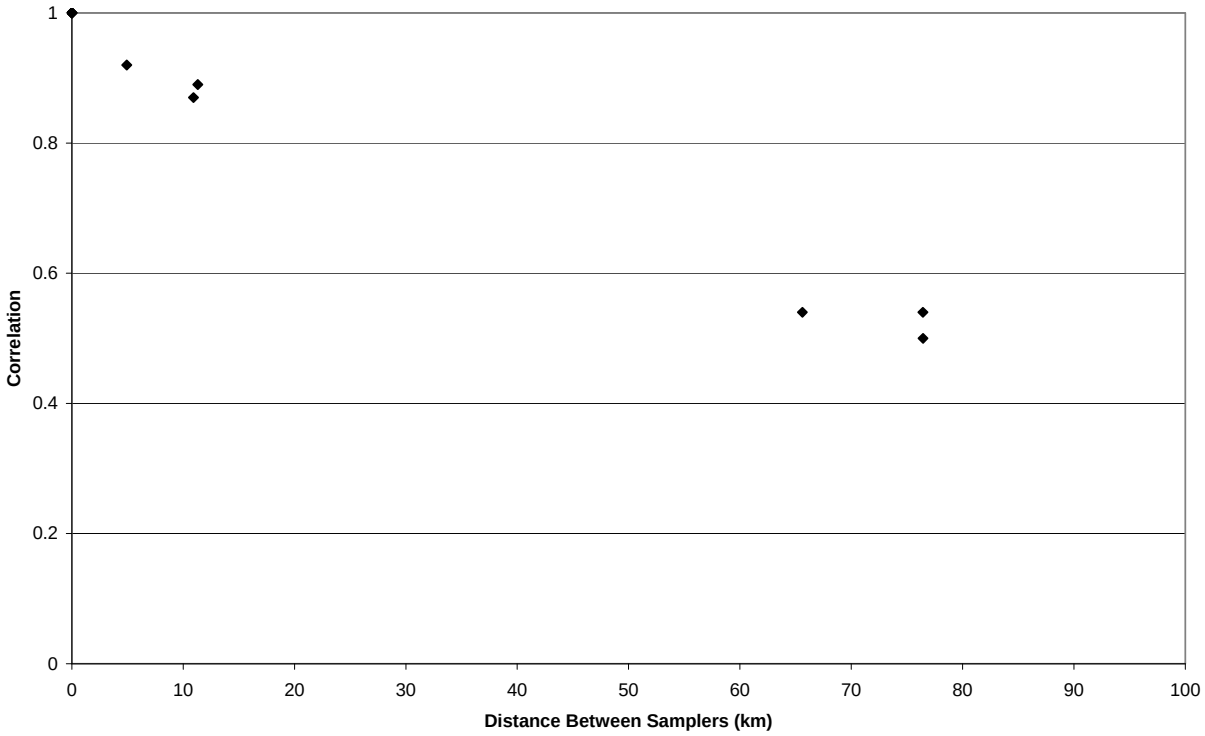


Figure A-61. $PM_{2.5}$ inter-sampler correlations as a function of distance between monitors for Phoenix, AZ.

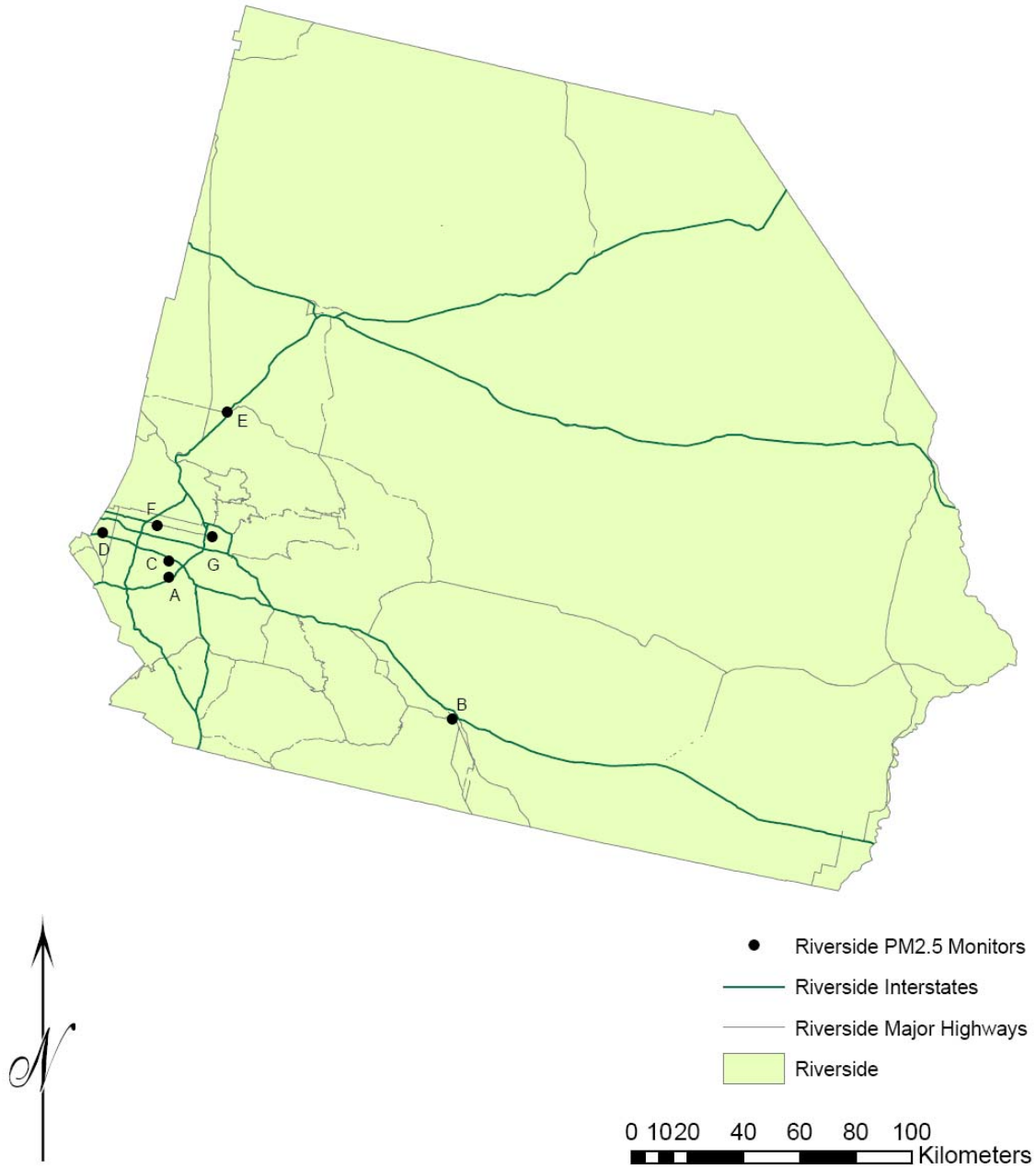


Figure A-62. $PM_{2.5}$ monitor distribution and major highways, Riverside, CA.

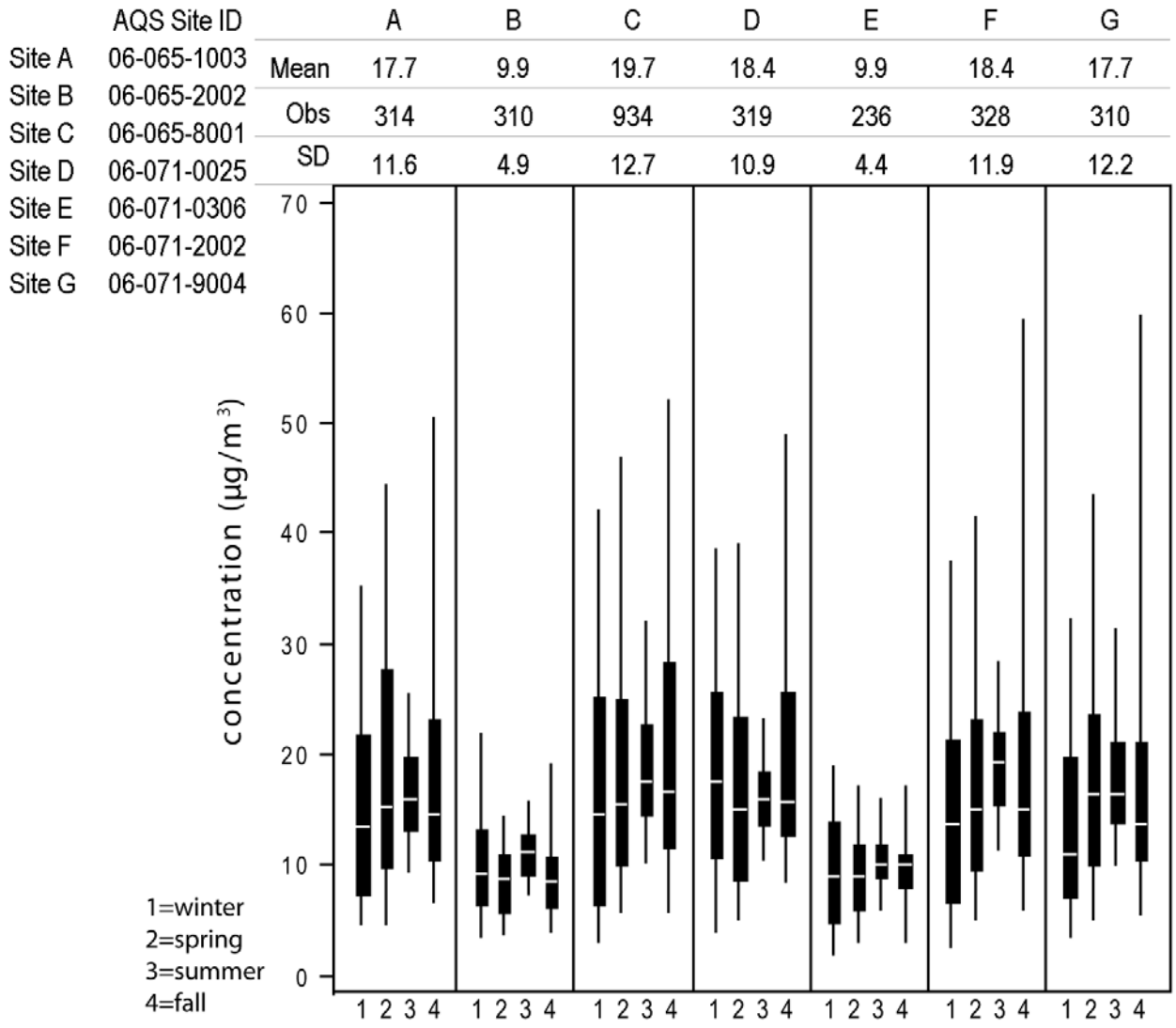


Figure A-63. Box plots illustrating the seasonal distribution of 24-h avg $PM_{2.5}$ concentrations for Riverside, CA.

Table A-10. Inter-sampler correlation statistics for each pair of $PM_{2.5}$ monitors reporting to AQS for Riverside, CA.

	A	B	C	D	E	F	G
A	1.00	0.45	0.96	0.92	0.36	0.94	0.90
	(0.0, 0.00)	(20.6, 0.32)	(5.0, 0.10)	(7.2, 0.13)	(22.1, 0.35)	(6.0, 0.12)	(5.7, 0.13)
	314	269	297	282	191	281	273
B		1.00	0.49	0.49	0.42	0.49	0.50
		(0.0, 0.00)	(22.7, 0.35)	(20.9, 0.34)	(8.2, 0.25)	(19.7, 0.33)	(18.8, 0.31)
		310	289	270	203	285	266
C			1.00	0.91	0.37	0.92	0.91
			(0.0, 0.00)	(8.2, 0.14)	(26.6, 0.37)	(6.9, 0.12)	(7.6, 0.12)
			934	300	227	302	287
D				1.00	0.36	0.93	0.82
				(0.0, 0.00)	(20.1, 0.35)	(6.7, 0.14)	(9.6, 0.17)
				319	195	289	274
E		R			1.00	0.40	0.41
		(P90, COD)			(0.0, 0.00)	(21.1, 0.36)	(21.6, 0.34)
		N			236	201	190
F						1.00	0.90
						(0.0, 0.00)	(6.7, 0.12)
						328	276
G							1.00
							(0.0, 0.00)
							310

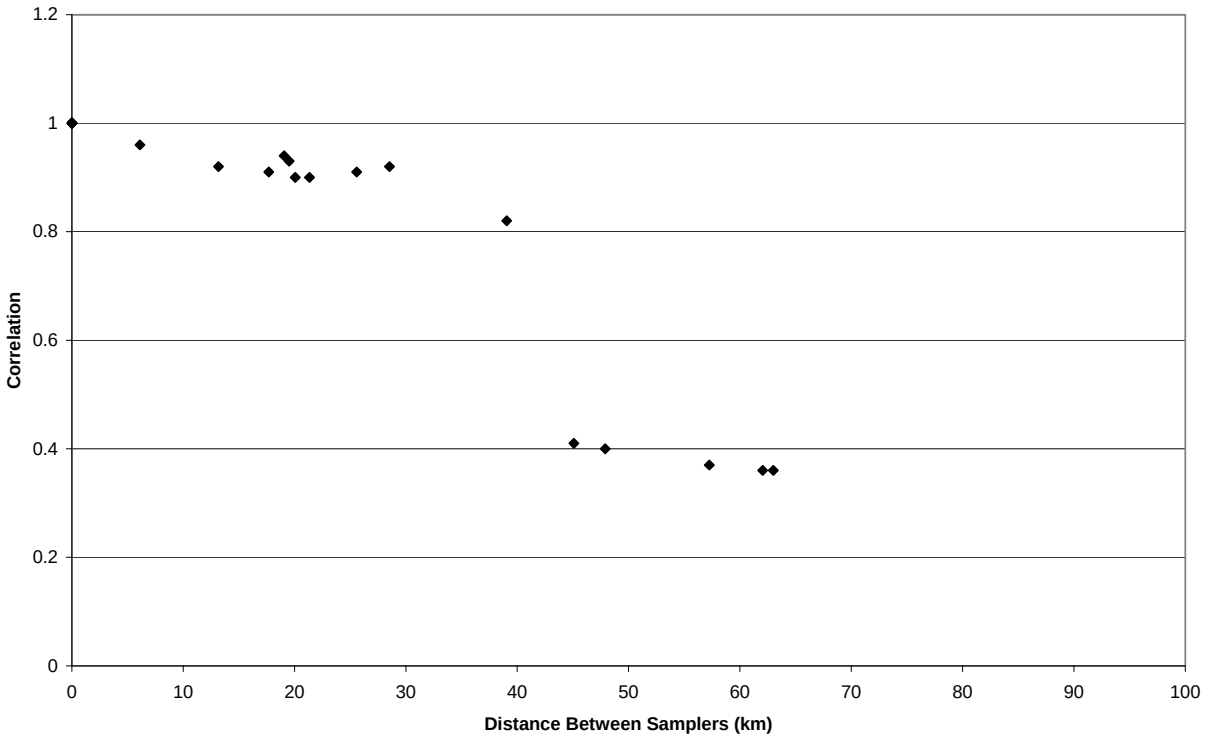


Figure A-64. $PM_{2.5}$ inter-sampler correlations as a function of distance between monitors for Riverside CA.

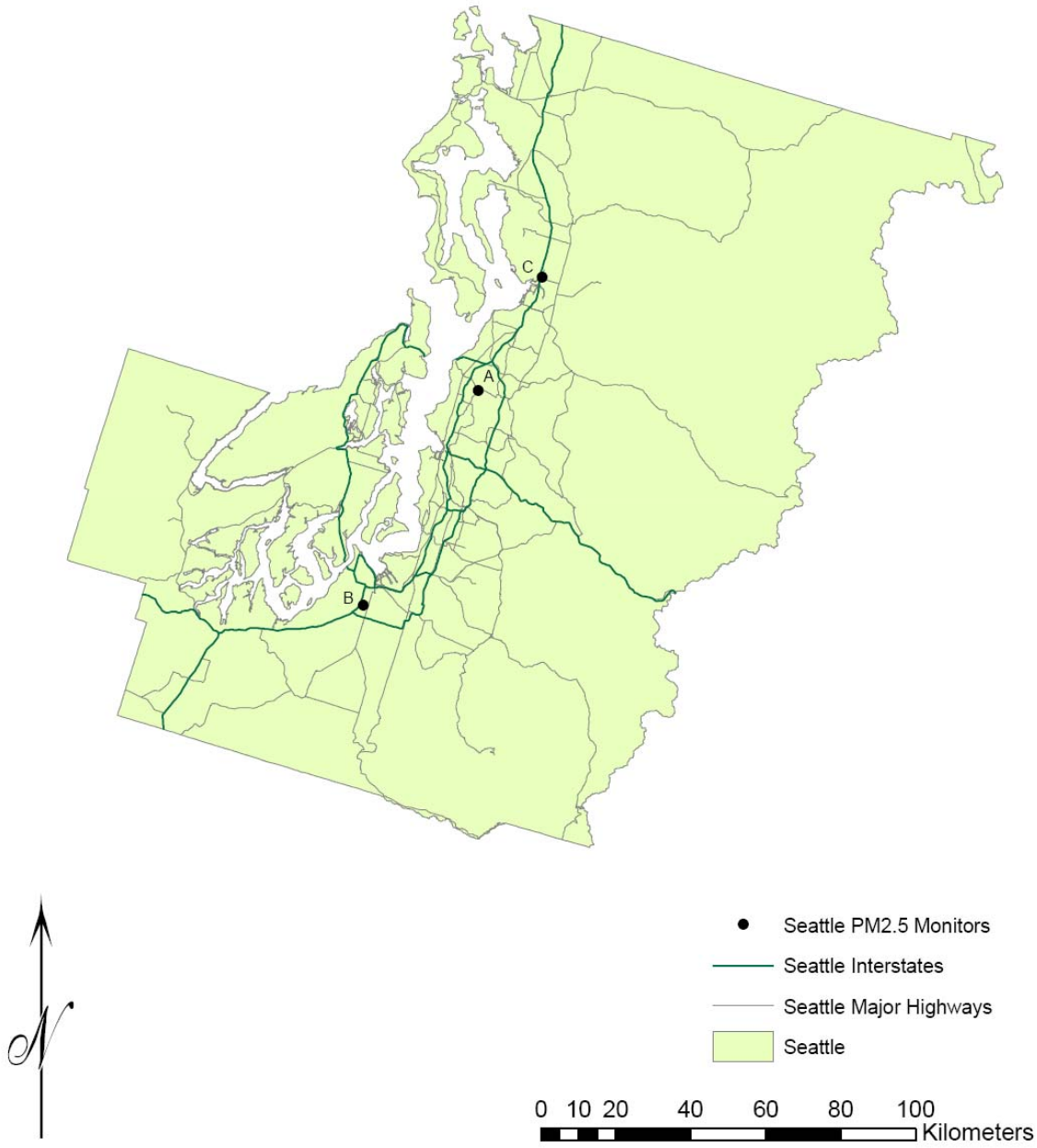


Figure A-65. $PM_{2.5}$ monitor figudistribution and major highways, Seattle, WA.

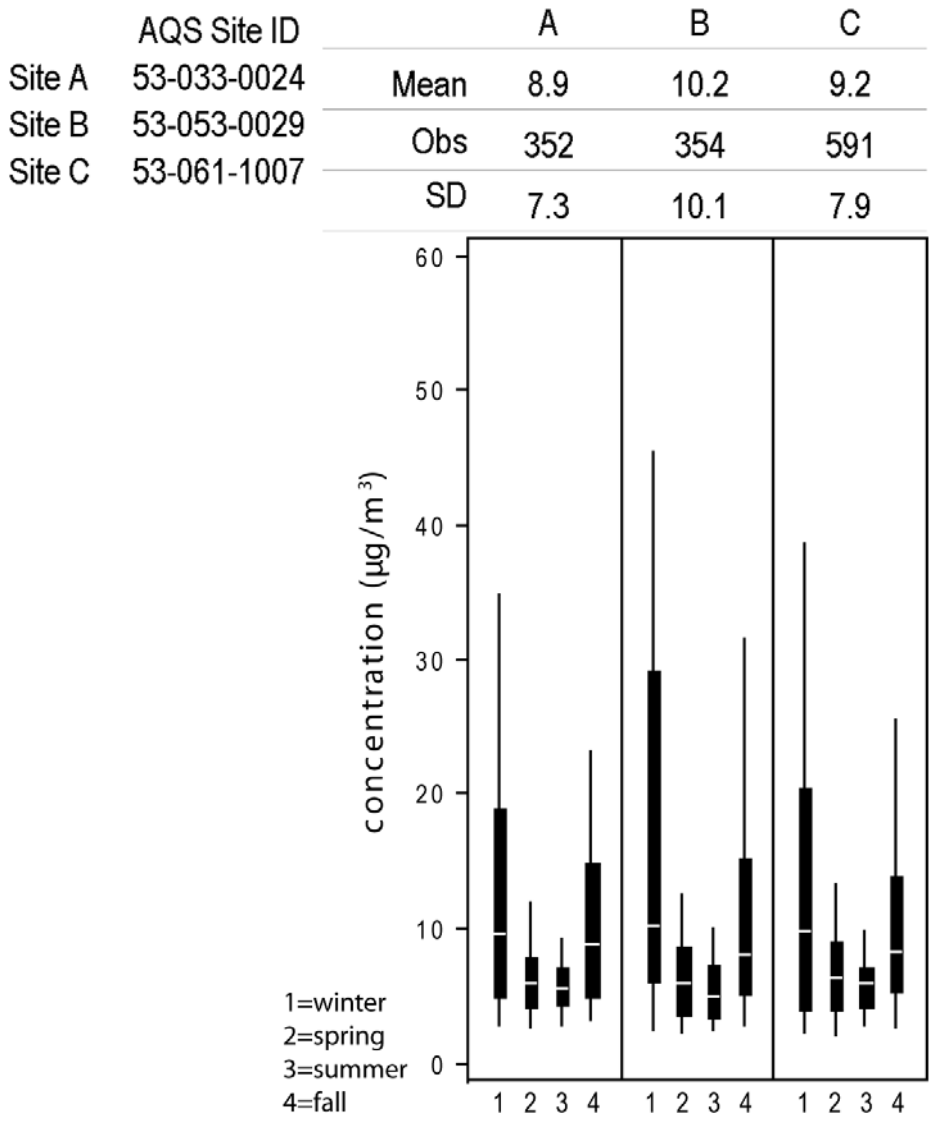


Figure A-66. Box plots illustrating the seasonal distribution of 24-h avg $PM_{2.5}$ concentrations for Seattle, WA.

Table A-11. Inter-sampler correlation statistics for each pair of $PM_{2.5}$ monitors reporting to AQS for Seattle, WA.

			A	B	C
A			1.00	0.89	0.86
			(0.0, 0.00)	(6.3, 0.16)	(4.5, 0.14)
			352	337	331
B	R			1.00	0.80
	(P90, COD)			(0.0, 0.00)	(7.8, 0.20)
	N			354	335
C					1.00
					(0.0, 0.00)
					591

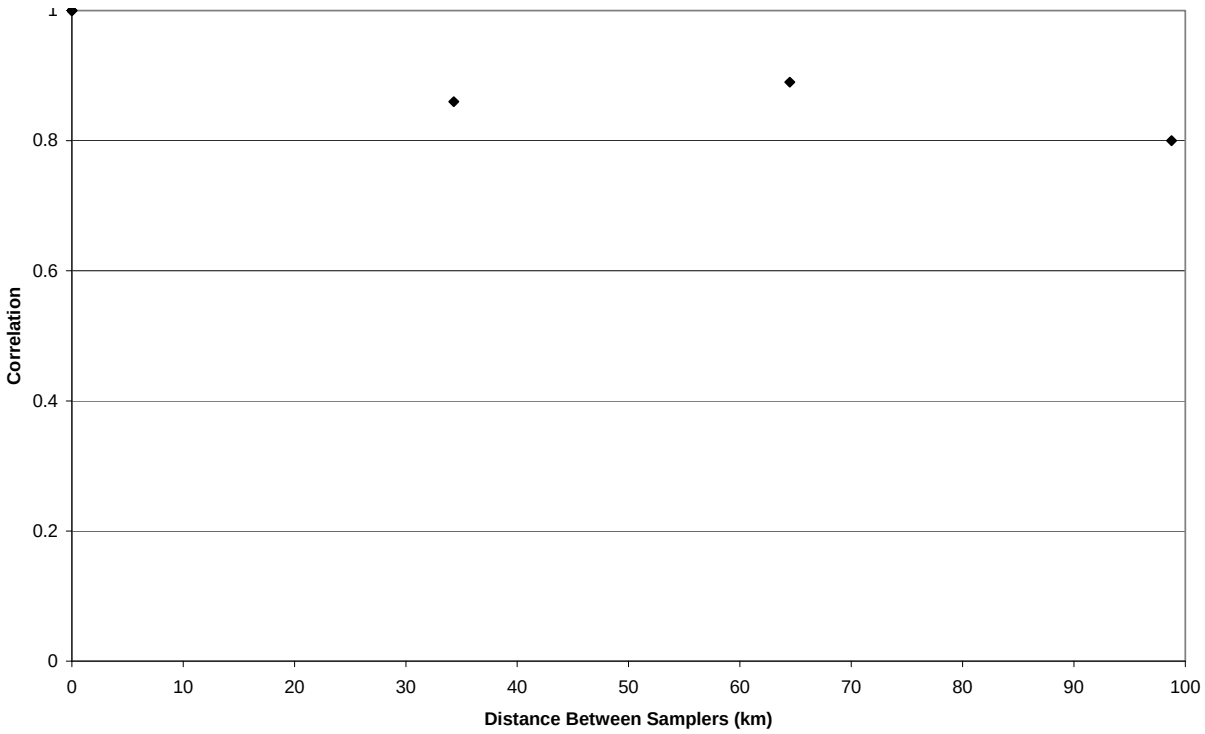


Figure A-67. $PM_{2.5}$ inter-sampler correlations as a function of distance between monitors for Seattle, WA.

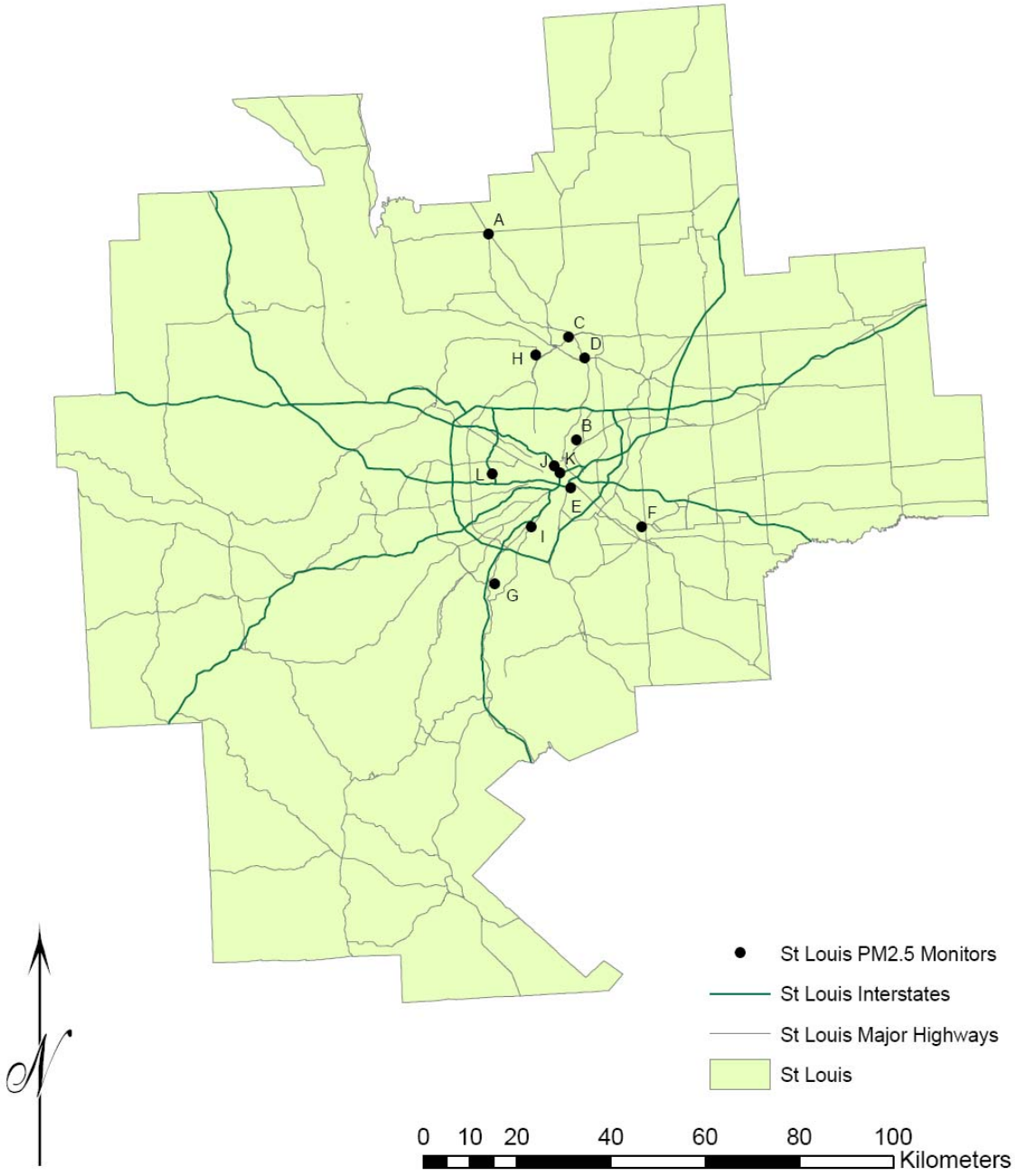


Figure A-68. $PM_{2.5}$ monitor distribution and major highways, St. Louis, MO.

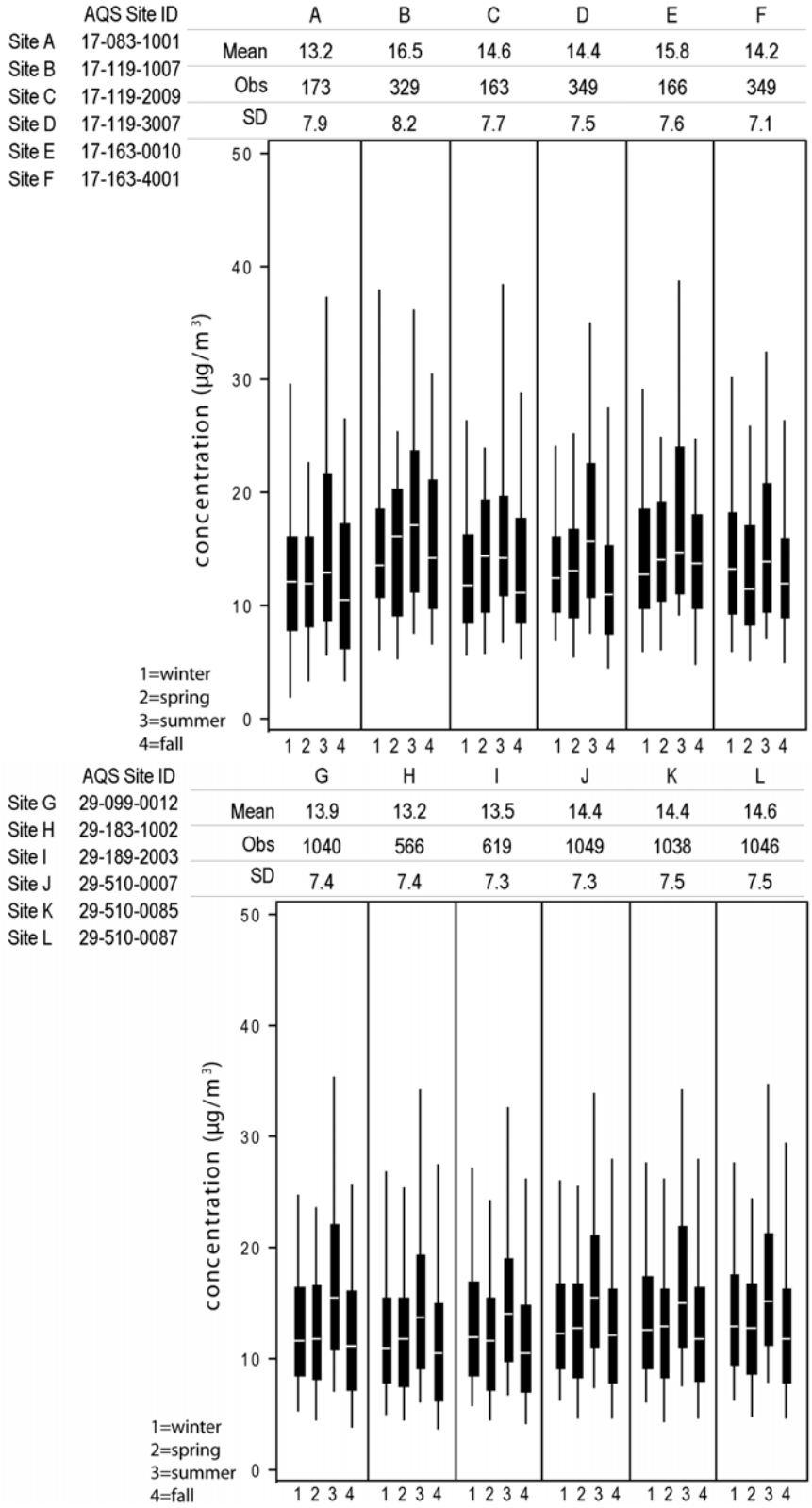


Figure A-69. Box plots illustrating the seasonal distribution of 24-h avg $PM_{2.5}$ concentrations for St. Louis, MO.

Table A-12. Inter-sampler correlation statistics for each pair of $PM_{2.5}$ monitors reporting to AQS for St. Louis, MO.

Site	A	B	C	D	E	F	G	H	I	J	K	L
A	1.00	0.85	0.93	0.89	0.88	0.86	0.85	0.93	0.86	0.84	0.84	0.88
	(0.0, 0.00)	(10.5, 0.23)	(4.7, 0.17)	(5.0, 0.17)	(7.3, 0.20)	(6.2, 0.18)	(4.8, 0.17)	(4.1, 0.13)	(4.4, 0.16)	(6.0, 0.18)	(5.7, 0.19)	(5.3, 0.17)
	173	156	129	162	146	156	167	158	162	168	169	166
B		1.00	0.89	0.86	0.85	0.82	0.88	0.89	0.88	0.86	0.87	0.89
		(0.0, 0.00)	(8.6, 0.16)	(7.4, 0.16)	(7.7, 0.16)	(8.6, 0.17)	(7.8, 0.17)	(8.2, 0.18)	(7.9, 0.17)	(7.7, 0.17)	(7.5, 0.16)	(6.8, 0.14)
		329	135	301	156	306	312	305	318	316	316	315
C			1.00	0.94	0.91	0.88	0.90	0.96	0.94	0.90	0.89	0.94
			(0.0, 0.00)	(4.0, 0.11)	(6.4, 0.13)	(5.7, 0.13)	(5.5, 0.13)	(3.9, 0.11)	(5.3, 0.11)	(5.7, 0.13)	(5.6, 0.14)	(4.4, 0.11)
			163	139	124	133	158	141	144	158	160	156
D				1.00	0.89	0.84	0.89	0.94	0.92	0.89	0.88	0.92
				(0.0, 0.00)	(5.7, 0.13)	(6.0, 0.15)	(4.9, 0.12)	(4.3, 0.12)	(4.5, 0.11)	(4.7, 0.13)	(4.6, 0.12)	(3.9, 0.11)
				349	156	314	331	315	326	335	332	336
E					1.00	0.90	0.91	0.90	0.91	0.93	0.91	0.95
					(0.0, 0.00)	(5.5, 0.12)	(6.2, 0.13)	(5.8, 0.16)	(5.3, 0.14)	(5.1, 0.13)	(4.9, 0.13)	(3.7, 0.10)
					166	152	159	153	157	160	163	160
F						1.00	0.89	0.86	0.88	0.88	0.85	0.88
						(0.0, 0.00)	(5.4, 0.12)	(6.1, 0.16)	(5.4, 0.13)	(5.3, 0.14)	(5.6, 0.14)	(5.4, 0.13)
						349	333	317	332	337	332	334
G		R					1.00	0.93	0.94	0.96	0.93	0.94
		(P90, COD)					(0.0, 0.00)	(4.3, 0.10)	(3.3, 0.08)	(2.9, 0.08)	(3.9, 0.10)	(3.8, 0.10)
		N					1040	533	586	994	987	992
H								1.00	0.96	0.95	0.95	0.96
								(0.0, 0.00)	(3.0, 0.08)	(4.1, 0.12)	(3.8, 0.12)	(4.0, 0.11)
								566	550	552	546	544
I									1.00	0.96	0.95	0.96
									(0.0, 0.00)	(3.1, 0.09)	(3.1, 0.10)	(3.4, 0.09)
									619	605	599	598
J										1.00	0.96	0.97
										(0.0, 0.00)	(2.5, 0.09)	(2.5, 0.08)
										1049	1001	1007
K											1.00	0.97
											(0.0, 0.00)	(1.9, 0.07)
											1038	991
L												1.00
												(0.0, 0.00)
												1046

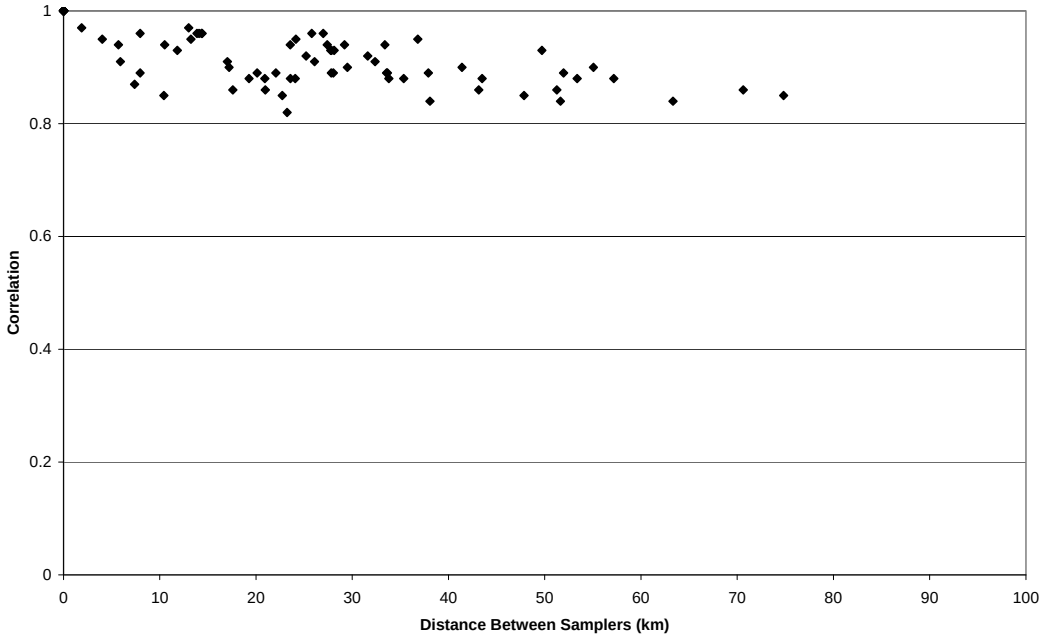


Figure A-70 $PM_{2.5}$ inter-sampler correlations as a function of distance between monitors for St. Louis, MO.

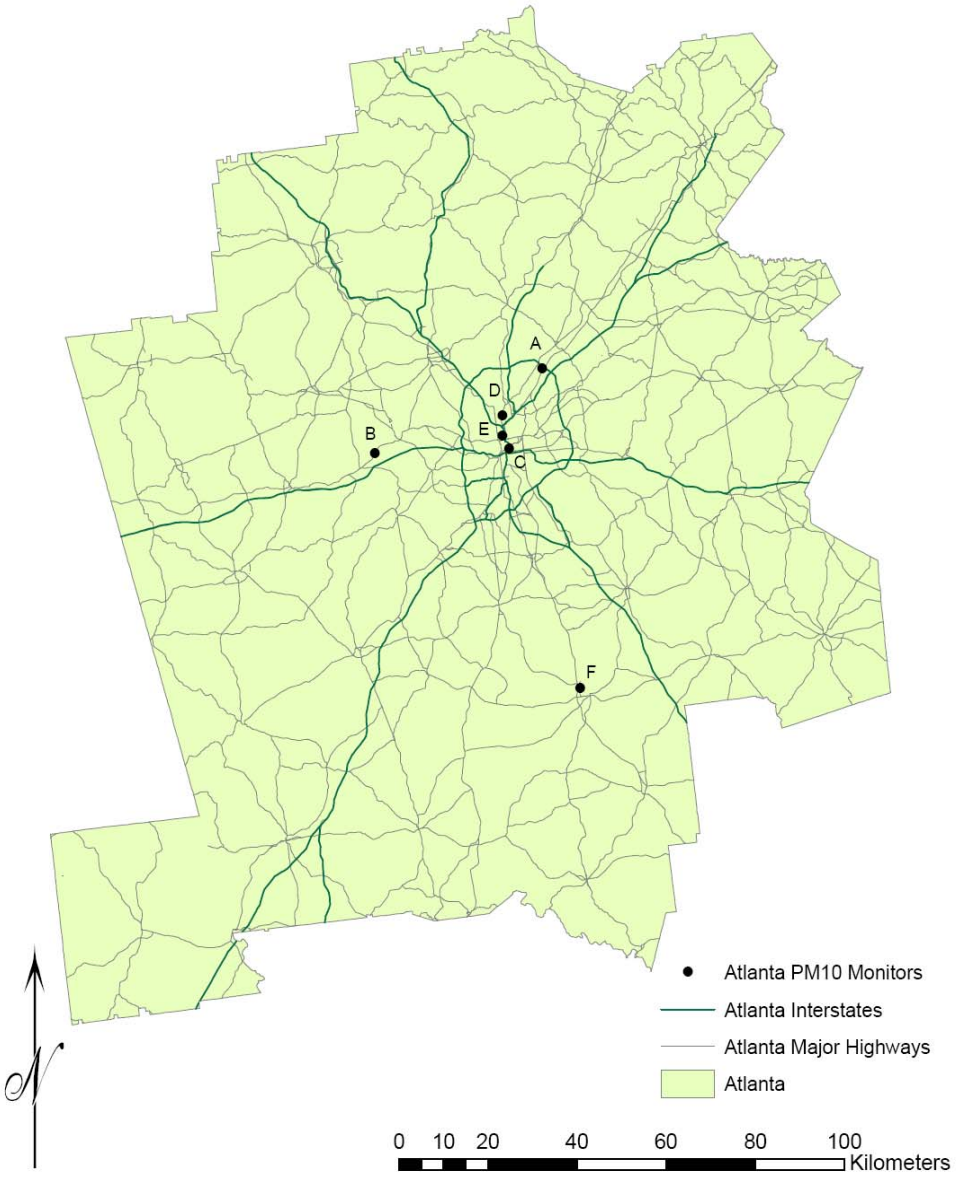


Figure A-71. PM_{10} monitor distribution and major highways, Atlanta, GA.

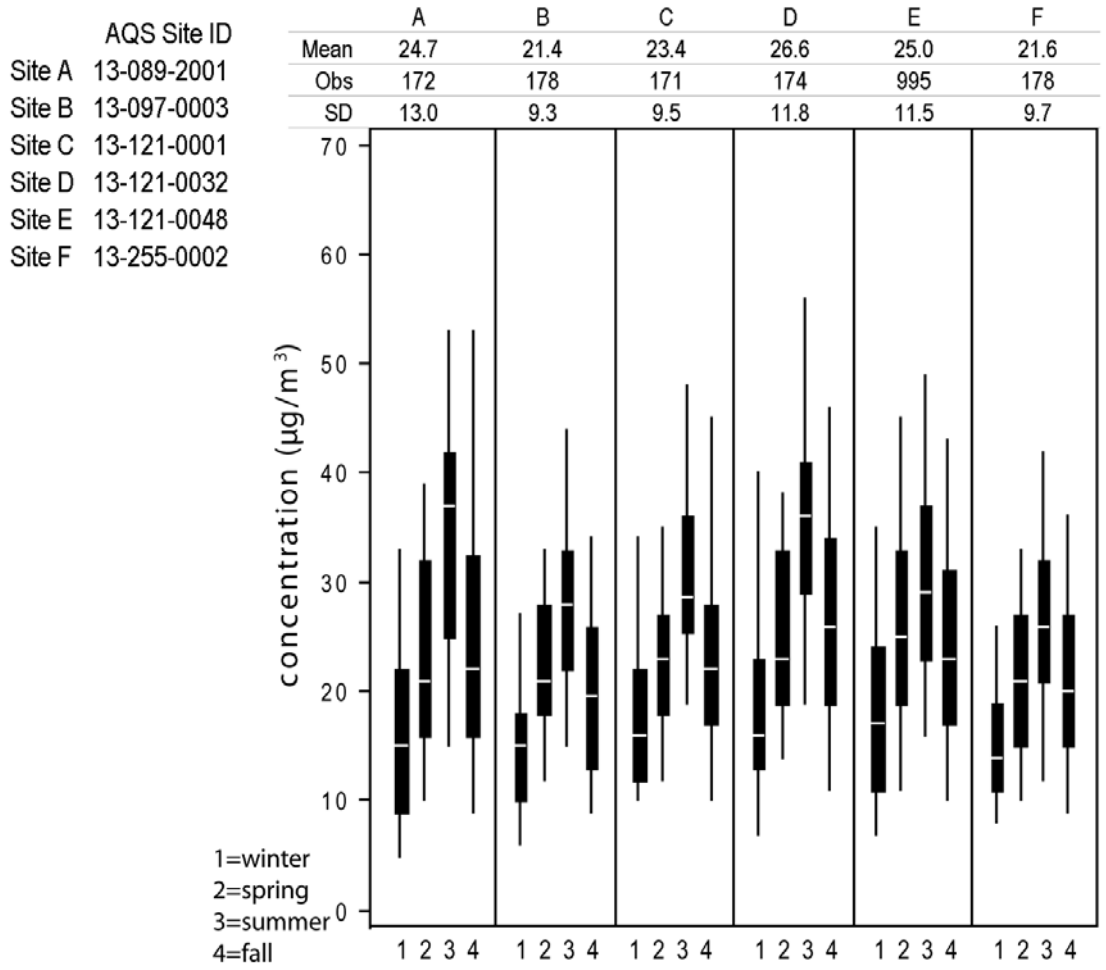


Figure A-72. Box plots illustrating the seasonal distribution of 24-h avg PM_{10} concentrations for Atlanta, GA.

Table A-13. Inter-sampler correlation statistics for each pair of PM_{10} monitors reporting to AQS for Atlanta, GA.

Site	A	B	C	D	E	F
A	1.00	0.69	0.74	0.78	0.70	0.59
	(0.0, 0.00)	(18.0, 0.22)	(15.0, 0.20)	(13.0, 0.20)	(16.0, 0.22)	(20.0, 0.24)
	172	169	162	165	158	164
B		1.00	0.88	0.79	0.71	0.82
		(0.0, 0.00)	(6.0, 0.12)	(14.5, 0.17)	(16.0, 0.18)	(10.0, 0.14)
		178	167	170	162	169
C			1.00	0.88	0.84	0.82
			(0.0, 0.00)	(9.0, 0.13)	(10.0, 0.13)	(9.0, 0.15)
			171	162	155	161
D				1.00	0.75	0.74
	R			(0.0, 0.00)	(12.0, 0.15)	(15.0, 0.20)
	(P90, COD)			174	158	166
E	N				1.00	0.67
					(0.0, 0.00)	(17.0, 0.19)
					995	163
F						1.00
						(0.0, 0.00)
						178

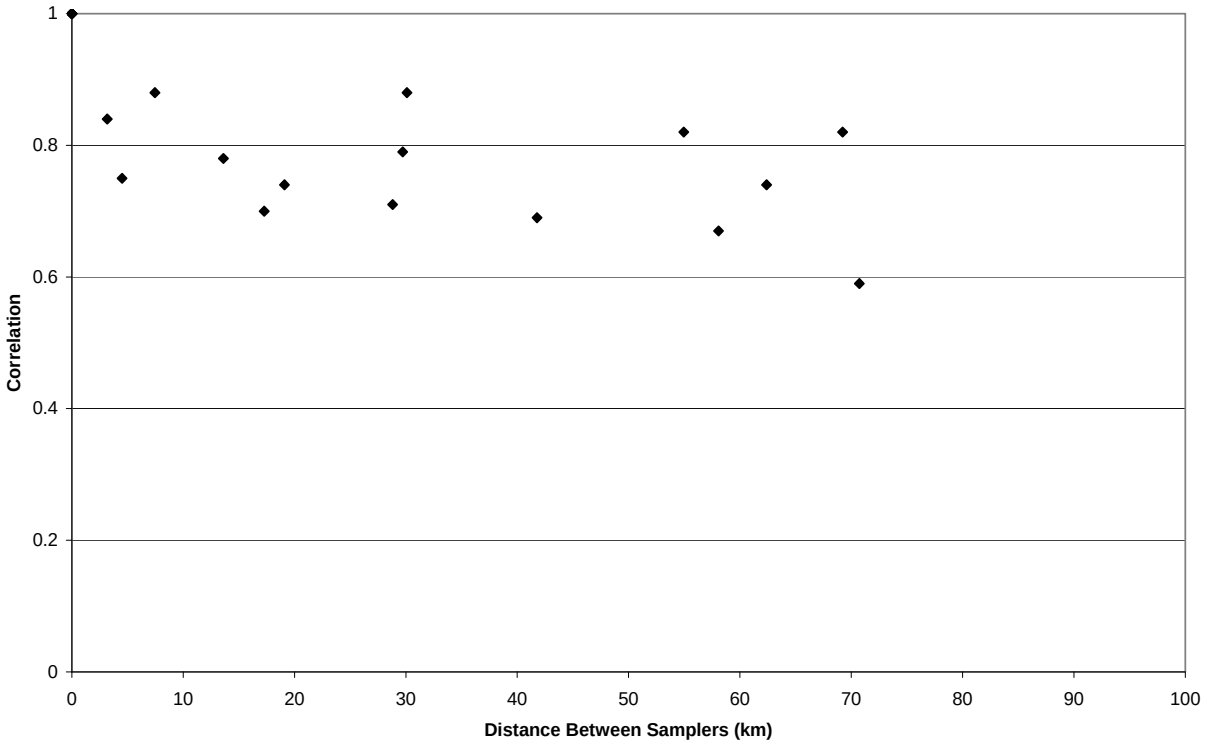


Figure A-73. PM_{10} inter-sampler correlations as a function of distance between monitors for Atlanta, GA.

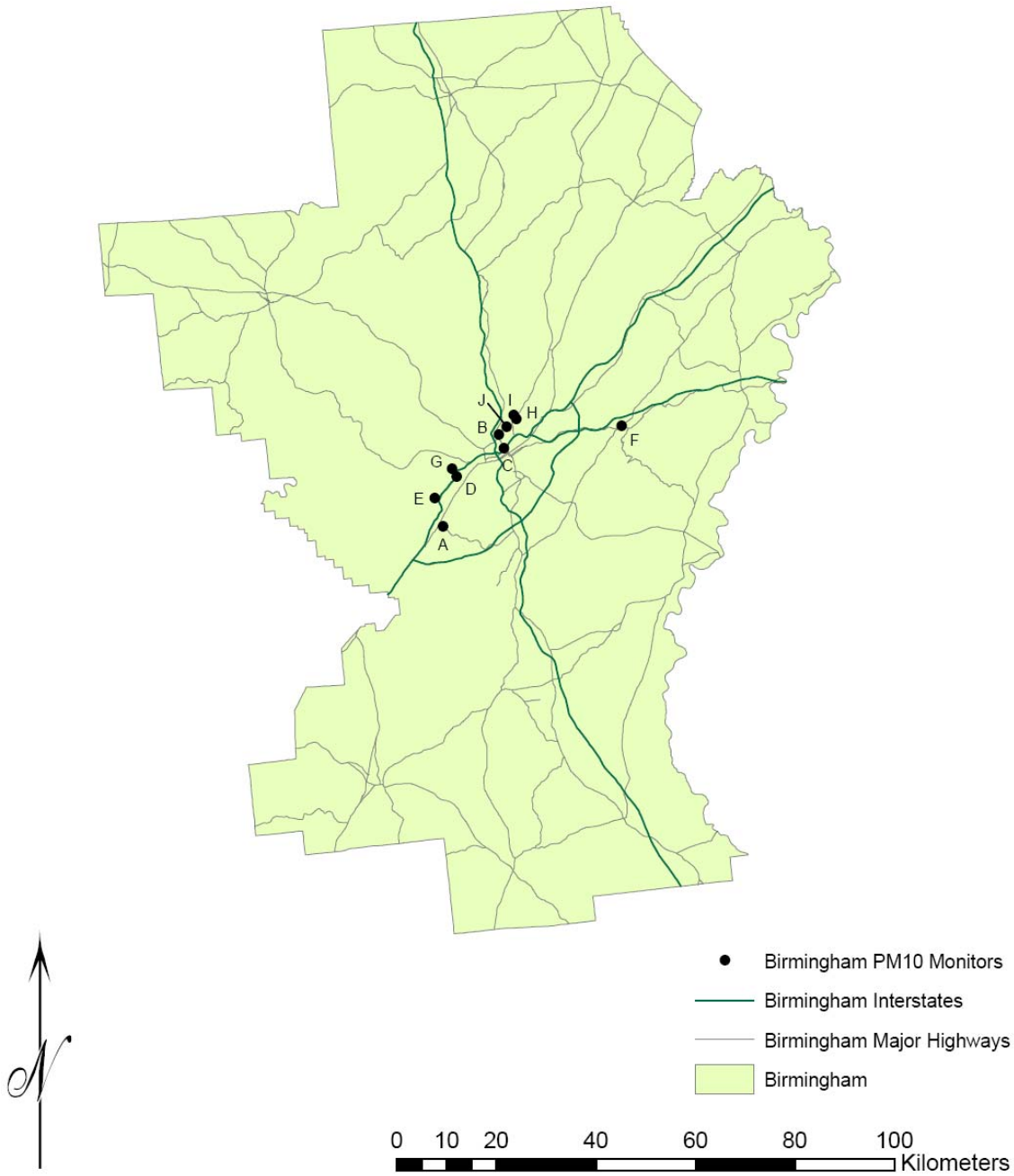


Figure A-74. PM_{10} monitor distribution and major highways, Birmingham, AL.

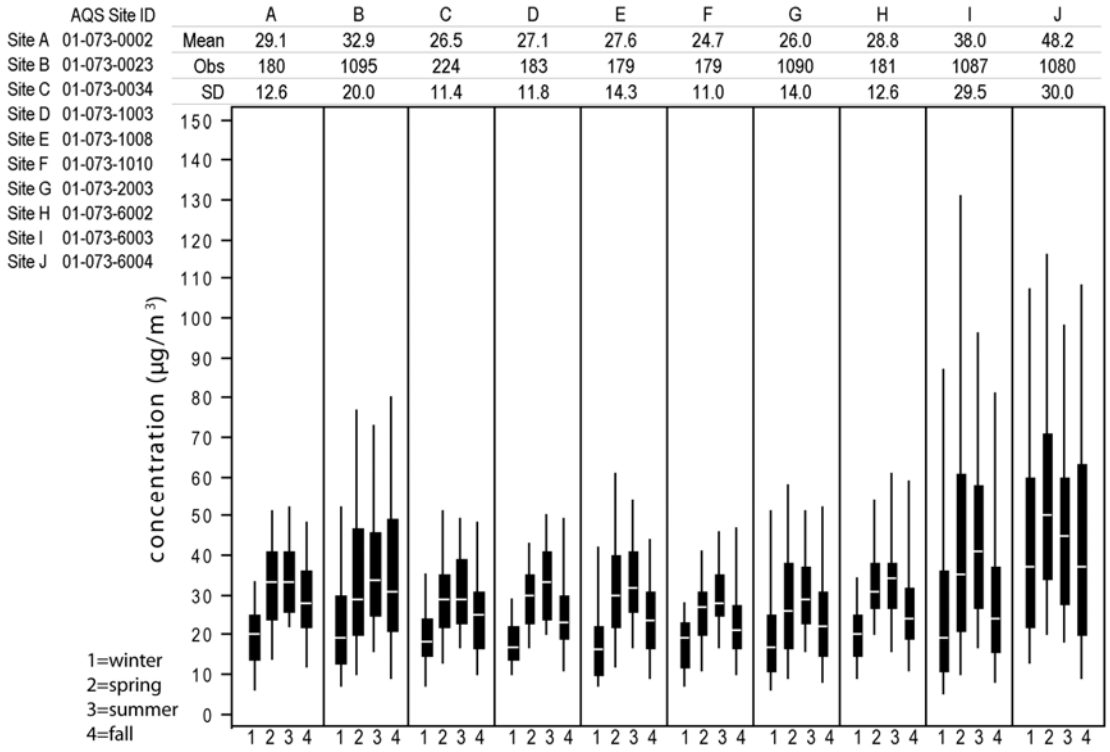


Figure A-75. Box plots illustrating the seasonal distribution of 24-h avg PM_{10} concentrations for Birmingham, AL.

Table A-14. Inter-sampler correlation statistics for each pair of PM_{10} monitors reporting to AQS for Birmingham, AL.

	A	B	C	D	E	F	G	H	I	J
A	1.00	0.80	0.88	0.86	0.78	0.84	0.77	0.78	0.41	0.29
	(0.0, 0.00)	(23.0, 0.16)	(11.0, 0.11)	(12.0, 0.13)	(12.0, 0.14)	(13.0, 0.13)	(15.0, 0.18)	(14.0, 0.15)	(41.0, 0.30)	(68.0, 0.34)
	180	180	174	180	176	171	180	178	179	177
B		1.00	0.82	0.74	0.61	0.73	0.75	0.71	0.26	0.23
		(0.0, 0.00)	(23.0, 0.17)	(25.0, 0.21)	(26.0, 0.20)	(26.0, 0.19)	(25.0, 0.20)	(25.0, 0.22)	(51.0, 0.33)	(57.0, 0.36)
		1095	224	183	179	179	1090	181	1087	1080
C			1.00	0.84	0.66	0.78	0.74	0.80	0.33	0.41
			(0.0, 0.00)	(10.0, 0.12)	(15.0, 0.16)	(12.0, 0.14)	(14.0, 0.17)	(13.0, 0.15)	(43.0, 0.32)	(62.0, 0.34)
			224	175	171	168	224	173	222	221
D				1.00	0.67	0.79	0.76	0.84	0.45	0.41
				(0.0, 0.00)	(15.0, 0.17)	(12.0, 0.15)	(14.0, 0.17)	(11.0, 0.12)	(42.0, 0.30)	(65.5, 0.34)
				183	178	173	183	180	182	180
E					1.00	0.67	0.64	0.56	0.33	0.12
					(0.0, 0.00)	(16.0, 0.15)	(18.0, 0.18)	(19.0, 0.20)	(45.0, 0.32)	(71.0, 0.39)
					179	169	179	176	178	176
F						1.00	0.75	0.74	0.36	0.21
						(0.0, 0.00)	(14.0, 0.16)	(15.0, 0.17)	(43.0, 0.32)	(71.0, 0.38)
		R				179	179	171	178	177
G		(P90, COD)					1.00	0.76	0.59	0.15
		N					(0.0, 0.00)	(15.0, 0.19)	(43.0, 0.27)	(63.0, 0.39)
							1090	181	1083	1075
H								1.00	0.58	0.50
								(0.0, 0.00)	(38.0, 0.27)	(59.0, 0.31)
								181	180	178
I									1.00	0.05
									(0.0, 0.00)	(72.0, 0.40)
									1087	1072
J										1.00
										(0.0, 0.00)
										1080

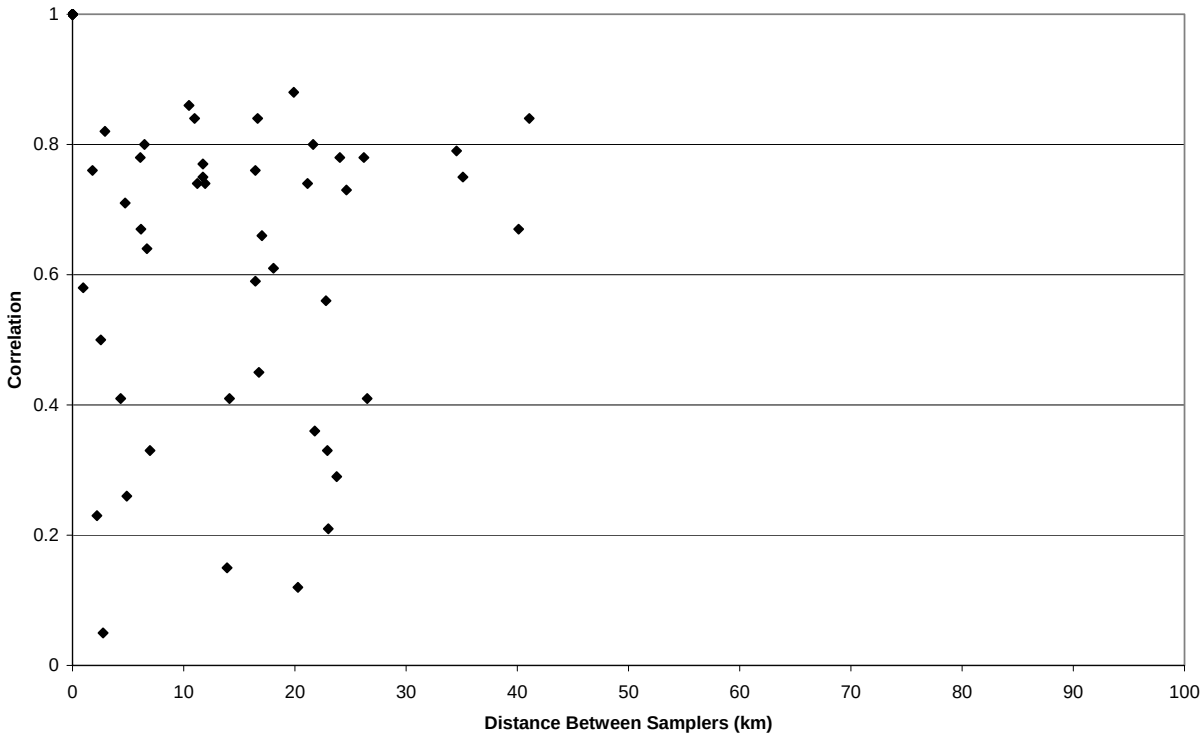


Figure A-76 PM_{10} inter-sampler correlations as a function of distance between monitors for Birmingham, AL.

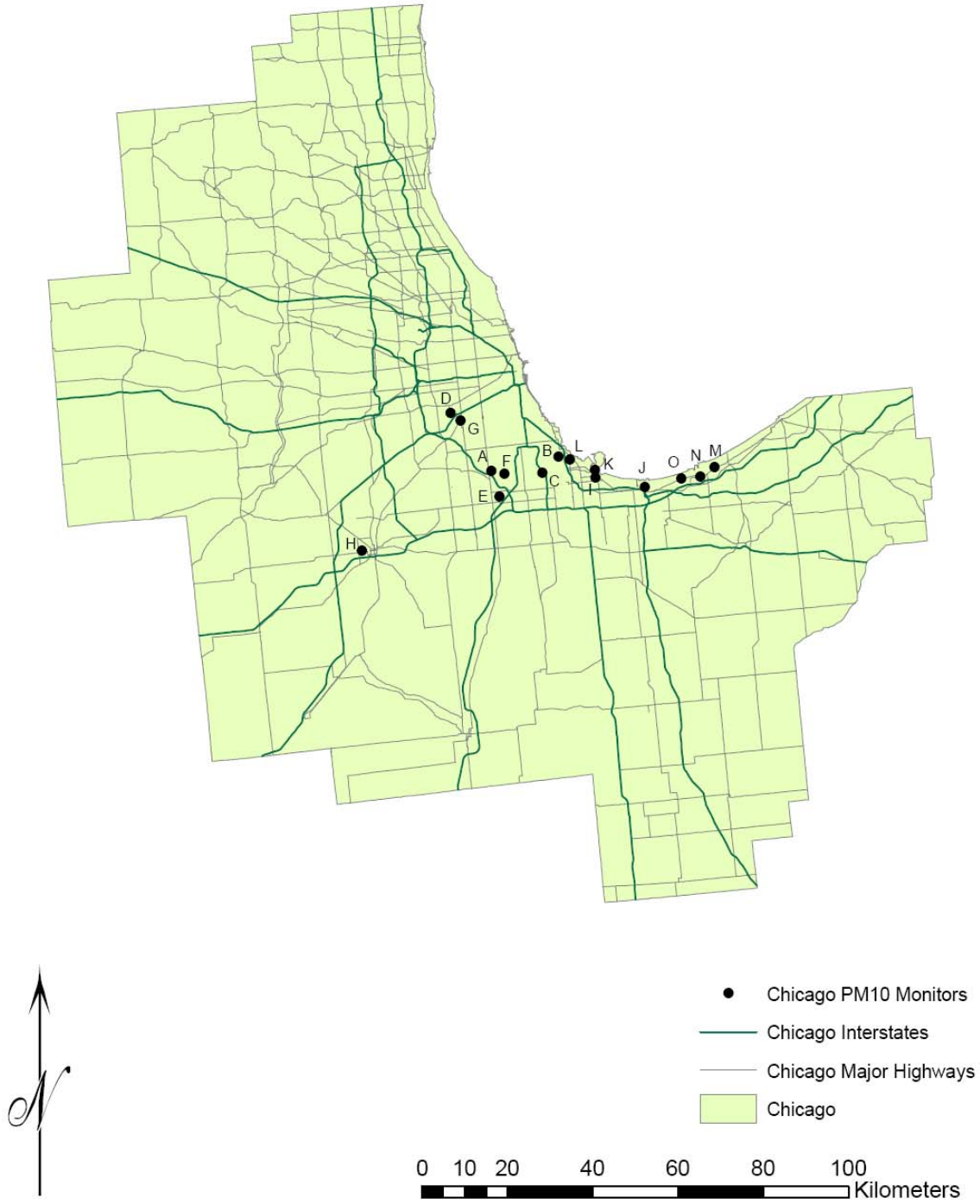


Figure A-77. PM_{10} monitor distribution and major highways, Chicago, IL.

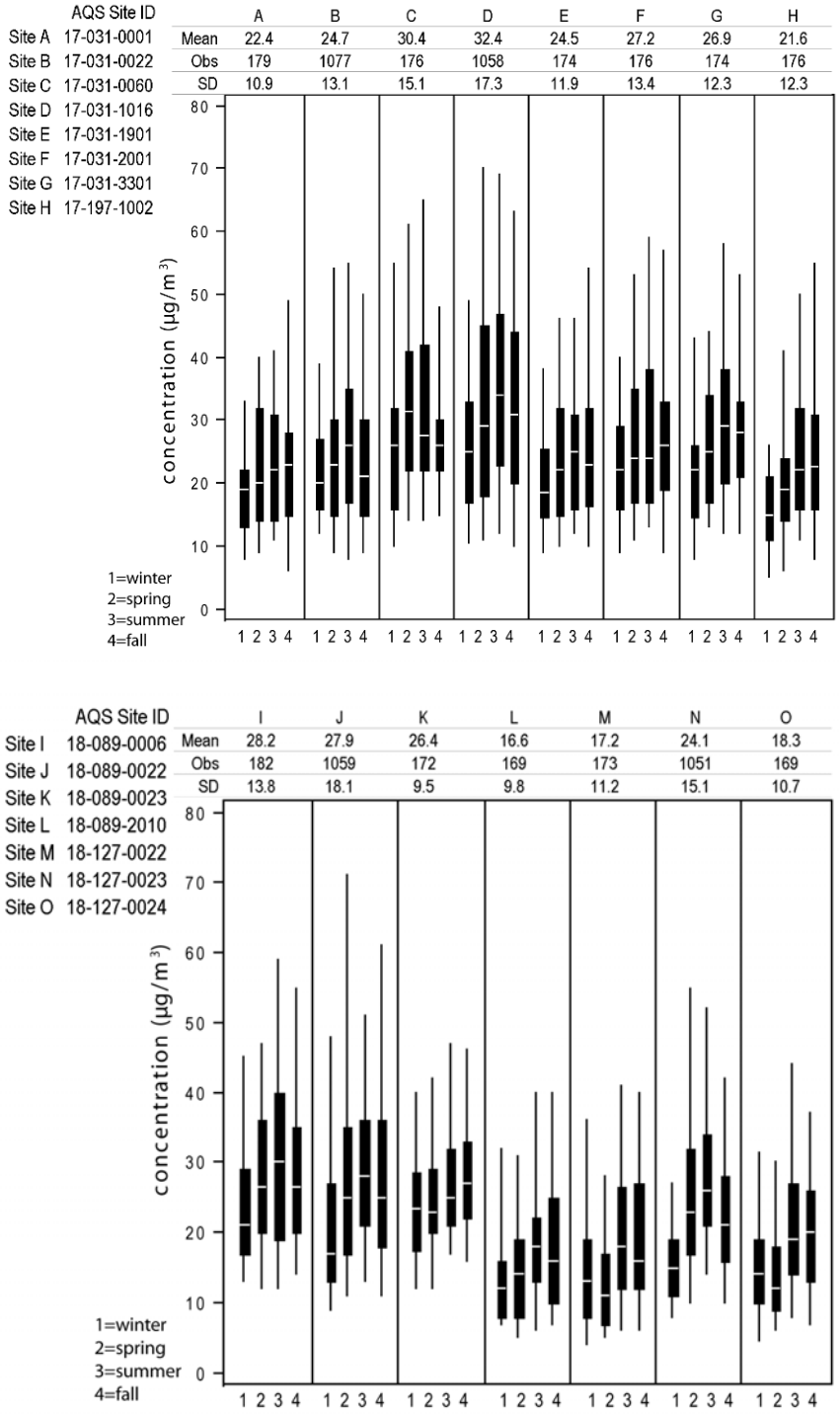


Figure A-78. Box plots illustrating the seasonal distribution of 24-h avg PM_{10} concentrations for Chicago, IL.

Table A-15. Inter-sampler correlation statistics for each pair of PM_{10} monitors reporting to AQS for Chicago, IL.

Site	A	B	C	D	E	F	G	H	I	J	K	L	M	N	O
A	1.00	0.78	0.68	0.83	0.93	0.92	0.86	0.79	0.75	0.14	0.69	0.89	0.55	0.27	0.75
	(0.0, 0.00)	(15.0, 0.18)	(23.0, 0.24)	(25.0, 0.22)	(8.0, 0.10)	(11.0, 0.13)	(12.0, 0.17)	(12.0, 0.18)	(13.0, 0.18)	(22.0, 0.28)	(15.0, 0.21)	(13.0, 0.22)	(21.0, 0.30)	(16.0, 0.24)	(15.0, 0.23)
	179	176	173	174	171	173	171	167	179	173	169	166	170	171	166
B		1.00	0.66	0.74	0.76	0.84	0.79	0.74	0.68	0.36	0.73	0.81	0.66	0.33	0.77
		(0.0, 0.00)	(23.0, 0.23)	(23.0, 0.21)	(14.0, 0.17)	(12.0, 0.15)	(13.0, 0.18)	(17.0, 0.23)	(16.0, 0.19)	(22.0, 0.24)	(16.0, 0.19)	(18.0, 0.27)	(23.0, 0.31)	(19.0, 0.25)	(20.0, 0.26)
		1077	173	1040	171	173	171	173	179	1041	169	166	170	1033	166
C			1.00	0.63	0.72	0.74	0.64	0.62	0.62	0.19	0.49	0.66	0.39	0.27	0.61
			(0.0, 0.00)	(26.0, 0.23)	(21.0, 0.21)	(18.5, 0.19)	(19.0, 0.21)	(22.0, 0.27)	(23.0, 0.20)	(26.5, 0.28)	(24.0, 0.23)	(29.0, 0.37)	(33.0, 0.40)	(26.0, 0.26)	(31.0, 0.35)
			176	171	169	170	168	164	176	170	166	163	167	168	163
D				1.00	0.79	0.85	0.79	0.74	0.70	0.23	0.69	0.82	0.61	0.29	0.76
				(0.0, 0.00)	(27.0, 0.21)	(19.0, 0.17)	(23.0, 0.19)	(27.0, 0.28)	(20.0, 0.19)	(32.0, 0.29)	(24.0, 0.23)	(31.0, 0.36)	(36.0, 0.39)	(31.0, 0.29)	(31.0, 0.33)
				1058	169	171	169	171	177	1022	168	166	168	1020	164
E					1.00	0.93	0.84	0.86	0.74	0.17	0.70	0.89	0.53	0.34	0.73
					(0.0, 0.00)	(9.0, 0.10)	(13.0, 0.16)	(10.0, 0.16)	(13.0, 0.16)	(22.0, 0.26)	(15.0, 0.19)	(15.0, 0.25)	(22.0, 0.33)	(17.0, 0.22)	(18.0, 0.25)
					174	168	166	163	174	168	164	161	166	166	163
F						1.00	0.84	0.86	0.77	0.21	0.75	0.89	0.62	0.32	0.80
						(0.0, 0.00)	(12.0, 0.15)	(13.0, 0.19)	(12.0, 0.14)	(23.0, 0.25)	(16.0, 0.17)	(18.0, 0.28)	(25.0, 0.34)	(20.0, 0.23)	(20.0, 0.27)
						176	169	165	176	170	166	163	167	168	163
G							1.00	0.77	0.69	0.28	0.74	0.86	0.52	0.33	0.70
							(0.0, 0.00)	(15.0, 0.22)	(14.0, 0.18)	(23.0, 0.26)	(14.0, 0.18)	(19.0, 0.31)	(24.0, 0.36)	(19.0, 0.24)	(22.0, 0.30)
							174	162	174	168	165	161	165	166	163
H								1.00	0.71	0.18	0.66	0.83	0.59	0.36	0.76
								(0.0, 0.00)	(16.0, 0.23)	(27.0, 0.30)	(18.0, 0.25)	(13.0, 0.23)	(19.0, 0.29)	(17.0, 0.25)	(14.0, 0.22)
								176	170	169	161	157	161	168	157
I									1.00	0.24	0.69	0.75	0.50	0.39	0.68
									(0.0, 0.00)	(22.0, 0.24)	(12.0, 0.15)	(20.0, 0.32)	(26.0, 0.37)	(16.0, 0.21)	(21.0, 0.30)
									182	176	172	169	173	174	169
J			R							1.00	0.49	0.38	0.22	0.48	0.22
			(P90, COD)							(0.0, 0.00)	(15.0, 0.20)	(25.0, 0.34)	(28.0, 0.36)	(22.0, 0.21)	(27.0, 0.33)
			N							1059	166	163	168	1018	164
K											1.00	0.80	0.54	0.49	0.65
											(0.0, 0.00)	(17.0, 0.32)	(24.0, 0.35)	(14.0, 0.19)	(21.0, 0.31)
											172	161	165	164	162
L												1.00	0.60	0.33	0.78
												(0.0, 0.00)	(15.0, 0.26)	(19.0, 0.31)	(10.0, 0.20)
												169	161	161	158
M													1.00	0.24	0.84
													(0.0, 0.00)	(21.0, 0.35)	(8.0, 0.16)
													173	165	161
N														1.00	0.31
														(0.0, 0.00)	(19.0, 0.29)
														1051	161
O															1.00
															(0.0, 0.00)
															169

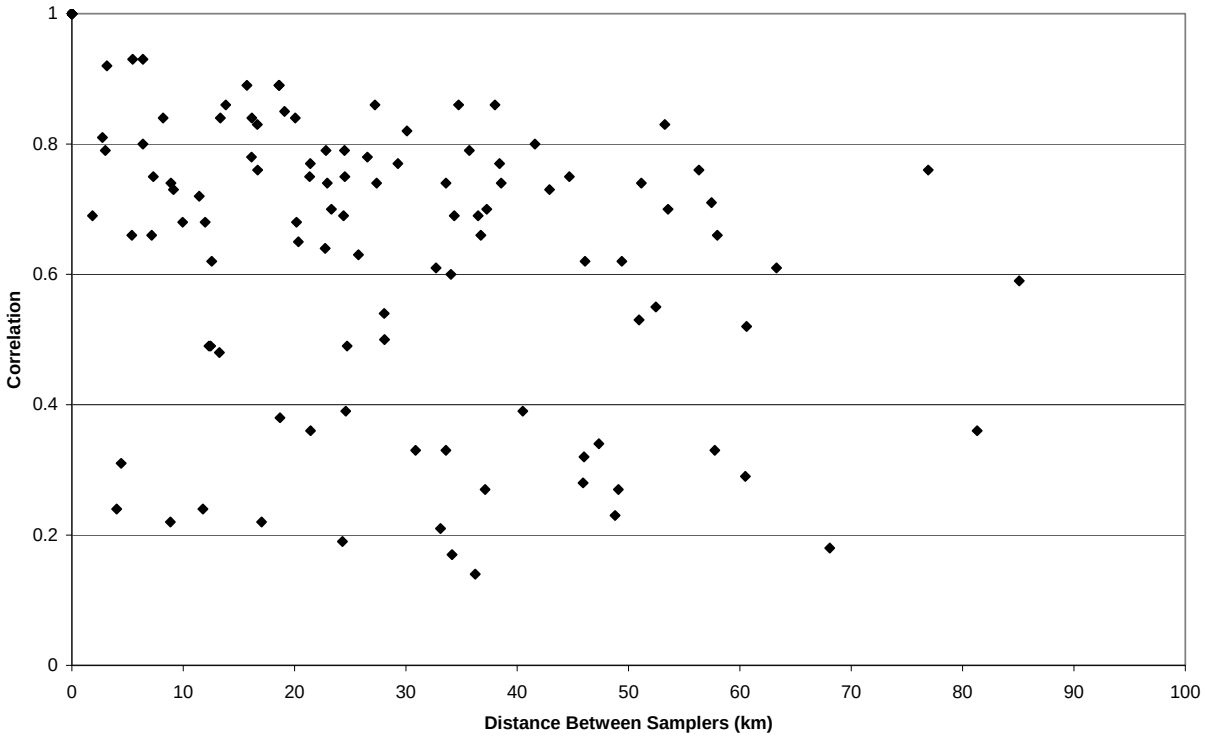


Figure A-79. PM_{10} inter-sampler correlations as a function of distance between monitors for Chicago, IL.

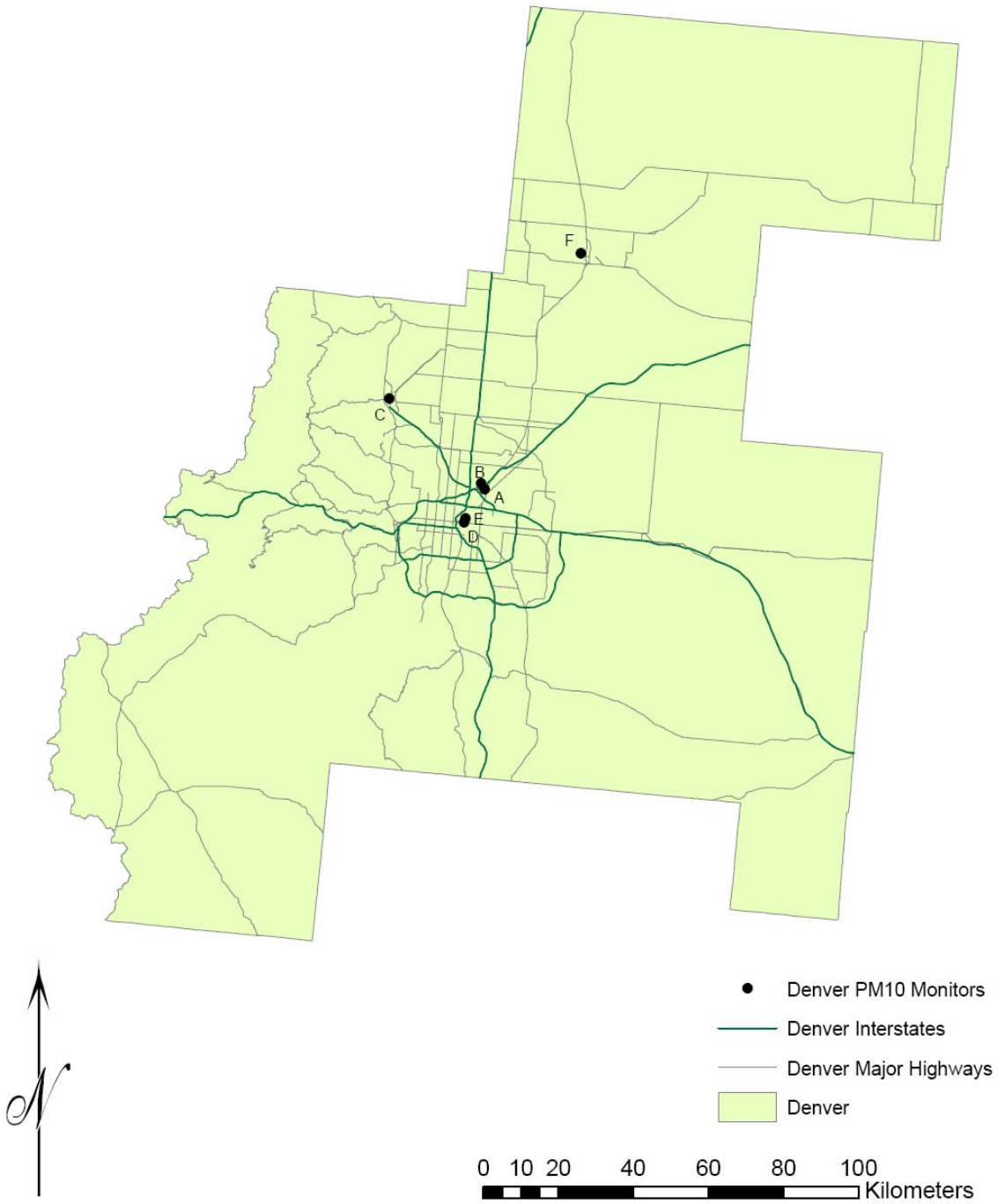


Figure A-80. PM_{10} monitor distribution and major highways, Denver, CO.

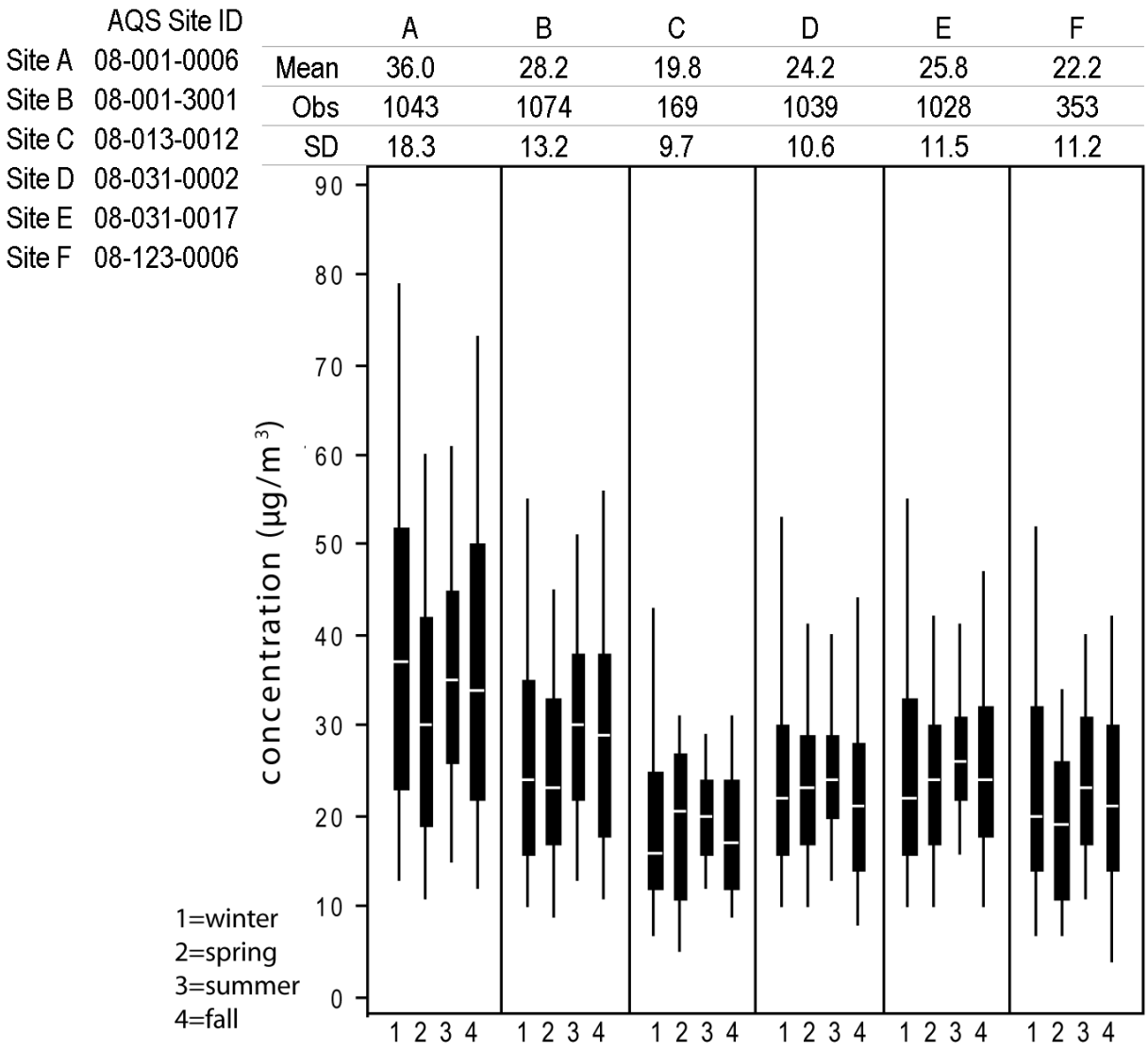


Figure A-81. Box plots illustrating the seasonal distribution of 24-h avg PM_{10} concentrations for Denver, CO.

Table A-16. Inter-sampler correlation statistics for each pair of PM_{10} monitors reporting to AQS for Denver, CO.

Site	A	B	C	D	E	F
A	1.00	0.84	0.43	0.70	0.72	0.67
	(0.0, 0.00)	(20.0, 0.16)	(36.0, 0.34)	(29.0, 0.24)	(26.0, 0.21)	(27.0, 0.28)
	1043	1022	164	987	980	339
B		1.00	0.57	0.72	0.74	0.72
		(0.0, 0.00)	(28.0, 0.27)	(17.0, 0.18)	(15.0, 0.16)	(18.0, 0.22)
		1074	169	1019	1007	348
C			1.00	0.75	0.72	0.51
			(0.0, 0.00)	(17.0, 0.23)	(16.0, 0.23)	(16.0, 0.23)
			169	169	156	164
D	R			1.00	0.89	0.52
	(P90, COD)			(0.0, 0.00)	(9.0, 0.13)	(17.0, 0.22)
	N			1039	976	341
E					1.00	0.58
					(0.0, 0.00)	(17.0, 0.23)
					1028	330
F						1.00

Site	A	B	C	D	E	F
						(0.0, 0.00)
						353

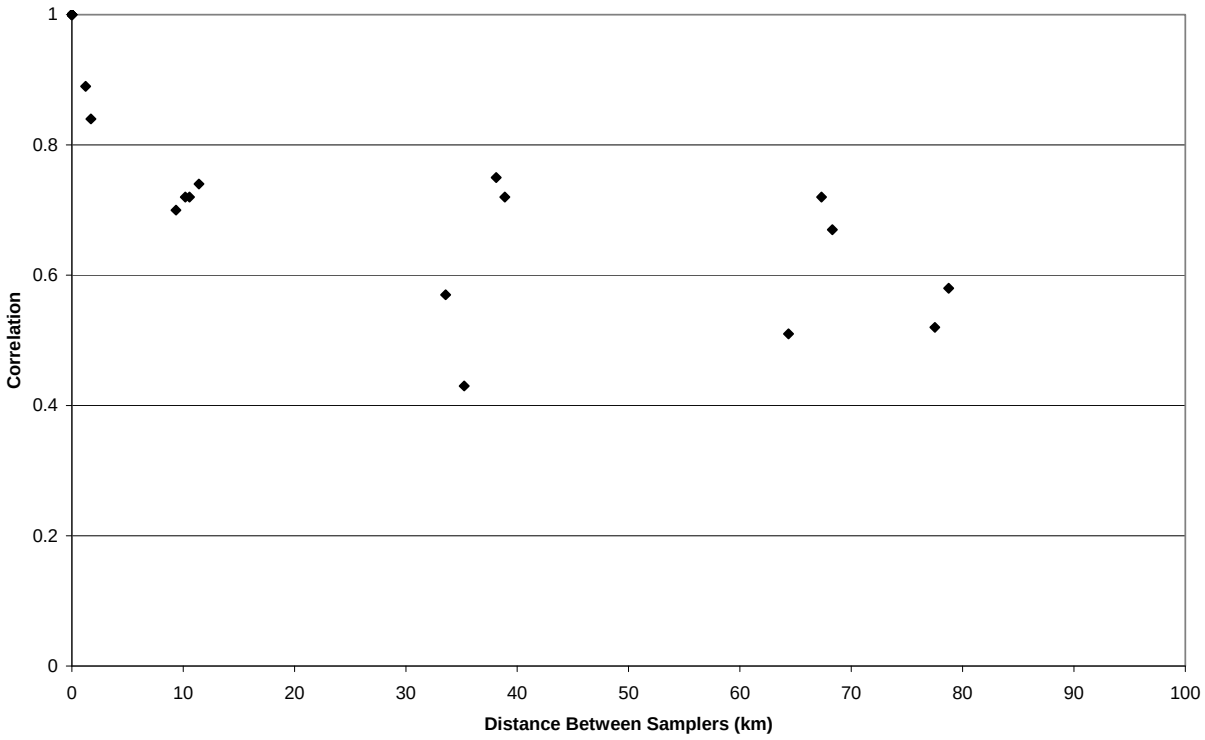


Figure A-82. PM_{10} inter-sampler correlations as a function of distance between monitors for Denver, CO.

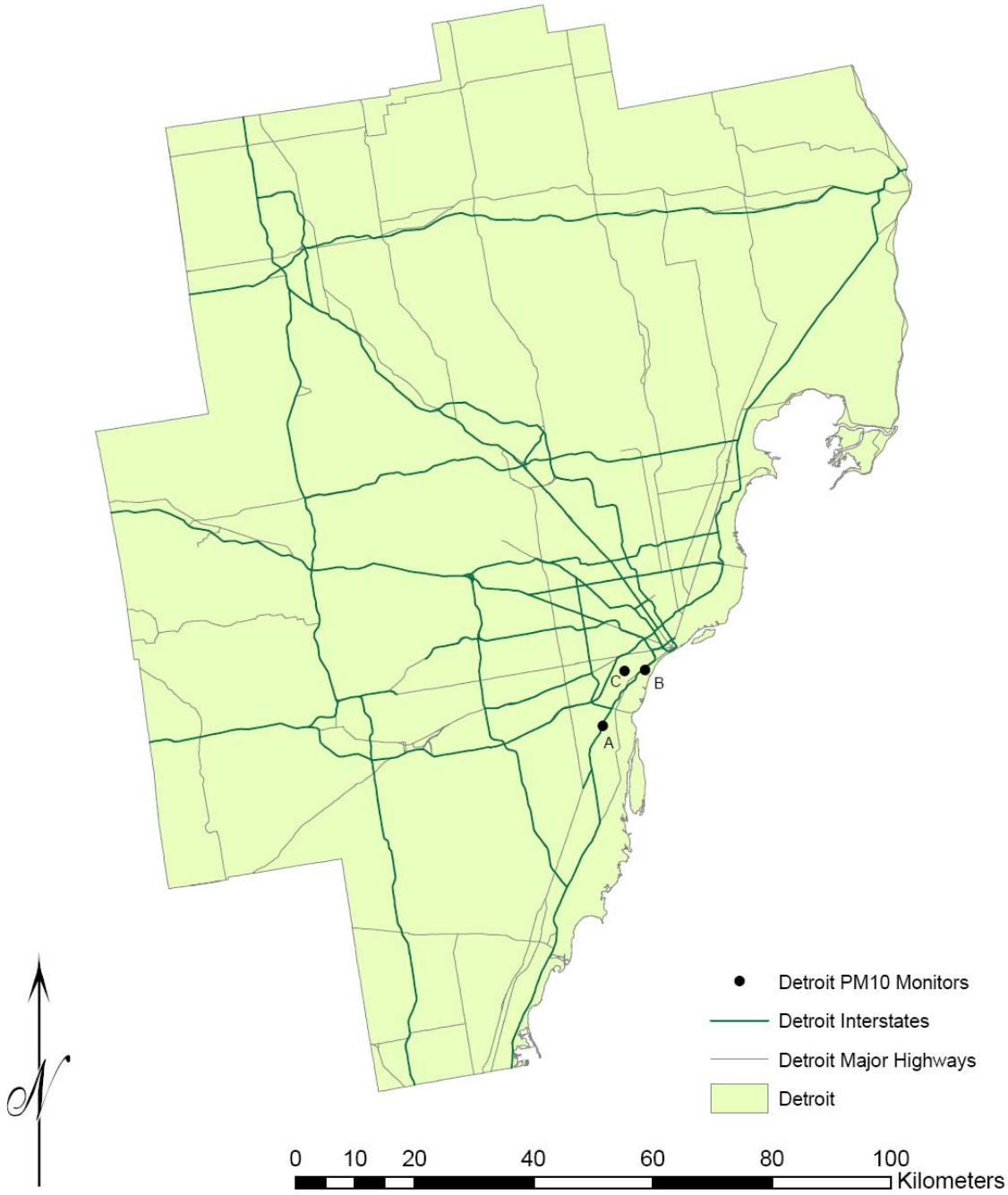


Figure A-83. PM_{10} monitor distribution and major highways, Detroit, MI.

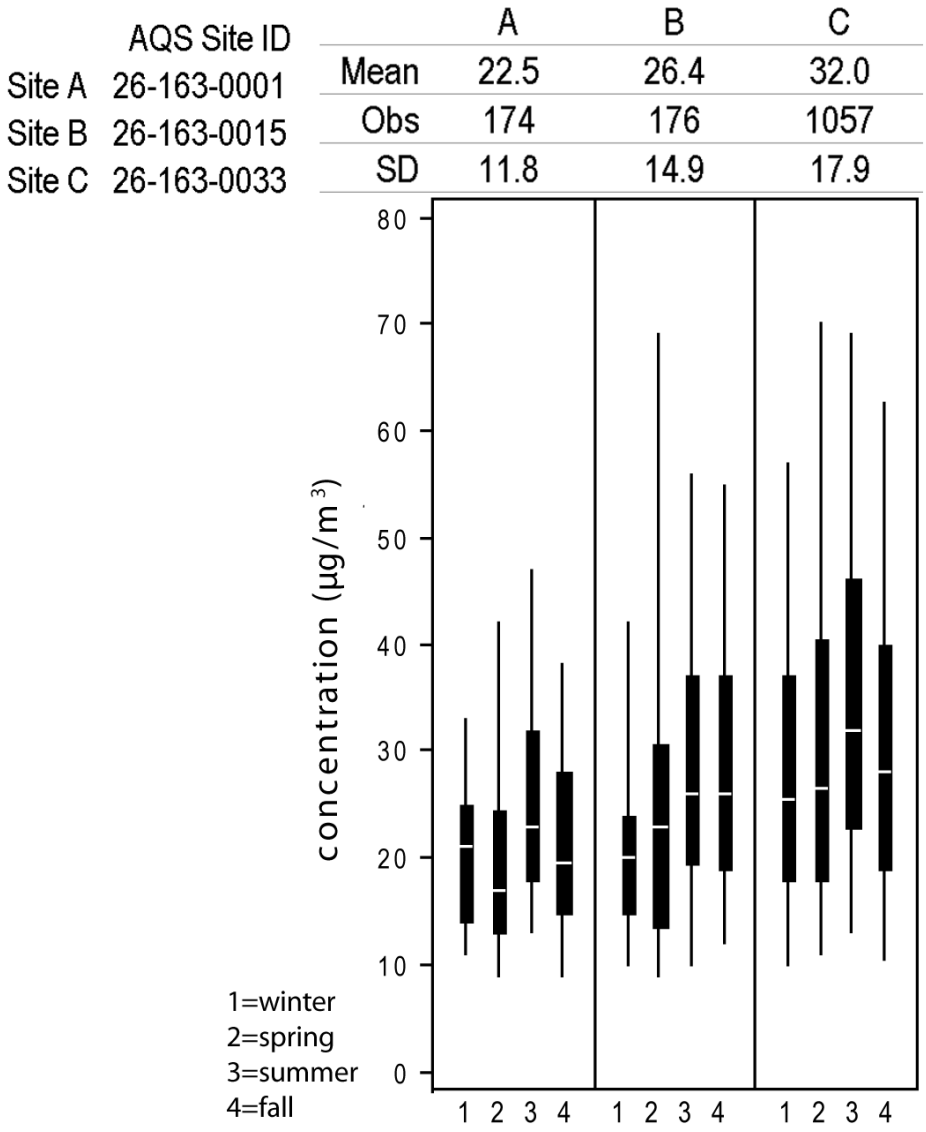


Figure A-84. Box plots illustrating the seasonal distribution of 24-h avg PM_{10} concentrations for Detroit, MI.

Table A-17. Inter-sampler correlation statistics for each pair of PM_{10} monitors reporting to AQS for Detroit, MI.

Site	A	B	C
A	1.00	0.77	0.74
	(0.0, 0.00)	(14.0, 0.18)	(28.0, 0.26)
	174	169	172
B		1.00	0.79
		(0.0, 0.00)	(21.0, 0.21)
		176	174
C	R		1.00
	(P90, COD)		(0.0, 0.00)
	N		
			1057

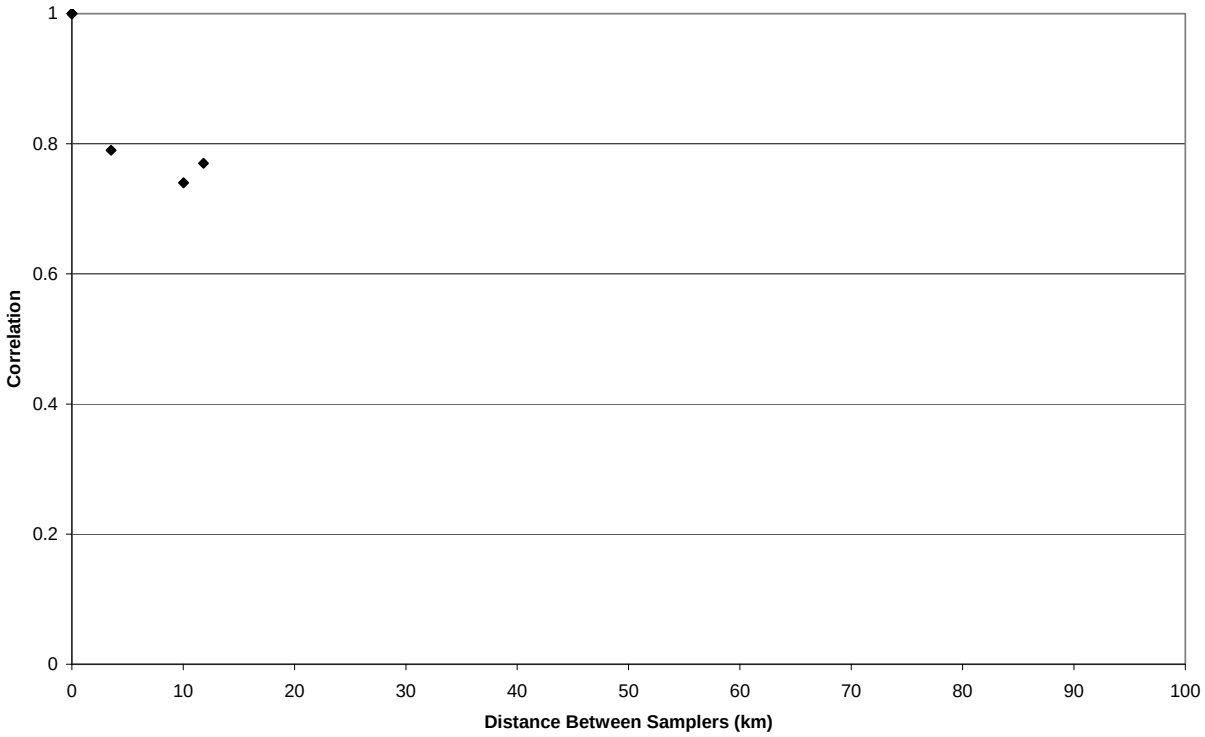


Figure A-85. PM_{10} inter-sampler correlations as a function of distance between monitors for Detroit, MI.

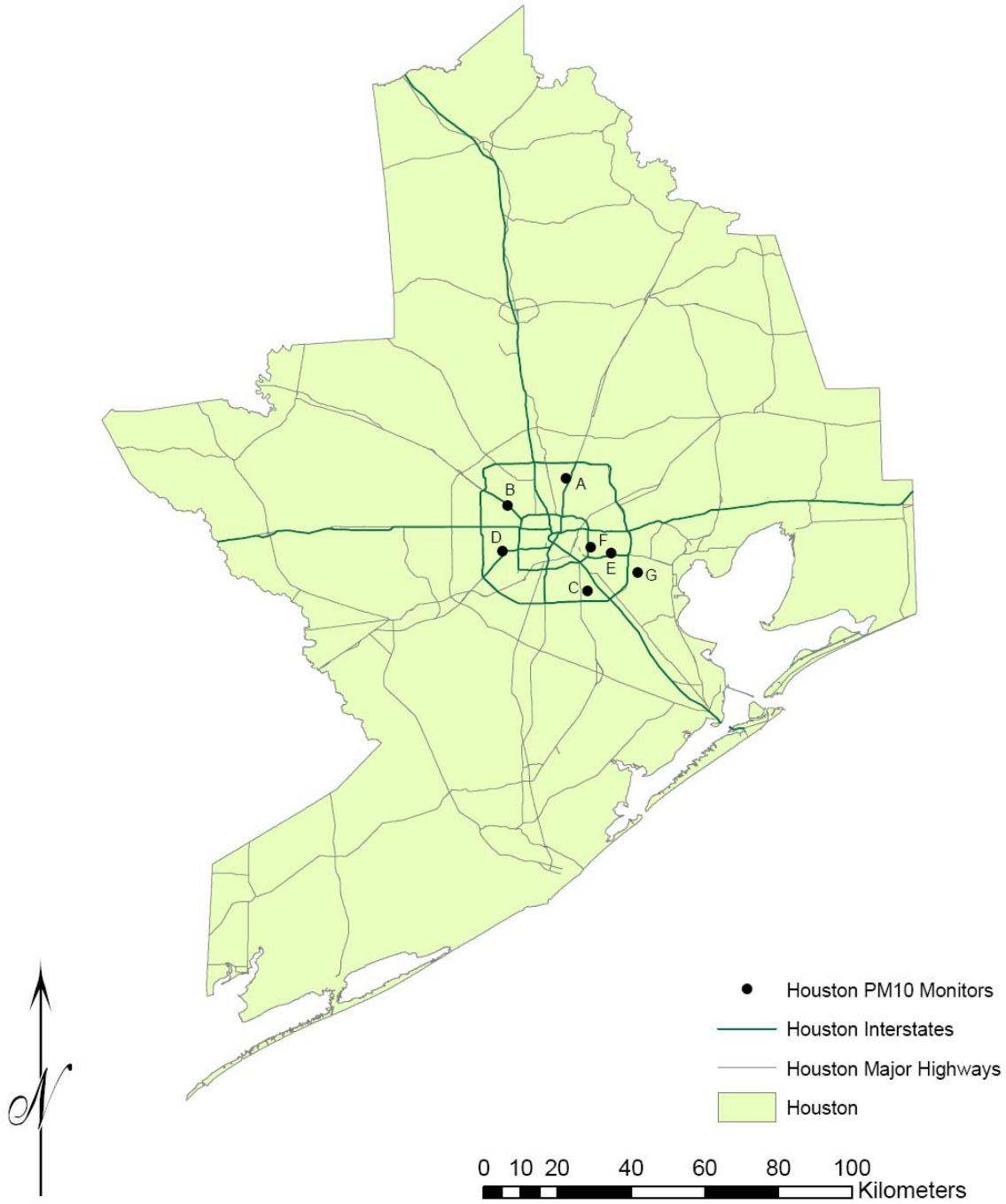


Figure A-86. PM_{10} monitor distribution and major highways, Houston, TX.

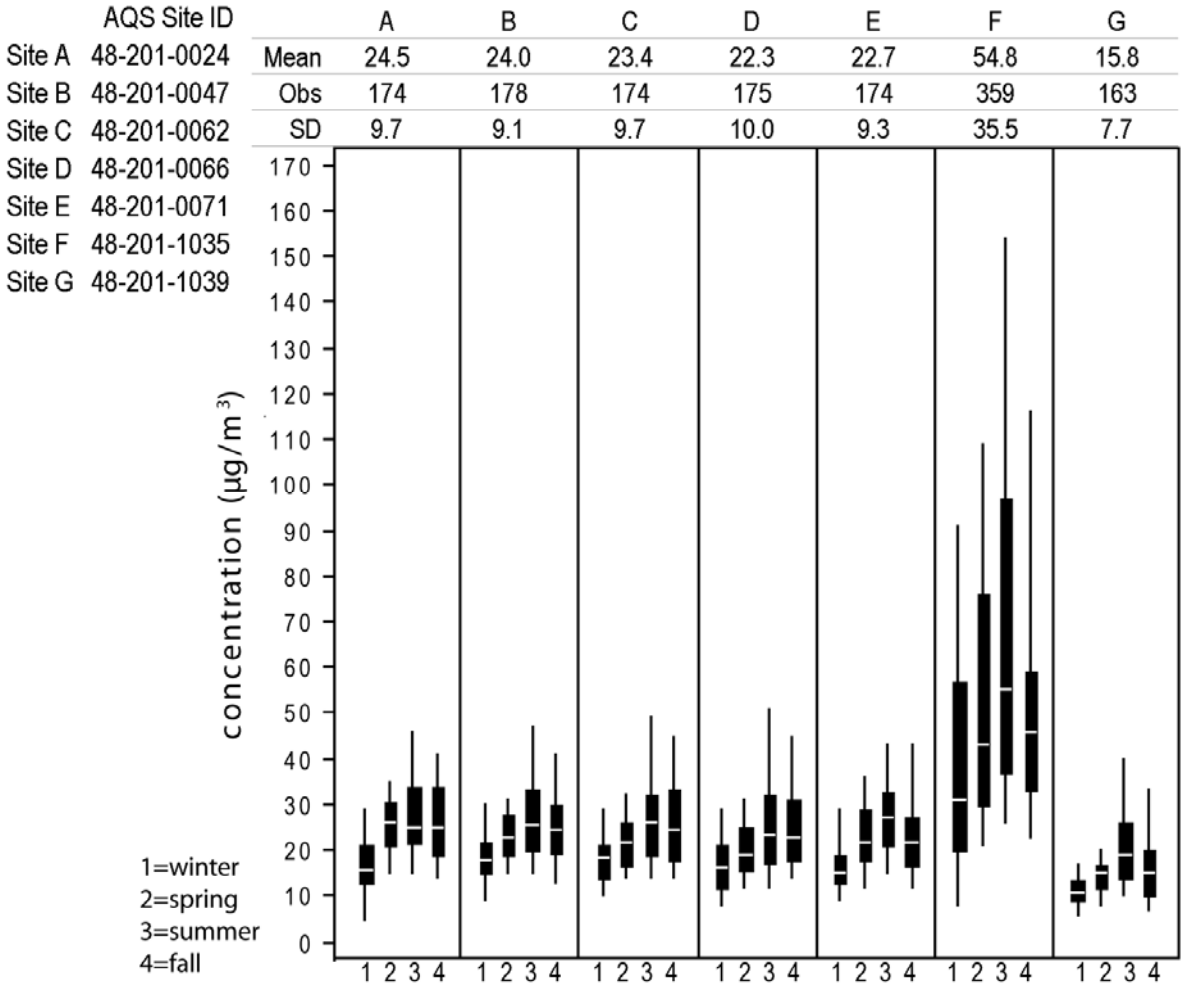


Figure A-87. Box plots illustrating the seasonal distribution of 24-h avg PM_{10} concentrations for Houston, TX.

Table A-18. Inter-sampler correlation statistics for each pair of PM_{10} monitors reporting to AQS for Houston, TX.

SITE	A	B	C	D	E	F	G
A	1.00	0.84	0.78	0.76	0.43	0.56	0.75
	(0.0, 0.00)	(9.0, 0.12)	(11.0, 0.16)	(12.0, 0.16)	(15.0, 0.20)	(77.0, 0.37)	(17.0, 0.28)
	174	163	158	165	167	159	156
B		1.00	0.86	0.86	0.38	0.52	0.79
		(0.0, 0.00)	(9.0, 0.11)	(9.0, 0.12)	(15.0, 0.19)	(74.0, 0.39)	(16.0, 0.26)
		178	156	160	163	158	152
C			1.00	0.83	0.41	0.38	0.85
			(0.0, 0.00)	(10.0, 0.14)	(17.0, 0.19)	(74.0, 0.40)	(14.5, 0.25)
			174	156	159	151	150
D				1.00	0.32	0.43	0.76
				(0.0, 0.00)	(18.0, 0.20)	(81.0, 0.43)	(16.0, 0.23)
				175	163	155	154
E					1.00	0.15	0.38
					(0.0, 0.00)	(78.0, 0.43)	(20.0, 0.28)
	R				174	158	157
F	(P90, COD)					1.00	0.37
	N					(0.0, 0.00)	(92.0, 0.54)
						359	149
G							1.00
							(0.0, 0.00)
							163

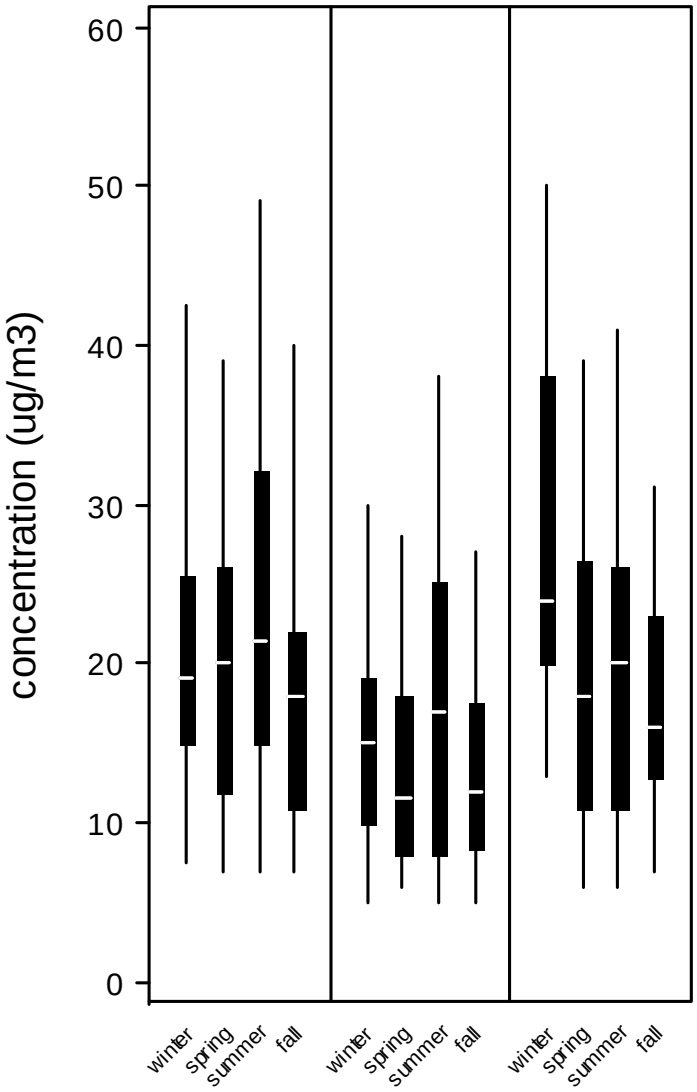


Figure A-88. Box plots illustrating the seasonal distribution of 24-h avg PM_{10} concentrations for New York City, NY.

Table A-19. Inter-sampler correlation statistics for each pair of PM_{10} monitors reporting to AQS for New York City, NY.

Site	A	B	C
A	1.00	0.88	0.82
	(0.0, 0.00)	(11.0, 0.20)	(12.0, 0.16)
	167	156	164
B		1.00	0.74
		(0.0, 0.00)	(18.0, 0.25)
		169	166
C	R (P90, COD) N		1.00
			(0.0, 0.00)
			178

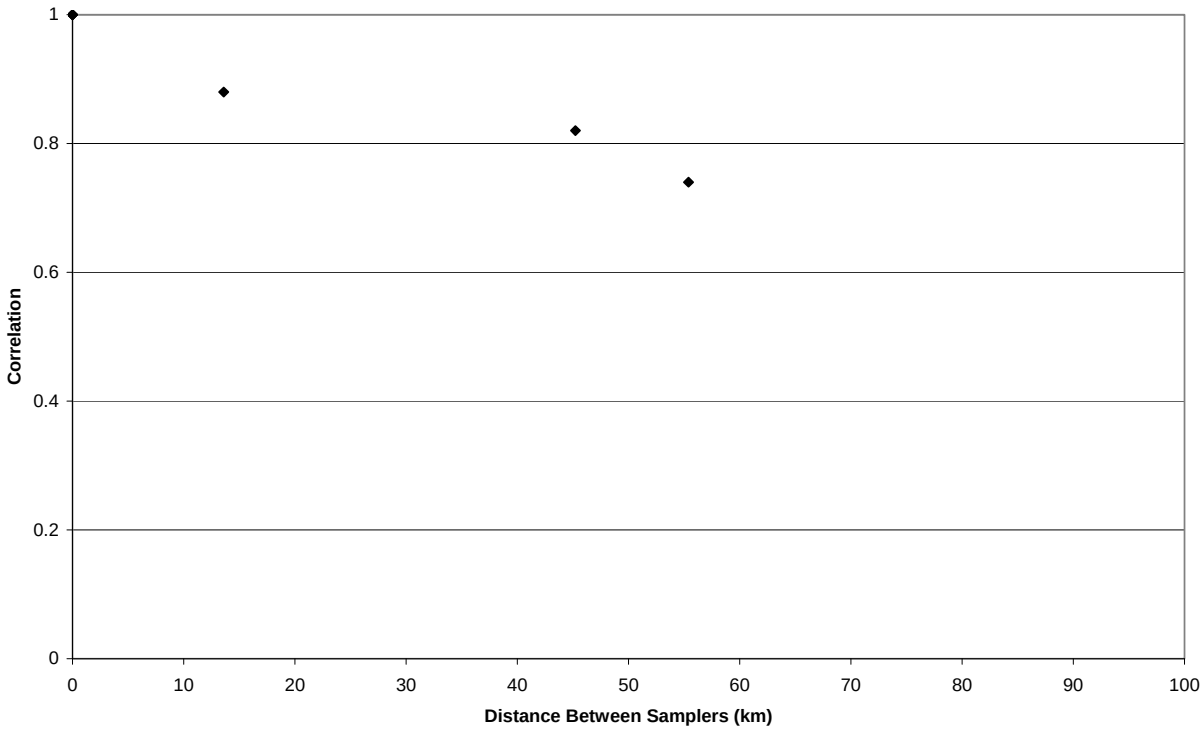


Figure A-89. PM_{10} inter-sampler correlations as a function of distance between monitors for New York City, NY.

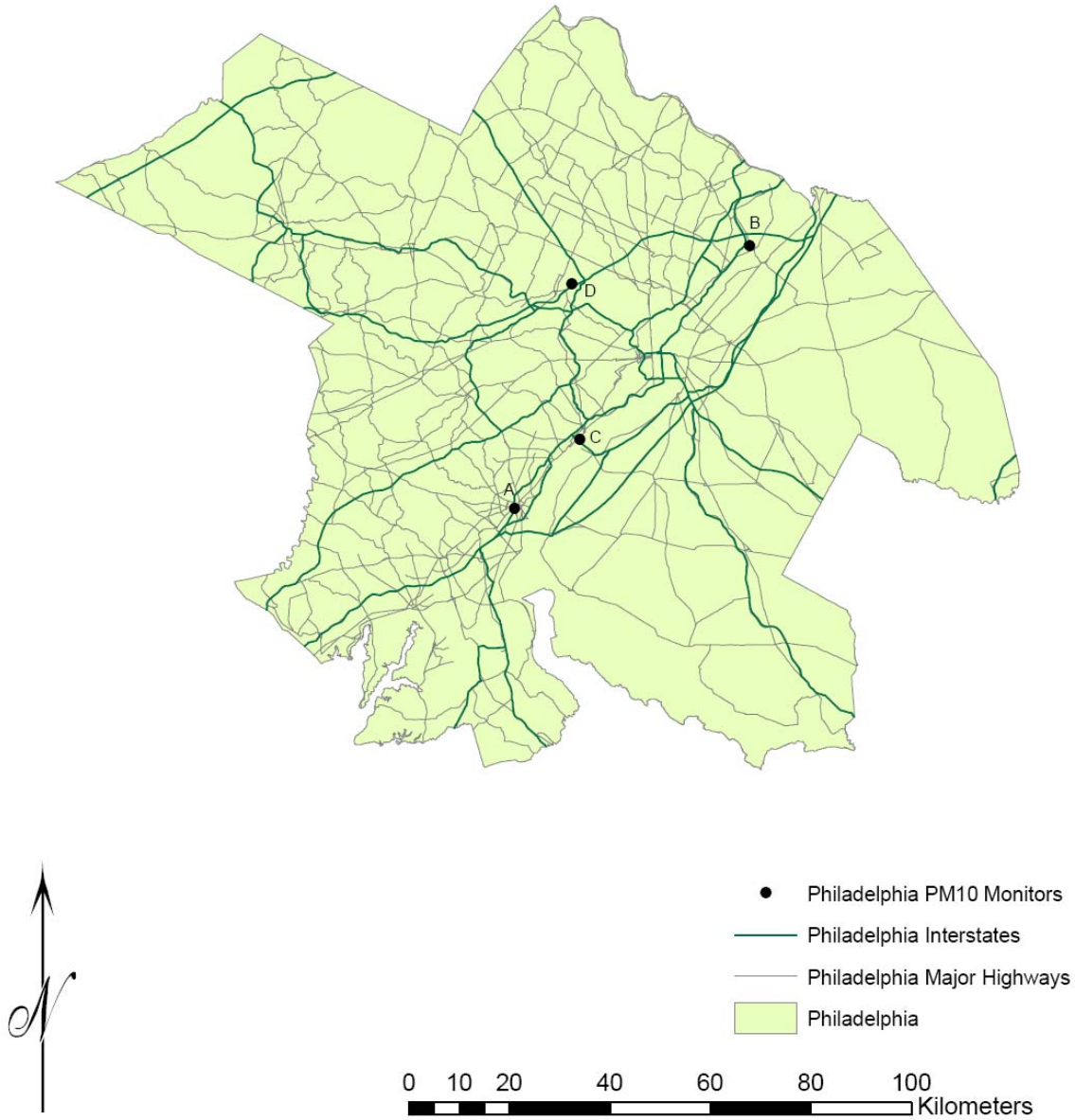


Figure A-90. PM_{10} monitor distribution and major highways, Philadelphia, PA.

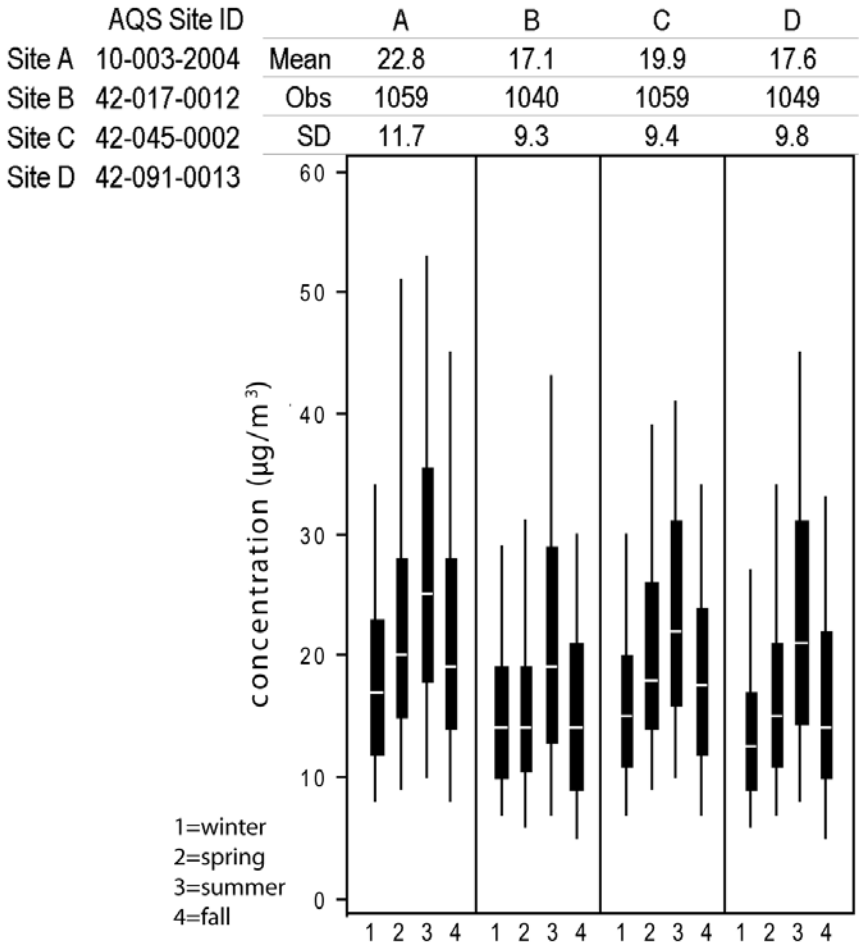


Figure A-91. Box plots illustrating the seasonal distribution of 24-h avg PM_{10} concentrations for Philadelphia, PA.

Table A-20. Inter-sampler correlation statistics for each pair of PM_{10} monitors reporting to AQS for Philadelphia, PA.

Site	A	B	C	D
A	1.00	0.81	0.64	0.84
	(0.0, 0.00)	(13.0, 0.21)	(14.0, 0.19)	(12.0, 0.20)
	1059	1005	1025	1013
B		1.00	0.71	0.93
		(0.0, 0.00)	(11.0, 0.20)	(6.0, 0.12)
		1040	1006	994
C			1.00	0.73
			(0.0, 0.00)	(11.0, 0.19)
			1059	1014
D	R (P90, COD)			1.00
	N			(0.0, 0.00)
				1049

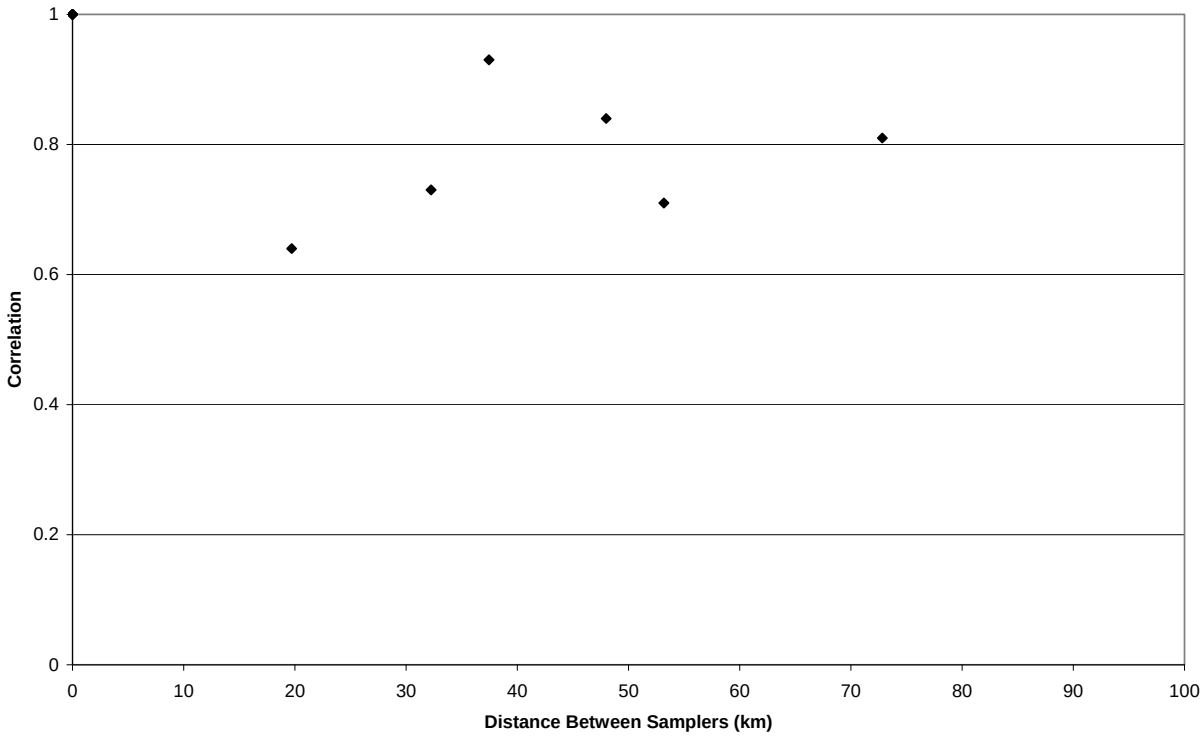


Figure A-92. PM_{10} inter-sampler correlations as a function of distance between monitors for Philadelphia, PA.

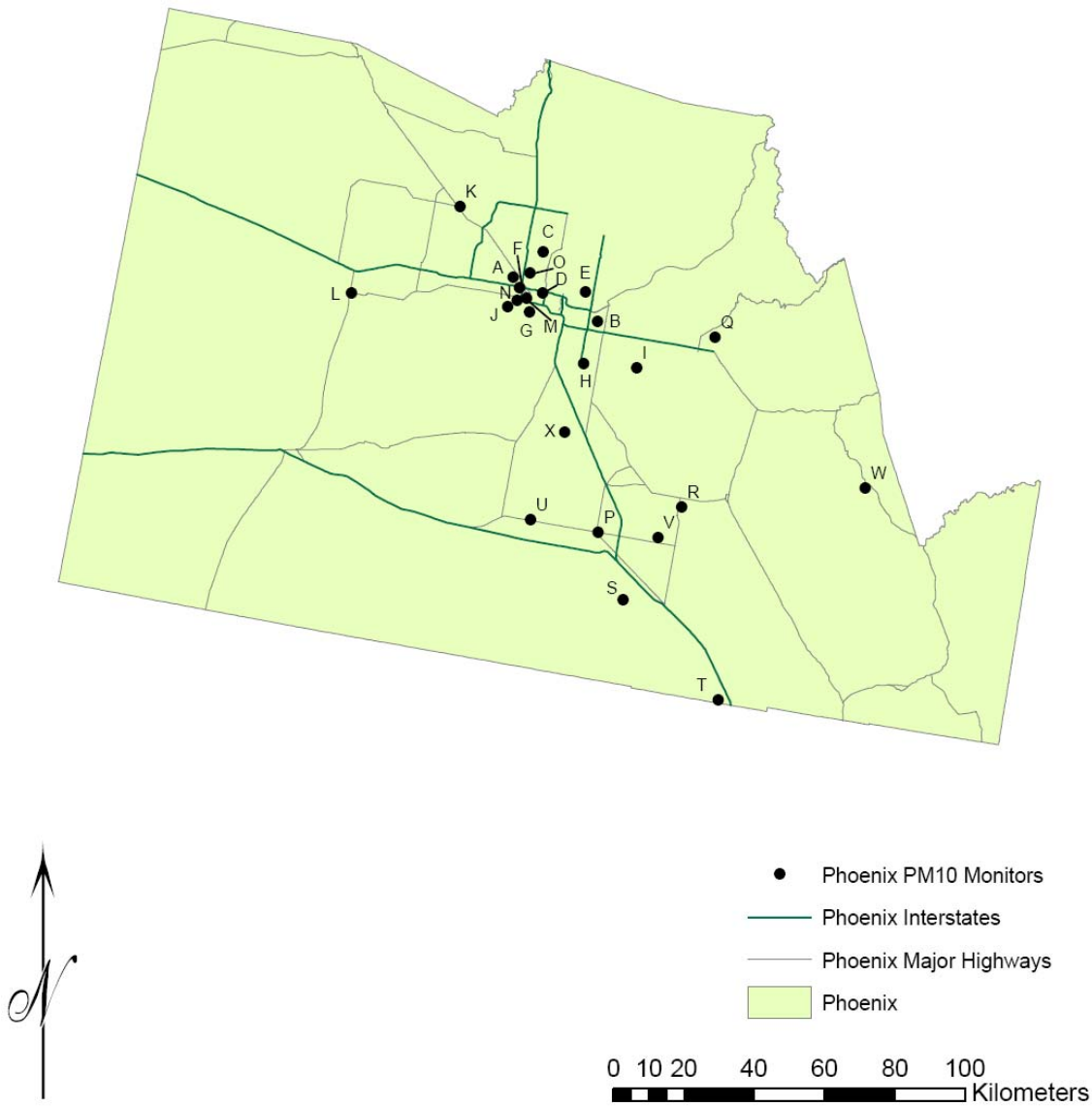
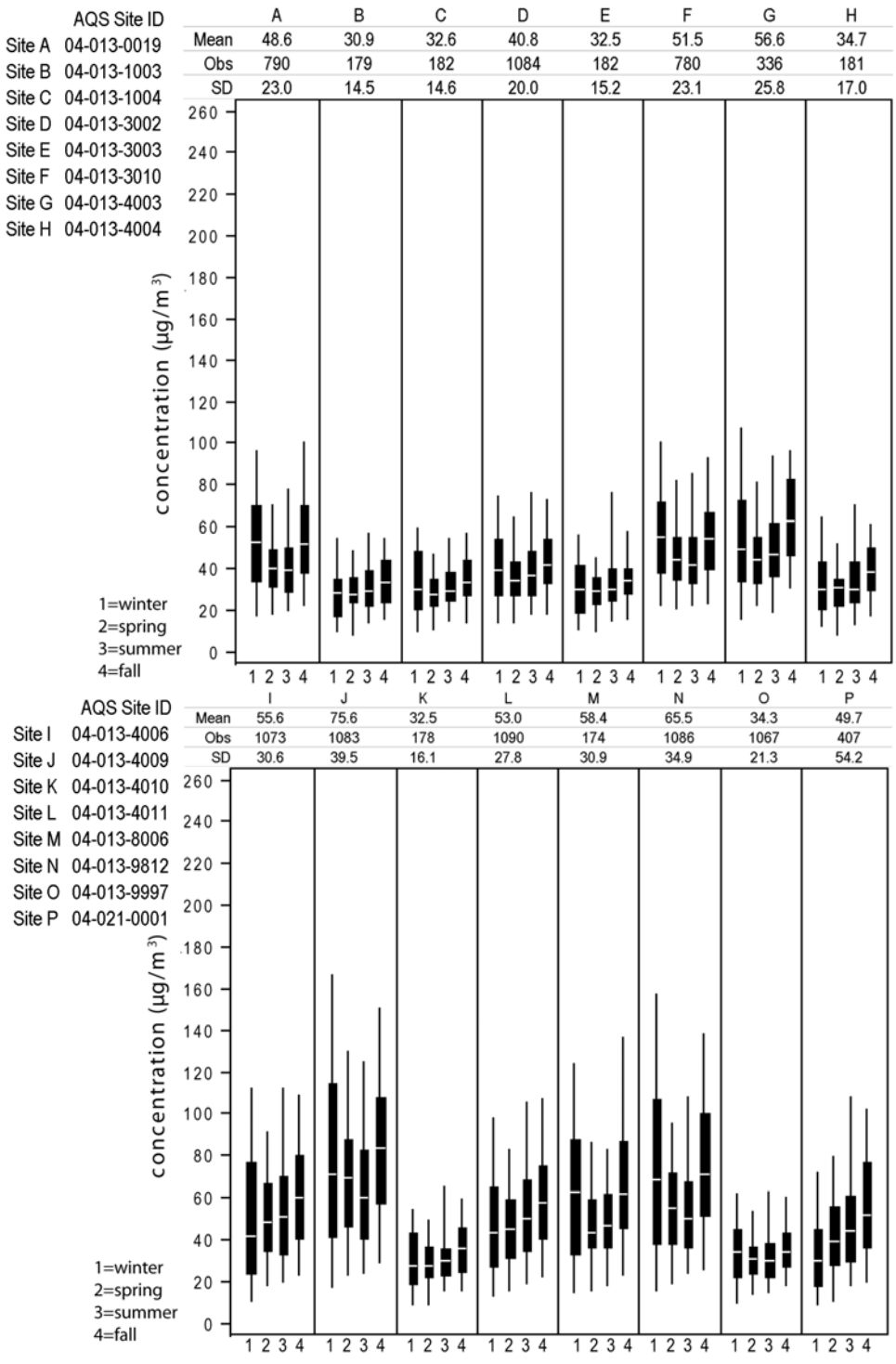


Figure A-93. PM_{10} monitor distribution and major highways, Phoenix, AZ.


AQS Site ID
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Site B 04-013-1003
Site C 04-013-1004
Site D 04-013-3002
Site E 04-013-3003
Site F 04-013-3010
Site G 04-013-4003
Site H 04-013-4004
A B C D E F G H
Mean 48.6 30.9 32.6 40.8 32.5 51.5 56.6 34.7
Obs 790 179 182 1084 182 780 336 181
SD 23.0 14.5 14.6 20.0 15.2 23.1 25.8 17.0
concentration (µg/m³)
1=winter
2=spring
3=summer
4=fall
AQS Site ID
Site I 04-013-4006
Site J 04-013-4009
Site K 04-013-4010
Site L 04-013-4011
Site M 04-013-8006
Site N 04-013-9812
Site O 04-013-9997
Site P 04-021-0001
I J K L M N O P
Mean 55.6 75.6 32.5 53.0 58.4 65.5 34.3 49.7
Obs 1073 1083 178 1090 174 1086 1067 407
SD 30.6 39.5 16.1 27.8 30.9 34.9 21.3 54.2
concentration (µg/m³)
1=winter
2=spring
3=summer
4=fall

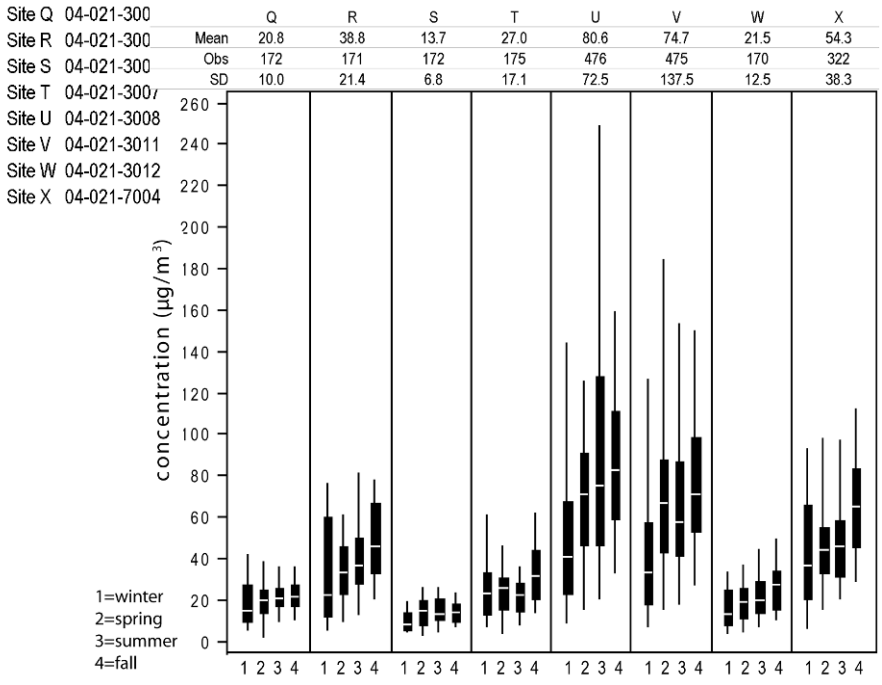

Figure A-94. Box plots illustrating the seasonal distribution of 24-h avg PM_{10} concentrations for Phoenix, AZ.

Table A-21. Inter-sampler correlation statistics for each pair of PM_{10} monitors reporting to AQS for Phoenix, AZ.

Site	A	B	C	D	E	F	G	H	I	J	K	L	M
A	1.00	0.71	0.85	0.85	0.67	0.94	0.86	0.77	0.73	0.83	0.77	0.70	0.87
	(0.0, 0.00)	(38.0, 0.25)	(33.0, 0.21)	(21.0, 0.12)	(38.0, 0.23)	(14.0, 0.09)	(22.0, 0.13)	(34.0, 0.21)	(35.0, 0.18)	(59.0, 0.24)	(34.0, 0.24)	(30.0, 0.17)	(28.5, 0.16)
	790	178	181	788	181	779	335	180	772	781	177	789	170
B		1.00	0.84	0.82	0.85	0.67	0.74	0.81	0.67	0.68	0.75	0.60	0.63
		(0.0, 0.00)	(13.0, 0.12)	(23.0, 0.19)	(11.0, 0.11)	(37.0, 0.29)	(47.0, 0.30)	(13.0, 0.13)	(49.0, 0.30)	(84.0, 0.43)	(16.0, 0.15)	(51.0, 0.31)	(56.0, 0.32)
		179	179	177	179	175	179	178	175	176	175	178	164
C			1.00	0.88	0.81	0.78	0.80	0.81	0.70	0.73	0.81	0.63	0.75
			(0.0, 0.00)	(20.0, 0.16)	(12.0, 0.11)	(38.0, 0.27)	(44.0, 0.28)	(13.0, 0.13)	(48.0, 0.29)	(84.0, 0.41)	(15.0, 0.14)	(49.0, 0.29)	(55.0, 0.30)
			182	180	182	178	182	181	178	179	178	181	167
D				1.00	0.76	0.88	0.81	0.82	0.76	0.78	0.79	0.65	0.83
				(0.0, 0.00)	(23.0, 0.17)	(22.0, 0.14)	(29.0, 0.16)	(18.0, 0.17)	(39.0, 0.20)	(71.0, 0.31)	(22.0, 0.19)	(35.0, 0.20)	(42.0, 0.21)
				1084	180	778	334	179	1062	1072	176	1080	172
E					1.00	0.64	0.68	0.74	0.66	0.59	0.67	0.51	0.61
					(0.0, 0.00)	(40.0, 0.27)	(47.0, 0.29)	(16.0, 0.14)	(48.0, 0.29)	(88.0, 0.42)	(15.0, 0.15)	(49.0, 0.30)	(58.0, 0.31)
					182	178	182	181	178	179	178	181	167
F						1.00	0.83	0.76	0.75	0.86	0.74	0.69	0.87
						(0.0, 0.00)	(22.0, 0.13)	(36.0, 0.25)	(32.0, 0.17)	(54.0, 0.21)	(41.0, 0.28)	(30.0, 0.17)	(25.0, 0.15)
						780	331	177	762	772	175	779	167
G							1.00	0.77	0.65	0.78	0.71	0.65	0.80
							(0.0, 0.00)	(44.0, 0.26)	(38.0, 0.19)	(48.0, 0.19)	(46.0, 0.30)	(36.0, 0.19)	(33.0, 0.16)
							336	181	326	333	178	335	169
H								1.00	0.79	0.81	0.79	0.69	0.72
								(0.0, 0.00)	(47.0, 0.26)	(79.0, 0.39)	(16.0, 0.14)	(43.0, 0.27)	(53.0, 0.29)
			R					181	177	178	177	180	167
I			(P90, COD)						1.00	0.79	0.76	0.69	0.68
			N						(0.0, 0.00)	(52.0, 0.22)	(48.0, 0.29)	(33.0, 0.17)	(38.0, 0.20)
									1073	1061	174	1068	171
J										1.00	0.78	0.73	0.80
										(0.0, 0.00)	(83.0, 0.42)	(57.0, 0.23)	(51.0, 0.22)
										1083	175	1078	171
K											1.00	0.72	0.68
											(0.0, 0.00)	(45.0, 0.29)	(56.0, 0.32)
											178	177	164
L												1.00	0.63
												(0.0, 0.00)	(42.0, 0.20)
												1090	173
M													1.00
													(0.0, 0.00)
													174

Table A-40, continued

	N	O	P	Q	R	S	T	U	V	W	X
A	0.87	0.68	0.47	0.53	0.68	0.40	0.69	0.50	0.27	0.56	0.65
	(39.0, 0.18)	(28.0, 0.17)	(29.0, 0.19)	(49.0, 0.42)	(34.0, 0.27)	(64.0, 0.57)	(40.0, 0.34)	(82.0, 0.31)	(49.0, 0.27)	(48.0, 0.43)	(31.0, 0.20)
	784	783	406	171	171	171	174	475	474	169	262
B	0.59	0.75	0.75	0.73	0.63	0.55	0.59	0.53	0.66	0.65	0.64
	(67.0, 0.37)	(15.0, 0.15)	(22.0, 0.17)	(23.0, 0.27)	(30.0, 0.25)	(32.0, 0.43)	(21.0, 0.24)	(94.0, 0.41)	(62.0, 0.34)	(24.0, 0.30)	(46.0, 0.29)
	178	179	175	169	168	169	172	172	177	167	155
C	0.70	0.87	0.80	0.70	0.71	0.48	0.64	0.56	0.71	0.62	0.60
	(69.0, 0.35)	(11.0, 0.12)	(19.0, 0.15)	(24.0, 0.28)	(26.0, 0.24)	(36.0, 0.44)	(22.0, 0.24)	(91.0, 0.40)	(59.0, 0.32)	(28.0, 0.31)	(43.0, 0.28)
	181	182	178	172	171	172	175	175	180	170	157
D	0.78	0.86	0.73	0.63	0.68	0.49	0.65	0.66	0.45	0.58	0.70
	(57.0, 0.25)	(15.0, 0.12)	(30.0, 0.19)	(38.0, 0.38)	(27.0, 0.25)	(46.0, 0.53)	(31.0, 0.31)	(87.0, 0.34)	(59.0, 0.30)	(38.0, 0.39)	(32.0, 0.21)
	1075	1056	405	170	169	170	173	474	473	168	318
E	0.60	0.73	0.68	0.72	0.64	0.43	0.48	0.42	0.69	0.51	0.52
	(67.0, 0.35)	(14.0, 0.14)	(21.0, 0.17)	(21.0, 0.28)	(27.0, 0.24)	(33.0, 0.44)	(21.0, 0.25)	(93.0, 0.41)	(63.0, 0.32)	(25.0, 0.32)	(46.0, 0.28)
	181	182	178	172	171	172	175	175	180	170	157
F	0.91	0.68	0.46	0.48	0.63	0.38	0.63	0.47	0.28	0.42	0.66
	(35.0, 0.14)	(31.0, 0.21)	(30.0, 0.22)	(60.0, 0.46)	(37.0, 0.30)	(68.0, 0.60)	(45.0, 0.39)	(80.0, 0.31)	(50.0, 0.27)	(57.0, 0.47)	(34.0, 0.22)
	774	773	403	169	167	168	172	470	469	166	259
G	0.77	0.57	0.47	0.55	0.65	0.46	0.62	0.49	0.44	0.57	0.64
	(35.0, 0.16)	(41.0, 0.25)	(36.5, 0.24)	(61.0, 0.47)	(41.0, 0.30)	(73.0, 0.61)	(58.0, 0.41)	(78.0, 0.28)	(45.0, 0.24)	(59.0, 0.48)	(32.0, 0.22)
	332	336	330	172	171	172	175	329	334	170	185
H	0.70	0.75	0.82	0.63	0.74	0.55	0.62	0.60	0.76	0.64	0.76
	(66.0, 0.33)	(15.0, 0.14)	(18.0, 0.15)	(29.0, 0.31)	(24.5, 0.22)	(37.0, 0.46)	(24.0, 0.25)	(84.0, 0.38)	(58.0, 0.29)	(30.0, 0.33)	(39.0, 0.25)
	180	181	177	171	170	171	174	174	179	169	156
I	0.76	0.61	0.52	0.57	0.71	0.51	0.58	0.59	0.37	0.51	0.80
	(42.0, 0.18)	(49.0, 0.27)	(39.0, 0.22)	(66.0, 0.47)	(41.0, 0.27)	(77.0, 0.60)	(60.0, 0.40)	(72.0, 0.27)	(46.0, 0.23)	(63.0, 0.47)	(30.0, 0.16)
	1064	1045	397	169	168	168	171	461	461	167	314
J	0.91	0.58	0.41	0.48	0.65	0.48	0.65	0.51	0.28	0.46	0.74
	(29.0, 0.12)	(83.0, 0.38)	(68.0, 0.31)	(103.0, 0.58)	(75.0, 0.40)	(115.0, 0.69)	(92.0, 0.51)	(69.0, 0.26)	(59.0, 0.27)	(101.0, 0.58)	(62.0, 0.27)
	1074	1055	404	169	168	169	172	473	472	167	319
K	0.69	0.71	0.75	0.52	0.64	0.52	0.62	0.71	0.68	0.55	0.68
	(73.0, 0.36)	(16.0, 0.16)	(19.0, 0.18)	(28.0, 0.29)	(27.0, 0.23)	(34.0, 0.44)	(22.0, 0.24)	(89.0, 0.40)	(59.0, 0.33)	(28.0, 0.32)	(44.0, 0.29)
	177	178	174	168	167	168	171	171	176	166	153
L	0.68	0.55	0.51	0.47	0.57	0.48	0.49	0.59	0.33	0.50	0.68
	(48.0, 0.20)	(44.0, 0.26)	(37.0, 0.22)	(66.0, 0.47)	(44.5, 0.29)	(71.0, 0.60)	(62.0, 0.40)	(75.0, 0.27)	(53.0, 0.24)	(67.0, 0.48)	(29.0, 0.18)
	1081	1063	406	171	170	171	174	475	474	169	321
M	0.86	0.81	0.75	0.48	0.64	0.37	0.62	0.46	0.65	0.44	0.59
	(32.0, 0.16)	(53.0, 0.29)	(47.0, 0.30)	(74.0, 0.48)	(51.0, 0.32)	(80.0, 0.61)	(58.5, 0.41)	(62.0, 0.31)	(48.0, 0.26)	(68.0, 0.49)	(42.0, 0.24)
	173	174	165	157	158	158	160	165	168	156	145
N	1.00	0.58	0.41	0.48	0.67	0.42	0.63	0.42	0.26	0.40	0.60
	(0.0, 0.00)	(66.0, 0.32)	(51.0, 0.27)	(88.0, 0.53)	(62.5, 0.35)	(98.0, 0.65)	(75.0, 0.46)	(71.0, 0.29)	(55.0, 0.27)	(88.0, 0.54)	(48.0, 0.24)
	1086	1059	403	171	170	171	174	470	469	169	319
O		1.00	0.90	0.61	0.64	0.39	0.60	0.72	0.59	0.55	0.64
		(0.0, 0.00)	(35.0, 0.22)	(28.0, 0.31)	(25.0, 0.24)	(38.0, 0.47)	(22.0, 0.26)	(94.0, 0.39)	(69.0, 0.35)	(29.0, 0.33)	(44.0, 0.26)
		1067	407	172	171	172	175	475	473	170	317
P			1.00	0.67	0.81	0.58	0.78	0.82	0.64	0.71	0.67
			(0.0, 0.00)	(32.0, 0.29)	(22.0, 0.19)	(44.0, 0.45)	(21.0, 0.21)	(80.0, 0.30)	(52.0, 0.23)	(32.0, 0.31)	(39.0, 0.24)
			407	169	170	169	172	400	404	167	197
Q				1.00	0.72	0.65	0.57	0.36	0.58	0.68	0.47
				(0.0, 0.00)	(40.0, 0.33)	(15.0, 0.28)	(23.0, 0.24)	(104.0, 0.53)	(78.0, 0.46)	(15.0, 0.22)	(62.0, 0.43)
				172	162	163	167	165	171	161	148
R					1.00	0.66	0.68	0.53	0.82	0.68	0.68
					(0.0, 0.00)	(55.0, 0.48)	(32.0, 0.27)	(75.0, 0.35)	(47.0, 0.25)	(40.0, 0.34)	(39.0, 0.24)
					171	162	165	164	171	160	148
S						1.00	0.60	0.46	0.59	0.72	0.52
						(0.0, 0.00)	(28.0, 0.35)	(115.0, 0.65)	(86.0, 0.59)	(19.0, 0.28)	(74.0, 0.58)
						172	167	165	171	162	149
T							1.00	0.56	0.66	0.68	0.61
							(0.0, 0.00)	(94.0, 0.47)	(71.0, 0.39)	(18.0, 0.24)	(51.5, 0.37)
							175	169	174	165	150
U								1.00	0.54	0.52	0.71
								(0.0, 0.00)	(66.0, 0.24)	(101.0, 0.53)	(61.0, 0.25)
								476	464	165	204
V									1.00	0.60	0.64
									(0.0, 0.00)	(78.0, 0.47)	(35.0, 0.20)
									475	169	206
W										1.00	0.56
										(0.0, 0.00)	(63.0, 0.44)
										170	145
											1.00
											(0.0, 0.00)
											322

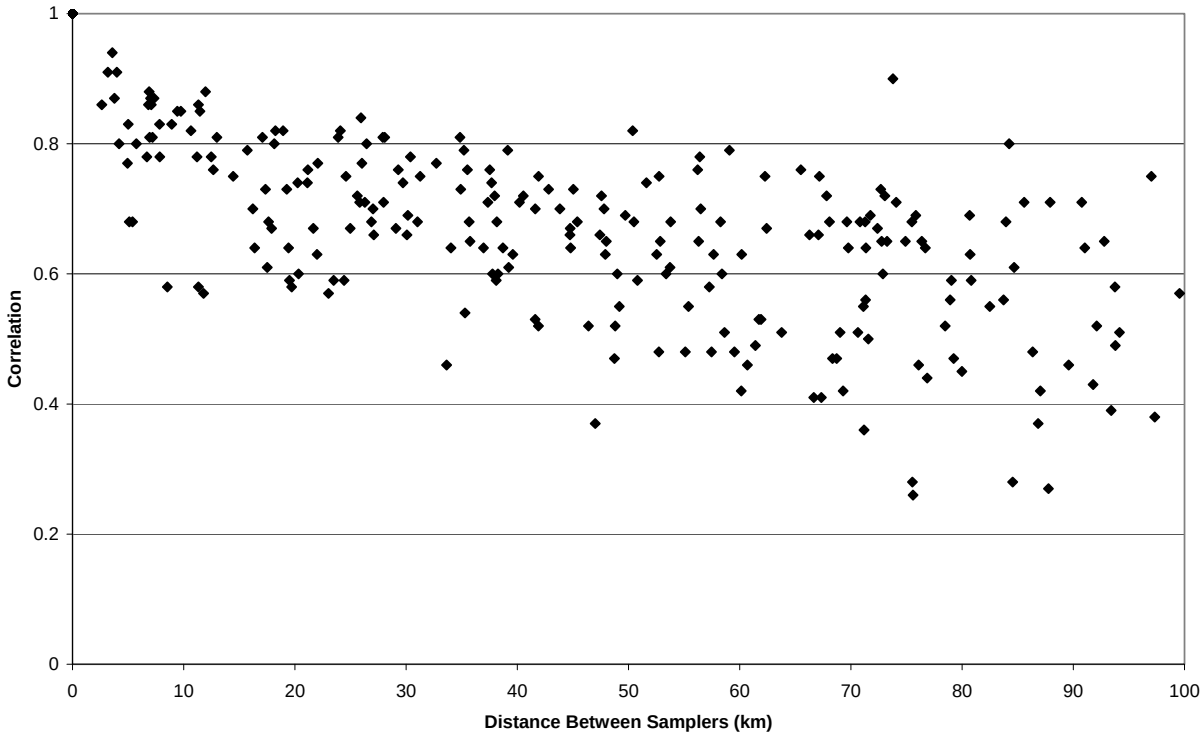

Figure A-95. PM_{10} inter-sampler correlations as a function of distance between monitors for Phoenix, AZ.

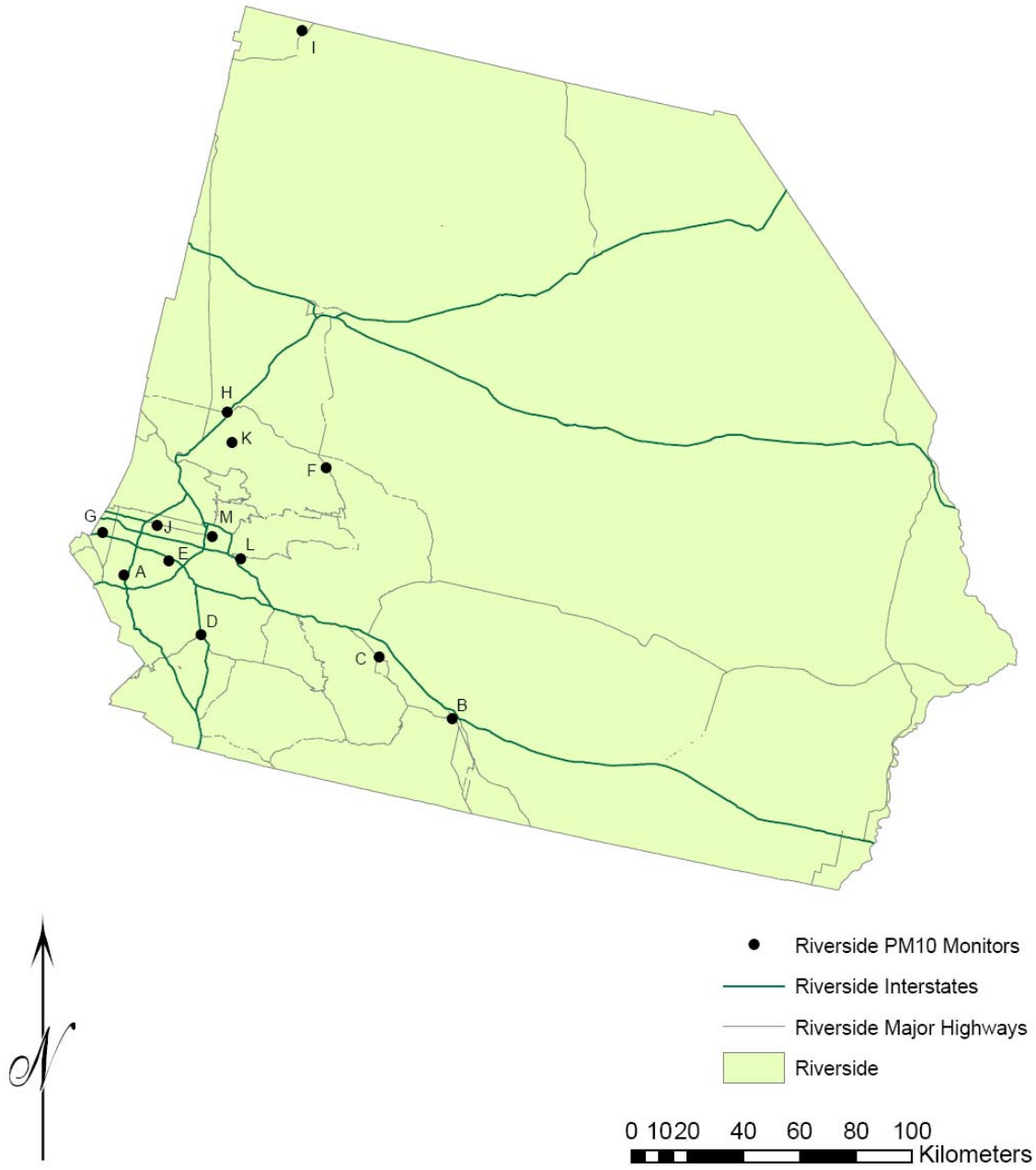

Figure A-96. PM_{10} monitor distribution and major highways, Riverside, CA.

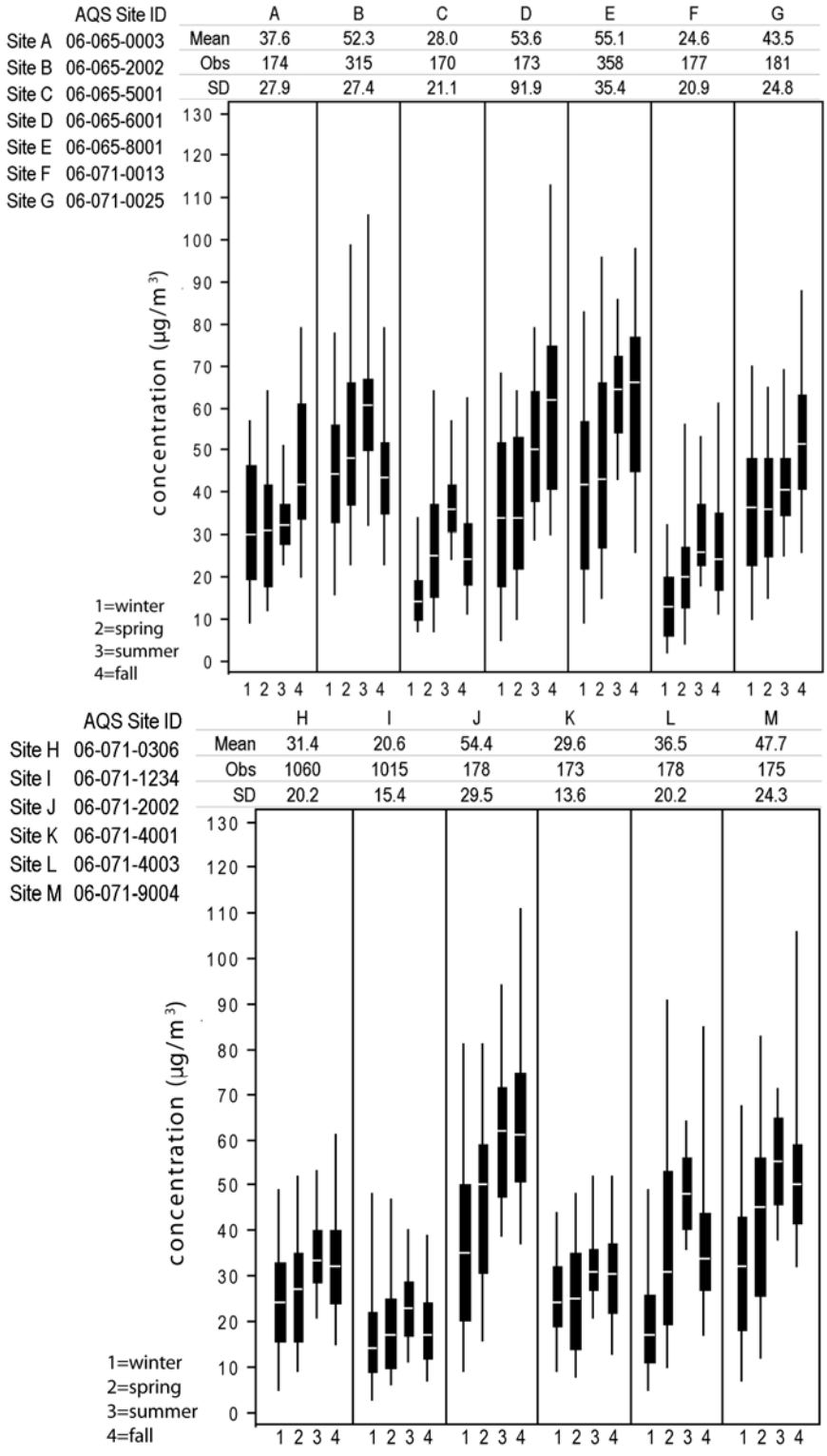

Figure A-97. Box plots illustrating the seasonal distribution of 24-h avg PM_{10} concentrations for Riverside, CA.

Table A-22. Inter-sampler correlation statistics for each pair of PM_{10} monitors reporting to AQS for Riverside, CA.

	A	B	C	D	E	F	G	H	I	J	K	L	M
A	1.00	0.09	0.15	0.90	0.94	0.25	0.94	0.24	0.12	0.83	0.27	0.46	0.78
	(0.0, 0.00)	(50.0, 0.31)	(36.0, 0.32)	(33.0, 0.19)	(37.0, 0.24)	(41.0, 0.38)	(16.0, 0.13)	(25.0, 0.22)	(40.0, 0.39)	(38.5, 0.24)	(30.0, 0.23)	(32.0, 0.25)	(33.0, 0.21)
	174	170	155	165	172	169	171	174	173	160	158	169	164
B		1.00	0.86	0.07	0.13	0.31	0.12	0.32	0.29	0.13	0.31	0.35	0.29
		(0.0, 0.00)	(48.0, 0.37)	(47.0, 0.28)	(45.0, 0.27)	(57.0, 0.47)	(49.0, 0.26)	(48.0, 0.33)	(55.0, 0.49)	(51.0, 0.25)	(49.0, 0.35)	(51.0, 0.31)	(44.0, 0.24)
		315	161	167	298	173	176	309	302	172	163	173	168
C			1.00	0.13	0.21	0.36	0.20	0.34	0.36	0.23	0.38	0.50	0.40
			(0.0, 0.00)	(49.0, 0.37)	(58.0, 0.42)	(24.0, 0.31)	(40.0, 0.35)	(27.0, 0.28)	(24.0, 0.30)	(57.5, 0.41)	(24.0, 0.27)	(30.0, 0.25)	(41.0, 0.34)
			170	151	162	156	160	170	168	150	147	159	154
D				1.00	0.93	0.19	0.83	0.11	0.05	0.73	0.13	0.38	0.69
				(0.0, 0.00)	(29.0, 0.17)	(52.0, 0.43)	(23.0, 0.17)	(38.0, 0.27)	(52.0, 0.46)	(26.0, 0.18)	(43.0, 0.30)	(40.0, 0.26)	(24.5, 0.16)
				173	169	167	168	173	172	157	155	165	160
E					1.00	0.23	0.93	0.26	0.16	0.86	0.27	0.57	0.82
					(0.0, 0.00)	(63.0, 0.48)	(27.0, 0.17)	(46.0, 0.33)	(63.5, 0.51)	(18.0, 0.13)	(54.0, 0.36)	(40.0, 0.28)	(26.0, 0.15)
					358	174	179	351	340	175	165	175	171
F						1.00	0.27	0.73	0.32	0.35	0.43	0.44	0.48
						(0.0, 0.00)	(44.0, 0.41)	(28.0, 0.33)	(27.0, 0.32)	(57.0, 0.46)	(24.5, 0.32)	(35.0, 0.35)	(46.0, 0.43)
						177	173	177	176	162	160	170	164
G							1.00	0.27	0.20	0.90	0.35	0.58	0.85
							(0.0, 0.00)	(30.0, 0.25)	(46.5, 0.45)	(25.0, 0.16)	(34.0, 0.27)	(29.0, 0.24)	(24.0, 0.15)
							181	181	180	165	163	174	168
H								1.00	0.26	0.47	0.48	0.40	0.44
								(0.0, 0.00)	(27.0, 0.33)	(45.0, 0.32)	(18.0, 0.18)	(29.0, 0.25)	(34.0, 0.26)
								1060	983	178	172	178	175
I									1.00	0.20	0.45	0.38	0.35
									(0.0, 0.00)	(62.0, 0.51)	(25.0, 0.32)	(41.0, 0.39)	(48.0, 0.46)
									1015	177	172	177	173
J		R								1.00	0.42	0.70	0.85
		(P90, COD)								(0.0, 0.00)	(49.0, 0.35)	(37.0, 0.27)	(20.0, 0.15)
		N								178	155	163	157
K											1.00	0.49	0.48
											(0.0, 0.00)	(30.0, 0.26)	(38.0, 0.29)
											173	162	157
L												1.00	0.84
												(0.0, 0.00)	(24.0, 0.20)
												178	167
M													1.00
													(0.0, 0.00)
													175

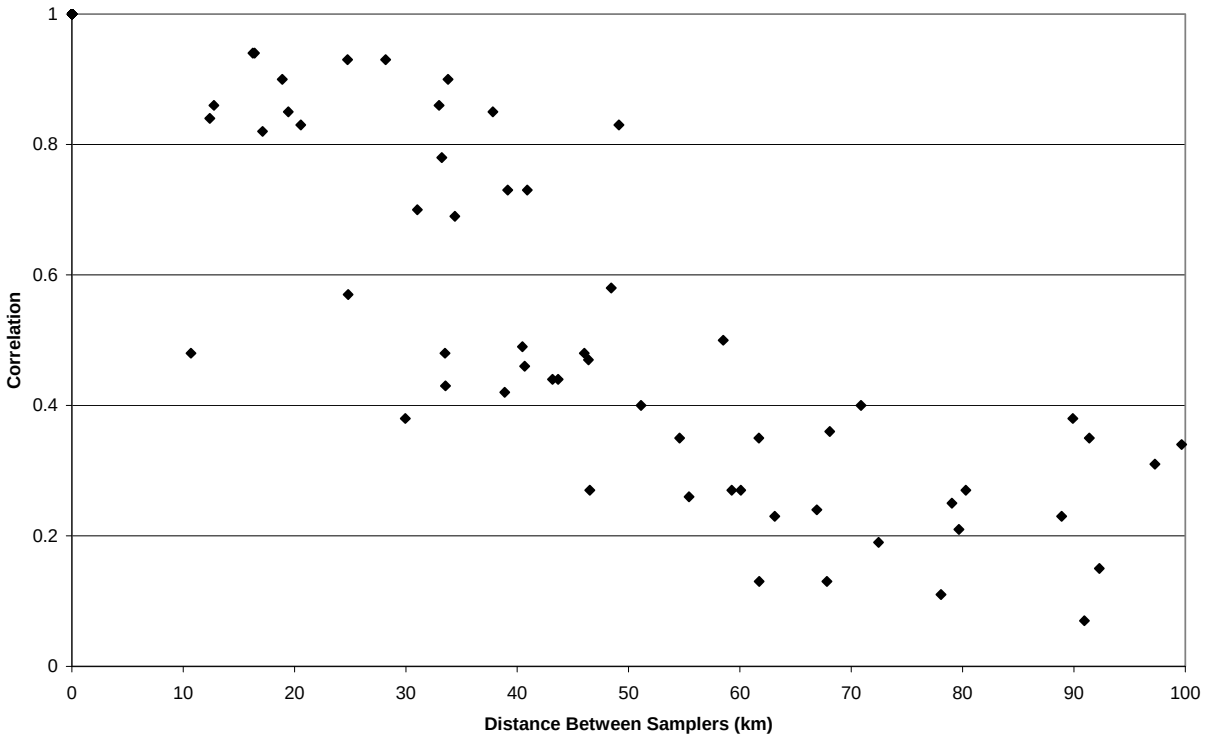

Figure A-98. PM_{10} inter-sampler correlations as a function of distance between monitors for Riverside, CA.

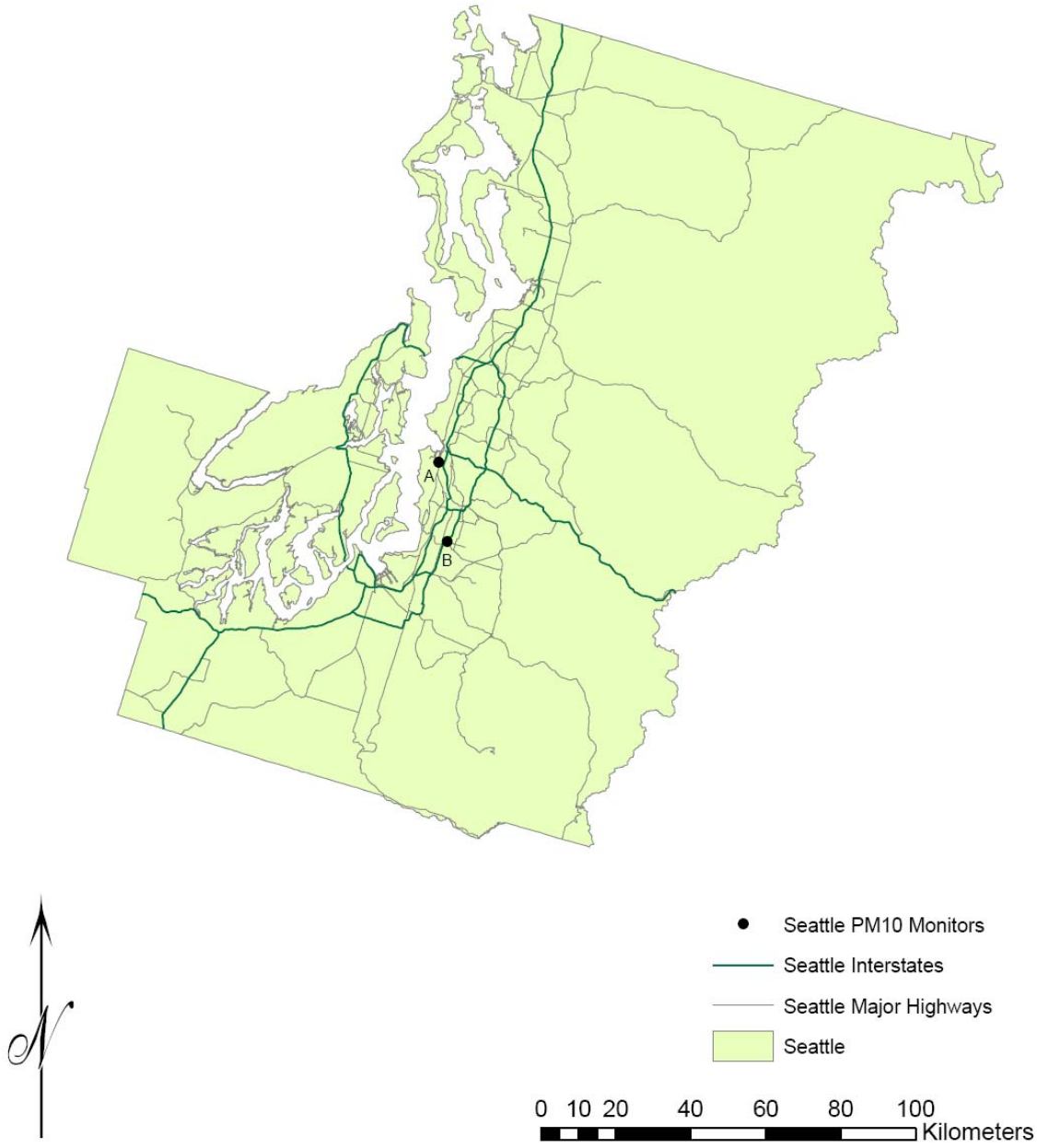

Figure A-99. PM_{10} monitor distribution and major highways, Seattle, WA.

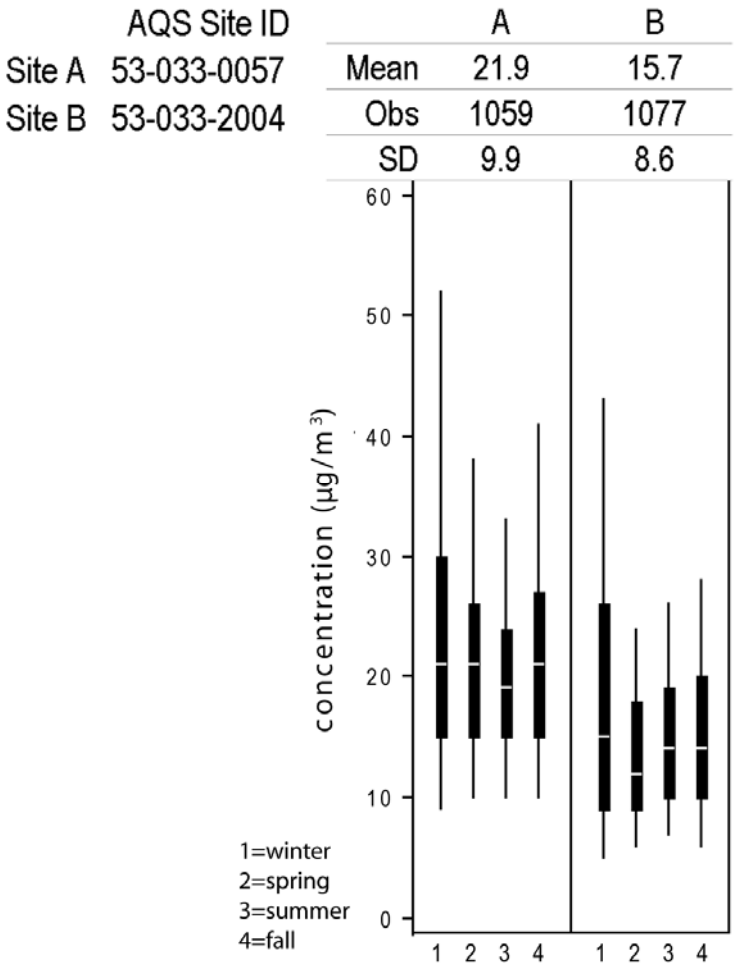

Figure A-100. Box plots illustrating the seasonal distribution of 24-h avg PM_{10} concentrations for Seattle, WA.

Table A-23. Inter-sampler correlation statistics for each pair of PM_{10} monitors reporting to AQS for Seattle, WA.

	A	B
A	1.00	0.77
	(0.0, 0.00)	(14.0, 0.24)
	1059	1041
B		1.00
	R	(0.0, 0.00)
	(P90, COD)	1077
	N	

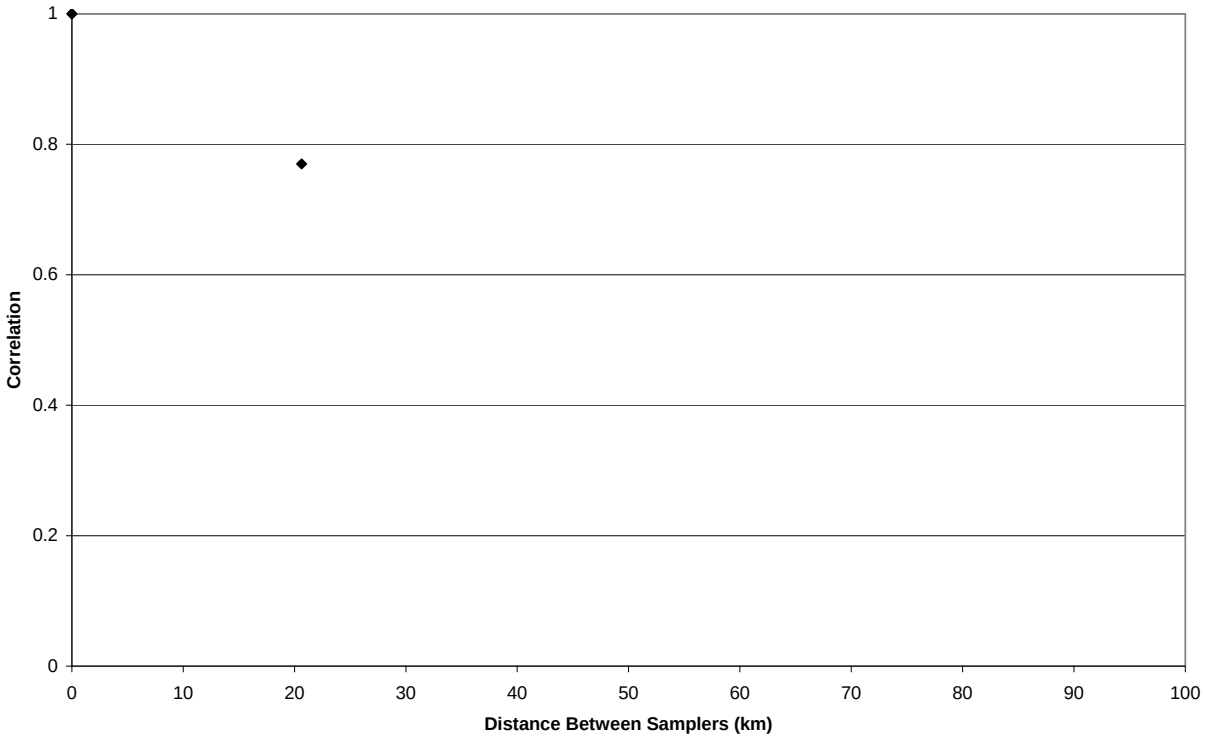

Figure A-101. PM_{10} inter-sampler correlations as a function of distance between monitors for Seattle, WA.

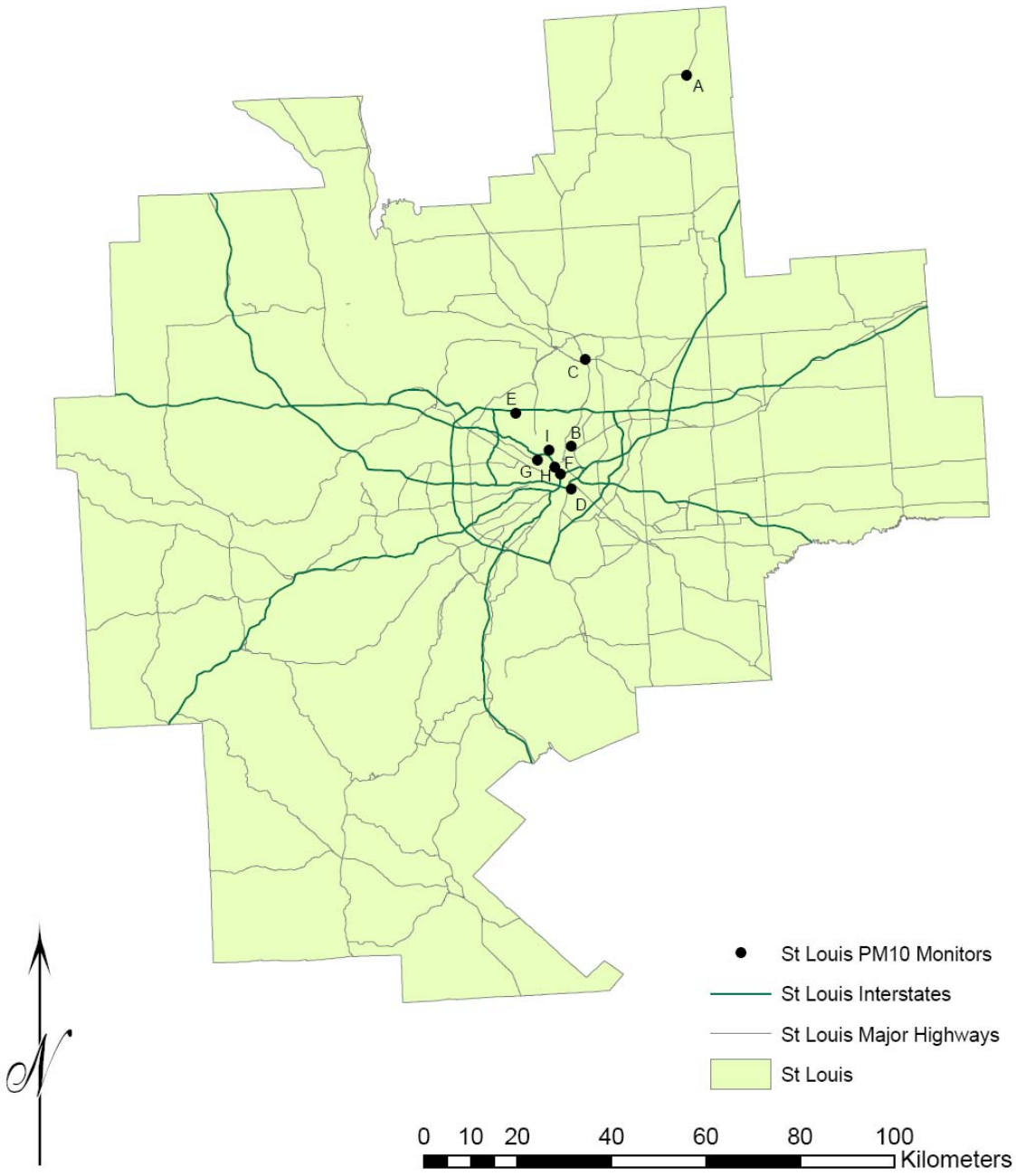

Figure A-102. PM_{10} monitor distribution and major highways, St. Louis, MO.

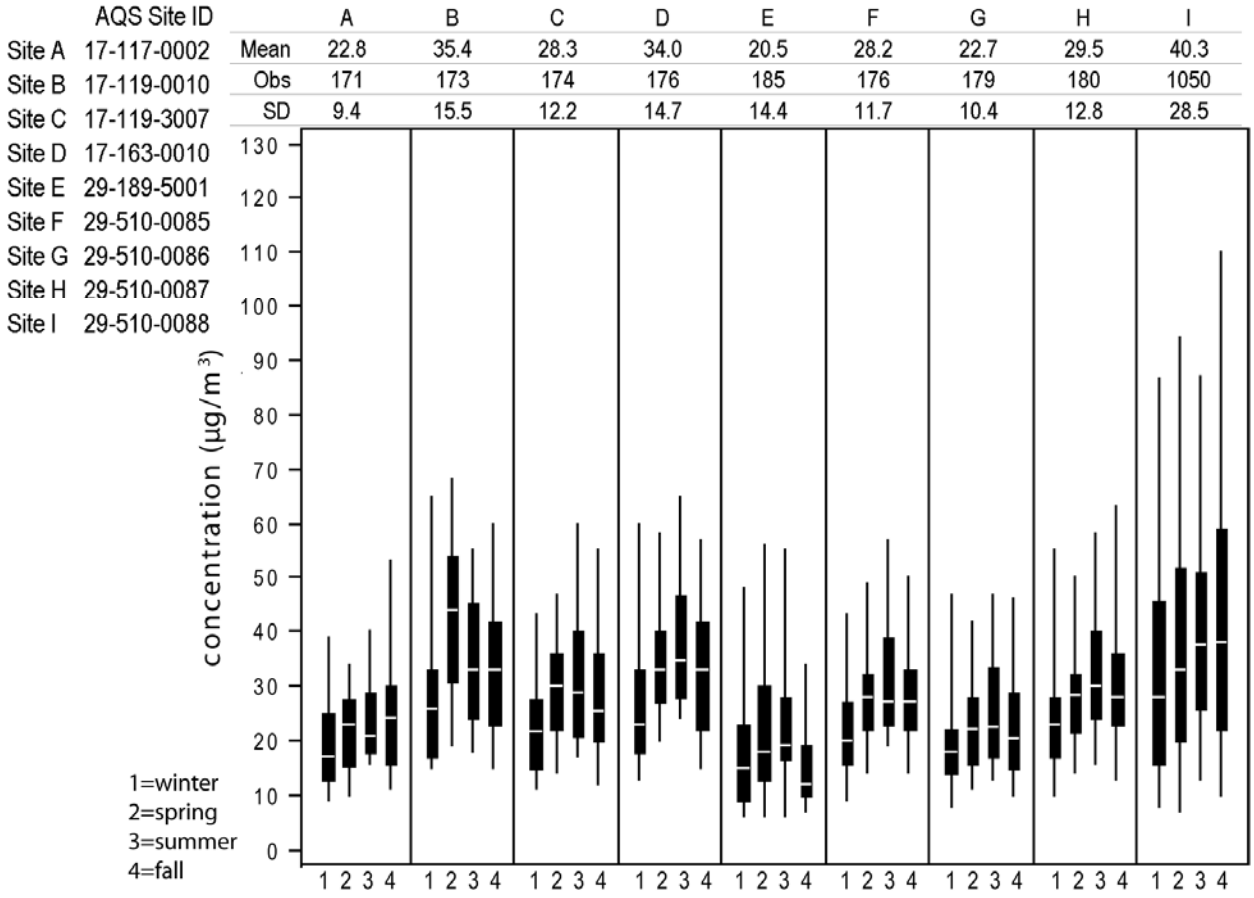

Figure A-103. Box plots illustrating the seasonal distribution of 24-h avg PM_{10} concentrations for St. Louis, MO.

Table A-24. Inter-sampler correlation statistics for each pair of PM_{10} monitors reporting to AQS for St. Louis, MO.

	A	B	C	D	E	F	G	H	I
A	1.00	0.50	0.75	0.67	0.47	0.65	0.67	0.73	0.55
	(0.0, 0.00)	(30.0, 0.28)	(14.0, 0.17)	(23.0, 0.24)	(16.0, 0.29)	(16.0, 0.18)	(13.0, 0.17)	(18.0, 0.19)	(52.0, 0.33)
	171	161	158	156	158	163	166	168	164
B		1.00	0.65	0.63	0.46	0.68	0.68	0.64	0.52
		(0.0, 0.00)	(20.0, 0.21)	(20.0, 0.19)	(37.0, 0.42)	(23.0, 0.20)	(28.0, 0.28)	(22.0, 0.20)	(36.0, 0.28)
		173	161	158	160	167	169	170	166
C			1.00	0.75	0.57	0.80	0.76	0.82	0.65
			(0.0, 0.00)	(17.0, 0.17)	(23.0, 0.33)	(12.0, 0.13)	(13.0, 0.18)	(12.0, 0.13)	(41.0, 0.27)
			174	157	158	165	169	169	168
D				1.00	0.44	0.82	0.81	0.80	0.59
				(0.0, 0.00)	(30.0, 0.40)	(16.0, 0.15)	(21.0, 0.24)	(14.0, 0.15)	(36.0, 0.27)
				176	157	163	165	166	169
E					1.00	0.53	0.62	0.56	0.34
					(0.0, 0.00)	(22.0, 0.34)	(17.0, 0.26)	(25.0, 0.35)	(55.0, 0.42)
					185	164	166	167	179
F						1.00	0.89	0.86	0.67
						(0.0, 0.00)	(11.0, 0.16)	(12.0, 0.11)	(41.0, 0.27)
		R				176	173	174	169
G		(P90, COD)					1.00	0.83	0.65
		N					(0.0, 0.00)	(16.0, 0.19)	(47.0, 0.32)
							179	177	173
H								1.00	0.64
								(0.0, 0.00)	(41.0, 0.27)
								180	173
I									1.00
									(0.0, 0.00)
									1050

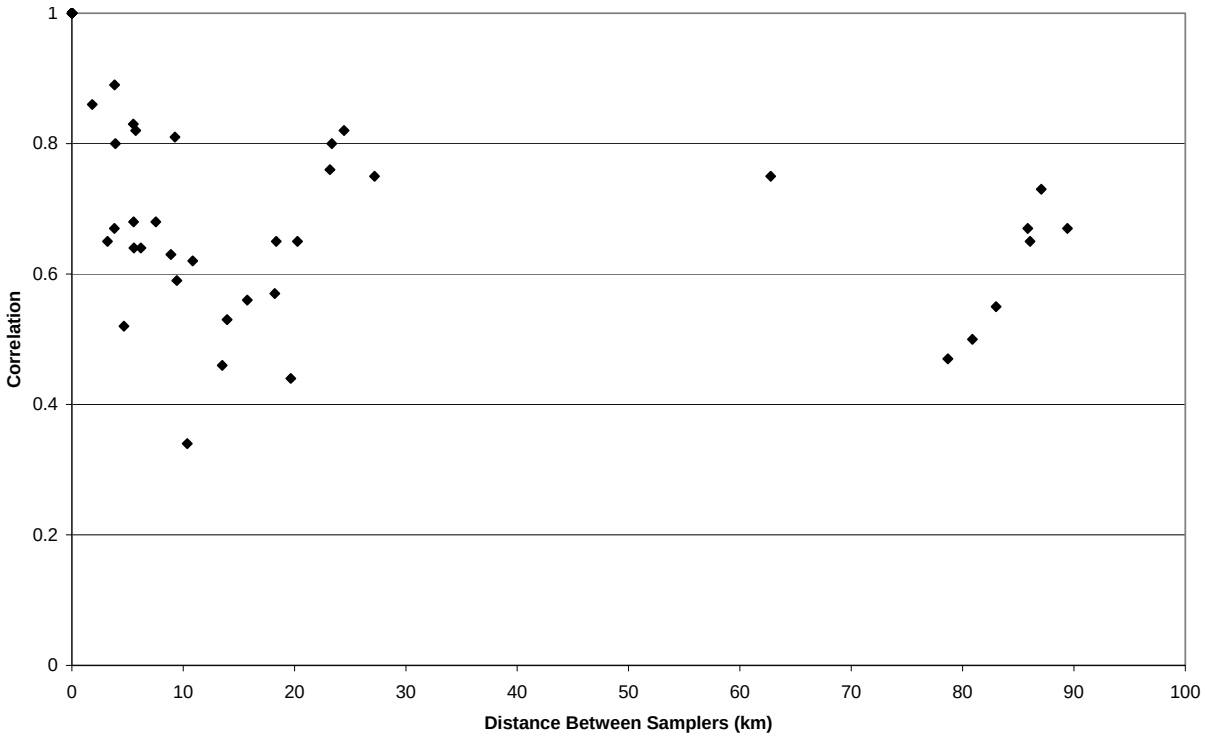

Figure A-104. PM_{10} inter-sampler correlations as a function of distance between monitors for St. Louis, MO.

Table A-25. Correlation coefficients of hourly and daily average particle number, surface and volume concentrations in selected particle size ranges.

Size range (nm)	Hourly averages					Daily avg
	All days (N = 5481)	Sundays (N = 701)	Weekdays (N = 3227)	Event days (N = 577)	No events (N = 4904)	All days (N = 263)
3–10	0.40	0.24	0.42	0.73	0.37	0.32
10–30	0.35	0.22	0.31	0.57	0.33	0.27
30–50	0.38	0.42	0.29	0.56	0.36	0.36
50–100	0.46	0.56	0.39	0.57	0.45	0.46
100–500	0.55	0.65	0.49	0.62	0.55	0.55
500–800	0.73	0.75	0.70	0.76	0.72	0.71
10–100	0.31	0.28	0.24	0.52	0.29	0.24
10–800	0.55	0.65	0.49	0.62	0.55	0.55
Total number	0.30	0.24	0.24	0.58	0.28	0.20
Total surface	0.57	0.63	0.51	0.65	0.56	0.57
Total volume	0.66	0.69	0.62	0.73	0.65	0.67

Source: Tuch et al. (2006)

A.2.3. Speciation

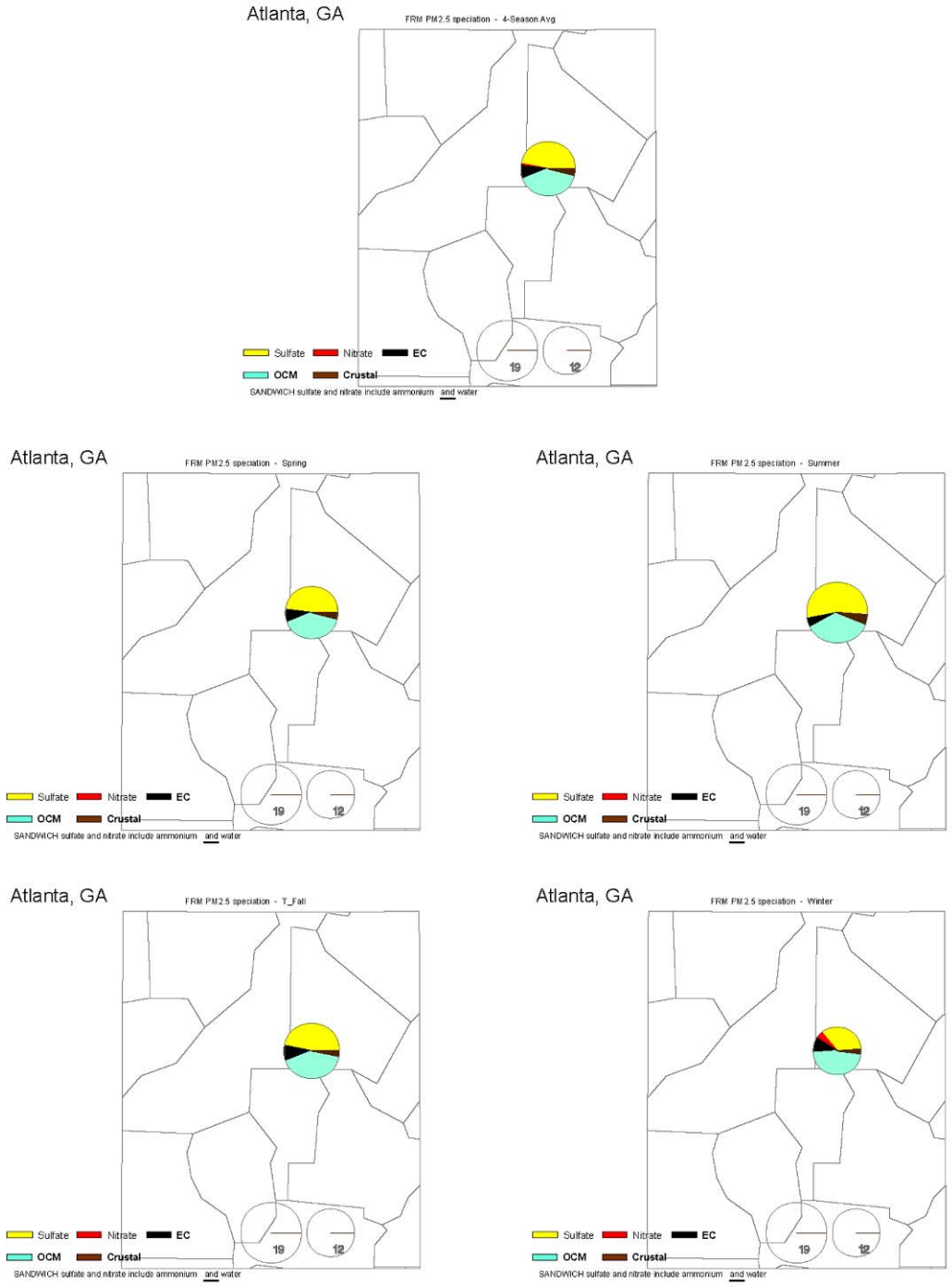

Figure A-105. Seasonally averaged $PM_{2.5}$ speciation data for 2005-2007 for a) annual, b) winter, c) spring, d) summer and e) fall derived using the SANDWICH method in Atlanta, GA.

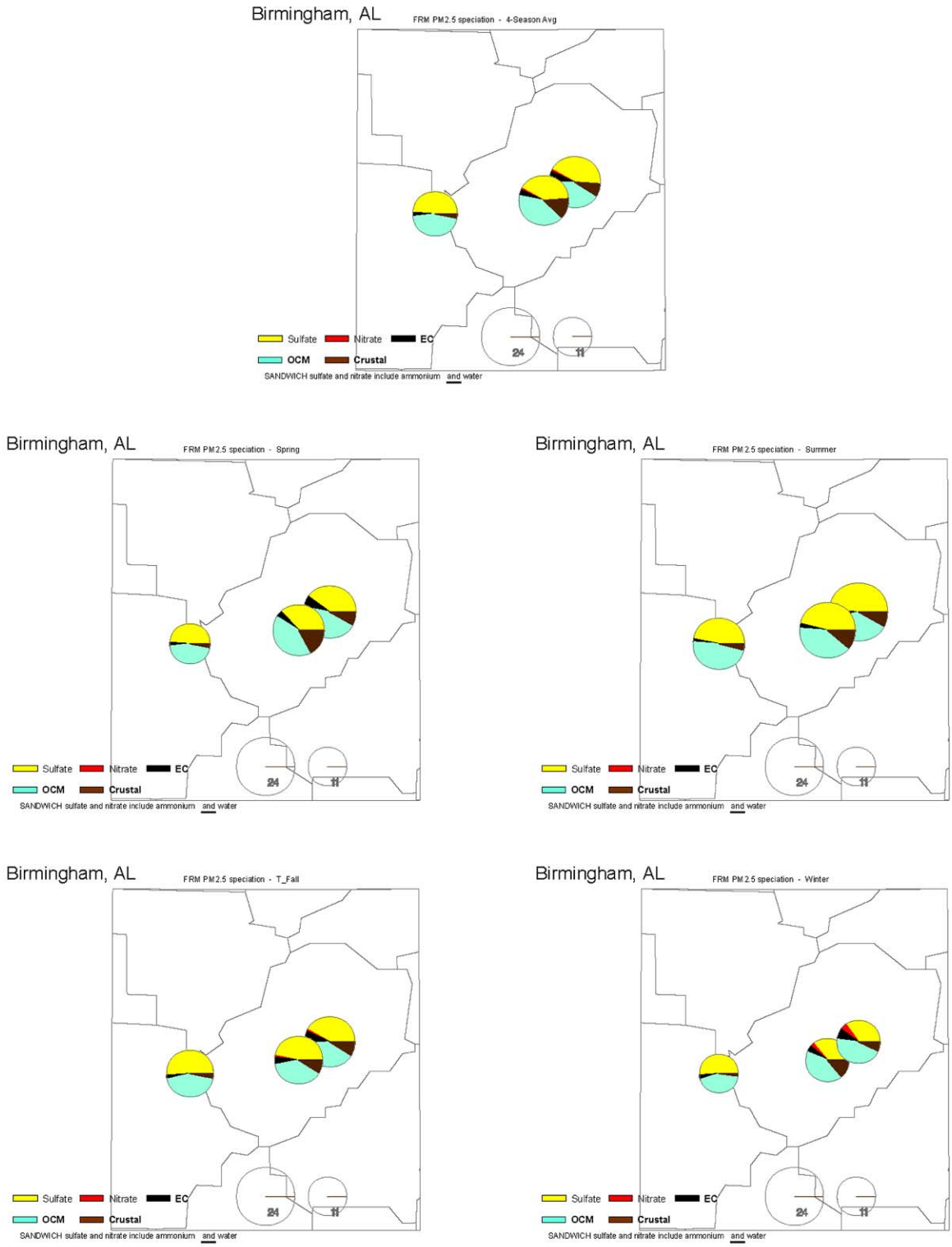

Figure A-106. Seasonally averaged $PM_{2.5}$ speciation data for 2005-2007 for a) annual, b) winter, c) spring, d) summer and e) fall derived using the SANDWICH method in Birmingham, AL.

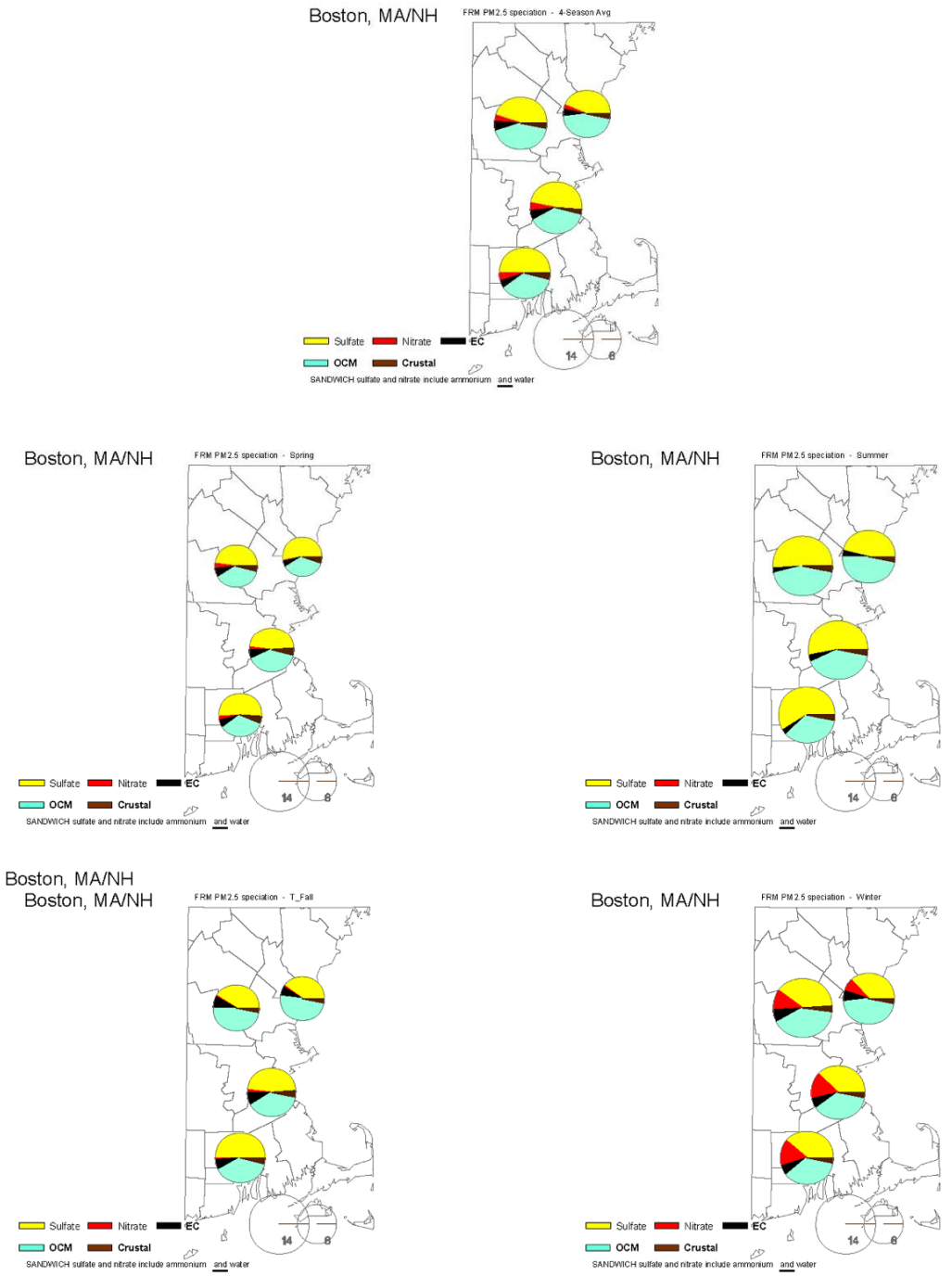

Figure A-107. Seasonally averaged $PM_{2.5}$ speciation data for 2005-2007 for a) annual, b) winter, c) spring, d) summer and e) fall derived using the SANDWICH method in Boston, MA.

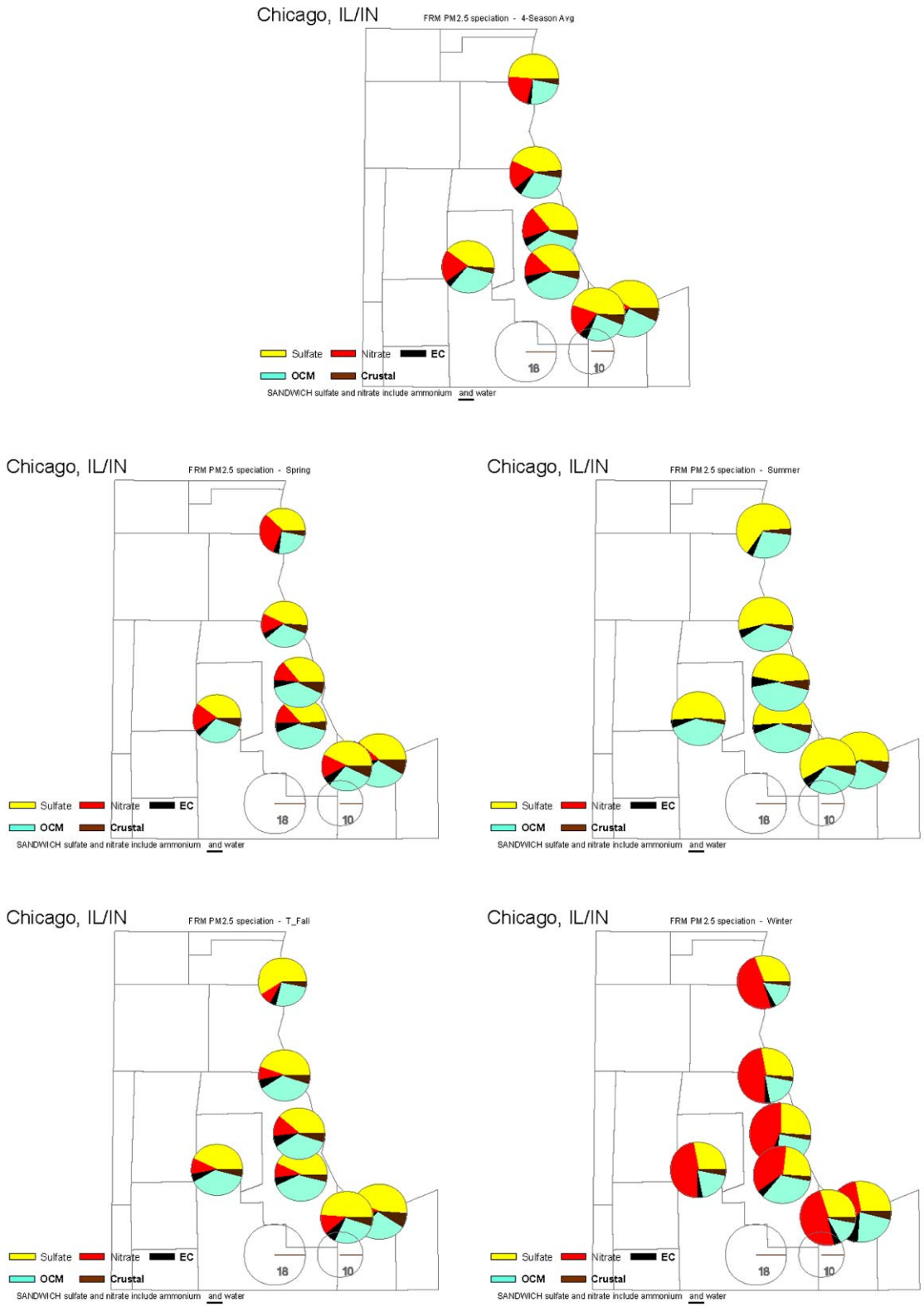

Figure A-108. Seasonally averaged $PM_{2.5}$ speciation data for 2005-2007 for a) annual, b) winter, c) spring, d) summer and e) fall derived using the SANDWICH method in Chicago, IL.

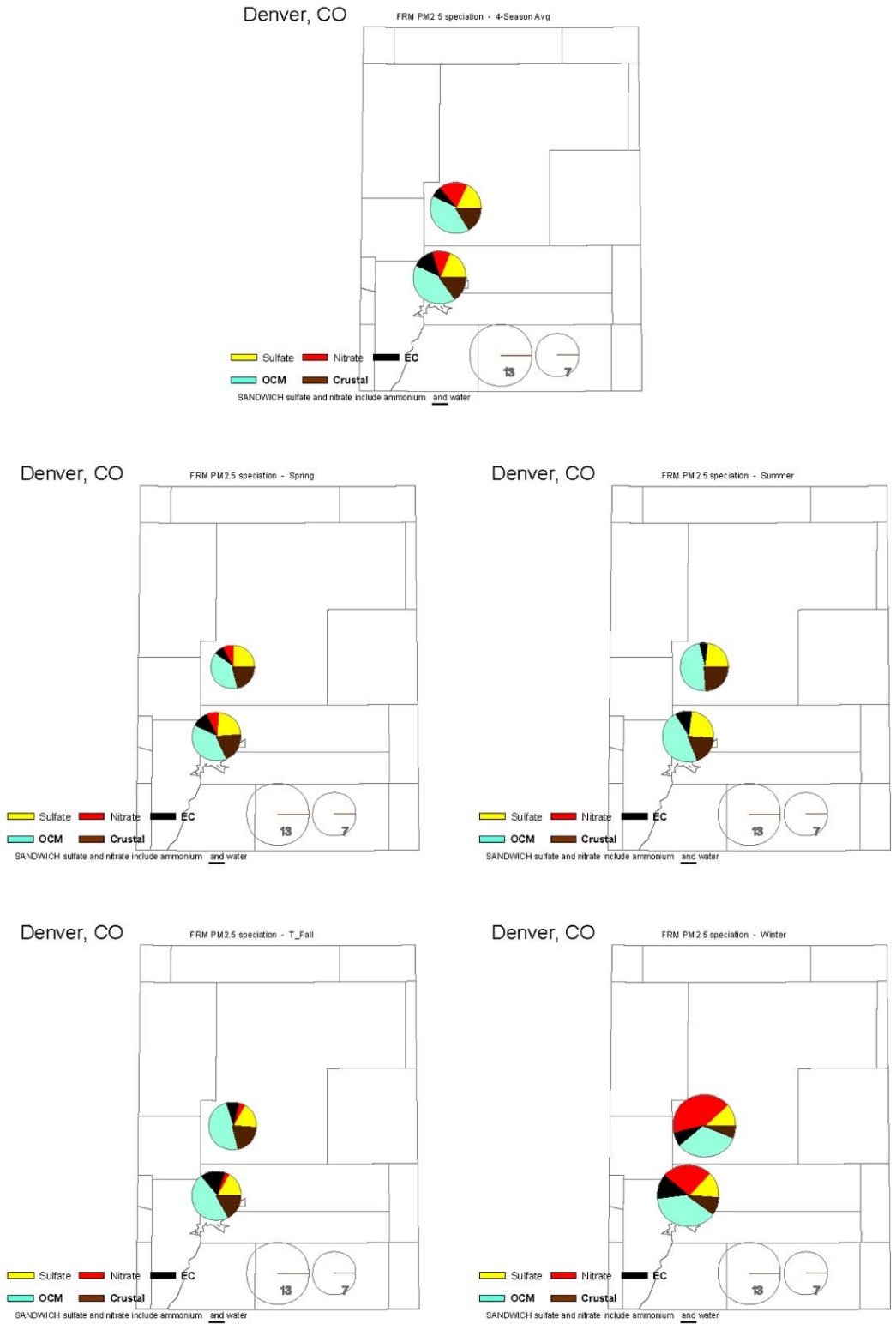

Figure A-109. Seasonally averaged $PM_{2.5}$ speciation data for 2005-2007 for a) annual, b) winter, c) spring, d) summer and e) fall derived using the SANDWICH method in Denver, CO.

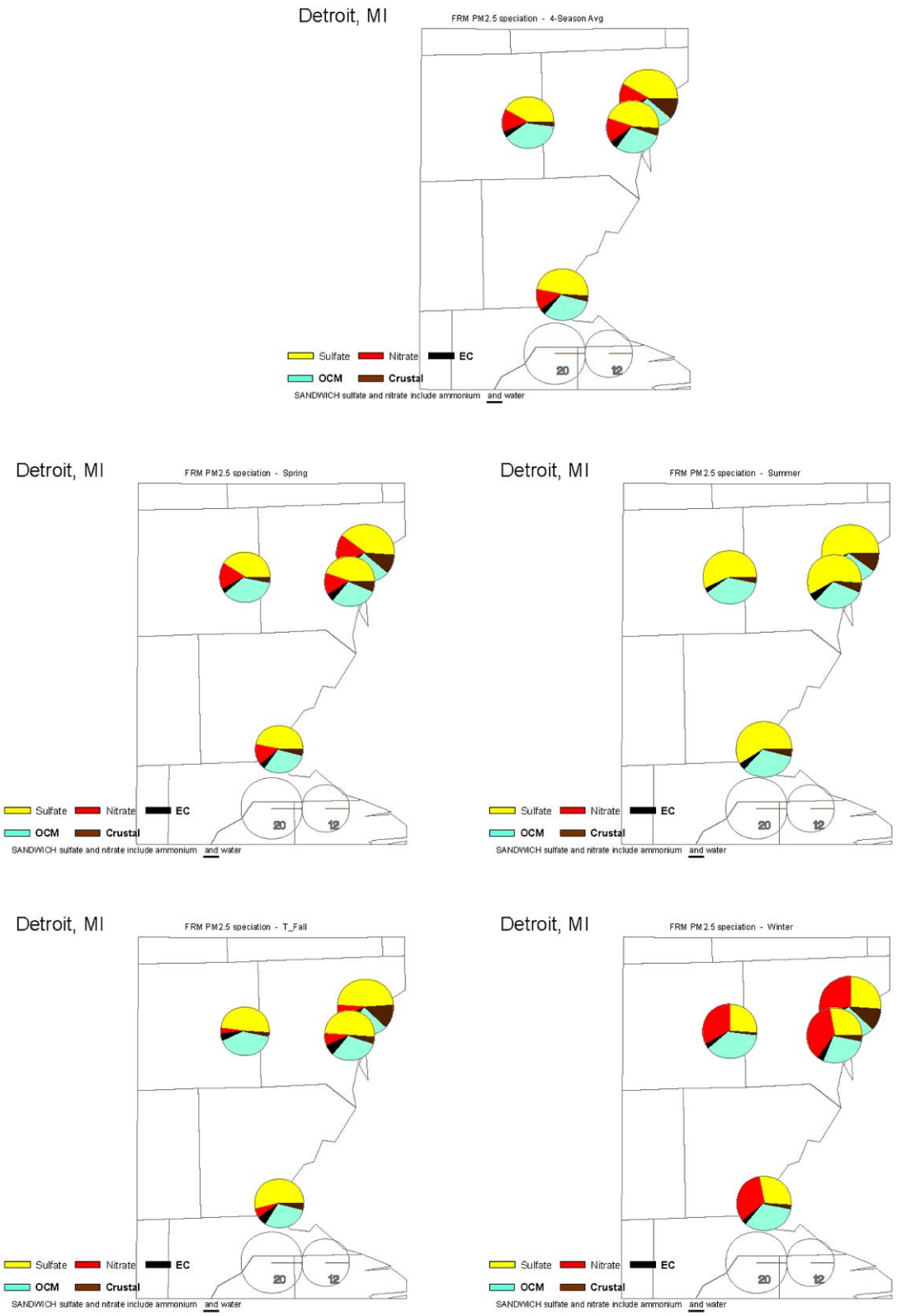

Figure A-110. Seasonally averaged $PM_{2.5}$ speciation data for 2005-2007 for a) annual, b) winter, c) spring, d) summer and e) fall derived using the SANDWICH method in Detroit, MI.

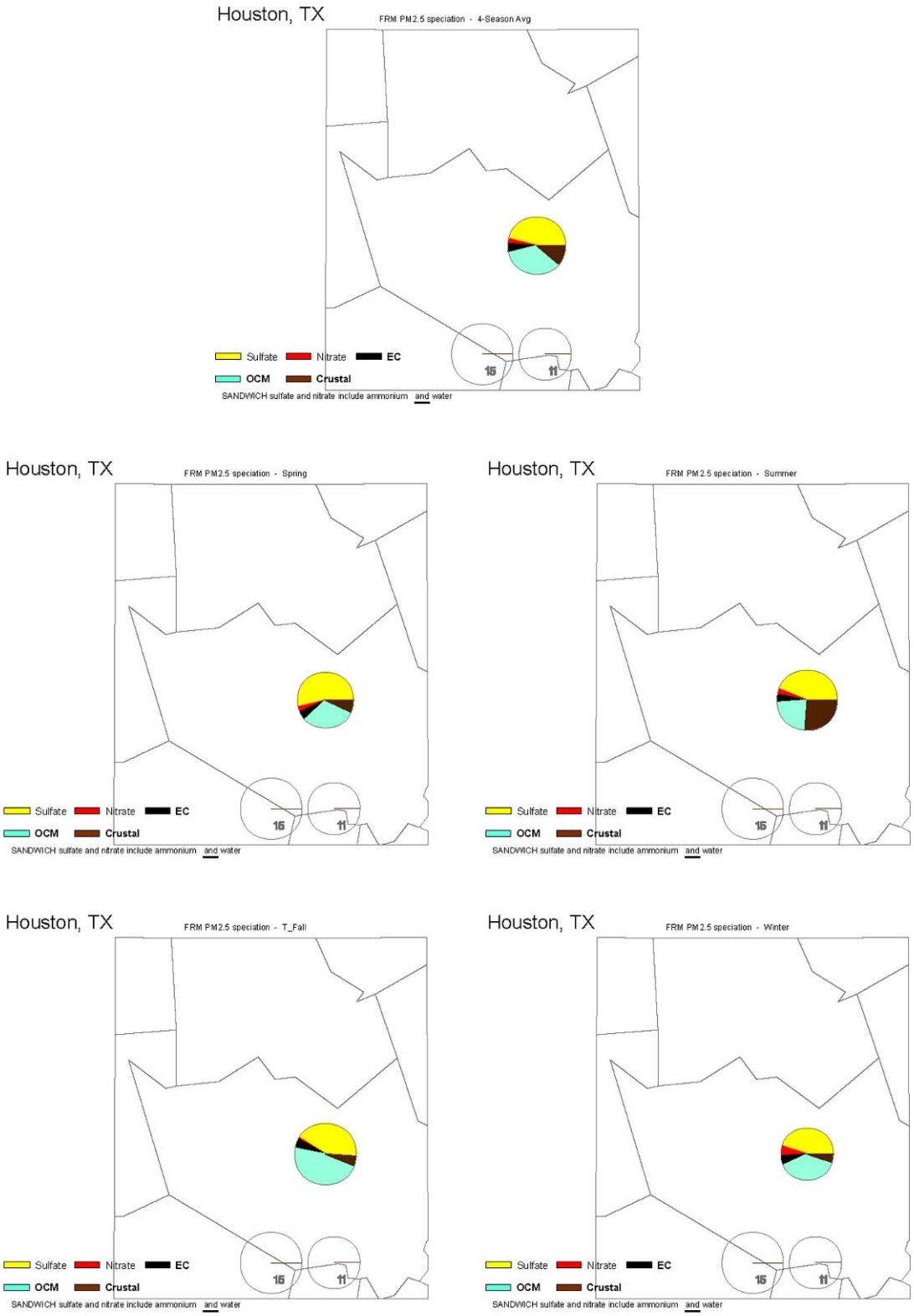

Figure A-111. Seasonally averaged $PM_{2.5}$ speciation data for 2005-2007 for a) annual, b) winter, c) spring, d) summer and e) fall derived using the SANDWICH method in Houston, TX.

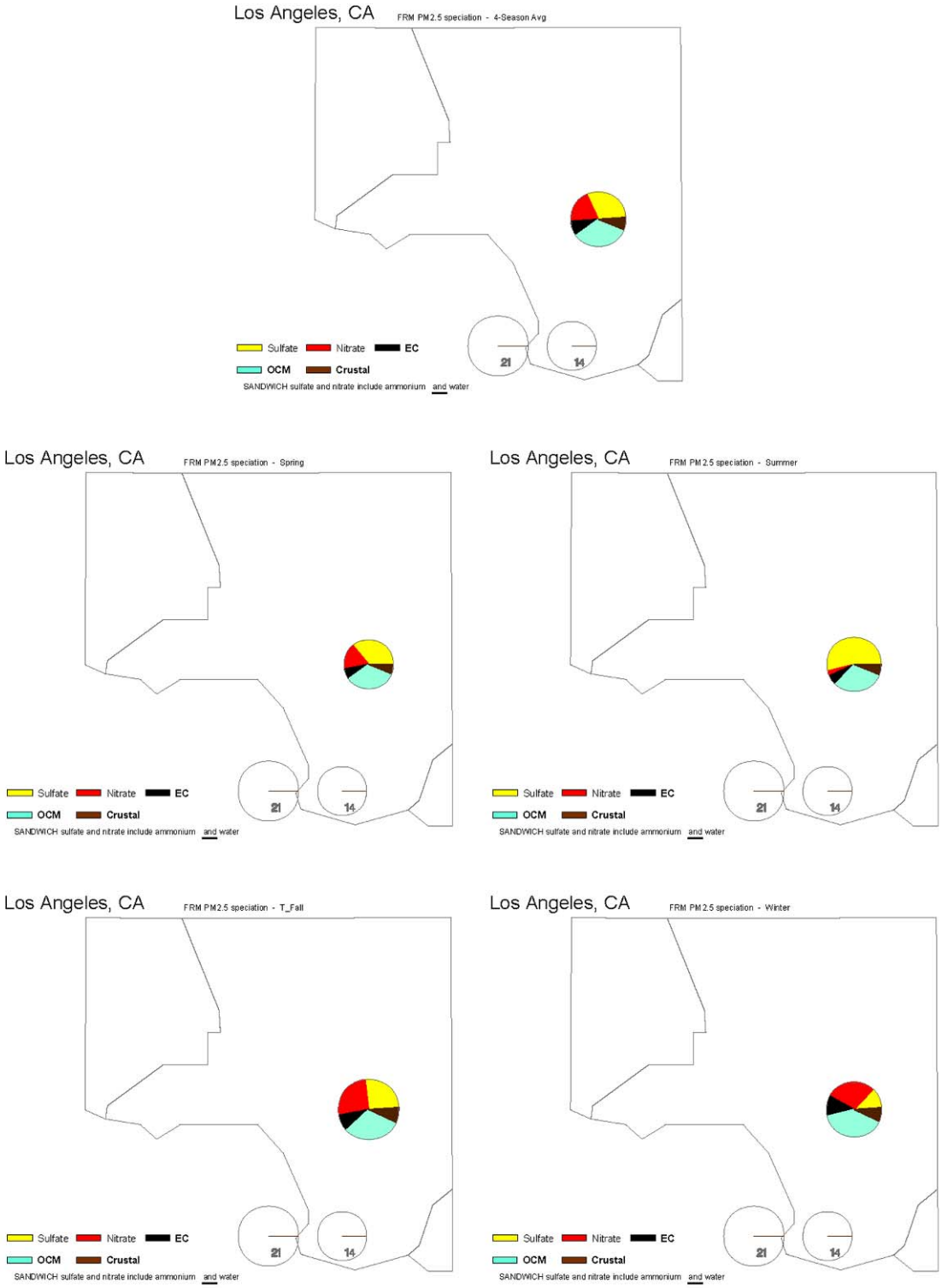

Figure A-112. Seasonally averaged $PM_{2.5}$ speciation data for 2005-2007 for a) annual, b) winter, c) spring, d) summer and e) fall derived using the SANDWICH method in Los Angeles, CA.

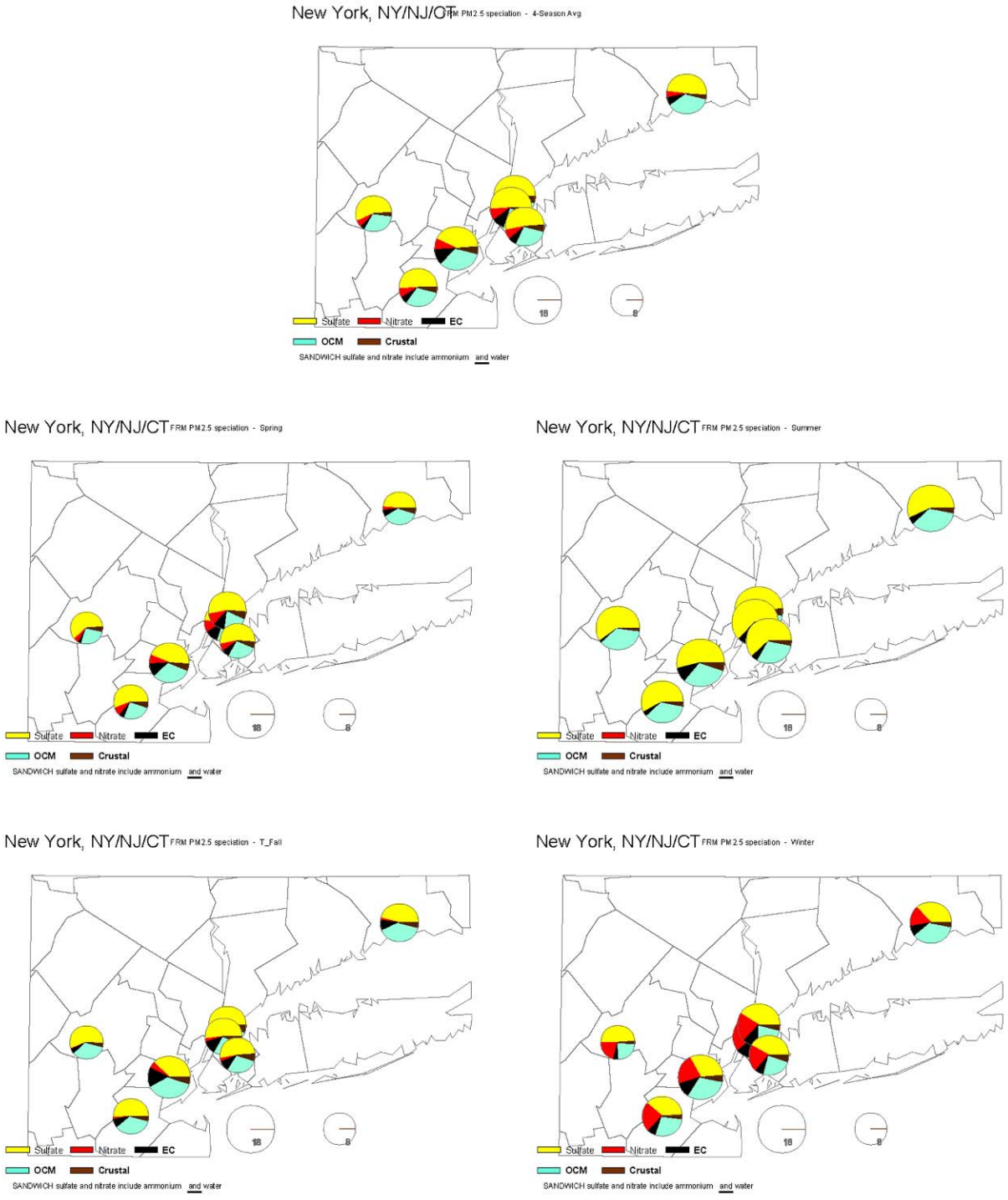

Figure A-113. Seasonally averaged $PM_{2.5}$ speciation data for 2005-2007 for a) annual, b) winter, c) spring, d) summer and e) fall derived using the SANDWICH method in New York City, NY.

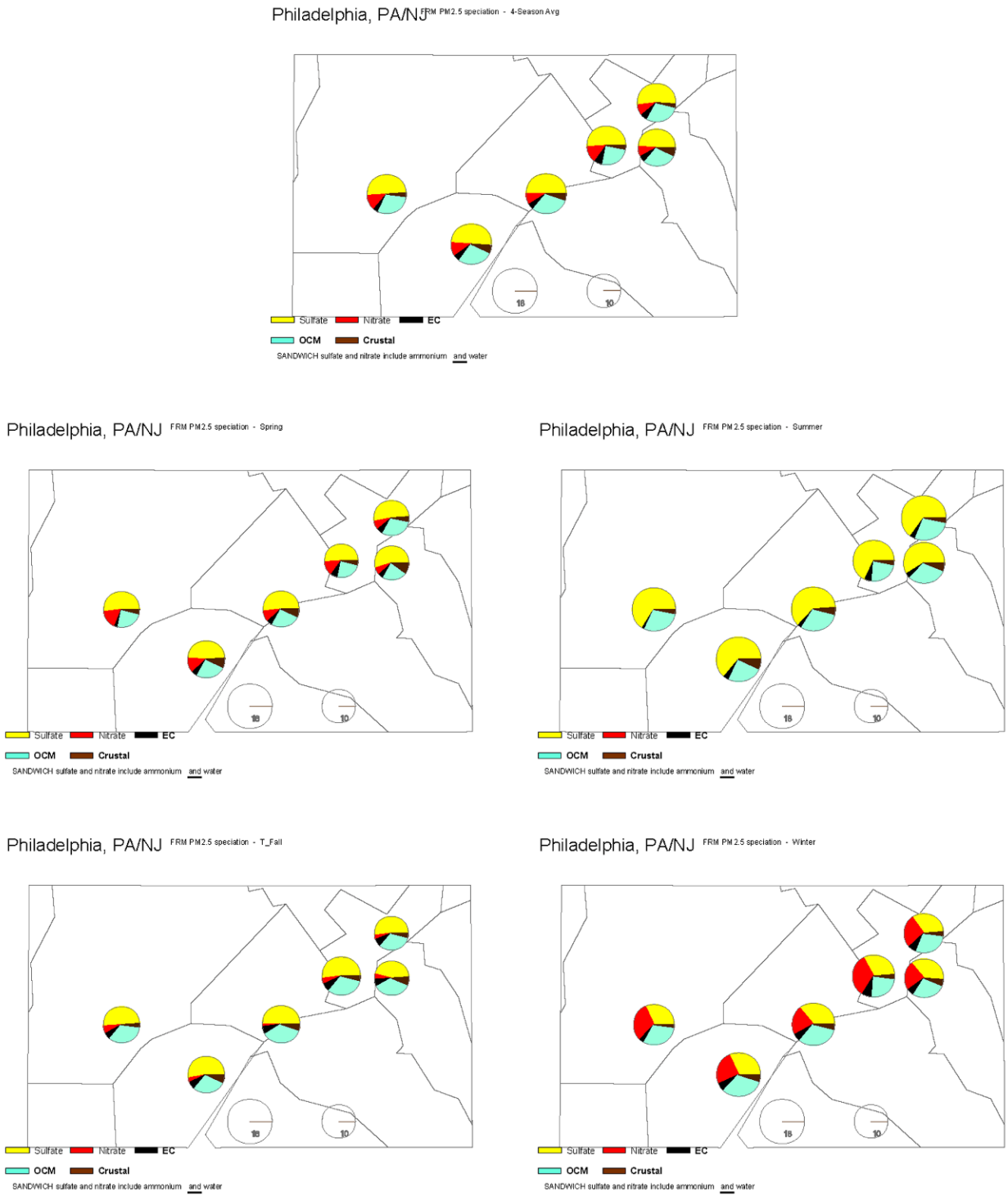

Figure A-114. Seasonally averaged $PM_{2.5}$ speciation data for 2005-2007 for a) annual, b) winter, c) spring, d) summer and e) fall derived using the SANDWICH method in Philadelphia.

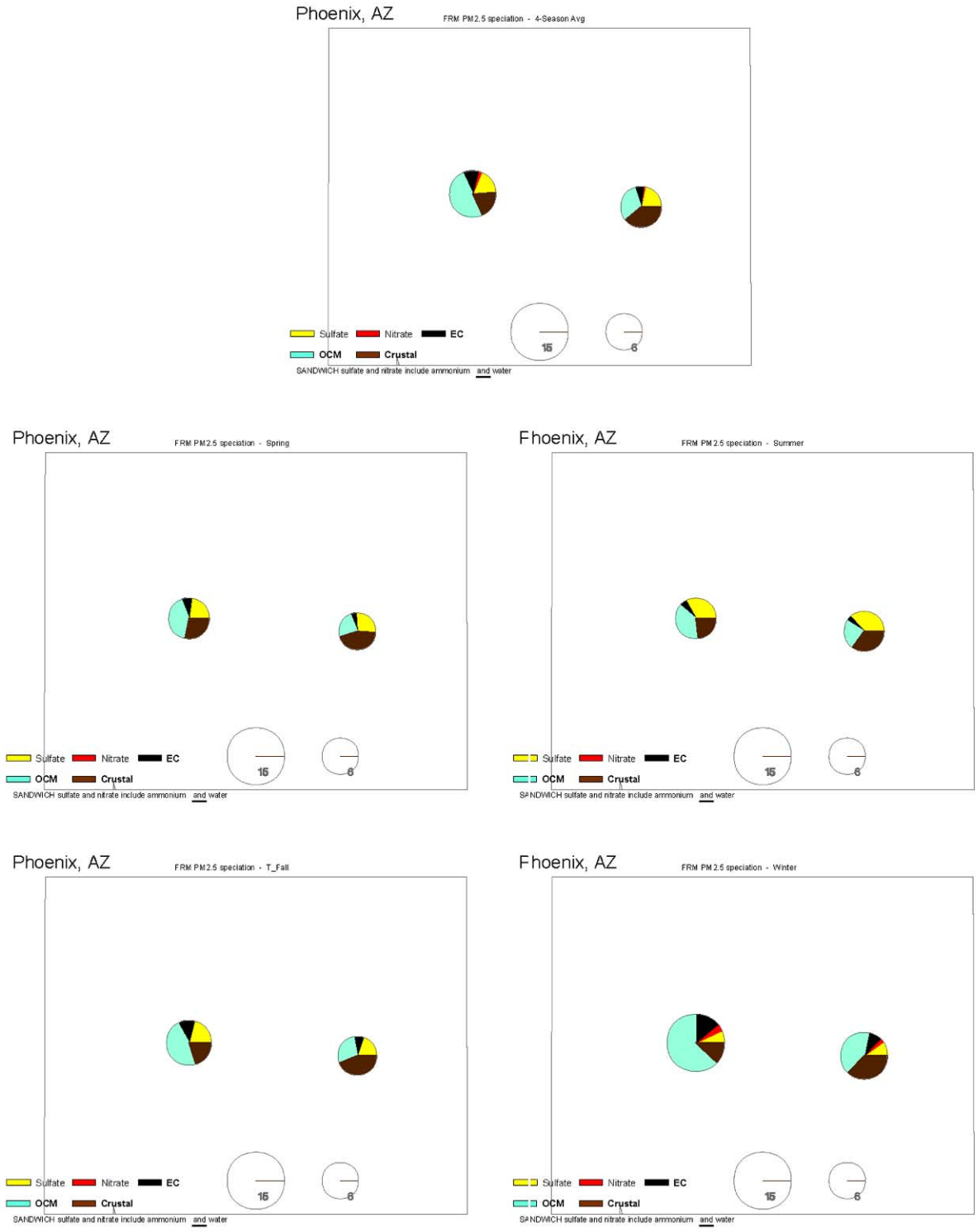

Figure A-115. Seasonally averaged $PM_{2.5}$ speciation data for 2005-2007 for a) annual, b) winter, c) spring, d) summer and e) fall derived using the SANDWICH method in Phoenix, AZ.

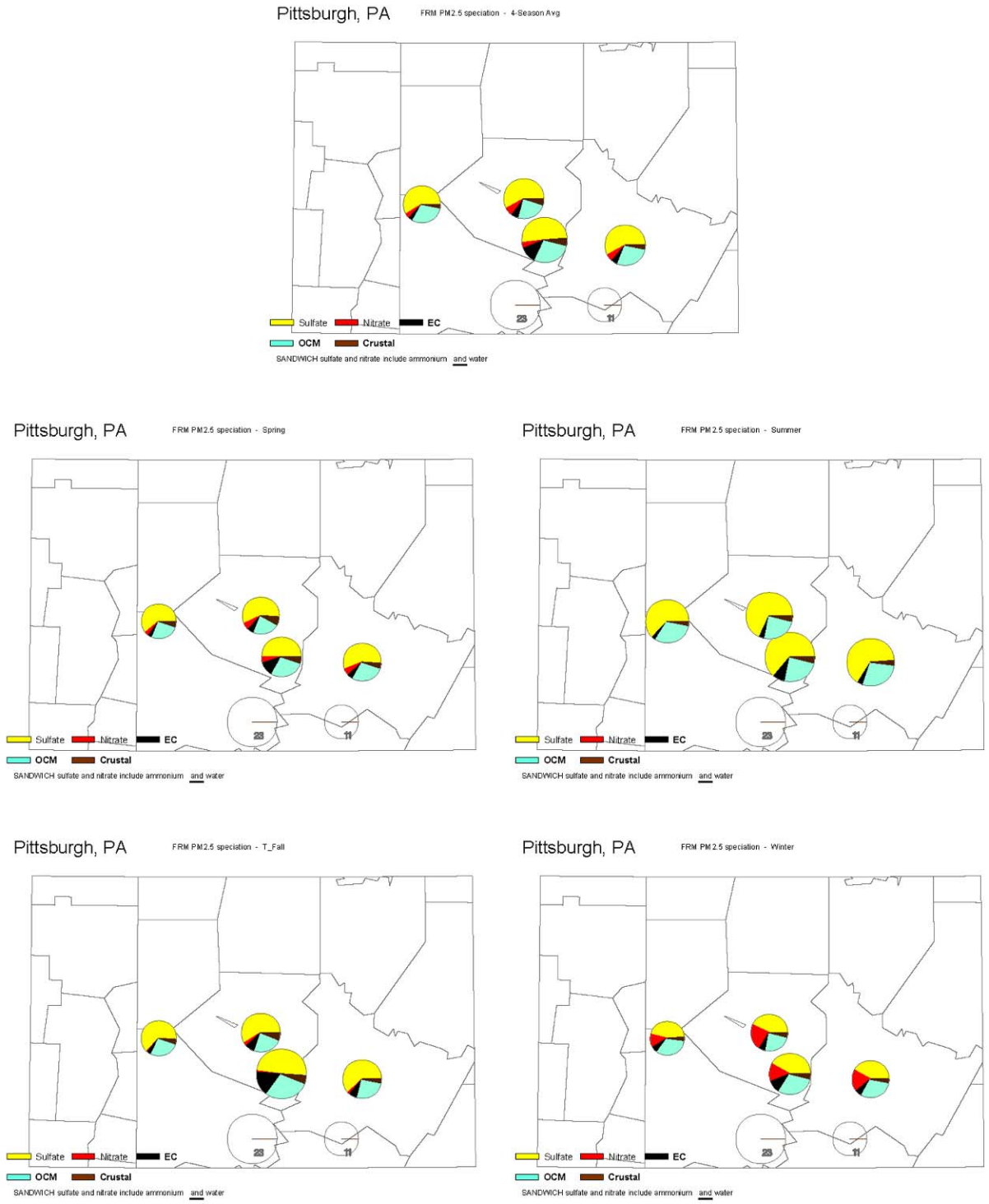

Figure A-116. Seasonally averaged $PM_{2.5}$ speciation data for 2005-2007 for a) annual, b) winter, c) spring, d) summer and e) fall derived using the SANDWICH method in Pittsburgh, PA.

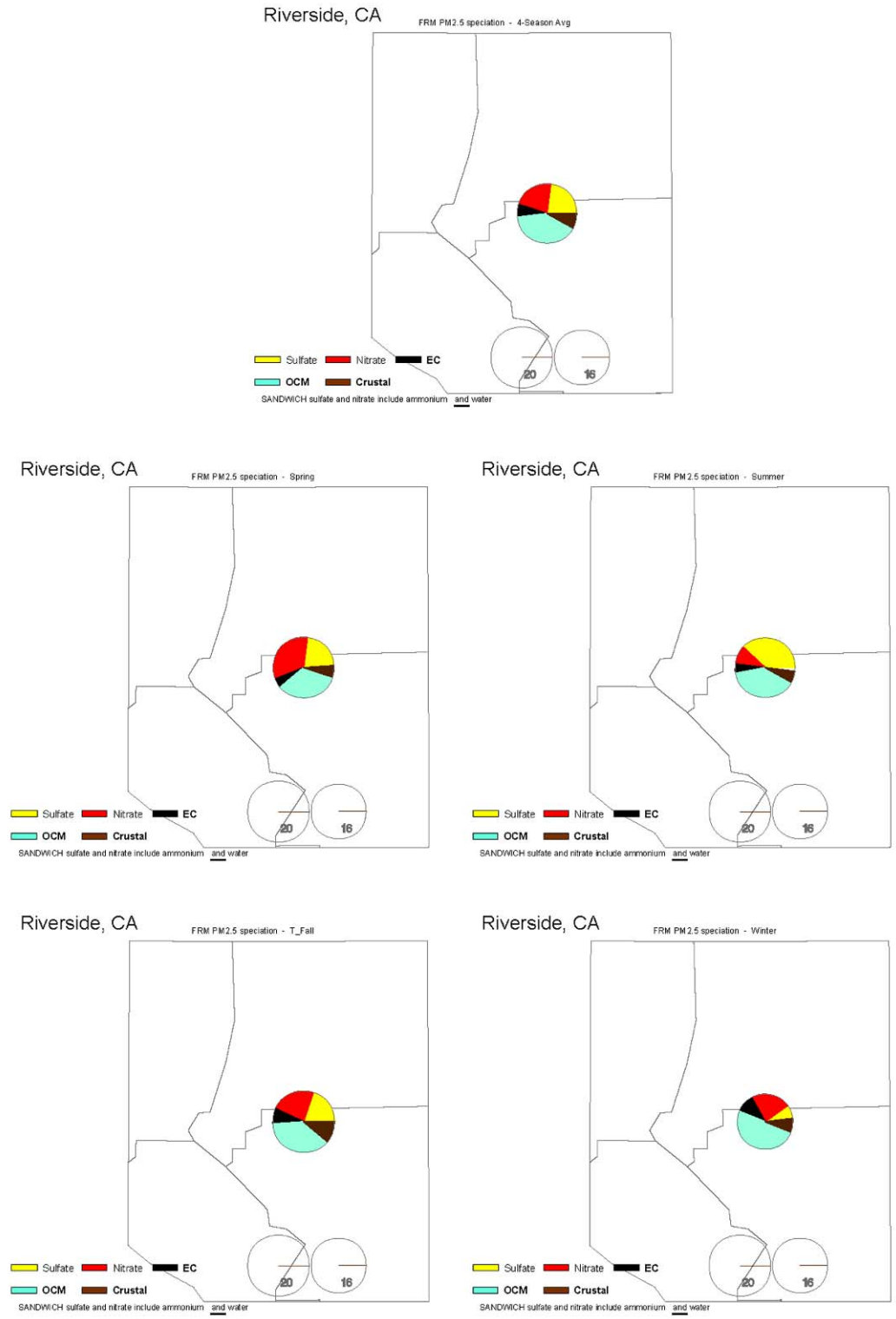

Figure A-117. Seasonally averaged $PM_{2.5}$ speciation data for 2005-2007 for a) annual, b) winter, c) spring, d) summer and e) fall derived using the SANDWICH method in Riverside, CA.

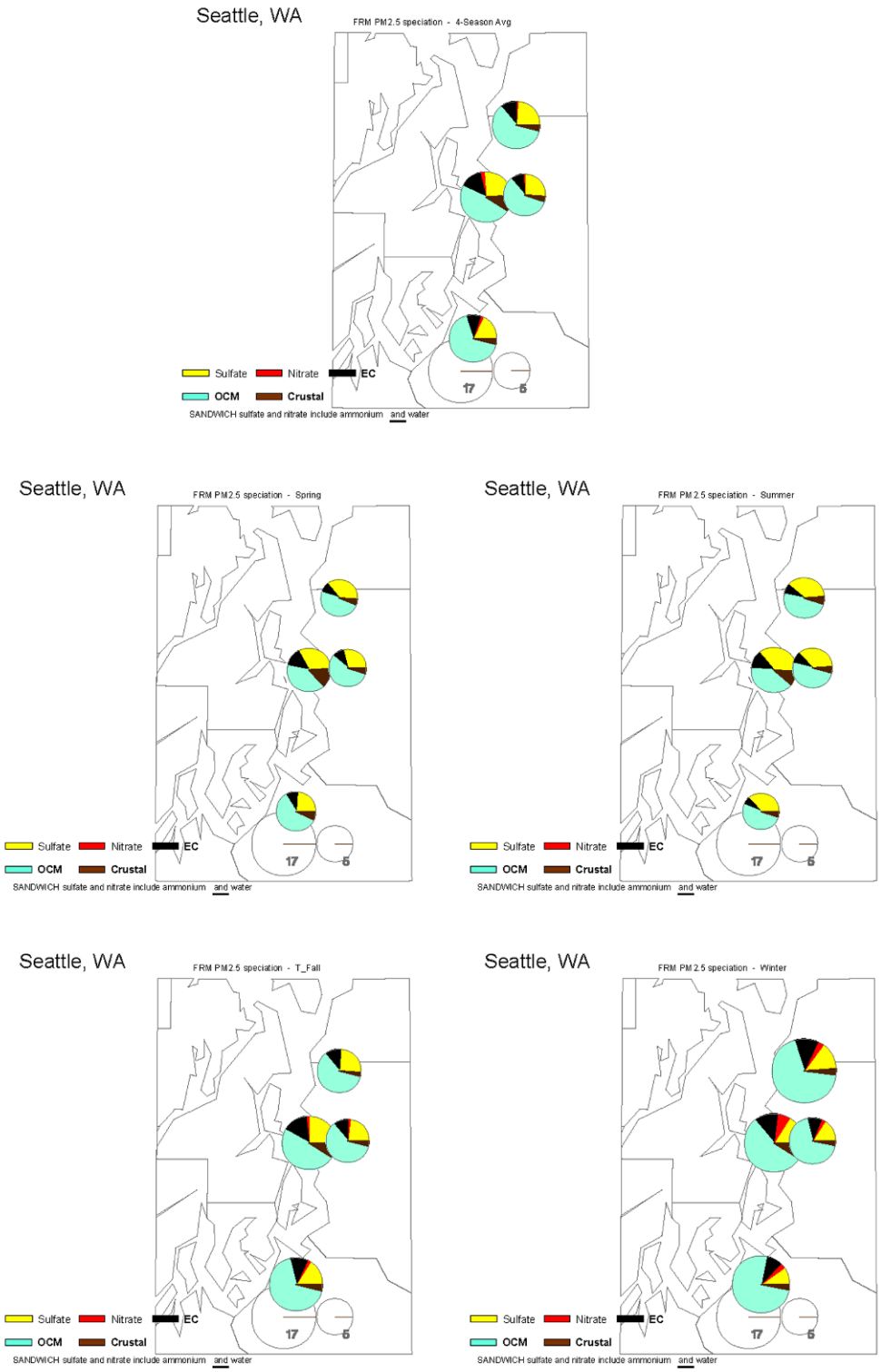

Figure A-118. Seasonally averaged $PM_{2.5}$ speciation data for 2005-2007 for a) annual, b) winter, c) spring, d) summer and e) fall derived using the SANDWICH method in Seattle, WA.

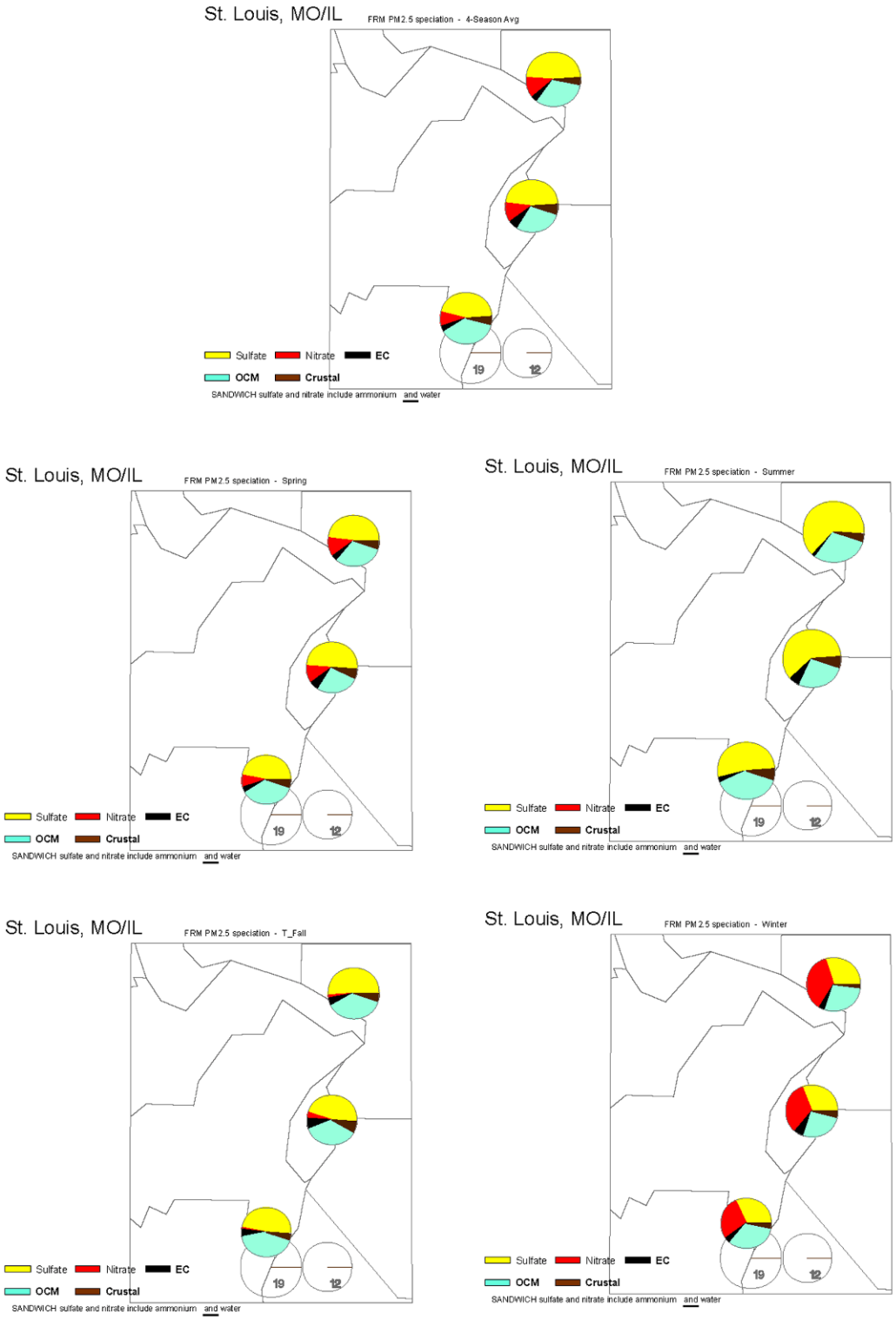

Figure A-119. Seasonally averaged $PM_{2.5}$ speciation data for 2005-2007 for a) annual, b) winter, c) spring, d) summer and e) fall derived using the SANDWICH method in St. Louis, MO.

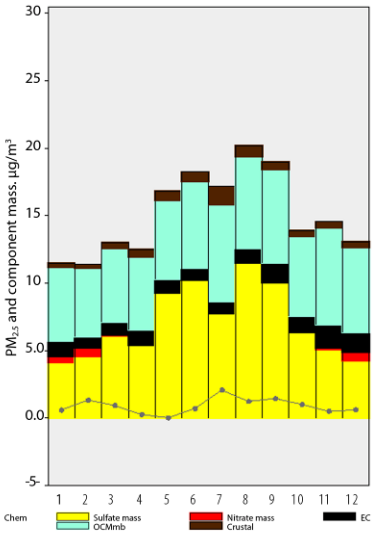

Figure A-120. Seasonal patterns in $PM_{2.5}$ chemical composition from city-wide monthly average values for Atlanta, GA, 2005-2007. The gray line represents the difference in OCM calculated using material balance and blank corrected OC x 1.4.

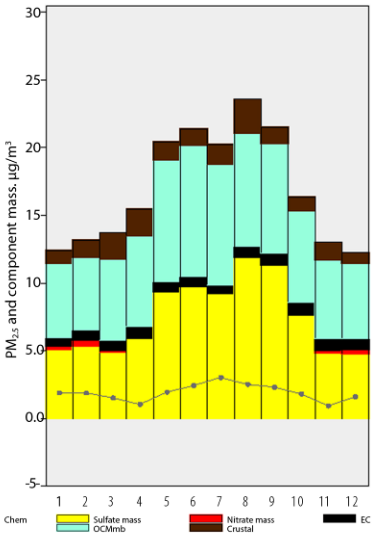

Figure A-121. Seasonal patterns in $PM_{2.5}$ chemical composition from city-wide monthly average values for Birmingham, AL, 2005-2007. The gray line represents the difference in OCM calculated using material balance and blank corrected OC x 1.4.

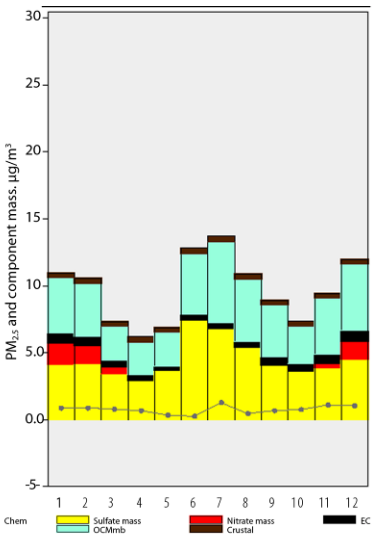

Figure A-122. Seasonal patterns in $PM_{2.5}$ chemical composition from city-wide monthly average values for Boston, MA, 2005-2007. The gray line represents the difference in OCM calculated using material balance and blank corrected OC x 1.4.

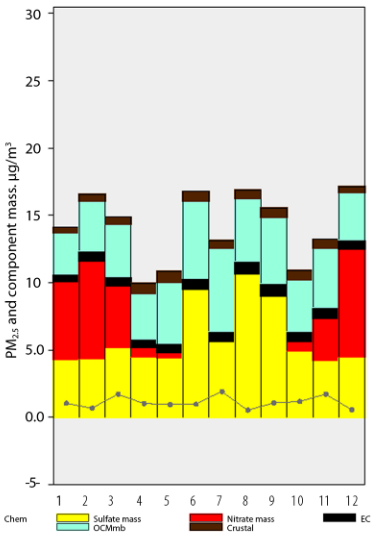

Figure A-123. Seasonal patterns in $PM_{2.5}$ chemical composition from city-wide monthly average values for Chicago, IL, 2005-2007. The gray line represents the difference in OCM calculated using material balance and blank corrected OC x 1.4.

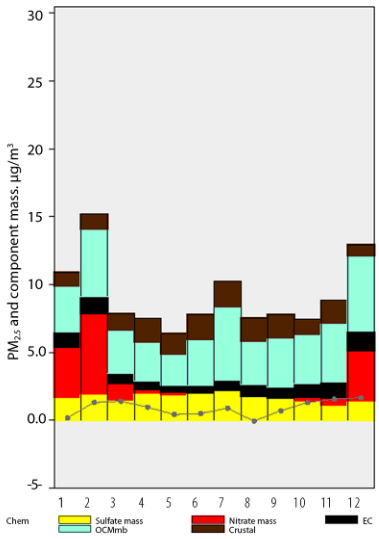

Figure A-124. Seasonal patterns in $PM_{2.5}$ chemical composition from city-wide monthly average values for Denver, CO, 2005-2007. The gray line represents the difference in OCM calculated using material balance and blank corrected OC x 1.4.

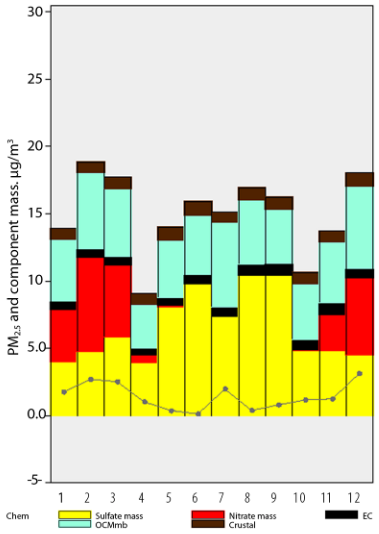

Figure A-125. Seasonal patterns in $PM_{2.5}$ chemical composition from city-wide monthly average values for Detroit, MI, 2005-2007. The gray line represents the difference in OCM calculated using material balance and blank corrected OC x 1.4.

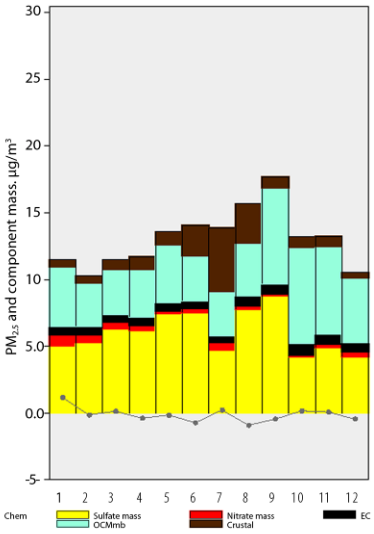

Figure A-126. Seasonal patterns in $PM_{2.5}$ chemical composition from city-wide monthly average values for Houston, TX, 2005-2007. The gray line represents the difference in OCM calculated using material balance and blank corrected OC x 1.4.

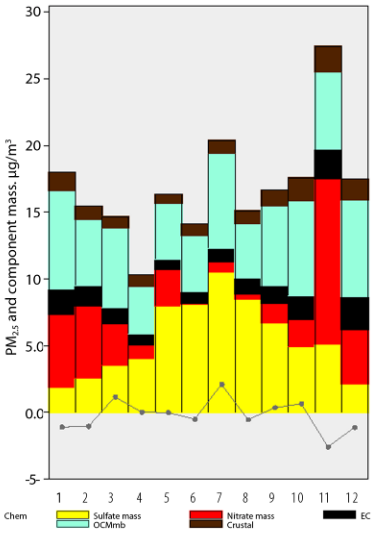

Figure A-127. Seasonal patterns in $PM_{2.5}$ chemical composition from city-wide monthly average values for Los Angeles, CA, 2005-2007. The gray line represents the difference in OCM calculated using material balance and blank corrected OC x 1.4.

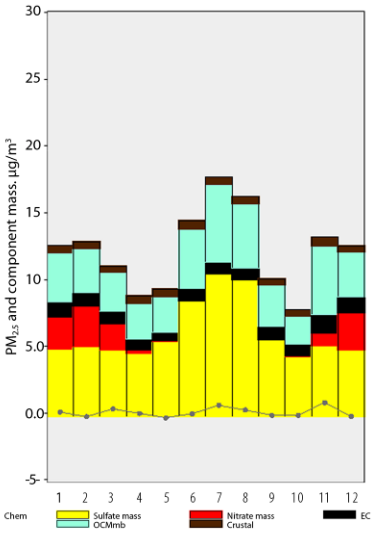

Figure A-128. Seasonal patterns in $PM_{2.5}$ chemical composition from city-wide monthly average values for New York City, NY, 2005-2007. The gray line represents the difference in OCM calculated using material balance and blank corrected OC x 1.4.

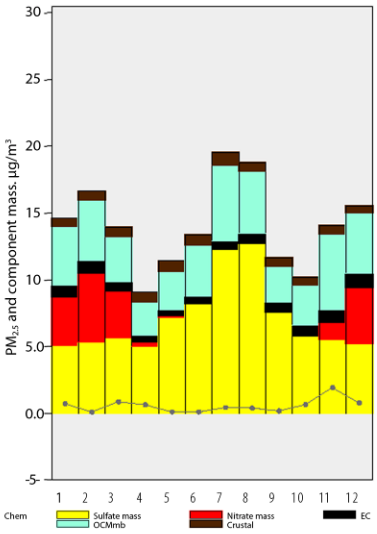

Figure A-129. Seasonal patterns in $PM_{2.5}$ chemical composition from city-wide monthly average values for Philadelphia, PA, 2005-2007. The gray line represents the difference in OCM calculated using material balance and blank corrected OC x 1.4.

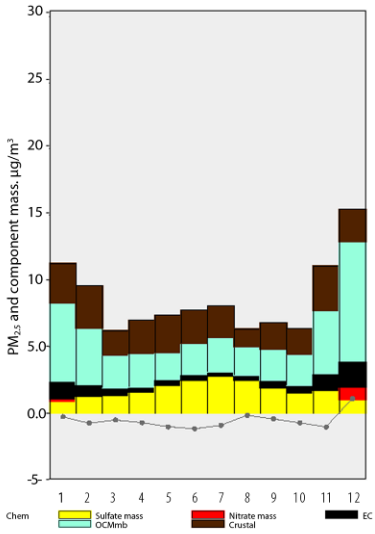

Figure A-130. Seasonal patterns in $PM_{2.5}$ chemical composition from city-wide monthly average values for Phoenix, AZ, 2005-2007. The gray line represents the difference in OCM calculated using material balance and blank corrected OC x 1.4.

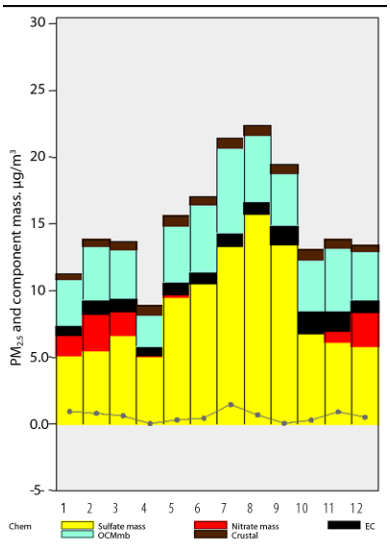

Figure A-131. Seasonal patterns in $PM_{2.5}$ chemical composition from city-wide monthly average values for Pittsburgh, PA, 2005-2007. The gray line represents the difference in OCM calculated using material balance and blank corrected OC x 1.4.

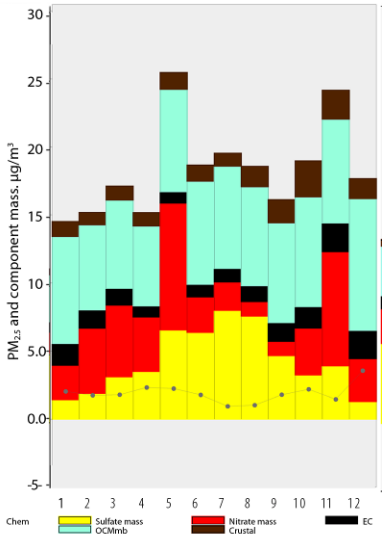

Figure A-132. Seasonal patterns in $PM_{2.5}$ chemical composition from city-wide monthly average values for Riverside, CA, 2005-2007. The gray line represents the difference in OCM calculated using material balance and blank corrected OC x 1.4.

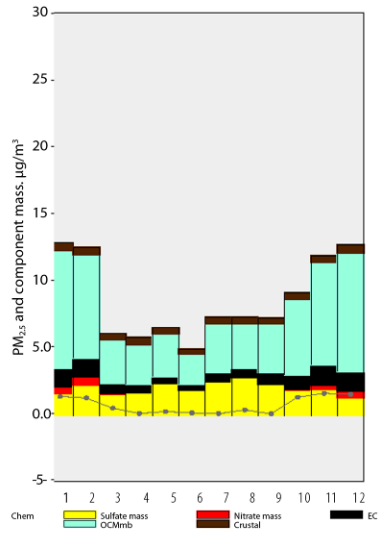

Figure A-133. Seasonal patterns in $PM_{2.5}$ chemical composition from city-wide monthly average values for Seattle, WA, 2005-2007. The gray line represents the difference in OCM calculated using material balance and blank corrected OC x 1.4.

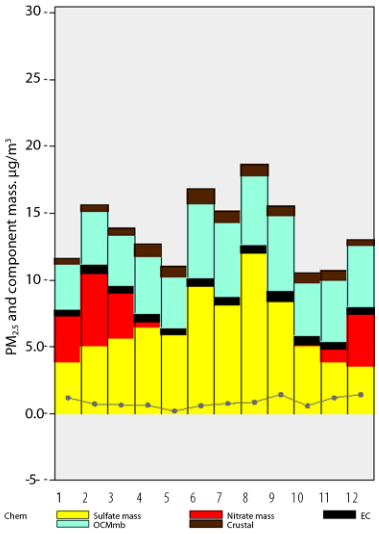

Figure A-134. Seasonal patterns in $PM_{2.5}$ chemical composition from city-wide monthly average values for St. Louis, MO, 2005-2007. The gray line represents the difference in OCM calculated using material balance and blank corrected OC x 1.4.

A.2.4. Diel Trends

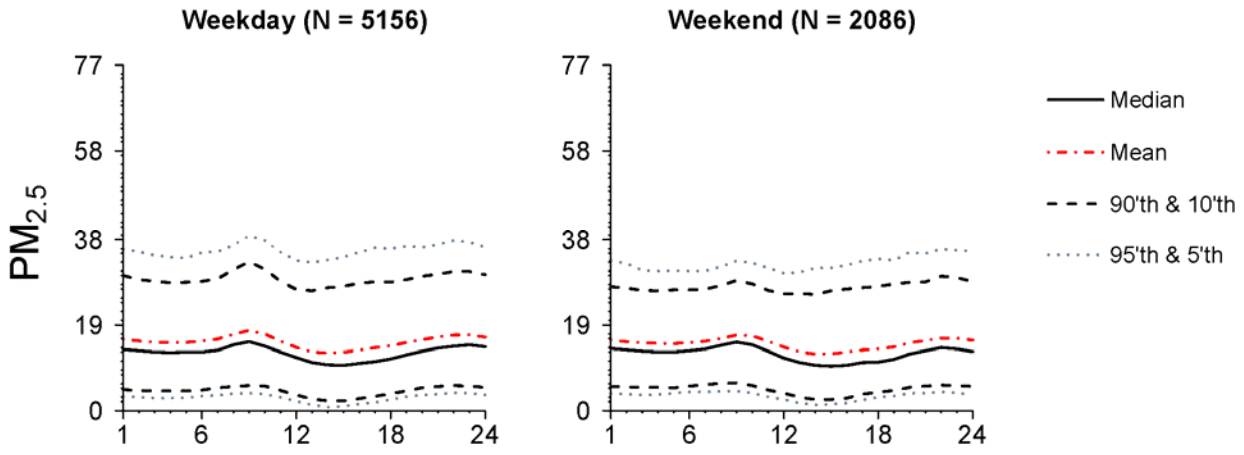

Figure A-135. Diel plots generated from all available hourly FRM-like $PM_{2.5}$ data, stratified by weekday (left) and weekend (right), in Atlanta, GA. Included are the number of monitor days (N) and the median, mean, 5th, 10th, 90th and 95th percentiles for each hour.

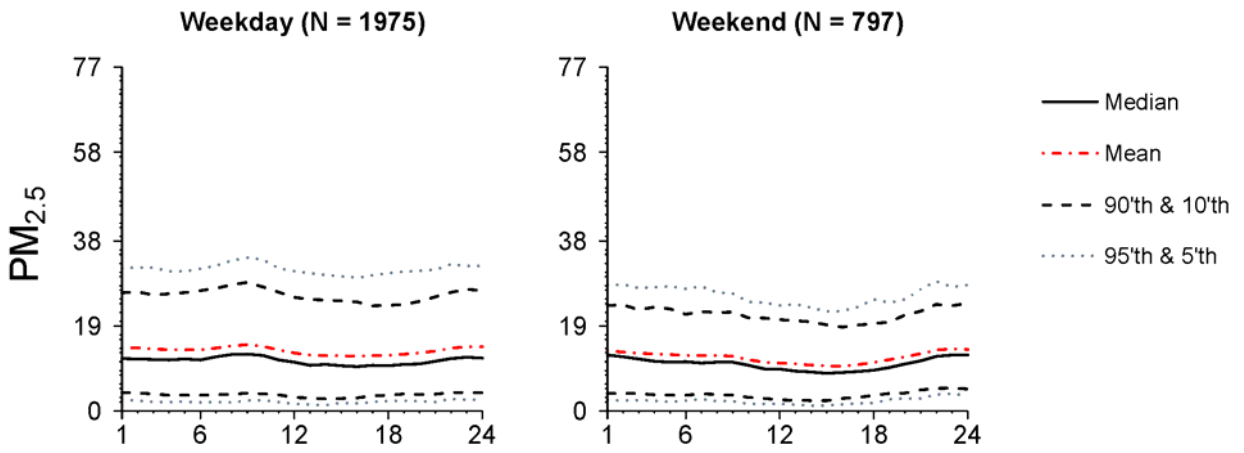

Figure A-136. Diel plots generated from all available hourly FRM-like $PM_{2.5}$ data, stratified by weekday (left) and weekend (right), in Chicago, IL. Included are the number of monitor days (N) and the median, mean, 5th, 10th, 90th and 95th percentiles for each hour.

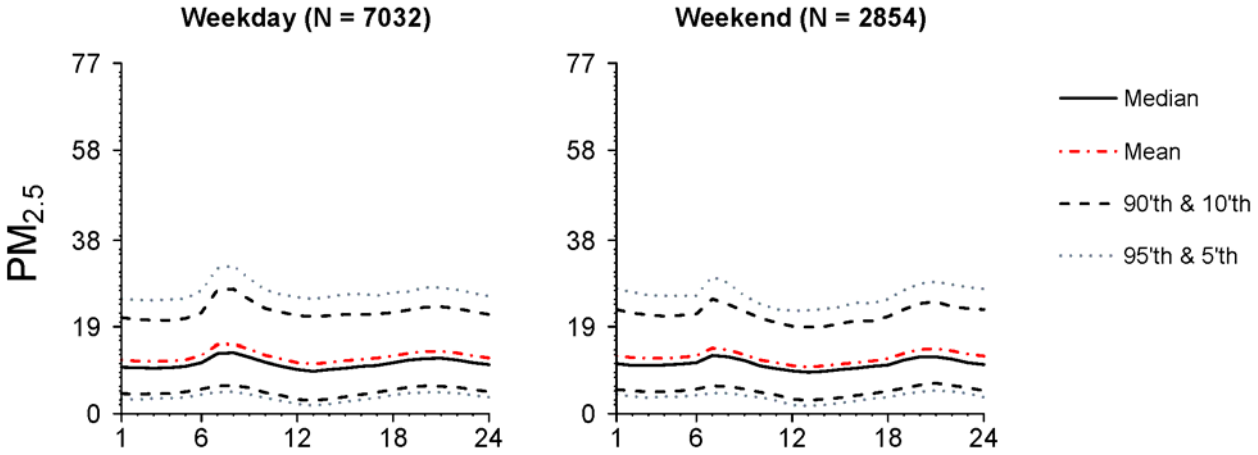

Figure A-137. Diel plots generated from all available hourly FRM-like $PM_{2.5}$ data, stratified by weekday (left) and weekend (right), in Houston, TX. Included are the number of monitor days (N) and the median, mean, 5th, 10th, 90th and 95th percentiles for each hour.

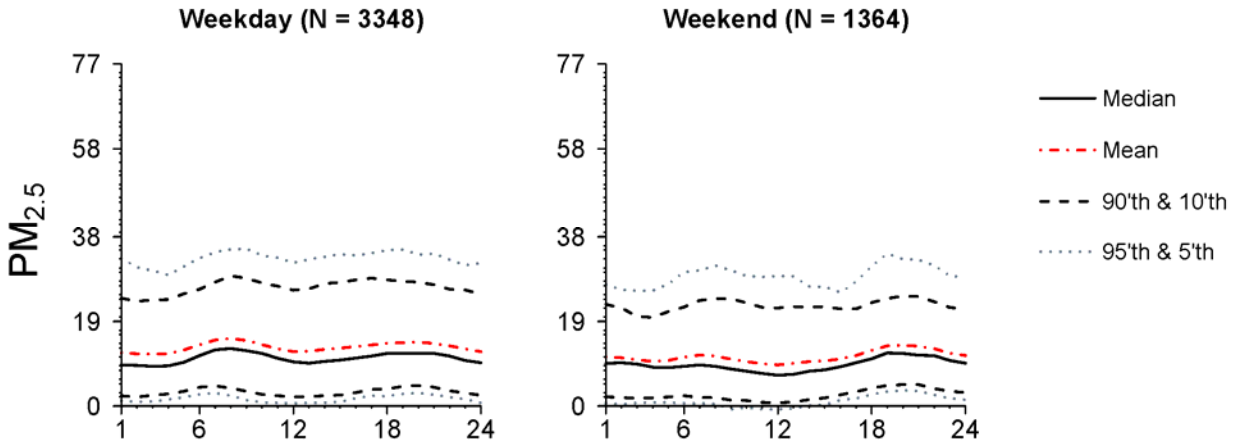

Figure A-138. Diel plots generated from all available hourly FRM-like $PM_{2.5}$ data, stratified by weekday (left) and weekend (right), in New York City, NY. Included are the number of monitor days (N) and the median, mean, 5th, 10th, 90th and 95th percentiles for each hour.

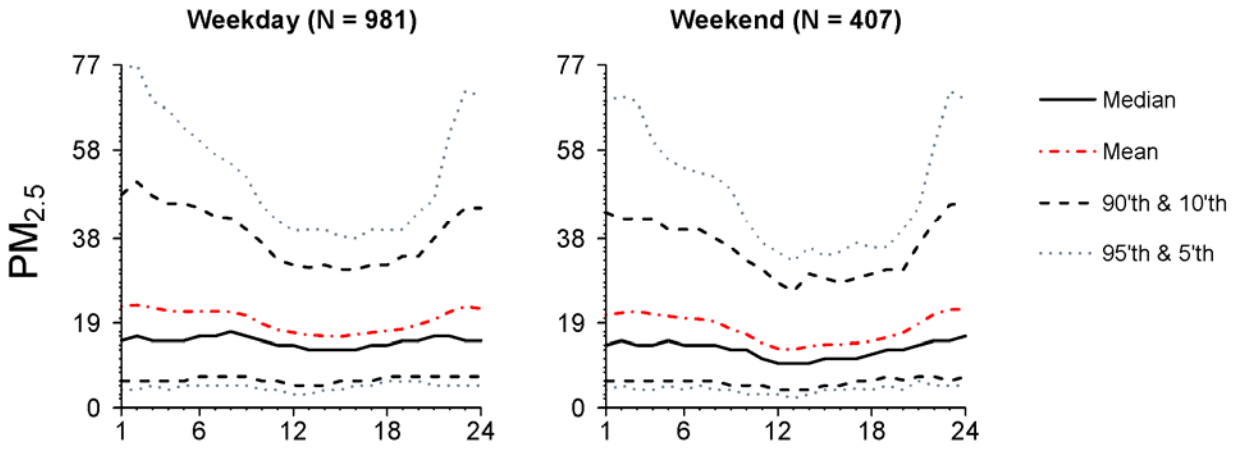

Figure A-139. Diel plots generated from all available hourly FRM-like $PM_{2.5}$ data, stratified by weekday (left) and weekend (right), in Pittsburgh, PA. Included are the number of monitor days (N) and the median, mean, 5th, 10th, 90th and 95th percentiles for each hour.

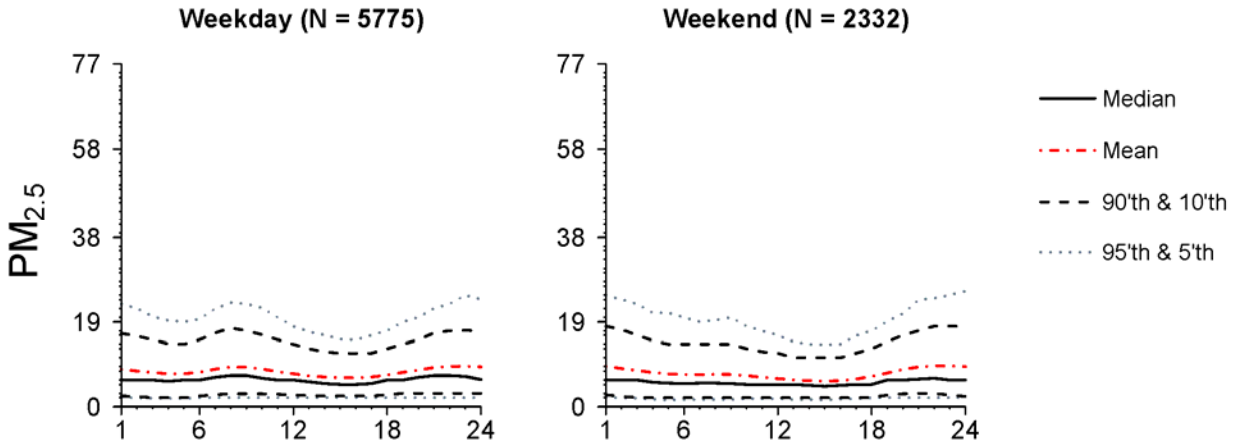

Figure A-140. Diel plots generated from all available hourly FRM-like $PM_{2.5}$ data, stratified by weekday (left) and weekend (right), in Seattle, WA. Included are the number of monitor days (N) and the median, mean, 5th, 10th, 90th and 95th percentiles for each hour.

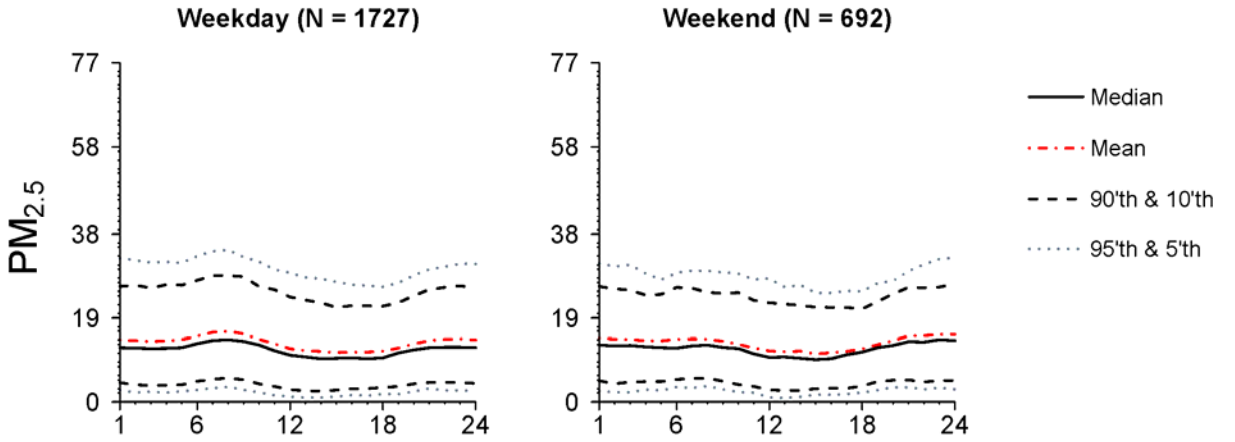

Figure A-141. Diel plots generated from all available hourly FRM-like $PM_{2.5}$ data, stratified by weekday (left) and weekend (right), in St. Louis, MO. Included are the number of monitor days (N) and the median, mean, 5th, 10th, 90th and 95th percentiles for each hour.

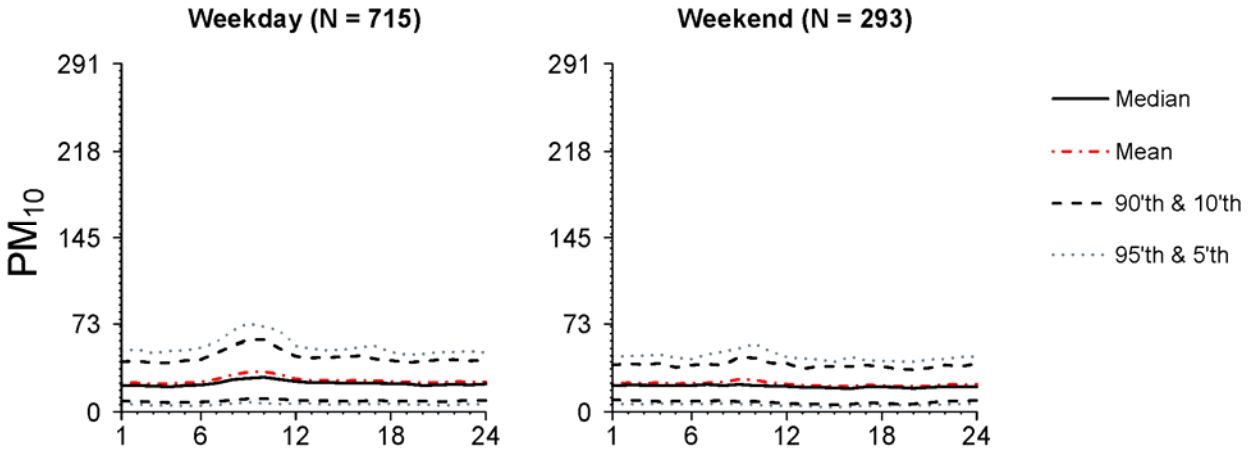

Figure A-142. Diel plot generated from all available hourly FRM/FEM PM_{10} data, stratified by weekday (left) and weekend (right), in Atlanta, GA. Included are the number of monitor days (N) and the median, mean, 5th, 10th, 90th and 95th percentiles for each hour.

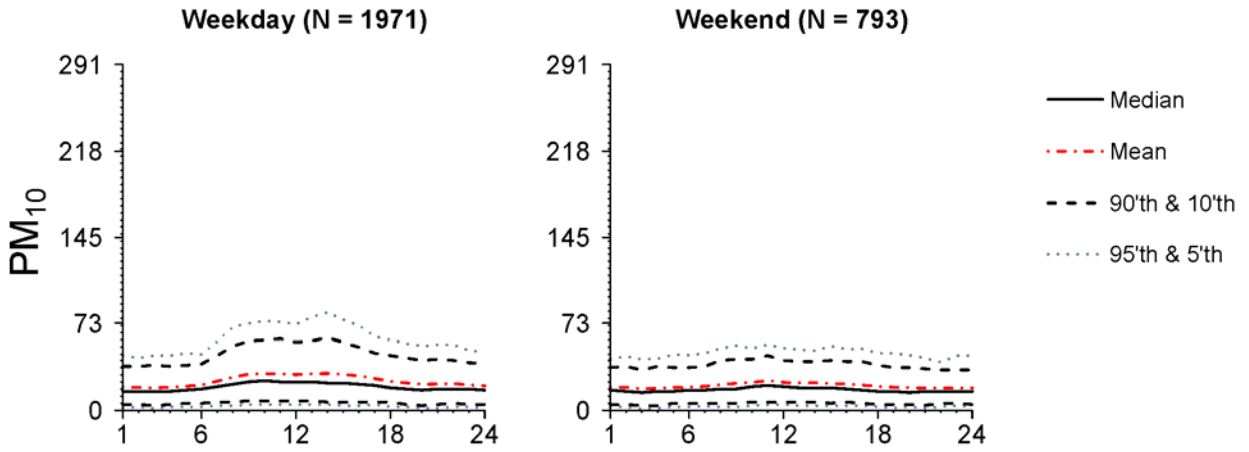

Figure A-143. Diel plot generated from all available hourly FRM/FEM PM_{10} data, stratified by weekday (left) and weekend (right), in Chicago, IL. Included are the number of monitor days (N) and the median, mean, 5th, 10th, 90th and 95th percentiles for each hour.

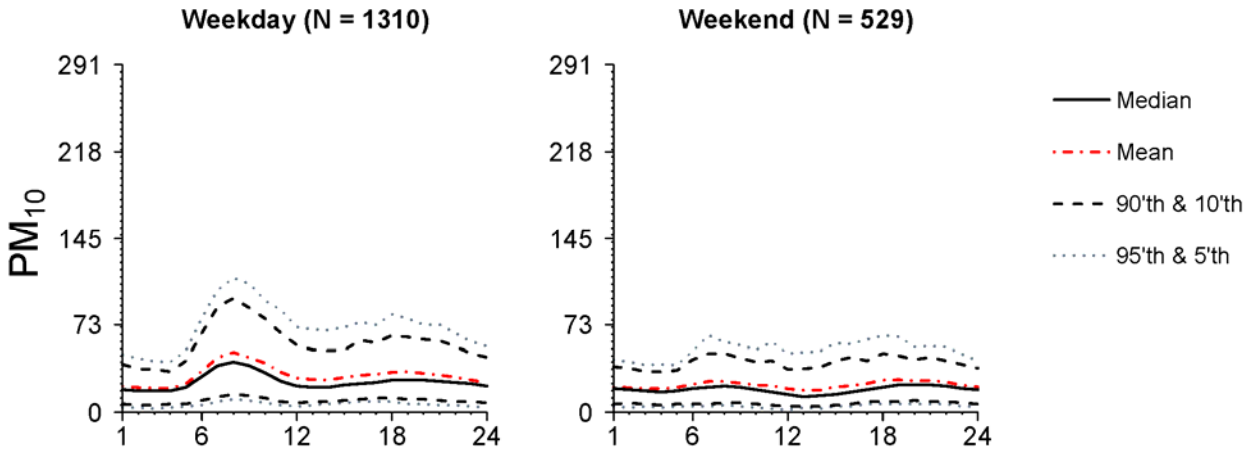

Figure A-144. Diel plot generated from all available hourly FRM/FEM PM_{10} data, stratified by weekday (left) and weekend (right), in Denver, CO. Included are the number of monitor days (N) and the median, mean, 5th, 10th, 90th and 95th percentiles for each hour.

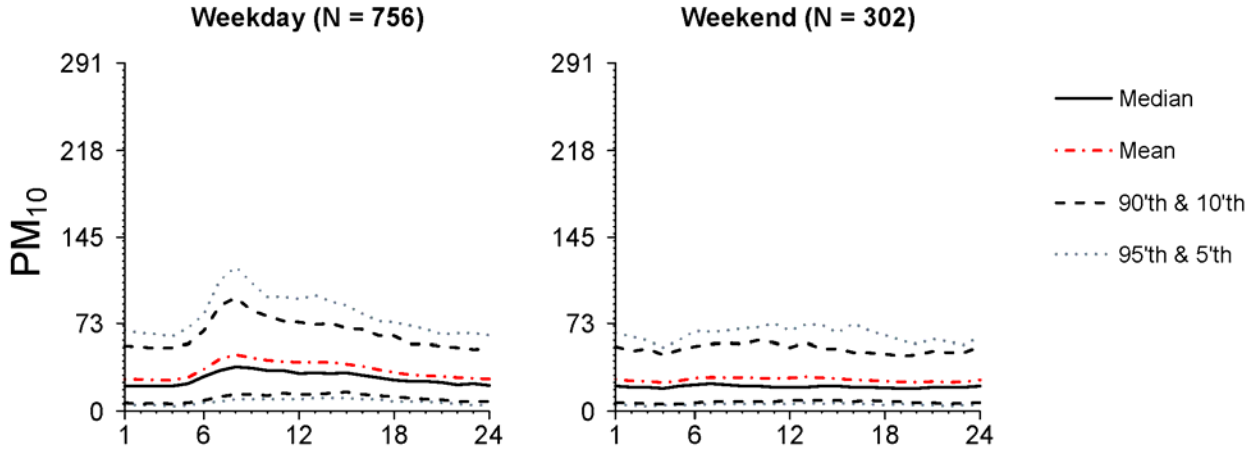

Figure A-145. Diel plot generated from all available hourly FRM/FEM PM_{10} data, stratified by weekday (left) and weekend (right), in Detroit, MI. Included are the number of monitor days (N) and the median, mean, 5th, 10th, 90th and 95th percentiles for each hour.

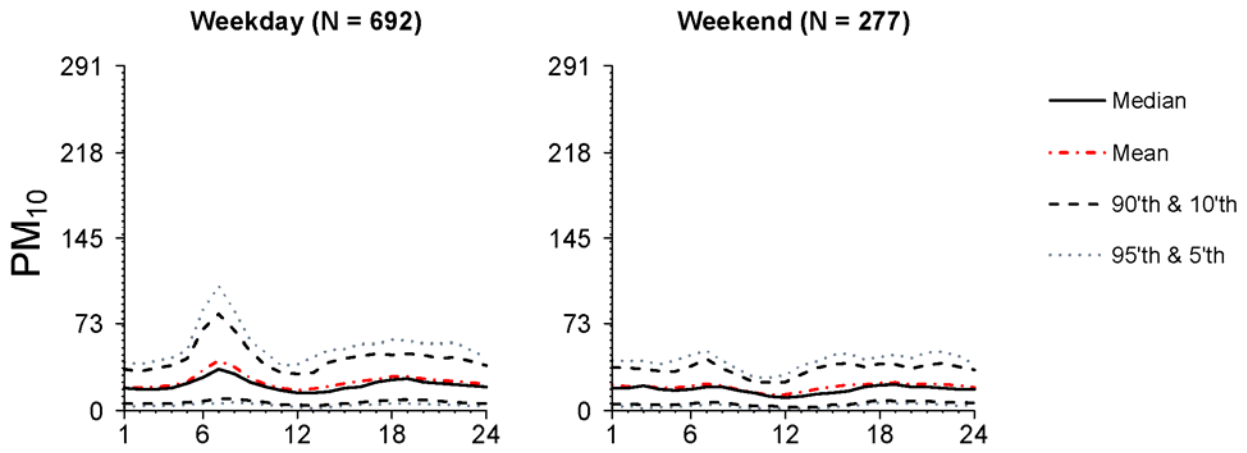

Figure A-146. Diel plot generated from all available hourly FRM/FEM PM_{10} data, stratified by weekday (left) and weekend (right), in Los Angeles, CA. Included are the number of monitor days (N) and the median, mean, 5th, 10th, 90th and 95th percentiles for each hour.

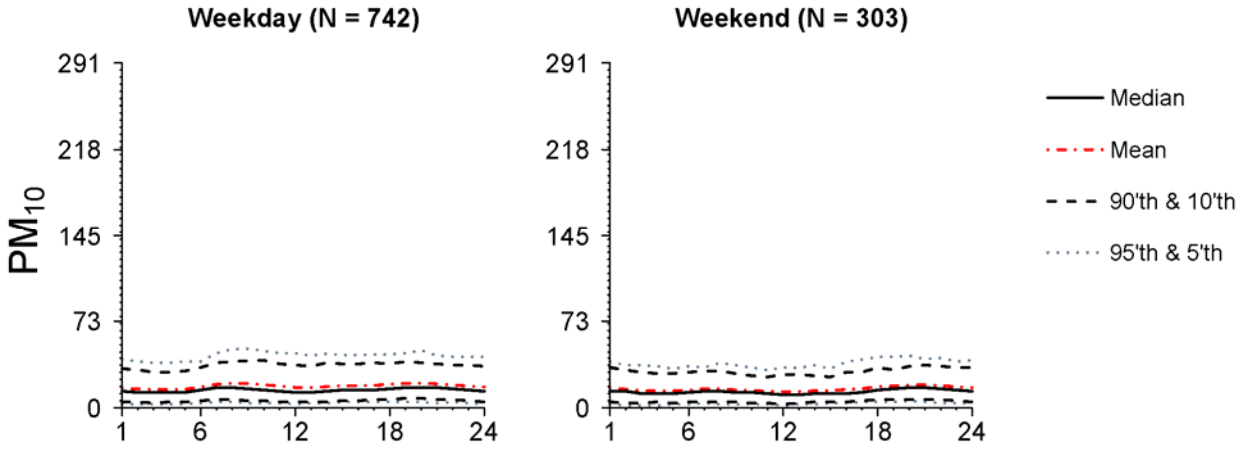

Figure A-147. Diel plot generated from all available hourly FRM/FEM PM_{10} data, stratified by weekday (left) and weekend (right), in Philadelphia, PA. Included are the number of monitor days (N) and the median, mean, 5th, 10th, 90th and 95th percentiles for each hour.

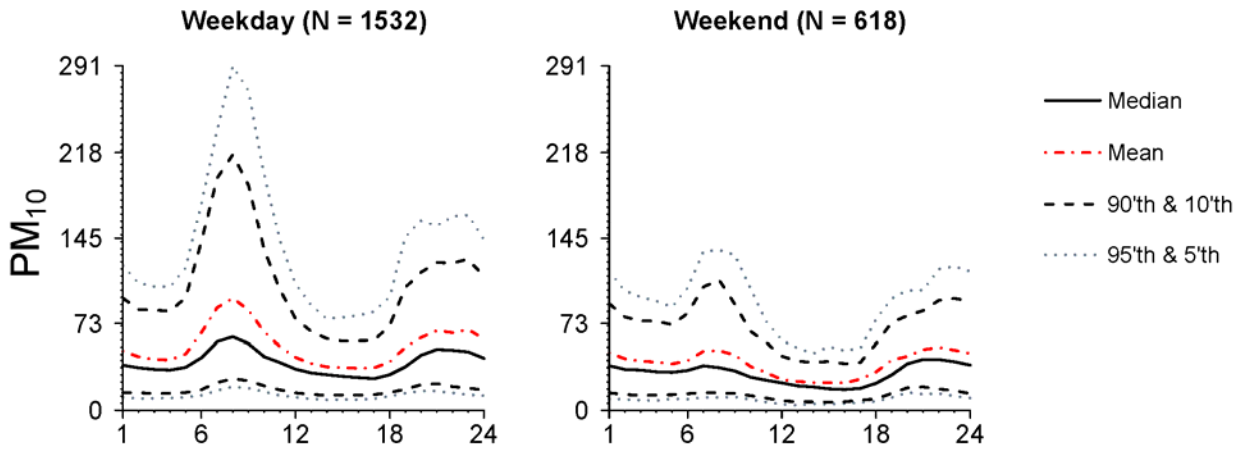

Figure A-148. Diel plot generated from all available hourly FRM/FEM PM_{10} data, stratified by weekday (left) and weekend (right), in Phoenix, AZ. Included are the number of monitor days (N) and the median, mean, 5th, 10th, 90th and 95th percentiles for each hour.

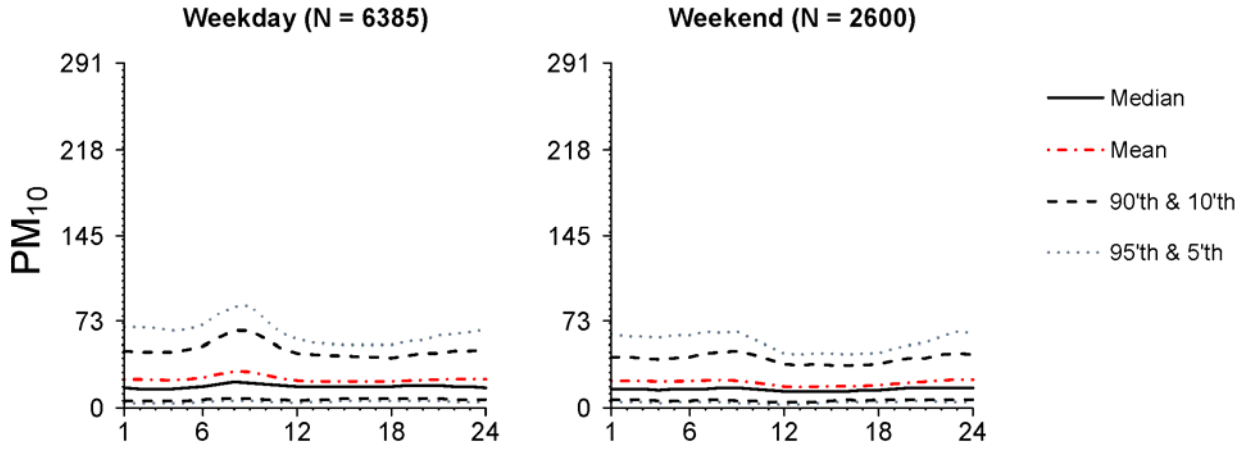

Figure A-149. Diel plot generated from all available hourly FRM/FEM PM_{10} data, stratified by weekday (left) and weekend (right), in Pittsburgh, PA. Included are the number of monitor days (N) and the median, mean, 5th, 10th, 90th and 95th percentiles for each hour.

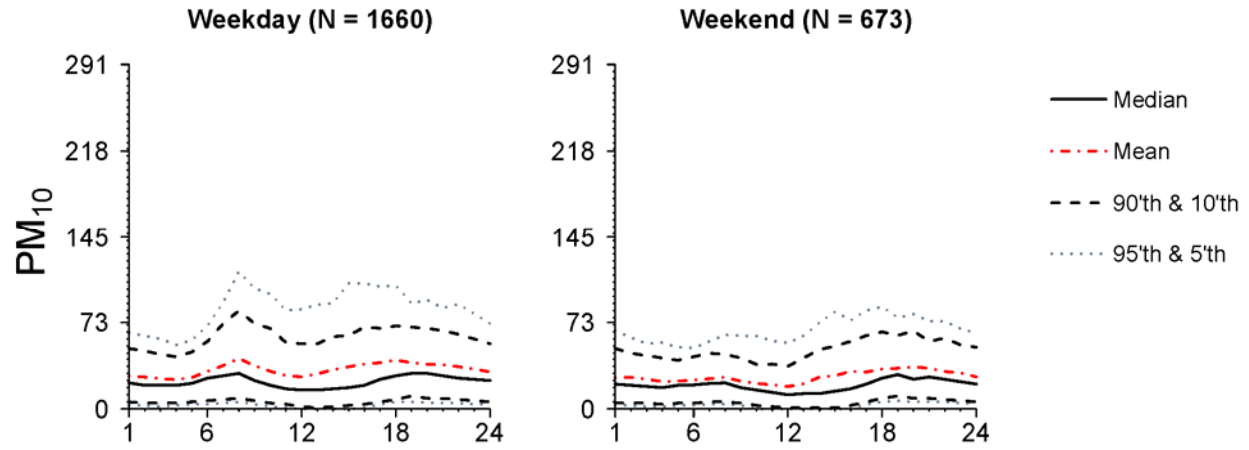

Figure A-150. Diel plot generated from all available hourly FRM/FEM PM_{10} data, stratified by weekday (left) and weekend (right), in Riverside, CA. Included are the number of monitor days (N) and the median, mean, 5th, 10th, 90th and 95th percentiles for each hour.

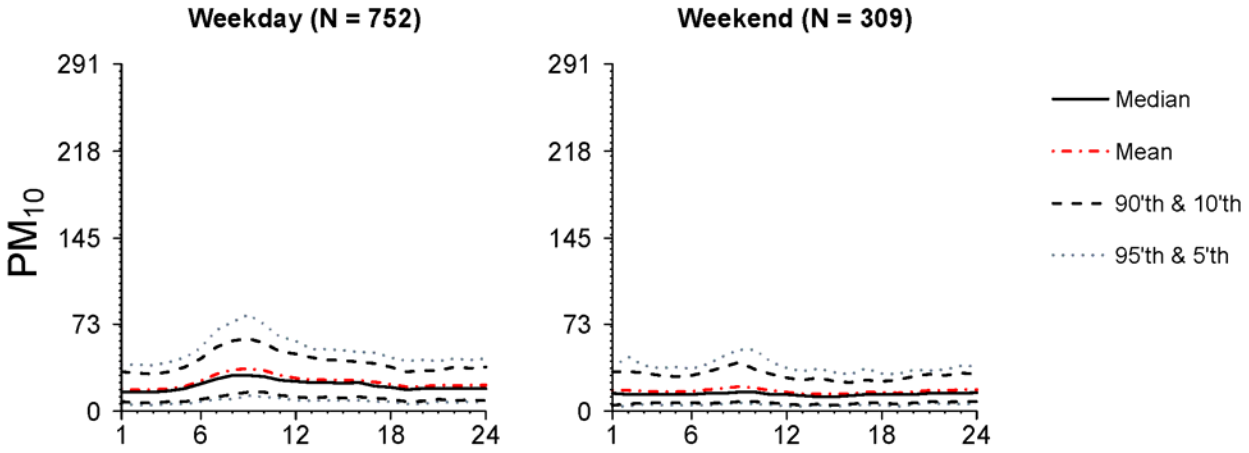

Figure A-151. Diel plot generated from all available hourly FRM/FEM PM_{10} data, stratified by weekday (left) and weekend (right), in Seattle, WA. Included are the number of monitor days (N) and the median, mean, 5th, 10th, 90th and 95th percentiles for each hour.

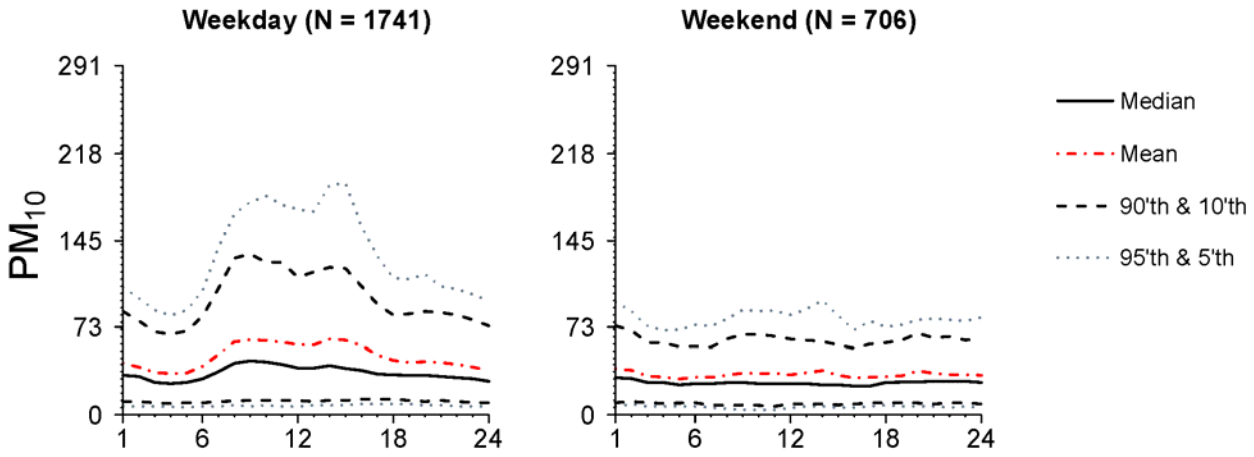

Figure A-152. Diel plot generated from all available hourly FRM/FEM PM_{10} data, stratified by weekday (left) and weekend (right), in St. Louis, MO. Included are the number of monitor days (N) and the median, mean, 5th, 10th, 90th and 95th percentiles for each hour.

A.2.5. Copollutant Measurements

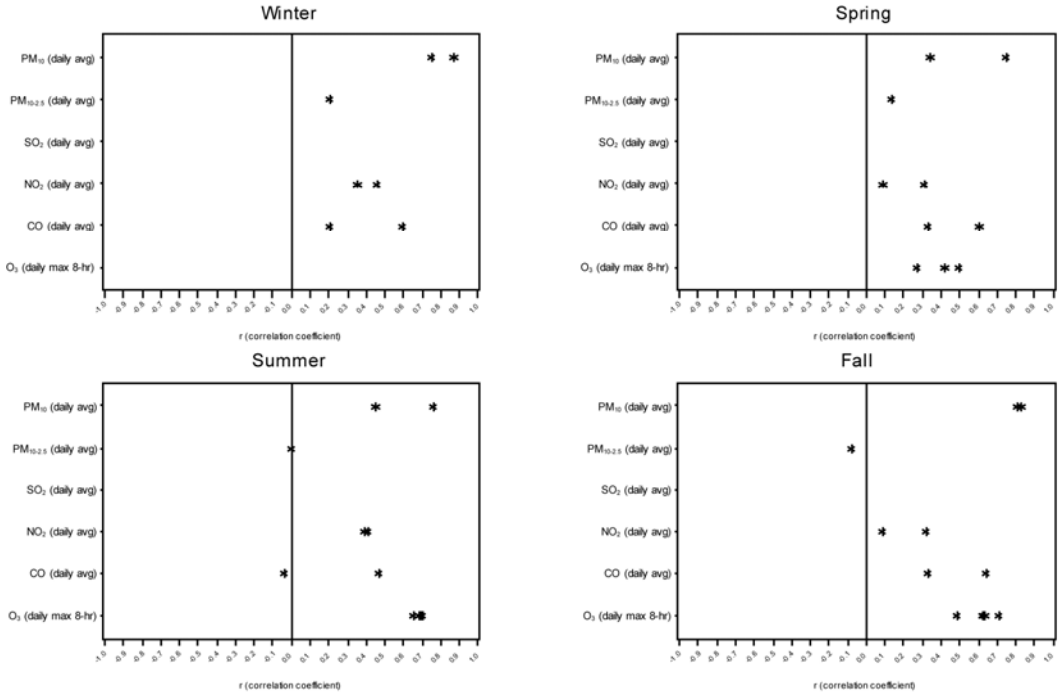

Figure A-153. Correlations between 24-h $PM_{2.5}$ and co-located 24-h avg PM_{10}, $PM_{10\text{-}2.5}$, SO_2, NO_2 and CO and daily maximum 8-h avg O_3 for Atlanta, GA, stratified by season (2005-2007). One point is included for each available monitor pair.

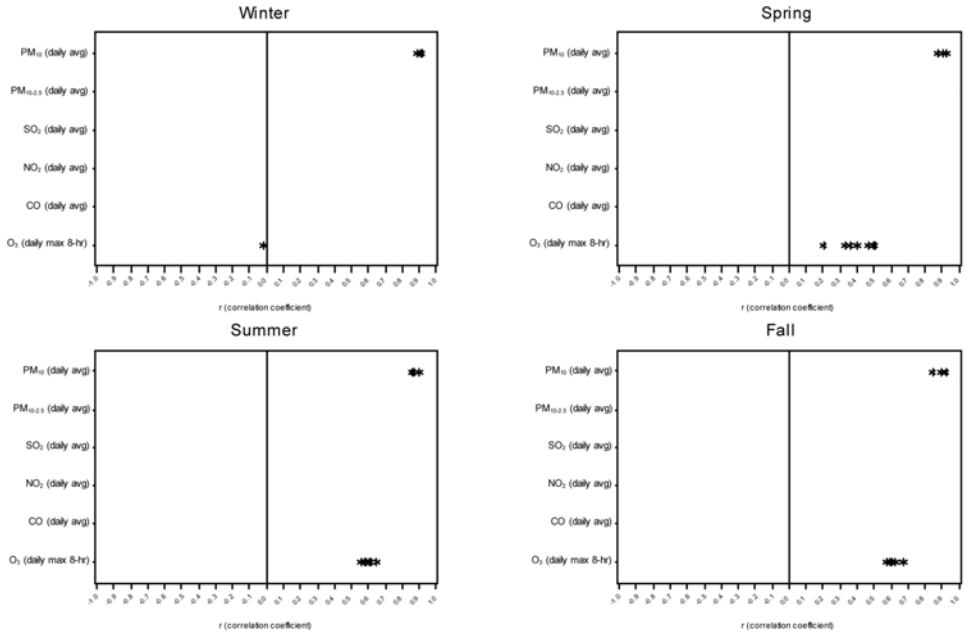

Figure A-154. Correlations between 24-h $PM_{2.5}$ and co-located 24-h avg PM_{10}, $PM_{10\text{-}2.5}$, SO_2, NO_2 and CO and daily maximum 8-h avg O_3 for Birmingham, AL, stratified by season (2005-2007). One point is included for each available monitor pair.

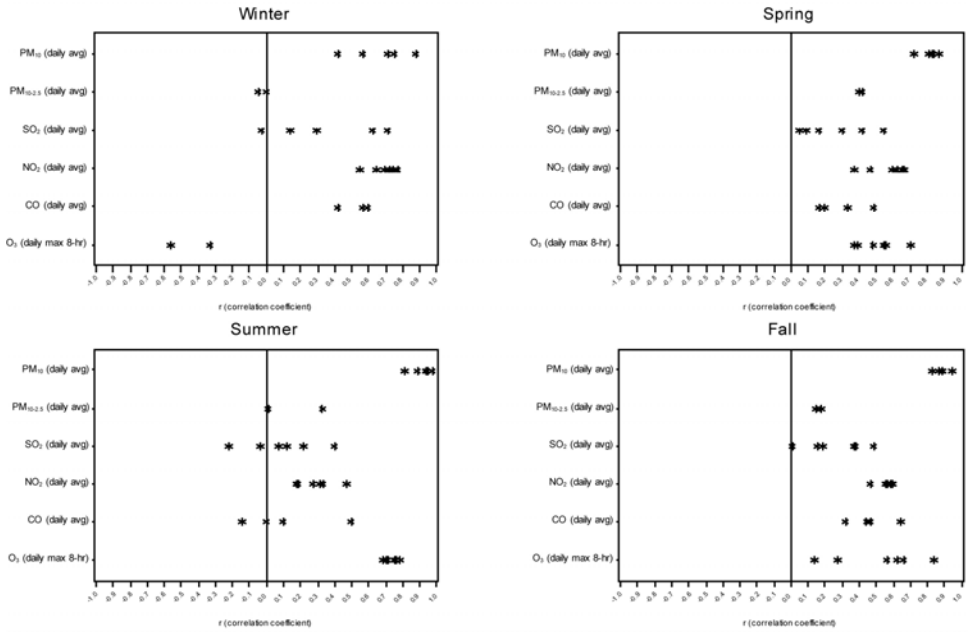

Figure A-155. Correlations between 24-h $PM_{2.5}$ and co-located 24-h avg PM_{10}, $PM_{10\text{-}2.5}$, SO_2, NO_2 and CO and daily maximum 8-h avg O_3 for Boston, MA, stratified by season (2005-2007). One point is included for each available monitor pair.

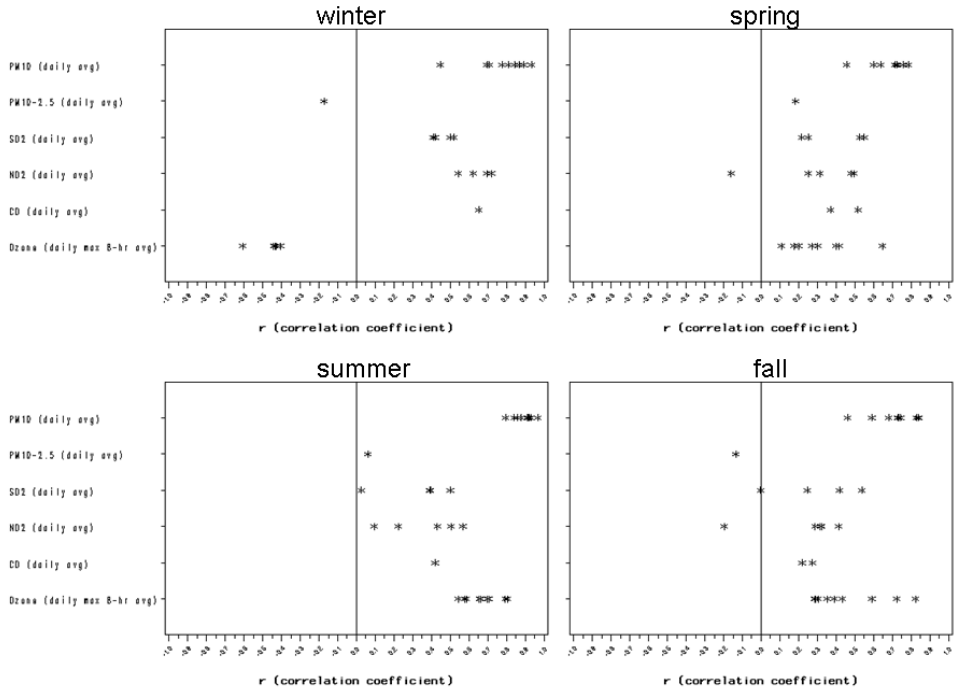

Figure A-156. Correlations between 24-h $PM_{2.5}$ and co-located 24-h avg PM_{10}, $PM_{10-2.5}$, SO_2, NO_2 and CO and daily maximum 8-h avg O_3 for Chicago, IL, stratified by season (2005-2007). One point is included for each available monitor pair.

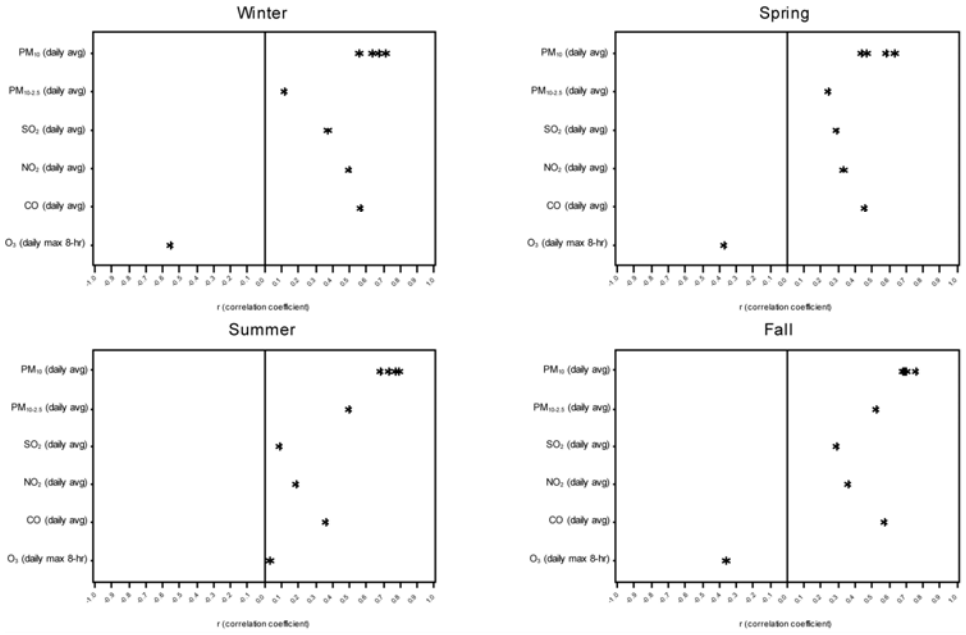

Figure A-157. Correlations between 24-h $PM_{2.5}$ and co-located 24-h avg PM_{10}, $PM_{10-2.5}$, SO_2, NO_2 and CO and daily maximum 8-h avg O_3 for Denver, CO, stratified by season (2005-2007). One point is included for each available monitor pair.

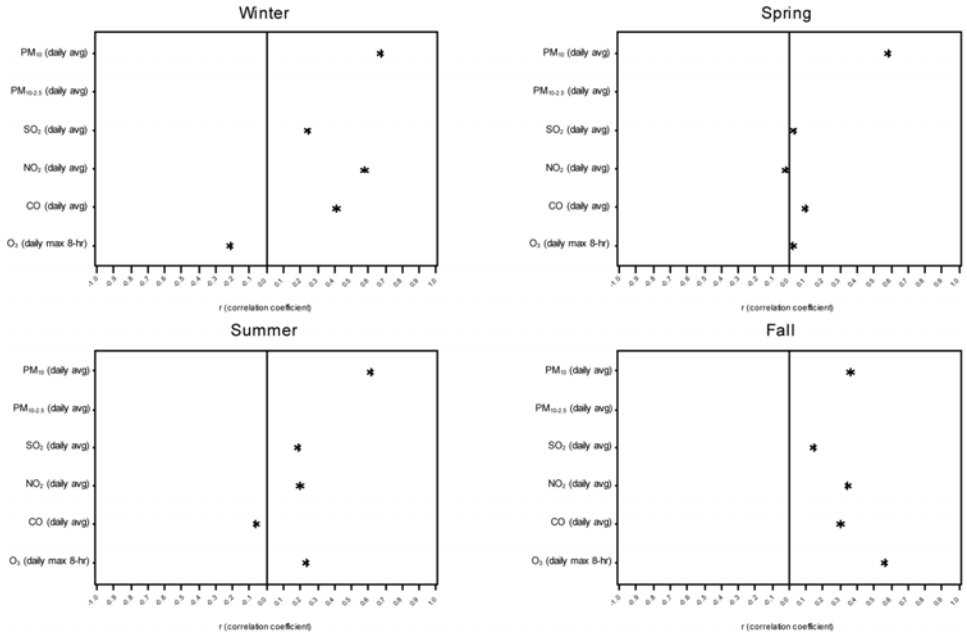

Figure A-158. Correlations between 24-h $PM_{2.5}$ and co-located 24-h avg PM_{10}, $PM_{10-2.5}$, SO_2, NO_2 and CO and daily maximum 8-h avg O_3 for Houston, TX, stratified by season (2005-2007). One point is included for each available monitor pair.

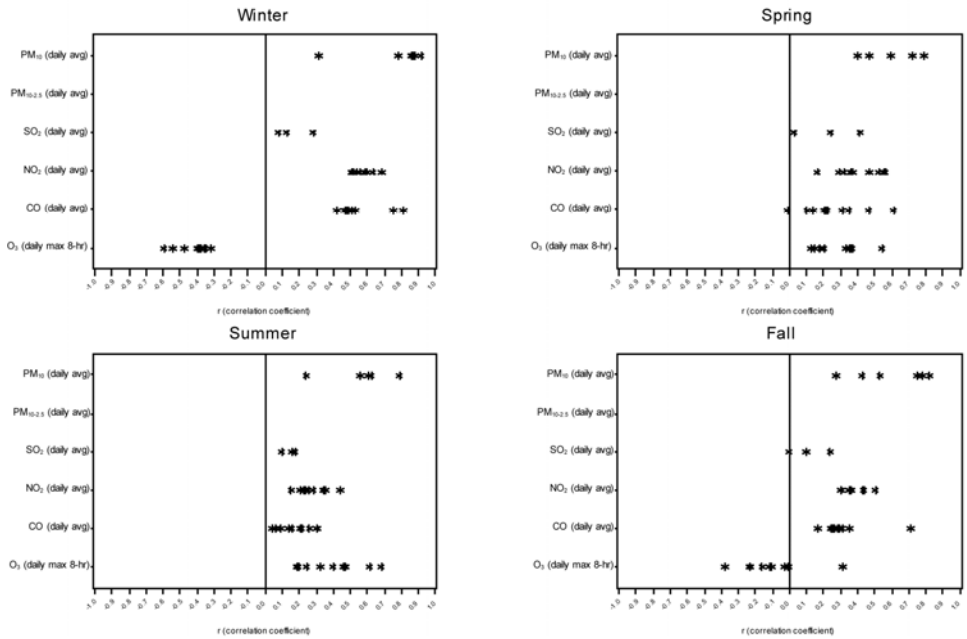

Figure A-159. Correlations between 24-h $PM_{2.5}$ and co-located 24-h avg PM_{10}, $PM_{10-2.5}$, SO_2, NO_2 and CO and daily maximum 8-h avg O_3 for Los Angeles, CA, stratified by season (2005-2007). One point is included for each available monitor pair.

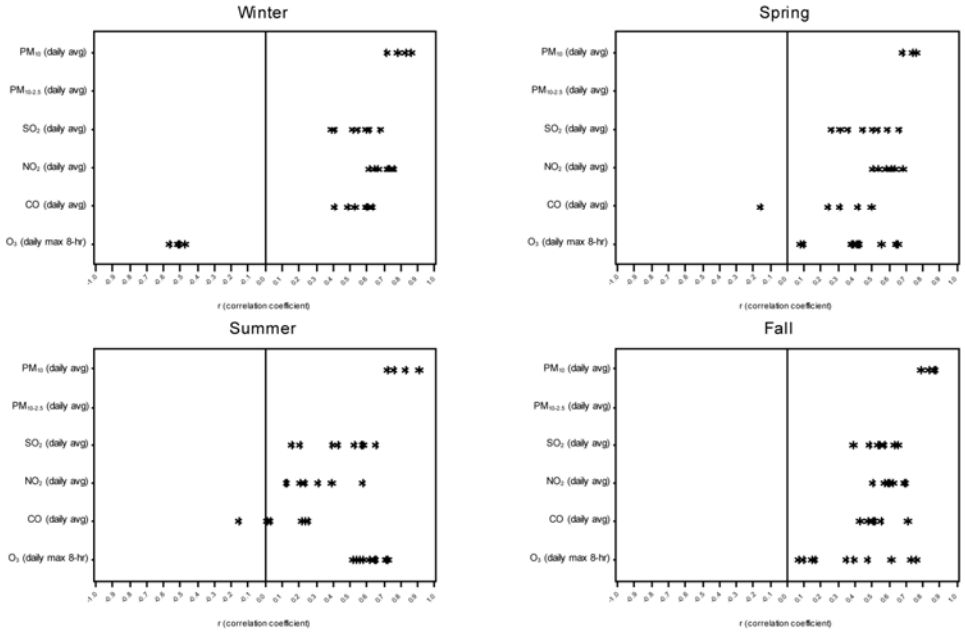

Figure A-160. Correlations between 24-h $PM_{2.5}$ and co-located 24-h avg PM_{10}, $PM_{10-2.5}$, SO_2, NO_2 and CO and daily maximum 8-h avg O_3 for Philadelphia, PA, stratified by season (2005-2007). One point is included for each available monitor pair.

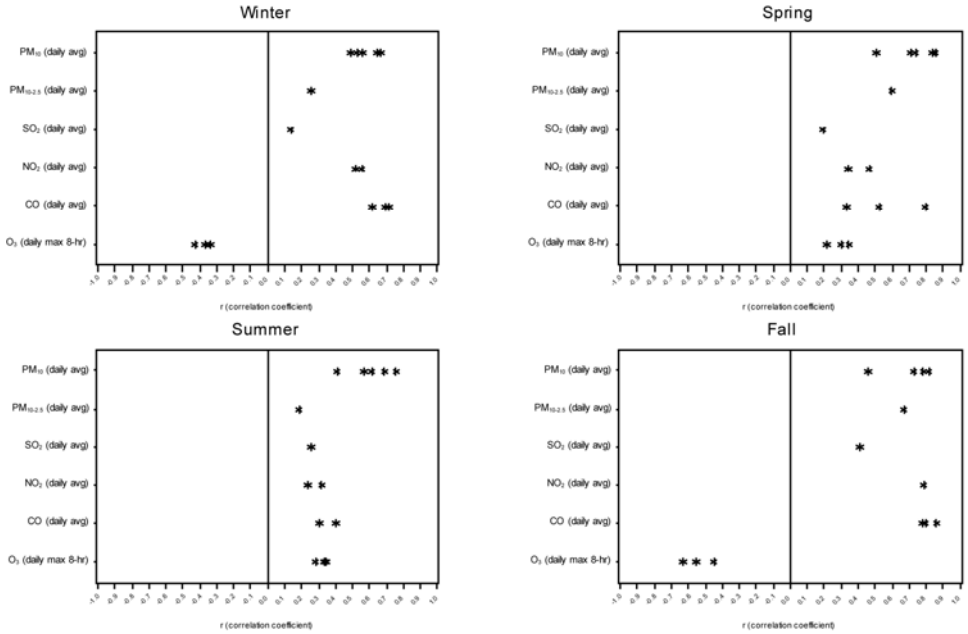

Figure A-161. Correlations between 24-h $PM_{2.5}$ and co-located 24-h avg PM_{10}, $PM_{10-2.5}$, SO_2, NO_2 and CO and daily maximum 8-h avg O_3 for Phoenix, AZ, stratified by season (2005-2007). One point is included for each available monitor pair.

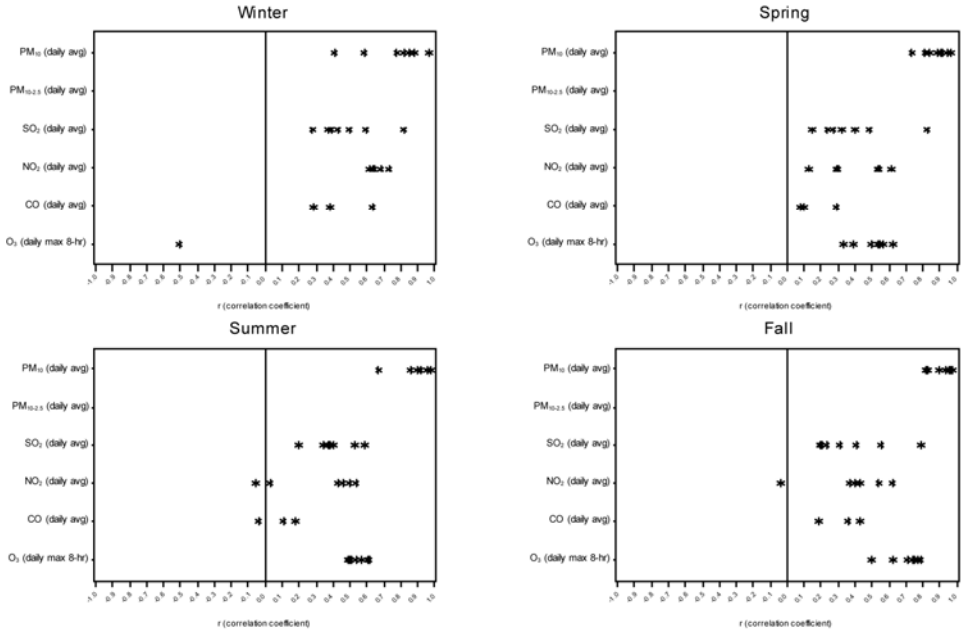

Figure A-162. Correlations between 24-h $PM_{2.5}$ and co-located 24-h avg PM_{10}, $PM_{10-2.5}$, SO_2, NO_2 and CO and daily maximum 8-h avg O_3 for Pittsburgh, PA, stratified by season (2005-2007). One point is included for each available monitor pair.

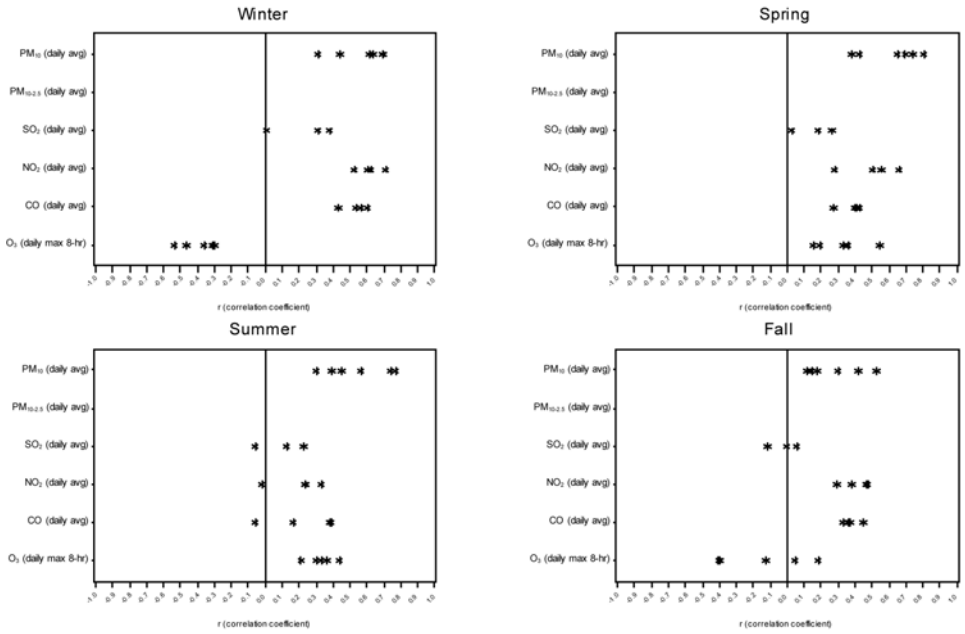

Figure A-163. Correlations between 24-h $PM_{2.5}$ and co-located 24-h avg PM_{10}, $PM_{10-2.5}$, SO_2, NO_2 and CO and daily maximum 8-h avg O_3 for Riverside, CA, stratified by season (2005-2007). One point is included for each available monitor pair.

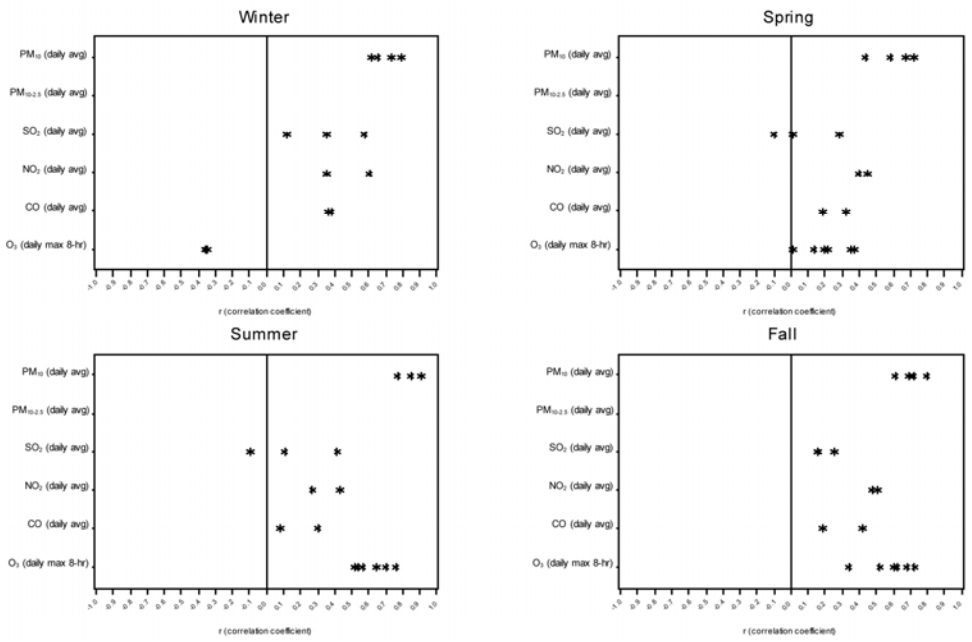

Figure A-164. Correlations between 24-h $PM_{2.5}$ and co-located 24-h avg PM_{10}, $PM_{10\text{-}2.5}$, SO_2, NO_2 and CO and daily maximum 8-h avg O_3 for St. Louis, MO, stratified by season (2005-2007). One point is included for each available monitor pair.

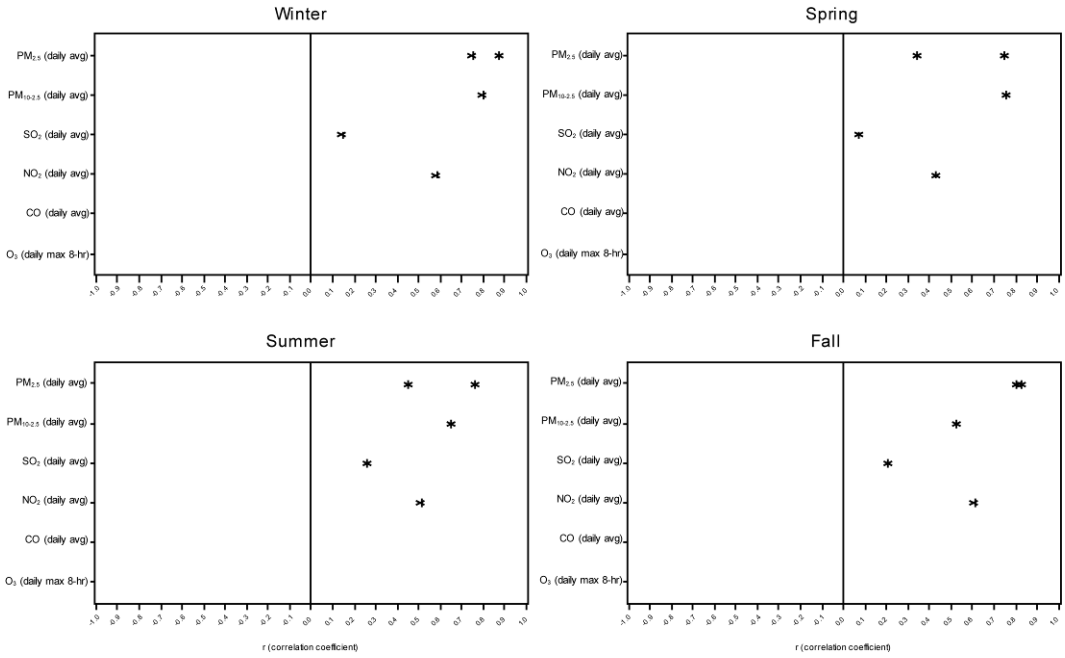

Figure A-165. Correlations between 24-h PM_{10} and co-located 24-h avg $PM_{2.5}$, $PM_{10\text{-}2.5}$ SO_2, NO_2 and CO and daily maximum 8-h avg O_3 for Atlanta, GA, stratified by season (2005-2007). One point is included for each available monitor pair.

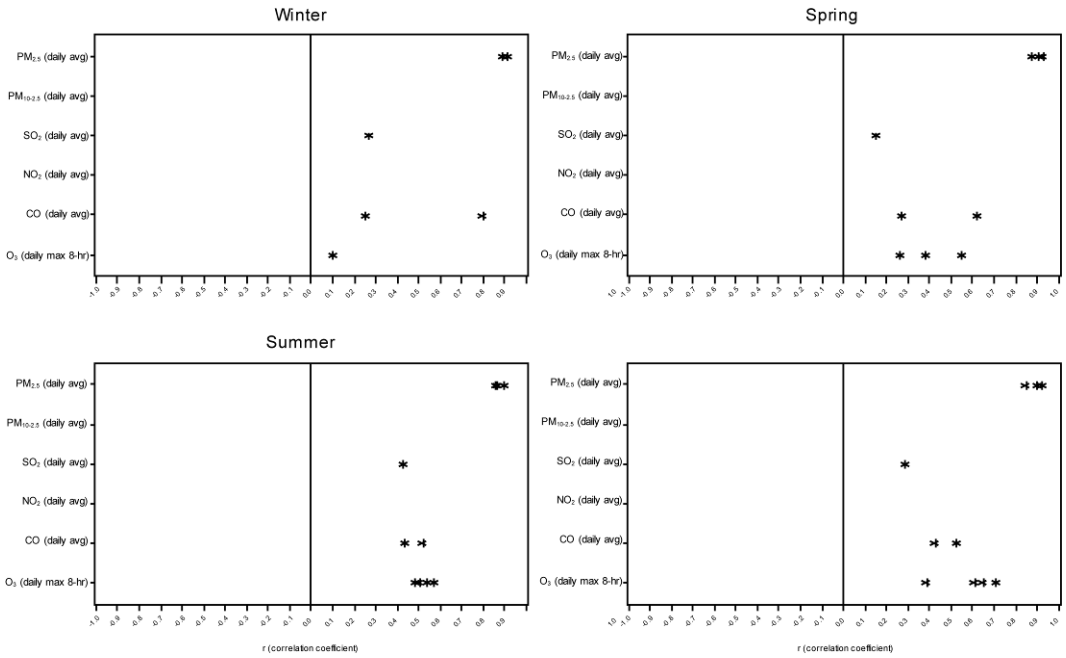

Figure A-166. Correlations between 24-h PM_{10} and co-located 24-h avg $PM_{2.5}$, $PM_{10-2.5}$ SO_2, NO_2 and CO and daily maximum 8-h avg O_3 for Birmingham, AL, stratified by season (2005-2007). One point is included for each available monitor pair.

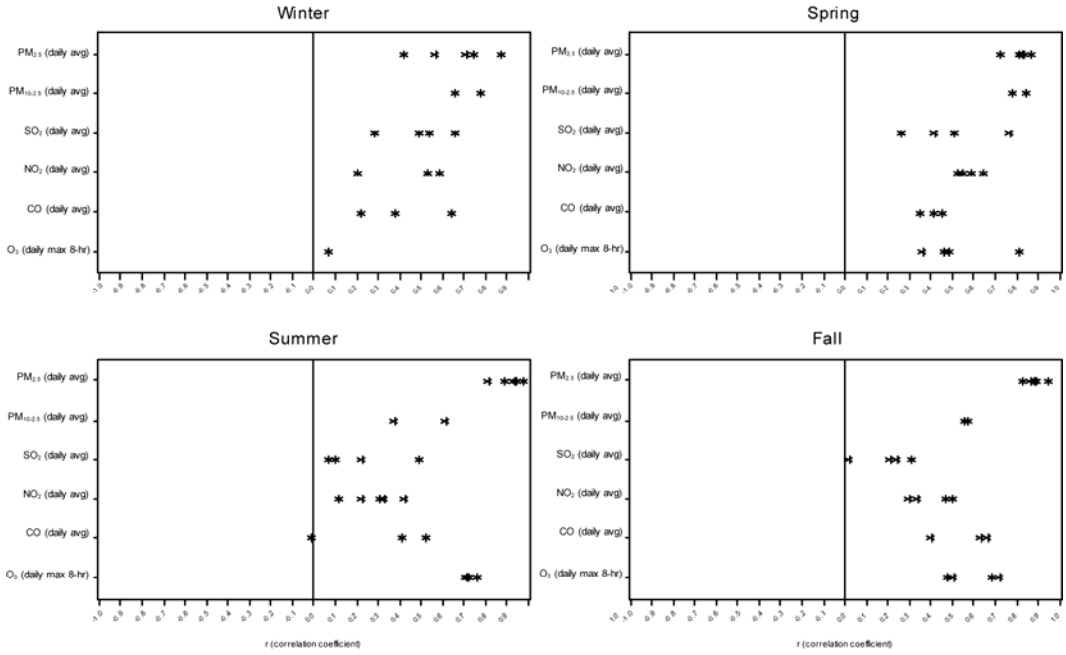

Figure A-167. Correlations between 24-h PM_{10} and co-located 24-h avg $PM_{2.5}$, $PM_{10-2.5}$ SO_2, NO_2 and CO and daily maximum 8-h avg O_3 for Boston, MA, stratified by season (2005-2007). One point is included for each available monitor pair.

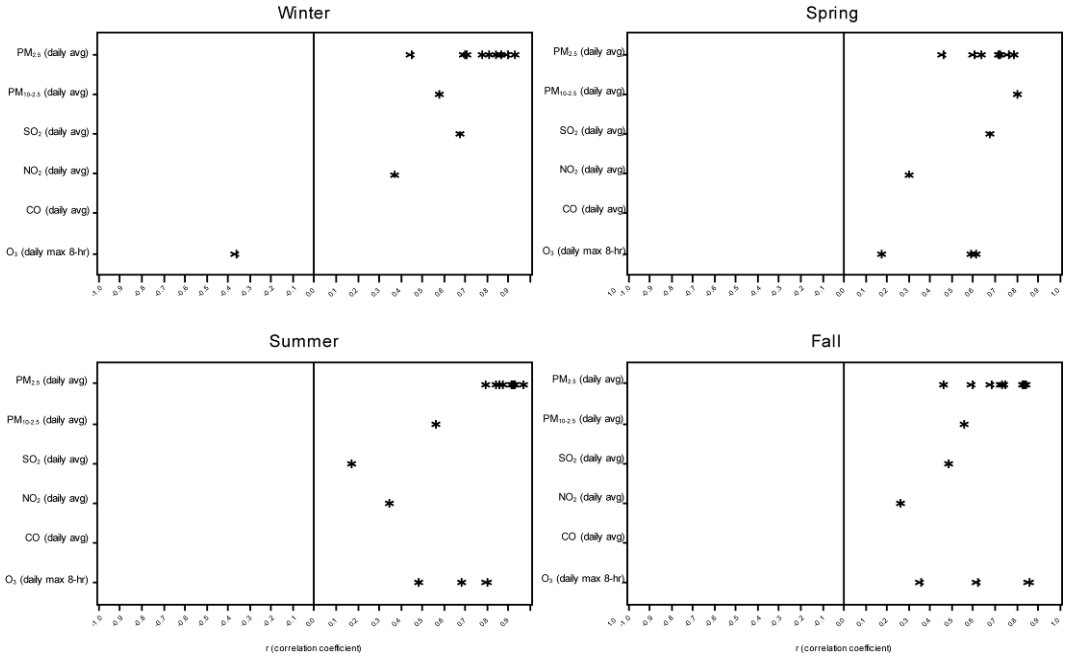

Figure A-168. Correlations between 24-h PM_{10} and co-located 24-h avg $PM_{2.5}$, $PM_{10-2.5}$ SO_2, NO_2 and CO and daily maximum 8-h avg O_3 for Chicago, IL, stratified by season (2005-2007). One point is included for each available monitor pair.

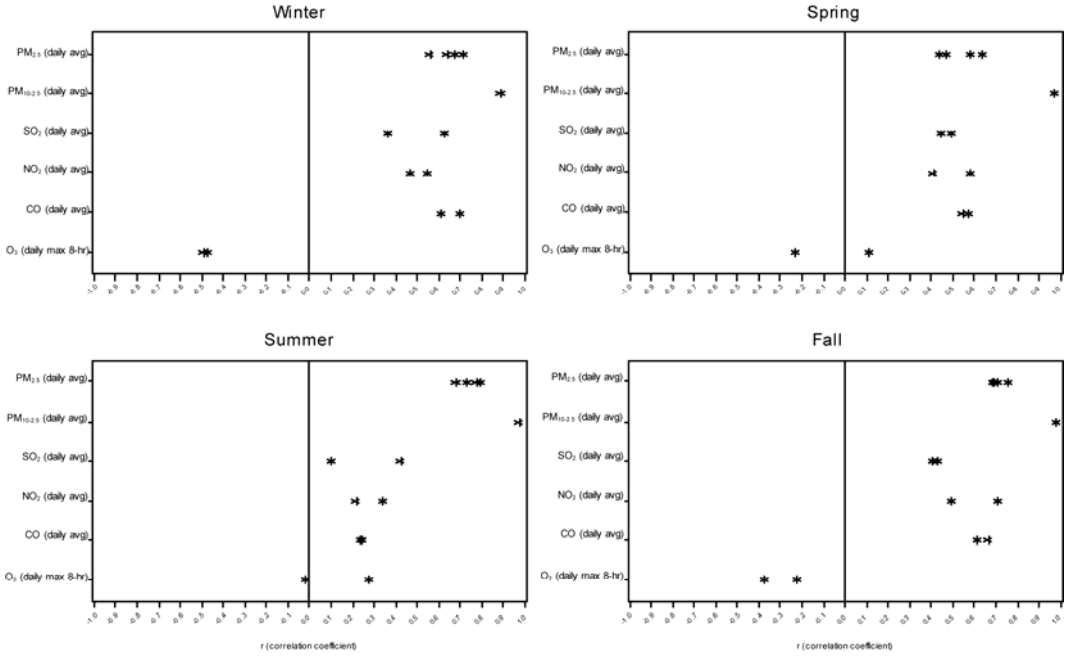

Figure A-169. Correlations between 24-h PM_{10} and co-located 24-h avg $PM_{2.5}$, $PM_{10-2.5}$ SO_2, NO_2 and CO and daily maximum 8-h avg O_3 for Denver, CO, stratified by season (2005-2007). One point is included for each available monitor pair.

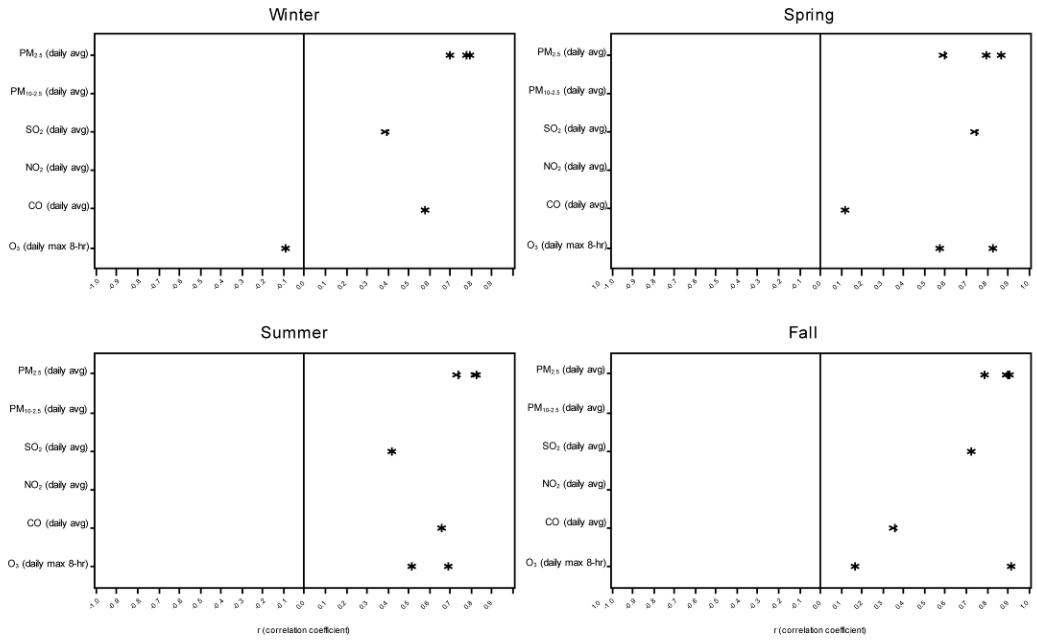

Figure A-170. Correlations between 24-h PM_{10} and co-located 24-h avg $PM_{2.5}$, $PM_{10\text{-}2.5}$ SO_2, NO_2 and CO and daily maximum 8-h avg O_3 for Detroit, MI, stratified by season (2005-2007). One point is included for each available monitor pair.

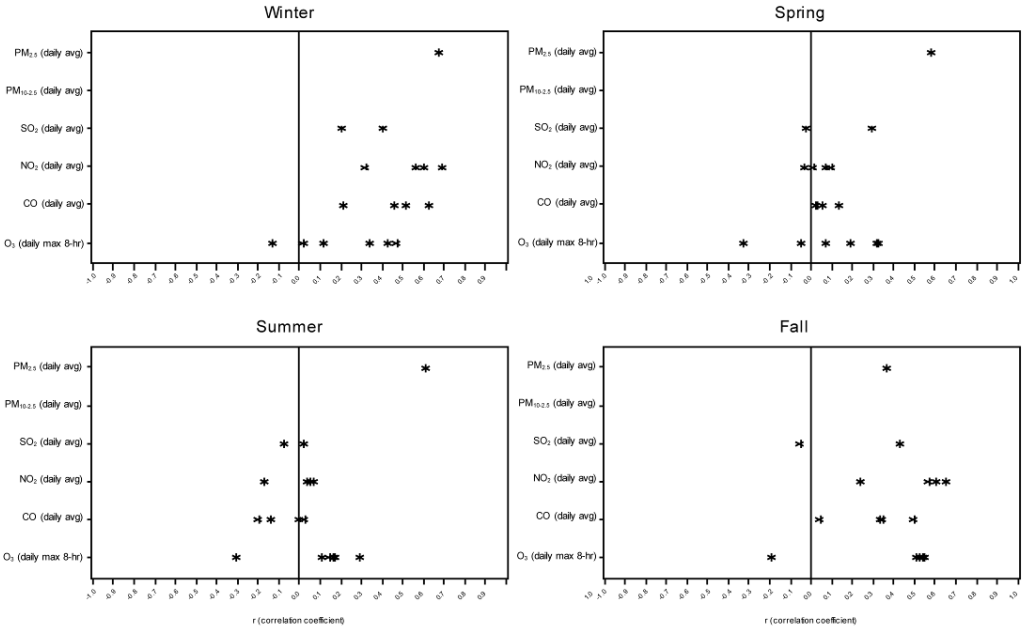

Figure A-171. Correlations between 24-h PM_{10} and co-located 24-h avg $PM_{2.5}$, $PM_{10\text{-}2.5}$ SO_2, NO_2 and CO and daily maximum 8-h avg O_3 for Houston, TX, stratified by season (2005-2007). One point is included for each available monitor pair.

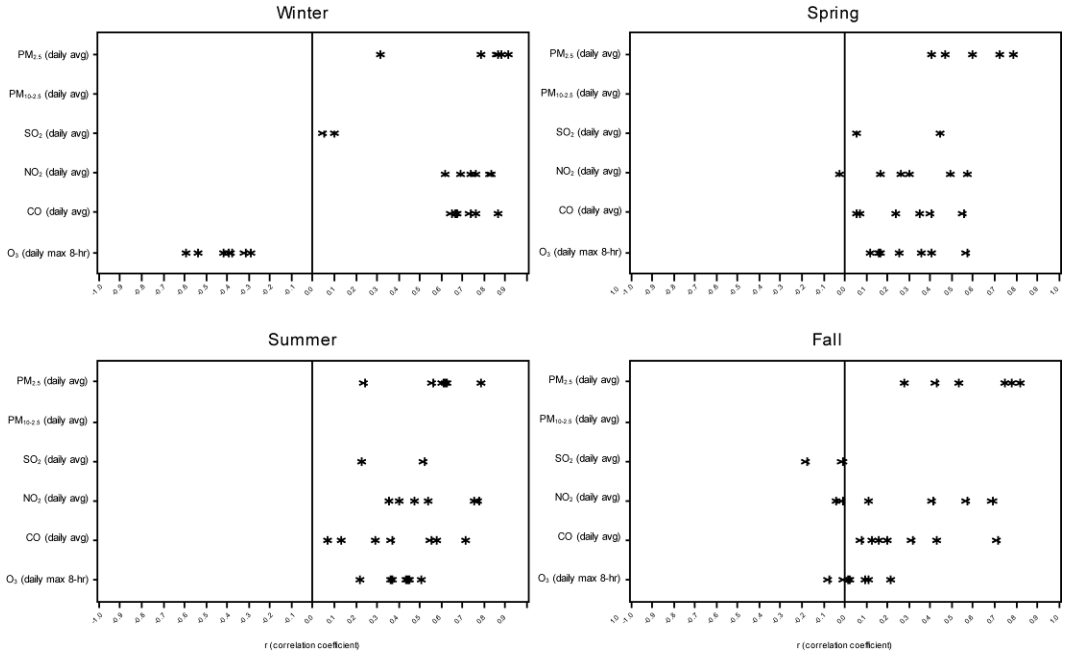

Figure A-172. Correlations between 24-h PM_{10} and co-located 24-h avg $PM_{2.5}$, $PM_{10\text{-}2.5}$ SO_2, NO_2 and CO and daily maximum 8-h avg O_3 for Los Angeles, CA, stratified by season (2005-2007). One point is included for each available monitor pair.

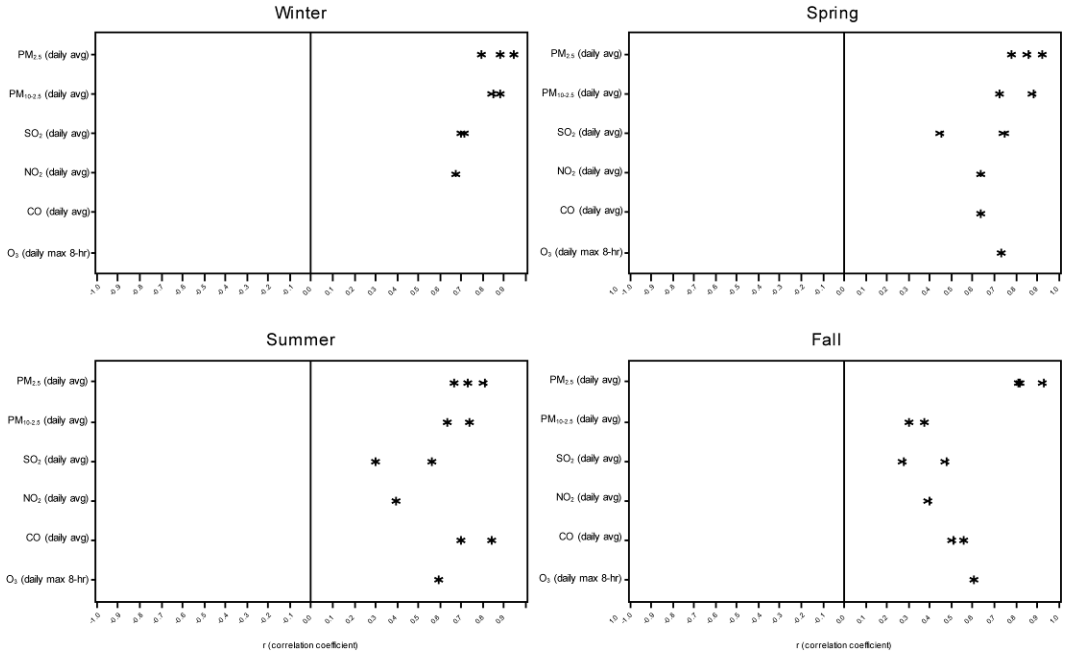

Figure A-173. Correlations between 24-h PM_{10} and co-located 24-h avg $PM_{2.5}$, $PM_{10\text{-}2.5}$ SO_2, NO_2 and CO and daily maximum 8-h avg O_3 for New York City, NY, stratified by season (2005-2007). One point is included for each available monitor pair.

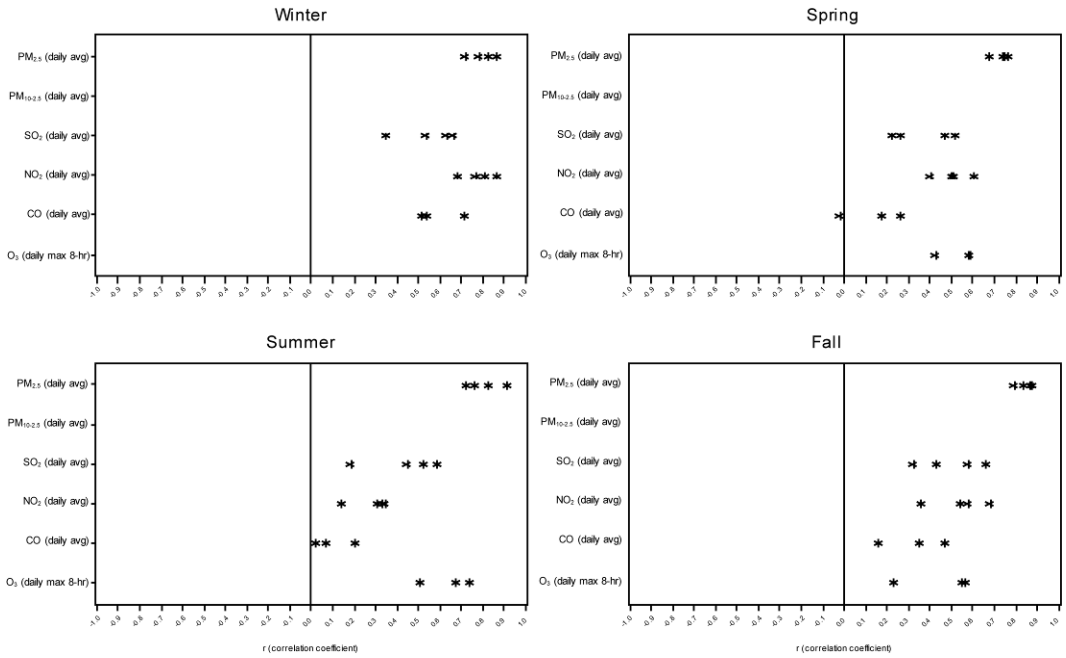

Figure A-174. Correlations between 24-h PM_{10} and co-located 24-h avg $PM_{2.5}$, $PM_{10\text{-}2.5}$ SO_2, NO_2 and CO and daily maximum 8-h avg O_3 for Philadelphia, PA, stratified by season (2005-2007). One point is included for each available monitor pair.

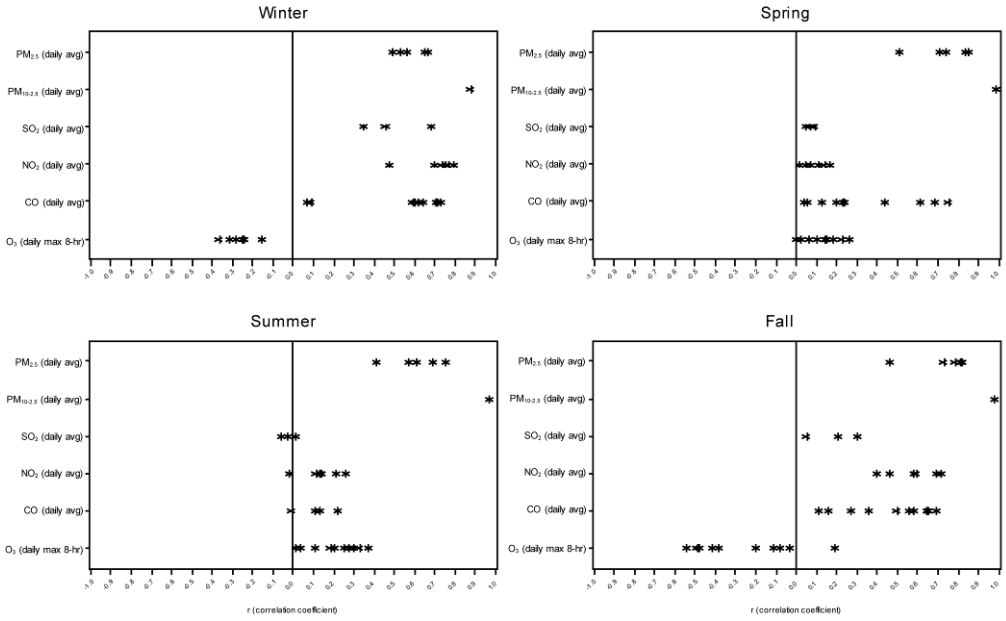

Figure A-175. Correlations between 24-h PM_{10} and co-located 24-h avg $PM_{2.5}$, $PM_{10\text{-}2.5}$ SO_2, NO_2 and CO and daily maximum 8-h avg O_3 for Phoenix, AZ, stratified by season (2005-2007). One point is included for each available monitor pair.

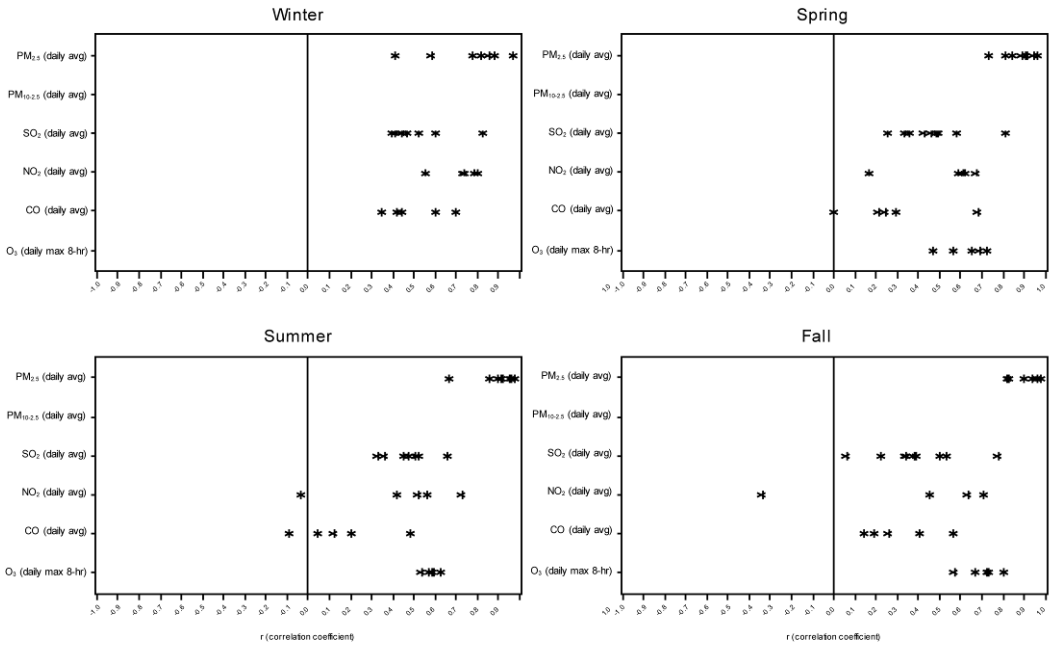

Figure A-176. Correlations between 24-h PM_{10} and co-located 24-h avg $PM_{2.5}$, $PM_{10-2.5}$ SO_2, NO_2 and CO and daily maximum 8-h avg O_3 for Pittsburgh, PA, stratified by season (2005-2007). One point is included for each available monitor pair.

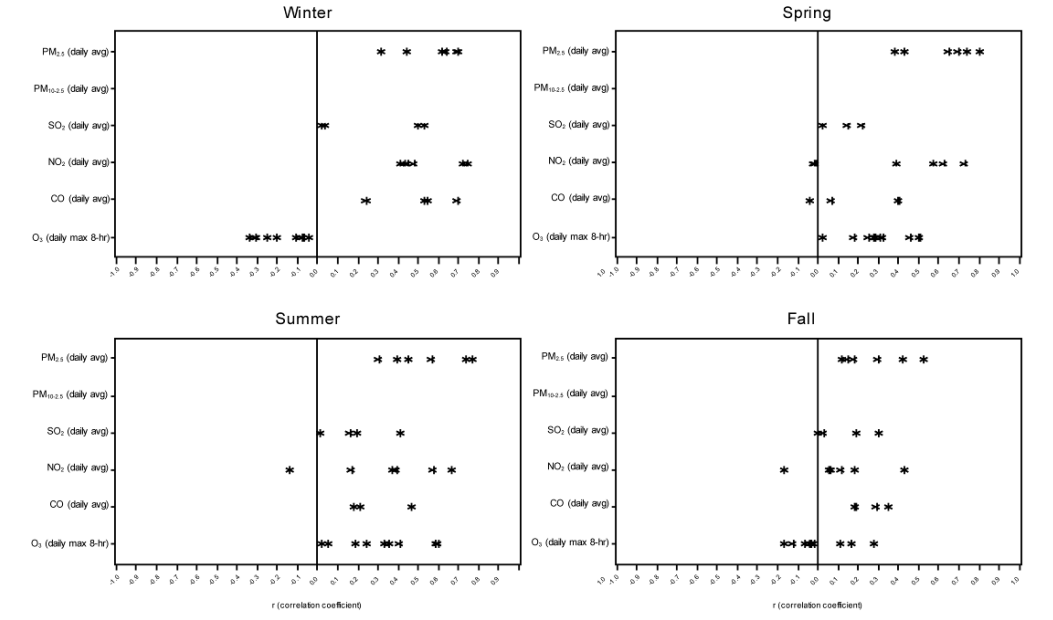

Figure A-177. Correlations between 24-h PM_{10} and co-located 24-h avg $PM_{2.5}$, $PM_{10-2.5}$ SO_2, NO_2 and CO and daily maximum 8-h avg O_3 for Riverside, CA, stratified by season (2005-2007). One point is included for each available monitor pair.

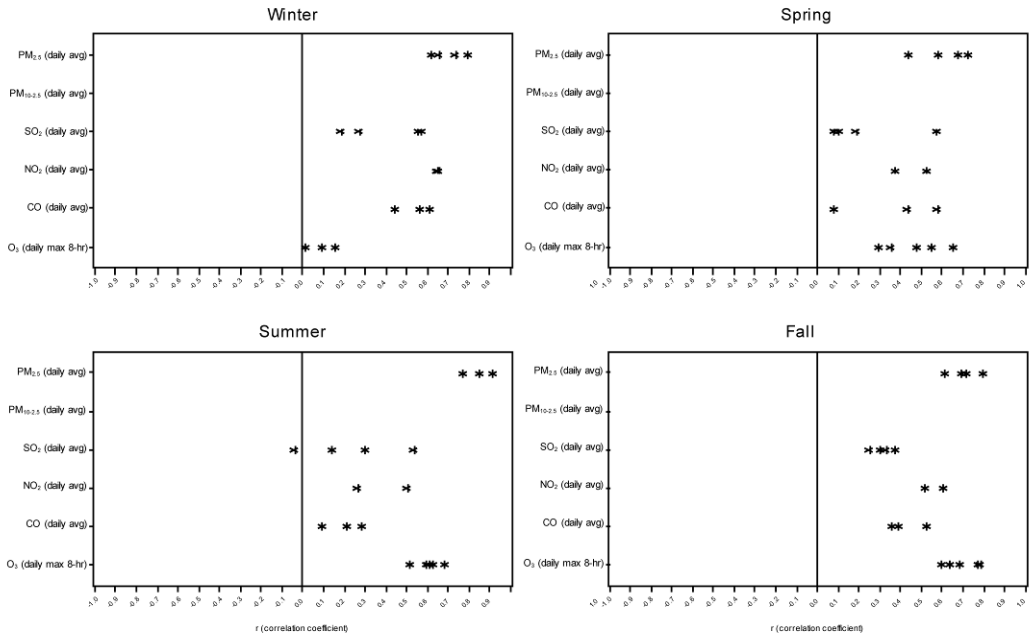

Figure A-178. Correlations between 24-h PM_{10} and co-located 24-h avg $PM_{2.5}$, $PM_{10\text{-}2.5}$ SO_2, NO_2 and CO and daily maximum 8-h avg O_3 for St. Louis, MO, stratified by season (2005-2007). One point is included for each available monitor pair.

A.3. Source Apportionment

A.3.1. Type of Receptor Models

Table A-46. Different receptor models used in the Supersite source apportionment studies: chemical mass balance.

Receptor Model	Description	Strengths and Weaknesses
Effective Variance CMB 42,121 (Note that all models based on eq 1 or 2 are CMB equations. The term CMB used here re□ects the historical solution in which source pro□les are explicitly used as model input and a single sample effective variance solution is reported.) CMB software is currently distributed by EPA. The most recent version is the CMB 8.2, which is run in the Microsoft Windows system.	Principle Ambient chemical concentrations are expressed as the sum of products of species abundances in source emissions and source contributions (eq 1 or 2). These equations are solved for the source contribution estimates when ambient concentrations and source pro□les are input. The single-sample effective variance least squares[122] is the most commonly used solution method because it incorporates uncertainties of ambient concentrations and source pro□les in the estimate of source contributions and their uncertainties. This reduced to the tracer solution when it is assumed that there is one unique species for each source. Choices of source pro□les should avoid collinearity, which occurs when chemical compositions of various source emissions are not sufficiently different.[121] Data Needs CMB requires source pro□les, which are the mass fractions of particulate or gas species in source emissions. The species and particle size fraction measured in source emissions should match those in ambient samples to be apportioned. Several sampling and analysis methods provide time-integrated speciation of $PM_{2.5}$ and volatile organic compounds (VOCs) for CMB. Source pro□les are preferably obtained in the same geographical region as the ambient samples, although using source pro□les from different regions is commonly practiced in the literature. The practitioner needs to decide the source pro□les and species being included in the model, on the basis of the conceptual model and model performance measures. Output Effective variance CMB determines, if converged, source contributions to each sample in terms of PM or VOC mass. CMB also generates various model performance measures, including correlation R2, deviation $□^2$, residue/ uncertainty ratio, and MPIN matrix that are useful for re□ning the model inputs to obtain the best and most meaningful source apportionment resolution.	Strengths Software available providing a good user interface. Provides quantitative uncertainties on source contribution estimates based on input concentrations, measurement uncertainties, and collinearity of source pro□les. Quanti□es contributions from source types with single particle and organic compound measurements. Weaknesses Completely compatible source and receptor measurements are not commonly available. Assumes all observed mass is due to the sources selected in advance, which involves some subjectivity. Chemically similar sources may result in collinearity without more speci□c chemical markers. Typically does not apportion secondary particle constituents to sources. Must be combined with pro□le aging model to estimate secondary PM.

[42] Hidy and Friedlander (1972, 156546)
[121] Watson et al. (1997, 157121) [122](1984, 045693)

Source: Watson et al. (2008, 157128)

Table A-47. Different receptor models used in the Supersites source apportionment studies: factor analysis.

Receptor Model	Description	Strengths and Weaknesses
PMF123,124 PMFx (PMF2 and PMF3) software is available from Dr. Pentti Paatero at the University of Helsinki, Finland. This software is a Microsoft DOS application. EPA distributes EPA PMF76 version 1.1 as a Microsoft Windows application with better user interface.	Principle PMFx contains PMF2 and PMF3. PMF2 solves the CMB equations (i.e., eqs 2 and 3) using an iterative minimization algorithm. Source pro□les F_{ij} and contribution S_{jt} are solved simultaneously. The non-negativity constraint is implemented in the algorithm to decrease the number of possible solutions (local minimums) in the PMF analyses, because both source pro□le and contribution should not contain negative values. There is rotational ambiguity in all two-way factor analyses (i.e., F_{ij} and S_{jt} matrices may be rotated and still □t the data). PMF2 allows using the FPEAK parameter to control the rotation. A positive FPEAK value forces the program to search such solutions where there are many zeros and large values but few intermediate values in the source matrix F_{ij}.F_{key} can further bind individual elements in F_{ij} to zero. On the basis of a similar algorithm, PMF3 solves a three-way problem. PMFx and UNMIX estimate F_{ij} and S_{jt} by minimizing: $$Q \text{ or } \chi^2 = \sum_i \sum_t [E_{it}/\sigma_{it}]^2 = \sum_i \sum_t [(C_{it} - \sum_j F_{ij}S_{jt})/\sigma_{it}]^2$$ (A-1) Where the weighing factor, σ_{it}, represents the magnitude of E_{it}, PMFx limits solutions of eq 2 to non-negative F_{ij} and S_{jt}. Data Needs A large number of ambient samples (usually much more than the number of factors in the model) are required to produce a meaningful solution. Species commonly used in PMF are also those in CMB. Weighting factors associated with each measurement need to be assigned before analysis. The practitioner also needs to decide the number of factors, FPEAK, and F_{key} in the model. Output PMFx reports all the elements in F_{ij} and S_{jt} matrices (PMF2). It also calculates model performance measures such as deviation $□^2$ and standard deviation of each matrix element. The practitioner needs to interpret the results linking them to source pro□les and source contributions.	Strengths Software available. Can handle missing or below-detection-limit data. Weights species concentrations by their analytical precisions. Downweight outliers in the robust mode. Derives source pro□les from ambient measurements as they would appear at the receptor (does not require source measurements). Weaknesses Requires large (> 100) ambient datasets. Need to determine the number of retaining factors. Requires knowledge of source pro□les or existing pro□les to verify the representativeness of calculated factor pro□les and uncertainties of factor contributions. Relies on many parameters/initial conditions adjustable to model input; sensitive to the preset parameters.
ME2125 ME2 code is available from Dr. Pentti Paatero at the University of Helsinki, Finland as a Microsoft DOS application.	Principle The PMFx algorithm is derived from ME2. Unlike PMFx that is limited to questions in the form of eq 1 or 2, ME2 solves all models in which the data values are □tted by sums of products of unknown (and known) factor elements. The □rst part of the algorithm interprets instructions from the user and generates a table that speci□es the model. The second part solves the model using an iterative minimization approach. Additional constraints could be programmed into the model to reduce the ambiguity in source apportionment. These constraints may include known source pro□les and/or contributions (e.g., contributions are known to be zero in some cases). Data Needs Data needs are similar to those of PMFx but are more □exible. In theory, any measured or unknown variables may be included in the model as long as they satisfy linear relationships. The users need to specify the model structure, the input, and the output. Output ME2 calculates and reports all unknown variables in the model.	Strengths Software available. Can handle user-speci□ed models. Possibility to include all measured variables into the model, such as speciated concentration over different time scales, size distributions, meteorological variables, and noise parameters. Weaknesses Require substantial training to access the full feature of the software and develop a model. Generally requires large ambient datasets. Need to assume linear relationships between all variables. Relies on many parameters/initial conditions adjustable to model input; sensitive to the preset parameters.

Receptor Model	Description	Strengths and Weaknesses
UNMIX [29,44,126] UNMIX code is available from Dr. Ron Henry at the University of Southern California as an MatLab application. A stand-alone version (UNMIX version 6) is also available from EPA.	Principle UNMIX views each sample as a data point in a multidimensional space with each dimension representing a measured species. UNMIX solves eqs 2 and 3 by using a principle component analysis (PCA) approach to reduce the number of dimensions in the space to the number of factors that produce the data, followed by an unique "edge detection" technique to identify "edges" de□ned by the data points in the space of reduced dimension (e.g., Figures 1 and 3). The number of factors is estimated by the NUMFACT algorithm in advance127, which reports the R2 and signal-to-noise (S/N) ratio associated with the □rst N principle components (PCs) in the data matrix. The number of factors should coincide with the number of PCs with S/N ratio >2. Once the data are plotted on the reduced space, an edge is actually a hyperplan that signi□es missing or small contribution from one or more factors. Therefore, UNMIX searches all the edges and uses them to calculate the vertices of the simplex, which are then converted back to source composition and contributions. Geometrical concepts of self-modeling curve resolution are used to ensure that the results obey (to within error) non-negativity constraints on source compositions and contributions. Data Needs A large number of ambient samples (usually much more than the number of factors in the model) are required to achieve a meaningful solution. Species commonly used in UNMIX are also those in CMB. The measurement precision is not required. The practitioner needs to specify the number of factors on the basis of the NUMFACT results. Output UNMIX determines all the elements in the factor (Fij) and contribution (Sjt) matrices. It also calculates the uncertainty associated with the factor elements and model performance measures including: (1) R2, (2) S/N ratio, and (3) strength.	Strengths Software available with graphical user interface. Does not require source measurements. Provide graphical problem diagnostic tools (e.g., species scatter plot). Provide evaluation tools (e.g., R2, S/N ratio). Weaknesses Requires large (>100) ambient datasets. Need to assume or predetermine number of retained factors. Does not make explicit use of errors or uncertainties in ambient measurements. Cannot use samples containing missing data in any species. Limited to a maximum of 7 or 14 (UNMIX version 6) factors. Can report multiple or no solutions. Requires knowledge of existing source pro□les to evaluate the solutions.

Receptor Model	Description	Strengths and Weaknesses
PDRM[97] PDRM was developed under the Supersites Program and requires MatLab or equivalent software to perform the calculation.	Principle PDRM estimates contributions from selected stationary sources for a receptor site using high time-resolution measurements and meteorological data. In PDRM, eq 2 is modi□ed to: $$C_k = \sum_j ER_{ij}\left(\frac{\chi}{Q}\right)_{j,t} + E_k \quad \text{(A-2)}$$ where $ER_{i,j}$ is interpreted as the emission rate of species i from stationary source j and (χ/Q)$_{j,t}$ is the meteorological dispersion factor averaged over the time interval t. Equation 4 is solved for $ER_{i,j}$ and (χ/Q)$_{j,t}$ simultaneously by a nonlinear □t minimizing the objective function, FUN: $$FUN = \sum_{i=1}^{I}\sum_{t=1}^{m}\sum_{j=1}^{n}\left[ER_{ij}\left(\frac{\chi}{Q}\right)_{j,t}^{PDRM} - C_k\right]^2 \quad \text{(A-3)}$$ Because the number of solutions for a product of unknowns is in□nite, additional constraints are set up for (χ/Q)$_{j,t}$ on the basis of the Gaussian plume model, thus: $$LB\left(\frac{\chi}{Q}\right)_{j,t}^{Met} \le \left(\frac{\chi}{Q}\right)_{j,t}^{PDRM} \le UB\left(\frac{\chi}{Q}\right)_{j,t}^{Met}$$ $$\left(\frac{\chi}{Q}\right)_{j,t}^{Met} = \frac{1}{2\pi\sigma_y\sigma_z u}\exp\left(-\frac{1}{2}\frac{y^2}{\sigma_y^2}\right)\left\{\exp\left[-\frac{1}{2}\left(\frac{z-h}{\sigma_z}\right)^2\right] + \exp\left[-\frac{1}{2}\left(\frac{z+h}{\sigma_z}\right)^2\right]\right\} \quad \text{(A-4)}$$ Eqs 6 and 7 limit the solution of eq 5 within the lower (LB) and upper (UB) bound of those predicted by the Gaussian plume model using different parameterizations. Data Needs PDRM requires speciated measurements at a higher time-resolution than typical CMB or PMF applications because of the fast-changing meteorological parameters. PDRM also requires data for eq 7: transport speed (u), lateral and vertical dispersion parameters (σ_Y and σ_z), and stack height (h). Output PDRM determines emission rates and contributions from each point source considered in the model at the same time resolution as the measurement.	Strengths Explicitly include meteorological information and stack con□guration of stationary sources into the model. Do not require source measurements. Do not need to interpret the relations between factors and sources. Commercial software (e.g., MatLab) available for performing nonlinear □t. Suitable for high time-resolution measurement. Weaknesses Can only handle stationary sources but not area or mobile sources. Need to assume that only stationary sources are considered in the model contribute signi□cantly for a measurement at the receptor site. Do not account for uncertainty in the measurement. Meteorological data may not be always available or accurate. Gaussian plume model may not be representative of the actual atmospheric dispersion. Sensitive to the imposed constraints (UB and LB).

Receptor Model	Description	Strengths and Weaknesses
PLS[128]	Principle PLS examines the relationships between a set of predictor (independent) and response (dependent) variables. It assumes that the predictor and response variables are controlled by independent "latent variables" less in number than either the predictor or the response variables. In recent applications,[96] PM chemical composition and size distribution are used as predictor (X) and response (Y) variables, respectively. Eq 2 is modified to: $$X_k = \sum_i T_i P_{ik} + E_k \quad \text{(A-5)}$$ $$Y_{ik} = \sum_i U_i C_{ik} + D_k \quad \text{(A-6)}$$ where T and U are matrices of so-called "latent variables," and P and C are loading matrices. If X and Y are correlated to some degree, T and U would show some similarity. Equations 8 and 9 are solved by an iterative algorithm "NIPALS," which attempts to minimize E,D, and the difference between T and U simultaneously. If T and U end up being close enough, the X and Y variables can be explained by the same latent variables. These latent variables may then be interpreted as source or source categories. Data Needs Typical applications of PLS require both chemical speciated and size-segregated measurements. The practitioner needs to decide the number of latent variables on the basis of the correlation of resulting T and U matrices. Output PLS calculates latent variables, which are common factors best explaining the predictor and response variables, and the residues from fitting. R_x and R_y, $$R_x = 1 - \text{var}(E)/\text{var}(X) \quad \text{(A-7)}$$ $$R_y = 1 - \text{var}(D)/\text{var}(Y) \quad \text{(A-8)}$$ indicate the degree to which variables X and Y are explained by the latent variables.	Strengths Fit two types of measurements (e.g., chemistry and size) with common factors. Provide more information to identify sources. Analyze strongly collinear and noisy dataset. Do not require source measurements. Weaknesses Requires large (> 100) ambient datasets. Difficult to relate latent variables to any physical quantities. Do not provide quantitative source contribution estimates. Need to decide the number of latent variables. Do not explicitly make use of measurement uncertainties. Can result in no solution.

[29] Henry (1997, 020941)
[44] Lewis et al. (2003, 088413)
[96] Ogulei et al. (2006, 119975)
[97] Park et al. (2005, 156844)
[123] Paatero (1997, 087001)
[124] Paatero et al. (2002, 156836)
[125] Paatero (1999, 156835)
[128] Henry (2003, 156540)

Source: Watson et al. (2008, 157128)

Table A-48. Different receptor models used in the Supersites source apportionment studies: tracer-based methods.

Receptor Model	Description	Strengths and Weaknesses
EF [129,130] The EF method may use a MLR algorithm, which is available in most statistical and spreadsheet software	Principle A tracer (or marker) for a particular source or source category is a species enriched heavily in the source emission against other species and other sources. Using EFs-, concentration of the ith pollutant at a receptor site at time t (i.e., $C_{i,t}$) can be expressed as: $$C_{i,t} = \sum_{j} \frac{1}{EF_{i,pj}} C_{pj,t} + Z_{i,t} = \sum_{j} \left(\frac{F_i}{F_{pj}}\right) C_{pj,t} + Z_{i,t} \quad \text{(A-7)}$$ where the enrichment factor $EF_{i,pj}$ is the ratio of emission rate of the pollutant of interest (F_{ij}) and tracer species (F_{pj}) from source j. $C_{pj,t}$ is the concentration of tracer species for source j at time t, and $Z_{i,t}$ represents contributions from all other sources (including the background level). The solution for eq 12 is situation-dependent. $EF_{i,pj}$ is usually unknown but may be estimated from source pro□les, edges of a two-way scatter plot (e.g., Figures 1 and 3), or the ratio of $C_{i,t}$ to $C_{pj,t}$ for a particular period when it is believed that a single source is dominant. In cases where $Z_{i,t}$ is a constant, $EF_{i,pj}$ may be derived from MLR. Data Needs The minimum data needs include concentrations of all primary tracers at the receptor site. Known EFs or background levels are helpful. Output The EF method determines contributions to species i from each source considered in the model.	Strengths No special software needed. Indicate presence or absence of particular emitters. Provides evidence of secondary PM formation and changes in source impacts by changes in ambient composition. Could use a large (>100) dataset or a small (e.g., <10) dataset. Weaknesses Semiquantitative method, not speci□c especially when the EFs are unknown in advance. Limited to sources with unique markers. Tracer species must be exclusively from the sources or source categories examined. Provide very limited error estimates. More useful for source/process identi□cation than for quanti□cation.
NNLS [131,132] The MatLab Optimization Toolbox provides a function "lsqnonneg" for performing the NNLS calculation.	Principle NNLS also solves the EF equation (eq 12 or equivalent) with known target species and tracer concentrations. Conventional MLR solutions to eq 12 may lead to negative EFs due to the uncertainty in measurements or colinearity in source contributions. This is avoided in the NNLS approach since additional non-negative constraints are built into the algorithm, i.e.: $$EF_{i,pj} \geq 0 \quad \text{(A-8)}$$ Utilizing orthogonal decomposition, a NNLS problem can be reduced to the more familiar least-distance programming and solved by a set of iterative subroutines developed and tested by Lawson and Hanson.[131] In a more general sense, NNLS linearly relates a response variable to a set of independent variables with only non-negative coef□cients. Data Needs When applied to EF or MLR problems, NNLS requires the concentration of target (response) and tracer (independent) species. Output NNLS generates non-negative regression coef□cients for an EF/MLR problem and these coef□cients can be related to the source contributions.	Strengths Implemented by many statistical software packages. Generate only non-negative EFs or regression coef□cients. Do not require source measurements. Possible to include meteorological or other (besides chemistry) data into the model. Weaknesses Require a large (>100) set of ambient measurements. Semiquantitative method, not speci□c. Do not explicitly consider measurement uncertainties. Tracer species must be exclusively from the sources or source categories examined. Non-negative constraints may not be appropriate in some cases.

Receptor Model	Description	Strengths and Weaknesses
FAC[111]	Principle FAC provides a simple mean of estimating the SOA production rate using the emission inventories of primary precursor VOCs. FAC is actually a source-oriented modeling technique but it does not take into account all the atmospheric processes. FAC is de□ned as the fraction of SOA that would result from the reactions of a particular VOC: $$[\text{SOA}] = \sum_i FAC_i \times ([\text{VOC}_i]_0 \times \text{Fraction of VOC } i \text{ reacted}) \quad \text{(A-9)}$$ where $[\text{VOC}_i]_0$ is the emission rate of VOC_i and [SOA] is the formation rate of SOA. Equation 14 can be viewed as an extension of eq 12 but concentrations are replaced with emission rates and EFs are replaced with FACs. FAC and the fraction of VOC reacted under typical ambient conditions have been developed for a large number of hydrocarbons $> C_6$.[111] The most signi□cant SOA precursors are aromatic compounds (especially toluene, xylene, and trimethylbenzenes) and terpenes. In most applications, these FACs are used directly to estimate SOA. Data Needs FAC requires the VOC emission inventory in the region of interest. The knowledge of O_3 and radiation intensity is also helpful for slight modi□cations of the FACs. Output FAC method estimates the total production rate of SOA.	Strengths Link SOA to primary VOC emissions so that SOA can also be treated as primary particles in the PM modeling. Simple and inexpensive. Weaknesses Ignore the in□uence of aerosol concentration and temperature-dependent gas-particle partitioning on SOA yield. Limited by the accuracy of VOC emission inventory. Do not directly infer the contribution of each source to ambient SOA concentration. Dif□cult to verify.

[111]Grosjean and Seinfeld (1989, 045643)
[129]Darns et al. (1970, 156379)
[130]Reimann and De Caritat (2000, 013269)
[131]Lawson and Hanson (1974)
[132] Wang and Hopke (1989)

Source: Watson et al. (2008)

Table A-49. Different receptor models used in the Supersites source apportionment studies: meteorology-based methods.

Receptor Model	Description	Strengths and Weaknesses
CPF [134,135]	Principle CPF estimates the probability that a given source contribution from a given wind direction will exceed a predetermined threshold criterion (e.g., upper 25th percentile of the fractional contribution from the source of interest). The calculation of CPF uses source contributions (i.e., S_{jt} in eq 2) determined for the receptor site and local wind direction data matching each of the source contributions in time. These data are then segregated to several sectors according to wind direction and the desired resolution (usually 36 sectors at a 10° resolution). Data with very low wind speed (e.g., $<$ 0.1 m/sec) are usually excluded from analysis because of the uncertain wind direction. CPF is then determined by: $$CPF(\theta) = \frac{m_{\Delta\theta}}{n_{\Delta\theta}} \quad \text{(A-10)}$$ where $m_{\Delta\theta}$ is the number of occurrences in the direction sector □ □ □ + □□ that exceeds the speci□ed threshold, and $n_{\Delta\theta}$ is the total number of wind occurrences in that sector. Because wind direction is changing rapidly, high-time resolution measurements (e.g., minutes to hours) are preferred for a CPF analysis. If the calculated source contributions represent long-term averages, wind direction needs to be averaged over the same duration. In addition to source contribution, CPF can be applied directly to pollutant concentration measurements at a receptor site. Data Needs CPF requires the time series of source contributions at a receptor site, which is usually determined by CMB or factor analysis methods using speciated measurements at the site. CPF also requires wind direction and wind speed data averaged over the same time resolution as the sampling duration. Output CPF reports the probability of "high" contribution from a particular source or factor occurring within each wind direction sector. The results are often presented in a wind rose plot (e.g., Figure 6a).	Strengths Infer the direction of sources or factors relative to the receptor site. Provide veri□cation for the source identi□cation made by factor analysis method. Easy to implement. Weaknesses Criterion for the threshold is subjective. Absolute source contribution (or fractional contribution) may be in□uenced by other factors besides wind direction (e.g., wind speed, mixing height). Local and near-surface wind direction only has a limited implication for long-range transport. Easy to be biased by a small number of wind occurrences in a particular sector. Work better for stationary sources than area or mobile sources.

Receptor Model	Description	Strengths and Weaknesses
NPR [136,137]	Principle NPR calculates the expected (averaged) source contribution as a function of wind direction following: $$S(\theta) = \frac{\sum_i K\left(\frac{\theta - W_i}{\Delta\theta}\right) \times S_i}{\sum_i K\left(\frac{\theta - W_i}{\Delta\theta}\right)} \quad \text{(A-11)}$$ where W_i is the wind direction for the ith sample and S_i is the contribution from a speci□c source to that sample, determined from measurements at the receptor site. K is a weighting function called the kernel estimator. There are many possible choices for K. Henry et al.[136] recommend either Gaussian or Epanechnikov functions. The most important decision in NPR is the choice of the smoothing parameter □□. If □□ is too large, S(□) will be too smooth and meaningful peaks could be lost. If it is too small, S(□) will have too many small, meaningless peaks. □□ needs to be chosen according to the project-speci□c spatial distribution of sources. NPR also estimates the con□dence intervals of S(□) based on the asymptotic normal distribution of the kernel estimates, thus: $$\Delta S(\theta) = \frac{\sum_i K\left(\frac{\theta - W_i}{\Delta\theta}\right) \times (S_i - S(\theta))^2}{\left(\sum_i K\left(\frac{\theta - W_i}{\Delta\theta}\right)\right)^2} \quad \text{(A-12)}$$ Data Needs NPR requires the same data as the CPF method, including the time series of source/factor contributions (or fractional contributions) at the receptor site and local wind direction data matching the sampling duration in time. Output NPR reports the distribution of source contribution as a function of wind direction and the con□dence level associated with it.	Strengths Infer the direction of sources or factors relative to the receptor site. Provide veri□cation for the source identi□cation made by factor analysis method. Require no assumption about the function form of the relationship between wind direction and source contribution. Provide uncertainty estimates. Easy to implement. Weaknesses Choices for the kernel estimator and smoothing factor are subjective. Absolute source contribution (or fractional contribution) may be in□uenced by other factors besides wind direction (e.g., wind speed, mixing height). Local and near-surface wind direction only has a limited implication for long-range transport. Easy to be biased by a small number of wind occurrences in a particular sector. Work better for stationary sources than area or mobile sources.

Receptor Model	Description	Strengths and Weaknesses
TSA [138] TSA requires the calculation of air parcel back trajectory, which is often accomplished using the HY-SPLIT model.115,139 HY-SPLIT version 4.5 is available at http://www.arl.noaa.gov/-ready/hysplit4.html.	Principle Similar to CPF, TSA clusters the measured pollutant concentration or calculated source contribution according to the wind pattern. However, air parcel back trajectory, rather than local wind direction, is used. A back trajectory traces the air parcel backward in time from a receptor. The initial height is often between 200 and 1000 m above ground level where the wind direction could differ from the surface wind direction substantively. For each sample i, TSA obtains one or more trajectories and calculates their total residence time in the jth directional sector (τ_{ij}, i.e., the total number of 1-h trajectory end points that fall into the sector). The pollutant concentration or source contribution in the sample, S_i is then linearly apportioned into each directional sector according to τ_{ij} and averaged over all samples to produce the directional dependent pollutant concentration/source contribution for the period of interest: $$\overline{S_j} = \sum_i S_i \left(\frac{\tau_{ij}}{\sum_i \tau_{ij}} \right) / N \quad \text{(A-13)}$$ where N is the number of samples. Compared with CPF and NPR, TSA considers the entire air mass history rather than just the wind direction at the receptor. Data Needs TSA requires the time series of pollutant concentration or source contribution at the receptor site, and back trajectories initiated over the site during the sampling duration. Trajectory is usually calculated once every hour so TSA is more suitable for analyzing measurements of $>$1-h resolution. Output TSA reports the avg pollutant concentration or source contribution as a function of wind direction based on back trajectory calculations.	Strengths Infer the direction of sources or factors relative to the sampling site. Provide veri□cation for the source identi□cation made by factor analysis method. Account for air mass transport over hundreds to thousands of kilometers and on the order of several days. Can represent plume spread from vertical wind shear at different hours of day by adjusting the initial height of back trajectories. Weaknesses Need to generate and analyze the back trajectory data. Uncertainty in back trajectory calculation increases with its length in time. Source contribution depends on not only trajectory residence time but also entrainment ef□ciency, dispersion, and deposition. Dif□cult to resolve the direction of more localized sources.

Receptor Model	Description	Strengths and Weaknesses
PSCF [140] PSCF requires the calculation of air parcel back trajectory, which is often accomplished using the HY-SPLIT model.115,139 HY-SPLIT version 4.5 is available at http://www.arl.noaa.gov/-ready/hysplit4.html.	Principle Ensemble air parcel trajectory analysis refers to the statistical analysis on a group of trajectories to retrieve useful patterns regarding the spatial distribution of sources. Uncertainties associated with individual trajectory calculations largely cancel out for a sufficient number of trajectories or trajectory segments. As a popular ensemble back trajectory analysis, PSCF estimates the probability that an upwind area contributes to high pollutant concentration or source contribution. Back trajectories are first calculated for each sample at the receptor site. To determine the PSCF, a study domain containing the receptor site is divided into an array of grid cells. Trajectory residence time (the time it spends) in each grid cell is calculated for all back trajectories and for a subset of trajectories corresponding to "high" pollutant concentration or source contribution at the site. PSCF in cell (i,j) is then defined as: $$PSCF_{ij} = \frac{\text{Sum of "high" residence time in cell } (i, j)}{\text{Sum of all residence time in cell } (i, j)} \quad \text{(A-14)}$$ The criterion for high pollutant concentration or source contribution is critical for the PSCF calculation. The 75th or 90th percentile of the concentration or factor is often used.[113,141,142] Residence time can be represented by the number of trajectory end points in a cell. Data Needs Similar to TSA, PSCF calculation requires the time series of pollutant concentration or source contribution at the receptor site, and back trajectories initiated over the site during the sampling period. Trajectories should be calculated with 1-to 3-h segment to reduce the uncertainty from interpolation (if needed). Output PSCF reports the probability that an upwind area contributes to high pollutant concentrations or source contribution at the downwind receptor site. The results are often presented as a contour plot on the map. A high probability usually suggests potential source region (e.g., Figure 4b).	Strengths Infer the location of sources or factors relative to the sampling site. Provide verification for the source identification made by factor analysis method Account for air mass transport over hundreds to thousands of kilometers and on the order of several days. Resolve the spatial distribution of source strength (qualitatively). Weaknesses Need to generate and analyze the back trajectory data. Need to correct for the central tendency (residence time always increases toward the receptor site regardless of source contribution). Uncertainty in back trajectory calculation increases with its length in time. Source contribution depends on not only trajectory residence time but also entrainment efficiency, dispersion, and deposition. Difficult to resolve the location of more localized sources.

Receptor Model	Description	Strengths and Weaknesses
SQTBA [117, 143] SQTBA requires the calculation of air parcel back trajectory, which is often accomplished using the HY-SPLIT model.115,139 HY-SPLIT version 4.5 is available at http://www.arl.noaa.gov/ready/hysplit4.html.	Principle SQTBA is another type of ensemble air parcel trajectory analysis. The concept of SQTBA is to estimate the "transport □eld" for each trajectory ignoring the effects of chemical reactions and deposition. Back trajectories are □rst calculated for each sample at the receptor site, and a study domain containing the receptor site is divided into an array of grid cells. SQTBA assumes that the transition probability that an air parcel at (x',y',t'), where x' and y' are spatial coordinates and t' means time, will reach a receptor site at (x,y,t) is approximately normally distributed along the trajectory with a standard deviation that increases linearly with time upwind[144,145], thus: $$Q(x, y, t|x', y', z') = \frac{1}{2\pi(at')^2}\exp\left[-\frac{1}{2}\left(\left(\frac{X - x'(t')}{at'}\right)^2 + \left(\frac{Y - y'(t')}{at'}\right)^2\right)\right] \quad \text{(A-15)}$$ where (X,Y) is the coordinate of the grid center, a is the dispersion speed, and x'(t') and x' (t') represent the trajectory. The probability □eld, Q, for a given trajectory is then integrated over the upwind period, □, to produce a two-dimensional "natural" (nonweighted) transport □eld: $$T_k(x, y|x', y') = \frac{\int_{-\tau}^{0} Q(x, y, t|x', y', z')}{\int_{-\tau}^{0} dt'} \quad \text{(A-16)}$$ After the transport □eld for each trajectory is established, they are weighted by the corresponding pollutant concentration or source contribution at the receptor site and summed to yield the overall SQTBA □eld.[117] Data Needs SQTBA requires the time series of pollutant concentration or source contribution at the receptor site, and back trajectories initiated over the site during the sampling period. Trajectories should be calculated with 1to 3-h segment to reduce the uncertainty from interpolation (if needed). Output SQTBA put more weight on trajectories associated higher pollutant concentration or source contribution and therefore the resulting □eld may imply the major transport path.	Strengths Imply the location of sources or factors relative to the sampling site. Account for air mass transport over hundreds to thousands of kilometers and on the order of several days. Resolve the spatial distribution of source strength (qualitatively). Weaknesses Need to generate and analyze the back trajectory data. Need to correct for the central tendency (residence time always increases toward the receptor site regardless of source contribution). Need to estimate dispersion velocity. Involve complicated calculations. Physical meaning of the SQTBA □eld is unclear. Dif□cult to resolve the location of more localized sources.

Receptor Model	Description	Strengths and Weaknesses
RTWC [146] RTWC requires the calculation of air parcel back trajectory, which is often accomplished using the HY-SPLIT model.115,139 HY-SPLIT version 4.5 is available at http://www.arl.noaa.gov/ready/hysplit4.html	Principle As an ensemble air parcel trajectory analysis, RTWC requires back trajectories calculated for each sample at the receptor site, and a study domain containing the receptor site divided into an array of grid cells. RTWC assumes that no major pollutant sources are located along "clean" (associated with low pollutant concentrations) trajectories and that "polluted" trajectories picked up emissions along their paths. In practice, RTWC distributes pollutant concentrations at the receptor to upwind grid cells along the back trajectories according to the trajectory residence times in those cells.[117,146] $$S_{i,k} = S_k \frac{\text{resident time in cell } i}{\text{average residence time in each cell}} \quad \text{(A-17)}$$ where S_k is the pollutant concentration or source contribution determined upon the arrival of trajectory k and $S_{i,k}$ is the redistributed pollutant concentration or source contribution for cell i upwind. RTWC is known for the problem of "tailing effect," i.e., spurious source areas can be identi□ed when cells are crossed by a very small number of trajectories. Although some corrections were proposed[147] these approaches are purely empirical.	Strengths Imply the location of sources or factors relative to the sampling site. Account for air mass transport over hundreds to thousands of kilometers and on the order of several days. Resolve the spatial distribution of source strength (qualitatively). Weaknesses Need to generate and analyze the back trajectory data. Need to correct for the central tendency and tailing effect. The amount of emission entrainment should not be proportional to the residence time of trajectories (so there is no linear relationship between RTWC □eld and source strength). Physical meaning of the RTWC □eld is unclear. Dif□cult to resolve the location of more localized sources.

113 (Pekney et al., 2006, 086115)
117 (Zhou et al., 2004, 157190)
134 (Ashbaugh, 1983, 156229)
135 (Ashbaugh et al., 1984, 045148)
136 (Henry et al., 2002, 136097)
137 (Yu et al., 2004, 101779)
138 (Parekh and Husain, 1981, 156840)
140 (Hopke et al., 1995, 156566)
143 (Keeler and Samson, 1989, 156633)
144 (Samson, 1978, 156941)
145 (Samson, 1980, 073010)
146 (Stohl, 1996, 157014)
147 (Cheng et al., 1993, 052294)

Source: (Watson et al., 2008, 157128)

A.3.2. Source Profiles

Table A-50. Source Profiles: Part I

Element	Symbol	Motor Vehicle Exhaust - Gasoline		Coal Combustion		Highway Road Dust		Unpaved Road Dust		Refinery	
		Weight %	Uncertainty	Weight %	Uncertainty	Weight %	Uncertainty	Weight %	Uncertainty	Weight %	Uncertainty
Aluminum	Al	0.1	-99	5.968	0.5247	5.729	0.4058	7.4822	0.9315	8.4853	2.3478
Antimony	Sb	0.01	-99	0	0.0625	0	0.0335	0	0.1601	0	0.0285
Arsenic	As			0	0.0164	0	0.0123	0	0.0226	0	0.0045
Barium	Ba	0.01	-99	1.3315	1.0801	0.1377	0.1027	0	0.5473	0	0.0979
Cadmium	Cd			0	0.0341	0	0.019	0	0.0881	0	0.0155
Calcium	Ca	0.42	-99	3.4536	1.0411	2.5657	0.1388	2.163	1.0444	0.1236	0.056
Chloride ion	Cl^-	0.39	-99								
Chromium	Cr	0.01	-99	0.0176	0.0041	0.0271	0.0023	0.0312	0.0161	0.0443	0.0127
Cobalt	Co			0	0.0432	0	0.0668	0	0.0869	0	0.0218
Copper	Cu	0.02	-99	0.0179	0.0112	0.0219	0.0101	0.0474	0.0307	0.0299	0.0082
Total carbon	TC			4.2763	4.2579	14.3927	2.3449	4.2671	3.7193	0	1.6175
Gallium	Ga			0.014	0.014	0	0.005	0	0.0233	0	0.0059
Gold	Au										
Indium	In	0	-99	0	0.0404	0	0.022	0	0.1041	0	0.0183
Iron	Fe	1.27	-99	2.916	0.3827	4.5713	0.2661	5.5128	2.1152	1.4708	0.2216
Lanthanum	La	0	-99	0	0.2462	0	0.1341	0	0.6521	0	0.1146
Lead	Pb	0.08	-99	0.068	0.0336	0.067	0.0074	0.0288	0.0284	0.0097	0.0063
Magnesium	Mg	0.14	-99								
Manganese	Mn	0.01	-99	0.0284	0.0139	0.087	0.009	0.1372	0.0509	0.016	0.002
Mercury	Hg	0	-99	0	0.0154	0	0.0083	0	0.0383	0	0.0073
Molybdenum	Mo			0	0.0134	0	0.0071	0	0.0331	0.0079	0.0088
Nickel	Ni	0.01	-99	0.0072	0.0019	0.0081	0.0015	0.0091	0.0057	0.04	0.0065
Nitrate	NO_3^-	0.06	-99	0	0.2116	0	0.094	0	0.6371	0	0.0772
Organic carbon	OC	59.37	-99	0	2.9263	12.7127	2.1296	4.2671	2.2637	0	1.5288
Palladium	Pd			0	0.0263	0	0.0151	0	0.0701	0	0.0127
Phosphorus	P	0.27	-99	0.9372	0.6322	0	0.0324	0.1603	0.044	0.0689	0.0144
Potassium	K	0.01	-99	0.4644	0.0602	2.7161	0.3069	2.8299	0.4949	0.0825	0.0234
Rubidium	Rb			0.0053	0.0043	0.0184	0.0023	0.0184	0.0093	0	0.002
Selenium	Se			0.0406	0.0407	0	0.0024	0	0.0108	0	0.0021
Silicon	Si	1.61	-99	9.0112	0.5675	17.596	1.4183	24.2969	4.0089	17.9733	5.1834
Silver	Ag			0	0.0312	0	0.0175	0	0.083	0	0.0151
Sodium	Na	0.01	-99								
Strontium	Sr			0.1964	0.0686	0.0395	0.0078	0.0313	0.0112	0.0094	0.0031

		Motor Vehicle Exhaust - Gasoline		Coal Combustion		Highway Road Dust		Unpaved Road Dust		Refinery	
Sulfate	$SO_4^{=}$			10.1716	8.9405	1.1604	0.2003	0.8688	1.3788	2.3243	3.4523
Sulfur	S	0.37	-99	2.948	2.729	0.598	0.0509	0.2808	0.3884	0.6304	0.9627
Thallium	Tl										
Tin	Sn			0	0.0527	0	0.0298	0	0.1464	0	0.0254
Titanium	Ti			0.4315	0.0651	0.3612	0.0313	0.5258	0.1289	0.6178	0.0711
Uranium	U										
Vanadium	V			0	0.0734	0.0288	0.0074	0	0.0646	0.0432	0.0084
Yttrium	Y			0	0.006	0.0046	0.0012	0	0.0146	0	0.0029
Zinc	Zn	0.49	-99	0.0797	0.0341	0.0932	0.0256	0.0502	0.021	0.0166	0.003
Zirconium	Zr			0.0247	0.0043	0.0128	0.0025	0.0219	0.0168	0.0166	0.0022
Ammonium	NH_4^+	0.34	-99	0.3476	0.1352	0	0.025	0	0.1317	0.3281	0.5565
Sodium ion	Na^+										
Carbonate	CO3 =										
Organic carbon II	OC2										
Organic carbon III	OC3										
Organic carbon IV	OC4										
EC I	EC1										
Chlorine atom	Cl^-			0.0629	0.0221	3.4403	0.5505	0.1519	0.0755	0.0186	0.0074
EC III	EC3										
EC	EC	16.44	-99	4.2763	3.0931	1.68	0.9817	0	2.9512	0	0.5283
Bromine Atom	Br			0.0147	0.0154	0.0037	0.0011	0	0.0078	0	0.0017
Organic carbon I	OC1										
EC II	EC2										
Sulfur dioxide	SO_2			7262.6687	7677.5681						
Potassium ion	K^+			0.1109	0.0571	0.2295	0.1046	0.1263	0.0744	0.0115	0.0059

Source: USA EPA Speciate database http://www.epa.gov/ttnchie1/software/speciate/index.html

Part II

Element	Symbol	Residential Wood Burning		Oil Combustion		DE		Fly Ash		Incinerator	
		Weight %	Uncert-ainty	Weight %	Uncert-ainty	Weight %	Uncert-ainty	Weight %	Uncert-ainty	Weight %	Uncert-ainty
Aluminum	Al	0.0034	0.0103	0	0.05	0	0.01	1.5708	0.4755	1.15	0.83
Antimony	Sb	0.0002	0.0108	0	0.01	0	0.01	0.007	0.0218	0.01	0.15
Arsenic	As	0.0003	0.0016	0.02	0	0	0	0.001	0.0023	0	0.04
Barium	Ba	0.0093	0.0369	0	0.03	0.01	0.04	0.0303	0.0655	0.14	0.55
Cadmium	Cd	0.0013	0.0058	0	0.01	0	0.01	0	0.0154	0.01	0.08
Calcium	Ca	0.0664	0.0165	0	0.04	0.01	0.01	10.1398	1.7825	2.37	0.62
Chloride ion	Cl^-	0.0028	0.0004					17.5498	1.5419		

		Residential Wood Burning		Oil Combustion		DE		Fly Ash		Incinerator	
Chromium	Cr	0.0003	0.0012	0.01	0.01	0	0	0.0054	0.001	0.02	0.02
Cobalt	Co	0.0005	0.0005	0.05	0.01	0	0	0.0015	0.0128	0	0.03
Copper	Cu	0.0002	0.0007	0.01	0.01	0	0	0.017	0.0013	0.08	0.1
Total carbon	TC	70.6416	7.1435	3.55	1.0855	98.94	17.859	1.4329	0.2009	55.79	27.5948
Gallium	Ga	0	0.0016	0.01	0	0	0	0.0013	0.0018	0	0.02
Gold	Au							0.0008	0.0033		
Indium	In	0.0021	0.0069	0	0.01	0	0.01	0	0.0164	0.01	0.1
Iron	Fe	0.0038	0.0017	0.68	0.1	0	0	0.8306	0.059	1.72	0.31
Lanthanum	La	0.0086	0.0431	0	0.04	0.02	0.05	0.0046	0.0868	8.43	61.15
Lead	Pb	0.0031	0.0018	0	0	0	0	0.0031	0.0031	14.56	11.69
Magnesium	Mg							0.4455	0.0465		
Manganese	Mn	0.003	0.0013	0	0	0	0	0.0426	0.0033	0.04	0.01
Mercury	Hg	0.0004	0.0027	0	0	0	0	0.0008	0.0025	27.63	47.27
Molybdenum	Mo	0	0.0024	0	0	0	0	0.0041	0.001	0.01	0.04
Nickel	Ni	0.0002	0.0005	2.36	0.23	0	0	0.0028	0.0004	0.01	0
Nitrate	NO_3^-	0.2025	0.0156	0	0	0.06	0.01	0	0.2192	5.5	4.55
Organic carbon	OC	49.4961	5.481	1.71	0.56	90.8	14.79	1.4329	0.1592	37.21	18.03
Palladium	Pd	0.0006	0.0047	0	0	0	0	0	0.0126	0.02	0.07
Phosphorus	P	0	0.0051	0	0.65	0.01	0.02	0.5808	0.2447	0.05	0.16
Potassium	K	0.6346	0.1008	0	0	0	0	24.4341	5.0076	1.28	0.86
Rubidium	Rb	0.0007	0.0007	0	0	0	0	0.0351	0.0026	0	0.02
Selenium	Se	0.0001	0.0008	0.03	0	0	0	0.0018	0.0003	0.01	0.01
Silicon	Si	0.0443	0.0167	0	0.09	0.01	0.01	4.0201	1.2886	4.42	1.82
Silver	Ag	0.0023	0.0054	0	0	0	0.01	0	0.0143	0.02	0.08
Sodium	Na							2.8137	0.2174		
Strontium	Sr	0.0006	0.0009	0	0	0	0	0.0406	0.0029	0.02	0.01
Sulfate	SO_4^{2-}	0.4553	0.0359	25.29	5.62	0.53	0.07	8.0717	0.6409	10.46	2.6
Sulfur	S	0.1533	0.0173	16.48	1.62	0.59	0.21	2.6349	0.1873	3.16	0.63
Thallium	Tl							0.0011	0.0025		
Tin	Sn	0.0006	0.0092	0	0.01	0	0.01	0.0067	0.0198	0.04	0.14
Titanium	Ti	0.001	0.012	0.01	0.01	0	0.01	0.058	0.0093	0.11	0.17
Uranium	U							0.0021	0.0052		
Vanadium	V	0.0007	0.005	0.4	0.04	0	0.01	0.0038	0.011	0.01	0.07
Yttrium	Y	0.0001	0.0011	0	0	0	0	0.0013	0.0021	0	0.02
Zinc	Zn	0.0762	0.0054	0.01	0	0.02	0.02	0.031	0.0023	0.57	0.39
Zirconium	Zr	0	0.0014	0	0	0	0	0.0039	0.0008	0	0.02
Ammonium	NH_4^+	0.1132	0.014	0.84	0.24	0.03	0.01	0.0234	0.022	7.41	7.81
Sodium ion	Na^+			0.11	0.02	0	0.01	4.7518	0.3438	1.81	2.63
Carbonate	CO3 =			0	0.0214	0.2577	0.4463				

		Residential Wood Burning		Oil Combustion		DE		Fly Ash		Incinerator	
Organic carbon II	OC2	7.513	0.6675								
Organic carbon III	OC3	8.9627	1.4665								
Organic carbon IV	OC4	2.7683	1.1919								
EC I	EC1	20.342	2.9324								
Chlorine atom	Cl^-	0.2874	0.0404	0.05	0.01	0.03	0.01	27.5797	8.1193	6.35	10.46
EC III	EC3	2.2878	0.4252								
EC	EC	21.1455	4.5813	1.84	0.93	8.14	10.01	0	0.1227	18.58	20.89
Bromine Atom	Br	0.0029	0.0011	0	0	0	0	0.0441	0.0032	0.19	0.3
Organic carbon I	OC1	25.1452	4.6648								
EC II	EC2	2.9362	1.2422								
Sulfur dioxide	SO_2										
Potassium ion	K^+	0.5208	0.0795	0.01	0.01	0	0.01	14.5473	1.3393	1.01	0.42

Source: U.S. EPA SPECIATE database http://www.epa.gov/ttnchie1/software/speciate/index.html

A.3.3. Receptor Model Results

Table A-51. PM_{10} receptor model results

	% Contribution						
Sampling Site	Wood Smoke	Diesel	Gasoline Vehicles	Natural Gas Combustion	Vegetative Detritus	Tire Wear Debris	Total % Allocated
Apline, CA, 1994-1995	15.00	33.19	46.46		5.31	6.91	99.955752
Apline, CA, 1995	9.92	58.78	11.47		19.63	9.43	99.795918
Apline, CA, 1995	10.97	65.64	10.81		12.66	5.31	100.07722
Atascadero, CA, 1994-1995	44.22	22.16	26.44				99.733333
Atascadero, CA, 1995	21.36	38.99	12.41		17.89		100.08772
Atascadero, CA, 1995	73.45	18.11			3.14		100.01241
Lake Arrowhead, CA, 1994-1995	6.86	46.55	33.92	2.73	9.85	20.42	99.896907
Lake Arrowhead, CA, 1995	4.85	65.20	7.40	4.95	17.65		100.04902
Lake Arrowhead, CA, 1995	9.91	38.90	46.70	0.79	3.66	28.01	99.955947
Lake Elsinore, CA, 1994-1995	12.72	44.01	18.61		4.21	9.78	99.967638
Lake Elsinore, CA, 1995	17.13	74.72		0.26	7.81	29.17	99.924528
Lake Elsinore, CA, 1995[2]	6.84	38.48	10.85	0.21	15.55	11.93	99.946809
Lancaster, CA, 1994-1995	22.49	43.14	20.56	0.45	3.73	26.38	100.14006
Lancaster, CA, 1995	3.69	46.18	12.66	0.20	8.21		100.09967
Lancaster, CA, 1995	34.89	37.30	7.33	0.61	7.78		99.839228
Lompoc, CA, 1994-1995		18.16	49.65		5.89	26.00	100.07092
Lompoc, CA, 1995	13.09	51.27	14.73		20.73	14.11	99.818182

	% Contribution						
Lompoc, CA, 1995		79.42	10.19		10.87	16.61	100.48077
Long Beach, CA, 1994-1995	10.12	43.24	16.49	0.13	3.97	19.52	99.955423
Long Beach, CA, 1995	2.38	70.25	5.47	0.86	6.79	15.71	99.865643
Long Beach, CA, 1995	14.32	56.80	6.15	0.72	5.34	9.85	99.939832
Mira Loma, CA, 1994-1995	4.68	48.87	18.10		8.82	20.31	100
Mira Loma, CA, 1995	5.20	53.72	6.65		18.79	19.06	100.07092
Mira Loma, CA, 1995	27.97	41.88	8.87		11.50	20.17	100.07519
Riverside, CA, 1994-1995	14.14	46.67	12.03		6.83		99.972222
Riverside, CA, 1995	6.20	52.15	7.93	0.16	14.54	7.85	100.0409
Riverside, CA, 1995	25.28	47.65			6.91	8.15	100
San Dimas, CA, 1995	7.62	71.35	4.87	0.15	8.35	12.78	100.17308
San Dimas, CA, 1995	22.01	61.34	4.48	0.23	3.70	15.05	99.919463
Santa Maria, CA, 1994-1995	18.66	23.99	22.03		5.58	14.70	100.14493
Santa Maria, CA, 1995	12.94	52.57	11.87	0.27	9.63	11.25	100.05348
Santa Maria, CA, 1995	12.24	48.13	10.79	0.47	18.04	9.81	104.71963
Upland, CA, 1994-1995	20.33	46.39	14.08		4.49		100
Upland, CA, 1995	7.33	68.69	3.50	0.17	9.19		100.12891
Upland, CA, 1995	28.10	46.52	4.90	0.33	10.30		99.952774

Source: Manchester-Neesvig et al. (2003, 098102)

Table A-52. $PM_{2.5}$ receptor model results

Sampling Site	Measured $PM_{2.5}$ Concentration	Vegetative Burning	Road Dust, Soil	$(NH_4)_2SO_4$	NH_4NO3	NaCL	Tailpipe	Brake Wear	Total % Allocated
Albany, NY 2000-2004	34.9	7.60	11.70	2.70	4.90	11.70	2.90		118.91
Birmingham, AL, 2000-2004	24.1	3.90	8.40	3.70	2.70	0.10	5.70		101.66
Houston, TX, 2000-2004	17.6	3.10	6.90	1.60	2.50	0.10	3.80		106.25
Long Beach, CA, 2000-2004	46.8	4.60	9.60	2.10	18.90	0.80	6.50	3.50	98.29

Source: Abu-Allaban et al. (2007, 098575)

A.4. Exposure Assessment

A.4.1. Exposure Assessment Study Findings

Table A-53. Exposure Assessment Study Summaries

Abou Chakra et al. (2007, 098819)

Study Design	Experimental, in vitro. HeLa cells incubated with organic extracts of personal PM_{10} and $PM_{2.5}$ samples
Period	NR
Location	3 French metropolitan areas (Paris, Rouen, Strasbourg) with varying air quality and emission sources
Population	Individuals from urban areas with varying air pollution levels and emissions sources
Age Groups	Children ages 6-13. Ages of adults not given
Indoor Source	NR
Personal Method	Harvard multi-pollutant Chempass personal exposure sampler placed in backpacks with BGI pump operating at 1.8 l/min.
Personal Size	PM_{10}, $PM_{2.5}$
Microenvironment Size	NR
Ambient Size	NR
Component(s)	Organic extracts of PM_{10}, organic extracts of $PM_{2.5}$
Primary Findings	Genotoxic effects were stronger for organic extracts of $PM_{2.5}$ than for PM_{10} and greater in winter than summer. Greater effects for winter samples may be attributed to elevated winter PAH concentrations.

Abu-Allaban et al. (2004, 156187)

Study Design	Exposure assessment of real world motor vehicle emissions
Period	May 18-22, 1999
Location	Tuscarosa Mountain Tunnel, Pennsylvania Turnpike
Population	Highway tunnel
Age Groups	NR
Indoor Source	NR
Personal Method	No personal exposure assessment was conducted.
Personal Size	NR
Microenvironment Size	0.01-0.5 μm
Ambient Size	NR
Component(s)	NR
Primary Findings	Monitoring sessions with the highest fraction heavy-duty vehicles had the highest particle concentrations; Observed particle size distribution was a combination of 2 bimodal log-normal distributions a dominant nucleation mode (86% of area under the curve).

Adar et al. (2007, 098635)

Study Design	Cohort
Period	March 2002-June 2002
Location	St. Louis, Missouri
Population	Senior citizens exposed to traffic-related PM
Age Groups	60
Indoor Source	NR
Personal Method	Samples of FeNO were collected between 8:00 and 9:00 a.m. on the mornings before and after each trip. In the hours surrounding these samples, group-level measurements of particle concentrations also were collected using several continuous instruments installed on two portable carts. These carts were first positioned in a central location inside the participants' living facilities 24-h before each trip. The carts remained at the facilities until it was time for the trips, at which point they followed the participants from the health testing room, onto the bus, to the group activity, and to lunch. After the trip home aboard the bus, the carts were returned to the central location in the living facility where they remained until the conclusion of the health testing on the following morning. Continuous measurements of ambient particles and gases also were collected from a central monitoring station in East St. Louis, Illinois.; Specifics-; Two portable carts containing continuous air pollution monitors were used to measure group-level micro-environmental exposures to traffic related pollutants, including fine particulate mass ($<$ 2.5 µm aerodynamic diameter; $PM_{2.5}$), BC, and size-specific particle counts. $PM_{2.5}$ concentrations were measured continuously using a DustTrak aerosol monitor model 8520 with a Nafion diffusion dryer. Integrated samples of $PM_{2.5}$ mass also were collected using a Harvard Impactor for daily calibration of the trip and facility (missing items?)
Periods	Continuous BC concentrations were measured using a portable aethalometer with a 2.5-µm impaction inlet. Particle counts were measured using a model CI500 optical particle counter with a modified flow rate of 0.1 cubic feet per minute.
Personal Size	NR
Microenvironment Size	$PM_{2.5}$

Ambient Size $PM_{2.5}$; PM_{10}
Component(s) BC; Pollen and Mold also assessed
Primary Findings Fine particle exposures resulted in increased levels of FeNO in elderly adults, suggestive of increased airway inflammation. These associations were best assessed by microenvironmental exposure measurements during periods of high personal particle exposures.In pre-trip samples, both microenvironmental and ambient exposures to fine particles were positively associated with FeNO. For example, an interquartile increase of 4 $\mu g/m^3$ in the daily microenvironmental $PM_{2.5}$ concentration was associated with a 13% [95% CI: 2-24) increase in FeNO. After the trips, however, FeNO concentrations were associated predominantly with microenvironmental exposures, with significant associations for concentrations measured throughout the whole day. Associations with exposures during the trip also were strong and statistically significant with a 24% (95% CI: 15-34) increase in FeNO predicted per interquartile increase of 9 $\mu g/m^3$ in $PM_{2.5}$. Although pre-trip findings were generally robust and the post-trip findings were generally robust, the post-trip findings were sensitive to several influential days.

Adgate et al. (2002, 030676)

Study Design Comparison of outdoor, indoor and personal $PM_{2.5}$ in three communities.
Period April-June, June-August, September-November, 1999
Location Battle Creek, East St. Paul, and Phillips, Minnesota, constituting the Minneapolis-St. Paul metropolitan area.
Population adults in urban areas
Age Groups mean age 42 ± 10, range 24-64 yr
Indoor Source No
Personal Method Inertial impactors (PEM) in a foam-insulated bag with shoulder strap with the inlet mounted on the front.
Personal Size $PM_{2.5}$
Microenvironment Size $PM_{2.5}$
Ambient Size $PM_{2.5}$
Component(s) NR
Primary Findings The relative level of concentrations report in other studies was duplicated. Outdoor < indoor < personal. On days with paired samples (n = 29), outdoor concentrations were significantly lower (mean difference 2.9 $\mu g/m^3$, p = 0.026) than outdoor at home.

Adgate et al. (2007, 156196)

Study Design NR
Period 1999-; April 26-June 20, June 21-August 11, September 23-November 21
Location Minneapolis-St. Paul metropolitan area
Population NR
Indoor Source Cigarette smoke, resuspension of house dust from carpets, furniture and clothes, and emissions from stoves and kerosene heaters (Leaderer et al., 1993; Ferro et al., 2004).
Personal Method Personal monitoring was conducted using a and consisted of two consecutive days, and was conducted so that the two 24-h averages matched indoor (I) and personal (P) measurements were collected in concert with O samples in each community. Gravimetric concentrations for P and I were collected using inertial impactor environmental monitoring inlets and air sampling pumps. To obtain I measurements, monitors were placed inside each residence in a room where the participants reported spending the most waking hours. P measurements were obtained by carrying personal pumps in small bags.; Outdoor central site samples (O) were collected near the approximate geographic center of each neighborhood and monitors ran from midnight to midnight for two consecutive 24-h periods, followed by a day to change filters. Gravimetric O $PM_{2.5}$ concentrations were obtained using a federal reference method sampler.
Personal Size $PM_{2.5}$-broken down into TE
Microenvironment Size $PM_{2.5}$-broken down into TE
Ambient Size $PM_{2.5}$-broken down into TE
Component(s) Ag, Al, Ca, Cd, Co, Cr, Cs, Cu, Fe, K, La, Mg, Mn, Na, Ni, Pb, S, Sb, Sc, Ti, Tl, V, Zn
Primary Findings The relationships among P, I, and O concentrations varied across TEs. Unadjusted mixed-model results demonstrated that O monitors are more likely to underestimate than overestimate exposure to many of the TEs that are suspected to play a role in the causation of air pollution related health effects. These data also support the conclusion that TE exposures are more likely to be underestimated in the lower income and centrally located PHI community than in the comparatively higher income BC K community. Within the limits of statistical power for this sample size, the adjusted models indicated clear seasonal and community related effects that should be incorporated in long-term exposure estimates for this population.

Adgate et al. (2003, 040341)

Study Design Time-series epidemiologic study
Period April-November 1999; spring: 26 April-20 June; summer: 21 June-11 August; fall: 23 September-21 November
Location Minneapolis-St. Paul, Minnesota
Population Healthy non-smoking results
Age Groups 24-64 yr (mean age 42 ± 10)
Indoor Source NR

Personal Method Personal and indoor gravimetric PM concentrations were collected using $PM_{2.5}$ inertial impactor environmental monitoring inlets and air sampling pumps. Monitors were placed inside each participant's residence in the room where he/she reported spending the majority of their waking hours to obtain indoor (I) measurements. Participants also carried personal pumps in small bags to obtain personal (P) measurements. Start times for indoor and personal monitors were always within a few minutes of each other. Gravimetric outdoor (O) and central site $PM_{2.5}$ concentrations were obtained using a federal reference method sampler and EPA site requirements for ambient sampling. Gravometric samples were collected near the approximate geographic center of each neighborhood, and monitors ran from midnight to midnight for 2 consecutive 24-h periods, followed by a day to change filters.

Personal Size NR

Microenvironment Size NR

Ambient Size NR

Component(s) NR

Primary Findings Personal (P) $PM_{2.5}$ concentrations were higher than indoor (I) concentrations, which were higher than outdoor (O) concentrations. In healthy non-smoking adults, moderate median PI; modest median IO; and minimal median PO longitudinal correlation coefficients were observed for $PM_{2.5}$ measurements. A sensitivity analysis indicated that correlations did not increase if the days with exposures to environmental tobacco smoke or occupational exposures were excluded. In the sample population neither P nor I monitors provided a highly correlated estimate of exposure to O $PM_{2.5}$ over time. These results suggest that the studies showing relatively strong longitudinal correlation coefficients between P and O $PM_{2.5}$ for individuals sensitive to air pollution health effects do not necessarily predict exposure to $PM_{2.5}$ in the general population.

Alander et al. (2004, 055650)

Study Design Exposure assessment, characterization of effects of fuel reformulation, engine design, and exhaust after-treatment on PM emissions

Period NR

Location Laboratory

Population Diesel-powered passenger cars of different engine types and different formulations of diesel fuel

Age Groups NR

Indoor Source NR

Personal Method No personal exposure assessment was conducted

Personal Size NR

Microenvironment Size NR

Ambient Size NR

Component(s) Total carbon, organic carbon, elemental carbon, sulfate, nitrate, chloride

Primary Findings Reformulated low sulfur diesel fuel produced 40% less total carbon mass compared to standard fuel. Organic carbon constituted 27-61% carbon mass from an indirect ignition engine. Low sulfur fuel reduced organic carbon mass by 10-55%, depending on engine.

Allen et al. (2003, 053578)

Study Design Use of continuous light scattering data to separate indoor PM into indoor- and outdoor-generated components to enhance knowledge of the outdoor contribution to total indoor and personal PM exposures.

Period November 1999-May 2001

Location Seattle, WA

Population Elderly people and children spending most of their time (up tp 70%) indoors.; The study included healthy elderly subjects, elderly with COPD and coronary heart disease (CHD), and child subjects with asthma.

Age Groups Age n; 0-29 25; 30-59 36; > 60 22; unknown 2

Indoor Source Suggested (not identified)

Personal Method NR. Indoor and outdoor sampling conducted

Personal Size NR

Microenvironment Size $PM_{2.5}$

Ambient Size $PM_{2.5}$

Component(s) Sulfur

Primary Findings A recursive mass balance model can be successfully used to attribute indoor PM to its outdoor and indoor components and to estimate an avg P, a, k, and NH_4^+ for each residence.

Allen et al. (2007, 154226)

Period Heating season October-February; Non-heating season March-September; (Year not specified)

Location Seattle, WA

Population NR

Age Groups NR

Indoor Source NR

Personal Method Indoor and outdoor $PM_{2.5}$ was measured using a 10-l/min single-stage Harvard Impactor (HI) with 37-mm Teflon filters. The relationship between particle mass concentration and light scattering coefficient (bsp) was also measured on a continuous basis indoors and outdoors using nephelometers (model 902 and 903).

Personal Size NR

Microenvironment Size $PM_{2.5}$

Ambient Size $PM_{2.5}$

Component(s) Sulfur (measured by XRF)

Primary Findings The authors showed that RM can reliably estimate F_{inf}. Simulation results suggest that the RM F_{inf} estimates are minimally impacted by measurement error. In addition, the average light scattering response per unit mass concentration was greater indoors than outdoors. Results show that the RM method is unable to provide satisfactory estimates of the individual components of F_{inf}. Iliu et ndividual homes vary in their infiltration efficiencies, thereby contributing to exposure misclassification in epidemiologic studies that assign exposures using ambient monitoring data. This variation across homes indicates the need for home-specific estimation methods, such as RM or sulfur tracer, instead of techniques that give average estimates of infiltration across homes.

Allen et al. (2007, 156207)

Study Design Primarily a study of exposure to indoor PBDE congeners.
Period Jan-Mar 2006
Location Greater Boston area, Massachusetts
Population Urban dwellers
Age Groups NR
Indoor Source NR
Personal Method No particulate sampled
Personal Size No particulate sampled
Microenvironment Size No particulate sampled
Component(s) Polybrominated diphenyls (PBDEs), divided into 13 congeners and total BDE (SBDE), which includes both vapor and particulate phase.
Primary Findings Total personal air concentrations (particulate ± vapor) were 469 pg/m^3 for non-209 BDEs and 174 pg/m^3 for BDE 209, significantly higher than bedroom and main living room concentrations ($p = 0.01$). The ratio of personal air to room air increased from 1 for vapor-phase congeners to 4 for fully particulate-bound congeners, indicating a personal cloud effect.

Andresen et al. (2005, 156216)

Study Design Residential exposure assessment personal and indoor
Period June-July 2002 to December 2002
Location Mysore, India
Population Women working at home, non-smoking, and primary household cook
Age Groups 18-50 yr old
Indoor Source Cooking fuel source
Personal Method $PM_{2.5}$ gravimetric filter measurement
Personal Size $PM_{2.5}$
Microenvironment Size NR
Ambient Size NR
Component(s) NR
Primary Findings Using kerosene for cooking was associated with higher personal $PM_{2.5}$ exposure in both winter and summer compared to LPG.; Kerosene use during winter was associated with higher personal $PM_{2.5}$ compared to summer.; LPG use was associated with comparable personal $PM_{2.5}$ across both seasons.; Indoor $PM_{2.5}$ measurements followed similar patterns by fuel-type and season.; Socioeconomic status, age, season, and income were significant predictors of cooking fuel choice.

Annesi-Maesano et al. (2007, 093180)

Study Design Population based
Period March 1999 to October 2000
Location Bordeaux, France; Clermont-Ferrand, France; Créteil, France; Marseille, France; Strasbourg, France; Reims, France
Population School children
Age Groups 10.4 ± 0.7 yr
Indoor Source NR
Personal Method $PM_{2.5}$ was monitored simultaneously in both schoolyards (proximity level) and fixed-site monitoring stations (city level) using 4L/min battery operated pumps attached to polyethylene filter sampling cartridges.
Personal Size NR
Microenvironment Size NR
Ambient Size $PM_{2.5}$
Component(s) NR
Primary Findings Results show an increased risk for EIB and flexural dermatitis at the period of the survey, past year atopic asthma and SPT positivity to indoor allergens in children exposed to high levels of traffic-related air pollution ($PM_{2.5}$ concentrations exceeding 10 μg/m^3). Population based findings are also consistent with experimental data that have demonstrated that inhalation of traffic-related air pollutants either individually or in combination, can enhance the immune responses and airway response to inhaled allergens, such as pollens or house dust mites, in atopic subjects.

Balakrishnan et al. (2002, 156247)

Study Design Exposure assessment
Period July-December 1999 (20 weeks)
Location 50 villages, Tamil Nadu, India
Population Men and women in rural households; children exempt

Age Groups All, children exempt
Indoor Source Yes
Personal Method Personal sampler for cooks during cooking time
Personal Size Respirable Particulates (based on NIOSH protocol)
Microenvironment Size Respirable Particulates (based on NIOSH protocol)
Ambient Size Respirable Particulates (based on NIOSH protocol)
Component(s) NR
Primary Findings Fuel type, type and location of the kitchen, and the time spent near the kitchen while cooking are the most important determinants of exposure across rural households.

Balasubramanian and Lee (2007, 156248)

Study Design Case study of 3 rooms of 1 flat on the 8th floor, and "outside the home."
Period May 12-23, 2004
Location Singapore
Population Residents of an urban area in a densely populated country.
Age Groups NR
Indoor Source Time-activity logs identified tobacco smoking, cooking, household cleaning and general resident movements.
Personal Method NR
Personal Size NR
Microenvironment Size $PM_{2.5}$
Ambient Size $PM_{2.5}$
Primary Findings Indoor/outdoor ratios (I/O) suggest that chemicals such as chloride, sodium aluminum, cobalt, copper, iron, manganese, titanium vanadium, zinc, elemental carbon were derived from the migration of outdoor particles (I/O < 1 or ~ 1).

Barn et al. (2008, 156252)

Study Design Measure indoor infiltration factor (FiaC) of $PM_{2.5}$ from forest fires/wood smoke, effectiveness of high-efficiency particulate air (HEPA) filter air cleaners in reducing indoor $PM_{2.5}$, and to analyze the home determinants of Fia and air cleaner effectiveness (ACE).
Period 2004-2005 (summer 2004 and 2005, winter 2004)
Location British Columbia, Canada
Population homes affected by either forest fire smoke or residential wood smoke
Age Groups NR
Indoor Source NR
Personal Method Pdr (Personal Data Ram) for ambient air sampling
Personal Size Indoor home $PM_{2.5}$
Microenvironment Size NR
Ambient Size Outdoor home $PM_{2.5}$
Component(s) NR
Primary Findings Use of HEPA filter air cleaners can dramatically reduce indoor $PM_{2.5}$ concentrations. Number of windows and season predict indoor infiltration Fia ($p < 0.001$).

Baxter et al. (2007, 092726)

Study Design Part of a prospective birth cohort study performed by the Asthma Coalition for Community, Environment, and Social Stress (ACCESS)
Period 2003-2005. Non-heating season- May to October; Heating season- December to March
Location Boston (urban)
Population Lower socio-economic status (SES) households
Age Groups NR
Indoor Source NR
Personal Method $PM_{2.5}$ samples were collected with Harvard personal environmental monitors (PEM).; NO concentrations were measured using Yanagisawa passive filter badges.
Personal Size NR
Microenvironment Size $PM_{2.5}$
Ambient Size $PM_{2.5}$
Component(s) EC
Primary Findings The authors' regression models indicated that $PM_{2.5}$ was influenced less by local traffic but had significant indoor sources, while EC was associated with local traffic and NO_2 was associated with both traffic and indoor sources. However, local traffic was found to be a larger contributor to indoor NO_2 where traffic density is high and windows are opened, whereas indoor sources are a larger contributor when traffic density is low or windows are closed. Similarly, traffic contributed up to 0.2 mg/m^3 to indoor EC for homes with open windows, with an insignificant contribution for homes where windows were closed.; Comparing models based on p-values and using a Bayesian approach yielded similar results, with traffic density volume within a 50m buffer of a home and distance from a designated truck route as important contributors to indoor levels of NO_2 and EC, respectively. However, results from the Bayesian approach also suggested a high degree of uncertainty in selecting the best model. The authors concluded that by utilizing public databases and focused questionnaire data they could identify important predictors of indoor concentrations for multiple air pollutants in a high-risk population.

Baxter et al. (2007, 092725)

Study Design	Simultaneous indoor and outdoor samples taken in 43 low SES homes in heating and non-heating seasons. Homes were selected from a prospective birth cohort study of asthma etiology (n = 25). Non-cohort homes were in similar neighborhoods (n = 18).
Period	2003-2005
Location	Boston, Massachusetts
Population	Lower SES populations in urban areas
Indoor Source	Home type, year built, tobacco smoke, opening windows, time spent cooking, use of candles or air freshener, cleaning activities, air conditioner use.
Personal Method	NR
Personal Size	NR
Microenvironment Size	$PM_{2.5}$
Ambient Size	NR
Component(s)	EC (m^{-1} x 10^{-5}); Ca (ng/m^3); Fe (ng/m^3); K (ng/m^3); Si (ng/m^3); Na (ng/m^3); Cl (ng/m^3); Zn (ng/m^3); S (ng/m^3); V (ng/m^3)
Copollutant(s)	NO_2
Primary Findings	The effect of Indoor Sources may be more pronounced in high-density multi-unit dwellings. Cooking times, gas stoves, occupant density and humidifiers contributed to indoor pollutants.

BéruBé et al. (2004, 189731)

Study Design	6 homes in Wales and Cornwall were monitored four times per year, inside samples in the living areas and outside the home.
Period	NR but < 2003
Location	Wales and Cornwall, UK
Population	urban, suburban, and rural homes
Indoor Source	Tobacco smoke, pets, cleaning, traffic load
Personal Method	NR
Personal Size	NR
Microenvironment Size	PM_{10} mass
Ambient Size	NR
Component(s)	NR
Primary Findings	There are greater masses of PM_{10} indoors, and that the composition of the indoor PM_{10} is controlled by outdoor sources and to a lesser extent by indoor anthropogenic activities, except in the presence of tobacco smokers. The indoor and outdoor PM_{10} collected was characterized as being a heterogeneous mixture of particles (soot, fibers, sea salt, smelter, gypsum, pollen and fungal spores).

Branis et al. (2005, 156290)

Study Design	Human exposure assessment in a university lecture hall
Period	Oct. 8, 2001-Nov. 11, 2001
Location	Prague, Czech Republic
Population	University students
Age Groups	NR
Indoor Source	Presence of people identified as a source of course ($PM_{2.5}$-10) particles; outdoor air identified as a source of indoor fine particles ($PM_{1.0}$ and $PM_{2.5}$)
Personal Method	Harvard impactors with membrane Teflon filters
Personal Size	PM_1, $PM_{2.5}$, PM_{10}
Microenvironment Size	PM_1, $PM_{2.5}$, PM_{10}
Ambient Size	PM_{10}
Component(s)	NR
Primary Findings	Presence of people is an important source of coarse particles indoors; Outdoor air may be an important source of fine indoor particles

Brauer et al. (2006, 090757)

Study Design	Cohort study of otitis media and traffic related air pollution
Period	Dec. 1997-Jan 1999
Location	Netherlands and Munich, Germany
Population	Children living near high traffic roads
Age Groups	0-2 yr
Indoor Source	Environmental tobacco smoke at home, gas cooking, indoor moulds and dampness, number of siblings, breast-feeding, and pets indoor.
Personal Method	NR
Personal Size	NR
Microenvironment Size	NR
Ambient Size	$PM_{2.5}$; Light absorbing carbon
Component(s)	Light absorbing carbon
Copollutant(s)	NO_2
Primary Findings	These findings indicate an association between exposure to traffic-related air pollutants and the incidence of otitis media.

Brauer et al. (2007, 090691)

Study Design Cohort study from birth to 5 yr Exposure obtained from stationery monitors identified as closest to birth home.
Location The Netherlands
Population Children
Age Groups 0-5 yr
Indoor Source NR
Personal Method NR
Personal Size NR
Microenvironment Size $PM_{2.5}$ and Soot/filter absorbance
Copollutant(s) NO_2
Primary Findings Adjusted odds ratios for wheeze, doctor-diagnosed asthma, ENT infections and flu indicated positive associations with air pollution. No associations for eczema and bronchitis. Findings at age 4 confirm findings at age 2 in the cohort.

Brunekreef et al. (2005, 090486)

Study Design Exposure assessment
Period Winter and spring 1998-1999
Location Amsterdam and Helsinki
Population Elderly
Age Groups 50-84 yr
Indoor Source NR
Personal Method Amsterdam Gillian with made to fit bags with belt with GK2.05 cyclone samplers 4L/min; Helsinki BGI with shoulder strap or backpack with GK2.05 cyclone samplers 4L/min
Personal Size $PM_{2.5}$
Microenvironment Size $PM_{2.5}$
Ambient Size $PM_{2.5}$
Component(s) Sulfate
Primary Findings In both cities personal and indoor $PM_{2.5}$ were lower than highly correlated with outdoor concentrations. For most elements, personal and indoor concentrations were also highly correlated with outdoor concentrations.

Cao et al. (2005, 156321)

Study Design Case study: 2 roadside homes (RS), 2 urban (UR), 2 rural (RU).
Period March-April 2004
Location Hong Kong, China
Population All
Age Groups NR
Indoor Source NR
Personal Size $PM_{2.5}$
Microenvironment Size $PM_{2.5}$
Component(s) OC, EC
Primary Findings $PM_{2.5}$ concentrations were roadside >urban >rural. Indoor $PM_{2.5}$ has an avg of 24.4-36.8% OC and 3.6-6.9% EC.

Chakrabarti et al. (2004, 147867)

Study Design This is an evaluation of the active-flow pDR for $PM_{2.5}$ against the Attenuation Monitor (BAM) and the gravimetric pDR
Period NR
Location Los Angeles, California
Population NR
Age Groups NR
Indoor Source NR
Personal Method NR
Personal Size NR
Microenvironment Size NR
Ambient Size $PM_{2.5}$
Component(s) NR
Primary Findings The personal pDR can be deployed as a personal monitor. The $PM_{2.5}$ cyclone prevents larger particles from biasing the results. Along with a wearable humidity and temperature monitor for correcting the readings, the results correlate highly with other methods. The samples can be taken every 15 min to provide a more accurate picture of personal exposure in various settings.

Chang et al. (2007, 156331)

Study Design Panel study
Period 2003 to 2005
Location Taipei County, Taipei
Population Elderly people

Age Groups 53-75yr (median = 66.2 ± 6.5)
Indoor Source NR
Personal Method Personal exposures to PM were measured simultaneously with ECG in real-time for twenty-four hours by using a personal dust monitor (DUSTcheck portable dust monitor, model 1.108) which recorded 1-min mass concentrations of PM_1, $PM_{2.5}$, and PM_{10}, as well as ambient temperature and relative humidity. To measure subjects' personal PM exposures, all subjects were instructed to keep the DUSTt check monitor with them at all times.; Details were reported previously (Chuang et al. 2005)
Personal Size PM_{10}; $PM_{2.5-10}$; $PM_{2.5}$; $PM_{1-2.5}$; PM_1
Microenvironment Size NR
Ambient Size NR
Component(s) NR
Primary Findings Short-term and medium-term PM exposures were associated with the reduction of HRV in the elderly, with stronger effects found for coarse particles in comparison with particles of other size ranges. In general, increase was observed with PM for H and the LF/HF ratio, where the strongest significant effects on H were found at short-term intervals (1-4 h) for $PM_{2.5-10}$ and at medium-term duration (5–8 h) for particles smaller than 2.5 lm in diameter. On the other hand, among the different-sized particles, $PM_{2.5-10}$ exposures showed the strongest significant association with decreases in time-domain (SDNN, r- MSSD) and frequency-domain parameters (LF, HF) in most averaging Periods. Especially for the longer duration of 5–8 h, the strongest association was found for the 6-h moving average of $PM_{2.5-10}$ exposures.

Charron et al. (2007, 156333)

Study Design Environmental PM exposure assessment. In this article, a total of 185 days with daily PM_{10} concentrations exceeding the limit value of 50 µg/m³ measured between January 2002 and December 2004 are discussed.
Period January 2002 and December 2004
Location Marylebone Road, Westminster, London
Population NR
Age Groups NR
Indoor Source NR
Personal Method NR
Personal Size NR
Microenvironment Size $PM_{2.5}$ PM_{10}
Ambient Size NO_3^-; SO_4^{2-}; OC; EC
Copollutant(s) NO_X; CO
Primary Finding(s) The regional background was the largest contributor to PM_{10} concentrations measured at Marylebone Road between January 2002 and December 2004

Chillrud et al. (2004, 054799)

Study Design Repeated measures on a cohort of high school students in New York City
Period Summer and winter of 1999 (eight weeks each)
Location Manhattan, Bronx, Queens, Brooklyn, NY
Population Persons traveling the subway
Age Groups 14-18 yr
Indoor Source No
Personal Method Sampling packs carried by subjects
Personal Size $PM_{2.5}$
Microenvironment Size $PM_{2.5}$; Home indoor and home outdoor
Ambient Size $PM_{2.5}$. Urban fixed-site and upwind fixed site operated for three consecutive 48-h periods each week.
Component(s) Elemental iron, manganese, and chromium are reported in this study out of 28 elements sampled.
Primary Findings Personal samples had significantly higher concentration of iron, manganese, and chromium than home indoor and ambient samples. The ratios of Fe (ng/µg of $PM_{2.5}$) vs Mn (pg/µg $PM_{2.5}$) showed personal samples to be twice the ratio for crustal material. Similarly for the Cr/Mn ratio. The ratios and strong correlations between pairs of elements suggested steel dust as the source. Time-activity data suggested subways as a source of the elevated personal metal levels.

Chuang et al. (2005, 087989)

Study Design Panel Study
Period Taipei, Taiwan
Location Individuals with CHD, prehypertension, and hypertension
Population No
Personal Method Yes, a technician carrying a DUSTcheck; monitor accompanied each patient
Personal Size $PM_{0.3-1.0}$; $PM_{1.0-2.5}$; $PM_{2.5-10}$
Microenvironment Size NR
Ambient Size NR
Component(s) NR
Primary Findings HRV reduction in susceptible population was associated with $PM_{0.3-1.0}$ but was not; associated with either $PM_{1.0-2.5}$ or $PM_{2.5-10}$.; $PM_{0.3-1.0}$ exposures at 1- to 4-h moving averages were associated with SDNN and r-MSSD in both cardiac and hypertensive; patients. For an interquartile increase in $PM_{0.3-1.0}$, there were 1.49–4.88% decreases in SDNN and 2.73-8.25% decreases in r-

MSSD. $PM_{0.3-1.0}$ exposures were also associated with decreases in LF; and HF for hypertensive patients at 1- to 3-h moving averages except for cardiac patients at moving averages of 2 or 3 h.

Cohen et al (2004, 056909)

Study Design	Field evaluation study to test performance of new technology to measure number concentration of acidic ultrafine particles (UFP)
Period	July 1999-September 2000
Location	New York City and nearby suburban location
Population	4 outdoor rural sites and 1 indoor rural site (cafeteria) in Tuxedo, NY. 1 suburban residential site in Newburgh, NY. 1 outdoor urban site in New York City
Age Groups	NR
Indoor Source	NR
Personal Method	No personal exposure assessment was conducted.
Personal Size	NR
Microenvironment Size	Ultrafine (UFP)
Ambient Size	Ultrafine (UFP)
Component(s)	Acidic UFP, Hydrogen ions, sulfate ions, ammonium ions
Primary Findings	Iron nanofilm detectors can be used with confidence under a range of ambient conditions. Concentrations of UFP determined by atomic force microscopy analysis of detectors in MOI-EAS and UDM appeared to underestimate number concentrations of total UFP and [missing items?]

Connell et al. (2005, 089458)

Study Design	Times-series
Period	May 2000-May 2002
Location	Steubenville, Ohio = ST; Saint Vincent College, Latrobe, PA (eastern site) = E; Tomlinson Run State Park, New Manchester, WV (northern site) = N; Hopedale, OH (western site) = W; Jesuit Univ., Wheeling, WV (southern site) = S
Population	NR
Age Groups	No
Indoor Source	NR
Personal Method	NR
Personal Size	NR
Ambient Size	PM_{10} & $PM_{2.5}$
Component(s)	Ammonium, sulfate, nitrate, chloride, and 21 elements, elemental carbon and organic carbon.
Copollutant(s)	SO_X, NO_X, Co, and O_3.
Primary Findings	The average $PM_{2.5}$ in Steubenville was 18.4 µg/m³, 3.4 µg/m³ above the annual $PM_{2.5}$ NAAQS.

Conner and Williams (2004, 156364)

Study Design	This is part of the EPA Baltimore PM Study of the Elderly.
Period	July-August, 1998
Location	Towson, Maryland
Population	65+ adults
Age Groups	65+ yr
Indoor Source	Personal sampling devices (PEM)
Personal Method	$PM_{2.5}$
Personal Size	$PM_{2.5}$
Microenvironment Size	NR
Ambient Size	NR
Primary Finding(s)	A greater variety of particles was observed in the personal samples compared to the fixed-location apartment samples.

Cortez-Lugo et al. (2008, 156368)

Study Design	Cohort
Period	Feb-Nov 2000
Location	Mexico City, Mexico
Population	Ambulatory adults with moderate to severe COPD, active smokers excluded
Age Groups	Adults
Indoor Source	carpeting, aerosol sprays used, boiler use and location, animals, mold, tobacco smoking, windows closed
Personal Method	Personal pumps with 37-mm Teflon filters, flow rate 4 l/min in a bag with shoulder strap. The impactor was near the breathing zone
Personal Size	$PM_{2.5}$
Microenvironment Size	$PM_{2.5}$ & PM_{10}
Ambient Size	$PM_{2.5}$ & PM_{10}
Component(s)	NR
Primary Findings	Indoor $PM_{2.5}$ concentrations explained 40% of the variability of personal exposure.; The best predictors of personal exposure were indoor contact with animals (12%, 1-25), mold (27%, 11-48), being present during cooking (27, 12-43), and aerosol use (17%, 4-31).

Crist et al. (2008, 156372)

Study Design Indoor, outdoor, and personal monitoring
Period January 1999-August 2000
Location Ohio
Population Fourth & fifth-grade children
Age Groups 9-11 yr old
Indoor Source Filter, portable pump
Personal Method Filter, $PM_{2.5}$
Personal Size Indoor school; Filter, $PM_{2.5}$
Microenvironment Size Outdoor school; Filter, $PM_{2.5}$
Ambient Size NR

Cyrys et al. (2003, 042232)

Study Design Exposure assessment, source apportionment of urban aerosol
Period September 1, 1995-December 21, 1998
Location Erfurt, Germany
Population Urban Populations
Age Groups NR
Indoor Source NR
Personal Method No personal exposure assessment was conducted
Personal Size NR
Microenvironment Size NR
Ambient Size Ultrafine (UFP, 0.01-2.5 µm), $PM_{2.5}$, PM_{10}
Component(s) Si, Al, Ti, Ca, Fe, Cr, Mg, Na, K, Mn, Ni, V, Co, Sc, Cu, Zn, Pb, Br, S
Primary Findings Low correlation between UFP number concentration and fine particle mass and differences in their diurnal patterns suggest that different sources contribute to particles in the 2 size ranges. Elements Si, Al, Ti and Ca were highly correlated and had low e

Cyrys et al. (2006, 156376)

Study Design Exposure assessment, evaluation of sampling methodologies
Period Sept 1 2000-August 31, 2001
Indoor Source Indoor
Personal Method No personal monitoring was conducted
Personal Size NR
Microenvironment Size NR
Ambient Size NR
Component(s) Restricted sampling scheme (covering 23% of study period) was able to estimate reliable annual and winter averages in Erfurt, Germany. Daily $PM_{2.5}$ means measured by EPA-WINS were higher than those measured by HI, but differences between samplers were small.

Delfina et al. (2004)

Study Design Panel study with repeated measures
Period Sep-Oct 1999 or Apr-Jun 2000
Location Alpine, California
Population Children
Age Groups 9-19 yr
Indoor Source No
Personal Method Personal dataRAM (pDR) carried at waist level using a fanny pack, shoulder harness, or vest.
Personal Size $PM_{2.5}$ (approximate); 0.1-10 range
Microenvironment Size PM_{10} & $PM_{2.5}$; measured immediately outside the house and in the living room of the home.
Ambient Size PM_{10}
Copollutant(s) O_3 and NO_2 measured at central site
Primary Findings Percent predicted FEV_1 was inversely associated with personal exposure to fine particles. Also with indoor, outdoor and central site gravimetric $PM_{2.4}$, PM_{10}, and with hourly TEOM PM_{10}.

Delfino et al. (2006, 090745)

Study Design Cohort. Measured daily expired NO (FeNO)
Period Aug-Dec 2003
Location Riverside and Whittier, California
Population Children with asthma exacerbations in previous 12 months, non-smokers, non-smoking households
Age Groups 9-18 yr
Indoor Source No

Personal Method	Wore a backpack during waking hours for $PM_{2.5}$, EC and OC, NO_2, temperature, and relative humidity. Exhaled air collected in Mylar bags to analyze for NO.
Personal Size	24-h $PM_{2.5}$; 1-h max $PM_{2.5}$; 8-h max $PM_{2.5}$; 24-h NO_2
Microenvironment Size	NR
Ambient Size	24-h $PM_{2.5}$; 24-h PM_{10}; 8-h max O_3; 8-h max NO_2; 24-h NO_2; 8-h max CO
Component(s)	24-h $PM_{2.5}$ EC; 24-h $PM_{2.5}$ OC
Primary Findings	PM associations with airway inflammation in asthmatics may be missed using ambient particle mass.; The strongest positive associations were between eNO and 2-day avg pollutant concentrations. Per IQR increases 1.1 ppb FeNO/24 µg/m³ personal $PM_{2.5}$.; 0.7 ppb FeNO/0.6 µg/m³ personal EC; 1.6 ppb FeNO / 17 ppb personal NO_2; Ambient $PM_{2.5}$ and personal and ambient EC were significant only when subjects were taking inhaled corticosteroids.; Subjects taking both inhaled steroids and antileukotrienes had no significant associations.; Distributed lag models showed personal $PM_{2.5}$ in the preceding 5 h was associated with FeNO.

Demokritou et al. (2002, 156393)

Study Design	Exposure assessment, evaluation of newly developed personal cascase impactor
Period	NR
Location	Laboratory chamber
Population	Newly developed personal PM sampler
Age Groups	NR
Indoor Source	NR
Personal Method	No personal exposure assessment was conducted
Personal Size	NR
Microenvironment Size	9.6-20 µm, 2.6-9.6 µm, 1.0-2.6, 0.5-1.0
Ambient Size	NR
Component(s)	NR
Primary Findings	The first stage showed excellent separation of particles larger than 9.6 µm from the airstream. In the second stage, for particles above 4.0 µm, the collection efficiency was greater than 95%. In the third stage, the collection efficiency for particles a

Dermentzoglou et al. (2003, 156395)

Study Design	Sampled rooms in 1 apartment for 2 h and compared to ambient air.
Period	NR, but winter < 2003
Location	Thessaloniki, Greece
Population	Urban apartment dwellers
Age Groups	NR
Indoor Source	Woodburing fireplace, cigarette smoking, cooking fish, chicken, sausage & potato.
Personal Method	NR
Personal Size	NR
Microenvironment Size	PM3.0
Ambient Size	NR
Component(s)	PAHs, Pb, Cd, Cu, Mn, Ni, V As
Primary Findings	Smoking could be associated with the highest indoor concentration of total and carcinogenic PAHs.The highest level of pyrens, and phnanthrenes were during fish frying. Smoking and fish frying had significant effect on Cd in indoor air, while woodburning had no effect of PAH or heavy metal levels.

Diapouli et al. (2007, 156397)

Study Design	Exposure assessment. Sampling of schools, residence, private vehicle
Period	Schools- 11/2003-02/2004 and 10/2004-12/2004.; Residence- 10/2004; Vehicle- 10/204-12/2004
Location	Athens, Greece
Population	Primary school children
Age Groups	NR
Indoor Source	NR
Personal Method	Handheld portable Condensation Particle Counters (TSI, Model 3007) were used for all sampling locations. Primary schools indoor measurements were primarily conducted inside classrooms, at table height. However, at three of the schools, rooms of different uses were selected. These included a teachers' office (where smoking was permitted), a computer day lab (used by students only part of the day), and a library and gymnasium (where intense activity took place almost all day long). Outdoor measurements took place in the yard of each school. Residence samples were taken in a bedroom at breathing height and on the terrace, for indoor and outdoor samples, respectively. In-vehicle samples were taken by placing the CPC 3700 on the passenger seat while the vehicle drove along predetermined routes.
Personal Size	NR
Microenvironment Size	0.01-1 µm
Ambient Size	0.01-1 µm
Component(s)	NR
Primary Findings	The results showed that children attending primary schools in the Athens area are exposed to significant PM concentration levels, both indoors and outdoors, Vehicular emissions seem to be a major contributor to the measured outdoor concentration levels at the studied sites. Indoor PM concentrations appeared to be influenced by both vehicular emissions and indoor sources including

cleaning activities, smoking, a high number of people in relation to room volume and furniture material (i.e., carpet.).; UFPs concentrations diurnal variation, both outside the schools and the residence, supports the close relation of UFPs levels with traffic density. Indoor concentrations within schools exhibited variability during the school day only when there were significant changes in room occupancy. 24-h variation of indoor concentrations at the residence followed quite well (R2 = 0.89) the outdoor one, with a delay of 1-h or less.

Diapouli et al. (2008, 190893)

Study Design Indoor, outdoor air monitoring of PM. To determine children exposure in school environment. To evaluate relationship between indoor and outdoor levels.
Period Athens, Greece
Location Primary schools
Population NR
Indoor Source Indoor $PM_{2.5}$ and PM_{10} presence of children and activities of children in classrooms PM_1vehicles
Personal Method Harvard PEM, Teflon filters Dust Trak Condensation particle counter
Personal Size NR
Microenvironment Size Weight concentration $PM_{2.5}$, PM_{10} Number concentration PM_1
Ambient Size Weight concentration $PM_{2.5}$, PM_{10} Number concentration PM_1
Component(s) NO3-, SO_4^{2-}
Primary Findings High levels of PM_{10} and $PM_{2.5}$ measured indoors and outdoors. PM_{10} more variable spatially than $PM_{2.5}$. Indoor/Outdoor ratio for PM_{10} and $PM_{2.5}$ close to 1 at almost all sites. Ratio of PM_1smaller than 1 in all cases. Vehicular traffic presumed to be the main source of PM_1. Indoor $PM_{2.5}$ and PM_{10} levels dependent on the amount of activity in classroom and outdoor levels. Indoor SO_4^{2-} concentrations strongly associated with outdoor levels. Result suggests that SO_4^{2-} can be used as a proper surrogate for indoor PM of outdoor origin.

Dills et al. (2006, 156402)

Study Design Dose-response, variability, and applicability of methoxyphenols as biomarkers in a realistic exposure situation mimicking indoor open fire cooking
Period August. Year not specified
Location Seattle, WA
Population Non-smokers exposed to woodsmoke
Age Groups 20-65 yr
Indoor Source Not required. Subjects exposed to wood smoke. One subject fitted with an integrating nephelometer for a continuous estimate of particle exposure, and a continuous monitor for CO, CO_2, and temperature. For 24-h prior to the exposure, subjects collected all urine voids at 'will in separate containers for a baseline ofmethoxyphenol excretion. Subjects then collected all urine voids at will for 48 h postexposure for measuring wood smoke biomarker elimination.
Personal Method Air collected at breathing level using Harvard Personal Environmental Monitor for $PM_{2.5}$ (HPEM2.5)
Personal Size $PM_{2.5}$
Microenvironment Size NA. No microenvironmental studied
Ambient Size NR
Component(s) 22 methoxyphenols, levoglucosan, and 17 polynuclear hydrocarbons for personal filter samples and urine samples.
Primary Findings According to the authors "A 12-h avg creatinine-adjusted methoxyphenol concentration is a practical metric for the biomarker exposure to woodsmoke." Propylguaiacol, syringol, methylsyringol, ethylsyringol, and ropylsyringol had peak urinary concentrations after the woodsmoke exposure.

Dimitroulopoulou et al. (2006, 090302)

Study Design Exposure assessment. Development of a model to predict indoor $PM_{2.5}$ concentrations under various emissions scenarios
Period 1997-1999
Location 5 sites in the UK Harwell, Birmingham East, Bradford, Bloomsbury, Marylebone Rd.
Population Indoor environments within homes
Age Groups NR
Indoor Source Smoking, cooking
Personal Method No personal exposure assessment was conducted.
Personal Size NR
Microenvironment Size PM_{10}, $PM_{2.5}$
Ambient Size PM_{10}, $PM_{2.5}$
Component(s) NR
Primary Findings Modeled mean concentrations were most sensitive to variation in outdoor concentrations, air exchange rates, and deposition velocity. Modeled peak concentrations were most sensitive to variations in emissions rates and room size. Cooking activities incre

Ebelt et al. (2005, 056907)

Study Design Personal exposure assessment related to health outcomes for a sensitive sub-population
Period Summer 1998
Location Vancouver, British Columbia, Canada

Population	16 persons who had COPD
Age Groups	Mean subject age 74 yr, Range 54 to 86
Indoor Source	Separated total personal exposure into "ambient" and "non-ambient" based on sulfate results and modeling.
Personal Method	"Subjects wore a $PM_{2.5}$ sampler that provided 24-h integrated personal $PM_{2.5}$ exposure data." No other details reported.
Personal Size	$PM_{2.5}$
Microenvironment Size	"ambient exposure" $PM_{2.5}$, PM_{10}, $PM_{2.5-10}$; "non-ambient exposure" $PM_{2.5}$
Ambient Size	$PM_{2.5}$, PM_{10}, $PM_{2.5-10}$
Component(s)	Ambient sulfate,; ambient non-sulfate,; personal sulfate,; personal ambient non-sulfate
Primary Findings	Ambient exposures and (to a lesser extent) ambient concentrations were associated with health outcomes; total and nonambient particle exposures were not.

Farmer et al. (2003, 089017)

Study Design	case control
Period	12 months
Location	Prague, Czech Republic (2 sites); Košice, Slovak republic; Sofia, Bulgaria
Population	Policeman and Busdrivers usually working through busy streets in 8-10h shifts and a Control Population.
Age Groups	Variable, range not stated
Indoor Source	NR
Personal Method	Personal Monitoring Devices; Blood and Urine Samples; Stationary Versatile Air Pollution Samplers (VAPS)
Personal Size	PM_{10}
Microenvironment Size	NR
Ambient Size	PM_{10}; $PM_{2.5}$ (not reported)
Component(s)	EOM; EOM2; B[a]P; c-PAHs
Primary Findings	Extractable organic matter (EOM) per PM_{10} was at least 2-fold higher in winter than in summer, and c-PAHs over 10-fold higher in winter than in summer. Personal exposure to B[a]P and to total c-PAHs in Prague ca. was 2-fold higher in the exposed group compared to the control group, in Košice ca. 3-fold higher, and in Sofia ca. 2.5-fold higher.

Farmer et al. (2003, 089017)

Study Design	Molecular epidemiology studies of carcinogenic environmental pollutants, particularly PAHs
Period	NR
Location	Prague, Czech Rep.; Kosice, Slovak Rep.; Sofia, Bulgaria
Population	Policemen and bus drivers
Age Groups	NR
Indoor Source	No
Personal Method	"Personal monitors for PM_{10}"; Extraction by dichloromethane and analyzed for PAH by HPLC with fluorimetric detection.
Personal Size	PM_{10}
Microenvironment Size	NR
Ambient Size	PM_{10}; Extractable organic material (EOM); B[a]P; cPAHs
Component(s)	Benzo[a]pyrene (B[a]P); Carcinogenic polycyclic aromatic hydrocarbons (cPAHs)
Primary Findings	Personal exposure to B[a]P and to total carcinogenic PAHs in Prague was twofold higher in the exposed group compared to controls, in Kosice three fold higher, and in Sofia 2.5-fold higher.

Ferro et al. (2004, 055387)

Study Design	Case study, 1 home
Period	Redwood City, California
Location	NR
Population	NR
Age Groups	NR
Personal Method	Co-located real-time particle counters and integrated filter samplers (Met-One Model 237B) were used to measure personal (PEM), indoor (SIM) and outdoor (SAM) PM concentrations. The PEM was attached to a backpack frame and worn by the investigator while performing prescribed activities. The SIM was attached to a six foot step-ladder with the intake at breathing height. The SAM was located under a two-sided roofed shed in the backyard of the home with the filter samplers supported by a metal stand and the real-time particle counters sitting on a table.
Personal Size	PM_5
Microenvironment Size	$PM_{2.5}$; PM_5
Ambient Size	$PM_{2.5}$; PM_5
Component(s)	NR
Primary Findings	The results of this study indicate that house dust resuspended from a range of human activities increases personal PM concentrations and this resuspension effect significantly contributes to the personal cloud. The results of this study also suggest that normal human activities that resuspend house dust may contribute significantly to the strong correlations found between personal exposure and indoor PM concentrations in previous studies. The PEM/SIM ratios for human activity presented in this paper are also in the range of those reported by previous studies.

Ferro et al. (2004, 055676)

Study Design	Modeling of PM source strengths from human activities
Period	April 2000
Location	Redwood City, CA
Population	Residential home occupants
Age Groups	NR
Indoor Source	Yes. Vacuuming resulted in the maximum $PM_{2.5}$ source strength while two persons walking around and sitting on furniture resulted in the maximum PM_5 source strength.
Personal Method	Met-One Model 237B laser particle counters (2.8 Lpm); AIHL design cyclone samplers with filters (21 and 11 Lpm for $PM_{2.5}$ and PM_5 respectively)
Personal Size	$PM_{2.5}$, PM_5
Microenvironment Size	$PM_{2.5}$, PM_5
Ambient Size	$PM_{2.5}$, PM_5
Component(s)	NR
Primary Findings	The source strengths were found to be a function of the number of persons performing the activity, the vigor of the activity, and the type of flooring. Proximity to the source played a large role in the observed differences between indoor concentration and personal exposure.

Fromme et al. (2007, 156453)

Study Design	Explorative analysis
Period	Winter session December 2004-March 2005; summer session May to July 2005
Location	Munich (and surrounding districts), Germany
Population	Primary and secondary school children
Age Groups	NR
Indoor Source	NR
Personal Method	Filter-based measurements of $PM_{2.5}$ in the classrooms were conducted with a medium volume sampler using a flow controlled pump (Derenda, Teltow, Germany). The sample inlet was a $PM_{2.5}$ sampler, having a 50% collection efficiency for particles with a 2.5 mm aerodynamic diameter. A Munktell 47mm binder free glass fibre filter with a pore size of 2 mm was used. Continuous measurements of PM (e.g. PM_{10}, PM_4, $PM_{2.5}$) were also done using an optical laser aerosol spectrometer (LAS) (Dust monitor 1.108). A TSI model 3034 scanning mobility particle sizer (SMPS) (TSI Inc., Shoreview, MN, USA) was used to measure particle number concentrations (PNC) for a discrete size distribution of aerosols. Indoor carbon dioxide was measured using a continuously monitoring infrared sensor (Testo 445).; The sampling and measuring position in the classroom was opposite to the black board, about one meter above floor level, the level at which the pupils would normally inhale.
Personal Size	NR
Microenvironment Size	$PM_{2.5}$; PM_4; PM_{10}
Ambient Size	PM_{10}, PN (particle number)
Copollutant(s)	CO_2
Primary Findings	Results clearly showed that exposure to PM in school is high. This study identified parameters correlated with increased concentrations of PM such as high CO_2 concentrations and low class level. Strong seasonal variability was observed, with air quality being particularly poor in winter. The influence of season on PM concentrations observed has been reported before from the US (Keeler et al., 2002). This difference is most likely due to the different ventilation practice in summer and winter. Further parameters correlated with increased concentrations of PM were small room size, high number of occupants, high CO_2 concentrations and low class level. No significant differences between PM and values in classrooms with carpets and those with hard surface floorings were reported. The number of fine and ultra fine particles measured in classrooms was in the same range or lower as the results from residences or outdoor monitoring sites (reported in similar studies) and show little variation.

Gadkari and Pervez (2007, 156459)

Study Design	Evaluation of relative source contribution estimates of various routes of personal RPM in different urban residential environments.
Period	Summer 2004 (March 15-June 15)
Location	Chattisgarh, India
Population	All likely. Not specified
Age Groups	21-61 yr, average age 40 ± 15 yr
Indoor Source	No
Personal Method	Personal respirable dust samplers (RDS) with GFF
Personal Size	RPM
Microenvironment Size	NR
Ambient Size	RPM
Component(s)	Fe, Ca, Mg, Na K, Cd, Hg, Ni, Cr, Zn, As, Pb, Mn and Li
Primary Findings	Authors concluded that "(1) indoor activities and poor ventilation qualities are responsible for major portion of high level of indoor RPM, (2) majority of personal RPM is greatly correlated with residential indoor RPM, (3) time–activity diary of individuals has much impact on relationship investigations of their personal RPM with their respective indoor and ambient-outdoor RPM levels; as reported in earlier reports and (4) residential indoors, local road-traffic and soil-borne RPMs are the dominating routes of personal exposure compared to ambient outdoor RPM levels."

Gauvin et al. (2002, 034893)

Study Design	Fine particle exposure assessment for children in French urban environments, part of VESTA study
Period	March 1998-December 2000
Location	Paris, Grenoble, Toulouse, France
Population	Children aged 8-14 yr
Indoor Source	Yes-ETS from mother, rodents at home.
Personal Method	SKC pump 4 Lpm with $PM_{2.5}$ inlet and 37 mm, 2 micron Teflon filter
Personal Size	$PM_{2.5}$
Microenvironment Size	NR
Ambient Size	PM_{10}
Component(s)	NR
Primary Findings	The final model explains 36% of the between subjects variance in $PM_{2.5}$ exposure, with ETS contributing more than a third to this.

Geyh et al. (2004, 156467)

Study Design	An evaluation of a modified personal monitoring pump (PMASS)
Period	NR
Location	Fresno, CA and Baltimore, Maryland
Population	Persons for personal sampling
Age Groups	NR
Indoor Source	PMASS and PEM "adjusted mass measurements downward by 22% to eliminate measurement bias with the Harvard impactor."
Personal Method	Particle mass
Personal Size	Particle mass
Microenvironment Size	NR
Ambient Size	EC, OC, nitrate
Primary Finding(s)	PMASS measurements of mass showed a significant bias of -24% compared to the reference sampler.; For microenvironmental sampling the PMASS for mass concentrations again had a bias of -34%, but for EC, OC and nitrate were much closer but still with a bias of 6.6-17.5%.

Geyh et al. (2005, 186949)

Study Design	Exposure assessment- representative population (WTC truck drivers) study
Period	October 2001 and April 2002
Indoor Source	Indoor
Personal Method	Each driver was given two monitors consisting of small portable pumps and battery packs worn at the waist, and sampling cartridges worn on the shoulder within the breathing zone. Monitoring was conducted across a work shift on all days of the week during both day and night shifts (6:00am to 6:00pm, and 6:00pm to 6:00am, respectively). Drivers were asked to wear their monitors at all times. If they were planning to sleep in their trucks, they were told they could remove the pumps from their belts and place them on the seat beside them.; Area monitoring was also conducted at the site at four locations around the perimeter of the disaster site on streets approximately representing the north, east, and south/southwest boundaries of the debris field. In addition, monitoring was also conducted directly in the debris pile for several days. The set of monitors were hung at head height either from scaffolding, from a chain link fence, or placed on supports, such as tank cages in the debris pile.; Sampling pumps used for particle sampling were either SKC Universal pumps(model 223-PCXR4), BGI personal sampling pumps (model 400S0, or ELF personal sampling pumps (MSA Inc). VOC sampling was conducted with SKC pocket pumps (Personal Packet Pump 210 series).
Personal Size	TD; PM_{10}; $PM_{2.5}$
Microenvironment Size	NR
Ambient Size	TD; PM_{10}; $PM_{2.5}$
Component(s)	EC; OC
Copollutant(s)	VOC(s)
Primary Findings	During October, the median personal exposure to TD was 346 $\mu g/m^3$. The maximum area concentration 1742 $\mu g/m^3$, was found in the middle of the debris. The maximum TD concentration found at the perimeter was 392 $\mu g/m^3$ implying a strong concentration gradient from the middle of debris outward. $PM_{2.5}$ /PM_{10} ratios ranged from 23% to 100% suggesting significant fire activity during some of the sampled shifts. During April, the median personal exposure to TD was 144 $\mu g/m^3$, and the highest area concentration, 195 $\mu g/m^3$, was found at the perimeter. Although the overall concentrations on PM at the site were significantly lower in April, the relative contributions of fine particles to the PM_{10}, and EC and OC to the TD were similar. During both months, volatile organic compounds concentrations were low. Comparison of recorded EC and OC values from October 2001 and April 2002 with previous studies suggests that the primary source of exposure to EC for the WTC truck drivers was emissions from their own vehicles.

Goyal and Sidhartha (2004, 156487)

Study Design	Actual air monitoring measurements are compared with a model.
Period	1998-1999
Location	Delhi, India
Population	Residents near coal-fired power plants (BTPS)
Age Groups	NR
Indoor Source	NR

Personal Method	NR
Personal Size	NR
Microenvironment Size	NR
Ambient Size	Suspended PM (SPM)
Component(s)	NR
Primary Findings	Measured SPM values are higher during the day than at night. This is because "point sources dominate during the daytime convective conditions. At night the small depth of the nocturnal boundary layer prevents the dispersion of the pollutants from the elevated point source to reach the surface. Convective turbulence breaks up the surface-based inversion and the fumigation process leads to an increased contribution from the point sources."

Graney et al. (2004, 053756)

Study Design	The study was designed to assess the trace metal quantification abilities of several analytical methods to measure the total as well as soluble amounts of metals with $PM_{2.5}$ collected from indoor and PM samples. (X-ray fluorescence and instrumental neutron activation analysis)
Location	Retirement facility in Towson, Maryland
Population	Retirement facility with subjects who spent 94% of their time indoors
Age Groups	Mean age = 84 yr
Indoor Source	No, this was not the objective of the study
Personal Method	Measured using personal exposure monitors (MSP Inc) with nozzle to remove particles >4 μg/m^3
Personal Size	$PM_{2.5}$
Microenvironment Size	$PM_{2.5}$
Ambient Size	NR
Component(s)	42 elements were analyzed for in the $PM_{2.5}$ samples collected from personal and well as indoor samples
Primary Findings	1) Most of the extractable components of the metals were in a water-soluble form suggesting a high potential for bioavailability of elements from respiratory exposure to $PM_{2.5}$. 2) based on comparison of trace metals in central indoor site vs. PE samples, resident activities result in exposure to higher conc of soluble trace metals.

Guo et al. (2004, 156506)

Study Design	Human exposure assessment
Period	Sept. 2001-Jan. 2002
Location	Hong Kong
Population	Shoppers at food markets
Age Groups	NR
Indoor Source	Yes. Elevated concentrations of PM at three markets probably due to outdoor particulates from vehicular exhaust. Poultry stalls in the markets had higher PM_{10} due to live chickens.
Personal Method	TSI Dust Trak Model 8520. In some locations an Anderson Hi-Vol sampler with filters weighed by electronic microbalance were used to calibrate the Dust Trak.
Personal Size	PM_{10}
Microenvironment Size	PM_{10}
Ambient Size	PM_{10}
Component(s)	NR
Primary Findings	Indoor PM_{10} concentrations at the markets were generally below Hong Kong Indoor Air Quality Objectives. Outdoor sources were dominant at the five markets, with elevated levels at three markets due to vehicular exhaust.

Hanninen et al. (2004, 056812)

Study Design	EXPOLIS-human exposure assessment
Period	1996-2000
Location	Athens, Greece; Basle, Switzerland; Helsinki, Finland; Prague, Czech Republic
Population	Residential homes
Age Groups	NR
Indoor Source	Yes. Sources identified in statistical analysis wooden building material, use of stove other than electric, PVC floors, attached garage
Personal Method	Pump & filter with gravimetric analysis; Elemental composition using energy dispersive X-ray fluorescence
Personal Size	$PM_{2.5}$
Microenvironment Size	NR
Ambient Size	$PM_{2.5}$
Component(s)	$PM_{2.5}$ -bound sulfur
Primary Findings	Associated with indoor concentration wooden building material, city, building age, floor of residence (ground, 1st, etc.), and use of stove other than electric.

Haverinen-Shaughnessy et al. (2007, 156526)

Study Design	Cross-sectional
Period	Winter, year not reported
Location	Eastern Sweden

Population Elementary school teachers
Age Groups NR
Personal Method Button inhalable aerosol samplers
Personal Size Particle mass
Microenvironment Size Particle mass
Ambient Size NR
Component(s) Absorbance coefficient/m x 10^{-5}; Total fungi (spores/m^3); Total bacteria (cells/m^3); Viable fungi MEA (CFU/m^3); Viable fungi DG18 (CFU/m^3); Viable bacteria (CFU/m^3)
Primary Findings The recall period of 7 days provided the most reliable data for health effect assessment. Both personal exposure and concentrations of pollutants at home were more frequently associated with health symptoms than work exposures.

Hazenkamp-von Arx et al. (2003, 136487)

Study Design Exposure assessment
Period November 2000-February 2001
Location 21 European cities Antwerp City, Antwerp South, Albacete, Barcelona, Basel, Erfurt, Galdakao, Grenoble, Goteborg, Huelva, Ipswich, Norwich, Ovledo, Pavia, Paris, Reykjavik, Tartu, Turin, Umea, Uppsala, Verona
Population European urban environments
Age Groups NR
Indoor Source NR
Personal Method No personal exposure assessment was conducted
Personal Size NR
Microenvironment Size NR
Ambient Size NR
Component(s) NR
Primary Findings Winter mean $PM_{2.5}$ concentrations were lowest in Iceland and Sweden and highest in Northern Italy (Turin, Verona). Cities also varied in daily concentrations. Geographical differences may be explained by differences in emissions (proximity of monitor to traffic.

Henderson et al. (2007, 090675)

Study Design Land use regression was employed to model oxides of nitrogen and fine particulates using two measures of traffic (road length and vehicle density)
Period Sampling was conducted from Feb 24 through Mar 14 and Sep 8 through Sep 26, 2003
Location Vancouver, British Columbia, Canada
Population NR
Age Groups NA
Indoor Source NR
Personal Method Personal monitoring was not conducted. Ambient fine particles were collected on PTFE filters using Harvard Impactors. Flow rate was 4 L/min Absorption coefficients were also calculated
Personal Size NR
Microenvironment Size NR
Ambient Size $PM_{2.5}$
Copollutant(s) NO, NO_2
Primary Findings Adjusted R2 for the linear regression models predicting NO, NO_2, $PM_{2.5}$, and ABS from fifty-five variables describing each sampling site ranged from 0.39 to 0.62. The resulting maps show the distribution of NO to be more heterogeneous than that of NO_2, supporting the usefulness of land use regression for assessing spatial patterns of traffic-related pollution

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Hertel et al. (2008, 156543)

Study Design Exposure assessment
Location Denmark
Population Bicycle commuters
Age Groups NR
Indoor Source No
Personal Method NR
Personal Size $PM_{2.5}$, PM_{10}
Microenvironment Size NR
Ambient Size NR
Component(s) NO_2
Primary Findings It is possible to significantly reduce the accumulated air pollution exposure during the daily bicycle route between home and work by following the low exposure route. Travelling outside the rush hour time periods significantly reduced the accumulated air pollution exposure along the routes through the city.

Ho et al. (2004, 056804)

Study Design Human exposure assessment

Period 25 Sept. 2002 to 8 March 2003
Location Hong Kong
Population Occupied buildings located near major roadways
Age Groups NR
Indoor Source Yes. Regression of indoor versus outdoor concentrations of OC and EC revealed an indoor source of OC not present for EC, presumably due to such activities of cooking, smoking, and cleaning.
Personal Method Co-located mini-volume samplers (flow rate 5 L/min) and Partisol model 2000 sampler with 2.5 micron inlet. All samples on 47 mm Whatman quartz microfiber filters, weighed on an electronic microbalance. Analyzed for OC and EC using DRI Model 2001 Thermal/Optical Carbon Analyzer.
Personal Size $PM_{2.5}$
Microenvironment Size NR
Ambient Size $PM_{2.5}$
Component(s) OC; EC; OM; TCA
Primary Findings The major source of indoor EC, OC, and $PM_{2.5}$ appears to be penetration of outdoor air, with a much greater attenuation in mechanically ventilated buildings.

Hoek et al. (2008, 156554)

Study Design Exposure assessment, characterizing indoor/outdoor particle relationships
Period October 2002-March 2004
Location 4 European cities Amsterdam, Athens, Birmingham, Helsinki
Population Urban populations
Age Groups NR
Indoor Source Smoking, candle burning, cooking/frying
Personal Method No personal exposure assessment was conducted
Personal Size NR
Microenvironment Size PM_{10}, $PM_{2.5}$, PM_{10}-2.5, Ultrafine (UFP)
Ambient Size PM_{10}, $PM_{2.5}$, PM_{10}-2.5, Ultrafine (UFP)
Component(s) soot, sulfate
Primary Findings Correlation between 24-avg [should this be 24-h avg?] central site and indoor concentrations was lower for UFP than for $PM_{2.5}$, soot, or sulfate, probably related to greater losses during infiltration due to smaller particle size. Infiltration factors for UFP and $PM_{2.5}$ were low.

Holguin et al. (2003, 057326)

Study Design Longitudinal analysis (repeated measures) of local $PM_{2.5}$ and biological markers of cardiovascular dysregulation
Period 3 months (Feb 8-Apr 30, 2000)
Location Mexico City, Mexico
Population Elderly residents of a nursing home (non-smokers)
Age Groups 60-96
Indoor Source Sources of indoor PM concentrations may be idling buses parked for a few hours close to living areas at least 3 times per week
Personal Method Mini-vol portable air samplers operating at 4 l/min used to monitor outdoor and indoor $PM_{2.5}$ concentrations at a nursing home. Gravimetric analysis of filters. Personal exposure calculated using time-weighted averages of outdoor and indoor concentration
Personal Size NR
Microenvironment Size NR
Ambient Size NR
Component(s) NR
Primary Findings Increases in personal $PM_{2.5}$ concentrations were associated with significant decreases in the high-frequency component of heart rate variability (HRV-hF) among elderly. Associations remained significant after adjusting for ozone; Indoor and outdoor $PM_{2.5}$

Hopke et al. (2003, 095544)

Study Design Epidemiology-Exposure study
Period 26 July to 22 August 1998
Location Retirement facility in Towson, MD
Population "A potentially susceptible elderly subpopulation"
Age Groups Mean age of 84
Indoor Source Ammonium sulfate and ammonium nitrate, secondary sulfate, OC, and motor vehicle exhaust
Personal Method Personal exposure samples were collected on 37mm Teflon filters using inertial impactor PEM in the breathing zone of the subjects. A "scalper" (MSP, PEM-019) nozzle was used on the PEM to exclude particles >4mm in order to reduce the potential of overloading the impactor. Centralized indoor sampling was conducted in an unoccupied apartment on the fifth floor of the retirement facility (central indoor). The windows of the apartment were kept closed and the front door was kept open to the common hallway with a small fan providing active air exchange. Residential outdoor sampling at the retirement facility was conducted from the rooftop of an attached three-story nursing care facility (outdoor). $PM_{2.5}$ measurements at an ambient site in Towson, MD were made on the roof of a sampling shelter approximately four meters off the ground (community). Daily community, outdoor, and central indoor $PM_{2.5}$ samples were collected with VAPS samplers.
Personal Size $PM_{2.5}$

Microenvironment Size $PM_{2.5}$
Ambient Size $PM_{2.5}$
Component(s) NR
Primary Findings VAPS and PEM data from the BPMEES were separately analyzed by different receptor models. These two approaches were complementary and allowed for evaluation of all of the available data. A three-way analysis of the VAPS data provided four sources of $PM_{2.5}$, nitrate–sulfate, sulfate, OC, and MV exhaust. The largest contribution to the community, outdoor, and central indoor sampling locations was the sulfate source. Infiltration of the sources varied depending on the source and ranged from 38% to 4% for the Sulfate, and Nitrate–Sulfate sources, respectively. In addition, MV exhaust had a penetration rate similar to Sulfate (32%). The OC source had little variability compared to the other sources and contributed approximately 8% of the community and outdoor PM and 18% of the central indoor PM.; The PEM data were analyzed using a complex model with a target for soil that included factors that are common to all of the types of samples (external factors) and factors that only apply to the data from the individual and apartment samples (internal factors). From these results, the impact of outdoor sources and indoor sources on indoor concentrations were assessed. The identified external factors were sulfate, soil, and an unknown factor. Internal factors were identified as gypsum or wall board, personal care products, and a factor representing variability not explained by the other indoor sources. The latter factor had a composition similar to outdoor particulate matter and explained 36% of the personal exposure. External factors contributed 63% to personal exposure with the largest contribution from sulfate (48%).

Jacquemin et al. (2007, 156600)

Study Design Assessment of relationship between outdoor and personal concentrations of PM25] absorbance and sulfur among post-myocardial infarction patients
Period January 2004-June 2004
Location Barcelona, Spain
Population Survivors of a myocardial infarction exposed to environmental tobacco smoke (ETS)
Age Groups n = 38 (males 32 (84%), age over or equal to 65 [something missing here] 15 (39%)
Indoor Source Not identified in this study. Results from other studies discussed.
Personal Method Personal samplers (BGI GK2.05 cyclones and battery operated BGI AFC400S pumps)
Personal Size $PM_{2.5}$
Microenvironment Size NA
Ambient Size $PM_{2.5}$
Component(s) Sulfur (S)
Primary Findings Authors suggest that "outdoor measurements of absorbance and sulfur can be used to estimate both the daily variation and levels of personal exposures also in Southern European countries, especially when exposure to ETS has been taken into account. For $PM_{2.5}$, indoor sources need to be carefully considered."

Janssen et al. (2005, 088692)

Study Design Panel Study
Period Amsterdam 11/2/1998-6/18/1999; Helsinki 11/1/1998-4/30/1999
Location Amsterdam, The Netherlands; Helsinki, Finland
Population Elderly Cardiovascular Patients
Age Groups 50-84 yr
Indoor Source No
Personal Method Personal $PM_{2.5}$ GK2.05; cyclones; indoor & outdoor Harvard Impactors; Reflectance EEL 43 reflectometers; Elemental Composition Tracor Spectrace 5000 ED-XRF system
Personal Size $PM_{2.5}$
Microenvironment Size $PM_{2.5}$
Ambient Size $PM_{2.5}$
Component(s) Estimated Elemental Carbon (Abs); Elemental composition of a subset of personal, indoor and outdoor samples
Primary Findings For most elements, personal and indoor; concentrations were lower than and highly correlated with outdoor concentrations. The highest correlations (median r.0.9) were found for sulfur and particle absorbance (EC), which both represent fine; mode particles from outdoor origin. Low correlations were observed for elements that represent the coarser part of the $PM_{2.5}$ particles (Ca, Cu, Si, Cl^-).

Jansen et al. (2005, 082236)

Study Design Panel Study
Period Winter 2002-2003
Location Seattle, Washington, USA
Population Elderly Respiratory Disease Patients (asthma/COPD)
Age Groups 71-86 yr
Indoor Source No
Personal Method Personal PM_{10} MPEM10; Indoor home and Outdoor home $PM_{2.5}$, PM_{10} Single-stage inertial Harvard Impactors and 37-mm Teflon filters
Personal Size PM_{10}
Microenvironment Size PM_{10}, $PM_{2.5}$, fine particles (~PM1)
Ambient Size PM_{10}, $PM_{2.5}$

Component(s)	BC, as an estimate of elemental carbon (EC)
Primary Findings	For 7 subjects with asthma, a 10 μg/m^3 increase in 24-h avg outdoor PM_{10}; and $PM_{2.5}$ was associated with a 5.9 [95% CI: 2.9-8.9] and 4.2 ppb (95% CI: 1.3-7.1) increase in FeNO, respectively. A 1 μg/m^3 increase in outdoor, indoor, and personal BC was associated with increases in FeNO of 2.3 ppb (95% CI: 1.1-3.6), 4.0 ppb (95% CI: 2.0-5.9), and 1.2 ppb (95% CI: 0.2-2.2), respectively. No significant association was found between; PM or BC measures and changes in spirometry, blood pressure, pulse rate, or SaO_2 in these subjects.

Jaques et al. (2004, 155878)

Study Design	Exposure assessment, field evaluation of continuous $PM_{2.5}$ monitor in comparison to integrated samplers
Period	February-August 2002
Location	Claremont, California
Population	Continuous $PM_{2.5}$ sampler, time-integrated $PM_{2.5}$ samplers
Age Groups	NR
Indoor Source	NR
Personal Method	No personal exposure assessment was conducted
Personal Size	NR
Microenvironment Size	NR
Ambient Size	$PM_{2.5}$
Component(s)	NR
Primary Findings	$PM_{2.5}$ mass measurements using the Differential TEOM monitor are consistent with those of the MOUDI and Partisol. Differences can be explained by loss of ammonium nitrate from reference time-integrated samplers. Partisol underestimates MOUDI measured mass.

Jedrychowski et al. (2006, 156606)

Study Design	Prospective cohort
Period	11/2000-3/2003
Location	Krakow, Poland
Population	Non-smoking pregnant women
Age Groups	Yes
Personal Method	Personal; Exposure Monitor Sampler (PEMS, Harvard; School of Public Health) with
Personal Size	$PM_{2.5}$
Microenvironment Size	NR
Ambient Size	PM_{10}
Component(s)	NR
Primary Findings	The contribution of the background ambient PM_{10} level was very strong determinant of the total personal exposure to $PM_{2.5}$ and it explained about 31% of variance between the subjects.

Jo and Lee (2006, 156613)

Study Design	Case study
Period	Winter of 2004 and summer of 2005
Location	Daegu, Korea
Population	Residents of high-rise apartment buildings
Age Groups	NR
Indoor Source	All of the surveyed apartments were constructed with concrete and iron frames. The apartments used liquid petroleum gas for cooking and as their primary heating system. The exhaust gas generated from heating or cooking was mechanically vented out of the apartments.
Personal Method	The PM_{10} concentrations were measured using real-time light scattering PM_{10} monitors (HAZDUST Model EPAM-500). The CO concentrations were measured using a CO dosimeter (CMCD-10P) equipped with an activated charcoal-Purafil prefilter.; From each building, one lower-floor apartment (first or second floor) and one higher-floor apartment (between 10th and 15th floor) were simultaneously surveyed. The concentrations of CO and PM_{10} were measured at the breathing height in the main living area where the participants spent most of their time and from the apartment balconies outdoors.
Personal Size	NR
Microenvironment Size	PM_{10}
Ambient Size	PM_{10}
Component(s)	CO
Primary Findings	This study found that the outdoor air concentrations of CO and PM_{10} were higher for lower-floor apartments than for higher-floor apartments situated in residential areas. In addition, the concentrations were significantly higher in winter and in summer, regardless of the floor height of the apartments. The indoor concentrations in the lower- and higher-floor apartments, however, were not consistent with the outdoor concentrations. Proximity to a major roadway was found to increase the indoor and outdoor concentrations of PM_{10} in high-rise apartments and therefore cause elevated exposures of the residents during presence at home. This was not observed for CO. Atmospheric stability was found to elevate indoor and outdoor air pollutant concentrations and was therefore determined to be another important factor regarding the level of exposure to CO and PM_{10}.

Johannesson et al. (2007, 156614)

Study Design Cohort
Period Spring and fall seasons of 2002 and 2003
Location Gothenburg, Sweden
Population General adult population
Age Groups 23-51 yr
Indoor Source NR
Personal Method Fine particles were measured for 24 h using both personal and stationary monitoring equipment. Personal monitoring of $PM_{2.5}$ and PM_1 was carried out simultaneously with parallel measurements of $PM_{2.5}$ and PM_1 indoors in living rooms and outside the house on a balcony, porch, etc. In addition, urban background $PM_{2.5}$ levels were measured. Personal monitoring was performed in two ways. The 20 randomly selected subjects carried personal monitoring equipment for $PM_{2.5}$ only, while the 10 staff members carried two pieces of personal monitoring equipment at the same time. On the first measuring occasion, the staff members carried one $PM_{2.5}$ cyclone and one PM_1 cyclone. On the second occasion, duplicate monitors for $PM_{2.5}$ were used. For personal and residential monitoring, the BGI Personal Sampling Pump was used together with the GK2.05 cyclone for $PM_{2.5}$ sampling and the Triplex cyclone SCC1.062 for PM_1sampling. The personal sampling pump was placed in a small shoulder bag and the cyclone attached to the shoulder strap near the subject's breathing zone. The personal monitoring equipment was carried by the subject during awake time. During the night, it was placed in the living room. For indoor monitoring in living rooms, cyclones ($PM_{2.5}$ and PM_1) were placed at about 1.5 m above the floor. The same setup was used for residential outdoor monitoring. The urban background monitor was placed on top of a roof somewhat south of the city center but not near any major highway.
Personal Size $PM_{2.5}$; PM_1
Microenvironment Size $PM_{2.5}$; PM_1
Ambient Size $PM_{2.5}$; PM_1
Component(s) BS
Primary Findings Personal exposure of $PM_{2.5}$ correlated well with indoor levels, and the associations with residential outdoor and urban background concentrations were also acceptable. Statistically significantly higher personal exposure compared with residential outdoor levels of $PM_{2.5}$ was found for nonsmokers. PM_1 made up a considerable proportion (about 70–80%) of $PM_{2.5}$. For BS, significantly higher levels were found outdoors compared with indoors, and levels were higher outdoors during the fall than during spring. There were relatively low correlations between particle mass and BS. The urban background station provided a good estimate of the residential outdoor concentrations of both $PM_{2.5}$ and $BS_{2.5}$ within the city. The air mass origin affected the outdoor levels of both $PM_{2.5}$ and $BS_{2.5}$; however, no effect was seen on personal exposure or indoor levels.

Jones and Harrison (2006, 155886)

Study Design NR
Period January 2002-December 2004
Location England London Marylebone Road (Located beside arterial road in street canyon carrying approximately 80,000 vehicles per day); London North Kensington (In grounds of school in west London suburb); Harwell (On western side of business park surrounded by agricultural land)
Population NR
Age Groups NR
Indoor Source NR
Personal Method No personal monitoring.
Personal Size NR
Microenvironment Size NR
Ambient Size PM_{10}
Component(s) NaCl; Strongly bound H2O; Secondary organic material
Primary Findings Using existing co-located and coincident data it has been possible to show that the removal of three natural components—sea salt (NaCl), strongly bound water and secondary organic matter—would reduce the number of days on which the PM_{10} concentration exceeds 50 $\mu g/m^3$ by about 50%. At each site, amongst the three natural components considered, the strongly bound water makes the greatest contribution to the mean or median concentrations of PM_{10}, followed by NaCl, and SOC respectively. Strongly bound water was shown to have the biggest effect on the number of days on which the PM_{10} concentrations exceeded a value of 50 $\mu g/m^3$; however, removal of estimated NaCl and SOC components also had a noteworthy effect on reducing PM_{10} concentrations. Therefore, application of this proposed measure would make a very major difference to the likelihood of compliance or otherwise with the 24-h limit value for PM_{10}.

Jones et al. (2007, 156615)

Study Design Monitoring living room, child's bedroom, cot, and at 2 heights 1.4 & 0.2 m above the floor
Period NR but probably 2006
Location Perth, Australia
Population Children 0-2 yr
Age Groups 0-2 yr
Indoor Source House age, house type, building material, # of stories, attached garage, main heating fuel, air conditioning
Personal Method NR
Personal Size NR
Microenvironment Size PM_{10}, $PM_{2.5}$, PMT

Ambient Size NR
Component(s) NR
Primary Findings No difference between samples at 0.2 and 1.4 m from floor in 3 PM fractions, no difference between living room, child's bedroom, and crib. Large variability between homes.

Kaur et al. (2005, 086504)

Study Design Exposure assessment, evaluation of exposures between modes of transport, routes, timing
Period April 28-May 23, 2003
Location Street canyon intersection in Central London, UK
Population Users of an urban street canyon intersection
Age Groups NR
Indoor Source NR
Personal Method $PM_{2.5}$ measured usnig high-flow gravimetric personal samplers ($PM_{2.5}$) operating at a flow rate of 16 l/min carried in a backpack with sampling head positioned in personal breathing zone; Ultrafine particles measured using TSI P-TRAK Ultrafine Particle Counters in which ambient aerosol mixes with isopropyl alcohol. Alcohol condenses to form droplets that can be easily counted by a photodetector as they pass through a laser beam.
Personal Size $PM_{2.5}$, Ultrafine particles (UFP, 0.02-1.0um)
Microenvironment Size $PM_{2.5}$, Ultrafine particles (UFP, 0.02-1.0um)
Ambient Size $PM_{2.5}$
Component(s) NR
Primary Findings Personal exposures to $PM_{2.5}$ while walking were significantly lower then while riding in a car or taxi, likely a function of greater distance to roadside. No significant differences in $PM_{2.5}$ were observed between exposures on the high traffic road compared with the backroad. Personal exposure levels were lowest during midday measurements for $PM_{2.5}$ and highest in the early evening. Personal exposures to ultrafine particles were lowest while walking and highest while riding the bus. Exposures to ultrafine particles were also significantly higher on the high traffic road and during morning measurements. Exposure to ultrafine particles were highest in the morning, likely the result of peak traffic density in the morning. Exposure assessment also revealed that the background and curbside monitoring stations were not representative of the personal exposure of individuals to $PM_{2.5}$ and CO at and around a street canyon intersection.

Kaur, et al. (2005, 088175)

Study Design Personal exposure assessment of pedestrians walking along high-traffic urban road
Period April 19, 2004-June 11, 2004
Location Central London, UK
Population Pedestrians
Age Groups NR (adults, presumably)
Indoor Source NR
Personal Method $PM_{2.5}$ gravimetric filter measurement; Ultrafine PM (0.02-1 µm) P-TRAK device; Reflectance reflectometer measurement of $PM_{2.5}$ filter
Personal Size $PM_{2.5}$; Ultrafine PM (0.02-1 µm); Reflectance ("blackness") of $PM_{2.5}$ filter
Microenvironment Size NR
Ambient Size $PM_{2.5}$; Ultrafine PM (0.02-1 µm); Reflectance ("blackness") of $PM_{2.5}$ filter
Component(s) NR
Primary Findings $PM_{2.5}$ pedestrian exposure was well correlated with and above background fixed-site monitoring levels. PM pedestrian exposure was influenced by proximity to curbside and the side of the road walked on.

Kim et al. (2005, 156640)

Study Design Panel study
Period 8/1999-11/2001
Location Toronto, Canada
Population Cardiac-compromised patients
Age Groups Mean age 64 yr
Indoor Source Yes. Tracer molecules/elements were used to investigate sources of indoor PM, including regional long range transport, combustion, local crustal materials. All were statistically significantly associated with indoor $PM_{2.5}$
Personal Method Rupprecht and Patashnick ChemPass Personal Sampling System
Personal Size $PM_{2.5}$
Microenvironment Size NR
Ambient Size $PM_{2.5}$
Component(s) Sulfate, Elemental carbon (EC), Calcium, Magnesium, Potassium, Sodium.

Kim et al. (2005, 156640)

Study Design Panel study
Period 8/1999-11/2001
Location Toronto, Canada

Population	Cardiac-compromised patients
Age Groups	Mean age 64 yr
Indoor Source	Gas range (68%); indoor grill (11%); outdoor barbeque (30%); Gas heating fuel (68%); Oil heating fuel (7%)
Personal Method	Rupprecht and Patashnick ChemPass Personal Sampling System
Personal Size	$PM_{2.5}$
Microenvironment Size	NR
Ambient Size	$PM_{2.5}$
Component(s)	NR
Primary Findings	Personal $PM_{2.5}$ exposures were higher than outdoor ambient levels. Personal $PM_{2.5}$ exposures levels were correlated with ambient levels, mean r = 0.58

Koenig et al. (2003, 156653)

Study Design	Comprehensive exposure assessment. "The research was part of an intensive exposure assessment and health effects panel study of susceptible sub-populations in Seattle from; 1999 through 2002 (Liu et al., 2003, 073841)."
Period	10-day monitoring session winter 2000-2001; 10-day monitoring session spring 2001
Location	Seattle, WA
Population	Asmatic children
Age Groups	6-13 yr
Personal Method	Harvard personal environmental monitors; Continuous PM monitors (nephelometers); Harvard Impactors; TEOM monitors; and exhaled breath measurements into an NO-inert Mylar balloon
Personal Size	$PM_{2.5}$
Microenvironment Size	$PM_{2.5}$
Ambient Size	$PM_{2.5}$
Component(s)	NR
Primary Findings	This study found a consistent relationship between daily eNO values in children with asthma and daily $PM_{2.5}$ measured at fixed sites and on subjects. As hypothesized, the authors found that the use of ICS therapy modified the association between eNO and $PM_{2.5}$. Including ambient NO values for the hour of the home visit from a central site in the model and discarding high NO days (>100 ppb) attenuated the magnitude but did not alter the association between $PM_{2.5}$ and eNO in all analyses. Same-day outdoor, indoor, personal, and central $PM_{2.5}$ levels were associated with eNO in either analysis. These data suggest ambient $PM_{2.5}$ exposure in Seattle is associated with an increase in eNO in children with asthma. Because eNO is a marker of airway inflammation, and PM has been shown to cause inflammation in animal studies, this result is biologically plausible. This finding also agrees with previous asthma research in Seattle that showed associations between $PM_{2.5}$ and lung function decrements in children with asthma.

Koistinen et al. (2004, 156655)

Study Design	Representative Population-based study
Period	Oct 1996-Dec 1997
Location	Helsinki, Finland
Population	Non-smoking adults not exposed to environmental tobacco smoke.
Age Groups	Adults 25-55 yr
Indoor Source	Soil from outdoors, cooking, smoking, aerosol cleaners, sea salt, combustion sources
Personal Method	Aluminum briefcase with personal sampling monitor
Personal Size	$PM_{2.5}$; BS
Microenvironment Size	$PM_{2.5}$; BS
Ambient Size	$PM_{2.5}$; BS
Component(s)	% contribution to $PM_{2.5}$; Outdoor-Indoor-Work-Personal; CoPM * 35 28 32 33; Secondary** 46 36 37 31; Soil 16 27 27 27; Detergents 0 6 2 6; Sea Salt 3 2 1 2
Primary Findings	Population exposure assessment of $PM_{2.5}$, based on outdoor fixed-site monitoring, overestimates exposures to outdoor sources like traffic and long-range transport and does not account for the contribution of significant indoor sources.

Kousa et al. (2001, 025270)

Study Design	Population based exposure assessment
Period	October 1996 to June 1998
Location	Helsinki, Finland; Basel, Switzerland; Prague, Czech Republic; Athens, Greece
Population	Adult urban populations
Age Groups	25-55 yr
Indoor Source	NR, Workplace and residential outdoor samples could not be collected for every participant. The number of non-ETS-exposed participants was particularly small in Prague (12) and Athens (29), and therefore, these results from these cities should be interpreted with caution. The population sampling and sample representativity issues are described in detail in Rotko et al. (2000, 012118), and, for the Basel sample, in Oglesby et al. (2000, 001832).
Personal Method	The 48 h $PM_{2.5}$ exposure was measured by a personal exposure monitor (PEM). Two filter holders were provided for each participant. One 'workday filter' for work and commuting, about 2 8–10 h, and one "leisure time filter" for the remaining time. Microenvironmental $PM_{2.5}$ monitors (MEMs) were placed at the participant's home outdoors and indoors and in their workplaces. The pumps were programmed to run at home outdoors and indoors during the expected leisure time hours and at workplace during

expected working hours of each participant. The personal and microenvironmental $PM_{2.5}$ sampling methods and QA/QC results are presented in detail in Koistinen et al. (1999, 010628).

Personal Size $PM_{2.5}$
Microenvironment Size $PM_{2.5}$
Ambient Size $PM_{2.5}$; PM_{10}
Component(s) NR
Primary Findings In Helsinki the concentration associations are high between the outdoor air concentrations of $PM_{2.5}$ and PM_{10} measured simultaneously at different locations. The highest exposure correlations are observed between the personal exposures and the respective indoor air concentrations. Correlations between the personal exposures and outdoor/ambient air concentrations are considerably lower (all centers). Personal exposures during leisure time correlate better with outdoor/ambient concentrations than during the workday (Helsinki and Prague). Leisure time and workday exposures correlate poorly with each other (all centers). Removing ETS improved the correlations between personal (indoor) air and ambient (outdoor) air, but decreased the correlations between personal exposures and indoor air concentrations and also between the personal exposures during workday and leisure time. In spite of these generalizations, there are considerable differences between the cities.

Koutrakis et al. (2005, 095800)

Study Design Panel study
Period Baltimore 6/28/98-8/22/98 (summer), 2/1/99-3/16/99 (winter); Boston 6/13/99-7/23/99 (summer), 2/1/00-3/12/00 (winter)
Location Baltimore, MD Boston, MA
Population Healthy older adults, children, adults with COPD
Age Groups Children 9-13 y/o; Seniors 65+ y/o; COPD Subjects NR
Indoor Source No
Personal Method Personal exposure samples of $PM_{2.5}$; were collected using a specially designed multipollutant sampler (Demokritou et al. 2001). $PM_{2.5}$ was collected using personal environmental monitors (PEMs) and 37-mm; Teflon filters (Teflo, Gelman Sciences, Ann Arbor MI).
Personal Size $PM_{2.5}$
Microenvironment Size NR
Ambient Size $PM_{2.5}$
Component(s) Elemental Carbon (EC); SO_4^{2-}
Primary Findings Ambient $PM_{2.5}$ and SO_4 are strong predictors of respective personal exposures. Ambient SO_4 is a strong predictor of personal exposure to $PM_{2.5}$. Because $PM_{2.5}$ has substantial indoor sources and SO_4 does not, the investigators; concluded that personal exposure to SO_4 accurately reflects exposure to ambient $PM_{2.5}$ and therefore the ambient component of personal exposure to $PM_{2.5}$ as well.

Kulkarni and Patil (2003, 156664)

Study Design Personal exposure assessment of toxic metals
Period NR
Location Mumbai, India; Two localities or sites, namely, Marol and Sakinaka, denoted as Sites 1 and 2 respectively
Population Outdoor workers- low-income group population working and residing in industrial areas
Age Groups NR
Indoor Source low grade cooking fuel and inadequate ventilation
Personal Method A personal sampler (Cassela/ SKC make), which consists of a diaphragm pump and operates on rechargeable batteries, was used along with a cyclone to measure personal exposure to respirable PM (RPM). The device was fitted to the waist belt of the respondent and connected by a flexible tube to the cyclone, which can be clipped to the shirt collar. The inlet of the cyclone was kept near the breathing level of the respondent. After working hours, the personal sampler was worn by the respondent in his/ her residence. Before sleeping, the sampler was removed from the waist and kept in the "on" condition as close to the breathing level as possible.; The RPM in ambient air was measured simultaneously by using high volume sampler (HVS) with a cyclone attachment for removal of particles with size greater than 10 µm.
Personal Size PM5
Microenvironment Size NR
Ambient Size PM5
Component(s) Lead; Nickel; Cadmium; Copper; Chromium; Potassium; Iron; Manganese
Primary Findings All listed metals were detected in the ambient air where as only Lead, Cadmium, Manganese, and Potassium were detected in personal exposures. Mean daily exposure to lead exceeds the Indian NAAQS by a factor of 4.2. However, ambient concentration of lead conforms to this standard. There is a rising trend in the personal exposures and ambient levels of cadmium. However, they are low and do not pose any major health risk as yet. Personal exposures to toxic metals exceed the corresponding ambient levels by a large factor ranging from 6.1 to 13.2. Thus, ambient concentrations may underestimate health risk due to personal exposure of toxic metals. Outdoor exposure to toxic metals is greater than the indoor (ratios ranging from 2.3 to 1.1) except for potassium (ratio 0.77). However, there is no significant correlation between these two.

Kumar et al. (2006, 129347)

Study Design Use of one year's 24-h monitoring data to model exposure to vehicular emissions.
Period Apr 1991-Feb 1992
Location Mumbai, India

Population	exposure to lead at busy intersections
Age Groups	NR
Indoor Source	NR
Personal Method	NR
Personal Size	NR
Microenvironment Size	NR
Ambient Size	Suspended PM (SPM)
Component(s)	Al As Ca Cu Cr Fe Hg K Mg Mn Na Ni NO_2 Pb O_2 SO_4 SPM *Concentration in ng/m^3, number of samples = 45.
Primary Findings	Application of a hybrid, receptor cum dispersion model is one possible way to evaluate effective emission factors for vehicles in different operating conditions like those at traffic-junctions. The composite approach of receptor and dispersion model gives realistic effective emission factors and will be useful for air quality management.

Lai et al. (2006, 090262)

Study Design	Population-based assessment of urban adult exposures. Identifying determinants of indoor PM concentrations
Period	1996-2000
Location	Athens, Basel, Helsinki, Milan, Oxford, Prague
Population	Homes of urban adults
Age Groups	NR
Indoor Source	Number of people smoking at home, duration of gas stove use. A previous paper is cited for full details on sampling methodology (Jantunen et al, 1998)
Personal Method	No personal exposure assessment was conducted.
Personal Size	NR
Microenvironment Size	NR
Ambient Size	NR
Component(s)	BS
Primary Findings	Number of people smoking at home, outdoor $PM_{2.5}$ conc., wind speed, duration of gas stove use, and outdoor temperature were significant determinants of indoor $PM_{2.5}$. City-specific effects included outdoor $PM_{2.5}$ conc., smoking, and wind speed. Outdoor BS,

Lai et al. (2004, 056811)

Study Design	Personal exposure study
Period	December 1998-February 2000
Location	Oxford, UK
Population	Adults
Age Groups	25-55 yr (avg = 41)
Indoor Source	Cooking, active smoking, passive smoking heating by gas heater
Personal Method	Personal exposure monitors (PEM) were carried by the participant for 48-h personal sampling, and microenvironmental monitors (MEM) were placed inside the participant's home (residential indoor), outside the home (residential outdoor) and in the participant's workplace (workplace indoor).; The $PM_{2.5}$ samplers used were GK2.05 cyclones (KTL) with 2um pore Gelman Teflo filters, and WINS $PM_{2.5}$ impactors for personal exposure and residential outdoor samples, respectively. VOC sampling was accomplished with Perkin Elmer Tenax-TA tubes, CO Enhanced Measurer T15s were used for CO samples, and NO_2 passive sampling badges were used to sample NO_2. No residential outdoor CO or NO_2 samples were taken.
Personal Size	$PM_{2.5}$
Microenvironment Size	$PM_{2.5}$
Ambient Size	$PM_{2.5}$
Component(s)	Ag Cr Mn Si; Al Cu Na Sm; As Fe Ni Sn; Ba Ga P Sr; Br Ge Pb Ti; Ca Hg Rb Tl; Cd I S Tm; Cl K Sb V; Co Mg Se Zn; Zr
Primary Findings	Both the indoor and outdoor environments have sources that elevated the indoor concentrations in a different extent, in turn led to higher personal exposures to various pollutants.; Geometric mean (GM) of personal and home indoor levels of $PM_{2.5}$, 14 elements, total VOC (TVOC) and 8 individual compounds were over 20% higher than their GM outdoor levels. Those of NO_2, 5 aromatic VOCs, and 5 other elements were close to their GM outdoor levels. For $PM_{2.5}$ and TVOC, personal exposures and residential indoor levels (in GM) were about 2 times higher among the tobacco-smoke exposed group compared to the non-smoke exposed group, suggesting that smoking is an important determinant of these exposures. Determinants for CO were visualized by real-time monitoring and that the peak levels of personal exposure to CO were associated with smoking, cooking and transportation activities. Moderate to good correlations were only found between the personal exposures and residential indoor levels for both $PM_{2.5}$ (r = 060; p< 0 001) and NO_2 (r = 047; p = 0 003).

Lai et al. (2004, 156666)

Study Design	Longitudinal exposure assessment
Period	January 4-14, 2001
Location	Taipei, Taiwan; (highway toll station)
Population	Highway toll station workers
Age Groups	19.3-43.6 yr; mean = 25.7 ± 5.71
Indoor Source	Indirect exposure assessment was based on information on (a) lane-specific traffic density (available for all lanes throughout the study period), (b) estimated relationships between lane- and shift-specific traffic density and the average $PM_{2.5}$ concentrations, and (c) information on time periods spent by individuals in different working environments.

Personal Method Direct exposure assessments were conducted by installing battery-operated personal $PM_{2.5}$ monitors (University Research Glassware Corp.) in the booth near the breathing zone of the workers.
Personal Size $PM_{2.5}$
Microenvironment Size NR
Ambient Size NR
Component(s) NR
Primary Findings Toll workers on Taipei highways are exposed high concentrations of $PM_{2.5}$. Mean $PM_{2.5}$ concentration per vehicle in the truck and bus lanes was 6.4 and 3.7 times higher, respectively, than that of ticket- or car-payment car lanes. There was a statistically significant correlation between traffic density and $PM_{2.5}$ concentrations in car lanes with ticket payments and truck and bus lanes. Wind speed and humidity had a significant inverse association with$PM_{2.5}$ concentration in car lanes with ticket and cash payments. Bus and truck lane was the strongest determinant of log ($PM_{2.5}$). The results of this study show that personal exposure to $PM_{2.5}$ can be reliably estimated using indirect traffic approaches.

Larson et al. (2004, 098145)

Study Design Time-series epidemiologic study
Period Sep 26, 2000-May 25, 2001
Location Seattle, Washington
Population "Susceptible Populations"
Age Groups Time-activity diary
Personal Method Harvard Personal Environmental Monitor
Personal Size $PM_{2.5}$
Microenvironment Size $PM_{2.5}$ outside subject's residence, and inside residence
Ambient Size $PM_{2.5}$ at Central outdoor site (downtown Seattle)
Component(s) Light absorbing carbon (LAC) and trace elements
Primary Findings Five sources of $PM_{2.5}$ identified vegetative burning, mobile emissions, secondary sulfate, a source rich in chlorine, and crustal-derived material. The burning of vegetation (in homes) contributed more $PM_{2.5}$ mass on average than any other sources in all microenvironments.

Lee et al. (2006, 098249)

Study Design Exposure assessment for instrument development
Period 11/2003, 5/2004
Location Boston, MA
Population NR
Age Groups No
Personal Method A new personal respirable particulate sampler (PRPS), operating at 5L/min. Sampler is designed to collect PM0.5, PM1.0, $PM_{2.5}$, PM4.5; and PM_{10} as well as O_3, SO_2, and NO_2. Sampler consists of 5 impaction stages, a backup filter, and two diffusion passive samplers. Particles are collected onto a polyurethane foam (PUF) substrate.
Personal Size NR
Microenvironment Size NR
Ambient Size PM>10, $PM_{10\text{-}2.5}$, $PM_{2.5}$
Component(s) NR
Primary Findings In the field, the PM_{10}, $PM_{2.5}$, and sulfate concentrations measured by PRPS were in a very good agreement with those obtained from the; reference samplers.; In the lab, the size distributions measured by the PRPS were found to be much closer to those; measured by the real-time particle sizing instruments than to those measured by the MOI.

Lee et al. (2006, 188450)

Study Design Cross-sectional
Period NR, but prior to 2006
Location Charleston, Ottawa, Clarksville, Ohio
Population Farmers
Age Groups NR, but prior to 2006
Indoor Source Hogs, poultry, cattle, feed, bedding
Personal Method The dust & microorganisms passed thru an optical particle counter and a filter sampler to collect airborne microorganisms.
Personal Size 0.7-1 µm; 1-2 µm; 2-3 µm; 3-5 µm; 5-10 µm; Total dust
Microenvironment Size NR
Ambient Size NR
Component(s) Fungal spores and bacteria
Primary Findings The highest contribution of large particles (3-10 µm) in total particles was found during grain harvesting. In animal confinements the particles were dominated by smaller particles <3 µm. A high proportion of the particles between 2 and 10 µm were fungal spores.

Lewne et al. (2006, 189293)

Study Design Personal exposure study to investigate the occurrence of systematic differences in the PE exposure to motor exhaust and to study if these are influenced by the choice of exposure indicator gaseous or particulate

Period	Sep 1997 to Oct 1999
Location	Stockholm, Sweden
Population	Taxi, bus, and lorry drivers
Age Groups	NR
Indoor Source	NR
Personal Method	PM was measured with a logging instrument Data-RAM, using nephelometric monitoring (Data-RAM measures PM 0.1 to 10 µm)
Personal Size	PM_{10}
Microenvironment Size	NR
Ambient Size	PM_{10}
Component(s)	NR
Component(s)	NO_2
Primary Findings	1) Lorry drivers experienced the highest exposure and taxi drivers the lowest with bus drivers in an intermediate position, regardless of whether NO_2 or particles were used as exposure indicator; 2) The levels of both NO_2 and particles were higher for bus drivers in the city than for them driving in the suburbs; 3) Using diesel or petrol as a fuel for taxis had no influence on the exposure for the drivers, indicating that the taxi drivers' exposure mainly depends on exhaust from surrounding traffic.

Lewne et al. (2007, 156690)

Study Design	7 groups of occupations defined by common or high exposure to DE
Period	Oct 2002-June 2004
Location	Stockholm, Sweden
Population	Persons exposed to DE
Indoor Source	Vehicle exhaust
Personal Method	Pump units and gravitmetric for PM_1& $PM_{2.5}$ and real-time monitoring of elemental carbon and total carbon. Diffusive samplers for NO_2 as an indicator of the gas phase of exhaust.
Personal Size	PM_1, $PM_{2.5}$, and DataRAM ($PM_{0.1-10}$)
Microenvironment Size	NR
Ambient Size	NR
Component(s)	elemental carbon (EC), total carbon (TC)
Primary Findings	Tunnel construction workers had the highest levels of exposure for all indicators, followed by diesel-exposed garage workers. The other 5 groups were significantly lower with no difference between the groups.

Li et al. (2003, 047845)

Study Design	Concurrent 10-min avg indoor and outdoor concentrations of PM_{10} and $PM_{2.5}$ were recorded for 2 days each in 10 homes with swamp coolers
Period	Summer 2001
Location	El Paso, Texas
Population	Cooking, cleaning, walking
Age Groups	NR
Indoor Source	NR
Personal Method	$PM_{2.5}$ and PM_{10}; indoor and outdoor; tapered element oscillating microbalance (TEOM) instruments. 2 days were monitored for $PM_{2.5}$, and 2 for PM_{10}.
Personal Size	NR
Microenvironment Size	NR
Primary Findings	Evaporative coolers were found to act as PM filters, creating indoor concentrations approximately 40% of outdoor PM_{10} and 35% of outdoor $PM_{2.5}$, regardless of cooler type.

Liao et al. (2006, 188451)

Study Design	Case study
Period	January 18-27, 2003
Location	Changhwa, Central Taiwan
Population	Traditional Taiwanese residences
Indoor Source	Chinese style cooking, incense burning, cleaning, and people's moving
Personal Method	A portable laser dust monitor (DM1100) was used to analyze the indoor and outdoor PM characteristics. The DM1100 was placed in a single indoor location, 1.5 m above the floor, adjacent to areas of the kitchen, altar, and living room where the housing activities occurred.
Personal Size	NR
Microenvironment Size	$PM_{0.5-5}$
Ambient Size	$PM_{0.5-5}$
Component(s)	NR
Primary Findings	Results indicate that only 2.6-8% of indoor particles are from outdoor sources. Both indoor and outdoor PM concentrations increase with PM size intervals, as do the deposition rates from cooking events; Authors suggest that "results revealed that cooking and incense burning events were major contributors to indoor concentrations for the particle sizes 1-5 µm. Results demonstrated the importance of knowing the time-activity data and the real-time indoor and outdoor particle size distribution information for

understanding exposure to particles of indoor sources. More importantly, this research illustrates that an exposure assessment based on $PM_{0.5-5}$ measured indoors can provide valuable information on the fate of indoor particles and hazard to human health."

Liu et al. (2003, 073841)

Study Design Part of a larger exposure assessment and health effect panel study
Period Winter 2000-2001 and spring 2001
Location Seattle, WA
Population Children with asthma
Age Groups 6-13 yr
Indoor Source NR
Personal Method Personal $PM_{2.5}$ measurements were collected from each subject using the Harvard personal environmental monitors.
Personal Size $PM_{2.5}$
Microenvironment Size $PM_{2.5}$
Ambient Size $PM_{2.5}$
Component(s) NR
Primary Findings The ambient-generated component of $PM_{2.5}$ exposure was consistently associated with increases in eNO and the indoor-generated component was less strongly associated with eNO. As a result, the eNO results support the hypothesis that $PM_{2.5}$ of outdoor origin could be more potent per unit mass than particles of indoor origin. However, the lung function data indicate that $PM_{2.5}$ of indoor origin might be more potent per unit mass in resulting in decrements of lung functions, although the results across functional tests were not consistent. The authors tentatively concluded that partitioning personal exposure into indoor- versus outdoor-generated particles is useful in understanding the health effects of sources of personal $PM_{2.5}$ and that the effects of indoor- versus outdoor-generated particles differ for different health points.

Liu et al. (2005, 156704)

Study Design Exposure assessment, validation set within a prospective occupational cohort (boiler workers)
Period NR (healthy working adults)
Population
Personal Method Yes, A personal environmental monitor (PEM, Model 200, MSP Co, Shoreview, MN) with a pump at 4L/min
Personal Size PM_{10}
Microenvironment Size PM_{10}
Ambient Size NR
Component(s) Metals Vanadium (V), Nickel (Ni), Iron (Fe), Chromium (Cr), Cadmium (Cd), lead (Pb), Manganese (Mn)
Primary Findings The validation demonstrated good approximations of actual exposures with differences less than 5% for PM.

Liu et al. (2003, 073841)

Study Design Comprehensive exposure assessment
Period 1999-2001
Location Seattle, WA
Population High-risk sub populations
Age Groups Children 6-13 yr; elderly 65-90 yr (one person was below 65 but not specified)
Personal Method Personal $PM_{2.5}$ exposures were determined using the Harvard Personal Environmental Monitor for $PM_{2.5}$ ($HPEM_{2.5}$). Each subject carried an $HPEM_{2.5}$ in the breathing zone for 24 h, except while sleeping, showering, or using the restroom. The monitor was attached to the shoulder strap of either a backpack or a fanny pack that contained the air pump. When the monitor was not worn, it was placed at an elevation of 3–5 feet (e.g., on a table) close to the subjects.; The indoor and outdoor PM concentrations were measured with single-stage inertial Harvard Impactors (HI) and 37 mm Teflon filters for PM_{10} and $PM_{2.5}$. One $HI_{2.5}$-HI_{10} pair was located inside each home in the main activity room and connected to a Medo pump (model vp0935a). Concurrently, one $HI_{2.5}$-HI_{10} pair was located outside each home and connected to a Gast pump (model DOA-V191-AA). All HI sampling periods were for 24 h at a flow rate of 10 L/min. $HI_{2.5}$, HI_{10}, and $HPEM_{2.5}$ were also co-located with the federal reference method monitor for $PM_{2.5}$ ($FRM_{2.5}$) at the central Beacon Hill site, which is located in a semiresidential area (elevation, 300 feet) and is maintained by the Washington State Department of Ecology.
Personal Size $PM_{2.5}$; PM_{10}
Microenvironment Size $PM_{2.5}$; PM_{10}
Ambient Size $PM_{2.5}$; PM_{10}
Primary Findings The average personal exposures to $PM_{2.5}$ were similar to the average outdoor $PM_{2.5}$ concentrations but significantly higher than the average indoor concentrations. Indoor and outdoor $PM_{2.5}$, PM_{10}, and the ratio of $PM_{2.5}$ to PM_{10} were significantly higher during the heating season. The increase in outdoor PM_{10} in winter was primarily due to an increase in the $PM_{2.5}$ fraction. A similar seasonal variation was found for personal $PM_{2.5}$. The children in the study experienced the highest indoor $PM_{2.5}$ and PM_{10} concentrations. Personal $PM_{2.5}$ exposures varied by study group, with elderly healthy and CHD subjects having the lowest exposures and asthmatic children having the highest exposures. Within study groups, the $PM_{2.5}$ exposure varied depending on residence because of different particle infiltration efficiencies; $PM_{2.5}$ exposures among the COPD and CHD subjects can be predicted with relatively good power with a microenvironmental model composed of three microenvironments. The prediction power is the lowest for the asthmatic children.

Lonati et al. (2005, 126171)

Study Design	Comparison sampling of an urban background site, UB during cold season and warm season with no traffic and a vehicle tunnel (TU) cold season.
Period	Aug 2002-Nov 2003
Location	Milan, Italy
Population	Urban population
Age Groups	NR
Indoor Source	NR
Personal Method	NR
Personal Size	NR
Microenvironment Size	NR
Ambient Size	$PM_{2.5}$
Component(s)	EC, OC, Particulate organic matter (OM); Total mass; Chloride, Nitrate, Sulfate, Ammonium, Crustal elements, Metals, undefined+F12
Primary Findings	Higher $PM_{2.5}$ during the cold season, about twice the warm season. Tunnel data are 7 times the urban background. The vehicle contribution to $PM_{2.5}$ is 11% in the warm season and 6% in the cold season.

Lung et al. (2007, 156719)

Period	Weekdays between Nov 1998 and Feb 1999
Location	6 communities in Taiwan, China 2 in Taipei, 2 in Taichung, and 2 in Kaohsiung. Sites are industrial, commercial, residential and mixed.
Age Groups	18 to >70
Indoor Source	Being in kitchen, park, major boulevard, stadium, incense burning, household work, factory, environmental tobacco smoke, traffic, ventilation conditions
Personal Method	Personal Environmental Monitor with a SKC personal pump at 2 L/min, 37 mm Teflon filters
Personal Size	PM_{10}
Microenvironment Size	PM_{10}
Ambient Size	PM_{10}
Component(s)	None
Primary Findings	Outdoor rather than indoor levels contributed significantly to personal exposure.; Important factors include time spend outdoors and on transportation, riding a motorcycle, passing by factories, cooking or being in the kitchen, incense burning at home.

Magari et al. (2002, 034813)

Study Design	Cross-sectional study of boilermakers
Period	NR
Location	NR
Population	NR, probably metal tradesmen
Age Groups	No
Personal Method	Gil-Air 5 personal pump
Personal Size	$PM_{2.5}$
Microenvironment Size	NR
Ambient Size	NR
Component(s)	V, Nr, Cr, Mn, Cu, Pb
Primary Findings	There were statistically significant mean increase in the standard deviation of the normal-to-normal heart rate index (SDNN) of 11.30 msec and 3.98 msec for every 1 $\mu g/m^3$ increase in the lead and vanadium concentrations after adjusting for mean heart rate, age, and smoking status.

Maitre et al. (2002, 156726)

Study Design	Personal (occupational) and ambient (in traffic area) PM and particle-bound PAH exposure assessment. This study evaluates individual airborne exposure to gaseous and particulate carcinogenic pollutants in a group of policemen working close to traffic in the center of Grenoble, France.
Period	Summer (June); winter (January) (year not indicated)
Location	City of Grenoble, located in the southeast of France
Population	Non-smoking policemen working outdoors on foot
Age Groups	NR
Indoor Source	NR
Personal Method	Cyclone and filter with personal sampling pump (SKC, United Kingdom)
Personal Size	Respirable particles (defined in this paper as the mass of particles that pass through a size selective orifice with a 50% collection efficiency at a cut-off aerodynamic diameter of 4 µm)
Microenvironment Size	NR
Ambient Size	Respirable particles (defined in this paper as the mass of particles that pass through a size selective orifice with a 50% collection efficiency at a cut-off aerodynamic diameter of 4 µm)

Component(s) PAH, benxene-toluene-xylenes (BTX), aldehydes; Personal BaP; Personal PAHc; Personal PAH; Personal Benzene; Personal Toluene; Personal Xylene; Personal BTX; Personal Formaldehyde; Personal Acetaldehyde; Personal Aldehydes; Stationary BaP; Stationary PAHc; Stationary PAH; Stationary Benzene; Stationary Toluene; Stationary Xylene; Stationary BTX; Stationary Formaldehyde; Stationary Acetaldehyde; Stationary Aldehydes

Primary Findings The occupational exposure of policemen does not exceed any currently applicable occupational or medical exposure limits. Individual particulate levels should preferably be monitored in Grenoble in winter to avoid underestimations.

Malm et al. (2005, 156729)

Study Design Exposure assessment, characterization of physical and optical properties of carbonaceous aerosol species, and comparison of several semi-continuous monitoring systems

Period July 15-September 4, 2002

Location Yosemite National Park at the Interagency Monitoring of Protected Visual Environments (IMPROVE) monitoring site

Population Different monitoring instruments to quantify ambient aerosol concentrations

Age Groups NR

Indoor Source No personal exposure assessment was conducted

Personal Method NR

Personal Size NR

Microenvironment Size NR

Ambient Size Inorganic ions (sulfate, nitrate), organic carbon in PM_{10} and $PM_{2.5}$ size ranges, elemental carbon

Primary Finding(s) 70% of the organic mass was made up of elemental carbon. 24-h bulk measurements of various aerosol species compared more favorably with each other than with the semi-continuous data.; Semi-continuous sulfate (PILS) correlated well with 24-h measurem

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Mar et al. (2005, 087566)

Period 1999-2001

Location Seattle, WA USA

Population "Older subjects" (< 57 y/o), non-smokers

Age Groups Age 57+ yr

Indoor Source No

Personal Method Harvard impactor

Personal Size $PM_{2.5}$

Microenvironment Size $PM_{2.5}$, PM_{10}

Ambient Size $PM_{2.5}$, PM_{10}

Component(s) NR

Primary Findings Healthy subjects; taking no medications had decreases in heart rate associated with; indoor and outdoor $PM_{2.5}$ and PM_{10}. Healthy subjects on medication; had small increases in systolic blood pressure associated with indoor; $PM_{2.5}$ and outdoor PM_{10}.

McCormack, et al. (2007, 156745)

Study Design Stratified analysis of subjects in the BIESAK study

Period NR but < 2003

Location East Baltimore, Maryland

Population low-income children with asthma

Age Groups 2-6 yr

Indoor Source sweeping, vacuuming, smoking, stove use, burned food, oven, candles/incense, open windows, space heater

Personal Method NR

Personal Size NR

Microenvironment Size PM_{10} and $PM_{2.5}$; in child's bedroom

Ambient Size PM_{10} and $PM_{2.5}$; central monitoring site

Primary Findings Indoor concentrations of $PM_{2.5}$ and PM_{10} were twice as high as the ambient; Sweeping, smoking, and ambient PM contributed significantly to higher indoor concentrations. Sweeping (not vacuuming) increased the PM_{10} by 3-4 $\mu g/m^3$.

Meng et al. (2005, 081194)

Study Design 3 Cohorts, one in New Jersey, 1 in Los Angeles, and 1 in Houston.; Personal, home indoor, and home outdoor samples taken for $PM_{2.5}$.

Period Summer, 1999-spring, 2001

Location Houston, Texas; Los Angeles, California; and Elizabeth, New Jersey

Population

Personal Method MSP monitors on the front strap of the sampling bag near the breathing zone. Pump, battery, and motion sensor were on the hip or back.

Personal Size $PM_{2.5}$

Microenvironment Size $PM_{2.5}$

Ambient Size NR

Component(s) NR

Primary Findings The median contribution of ambient sources to indoor $PM_{2.5}$ using the mass balance approach was 56% for all study homes, 63% for California, 52% for New Jersey, and 33% for Texas.

Meng et al. . (2005, 081194)

Study Design Evaluation of the use of central-site PM, rather than actual exposure, in PM epidemiology
Period Summer 1999-spring 2001
Location 3 cities: Houston (TX), Los Angeles County (CA), and Elizabeth (NJ)
Population People suffering from cardiovascular and respiratory morbidity likely. Not specified
Age Groups All age groups possible. not specified
Indoor Source Likely sources mentioned. not identified
Personal Method NR. Indoor and outdoor sampling conducted
Personal Size $PM_{2.5}$
Microenvironment Size NA
Ambient Size NR
Component(s) EC, OC, S, Si
Primary Findings Use of central-site $PM_{2.5}$ as an exposure surrogate underestimates the bandwidth of the distribution of exposures to PM of ambient origin.

Meng et al. (2005, 058595)

Study Design RIOPA study matched indoor home & outdoor exposure assessment
Period May-October (hot); November-April (cool); (1999-2001)
Location Los Angeles County, CA; Elizabeth, NJ; Houston, TX
Population Non-smoking homes
Indoor Source Combustion (primary); atmospheric (secondary); sulfate, organics, nitrates; mechanically (abrasion) generated.
Personal Method Filter (not specified)
Personal Size NR
Microenvironment Size Indoor home.; $PM_{2.5}$
Ambient Size $PM_{2.5}$, outdoor home
Component(s) Organic and elemental carbon; 24 elements (metals).

Mihaltan et al. (2006, 156761)

Study Design Indoor air monitoring. To assess the effect of smoking on air quality in hospitality venues (restaurant, pubs and bars).
Period NR
Location Romania
Population Restaurant/pubs/bars
Age Groups NA
Indoor Source Smoking
Personal Method Personal aerosol monitor
Personal Size NR
Microenvironment Size Respirable suspended particles, $PM_{2.5}$
Ambient Size NR
Component(s) NR
Primary Findings Hospitality venues allowing indoor smoking are significantly more polluted than indoor smoke-free venues and outdoor air in Romania.

Miller et al. (2007, 156765)

Study Design Exposure Assessment, evaluation study of effectiveness and accuracy of a nephelometer (portable, direct reading photometer) to measure tailpipe emissions of elemental carbon from diesel engines
Period NR
Location In laboratory
Population 2 Exposure assessment methods to measure elemental carbon
Age Groups NR
Indoor Source NR
Personal Method No personal exposure assessment was conducted.
Personal Size NR
Microenvironment Size NR
Ambient Size NR
Component(s) EC, Total Carbon
Primary Findings EC measurements made with a Thermo Electron Personal DataRAM 1200 direct reading nephelometer showed good correlation with EC mass concentrations quantified by thermal optical analysis of $PM_{2.5}$ and PM_1 samples collected on quartz filters (reference NIOS

Miller et al. (2007, 156764)

Study Design	Comprehensive study of key contaminants
Period	Ottawa, Ontario, Canada
Location	NR
Population	NR
Age Groups	NR
Personal Method	$PM_{2.5}$ and PM_{10} filter samples were collected in the living room of each home for 7 days, using SKC sampler model 200 PEM on tared teflon filters. Concurrent $PM_{2.5}$ samples were collected on 47-mm Teflo 2- µm filters with MiniVol air samplers mounted on a tripod ~2 m in front of the house. Particulate samples for analysis of airborne endotoxin, ergosterol, and ß1, 3-D-glucan were collected on a three-piece cartridge equipped with an endotoxin-free polycarbonate filter in the living room sand bedrooms of each home. In the living room, samplers were located at a height between 1.22 m and 1.83 m from the floor and no closer than 0.5 m to surfaces. In bedrooms, the samples were collected form as close to the beds as feasible. BC concentrations were continuously recorded for 7 days with a Magee Scientific Aethalometer in the living room of each house.
Personal Size	NR
Microenvironment Size	$PM_{2.5}$; PM_{10}
Ambient Size	NR
Component(s)	BC; Also assessed: Endotoxin; Ergosterol; Glucan; Dust samples Dust >300; Der p1; Der f1; Fel d1;
Primary Findings	Airborne concentrations of the contaminants measured generally were unremarkable, although the mass of settled dust per square meter was well above that associated with increased asthma and comfort symptoms clinical response, particularly in urban homes. When co-occurrence of inflammatory agents and dust mite allergen burdens in the houses was considered, three homes had higher relative amounts of the agents considered. Based on what is known about clinical interactions between, for example, endotoxin and dust mite allergens, a combined exposure possibly results in an increased relative risk of allergic disease.

Molnár et al. (2005, 156772)

Study Design	Indoor/outdoor exposure assessment related to domestic wood burning
Period	10 February to 12 March 2003
Location	Hagfors, Sweden
Population	Adult residents of Hagfors
Age Groups	NR
Indoor Source	NR
Personal Method	Identical sets of equipment were used for both personal exposure and indoor sampling a GK2.05 (KTL) cyclone connected to a BGI 400S Personal Sampling Pump with a flow rate of 4 l min 1. Each person was equipped with an easily carried shoulder bag with the cyclone and pump attached to it. The cyclone was attached to the shoulder strap and placed near the breathing zone. The personal sampler was worn all day, and at night it was placed next to the stationary indoor sampler in the living room, owing to the noise of the pump.; Two different types of impactors were used for the outdoor sampling one Sierra Andersen series 240, dichotomous virtual impactor that separates particles into two size ranges, coarse and fine particles ($PM_{10\text{-}2.5}$ and $PM_{2.5}$, respectively); and one EPA-WINS impactor (PQ100 EPA-WINS Basel $PM_{2.5}$ Sampler) for collecting $PM_{2.5}$ particles. The outdoor measurements were made at a single location on the roof of a single car garage, belonging to one of the subjects, in the middle of the study area.
Personal Size	$PM_{2.5}$
Microenvironment Size	$PM_{10\text{-}2.5}$; $PM_{2.5}$
Ambient Size	$PM_{10\text{-}2.5}$; $PM_{2.5}$
Component(s)	BS; S; Cl; K; Ca; Mn; Fe; Cu; Zn; Br; Rb; Pb
Primary Findings	Statistically significant contributions of wood burning to personal exposure and indoor concentrations have been shown for K, Ca, and Zn. Increases of 66–80% were found for these elements, which seem to be good wood-smoke markers. In addition, Cl, Mn, Cu, Rb, Pb, and BS were found to be possible wood-smoke markers, though not always to a statistically significant degree for personal exposure and indoor concentrations. For some of these elements subgroups of wood burners had clearly higher levels which could not be explained by the information available. Sulfur, one of the more typical elements mentioned as a wood-smoke marker, showed no relation to wood smoke in this study due to the large variations in outdoor concentrations from LDT air pollution. This was also the case for $PM_{2.5}$ mass. Personal exposures and indoor levels correlated well among the subjects for all investigated species, and personal exposures were generally higher than indoor levels. The correlations between the outdoor and personal or ind

Molnár et al. (2006, 156773)

Study Design	Cross-sectional
Period	Autumn and spring in 2002 and 2003
Location	Goteborg, Sweden,
Population	Persons living in urban settings
Age Groups	20 subjects 20-50 yr randomly selected from the population and 10 from departmental colleagues.
Indoor Source	Nr
Personal Method	The volunteer subjects had a small shoulder bag with one $PM_{2.5}$ cyclone and a pump attached. Intake was in the breathing zone. Pump was carried during the day and placed next to the indoor cyclone during the night.; Ten subjects from their staff wore 2 sets of sampling equipment near the breathing zone. A GK2.05 cyclone for $PM_{2.5}$ and a Triplex cyclone for PM_1in a small shoulder bag.
Personal Size	$PM_{2.5}$ and PM_1
Microenvironment Size	NR

Ambient Size NR
Component(s) S; Cl; K; Ca; Ti; V; Mn; Fe; Ni Cu; Zn Br Pb.
Primary Findings $PM_{2.5}$ personal exposures were significantly higher than both outdoor and urban background for the elements Cl, K, Ca, Ti, Fe, and Cu.; Personal exposure was also higher tan indoor levels of Cl, Ca, Ti, Fe, and Br, but lower than outdoor Pb. Residential outdoor levels were significantly higher than the corresponding indoor levels for Br and Pb, but lower for Ti and Cu. The residential levels were also significantly higher than the urban background for most elements.

Molnár et al. (2007, 156774)

Study Design Microenvironmental monitoring of PM and elements in 10 schools, 10 preschools, and 20 non-smoking homes.
Period 1 Dec 2003- 1 July 2004
Location Stockholm, Sweden
Population Children
Age Groups 6-11 yr (no pre-school children) but sampling was conducted at 10 preschools.
Indoor Source Smoking, gas stoves,
Personal Method NR
Personal Size NR
Microenvironment Size NR
Ambient Size NR
Component(s) S; K; Ca; Ti; V; Cr; Mn; Fe; Ni; Cu; Zn; Br; Pb
Primary Findings Significantly lower indoor concentrations of S, Ni, Br and Pb, elements from long-range transported air masses, were found in all locations. Only Ti was significantly higher indoors in all locations, probably because of TiO2 in paint pigment. Similar differences were found during both seasons for homes and schools. At preschools the infiltration of the long-range transported elements S, Br and Pb was lower in winter than in spring, and also the crustal elements Ti, Mn and Fe had higher indoor concentrations during spring. There were spatial differences outdoors, with significantly lower concentrations of elements of crustal and traffic origin in the background area community.

Monkkonen et al. (2005, 156775)

Study Design Human exposure assessment in homes in India
Period Nagpur Mar to Oct 2002; Mysore Aug to Dec 2002
Location Nagpur and Mysore, India
Population Residential homes in India
Age Groups NR
Indoor Source Yes; cooking w/ kero. and LPG; Toaster; Burning incense; Infiltration of outdoor air; Burning coconut husks
Personal Method TSI Condensation Particle Counter Model 3007 (CPC counts all particles >10 nm); TSI Model 8520 Dust Trak; $PM_{2.5}$ Environmental Monitor with Whatman PTFE membrane filters and gravimetric analysis
Personal Size $PM_{2.5}$ for mass ($\mu g/m^3$); Total PM for counts ($particles/cm^3$)
Microenvironment Size $PM_{2.5}$ for mass ($\mu g/m^3$); Total PM for counts ($particles/cm^3$)
Ambient Size Total PM for counts ($particles/cm^3$)
Component(s) NR
Primary Findings The maximum concentrations observed in most cases were due to indoor combustion sources. Besides cooking stoves that use LPG or kerosene as the main fuel, high indoor concentrations can be explained by poor ventilation systems.

Mwaiselage et al. (2006, 156789)

Study Design Cross-sectional; personal monitoring. To determine the effects of cement exposure on acute respiratory health.
Period June-August 2001
Location Dar es Salaam, Tanzania
Population Cement factory workers
Age Groups NR
Indoor Source Cement production
Personal Method Cellulose Acetate Filter, Sidekick pump
Personal Size Respirable dust, total dust
Microenvironment Size NR
Ambient Size NR
Component(s) Ca, Al, Fe, K
Primary Findings Results of Cox Regression analysis showed that prevalence ratios for cough, short breathness and stuffy nose for high exposed workers in the production department compared to low exposed workers in the low exposed workers working in the maintenance department and the administration building are 6.7, 4.5 and 1.9 respectively. Cross shift decrease in PEF was more in the higher among high exposed workers (7.6%) than low exposed workers (2.7%). The observed acute acute respiratory health effects are most likely related to exposure of workers to high concentrations of irritant cement dust.

Na and Cocker (2005, 156790)

Study Design Human exposure assessment
Period Sept. 2001-January 2002

Location	Mira Loma, CA
Population	Residential homes and a high school
Age Groups	NR
Indoor Source	Indoor EC (elemental carbon) concentrations primarily of outside origin; Indoor $PM_{2.5}$ significantly influenced by indoor OC (organic carbon) sources, including indoor smoking.
Personal Method	$PM_{2.5}$ Particle trap impactor with 47 mm Teflo substrates; EC/OC Particle trap impactor with 47 mm QAT Tissuquartz quartz fiber filter, analysis by thermal.optical carbon aerosol analyzer (NIOSH Method 5040)
Personal Size	$PM_{2.5}$
Microenvironment Size	NR
Ambient Size	$PM_{2.5}$
Component(s)	EC (Elemental carbon); OC (Organic carbon)
Primary Findings	Indoor $PM_{2.5}$ was significant influenced by indoor OC sources. Indoor EC sources were predominantly of outdoor origin.

Naumova et al. (2002, 026105)

Study Design	Exposure assessment
Period	6/1999-5/2000
Location	Los Angeles County, CA; Houston, TX; Elizabeth, NJ
Population	US. General population
Age Groups	NR
Indoor Source	No
Personal Method	None-area sampling only (in home and outdoors)
Personal Size	NR
Microenvironment Size	$PM_{2.5}$
Ambient Size	$PM_{2.5}$
Component(s)	PAH (total and specific)
Primary Findings	See Component Column. Many of the study findings pertain to combined particle-bound and gas-phase PAHs, and are not presented here.

Naumova et al. (2003, 089213)

Study Design	RIOPA Study-PAH partitioning indoor and outdoor. To evaluate the hypothesis that outdoor air pollution contributed strongly to indoor air pollution.
Period	July 1999-June 2000
Location	Los Angeles, CA, Houston, TX, Elizabeth, NJ
Population	Houses
Age Groups	NR
Indoor Source	NR
Personal Method	Modified MSP Samplers, 37 mm quartz filter
Personal Size	NR
Microenvironment Size	Filter, $PM_{2.5}$
Ambient Size	Filter, $PM_{2.5}$
Component(s)	Organic Carbon (OC), Elemental Carbon (EC)
Primary Findings	Multiple linear regression (MLR) log PAH particulate partition coefficient (kp) vs log vapor pressure coefficient (std) 0.888 (0.009) fraction of elemental carbon in PM coefficient (std) 3.686 (0.238) fraction of elemental carbon in PM coefficient (std) 0.469 (0.055) temperature coefficient (std) -0.0456 (0.002) intercept (std) 8.398 (0.604) R2 = 0.845. Both EC and OC carbon are important predictors of gas/particle partitioning of PAHs, with EC being a better predictor. Because EC is highly correlated with (and is a good tracer of) primary combustion-generated OC, this result suggests that PAHs more readily sorb on combustion-generated aerosol containing EC. Enrichment of the indoor aerosol in nin-combustion OC suggests that sorption of PAHs is more important in the indoor air compared to the outdoor air. The MLR developed in this work will improve prediction of gas/particle partitioning of PAHs in indoor and outdoor air.

Nerriere et al. (2005, 088630)

Study Design	Exposure assessment
Period	2001-2003; Lung cancer mortality 1999
Location	4 French Cities (Grenoble, Paris, Rouen, Strasbourg)
Population	6-13 y/o children not exposed to passive smoke; 30-71 y/o adults (average age ~40y/o) not occupationally exposed
Personal Method	yes, using Harvard Chempass worn in a backpack
Personal Size	$PM_{2.5}$
Microenvironment Size	NR
Ambient Size	$PM_{2.5}$
Component(s)	NR
Primary Findings	Number of cases attributable to $PM_{2.5}$ exposure (95% CI); attributable Fraction (%) (95% CI) Paris 303 (42-553); 8 (1-16); Grenoble 12 (3-22); 10 (3-19); Rouen 19 (3-35); 10 (2-19); Strasbourg 43 (7-71); 24 (4-10).

Nerriere et al. (2005, 089481)

Study Design Exposure assessment. stratified sampling of children and adults in 3 environments high traffic emissions, local industrial sources, and urban background.
Period "Hot" season May-June and "cold" season Feb-Mar. Grenoble in 2001, Paris in 2002, Rouen in 2002-2003, Strasbourg 2003.
Location Grenoble, Paris, Rouen, and Strasbourg, France
Population Persons living, working, or going to school in 3 urban areas one highly exposed to traffic emissions, one influenced by local industrial sources, and a background urban environment. Industrial sources of pollution were present in each city.
Age Groups 6-13 yr and 20-71 yr. All non-smokers and not exposed to environmental tobacco smoke or industrial air pollution.
Indoor Source Daily activity diaries used to do
Personal Method Rucksack with Harvard ChemPass
Personal Size $PM_{2.5}$, PM_{10}
Microenvironment Size NR
Ambient Size $PM_{2.5}$, PM_{10}
Copollutant(s) NO_2
Primary Findings The difference between ambient air concentrations and average total exposure is pollutant specific. $PM_{2.5}$ and PM_{10} concentrations underestimate population exposures across almost all cities, season, and age groups, the opposite is true for NO_2.

Ng et al. (2005, 155996)

Study Design Study is to model the dispersion of the 911 WTC destruction cloud to areas of the city and boroughs using "representative persons." Input data are from extant monitoring stations throughout the area.
Period 14 Sep., 2001 to 30 Sep., 2001
Location Lower Manhattan, New York City, NY
Population NYC residents
Age Groups NR, but both adults and children
Indoor Source Smoking, cooking
Personal Method Simulated
Personal Size $PM_{2.5}$
Microenvironment Size $PM_{2.5}$
Ambient Size $PM_{2.5}$
Component(s) NR
Primary Findings Although the outdoor $PM_{2.5}$ was lower than the NAAQS value, personal exposure levels were higher than outdoor and might be of concern.

Nikasinovic et al. (2006, 156807)

Study Design cross-sectional
Period Oct 1999-Jun 2002
Location Paris, France
Population Asthmatic children
Age Groups 7-14 yr
Indoor Source Presence of pets, smoking in the home, house dust mites, home ventilation frequency, allergies to grass, cats, pollen, gas cooking, barometric pressure.
Personal Method Active sampler in a rucksack carried by the children whenever they moved.
Personal Size $PM_{2.5}$
Microenvironment Size NR
Ambient Size PM_{10}
Copollutant(s) Ozone
Primary Findings Pollutant concentrations did not differ between the 2 groups. In asthmatic children only personal $PM_{2.5}$ levels were correlated to nasal markers after adjustment for age, sex, house mites, pollens, cat, tobacco smoke, barometric pressure, and respiratory infection.

Noullett et al (2006, 155999)

Study Design Cohort
Period 5 February to 16 March 2001
Location Prince George, British Columbia
Population Children
Age Groups 10-12 yr
Indoor Source NR, Each child completed a time activity diary every 30 min on the days that they carried the monitor. A motion sensor (HOBO, Onset Computer Corporation) was also placed in each pack and data from the sensor was downloaded each morning and then compared to each child's time activity diary as a quality assurance measure.
Personal Method $PM_{2.5}$ Harvard Personal Environment Monitors (HPEM2.5) with a PTFE Teflon filter (Pall Gelman R2PJ037) were used for both the ambient and personal sampling. At ambient sampling sites, the HPEM2.5 was suspended approximately 4 ft above the school rooftop (20 ft from the ground at all schools), connected to a large flow controlled pump and situated in an open area on the roof free of air vents, exhausts or intakes. BGI air sampling pumps and battery packs (BGI-400S and BGI-401) were used for the

personal monitoring and were contained in a child-size backpack. The sampler was attached to the strap of the backpack in the breathing zone of the child with the inlet facing downwards and protected by a 4-in piece of plastic tubing. Subjects were required to wear the pack whenever possible and otherwise to keep the pack close to them and as close to their breathing zone as possible

Personal Size $PM_{2.5}$
Microenvironment Size NR
Ambient Size $PM_{2.5}$
Component(s) SO_4; ABS (light absorbing carbon)
Primary Findings In Prince George, a combination of topography, meteorological conditions and location of ambient sources resulted in episodic levels of fine PM during the short study period in the winter of 2001. Thermal inversions were moderately associated with both high ambient levels and personal exposures and were likely responsible for the spatial variation and, in combination with wind, the temporal variation in ambient concentrations throughout the city. The clear link between thermal inversions and both high ambient levels and measured personal exposures during $PM_{2.5}$ episodes support management strategies to reduce ambient sources during periods of limited dispersion in an effort to reduce exposure levels. Despite the significant spatial variation found in ambient levels throughout the city for all three measures, there was a high correlation between the outdoor sites suggesting that a single monitor would represent temporal trends. Similar to the findings in other studies, both sulfate and light

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O'Neill et al. (2004, 087429)

Study Design Time-series epidemiologic study of PM_{10}-associated mortality, comparison of different samplers
Period January 1, 1994-December 30, 1998
Location 5 sites in Mexico City, Mexico
Population Urban environments
Age Groups NR
Indoor Source NR
Personal Method No personal exposure assessment was conducted
Personal Size NR
Microenvironment Size NR
Ambient Size PM_{10}, $PM_{2.5}$
Component(s) NR
Primary Findings PM_{10} levels were higher in the winter. PM_{10} levels measured using different methods were not well correlated with each other. Re-analysis of associations between $PM_{2.5}$ and mortality with sensitivity analyses (non-parametric modeling) produced lower eff

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Offenberg et al. (2004, 156821)

Study Design Exposure assessment
Period 6/1999-5/2000
Location Los Angeles County, CA; Houston, TX; Elizabeth, Nj
Population US. General population
Age Groups NR
Indoor Source No
Personal Method None-area sampling only (in home and outdoors)
Personal Size NR
Microenvironment Size $PM_{2.5}$
Ambient Size $PM_{2.5}$
Component(s) Chlordane
Primary Findings Geometric mean particle-bound chlordane concentrations were higher indoors relative to outdoors, suggesting indoor sources.

Ogulei et al. (2006, 156823)

Study Design Exposure Assessment
Period 11/1999-3/2000
Location Reston, VA
Population US homes
Age Groups NR
Indoor Source Yes. Nine primary sources of PM were identified gas burner; use (boiling water), deep-frying tortillas and miscellaneous; cooking of dinner, burning of citronella candle, combined gas burner and gas oven use (broiling salmon), sweeping/vacuuming, use of electric toaster; oven, traffic, wood smoke, and pouring of kitty litter.
Personal Method None
Personal Size NR
Microenvironment Size A range 0.01-20.0 mm
Ambient Size NR
Component(s) NR
Primary Findings Each particle source identified in the study produces distinct particle size distributions.

Pang et al. (2002, 037057)

Study Design Field test of prototype Personal Particulate Organic and Mass Sampler

Period November 2000-May 2001
Location Seattle, WA
Population NR
Age Groups NR
Indoor Source NR
Personal Method Outdoor sampling for $PM_{2.5}$ massThe PPOMS was co-located with two Federal Reference Method (FRM) samplers and a HPEM sampler at the Beacon Hill EPA Air Quality and Particulate Speciation Monitoring Site. Samples were collected on each sample day for 24 h, starting at 0:00 PST. Indoor sampling for particulate carbon the PPOMS was co-located with an integrated particle sampler and a Harvard impactor ($HI_{2.5}$) inside of two residences. Five samples were collected at each house over the course of several days.
Personal Size NR
Microenvironment Size NR
Ambient Size $PM_{2.5}$
Component(s) EC; OC
Primary Findings "This study shows that the PPOMS design provides a 2.5 µm size-selective inlet that also prevents the adsorption of gas-phase SVOC onto quartz filters, thus eliminating the filter positive artifacts. The PPOMS meets a significant current challenge for indoor and personal sampling of particulate organic carbon. The PPOMS design can also simplify accurate ambient sampling for $PM_{2.5}$."

Paschold et al. (2003, 156847)

Study Design Concurrent 48-h indoor and outdoor concentrations of PM_{10} and $PM_{2.5}$ in 10 homes with swamp coolers
Period Summer of 2001
Location El Paso, Texas
Population Homes with evaporative coolers
Age Groups NR
Indoor Source NR
Personal Method NR
Personal Size NR
Microenvironment Size PM_{10} and $PM_{2.5}$
Ambient Size NR
Component(s) Geologic material; Sodium; Magnesium; Aluminum; Potassium; Calcium; Titanium; Manganese; Iron; Trace metals; Copper; Zinc; Barium; Lead
Primary Findings Indoor elemental concentrations in PM_{10} were approximately 50–70% lower than outdoor concentrations in 9 of 10 homes, consistent with the PM_{10} indoor/outdoor (I/O) mass concentrations previously reported. $PM_{2.5}$ I/ O ratio correlations were not as strong as for PM_{10}; however, reduced correlations could be attributed to a pattern of recurring outlier data pairs, consisting of the same 3 or 4 elements in all 10 homes.

Polidori et al. (2007, 156877)

Study Design time-series epidemiologic study
Period Site A (Group 1 [G1]); -Phase 1 July 6 to August 20, 2005; -Phase 2 October 19 to December 10, 2005; Site B Group 2 [G2]); -Phase 1 August 24 to October 15, 2005; -Phase 2 January 4 to February 18, 2006
Location Los Angeles, California (Two Retirement homes)
Population Elderly residents of Los Angeles, California retirement homes
Age Groups NR
Indoor Source No
Personal Method Two identical sampling stations were installed at each location, one indoors and one outdoors. The indoor sampling station at site A was located in the recreational area of the first community's main building, adjacent to a construction site where work was ongoing. The indoor sampling area at site B was situated in the dining room of the second community's main building. At both sites, the outdoor station, set up inside a movable trailer, was positioned within 300m from the indoor station. Two □-attenuation mass monitors (BAMS) (Model 1020) were used at each indoor and outdoor sampling station to measure hourly $PM_{2.5}$ mass concentrations. Continuous NO, NO_2, and CO measurements were taken indoors and outdoors using Thermo Environmental NOX analyzers (Model 42), and Dasibi CO Analyzers (Model 3008) respectively. O_3 concentrations were also monitored at each sampling station by using API Ozone Analyzers (Model 400A). At both indoor and outdoor sampling areas, a water-based condensation particle counter (CPC model 3785), and a semicontinuous OC-EC analyzer (Model 3F) were operated side by side. A modified National Institute for Occupational Safety and Health (NIOSH) analysis protocol was used here to evolve particulate OC and EC.
Personal Size NR
Microenvironment Size $PM_{2.5}$
Ambient Size $PM_{2.5}$, PN
Component(s) OC; EC; OC1; OC2-4
Primary Findings Measured indoor and outdoor concentrations of $PM_{2.5}$, OC, EC, PN, O_3, CO, and NOX were generally comparable, although at G2, a substantial peak in indoor OC, PN, and $PM_{2.5}$ (probably from cooking) was typically observed between 6:00 and 9:00 am. The study average percentage contribution of outdoor SOA to outdoor particulate OC was 40% and varied between 40% and 45% in the summer (during G1P1) and 32% and 40% in the winter (during G2P2). The low AERs (0.25-0.33 h-1) calculated for G1 and G2 are consistent with the structural characteristics of the sampling sites, the low number of open windows and doors, and the presence of central air conditioners. F_{inf} estimates were highest for EC and also for OC. Lower F_{inf} values were obtained for $PM_{2.5}$ and PN,

because these compounds are composed of both volatile and nonvolatile inorganic and organic compounds. Based on a single compartment mass balance model, it was found that 13-17% (G2P2) to 16-26% (G1P1) of measured indoor OC was emitted or formed indoors. Although the G2 indoor site was characterized by higher indoor morning OC peaks because of cooking, the overall contribution of indoor sources to measured indoor OC was higher at the G1 site. The modeling results also showed that the measured indoor $PM_{2.5}$ emitted or formed indoors was highly variable (from 6-21% at G1P1 to 45-51% at G1P2). The average percentage contribution of indoor SOA of outdoor origin to measured indoor OC varied from ~35% (at site 1) to ~45% (at site 2). Also, outdoor-generated primary OC composed, on average, 36-44% of measured indoor OC during G2P1 and G1P1 respectively.

Poupard et al. (2005, 074025)

Study Design	Explore relationships between indoor and outdoor air quality
Period	NR
Location	La Rochelle, France
Population	School buildings
Age Groups	NR
Indoor Source	No
Personal Method	GRIMM 1.108 analyzer
Personal Size	15 size intervals from 0.3 to 15 microns
Microenvironment Size	NR
Ambient Size	15 size intervals from 0.3 to 15 microns
Component(s)	NR
Primary Findings	Influence of room occupancy on particle concentrations indoors changes with particle size; Indoor ozone and particle concentrations are negatively correlated.

Price et al. (2003, 098082)

Study Design	Exposure assessment, comparison of PM_{10} samplers
Period	November 2000 to August 2001
Location	Sunderland, England (northeast England), monitoring at curbside
Population	Urban Populations near high traffic areas
Age Groups	NR
Indoor Source	NR
Personal Method	No personal exposure assessment was conducted
Personal Size	NR
Microenvironment Size	NR
Ambient Size	PM_{10}
Component(s)	NR
Primary Findings	Correlation between TEOM and partisol appeared to be seasonal, with strongest correlation in the summer when ambient PM_{10} concentrations were relatively low. In the winter and spring, when PM_{10} levels are higher, the Partisol sampler records grea

Ramachandran et al. (2003, 190112)

Study Design	Matched $PM_{2.5}$ 24-h and 15-min averages at 9-10 residences in each of 3 communities and at 3 central sites, in 3 seasons.
Period	The measurements were made over 3 seasons—spring (April 26–June 2), summer (June 20–August 10), and fall (September 23–November 20) of 1999.
Location	Phillips, East St. Paul, and Battle Creek, Metropolitan Minneapolis-St. Paul, Minnesota
Population	Urban residential communities
Age Groups	23 females, 9 males; mean age 42 ± 10, range 24–64 yr
Indoor Source	No
Personal Method	NR
Personal Size	NR
Microenvironment Size	$PM_{2.5}$ in residences
Ambient Size	At 3 central sites, $PM_{2.5}$
Component(s)	NR
Primary Findings	Outdoor $PM_{2.5}$ concentrations across the Minneapolis– St. Paul area appear to be spatially homogeneous on a 24-h time scale as well as on a 15-min time scale. Short-term average outdoor $PM_{2.5}$ concentrations can vary by as much as an order of magnitude within a day.

Riojas-Rodríguez et al. (2006, 156913)

Study Design	Panel Study
Period	12/2001-4/2002
Location	Mexico City, Mexico
Population	Patients with heart disease
Age Groups	Avg Age 55 yr (range 25-76)
Indoor Source	Nub

Personal Method	Yes, using nephelometers (personal data ram (PDR) model 1200, Monitoring Instruments for the Environment, Inc) connected to a 4L/min pump
Personal Size	$PM_{2.5}$
Microenvironment Size	NR
Ambient Size	NR
Component(s)	NR
Primary Findings	Authors found a decrease in HRV measured as high frequency (Ln) (coefficient = -0.008, 95% confidence interval (CI) to -0.015, 0.0004) for each 10 microg/m3 increase of personal $PM_{2.5}$ exposure.

Robinson et al. (2007, 156054)

Study Design	A pollution mapping exercise was undertaken to measure average pollution levels on a number of transects across the New South Wales Valley and the variation with height and land use was determined. Spatial variation was then used to predict population exposure to $PM_{2.5}$ pollution and the effect on health.
Period	Pollution measurements were made between 17 July 1996 and 10 September 1996
Location	Armidale, New South Wales, Australia
Population	Armidale, New South Wales, Australia
Age Groups	NA
Indoor Source	NR
Personal Method	A portable Radiance Research M903 integrating nephelometer was used to measure ambient air pollution at four transects; Ambient air pollution was also measured using a fixed site Belfort nephelometer
Personal Size	NR
Microenvironment Size	NR
Ambient Size	$PM_{2.5}$
Component(s)	NR
Primary Findings	1) Considerable variability was observed in winter woodsmoke pollution levels; 2) A small number of badly operated heaters can have a large influence on local air quality; 3) Pollution was generally higher in residential areas; 4) Annual exposure to $PM_{2.5}$ pollution in Armidale from woodsmoke was double that from all sources in Sydney.

Rojas-Bracho et al. (2004, 054772)

Study Design	Cohort, repeated measures. 18 COPD patients in non-smoking homes were sampled either in winter 1996 or 1997. 16 of these also were sampled in the summer. All subjects were sampled for 6 consecutive days in winter, and one or two sets of 6 consecutive days in the summer.
Period	1996-1997
Location	Boston, Massachusetts
Population	COPD patients
Age Groups	**H**ousecleaning, cooking, transport in motor vehicles, low-effort home activities, moderate-effort home activities, activities in public places, and resting or sleeping.
Personal Method	PEM attached to shoulder strap of a bag (near breathing zone) containing the pump and batteries.
Personal Size	$PM_{2.5}$, PM_{10}, & $PM_{2.5-10}$
Microenvironment Size	$PM_{2.5}$, PM_{10}, & $PM_{2.5-10}$
Ambient Size	NR
Component(s)	NR
Primary Findings	During both seasons personal exposures were higher than indoor or outdoor means, except the winter indoor concentrations were higher than the personal or outdoor.

Rotko et al. (2002, 037240)

Study Design	European multi-city air pollution study
Period	Athens, Greece (A) 26 January 1997–4 June 1998; Basel, Switzerland (B) 3 February 1997–23 January 1998; Milan, Italy (M) 10 March 1997–23 May 1998; Oxford, UK (O) November 1998–7 October 1999; Prague, Czech Republic (P) 3 June 1997–4 June 1998; Helsinki, Finland (H) 26 September 1996–10 December 1997
Location	Athens, Greece (A); Basel, Switzerland (B); Milan, Italy (M); Oxford, UK (O); Prague, Czech Republic (P); Helsinki, Finland (H)
Population	Adults
Age Groups	25-55 yr
Indoor Source	NR
Personal Method	Personal $PM_{2.5}$ exposures were collected on two different filters one for the working hours including commuting (personal work) and the other for the remaining hours of 48-h measurement period (personal leisure time). In addition to personal exposure monitoring, $PM_{2.5}$ concentrations were measured in each home (indoors and outdoors) and workplace (indoors). The $PM_{2.5}$ concentration measured at work was the avg of two consecutive workdays and at home of the remaining hours of the 48-h monitoring period. $PM_{2.5}$ personal cyclones were used as pre-separators at flow rate of 4 l min 1 [this seems wrong] and the EPA-WINS impactors were employed at 16.7 l min 1 for the microenvironment measurements with Gelman Teflo filters (37 and 47 mm, respectively).
Personal Size	$PM_{2.5}$
Microenvironment Size	$PM_{2.5}$
Ambient Size	$PM_{2.5}$
Copollutant(s)	NO_2

Primary Findings * There was a large variation in the levels of air pollution annoyance between the six studied cities. The highest annoyance levels were experienced while in traffic. *The significant determinants of air pollution annoyance were the city, self-reported sensitivity to air pollution and respiratory symptoms, downtown residence and gender of the subject.; * No consistent or significant correlations were seen between the individual levels of annoyance and exposure concentrations to either $PM_{2.5}$ or NO_2. * High air pollution annoyance in traffic, however, was significantly associated with home outdoor concentrations of both $PM_{2.5}$ and NO_2 and downtown living (NO_2^- model). *When average annoyance levels and concentrations were averaged for each city, the perceived annoyance levels at home correlated highly with the measured personal 48-h $PM_{2.5}$ and NO_2 exposures and home indoor NO_2 concentration, annoyance at work correlated with personal workday exposure and workplace $PM_{2.5}$ concentrations, and annoyance in traffic wi

Salma et al. (2005, 156937)

Study Design 2 types of samplers collected aerosols in an urban area. 23 samples were collected with each device separately for day and night.
Period Spring 2002
Location Budapest, Hungary
Population urban dwellers
Age Groups NR
Indoor Source NR
Personal Method NR
Personal Size NR
Microenvironment Size NR
Ambient Size See direct quote in the note below
Component(s) Al; Si; Ca; Ti; Fe; Cl; Zn; Na; Mn; Ni; Cu; Pb; K; S; Br
Primary Findings The variation in the overall size distributions and RMCs for the various elements indicated the existence of multiple sources, including vehicular (both combustion and frictional) and industrial emissions, resuspension of road and soil dust, and long-range transport of air masses. The significant coarse mode for some typical anthropogenic elements (Cu and Zn) and the observed coarse mode concentration differences between daytime periods and nights (e.g., for Ca) point to the importance of frictional sources and road dust resuspension in cities, which are both primarily related to road traffic.

Salma et al. (2007, 113850)

Study Design examination of aerosol air quality and its temporal variation in the Budapest metro
Period April 20 and 21, 2006
Location Budapest, Hungary
Population underground metro commuters
Age Groups NR
Indoor Source No. Air monitoring equipment consisted of a tapered element oscillating microbalance (TEOM), a wind monitor (Campbell), and a laboratory-made Gent-type stacked filter unit (SFU) aerosol sampler. OM was equipped with a PM_{10} inlet facing upwards and was operated with the filter heated to 40 1C to prevent moistening. The sampling station was ventilated without filtration by drawing air from the opposite platform to the roof level of a 12-story building next to the station.
Personal Method No personal monitoring. In situ aerosol measurement and sample collection at the metro station.
Personal Size PM_{10}-2.0 and PM2.0
Microenvironment Size NA
Ambient Size NR
Component(s) 30 elements (Na, Mg, Al, Si, P, S, Cl^-, K, Ca, Ti, V, Cr, Mn, Fe, Ni, Cu, Zn, Ga, Ge, As, Se, Br, Rb, Sr, Y, Zr, Nb, Mo, Ba, and Pb)
Primary Findings The concentrations observed in the Astoria underground station were clearly lower (by several orders of magnitude) than the corresponding workplace limits.

Sanderson and Farant (2004, 156942)

Study Design Indoor and outdoor air monitoring of PAH. Investigate the relationship between indoor and outdoor PAH.
Period NR
Location Canada
Population Residential homes in neighborhoods around aluminum smelting plant
Age Groups NR
Indoor Source NR
Personal Method Indoor quartz filter, XAD-2 Resin Outdoor glass fiber filter
Personal Size NR
Microenvironment Size NR
Ambient Size NR
Component(s) 4-6 ring PAHs on indoor particle
Primary Findings Indoor concentration of 4-6-ring PAH linked to outdoor sources in residences without any major indoor source, but with industrial facility as the main outdoor source. Study suggests that simultaneous measurements of indoor and outdoor concentrations of PAH >4 rings predominantly associated with fine PM could provide useful estimates of particle infiltration efficiency.

Sarnat et al. (2006, 089166)

Study Design Outdoor-indoor pollutant infiltration, occupied residences

Period July 28, 2001-February 25, 2002
Location Los Angeles, CA
Population NR
Indoor Source Yes; cleaning, cooking, home ventilation (open windows/doors), kitchen fans, air conditioner/heating usage, number of occupants, nearby roadways
Personal Method NR
Personal Size NR
Microenvironment Size $PM_{2.5}$, Particle number
Ambient Size $PM_{2.5}$
Component(s) BC (nonvolatile component); NO3 (volatile component)
Primary Findings 1) Infiltration rate for $PM_{2.5}$ was intermediate, while BC was highest, NO3 lowest; 2) Infiltration rate varied with particle size, air exchange rate, outdoor NO3; 3) $PM_{2.5}$ infiltration was lowest for volatile component; 4) Outdoor volatile $PM_{2.5}$ components may be less representative of indoor exposure to volatile $PM_{2.5}$ of ambient origin; 5) Outdoor nonvolatile $PM_{2.5}$ components may be more representative of indoor exposure to nonvolatile $PM_{2.5}$ of ambient origin.

Sarnat et al. (2006, 090489)

Study Design Personal and ambient exposure assessment
Period June 14-August 18 (summer); Sep 24-Dec 15 (fall), 2000
Location Steubenville, OH
Population Non-smoking, older adults
Age Groups No
Personal Method Integrated filter gravimetric measurement
Personal Size $PM_{2.5}$
Microenvironment Size NR
Ambient Size $PM_{2.5}$
Component(s) SO_4; EC
Primary Findings 1) 24-h ambient measurements more representative of personal particle exposure than gases; 2) ventilation is an important exposure modifier.

Sarnat et al. (2005, 087531)

Study Design Time-series epidemiologic study
Period Summer 1999 and winter 2000
Location Boston, MA. Comparisons to a previous study in Baltimore are made.
Population School children and seniors
Age Groups NR
Indoor Source $PM_{2.5}$
Personal Method NR
Personal Size $PM_{2.5}$
Microenvironment Size
Ambient Size
Component(s) SO_4,
Copollutant(s) O_3, NO_2, SO_2
Primary Findings Substantial correlations between ambient $PM_{2.5}$ concentrations and corresponding personal exposures.; Summertime gaseous pollutant concentrations may be better surrogates of personal $PM_{2.5}$ exposures (especially personal exposures to $PM_{2.5}$ of ambient origin) than they are surrogates of personal exposures to the gases themselves.

Sax et al. (2006, 156950)

Study Design 2 Cohorts, one in NYC and 1 in LA.; Personal, home indoor, and home outdoor samples taken for $PM_{2.5}$, VOCs, and aldehydes.
Period 1999-2000, winter and summer in NYC, winter and fall in LA.
Location New York City, New York, and Los Angeles, California
Population 13-19 yr
Age Groups No
Indoor Source Customized backpack
Personal Method NR
Personal Size NR
Microenvironment Size NR
Ambient Size NR
Primary Finding(s) Most VOCs has median upper-bound lifetime cancer risks that exceeded the USEPA benchmark of 1×10^{-6} and were generally greater that the EPA modeled estimates, more so for compounds with predominant indoor sources. chromium, nickel, and arsenic had median personal cancer risks above the benchmark with exposures largely from outdoors and other microenvironments. The EPA model overestimate risks for beryllium and chromium and underestimate risks for nickel and arsenic.

Scheepers et al. (2003, 156955)

Study Design	Field Study
Period	Pilot Study; March 15-18, 1999, and March 22-25, 1999 (coal mine); April 12-14, 1999 (oil shale mine); Main Study; June 5-22, 2000
Location	Pilot Study; Ostrava, Czech Republic (black coal mine); Kohtla-Järve, Estonia (oil shale mine); Main Study; Kohtla-Järve, Estonia (oil shale mine)
Population	Coal miners with high exposures to Diesel-powered machinery
Age Groups	NR
Indoor Source	NR
Personal Method	Personal sampling was accomplished by each individual worker carrying personal air sampling equipment (GSA 200 or Gillian) during two shifts in the same work week (1 shift for the main study). The air sampling pumps operated in the breathing zones of the individual workers and operated at an electronically controlled flow rate of 2.0l/min.; Inhalable dust samples were collected using a sampler head developed by the Institute für Gefahrstoff Forschung der Bergbau Berufsgenossenschaft (IGF). Respirable dust was collected using an elutriator pre-separator type MPGII (IGF). Particles were collected on polystyrene membrane filters with a Teflon coating. All samples were taken at a height of ~1.5 m above the floor.
Personal Size	NR
Microenvironment Size	NR
Ambient Size	NR
Component(s)	1-nitropyrene (1-NP)
Primary Findings	This study confirms that 1-NP in black coal and oil shale mines is mostly associated with respirable particles and that mining operations involving diesel-powered engines exposures to DEP may be 3- to 10-fold higher for underground miners than workers on the surface. Furthermore, measurements of particle-associated 1-NP is a more sensitive and discriminating indicator o exposure to DEP than inhalable or respirable particles because of the relatively high concentrations of mine dust in mining operations.; Respirable dust concentration were 2- to 3-fold higher in the breathing zone than at fixed sampling locations while 1-NP concentrations were found to be 2.5-fold and 10-fold higher in the coal mine and oil shale mine respectively. This is thought to be due to location of fixed sampling points as well as wind and humidity levels within the mines themselves. For these reasons and others, personal air sampling is preferred over air sampling at fixed sites.

Shalat et al. (2007, 156971)

Study Design	Indoor home exposure assessment; sampling technology demonstration
Period	Winter heating season
Location	Residential home
Population	Children
Age Groups	Pre-toddler (6- to 12-month-old) children
Indoor Source	Mobile robotic and stationary. Filter and real-time nephelometer.
Personal Method	Floor; Filter inhalable particles (approximately < 100 µm)
Personal Size	Indoor home; Nephelometer total suspended particles.
Microenvironment Size	NR
Ambient Size	NR

Shao et.al. (2007, 156973)

Study Design	Exposure assessment
Period	July and Winter 2003
Location	Beijing, China
Population	General population
Age Groups	NR
Indoor Source	Soot aggregates, coal fly ash, minerals, unknown fine particles
Personal Method	PM_{10} selective inlet heads 30L/min flow rate with polycarbonate filters
Personal Size	PM_{10}
Microenvironment Size	PM_{10}
Ambient Size	PM_{10}
Component(s)	NR
Primary Findings	Plasmid scission assay, coupled with the image analysis, can be used to evaluate the relationship between particle physico-chemistry and toxicity.

Shilton et al. (2002, 049602)

Study Design	Respirable particulates inside and outside of a building were collected and compared
Period	24-h sampling from 12:45 pm Mondays to Fridays between 9/19/00 to 5/01/01
Location	Wolverhampton city center, University of Wolverhamptom, UK
Population	Building near traffic dominated by heavy-duty diesel vehicles
Indoor Source	Outdoor (primary); Mn,Al, NO3, Cl^- (wind-blown dust); Cu and Zn-(traffic emissions)
Personal Method	Active sampling using Casella sampler (filter)-
Personal Size	Respirable PM (inside and outside)
Microenvironment Size	Respirable PM (inside)

Ambient Size Respirable PM (outside)
Component(s) Respirable PM, metals (Zn, Cu, Mn, Al), sulfate, nitrate, and chloride
Primary Findings The indoor particulate concentration was driven by ambient concentration; meteorological-induced changes in ambient PM were detected indoors.

Simons et al. (2007, 156982)

Study Design NR
Period Baltimore, Maryland; and surrounding counties
Location Children with asthma
Population Inner city-6-12 yr; Surrounding counties 6-17 yr
Age Groups Gas stoves, cats, dogs, smokers, mold/mildew carpet, outside PM, dryer vents
Indoor Source Indoor air was collected from the child's bedroom with 4 L/min MSP impactors over a 72-h Period.
Personal Method NR
Personal Size $PM_{2.5}$; PM_{10}
Microenvironment Size $PM_{2.5}$; PM_{10}
Ambient Size Allergens were also assessed Dust mite; Bla/g; Mus/m; Fel/d; Can/ f; Airborne Mus/m
Primary Finding(s) Compared with the homes of suburban children with asthma, the homes of inner city Baltimore children with asthma had higher levels of airborne pollutants (including PM, NO_2 and O_3 amongst others) and home characteristics that predispose to greater asthma morbidity. In the inner city homes, median and GM PM_{10} levels were almost three times as high and the GM $PM_{2.5}$ levels were more than three times higher than in the suburban homes. Median GM NO_2 and GM O_3 levels were found in similar ratios. It is important to note that PM_{10} levels were found to be markedly higher in homes on arterial streets compared to those not on arterial streets. Although standards specific for home indoor air quality have not been established, the authors found that the inner city children were exposed to home pollutant levels in excess of the Environmental Protection Agency's National Ambient Air Quality standards.

Smith et al. (2006, 156990)

Study Design Location U.S.
Population Trucking industry
Age Groups working age
Indoor Source Diesel tractors, cigarette smoking, site pollution
Personal Method Terminal workers had samplers in a special harness; Drivers had a sampling box placed in the cab.
Personal Size $PM_{2.5}$
Microenvironment Size $PM_{2.5}$; Area samplers in the offices, freight dock, or shop.
Ambient Size $PM_{2.5}$; Samplers were located in the yard upwind of the terminal.
Component(s) Elemental carbon (EC); Organic carbon (OC)

Sørensen et al. (2005, 083053)

Study Design panel study
Period 11/1999, 8/2000
Location Copenhagen, Denmark
Population Healthy young adults, nonsmokers
Age Groups 20-33 yr, median age = 24yr
Indoor Source No
Personal Method International Gravity Bureau (BGI, Toulouse; France) (Kenny and Gussman 1997), a KTL; $PM_{2.5}$ cyclone (Jantunen et al. 1998), a; BGI400 pump (BGI Inc., Waltham, MA; USA) (flow 4 L/min)
Personal Size $PM_{2.5}$
Microenvironment Size NR
Ambient Size NR
Component(s) Transition metals (vanadium; chromium, iron, nickel, copper, and platinum)
Primary Findings The 8-oxodG concentration in; lymphocytes was significantly associated with vanadium and chromium concentrations with a 1.9% increase in; 8-oxodG per 1 Mg/L increase in vanadium and a 2.2% increase in 8-oxodG per 1 Mg/L increase chromium.

Sørensen et al. (2003, 042700)

Study Design Epidemiologic personal exposure study
Period Autumn- November 1999; Winter- January to February 2000; spring- April to May 2000; summer- August 2000
Location Central Copenhagen
Population University students
Age Groups 20-33 yr (median = 24 yr)
Indoor Source NR
Personal Method Particles were sampled using a KTL $PM_{2.5}$ cyclone developed for the European EXPOLIS study (17), a International Gravity Bureau 400 pump (4l/min), and a battery pack. The equipment was placed in a backpack, which the subjects carried or placed nearby when they were indoor. Sampling was done on 37-mm Teflon filters.; Urban background concentrations of $PM_{2.5}$ were measured on the rooftop of a building (20 meters above the ground) on the Copenhagen University campus.

Personal Size $PM_{2.5}$
Microenvironment Size NR
Ambient Size $PM_{2.5}$
Component(s) BS
Primary Findings Personal $PM_{2.5}$ exposure was found to be a predictor of 8-oxodG in lymphocyte DNA. No other associations between exposure markers and biomarkers could be distinguished. ETS was not a predictor of any biomarker in the present study. The current study suggests that exposure to $PM_{2.5}$ at modest levels can induce oxidative DNA damage and that the association to oxidative DNA damage was confined to the personal exposure, whereas the ambient background concentrations showed no significant association.; For most of the biomarkers and external exposure markers, significant differences between the seasons were found. Similarly, season was a significant predictor of SBs and PAH adducts, with average outdoor temperature as an additional significant predictor.

Sørensen et al. (2005, 089428)

Study Design Repeated measures cohort study.
Period Nov 1999-Aug 2000
Location Copenhagen, Denmark
Population residents of downtown Copenhagen
Age Groups 20-33 yr old, all non-smokers
Indoor Source Used a questionnaire to get time exposed to environmental tobacco smoke, burning candles, frying food, open windows
Personal Method wore a backpack, or placed nearby when indoors.
Personal Size $PM_{2.5}$ and BS
Microenvironment Size Bedroom and front door; $PM_{2.5}$ and BS
Ambient Size Street monitoring station and roof of a campus building; $PM_{2.5}$ and BS
Copollutant(s) NO_2
Primary Findings For NO_2 there was a significant association between personal exposure and the bedroom, the front door and the background, whereas for $PM_{2.5}$ and BS only the bedroom and the front door concentrations, and not the background, were significantly associated with personal exposure. The bedroom concentration was the strongest predictor of all three pollution measurements. The association between the bedroom and front door concentrations was significant for all three measurements, and the association between the front door and the background concentrations was significant for $PM_{2.5}$ and NO_2, but not for BS, indicating greater spatial variation tbr BS than for $PM_{2.5}$ and NO_2. For NO_2, the relationship between the personal exposure and the front door concentration was dependent upon the "season," with a stronger association in the warm season compared with the cold season, and for $PM_{2.5}$ and BS the same tendency was seen. Time exposed to burning candles was a significant predictor of personal $PM_{2.5}$, BS and NO_2 exposure, and time exposed to ETS only associated with personal $PM_{2.5}$ exposure. These findings imply that the personal exposure to $PM_{2.5}$, BS and NO_2 depends on many factors besides the outdoor levels, and that information on season or outdoor temperature and residence exposure, could improve the accuracy of the personal exposure estimation. Regression coefficients for personal exposure and front door $PM_{2.5}$ in warm season was 0.67 *, and in the cold season, 0.28. Front door BS in warm season was 0.86 *, and in the cold season, 0.45.* Front door NO_2 in warn season was 0.68 *, and cold season 0.32.*

Sram et al. (2007, 188457)

Study Design Exposure-Control study 53 policemen (exposed) and 52 age- and sex-matched healthy volunteers (control) were enrolled. Ambient and PE PM_{10}, $PM_{2.5}$, and c-PAHs were monitored and chromosomal aberrations were analyzed.
Period Feb 6-20, 2001
Location Prague, Czech Republic
Population Policemen working outdoors in Prague
Age Groups NA
Indoor Source Personal monitoring using personal samplers (name of instrument not stated)
Personal Method PM_{10} $PM_{2.5}$
Personal Size NR
Microenvironment Size PM_{10} $PM_{2.5}$
Ambient Size c-PAHs, B[a]P
Component(s) Ambient air exposure to c-PAHs increased fluorescent in situ hybridization (FISH) cytogenetic parameters in non-smoking policemen exposed to ambient PM

Srivasta et al (2007, 157004)

Study Design Exposure assessment of indoor environment
Period April 5-June 26, 2000
Location Laboratory in Delhi, India
Population Building occupants
Age Groups NR
Indoor Source Re-entrainment of existing dust on floor and other surfaces
Personal Method No personal exposure assessment was conducted
Personal Size NR
Microenvironment Size Suspended PM (SPM)
Ambient Size NR

Component(s)	metals Ca, Mg. Cu, Zn, Cd, Pb, Cr, Mn, Fe, Co, Ni
Primary Findings	Gravimetric analysis and atomic absorption spectrometry results indicated that the suspended PM (SPM) and metal (lead) concentrations were higher than the National Ambient Air Quality Standards for Delhi, India and SPM standards for residential and sensitive areas. The maximum concentrations of SPM were observed to be due to penetration of outdoor particles originating from wind-blown crustal dust and vehicular pollution. Scanning electron microscopy analysis of particles showed dominance of crystalline silicon and spherical soot particles in samples.

Stein et al. (2002, 157008)

Study Design	Exposure assessment, evaluation of an aerodynamic particle sizer to accurately measure size-distributed particle mass from number concentrations
Period	NR
Location	laboratory
Population	PM monitoring devices
Age Groups	NR
Indoor Source	No personal exposure assessment was conducted
Personal Method	NR
Personal Size	1.0-13 µm
Microenvironment Size	NR
Ambient Size	NR
Primary Finding(s)	Significant errors were observed in APS size-distribution measurements with measured mass median diameters (MMAD) as much as 17 times higher than from cascade impactors. Analysis of APS-correlated time of flight and light scattering data indicated that th

Strand et al. (2007, 157018)

Study Design	Cohort
Period	Winter of 1999-2000; winter of 2000-2001
Location	Denver, Colorado, USA
Population	Asthmatic Children
Indoor Source	No
Personal Method	Modeling/Extrapolation from fixed-site ambient monitoring (multiple methods)
Personal Size	No
Microenvironment Size	NR
Ambient Size	$PM_{2.5}$
Component(s)	NR
Primary Findings	Using modeled or extrapolated personal ambient PM exposure results in a deattenuation of decrements in FEV_1 associated with PM exposure, relative to use of fixed-site ambient monitoring PM levels. Associations between FEV_1 decrements and the various estimation procedures (modeling and extrapolation) were similar to each other.

Tang et al. (2007, 091269)

Study Design	Cohort Study
Period	12/2003-2/2005
Location	Sin-Chung City, Taiwan
Population	Asthmatic children
Age Groups	6-12 yr
Indoor Source	No
Personal Method	Portable particle monitor; DUSTcheck Portable Dust Monitor, model 1.108, GRIMM Labortechnik Ltd., Germany
Personal Size	PM_{10}, $PM_{2.5}$, PM1, $PM_{2.5}$-10, $PM_{1\text{-}2.5}$
Microenvironment Size	NR
Ambient Size	PM_{10}, $PM_{2.5}$, $PM_{2.5\text{-}10}$
Component(s)	NR
Primary Findings	Results of linear mixed-effect model analysis suggested that personal PM data was more suitable for the assessment of change in children's PEFR than ambient monitoring data.

Tatum et al. (2002, 157046)

Study Design	Methodological in this study, the performance of the gravimetric version of the RespiCon was examined in various forest products industry facilities. The precision of the RespiCon was assessed and its performance was compared with that of both a respirable cyclone and an inhalable dust sampler. In addition, some RespiCon samples were examined using scanning electron microscopy to determine physical particle size distribution.
Period	NA
Location	Various forest products industry facilities
Population	occupational
Age Groups	NA
Indoor Source	No
Personal Method	NR

Personal Size NR
Microenvironment Size Respirable (< PM_4 µm), Thoracic (< PM_{10} um), and Inhalable fractions (all PM) of airborne PM.
Ambient Size NR
Component(s) NR
Primary Findings The RespiCon is a useful sampling device for those situations in which it is important to simultaneously collect either personal or area samples of the respirable, thoracic, and inhalable fractions of airborne PM.

Thomaidis et al. (2003, 044193)

Study Design Exposure assessment (chemical characterization of $PM_{2.5}$ aerosols, source apportionment)
Period March 1995-March 1995
Location Two sites in Athens, Greece 1) Patisson in Athens city center and mainly affected by local traffic; 2) Rentis located in a semi-urban industrial area 5 km outside city center and mainly influenced by small industries
Population Urban Populations
Age Groups NR
Indoor Source NR
Personal Method No personal exposure sampling.
Personal Size NR
Microenvironment Size NR
Ambient Size $PM_{2.5}$
Component(s) Pb, Cd, Ni, As
Primary Findings Pb exhibited higher values during the winter, possibly due to increased diesel oil combustion from central heating and motor vehicles. No seasonal variation was observed for other metals. Annual mean levels of Pb at both sites were below the European Union guidelines.

Thornburg et al. (2004, 157052)

Study Design PM exposure studies RTP PM Panel Study; Tampa Asthmatic Children's Study
Period RTP summer 2000-spring 2001; Tampa October-November 2002
Location Research Triangle Park (RTP), NC and; Tampa, FL
Population Residential home occupants
Age Groups NR
Indoor Source Yes. Resuspension of PM_{10} from a carpet was identified as a major source in one home (a trailer), while cooking was identified as a source in many homes.
Personal Method 20 Lpm Harvard impactors and 2 Lpm Personal Exposure Monitors both with 37 mm Teflo filters and gravimetric analysis.; Also, MIE pdr1000 nepholometer.
Personal Size $PM_{2.5}$, PM_{10}
Microenvironment Size NR
Ambient Size $PM_{2.5}$, PM_{10}
Component(s) NR
Primary Findings The association of duty cycle with indoor-outdoor (I/O) ratio was confounded by the short time span of ventilation system operation and the presence of strong indoor sources.

Toivola et al. (2002, 026571)

Study Design Random sample of teachers
Period Nov 1998-Mar 1999 and Nov-Dec 1999
Location 2 cities in eastern Finland
Population Elementary school teachers
Personal Method Button inhalable aerosol sampler
Personal Size Particle Mass; BS
Microenvironment Size Particle Mass; BS
Ambient Size NR
Component(s) Total fungi, total bacteria, viable fungi, viable bacteria
Primary Findings Personal BS exposure correlated with both home and work BS exposures. BS concentrations explained best the variation of particle mass in personal and home concentrations.

Tovalin et al. (2006, 091322)

Study Design Biomarker (DNA damage in blood) exposure assessment
Period Mexico City and Puebla, Mexico
Location Occupationally exposed outdoor workers
Population 18-60 years old
Age Groups NA
Personal Method Personal integrated filter gravimetric measurement. Questionnaire
Personal Size $PM_{2.5}$
Microenvironment Size NR

Ambient Size NR
Component(s) NR
Primary Findings 1) In Mexico City, outdoor workers had greater DNA damage than indoor (median tail length 46.8 vs. 30.1 μm); 2) In Mexico City, outdoor workers had a greater proportion of cells with high DNA damage (tail length = 41 μm); 3) In Puebla, outdoor and indoor workers had similar damage; 4) DNA damage was correlated with $PM_{2.5}$ and ozone exposure; 5) High DNA damage in workers was associated with ozone, $PM_{2.5}$, and 1-ethyl-2-methyl benzene (VOC) exposure.

Tovalin-Ahumada et al. (2007, 190165)

Study Design Point study
Period April and May, 2002
Location Mexico City (Ne and SE) and Puebla, Mexico
Population Indoor and outdoor workers in large urban areas
Age Groups 18 years of age and older
Indoor Source NR, The exposures described in this report were monitored as part of a larger study directed at evaluating the association between personal exposures to $PM_{2.5}$ and VOCs and genetic damage in outdoor and indoor workers reported elsewhere (Tovalin et al., 2006).
Personal Method Personal exposures to $PM_{2.5}$ were monitored using 37mm Teflon filters (Model 225-9006, SKC Inc.), fitted to a single stage personal impactor (Model PEM-761-203A, SKC) and personal sampling pumps (Model PCXR4, SKC). Two $PM_{2.5}$ personal air samples (occupational and nonoccupational) were obtained during a 24-h period for each worker in Mexico City and for the indoor workers in Puebla. Only one $PM_{2.5}$ personal sample could be obtained during a 24-h period (an overall exposure) for the bus drivers in Puebla because of their work shift (from 4AM to 8 PM) with rotating start and end times. At the beginning of the work shift, each participant was asked to carry a backpack holding the pump; the impactor was attached to the backpack strap, in the breathing zone. At the end of the work shift, the impactor and pump were removed, and replaced with a new sampling setup that was worn by the worker until the beginning of the next day work shift.
Personal Size $PM_{2.5}$
Microenvironment Size NR
Ambient Size NR
Component(s) Si; S; K; Ca; Cl(e); Ti; V; Cr; Mn; Fe; Co; Ni; Cu; Zn; Mo(e); Cd(e); Se; Br; Rb; Sr(e); As; Pb
Primary Findings The results of this study suggest that outdoor workers in both Mexican cities experienced higher exposures to $PM_{2.5}$ than indoor workers, and that these exposures are well above either the 35 $\mu g/m^3$ US-EPA or the 65 $\mu g/m^3$ 24-h Mexican standards for $PM_{2.5}$. Both subgroups experienced higher occupational than non-occupational exposures. Mexico City outdoor workers had higher exposures to Soil, Fuels and Industrial emission-related elements than Puebla outdoor workers did. However, Mexico City outdoor workers had half the exposure to soil dust-related elements and fuel related elements than Puebla outdoor workers. However the S exposure was similar in all groups but high, product of the high vehicles density in the areas, responsible for 60% of the emission in Mexico City (Secretaria del Medio Ambiente, 2005). This study of Mexico City results correlates well with a previous $PM_{2.5}$ emissions inventory results, which determined that 81.14% of particles are released from mobile sources

Trenga et al. (2006, 155209)

Study Design Panel study with repeated measures
Period 3 sampling periods Oct 1999-Aug 2000, Oct 2000-May 2001, Oct 2001-Feb 2002
Location Seattle, Washington
Population Adults with and without COPD and children with asthma
Age Groups adults ages 56-89 and children ages 6-13
Indoor Source NR
Personal Method Carrying personal monitor (Harvard Personal Environmental Monitor for $PM_{2.5}$)
Personal Size $PM_{2.5}$
Microenvironment Size $PM_{2.5}$
Ambient Size Coarse (PM_{10}-$PM_{2.5}$) and $PM_{2.5}$ for residential outdoor, $PM_{2.5}$ for central site
Component(s) NR
Primary Findings FEV_1 decrements associated with 1-day lagged central site $PM_{2.5}$ in adult subjects with COPD. Associations between PM and lung function decrements were significant only in asthmatic children not receiving anti-inflammatory medication. Same day central s

Turpin et al. (2007, 157062)

Study Design RIOPA Study 24-h integrated indoor, outdoor, and personal samples collected in 3 cites.
Period Summer 1991-spring 2001
Location Elizabeth, NJ, Houston, TX, and Los Angeles County, CA
Population 309 adults and 118 children (89-18)
Indoor Source NR
Personal Method PEM on the front strap of a harness near the breathing zone. The bag on the hip contained the pump, battery pack, and motion sensor
Personal Size $PM_{2.5}$
Microenvironment Size $PM_{2.5}$, in the main living area (not kitchen)
Ambient Size $PM_{2.5}$, in the front or back yard
Component(s) 18 volatile organics, 17 carbonyl, $PM_{2.5}$ mass and >23 $PM_{2.5}$ species, organic carbon, elemental carbon, and PAHs

Primary Findings The best estimate of the mean contribution of outdoor to indoor $PM_{2.5}$ was 73% and the outdoor contribution to personal was 26%.

Urch et al. (2004, 055629)

Study Design The study was a crossover design in which each participant received a 2-h exposure to filtered air (FA) and CAP+O_3, assigned randomly and on separate occasions. Study objective is to examine the relationship between total and constituent $PM_{2.5}$ mass concentrations and acute vascular response.
Period 2000-2001; not explicitly stated
Location Downtown Toronto, Canada
Population 24 young (35 ± 10 yr) healthy, nonasthmatic, nonsmoking people
Age Groups 35 ± 10years
Indoor Source NR
Personal Method During exposures a sample was collected immediately upstream to the participant on a 47 mm Gelman Teflon filter with a 2 µm pore size at an airflow of 15 L/min.
Personal Size $PM_{2.5}$
Microenvironment Size NR
Ambient Size $PM_{2.5}$
Component(s) Total carbon (elemental carbon, organic carbon), NO3-, SO_4^{2-}. NH_4^+, K^+, Cl-, Ca, Fe, Al, Mg, Zn, Mn, Pb, Cu, Ba, Se, Cr, Ni, V, Ar, Cd all have median, min, and max reported for ambient levels. NO3-, SO_4^{2-}. NH_4^+ all have median, min, and max reported for directly measured personal exposure levels. Total carbon (elemental carbon, organic carbon), Ca, K^+, Fe, Cl^-, Al, Mg, Zn, Pb, Mn, Cu, Ba, Se, Cr, Ni, V, Ar, Cd all have median, min, and max reported for estimated personal exposure
Primary Findings A significant negative association between both the organic and elemental carbon concentrations and the difference in the post-exposure change in the BAD between CAP+O_3 and FA exposure days.

Vallejo et al. (2006, 157081)

Study Design 4/2002-8/2002
Period Mexico City, Mexico
Location Health young, non-smoking adults
Population Mean age 27 yr
Age Groups No
Personal Method pDR nephelometric method (personal DataRam, pDR1200)
Personal Size $PM_{2.5}$
Microenvironment Size NR
Ambient Size NR
Component(s) NR
Primary Findings Mean personal $PM_{2.5}$ level was 74 mg/m^3

Vallejo et al. (2006, 157081)

Study Design Pilot Study
Period April-August 2002
Location Mexico City, Mexico
Population Healthy residents of Mexico City
Age Groups 21-40 yr
Indoor Source NR
Personal Method The participant carried a personal $PM_{2.5}$ monitor (DataRAM 1200) during a single 13-h period starting at 9:00 a.m. Indoor situations included activities at home, at work, at school, or in public places such as theaters, stores, restaurants coffee shops, and subway transportation. Outdoor activities included walking, standing, or sitting in an open space, driving a car or using public transportation (bus or taxi).
Personal Size NR
Microenvironment Size $PM_{2.5}$
Ambient Size $PM_{2.5}$
Component(s) NR
Primary Findings The descriptive analysis showed that overall outdoor median concentration of $PM_{2.5}$ was higher than the indoor one. In the indoor microenvironment, the highest concentrations occurred in the subway followed by the school, and the lowest was at home. The outdoor microenvironment with the highest concentrations was the public transportation (bus), while the automobile had the lowest. It was found that $PM_{2.5}$ concentration levels had a circadian-like behavior probably related to an increase in the population daily activities during the morning hours, which decrease in the evening, especially at indoor microenvironments. The Center city area was found to have the highest concentrations of $PM_{2.5}$.; Multivariate analysis corroborated that $PM_{2.5}$ concentrations are mainly determined by geographical locations and hour of the day, but not by the type of microenvironment.

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van Roosbroeck et al. (2006, 090773)

Study Design Personal exposure assessment, effect of traffic-related pollutants
Period March-June 2003
Location Amsterdam, The Netherlands

Population	Schoolchildren
Age Groups	9-12 yr
Indoor Source	Environmental tobacco smoke, cooking
Personal Method	Integrated filter gravimetric measurement. Light absorbance.
Personal Size	$PM_{2.5}$ absorbance = "soot" ~ EC (see Notes)
Microenvironment Size	NR
Ambient Size	$PM_{2.5}$ absorbance = "soot" ~ EC (see Notes)
Component(s)	NR
Primary Findings	Children living near busy roads had 35% higher personal exposure to 'soot' than children living in urban background locations.

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van Roosbroeck et.al. (2006, 090773)

Study Design	Exposure assessment
Period	9 months (no year provided)
Location	Utrecht, The Netherlands
Population	School children
Age Groups	10-12 yr
Indoor Source	NR
Personal Method	$PM_{2.5}$ GK2.05 cyclones 4 L/min in a custom made backpack; NO_2 and NOX Ogawa passive samplers
Personal Size	$PM_{2.5}$
Microenvironment Size	NR
Ambient Size	$PM_{2.5}$
Copollutant(s)	NO_2
Primary Findings	Increased personal exposure to the traffic-related air pollutants soot and NOX was seen in children at the Freeway school. No increased personal exposure in any of the studied air pollutants was found for children at Ring School.

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van Roosbroeck et.al. (2006, 090773)

Study Design	Exposure assessment
Period	9 months (no year provided)
Location	Utrecht, The Netherlands
Population	School children
Age Groups	10-12 yr
Indoor Source	NR
Personal Method	$PM_{2.5}$ GK2.05 cyclones 4 L/min in a custom made backpack; NO_2 and NOX Ogawa passive samplers
Personal Size	$PM_{2.5}$
Microenvironment Size	NR
Ambient Size	$PM_{2.5}$
Copollutant(s)	NOX
Primary Findings	Increased personal exposure to the traffic-related air pollutants soot and NOX was seen in children at the Freeway school. No increased personal exposure in any of the studied air pollutants was found for children at Ring School.

Verma et al. (2003, 157093)

Study Design	Task-based exposure assessment of current occupational exposures to chemical agents of Ontario construction workers
Period	June 2000
Location	Ontario, Canada
Population	Ontario construction workers
Age Groups	NR
Indoor Source	known source construction activities
Personal Method	Air samples personal sampling pumps and collection media. Direct-reading particulate monitor DustTrak
Personal Size	respirable, inhalable, total, and silica dust; man-made mineral fibers (MMMF)
Microenvironment Size	NR
Ambient Size	NR
Component(s)	NR
Primary Findings	Authors state "Ontario construction workers are exposed to potentially hazardous levels of chemical agents."

Vinzents et al. (2005, 087482)

Study Design	Panel Study
Period	3/2003-6/2003
Location	Copenhagen, Denmark
Population	Healthy young adults
Age Groups	Mean age = 25 yr
Indoor Source	No
Personal Method	

Personal Size Ultra-fine particles (UFP) condensation particle counters; (TSI 3007; TSI, St. Paul, MN, USA)
Microenvironment Size UFP (10–100 nm)
Ambient Size PM_{10}
Primary Findings NR

Wallace (2005, 157102)

Study Design Exposure Assessment (Indoor, outdoor air monitoring for concentration of ultrafine particles. To determine indoor source of ultrafine particles-determine the contribution of vented gas clothes dryer)
Period Not specifically stated. An 18 month period including 2000.
Location NR
Population NR
Age Groups NR
Indoor Source Vented gas clothes dryer
Personal Method Scanning mobility particle sizer, differential mobility analyzer, condensation particle counter
Personal Size NR
Microenvironment Size Ultrafine (PM 0.01-0.45)
Ambient Size Ultrafine (PM 0.01-0.45)
Component(s) NR
Primary Findings Vented gas clothes dryer was determined to be a major source of indoor ultrafine particles. It consistently produced an order of magnitude increase in ultrafine particle concentration compared to times when there was no indoor source.

Wallace and Williams (2005, 057485)

Study Design Cohort
Period 2000-2001
Location Raleigh, North Carolina
Population African-American persons with elevated risk from exposure to particles.
Age Groups NR
Indoor Source NR
Personal Method PEM $PM_{2.5}$ monitor
Personal Size $PM_{2.5}$
Microenvironment Size Indoors $PM_{2.5}$
Ambient Size Outdoors near residence $PM_{2.5}$ $PM_{2.5}$
Component(s) Sulfur
Primary Findings Using outdoor particles to determine the effect on health is not accurate. The infiltration factor is a good estimator for personal exposure. Indoor and outdoor measurements of sulfur could be used in the absence of personal exposure measurement to estimate the contribution of outdoor fine particles to personal exposures.

Wallace et al. (2006, 088211)

Study Design Time series continuous monitoring of subjects with controlled hypertension or implanted defibrillators were monitored for 7 consecutive days in 4 seasons.
Period 2000-2001
Location North Carolina, probably near Research Triangle Park
Population Health-compromised adults, non-smokers
Age Groups Adults [range not specified]
Indoor Source Cooking, cleaning, personal care, smoking
Personal Method PEM
Personal Size $PM_{2.5}$
Microenvironment Size $PM_{2.5}$; Indoor and outdoor
Ambient Size NR
Component(s) NR
Primary Findings Use of continuous particle measuring instruments allowed more precise identification of sources, frequency and magnitude of short-term peaks, and more accurate calculation of individual personal clouds.

Wallace et al. (2003, 053553)

Study Design Inner City Air Pollution (ICAP) study- Randomized controlled trial
Period NR
Location Bronx, NY; Manhattan, NY; Boston, MA; Chicago, IL; Dallas, TX; Seattle, WA; Tucson, AZ
Population Asthmatic children and their residences
Age Groups 5-11 yr
Indoor Source Combustion-related particles smoking, cooking, use of a wood stove or fireplace, use of candles or incense, gas or kerosene space heaters or stoves.
Personal Method NR

Personal Size NR
Microenvironment Size 0.10 µg-5 µg; (see note below)
Ambient Size $PM_{2.5}$
Component(s) NR
Primary Findings Geometric mean values of indoor concentrations in the seven locations differed by less than a factor of 2, and the shape of the distributions was very similar across cities, both for the nominal 2-week averages and for hourly averages. The hourly averages exceeded 100 µg/m³ for at least 2% of all measurements in all cities, and exceeded 1,000 µg/m³ on at least a few occasions in each city. The most important particle source in these homes was smoking. A second, less powerful source was cooking, particularly frying/ sautéing or reporting a smoky cooking event. Use of incense also led to significant increases in particle concentrations. Dusting frequently also led to higher concentrations, possibly considerably higher than indicated by the pDR because of its lack of sensitivity for coarse particles. Infiltration of outdoor air added about half of the outdoor air concentration to the concentrations produced by the indoor sources, a result similar to that found by previous studies. Most of the observed variance in indoor concentrations was day to day, with roughly similar contributions to the variance from visit to visit and home to home within a city and only a small contribution made by variance among cities. The small variation among cities and the similarity across cities of the observed indoor air particle distributions suggest that sources of indoor concentrations do not vary considerably from one city to the next, and thus that simple models can predict indoor air concentrations in cities having only outdoor measurements. A new finding from this study was the observation that concentrations of fine particles peak in the late evening in homes with smoking, perhaps reflecting the influence of after dinner smoking.

Wang et al. (2006, 157108)

Study Design Exposure assessment, identification of sources of outdoor and indoor PM and trace elements
Period Aug 4 -Sep 10, 2004
Location Guangzhou, China
Population 4 hospitals
Age Groups NR
Indoor Source NR
Personal Method No personal exposure assessment was conducted.
Personal Size NR
Microenvironment Size PM_{10}, $PM_{2.5}$
Ambient Size PM_{10}, $PM_{2.5}$
Component(s) Trace elements Na, Al, Ca, Fe, Mg, Mn, Ti, K, V, Cr, Ni, Cu, Zn, Cd, Sn, Pb, As, Se
Primary Findings High correlation between $PM_{2.5}$ and PM_{10} suggest that they came from similar emission sources. Outdoor infiltration could lead to direct transportation of PM indoors. Human activities and ventilation types could also influence indoor PM. levels.

Ward et al. (2007, 157112)

Study Design Indoor air sampling to determine size fractionated concentrations of PM, OC, EC, and total carbon
Period Jan-Mar 2005
Location Libby, Montana
Population Children exposed to wood-burning stoves in elementary and middle schools
Indoor Source Burning wood in stoves for heating
Personal Method NR
Personal Size NR
Microenvironment Size PM >2.5, 1.0-2.5, 0.5-1.0, 0.25-0.5, and < 0.25 µm
Ambient Size PM >2.5, 1.0-2.5, 0.5-1.0, 0.25-0.5, and < 0.25 µm
Component(s) Organic carbon (OC) and elemental carbon (EC) in 5 size fractions >2.5, 1.0-2.5, 0.5-1.0, 0.25-0.5, and < 0.25 µm
Primary Findings Total measured PM mass concentrations were much higher inside the elementary schools, with particle size fraction (>2.5, 0.5-1.0, 0.25-0.5, and < 0.25 mm) concentrations between 2 and 5 times higher when compared to the middle school. The 1.0-2.5 mm fraction had the largest difference between the two sites, with elementary school concentrations nearly 10 times higher than the; middle school values. Weichenthal et al. (2006)
Study Design Cross-sectional survey comparing heating systems
Period Dec 2005-Mar 2006
Location Montreal, Quebec, Pembroke, Ontario, Canada
Population NR
Personal Method Yes, by questionnaire on age/size of home, cleaning frequency, type of stove and other cooking appliances, use of kitchen exhaust fan, number of smokers, burning candles, use of candles, portable heaters, natural gas clothes dryer.
Personal Size NR
Microenvironment Size NR
Ambient Size Ultrafine Particles < 100 nm in diameter, PM_4
Component(s) NR
Primary Findings Ultrafine Particles < 100 nm in diameter

Weisel et al. (2005, 157131)

Study Design Matched indoor, outdoor, and personal concentrations in proximity to pollution sources.
Period May 1999-Feb 2001

Location Elizabeth, NJ, Houston, TX, and Los Angeles County, CA
Population urban children and adults
Age Groups Child 6-19 yr; adult 17-89 yr
Indoor Source Age of house, recent renovations (< 1 yr), type of home (single, multiple family), attached garage, carpet indoors, local pollution sources.
Personal Method PEM on a harness with inlet near breathing zone.
Personal Size $PM_{2.5}$
Microenvironment Size $PM_{2.5}$
Ambient Size $PM_{2.5}$
Component(s) NR
Primary Findings Personal $PM_{2.5}$ was significantly higher than indoor and outdoor by one-way ANOVA and Sheffe test ($p < 0.001$).

Wichmann et al. (2005, 086240)

Study Design Exposure assessment; Ambient (indoor); Personal
Period November 29, 1993-March 30, 1994; October 17, 1994-December 22, 1994
Location Amsterdam, The Netherlands
Population Adults and schoolchildren living near high-traffic or low-traffic roads.
Age Groups Adults (50-70 yr); Schoolchildren (10-12 yr)
Indoor Source No
Personal Method Personal impactor
Personal Size Absorbance coefficient measurements of PM_{10} filter samples
Microenvironment Size Absorbance coefficient measurements of PM_{10} filter samples
Ambient Size Absorbance coefficient measurements
Component(s) NR
Primary Findings Found tentative support for using type of road as a proxy for indoor and personal exposure to traffic-related absorbance (PM).

Williams et al. (2003, 053338)

Study Design Cohort study, longitudinal
Period Summer 2000, fall 2000, winter 2001, and spring 2001
Location SE Raleigh, North Carolina; Chapel Hill, North Carolina
Population Elderly persons
Age Groups 50 yr
Indoor Source NR. While no smokers were enrolled into the study, 18 participants occasionally recorded passive exposures to environmental tobacco smoke. Since this study attempted to determine the effects of ambient PM upon personal and residential settings, and ETS exposures typically overwhelm ambient contributions, gravimetric values believed to have been heavily influenced by ETS were excluded from the analysis.
Personal Method A number of filter-based PM monitors widely used in other PM studies were employed here as described below in Table 1. A nylon vest, matched to the body size of the participant, was used to support and retain all of the personal monitoring equipment. All of the personal monitoring equipment was located in the participants breathing zone (chest area) with the exception of the nephelometer which was secured to the front pocket of the vest with the inlet fully exposed. Each participant was asked to wear the vest at all times with the exception of sleeping, bathing or the changing of clothes. In those instances, they were asked to secure the vest on nearby furniture or fixture. A local State of North Carolina AIRS monitoring platform in Raleigh, NC was selected to serve as the ambient monitoring site.
Personal Size $PM_{2.5}$
Microenvironment Size $PM_{2.5}$; PM_{10}; $PM_{10-2.5}$
Ambient Size $PM_{2.5}$; PM_{10}; $PM_{10-2.5}$
Component(s) NR
Primary Findings No statistical difference in personal $PM_{2.5}$ concentration exposures existed between the two cohorts. Neither seasonality nor community settings were determined to be critical factors in aggregate personal $PM_{2.5}$ exposures in the two subpopulations. $PM_{2.5}$, and to a lesser extent PM_{10}, mass concentrations were determined to be generally homogeneous across a large spatial area. The lack of a seasonal effect observed in the RTP was unexpected due to the historically higher $PM_{2.5}$ levels observed in central North Carolina during the spring and summertime when automotive traffic is highest and regional power plant demands for electricity are greatest (and subsequent release of emissions). $PM_{2.5}$ personal cloud estimates in the current study were in agreement with those observed in other PM studies involving susceptible subpopulations having more sedentary lifestyles. Mean personal $PM_{2.5}$ exposures in the current study had a moderate Pearson correlation relative to ambient or residential outdoor mass concentrations i

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Wilson and Zawar-Resa (2006, 088292)

Study Design Exposure assessment using Advanced Dispersion Modeling to estimate long-term personal PM exposures in small areas within a city
Period July 2003 and June 2004-2 winter months
Location Christchurch, New Zealand
Population urban environments
Age Groups NR
Indoor Source NR

Personal Method No personal exposure assessment was conducted
Personal Size NR
Microenvironment Size NR
Ambient Size PM_{10}
Component(s) NR
Primary Findings Despite the area's high intraurban PM concentration variability and meteorological and topographical complexity, the model performed satisfactorily overall, except for the Mount Pleasant site. The mean of observed measurements across all sites was close

Wilson and Brauer (2006, 088933)

Study Design Exposure assessment; Ambient (outdoor); Ambient (indoor infiltrating from outdoors); Non-ambient (indoor from indoor sources and "personal cloud')
Period April-September 1998
Location Vancouver, Canada
Population Subjects with physician-diagnosed COPD
Age Groups 54-86-years-old
Indoor Source No
Personal Method Personal integrated filter gravimetric measurement; TEOM outdoor ambient
Personal Size $PM_{2.5}$
Microenvironment Size NR
Ambient Size NR
Component(s) NR
Primary Findings 1) Measured ambient $PM_{2.5}$ exposure comprised 71% ambient $PM_{2.5}$ exposure was 71% of measured ambient concentration and 44% of measured total personal exposure; 2) Non-ambient exposure was independent of ambient exposure; 3) Pearson correlations of longitudinal estimated ambient exposure with ambient concentration averaged 0.88 (0.77-0.92).

Wu et al. (2006, 157156)

Study Design Panel study/exposure assessment
Period 9/3/2002-11/1/2002 (The fall agricultural burning season
Location Pullman, WA
Population Asthmatic adults
Age Groups (mean age = 27y/o, min = 18, max = 52 y/o)
Indoor Source No
Personal Method Yes, using two co-located Harvard; Personal Environmental Monitors (HPEM2.5; Harvard School of Public Health, Boston, MA), each connected to its own pump (BGI; AFC 400S, Waltham, MA) operated at 4 L/min
Personal Size $PM_{2.5}$
Microenvironment Size $PM_{2.5}$
Ambient Size $PM_{2.5}$
Component(s) Levoglucosan (LG); Elemental Carbon (EC); Organic Carbon (OC)

Wu et al. (2005, 086397)

Study Design Panel study with repeated measures
Period 1999-2000
Location Alpine, CA
Population Asthmatic children
Age Groups 9-17 yr
Indoor Source No
Personal Method pDR, continuously and 1-min concentrations (passive), in a fanny pack.
Personal Size $PM_{2.5}$
Microenvironment Size $PM_{2.5}$, Home inside & home outside
Ambient Size $PM_{2.5}$
Component(s) NR
Primary Findings Personal exposure was higher than those at fixed sites. Subjects received only 45.0% of their exposure indoors at, even though they spent more than 60% of their time there. In contrast, 29.2% of their exposure was received at school where they spent only 16.4% of their time. Thus, exposures in microenvironments with high PM levels where less time is spent can make significant contributions to the total exposure.

Wu et al. (2005, 157155)

Study Design Modeling of individual exposure using ambient data from a 10-yr longitudinal study.
Location Southern California Lancaster, San Dimas, Upland, Mira Loma, Riverside, Long Beach and Lake Elsinore.
Population Children
Age Groups NR
Personal Size No measurements presented in this study
Microenvironment Size NR

Yeh and Small (2002, 040077)

Study Design Comparative assessment of AME and IES models
Period 1997 (364 days) spring March-May, summer June-August, Fall September-November, winter December-February
Location Los Angeles County, CA
Population General population; ETS and Non ETS Homes
Age Groups NR
Indoor Source Indoor Cooking, ETS, Other sources and unexplained particulates that maybe generated with engaging in various activities
Personal Method NR
Personal Size PM_{10} $PM_{2.5}$
Microenvironment Size NR
Ambient Size PM_{10} $PM_{2.5}$
Component(s) NR
Primary Findings Adjusting from outdoor concentrations to personal exposures and correcting dose-response bias are nearly equal. Roughly the same premature mortalities associated with short-term exposure to both ambient $PM_{2.5}$ and PM_{10} are predicted by both models

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Personal Method NR
Personal Size PM_{10} $PM_{2.5}$
Microenvironment Size NR
Ambient Size PM_{10} $PM_{2.5}$
Component(s) NR
Primary Findings Adjusting from outdoor concentrations to personal exposures and correcting dose-response bias are nearly equal. Roughly the same premature mortalities associated with short-term exposure to both ambient $PM_{2.5}$ and PM_{10} are predicted by both models

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Period 1997 (364 days) spring March-May, summer June-August, Fall September-November, winter December-February
Location Los Angeles County, CA
Population General population; ETS and Non ETS Homes
Age Groups NR
Indoor Source Indoor Cooking, ETS, Other sources and unexplained particulates that maybe generated with engaging in various activities
Personal Method NR
Personal Size PM_{10}
Microenvironment Size NR
Ambient Size PM_{10}
Component(s) NR
Primary Findings Adjusting from outdoor concentrations to personal exposures and correcting dose-response bias are nearly equal. Roughly the same premature mortalities associated with short-term exposure to both ambient $PM_{2.5}$ and PM_{10} are predicted by both models

Yip et al. (2004, 157166)

Study Design A panel study with repeated measures with personal & home monitoring for 8 2-week Periods. Children were stratified into smoking and non-smoking households.
Period 2000-2001
Location Detroit, Michigan
Population School-age children with asthma
Age Groups 7-11 yr
Personal Method PEM in a backpack
Personal Size PM_{10}
Microenvironment Size PM_{10}; indoor at home & indoor at school
Ambient Size PM_{10}
Component(s) NR
Primary Findings Personal PM concentrations were significantly correlated with home environment (r = 0.38 to 0.70), with the strongest relationships in home with non-smokers.

Zhang et al. (2005, 157185)

Study Design Several co-located instruments were used to simultaneously sample air at the Pittsburgh EPA Supersite for 15 days

Period	7-22 Sep 2002
Location	Pittsburgh, Pennsylvania
Population	urban Population
Age Groups	NR
Indoor Source	NR
Personal Method	NR
Personal Size	NR
Microenvironment Size	NR
Ambient Size	Nonrefractory-PM1
Component(s)	Sulfate, Ammonium, Nitrate, Organics, Chloride
Primary Findings	Reasonably good agreement was observed on particle concentrations, composition, and size distributions between the AMS data and measurements from co-located instruments (given the difference between the $PM_1$and $PM_{2.5}$ size cuts), including TEOM, semicontinuous sulfate, 2-h- and 24-h-averaged organic carbon, SMPS, 4-h-averaged ammonium, and micro-orifice uniform deposit impactor.

Zhao et al. (2006, 156181)

Study Design	Aerosol source apportionment under four environments (personal, residential indoor, residential outdoor and ambient) to evaluate the relationship between different environments through exposure analysis, and to demonstrate the utility of the combined receptor model on air quality studies of various environments.
Period	June 2000 to May 2001
Location	Raleigh and Chapel Hill, NC
Population	NR. People with respiratory ailments most likely.
Age Groups	NR
Indoor Source	Yes (4 main sources to residential indoor PM Cu-factor mixed with indoor soil, secondary sulfate, Personal care and activity, ETS and its mixture)
Personal Method	Personal Exposure Monitors (PEM) and Harvard Impactor monitors (HI)
Personal Size	NR
Microenvironment Size	NR
Ambient Size	NR
Component(s)	OC, EC, and elements
Primary Findings	As per the authors “Secondary sulfate was the largest source for both residential outdoor and ambient PM. Cooking and personal care activity were two major internal sources for personal and residential indoor PM samples. In this study, secondary sulfate and motor-vehicle emission contributed significantly to the personal and residential indoor PM.

Zhao et al. (2007, 156182)

Study Design	Comprehensive analysis of the sources of $PM1_5$ exposure on children with moderate to severe asthma in urban-poor settings.
Period	**T**wo winter periods (October 2002-March 2003 and October 2003-March 2004)
Location	Elementary school for children with significant asthma, Denver, CO
Population	Schoolchildren in urban-poor settings suffering from moderate to severe asthma
Age Groups	6-13 yr (60% in the range 10-13 yr, rest in the range 6-9 yr)
Indoor Source	Yes, House cleaning compounds, and smoking were identified as primary internal sources.
Personal Method	Personal Exposure Monitor (PEM)
Personal Size	$PM_{2.5}$
Microenvironment Size	$PM_{2.5}$
Ambient Size	$PM_{2.5}$
Component(s)	EC, Cl, Si, NO3
Primary Findings	Four external sources and three internal sources were resolved in this study. Secondary nitrate and motor vehicle were two major outdoor $PM_{2.5}$ sources. Cooking was the largest contributor to the personal and indoor samples. Indoor environmental tobacco smoking also has an important impact on the composition of the personal exposure samples.

Zhu et al. (2005, 157191)

Study Design	4 apartments near the freeway were monitored at 2 times for 6 consecutive days, 24 h per day. Subjects did not enter the bedrooms where the samplers were, no cooking, cleaning, children, or pets.
Period	Oct. 2003-Dec. 2003 and Dec. 2003-Jan. 2004
Location	Los Angles, CA
Population	Urban Populations near major freeways.
Age Groups	NR
Indoor Source	NR
Personal Method	NR
Personal Size	Indoor and Outdoor ultrafine particles (6-220 nm)
Microenvironment Size	NR
Component(s)	CO
Primary Findings	The size distributions of indoor aerosols showed less variability than the adjacent outdoor aerosols. Indoor to outdoor ratios for ultrafine particle concentrations depended strongly on particle size. I/O ratios were dependent on the indoor ventilation mechanisms

applied. Size-dependent particle penetration factors and deposition rates were predicted from data by fitting a dynamic mass balance model.

Zöllner et al. (2007, 157192)

Study Design	PM exposure was investigated in and outside of schools
Period	Winter Period of 2005 and 2006
Location	Baden-Wuerttemberg, Germany
Population	School children
Age Groups	NR
Personal Method	No personal monitoring done. $PM_{2.5}$ was collected with filter device LVS 6.01 and analyzed gravimetrically; fifteen particle fractions (0.30 µm to >20 µm) were recorded with laser particle counters; >0.02 µm particles were recorded using condensation particle counters
Personal Size	NR
Microenvironment Size	They only reported concentrations for $PM_{2.5}$. PM 0.02 to >20 were collected and analyzed but only $PM_{2.5}$ concentration were reported.
Ambient Size	They only reported concentrations for $PM_{2.5}$. PM 0.02 to >20 were collected and analyzed but only $PM_{2.5}$ concentration were reported.
Primary Findings	1) The impaction of PM was strongly influenced by specific weather conditions; 2) Time resolution of measurements in classrooms showed variation in particle concentration depending on the type of building and indoor activities; 3) Concentrations of very small particles indoors and in ambient air measured by condensation particle counter were influenced by traffic emissions.

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Table A-53. Examples of studies showing developments with UFP sampling methods since the 2004 PM AQCD.

Reference	PM Size Ranges	PM Constituents	Instruments	Primary Findings
Biswas et al. (2005, 150694)			CPC (water)	Water-based CPC performance eval
Feldpausch et al. (2006, 155773)	20 – 100 nm	Carbonaceous aerosols	DS with CPC, compared with DMA	The DS with CPC compared fairly well with the DMA for particle sizes up to 40 nm with 20 – 40% underestimation depending on discharge frequency settings. The DS sampling period is 3 – 5 s in comparison with the 1 min scanning time of the DMA.
Hering et al. (2005, 155838)			CPC (water)	Water-based CPC performance eval
Hermann et al. (2007, 155840)	3 – 40 nm	Ag, NaCl	CPC (water and butanol)	Roughly 95% collection efficiency for d > 5 nm for TSI models 3776 and 3786, 95% efficiency for d > 20 nm for model 3775, near 90% efficiency for d > 20 nm for model 3785, near 90% efficiency for d > 25 nm for model 3772.
Kinsey et al. (2006, 130654)	10 nm – 5 µm	DE	TEOM, SMPS, CPC, DustTrak, E-BAM, ELPI, integrated filter samples	TEOM best comparison with gravimetric filter among mass concentration analyzers, ELPI and SMPS comparable for differential number distribution but ELPI not useful for gravimetric analysis because mass is not significant at small end of distribution.
Kulmala et al. (2007, 097838)			CPC	Changing temperature difference between saturator and condenser within CPC allowed for differences in cut-off diameters.
Kulmala et al. (2007, 155911)	2 – 20 nm	Atmospheric aerosol, Ag	Battery of CPCs (water, butanol, n-butanol)	Used the battery to discriminate between water-soluble, water-insoluble, butanol-soluble, and butanol-insoluble nucleation-mode particles
Ntziachristos and Samaras (2006, 116722)	7 nm – 1 µm	Automobile exhaust	5 instruments used simultaneously to reduce uncertainty: Teflon-coated filter downstream of constant volume sampling, ELPI with thermodenuder, CPC, SMPS, diffusion charger	Use of four reduced variables combining output from all instruments (ratio of particle number concentration from CPC and ELPI, estimated mean geometric mobility diameter from signal of diffusion charger and number concentration from CPC, ratio of signal of diffusion charger to constant volume sampler mass, ratio of constant volume sampler mass to volume collected by ELPI) resulted in identification of clear outliers and factors related to driving and fuel properties rather than measurement errors.
Olfert et al. (2008, 156004)	30 – 100 nm	NaCl, ambient	FIMS (compared with SMPS)	Particle number concentrations reported by the FIMS were 8 – 23% higher than the SMPS using an inversion technique designed to correct for particle residence time in the FIMS, which operates at 0.1 s resolution.

Reference	PM Size Ranges	PM Constituents	Instruments	Primary Findings
Petäjä et al. (2006, 156021)			CPC (water)	Water-based CPC performance eval
Winkler et al. (2008, 156160)	1.5 – 4 nm	Tungsten oxide	CPC (n-Propanol)	Authors remove excess charge on particles with ion trap to detect particles down to ~ 1 nm (by eliminating electrostatic attraction to agglomerate).

Table A-54. Summary of in-vehicle studies of exposure assessment.

Reference	Study Design	Mode of Transport	Exposures	Primary Findings
Rossner et al. (2008, 156927)	Measured $PM_{2.5}$ exposure of 50 city bus drivers and 50 controls in Prague, Czech Republic using personal samplers (type not specified) and VOCs using passive samplers. $PM_{2.5}$ filters analyzed for c-PAHs. Focus of study is oxidative stress biomarkers in drivers. Study period: winter 2005, summer 2006, winter 2006.	Bus	Units: ng/m³ winter 2005: Bus c-PAH **7.1 (3.7)** B[a]P **1.3 (0.7)** summer 2006: Bus c-PAH **1.8 (0.5)** B[a]P 0.2 (0.1) winter 2006: Bus c-PAH **5.4 (3.5)** B[a]P **1.0 (0.5)**	c-PAH and B[a]P exposure to bus drivers was significantly higher in Winter 2006, but control exposure was significantly higher in Winter 2005 for c-PAH and B[a]P and in summer 2006 for c-PAH. No significant difference in VOC exposure between bus drivers and controls was observed. Oxidative stress markers were significantly higher in bus drivers than controls for all seasons.
Fruin et al. (2008, 097183); Westerdahl et al. (2005, 086502) [Note: same data presented.]	On-road zero emissions vehicle driven on 33-mi arterial road and 75-mi freeway was equipped measured UFP (CPCs, SMPS, EAD), BC (aethalometer), NO_x (chemiluminescence), PM-bound PAHs (UV-photoionization), CO (Q-Trak). DVD analysis of traffic density and car speed. Study Period: Feb-Apr 2003 for 2- to 4-h periods.	Car	Arterial range of medians: UFP (1000p/cm³) 13-43 $PM_{2.5}$ (µg/m³) 7.9-45 BC (µg/m³) 0.74-3.3 Freeway range of medians: UFP (1000p/cm³) 47-190 $PM_{2.5}$ (µg/m³) 25-110 BC (µg/m³) 2.4-13	Measurements of freeway UFP, BC, PM-bound PAH, and NO concentrations were roughly one order of magnitude higher than ambient measurements. Multiple regression analysis suggests these concentrations were a function of truck density and total truck count. (Only PM measurements reported here).
Briggs et al. (2008, 156294)	UFP (P-Trak) and PM_{10}, $PM_{2.5}$, and PM1 (OSIRIS light scatter) were operated in a car while driving or walking on one of 48 routes in London. Trips ranged 1.5-15 min by car and were repeated up to 5 times to improve statistics. Study Period: Weekdays in May and June 2005.	Car Walking	Units: PM1 – PM_{10} (µg/m³), UFP (p cm-3) Avg Car Exposure: PM_{10} 5.87 (3.09) $PM_{2.5}$ 3.01 (1.10) PM1 1.82 (1.10) UFP 21639 (14379) Avg Walking Exposure: PM_{10} 27.56 (13.16) $PM_{2.5}$ 6.59 (3.12) PM1 3.37 (3.40) UFP 30334 (17245)	In-car concentrations of $PM_{2.5}$, PM1, and UFP correlated well with walking concentrations (R = 0.806, 0.800, 0.799 respectively). Avg walking concentrations were 1.4 – 4.7 times higher than avg in-car concentrations. Cumulative walking exposures (not shown here) were 4.4 – 15.2 times higher than those in cars, likely resulting from longer transit times for walking.

Reference	Study Design	Mode of Transport	Exposures		Primary Findings
Gomez-Perales et al. (2007, 138816)	$PM_{2.5}$ (personal filter pump), CO (T15 electrochemical cell), and benzene (canister) were measured on transit routes, and $PM_{2.5}$ filters were analyzed for mass, OC/EC, SO_4^{2-}, NO_3^-, and trace metals. Study period: 3-h morning and evening rush hour Jan – March 2003	Bus Minibus Metro	Units: $PM_{2.5}$ mass (µg/m^3), components (% of mass) Bus: $PM_{2.5}$ $(NH_4O_3$ $(NH_4)_2SO_4$ OC EC Crustal Non-crustal Unknown Minibus: $PM_{2.5}$ $(NH_4O_3$ $(NH_4)_2SO_4$ OC EC Crustal Non-crustal Unknown Metro: $PM_{2.5}$ $(NH_4O_3$ $(NH_4)_2SO_4$ OC EC Crustal Non-crustal Unknown	 20-58 5-8 10-18 17-39 8-20 15-18 2-3 6-24 25-55 4-13 7-22 22-37 9-19 12-13 3-3 4-26 24-41 5-8 10-21 35-42 9-13 10-16 2-4 5-20	Buses and minibuses had similar concentration levels for $PM_{2.5}$ mass, and metro exposures were lower. CO and benzene concentrations were higher on minibuses than buses. OC was the largest PM constituent for all modes of transport. Measured concentrations were higher in the morning than in the evening rush hour periods. Maximum historical wind speeds (1995-2003) appeared to be inversely associated with measured concentration.
Diapouli et al. (2007, 156397)	UFP (CPC) concentrations were measured at school, residential, and in-vehicle environments in Athens, Greece. Study Period: school hours, Nov 2003 – Feb 2004 and Oct – Dec 2004	"In-vehicle" (not specified)	15-min median (1000p/cm^3): School indoor School outdoor Residence indoor Residence outdoor In-vehicle	 13.6 16.6 11.2 24.0 78.0	In-vehicle UFP concentrations were roughly 3.5 – 7 times higher than school or residence concentrations. Indoor concentration diel patterns were also shown to follow outdoor levels, which suggests that indoor levels are of outdoor origin.
Gulliver and Briggs (2007, 155814)	TSP, PM_{10}, $PM_{2.5}$, and PM1 sampled (OSIRIS light-scatter devices) in a car while driving or walking on one of 48 routes in London. Trips ranged 1.5-15 min by car and were repeated up to 4 times to improve statistics. Study Period: Jan – Mar 2005.	Car Walk	Mean conc (µg/m^3): TSP-PM_{10} PM_{10}-2.5 $PM_{2.5}$ -1 PM1	 Walk 19.1 (19.8) 22.1 (22.8) 10.9 (10.4) 4.8 (3.4)	Walking exposures larger than car and background, and car exposures were generally larger than background except for PM1. Peak exposures during walking were significantly higher than peak in-car exposures.

Reference	Study Design	Mode of Transport	Exposures	Primary Findings
Sabin et al. (2005, 088300)	BC (aethalometer), particle-bound PAH (UV-photoionization), and NO (luminol reaction) were measured on 3 diesel school buses, 1 diesel school bus with a particle trap, and one compressed gas bus during before- and after-school commutes. Study Period: May – June 2002.	School bus (diesel, diesel with particle trap (TO), compressed gas (CNG))	In-bus mean concentration, Units: BC (μg/m^3), PAH (ng/m^3) Windows closed: BC BG 2.5 CNG 2.3 TO 7.1 diesel 11 Windows open: BC BG 1.9 CNG 1.5 TO 2.3 diesel 3.9	Mean concentrations on diesel buses without newer emissions control technologies were 2 – 4.4 times higher than background. On buses with particle traps, concentrations were 1.2 – 2.5 times higher than background, while concentrations on compressed gas-fueled school buses were actually lower than background.
Gulliver and Briggs (2004, 053238)	PM_{10}, $PM_{2.5}$, and PM1 sampled (OSIRIS light-scatter devices) in a car while driving or walking on northern corridor of Northhampton UK. Study Period: 1-h interval of morning and evening rush hour during Winter 1999 – 2000.	Car Walk	Walk PM_{10} 38.2 $PM_{2.5}$ 15.1 PM1 7.1	In-car PM_{10} concentrations were elevated compared with walking and background. $PM_{2.5}$ and PM1 concentrations were comparable for walking and background. Periods of elevated $PM_{2.5}$ compared with PM_{10} generally corresponded to times when SO_4^{2-} levels were also high.
Gomez-Perales et al. (2004, 054418)	$PM_{2.5}$ (personal filter pump), CO (T15 electrochemical cell), and benzene (canister) were measured on transit routes, and $PM_{2.5}$ filters were analyzed for mass, OC/EC, SO_4^{2-}, NO_3^-, and trace metals. Study period: 3-h morning and evening rush hour May – June 2002	Bus Minibus Metro	$PM_{2.5}$ (μg/m^3): Bus 68 Minibus 71 Metro 61	Generally, $PM_{2.5}$ concentration was higher in the morning than evening rush hour, but variability was higher for minibuses than other modes of transport. Wind speed was found to be associated with $PM_{2.5}$ concentration on minibuses.

Table A-55. Summary of personal PM exposure studies with no indoor source during 2002-2008.

Reference / Location	Personal	Micro	Ambient
SOUTH WEST			
Delfino et al. (2006, 090745) Riverside and Whittier, California	Method: PEM Riverside: n 13 24-h $PM_{2.5}$ 32.78 (21.84) 1-h max $PM_{2.5}$ 97.94 (70.29) 8-h max $PM_{2.5}$ 47.21 (30.0) Whittier: n 32 24-h $PM_{2.5}$ 36.2 (21.84) 1-h max $PM_{2.5}$ 93.63 (75.19) 8-h max $PM_{2.5}$ 51.75 (36.88)		Method: FRM Riverside: 24-h $PM_{2.5}$ (23.46) 36.63 24-h PM_{10} (29.36) 70.82 Whittier: 24-h $PM_{2.5}$ (12.14) 18.0 24-h PM_{10} (16.6) 35.73
Delfino et al. (2004, 056897) Alpine, California	Method: pDR Last 2-h $PM_{2.5}$ (33.7) 34.4 Diurnal $PM_{2.5}$ (31.6) 55.7 Nocturnal $PM_{2.5}$ (13.6) 22.3 1-h max $PM_{2.5}$ (120.3) 151.0 4-h max $PM_{2.5}$ (55.3) 87.5 8-h max $PM_{2.5}$ (39.0) 67.6 24-h $PM_{2.5}$ (19.9) 37.9	Method: HI Indoor 24-h PM_{10} 30. 3 (11.9) Indoor 24-h $PM_{2.5}$ 12. 1 (5.4) Outdoor 24-h PM_{10} 25. 9 (10.4) Outdoor 24-h $PM_{2.5}$ 11. 0 (5.4)	Method: TEOM Diurnal PM_{10} 3 5.1 (11.3) Nocturnal PM_{10} 2 3.3 (8.4) 1-h max PM_{10} 5 4.4 (13.8) 4-h max PM_{10} 4 4.5 (12.4) 8-h max PM_{10} 3 9.8 (11.2) 24-h PM_{10} 2 3.6 (9.1) 24-h $PM_{2.5}$ 1 0.3 (5.6)
Wu et al. (2005, 157155) Alpine, CA	Method: pDR n 11 Avg of 24-h $PM_{2.5}$ 11.4 (7.8)	Method: pDR n 14 Avg of 24-h $PM_{2.5}$ 5.6 (2.9) Method: HI n 14 Avg of 24-h $PM_{2.5}$ 9.8 (2.5)	Method: pDR n 8 Avg of 24-h $PM_{2.5}$ (11.4) 14.0 Method: HI n 8 Avg of 24-h $PM_{2.5}$ (7.8) 14.3

Reference / Location	Personal	Micro	Ambient
Turpin et al. (2007, 157062) Los Angeles County, CA (and Elizabeth, NJ, Houston, TX)	Method: PEM Avg of 48-h $PM_{2.5}$ Child 40.2 Adult 29.2	Method: HI Avg of 48-h $PM_{2.5}$: 16.2	Method: HI Avg of 48-h $PM_{2.5}$: 19.2
NORT WEST			
Jansen et al. (2005, 082236) Seattle, Washington, USA	Method: PM Results	Method: HI Indoor home: PM_{10} 11.93 $PM_{2.5}$ 7.29 Outdoor home: PM_{10} 13.47 $PM_{2.5}$ 10.47	Method: HI PM_{10} 18.0 $PM_{2.5}$ 14.0
Mar et al. (2005, 087566) Seattle, WA USA	Method: HI $PM_{2.5}$: Healthy: 9.3 (8.4) CVD: 10.8 (8.4) COPD: 10.5 (7.2)	Method: HI $PM_{2.5}$: Healthy: 7.4 (4.8) CVD: 9.5 (6.8) COPD: 8.5 (5.1) PM_{10}: Healthy: 12. 7 (7.8) CVD: 16. 2 (11.3) COPD: 14. 1 (6.6)	Method: HI $PM_{2.5}$: Healthy: 9.0 (4.6) CVD: 12. 7 (7.9) COPD: 9.2 (5.1) PM_{10}: Healthy: 14. 5 (7.0) CVD: 18. 0 (9.0) COPD: 14. 3 (6.8)
Wu et al. (2006, 157156) Pullman, WA	During non-burning times: 13.8 (11.1) During burning episodes: 19.0 (11.8)		

Reference / Location	Personal	Micro	Ambient
Trenga et al. (2006, 155209) Seattle, Washington	Method: PEM Median $PM_{2.5}$ Child 11.3 Adult 8.5	Method: HI Median $PM_{2.5}$ Child 7.5 Adult 7.6	Method: HI Residential Outdoor Median $PM_{2.5}$ Child 9.6 Adult 8.6 Residential Outdoor Median PMcoarse Child 4.7 Adult 5.0 Residential Outdoor Median $PM_{2.5}$ central site Child 11.2 Adult 10.3
Koenig et al. (2003, 156653) Seattle, WA	13.4 ± 3.2 $\mu g/m^3$	Inside homes = 11.1 ± 4.9	Outside homes = 13.3 ± 1.4 3 Central-sites = 10.1 ± 5.7
Liu S et al. (2003, 073841) Seattle, WA	Summary of PM concentrations ($\mu g/m^3$) between October 1999 and May 2001 by study group. Group Mean ± SD Personal $PM_{2.5}$ COPD 10.5 ± 7.2 Healthy 9.3 ± 8.4 Asthmatic 13.3 ± 8.2 CHD 10.8 ± 8.4	Summary of PM concentrations ($\mu g/m^3$) between October 1999 and May 2001 by study group. Group Mean ± SD Indoor $PM_{2.5}$ COPD 8.5 ± 5.1 Healthy 7.4 ± 4.8 Asthmatic 9.2 ± 6.0 CHD 9.5 ± 6.8 PM_{10} COPD 14.1 ± 6.6 Healthy 12.7 ± 7.8 Asthmatic 19.4 ± 11.1 CHD 16.2 ± 11.3	Summary of PM concentrations ($\mu g/m^3$) between October 1999 and May 2001 by study group. Location Pollutant Group Mean ± SD Outdoor $PM_{2.5}$ COPD 9.2 ± 5.1 Healthy 9.0 ± 4.6 Asthmatic 11.3 ± 6.4 CHD 12.7 ± 7.9 PM_{10} COPD 14.3 ± 6.8 Healthy 14.5 ± 7.0 Asthmatic 16.4 ± 7.4 CHD 18.0 ± 9.0
SOUTH CENTRAL			
Turpin et al. (2007, 157062) Houston (and Elizabeth, NJ, and Los Angeles County, CA)	Houston Child: 36.6 Adult: 37.2	Houston: 17.1	Houston: 14.7
MID-WEST			
Sarnat et al. (2006, 089784) Steubenville, OH	Mean (SD): $PM_{2.5}$ Summer n = 169 mean (SD) = 19.9 (9.4) Fall mean (SD) = 20.1 (11.6)		Mean (SD): $PM_{2.5}$ Summer n = 65 mean (SD) = 20.1 (9.3) Fall mean (SD) = 19.3 (12.2)

Reference / Location	Personal	Micro	Ambient
Adgate et al. (2002, 030676) Battle Creek, East St. Paul, and Phillips, Minnesota, constituting the Minneapolis-St. Paul metropolitan area.	Battle Creek All Seasons: 118, 22.7, (25.7), 16.2 (2.2) Spring: 41, 26.3 (25.7), 19.4 (2.1) summer: 31, 28.5 (36.1), 20.3 (2.1) Fall 46, 15.5 (13.4), 11.9 (2.1) E. St. Paul All Seasons: 107, 30.5 (38.7), 20.6 (2.3) Spring: 44, 33.9 (34.4), 23.9 (2.3) summer: 25, 20.5 (15.0), 17.2 (1.8) Fall: 38, 33.1(51.9), 19.5 (2.5) Phillips All Seasons: 107, 26.5 (24.3), 20.9 (2.0) Spring: 28, 37.5 (37.6), 30.0 (1.8) summer: 40, 22.7 (15.3), 19.2 (1.7) Fall: 39, 22.7 (16.7), 17.6 (2.1)	Battle Creek All Seasons: 108, 10.6 (6.6), 9.0 (1.8) Spring: 25, 12.7 (7.7), 11.0 (1.7) summer: 36, 8.9 (3.8), 8.1 (1.5) Fall: 47, 10.9 (7.4), 8.8 (2.0) E. St. Paul All Seasons: 97, 17.4 (20.3), 12.2 (2.2) Spring: 30, 20.7 (26.4), 13.6 (2.4) summer: 26, 15.8 (11.4), 13.7 (1.6) Fall 41 16.0 19.6 10.4 2.4 Phillips All Seasons: 89, 14.2 (13.0), 11.3 (1.9) Spring: 15, 16.9 (14.2), 13.0 (2.1) summer: 36, 13.2 (6.4), 11.4 (1.7) Fall: 38,14.4 (16.7), 10.6 (2.0)	Battle Creek All Seasons: 88 9.4 (6.2), 7.8 (1.8) Spring: 36, 10.5 (7.1), 8.5 (2.0) summer: 22, 8.7 (4.4), 7.8 (1.6) Fall: 30, 8.4 (6.2), 7.1 (1.7) E. St. Paul All Seasons: 95, 10.8 (6.6), 9.3 (1.8) Spring: 36, 12.0 (7.3), 10.1 (1.9) summer: 25, 8.5 (3.2), 7.8 (1.6) Fall: 34, 11.3 (7.5), 9.6 (1.8) Phillips All Seasons: 88, 10.0 (5.8), 8.7, (1.7) Spring: 30 (12.1), 7.2 (10.5) summer: 30, 8.6 (3.8), 7.8 (1.6) Fall: 28, 9.3 (5.5), 8.1 (1.7)
Crist et al. (2008, 156372) Ohio River Valley near Columbus	Athens (rural): 17.61 (17.81) Koebel (urban): 14.59 (13.05) New Albany (suburb): 13.93 (12.25)	Indoor Athens (rural): 17.20 (13.56) Koebel (urban): 14.98 (12.30) New Albany (suburb): 16.52 (13.53)	Outdoor Athens (rural): 13.66 (8.91) Koebel (urban): 13.89 (9.29) New Albany (suburb): 12.72 (8.86)
SOUTH EAST			
Wallace and Williams (2005, 057485) Raleigh, North Carolina	$PM_{2.5}$ pers = 23.0 (16.4) $PM_{2.5}$ pers/$PM_{2.5}$ out = 1.31 (0.99)	$PM_{2.5}$ in = 19.4 (16.5) $PM_{2.5}$ in/$PM_{2.5}$ out = 1.08 (1.05)	$PM_{2.5}$ out = 19.5 (8.6) 18.1 (8.1)
Williams et al. (2003, 053338) SE Raleigh, North Carolina Chapel Hill, North Carolina	Pooled PM mass concentrations ($\mu g/m^3$) across all subjects, residences, seasons, and cohorts Variable N Geo mean Mean RSD(a) Personal $PM_{2.5}$ (b) 712 19.2 23.0 70.1 (a) Relative standard deviation of the presented arithmetic mean. (beasured using PEMs.	Pooled PM mass concentrations ($\mu g/m^3$) across all subjects, residences, seasons, and cohorts Variable N Geo mean Mean RSD(a) Indoor $PM_{2.5}$ (c) 761 15.3 19.1 80.1 Outdoor $PM_{2.5}$ (c) 761 17.5 19.3 43.7 Indoor PM_{10}(b) 761 23.2 27.7 70.6 Outdoor PM_{10}(b) 761 27.5 30.4 46.4 Indoor PM_{10} 2.5(d) 761 6.3 8.6 111.8 Outdoor PM_{10} 2.5(d) 761 8.5 11.1 86.9 (a) Relative standard deviation of the presented arithmetic mean. (beasured using PEMs. (ceasured using HI samplers. (deasured by difference in PEM PM_{10} monitor and co-located HI $PM_{2.5}$ mass concentrations.	Pooled PM mass concentrations ($\mu g/m^3$) across all subjects, residences, seasons, and cohorts Variable N Geo mean Mean RSD(a) Ambient $PM_{2.5}$ (c) 746 17.3 19.2 44.9 Ambient PM_{10}(b) 752 27.9 31.4 51.5 Ambient $PM_{10\text{-}2.5}$(d) 210 8.6 10.0 62.3 (a) Relative standard deviation of the presented arithmetic mean. (beasured using PEMs. (ceasured using HI samplers. (deasured by difference in PEM PM_{10} monitor and co-located HI $PM_{2.5}$ mass concentrations.

Reference / Location	Personal	Micro	Ambient
NORTH EAST			
Koutrakis et al. (2005, 095800) Baltimore, MD Boston, MA	$PM_{2.5}$: (Baltimore, Boston) Winter: Seniors: 15.1 (14.6), 14.1 (6.0) Children: 24.0 (21.8), 18.5 (12.8) COPD: 16.4 (12.7), NR Summer: Seniors: 22.1 (10.1), 18.8 (9.7) Children: 18.6 (8.1), 30.3 (14.2) COPD: NR, NREC: (Baltimore, Boston) Winter: Seniors: NR, 1.4 (0.9) Children: 2.8 (1.8), 1.6 (1.6) COPD: 2.0 (1.2), NR Summer: Seniors: NR, NR Children: NR, NR COPD: NR, $NRSO_4$: (Baltimore, Boston) Winter: Seniors: 1.9 (1.1), 1.9 (1.2) Children: NR, 2.3 (1.7) COPD: 1.5 (0.8), NR Summer: Seniors: 5.7 (3.5), 2.9 (1.9) Children: NR, NR COPD: NR, NR		$PM_{2.5}$: (Baltimore, Boston) Winter: All: 20.1 (9.4), 11.6 (6.8) summer: Seniors: 25.2 (11.5), 12.7 (5.4) Children: 23.2 (14.0), 17.0 (11.5) COPD: NR, NREC: (Baltimore, Boston) Winter: All: 1.2 (0.6) summer: NR, $NRSO_4$: (Baltimore, Boston) Winter: All: 4.0 (1.7), 3.1 (1.8) summer: Seniors: 10.5 (7.1), 3.1 (1.8) Children: NR, 6.5 (6.0)
Turpin et al. (2007, 157062) Elizabeth, NJ, (and Houston, TX, and Los Angeles County, CA+	Elizabeth Child: 54.0 Adult: 44.8	Elizabeth: 20.1	Elizabeth: 20.4
Sarnat et al. (2005, 087531) Boston, Massachusetts. Comparisons to a previous study in Baltimore are made.	Winter-Children: $PM_{2.5}$: 17.4-25.8 SO_4: 1.6-3.3 Winter-Seniors: $PM_{2.5}$: 10.8-16.2 SO_4: 1.6-2.6 Summer-Children $PM_{2.5}$: 25.4-32.8 SO_4: 2.7-3.3 Summer-Seniors $PM_{2.5}$: 17.8-20.5 SO_4: 2.7-3.3	NR	Winter: $PM_{2.5}$: 6.5-15.5 SO_4: 1.7-4.2 Summer: $PM_{2.5}$: 11.9-21.4 SO_4: 3.6-9.0

Table A-56. Summary of PM species exposure studies.

Reference	Particle Sizes Measured	Component	Results	Primary Findings
Gadkari et al. (2007, 156459)	Personal: RPM Micro: NR Ambient: RPM	Fe, Ca, Mg, Na K, Cd, Hg, Ni, Cr, Zn, As, Pb, Mn and Li	Source contributions varied widely among 12 sites. Indoor: 0-95% Ambient: 0-26% Road: 0-94% Soil: 0-75%	Authors conclude that "(1) indoor activities and poor ventilation qualities are responsible for major portion of high level of indoor RPM, (2) majority of personal RPM is greatly correlated with residential indoor RPM, (3) time–activity diary of individuals has much impact on relationship investigations of their personal RPM with their

Reference	Particle Sizes Measured	Component	Results	Primary Findings
				respective indoor and ambient-outdoor RPM levels as reported in earlier reports and (4) residential indoors, local road-traffic and soil-borne RPMs are the dominating routes of personal exposure compared to ambient outdoor RPM levels.
Koistinen et al. (2004, 156655)	Personal, Micro, and Ambient: $PM_{2.5}$	Black smoke, SO_4^{2-}, NO_{3-}, NH_4^+, Al, Ca, Cl, Cu, K, Mg, P, S, Si, Zn	% contribution to $PM_{2.5}$ Outdoor - Indoor - Work - Pers CoPM * 35 28 32 33 Secondary** 46 36 37 31 Soil 16 27 27 27 Detergents 0 6 2 6 Sea Salt 3 2 1 2 * CoPM is the difference between total mass and other identified components; i.e., primary combustion particles, nonvolatile primary and secondary organic particles, and particles from tire wear, water, etc. ** Secondary particles are the sum of sulfate, nitrate, and ammonium.4 factors were identified for each exposure type (residential indoor, residential outdoor, workplace indoor, and personal). The factors contained the elements Al, Ca, Cl, Cu, K, Mg, P, S, Si, Zn, and black smoke. (insert in cell to left after consolidating PM size)	Population exposure assessment of $PM_{2.5}$, based on outdoor fixed-site monitoring, overestimates exposures to outdoor sources like traffic and long-range transport and does not account for the contribution of significant indoor sources.
Turpin et al. (2007, 157062)	Personal: $PM_{2.5}$ Micro: $PM_{2.5}$, in the main living area (not kitchen) Ambient: $PM_{2.5}$, in the front or back yard	18 volatile organics, 17 carbonyl, $PM_{2.5}$ mass and >23 $PM_{2.5}$ species, organic carbon, elemental carbon, and PAHs	For Los Angeles Carbon ($\mu gC/m^3$) EC 1.4 OC 4.1 Elements (ng/m^3) Ag 0.5; Al 24.7; As 0.5; Ba 22.9; Br 5.3; Ca 80.9; Cd 0.4; Cl 62.0; Co ND; Cr 0.6; Cu 5.5; Fe 162.9; Ga 0.1; Ge 0.1; Hg 0.1; In 0.3; K 74.1; La 2.3; Mn 2.9; Mo 0.4; Ni 2.0; Pb 4.7; Pd 0.3; P 0.1; Rb 0.1; S 1022.9; Sb 2.1; Se 1.4; Si 128.9; Sn 7.9; Sr 1.8; Ti 10.4; V 5.3; Y 0.1; ; Zn 16.4; Zr 0.5	The best estimate of the mean contribution of outdoor to indoor $PM_{2.5}$ was 73% and the outdoor contribution to personal was 26%.
Delfino et al. (2006, 090745)	Personal: 24-h $PM_{2.5}$ 1-h max $PM_{2.5}$ 8-h max $PM_{2.5}$ Ambient: 24-h $PM_{2.5}$ 24-hPM_{10} (also 24-h NO_2, 8-h max O_3, 8-h max NO_2, 24-h NO_2, 8-h max CO)	24-h $PM_{2.5}$ EC 24-h $PM_{2.5}$ OC	Mean (SD), units: $\mu g/m^3$: Riverside 24-h $PM_{2.5}$ EC = 1.61 (0.78) 24-h $PM_{2.5}$ OC = 6.88 (1.86) Whittier 24-h $PM_{2.5}$ EC = 0.71 (0.43) 24-h $PM_{2.5}$ OC = 3.93 (1.49)	PM associations with airway inflammation in asthmatics may be missed using ambient particle mass. The strongest positive associations were between eNO and 2-day avg pollutant concentrations. Per IQR increases: 1.1 ppb FENO/24 $\mu g/m^3$ personal $PM_{2.5}$. 0.7 ppb FENO/0.6 $\mu g/m^3$ personal EC 1.6 ppb FENO / 17 ppb personal

Reference	Particle Sizes Measured	Component	Results	Primary Findings
				NO_2 Ambient $PM_{2.5}$ and personal and ambient EC were significant only when subjects were taking inhaled corticosteroids. Subjects taking both inhaled steroids and antileukotrienes had no significant associations. Distributed lag models showed personal $PM_{2.5}$ in the preceding 5 h was associated with FENO.
Salma et al. (2007, 113852)	Personal: PM_{10}-2.0 and PM2.0 Micro: NA Ambient: NR	30 elements (Na, Mg, Al, Si, P, S, Cl, K, Ca, Ti, V, Cr, Mn, Fe, Ni, Cu, Zn, Ga, Ge, As, Se, Br, Rb, Sr, Y, Zr, Nb, Mo, Ba, and Pb)	Units: ng/m^3: $PM_{10-2.0}$; $PM_{2.0}$; Mg 296 130; Al 531 93; Si 2.09 442; S 978 828; Cl 305 104; K 318 127; Ca 2.57 413; Ti 47 25; Cr 35 15; Mn 310 148; Fe 33.5 15.5; Ni 29 8; Cu 496 190; Zn 118 50; Br 13 DL; Ba 145 DL; Pb 47 21; PM 83.6 33.0	The concentrations observed in the Astoria underground station were clearly lower (by several orders of magnitude) than the corresponding workplace limits.
Lai et al. (2004, 056811)	Personal, Micro, and Ambient: $PM_{2.5}$	Ag Cr Mn Si Al Cu Na Sm As Fe Ni Sn Ba Ga P Sr Br Ge Pb Ti Ca Hg Rb Tl Cd I S Tm Cl K Sb V Co Mg Se Zn Zr	GM (GSD), units: ng/m^3 P RI RO WI I/O Al As Br Ca Cd Cl Cu Fe Ga K Mg Mn Na Ni	Both the indoor and outdoor environments have sources that elevated the indoor concentrations in a different extent, in turn led to higher personal exposures to various pollutants. Geometric mean (GM) of personal and home indoor levels of $PM_{2.5}$, 14 elements, total VOC (TVOC) and 8 individual compounds were over 20% higher than their GM outdoor levels. Those of NO_2, 5 aromatic VOCs, and 5 other elements were close to their GM outdoor levels. For $PM_{2.5}$ and TVOC, personal exposures and residential indoor levels (in GM) were about 2 times higher among the tobacco-smoke exposed group compared to the non-smoke exposed group, suggesting that smoking is an important determinant ofthese exposures. Determinants for CO were visualised by real-time monitoring, and the authors showed that the peak levels of personal exposure to CO were associated with smoking, cooking and transportation activities. Moderate to good correlations were only found between the personal exposures and residential indoor levels for both $PM_{2.5}$ ($r = 0.60$; $p < 0.001$) and NO_2 ($r = 0.47$; $p = 0.003$).

Reference	Particle Sizes Measured	Component	Results	Primary Findings
			P Pb S Se Si Sn Ti V Zn	
Adgate et al. (2007, 156196)	Personal, Micro, and Ambient: $PM_{2.5}$ - broken down into TE	Ag, Al, Ca, Cd, Co, Cr, Cs, Cu, Fe, K, La, Mg, Mn, Na, Ni, Pb, S, Sb, Sc, Ti, Tl, V, Zn	Median, units: ng/m^3: O I P S 334.4 272.1 351.6; Ca 232.285.0 174.1; Al 96.3 23.3 58.6; Na 33.1 20.6 31.9; Fe 12.6 43.1 78.6; Mg 10.9 16.3 27.5; K 3.2 38.4 47.5; Ti 3.0 0.8 1.4; Zn 2.7 6.5 9.6; Cu 2.4 1.5 4.9; Ni NA -0.1 1.8; Pb 1.5 2.4 3.2; Mn 0.6 1.5 2.3; Sb 0.08 0.21 0.30; Cd 0.05 0.12 0.14; V 0.05 0.12 0.16; La 0.02 0.05 0.11; Cs 0.00 0.00 0.00; Th 0.00 0.00 0.00; Sc 0.00 0.00 0.01; Ag 0.00 0.07 0.08; Co NA 0.02 0.07; Cr -0.09 1.2 2.6	The relationships among P, I, and O concentrations varied across TEs. Unadjusted mixed-model results demonstrated that O monitors are more likely to underestimate than overestimate exposure to many of the TEs that are suspected to play a role in the causation of air pollution related health effects. These data also support the conclusion that TE exposures are more likely to be underestimated in the lower income and centrally located PHI community than in the comparitively higher income BC K community. Within the limits of statistical power for this sample size, the adjusted models indicated clear seasonal and community related effects that should be incorporated in long-term exposure estimates for this population.
Ebelt et al. (2005, 056907)	Personal: $PM_{2.5}$ Micro: "ambient exposure": $PM_{2.5}$, PM_{10}, $PM_{2.5-10}$; "non-ambient exposure": $PM_{2.5}$ Ambient: $PM_{2.5}$, PM_{10}, $PM_{2.5-10}$	Ambient sulfate, ambient non-sulfate, personal sulfate, personal ambient non-sulfate	Mean (SD), units μg/m^3 Ambient sulfate: 2.0 (1.1), ambient non-sulfate: 9.3 (3.7), personal sulfate: 1.5 (0.9), personal ambient non-sulfate: 6.5 (3.0)	Ambient exposures and (to a lesser extent) ambient concentrations were associated with health outcomes; total and nonambient particle exposures were not.
Farmer et al. (2003, 089017)	Personal: PM_{10} Micro: NR Ambient: PM_{10} Extractable organic material (EOM)	Benzo[a]pyrene (B[a]P) Carcinogenic polycyclic aromatic hydrocarbons (cPAHs)	Units: ng/m^3: Exposed, controls: **Prague:** cPAHs = 12.04(11.10), 6.17 (3.48)	Personal exposure to B[a]P and to total carcinogenic PAHs in Prague was two fold higher in the exposed group compared to controls, in Kosice three fold higher, and in Sofia 2.5 fold

Reference	Particle Sizes Measured	Component	Results	Primary Findings
	B[a]P cPAHs		B[a]P = 1.79 (1.67), 0.84 (0.60) **Kosice:** cPAHs = 21.72 (3.12), 6.39 (1.56) B[a]P = 2.94 (1.44), 1.07 (0.66) **Sofia:** cPAHs = 93.84 (55.0) police, 94.74 (120.34) bus drivers, 41.65 (33.36) B[a]P = 4.31 (2.6) police, 5.4 (3.18) bus drivers, 1.96 (1.53)	higher.
Jansen et al. (2005, 082236)	Personal: PM_{10} Micro: PM_{10}, $PM_{2.5}$, fine particles (~ PM1) Ambient: PM_{10}, $PM_{2.5}$	BC, as an estimate of elemental carbon (EC)	Mean (IQ Range), units: $\mu g/m^3$: BC Indoor: 1.34 (1.12) Outdoor 2.01 (1.68) Personal 1.64 (2.05)	For 7 subjects with asthma, a 10 $\mu g/m^3$ increase in 24-h avg outdoor PM_{10} and $PM_{2.5}$ was associated with a 5.9 [95% CI, 2.9–8.9] and 4.2 ppb (95% CI, 1.3–7.1) increase in FENO, respectively. A 1 $\mu g/m^3$ increase in outdoor, indoor, and personal BC was associated with increases in FENO of 2.3 ppb (95% CI, 1.1–3.6), 4.0 ppb (95% CI,2.0–5.9), and 1.2 ppb (95% CI, 0.2–2.2), respectively. No significant association was found between PM or BC measures and changes in spirometry, blood pressure, pulse rate, or SaO2 in these subjects.
Sørensen et al. (2003, 157000)	Personal: $PM_{2.5}$ Micro: NR Ambient: $PM_{2.5}$	BS (black smoke)	Units: $10^{-6}/m$ n Median Q25-Q75 All 177 6.8 (5.0-13.2) Autumn 42 7.1 (6.5-17.2) Winter 46 8.2 (5.1-13.3) Spring 46 12.6 (5.4-10.4) Summer 47 8.1 (3.4-9.0)	Personal $PM_{2.5}$ exposure was found to be a predictor of 8-oxodG in lymphocyte DNA. No other associations between exposure markers and biomarkers could be distinguished. ETS was not a predictor of any biomarker in the present study. The current study suggests that exposure to $PM_{2.5}$ at modest levels can induce oxidative DNA damage and that the association to oxidative DNA damage was confined to the personal exposure, whereas the ambient background concentrations showed no significant association. For most of the biomarkers and external exposure markers, significant differences between the seasons were found. Similarly, season was a significant predictor of SBs and PAH adducts, with avg outdoor temperature as an additional significant predictor.
Molnár et al. (2005, 156772)	Personal: 2.5 Micro and Ambient: $PM_{10-2.5}$ and $PM_{2.5}$	BS (black smoke) S Cl K Ca	Median, unit = ng/m^3 Wood burners Ref 1-sided p-value BS 0.97 0.74 0.053	Statistically significant contributions of wood burning to personal exposure and indoor concentrations have been shown for K, Ca, and Zn. Increases of

Reference	Particle Sizes Measured	Component	Results	Primary Findings
		Mn Fe Cu Zn Br Rb Pb	S 880 650 0.500 Cl 200 160 0.036 K 240 140 0.024 Ca 76 43 0.033 Mn 4.8 3.5 0.250 Fe 64 49 0.139 Cu 8.9 2.4 0.016 Zn 38 22 0.033	66–80% were found for these elements, which seem to be good wood-smoke markers. In addition, Cl, Mn, Cu, Rb, Pb, and BS were found to be possible wood-smoke markers, though not always to a statistically significant degree for personal exposure and indoor concentrations. For some of these elements subgroups of wood burners had clearly higher levels which could not be explained by the information available. Sulphur, one of the more typical elements mentioned as a wood-smoke marker, showed no relation to wood smoke in this study due to the large variations in outdoor concentrations from LDT air pollution. This was also the case for $PM_{2.5}$ mass. Personal exposures and indoor levels correlated well among the subjects for all investigated species, and personal exposures were generally higher than indoor levels. The correlations between the outdoor and personal or ind
Johannesson et al. (2007, 156614)	Personal, Micro, and Ambient: $PM_{2.5}$, PM1	BS- Black Smoke	BS2.5 Mean SD Personal 0.65 0.47 Exclusively smokers 0.62 0.47 Residential indoor 0.56 0.47 Exclusively smokers 0.52 0.46 Residential outdoor 0.68 0.51 Exclusively smokers 0.71 0.54 Urban background 0.63 0.37 All measurements 0.68 0.40 PM1/BS1 Personal 0.55 0.20 Residential indoor 0.54 0.45 Exclusively smokers 0.49 0.43 Residential outdoor 0.66 0.51 Exclusively smokers 0.68	Personal exposure of $PM_{2.5}$ correlated well with indoor levels, and the associations with residential outdoor and urban background concentrations were also acceptable. Statistically significantly higher personal exposure compared with residential outdoor levels of $PM_{2.5}$ was found for nonsmokers. PM1 made up a considerable proportion (about 70–80%) of $PM_{2.5}$. For BS, significantly higher levels were found outdoors compared with indoors, and levels were higher outdoors during the fall than during spring. There were relatively low correlations between particle mass and BS. The urban background station provided a good estimate of the residential outdoor concentrations of both $PM_{2.5}$ and BS2.5 within the city. The air mass origin affected the outdoor levels of both $PM_{2.5}$ and BS2.5; however, no effect was seen on personal exposure or indoor levels.
Sram et al. (2007, 192084)	Personal: PM_{10}, $PM_{2.5}$ Micro: NR	c-PAHs, B[a]P	B[a]P: exposed 1.6 ng/m^3, control 0.8 ng/m^3; c-PAHs: exposed 9.7 ng/m^3, control 5.8	Ambient air exposure to c-PAHs increased fluorescent in situ hybridization (FISH) cytogenetic

Reference	Particle Sizes Measured	Component	Results	Primary Findings
	Ambient: PM_{10}, $PM_{2.5}$		ng/m^3	parameters in non-smoking policemen exposed to ambient PM
Na and Cocker (2005, 156790)	Personal: $PM_{2.5}$ Micro: NR Ambient: $PM_{2.5}$	EC (Elemental carbon) OC (Organic carbon)	Mean (SD), units = µg/m^3 Residential homes: EC 2.0 (NR) OC 14.8 (NR) High school (EC): Weekday samples 1.1 (0.9) Weekend samples 1.0 (0.5) High school (OC): Weekday samples 8.8 (4.7) Weekend samples 7.4 (2.4)	Indoor $PM_{2.5}$ was significant influenced by indoor OC sources. Indoor EC sources were predominantly of outdoor origin.
Geyh et al. (2005, 186949)	Personal: TD, PM_{10}, $PM_{2.5}$ Micro: NR Ambient: TD, PM_{10}, $PM_{2.5}$	EC OC VOC also assessed	Mean (SD), units = µg/m^3: Summary Statistics by Area Location October 2001: Albany and West EC 5.9 (NA) OC 36 (NA) Liberty and Greenwich EC 5.3 (59) OC 30 (56) Park Place and Greenwich EC 14.5 (5.4) OC 72 (26) Church and Dey EC 7.9 (3.3) OC 48 (15) April 2002: Liberty and West EC 4.2 (2.1) OC 26 (13) Barclay and Greenwich EC 4.0 (2.6) OC 18 (14) Church and Dey EC 4.5 (1.9) OC 27 (15) Middle of the Pile EC 6.7 (1.0) OC 40 (25)	During October, the median personal exposure to TD was 346 µg/m^3. The maximum area concentration 1742 µg/m^3, was found in the middle of the debris. The maximum TD concentration found at the perimeter was 392 µg/m^3 implying a strong concentration gradient from the middle of debris outward. $PM_{2.5}$ /PM_{10} ratios ranged from 23% to 100% suggesting significant fire activity during some of the sampled shifts. During April, the median personal exposure to TD was 144 µg/m^3, and the highest area concentration, 195 µg/m^3, was found at the perimeter. Although the overall concentrations on PM at the site were significantly lower in April, the relative contributions of fine particles to the PM_{10}, and EC and OC to the TD were similar. During both months, volatile organic compounds concentrations were low. Comparison of recorded EC and OC values from October 2001 and April 2002 with previous studies suggests that the primary source of exposure to EC for the WTC truck drivers was emissions from their own vehicles.
Zhao et al. (2007, 156182)	Personal, Micro, and Ambient: $PM_{2.5}$	EC, Cl, Si, NO_3	Units = µg/m^3: Personal: EC: 1.64 NO_3: 0.135, Si: 0.176, Cl: 0.116; Indoor: EC: 1.819 NO_3: 0.013, Si: 0.051, Cl: 0.024; Outdoor: EC: 1.876 NO_3: 0.292, Si: 0.115, Cl: 0.013	Four external sources and three internal sources were resolved in this study. Secondary nitrate and motor vehicle were two major outdoor $PM_{2.5}$ sources. Cooking was the largest contributor to the personal and indoor samples. Indoor environmental tobacco smoking also has an important impact on the composition of the personal exposure samples.
Meng et al. (2005, 081194)	Personal: $PM_{2.5}$ Micro: NA Ambient: NR	EC, OC, S, Si	Mean (SD), units = ng/m^3: Indoor: EC: 1165.9 (2081.0) OC: 7725.5 (9359.3) S: 902.3 (602.2) Si: 124.0 (79.0)	Use of central-site $PM_{2.5}$ as an exposure surrogate underestimates the bandwidth of the distribution of exposures to

Reference	Particle Sizes Measured	Component	Results	Primary Findings
			Outdoor: EC: 1144.1 (968.1) OC: 3777.7 (2520.1) S: 1232.3 (633.2) Si: 141.1 (171.3)	PM of ambient origin.
Smith et al. (2006, 156990)	Personal: $PM_{2.5}$ Micro: $PM_{2.5}$ Area samplers in the offices, freight dock, or shop. Ambient: $PM_{2.5}$ Samplers were located in the yard upwind of the terminal.	Elemental carbon (EC) Organic carbon (OC)	Work Area Office Dock Yard Shop Non-smokers on-site: Clerk Dock worker Mechanic Hostler Non-smokers off-site Pickup/deliver driver Long haul driver Smokers On-Site Clerk Dock worker Mechanic Hostler Smokers off-site Pickup & Delivery drivers Long haul drivers	
Koutrakis et al. (2005, 095800)	Personal: $PM_{2.5}$ Micro: NR Ambient: $PM_{2.5}$	Elemental Carbon (EC), SO_4^{2-}	Mean (SD) data are provided for Baltimore and Boston, units = $\mu g/m^3$: EC: (Baltimore, Boston) Winter: Seniors: NR, 1.4 (0.9) Children: 2.8 (1.8), 1.6 (1.6) COPD: 2.0 (1.2), NR SO_4: (Baltimore, Boston) Winter: Seniors: 1.9 (1.1), 1.9 (1.2) Children: NR, 2.3 (1.7) COPD: 1.5 (0.8), NR Summer: Seniors: 5.7 (3.5), 2.9 (1.9)	Ambient $PM_{2.5}$ and SO_4 are strong predictors of respective personal exposures. Ambient SO_4 is a strong predictor of personal exposure to $PM_{2.5}$. Because $PM_{2.5}$ has substantial indoor sources and SO_4 does not, the investigators concluded that personal exposure to SO_4 accurately reflects exposure to ambient $PM_{2.5}$ and therefore the ambient component of personal exposure to $PM_{2.5}$ as well.
Chillrud et al. (2004, 054799)	Personal: $PM_{2.5}$ Micro: $PM_{2.5}$ Home indoor and home outdoor Ambient: Urban fixed-site and upwind fixed site operated for three consecutive 48-h periods each week.	Elemental iron, manganese, and chromium are reported in this study out of 28 elements sampled.	Mean of duplicate samples: $PM_{2.5}$: 62 $\mu g/m^3$ Fe: 26 $\mu g/m^3$ Mn: 240 ng/m^3 Cr: 84 ng/m^3 Variability: 1-15%	Personal samples had significantly higher concentration of iron, manganese, and chromium than home indoor and ambient samples. The ratios of Fe (ng/ μg of $PM_{2.5}$) vs Mn (pg/ μg $PM_{2.5}$) showed personal samples to be twice the ratio for crustal material. Similarly for the Cr/Mn ratio. The ratios and strong correlations between pairs of elements suggested steel dust as the source. Time-activity data suggested subways as a source

Reference	Particle Sizes Measured	Component	Results	Primary Findings
				of the elevated personal metal levels.
Jansen et al. (2005, 082236)	Personal, Micro, and Ambient: $PM_{2.5}$	Estimated Elemental Carbon (Abs) Elemental composition of a subset of personal, indoor and outdoor samples	Mean (SD), units = $\mu g/m^3$: $PM_{2.5}$ Abs S Zn Fe K Ca Cu Si Cl	For most elements, personal and indoor concentrations were lower than and highly correlated with outdoor concentrations. The highest correlations (median r.0.9) were found for sulfur and particle absorbance (EC), which both represent fine mode particles from outdoor origin. Low correlations were observed for elements that represent the coarser part of the $PM_{2.5}$ particles (Ca, Cu, Si, Cl).
Molnar et al. (2006, 156773)	Personal: $PM_{2.5}$ and PM1 Micro and Ambient: NR	S Cl K Ca Ti V Mn Fe Ni Cu Zn Br Pb	Urban background $PM_{2.5}$ mean, median, range S 620 320 95-1900 Cl 97 54 25-460 K 55 50 32-130 Ca 21 17 6.6-6.2 Ti 2.1 1.9 1.3-3.8 V 3.4 2.4 1.0-13 Mn 1.6 1.4 0.67- 3.8 Fe 36 33 7.1-100 Ni 1.6 1.2 0.33- 5.7 Cu 2.1 1.4 0.33-11 Zn 14 11 2.8-38 Br 1.7 1.4 0.47-44.3 Pb 3.3 2.1 0.94-11 Personal $PM_{2.5}$ mean, median, range S - < 470 270-1400 Cl 270 170 60-920 K 140 96 39-690 Ca 110 80 27-670 Ti 11 9.5 3.7-27 V 4.7 4.0 2.7-9.4 Mn - - - Fe 68 69 23-150 Ni 4.2 2.6 0.89-46 Cu 10 6.6 1.1-81 Zn 21 16 6.6-70 Br 2.0 1.3 0.91-14 Pb 2.9 2.6 0.92-8.3 Personal PM1 S - < 470 240-1200 Cl - < 110 54-160 K 80 82 50-130 Ca 32 23 8.4-87 Ti 6.5 6.3 3.7-11 V - < 4.2 2.8-8.9 Mn - - - Fe 28 25 7.6-68 Ni 8.2 1.2 0.83-58 Cu 5.0 4.4 1.6-14 Zn 15 14 7.6-37	$PM_{2.5}$ personal exposures were significantly higher than both outdoor and urban background for the elements Cl, K, Ca, Ti, Fe, and Cu. Personal exposure was also higher tan indoor levels of Cl, Ca, Ti, Fe, and Br, but lower than outdoor Pb./ Residential outdoor levels were significantly higher than the corresponding indoor levels for Br and Pb, but lower for Ti and Cu. The residential levels were also significantly higher than the urban background for most elements.

Reference	Particle Sizes Measured	Component	Results	Primary Findings
			Br 1.6 1.5 0.83-4.4 Pb 3.6 2.8 1.1-11 Residential Outdoor $PM_{2.5}$ mean, median, range S 640 460 190-1800 Cl 6.3 140 57-840 K 200 78 32-200 Ca 82 28 4.6-85 Ti 34 5.2 3.3-21 V 6.3 3.9 2.1-14 Mn Fe 5.5 31 8.8-200 Ni 45 < 1.6 0.65-5.5 Cu 2.6 1.3 0.65-17 Zn 22 15 5.5-85 Br 2.0 >450 0.91-51 Pb 4.6 2.6 0.90-20 Residential Outdoor PM1 S - 1.3 24-2000 Cl - < 110 44-170 K 76 68 34-170 Ca - < 12 5.1-78 Ti - < 5.0 2.2-9.5 V 5.6 4.47 2.2-14 Mn Fe 23 14 3.7-140 Ni 3.3 1.4 0.73-28 Cu - < 1.1 0.73-12 Zn 15 14 5.2-30 Br 1.5 1.4 0.78-4.3 Pb 4.1 1.5 1.0-17	
Kulkarni and Patil (2003, 156664)	Personal: PM5 Micro: NR Ambient: PM5	Lead Nickel Cadmium Copper Chromium Potassium Iron Manganese	Personal samples: Mean ± SD Type Lead Occupational 4.384 ± 7.766 $\mu g/m^3$ Residential 4.093 ± 5.925 $\mu g/m^3$ 24-h integrated 4.205 ± 1.523 $\mu g/m^3$ Cadmium Occupational 0.201 ± 0.158 $\mu g/m^3$ Residential 0.111 ± 0.165 $\mu g/m^3$ 24-h integrated 0.134 ± 0.140 $\mu g/m^3$ Manganese Occupational 1.979 ± 7.842 $\mu g/m^3$ Residential 0.180 ± 0.261 $\mu g/m^3$ 24-h integrated 1.983 ± 6.824 $\mu g/m^3$ Potassium Occupational 3.473 ± 4.691 $\mu g/m^3$	All listed metals were detected in the ambient air where as only Lead, Cadmium, Manganese, and Potassium were detected in personal exposures. Mean daily exposure to lead exceeds the Indian NAAQS by a factor of 4.2. However, ambient concentration of lead conforms to this standard. There is a rising trend in the personal exposures and ambient levels of cadmium. However, they are low and do not pose any major health risk as yet. Personal exposures to toxic metals exceed the corresponding ambient levels by a large factor ranging from 6.1 to 13.2. Thus, ambient concentrations may underestimate health risk due to personal exposure of toxic metals. Outdoor exposure to toxic metals is greater than the indoor (ratios ranging from 2.3 to 1.1) except for potassium (ratio 0.77). However, there is no significant correlation between these two.

Reference	Particle Sizes Measured	Component	Results	Primary Findings
			Residential 4.589 ± 4.619 $\mu g/m^3$ 24-h integrated Check	
Wu et al. (2006, 179950)	Personal: $PM_{2.5}$ Micro: $PM_{2.5}$ Ambient: $PM_{2.5}$	levoglucosan (LG) Elemental Carbon (EC) Organic Carbon (OC)	Mean personal exposure: LG: 0.018 (0.024) EC: 0.4 (0.5) OC: 8.5 (2.7). Ambient: check component During non-burning times: 0.026 (0.030) During burning episodes: 0.010 (0.012)	Authors "found a significant between-subject variation between episodes and non-episodes in both the Exposure during agricultural burning estimates and subjects' activity patterns. This suggests that the LG measurements at the central site may not always represent individual exposures to agricultural burning smoke "Evidence of "Hawthorne Effect": During declared episodes (i.e. real and sham), subjects spent less time indoors at home and more time in transit or indoors away from home than during non-declared episode periods. The differences remained even when limited to weekdays only.
Larson et al. (2004, 098145)	Personal: $PM_{2.5}$ Micro: $PM_{2.5}$ outside subject's residence, and inside residence Ambient: $PM_{2.5}$ at Central outdoor site (downtown Seattle)	Light absorbing carbon (LAC) and trace elements	Personal RI RO Central Mass 10,500 10,250 12,693 11,970 Al 32 19 21 31 As 1 1 2 2 LAC * 1439 01105 1830 1741 Br 3 2 3 3 Ca 72 46 36 50 Cl 248 173 75 78 Cr 2 2 1 2 Cu 3 4 2 3 Fe 63 35 61 95 K 57 54 78 67 Mn 2 2 3 6 Ni	Five sources of $PM_{2.5}$ identified: vegetative burning, mobile emissions, secondary sulfate, a source rich in chlorine, and crustal-derived material. The burning of vegetation (in homes) contributed more $PM_{2.5}$ mass on avg than any other sources in all microenvironments.
Brunekreef et al. (2005, 090486)	Personal, Micro & Ambient: $PM_{2.5}$	Nitrate	Mean (SD), units = ng/m^3: **Amsterdam:** Personal 1389(1965) Indoor 1348(1843) outdoor 4063(4435) **Helsinki:** Personal 161(202) Indoor 267(215) Outdoor 1276(1181)	In both cities personal and indoor $PM_{2.5}$ were lower than highly correlated with outdoor concentrations. For most elements, personal and indoor concentrations were also highly correlated with outdoor concentrations.
Sorenson et al. (2005, 089428)	Personal: $PM_{2.5}$ & Black smoke (BS) Micro: $PM_{2.5}$ & Black smoke (BS) Ambient: Street monitoring station and roof of a campus building $PM_{2.5}$ & Black smoke (BS)	Black Smoke (also NO_2)	Mean, IQR, Units = $\mu g/m^3$: Personal: Cold Season: 10.2 (5.6-14.8) Warm Season: 7.1 (5.5-11.4) Micro: Cold Season Home Indoor: 6.2 (5.5-11.4) Home front door: 10.8 (7.4-16.3) Warm Season Home Indoor: 6.1 (3.7-7.6) Home front door: 8.8 (5.6-11.54) Ambient: Cold Season: Street Station:	Indoor sources of PM and BS (as well as NO_2) were shown to be greatly influenced by indoor sources.

Reference	Particle Sizes Measured	Component	Results	Primary Findings
			31.6 (27.5-34.0) Urban Background: 7.7 (5.9-11.0) Warm Season: Street Station: 30.6 (24.7-36.0) Urban Background: 6.8 (4.6-8.6)	
Ho et al. (2004, 056804)	Personal: $PM_{2.5}$ Micro: NR Ambient: $PM_{2.5}$	OC EC OM TCA	Mean, unit = $\mu g/m^3$ Indoors: OM = 18.1; TCA = 22.9 Outdoors: OM = 20.1; TCA = 26.5	The major source of indoor EC, OC, and $PM_{2.5}$ appears to be penetration of outdoor air, with a much greater attenuation in mechanically ventilated buildings.
Maitre et al. (2002, 156726)	Personal: PM4 Micro: NR Ambient: PM4	PAH, benxene-toluene-xylenes (BTX), aldehydes, BaP PAHc, formaldehyde, acetaldehyde	Median Resp $\mu g/m^3$ BaP ng/m^3 PAHc ng/m^3 PAH ng/m^3 Benzene $\mu g/m^3$ Toluene $\mu g/m^3$ Xylene $\mu g/m^3$ BTX $\mu g/m^3$ Formaldehyde $\mu g/m^3$ Acetaldehyde $\mu g/m^3$ Aldehyde $\mu g/m^3$	The occupational exposure of policemen does not exceed any currently applicable occupational or medical exposure limits. Individual particulate levels should preferably be monitored in Grenoble in winter to avoid underestimations.
Farmer et al. (2003, 089017)	Personal: PM_{10} Micro: NR Ambient: PM_{10} $PM_{2.5}$ (not reported)	PM_{10} EOM EOM2 B[a]P c-PAHsb	Prague-SM EOM ($\mu g/m^3$) EOM2 (%) B[a]P ($\mu g/m^3$) c-PAHsb ($\mu g/m^3$) Prague-LB EOM ($\mu g/m^3$) EOM2 (%) B[a]P ($\mu g/m^3$) c-PAHsb ($\mu g/m^3$) Košice EOM ($\mu g/m^3$) EOM2 (%) B[a]P ($\mu g/m^3$) c-PAHsb ($\mu g/m^3$) Sofia EOM ($\mu g/m^3$) EOM2 (%) B[a]P ($\mu g/m^3$) c-PAHsb ($\mu g/m^3$)	Extractable organic matter (EOM) per PM_{10} was at least 2-fold higher in winter than in summer, and c-PAHs over 10-fold higher in winter than in summer. Personal exposure to B[a]P and to total c-PAHs in Prague ca. was 2-fold higher in the exposed group compared to the control group, in Košice ca. 3-fold higher, and in Sofia ca. 2.5-fold higher.
Hanninen et al. (2004, 056812)	Personal: $PM_{2.5}$ Micro: NR Ambient: $PM_{2.5}$	$PM_{2.5}$ -bound sulphur	Athens Basel Helsinki	Associated with indoor concentration: wooden building material, city, building age, floor of residence (i.e. ground, 1st, etc.), and use of stove other than electric.

Reference	Particle Sizes Measured	Component	Results	Primary Findings
			Prague	
Shilton et al. (2002, 049602)	Personal, Micro, and Ambient: Respirable PM	Respirable PM, metals (Zn, Cu, Mn, Al), sulphate, nitrate, and chloride	Indoor Outdoor Zn (ng/m^3) 241.1 179.5 Cu (ng/m^3) 43.3 24.99 Mn (ng/m^3) 15.6 4.18 Al (ng/m^3) 305.2 52.90 SO_4 (ng/m^3) 4.72 3.47 Cl (ng/m^3) 1.08 0.15 NO_3 (ng/m^3).35 1.08	The indoor particulate conc was driven by ambient conc; meteorological-induced changes in ambient PM were detected indoors;
Noulett et al. (2006, 155999)	Personal: $PM_{2.5}$ Micro: NR Ambient: $PM_{2.5}$	SO_4 ABS (light absorbing carbon)	Measurement Mean s.d. Ambient SO_4 2.72* 3.11 Ambient ABS 1.4** 1.0 Personal SO_4 1.33* 1.47 Personal ABS 1.0** 1.7 * Mean SO_4 values reported in $\mu g/m^3$ ** Mean ABS values reported in $10^{-5}/m{\cdot}1$	SO_4 and light absorbing carbon concentrations had higher personal-ambient correlations and less variability. This indicates that SO_4 and ABS were of outdoor origin, while $PM_{2.5}$ mass was of varied indoor and outdoor origin.
Sarnat et al. (2006, 089784)	Personal: $PM_{2.5}$ Micro: NR Ambient: $PM_{2.5}$	SO_4 EC	Mean (SD), units = $\mu g/m^3$: Personal Ambient SO_4 Summer 5.9 (4.2) 7.7 (4.8) Fall 4.4 (3.3) 6.2 (4.7) EC Summer 1.1 (0.6) 1.1 (0.5) Fall 1.2 (0.7) 1.1 (0.7)	High association between personal and ambient SO_4^{2-} and EC, especially for SO_4^{2-} for which there is no significant indoor source.
Sarnat et al. (2005 RMID 9171) (2005, 087531)	Personal: $PM_{2.5}$ Micro: n/a Ambient: $PM_{2.5}$	SO_4, O_3, NO_2, SO_2	Correlations between personal $PM_{2.5}$ and ambient gas O_3 correlated in summer. Spearman's R ◘ 0.4, anti-correlated in winter, R ◘ 0.3-0.1. NO_X somewhat correlated in summer. R ~ 0.3 Winter, R ~ 0.2-0.4 SO_2 not well correlated in summer or winter. R ~ 0-0.1. CO somewhat correlated in summer. R ◘0.1-0.3. Correlated in winter R ~ 0.2-0.3. No results were significant.	Substantial correlations between ambient $PM_{2.5}$ concentrations and corresponding personal exposures. Summertime gaseous pollutant concentrations may be better surrogates of personal $PM_{2.5}$ exposures (especially personal exposures to $PM_{2.5}$ of ambient origin) than they are surrogates of personal exposures to the gases themselves.
Brunekreef et al. (2005,	Personal, Micro, and Ambient:	SO_4^{2-}, NO_3-	Mean, units = $\mu g/m^3$:	In both cities personal and indoor

Reference	Particle Sizes Measured	Component	Results	Primary Findings
090486)	$PM_{2.5}$		SO_4^{2-}: Amsterdam Helsinki NO_3-: Amsterdam Helsinki	$PM_{2.5}$ were lower than highly correlated with outdoor concentrations. For most elements, personal and indoor concentrations were also highly correlated with outdoor concentrations.
Kim et al. (2005, 156640)	Personal: $PM_{2.5}$ Micro: NR Ambient: $PM_{2.5}$	Sulfate, Elemental carbon (EC), Calcium, Magnesium, Potassium, Sodium	Mean (SD), units = µg/m^3: SO_4^{2-}: 2.7 (3.2) Ca^{2+}: 0.12 (0.12) Mg^{2+}: 0.02 (0.01) K: 0.07 (0.08) Na^+: 0.09 (0.20) EC: 0.60 (0.54)	Traffic-related combustion, regional, and local crustal materials were found to contribute 19% ± 17%, 52% ± 22%, and 10% ± 7%, respectively. Among participants that spent considerable time indoors, exposure to outdoor $PM_{2.5}$ includes a greater relative contribution from combustion sources, compared with outdoor (ambient) $PM_{2.5}$ measurements.
Wallace and Williams (2005, 057485)	Personal: $PM_{2.5}$ Indoor Micro: $PM_{2.5}$ Outdoor Micro: $PM_{2.5}$	Sulfur	Mean (SD), units = ng/m^3: Personal: 1046 (633) Indoor: 1098 (652) Outdoor: 1951 (1137)	Generally, infiltration factor provides a reliable estimate of personal exposure. Sulfur can be used in lieu of personal exposure to PM because it is derived from outdoors.
Jacquemin et al. (2007, 192372)	Personal: $PM_{2.5}$ Micro: NA Ambient: $PM_{2.5}$	Sulfur	Mean, units = µg/m^3: Personal: 1.3 outdoor: 1.2	Authors suggest that "outdoor measurements of absorbance and sulphur can be used to estimate both the daily variation and levels of personal exposures also in Southern European countries, especially when exposure to ETS has been taken into account. For $PM_{2.5}$, indoor sources need to be carefully considered."

Table A-57. Summary of personal PM exposure source apportionment studies.

Reference	Study Design	Results	Primary Findings
Hopke et al. (2003, 095544)	Source apportionment of personal (PEM) and indoor central and apartment (VAPS) and outdoor (VAPS) $PM_{2.5}$, Baltimore retirement home with 10 elderly subjects, July-Aug 1998.	% contr External Secondary SO_4^{2-} Unknown Soil Internal Gypsum Activity Personal care	63% of personal exposure could be attributed to outdoor sources (with 46% from sulfate), and resuspension of indoor PM during vacuuming, cleaning, or other activities contributed 36% of personal exposure.
Larson et al. (2004, 098145)	Source apportionment of personal (PEM) and residences (HI) and central outdoor (HI) $PM_{2.5}$ around Seattle with 10 elderly subjects and 10 asthmatic children, Sep 2000 and May 2001. The purpose of the article was to compare PMF2 and PMF3 methods.	PMF2: % contr Veg burn Mobile Fuel oil S, Mn, Fe Secondary Cl-rich Crustal Crustal2 PMF3: % contr Veg burn Mobile Secondary Crustal	Results showed that vegetative burning was the largest contributor to personal exposure and that was related to outdoor combustion. Crustal exposures were related to indoor activities.
Zhao et al. (2006, 156181)	Source apportionment of personal (PEM) and residential indoor (HI) and residential outdoor (HI) and central outdoor (HI) $PM_{2.5}$, Raleigh and Chapel Hill NC with 38 subjects, summer 2000 and Spring 2001.	% contr Motor vehicle Soil Secondary SO_4^{2-} Secondary NO_3^- ETS Personal care and activity CU-factor mix w indoor soil Cooking Secondary sulfate was the largest ambient source and the largest ambient contribution to personal exposure. Cooking produced the largest contribution to personal and indoor concentrations. Note that sums over 100% because multiple sources obscured PMF resolution	

Reference	Study Design	Results	Primary Findings
Meng et al. (2007, 091197)	Source apportioned infiltration for personal (PEM) and residential indoor (HI) and residential outdoor (HI) and central outdoor (HI) $PM_{2.5}$, Los Angeles, Houston, and Elizabeth, NJ with 100 non-smoking residences and residents in each city, in each season between summer 1999 and spring 2001 (RIOPA).	% contr (Outdoor Origin) Mechanically generated Primary Combustion Secondary Formation* *excludes nitrates	Differential infiltration of the $PM_{2.5}$ resulted in a reduction of secondary formation products relative to outdoors.
Reff et al. (2007, 156045)	Functional group distinction for personal (PEM) and residential indoor (HI) and residential outdoor (HI) and central outdoor (HI) $PM_{2.5}$, Los Angeles, Houston, and Elizabeth, NJ with 100 non-smoking residences and residents in each city, in each season between summer 1999 and spring 2001 (RIOPA). $PM_{2.5}$ samples from 219 homes were used for this analysis.	SO_4^{2-}: R O I P C = O: R O I P CH: R O I P	The main finding was that indoor and personal levels of CH in organic carbons were found to be substantially higher than outdoors. This reduced the polarity of indoor and personal organic carbons
Zhao et al. (2007, 156182)	Source apportionment of personal (PEM) and indoor school (FRM) and outdoor school (FRM) $PM_{2.5}$, Denver with 56 asthmatic children, Oct 2002-March 2003 and Oct 2003-March 2004.	% contr Secondary SO_4^{2-} Soil Secondary NO_3^- Motor vehicle Cl-based cleaning Cooking ETS	The largest personal exposure was from cooking (54.8%), but motor vehicle emissions were the largest outdoor contributor (13.3%) to personal exposure. Secondary nitrate comprised the largest outdoor source but accounted for only 9.4% of personal exposure.
Strand et al. (2006, 089203)	Using positive matrix factorization and an extrapolation method to estimate $PM_{2.5}$ based on SO_4^{2-} and Fe components.	Estimation method, Mean (SD, range): PMF: 7.42 (1.93, 3.43 - 12.89) Extrapolation Method: Using sulphate: 6.38 (1.60, 3.20 - 10.97) Using sulphate & iron: 6.50 (1.36, 3.54 - 10.12) Using sulphate & iron, temperature adjusted: 7.02 (1.48, 3.79 - 11.02) Using sulphate (no gamma): 8.23 (2.06, 4.12 - 14.14)	Similar results were found with each technique.

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Table A-54. Summary of PM infiltration studies.

Reference	Study Design	F_{inf}	I/O
Allen et al. (2003, 053578)	**Objective:** Enhance knowledge of the outdoor contribution to total indoor and personal PM exposures. **Methods:** Continuous light scattering monitoring. **Subjects:** Elderly and children spending most of their time indoors. Healthy individuals, elderly with COPD or CHD and children with asthma. 44 residences measured for 55 10-day sessions. Seattle, WA.	$PM_{2.5}$ avg- 0.65 ± 0.21; Non-heating season- 0.79 ± 0.18; Heating season- 0.53 ± 0.16; Open windows (mean)- 0.69; Closed windows (mean)- 0.58; All days (mean)- 0.65	Light scattering (whole peak): 0.75 ± 0.25; Light scattering (uncensored data): 0.77 ± 0.24; Sulfur concentration (slope): 0.65 ± 0.01
Arhami et al. (2009, 190096)	**Objective:** To examine associations between size-segregated PM, their particle components, and gaseous co-pollutants. **Methods:** Data analyzed with linear mixed-effect models. **Subjects:** Four different retirement communities in San Gabriel Valley, CA and Riverside, CA. 2005-2007.	$PM_{2.5}$: 0.38-0.57 EC: 0.64-0.82 OC: 0.60-0.98	n/a
Balasubramanian et al. (2007, 156248)	**Objective:** PM monitoring and assessment based on analysis of chemical and physical characteristics or indoor and outdoor particles. **Methods:** Particle number and mass concentrations measured using real-time particle counter and low-volume particulate sampler. **Subjects:** 3 residential indoor and 1 residential outdoor environments in Choa Chu Kang, Singapore. May 12-May 23, 2004.	n/a	$PM_{2.5}$: 0.93-1.90 Chemical Species: Cl^-: 0.35-0.45, NO_2-: 2.50-4.13, NO_3^-: 1.41-5.41, SO_4^{2-}: 1.21-1.70, Na^+: 0.43-0.74, NH_4: 1.43-2.39, EC: 0.75-0.96, OC: 1.04-1.92, Al: 1.04-1.92, Co: 0.86-1.32, Cr: 1.35-2.90, Cu: 0.50-0.69, Fe: 0.30-0.42, Mn: 0.23-0.42, Pb: 0.40-2.47, Zn: 0.59-0.81, Cd: 0.74-1.75, Ni: 0.71-1.32, Ti: 0.73-0.78, V: 1.01-1.05
Barn et al. (2008, 156252)	**Objective:** Measure infiltration factor from $PM_{2.5}$ from forest fires and determine effectiveness of HEPA filter. **Methods:** pDR for ambient air sampling. **Subjects:** Homes affected by forest fire or residential wood smoke. British Columbia, Canada. 38 homes sampled (valid samples: 19 winter, 13 summer).	$PM_{2.5}$ (mean)- Summer: HEPA- 0.19 ± 0.20, Unfiltered- 0.61 ± 0.27 Winter: HEPA- 0.10 ± 0.08, Unfiltered- 0.28 ± 0.18 Both: HEPA- 0.13 ± 0.14, Unfiltered- 0.42 ± 0.27	Mean- Summer: HEPA- 0.43, Unfiltered- 0.77 Winter: HEPA- 0.21, Unfiltered- 0.36 Both: HEPA- 0.25, Unfiltered- 0.47

Reference	Study Design	F_{inf}	I/O
Baxter et al. (2007, 092726)	**Objective:** To develop predictive models of residential indoor air pollutant concentrations for lower SES, urban households. Part of ACCESS cohort study of asthma etiology. **Methods:** Regression analysis; mass balance model; F_{inf} from slope in univariate regression analyses. **Subjects:** Lower SES populations. 43 homes, 23 homes monitored in both seasons, 15 in the non-heating season (May-Oct) only, 5 in heating season (Dec-Mar) only; 2003-2005.	$PM_{2.5}$: 0.91 ± 0.23 Chemical species: EC: 0.72 ± 0.49; Ca: 0.56 ± 0.30; Fe: 0.38 ± 0.26; K: 0.83 ± 0.52; Si: 0.02 ± 0.00; Na: 0.46 ± 0.43; Cl: 0.40 ± 0.12; Zn: 0.85 ± 0.28; S: 0.95 ± 0.78; V: 0.60 ± 0.77	$PM_{2.5}$ (coefficient of variation (CV)): 1.14 (0.71) Chemical species (coefficient of variation (CV)): EC (CV): 0.89 (0.64); Ca (CV): 1.16 (1.90); Fe (CV): 0.69 (1.40); K (CV): 1.10 (0.95); Si (CV): 1.04 (1.31); Na (CV): 1.05 (1.84); Cl (CV): 3.18 (3.79); Zn (CV): 0.83 ± (1.13); S (CV): 0.76 ± (0.32); V (CV): 0.76 ± (0.46)
Baxter et al. (2007, 092725)	**Objective:** To predict residential indoor concentrations of traffic-related air pollutants in lower SES urban households. Part of ACCESS cohort study of asthma etiology. **Methods:** Regression modeling, Bayesian variable selection I/O is slope from multivariate model **Subjects:** Lower statuses, urban households in Boston, MA. 43 sites among 39 households, 66 sampling sessions, nonheating (May-Oct) and heating (Dec-Mar) 2003-2005	n/a	$PM_{2.5}$: Ambient Concentrations X Open Windows- 0.98; Ambient Concentrations X Closed Windows- 0.64 EC: Ambient Concentrations- 0.38
Brown et al. (2008, 190894)	**Objective:** To examine if ambient, home outdoor, and home indoor particle concentrations can be used as proxies of corresponding personal exposure. **Methods:** Associations characterized using univariate mixed effects models that included a random subject term. **Subjects:** 15 participants in Boston, MA in winter (Nov. 1999-Jan. 2000) and summer (June-July 2000).	n/a	$PM_{2.5}$: Winter- Median: 1.2, Range: 0.8-1.8; Summer- Median: 0.9, Range: 0.6-1.2 EC: Winter- Median: 1.1, Range: 0.7-4.5; Summer- Median- 1.0, Range: 0.9-1.3 SO_4^{2-}: Winter- Median: 0.5, Range: 0.3-0.8; Summer- Median: 0.8, Range: 0.4-1.0
Cao et al. (2005, 156321)	**Objective:** To determine relationships and distributions of indoor and outdoor $PM_{2.5}$, OC, and EC. To determine indoor/outdoor sources to indoor carbonaceous aerosol. **Methods:** Gravimetric analysis to determine $PM_{2.5}$ concentrations. OC and EC determined by TOR following IMPROVE protocol. **Subjects:** 6 residences in Hong Kong (2 roadside, 2 urban, 2 rural). March 6-April 18, 2004.	n/a	20min $PM_{2.5}$- Roadside: 0.7-4.0, Urban: 0.9-6.7, Rural: 0.5-1.7 24h $PM_{2.5}$- Roadside: 0.8-1.4, Urban: 1.2-2.0, Rural: 1.0-1.8 OC (average and range)- Roadside: 1.9 (1.1-2.3), Urban: 2.3 (1.5-4.0), Rural: 1.3 (1.2-2.2) EC (average and range)- Roadside: 1.0 (0.9-1.1), Urban: 1.1 (0.8-1.3), Rural: 1.1 (0.9-1.8)

Reference	Study Design	F_{inf}	I/O
Cortez-Lugo et al. (2008, 156368)	**Objective:** To determine personal $PM_{2.5}$ and its relationship with outdoor and indoor $PM_{2.5}$ and PM_{10}. **Methods:** Linear regression model used to compare personal and indoor $PM_{2.5}$. I/O variation studied using analysis of variance and predictors determined by generalized estimating equation models. I/O $PM_{2.5}$ ratio transformed into natural logarithm. **Subjects:** 38 nonsmoking long-time Mexico residents with COPD. Mexico City, Mexico. Feb-Nov 2000.	n/a	$PM_{2.5}$: Average- 1.2, Range- 0.05-6.1
Crist et al. (2008, 156372)	**Objective:** To examine correlations between personal, indoor, and outdoor $PM_{2.5}$ exposures in children. **Methods:** Indoor and personal samples by Whatman Teflon filters. Ambient samples taken by TEOMs. **Subjects:** Fourth and fifth-grade children. 1 school in Athens, OH and 2 schools inColumbus, OH. No pre-existing health conditions. 90 children (30 at each site), 3 of which had personal monitors. 194-332 days of indoor, outdoor, and personal samples. N samples taken at schools range 31-235.	n/a	Mean $PM_{2.5}$ Mass Concentrations ($\mu g/m^3$)- Athens: school-day- 2.61 ± 5.76, Non-school-day- 0.8 ± 0.7 Koebel: school-day- 1.71 ± 3.17, non-school-day- 1.27 ± 1.16 New Albany: school-day- 2.98 ± 5.47, non-school-day- 0.82 ± 0.6
Diapouli et al. (2008, 190893)	**Objective:** To characterize the PM_{10}, $PM_{2.5}$, UFP concentrations at primary schools. To examine the relationship between indoor and outdoor concentrations. **Methods:** Chemical analysis of collected filters. Regressions to examine correlations between indoor and outdoor concentrations. **Subjects:** 7 primary schools with different characterizations of urbanization and traffic density in Athens, Greece. No ventilation system. Nov. 2003-Feb. 2004 and Oct.-Dec. 2004.	n/a	PM_{10}: 0.54-2.46 $PM_{2.5}$- 0.67-2.77 UFP- 0.33-0.74
Dimitroulopoulou et al. (2006, 090302)	**Objective:** To develop a probabilistic indoor air model (INDAIR). **Methods:** INDAIR predicts frequency distributions of concentrations of up to 4 pollutants simultaneously (NO_2, CO, PM_{10}, $PM_{2.5}$). 3 scenarios- no source, gas cooking, smoking. **Subjects:** 5 UK sites- Harwell (rural), Birmingham East (urban background), Bradford (urban center), Bloomsbury (urban center), Marylebone Road (roadside). Winter (October 1-March 31), summer (April 1-September 30), 1997-1999.	n/a	No source- PM_{10}: 0.5-0.65; $PM_{2.5}$: 0.6-0.7 Gas cooking- PM_{10}: 0.6-0.9 (bedroom), 1.0-2.0 (lounge), 1.6-4.3 (kitchen); $PM_{2.5}$: 0.74-0.9 (bedroom), 0.9-1.6 (lounge), 1.6-2.9 (kitchen) Smoking- PM_{10}: 0.7-1.1 (bedroom), 1.1-2.7 (lounge), 1.1-2.5 (kitchen); $PM_{2.5}$: 0.8-1.3 (bedroom), 1.3-2.8 (lounge), 1.4-2.6 (kitchen)

Reference	Study Design	F_{inf}	I/O
Fromme et al. (2008, 155147)	**Objective:** To characterize the chemical and morphological properties of PM in classrooms and in corresponding outdoor air. **Methods:** PM F_{inf} derived from sulfate F_{inf} and a correction factor that results from division of B^{PM} (increase of indoor PM per outdoor PM, linear relationship) by B^{sulf} (increase of indoor sulfate per outdoor sulfate, linear relationship). If no indoor source, the sulfate F_{inf} is equal to the sulfate I/O. **Subjects:** Primary school in northern Munich. Densely populated residential area 160m away from a very busy street. Classrooms had 21-23 students. Sampling during teaching hours. Oct.-Nov. 2005.	n/a	PM_{10}: Sulfate- 0.3, Nitrate- 0.1, Cl- 0.6, Na- 0.9, Ammonium- 0.1, Mg- 0.6, Ca- 1.4, EC- 0.7, OC- 1.1 $PM_{2.5}$: Sulfate- 0.4, Nitrate- 0.2, Cl- 0.5, Na- 0.6, Ammonium- 0.3, Mg- 0.5, Ca- 1.6
Guo et al. (2004, 156506)[1]	**Objective:** To investigate pollutant concentrations at air-conditioned and non-air-conditioned markets. To compare indoor air quality with the Hong Kong standard. **Methods:** PM_{10} concentrations measured by Hi-Vol sampler correlated with corresponding levels measured by Dust-Trak monitor. **Subjects:** 3 non-air-conditioned and 2 air-conditioned markets in Hong Kong. Sept. 2001-Jan. 2002.	n/a	PM_{10}: Non-air-conditioned- ~0.7, Air-conditioned- ~0.98
Hänninen et al. (2004, 056812)[2]	**Objective:** To assess indoor $PM_{2.5}$ by origin and potential determinants. **Methods:** Part of EXPOLIS study. Pump and filter with gravimetric analysis. Univariate single and stepwise-multiple regression analyses. **Subjects:** Residential homes in Athens, Greece; Basle, Switzerland; Helsinki, Finland; Prague, Czech Republic. Homes by city: Athens 50, Basle 50, Helsinki 189, Prague 49.	$PM_{2.5}$ (mean): Athens- 0.70 ± 0.12,Basle- 0.63 ± 0.15,Helsinki- 0.59 ± 0.17,Prague- 0.61 ± 0.14 Sulfur (mean): Athens- 0.82 ± 0.14, Basle- 0.80 ± 0.19, Helsinki- 0.70 ± 0.20, Prague- 0.72 ± 0.16	$PM_{2.5}$: Athens- ~0.84, Basle- ~1.37, Helsinki- ~1.30, Prague- ~1.33 Sulfur: Athens- ~0.70, Basle- ~0.80, Helsinki- ~0.74, Prague- ~0.77
Ho et al. (2004, 056804)[3]	**Objective:** $PM_{2.5}$, OC, and EC exposure assessment of occupied buildings located near major roadways under natural ventilation (NV) and mechanical ventilation (MV). **Methods:** Co-located mini-volume samplers and Partisol model 2000 sampler with 2.5 micron inlet. IMPROVE TOR carbon analysis. **Subjects:** Occupants of MV (1 classroom and office) and NV (3 residences) buildings located within 10m of major roadway; Hong Kong, China. Sep. 2002-Feb. 2003.	$PM_{2.5}$: 0.42 EC- MV: 0.42, NV: 0.76 OC- MV: 0.66, NV: 0.71	$PM_{2.5}$ (average)- 0.2-1.6; MV (average)- <0.7; NV (average)- 0.6-1.6 EC- Range: 0.5±0.1 – 1.1±0.4 OC- Range: 0.6±0.2 – 1.5±1.0

Reference	Study Design	F_{inf}	I/O
Hoek et al. (2008, 156554)	**Objective:** Exposure assessmentof indoor/outdoor particle relationships.RUPIOH study. **Methods:** Sampling by condensation particle counters and Harvard impactors. Gravimetric analysis and reflectance. Calculations performed for 24h avg concentrations. F_{inf} estimated by linear regression analysis. **Subjects:** 4 European cities (Helsinki, Finland; Athens, Greece; Amsterdam, The Netherlands; Birmingham, England). Urban populations. > 35yrs. Asthma or COPD. Non-smoking households. Work < 16h/wk outside home. 153 homes sampled Oct. 2002-Mar. 2004.	Regression slope for indoor vs. central site outdoor- $PM_{2.5}$: 0.30-0.51; PM_{10}: 0.17-0.41; $PM_{10-2.5}$: 0.01-0.17; Sulfate: 0.59-0.78,; Soot: 0.43-0.87 Regression slope for indoor vs. residential outdoor- $PM_{2.5}$: 0.34-0.48; PM_{10}: 0.26-0.44; $PM_{10-2.5}$: 0.11-0.16; Soot: 0.63-0.84	n/a
Hopke et al. (2003, 095544)	**Objective:** To use advanced factor analysis models to identify and quantify PM sources. 1998 BPMEES data. **Methods:** PEM, outdoor and indoor sampling of unoccupied apartment in retirement facility. PMF used to derive source contributions. Multilinear Engine used to derive joint factors. **Subjects:** 10 non-smoking elderly subjects of mean age 84 who did not cook.Towson, MD. July 26-Aug. 22, 1998.	Nitrate-sulfate- 0.03; Sulfate- 0.38; OC- 0.77; MV Exhaust- 0.32	n/a
Hystad et al. (2008, 190890)	**Objective:** To explore the feasibility of modeling residential $PM_{2.5}$ F_{inf} for occupied residences using data readily available for most of North America. **Methods:** F_{inf} calculated by recursive mass balance model where F_{inf} is a function of penetration efficiency, particle removal rate, and air exchange. **Subjects:** 46 residences in Seattle, WA 1999-2003. 38 nonsmoking residences in Victoria, British Columbia, Canada 2006. Heating (Oct.-Feb.) and nonheating (March-Sept.).	Seattle: Mean (all)- 0.59 ± 0.21, Mean (detached residences)- 0.60 ± 0.20 Victoria: Mean (all)- 0.62 ± 0.22, Mean (detached residences)- 0.59 ± 0.22	n/a
Klinmalee et al. (2008, 190888)	**Objective:** To monitor indoor and outdoor pollution in an university campus and shopping center. **Methods:** PM measured by PEM and quartz filters. Analyzed for mass, water soluble ions by ion chromatography, and black carbon by a smokestain reflectometer. I/O calculated for each sample pair then average taken. **Subjects:** University campus and shopping center in northern suburb of Bangkok, Thailand. Dec. 2005-Feb. 2006.	n/a	$PM_{2.5}$: University- Weekdays: 0.6, Weekends: 0.5; Shopping center- Weekdays: 1.5, Weekends: 2.0 BC in $PM_{2.5}$: University- 0.9, Shopping center- 0.67

Reference	Study Design	F_{inf}	I/O
Koistinen et al. (2004, 156655)	**Objective:** To identify $PM_{2.5}$ sources in personal exposures with principal component analysis of the elemental compositions in residential outdoor, indoor, and workplace indoor microenvironments. Part of EXPOLIS study. **Methods:** Principal component analysis to identify sources of microenvironmental and personal $PM_{2.5}$ exposure. Specific mass contributions of sources calculated by source reconstruction. **Subjects:** Non-smoking, 25-55yrs. Helsinki, Finland. Oct. 1996-Dec. 1997.	n/a	Median seasonal- $PM_{2.5}$: Winter- 0.77, Spring- 1.03, Summer- 0.95, Fall- 0.92, Total- 0.92 Pb: Winter- 0.67, Spring- 0.56, Summer- 0.86, Fall- 0.69, Total- 0.67 S: Winter- 0.60, Spring- 0.63, Summer- 0.90, Fall- 0.75, Total- 0.69 Br: Winter- 0.57, Spring- 0.72, Summer- 0.98, Fall- 0.89, Total- 0.77 Black Smoke: Winter- 0.65, Spring- 0.67, Summer- 0.91, Fall- 0.88, Total- 0.79 Zn: Winter- 0.58, Spring- 0.75, Summer- 0.66, Fall- 0.75, Total- 0.68 Fe: Winter- 0.52, Spring- 0.96, Summer- 0.90, Fall- 0.95, Total- 0.83 K: Winter- 0.95, Spring- 1.05, Summer- 1.01, Fall- 1.08, Total- 1.05 Cl: Winter- 1.01, Spring- 1.24, Summer- 1.37, Fall- 1.74, Total- 1.24 Al: Winter- 1.19, Spring- 1.08, Summer- 1.41, Fall- 2.20, Total- 1.27
Li et al. (2003, 047845)	**Objective:** To establish effects of evaporative coolers on indoor PM concentrations. **Methods:** Concurrent 10min avg indoor and outdoor concentrations recorded for 2 days.I/O determined by equation based on mass conversation principles. **Subjects:** 10 homes with evaporative coolers. El Paso, TX. June 22-Aug. 23, 2001.	n/a	PM_{10}- All: 0.60, Cooler On: 0.57, Cooler Off: 0.66 $PM_{2.5}$- All: 0.65, Cooler On: 0.63, Cooler Off: 0.73
Lunden et al. (2008, 155949)	**Objective:** To investigate the physiochemical processes that influence the transport and fate of outdoor particles to the indoor environment. **Methods:** I/O calculated from measurements of aerosols collected on quartz filters. **Subjects:** 3-bedroom single-story unoccupied house in Clovis, CA. 3 periods: Oct. 9-23, 2000; Dec. 1-19, 2000; Jan. 12-23, 2001.	n/a	$PM_{2.5}$: Oct.- 0.46 ± 0.2, Dec.- 0.39 ± 0.2, Jan.- 0.38 ± 0.3, All periods- 0.41 ± 0.2 Carbon: Oct.- 0.50 ± 0.1, Dec.- 0.46 ± 0.1, Jan.- 0.52 ± 0.2, All periods- 0.50 ± 0.2 OC: Oct.- 0.48 ± 0.1, Dec.- 0.44 ± 0.1, Jan.- 0.50 ± 0.2, All periods- 0.47 ± 0.2 Black carbon: Oct.- 0.60 ± 0.2, Dec.- 0.60 ± 0.2, Jan.- 0.65 ± 0.2, All periods- 0.61 ± 0.2
MacIntosh et al. (2009, 190887)	**Objective:** To estimate the potential for residential air cleaning systems to mitigate exposure to fine particles of ambient origin. **Methods:** Multi-zone indoor air quality model to examine annual, 24h avg and diurnal concentrations of outdoor $PM_{2.5}$ in residential indoor air. **Subjects:** Homes in Cincinnati, Cleveland, and Columbus, OH that have natural ventilation, forced air heating and cooling with conventional in-duct filtration, or forces air heating and cooling with high-efficiency in-duct air cleaning. 2005.	n/a	$PM_{2.5}$ (range): Natural ventilation- 0.23-0.97; Forced air – conventional filtration- 0.13-0.94; Forced air – high-efficiency electrostatic- 0.02-0.80

Reference	Study Design	F_{inf}	I/O
Martuzevicius et al. (2008, 190886)	**Objective:** To determine the contribution of traffic-related PM to the indoor aerosols. **Methods:** Receptor modeling based on a PARAFAC model. **Subjects:** 6 houses 30-300m from a highway, with conventional windows, central HVAC, and with smoking and cooking allowed. Spring: Mar. 30-May 14, 2004. Fall: Sept. 13-Oct. 22, 2004. Cincinnati, OH.	n/a	Range- $PM_{2.5}$: Spring- 0.5 ± 0.2 – 2.9 ± 1.2; Fall- 0.7 ± 0.1 – 4.7 ± 6.9 EC: Spring- 0.3 ± 0.1 – 2.2 ± 1.7; Winter- 0.6 ± 0.1 – 1.3 ± 0.7 OC: Spring- 1.0 ± 0.7 – 6.9 ± 3.9; Winter- 1.2 ± 0.1 – 7.6 ± 10 Si: Spring- 0.4 ± 0.1 – 5.1 ± 3.9; Winter- 0.5 ± 0.1 – 5.3 ± 4.5 S: Spring- 0.4 ± 0.1 – 0.7 ± 0.1; Winter- 0.5 ± 0.1 – 0.9 ± 0.4 Mn: Spring- 0.3 ± 0.2 – 0.8 ± 0.6; Winter- 0.3 ± 0.2 – 1.0 ± 0.2 Fe: Spring- 0.3 ± 0.0 – 1.3 ± 0.8; Winter- 0.4 ± 0.1 – 0.9 ± 0.6 Zn: Spring- 0.3 ± 0.1 – 0.7 ± 0.6; Winter- 0.6 ± 0.1 – 1.1 ± 0.8 Br: Spring- 0.3 ± 0.1 – 1.0 ± 0.5; Winter- 0.2 ± 0.1 – 0.9 ± 0.6 Pb: Spring- 0.3 ± 0.3 – 0.9 ± 0.6; Winter- 0.2 ± 0.2 – 1.9 ± 2.3
Meng et al. (2005, 058595)	**Objective:** Analyses of RIOPA data, which investigated relationships between indoor, outdoor, and personal exposure for several air pollutants. **Methods:** PM measured on Teflon filters collected by PEMs for 48h. The mass balance model and RCS statistical model used to estimate indoor and and personal PM concentrations. **Subjects:** 212 nonsmoking homes sampled. Houston, TX; Los Angeles County, CA; Elizabeth, NJ. Summer 1999-spring 2001, all 4 seasons.	$PM_{2.5}$- 0.46	Los Angeles: $PM_{2.5}$- Mean: 0.84, Median: 0.90; EC- Mean: 0.93, Median: 0.92; OC- Mean: 1.32, Median: 1.31 Elizabeth: $PM_{2.5}$- Mean: 0.99, Median: 0.86; EC- Mean: 1.0, Median: 0.85; OC- Mean: 2.4, Median: 1.8 Houston: $PM_{2.5}$- Mean: 1.16, Median: 1.02; EC- Mean: 1.0, Median: 0.71; OC- Mean: 2.25, Median: 2.35
Molnár et al. (2007, 156774)	**Objective:** To characterize and compare indoor and outdoor $PM_{2.5}$ trace element concentrations in difference microenvironments related to children. **Methods:** Elemental concentrations analyzed using X-ray fluorescence spectroscopy. **Subjects:** 40 sampling sites (10 classrooms in 5 schools, 10 preschools, 20 non-smoking homes). 3 communities in Stockholm, Sweden. Sampled once during spring and once during winter. Dec. 1, 2003-July 1, 2004.	$PM_{2.5}$ (containing sulfur or lead): 0.4-0.9	Sulfur (median): Both seasons- 0.61 (homes), 0.53 (schools), 0.69 (preschools); Winter- 0.47 (homes), 0.36 (schools), 0.63 (preschools); Spring- 0.63 (homes), 0.55 (schools), 0.90 (preschools) Lead (median): Both seasons- 0.70 (homes), 0.59 (schools), 0.70 (preschools); Winter- 0.62 (homes), 0.43 (schools), 0.63 (preschools); Spring- 0.70 (homes), 0.64 (schools), 0.75 (preschools)

Reference	Study Design	F_{inf}	I/O
Ng et al. (2005, 155996)	**Objective:** To estimate PM exposures following the September 11, 2001 attack in NYC. **Methods:** Outdoor $PM_{2.5}$ interpolated and used in a deterministic micro-environmental model (INTAIR) to simulate analytically concentrations in indoor micro-environments. Linear regression equations used. **Subjects:** Lower Manhattan residents divided into representative individuals – home-maker, office/shop-worker, student/child. Estimates Sept. 14-31.	n/a	Mean I/O in home simulated with INTAIR- No Source: 0.6, Smoking: 1.9, Cooking: 1.3, Smoking and Cooking: 2.3; I/O of micro-environments simulated by analytical and empirical methods (no indoor source)- Office/Shop: 0.4, Classroom: 0.9, Transport Area: 1.9, Store: 1.2
Paschold et al. (2003, 156847)	**Objective:** To identify PM sources inside homes with evaporative coolers. **Methods:** PM element composition analysis by ICP-MS. **Subjects:** 10 residences. El Paso, TX. Summer 2001.	n/a	PM_{10}: Na- 0.33, Mg- 0.43, Al- 0.50, K- 0.48, Ca- 0.40, Ti- 0.52, Mn- 0.48, Fe- 0.46, Cu- 0.74, Zn- 0.52, Ba- 0.54, Pb- 0.76 $PM_{2.5}$: Na- 0.20, Mg- 0.29, Al- 0.34, K- 0.30, Ca- 0.52, Ti- 0.40, Mn- 0.35, Fe- 0.30, Cu- 0.67, Zn- 0.34, Ba- 0.47, Pb- 0.51
Polidori et al. (2007, 156877)	**Objective:** To investigate the relationships of indoor and outdoor $PM_{2.5}$, its components, seasonal variations, and gaseous copollutants. **Methods:** F_{inf} estimated by analysis of I/O's and a recursive model technique. **Subjects:** 2 retirement facilities in Los Angeles, CA. July 6-Aug. 20, 2005. Aug. 24-Oct. 15, 2005. Oct. 19-Dec. 10, 2005. Jan. 4-Feb. 18, 2006.	$PM_{2.5}$: July 6-Aug. 20- 0.71 ± 0.10; Aug. 24-Oct. 15- 0.60 ± 0.05; Oct. 19-Dec. 10- 0.59 ± 0.07; Jan. 4-Feb. 18- 0.45 ± 0.06 OC: July 6-Aug. 20- 0.86 ± 0.05; Aug. 24-Oct. 15- 0.77 ± 0.09; Oct. 19-Dec. 10- 0.82 ± 0.07; Jan. 4-Feb. 18- 0.64 ± 0.10 EC: July 6-Aug. 20- 0.73 ± 0.07; Aug. 24-Oct. 15- 0.71 ± 0.05; Oct. 19-Dec. 10- 0.77 ± 0.06; Jan. 4-Feb. 18- 0.64 ± 0.10	Only I/O's ◘1considered
Ramachandran et al. (2003, 188454)	**Objective:** To examine variability in measurements of 24h avg and 15min avg $PM_{2.5}$ concentrations. **Methods:** Linear regression of gravimetric measurements. **Subjects:** 3 urban residential neighborhoods in Minneaopolis-St. Paul, MN. 9-10 nonsmoking residences. Spring (April 26-June 2), summer (June 20-Aug. 10), fall (Sept. 23-Nov. 20) of 1999.	n/a	24h avg: Mean- 1.7, Median- 1.3, Standard deviation- 1.6 15min avg: Mean- 2.7, Median- 1.2, Standard deviation- 8.7
Rojas-Bracho et al. (2004, 054772)	**Objective:** To examine determinants of personal exposure to $PM_{2.5}$, PM_{10}, $PM_{2.5-10}$. **Methods:** 2 sets of mixed models. Personal exposures modeled as dependent variables. Subject variability modeled using random effects. Explanatory variables and season modeled as fixed effects. **Subjects:** 18 COPD subjects in nonsmoking households. Boston, MA. Winters of 1996 and 1997, summer of 1996.	n/a	$PM_{2.5}$: Winter- Mean: 1.58, Median: 2.11; Summer- Mean: 1.08, Median: 0.88 PM_{10}: Winter- Mean: 2.02, Median: 3.77; Summer-Mean: 1.14, Median: 1.05 $PM_{2.5-10}$: Winter- Mean: 2.65, Median: 3.59; Summer- Mean: 1.26, Median: 1.39

Reference	Study Design	F_{inf}	I/O
Sarnat et al. (2006, 089166)	**Objective:** To assess the ability of outdoor $PM_{2.5}$, its volatile and nonvolatile components and particle sizes to infiltrate indoors. **Methods:** $PM_{2.5}$ mass contributions estimated by the mean concentration ratio between each component and $PM_{2.5}$. Indoor and outdoor particle concentrations relationships examined by Spearman correlation coefficient. I/O concentration ratios used during overnight (nonsource) period to estimate fraction of ambient particles remaining airborne indoors (F_{inf}). **Subjects:** 17 occupied, nonsmoking Los Angeles, CA residences. July 28, 2001-Feb. 25, 2002.	$PM_{2.5}$: Median- 0.48, Interquartile range- 0.39-0.57 BC: Median- 0.84, Interquartile range- 0.70-0.96 UFP (0.02-0.03 μm): Median- 0.50, Interquartile range- 0.39-0.60 UFP (0.08-0.3 μm): Median- ~0.75 Coarse particles (5-10 μm): Median- <0.17	$PM_{2.5}$: Overnight- 0.40-0.57, Morning- 0.43-0.74, Afternoon- 0.45-0.90, Evening- 0.42-0.82 BC: Overnight- 0.70-0.97, Morning- 0.67-0.98, Afternoon- 0.77-1.04, Evening- 0.70-1.01
Stranger et al. (2008, 190884)	**Objective:** To assess indoor air quality by determining indoor and outdoor $PM_{2.5}$ mass concentrations, elemental composition, and gaseous compounds. **Methods:** PM mass concentrations determined gravimetrically. **Subjects:** 27 primary schools in city center and suburbs of Antwerp, Belgium. Dec. 2002 and June 2003.	n/a	$PM_{2.5}$: Urban- Range: 0.3-6.9, Average: 1.3; Suburban- Range: 0.2-8.8, Average: 2.3 V, Ni, Zn, Pb, Br, Mn: <1 Cl, Ca, Al, Si, K, Ti, Fe: >1 Black smoke: Urban- Average: Dec.- 0.7 ± 0.1, June- 1.1 ± 0.3; Suburban: Dec.- 0.8 ± 0.2, June-1.0 ± 0.4
Stranger et al. (2009, 190883)	**Objective:** To assess indoor air quality in residences by quantifying various gaseous pollutants, and PM mass concentrations, elemental composition, and water-soluble ionic content. **Methods:** PM mass concentrations gravimetrically determined. Elemental bulk analysis on filters. **Subjects:** 19 residential homes in Antwerp, Belgium that were a subset of participants in the ECRHS II study.	n/a	PM_1: Houses 1-15- Average: 2.0, Range: 0.3-9.6; Smokers: Average- 3.9, Range- 1.2-9.7; Non-smokers: Average: 0.8, Range- 0.3-14 $PM_{2.5}$: Houses 1-15- Average: 1.5, Range: 0.4-5.4, Smokers average: 2.5, Smokers range: 1.2-5.4, Non-smokers average: 0.8, Non-smokers range: 0.4-1.3; Houses 16-19- Average: 2.6, Range: 0.3-3.9 PM_{10}: Houses 1-15- Average: 1.3, Range: 0.4-4.1, Smokers average: 2.1, Smokers range: 1.1-4.1, Non-smokers average: 0.8, Non-smokers range: 0.4-1.2 Ca, Ti, V, Cr, Mn, Fe, Ni, Zn, Pb, Si, S, Cl: <1 K, Cu, Br, Al: >1
Turpin et al. (2007, 157062)	**Objective:** To characterize and compare outdoor, indoor, personal $PM_{2.5}$ exposure. Identify indoor and personal $PM_{2.5}$ sources. Estimate outdoor $PM_{2.5}$ effect on indoor and personal $PM_{2.5}$. RIOPA study. **Methods:** F_{inf} calculated in three ways: RCS model used to obtain constant F_{inf}. Mass balance model shows F_{inf} varying with AER. Robust regression uses major $PM_{2.5}$ species for home-specific F_{inf}. **Subjects:** 309 nonsmoking adults and 118 children with no preexisting conditions. 219 homes sampled. Elizabeth NJ, Houston TX, and Los Angeles County CA.	$PM_{2.5}$- RCS model: 0.46; Least-Trimmed Squared Regression- Mean: 0.69, Median- 0.70, SD: 0.23; Mass Balance Model- ~0.08- ~0.85	Los Angeles: $PM_{2.5}$- Mean: 0.84, Median: 0.90; EC- Mean: 0.93, Median: 0.92; OC- Mean: 1.32, Median: 1.31 Elizabeth: $PM_{2.5}$- Mean: 0.99, Median: 0.86; EC- Mean: 1.0, Median: 0.85; OC- Mean: 2.4, Median: 1.8 Houston: $PM_{2.5}$- Mean: 1.16, Median: 1.02; EC- Mean: 1.0, Median: 0.71; OC- Mean: 2.25, Median: 2.35

Reference	Study Design	F_{inf}	I/O
Wallace and Williams (2005, 057485)	**Objective:** To estimate the contribution of outdoor $PM_{2.5}$ to personal exposure in high-risk subpopulations. **Methods:** Longitudinal regressions of estimated indoor and outdoor $PM_{2.5}$ for F_{inf}. **Subjects:** 29 African-Americans with hypertension and 8 with implanted cardiac defibrillators. Measured 7d/season, 4 seasons in 2000-2001. Raleigh, NC.	Range: 0.35-0.87	$PM_{2.5}$: Mean- 1.08 ± 1.05, Median- 0.75, Range- 0.24-9.48 Sulfur: Mean- 0.59 ± 0.16, Median- 0.58, Range- 0.17-1.06
Williams et al. (2003, 053338)	**Objective:** To estimate ambient $PM_{2.5}$ contributions to personal and indoor residential PM mass concentrations. **Methods:** F_{inf} estimated from least squares, regression analysis, and mixed model slope. **Subjects:** Nonsmoking, ambulatory, ≥ 50yrs. 2 cohorts: mostly Caucasian with implanted cardiac defibrillators in Chapel, NC; 30 African-Americans with controlled hypertension in low-to-moderate SES neighborhoods in Raleigh, NC. 7d/season, 4 seasons in 2000-2001.	Least squares estimate of indoor filtration factors: Mean- 0.42 ± 0.38, Range- -0.55 to 1.62 Regression analysis: 0.43 ± 0.06 Mixed model slope: Mean- 0.45 ± 0.21, Range- 0.05-0.94	n/a
Williams et al. (2008, 191201)	**Objective:** To examine the spatial variability of $PM_{2.5}$ and $PM_{10-2.5}$ and their components to determine the suitability of conducting health outcome studies using a central site monitor in a metropolitan area having multiple source impacts. **Methods:** Gravimetric analysis of PM mass concentrations. ED-XRF analysis of PM elements. **Subjects:** Non-smoking, ambulatory, and living in detached homes and non-smoking households. Detroit, MI.	$PM_{2.5}$: Range- 0.16-6.45, Mean- 0.7 ± 0.33, Median- 0.70 (indicate indoor sulfur source when $F_{inf} > 1$)	n/a
Wilson and Brauer (2006, 088933)	**Objective:** To provide additional insight into factors affecting exposure to airborne PM and the resultant health effects. **Methods:** F_{inf} estimated by mass balance equation. **Subjects:** 16 nonsmoking subjects with COPD. 54-86yrs. Vancouver, British Columbia. April-Sept. 1998.	Sulfate: 0.72	n/a
Wu et al. (2006, 179950)	**Objective:** To assess personal $PM_{2.5}$ exposures from ambient sources and agriculture burning smoke. **Methods:** F_{inf} estimated by RCS model. Application of Robust regression algorithm. **Subjects:** 33 adult asthmatics. 18-52yrs. Pullman, WA. Sept. 3, 2002-Nov. 1, 2002.	Range: 0.25-0.94	n/a

Reference	Study Design	F_{inf}	I/O
Yang et al. (2009, 190885)	**Objective:** To characterize the concentrations of different indoor air pollutants. **Methods:** PM_{10} collected on pall flex membrane filer using MiniVol portable air samplers. Arithmetic and geometric means calculated for indoor concentrations. Differences in concentrations measured by Kruskal-Wallis test. **Subjects:** 55 schools in 6 metropolitan areas in Korea. Samples from a classroom, laboratory, and computer classroom. 3 seasons, July-Dec. 2004.	n/a	PM_{10}: Classroom- Summer: 1.98, Autumn: 2.25, Winter: 2.07, Total: 2.06 Laboratory- Summer: 1.33, Autumn: 1.32, Winter: 1.72, Total: 1.46 Computer classroom: Summer- 0.77, Autumn: 1.43, Winter: 2.08, Total: 1.43
Zhao and Hopke (2004, 100956)	**Objective:** To characterize airborne particle sources that are common to personal, residential indoor, residential outdoor, and ambient environments. To study relationships between $PM_{2.5}$ exposure and emission sources. **Methods:** Expanded receptor model. **Subjects:** Nonsmoking, ambulatory, ≥50yrs. 2 cohorts: mostly Caucasian with implanted cardiac defibrillators in Chapel, NC; 30 African-Americans with controlled hypertension in low-to-moderate SES neighborhoods in Raleigh, NC. 7d/season, 4 seasons in 2000-2001.	n/a	n/a
Zhu et al. (2005, 190081)	**Objective:** To determine penetration behavior of outdoor ultrafine particles into indoor environments in areas close to freeways. **Methods:** Dynamic mass balance model. **Subjects:** 4 2-bedroom apartments within 60m from the center of the 405 Freeway in Los Angeles, CA. Non-smoking tenants. 2 sampling periods (non-cooking, non-cleaning): Oct.-Dec. 2003 and Dec. 2003-Jan. 2004.	n/a	Highest (largest ultrafine particles- 70-100nm): 0.6-0.9 Lowest (smallest ultrafine particles- 10-20nm): 0.1-0.4

1. I/O estimated from Figure 8 in study.

2. I/O calculated from indoor and outdoor concentrations in Table 1 in study.

3. F_{inf} measured by coefficient of determination, R^2.

4. RIOPA calculated I/O's.

5. I/O calculated from mean and median indoor and outdoor concentrations listed in Table 1 of study.

6. I/O's estimated from Figure 3 in study.

7. Mean and median I/O concentrations calculated from all residences in study.

8. F_{inf} estimated from Figure 2 in study.

9. F_{inf} presented in box plot (Figure 8), however data is difficult to deduce. No numeric values reported.

Table A-55. Summary of PM – copollutant exposure studies.

Reference	PM metric	Copollutant metric	Association between PM and copollutant	Primary findings
Fruin et al. (2008, 097183)	In-vehicle UFP, BC, PM-bound PAH	In-vehicle NO_x, CO	R UFP $PM_{2.5}$ NO BC CO CO_2 UFP 1 0.71 0.97 0.95 0.63 0.72 $PM_{2.5}$ 1 0.69 0.89 0.66 0.68 NO 1 0.91 0.78 0.85 BC 1 0.65 0.74 CO 1 0.94 CO_2 1 Note that these correlations are computed from data presented by Fruin et al. (2008, 097183) for mean concentrations at different loc ations.	Measurements of freeway UFP, BC, PM-bound PAH, and NO concentrations were roughly one order of magnitude higher than ambient measurements. Multiple regression analysis suggests these concentrations were a function of truck density and total truck count.
Schwartz et al. (2007, 090220)	Ambient and personal $PM_{2.5}$ data from the Baltimore panel study	Ambient and personal O_3 and NO_2 data from the Baltimore panel study.	Median β for regressions: Ambient $PM_{2.5}$ Ambient O_3 Ambient NO_2 Personal $PM_{2.5}$ 0.0143 - 0.0016 0.0115 Personal $PM_{2.5}$ of ambient origin 0.0183 -0.0037 0.0124 Personal SO_4^{2-} 0.0051 0.0035 0.0006 Personal O_3 0.0014 0.0010 0.0009 Personal NO_2 0.0015 0.0009 0.0010	Results suggest that ambient O_3 exposure may be related to personal SO_4^{2-} exposure but not to personal $PM_{2.5}$ exposure on the whole. Ambient NO_2 exposure was associated with personal $PM_{2.5}$ exposure, possibly because both have traffic sources.

Reference	PM metric	Copollutant metric	Association between PM and copollutant	Primary findings
Tolbert et al. (2007, 090316)	Ambient PM_{10}, $PM_{10-2.5}$, $PM_{2.5}$, EC, OC, TC, SO_4^{2-}, water-soluble metals, oxygenated hydrocarbons	Ambient O_3, NO_2, CO, SO_2	PM_{10} PMc $PM_{2.5}$ O_3 NO_2 CO SO_2 PM_{10} 1.0 O_3 0.6 1.0 NO_2 0.5 0.4 1.0 CO 0.5 0.3 0.7 1.0 SO_2 0.2 0.2 0.4 0.3 1.0 PMc 0.7 0.4 0.5 0.4 0.2 1.0 $PM_{2.5}$ 0.8 0.6 0.6 0.4 0.2 0.5 1.0 SO_4^{2-} 0.7 0.6 0.1 0.1 0.1 0.3 0.8 EC 0.6 0.4 0.6 0.7 0.2 0.5 0.7 OC 0.7 0.5 0.6 0.6 0.2 0.5 0.7 TC 0.7 0.5 0.7 0.6 0.2 0.5 0.7 Metals 0.7 0.4 0.3 0.4 0.1 0.5 0.7 OHC 0.5 0.4 0.2 0.3 0.1 0.4 0.5 SO_4^{2-} EC OC TC Metals OHC SO_4^{2-} 1.0 EC 0.3 1.0 OC 0.3 0.8 1.0 TC 0.3 0.9 1.0 1.0 Metals 0.7 0.5 0.5 0.5 1.0 OHC 0.5 0.4 0.4 0.4 0.5 1.0	Low correlations were seen between SO_2 and PM constituents. Components were used in a multi-pollutant model to predict emergency department visits in Atlanta. CO was found to be the most significant predictor of cardiovascular disease visits in one-, two-, and three-pollutant models, and O_3 was the most significant predictor of respiratory disease visits in one-, two-, and three-pollutant models.

Reference	PM metric	Copollutant metric	Association between PM and copollutant	Primary findings
Brook et al. (2007, 091153)	Anbient PM_{10}, $PM_{10-2.5}$, $PM_{2.5}$, SO_4^{2-}, and trace metals in 10 Canadian cities.	Ambient NO_2, NO	R with NO_2 (min, Max) NO_2 1.00 (1.00, 1.00) NO 0.67 (0.51, 0.77) $PM_{2.5}$ 0.54 (0.45, 0.71) $PM_{10-2.5}$ 0.31 (0.04, 0.50) PM_{10} 0.50 (0.23, 0.70) SO_4^{2-} 0.33 (0.10, 0.48) Fe 0.44 (0.29, 0.56) Zn 0.39 (0.28, 0.52) Ni 0.20 (0.06, 0.40) Mn 0.51 (0.37, 0.62) As 0.21 (0.07, 0.39) Al 0.07 (-0.17, 0.18) Cu 0.03 (-0.07, 0.15) Pb 0.28 (0.16, 0.39) Si 0.19 (0.00, 0.32) Se 0.14 (-0.04, 0.35)	NO_2 showed the strongest association with mortality, but it is unclear if this association is due to health effects of NO_2 or health effects of copollutant PM.
Ito et al. (2007, 156594)	Ambient $PM_{2.5}$	Ambient O_3, NO_2, SO_2, CO	Shown in figure format only.	Authors tested relationship between meteorological variables and copollutants to determine if multi-pollutant models are impacted by spatial or temporal variation or by meteorological conditions. Multicollinearity varied by pollutant and season.
Kaur et al. (2005, 086504)	Fixed-site and personal $PM_{2.5}$, personal UFP	Fixed site and personal CO	Personal R: $PM_{2.5}$ UFP CO $PM_{2.5}$ 1 0.5 0.2 UFP 0.5 1 0.7 CO 0.2 0.7 1	Fairly low correlation was observed between $PM_{2.5}$ and CO and between $PM_{2.5}$ and UFP, stronger correlations between UFP and CO.
Kaur et al. (2005, 088175)	Fixed-site and personal $PM_{2.5}$ analyzed post-sample for light absorbance (as indicator for carbonaceous aerosol), personal UFP	Fixed site and personal CO	Personal R: R $PM_{2.5}$ Abs CO UFP $PM_{2.5}$ 1 0.3 -0.1 0.0 Abs 0.3 1 0.2 0.7 CO -0.1 0.2 1 0.1 UFP 0.0 0.7 0.1 1	Strongest correlation observed between UFP and absorption, which is reasonable given that much absorptive carbonaceous aerosol is in the ultrafine range.
Sørenson et al. (2005, 089428)	Personal, indoor residential, and outdoor residential $PM_{2.5}$ and BC	Personal, indoor residential, and outdoor residential NO_2	Personal exposure regression coefficients to: $PM_{2.5}$ BC NO_2 Bedroom 0.72 0.47 0.70 Front door 0.46 0.61 0.60 Background 0.29 0.03 0.56	Personal NO_2 concentration is more strongly influenced by background than $PM_{2.5}$ or BC.
Sabin et al. (2005, 087728)	BC, particle-bound PAH on a school bus.	NO_2 on a school bus.	BC PB-PAH NO_2 BC 1 0.94 0.49 PB-PAH 1 0.37 NO_2 1 Note that these correlations are computed from data presented by Sabin et al. for mean concentrations when the test bus travelled behind different vehicles.	Less correlation was observed between NO_2 and PM species. This study was aimed more at fuel choices and control technologies for children's exposures on school buses.

Reference	PM metric	Copollutant metric	Association between PM and copollutant	Primary findings
Lai et al. (2004, 056811)	Microenvironmental and personal $PM_{2.5}$ and trace elements	Microenvironmental and personal VOCs, NO_2, and CO.	R $PM_{2.5}$ TVOC NO_2 TVOC 0.21 0.21 0.41 -0.32 NO_2 -0.1 -0.02 -0.16 0.09 -0.11 -0.01 -0.23 0.03 CO -0.07 NR NR NR 0.07 NR NR NR 0.3 NR NR NR Correlation coefficients listed (in order) for personal exposure, residential indoor, residential outdoor, and workplace indoor.	The EXPOLIS Oxford study was more focused on the indoor-outdoor exposure relationship, but the correlation results showed no important relationships between the pollutants shown.
Gomez-Perales et al. (2007, 138816; 2004, 054418)	Microenvironmental $PM_{2.5}$ with SO_4^{2-}, NO_3^-, EC, OC.	Microenvironmental CO.	Ratio of Conc $PM_{2.5}$ CO Benzene Minibus/Bus 1.04 1.54 2.01 1.20 1.40 1.33 Minibus/Metro 1.70 2.02 3.20 1.43 3.03 3.10	Morning and evening measurements of $PM_{2.5}$ were on avg higher and more variable than for benzene and CO (in order). Benzene and CO had higher and more variable concentrations for minibuses than for buses and metros, respectively, while $PM_{2.5}$ concentrations were not substantially different for buses and minibuses.
Sarnat et al. (2001, 019401)	Fixed site and personal $PM_{2.5}$ monitors.	Ambient O_3, NO_2, SO_2, and CO	R $PM_{2.5}$ O_3 NO_2 SO_2 CO $PM_{2.5}$ 1 0.67 0.37 --- 0.15 O_3 -0.72 1 0.02 --- -0.06 NO_2 0.75 -0.71 1 --- 0.75 SO_2 -0.17 0.41 -0.17 1 -0.32 CO 0.69 -0.67 0.76 -0.12 1	Strong association between ambient NO_2 and personal $PM_{2.5}$ suggests that ambient gas may be a suitable surrogate for personal exposure.

Table A-56. Summary of studies relating PM, SES, and mortality and/or morbidity.

Reference	Population Studied	Data interval	Metrics Used (health; pollutant; SES variable)	Study Outcome
Bateson and Schwartz (2004, 086244)	Residents (>65) of Cook Co. IL with prior cardiac or respiratory hospitalization, 1988-1991	Days	All-cause mortality; PM_{10}; median household income, % with bachelor's degree, % not speaking English at home	No significant change in mortality with a 10 $\mu g/m^3$ increase in PM_{10} with SES variables.
Cifuentes et al. (2000, 010351)	Residents (aged 25-64) of Santiago, Chile, 1988-1996	Days	Non-trauma mortality; $PM_{2.5}$; educational level	Relative risks of non-trauma mortality were at or near significance in the group having only elementary education.
Filleul et al. (2003, 087403)	Residents (aged >65) of Bordeaux, France, 1988-1997	Days	Non-trauma mortality; BC (10th or 90th percentile levels); education level, previous occupation (domestic, skilled, intellectual)	No significant effect between BC and non-trauma mortality was observed for either SES variable.
Filleul et al. (2004, 087404)	Residents (aged >65) of Bordeaux, France, 1988-1997	Days	Non-trauma mortality, cardio-respiratory mortality; BC; educational level, previous occupation (never worked, white-collar, blue collar)	Blue collar SES group had a significant odds ratio of non-trauma mortality; high education level had a significant odds ratio for cardio-respiratory mortality.
Filluel et al. (2005, 087357)	Adults (aged 25-59 at enrollment) in 7 French cities, 1974-2000	Years	Non-trauma mortality; BC, TSP; educational level	No trend as a function of education level.
Finkelstein et al. (2003, 056117)	Adults (aged >40) in Hamilton-Burlington, Canada, 1992-2001	Years	Non-trauma mortality; TSP; mean household income	Significantly higher relative risk as a function of TSP exposure for low and high income strata
Finkelstein et al. (2003, 056117)	Adults (aged >40) in Hamilton-Burlington, Canada, 1992-2001	Years	Cardio-vascular mortality; Pollution index (TSP and SO_2) (regional, urban, near-road), traffic proximity; deprivation index	No significant relative risk as a function of pollution index or traffic proximity.
Gouveia & Fletcher (2000, 012132)	Residents (aged >65) of Sao Paulo, Brazil, 1991-1993	Days	Non-trauma mortality; PM_{10}; composite SES index	Non-significant results show relative risk of non-trauma mortality as a function of PM_{10} slightly higher in advantaged neighborhoods.
Gwynn & Thurston (2001, 017206)	Residents of NY City, 1988-1990	Days	Respiratory hospital admissions; PM_{10}, sulfate; race, insurance	Higher but non-significant relative risk for non-whites than whites but neither with relative risk significantly different from 1 for PM_{10}; relative risk significantly higher than 1 for sulfate among non-whites.
Hoel et al. (2002, 042364)	Adults (aged 55-69 at enrollment) in The Netherlands, 1992-2000	Years	Non-trauma mortality; BC (regional, urban, near-road); educational level	No significant difference in relative risk as a function of BC exposure for education level
Ito and Thurston (1996, 078841)	Residents of Cook County, IL, 1985-1990	Days	Mortality; PM_{10}; race, sex	Mortality increased with PM_{10}, effects of sex and race were noted with black females > white females > black males > white males
Krewski et al. (2000, 012281)	Adults (aged 25-74 at enrollment) in Six Cities cohort, 1974-1991	Years	Non-trauma mortality, cardio-pulmonary mortality; $PM_{2.5}$, sulfates; educational level	Relative risk significantly greater than 1 for non-trauma mortality among those with less than high school education caused by increased $PM_{2.5}$ and sulfate exposures
Krewski et al. (2000, 012281)	Adults (aged >30 at enrollment) in American Cancer Society cohort, follow-up 1982-1989	Years	Non-trauma mortality, cardio-pulmonary mortality; $PM_{2.5}$, sulfates; educational level	Relative risk significantly greater than 1 for non-trauma and cardio-pulmonary mortality as a function of $PM_{2.5}$ exposure for less than high school and high school education; relative risk significantly greater than 1 for non-trauma and cardio-pulmonary mortality as a function of sulfate exposure for less than high school education.
Lee et al. (2006, 098248)	Children (aged < 15) in Seoul, Korea, 2002	Days	Hospitalized for asthma; PM_{10}; SES (listed as "high," "medium," or "low" of monitor site without explanation of criteria)	PM_{10} level does not vary linearly with increasing SES. Relative risk significantly greater than 1 for high and low SES.

Reference	Population Studied	Data interval	Metrics Used (health; pollutant; SES variable)	Study Outcome
Linn et al. (1999, 011680)	Residents of South Coast Air Basin, CA, 1992-1995	Days	Respiratory and cardiovascular hospital admissions; PM_{10}; sex, ethnicity (white, black, Hispanic, other)	Impact of PM_{10} on cardiovascular effects increased in blacks and whites relative to Hispanics and others.
Martins et al. (2004, 087457)	Residents (aged > 60) of six zones of Sao Paulo, Brazil, 1997-1999	Days	Respiratory mortality; PM_{10}; % with college education, % families with monthly income > $3500, % living in slums	% with college education and % families with monthly income > $3500 have negative impact of effect of PM_{10} on respiratory mortality, % people living in slums had positive effect.
Norris et al. (2000, 087104)	Children (aged < 18) in Seattle, WA, 1995-1996	Days	Emergency room visits for asthma; PM_{10}; high vs. low emergency room use	Relationship between PM_{10} and emergency room visits not significantly impacted by overall emergency room use.
O'Neill et al. (O'Neill et al., 2004, 055597)	Residents (aged > 65) of Mexico City, Mexico, 1996-1998	Days	Non-trauma mortality; PM_{10} and O_3; % homes with electricity, % homes with piped water, % literacy, % indigenous language speakers	PM_{10} not associated with non-trauma mortality (but significant associations for O_3).
Ou et al. (2008, 189955)	Residents (aged > 30) of Hong Kong, 1998	Days	Non-trauma mortality; PM_{10}; housing type, occupational level, education level	Housing type and blue-collar caused significantly greater impact of PM_{10} on mortality compared with single family housing or white-collar and never employed, respectively.
Pope et al. (2002, 024689)	Adults (aged > 30 at enrollment) in American Cancer Society cohort, follow-up 1982-1989	Years	Mortality; $PM_{2.5}$; education level	Non-trauma mortality increased with $PM_{2.5}$ increase; greatest impact among those with less than high school education.
Romieu (2004, 093074)	Children (1 mo. – 1 yr.) in Ciudad Juarez, Mexico, 1997-2001	Days	Total mortality, respiratory mortality; PM_{10}; composite SES index	No significant association between pollutants and total mortality; significant odds ratio for respiratory mortality and PM_{10} for lowest SES; nearly significant association between SES and PM_{10}
Samet et al. (2000, 013132)	Residents (all ages) of 20 US cities, 1987-1994	Days	Non-trauma mortality; PM_{10} (adj for O_3, SO_2, NO_2, CO); % high school graduates, % annual income < $12,675, % annual income > $100,000	No significant association between PM_{10}-related non-trauma mortality and SES variables.
Schwartz (2000, 002470)	Residents (all ages) of 10 US cities, 1986-1993	Days	Non-trauma mortality; PM_{10}; unemployment rate, % below poverty level; % with college degree	No significant difference in the effect of poverty, college degree, or unemployment rate on the influence of PM on mortality, but unemployment rate effect slightly higher.
Tolbert et al. (2000, 001993)	Children (aged < 16) in Atlanta, GA, 1993-1995	Days	Emergency room visits for asthma; PM_{10}; race, Medicaid status, sex	Impact of PM_{10} on asthma emergency room visits was not impacted by any SES variable.
Villeneuve et al. (2003, 055051)	Residents (aged > 65) of Vancouver, Canada, 1986-1999	Days	Non-trauma mortality; TSP, PM_{10}, and $PM_{2.5}$; mean family income	Significantly higher non-trauma mortality as a function of TSP for high and low income.
Wheeler & Ben-Schlomo (2005, 089860)	Respondents to Health Survey of England, 1995-1997	n/a	Decreased lung function, asthma prevalence; air quality index based on PM_{10}, NO_2, SO_2, benzene; social class, sex	In urban areas, lower SES significantly associated with poor air quality; in rural areas, higher SES significantly associated with poor air quality. Lower SES was shown to impact the relationship between PM_{10} and lung function among men but not women.
Wilson et al. (2007, 157149)	Residents (all ages) of Phoenix, AZ, 1995-1997	Days; lag 0-5, 6-day moving avg	Non-trauma mortality, cardiovascular mortality; $PM_{2.5}$, $PM_{10-2.5}$; % < HS diploma, % below poverty level, location within city	The lower SES region of Central Phoenix had higher risk of mortality as a function of $PM_{2.5}$ exposure. Modification of the effect of $PM_{10-2.5}$ on mortality was observed for the higher SES region.
Wojtyniak et al. (2001, 090372)	Residents (aged 0-70 or > 70) of Cracow, Lodz, Poznan, and Wrockrw (Poland), 1990-1996	Days	Non-trauma and cardiovascular mortality; BC; educational level	Non-trauma and cardiovascular mortality was significantly associated with BC for those with less than secondary education.

Reference	Population Studied	Data interval	Metrics Used (health; pollutant; SES variable)	Study Outcome
Wong et al. (2008, 157151)	Residents of 209 tertiary planning units (smallest classification for a town), 1996-2002	Days	Non-trauma and cardiovascular mortality; PM_{10}; social deprivation index	Significant associations between PM_{10} and non-trauma and cardiovascular mortality for medium and high social deprivation index.
Zanobetti et al. (2000, 011979)	Residents of 10 US cities, 1985-1994	Days	Respiratory and cardiovascular hospital admissions; PM_{10}; % poverty, % non-white	No significant effect of SES factors on relationship between hospital admissions and PM_{10}.
Zanobetti et al. (2000, 012187)	Medicare recipients in Cook County, IL, 1985-1994	Days	Respiratory and cardiovascular hospital admissions; PM_{10}; race, sex	No significant effect of SES factors on relationship between hospital admissions and PM_{10}.
Zanobetti & Schwartz (2000, 010198)	Residents (all ages) of Chicago, Detroit, Minneapolis-St. Paul, and Pittsburgh, 1986-1993	Days	Non-trauma mortality; PM_{10} (excluding days when concentrations exceeded 150 $\mu g/m^3$); education below or above high school	Higher but non-significant % increase in non-trauma mortality with 10 $\mu g/m^3$ increase in PM_{10} for people with less than high school education.
Zeka et al. (2006, 088749)	Residents (all ages) of 20 US cities, 1989-2000	Days	Non-trauma mortality, respiratory mortality, cardiac mortality, mortality from infarction, mortality from stroke; PM_{10}; educational level	No significant relationship between increased mortality (any type) with 10 $\mu g/m^3$ increase in PM_{10} for any SES factors.

Some studies measured constituents other than PM; those metrics and results are not reported here.

Source: Adapted from Laurent et al. (2008, 156672) and O'Neill et al. (2003, 090310).

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Annex B. Dosimetry

B.1. Ultrafine Disposition

Table B-1. Ultrafine disposition in humans.

Reference	Study Group	Aerosol	Study Protocol	Observations
Mills et al.(2006, 088770)	Healthy nonsmokers (5 M, 5 F; 21-24 yrs)	Carbon - 99mTc 108 nm CMD (σ_g = 2.2) Technegas Generator	Lung activity in the lung was measured at 0, 1, and 6 h post aerosol inhalation.	On avg, lung activity decreased 3.2 ± 0.7% during the first h and 1.2 ± 1.7% over the next 5 h. With 95.6% of the particles in the lungs at 6 h post inhalation and no accumulation of radioactivity detected over the liver or spleen, findings did not support rapid translocation from the lungs into systemic circulation.
Möller et al. (2008)	Healthy nonsmokers (n = 9; 50 ± 11 yrs) Smokers (n = 10; 51 ± 10 yrs) COPD patients (n = 7; 69 ± 10 yrs)	Carbon - 99mTc ~100 nm CMD Technegas Generator	On two separate occasions, subjects inhaled 100 mL aerosol boli to target front depths of 150 and 800 mL into the lungs to target the airways and alveoli, respectively. Retention measured at 10 min, 1.5, 5.5, 24 and 48 h post inhalation. Isotope (99mTc) leaching from particles assessed via filters in saline, blood, and urine. ^{81m}Kr utilized to assess ventilation.	*Shallow airways boli* – Total deposition in airways (shallow boli) similar between groups. Pattern of deposition was significantly more central in the healthy subjects which was thought due to non-uniform ventilation distribution in smokers and COPD patients as visualized by gamma-camera scans. Airway retention after 1.5 h was significantly lower in healthy subjects (89 ± 6%) than smokers (97 ± 3%) or COPD patients (96 ± 6%). At 24 and 48 h, retention significantly remained higher in COPD patients (86 ± 6% and 82 ± 6%) than healthy subjects (75 ± 10% and 70 ± 9%). *Deep alveolar boli* – Total deposition in alveoli (deep boli) significantly greater in smokers (64 ± 11%) and COPD patients (62 ± 5%) than healthy subjects (50 ± 8%). Alveolar retention of particles similar at all times between groups. For example, at 48 h, 97 ± 3% in healthy subject, 96 ± 3% in smokers, and 96 ± 2% in COPD patients. Retention at 24 and 48 correlated with isotope leaching, suggesting that the small amount of clearance primarily reflected the disassociation of 99mTc from the particles with little transport of particles from the lungs.
Wiebert et al. (2006, 156154)	Subjects having varied health status (9M, 6F; 46-74 yrs) 6 healthy 5 asthmatic 4 smokers	Carbon - 99mTc 87 nm CMD (σ_g = 1.7) Technegas Generator	Technegas system was modified to reduce leaching of 99mTc radiolabel from particles. The avg tidal volume during aerosol inhalation was 1.8 L (range 0.8 – 3.3). Activity in chest region measured at 0, 2, 24, 46, and 70 h after inhalation. Leaching assessed in vitro and via urine collection.	Lung function not significantly different between healthy and affected lungs. The aerosol deposition fraction was 41 ± 10%. Lung retention was 99 ± 3%, 99 ± 5%, and 99 ± 10% at 24, 46, and 70 h post inhalation. Cumulative in vitro leaching by 70 h was 2.6 ± 0.96%. Except for radiotracer leaching from particles (1.0 ± 0.6% of initially deposited activity in urine by 24 h), there was not significant clearance from the lungs by 70 h. Individual leaching was not correlated with individual retention.

Note: Hyperlinks to the reference citations throughout this document will take you to the NCEA HERO database (Health and Environmental Research Online) at http://epa.gov/hero. HERO is a database of scientific literature used by U.S. EPA in the process of developing science assessments such as the Integrated Science Assessments (ISA) and the Integrated Risk Information System (IRIS).

Reference	Study Group	Aerosol	Study Protocol	Observations
Wiebert et al. (2006, 157146)	Healthy subjects (4M, 5F; 56 ± 9 yrs) Asthmatics (2M, 3F; 59 ± 6 yrs) Control (1M; 50 yrs)	Carbon - 99mTc 34 nm CMD (σ_g = 1.5) Technegas Generator	Slow deep aerosol inhalations with 10s breath hold. Mean inhalation time of 6 min. Control subject inhaled aerosol with loosely bound radiolabel. Retention scans at 10 min, 60 min, 100 min, and 24 h post inhalation. Leaching assessed in vitro and via collection of blood and urine.	Avg deposition fraction of 60 ± 17% which was correlated with tidal volume during aerosol inhalation (p = 0.01). Activity excreted in urine over 24-h post inhalation was 51% in the control subject (high 99mTc disassociation) and 3.6 ± 0.9% of deposited activity. In the blood of the control subject, activity was 30%, 31%, and 5% of the deposited activity at 20 min, 80 min, and 24-h (respectively), whereas it was only 0.9 ± 0.6%, 1.1 ± 0.4%, and 1.5 ± 0.5% the other 13 subjects at these times. Lung retention in the control subject was 30% at 1-h and 18% at 24 h. In the remainder of subjects, lung retention was approximately 100% through 24 h.

Table B- 2. Ultrafine disposition in animals.

Reference	Study Group	Aerosol	Study Protocol	Observations
Bermudez et al. (2004, 189730)	Fischer 344 rats, females (6 wks) B3C3F1 mice, females (6 wks) Hamsters, females (6 wks)	TiO_2: 1.29–1.44 μm MMAD (σg = 2.46-3.65), 21 nm primary particles	Animals exposed 6 h/day, 5 day/wk, for 13 weeks to 0.5, 2 and 10 mg/m^3. Control animals exposed to filtered air. Animals sacrificed at 0, 4, 13, 26, and 56 (49 for hamsters) postexposure. Groups of 25 animals per species and time point.	TiO_2 pulmonary retention half-times for the low-, mid-, and high-exposure groups, respectively: 63, 132, and 365 days in rats; 48, 40, and 319 days in mice; and 33, 37, and 39 days in hamsters. Burden of TiO_2 in lymph nodes increase with time postexposure in mid- and high-dosed rats; in high-dosed mice; but was unaffected in hamsters at any time or dosage group. In high-exposure groups of mice, epithelial permeability remained elevated ($\sim$2× control groups) out to 52 weeks without signs of recovery. Epithelial permeability was 3-4× control in high exposed rats through 4 weeks post exposure, but approached control by 13 weeks. Epithelial permeability was unaffected in all groups of hamsters.
Chen et al. (2006, 087947)	Sprague-Dawley male rats (220 ± 20 g)	Polystyrene 125I radiolabel Ultrafine: 56.4 nm Fine: 202 nm	Intratracheal instillation of particles in healthy rats or those pretreated with LPS (12 h before particle instillation). Healthy rats sacrificed between 0.5-2 h and at 24 or 48 h post-instillation. LPS treated rats were sacrificed 0.5-2 h post-instillation.	In healthy rats, there were no marked differences in lung retention or systemic distribution between the ultrafine and fine particles. Results for healthy animals focused on ultrafine particles which were primarily retained in lungs (72 ± 10% at 0.5-2 h; 65 ± 1% at 1 day; 62 ± 5% at 5 days). Initially, there was rapid particle movement into the blood (2 ± 1% at 0.5-2 h; 0.1 ± 0.1% at 5 days) and liver (3 ± 2% at 0.5-2 h; 1 ± 0.1% at 5 days). At 1 day post-instillation, about 13% of the particles where in the urine or feces. Following LPS treatment, ultrafine accessed the blood (5 vs. 2%) and liver (11 vs. 4%) to a significantly greater extent than fine particles.
Geiser et al. (2005, 087362) Also included in vitro study	Wistar rats 20 adult males (250 ± 10 g)	TiO_2 (22 nm CMD, 1.7 σg) Spark generated 0.11 mg/m^3 7.3 × 106 particles/cm^3	Rats exposed 1-h via endotracheal tube while anesthetized and ventilated at constant rate. Lungs fixed at 1 or 24-h postexposure.	Distributions of particles among lung compartments followed the volume distribution of compartments and did not differ significantly between 1 and 24-h post-inhalation. On avg, 79.3 ± 7.6% of particles were on the luminal side of the airway surfaces, 4.6 ± 2.6% in epithelial or endothelial cells, 4.8 ± 4.5% in connective tissues, and 11.3 ± 3.9% within capillaries. Particles within cells were not membrane-bound.
Kapp et al. (2004, 156624)	Charles River rats 5 young adult male (250 ± 10 g)	TiO_2 (22 nm CMD, 1.7 σg) Spark generated	--Rats exposed 1-h via endotracheal tube while anesthetized and ventilated at constant rate. Lungs fixed immediately postexposure.	Of particles in tissues, 72% were aggregates of 2 or more particles; 93% of aggregates were in round or oval shape aggregates, 7% were needle-like. The size distribution of particles in lung tissues (29 nm CMD, 1.7 σg) was remarkably similar to the aerosol; the small discrepancy may have been due to differences sizing techniques. A large 350 nm aggregate was found in a type II pneumocyte, a 37 nm particle in a capillary close to the endothelial cells, and a 106 nm particle within the surface-lining layer close to the alveolar epithelium.

Table B- 3. In vitro studies of ultrafine disposition.

Reference	Animal	Particles	Study Protocol	Observations
Edetsberger et al. (2005, 155759)	Human cervix carcinoma cells (HeLa cells)	Polystyrene spheres (0.020 μm)	Cells incubated with polystyrene particles having negative surface charges. Cell cultures were naïve or treated with Genistein or Cytochalasin B (CytB) prior to particle application. Genistein inhibits endocytotic processes, expecially caveolae internalization. CytB inhibits actin polymerization and phagocytosis.	Particles translocated into cells by first measurement (~ 1 min after particle application) independent of treatment group. In naïve cells, agglomerates of 88-117 nm were seen by 15-20 minutes and of 253-675 nm by 50-60 minutes after particle application. Intracellular aggregates thought to be result from particle incorporation into endosomes or similar structures. In treated cells, only a small number of agglomerates (161-308 nm) were found and only by 50-60 minutes. At 50-60 minutes, 90% and 98% of particles were in the 20-40 nm range in naïve and treated cells, respectively. Particles did not translocate into dead cells, rather they attached to outside of the cell membrane.
Geiser et al. (2005, 087362) *Also included inhalation study*	Porcine lung macrophages (10^6 cell/mLuman red blood cells (RBC; 8 × 10^6 cells/mL)	Fluorescent polystyrene spheres (0.078, 0.2, and 1 μm) Gold shheres (0.025 μm)	Cells cultured for 4 h with each sized polystyrene spheres. RBC were employed as a model of nonphagocytic cells. Some macrophages cultures were treated with cytochalasin D (cytD) to inhibit phagocytosis. In addition, RBC were also cultured with gold particles.	Of the non-cytD treated macrophages, 77 ± 15%, 21 ± 11%, and 56 ± 30% contained 0.078, 0.2, and 1 μm particles, respectively. CytD treatment of macrophages effectively blocked the phagocytosis of 1 μm particles, but did not alter the uptake of the 0.078 and 0.2 μm particles. Human RBC were found to contain 0.078 and 0.2 μm polystyrene spheres as well as the 0.025 μm gold particles, which were not membrane bound. In contrast, the RBC did not contain the larger 1 μm polystyrene spheres. Results suggest that ultrafine and fine (0.078 and 0.2 μm diameter) particles cross cellular membranes by a non-endocytic (i.e. not involving vesicle formation) mechanisms such as adhesive interactions and diffusion.
Geys et al. (2006, 155789)	Human alveolar (A549) and bronchial (Calu-3) epithelial cellsRat primary type II pneumocytes	Amine- and carboxyl-modified fluorescent polystyrene (46 nm)	Cells cultured in clear polyester transwells with 0.4 or 3 μm pores. Monolayer considered "tight" when < 1% sodium fluorescein moved from apical to basolateral compartment. Particle translocation assessed in transwells with and without cells. Cells incubated with particles for 14-16 h to assess translocation from apical to basolateral compartment.	Without cells, 13.5% of carboxyl-modified particles passed through the 0.4 μm pores (n = 7) and 67.5% through 3 μm pores (n = 3). Movement of the amine-modified particles was 4.2% through 0.4 μm pores (n = 7) and 52.7% through 3 μm pores (n = 3). The integrity of the monolayer was insufficient for translocation studies using the A549 cells (0.4 and 4 μm pore size) and rat pneumocytes (0.3 μm pore). Using 0.4 μm pores, there was no detectable translocation through either Calu-3 or rat pneumocyte monolayers. Using 3 μm pores, ~ 6% of both particle types passed through the Calu-3 monolayer; however, results were highly variable with no translocation in 2 (of 5) and 3 (of 6) trials with carboxyl- or amine-modified particles, respectively.

B.2. Olfactory Translocation

Table B-4. Olfactory particle translocation.

Reference	Study Group	Aerosol	Study Protocol	Observations
DeLorenzo (1970, 156391)	Squirrel monkeys young males (1 kg)	Silver-coated colloidal gold (50 nm)	Intranasal instillation of 1 mL particle suspension. Animals sacrificed at 0.25, 0.5, 1, and 24-h after instillation.	Rapid movement (30-60 min) into olfactory bulbs. Within 30 min of being placed on nasal mucosa, particle aggregates were seen in axoplasm of the fila olfactoria Within 1 h, particles were in olfactory glomerulus. Particles in the olfactory bulb were located preferentially in mitochondria and not free in the cytoplasm.
Dorman et al. (2001, 055433)	Crl: CD rats Males (6 wks old)	Soluble and insoluble Mn particle types; MMAD = 1.3-2.1 μm; GSD < 2	Whole body exposure (6 h/day, 14 consecutive days) to 0, 0.03, 0.3, and 3 mg Mn/m^3. Tissues analyzed in six animals per concentration exposed to soluble ($MnSO_4$) or insoluble (Mn_3O_4) aerosols.	Increased Mn levels in olfactory bulb observed following $MnSO_4$ of ≥ 0.3 mg Mn/m^3 and following Mn_3O_4 of 3 mg Mn/m^3. At 3 mg Mn/m^3, Mn levels were significantly greater in olfactory bulb (1.4-fold) and striatum (2.7-fold) following soluble MnO_4 than insoluble Mn_3O_4. Mn levels in the cerebellum were unaffected following all exposures.
Dorman et al. (2004, 155752)	Crl: CD rats Males (6 wks old)	Soluble and insoluble Mn particle types; MMAD = 1.5-2 μm; GSD = 1.4-1.6	Whole body exposure (6 h/day, 5 days/wk, 13 wks) to $MnSO_4$ at 0, 0.01, 0.1, and 0.5 mg Mn/m^3. Compared to Mn phosphate (as hureaulite) exposure of 0.1 mg Mn/m^3. Brain Mn levels assessed immediately following 90 days of exposure or 45 days postexposure.	Relative to air, the insoluble hureaulite was significantly increased at 90 days of exposure in the olfactory bulb, but not striatum or cerebellum. The soluble Mn phosphate showed a dose dependent increase in olfactory bulb Mn levels at 90 days. At 0.1 mg Mn/m^3, Mn levels following Mn phosphate were significantly increased in the olfactory bulb and striatum relative to hureaulite and air exposures. At 45 days postexposure, relative to air, olfactory bulb Mn levels only remained increased Mn phosphate group at 0.5 mg Mn/m^3.
Elder et al. (2006, 089253)	Fisher 344 rats Males (200-250 g)	Mn oxide (~30 nm equivalent sphere with 3-8 nm primary particles) Spark generated 0.5 mg/m^3 18×10^6 particles/cm^3	Whole body inhalation exposure to either filtered air or Mn oxide for 12 days (6 h/day, 5 days/wk) with both nares open or Mn oxide for 2 days (6 h/day) with right nostril blocked. Intranasal instillation in left nostril of Mn oxide particles or soluble $MnCl_2$ suspended in 30 µL saline. Analyzed Mn in the lung, liver, olfactory bulb, and other brain regions.	After 12 day exposure via both nostrils, Mn in the olfactory bulb increased 3.5-fold, whereas in the lung Mn concentrations doubled; there were also increases in the striatum, frontal cortex, and cerebellum. After the 2 days exposure with the right nostril blocked, Mn accumulated in the mainly in the left olfactory bulb (~2.4-fold increase) in to a lesser extent in the right olfactory bulb (1.2-fold increase). At 24-h post instillation, the left olfactory bulb contained similar amounts of the poorly soluble Mn oxide (8.2 ± 0.7%) and soluble $MnCl_2$ (8.2 ± 3.6%) as a percent of the amount instilled.
Oberdörster et al. (2004, 055639)	Fisher 344 rats Males (14 wks; 284 ± 9 g)	^{13}C (36 nm CMD, 1.7 σ_g) Spark generated	Rats (n = 12, 3 per time point) exposed to 160 µg/m^3 for 6 h in whole-body chamber and sacrificed at 1, 3, 5, and 7 day postexposure. Lung, olfactory bulb, cerebrum, and cerebellum removed for ^{13}C analysis. Tissue ^{13}C-levels were determined by isotope ratio mass spectroscopy and background corrected for ^{13}C levels in unexposed controls (n = 3).	At 1 day postexposure, the lungs of rats exposed to ultrafine ^{13}C particles contained 1.34 ± 0.22 µg of ^{13}C (1.39 µg/g-lung) following background corrected. By 7 days postexposure, the ^{13}C concentration had decreased to 0.59 µg/g-lung. There was a significant and persistent increase in ^{13}C in the olfactory bulb of 0.35 µg/g on day 1, which increased to 0.43 µg/g by day 7. Day 1 concentrations of ^{13}C in the cerebrum and cerebellum were also significantly increased but the increase was inconsistent, possibly reflecting translocation of particles from the blood across the blood–brain barrier into brain regions.
Persson et al. (2003, 051846)	Sprague-Dawley male rats (150 g) Freshwater Pike female (3 kg)	$^{65}ZnCl_2$ dissolved in 0.1 M HCl	Rats: intransal (0.03 μg Zn in 10 μL) or intraperitoneally (0.03 μg Zn in 100 μL); autoradiography and γ spec at 1 day or 1, 3, or 6 weeks postexposure. Pike: instilled (0.12 μg Zn in 10 μL) in right or both olfactory chambers, assayed 2 weeks postexposure	Zn uptake in olfactory epithelium and transport along olfactory neurons to olfactory bulb. Zn continued into interior of olfactory bulb and in rat went into anterior olfactory cortex. Zn found bound to both cellular constituents and cytosolic components. Some Zn bound to metallothionein in olfactory mucosa and olfactory bulb.

Reference	Study Group	Aerosol	Study Protocol	Observations
Wang et al. (2007, 156147)	CD-1 (ICR) mice	Rutile TiO_2 21 and 80 nm Anatase TiO_2 155 nm	Twenty mice (n = 5 per group) exposed 0 or 0.01 g-TiO_2 per mL DI. Instilled 25 μL each day for 5 days, then inhaled 10 μL every other day. Mice sacrificed after one month.	Rutile particles were observed to be column/fiber shaped, whereas anatase was octahedral. TiO_2 particles taken up by olfactory bulb via the olfactory nerve layer, olfactory ventricle, and granular cell layer of the olfactory bulb. Fine TiO_2 showed greater entry into the olfactory bulb presumably due to aggregation of smaller rutile particles that was not seen for the fine anatase particles.
Yu et al. (2003, 156171)	Sprague-Dawley male rats, 6 wks old (218 ± 10 g)	Stainless steel welding-fume < 0.5 μm	Whole body exposure 2 h/day for 1, 15, 30, or 60 days Low: 64 ± 4 mg/m^3 (1.6 mg/m^3 Mn) High: 107 ± 6 mg/m^3 (3.5 mg/m^3 Mn)	Significant increases in cerebellum Mn at 15 – 30 days of exposure. Slight increases in Mn in substantia nigra, basal ganglia, temporal cortex, and frontal cortex after 60 days. Significant increase at 30 days in basal ganglia at low dose. Authors suggested that pharmacokinetics and distribution of welding fume Mn differs from pure Mn.

B.3. Clearance and Age

Table B-5. Studies of respiratory tract mucosal and macrophage clearance as a function.

Reference	Animal	Particles	Study Protocol	Observed Effect(s)
NASAL AND TRACHEAL CLEARANCE				
Ho et al. (2001, 156549)	Human, males and females	Not applicable	Ninety subjects (47 M, 43 F; 52 ± 23 yrs) between 11 and 90 years of age were recruited to measure nasal saccharine clearance and ciliary beat frequency.	Ciliary beat frequency (n = 90; r = -0.48, p < 0.0001) and nasal mucociliary clearance time (n = 43; r = 0.64, $p < 0.001$) were correlated with subject age. Nasal clearance times were significantly ($p < 0.001$) faster in individuals under 40 years of age (9.3 ± 5.2 min) versus older subjects (15.4 ± 5.0 minutes). Results similar between males and females.
Goodman et al. (1978, 071130)	Humans, males and females	Radiolabed Teflon disks (1 mm diameter, 0.8 mm thick)	Tracheal mucus velocity following delivery via bronchoscope to the tracheal mucosa. Ten young (2 M, 8 F; 23 ± 3 yrs) and ten elderly (2 M, 5 F; 63 ± 5 yrs) nonsmokers served as control subjects. Measurements were also made in young smokers, ex-smokers, and individuals with chronic bronchitis.	Young nonsmokers had a tracheal mucus velocity of 10.1 ± 3.5 mm/min which was significantly faster than the velocity of 5.8 ± 2.6 observed in the elderly nonsmokers.
Whaley et al. (1987, 156153)	Beagle dogs, males and females	Macroaggregated albumin99mTc labelled	Intratracheal instillation of 10- μl droplet of labelled albumin in saline. Tracheal clearance followed 25 minutes. Longitudinal measure measurements in 5 males and 3 females when young adults (2.8-3 yr), middle-aged (6.7-6.9 yr), and mature (9.6-9.8 yr). Additional 5 females and 3 males comprised immature group (9-10 mo) and 4 males and 4 females used as aged group (13-16 yr).	Tracheal mucus velocity significantly ($p < 0.05$) greater in young (9.7 ± 0.6 [SE] mm/min) and middle-aged (6.9 ± 0.5) groups than in immature (3.6 ± 0.4), mature (3.5 ± 0.8), and aged (2.9 ± 0.5) dogs.

Reference	Animal	Particles	Study Protocol	Observed Effect(s)
Yeates et al. (1981, 095391)	Humans, males and females	Radioaerosols 99mTc labelled	Tracheal mucus velocities compiled for 74 healthy non-smoking subjects (60 M, 14 F; 10-65 yrs, mean 30 yrs) from prior studies. Forty-two (32 M, 10 F) inhaled albumin in saline droplets (6.2-6.5 μm MMAD), Yeates et al. (1975); twenty-two (21 M, 1 F) inhaled iron oxide (4.2 μm MMAD), Yeates et al. (1981b); and ten (7 M, 3 F) inhaled monodisperse iron oxide aerosol (7.5 μm MMAD), Leikauf et al. (1981). Inhalations were via a mouthpiece with an inspiratory flow of $\sim$1 liter/sec.	A lognormal distribution of tracheal mucus velocities was reported. Age did not appear to affect velocities, e.g., 4.7 ± 2.5 mm/min in 18-24 yrs olds vs. 4.6 ± 3.2 mm/min in individuals $>$30 yrs of age. However, it should be noted that only 2 subjects were greater than 45 yrs of age and that the data was complied from three studies using differing experimental techniques. Rather similar tracheal mucus velocities in males (4.7 ± 3.0 mm/min) and females (4.9 ± 2.4 mm/min).
BRONCHI AND BRONCHIOLES CLEARANCE				
Puchelle et al. (1979, 006863)	Human, males	7.4 μm MMAD99mTc labelled resin	Mucociliary clearance measured for 1 h post aerosol inhalation in 19 healthy non-smoking males (21-69 yrs of age). Clearance measure on two occasions in 16 individuals.	Negative correlation (r = -0.472, $p < 0.05$) between mucociliary clearance and age. Younger subjects (n = 9; 21-37 yrs) had 1-h clearance of 34 ± 14% which was significantly greater than the 22 ± 8% found in the older subjects (n = 5; $>$54 yrs). Separated by 5.4 wks (on avg), there was a good correlation between repeated clearance measurements (r = 0.65, $p < 0.001$)
Svartengren et al. (2005, 157034)	Humans, males and females	6 μm MMAD111In labelled Teflon	Small airway clearance measured in five age groups (□ 24 yrs, n = 13; 25-29 yrs, n = 8; 30-49 yrs, n = 7; 50-64, n = 9; $>$65 yrs, n = 9) of healthy subjects. Aerosol inhaled via mouthpiece at extremely slow rate of 0.05 L/s. Activity in lungs measured at 1 day, 2 days, and 1, 2, and 3 wks post-exposure. Under the presumption that most large airway clearance was complete by 24 h, retention at 24 h was normalized to 100%.	Large and small airway clearance slowed with increasing age. Clearance correlated with age at all times (r = -0.46 to -0.50, -0.55, -0.66, and -0.70 at 1 day, 2 days, 1 wk, 2 wks, and 3 wks, respectively). Based on linear regression, the clearance from 1 to 21 days post-exposure was 47% in a 20 yr-old versus 23% in an 80 yr-old. Lung function was not a significant predictor of clearance when age considered.
Vastag et al. (1985, 157088)	Humans, males and females	Monodisperseerythrocytes99mTc labelled	Clearance measured for 1-h post-inhalation in eighty healthy (59 M, 21 F; 43 ± 17 yrs) subjects who had never smoked. Smokers and ex-smokers also studied. Aerosol inhalation not described.	Clearance significantly associated with age. Based on linear regression, total mucociliary clearance at 1-h post-exposure was 46% in a 20-yr-old versus 23% in an 80 yr-old. Similar results for males and females.
ALVEOLAR CLEARANCE				
Muhle et al. (1990, 006853)	Fischer 344 rats	3.5 μm MMAD 85Sr labelled polystyrene latex	Control animals compared across several studies. Aerosol inhaled by short-term nose only exposure. Alveolar clearance determined by exponential fit to thoracic activity measured over 75-100 days excluding the first 15 days post-exposure.	Typical alveolar clearance half-time of 45 days in 5-month-old rats compared to 74 days in 23-month-old rats. Statistical significance of findings not proved.

Annex B References

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Annex C. Controlled Human Exposure Studies

Table C-1. Cardiovascular Effects

Study	Pollutant	Exposure	Findings
Reference: Barregard et al. (2006, 091381) **Subjects:** 13 healthy adults **Gender:** 6 M/ 7 F **Age:** 20-56 yrs	Wood smoke **Particle Size:** Session 1: GMD 42 nm; Session 2: GMD 112 nm **Particle Number/Count:** Session 1: 180,000/cm^3; Session 2: 95,000/cm^3 **Concentration:** Session 1: median: 279 $\mu g/m^3$; Session 2: median 243 $\mu g/m^3$	Subjects exposed in two groups for 4 h to filtered air, followed a wk later by a 4-h exposure to wood smoke. Exposures conducted with two 25-min periods of light exercise. Other measured combustion products: Session 1: NO_2 (0.08 ppm), CO (13 ppm), formaldehyde (114 $\mu g/m^3$), acetaldehyde (75 $\mu g/m^3$), benzene (30 $\mu g/m^3$), 1,3-butadiene (6.3 $\mu g/m^3$); Session 2: NO_2 (0.09 ppm), CO (9.1 ppm), formaldehyde (64 $\mu g/m^3$), acetaldehyde (40 $\mu g/m^3$), benzene (20 $\mu g/m^3$), 1,3-butadiene (3.9 $\mu g/m^3$). **Time to analysis**: Immediately following exposure as well as 3 and 20 h post-exposure.	Statistically significant increase in plasma factor VIII 20 h post wood smoke exposure relative to filtered air. The factor VIII/von Willebrand ratio in plasma was increased with wood smoke relative to filtered air at 0, 3, and 20 h post-exposure. Wood smoke exposure increased the urinary excretion of free 8-iso-prostaglandin$_{2\alpha}$ relative to clean air 20 h post-exposure (n = 9). These findings were more pronounced in session 1 than session 2 (similar mass concentration but higher number concentration in session 1).
Reference: Beckett et al. (2005, 156261) **Subjects:** 12 healthy adults **Gender:** 6 M/6 F **Age:** 23-52 yrs	Ultrafine and fine zinc oxide **Particle Size:** UF: < 0.1 μm; Fine: 0.1-1.0 μm **Particle Number/Count:** UF: $4.6 \times 10^7/cm^3$; Fine: $1.9 \times 10^5/cm^3$ **Concentration:** 500 $\mu g/m^3$	Subjects exposed via mouthpiece for 2 h during rest to filtered air, ultrafine, and fine zinc oxide in a randomized crossover study design. Exposures were separated by at least 3 wks. **Time to analysis:** Immediately following exposure and 3, 6, 11, 23, and 24 h after exposure.	Exposure to ultrafine and fine zinc oxide did not affect HRV (time and frequency domain parameters) relative to clean air immediately following exposure, or at 3, 6, 11, and 23 h post-exposure. Exposure did not affect blood pressure through 24 h post-exposure. No effects of exposure to either fine or ultrafine zinc oxide observed on factor VII, von Willebrand factor (vWf), tissue plasminogen activator (t-PA), or fibrinogen. No effect of exposure observed on peripheral blood cell counts or levels of pro-inflammatory cytokines.
Reference: Blomberg et al. (2005, 191991) **Subjects:** 15 older adults (former smokers) with COPD **Age:** 56-72 yrs	DE **Concentration:** 300 $\mu g/m^3$	Subjects exposed for 1 h with intermittent exercise to DE and filtered air in a randomized crossover study design. **Time to analysis:** 6 and 24 h post-exposure.	DE was not observed to affect blood levels of C-reactive protein, fibrinogen, D-Dimer, prothrombin factor 1-2, or von Willebrand factor activity at 6 and 24 h post-exposure.

Note: Hyperlinks to the reference citations throughout this document will take you to the NCEA HERO database (Health and Environmental Research Online) at http://epa.gov/hero. HERO is a database of scientific literature used by U.S. EPA in the process of developing science assessments such as the Integrated Science Assessments (ISA) and the Integrated Risk Information System (IRIS).

Study	Pollutant	Exposure	Findings
Reference: Brauner et al. (2007, 188507) **Subjects:** 29 healthy adults **Gender:** 20 M/9 F **Age:** 20-40 yrs	Urban traffic particles **Particle Number/Count:** 6-700nm: 10,067/cm^3 **Concentration:** $PM_{2.5}$: 9.7 $\mu g/m^3$; $PM_{2.5-10}$: 12.6 $\mu g/m^3$	Subjects exposed to urban traffic particles and filtered air for 24 h with and without two 90-min periods of light exercise in a randomized crossover study design. Concentrations of NO_X and NO were low and did not differ between filtered and unfiltered exposures. CO concentrations were higher with filtered air (0.35 and 0.41 ppm), while ozone concentrations were lower with filtered air (12.08 and 4.29 ppb). **Time to analysis:** 6 and 24 h after the start of exposure.	An increase in DNA strand breaks and formamidopyrimidine-DNA glycosylase sites in peripheral blood mononuclear cells were observed after 6 and 24 h of exposure to urban particulates. The particle concentration at the 57nm mode was shown to be the major contributor to these effects.
Reference: Brauner et al. (2008, 156293) **Subjects:** 42 healthy older adults (21 couples) **Age:** 60-75 yrs	Indoor air particles **Particle Number/Count:** 10-700 nm: 10,016/cm^3 **Concentration:** Coarse: 9.4 $\mu g/m^3$; Fine: 12.6 $\mu g/m^3$	Exposures consisted of two 48 h periods in the home of each subject with or without the use of a HEPA filter (randomized crossover design). HEPA filters reduced coarse concentration from 9.4 to 4.6 $\mu g/m^3$, and fine concentration from 12.6 to 4.7 $\mu g/m^3$. Concentrations of NO_2 did not differ between the 2 sessions (20 ppb). **Time to analysis:** After the completion of each 48 h session.	The use of HEPA filters significantly improved microvascular function ($p = 0.04$) after 48 h (reactive hyperemia-peripheral arterial tonometry). Microvascular function was assessed using a scoring system representing the extent of reactive hyperemia. The reduction in PM concentration through the use of HEPA filters did not significantly affect blood pressure following the 48-h exposures. Lowering PM concentration did not significantly affect inflammatory response markers in peripheral venous blood (IL-6, TNF-□, C-reactive protein, plasma amyloid A).
Reference: Brauner et al. (2008, 191966) **Subjects:** 29 healthy adults **Gender:** 20 M, 9 F **Age:** M avg 27 yrs, F avg 26 yrs	Urban traffic particles **Particle Number/Count:** 6-700 nm: 10,067/cm^3 **Concentration:** $PM_{2.5}$: 9.7 $\mu g/m^3$; $PM_{2.5-10}$: 12.6 $\mu g/m^3$	Subjects exposed to urban traffic particles and filtered air for 24 h with and without two 90-min periods of light exercise in a randomized crossover study design. Concentrations of NO_X and NO were low and did not differ between filtered and unfiltered exposures. CO concentrations were higher with filtered air (0.35 and 0.41 ppm), while ozone concentrations were lower with filtered air (12.08 and 4.29 ppb). **Time to analysis:** 6 and 24 h after the start of exposure.	Exposure to urban traffic particles was not observed to affect microvascular function (digital peripheral artery tone) at 6 or 24 h after the start of exposure. No difference in various blood markers of coagulation, inflammation, or protein oxidation (e.g., fibrinogen, platelet count, CRP, IL-6, TNF- □) were demonstrated between particle and filtered air exposure.
Reference: Carlsten et al. (2007, 155714) **Subjects:** 13 healthy adults **Gender:** 11 M/2 F **Age:** 20-42 yrs	DE 2002 Cummins B-series diesel engine (6BT5.9G6, 5.9 L) operating at load **Concentration:** Fine PM: 100, 200 $\mu g/m^3$	Subjects exposed for 2 h at rest to filtered air and each of the two DEPs concentrations in a randomized crossover study design. Exposures were separated by at least 2 wks. Other diesel emissions measured: NO_2 (10-35 ppb), CO (0.7-1.8 ppm). **Time to analysis:** 3, 6, and 22 h after the start of exposure.	No statistically significant changes in plasminogen activator inhibitor-1 (PAI-1), vWf, D-dimer, or platelet count observed 3, 6, or 22 h following exposure to DE relative to filtered air. Non-statistically significant increases in D-dimer, vWf, and platelet count was observed at 6 h following the start of exposure (4 h post-exposure). No diesel-induced increase in C-reative protein observed in relative to filtered air in peripheral venous blood at 1 or 20 h post-exposure.
Reference: Carlsten et al. (2008, 156323) **Subjects:** 16 adults with metabolic syndrome **Gender:** 10 M/6 F **Age:** 25-48 yrs	DE 2002 Cummins B-series diesel engine (6BT5.9G6, 5.9 L) **Concentration:** Fine PM: 100, 200 $\mu g/m^3$	Subjects exposed for 2 h at rest to filtered air and each of the two DE particle concentrations in a randomized crossover study design. Exposures were separated by at least 2 wks. Other diesel emissions measured: NO_2 (30 ppb), NO (1.69 ppm), CO (0.65 ppm). **Time to analysis:** 3, 7, and 22 h after the start of exposure.	At 5 h after the end of diesel exposure (fine particulate concentration 200 $\mu g/m^3$), the authors observed a significant decrease in vWf in peripheral venous blood. No other changes in thrombotic markers (vWf, D-dimer, PAI-1) were observed at either concentration between 1 and 20 h post-exposure.

Study	Pollutant	Exposure	Findings
Reference: Danielsen et al. (2008, 156382) **Subjects:** 13 healthy adults **Gender:** 6 M/ 7 F **Age:** 20-56 yrs	Wood smoke **Particle Size:** Session 1: GMD 42 nm; Session 2: GMD 112 nm **Particle Number/Count:** Session 1: 180,000/cm^3; Session 2: 95,000/cm^3 **Concentration:** Session 1: median: 279 $\mu g/m^3$; Session 2: median 243 $\mu g/m^3$	Subjects exposed in two groups for 4 h to filtered air, followed a wk later by a 4-h exposure to wood smoke. Exposures conducted with two 25-min periods of light exercise. Other measured combustion products: Session 1: NO_2 (0.08 ppm), CO (13 ppm), formaldehyde (114 $\mu g/m^3$), acetaldehyde (75 $\mu g/m^3$), benzene (30 $\mu g/m^3$), 1,3-butadiene (6.3 $\mu g/m^3$); Session 2: NO_2 (0.09 ppm), CO (9.1 ppm), formaldehyde (64 $\mu g/m^3$), acetaldehyde (40 $\mu g/m^3$), benzene (20 $\mu g/m^3$), 1,3-butadiene (3.9 $\mu g/m^3$). **Time to analysis:** 3 and 20 h post-exposure.	Exposure to wood smoke increased the mRNA levels of *hOGG1* in PBMCs relative to filtered air 20 h after exposure. DNA strand breaks were shown to decrease in PBMCs 20 h after wood smoke exposure.
Reference: Devlin et al. (2003, 087348) **Subjects:** 10 healthy older adults **Gender:** 7 M/ 3 F **Age:** Avg 66.9 yrs	Fine CAPs (Chapel Hill, NC) **Concentration:** Mean: 40.5 $\mu g/m^3$, Range: 21.2-80.3 $\mu g/m^3$	Exposures conducted for 2 h at rest to filtered air and CAPs in a randomized crossover study design. **Time to analysis:** Immediately following exposure and 24 h post-exposure.	CAPs exposure resulted in statistically significant reductions ($p < 0.05$) in time domain (PNN50) and frequency domain (HF power) parameters relative to clean air immediately following exposure. These relative decreases were still apparent 24 h after exposure ($p < 0.08$).
Reference: Fakhri et al. (2009, 191914) **Subjects:** 50 adults (40 healthy, 10 asthmatic) **Gender:** 24 M/26 F **Age:** 19-48 yrs	Fine CAPs (Toronto) **Concentration:** 127 ± 62 $\mu g/m^3$ with and without co-exposure to ozone (114 ± ppb)	Exposures conducted through a facemask which covered the subject's nose and mouth. Subjects were exposed to CAPs, ozone, CAPs + ozone and filtered air for 2 h at rest in a randomized crossover study design. **Time to analysis:** Every 30 min during exposure, with the final measurement made immediately prior to the end of the exposure.	Exposure to CAPs or ozone, alone or in combination, resulted in no significant changes in HRV or blood pressure relative to filtered air. However, a negative concentration response relationship was reported between CAPs concentration with co-exposure to ozone and SDNN, rMSSD, HF power and LF power (statistically significant for LF power). Diastolic blood pressure was observed to increase with exposure to CAPs + ozone, but not with either pollutant alone. There was no difference in response between asthmatics and healthy subjects.
Reference: Frampton et al. (2006, 088665) **Subjects:** 16 asthmatic adults, 40 healthy adults **Gender:** Asthmatics: 8 M/8 F, Healthy: 20 M/20 F **Age:** 18-40 yrs	Ultrafine elemental carbon **Particle Size:** CMD ~25 nm **Particle Number/Count:** 10 $\mu g/m^3$: ~2.0 × 10^6/cm^3; 25 $\mu g/m^3$: ~7.0 × 106/cm^3; 50 $\mu g/m^3$: ~10.8 × 106/cm^3 **Concentration:** 10, 25, and 50 $\mu g/m^3$	Study conducted using a randomized crossover design with 2-h exposures. Asthmatics (n = 16) exposed to filtered air and 10 $\mu g/m^3$. 12 healthy adults exposed to filtered air and 10 $\mu g/m^3$ at rest; 12 healthy adults exposed to filtered air, 10 and 25 $\mu g/m^3$ with intermittent exercise; 16 healthy adults exposed to filtered air and 50 $\mu g/m^3$ with intermittent exercise. Exposures were conducted via mouthpiece. **Time to analysis:** Immediately following exposure as well as 3.5, 21, and 45 h post-exposure.	No effect of ultrafine particle exposure on leukocyte counts or leukocyte expression of adhesion molecules observed in healthy subjects exposed at rest to 10 $\mu g/m^3$. Among healthy adults exposed to ultrafine carbon during exercise, monocyte expression of adhesion molecules CD54 and CD18 decreased relative to filtered air immediately following exposure. An ultrafine particle-induced decrease in PMN expression of CD18 was also observed 0-21 h post-exposure. Expression of CD11b on monocytes and eosinophils was reduced following exposure to ultrafine particles in exercising asthmatics 0-21 h post-exposure. A decrease in total leukocyte count was observed following ultrafine particle exposure in exercising healthy and asthmatic subjects.

Study	Pollutant	Exposure	Findings
Reference: Gong et al. (2004, 087964) **Subjects:** 13 older adults with COPD, 6 healthy older adults **Gender:** COPD: 5 M/ 8 F, Healthy: 2 M/4 F **Age:** COPD: avg 68 yrs, Healthy: avg 73 yrs	Fine CAPs (Los Angeles) **Particle Size:** 85% of mass between 0.1 and 2.5 μm **Concentration:** Mean: 194 $\mu g/m^3$, Range: 135-229 $\mu g/m^3$	Exposures to CAPs and filtered air (randomized crossoveronducted for 2 h with intermittent light exercise (four 15-min periods). Exposures were separated by at least 2 wks. **Time to analysis:** Immediately following exposure as well as 4 and 22 h post-exposure.	SDNN shown to decrease following CAPs exposure relative to filtered air in healthy older adults (4-22 h post-exposure). No CAPs-induced changes in HRV were observed in older adults with COPD. Ectopic heart beats were observed to increase slightly with CAPs relative to filtered air among healthy subjects, but decreased among subjects with COPD. Exposure to CAPs did not affect platelet or white blood cell count, or levels of fibrinogen, vWF, or factor VII.
Reference: Gong et al. (2004, 055628) **Subjects:** 12 adult asthmatics, 4 healthy adults **Gender:** Asthmatics: 4 M/8 F, Healthy: 2 M/2 F **Age:** Asthmatics: avg 38 yrs, Healthy: avg 32 yrs	Coarse CAPs (Los Angeles) **Particle Size:** 80% of mass between 2.5 and 10 μm, 20% of mass < 2.5 μm **Concentration:** Mean: 157 $\mu g/m^3$, Range: 56-218 $\mu g/m^3$	Exposures to CAPs and filtered air (randomized crossoveronducted for 2 h with intermittent light exercise (four 15-min periods). Exposures were separated by at least 2 wks. **Time to analysis:** Immediately following exposure as well as 4 and 22 h post-exposure.	SDNN shown to decrease following CAPs exposure relative to filtered air in healthy adults (4-22 h post-exposure). Decrease in PNN50 also observed in healthy adults at 4 h post-exposure. No CAPs-induced decreases in HRV demonstrated in asthmatics.
Reference: Gong et al. (2008, 156483) **Subjects:** 14 adult asthmatics, 17 healthy adults **Gender:** Asthmatics: 9 M/5 F, Healthy: 5 M/12 F **Age:** Asthmatics: 34 ± 12 yrs, Healthy: 24 ± 8 yrs	Ultrafine CAPs (Los Angeles) **Particle Number/Count:** 145,000/cm^3, Range 39,000-312,000/cm^3 **Concentration:** Mean-100 $\mu g/m^3$, Range-13-277 $\mu g/m^3$	Subjects exposed for 2 h during intermittent exercise (15-min periods) to both CAPs and filtered air in random order. The first 7 subjects underwent whole body exposure, while the remaining subjects were exposed through a facemask. Facemask exposures had higher particle counts but lower particle mass than whole body exposures. Exposures were separated by at least 2 wks. **Time to analysis:** Immediately following exposure as well as 4 and 22 h post-exposure.	Relative to filtered air, exposure to ultrafine CAPs resulted in a transient decrease in LF power 4 h post-exposure. This effect of CAPs on HRV was not influenced by health status. CAPs exposure was not observed to affect any other measures of HRV, blood pressure, or blood markers of inflammation or coagulation. There were no differences in response observed between facemask and whole body exposures.
Reference: Graff et al. (2009, 191981) **Subjects:** 14 healthy adults **Gender:** 8 M/6 F **Age:** 20-34 yrs	Coarse CAPs (Chapel Hill, NC) **Concentration:** 89 ± 49.5 $\mu g/m^3$ (estimated inhaled dose ◘ 67% of measured particle mass)	Subjects exposed for 2 h with intermittent exercise (15-min periods) to coarse CAPs and filtered air in a randomized crossover design. Exposures were separated by at least 2 mos. **Time to analysis:** 0-1 and 20 h post-exposure.	At 20 h post-exposure, tPA was observed to decrease by 32.9% from baseline (pre-exposure) per 10 $\mu g/m^3$ increase in CAPs concentration ($p = 0.01$). D-dimer concentration decreased 11.3% per 10 $\mu g/m^3$, a change of marginal statistical significance ($p = 0.07$). No other coarse CAPs-induced changes in blood biomarkers of coagulation (e.g., vWF, factor VII, plasminogen, fibrinogen, or PAI-1) or inflammation (e.g., CRP) were observed. At 20 h post-exposure, overall HRV (SDNN) was shown to decrease by 14.4% relative to pre-exposure measurements per 10 $\mu g/m^3$ increase in CAPs concentration. No other changes in HRV were observed following exposure to coarse CAPs.

Study	Pollutant	Exposure	Findings
Reference: Huang et al. (2003, 087377) **Subjects:** 38 healthy adults **Gender:** 36 M/2 F **Age:** Avg 26.2 ± 0.7 yrs	Fine CAPs (Chapel Hill, NC) **Concentration:** 23.1-311.1 μg/m^3	Subjects exposed to CAPs (n = 30) or filtered air (n = 8) for 2 h with intermittent exercise (subjects did not serve as their own controls). Component data of CAPs was available for 37 of the 38 subjects. **Time to analysis:** 18 h after exposure.	The increase in blood fibrinogen following exposure to fine CAPs reported by Ghio et al. (2000, 012140) was shown to be associated with copper, zinc, and vanadium content in the CAPs.
Reference: Larsson et al. (2007, 189320) **Subjects:** 16 healthy adults **Gender:** 10 M/6 F **Age:** 19-59 yrs	Traffic particles (road tunnel) **Particle Size:** $PM_{2.5}$, PM_{10}; $PM_{2.5}$ mass constituted ~36% of PM_{10} mass **Particle Number/Count:** 20-1,000 nm: 1.1 × 105/cm^3, < 100 nm: 0.85 × 105/cm^3 **Concentration:** $PM_{2.5}$-46-81 μg/m^3; PM_{10}-130-206 μg/m^3	Exposures were conducted for 2 h with intermittent exercise in a room adjacent to a busy road tunnel. Study used a randomized crossover design with each subject also exposed to normal air (control). Exposures were separated by 3-10 wks. No exposures to filtered air were conducted. Other traffic emissions measured: NO (874 μg/m^3), NO_2 (230 μg/m^3), CO (5.8 μg/m^3 reported, likely 5.8 mg/m^3). **Time to analysis:** 14 h post-exposure.	No change in plasma levels of fibrinogen or PAI-1 observed 14 h post-exposure.
Reference: Lucking et al. (2008, 191993) **Subjects:** 20 healthy adults **Gender:** M **Age:** 21-44 yrs	DE Protocol 1 (n=8): idling Deutz diesel engine (F3M2011, 2.2 L, 500 rpm) using gas oil Protocol 2 (n=12): idling Volvo diesel engine (TD45, 4.5 L, 4 cylinders, 680 rpm) using Gasoil E10 **Particle Number/Count:** Protocol 1: 1.2 × 10^6/cm^3; Protocol 2: 1.26 × 10^6/cm^3 **Concentration:** Protocol 1: 348 μg/m^3, Protocol 2: 330 μg/m^3	In both protocols, exposures were conducted with intermittent exercise (15-min periods) to DE and filtered air in a randomized crossover design with exposures separated by at least one wk. Protocol 1 (n=8): Exposures conducted for 2 h. Other diesel emissions measured: NO_X (0.58 ppm), NO_2 (0.23 ppm), NO (0.36 ppm), CO (3.54 ppm), total hydrocarbon (2.8 μg/m^3). **Time to analysis:** 6 h post-exposure. Protocol 2 (n=12): Exposures conducted for 1h. Other diesel emissions measured: NO_X (2.78 ppm), NO_2 (0.62 ppm), NO (2.15 ppm), CO (3.08 ppm), total hydrocarbon (1.58 μg/m^3). **Time to analysis:** 2 and 6 h post-exposure.	Thrombus formation was observed to increase with diesel 2 and 6 h post-exposure using an ex vivo perfusion chamber. Both platelet-neutrophil and platelet-monocyte aggregates increased relative to filtered air 2 h following exposure to diesel (only evaluated in Protocol 2). Plasma concentrations of soluble CD40L were also observed to increase with diesel. Exposure to diesel was not shown to affect total leukocyte, monocyte, or platelet counts.
Reference: Lund et al. (2009, 191159) **Subjects:** 10 healthy adults **Gender:** 4 M/6 F **Age:** 18-40 yrs	DE Idling Cummins diesel engine (5.9 L) using commercial No. 2 fuel **Particle Size:** MMAD 0.10 μm **Concentration:** 100 μg/m^3	Subjects exposed for 1 h with intermittent exercise (15-min periods) to DE and filtered air in a randomized crossover study design. Other diesel emissions measured: NO_X (4.7 ppm), NO_2 (0.8 ppm), CO (2.8 ppm), total hydrocarbons (2.4 ppm). **Time to analysis:** 30 min and 24 h post-exposure.	Exposure to diesel resulted in an increase in MMP-9 plasma concentration and activity as well as an increase in endothelin-1 plasma concentration at both 30 min and 24 h post-exposure.
Reference: Lundback et al. (2009, 191967) **Subjects:** 12 healthy adults **Gender:** M **Age:** 21-30 yrs	DE Idling Volvo diesel engine (TD45, 4.5 L, 4 cylinders, 680 rpm) using Gasoil E10 **Particle Number/Count:** 1.26 × 10^6/cm^3 **Concentration:** 330 μg/m^3	Subjects exposed for 1 h with intermittent exercise (15-min periods) to DE and filtered air in a randomized crossover study design. Exposures were separated by at least one wk. Other diesel emissions measured: NO_X (2.78 ppm), NO_2 (0.62 ppm), NO (2.15 ppm), CO (3.08 ppm), total hydrocarbon (1.58 μg/m^3). **Time to analysis:** 10, 20, 30, and 40 min post-exposure.	Diesel-induced increase in arterial stiffness (increases in augmentation pressure and augmentation index, as well as decrease in time to wave reflection) observed at 10 and 20 min post-exposure using radial artery pulse wave analysis. No effect of diesel observed on carotid-femoral pulse wave velocity which was assessed 40 min post-exposure, but not at earlier time points. No effect of diesel observed on blood pressure 10-30 min post-exposure.

Study	Pollutant	Exposure	Findings
Reference: Mills et al. (2005, 188557) **Subjects:** 30 healthy adults **Gender:** M **Age:** 20-38 yrs	DE Idling 1991 Volvo diesel engine (TD45, 4.5 L, 4 cylinders, 680 rpm) **Particle Number/Count:** 1.2 × 106/cm^3 **Concentration:** 300 $\mu g/m^3$	Subjects exposed for 1 h with intermittent exercise (15-min periods) to DE and filtered air in a randomized crossover study design. Exposures were separated by two wks. Other diesel emissions measured: NO_2 (1.6 ppm), NO (4.5 ppm), CO (7.5 ppm), total hydrocarbon (4.3 ppm), formaldehyde (0.26 $\mu g/m^3$). **Time to analysis:** 2-4 h post-exposure for 15 subject; 6-8 h post-exposure for the other 15 subjects.	Forearm blood flow increase (induced by bradykinin, acetylcholine, and sodium nitroprusside) was attenuated by DE 2 and 6 h post-exposure. A 6 mmHg increase in diastolic blood pressure (p = 0.08) 2 h following exposure to DE was observed relative to filtered air control. Bradykinin-induced release of t-PA was attenuated by diesel exposure 6 h post-exposure. DE did not affect the release of t-PA 2 h post-exposure. No diesel-induced changes in serum IL-6 or TNF-□ observed 6 h post-exposure.
Reference: Mills et al. (2007, 091206) **Subjects:** 20 older adults with prior myocardial infarction **Gender:** M **Age:** 60 ± 1 yrs	DE Idling 1991 Volvo diesel engine (TD45, 4.5 L, 4 cylinders, 680 rpm) using low sulfur gas-oil E10 **Particle Size:** Median particle diameter 54 nm, Range 20-120 nm **Particle Number/Count:** 1.26 × 106/cm^3 **Concentration:** 300 $\mu g/m^3$	Subjects exposed for 1 h with intermittent exercise (15-min periods) to DE and filtered air in a randomized crossover study design. Exposures were separated by at least two wks. Other diesel emissions measured: NO_x (4.45 ppm), NO_2 (1.01 ppm), NO (3.45 ppm), CO (2.9 ppm), total hydrocarbon (2.8 ppm). **Time to analysis:** During exposure and 6-8 h post-exposure.	A greater increase in exercise induced ST-segment depression and ischemic burden was observed during exposure to DE than clean air. No diesel-induced effects on vasomotor dysfunction observed 6 h post-exposure. Bradykinin-induced release of t-PA was attenuated by diesel exposure relative to filtered air 6 h post-exposure. Effect of diesel on t-PA release was not evaluated at earlier times post-exposure. No diesel-induced changes in blood leukocyte counts or serum C-reactive protein 6 h post-exposure.
Reference: Mills et al. (2008, 156766) **Subjects:** 12 adults with coronary heart disease, 12 healthy adults **Gender:** M **Age:** CHD: 59 ± 2 yrs, Healthy: 54 ± 2 yrs	Fine CAPs (Edinburgh, Scotland, UK) **Particle Size:** Mean 1.23 μm **Particle Number/Count:** 99,400/cm^3 **Concentration:** 190 ± 37 $\mu g/m^3$	Exposures conducted for 2 h with intermittent exercise. Subjects exposed to CAPs and filtered air using a randomized crossover design with exposures separated by at least 2 wks. **Time to analysis:** 2, 6-8, and 24 h post-exposure.	CAPs exposure had no significant effect on vascular function in healthy adults or adults with coronary heart disease 6-8 h post-exposure (i.e., no change in forearm blood flow as assessed using venous occlusion plethysmography). The authors attributed this lack of response to a low concentration of combustion-derived particles. Small increase in blood platelet and monocyte concentration observed following CAPs exposure. Exposure to CAPs did not affect serum CRP concentration or total leukocyte or neutrophil count.
Reference: Peretz et al. (2007, 189082) **Subjects:** 5 healthy adults **Gender:** M **Age:** 20-31 yrs	DE 2002 Cummins B-series diesel engine (6BT5.9G6, 5.9 L); operating at 75% of rated capacity **Concentration:** Fine PM 50, 100, 200 $\mu g/m^3$	Subjects exposed for 2 h at rest to filtered air and each of the three DE particle concentrations in a randomized crossover study design. Exposures were separated by at least 2 wks. Other diesel emissions measured, 200 $\mu g/m^3$ exposure: NO_2 (23 ppb), NO (1.75 ppm), CO (1.58 ppm). **Time to analysis:** 6 and 22 h after the start of exposure.	PBMC expression of 10 genes involved in the inflammatory response were observed to be significantly affected by exposure to DE at the highest concentration tested (8 upregulated, 2 downregulated) 6 h after the start of exposure. The expression of 4 genes (1 upregulated, 3 downregulated) associated with the inflammatory response showed significant changes 22 h after diesel exposure. PBMC expression of 5 genes involved in the oxidative stress pathways showed significant changes at 6 h after the start of diesel exposure at the highest concentration tested (4 upregulated, 1 downregulated). 7 genes involved in the oxidative stress pathways showed significant changes at 22 h following exposure (4 upregulated, 3 downregulated).

Study	Pollutant	Exposure	Findings
Reference: Peretz et al. (2008, 156854) **Subjects:** 17 adults with metabolic syndrome, 10 healthy adults **Gender:** Metabolic syndrome: 11 M/6 F, Healthy: 8 M/2 F **Age:** Metabolic syndrome: 20-48 yrs, Healthy: 20-42 yrs	DE 2002 Cummins B-series diesel engine (6BT5.9G6, 5.9 L) using No. 2 undyed on-highway fuel; operating at 75% of rated capacity **Particle Size:** Median particle diameter 1.04 μm **Concentration:** Fine PM 100, 200 $\mu g/m^3$	Subjects exposed for 2 h at rest to both concentrations of DE as well as filtered air in a randomized crossover design. Exposures were separated by at least 2 wks. Other diesel emissions measured, 100 $\mu g/m^3$ exposure: NO_2 (16.5 ppb), NO (0.96 ppm), CO (0.51 ppm); 200 $\mu g/m^3$ exposure: NO_2 (24.7 ppb), NO (1.54 ppm), CO (0.89 ppm). **Time to analysis:** Immediately following exposure (within 30 min post-exposure) and 3 h from the start of exposure.	Exposure to 200 $\mu g/m^3$ elicited a statistically significant decrease in brachial artery diameter relative to filtered air immediately following exposure. A smaller decrease in brachial artery diameter was also observed following exposure to DE at 100 $\mu g/m^3$. Plasma levels of endothelin-1 were observed to increase following DE exposure (200 $\mu g/m^3$). The observed effects were more pronounced in healthy subjects than in subjects with metabolic syndrome. DE did not affect endothelium-dependent flow-mediated dilatation. No effect of DE on blood pressure was demonstrated immediately following exposure.
Reference: Peretz et al. (2008, 156855) **Subjects:** 13 adults with metabolic syndrome, 3 healthy adults **Gender:** Metabolic syndrome: 8 M/5 F, Healthy: 3 M/0 F **Age:** Metabolic syndrome: 31-48 yrs, Healthy: 24-39 yrs	DE 2002 Cummins B-series diesel engine (6BT5.9G6, 5.9 L) using No. 2 undyed on-highway fuel; operating at 75% of rated capacity **Concentration:** Fine PM 100, 200 $\mu g/m^3$	Subjects exposed for 2 h at rest to both concentrations of DE as well as filtered air in a randomized crossover design. Exposures were separated by at least 2 wks. Other diesel emissions measured, 100 $\mu g/m^3$ exposure: NO_2 (20.6 ppb), NO (0.95 ppm), CO (0.47 ppm); 200 $\mu g/m^3$ exposure: NO_2 (28.3 ppb), NO (1.63 ppm), CO (0.74 ppm). **Time to analysis:** 1, 3, 6, and 22 h from the start of exposure.	Exposure to 200 $\mu g/m^3$ increased HF power and decreased the LF/HF ratio 1h post-exposure; however, this effect was not consistent across subjects. No effect of DE was observed at later time points. Subjects with metabolic syndrome did not experience greater changes in HRV than healthy subjects.
Reference: Power et al. (2008, 191982) **Subjects:** 5 adults with mild-to-moderate allergic asthma **Gender:** 1 M/4 F **Age:** 28-51 yrs	Carbon and ammonium nitrate particles **Concentration:** With co-exposure to 0.2ppm O_3: 255 $\mu g/m^3$, Without co-exposure to 0.2ppm O_3: 313 $\mu g/m^3$	Subjects exposed for 4 h with intermittent exercise (30-min periods) to filtered air, particles, and particles + ozone in a crossover study design. Exposures were separated by at least 3 wks. **Time to analysis:** 3 h 40 min from the start of exposure.	Time and frequency domain HRV parameters were not affected by particle exposure relative to filtered air. However, exposure to particles with ozone resulted in a significant decrease in SDNN as well high and low frequency power normalized to the difference between total and very low frequency power.

Study	Pollutant	Exposure	Findings
Reference: Routledge et al. (2006, 088674) **Subjects:** 20 older adults with coronary artery disease, 20 healthy older adults **Gender:** CAD: 17 M/3 F, Healthy: 10 M/10 F **Age:** CAD: 52-74 yrs, Healthy: 56-75 yrs	Ultrafine carbon **Particle Size:** < 10-300 nm; mode at 20-30 nm **Concentration:** Ultrafine carbon: 50 $\mu g/m^3$; SO_2: 200ppb	Exposures conducted (head dome system) to filtered air, ultrafine carbon, SO_2, and ultrafine carbon + SO_2 for 1 h at rest using a randomized crossover study design. **Time to analysis:** Immediately following exposure as well as 3 and 23 h post-exposure.	No PM-induced changes in HRV observed among subjects with coronary artery disease. Among healthy subjects, small increase in HRV (RR, SDNN, rMSSD, and LF power) demonstrated immediately post-carbon exposure. Relative to filtered air control, exposure to ultrafine carbon did not significantly affect blood pressure in healthy adults or adults with coronary artery disease 0-3 h post-exposure. Exposure to ultrafine carbon, either alone or with SO2, did not affect plasma levels of fibrinogen or D-dimer at 3 or 23 h post-exposure. Exposure to ultrafine carbon did not affect peripheral blood leukocyte count or C-reactive protein levels 3 or 23 h post-exposure.
Reference: Rundell and Caviston (2008, 191986) **Subjects:** 15 healthy college athletes **Gender:** M **Age:** Avg 19.5 yrs	Gasoline emissions 2.5 hp gasoline engine running 10 s each min during exposure and in the min prior to exposure **Particle Size:** $PM_{1.0}$ **Particle Number/Count:** Trial 1: 336,730 ± 149,206/cm^3; Trial 2: 396,200 ± 82,564/cm^3	Subjects were exposed twice to both clean air and dilute gasoline exhaust during 6-min periods of maximal exercise on a cycle ergometer. Clean air exposures occurred first and were separated by 3 days. Gasoline exhaust exposures were also separated by 3 days, with the first occurring 7 days after the second clean air exposure. Other emissions measured: CO (6.3 ± 3.4 ppm). **Time to analysis:** 6 min	There was no difference in total work done (kJ) between the clean air exposures or between the clean air exposures and the first exposure to gasoline exhaust. However, the second gasoline exhaust exposure was demonstrated to significantly decrease work accumulated over the 6min exercise period compared with either of the other exposure conditions ($p < 0.01$).
Reference: Samet et al. (2007, 156940) **Subjects:** Ultrafine CAPs: 20 healthy adults, Coarse CAPs: 14 healthy adults **Gender:** Ultrafine CAPs: 11 M/9 F, Coarse CAPs: 8 M/6 F **Age:** Ultrafine CAPs: 18-35 yrs, Coarse CAPs: 18-35 yrs	CAPs (Chapel Hill, NC) **Particle Size:** Ultrafine 0.049 ± 0.009 μm; Coarse 3.59 ± 0.58 μm **Concentration:** Ultrafine 47.0 ± 20.2 $\mu g/m^3$; Coarse 89.0 ± 49.5 $\mu g/m^3$	Preliminary report comparing effects of controlled exposures to coarse, fine, and ultrafine CAPs among healthy adults (3 separate studies). A randomized crossover design was used in evaluating effects of coarse CAPs (n=14) and ultrafine CAPs (n=20) relative to filtered air following 2-h exposures with intermittent exercise. Results compared with previous study of controlled exposure to fine CAPs (Chapel Hill, NC) where subjects did not serve as their own controls (Ghio et al., 2000, 012140). **Time to analysis:** 0-20 h post-exposure.	Statistically significant decrease in SDNN observed 20 h following exposure to coarse CAPs relative to filtered air. Subjects in the high ultrafine CAPs group experienced a decrease in SDNN based on an analysis of 24 h ambulatory Holter monitoring relative to filtered air. Fine CAPs did not significantly affect HRV. Increased levels of D-dimer observed 18 h following exposure to ultrafine CAPs. No CAPs-induced changes in plasma factor VII, plasminogen, fibrinogen, PAI-1, vWf, or t-PA. No CAPs-induced changes in C-reactive protein levels were observed.

Study	Pollutant	Exposure	Findings
Reference: Samet et al. (2009, 191913) **Subjects:** 19 healthy adults **Gender:** 10 M/9 F **Age:** 18-35 yrs	Ultrafine CAPs (Chapel Hill, NC) **Particle Size:** < 0.16 μm **Particle Number/Count:** 120,662 ± 48,232 particles/cm^3 **Concentration:** 49.8 ± 20 μg/m^3	Subjects exposed for 2 h with intermittent 15 periods of exercise to UF CAPs and filtered air using a randomized crossover study design. **Time to analysis:** Immediately following exposure and 1 and 18 h post-exposure.	UF CAPs exposure resulted in an increase in plasma concentrations of D-dimer both immediately following exposure (20.6% increase per 10^5 particles/cm^3) as well as 18 h post-exposure (18.2% increase per 10^5 particles/cm^3). Plasma concentration of PAI1 also increased with UF CAPs, although this increase was not statistically significant (24% increase, p = 0.1). No UF CAPs-induced changes observed in plasma concentrations of tPA, vWF, CRP, fibrinogen, plasminogen, or Factor VII. HF and LF power were both observed to increase with UF CAPs exposure relative to filtered air at 18 h post-exposure (41.8% and 36%, respectively, per 10^5 particles/cm^3 increase in UF CAPs). UF CAPs expressed as mass concentration was not observed have a statistically significant effect in HF total power. UF CAPs was not observed to affect time domain measures of HRV over 24 h. The QT interval was shown to decrease both immediately following and at 18 h post exposure (not statistically significant immediately following exposure).
Reference: Shah et al. (2008, 156970) **Subjects:** 16 healthy adults **Age:** 26.9 ± 6.9 yrs	Ultrafine elemental carbon **Particle Number/Count:** 10.8 ± 1.7 × 10^6 /cm^3 **Concentration:** 50 μg/m^3	Exposures conducted via mouthpiece for 2 h with intermittent exercise to filtered air and ultrafine carbon in a randomized crossover study design. **Time to analysis:** Immediately following exposure as well as 3.5, 21, and 45 h post-exposure.	Exposure to ultrafine carbon attenuated peak forearm blood flow after ischemia relative to filtered air 3.5 h post-exposure. Venous nitrate levels were significantly lower at 21 h following exposure to UF carbon compared with filtered air exposure. PM exposure was not observed to affect blood pressure relative to filtered air at times 0-45 h post-exposure.
Reference: Tornqvist et al. (2007, 091279) **Subjects:** 15 healthy adults **Gender:** M **Age:** 18-38 yrs	DE Idling 1991 Volvo diesel engine (TD45, 4.5 L, 4 cylinders, 680 rpm) **Concentration:** 300 μg/m^3	Subjects exposed for 1h with intermittent exercise (15-min periods) to DE and filtered air in a randomized crossover study design. Exposures were separated by at least two wks. Other diesel emissions measured: NO_X (4.44 ppm), NO_2 (0.82 ppm), NO (3.62 ppm), total hydrocarbon (2.21 ppm). **Time to analysis:** 24 h post-exposure.	DE was observed to significantly attenuate endothelium-dependent vasodilation 24 h post-exposure. Endothelium-independent vasodilation was not affected by diesel exposure. Exposure to DE did not affect blood pressure relative to filtered air 24 h after exposure. DE significantly increased plasma levels of IL-6 and TNF-¤ 24 h following exposure. Exposure to diesel resulted in an increase in total antioxidant capacity of plasma relative to filtered air 24 h post-exposure.
Reference: Urch et al. (2004, 055629) **Subjects:** 24 healthy adults **Gender:** 14 M/10 F **Age:** 35 ± 10 yrs	Fine CAPs (Toronto) **Concentration:** 150 μg/m^3 (range 101-257 μg/m^3) with 120 ppb ozone	Exposures conducted through a facemask which covered the subject's nose and mouth. Subjects were exposed to CAPs + ozone and filtered air for 2 h at rest in a randomized crossover study design. Exposures were separated by at least 2 days. **Time to analysis:** Immediately following exposure.	CAPs + ozone exposure resulted in a significant decrease in brachial artery diameter immediately post-exposure (Brook et al., 2002, 024987), which was demonstrated to be associated with both the organic and elemental carbon fractions of the CAPs.
Reference: Urch et al. (2005, 081080) **Subjects:** 23 healthy adults **Gender:** 13 M/10 F **Age:** 32 ± 10 yrs	Fine CAPs (Toronto); **Concentration:** 150 μg/m^3 (range 102-214 μg/m^3) with 120 ppb ozone	Exposures conducted through a facemask which covered the subject's nose and mouth. Subjects were exposed to CAPs + ozone and filtered air for 2 h at rest in a randomized crossover study design. **Time to analysis:** Every 30 min during exposure, with the final measurement made immediately prior to the end of the exposure.	An increase in diastolic blood pressure of 6 mmHg was observed at the end of CAPs + ozone exposure, which was statistically different from the change in blood pressure experienced during exposure to filtered air (1 mmHg). This effect was associated with the organic fraction of $PM_{2.5}$.

Study	Pollutant	Exposure	Findings
Reference: Zareba et al. (2009, 190101) **Subjects:** 24 healthy adults **Gender:** 12 M/12 F **Age:** 18-40 yrs	Ultrafine elemental carbon **Particle Size:** Count median diameter 25 nm **Particle Number/Count:** $2x10^6/cm^3$ (10 $\mu g/m^3$), $7x10^6/cm^3$ (25 $\mu g/m^3$) **Concentration:** 10 $\mu g/m^3$; 25 $\mu g/m^3$	Protocol 1 (n = 12, 6 M/6 F): Subjects exposed to 10 $\mu g/m^3$ UF carbon and filtered air for 2 h at rest in a randomized crossover design. Exposures were separated by at least 2 wks. Protocol 2 (n = 12, 6 M/6 F): Subjects exposed to 10 $\mu g/m^3$, 25 $\mu g/m^3$, and filtered air for 2 h with intermittent exercise (15-min periods) in a restricted randomized crossover design (all subjects exposed to 10 $\mu g/m^3$ before 25 $\mu g/m^3$). Exposures were separated by at least 2 wks. **Time to analysis (both protocols):** Immediately following exposure and 3.5 and 21 h post-exposure.	Exposure to 10 $\mu g/m^3$ at rest resulted in no change in HRV frequency domain parameters relative to filtered air exposure. Time domain parameters were observed to increase slightly with UF carbon exposure (10 $\mu g/m^3$ at rest), however, only the increase in rMSSD was statistically significant (p = 0.032). Some trends toward less shortening of QT interval, increase in ST segment, and increase in variability of repolarization (variability of T wave complexity) were observed with exposure to 10 $\mu g/m^3$ at rest, none of which were statistically significant. In Protocol 2, exposure to 10 $\mu g/m^3$ UF carbon was observed to slightly increase HRV time domain parameters as was demonstrated in Protocol 1. However, this was not observed at the higher concentration (25 $\mu g/m^3$). As with exposure at rest, exposure to UF carbon during exercise was observed to affect repolarization (reduction in QT duration and increase in T-wave amplitude), although this effect was not statistically significant.

Table C-2. Respiratory effects

Reference	Pollutant	Exposure	Findings
Reference: Alexis et al. (2006, 088636) **Subjects:** 9 healthy adults **Gender:** 3 M/6 F **Age:** 18-35 yrs	Coarse fraction particles (Chapel Hill, NC) Heat-treated (biologically inactive) and non-heated particles **Particle Size:** MMAD 5 μm **Concentration:** 0.65 mg per subject	Subjects were administered heat-treated $PM_{2.5-10}$, non-heated $PM_{2.5-10}$, and 0.9% saline (control) via nebulization in a randomized crossover study design. Exposures were separated by at least 1 wk. **Time to analysis:** 2-3 h post-inhalation.	Both heat-treated and non-heated coarse PM were observed to increase neutrophil counts in induced sputum 2-3 h post-inhalation. Biologically active PM (non-heated) induced an increase expression of macrophage TNF-α mRNA, eotaxin, and immune surface phenotypes on macrophages (mCD14, CD11b/CR3, and HLA-DR).
Reference: Barregard et al. (2008, 155675) **Subjects:** 13 healthy adults **Gender:** 6 M/ 7 F **Age:** 20-56 yrs	Wood smoke **Particle Size:** Session 1: geometric mean diameter 42 nm, Session 2: geometric mean diameter 112 nm **Particle Number/Count:** Session 1: 180,000/cm^3; Session 2: 95,000/cm^3 **Concentration:** Session 1: median 279 $\mu g/m^3$; Session 2: median 243 $\mu g/m^3$	Subjects exposed in two groups for 4 h to filtered air, followed a wk later by a 4-h exposure to wood smoke. Exposures conducted with two 25-min periods of light exercise. Other measured combustion products: Session 1: NO_2 (0.08 ppm), CO (13 ppm), formaldehyde (114 $\mu g/m^3$), acetaldehyde (75 $\mu g/m^3$), benzene (30 $\mu g/m^3$), 1,3-butadiene (6.3 $\mu g/m^3$); Session 2: NO_2 (0.09 ppm), CO (9.1 ppm), formaldehyde (64 $\mu g/m^3$), acetaldehyde (40 $\mu g/m^3$), benzene (20 $\mu g/m^3$), 1,3-butadiene (3.9 $\mu g/m^3$). **Time to analysis:** Immediately following exposure as well as 3 and 20 h post-exposure.	Relative to filtered air, exposure to wood smoke was observed to increase levels of eNO 3 h post-exposure. Serum Clara cell protein increased 20 h after wood smoke exposure. Wood smoke was observed to increase levels of malondialdehyde in breath condensate immediately after as well as 20 h post-exposure. Effects of wood smoke on eNO and malondialdehyde levels were similar between the two sessions of wood smoke exposure. However, serum Clara cell protein was significantly increased with wood smoke in session 1 (higher particle count) but not in session 2.
Reference: Bastain et al. (2003, 098690) **Subjects:** 18 nonsmoking adults with positive allergy skin test to short ragweed **Gender:** 7 M/11 F **Age:** 18-38 yrs	DEP Isuzu diesel engine, 4 cylinder, 4JB1 **Concentration:** 0.3 mg in 200 μl saline	Subjects underwent nasal provocation challenge (intranasal spray) with allergen and either DEP or placebo (saline) in a randomized crossover study design. Challenges were separated by 30 days. This protocol was then repeated 30 days after the last exposure. **Time to analysis:** 24 h post-exposure and 4 and 8 days after exposure.	DEP significantly increased allergic responses to short ragweed. Relative to allergen + placebo, allergen + DEP increased allergen specific IgE 4days following exposure, and increased IL-4 1 day post-exposure. The enhancement of allergic response with DEP was observed to be reproducible within subjects.

Reference	Pollutant	Exposure	Findings
Reference: Beckett et al. (2005, 156261) **Subjects:** 12 healthy adults **Gender:** 6 M/6 F **Age:** 23-52 yrs	Ultrafine and fine zinc oxide **Particle Size:** UF: $<$ 0.1 μm; Fine: 0.1-1.0 μm **Particle Number/Count:** UF: 4.6 $\times$ 10^7/cm^3; Fine: 1.9 $\times$ 10^5/cm^3 **Concentration:** 500 μg/m^3	Subjects exposed via mouthpiece for 2 h during rest to filtered air, ultrafine, and fine zinc oxide in a randomized crossover study design. Exposures were separated by at least 3 wks. **Time to analysis:** 11 and 24 h after exposure.	No changes observed in neutrophil count in induced sputum. No PM (zinc oxide)-induced changes in respiratory symptoms observed 0-24 h post-exposure.
Reference: Behndig et al. (2006, 088286) **Subjects:** 15 healthy adults **Gender:** 8 M/7 F **Age:** 21-27 yrs	DE Idling 1991 Volvo diesel engine (TD45, 4.5 L, 4 cylinders, 680 rpm) **Particle Size:** PM_{10}; majority of PM mass made up of particles $<$ 1 μm in diameter **Concentration:** 100 μg/m^3	Exposures conducted for 2 h with intermittent exercise to both DE and filtered air in a randomized crossover design. Exposures were separated by at least 3 wks. Other diesel emissions measured: NO_X (1.8 ppm), NO_2 (0.4 ppm), NO (1.3 ppm), CO (10.4 ppm), total hydrocarbons (1.3 ppm). **Time to analysis:** 18 h post-exposure.	Exposure to DE increased neutrophil and mast cell numbers in bronchial mucosa at 18 h post-exposure. Neutrophils, IL-8, and myeloperoxidase observed to increase in bronchial lavage fluid following exposure relative to filtered air. No inflammatory response observed in the alveolar compartment. Exposure to DE increased urate and reduced glutathione bronchoalveolar lavage at 18 h post-exposure.
Reference: Blomberg et al. (2005, 191991) **Subjects:** 15 older adults (former smokers) with COPD **Age:** 56-72 yrs	DE **Concentration:** 300 μg/m^3	Subjects exposed for 1 h with intermittent exercise to DE and filtered air in a randomized crossover study design. **Time to analysis:** 6 and 24 h post-exposure.	DE was not observed to affect levels of Clara cell protein in peripheral blood at 6 and 24 h post-exposure.
Reference: Bosson et al. (2007, 156286) **Subjects:** 16 healthy adults **Gender:** 7 M/9 F **Age:** 20-28 yrs	DE Idling Volvo diesel engine **Concentration:** PM 300 μg/m^3 followed by exposure to filtered air or 0.2 ppm ozone	Subjects exposed to DE for 1 h followed 5 h later by a 2-h exposure to either filtered air or ozone (0.2 ppm) using a randomized crossover study design. All exposures were conducted with subjects engaged in intermittent exercise. **Time to analysis:** 18 h after second exposure (filtered air or ozone).	The percentage of neutrophils and concentration of myeloperoxidase in induced sputum (18 h post-ozone/air exposure) was significantly higher following diesel + ozone than diesel + air.
Reference: Bosson et al. (2008, 156287) **Subjects:** 14 healthy adults **Gender:** 9 M/5 F **Age:** 21-29 yrs	DE Idling 1991 Volvo diesel engine (TD45, 4.5 L, 4 cylinders) **Concentration:** PM 300 μg/m^3 or filtered air followed by exposure to 0.2 ppm ozone	Subjects exposed to DE or filtered air for 1h followed 5 h later by a 2-h exposure to ozone (0.2 ppm) using a randomized crossover study design. All exposures were conducted with subjects engaged in intermittent exercise. Other diesel emissions measured: NO_2 (0.51 ppm), NO (1.65 ppm), total hydrocarbons (1.18 ppm). **Time to analysis:** 24 h after the start of the initial exposure.	Neutrophil and macrophage numbers in bronchial wash were significantly increased 16 h following ozone exposure when preceded by exposure to diesel, compared to ozone exposure preceded by exposure to filtered air.
Reference: Brauner et al. (2009, 190244) **Subjects:** 29 healthy adults **Gender:** 20 M, 9 F **Age:** M avg 27 yrs, F avg 26 yrs	Urban traffic particles **Particle Size:** $PM_{2.5}$, $PM_{2.5\text{ -}10}$ **Particle Number/Count:** 6-700 nm: 10,067/cm^3 **Concentration:** $PM_{2.5}$: 9.7 μg/m^3, $PM_{2.5\text{ -}10}$: 12.6 μg/m^3	Subjects exposed to urban traffic particles and filtered air for 24 h with and without two 90-min periods of light exercise in a randomized crossover study design. Concentrations of NO_X and NO were low and did not differ between filtered and unfiltered exposures. CO concentrations were higher with filtered air (0.35 and 0.41 ppm), while ozone concentrations were lower with filtered air (12.08 and 4.29 ppb). **Time to analysis:** 2.5, 6, and 24 h after the start of exposure.	Epithelial membrane integrity and blood-gas barrier permeability, assessed using pulmonary clearance of ^{99m}Tc-labeled diethylenetriamine pentaacetic acid (DTPA), was observed to increase with exercise, but was not affected by exposure to urban particles (2.5 h of exposure). Exposure to urban particles was not shown to affect plasma or urine concentration of Clara cell 16 protein at 6 and 24 h after the start of exposure. No relationship between exposure and pulmonary function was observed at 2.5 h.
Reference: Gilliland et al. (2004, 156471) **Subjects:** 19 adults with allergic rhinitis and positive skin test to ragweed, GSTM1 (14 null, 5 present); GSTT1 (9 null, 10 present); GSTP1 codon 105 variants (13 I/I, 6 I/V, 0 V/V) **Gender:** 7 M/12 F **Age:** 20-34 yrs	DEP Isuzu diesel engine, 4 cylinder, 4JB1 **Concentration:** 0.3 mg DEP in 300 μL saline	Subjects were challenged intranasally with allergen and placebo (saline) as well as allergen plus DEP in saline in a randomized crossover design. Challenges were separated by at least 6 wks. **Time to analysis:** 10 min, 24 h, and 72 h post-challenge.	Subjects who were GSTM1 null or homozygous for GSTP1 I105 wild-type allele experienced significantly greater increase in nasal IgE and histamine with diesel plus allergen compared to subjects with functional GSTM1 or who were heterozygous for GSTP1 I/V(105).

Reference	Pollutant	Exposure	Findings
Reference: Gong et al. (2004, 087964) **Subjects:** 13 older adults with COPD, 6 healthy older adults **Gender:** COPD: 5 M/ 8 F, Healthy: 2 M/4 F **Age:** COPD: avg 68 yrs, Healthy: avg 73 yrs	Fine CAPs (Los Angeles) **Particle Size:** 85% of mass between 0.1 and 2.5 μm **Concentration:** Mean: 194 $\mu g/m^3$, Range: 135-229 $\mu g/m^3$	Exposures to CAPs and filtered air (randomized crossover) for 2 h with intermittent light exercise (four 15-min periods). Exposures were separated by at least 2 wks. **Time to analysis:** Immediately following exposure as well as 4 and 22 h post-exposure.	No CAPs-induced respiratory symptoms observed in healthy older adults or older adults with COPD at 0, 4, or 22 h post-exposure. Exposure to CAPs did not significantly affect FVC or FEV_1. CAPs exposure caused a decrease in arterial oxygen saturation 4 h post-exposure which was more pronounced in healthy older adults than in older adults with COPD. Exposure to CAPs was not observed to affect the levels of white blood cells, columnar epithelial cells, IL-6, or IL-8 in induced sputum.
Reference: Gong et al. (2004, 055628) **Subjects:** 12 adult asthmatics, 4 healthy adults **Gender:** Asthmatic: 4 M/8 F, Healthy: 2 M/2 F **Age:** Asthmatic: avg 38 yrs, Healthy: avg 32 yrs	Coarse CAPs (Los Angeles) **Particle Size:** 80% of mass between 2.5 and 10 μm, 20% of mass < 2.5 μm **Concentration:** Mean: 157 $\mu g/m^3$; Range: 56-218 $\mu g/m^3$	Exposures to CAPs and filtered air (randomized crossover) for 2 h with intermittent light exercise (four 15-min periods). Exposures were separated by at least 2 wks. **Time to analysis:** Immediately following exposure as well as 4 and 22 h post-exposure.	No effect of CAPs exposure on spirometry or arterial oxygen saturation was observed 0, 4, or 22 h post-exposure. No respiratory symptoms reported 0-22 h post-exposure in either healthy or asthmatic adults. Sputum cell counts at 22 h post-exposure did not differ between CAPs and filtered air.
Reference: Gong et al. (2005, 087921) **Subjects:** 18 older adults with COPD, 6 healthy older adults **Gender:** COPD: 9 M/9 F, Healthy: 2 M/4 F **Age:** COPD: avg 72 yrs, Healthy: avg 68 yrs	Fine CAPs (Los Angeles) **Concentration:** CAPs: 200 $\mu g/m^3$; NO_2: 0.4 ppm	Each subject was exposed to CAPs, NO_2, CAPs + NO_2, and filtered air for 2 h with intermittent exercise. Exposure order was not fully counterbalanced as NO_2 exposures were conducted after the majority of the CAPs and filtered air exposures had been completed. Exposures were separated by at least 2 wks. **Time to analysis:** Immediately following exposure as well as 4 and 22 h post-exposure.	Exposure to CAPs was observed to decrease maximal mid-expiratory flow and arterial oxygen saturation relative to filtered air 4-22 h post-exposure. This response was more pronounced in healthy older adults than in older adults with COPD. Concomitant exposure to NO_2 did not enhance the response. No other respiratory responses (symptoms, spirometry, sputum cell counts) were affected by exposure to CAPs.
Reference: Gong et al. (2008, 156483) **Subjects:** 14 adult asthmatics, 17 healthy adults **Gender:** Asthmatics: 9 M/5 F, Healthy: 5 M/12 F **Age:** Asthmatics: 34 ± 12 yrs, Healthy:24 ± 8 yrs	Ultrafine CAPs (Los Angeles) **Particle Number/Count:** 145,000/cm^3, Range 39,000-312,000/cm^3 **Concentration:** Mean: 100 $\mu g/m^3$, Range: 13-277 $\mu g/m^3$	Subjects exposed for 2 h during intermittent exercise (15-min periods) to both CAPs and filtered air in random order. The first 7 subjects underwent whole body exposure, while the remaining subjects were exposed through a facemask. Facemask exposures had higher particle counts but lower particle mass than whole body exposures. Exposures were separated by at least 2 wks. **Time to analysis:** Immediately following exposure as well as 4 and 22 h post-exposure.	No significant differences in respiratory symptoms observed between filtered air and ultrafine CAPs exposures. Individuals exposed to higher particle counts tended to experience greater symptoms with CAPs than with filtered air. An ultrafine CAPs-induced decrease in arterial oxygen saturation (0.5%) was observed at 0, 4, and 22 h post-exposure. A decrease in FEV_1 (2%) was also observed 22 h post-exposure relative to filtered air. Responses were not significantly different between healthy and asthmatic adults. CAPs exposure was not observed to affect total sputum cell counts or cytokine levels. There were no differences in response observed between facemask and whole body exposures.
Reference: Graff et al. (2009, 191981) **Subjects:** 14 healthy adults **Gender:** 8 M/6 F **Age:** 20-34 yrs	Coarse CAPs (Chapel Hill, NC) **Concentration:** 89 ± 49.5 $\mu g/m^3$ (estimated inhaled dose ◘ 67% of measured particle mass)	Subjects exposed for 2 h with intermittent exercise (15-min periods) to coarse CAPs and filtered air in a randomized crossover design. Exposures were separated by at least 2 mos. **Time to analysis:** 0-1 and 20 h post-exposure.	Pulmonary function (FVC, FEV_1, and carbon moNOxide diffusing capacity) was not affected by exposure to coarse CAPs either immediately following exposure or 20 h post-exposure. A significant increase in percent PMNs (10.7% increase per 10 $\mu g/m^3$ coarse CAPs) was observed in BAL fluid 20 h post-exposure. Percent monocytes in BL fluid were slightly decreased at 20 h post-exposure (2.0% decrease per 10 $\mu g/m^3$ CAPs; $p = 0.05$). Total protein in BAL fluid was also observed to decrease following CAPs exposure (1.8% decrease per 10 $\mu g/m^3$ CAPs). Markers of inflammation in BAL and BL fluids including IL-6, IL-8, and PGE_2 were not affected by exposure to coarse CAPs.
Reference: Huang et al. (2003, 087377) **Subjects:** 38 healthy adults **Gender:** 36 M/2 F **Age:** Avg 26.2 ± 0.7 yrs	Fine CAPs (Chapel Hill, NC) **Concentration:** 23.1-311.1 $\mu g/m^3$	Subjects exposed to CAPs (n = 30) or filtered air (n = 8) for 2 h with intermittent exercise (subjects did not serve as their own controls). Component data of CAPs was available for 37 of the 38 subjects. **Time to analysis:** 18 h after exposure.	The increase in bronchoalveolar lavage fluid neutrophils observed by Ghio et al. (2000, 012140) following exposure to fine CAPs was shown to be associated with iron, selenium, and sulfate content of the CAPs.

Reference	Pollutant	Exposure	Findings
Reference: Kongerud et al. (2006, 156656) **Subjects:** 17 asthmatic adults, 46 healthy adults **Gender:** Asthmatics- 6 M/11 F, Healthy- 24 M/22 F **Age:** Asthmatics: avg 23 yrs, Healthy: avg 26 yrs	DEP NIST 1650, heavy duty diesel engine **Concentration:** Untreated and treated with 0.1 ppm ozone (48 h); 300 μg per nostril	DEP (with and without ozone pre-treatment) were intranasally instilled, using the saline vehicle as control. Subjects did not serve as their own controls (not a crossover design). **Time to analysis:** 4 and 96 h post-instillation.	Exposure to DEP was not observed to alter markers of inflammation in nasal lavage fluid (e.g., cell counts, IL-8, IL-6) at 4 or 96 h post-instillation.
Reference: Larsson et al. (2007, 189320) **Subjects:** 16 healthy adults **Gender:** 10 M/6 F **Age:** 19-59 yrs	Traffic particles (road tunnel) **Particle Size:** $PM_{2.5}$, PM_{10}; $PM_{2.5}$ mass constituted $\sim$36% of PM_{10} mass **Particle Number/Count:** 20-1,000 nm: 1.1 × 105/cm^3, < 100 nm: 0.85 × 105/cm^3 **Concentration:** $PM_{2.5}$-46-81 μg/m^3; PM_{10}-130-206 μg/m^3	Exposures were conducted for 2 h with intermittent exercise in a room adjacent to a busy road tunnel. Study used a randomized crossover design with each subject also exposed to normal air (control). Exposures were separated by 3-10wks. No exposures to filtered air were conducted. Other traffic emissions measured: NO (874 μg/m^3), NO_2 (230 μg/m^3), CO (5.8 μg/m^3 reported, likely 5.8 mg/m^3). **Time to analysis:** 14 h post-exposure.	An increase in bronchoaveolar lavage fluid cell number, lymphocytes, and alveolar macrophages were observed 14 h after road tunnel exposure relative to control. Traffic particulate exposure was not shown to effect cytokine or adhesion molecule expression in bronchial tissues. Respiratory symptoms were reported to increase during exposure to road tunnel air relative to pre-exposure symptom ratings. Exposure to road tunnel air was not shown to affect lung function.
Reference: Mudway et al. (2004, 180208) **Subjects:** 25 healthy adults **Gender:** 16 M/9 F **Age:** 19-42 yrs	DE Idling 1991 Volvo diesel engine (TD45, 4.5 L, 4 cylinders, 680 rpm) **Concentration:** PM_{10} 100 μg/m^3	Subjects exposed to DE and filtered air for 2 h with intermittent exercise (15-min periods) in a randomized crossover design. Exposures were separated by at least 3 wks. Other diesel emissions measured: NO_2 (0.2 ppm), NO (0.6 ppm), CO (1.7 ppm), total hydrocarbons (1.4 ppm), formaldehyde (43.5 μg/m^3). **Time to analysis:** 1 h after the start of exposure, immediately following exposure, and 6 h post-exposure.	DE caused mild throat irritation in some subjects and a significant increase in airways resistance (Raw) during or immediately following exposure. No changes in FEV_1 or FVC were observed following exposure to diesel. Neutrophil numbers in the bronchial airways tended to increase following exposure to DE, however, this increase was highly variable between subjects and did not reach statistical significance. Exposure to DE did not affect levels of SOD or malondialdehyde in the airways. An increase in levels of ascorbate and GSH in nasal lavage fluid was observed 6 h following exposure to DE.
Reference: Pietropaoli et al. (2004, 156025) **Subjects:** 16 asthmatic adults, 40 healthy adults **Gender:** Asthmatic: 8 M/8 F, Healthy: 20 M/20 F **Age:** 18-40 yrs	Ultrafine elemental carbon **Particle Size:** CMD $\sim$25 nm **Particle Number/Count:** 10 μg/m^3: $\sim$2.0 × 10^6/cm^3; 25 μg/m^3: $\sim$7.0 × 106/cm^3; 50 μg/m^3: $\sim$10.8 × 106/cm^3 **Concentration:** 10, 25, and 50 μg/m^3	Study conducted using a randomized crossover design with 2-h exposures. Asthmatics (n = 16) exposed to filtered air and 10 μg/m^3. 12 healthy adults exposed to filtered air and 10 μg/m^3 at rest; 12 healthy adults exposed to filtered air, 10 and 25 μg/m^3 with intermittent exercise; 16 healthy adults exposed to filtered air and 50 μg/m^3 with intermittent exercise. Exposures were conducted via mouthpiece. **Time to analysis:** Immediately following exposure as well as 3.5, 21, and 45 h post-exposure.	No PM-induced changes in eNO or cell counts, IL-6, or IL-8 in induced sputum were observed in any of the protocols 21 h following exposure. Ultrafine carbon was not observed to increase respiratory symptoms in any of the study protocols. Healthy adults experienced an ultrafine PM-induced reduction in maximal mid-expiratory flow and CO diffusing capacity relative to filtered air 21 h following exposure.
Reference: Pourazar et al. (2005, 088305) **Subjects:** 15 healthy adults **Gender:** 11 M/4 F **Age:** 21-28 yrs	DE Idling Volvo diesel engine **Particle Number/Count:** 4.3 × 106/cm^3 **Concentration:** PM_{10} 300 μg/m^3	Subjects exposed to DE and filtered air for 1 h with intermittent exercise (randomized crossover study design). Other diesel emissions measured: NO_2 (1.6 ppm), NO (4.5 ppm), CO (7.5 ppm), total hydrocarbons (4.3 ppm), formaldehyde (0.26 mg/m^3). **Time to analysis:** 6 h post-exposure.	Exposure to DE significantly increased nuclear translocation of NF-κB, AP-1, phosphorylated p38, and phosphorylated JNK in bronchial epithelium 6 h post-exposure.
Reference: Pourazar et al. (2008, 156884) **Subjects:** 15 healthy adults **Gender:** 11 M/4 F **Age:** 21-28 yrs	DE Idling Volvo diesel engine **Particle Number/Count:** 4.3 × 106/cm^3 **Concentration:** PM_{10} 300 μg/m^3	Subjects exposed to DE and filtered air for 1 h with intermittent exercise (randomized crossover study design). Other diesel emissions measured: NO_2 (1.6 ppm), NO (4.5 ppm), CO (7.5 ppm), total hydrocarbons (4.3 ppm), formaldehyde (0.26 mg/m^3). **Time to analysis:** 6 h post-exposure.	Exposure to DE observed to enhance epidermal growth factor receptor (EGFR) expression in bronchial epithelium 6 h post-exposure.
Reference: Riechelmann et al. (2004, 180120) **Subjects:** 30 healthy adults **Gender:** 11 M/19 F **Age:** 22-32 yrs	Urban dust NIST SRM 1649a **Concentration:** 150, 500 μg/m^3	Subjects exposed to both concentrations of urban dust (nose only exposure system) as well as filtered air for 3h at rest in a randomized crossover design. Exposures were separated by at least 1 wk. **Time to analysis:** 30 min, 8 h, and 24 h post-exposure.	An increase in nasal secretion (nasal cytology) of IL-6 and IL-8 were observed 24 h after exposure to 500 μg/m^3 urban dust.

Reference	Pollutant	Exposure	Findings
Reference: Samet et al. (2007, 156940) **Subjects:** Ultrafine CAPs: 20 healthy adults, Coarse CAPs: 14 healthy adults **Gender:** Ultrafine CAPs: 11 M/9 F, Coarse CAPs: 8 M/6 F **Age:** 18-35 yrs	CAPs (Chapel Hill, NC) **Particle Size:** Ultrafine: 0.049 ± 0.009 μm, Coarse: 3.59 ± 0.58 μm **Concentration:** Ultrafine: 47.0 ± 20.2 $\mu g/m^3$, Coarse: 89.0 ± 49.5 $\mu g/m^3$	Preliminary report comparing effects of controlled exposures to coarse, fine, and ultrafine CAPs among healthy adults (3 separate studies). A randomized crossover design was used in evaluating effects of coarse CAPs (n=14) and ultrafine CAPs (n=20) relative to filtered air following of 2-h exposures with intermittent exercise. Results compared with previous study of controlled exposure to fine CAPs (Chapel Hill, NC) where subjects did not serve as their own controls (Ghio et al., 2000, 012140) **Time to analysis:** 0-20 h post-exposure.	As was shown with fine CAPs, exposure to coarse CAPs increased the percentage of neutrophils in bronchoalveolar lavage fluid 20 h following exposure. Unlike fine CAPs, coarse CAPs did not increase the percent of monocytes in bronchoalveolar lavage fluid. Ultrafine CAPs were not shown to affect any markers of pulmonary inflammation in bronchoalveolar lavage fluid 18 h after exposure. No CAPs-induced changes in lung function were observed.
Reference: Samet et al. (2009, 191913) **Subjects:** 19 healthy adults **Gender:** 10 M/9 F **Age:** 18-35 yrs	Ultrafine CAPs (Chapel Hii, NC) **Particle Size:** < 0.16 μm **Particle Number/Count:** 120,662 ± 48,232 particles/cm^3 **Concentration:** 49.8 ± 20 $\mu g/m^3$	Subjects exposed for 2 h with intermittent 15 periods of exercise to UF CAPs and filtered air using a randomized crossover study design. **Time to analysis:** Immediately following exposure and 1 and 18 h post-exposure.	No effect of UF CAPs observed on pulmonary function immediately following exposure or 18 h post-exposure. IL-8 in BAL fluid was observed to increase with UF CAPs 18 h post-exposure. UF CAPs was not shown to alter any other inflammatory markers in BAL fluid.
Reference: Schaumann et al. (2004, 087966) **Subjects:** 12 healthy adults **Gender:** 4 M/8 F **Age:** Avg 27 ± 2.5 yrs	Fine PM Collected (filter) from industrialized and non-industrialized areas in Germany **Concentration:** 100 μg per subject	Bronchoscopic instillation of particles collected from both areas was conducted in contralateral lung segments for each subject. **Time to analysis:** 24 h post-instillation.	Particles collected from the industrialized area (transition metal-rich) increased the percentage of monocytes and oxidant radical generation in bronchoalveolar lavage fluid 24 h after exposure compared with PM containing less metal.
Reference: Stenfors et al. (2004, 157009) **Subjects:** 15 asthmatic adults, 25 healthy adults **Gender:** Asthmatic: 10 M/5 F, Healthy: 16 M/9 F **Age:** Asthmatic: 22-52 yrs, Healthy:19-42 yrs	DE Volvo diesel engine **Concentration:** PM_{10} 108 $\mu g/m^3$	Subjects were exposed for 2 h with intermittent exercise to DE and filtered air using a randomized crossover study design. Other diesel emissions measured: NO_2 (0.7 ppm). **Time to analysis:** 1 h after the start of exposure, immediately following exposure, and 6 h post-exposure.	DE was observed to increase neutrophilia and IL-8 in bronchial lavage fluid among healthy subjects 6 h after exposure. Among asthmatic subjects, exposure to DE did not cause an increase in inflammatory markers. No diesel-induced change in pulmonary function was observed during or immediately following exposure.
Reference: Tunnicliffe et al. (2003, 088744) **Subjects:** 12 asthmatic adults, 12 healthy adults **Gender:** Asthmatics: 7 M/5 F, Healthy: 5 M/7 F **Age:** Asthmatics: avg 35.7 yrs, Healthy: avg 34.5 yrs	Aerosols of ammonium bisulfate and sulfuric acid **Particle Size:** MMD 0.3 μm **Concentration:** 200, 2,000 $\mu g/m^3$	Subjects were exposed for 1 h at rest to ammonium bisulfate (low and high concentrations), sulfuric acid (low and high concentrations) and filtered air in a randomized crossover design. Exposures were separated by at least 2 wks and were conducted using a head dome exposure system. **Time to analysis:** Immediately following exposure as well as 5.5-6 h post-exposure.	Neither ammonium bisulfate nor aerosolized sulfuric acid were not observed to affect lung function or respiratory systems following exposures to 200 or 2,000 $\mu g/m^3$ among healthy or asthmatic adults. Exposures to ammonium bisulfate at both concentrations resulted in a significant increase in eNO in the asthmatic subjects.

Table C- 3. Central Nervous System Effects

Reference	Pollutant	Exposure	Findings
Reference: Cruts et al. (2008, 156374) **Subjects:** 10 healthy adults **Gender:** M **Age:** 18-39 yrs	DE Idling Volvo diesel engine (TD45, 4.5 L, 4 cylinders, 680 rpm) **Particle Number/Count:** 1.2 × 106/cm^3 **Concentration:** 300 $\mu g/m^3$	Subjects were exposed to DE and filtered air for 1 h at rest in a randomized crossover study design. Exposures were separated by 2-4 days. Other diesel emissions measured: NO_2 (1.6 ppm), NO (4.5 ppm), CO (7.5 ppm), total hydrocarbons (4.3 ppm). **Time to analysis:** From onset of exposure until 1 h post-exposure.	Exposure to DE was observed to significantly increase the median power frequency (MPF) in the frontal cortex during exposure, as well as in the hour following the completion of the exposure.

Annex C References

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Note: Hyperlinks to the reference citations throughout this document will take you to the NCEA HERO database (Health and Environmental Research Online) at http://epa.gov/hero. HERO is a database of scientific literature used by U.S. EPA in the process of developing science assessments such as the Integrated Science Assessments (ISA) and the Integrated Risk Information System (IRIS).

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Annex D. Toxicological Studies

Table D-1. Cardiovascular effects.

Study	Pollutant	Exposure	Effects
Reference: Anselme et al. (2007, 097084) **Species:** Rat **Gender:** Male **Strain:** Wistar **Age:** Adult **Weight:** 200-225g	**DE:** monocylinder Diesel engine using Euro 4 ELF 85A reference gasoline **Particle Size:** DE: 10-650 nm (85 nm mean mobility diameter)	**Route:** Whole-body Inhalation **Dose/Concentration:** DE: 0.5 mg/m^3; Other emissions measured: non-methane hydrocarbons (7.7 ppm), NO_2 (1.1 ppm), CO (4.3 ppm) **Time to Analysis:** Experiments started 3 months after L coronary artery ligation. ECG started at t0 and the DE exposure at t30 min for a 3h period; ventricular premature beats (VPBs) and RMSSD calculated every 30 min during clean room air exhaust and PE periods. Early (t210-300 min) and late (t480-540 min) PE were analyzed.	Immediate decrease in RMSSD was observed in both healthy and CHF rats PE. Immediate increase in VPBs observed in CHF rats only; which lasted 4-5h after exposure ceased. Whereas HRV progressively returned to baseline values within 2.5 h post-exposure (PE), the proarrhythmic effect persisted as late as 5 h PE termination in CHF rats
Reference: Bagate et al. (2004, 055638) **Species:** Rat **Gender:** Male **Strain:** SH **Age:** 13-15wks	**LPS and EHC-93 (PM):** Urban Air collected at the Health Effects Institute Ottawa, Canada **Particle Size:** EHC-93: 0.8-0.4 μm (mean) (range: $<$ 3 μm)	**Route:** IT Instillation **Dose/Concentration:** PM: 10 mg/kg of bw; LPS- 350 EU/animal **Time to Analysis:** Sacrificed 4 or 24 h post-instillation	PM and LPS elicited a significant increase in receptor-dependent vasorelaxation of the aorta compared to saline-instilled rats.
Reference: Bagate et al. (2004, 055638) **Species:** Rat **Gender:** Male **Strain:** SH **Age:** 13-15wks	EHC-93 (PM), CB-V or CB-Fe, LPS **Particle Size:** EHC-93: 0.8-0.4 μm (mean) (range: $<$ 3 μm)	**Route:** Aortic Suspension Fluid **Dose/Concentration:** Cumulative concentrations of EHC-93, CB-V and CB-Fe (10, 25, 50, 75, 100 μg/mL) CB 1.5-2.0 nm (mean) (range $<$ 5 μm) **Time to Analysis:** Immediately post-exposure of aortic rings to cumulative concentrations of EHC-93, CB-V, CB-Fe and LPS.	CB-V particles induced more relaxation than CB-Fe particles or EHC-93 in a dose-dependent manner. PM and LPS had an acute transient effect on the receptor dependent vasorelaxation. PM and LPS attenuated ACh-elicited vasocontraction in denuded aortic rings (DARs).

Note: Hyperlinks to the reference citations throughout this document will take you to the NCEA HERO database (Health and Environmental Research Online) at http://epa.gov/hero. HERO is a database of scientific literature used by U.S. EPA in the process of developing science assessments such as the Integrated Science Assessments (ISA) and the Integrated Risk Information System (IRIS).

Study	Pollutant	Exposure	Effects
Reference: Bagate et al. (2004, 055638) **Species:** Rat **Gender:** Male **Strain:** Wistar Kyoto **Age:** 13-15wks	EHC-93 (PM): Urban Air collected at the Health Effects Institute Ottawa, Canada. EHC-93 filtrate (PMF) Zn^{2+} and Cu^{2+} particles (10,000 and 845 μg PM respectively) **Particle Size:** PM: 4.6 μm (GSD = 3.2)	**Route:** In Vitro **Dose/Concentration:** PM Suspensions: 10-100 μg/mL; $CuSO_4$/$ZnSO_4$ 1-100 μmol; Phe 2 μm; arbacol: 10 μm **Time to Analysis:** Measured immediately after maximum response for each cumulative dose was achieved.	**PM-Induced Contraction:** No effect of suspension or filtrate seen on resting tension of aorta and SMRA. **PM- and Metal-Induced Vasorelaxation:** Cumulative concentrations (10-100 μg/mL) of PM suspension and its water soluble components (PMF) elicited dose-dependent relaxation in aorta. Relaxation induced by particle suspension was higher than relaxation induced by free filtrate. The difference was significant at 100 μg/mL. In SMRA, vasorelaxation similar to aorta's was observed, and the activity of the particle suspension was stronger than the filtrate, with the difference being significant starting at 30 μg/mL. Both Zn^{2+} and Cu^{2+} in sulfate salts (10-100 μmol) induced relaxation in pre-contracted aortic rings, with Cu^{2+} having a greater effect than Zn^{2+} at the same concentration. Ions didn't affect ACh relaxation. **Effect of PM on α-Adrenergic Contraction:** Phenylephrine-induced dose-response contraction, starting at 1μM with max at 100 μmol. Pre-treatment of SMRA did not change the phenylephrine-induced contraction.
Reference: Bagate et al. (2006, 097608) **Species:** Rat **Gender:** Male **Strain:** Wistar Kyoto (WKY) and SH **Age:** 13-15wks	EHC-93 (PM) EHC-93 (Filtrate) Cu^{2+} and Zn^{2+} solutions **Particle Size:** PM: 4.6 μm (GSD = 3.2)	**Route:** In Vitro **Dose/Concentration:** PM and PMF Suspensions: 10-100 μg/mL; $CuSO_4$ or $ZnSO_4$:10-100 μmol; Phenylephrine: 2 μm; Carbacol: 10 μm **Time to Analysis:** Responses evaluated at maximum of each dose-response.	PM and its soluble components elicited endothelium-independent vasodilation in rat aorta rings. This response is a result of the activation of sGC since its inhibition by NS2028 practically eliminated relaxation. PM suspensions stimulated cGMP production in purified isolated sGC. Neither receptor nor their signaling pathways played a significant role in the direct relaxation by PM or metals. Vasodilation responses were significantly higher in SH than WKY control rats.
Reference: Bagate et al. (2006, 096157) **Species:** Rat **Gender:** Male **Strain:** SH/NHsd **Age:** 11-12 wks **Weight:** 250-350g	EHC-93 (PM): Urban Air collected at the Health Effects Institute Ottawa, Canada. EHC-93 (Filtrate), Zinc (in PM), LPS **Particle Size:** PM: 4.6 μm (GSD = 3.2)	**Route:** IT Instillation **Dose/Concentration:** PM: 10 mg/kg of bw; LPS: 350 EU/animal (0.5 mL) **Time to Analysis:** 4h post-exposure	**Effect of Pretreatment on Baseline parameters of Isolated Perfused Heart:** After PM exposure a slight increase of baseline coronary flow (CF) and heart rate (HR) was noted. In contrast, a significant decrease of left developing ventricular pressure (LDVP) was observed in SH. LPS also elicited a non-significant decrease in LVDP. **Effect of Pretreatment and Ischemia on Cardiac Function:** When SH rats were pretreated with PM or LPS the isolated heart had a reduced ability to recover to baseline levels after occlusion, in comparison with saline treated rats. After occlusion was released CF went back to baseline values. Saline and LPS treated rats, showed a gradual decrease in CF noted during the reperfusion period. Isolated hearts from PM-exposed SH showed a complete restoration of CF and no gradual decrease. The increase of Zn2+ elicited a rapid decrease of LDVP and HR. The impairment of cardiac function measured by LDVP and HR started immediately upon Zn2+ infusion and remained the same during the perfusion period (no Zn2+ was present in the perfusate).
Reference: Bagate et al. (2006, 096157) **Species:** Rat **Strain:** H9c2 (EACC), cardiomyocyte cells	EHC-93 (PM) Filtrate: Urban Air collected at the Health Effects Institute Ottawa, Canada, $ZnSO_4$ **Particle Size:** PM: 4.6 μm (GSD = 3.2); Carbon Particles: 44nm	**Route:** In Vitro **Dose/Concentration:** PM: 1, 50, 100 μg/mL; $ZnSO_4$: 50 μmol **Time to Analysis:** 30min incubation	**Effect of EHC-93 filtrate on Ca2+ Uptake in Cardiomyocytes:** Both PMF and Zn2+ inhibited ATP or ionophore-stimulated Ca2+ influx in cardiomyocytes.

Study	Pollutant	Exposure	Effects
Reference: Bartoli et al. (2009, 156256) **Species:** Dog **Gender:** Female **Strain:** Mixed breed **Age:** 2-12yrs **Weight:** Average: 15.7kg, Range: 13.6-18.2kg	CAPs (Boston; Harvard Ambient Particle Concentrator) **Particle Size:** Diameter: 0.15-2.5 μm	**Route:** Permanent Tracheostomy **Dose/Concentration:** Concentration range and mean: CAPs: 94.1-1557(358.1 ± 306.7) $\mu g/m^3$, BC: 1.3-32(7.5 ± 6.1) $\mu g/m^3$, Particle count: 3000-69300(18230 ± 13.151articles/cm^3 **Time to Analysis:** Preanesthetized. Tracheostomy. 5h exposures separated by minimum 1wk. Prazosin administered in 8 of 13 dogs 30-60min before exposure. 55 exposure days.	CAPs significantly increased SBP, DBP, mean arterial pressure, HR and rate-pressure product. Prazosin (α-adrenergic antagonist) decreased these CAPs-induced effects. CAPs mass, BC, particle number concentrations were positively and significantly associated with each of the cardiovascular parameters except for pulse pressure.
Reference: Bartoli et al. (2009, 179904) **Species:** Dog **Gender:** Female **Strain:** Mixed breed **Age:** Adult **Weight:** 14-18kg	CAPs (Boston; Harvard Ambient Particle Concentration) **Particle Size:** Diameter: ¤2.5 μm	**Route:** Permanent Tracheostomy **Dose/Concentration:** Concentration range and mean: CAPs: 94.1-1556.8 (349 ± 282.6) $\mu g/m^3$, BC: 1.3-32 (7.5 ± 5.6) $\mu g/m^3$, Particle number: 3000-69300 (20381 ± 13075) $\mu g/m^3$ **Time to Analysis:** Tracheostomy. Minimum 3wk recovery. Acclimatized. Exposed 5h. 2 5min occlusions of LAD coronary artery separated by 20min rest. Exposure days separated by 1wk minimum.	During coronary artery occlusion, CAPs exposure reduced myocardial blood flow and increased coronary vascular resistance, SBP and DBP. CAPs effects were greater in ischemic tissue than nonischemic. Increases in CAPs mass, particle number and BC concentrations were significantly associated with decreased myocardial blood flow and increased coronary vascular resistance.
Reference: Campen et al. (2005, 083977) **Species:** Mouse **Gender:** Male **Strain:** C57BL/6J and Apo $E^{-/-}$ **Age:** 10-12wks	High Whole DE (HWDE); Low Whole DE (LWDE); High PM Filtered (HPMF); Low PM Filtered (LPMF) **Particle Size:** NR	**Route:** Whole-body Inhalation Chambers and Ex-vivo Exposures (isolated, pressurized septal coronary arteries) **Dose/Concentration:** HWDE: PM = 3.6 mg/m^3; NO_x = 102 ppm LWDE: PM = 0.512 mg/m^3; NO_x = 19 ppm; PM = 0.770 mg/m^3; NO_x = 105 ppm LPMF: PM = 0.006 mg/m^3; NO_x = 26 ppm **Time to Analysis:** Whole-body Exposures: DE or PFDE for 6h/day for 3 days, euthanized at the end of last exposure. Coronary Vessels Exposure: PSS bubbled with DE to expose coronary vessels to the soluble contents of DE. Analysis occurred immediately post exposure.	**Whole-body Exposure on ApoE$^{-/-}$:** During DE exposure, ApoE$^{-/-}$ mice HR consistently decreased during high concentration exposures, compared to the C57BL/6J strain. **Coronary Vascular Effects on ApoE$^{-/-}$:** DE had no significant effects on the resting myogenic tone of isolated septal coronary arteries. Control coronary arteries showed constrictive responses to ET-1 and dilatory responses to SNP. DE exposed PSS vessels responses to ET-1 enhanced compared to control. SNP-induced dilation blunted in vessels resting in diesel-exposed saline.
Reference: Campen et al.(2003, 055626) **Species:** Rat **Gender:** Male and Female **Strain:** SH **Age:** 4 m	DE: generated by either of two Cummins (2000 model) 5.9-L ISB turbo engines fueled by Number 2 Diesel Certification Fuel. **Particle Size:** 0.1-0.2 μm aerodynamic diameter	**Route:** Whole-body exposure **Dose/Concentration:** 0, 30, 100, 300, 1000 $\mu g/m^3$ **Time to Analysis:** 6 h/day for 7 days; ECG measurements taken 4 days post-exposure.	**HR:** Significantly higher in exposed animals and not concentration-dependent. More substantial results seen in male rats. **ECG:** The PQ interval was significantly prolonged among exposed animals in a concentration-dependent manner.
Reference: Campen et al. (2006, 096879) **Species:** Mouse **Gender:** Male **Strain:** ApoE$^{-/-}$ **Age:** 10wks	Road dust from paved surfaces (Reno, NV) Gasoline engine emissions, containing PM, NO_x, CO and HC **Particle Size:** Road dust: 1.6 μm (Standard Deviation 2.0) Gasoline engine emissions: Average particle diameter of 15 nm	**Route:** Whole-body inhalation **Dose/Concentration:** Road dust: 0.5 and 3.5 mg/m^3; Gasoline engine emissions: 5 to 60 $\mu g/m^3$ (at dilutions of 10:1, 15:1, and 90:1) Mean concentrations of PM: 61 $\mu g/m^3$; NO_x: 18.8 ppm; CO: 80 ppm. **Time to Analysis:** 6h/d for 3d. Sacrificed 18h post-exposure.	**ET-1:** Gasoline exhaust significantly upregulated ET-1 in a dose-dependent manner. ET-1 increased levels in the PM filtered group and decreased in the low levels of road dust. **ECG:** HR consistently decreased from beginning to end of exposure in all groups. No significant HR effects on road dust or gasoline exposure was observed. No significant effects on P-wave, PQ-interval, QRS-interval, or QT-interval were observed in either treatment. **T-wave:** Mice exposed to whole gasoline exhaust displayed significant increases in T-wave morphology from the beginning of exposures; this effect was consistent on all exposure days.

Study	Pollutant	Exposure	Effects
Reference: Cascio et al. (1987, 007583) **Species:** Mouse **Gender:** Male **Strain:** ICR **Age:** 6-10wks	UFPM: Ultra fine PM, EPA Chapel Hill, NC **Particle Size:** $< 0.1\ \mu m$	**Route:** IT Instillation **Dose/Concentration:** 100 μg in 100 ul **Time to Analysis:** 24h post-exposure (single exposure)	UFPM exposure double the size of myocardial infarction attendant to an episode of ischemia and reperfusion while increasing post ischemic oxidant stress. UFPM alters endothelium-dependent/independent regulation of systemic vascular tone; increases platelet number, plasma fibrinogen, and soluble P-selectin levels; reduces bleeding time.
Reference: Chang et al. (2007, 155720) **Species:** Rat **Gender:** Male **Strain:** SH **Age:** 60d	UfCB: Ultra fine carbon blackFerric sulfate $Fe_2(SO_4)_3$ Nickel sulfate $NiSO_4$ **Particle Size:** UfCB	**Route:** IT Instillation **Dose/Concentration:** UfCB: 415 and 830 μg Ferric Sulfate: 105 and 210 μg Nickel Sulfate: 263 and 526 μg Combined UfCB and ferric sulfate: 830 μg UfCB + 105 μg Ferric Sulfate Combined UfCB with Nickel Sulfate: 830 μg UfCB + 263 μg Nickel Sulfate **Time to Analysis:** Single dose, radiotelemetry readings recorded for 72h post exposure.	Both high/low-dose UfCB decreased ANN (normal-to-normal intervals) slightly around the 30th hour, concurrent increases of LnSDNN. LnRMSSD returned to baseline levels after small initial increases. Minor effects observed after low-dose Fe and Ni instilllation; biphasic changes occurred after high-dose instillations. Combined exposures of UfCB and either Fe or Ni resulted in HRV trends different from values estimated from individual-component effects.
Reference: Chang et al. (2007, 155720) **Species:** Rat **Gender:** Male **Strain:** SH **Age:** 10wks	CAPs: collected during a dust storm from Chung-Li, Taipei **Particle Size:** $PM_{2.5}$	**Route:** Nose-only Inhalation **Dose/Concentration:** 315.55 $\mu g/m^3$ **Time to Analysis:** 6h	A linear mixed-effects model revealed sigmoid increases in HR and a sigmoid decrease of QAI during exposure, after an initial incubation period.
Reference: Chang et al. (2004, 055637) **Species:** Rat **Gender:** Male **Strain:** SH **Age:** 60d	CAPs collected in Chung-Li, Taipei (spring and summer periods) **Particle Size:** $PM_{2.5}$	**Route:** Nose-only Inhalation **Dose/Concentration:** Spring exposure: 202.0 $\pm$ 68.8 $\mu g/m^3$; Mean number concentration: 2.30 x 10^5 particles/cm^3 (range: 7.12 x 10^3 - 8.26 x 10^5) Summer exposure: 141.0 $\pm$ 54.9 $\mu g/m^3$; Mean number concentration: 2.78 x 10^5 particles/cm^3 (range: 7.76 x 10^3 - 8.87 x 10^5) **Time to Analysis:** 4d of spring exposure and 6d of summer exposure for 5h each exposure. Parameters measured throughout duration of exposures.	During spring exposures, the maximum increase of heart rate (HR) and blood pressure (BP) were 51.6 bpm and 8.5 mmHg respectively. The maximum decrease of QAI (measures cardiac contractility) noted at the same time was 1.6 ms. Similar pattern was observed during summer exposure, however., the responses were less prominent.
Reference: Chang, et al. (2005, 097776) **Species:** Rat **Gender:** Male **Strain:** SH **Weight**: 200g	CAPs collected in Chung-Li, Taipei **Particle Size:** $PM_{2.5}$ (0.1-2.5μm)	**Route:** Nose-only Inhalation **Dose/Concentration:** 202.0 $\pm$ 68.8 $\mu g/m^3$ **Time to Analysis:** 5h/d for 4d	During the inhalation stage, crude effects of both LnSDNN and LnRMSSD for exposure and control groups decreased from the baseline values. Immediately after the experiments, both LnSDNN and LNRMSSD decreased due to stresses produced by release from the exposure system, then returned to the baseline values.
Reference: Chauhan et al. (2005, 155722) **Tumor Cell Line:** A549 derived from alveolar type II epithelial cells	SRM-1879 (SiO_2) and SRM-154b (TiO_2) from the NIST EHC-93 from Ontario, Canada (EHCsol, EHCinsol) **Particle Size:** EHC-93 median physical diameter: 0.4 μm; TiO_2 and SiO_2 particle size distribution: 0.3-0.6 μm	**Route:** Cell Culture **Dose/Concentration:** 0, 1, 4, and 8 mg EHC total equivalent per 5 mL **Time to Analysis:** Culture medium was removed from flasks and replaced w/ 5 mL of the particle suspension media. Plates were incubated for 24h. After 24h cell culture supernatants were collected and analyzed.	The decreased expression of preproET-1 in A549 cells suggests that epithelial cells may not be the source of higher pulmonary ET-1 spillover in the circulation measured in vivo in response to inhaled urban particles. However, higher levels ECE-1 in A549 post-exposure to particles suggests an increased ability to process bigET-1 into mature ET-1 peptide, while increased receptor expression implies responsiveness. The increased release of Il-8 and VEGF by epithelial cells in response to particles could possibly up regulate ET-1 production in the adjacent pulmonary capillary endothelial cells, with concomitant increased ET-1 spillover in the systemic circulation.

Study	Pollutant	Exposure	Effects
Reference: Chen et al. (2005, 087218) **Species:** Mouse **Strain:** Normal (C57) and ApoE$^{-/-}$	CAPs (NYU, NY) **Particle Size:** $PM_{2.5}$	**Route:** Whole-body Inhalation **Dose/Concentration:** 10 x ambient concentrations 19.7 $\mu g/m^3$ average concentration over 5 months (daily average exposure concentration was 110 $\mu g/m^3$) **Time to Analysis:** 6h/d, 5 d/wk, for 5m. Parameters measured continuously throughout.	Significant decreasing patterns of HR, body temperature, and physical activity for ApoE$^{-/-}$ mice, with nonsignificant changes for C57 mice. SDNN and RMSSD in the late afternoon and overnight for ApoE$^{-/-}$ mice showed a gradual increase for the first 6 weeks, a decline for about 12 more weeks, and a slight turn upward at the end of the study period. For C57 mice, there were no chronic effect changes in SDNN or RNSSD in the late afternoon, and a slight increase after 6 weeks for the overnight period.
Reference: Chen LC and Nadziejkov C, (2005, 087219) **Species:** Mouse **Strain:** Normal (C57), ApoE$^{-/-}$, **Age:** 26-28wks (C57), 39-41wks (ApoE $^{-/-}$), and 18-20wks (LDLr$^{-/-}$ [DK])	CAPs (NYU, NY) **Particle Size:** $PM_{2.5}$	**Route:** Whole-body Inhalation **Dose/Concentration:** Mean exposure concentration: 110 $\mu g/m^3$ **Time to Analysis:** 6h/d, 5d/wk for up to 5m. Sacrificed 3 to 6d after last exposure.	All DK mice developed extensive lesions in the aortic sinus regions. In male DK mice, the lesion areas appeared to be enhanced by CAPs exposure. Plaque cellularity was increased, but there were no CAPs-associated changes in the lipid content. ApoE $^{-/-}$ and DK mice showed prominent areas of severe atherosclerosis. Quantitative measurements showed that CAPs increased the percentage of aortic intimal surface covered by grossly discernible atherosclerotic lesion.
Reference: Corey LM et al. (2006, 156366) **Species:** Mouse **Gender:** Male **Strain:** ApoE$^{-/-}$ **Age:** 11-12m **Weight**: 32.84g (avg)	PM collected November – March between 1996-1999(Seattle, WA) Silica (U.S. Silica Company, Berkeley Springs, WV) **Particle Size:** $PM_{2.5}$	**Route:** Nasal Instillation **Dose/Concentration:** PM: 1.5 mg/kg; Saline: 50ul; Silica: Min-u-Sil 5 in 50 ul saline **Time to Analysis:** Mice monitored for 1d baseline prior to and for 4d following exposure.	After an initial increase in both HR and activity in all groups, there was delayed bradycardia with no change in activity of the animals in the PM and silica exposed groups. In addition, with PM and silica exposure, there was a decrease in HRV parameters.
Reference: Cozzi et al. (2006, 091380) **Species:** Mouse **Strain:** ICR **Age:** 6-10wks	Ultrafine PM (collected continuously over 7 day periods in Oct 2002 in Chapel Hill, NC) **Particle Size:** $<$ 150nm	**Route:** IT Instillation **Dose/Concentration:** 100 μg of PM in vehicle **Time to Analysis:** 24h post-exposure	**Ischemia-Reperfusion:** PM exposure doubled the relative size of myocardial infarction compared with the vehicle control. No difference was observed in the percentage of the vehicle at the risk of ischemia. PM exposure increased the level of oxidative stress in the myocardium after I-R. The density of neutrophils in the reperfused myocardium was increased by PM exposure, but differences in the numbers of blood leukocytes, expression of adhesion molecules on circulating neutrophils, and activation state of circulating neutrophils, 24 h after PM exposure, could not be correlated to the increase I-R injury observed. **Isolated Aortas:** Aortas isolated from PM-exposed animals exhibited a reduced endothelium-dependent relaxation response to ACh.
Reference: Dvonch JT et al. (2004, 055741) **Species:** Rat **Gender:** Male **Strain:** Brown Norway	CAPs, Detroit, MI **Particle Size:** $PM_{2.5}$	**Route:** Whole-body Inhalation Chamber **Dose/Concentration:** Average concentration 354 $\mu g/m^3$ **Time to Analysis:** 8h/d for 3 consecutive days; plasma samples collected 24h post-exposure.	Plasma concentrations of asymmetric dimethylarginine (ADMA) were significantly elevated in rats exposed to CAPs versus filtered air.

Study	Pollutant	Exposure	Effects
Reference: Elder et al. (2004, 055642) **Species:** Rat **Gender:** Male **Strain:** Fischer 344 and SH **Age:** 23m (Fischer); 11-14m (SH) **Weight:** NR	UFP - Ultrafine carbon particles; LPS (Sigma) **Particle Size:** UFP: 36nm (median size)	**Route:** Intraperitoneal Injection (ip) for saline and LPS Whole-body exposure for inhaled particles **Dose/Concentration:** Particles: 150 mg/m^3; LPS: 2mg/kg bw **Time to Analysis:** Single 6h exposure to particles. Sacrificed 24h after ip LPS exposure.	**BAL fluid cells:** Neither inhaled UFP nor ip LPS cause a significant increase in BAL fluid total cells or the percentage of neutrophils in either rat strain. No significant exposure-related alteration in total protein concentration or the activites of LDH and b-glucuronidase. **Peripheral blood:** In both rat strains ip LPS induced significant increase in the number and percentage of circulating PMNs. When combined with inhaled UFP, PMNs decreased, significantly for F-344 rats. Plasma fibrinogen increased with ip LPS in both rat strains with the magnitude of change greater in SH rats. UFP alone decreased plasma fibrinogen in SH rats. Combined UFP and LPS response was blunted, but significantly higher than controls. Hematocrit was not altered in either rat strain by any treatment. **TAT complexes:** With all exposure groups averaged, plasma TAT complexes in SH rats were 6.5 times higher than in F-344 rats. LPS caused an overall increase in TAT complexes for F-344 rats that was further augmented by inhaled UFP. UFP alone decreased response. In SH rats, UFP alone significant increased response and LPS decreased response. **ROS in BAL cells:** In F-344 rats both UFP and LPS have independent and significant effects on DCFD oxidation. Effects were in opposite directions; particles decreased ROS, LPS increased ROS.
Reference: Finnerty et al. (2007, 156434) **Species:** Mouse **Gender:** Male **Strain:** C57BL/6 **Age:** 9 wks **Weight**: 22-2g	Coal Fly Ash (U.S. EPA), Analysis: ($PM_{2.5}$ samples) low unburned carbon (0.53 wt%), moderate levels of transition metals, including (in μg/g): Fe (30, 400), Mg (31, 200), Ti (6, 180), Mn (907), and V (108). **Particle Size:** 1.8 and 2.5μm	**Route:** IT Instillation **Dose/Concentration:** PM: 200 μg; PM+10 μg LPS: 200 μg; PM+100 μg LPS: 200 μg **Time to Analysis:** 18h after IT instillation	**Plasma:** TNF-α significantly increased in both PM+LPS10 and PM+LPS100 treatments. For plasma IL-6, all groups tended to rise with a significant increase in the PM+LPS100 group.
Reference: Floyd et al. (2009, 190350) **Species:** Mouse **Gender:** Male **Strain:** ApoE$^{-/-}$ **Age:** 20wks	CAPs: $PM_{2.5}$ concentrated from Tuxedo, NY (April–Sept 2003) **Particle Size:** NR	**Route:** Whole-body Inhalation **Dose/Concentration:** Avg 120 μg/m^3 (n=6/group) **Time to Analysis:** 6h/d×5d/wk×5m	**Gene Expression:** Microarry gene expression identified 395 genes downregulated and 216 genes upregulated in the aortic plaques. Ontologic analysis identified a list of functional processes associated with gene expression and included: inflammation, tissue development, cellular movement, cellular growth and proliferation, hematological system development and function, lipid metabolism, cardiovascular system function, cellular assembly and organization, and cell death.
Reference: Floyd et al. (2009, 190350) **Species:** Mouse **Gender:** NR **Strain:** ApoE$^{-/-}$ **Age:** 6wks **Weight:** NR	CAPs (Tuxedo, NY) (April-September 2003) (modified VACES system) **Particle Size:** NR	**Route:** Exposure Chamber **Dose/Concentration:** Average concentration: 120 μg/m^3 **Time to Analysis:** Exposed 6h/d, 5d/wk, 5m.	CAPs altered 611 genes of which 306 were significantly related to alterations in molecular pathways and associated with biological pathways. The 50 most significant biological function alterations related to CVD. Further analysis of recent literature showed that some CAPs-altered genes were the same as genes altered by CVD or unstable-human plaque gene expression.
Reference: Folkmann et al. (2007, 097344) **Species:** Mouse **Gender:** Female **Strain:** Wild type and ApoE$^{-/-}$ **Age:** 11-13wks **Weight**: 21g (avg)	DEP: SRM2975 (particulate fraction of exhaust from a filtering system designed for diesel-powered forklifts). **Particle Size:** DEP: NR	**Route:** Intraperitoneal Injection **Dose/Concentration:** 0, 50, 500, 5,000 μg DEP/kg of bw **Time to Analysis:** 6 or 24 h post-ip injection	The expression of inducible nitric oxide synthase (iNOS) mRNA was increased in the liver 6h post-ip injection. The level of oxidized purine bases, determined by formami-dopyrimidine DNA glycosylase sites increased significantly in the liver after 24 h in in mice injected w/ 50μg/kg of bw. There was no indication of systemic inflammation determined as the serum concentration of nitric oxide and iNOS expression, and DNA damage was not increased in the aorta.

Study	Pollutant	Exposure	Effects
Reference: Furuyama et al. (2006, 097056) **Species:** Rat **Cell Type:** Heart Micro vessel Endothelial (RHMVE) Cells	OE-DEP, OE-UFP (from Urawa, Saitama, Japan) OE = Organic Extracts **Particle Size:** NR	**Route:** Cell Culture **Dose/Concentration:** 0, 5, 10, 25 μg/mL of OE-DEP or OE-UFP **Time to Analysis:** Exposed for 0, 6, 12, 24, or 36h	The cell monolayer exposed to 10 μg/mL OE-UFP produced a larger amount of HO-1 than cells exposed to 10 μg/mL OE-UFP. OE-DEP and OE-UFP exposure reduced PAI-1 production by the cells but did not affect the production of thrombomodulin, tissue-type PA, or urokinase-type PA. Increased PAI-1 synthesis in response to treatment with 1ng/mL TNF-α or 0.5ng/mL TGF-β1 was reduced by OE-DEP exposure. Suppression of PAI-1 production by OE-DEP exposure was mediated through oxidative stress and was independent of HO-1 activity.
Reference: Gerlofs-Nijland et al. (2009, 190353) **Species:** Rat **Gender:** Male **Strain:** SH **Age:** 12wks **Weight:** 200-300g	PM (Prague, Czech Republic; Duisburg, Germany; Barcelona, Spain) (Prague and Barcelona coarse PM organic extracts) **Particle Size:** Coarse: 2.5-10 μm, Fine: 0.2-2.5 μm	**Route:** IT Instillation **Dose/Concentration:** 7mg PM/kg body weight **Time to Analysis:** DTPA added to some PM samples preinstillation. Instilled with PM. Necropsy 24h postexposure.	Inflammation (LDH, protein, albumin), cytotoxicity (NAG, MPO, TNF-α), and fibrinogen were increased by PM, and were greatest in the coarse PM fraction. Metal-rich PM had greater inflammatory and cytotoxic effects, and increased fibrinogen and vWF and decreased ACE. PAH content influenced greater inflammation (including neutrophils), cytotoxicity, and fibrinogen. Generally, whole PM and coarse PM were more potent than organic extracts and fine PM, respectively.
Reference: Ghelfi et al. (2008, 156468) **Species:** Rat **Strain:** Sprague-Dawley **Age:** Adult	CAPs CPZ (Capsazepine) (Axxora LLC, San Diego, CA) **Particle Size:** $PM_{2.5}$	**Route:** CAPs: Inhalation via Whole-body Exposure; CPZ: IP Injection or Aerosol **Dose/Concentration:** CAPs: mean mass concentration: 218 $\pm$ 23 μg/m^3; CPZ: 10mg/kg (ip), 500 μmol (aerosol) **Time to Analysis:** Experiment 1: CPZ ip or 20min aerosol pretreatment immediately prior to CAPs exposure. Single CAPs exposure for 5h. Parameters measured immediately following exposure. Experiment 2: CPZ ip pretreatment prior to CAPs exposure. Exposed to CAPs for 5h/day for 4mths. Parameters measured throughout duration of experiment.	CPZ (ip or aerosol) decreased CAPs-induced chemiluminescence (CL), lipid thiobarbituric acid reactive substances (TBARS), and edema in the heart, indicating that blocking TRP receptors, systemically or locally, decreases heart CL. CAPs exposure led to significant decreases in HR and in the length of QT, RT, Pdur and Tpe intervals. These changes were observed immediately upon exposure, and were maintained throughout the 5h period of CAPs inhalation. Changes in cardiac rhythm and ECG morphology were prevented by CPZ.
Reference: Gilmour et al. (2004, 054175) **Species:** Rat **Gender:** Male **Strain:** Wistar (Crl: WI) BR	ufCB (Printex 90 from Frankfurt, Germany) fCB (Huber 990 from UK) **Particle Size:** ufCB: 114 nm (MMAD); fCB: 268 nm (MMAD).	**Route:** Whole-body Inhalation **Dose/Concentration:** ufCB: 1.66 mg/m^3; fCB: 1.40mg/m^3 **Time to Analysis:** Single exposure for 7h. Sacrified and samples taken at 0, 16, and 48h post-exposure.	Exposure to ultrafine, but not fine, CB particles was also associated with significant increases in the total number of blood leukocytes. Plasma fibrinogen factor VIII and vWF were unaffected by particle treatments as was plasma Trolox equivalent antioxidant status.
Reference: Gilmour PS et al.(2005, 087410) **Species:** Human **Cell Types:** Primary Human Monocyte Derived Macrophages (MP); Human Umbilical Vein Endothelial Cells (HUVEC); A549 cells; Human Bronchial Epithelial Cells (16HBE)	PM_{10}: (Carbon Black from Degussa Ltd, Frankfurt, Germany) **Particle Size:** PM_{10}	**Route:** Cell Culture **Dose/Concentration:** PM_{10}: 50 and 100μg/mL **Time to Analysis:** 6 and 20h	The culture media from MPs and 16HBE cells but not A549 cells, exposed to PM_{10} had an enhanced ability to cause clotting. H2O2 also increased clotting activity. Apoptosis was significantly increased in MPs exposed to PM_{10} and LPS as shown by annexin V binding. TF gene expression was enhanced in MPs exposed to PM_{10} and HUVEC tissue factor. tPA gene and protein expression were inhibited.

Study	Pollutant	Exposure	Effects
Reference: Gilmour et al. (2006, 156472) **Species:** Rat **Gender:** Male **Strain:** Wistar Kyoto **Age:** 12-14wks **Weight:** 280–340g	Zinc Sulfate ($ZnSO_4$ in saline solution) **Particle Size:** NR	**Route:** IT Instillation **Dose/Concentration:** 131 μg/kg of bw (2 μmol/kg) **Time to Analysis:** 1, 4, 24, 48h	**Zinc levels in plasma and tissue:** At 1-24 h post-exposure, zinc plasma levels increased to nearly 20% above baseline. **mRNA expression:** Cardiac tissues demonstrated similar temporal increases in expressions of TF, PAI-1 and thrombomodulin mRNA, following pulmonary instillation of Zn. **Cardiac histopathology:** Mild and focal acute, myocardial lesions developed in a few Zn exposed rats. No changes in fibrin deposition or troponin disappearance were observed. At 24 and 48h PE to Zn, increases occurred in levels of systemic fibrinogen an the activated partial thromboplastin time.
Reference: Gong et al. (2007, 091155) **Species:** Human and Mouse **Cell Type:** Human Microvascular Endothelial Cells (HMEC) **Strain:** C57BL/6J **Gender:** Male (mouse) **Age:** 2m (mouse)	Organic DEP extract: collected from exhaust in a 4JB1-type LD, 2.74 liter, 4-cylinder Isuzu diesel engine (provided by Masaru Sagai, Tsukuba, Japan) ox-PAPC: (provided by Judith Berliner, UCLA, CA) In vivo validation: Ultrafine (ufp) and fine (fp) particulate matter **Particle Size:** DEP < 1 μm (diameter)	**Route:** Cell culture; In vivo validation via Whole-body inhalation **Dose/Concentration:** ox-PAPC: 10, 20, and 40 μg/mL; DEP: 5, 15, and 25 μg/mL; DEP (5 μg/mL) + ox-PAPC: 10 or 20 μg/mL In Vivo Valudation: Ufp: $3.24x10^5/cm^3$; fp: $2.7x10^5/cm^3$ In vivo validation: Ufp: < 0.18 μm; fp: < 2.5 μm **Time to Analysis:** 4h In vivo validation: Exposed to CAPs for 5h/day, 3d/wk for 8wks. Sacrificed 24h after last CAPs exposure.	Gene-expression profiling showed that both DEP extract and ox-PAPC co-regulated a large number of genes. U sinfg network analysis to identify co-expressed gene modules, led to the discovery of three modules that were highly enriched in genes that were differentially regulated by the stimuli. These modules were also enriched in synergistically co-regulated genes and pathways relevant to vascular inflammation. **In vivo validation:** Results were validated by demonstrating that hypercholesterolemic mice exposed to ambient ultrafine particles inhibited significant upregulation of the module genes in the liver.
Reference: Goto et al. (2004, 088100) **Species:** Rabbit **Gender:** Female **Strains:** NZW **Age:** NR **Weight:** 2.3 kg	EHC-93 (Ottawa, ON,Canada) CC: Coilloidal Carbon (obtained from Hamburg, Germany) **Particle Size:** EHC-93: PM_{10}; CC: $<$ 1 μm	**Route:** Intrabronchial Instillation **Dose/Concentration:** AMs incubated with EHC-93 or CC: 0.6 ml/kg EHC-93 alone: 1 mL (500 μg/ mL) CC alone: 1mL (1% CC) **Time to Analysis:** WBC counts measured 4 - 168h after BrdU injection. Sacrificed 7d post instillation.	**Lung distribution of PM_{10}:** PM-containing AMs were distributed diffusely. PM-containing AMs were more prevalent in the PM exposed animals. There was no AM-containing particle difference between the CC-exposed and EHC-93-exposed groups. **Monocyte release from Bone Marrow:** EHC exposure increased WBC and band cell counts from 12hours after instillation. Monocyte count was not affected. Labeled monocytes peaked more quickly after DEP exposure (12h vs 16 h for control). There was no observed change in BM monocyte pool. **Cytokine release:** EHC stimulation increased the release of GM-CSF, IL-6, IL-1β, TNF-α, IL-8 and MCP-1. No effect on m-CSF and MIP-1β. CC particles induced increases in IL-6 and TNF-α; other cytokine levels did not differ from control. **Supernatant instillation:** AM + EHC increased circulating WBC and band cell counts. Circulating monocyte counts were unaffected. AM + EHC showed a major increase in fraction and amount of monocyte released as well as faster clearance when compared to control. The BM monocyte pool was similar in all groups. **Monocyte transit time through BM:** Exposure to EHC, CC only shortened the transit time of monocytes as compared to controls. AM + EHC also shortened monocyte transit time whereas AM + CC had a nonsignificant effect.

Study	Pollutant	Exposure	Effects
Reference: Gottipolu RR et al.(2009, 190360) **Species:** Rat **Gender:** Male **Strain:** Wistar Kyoto (WKY), SH **Age:** 14-16wks **Weight:** NR	DE (30-kW (40hp) 4-cylinder indirect injection Deutz diesel engine) (O_2- 20%, CO- 1.3-4.8ppm, NO- < 2.5-5.9ppm, NO_2- < 0.25-1.2ppm, SO_2- 0.2-0.3ppm, OC/EC- 0.3 ± 0.03) **Particle Size:** Number Median Diameter: Low- 83 ± 2nm, High- 88.2nm; Volume Median Diameter: Low- 207 ± 2nm, High- 225 ± 2nm	**Route:** Exposure Chamber **Dose/Concentration:** Low- 507 ± 4 $\mu g/m^3$, High- 2201 ± 14 $\mu g/m^3$ **Time to Analysis:** Exposed 4h/d, 5d/wk, 4wks. Necropsied 1d postexposure.	DE increased neutrophils in a concentration-dependent manner, and GGT activity at the high dose. Particle-laden macrophages were found in DE-exposed rats. DE dose-dependently inhibited mitochondrial aconitase activity. DE caused 377 genes to be differentially expressed within WKY rats, most of which were downregulated, but none in SH rats. However, WKY rats had an expression pattern shift that mimicked baseline expression of SH rats without DE. These genes regulated compensatory response, matrix metabolism, mitochondrial function, and oxidative stress response.
Reference: Graff et al. (2005, 087956) **Species:** Rat **Cell Type:** Ventricular Myocytes	Zinc (Zn); Vanadium (V) **Particle Size:** NR	**Route:** Cell Culture **Dose/Concentration:** 0, 6.25, 12.5, 25, or 50 μm **Time to Analysis:** Toxicity: 24h post exposure **Beat Rate:** 0.5, 1, 2, 4, and 24h PE **PCR:** 6 and 24h PE	**Beat Rate:** There were statistically significant reductions in spontaneous beat rate 4 and 24 h post-exposure (greater reductions were observed with Zn). **Inflammation:** Exposure to Zn or V (6.25-50 μm) for 6h produced significant increases in IL-6, IL-α, heat shock protein 70, and connexin 43 (Cx43). **Impulse Conduction:** 24 h post-exposure, Zn induced significant changes in the gene expression of Kv4.2 and KvQLt, α-1 subunit of L-type Ca channel, Cx43, IL-6, and IL-1α. V produced a greater effect on Cx43 and affected only KvLQT1.
Reference: Gunnison and Chen (2005, 087956) **Species:** Mouse **Gender:** Male **Strain:** F2 generation DK ($ApoE^{-/-}$, $LDLr^{-/-}$) **Age:** 18-20wks	CAPs (Tuxedo, NY) Copollutants measured: O_3 and NO_2. **Particle Size:** 389 ± 2nm	**Route:** Whole-body Inhalation **Dose/Concentration:** CAPs: 131 ± 99 $\mu g/m^3$ (range 13 - 441 $\mu g/m^3$) O_3: 10 ppb NO_2: 4.4 ppb **Time to Analysis:** 6h/d, 5d/wk for approximately 4m. Tissue collection was performed 3-4d after the last day of exposure.	**Gene expression:** In CAPs-exposed heart tissue, the expression of Limd1 and Rex3 were the most consistently affected genes among the exposed mice. Limd1 was down regulated by 1.5-fold or greater from moderate baseline expression. Rex3 showed a relatively small increase in absolute expression.
Reference: Gurgueira et al. (2002, 036535) **Species:** Rat **Gender:** Male **Strain:** Sprague-Dawley **Weight:** 250-300g	CAPs; Carbon Black (CB from Fisher Scientific, Pittsburgh, PA): C (85.9 ± 0.2%); O (13 ± 0.2%); S (1.17 ± 0.02%) ROFA: obtained from an oil-fired power plant (Boston, MA) **Particle Size:** CAPs size range: 0.1-2.5 μm; CB and ROFA ($PM_{2.5}$)	**Route:** Whole-body Exposure Chamber **Dose/Concentration:** CAPs: average mass concentration: 300 ± 60 $\mu g/m^3$; ROFA: 1.7 mg/m^3; CB: 170 $\mu g/m^3$ **Time to Analysis:** CAPs: 1, 3, and 5h; ROFA: 30min; CB: 5h	**Oxidative stress:** Rats breathing CAPs aerosols for 5 h showed significant oxidative stress, determined as in situ chemiluminescence (CL) in the lung, heart, but not in the liver. ROFA also triggered increases in oxidant levels but not particle-free air or CB. Increases in CL showed strong associations with the CAPs content of Fe, Al, Si and Ti in the heart. The oxidant stress imposed by 5h exposure to CAPs was associated with slight, but significant increases in the lung and heart water content, with increased serum levels of lactate dehydrogenase, indicating mild damage to tissues. CAPs inhalation also led to tissue-specific increases in the activities of SOD and catalase.
Reference: Gursinsky et al. (1976, 015607) **Species:** Rat **Cell Type:** Fibroblasts isolated from adult male Wistar rats hearts	Fly ash (TAF98) **Particle Size:** NR	**Route:** In Vitro **Dose/Concentration:** TAF98: 0, 1, 2 3, 10, 25, 50, 100, 200 $\mu g/mL$ **Time to Analysis:** 0, 5, 10, 30, 60, 120min	Brief treatment of fibroblasts with fly ash triggered the immediate formation of ROS. Using phosphospecific antibodies the activation of p38 MAP kinase, p44/42 MAP kinase (ERK1/2) and p70S6 kinase. Prolonged incubation with fly ash increased the expression of collagen 1 and TGF-β1, but decreased mRNA levels of MMP9 and TNF-α. Cell proliferation was inhibited at high concentrations of fly ash. An increase in the level of advanced glycation end product modification of various cellular proteins was observed.

Study	Pollutant	Exposure	Effects
Reference: Hansen et al. (2007, 090703) **Species:** Mouse **Gender:** Female **Strain:** ApoE$^{-/-}$ and C57BL/6J ApoE$^{+/+}$ **Age:** 11-13wks	DEP: SRM-2975 (NIST) **Particle Size:** DEP: 215nm (geometric mean diameter)	**Route:** Intraperitoneal Injection **Dose/Concentration:** DEP: 0, 0.5 and 5 mg/kg of bw; Aorta segments incubated with 0, 10 and 100 μg DEP/mL **Time to Analysis:** Sacrificed 1h after ip injection.	Exposure to 0.5 mg/kg DEP caused a decrease in the endothelium-dependent Ach elicited vasorelaxation in ApoE$^{-/-}$ mice, whereas the response was enhanced in ApoE+/+ mice. No significant changes were observed after administration of 5 mg/kg DEP. K+ or phenylephrine induced constriction was not affected.
Reference: Hansen et al. (2007, 090703) **Species:** Mouse **Gender:** Female **Strain:** ApoE$^{-/-}$ and C57BL/6J ApoE$^{+/+}$ **Use:** Aorta rings used for in-vitro studies	DEP: SRM-2975 (NIST) **Particle Size:** DEP: 215nm (geometric mean diameter)	**Route:** Cell Culture **Dose/Concentration:** 0, 10 and 100 μg DEP/mL **Time to Analysis:** Basal tone measured at 5 different points throughout experiment.	Exposure to 100 μg DEP/mL enhanced ACh-induced relaxation and attenuated phenylephrine-induced constriction. Vasodilatation induced by sodium nitroprusside was not affected by any DEP exposure.
Reference: Harder et al. (2005, 087371) **Species:** Rat **Gender:** Male **Strain:** Wistar Kyoto **Age:** 12-15wks	Carbon UFPs ((generated by Electric Spark Generator GFG 1000; Palas, Karlsruhe, Germany) **Particle Size:** 37.6 ± 0.7nm (mean)	**Route:** Whole-body Exposure Chamber**Dose/Concentration:** 180 μg/m^3 **Time to Analysis:** Days 1-3: baseline reading, Day 4: exposure to UFPs or filtered air for 4 or 24h then sacrificed immediately following exposure period OR Sacrificed following 1-3d recovery period.	**Cardiovascular Performance:** Mild but consistent increase in heart rate (HR), which was associated with a significant decrease in HR variability during exposure (particle-induced alteration of cardiac autonomic balance, mediated by a pulmonary receptor activation). **Lung Inflammation and Acute-Phase Response:** BALF revealed significant but low-grade pulmonary inflammation. **Effects on Blood:** There was no evidence of an inflammation-mediated increase in blood coagulability; no changes in plasma fibrinogen or factor VIIa. **Pulmonary and Cardiac Histopathology:** Sporadic accumulation of particle-laden macrophages found in the alveolar region. No signs of cardiac inflammation or cardiomyopathy. **mRNA Expression Levels:** No significant changes in the lung or heart.
Reference: Harder et al. (2005, 087371) **Species:** Rat **Gender:** Male **Strain:** Wistar Kyoto **Age:** 14-17wks **Weight:** NR	Carbon UFP **Particle Size:** Diameter: 37.6±0.7nm	**Route:** Exposure Chamber **Dose/Concentration:** 180 μg/m^3 **Time to Analysis:** Telemeter implanted into peritoneal cavity. 10d recovery. 3d baseline reading. 24h exposure. 3d recovery.	Carbon UFP mildly but significantly elevated HR compared to the control. SDNN was significantly decreased during exposure. UFP induced mild pulmonary inflammation, significantly increased PMN, and increased the total protein and albumin concentrations. Particle-laden macrophages sporadically accumulated in the alveolar region.
Reference: Hirano et al. (2003, 097345) **Species:** Rat **Cell Types:** Heart Microvessel Endothelial Cells (RHMVE)	Organic Extracts of DEP (DEP) and Organic Extracts of Ultra Fine Particles (UFP). (Urawa City, Saitama, Japan) **Particle Size:** DEP and UFP: < 2.0 μm	**Route:** Cells Culture **Dose/Concentration:** NAC effects on viability: DEP: 25 μg/ml; UFP: 50 μg/ml mRNA levels for DEP and UFP: 0,1,3,10 μg/ml cell monolayer exposed to DEP and UFP: 1,10,100 μg/ml **Time to Analysis:** mRNA levels measured after 6h incubation with DEP or UFP. Other parameters measured after 24h.	**Cytotoxicity and Oxidative Stress:** LC50 values were 17 and 34 μg/mL for DEP and UFP respectively. The viability of DEP and UFP exposed cells was ameliorated by N-acetyl-L-cysteine (NAC). **mRNA levels:** mRNA levels increased dose-dependently with DEP and HO-1 mRNA showed the most marked response to DEP. mRNA levels of antioxidant enzymes and heat shock protein 72 (HSP72) in DEP-exposed cells were higher than UFP exposed cells at the same concentration. The transcription levels of HO-1 and HSP72 in DEP and UFP-exposed cells were also reduced by NAC.

Study	Pollutant	Exposure	Effects
Reference: Hwang et al. (2005, 089454) **Species:** Mouse **Strain:** Normal (C57) and ApoE$^{-/-}$	CAPs (Tuxedo, NY) **Particle Size:** 389 ± 2nm	**Route:** Whole-body Inhalation **Dose/Concentration:** CAPs Range: 5-627 μg/m^3. Mean CAPs Concentration: 133μg/m^3. Mean Concentrations of Ozone and Nitrogen in CAPs: 10 and 4.4 ppb respectively. **Time to Analysis:** 6h/day, 5d/wk for 5m.	**Long-term Analysis:** Significant decreasing patterns of heart rate (HR), body temperature (T), and physical activity (PA) in ApoE$^{-/-}$ mice. Nonsignificant changes for C57 mice. The chronic effect changes for HR, T, and PA for ApoE$^{-/-}$ mice were maximal in the last three weeks. **Short-term Analysis:** Dose-dependent relationship for HR variations in ApoE$^{-/-}$ mice. **Heart Rate Fluctuation:** HR fluctuations in Apo E$^{-/-}$ mice during the period of 3-6 h increased by 1.35 fold at the end of the exposure and during a 15 min period increases by 0.7 fold at the end of the exposure.
Reference: Inoue et al. (2006, 190142) **Species:** Mouse **Gender:** Male **Strain:** ICR **Age:** 6-7wks	DEP (obtained from a 4Jb1-type light-duty, 4-cylinder, 2.74-L Isuzu diesel engine) Washed DEP (carbonaceous nuclei of DEP after extraction) and DEP-OC (organic chemicals in DEP extracted with CH_2Cl_2); Washed DEP+LPS and DEP-OC+LPS **Particle Size:** $PM_{2.5}$	**Route:** IT Instillation **Dose/Concentration:** Washed DEP: 4mg/kg bw. DEP-OC: 4mg/kg bw. LPS: 2.5mg/kg. Washed DEP+LPS and DEP-OC+LPS: respective additions of LPS to each component prior-experimentation. **Time to Analysis:** Sacrificed 24h post single dose instillation.	Both DEP components exacerbated vascular permeability. The increased fibrinogen and E-selectin levels induced by LPS. This exacerbation was more prominent with washed DEP than with DEP-OC. Washed DEP+LPS significantly decreased protein C and antithrombin-III and elevated circulatory levels of IL-6, KC and LPs without significance.
Reference: Ito et al. (2008, 096823) **Species:** Rat **Gender:** Male **Strain:** Wistar Kyoto (Specific pathogen-free) **Age:** 13-14 wks	CAPs (f-PM) from Inhalation Facility in Yokohama City, Japan. **Particle Size:** 0.1-2.5 μm	**Route:** Whole-body Inhalation **Dose/Concentration:** 0.6-1.5 mg/m^3 **Time to Analysis:** Three groups exposed to: (1) filtered air for 4d, (2) filtered air for 3d and CAPs for 1d or (3) CAPs for 4d. All groups exposed for a maximum of 4.5h/d for 4 consecutive days.	**mRNA expression and cardiovascular function:** In samples of heart tissue, the mRNA of cytochrome P450 (CYP) 1B1, heme oxygenase-1 (HO-1), and endothelin A (ET_A) receptor were up-regulated by CAPs; their levels were significantly correlated with the cumulative weight of CAPs in the exposure chamber. The up-regulation of ET_A receptor mRNA was significantly correlated with the increase in HO-1 mRNA and weakly with the increase in MBP.
Reference: Khandoga A et al. (2004, 087928) **Species:** Mouse **Gender:** Female **Strain:** C57B1/6 **Age:** 5-7wks	UFPs: Ultra fine carbon black particles (Printex 90) **Particle Size:** 14nm diameter (60 % $<$ 100nm)	**Route:** Aortic Infusion **Dose/Concentration:** 1 x 10^7 and 5 x 10^7 total particles infused 300 m^2/g surface area **Time to Analysis:** Single exposure, analysis 2h post exposure	**Platelet effects:** Application of UFPs caused significantly enhanced platelet accumulation on endothelium of postsinusodal venules and sinusoids in healthy mice. UFP-induced platelet adhesion was not preceded by platelet rolling but was strongly associated with fibrin deposition and an increase in vWF expression on the endothelial surface. **Inflammatory effects:** In contrast, inflammatory parameters such as the number of rolling/adherent leukocytes, P-selectin expression/translocation, and the number of apoptotic cells were not elevated. UFPs did not affect sinusoidal perfusion and Kupffer cell function.
Reference: Knuckles et al. (2007, 156652) **Species:** Rat **Gender:** Female (Pregnant, purchased at gestation day 19) **Strain** Sprague-Dawley: **Age:** 60-90d **Weight:** 300g **Use:** RMCs were harvest from 1d-old neonatal pups using the neonatal rat cardiomyocyte isolation kit	ROFA-L: Leachate **Particle Size:** $<$0.2 μm	**Route:** Cell Culture **Dose/Concentration:** 3.5 μg/mL **Time to Analysis:** 1h	**ROFA-L induced alterations to the RCM transcriptosome:** 38 genes were suppressed and 44 genes were induced PE. Genomic alterations in pathways related to IGF-1, VEGF, IL-2, PI3/AKT, CVD, and free radical scavenging were detected. Global gene expression was altered in a manner consistent with cardiac myocyte electrophysiological remodeling, cellular oxidative stress and apoptosis. **ROFA-L induced alterations to the RCM transcription factor proteome:** ROFA-L altered the transcription factor proteome by suppressing activity of 24 and activating 40 transcription factors out of 149.

Study	Pollutant	Exposure	Effects
Reference: Knuckles et al. (2008, 191987) **Species:** Mouse **Gender:** Male **Strain:** C57BL/6 **Age:** 8-10wks **Weight:** NR	DE (single cylinder Yanmar diesel generator burning #2 certified diesel fuel (Chevron-Phillips, Borger, TX) under 100% load) **Particle Size:** $PM_{2.5}$	**Route:** Whole-body Exposure. Ex Vivo. **Dose/Concentration:** In vivo: 350 $\mu g/m^3$; Ex vivo: $PM_{2.5}$ concentration 2-3mg/m^3 flow rate 500mL/min **Time to Analysis:** Exposed 4h. Ex vivo assays.	**Veins:** DE increased vascular reactivity to ET-1. Ex vivo exposed vessels had greater vasoconstriction. L-NAME (an arginine blocker) did not promote constriction in DE-exposed rats but did so in controls. **Arteries:** DE did not significantly alter vascular reactivity. Carbonyls or alkanes alone or with DE did not alter vasoconstriction.
Reference: Kodavanti et al. (2008, 155907) **Species:** Rat **Gender:** Male **Strain:** Wistar Kyoto (WKY) **Age:** 12-14wks	G1: saline (control); G2: Mount Saint Helen's ash (SH); G3: whole suspension of oil combustion PM at high concentration (PM-HD); G4: whole suspension of oil combustion PM at low concentration (PM-LD); G5: saline-leachable fraction of PM high-concentration suspension; G6: ZnS 7H20 **Particle Size:** $PM_{2.5}$	**Route:** IT Instillation **Dose/Concentration:** Doses (mg/kg/week) are for 8 and 16 weeks (PM-solid and soluble Zn) respectively. G1: 0.00-0.00 and 0.00-0.00; G2: 4.60-0.00 and 2.30-0.00; G3: 4.60-66.8 and 2.30-33.4; G4: 2.30-33.4 and 1.15-16.7; G5: 0.00-66.8 and 0.00-33.4; G6: 0.00-66.8 and 0.00-33.4 **Time to Analysis:** 1x/wk for 8 or 16wks; analyzed 48h after last instillation.	**DNA damage (left ventricular tissue):** All groups except MSH caused varying degrees of damage relative to control. Total cardiac aconitase activity was inhibited in rats receiving soluble Zn. Analysis of heart tissue revealed modest changes in mRNA for genes involved in signaling, ion channels function, oxidative stress, mitochondrial fatty acid metabolism, and cell cycle regulation in Zn, but not MSH-exposed rats.
Reference: Kyoso M et al. (2005, 186998) **Species:** Rat **Gender:** NR **Strain:** NR **Age:** 15m	DE PM and NO_x exposures **Particle Size:** NR	**Route:** Whole-body Inhalation **Dose/Concentration:** PM (mg/m^3): 0.01, 0.109, 0.54, 1.09, 0.01 (from 1.09 concentration w/o PM) NO_x (ppm): 0.19, 0.59, 2.60, 5.53, 5.47 (w/o PM) **Time to Analysis:** Exposed 16h/d (from 5pm-9am) for 7m	All of the resting R-R intervals before exposure were lower at night than during the day, but few changes were found after exposure.
Reference: Lei YC et al. (2005, 088660) **Species:** Rat **Gender:** Male **Strain:** Sprague-Dawley **Weight:** 200-250g (upon arrival) **Use:** ip STZ (60 mg/kg bw) dissolved in citric acid buffer administered to 8 rats to induce diabetes; ip citric acid buffer administered to 8 non-diabetic rats	CAPs: Hsin-Chuang, Taipei **Particle Size:** PM: 0.01 - 2.5 μm	**Route:** IT Instillation **Dose/Concentration:** $PM_{2.5}$: 200 μg in 0.5 mL saline. Components ($\mu g/m^3$): Organic Carbon (9.8-SD 2.4)), Elemental Carbon (3.6-SD 3.2), Sulfate (4.8-SD 1.2), Nitrate (6.3-SD 3.4) **Time to Analysis:** Single dose. Animals sacrificed 24h post instillation.	**Effects of Diabetes:** Body weight (bw) of diabetic (D) rats (397.5g) was lower than non-diabetic (ND) rats (483.1g). Mean plasma glucose level was 163 mg/dL in ND rats and 448.2 mg/dL in D rats. D rats had significant greater levels of 8-OHdG in plasma compared to ND rats. D rats had significantly increased levels of plasma [nitrate+nitrite]. No observable changes in TNF-α for D and ND rats. **Effects of PM Exposure ND Rats:** Increase in plasma levels of 8-OHdG and plasma IL-6, TNF-α, and serum CRP. Significant reduction of plasma [nitrate+nitrite]. No significant effect on plasma ET-1. **Effects of PM Exposure STZ-D Rats**: Significant elevation of plasma ET-1. Decrease in plasma [nitrate+nitrite] Plasma 8-OHdG and TNF-α significantly increased. No significant alterations in IL-6 and CRP.
Reference: Lemos M et al. (2006, 088594) **Species:** Mouse **Gender:** NR **Strain:** BALB/c **Age:** 1d (neonatal) **n:** 10 **Weight:** 4-6g	PM_{10}, CO, NO_2, and SO_2 from Universidade de Sao Paulo, Brazil. **Particle Size:** PM_{10}	**Route:** Inhalation via Whole-body exposure **Dose/Concentration:** Mean (± SD) concentrations were: CO_2: 2.06 ± 0.08ppm (8h mean); NO_2: 104.75 ± 42.62 $\mu g/m^3$ (24h mean); SO_2: 11.07 ± 5.32 $\mu g/m^3$ (24h mean); PM_{10}: 35.52 ± 12.84 $\mu g/m^3$ (24h mean) **Time to Analysis:** 24 h/d, 7d/wk for 4m	Morphometric measurements of the ratio between the lumen and the wall (L/W) areas were performed on transverse sections of renal, pulmonary and coronary arteries. A significant decrease of L/W with exposure to air pollution was detected in pulmonary and coronary arteries, whereas no effects of air pollution were observed in renal vessels.

Study	Pollutant	Exposure	Effects
Reference: Li et al. (2005, 088647) **Species:** Human and Rat **Strain:** Sprague-Dawley Rats **Tissues/Cell Types:** Cultured HPAECs; Pulmonary Artery Rings (PARs)	Urban Particles (UPs SRM 1648) Major Constituents (mass fraction in %): Al (3.4), Fe (3.9), K (1.1). Minor Constituents (mass fraction in %): Na (0.43), Pb (0.66), Zn (0.48). Trace Constituents (ng/mg): As (115), Cd (75), Cr (403), Cu (609), Mn (786), Ni (82), Se (27), U (5.5), V (127). **Particle Size:** NR	**Route:** PARs: In vitro organ model HPAECs: grown to 80% confluence **Dose/Concentration:** PARs and HPAECs: 1 to 100 μg/mL; Losartan treatment: 0.2 μmol Captopril treatment: 100 μmol **Time to Analysis:** PARs were exposed to increasing doses of UPs from 1 to 100 μg/mL. Maximum tension was recorded within 5min after each UPs dose. HPAECs: exposed to UPs from 1 to 100 μg/mL for up to 20min	Effects of UPs on the constriction of isolated rat pulmonary PARs and the activation of extracellular signal-regulated kinases 1 and 2 (ERK1/2) and p38 mitogen-activated protein kinases (MAPKs) in HPAECs with or without Losartan at 1-100 μg/mL induced acute vasoconstriction. UPs also produced a time- and dose-dependent increase in phosphorylation of ERK1/2 and p38 MAPK. Losartan pre-treatment inhibited both vasoconstriction and activation of ERK1/2 and p38. The water soluble fraction of UPs was sufficient for inducing ERK1/2 and p38 phosphorylation, which was also inhibited by Losartan. Cu ($CuSO_4$) and V ($VOSO_4$), induced pulmonary vasoconstriction and phosphorylation of ERK1/2 and p38, but only phosphorylation of p38 was inhibited by Losartan. UPs induced activation of ERK1/2 and p38 was attenuated by Captopril.
Reference: Li et al. (2006, 156693) **Species:** Rat, Rabbit, and Human **Tissues/Cell Types:** Pulmonary Artery Rings (PARs) (rat); isolated buffer-infused lungs (rabbits) and cultured HPAECs **Strain:** Sprague Dawley Rats, New Zealand White Rabbits **Weight:** Rat: 200-350g; Rabbit: 2.5-3.0kg	Urban Particles (UPs SRM 1648). Major Constituents (mass fraction in %): Al (3.4), Fe (3.9), K (1.1). Minor Constituents (mass fraction in %): Na (0.43), Pb (0.66), Zn (0.48). Trace Constituents (ng/mg): As (115), Cd (76), Cr (403), Cu (609), Mg (786), Ni (82), Se (27), U (5.5), V (127). **Particle Size:** NR	**Route:** In Vitro **Dose/Concentration:** PARs and HPAECs: 1 to 100 μg/mL; **Time to Analysis:** PARs: treatment given 15min prior to exposure. Exposed to increasing doses of UPs from 1 to 100 μg/mL. Maximum tension was recorded within 5min after each UPs dose. HPAECs: exposed to UPs from 1 to 100 μg/mL for 20 and 120min.	**Effects of UP on H_2O_2 release:** Within minutes after UPs treatment, HPAEC increased H2O2 production that could be inhibited by DPI, APO, and NaN_3. The water soluble fraction of UPs as well as its two transition metal components Cu and V, also stimulated H2O2 production. NaN_3 inhibited H2O2 production stimulated by Cu and V, whereas DPI and APO inhibited only Cu-stimulated H2O2 production. Inhibitors of other H2O2-producing enzymes, including N-methyl-L-arginine, indomethacin, allopurinol, cimetidine, rotenone, and antimycin, had no effects. **Effects of UP-induced H_2O_2 on MAPK activation:** DPI but not NaN_3 attenuated UPs-induced pulmonary vasoconstriction and phosphorylation of ERK1/2 and p38 MAPKs. Knockdown of p47phox gene expression by small interfering RNA attenuated UPs-induced H2O2 production and phosphorylation of ERK1/2 and p38 MAPKs.
Reference: Lippmann et al. (2005, 087453) **Species:** Mouse **Strain:** C57 and ApoE$^{-/-}$	(March-September 2003). Chemical Composition: regional secondary sulfate (SS) characterized by high S, Si, and organic C; resuspended soil (RS) characterized by high concentrations of Ca, Fe, Al, and Si; RO-fired powered emissions of the Eastern U.S. identified by the presence of V, Ni, and Se; and motor vehicle (MV) traffic and other sources. Contributors to Average Mass: SS (56.1%), RS (11.7%), RO combustion (1.4%), MV traffic and other sources (30.9%) **Particle Size:** $PM_{2.5}$	**Route:** Whole-body Inhalation **Dose/Concentration:** $PM_{2.5}$ concentrated ten-fold, producing an average of 113 μg/m^3 **Time to Analysis:** 6h/d, 5d/wk for 5m. Parameters measured daily: during exposure, the afternoon after exposure, and late at night	**Associations between sources and short-term Heart Rate changes**: There were no significant associations betweeb SS, RS, RO, and MV factors and HR in C57 mice at any of the three intervals. There were significant associations between $PM_{2.5}$ and the RS source factor and decreases in HR for the ApoE$^{-/-}$ mice during the daily CAPs exposures but no associations with the other factors. There was no residual association of HR with $PM_{2.5}$ or the RS factor later in the afternoon or late at night. In the afternoon, there was a significant association between decreases in HR and the SS factor for the ApoE$^{-/-}$ mice that had not been present during exposure and did not persist into the night time period. MV traffic and others were not significantly associated with HR during any of these three time periods. For the C57 mice, there were no significant associations of HR with $PM_{2.5}$ or any of its components during any of the three daily time periods. **Associations between sources and short-term HRV changes:** Signal noise during exposures did not permit reliable analyses of HRV changes during the hours of CAP exposure.

Study	Pollutant	Exposure	Effects
Reference: Lippmann et al. (2005, 087453) **Species:** Mouse **Gender:** NR **Strain:** ApoE$^{-/-}$, ApoE$^{-/-}$ LDLr$^{-/-}$, C57BL/6 **Age:** NR **Weight:** NR	CAPs (Sterling Forest, spring-summer 2003) **Particle Size:** $PM_{2.5}$	**Route:** Inhalation **Dose/Concentration:** $PM_{2.5}$ average concentration: 110 $\mu g/m^3$, Long-term average: 19.7 $\mu g/m^3$ **Time to Analysis:** Exposed 6h/d, 5d/wk, 5 or 6m. Semicontinuous EKG recordings.	HR increased in ApoE$^{-/-}$ mice but not C57 mice. HRF increased over the duration of the experiment. Atherosclerotic plaque deposits and coronary artery disease lesions occurred in both CAPs-exposed mice and controls, but invasive lesions were only present in CAPs-exposed mice. A gene affecting circadian rhythm was upregulated in double knockout mice. CAPs activated NF-κB. No inflammation occurred in the pulmonary system.
Reference: Lippman M et al. (2006, 091165) **Species:** Mouse **Gender:** Male **Strain:** ApoE$^{-/-}$ **Age:** 6wks	CAPs from Tuxedo, NY. Component of interest: Ni. **Particle Size:** $PM_{2.5}$	**Route:** Whole-body Inhalation **Dose/Concentration:** Average daily CAPs: 85.6 $\mu g/m^3$ Average daily Ni: 43 ng/m^3 **Time to Analysis:** 6h/d, 5d/wk, for 6m (July 2004-January 2005). 10-second ECG, HR, activity, and body temperature data were sampled every 5min for the duration of the experiment.	For the CAPs-exposed mice, on 14 days there were Ni peaks at approximately 175 ng/m^3 and usually low CAPs and V. For those days back-trajectory analysis identified a remote Ni point source. ECG measurements on CAPs-exposed and sham-exposed mice showed Ni to be significantly associated with acute changes in HR and HRV.
Reference: Lund et al. (2007, 125741) **Species:** Mouse **Gender:** Male **Strain:** ApoE$^{-/-}$ **Age:** 10wks **Use:** Mice were placed on a high fat at the beginning of the exposure.	Varying dilutions of gasoline emissions: (generated using two 1996 model 4.3L General Motors V-6 engines, fueld with conventional, unleaded, non-oxygenated gasoline, equipped with stock exhaust systems). Composition for Hi, Med, and Lo dilutions: PM, NO_x, CO, and Total Hydrocarbons (THC) **Particle Size:** NR	**Route:** Whole-body Inhalation **Dose/Concentration:** FA: PM (2 $\mu g/m^3$), NO_x (0 ppm), CO (0.1 ppm), HC (0.1 ppm); Low (1: 90 dilution of exhaust): PM (8 $\mu g/m^3$), NO_x (2 ppm), CO (9 ppm), HC (0.9 ppm); Mid (1: 20): PM (39 $\mu g/m^3$), NO_x (12 ppm), CO (50 ppm), HC (8.4 ppm); High (1: 12): PM (61 $\mu g/m^3$), NO_x (19 ppm), CO (80 ppm), HC (12 ppm); High-filtered (1:12): PM (2 $\mu g/m^3$), NO_x (18 ppm), CO (80 ppm), HC (12.7 ppm). **Time to Analysis:** 6h/d, 7d/wk for 7wks. Mice were sacrificed within 16h PE. During the study period all animals concurrently exposed to the following: FA: 8 $\mu g/m^3$ and 40 $\mu g/m^3$; PM Whole Exhaust: 60 $\mu g/m^3$; or Filtered Exhaust w/ gases matching the 60 $\mu g/m^3$ concentration.	Inhalation exposure to gasoline engine emissions resulted in increased aortic mRNA expression of matrix metalloproteinase-3 (MMP-3), MMP-7, and MMP-9, tissue inhibitor of MMP-2, ET-1 and HO-1 in ApoE$^{-/-}$ mice; increased aortic MMP-9 protein levels were confirmed through immunochemistry. Elevated ROS were also observed in arteries from exposed animals, despite absence of plasma markers. Similar findings were also observed in the aortas ApoE$^{-/-}$ mice exposed to particle filtered atmosphere, implicating the gaseous components of the whole exhaust in mediating the expression of markers associated with vasculopathy.
Reference: Lund AK et al. (2009, 191159) **Species:** Mouse **Gender:** Male **Strain:** ApoE$^{-/-}$ **Age:** 10wks **Weight:** NR	GEE (conventional unleaded, nonoxygenated, nonreformulated gasoline- ChevronPhillips Specialty Fuels Division) **Particle Size:** PM: MMAD- 0.150 μm	**Route:** Inhalation **Dose/Concentration:** PM: 60 $\mu g/m^3$, NO_2: 2ppm, NO: 16ppm, CO: 80ppm, THC: 12.7ppm **Time to Analysis:** Mice fed high-fat diet 30d before exposure. Exposed 6h/d, 1 or 7d. Some groups dosed with Tempol or BQ-123. Killed within 18h of last exposure.	Aorta gelatinase activity increased with GEE exposure time. MMP-2/9 activity spread throughout the vasculature by day 7. 7d GEE exposure significantly increased the aorta protein expression of MMP-9, MMP-2, TIMP-2, and plasma MMP-9. Generally, in GEE-exposed mice, Tempol decreased TBARS and vascular ET-1, and BQ-123 decreased vascular ROS, ET-1, MMP-9, and gelatinase activity.
Reference: Lund AK et al. (2009, 191159) **Species:** Human **Gender:** Male, Female **Strain:** -- **Age:** 18-40yrs **Weight:** NR	GEE (Cummins engine- 5.9L, 205hp, at or near idle conditions, ChevronPhillips- certified commercial #2 fuel) **Particle Size:** PM: MMAD- 0.10 ± 0.02 μm	**Route:** Inhalation **Dose/Concentration:** PM: 100 $\mu g/m^3$, NO_2: 0.4ppm, NO: 3.5ppm, CO: 9ppm, THC: 0.9ppm **Time to Analysis:** Exposed 2h on separate occasions. 4 cycles of 15min rest then 15min on exercise bike. Blood collected preexposure and 30min and 24h postexposure. Plasma samples collected.	GEE significantly increased plasma MMP-9 activity and concentration uniformly among the subjects. DE significantly upregulated plasma ET-1 and NO_x.
Reference: Montiel-Davalos et al.(2007, 156778) **Species:** Human **Cell Types:** HUVEC (from primary human endothelial cells) and U937 (human leukemia pro-monocytic) cell cultures.	$PM_{2.5}$ and PM_{10} from Mexico City **Particle Size:** $PM_{2.5}$, PM_{10}	**Route:** In Vitro **Dose/Concentration:** HUVEC TNF-α (10 ng/mL), and a PM range of 5, 10, 20, and 40 $\mu g/cm^2$ concentrations. **Time to Analysis:** 6 or 24 h (early and late adhesion molecules respectively)	Results showed that both $PM_{2.5}$ and PM_{10} induced the adhesion of U937 cells to HUVEC, and their maximal effect was observed at 20 $\mu g/cm^2$. This adhesion was associated w/ an increase in the expression of all adhesion molecules evaluated for PM_{10}, and E-selectin, P-selectin, and ICAM-1 for $PM_{2.5}$. In general the maximum expression of adhesion molecules induced by $PM_{2.5}$ and PM_{10} was obtained w/ 20 $\mu g/cm^2$; however PM_{10}-induced expression was observed from 5 $\mu g/cm^2$. E-selectin and ICAM-1 had the strongest expression in response to particles.

Study	Pollutant	Exposure	Effects
Reference: Moyer et al. (2002, 052222) **Species:** Mouse **Gender:** Male and Female **Strain:** B6C3F1	In phosphide (InP), Co sulfate heptahydrate ($CoSO_47H_2O$), Vanadium pentoxide(V_2O_5) Gallium arsenide (GaAs), Ni oxide (NiO), Ni subsulfide (Ni_3S_2), Ni sulfate hexahydrate ($NiSO_4 \cdot 6H_2O$), talc, and Mo trioxide ($(MoO_3$) **Particle Size:** MMAD particle size (μm): InP (1.1-1.3), $CoSO_47H_2O$ (1.5-1.8), V_2O_5: (1.0), GaAs: (1.0)	**Route:** Inhalation **Dose/Concentration:** High-Dose Concentration in Chronic Studies, Male ($\mu g/m^3$): InP: 0.3, $CoSO_47H_2O$: 3.0, V_2O_5: 4.0, GaAs: 1.0 High-Dose Concentration in Sub-Chronic Studies, Male or Female ($\mu g/m^3$): InP: 100, $CoSO_47H_2O$: 30, V_2O_5: 16, GaAs: 75 **Time to Analysis:** Phase One: Evaluation of heart, kidney and lung tissues from all control and high dose male B6C3F1 mice exposed by inhalation to 9 particulate compounds for a 2yr period. Phase Two: evaluated heart, lung, kidney and mesentery tissues of control and high dose male and female B6C3F1 mice from the 90d studies of the 4-compounds demonstrating arteritis after a 2yr period.	**Phase One:** High-dose males developed significantly increased incidences of arteritis over controls in 2 of the 9 studies (InP and $CoSO_47H_2O$), while marginal increases of arteritis were detected in 2 additional studies ($V_2O_5$and GaAs). In contrast, arteritis of the muscular arteries of the lung was not observed. Morphological features of arteritis in these studies included an influx of mixed inflammatory cells including neutrophils, lymphocytes, and macrophages. Partial and complete effacement of the normal vascular wall architecture, often with the extension of the inflammatory process into the periarterial connective tissue, was observed. **Phase Two:** Results showed that arteritis did not develop in the 90-day studies, suggesting that long-term chronic exposure to lower-dose metallic PM may be necessary to induce or exacerbate arteritis.
Reference: Mutlu GM et al. (2007, 121441) **Species:** Mouse **Gender:** Male **Strain:** 57BL/6 ($IL6^{+/+}$ and $IL6^{-/-}$) **Age:** 6-8wks **Weight:** 20-25g	PM_{10} from ambient air in Düsseldorf, Germany **Particle Size:** PM_{10}	**Route:** IT Instillation **Dose/Concentration:** PM_{10}: 10 μg; Clodronate: 120mg **Time to Analysis:** For alveolar macrophage depletion, clodronate instilled into mice lungs following endotracheal intubation 48h prior to instillation of PM. Parameters measured 24h post-exposure.	Mice treated with PM_{10} exhibited a shortened bleeding time, decreased prothrombin and partial thromboplastin times (decreased plasma clothing times), increased levels of fibrinogen, and increased activity of factors II, VIII, and X. This prothrombotic tendency was associated with increased generation of intravascular thrombin, an acceleration of arterial thrombosis, and an increase in BALF concentration of prothrombotic IL-6. IL-$6^{-/-}$ mice were protected against PM-induced intravascular thrombin formation and the acceleration of arterial thrombosis. Depletion of macrophages by the IT administration of liposomal clodronate attenuated PM-induced IL-6 production and the resultant prothrombotic tendency.
Reference: Nadziejko et al. (2002, 050587) **Species:** Rat **Gender:** Male **Strain:** SH Wistar Kyoto **Age:** 16wks	CAPs ($PM_{2.5}$) from Tuxedo, NY. (SO_2, NO_2 O_3 and NH3 were removed prior to exposure). H_2SO_4 (fine and ultrafine) **Particle Size:** Ultrafine H_2SO_4 mass median diameter: 50-75nm	**Route:** Nose-only Inhalation **Dose/Concentration:** CAPs: 80 and 66 $\mu g/m^3$ (avg 73); Fine H_2SO_4: 299, 280, 119, and 203 $\mu g/m^3$ (avg 225); Ultrafine H_2SO_4: 140, 565, 416, 750 $\mu g/m^3$ (avg 468) **Time to Analysis:** 4h/exposure	Exposure to CAPs caused a striking decrease in respiratory rate that was apparent soon after the start of exposure and stopped when exposure to CAPs ceased. The decrease in respiratory rate was accompanied by a decrease in HR. Exposure of the same animals to fine-particle-size sulfuric acid aerosol also caused a significant decrease in respiratory rate similar to the effect of CAPs. Ultrafine acid had the opposite effect on respiratory rate compared to CAPs.
Reference: Naziejko C et al. (2004, 055632) **Species:** Rat **Gender:** Male **Strain:** F344 **Age:** 18m	PM/CAPs (Tuxedo, NY) UFC (lab generated) SO_2 **Particle Size:** PM (Size Range): 0.5-2.5um; UFC (MMAD): 30-50nm	**Route:** Nose-only Inhalation **Dose/Concentration:** PM ($\mu g/m^3$): 161-200, avg. 180; UFC ($\mu g/m^3$): 500-1280, avg. 890; SO_2 (ppm): 1.2, 1.2, avg. 1.2 **Time to Analysis:** A total of 8 exposures were performed: 2 exposures to CAPs, 2 exposures to UFC, 4 exposures to SO_2. All three pollutants were tested w/ a crossover design so that each group alternated exposure to air and to pollutant. Exposures lasted 4h and were performed at least 1wk apart. Parameters measured throughout duration of experiment.	Old F344 rats had many spontaneous arrhythmias. There was a significant increase in the frequency of irregular and delayed beats after exposure to CAPs. The same rats were subsequently exposed to UFC, SO_2 or air with repeated crossover design. In these experiments there was no significant change in the frequency of any category of spontaneous arrhythmia following exposure to UFC or SO_2.
Reference: Nemmar et al. (2008, 096566) **Species:** Rat **Gender:** Male **Strain:** Wistar Kyoto **Weight:** 440 ± 14g	DEP (SRM 2975) **Particle Size:** < 1 μm	**Route:** Intravenous via the tail vein **Dose/Concentration:** DEP: 0.02mg or 0.1mg DEP/kg (corresponding to about 8 μg or 44 μg DEP/rat) **Time to Analysis:** 48h following systemic administration of saline or DEP	Intravenous administration of DEP (0.1 mg/kg) triggered systemic inflammation characterized by an increase in monocyte an granulocyte numbers. Both doses of DEP caused a reduction of RBC numbers and hemoglobin concentration. TEM analysis of RBC safter in vitro incubation (5 μg/mL) or in vivo administration of DEP, revealed the presence of ultrafine-sized aggregates of DEP within the RBC. Larger aggregates were also taken up by the RBC. The myocardial morphology and capillary bed were not affected by DEP exposure.

Study	Pollutant	Exposure	Effects
Reference: Nemmar A et al. (2007, 156800) **Species:** Rat **Gender:** Male **Strain:** Wistar Kyoto **Age:** 16wks **Weight:** 424 ± 8g	DEP (SRM 2975) **Particle Size:** NR	**Route:** Tail Vein Injection **Dose/Concentration:** 8, 42, or 212 μg DEP/rat (150ul of 0.02, 0.1, or 0.5 mg/kg bw) **Time to Analysis:** 24h	**Effect of DEP on Blood Pressure:** Significant decrease on BP in DEP-exposed rats at doses of 0.02 mg/kg bw, compared with mean BP observed in controls. **Effect of DEP onHR**: Doses of 0.02, 0.1, and 0.5 mg/kg bw in rats, resulted in significant reduction of HR compared to controls. **Effect of DEP on Tail Bleeding Time:** Shortening of tail bleeding time in rats exposed to 0.02, 0.1, and 0.5 mg/kg bw. The shortening was significant at the dose of 0.02 and 0.5 mg/kg compared w/ controls. Platelet counts in blood did not significantly increased post-DEP administration. **Effect of DEP on WBC and RBC numbers:** No significant effect of DEP at doses of 0.02, 0.1 and 0.5 mg/kg on the numbers of granulocytes, monocytes, or lymphocytes compared with control.
Reference: Nemmar et al. (2003, 096567) **Species:** Hamster **Gender:** Male and Female **Weight:** 100-110g	DEP (SRM 1650) **Particle Size:** NR	**Route:** IT Instillation **Dose/Concentration:** 120 μl (5, 50, or 500 μg/animal) **Time to Analysis:** In-vivo: formation and embolization of thrombus were continuously monitored for 40min. Ex-vivo: animals were ITly instilled w/ DEPs (0 or 50 μg per animal), and blood was collected 5, 15, 30, and 60min post-instillation. In-vitro: Saline or saline-containing DEPs (0.1, 0.5, 1, and 5 μg/mL) was added to venous blood from untreated hamsters, and closure time was measured in the PFA-100 after 5min/animal.	Doses of 5 – 500 μg enhanced experimental arterial and venous platelet-rich thrombus formation in-vivo. Blood samples taken from hamsters 30 and 60 min after instillation of 50 μg of DEPs yielded accelerated aperture closure (platelet activation) ex-vivo, when analyzed in the PFA-100. The direct addition of as little as 0.5 μg/mL DEPs to untreated hamster blood significantly shortened closure time in vitro.
Reference: Nemmar et al. (2004, 087959) **Species:** Hamster **Gender:** Male and Female **Weight:** 100-110g	DEP (SRM 1650); Positively Charged Polystyrene Particles (PCPSP) **Particle Size:** PCPSP: 400 nm; DEP: NR	**Route:** IT Instillation **Dose/Concentration:** DEP: 50 μg/animal, or PCPSP: 500 μg/animal **Time to Analysis:** Pretreatment Phase: Hamsters were pretreated w/ Dexametasone IP (5mg/kg) or IT (0.1 or 0.5mg/kg) or Sodium Cromoglycate given IP (40mg/kg), 1h before DEP or vehicle instillation. Thrombosis: In-vivo thrombogenesis assessed 24h post-instillation of DEP or vehicle.	DEP increased thrombosis without elevating plasma vWF. The IT instillation of PCPSP equally produced histamine release and enhanced thrombosis. Histamine in plasma resulted from basophil activation. IP pretreatment with Dexametasone abolished the DEP-induced histamine increase in BALF and plasma and abrogated airway inflammation and thrombogenicity. The IT pretreatment with Dexametasone showed a partial but parallel inhibition of all these parameters. Pretreatment with Sodium Cromoglycate strongly inhibited thrombogenicity, and histamine release.
Reference: Nemmar et al. (2003, 097487) **Species:** Hamster **Gender:** Male and Female **Weight:** 100-110g	Ultrafine Particles: Unmodified Polystyrene Particles (UPSPs); Negatively Charged Carboxylate-Modified Polystyrene Particles (NCC-MPSPs); Positively-Charged Amine Modified Polystyrene Particles (PCA-MPSPs) **Particle Size:** UPSPs: 60nm; NCC-MPSPs: 60nm; PCA-MPSPs: 60 or 400nm	**Route:** IT Instillation **Dose/Concentration:** 5, 50, and 500 μg/animal in 120 ul saline **Time to Analysis:** 1h post-instillation	Unmodified and negative UFPs did not modify thrombosis. Positive UFPs increased thrombosis at 500 and 50 μg/animal, but not at 5 μg/animal. Positive 400 nm particles (500 μg/animal) did not affect thrombosis. PFA-100 analysis showed that platelets were activated by the in-vitro addition of positive UFPs and 400 nm particles to blood.
Reference: Nemmar et al. (2003, 087931) **Species:** Hamster **Weight:** 100-110g	DEP (SRM 1650) **Particle Size:** NR	**Route:** IT Instillation **Dose/Concentration:** 50 μg/animal in 120 ul saline **Time to Analysis:** 1, 3, 6, and 24h	At 1, 6, and 24 h after instillation of 50 μg DEPs, the mean size of in-vivo induced and quantified venous thrombosis was increased by 480, 770, and 460%, respectively. Platelets activation in blood was confirmed by a shortened closure time in the PFA-100 analyzer. In plasma, histamine was increased only at 6 and 24 h. Pre-treatment with a H1 receptor antagonist (diphenhydramine, 30 mg/kg intraperitoneally) did not affect DEP-induced thrombosis or platelet activation at 1 h; however both were markedly reduced at 6 and 24 h.

Study	Pollutant	Exposure	Effects
Reference: Niwa et al. (2007, 091309) **Species:** Mouse **Gender:** Male **Strain:** LDLR/KO **Age, Use:** 6 weeks (n = 20), IT CB dispersion; 10-14wks acute effect of CB dispersion on circulating CRP	Carbon Black **Particle Size:** 23-470nm (mean size 120.7nm)	**Route:** IT Dispersion **Dose/Concentration:** IT CB Dispersion Study: 1 mg per animal/week; Acute Effect of CB Dispersion on Circulating CRP Study: 1mg/animal (single administration) **Time to Analysis:** IT CB Dispersion Study: 1x/wk for 10wks; Acute Effect of CB Dispersion on Circulating CRP Study: Single CB administration, blood samples collected 24h post-administration	**IT CB Dispersion Study:** Although no difference in body weight (bw) between the four groups was observed at baseline, and all mice experienced an increase in bw with advancing age, the mice treated with CB tended to be smaller than those treated with vehicle (air). No significant differences were observed in cholesterol and TG levels among the for groups. Development of aortic lipid-rich lesions occurred in mice under a 0.51% cholesterol diet with or without CB infusion, but not in the mice fed a 0% cholesterol diet. **Acute Effect of CB Dispersion on Circulating CRP Study:** Circulating levels of CRP were significantly higher in mice exposed to CB versus those exposed to air, indicating an acute inflammatory response. Although the presence of CB in pulmonary macrophage-like cells in CB treated mice under 0.51% cholesterol diet was confirmed, CB was not detected in aortas, livers, kidneys, or spleens.
Reference: Niwa et al. (2007, 091309) **Species:** Mouse **Cell Types:** Macrophages Cell Lines (RAW264.7)	Carbon Black (CB); Water-Soluble Fullerene (C60(OH)24); Fluoresbrite Carboxylate Microspheres; Ox-LDL; Acetylated-LDL **Particle Size:** Carbon Black and $C_{60}(OH)_{24}$: 7.1nm (SD 2.4); Fluoresbrite Carboxylate Microspheres: 6nm	**Route:** Cell Culture **Dose/Concentration:** CB: 1, 10, 100 μg/mL; $C_{60}(OH)_{24}$: 20, 100ng/mL **Time to Analysis:** RAW264.7 + CB for 24h, 13d, and 50d; RAW264.7 + C60(OH)24 for 24h or 10d; RAW264.7 + C60(OH)24 for 8d, then co-treated w/ Ox-LDL for an additional 48h; RAW264.7 + Ox-LDL for 5d, and then co-cultured w/ C60(OH)24 for an additional 48h; RAW264.7 + 6nm beads: 3d, the Ox-LDL or acetylated-LDL added for 24h	CB alone had no significant effects on RAW264.7 cell growth. C60(OH)24 alone or CB and C60(OH)24 together w/ Ox-LDL induced cytotoxic morphological changes, such as Ox-LDL uptake-induced foam cell-like formation and decreased cell growth, in a dose-dependent manner. C60(OH)24 induced LOX-1 protein expression, pro-matrix metalloprotease-9 protein secretion, and tissue factor mRNA expression in lipid-laden macrophages. Although CB or C60(OH)24 alone did not induce platelet aggregation, C60(OH)24 facilitated ADP-induced platelet aggregation. C60(OH)24 also acted as a competitive inhibitor of ADP receptor antagonists in ADP-mediated platelet aggregation.
Reference: Niwa Y et al. (2008, 156812) **Species:** Rat **Strain:** Sprague-Dawley **Age:** 6wks	CB from Kyoto, Japan **Particle Size:** Mean size (nm) ± SD determined at 1, 8, 15, 22, and 29d post-exposure was 118.1 ± 2.4, 119.1 ± 2.7, 122.2 ± 2.0, 122.4 ± 2.5 and 121.0 ± 3.6 respectively	**Route:** Whole-body Inhalation **Dose/Concentration:** 15.6 ± 3.5mg/m^3 **Time to Analysis:** 6h/d, 5d/wk, for a total of 4wks. BP and HR were measured by tail-cuff plethysmography at 1, 14, and 28d post - exposure. Sacrificed At 1, 7, 14, 28, and 30d post-exposure	Although the presence of CB was confirmed in pulmonary macrophages, electron microscopic survey did not detect CB in other tissues including, liver, spleen and aorta. CB exposure raised blood pressure levels in a exposure-time dependent manner. Levels of circulating inflammatory marker proteins, including monocyte chemo attractant protein-1, IL-6, andCRP, were higher in the CB treated groups than in control groups.
Reference: Nurkiewicz TR et al. (2004, 087968) **Species:** Rat **Gender:** Male **Strain:** Sprague-Dawley **Age:** 7-8wks	ROFA (from Everett, MA) Major metal contaminants are: Fe, Al, V, Ni, Ca, and Z. Main soluble metals are: Al, Ni, and Ca. **Particle Size:** ROFA mean count diameter: 2.2 μm	**Route:** IT Instillation **Dose/Concentration:** ROFA group: 0.1, 0.25, 1, or 2mg/rat. Vehicle control group: 300 ul saline. Particle control group: TiO_2 0.25mg/rat. **Time to Analysis:** After single IT instillation of a particular dose, all rats recovered for 24h.	**Saline Treated Rats:** A23187 dilated arterioles up to 72 ± 7% max. **ROFA and TiO_2 Exposed Rats:** A23187-induced dilation was significantly attenuated. **Sensitivity of Arteriolar Smooth Muscle to NO:** Similar in saline treated and ROFA exposed rats. **Other:** Significant increase in venular leuko-cyte-adhesion and rolling observed in ROFA exposed rats.

Study	Pollutant	Exposure	Effects
Reference: Nurkiewicz et al. (2006, 088611) **Species:** Rat **Gender:** Male **Strain:** Sprague-Dawley **Age:** 7-8wks	ROFA from Everett, MA **Particle Size:** ROFA mean count diameter: 2.2 μm; TiO_2 mean diameter: 1.0 μm	**Route:** IT Instillation **Dose/Concentration:** ROFA group: 0.1 or 0.25 mg/rat. Vehicle control group: 300 ul saline. Particle control group: TiO_2 0.1 or 0.25 mg/rat. **Time to Analysis:** After single IT instillation of a particular dose, all rats recovered for 24h.	**ROFA or TiO_2 Exposure and Arteriolar Dilation:** Exposure caused a dose-dependent impairment of endothelium-dependent arteriolar dilation. **ROFA or TiO_2 Exposure and Arteriolar Constriction:** Exposure did not affect microvascular constriction in response to PHE. **ROFA and TiO_2 and Leukocyte Rolling and Adhesion:** Exposure significantly increased leukocyte rolling and adhesion in aired venules, and these cells were identified as PMN leukocytes. **ROFA and TiO_2 and MPO:** MPO was found in PMN leukocytes, adhering to the systemic microvascular wall. Evidence suggests that some of this MPO had been deposited in the microvascular wall. There was also evidence of oxidative stress in the microvascular wall.
Reference: Nurkiewicz TR et al. (2008, 156816) **Species:** Rat **Gender:** Male **Strain:** SD **Age:** 6-7wks **Weight:** NR	TiO_2 (DeGussa, Sigma-Aldrich; powders put through fluidized-bed aerosol generator) **Particle Size:** Fine- 1 μm, UF- 21nm	**Route:** Whole-body Exposure **Dose/Concentration:** Concentrations: Fine- 3-16mg/m^3; UF- 1.5-12mg/m^3; Dose: Fine- 8, 20, 36, 67, 90 μg; UF- 4, 6, 10, 19, 30 μg **Time to Analysis:** Acclimated 5d. Exposed 4-12h. Sacrificed 24h postexposure.	Particle accumulation within AMs, anuclear macrophages, particle-laden AMs intimately associated with the alveolar wall were all present in exposed rats. Calcium ionophore impaired arteriolar dilation in a dose-dependent manner in UF and fine exposed rats. UF produced greater systemic microvascular dysfunction. Microvascular dysfunction was the same for three groups of rats exposed to 30 μg UF TiO_2 under different conditions.
Reference: Nurkiewicz et al. (2009, 191961) **Species:** Rat **Gender:** Male **Strain:** SD **Age:** 7-8wks **Weight:** NR	Fine TiO_2 (Sigma-Aldrich- (titanium (IV)) oxide, 224227, St. Louis, MO) ($\sim$99% rutile) TiO_2 nanoparticles (DeGussa-Aeroxide TiO_2 P25, Parsippany, NJ) (80% anatase, 20% rutile) **Particle Size:** Fine TiO_2- Primary size: <5 μm, MMAD: 402nm, CMD: 710nm; Nano-TiO_2- Primary size: 21nm, MMAD: 138nm, CMD: 100nm	**Route:** Aerosol Inhalation **Dose/Concentration:** 1.5-16mg/m^3 **Time to Analysis:** Acclimated 5d. Exposed 240-720min. Anesthetized 24h postexposure. Intravital microscopy, NO measurement, microvascular oxidative stress measurement, nitrotyrosine staining.	**Arteriolar Dilation:** Nano-TiO_2 significantly impaired endothelium-dependent arteriolar dilation. Equivalent levels of arteriolar dysfunction were found in fine and nano-TiO_2. Arteriolar dilation in response to abluminal microiontophretic application of SNP was not different from the controls or between the exposure groups. Arteriolar dilation was partially restored by radical scavenging with TEMPOL and catalase, NADPH oxidase with apocynin, and MPO inhibition with ABAH. **Microcirculation:** ROS increased in both groups. Nano-TiO_2 significantly increased the area of tissue containing nitrotyrosine in the lung and spinotrapezius microcirculation. **NO:** Fine and nano-TiO_2 significantly and dose-dependently decreased stimulated NO production in isolated microvessels. NO production was increased by radical scavenging with TEMPOL and catalase or NADPH oxidase with apocynin, and was largest in the fine TiO_2 group.

Study	Pollutant	Exposure	Effects
Reference: Okayama et al. (2006, 156824) **Species:** Rat **Cell Type:** Ventricular Cardiac Myocytes from Wistar Rats, approximately 3d old	DEP (Tsukuba, Japan) DEPE: 5g of DEP in 5 mL PBS containing 0.05% Tween 80. Others: Catalase, LDH, MPG and SOD. **Particle Size:** DEP mass median diameter: 0.34 μm.	**Route:** In Vitro **Dose/ Concentration:** DEPE: 0-100 μg/mL; MPG: 0-1 mM; SOD: 800 U/mL; Catalase: 500 U/mL **Time to Analysis:** Long-Term Exposure to DEPE: cells were incubated for 24 or 48h. Short-Term Exposure to DEPE: 1, 2, 4, or 8 h and then medium containing DEPE was replaced by serum-free medium, and incubated for an additional 24 h. LDH Activity of Supernatant: 24 h post-DEPE exposure. SOD, Catalase, MPG on DEPE-induced Toxicity: SOD, catalase or MPG was added to cells w/ or w/o DEPE & incubated for 4 or 24h. Medium then replaced w/serum-free & cells incubated for another 24h to analysis.	**Cytotoxic Effects of DEPE on Cardiac Myocytes:** DEPE above 20 μg/mL damaged cardiac myocytes in a time and concentration-dependent manner in both long- and short-term exposure conditions. However damage was greater after long-term exposure. LDH activity showed a concentration-dependent increase at higher levels of exposure (greater than 20 μg/mL). **Effects of ROS Scavenging Enzymes and Antioxidant on DEPE-induced Cell Damage:** SOD or catalase attenuated 50 μg/mL DEPE-induced cell damage compared with DEPE-treated groups lacking antioxidant enzymes. Co-incubation with SOD and catalase showed more protective effects towards DEPE-induced cell damage, although these effects were not statistically significant from cells treated with SOD only. MPG attenuated 50 μg/mL DEPE-induced cell damage in a concentration-dependent manner in both long and short-term exposure conditions. Especially in long-term exposure MPG showed strong protective effects against DEPE-induced cell damage. Cell viability was not affected by SOD, catalase, or MPG.
Reference: Proctor et al. (2006, 088480) **Species:** Rat **Gender:** Male **Age:** 12wks **Use:** Thoracic Aorta from cp/cp and +/? Male Rats cp/cp = homozygous for cp gene. Prone to obesity and insulin resistant. +/? = heterozygous for either +/cp or +/+. Lean and metabolically normal.	ROFA from Birmingham, AL **Particle Size:** 1.95 ± 0.18μm aerodynamic diameter of ROFA	**Route:** Protocol 1: Used two aorta rings per each experimental treatment group (4 groups total). Protocol 2: Used four rings. **Dose/Concentration:** Protocol 1: exposed to 12.5μg/mL ROFA-L (at 10mg/mL). Protocol 2: exposed to 1.56, 3.25, 6.26, 12.5 μg/mL ROFA-L (at 10mg/mL). **Time to Analysis:** Protocol 1: Cells treated with 12.5 μg/mL ROFA-L and/or 104mol/L L-NAME for 20min Protocol 2: Parameters measured after ROFA-L only treatment Contractile response to phenylephrine (PE) was measured	ROFA-L (12.5 μg/mL) increased PE-mediated contraction in obese, but not in lean rat aortae. Effect was exacerbated by L-NAME, and it reduced ACh-mediated relaxation in obese and lean aortae. Initial exposure of aortae to ROFA-L caused a small contractile response, which was markedly greater on second exposure in the obese aortae but marginal in lean.
Reference: Radomski A et al. (2005, 091377) **Species:** Rat **Strain:** Wistar-Kyoto **Use:** Vascular Thrombosis	Carbon Nano Particles (CNPs) (purchased from SES Research, Houston, TX): Multiplewall Nanotubes (MWNT); Singlewall Nanotubes (SWNT); C60 Fullerenes (C60CS); Mixed Carbon Nanoparticles (MCN) PM: (SRM1648) (NIST) **Particle Size:** CNPs: NR; PM: 1.4 μm average size	**Route:** Simultaneous single PM injection into femoral vein as $FeCl_3$ injected to induce carotid thrombosis **Dose/Concentration:** 0.5 mL suspension of 50 μg/mL of PM in 0.9% NaCl solution. **Time to Analysis:** Blood flow continuously monitored for 900 seconds.	**Vascular Thrombosis:** $FeCl_3$ induced carotid artery thrombosis and MCN had an amplifying effect in the development of thrombosis. Infusions of MCN, SWNT, and MWNT significantly accelerated the time and rate of development of carotid artery thrombosis in rats. SRM1648 was less effective than CNPs in inducing thrombosis, while C60CS exerted no significant effect on the development of vascular thrombosis.
Reference: Radomski A et al. (2005, 091377) **Species:** Human **Cell Types:** Platelets **Use:** Human platelet aggregation	Carbon Nano Particles (CNPs) (purchased from SES Research, Houston, TX): Multiplewall Nanotubes (MWNT); Singlewall Nanotubes (SWNT); C60 Fullerenes (C60CS); Mixed Carbon Nanoparticles (MCN); PM (SRM1648) **Particle Size:** CNPs: NR; PM: 1.4 μm average size	**Route:** Cell Culture ($2.5X10^8$ platelets/mL) **Dose/Concentration:** CNPs: 0.2-300 μg/mL; PM: 5 - 300 μg/mL **Time to Analysis:** Prostacyclin (PGI2), S-nitroso-glutathione (GSNO), aspirin, 2-methylthio-AMP, phenanthroline, EDTA and Go6976 were pre-incubated w/ platelets for1 min before particle addition. Particles added to platelets and platelet aggregation studied for 8min.	**Platelet Aggregation:** All CNPs, except C60CS, stimulated platelet aggregation (MCN ≥ SWNT > MWNT > SRM1648). All particles resulted in upregulation of GPIIb/IIIa in platelets. In contrast, particles differentially affected the release of platelet granules, as well as the activity of thromboxane-, ADP, matrix metalloproteinase- and protein kinase C-dependent pathways of aggregation. Particle-induced aggregation was inhibited by prostacyclin and GSNO, but not by aspirin.

Study	Pollutant	Exposure	Effects
Reference: Reed MD et al. (2008, 156903) **Species:** Rat **Gender:** Male, Female **Strain:** CDF (F344)/CrlBR, SHR **Age:** NR **Weight:** NR	GEE (two 1996 General Motors 4.3-L V-6 engines; regular, unleaded, non-oxygenated, non-reformulated Chevron-Phillips gasoline, U.S. average consumption for summer 2001 and winter 2001-2002) **Particle Size:** MMAD: 150nm	**Route:** Inhalation Exposure Chamber **Dose/Concentration:** PM: Low- 6.6 ± 3.7 $\mu g/m^3$, Medium- 30.3 ± 11.8 $\mu g/m^3$, High- 59.1 ± 28.3 $\mu g/m^3$ **Time to Analysis:** 2wk quarantine period in chamber. Exposed 6h/d, 7d/wk, 3d-6m. SHR-surgery to implant telemeter in peritoneal cavity. 4wks recovery. ECG data obtained every 15min beginning 3d preexposure, 7d exposure, 4d postexposure.	**Organ Weight:** At 6m exposure, the heart weights of male and female rats increased and male rats' seminal vesicle weight decreased. **Histopathology:** PM-containing macrophages increased by 6m. **Serum Chemistry:** Serum alanine aminotransferase, aspartate aminotransferase, and phosphorus decreased in medium and high-exposure females. **Hematology, Clotting Factors:** Hematocrit, red blood cell count, and hemoglobin dose-dependently increased for both genders at both time points. Plasma fibrinogen increased at 1wk in males. **Lung DNA Damage:** Hypermethylation occurred in medium- and high-exposure male rats at 6m. **BAL:** For both genders in the high-exposure group, LDH and MIP-2 significantly increased at 6m. ROS decreased at 1wk and 6m. Generally, the production of hydrogen peroxide and superoxide decreased in the high-exposure group and medium- and high-exposure groups, respectively. **CV effects in SHR:** Lipid peroxides were significantly increased in males in the high exposure group. TAT complexes decreased in females in the high exposure group. **Removal of Emission PM:** The removal of emission PM strongly linked PM to increased seminal vesicle weight, red blood cell counts, LDH, lipid peroxides, and methylation.
Reference: Rhoden et al. (2005, 087878) **Species:** Rat **Gender:** Male **Strain:** Sprague-Dawley **Age:**Adult **Weight:** 300g	Urban Ambient Particles (UAPs): SRM-1649; CAPs (from Boston, MA) **Particle Size:** NR	**Route:** UAPs: IT Instillation. CAPs: Inhalation **Dose/Concentration:** UAPs: 750 μg suspended in 300 ul saline; CAPs: 700 ± 180 $\mu g/m^3$ **Time to Analysis:** UAPs: 30min post-instillation. CAPs: immediately after 5h exposure period	**Oxidative Stress and HR Function:** UAPs instillation led to significant increases in heart oxidants. HRincreased immediately after exposure and returned to basal levels over the next 30 min. SDNN was unchanged immediately after exposure, but significantly increased during the recovery phase. **Role of ROS in Cardiac malfunction:** Rats were treated with 50 mg/kg NAC 1h prior to UAPs instillation or CAPs inhalation. NAC prevented changes in heart rate and SDNN in UAPs-exposed rats. **Role of the Autonomic Nervous System in PM-induced Oxidative Stress:** Rats were given 5 mg/kg atenolol, 0.30 mg/kg glycopyrrolate, or saline immediately before CAPs exposure. Both atenolol and glycopyrrolate effectively prevented CAPS-induced cardiac oxidative stress.
Reference: Rivero DH et al. (2005, 088653) **Species:** Rat **Gender:** Male **Strain:** Wistar **Age:** 3m **Weight:** ~250g	$PM_{2.5}$, collected from heavy traffic area in Sao Paulo, Brazil. $PM_{2.5}$ Composition (%): S (3.05), As (0.30), Br (0.21), Cl (2.09), Co (2.65), Fe (2.67), La (5.42), Mn (0.64), Sb (0.21), Sc (3.25), Th (8.14) **Particle Size:** $PM_{2.5}$	**Route:** IT Instillation **Dose/Concentration:** 100 or 500 μg of $PM_{2.5}$. **Time to Analysis:** 24h post-instillation	**Blood:** Total reticulocytes significantly increased at both $PM_{2.5}$ doses, while hematocrit levels increased in the 500 μg group. Quantification of segmented neutrophils and fibrinogen levels showed a significant decrease, while lymphocytes counting increased with 100 μg of $PM_{2.5}$. **Pulmonary vasculature:** Significant dose-dependent decrease of intra-acinar pulmonary arteriole lumen/wall ratio was observed in both $PM_{2.5}$ groups. **Wet-to Dry Weight Ratio:** Significant increase in heart wet-to-dry weight ratio was observed in the 500 μg group.

Study	Pollutant	Exposure	Effects
Reference: Rodriquez Ferreira Rivero DH et al. (2005, 088659) **Species:** Rat **Gender:** Male **Strain:** Wistar **Age:** 3m **Weight:** ~250g	$PM_{2.5}$, collected from heavy traffic area in Sao Paulo, Brazil. $PM_{2.5}$ Composition (%): S (3.05), As (0.30), Br (0.21), Cl (2.09), Co (2.65), Fe (2.67), La (5.42), Mn (0.64), Sb (0.21), Sc (3.25), Th (8.14) **Particle Size:** $PM_{2.5}$	**Route:** IT Instillation **Dose/Concentration:** 50 and 100 μg of $PM_{2.5}$. **Time to Analysis:** HR and SDNN were assessed immediately before instillation, 30 and 60min post-instillation.	HR decreased significantly with time, but no significant effect of treatment or interaction between time and treatment was observed. In contrast, there was a significant SDNN interaction between time and treatment. The SDNN decreased 60 min after instillation with $PM_{2.5}$ concentration of 50 and 100 μg.
Reference: Seagrave et al. (2008, 191990) **Species:** Rat **Gender:** Male **Strain:** SD **Age:** 10-12wks **Weight:** 250-300g	GEE (2 1996 General Motors 4.3-L V6 gasoline engines; conventional Chevron Phillips gasoline, U.S. average composition) (CO, NO, NO_2, SO_2, THC) ($PM_{2.5}$ composition- EC, OC, SO_4, NH_4, NO_3) Simulated downwind coal emission atmospheres (SDCAs) (fly ash, gas-phase pollutants, sulfate aerosols, NO, NO_2, SO_2) Paved Road Dust (RD) (Los Angeles, CA; New York City, NY; Atlanta, GA) **Particle Size:** GEE: MMAD- 150nm, RD: 2.6 ± 1.7 μm, SDCA: 0.1-1.0 μm	**Route:** Nose-only Inhalation **Dose/Concentration:** GEE: 60 $\mu g/m^3$, SDCAs: 317-1072 $\mu g/m^3$, RD: 306-954 $\mu g/m^3$; GEE: CO- 104ppm, NO- 16.7ppm, NO_2- 1.1ppm, SO_2- 1.0ppm, THC- 12ppm; SDCAs: CO- < 1ppm, NO- 0.19-0.62ppm, NO_2- 0.10-0.37ppm, SO_2- 0.07-0.24ppm, THC- < 1ppm **Time to Analysis:** Quarantined 2wks. 6h exposure then ip injected. Cannula ligated into trachea and connected to rodent ventilator. Thorax and abdomen opened. Killed after measurements taken.	GEE produced CL in the lungs, heart, and liver. RD produced a significant effect in the heart at the low dose. SDCAs had no effect on CL. RD significantly increased the heart's oxidative stress, as demonstrated by the TBARS levels. GEE did not affect the amount of macrophages or PMN. SDCAs increased macrophages. The RD low dose increased macrophages and PMN. SDCAs increased P_{enh} values and tidal volumes.
Reference: Simkhovich et al. (2007, 096594) **Species:** Rat **Gender:** Female **Strain:** Fischer 344 x Brown Norway hybrid **Age:** 4, 26 m **Use:** Study performed in isolated Langen-dorff-perfused rat hearts	Ultra Fine Particles (UFPs) isolated from industrial diesel reference PM 2975 **Particle Size:** UFPs ◘ 0.1 μm	**Route:** Heart Perfusion (ex-vivo) **Dose/Concentration:** UFPs 12.5, 25, and 37.5 mg. **Time to Analysis:** Hearts perfused w/ UFPs for 30 minutes and analysis conducted every 10min.	Young adult and old hearts demonstrated equal functional deterioration in response to direct infusion of UFPs. Developed pressure in young adult UFPs-treated hearts fell from 101 ± 4 to 68 ± 8 mmHg. In the old UFPs-treated hearts developed pressure fell by 35%. Positive dP/dt was equally affected in the young adult and old UFPs-treated hearts and was decreased by 28% in both groups.
Reference: Sun Q et al. (2005, 186814) **Species:** Mouse **Gender:** Male **Strain:** $ApoE^{-/-}$ **Age:** 16wks	CAPs: $PM_{2.5}$ from Tuxedo, NY. HFCD: High Fat Chow Diet NCD: Normal Chow Diet **Particle Size:** $PM_{2.5}$	**Route:** Whole-body Inhalation **Dose/Concentration:** $PM_{2.5}$: 85 $\mu g/m^3$; Daily concentration: 10.6 (SD 3.4) $\mu g/m^3$ (mean) Average exposure over 6 mth period: 15.2 $\mu g/m^3$. **Time to Analysis:** Study diets fed for at least 10wk prior to exposure to $PM_{2.5}$ or FA. Exposed for 6h/d, 5d/wk for 6m. Sacrificed 15-47d after exposure.	**Vasomotor Function:** Mice fed HFCD and exposed to $PM_{2.5}$ demonstrated an increase in the half-maximal dose for dilation to ACh with no changes in peak relaxation compared to the mice exposed to FA and fed HFCD and NCD. **Atherosclerosis Burden with $PM_{2.5}$:** In vivo MRI imaging of atherosclerosis burden in the abdominal aorta revealed significantly increased plaque burden in the mice fed HFCD compared with the mice fed NCD. Mean (SD) plaque areas in the mice exposed to $PM_{2.5}$ and fed HFCD vs. mice exposed to FA and fed HFCD were 33 (10) vs. 27 (13) units, respectively. **$PM_{2.5}$ and Vascular Inflammation:** A 2.6-fold higher inducible NOS content was apparent in the mice exposed to $PM_{2.5}$ and fed HFCD compared with the mice exposed to FA and fed HFCD chow and a 4-fold increase in the mice exposed to $PM_{2.5}$ and fed NCD compared with the mice exposed to FA and fed NCD.

Study	Pollutant	Exposure	Effects
Reference: Sun et al. (2008, 157033) **Species:** Mouse **Gender:** Male **Strain:** ApoE$^{-/-}$ **Age:** 6wks	CAPs $PM_{2.5}$ Collected from Sterling Forest State Park, Tuxedo NY (40 miles NW of Manhattan) **Particle Size:** $PM_{2.5}$	**Route:** Inhalation Chamber **Dose/Concentration:** Average Concentration of: 85 $\mu g/m^3$ CAPs in chamber. Average exposure over 6m = 15.2 $\mu g/m^3$. **Time to Analysis:** 6h/d, 5d/wk for 6m. Mice received two different diets, high-fat chow and normal-chow.	**Macrophage and Tissue Factor Expression in Aortic Segments**: Tissue Factor (TF) expression was noted predominantly in the extracellular matrix surrounding macrophages, foam cell-rich areas and around smooth muscle cells. **1. High-Fat Diet:** Increased TF and increased macrophage infiltration was observed in the plaques of high-fat chow mice exposed to PM compared to mice exposed to air and high fat diet. **2. Normal Diet:** PM-exposed mice saw an increase in CD68 expression compared to air-exposed. However TF expression was not significantly different in PM exposed normal diet mice compared to control normal diet mice.
Reference: Sun et al. (2008, 157033) **Species:** Human **Cell Lines:** Human Bronchial Epithelial Cells (BEAS-2B); Vascular Smooth Muscle Cells (hSMCs); and Monocytes (THP-1)	Ambient Particles collected from Sterling Forest State Park, Tuxedo, NY (24 h/d for 4wks) **Particle Size:** Particle size ranges: 1. <0.18 μm 2. 1.8 - 2.5 μm or 3. 2.5 - 10 μm	**Route:** In vitro **Dose/Concentration:** 10-300 $\mu g/ml$ **Time to Analysis:** Doses were tested for durations up to 24 h.	Dose durations tested for up to 24-h did not indicate detectable effects on cell viability. **Effect of PM on TF Expression and Activity in hSMCs:** In the PM size range of 1-3 μm, significant increases in TF expression was observed at doses of 100 and 300 μm /mL. In the <0.18 μm size range, significant increase in TF expression was observed at all doses. The particles with sizes 0.18 - 1.0 μm did not induce significant change in TF expression. **Effect of PM on TF Expression and Activity in Monocyte Cells:** TF protein expression increased with <0.18 μm and the 1- 3 μm range particles. Expression was increased in the 0.18-1.0 μm particle range but it was limited compared to the other PM size ranges. In general TF expression was higher in monocytes than in hSMCs cells, but not significantly. **Effect on TF Expression and Activity in Bronchial Epithelial Cells:** 100 $\mu g/mL$ of the 1-3 μm and <0.18 μm particles significantly increased TF expression. **TF mRNA Expression:** TF mRNA was increased rapidly within the first hour in response to SRM-1694a PM. The lowest dose of SRM PM_{10} $\mu g/mL$ induced highest levels of mRNA in hSMCs, no further increase was observed at higher concentrations.
Reference: Sun et al. (2008, 157032) **Species:** Rat **Gender:** Male **Strain:** Sprague-Dawley **Age:** 500-650g	$PM_{2.5}$, Ultra Fine Particle (UFP) or FA **Particle Size:** $PM_{2.5}$; UFP: <0.1 μm	**Route:** Inhalation via Whole-body Exposure **Dose/Concentration:** Mean $PM_{2.5}$ concentration: 79.1 $\pm$7.4 $\mu g/m^3$. Normalized $PM_{2.5}$ over 10wk period: 14.1 $\mu g/m^3$. **Time to Analysis:** 6h/day, 5d/week random exposure to $PM_{2.5}$, UFP, or FA for a total of 10 weeks. At the end of week 9 exposure, rats were infused w/ 0.75 mg/kg/d of AII for 7 days. $PM_{2.5}$, UFP, or FA, continued during AII infusion period. AII = angiotensin II	**Mean Arterial Pressure (MAP):** after AII infusion, MAP was significantly higher in $PM_{2.5}$ -AII vs. FA-AII group. Aortic Vasoconstriction to PE was potentiated with exaggerated relaxation to the Rho-kinase (ROCK) inhibitor Y-27632 and increase in ROCK-1 mRNA levels in the $PM_{2.5}$ -AII group. Superoxide production in the aorta was increased in the $PM_{2.5}$. AII group compared to FA-AII group, inhabitable by apocynin and L-NAME with coordinate upregulation of NAD(PR oxidase subunits p22phox and p47phox and depletion of tetrahydrobipterin.
Reference: Sun et al. (2008, 157032) **Species:** Rat **Gender:** Male **Strain:** Sprague-Dawley **Age:** 500-650g **Use:** Primary Rat Aortic Smooth Muscle Cells (RASMCs), passages 4 to 8 used for the experiment.	$PM_{2.5}$; Ultra Fine Particle (UFP) or Filtered Air (FA) **Particle Size:** $PM_{2.5}$; UFP: <0.1 μm	**Route:** Cell Culture **Dose/Concentration:** UFP, $PM_{2.5}$: 10 or 50 $\mu g/mL$; AII: 100 nmol/L **Time to Analysis:** Exposed to UFP or $PM_{2.5}$ and parameters measured at 0, 1, 3, 6, and 15min. AII = angiotensin II	Exposure to UFPs and $PM_{2.5}$ was associated with an increase in ROCK activity, phosphorylation of myosin light chain, and MYPT1. Pretreatment with N-Acetylcysteine and the Rho kinase inhibitors (Fasudil and Y-27632) prevented MLC and MYPT-1 phosphorylation by UFPs suggesting a Superoxide-mediated mechanism for $PM_{2.5}$ and UFPs effects.

Study	Pollutant	Exposure	Effects
Reference: Sun Q et al. (2009, 190487) **Species:** Mouse **Gender:** Male **Strain:** C57BL/6, c-*fms*^{YFP} (transgenic, yellow fluorescent protein under monocyte-specific promoter) **Age:** 8, 10wks **Weight:** NR	PM (concentrated- northeastern regional background; Tuxedo Park, NY) **Particle Size:** Diameter: 2.5 μm	**Route:** Whole-body Exposure. IT Instillation. **Dose/Concentration:** Exposure chamber (mean): 72.7 μg/m^3, IT: 1.6mg/kg **Time to Analysis:** C57BL/6 mice equilibrated 2wks, fed high-fat chow 10wks. Exposed in vivo 6h/d, 5d, 128d. *fms*^{YFP} rendered diabetic or fed normal chow 10wks. IT instilled with PM 5min, 2X/wk, 10wks.	**Metabolic Impairment:** PM induced insulin, homeostasis model assessment indexes, elevated glucose, and abnormalities in lipid profile consistent with the IR phenotype. **Vascular Endothelium:** PM decreased peak relaxation and ED_{50} to ACH and peak relaxation to insulin. Lower levels of NO release were seen. **Insulin Signaling:** PM reduced the phosphorylation of Akt in intact aorta. PKC-β11 was the only PKC isoform to increase. **Adipose Inflammation, Visceral Adiposity:** PM significantly increased TNF-α, IL-6, E-selectin, ICAM-1, plasminogen activator inhibitor-1, and restin. PM increased visceral and mesenteric fat mass. F4/80$^+$ macrophages in fat tissue and adipocyte size increased. PM downregulated IL-10 and glactose-N-acetylgalactosamine-specific lectin. **YFP Cell Adhesion and Infiltration:** PM increased YFP cells in the adipose tissue, YFP cell infiltration in the mesenteric fat, and YFP cell adhesion to endothelium.
Reference: Tamagawa et al. (2008, 191988) **Species:** Rabbit **Gender:** Female **Strain:** New Zealand White **Age:** 12wks **Weight:** Acute (average)- 2.4 ± 0.2kg, Chronic (average)- 2.7 ± 0.3kg	PM_{10} (urban; Ottawa, Canada) **Particle Size:** Mean diameter: 0.8 ± 0.4 μm	**Route:** Intrapharyngeal Instillation **Dose/Concentration:** Acute- 2.6mg/kg, Chronic- 2mg/kg **Time to Analysis:** Acute animals exposed days 1, 3, 5. Chronic animals exposed 2x 4wks. Killed postexposure.	**Inflammation:** PM_{10} induced more macrophages, AMs, positive and activated AMs, and fewer tissue macrophages. NO, WBC and PMN were only significantly higher in the first two weeks and IL-6 in the first week. **Vascular endothelial function:** PM_{10} significantly reduced Ach-stimulated relaxation and did not alter SNP-stimulated relaxation. A significant inverse relationship between IL-6 and Ach-induced relaxation occurred at week 1 in the acute model and weeks 1 and 2 in the chronic model. **AMs:** The chronic model had a significant correlation between IL-6 and both positive and activated AMs at week 1. A significant inverse relationship occurred between Ach and both the volume fraction of positive and activated AMs.
Reference: Tankersley et al. (2008, 157043) **Species:** Mouse **Gender:** Male **Strain:** C57BL/6, C3H/HeJ, B6C3F1 **Age:** 18, 28m **Weight:** NR	Carbon black (CB) (Wright dust feed particle generator – BGI, Waltham, MA) **Particle Size:** 0.1-1.0 μm	**Route:** Inhalation Chamber **Dose/Concentration:** Average $PM_{2.5}$ concentration- 401 ± 46 μg/m^3, Average PM_{10} concentration- 553 ± 49 μg/m^3 **Time to Analysis:** Exposed 3h/d 4d.	**Hemodynamics:** CB significantly elevated right atrial and ventricular pressures, pulmonary arterial pressure and vascular resistance, all of which were more pronounced in the 28m-old mice. RV contractility (specifically, the ejection fraction and maximum change in pressure over time) reduced in CB-exposed 28m-old mice. **Heart tissue:** CB significantly declined Ca^{2+}-dependent NOS activity and was more pronounced in 28m-old mice, who also had NOS2 upregulated. CB enhanced ROS generation and NOS-uncoupling and was greatest in 28m-old mice. CB also increased MMP2, MMP9, ANP, BNP, which were greatest in 28m-old mice. CB also reduced PKG-1 in 28m-old mice.

Study	Pollutant	Exposure	Effects
Reference: Tankersley et al. (2007, 097910) **Species:** Mouse **Gender:** Male **Strain:**C3H/HeJ and C57BL/6J **Age:** 10wks **Weight:** 22-26g	Carbon Black (CB) and Filtered Air (FA) **Particle Size:** CB: 2.4 μm (MMAD) (GSD 2.75 μm).	**Route:** CB: Whole-body Inhalation Chamber; Sympathetic (S) & Parasympathetic (PS) blockade: IP Injection **Dose/Concentration:** CB: 159 ± 12 μg/m^3; PS (atropine): 0.5mg/kg; S(propanolol): 1mg/kg **Time to Analysis:**Successive 3h CB and FA Exposures: conducted from 9 a.m. to 1 p.m., or at least 3h after dark-to-light transition (exposure period selected based on the nadir in circadian pattern in HR responses). Subgroups of both strains exposed to PS & S blockade.	**FA Exposure with Saline:** A significantly greater 3 h average response occurred in C3 compared with B6 mice. **PS Blockade:** No evident strain difference between C3 and B6 was observed. **S Blockade:** 3 h average HR responses for C3 mice were significantly reduced compared with saline. **CB Exposure:** HR responses were significantly elevated in C3 compared with B6 mice, but these HR responses were not different relative to FA exposure. **S Blockade:** HR was significantly elevated in B6 mice during CB relative to FA, but was not changed in C3 mice.
Reference: Tankersley CG et al. (2004, 094378) **Species:** Mice **Strain:**AKR/J **Age:** ~180d **Use:** Age matched animals	Carbon Black (CB) and Filtered Air (FA) **Particle Size:** CB: 0.1 to 1um.	**Route:** Whole-body Inhalation **Dose/Concentration:** CB average concentration: 160 ± 22 μg/m^3 **Time to Analysis:** FA exposure on day 1, CB exposure 3h/d for 3 consecutive days (days 2-4)	On day 1, HR was significantly depressed during FA in terminally senescent mice. By day 4, HR had significantly slowed due to the effects of 3 days CB exposure. The combined effects of terminal senescence and CB exposure acted to depress HR to an average (± SEM) 445 ± 40 bpm, ~ 80 bpm lower compared to healthy HR responses. The change in rMSSD was significantly greater on day 1 and day 4 in terminally senescent mice, compared to healthy mice. LF/HF ratio was significantly depressed in terminally senescent mice on day 1. By day 4, significant increases in LF/HF were evident in healthy mice during CB exposure. Terminally senescent mice modulated a lower HR without change in the LH/HF ratio during CB exposure.
Reference: Thomson et al. (2005, 087554) **Species:** Rat **Gender:** Male **Strain:** Fischer-344 **Weight:** 200-250g	Urban Ambient Particles (EHC-93) from Ottawa, Canada; Ozone **Particle Size:** Respirable Modes (aerodynamic diameter): 1.3 and 3.6 μm. Non-respirable Mode (aerodynamic diameter): 15 μm	**Route:** Nose-only Inhalation **Dose/Concentration:** EHC-93: 0, 5, 50 mg/m^3; Ozone: 0, 0.4, 0.8ppm **Time to Analysis:** 4h to particles, ozone, or combination of particles and ozone.	Both pollutants individually increased preproET-1, ET-1 and endothelia NOS mRNA levels in the lungs shortly after exposure, consistent w/ the concomitant increase in plasma of ET-1[1-21]. Prepro-ET1 mRNA remained elevated 24 h post-exposure to particles but no after ozone. Both pollutants transiently increased ET-B receptor mRNA expression, while ozone decreased ET-A receptor mRNA levels. Coexposure to particles plus ozone increased lung preproET-1 mRNA but not plasma ET-1[1-21], suggesting alternative processing or degradations of endothelins. This coincided w/ an increase of MMP-2 in the lungs (this enzyme cleaves bigET-1 to ET-1[1-32]).
Reference: Thomson E et al. (2006, 097483) **Species:** Rat **Gender:** Male **Strain:** Fischer-344 **Weight:** 200-250g	Urban Ambient Particles (EHC-93) from Ottawa, Canada; Ozone **Particle Size:** NR	**Route:** Nose-only Inhalation **Dose/Concentration:** EHC-93: 0, 50 mg/m^3; Ozone: 0, 0.8ppm **Time to Analysis:** 4h to particles, ozone, or combination of particles and ozone. Sacrificed immediately following exposure or following 24h recovery.	Circulating levels of both ET-1[1-21] and ET-3[1-21] were increased immediately after exposure to PM and ozone. While expression of preproET-1 mRNA in the lungs increased, expression of preproET-3 mRNA decreased immediately after exposure. PreproET-2 mRNA was not detected in the lungs, and exposure to either pollutant did not affect plasma ET-2 levels. Coexposure to ozone and particles, while altering lung preproET-1 and preproET-3 mRNA levels in a fashion similar to ozone alone, did not cause changes in the circulating levels of the two corresponding peptides.

Study	Pollutant	Exposure	Effects
Reference: Totlandsdal et al. (2008, 157056) **Species:** Rat **Gender:** Male **Strain:** WKY/NCrl and Crl: WI (Han) **Age:** Adult **Weight:** 220-300 g, WKY/NCrl; 250-300 g, Crl: WI (Han) **Use:** Isolation of Primary Rat Epithelial Lung Cells (PRELCs): from WKY/NCrl rats. Isolation of Rat Ventricular Cardiomyocytes and Cardiofibroblasts (RVCMs and RVCFBs) from Crl: WI (Han) rats.	Pigment Black Printex 90 (Frankfurt, Germany); PM: SRM 1648 **Particle Size:** Printex 90: 12-17nm; PM: NR	**Route:** Cell Culture **Dose/Concentration:** Printex 90: 0, 50, 100, 200 or 400 μg/mL; PM: 0, 200 μg/mL **Time to Analysis:** 20h	**Lung cell cultures:** Both particles induced release of IL-6 and IL-1 β, whereas TNF α was only detected upon exposure to PM. **Cardiac Cell Cultures:** IL-6 release was strongly enhanced upon exposure to conditioned media, and markedly exceeded the response to direct particle exposure. IL-1, but not TNF-α, seemed necessary, but not sufficient, for this enhanced IL-6 release. The role of IL-1 was demonstrated by use the use of an IL-1 receptor antagonist that partially reduced the effect of the conditioned media, and by a stimulating effect on the cardiac cell release of IL-6 by exogenous addition of IL-1 α and IL-1 β.
Reference: Tzeng H-P et al. (2007, 097883) **Species:** Rat **Strain:** Wistar **Cell Type:** Primary Vascular Smooth Muscle Cell Culture (VSMCs): isolated from thoracic aortas from 200-250g rats.	Motorcycle Exhaust Particulate Extract (MEPE) collected from a Yamaha motorcycle with a 50 cm^3 two-stroke engine using 95% octane unleaded gasoline. **Particle Size:** PM1, $PM_{2.5}$, PM_{10}	**Route:** In vitro **Dose/Concentration:** 10-100 μg/mL **Time to Analysis:** 3d	Exposure of VSMCs to MEPE (10-100 μg/mL), enhanced serum-induced VSMC proliferation. The expression of proliferating cell antinuclear antigen was also enhanced in the presence of MEPE. VSCMs treated with MEPE induced increase COX-2 mRNA, protein expression, and PGE2 production, whereas the level of COX-1 protein was unchanged. MEPE increased the production of ROS in VSMCs, in a dose-dependent manner. MEPE triggered time-dependent ERK1/2 phosphorylation in VSMCs which was attenuate by antioxidants (NAC, PDTC). The level of translocation of NF-kappaB-p65 in the nuclei of VSMCs was also increased during MEPE exposure. The potentiating effect of MEPE in serum-induced VSMC proliferation was abolished by COX-2 selective inhibitor NS-398, specific ERK inhibitor PD98059, and antioxidants (NAC, PTDC).
Reference: Tzeng H-P et al. (2003, 097247) **Species:** Rat **Strain:** Wistar **Cell Type:** Primary Vascular Smooth Muscle Cell Culture (VSMCs)	Motorcycle Exhaust Particulate Extract (MEPE) collected from a Yamaha motorcycle with a 50 cm^3 two-stroke engine using 95% octane unleaded gasoline. **Particle Size:** NR	**Route:** In vitro **Dose/Concentration:** MEPE: 10 μg/mL; Nifedipine: 10 μmol; Manganese Acetate: 100 μmol; Staurosporine: 1-2 nM; Chelerythrine: 1 μm **Time to Analysis:** 18h	MEPE induced a concentration-dependent enhancement of vasoconstriction elicited by phenylephrine in the organ cultures of intact and endothelium-denuded aortas for 18h. Nifedipine, manganese acetate, and staurosporine, but not chelerythrine, inhibited the enhancement of vasoconstriction by MEPE. ML-9 inhibited the enhancement of vasoconstriction by MEPE. MEPE enhanced the phosphorylation of 20k-Da in rat vascular smooth muscle cells. N-acetylcysteine significantly inhibited the enhancement of vasoconstriction by MEPE. A time-dependent increase in ROS production by MEPE was also detected in primary cultures of VSMCs.

Study	Pollutant	Exposure	Effects
Reference: Upadhay S et al. (2008, 159345) **Species:** Rat **Gender:** Male **Strain:** SHR **Age:** 6m **Weight:** NR	Ultrafine Carbon Particles (UFCP) **Particle Size:** Size- 31 ± 0.3nm, MMAD- 46nm, Surface area concentration- 0.139 m^2(particle)/m^3(air), Mass specific surface area- 807m^2/g	**Route:** Whole-body Exposure **Dose/Concentration:** 172 μg/m^3 **Time to Analysis:** Acclimatized 2d. 1d baseline. 24h exposure. 4d recovery. Sacrificed 1st or 3rd day of recovery.	**Cardiophysiology:** The mean arterial BP and HR increased but returned to baseline levels by the 4th recovery day. SDNN and HRV decreased. RMSSD and LF/HF decreased but were not significant. **Pulmonary Inflammation:** UFCP did not cause pulmonary inflammation. **Pulmonary and Cardiac Tissue:** HO-1, ET-1, ETA, ETB, TF, PAI-1 significantly increased in the lung on the 3rd recovery day. HO-1 was repressed in the heart, but the other markers had slight, nonsignificant increases. **Systemic Responses:** Neutrophil and lymphocyte cell differentials significantly increased on the 1st recovery day. Other blood parameters were unaffected. The plasma renin concentration increased on the first 2 recovery days. Ang I and II concentrations increased on the 1st recovery day but was not significant.
Reference: Wallenborn et al. (2008, 191171) **Species:** Rat **Gender:** Male **Strain:** Wistar Kyoto (WKY) **Age:** 13wks **Weight:** NR	Zinc Sulfate ($ZnSO_4$, aerosolized) **Particle Size:** NR	**Route:** Nose-only Inhalation **Dose/Concentration:** 9.0 ± 2.1 μg zinc/m^3, 35 ± 8.1 μg zinc/m^3, 123.2 ± 29.6 μg zinc/m^3 **Time to Analysis:** Exposed 5h/d, 3d/wk, 16wks. Half of the rats used for plasma/serum analysis, other half for isolation of cardiac mitochondria.	A trend toward increased BALF protein was seen. Cardiac mitochondrial ferritin had a small, significant increase. Mitochondrial succinate dehydrogenase and glutathione peroxidase had small, significant decreases. Subchronic exposure to 100 μg/m^3 caused expression changes of cardiac genes involved with cell signaling events, ion channels regulation, and coagulation. No pulmonary-related effects were seen.
Reference: Wellenius GA et al. (2003, 055691) **Species:** Dog **Gender:** Female **Strain:** Mixed mongrel **Age:** NR **Weight:** 14-17kg	CAPs **Particle Size:** 0.26 ± 0.04 μm	**Route:** Permanent Tracheostomy **Dose/Concentration:** Median: 285.7 μg/m^3, Range: 161.3-957.3 μg/m^3 **Time to Analysis:** Thoracotomy and tracheostomy performed. 5-13wks recovery. Pairs of subjects: exposed 6h/d either 2nd or 3rd exposure time and filtered air other days. 5min preconditioning occlusion. 20min rest interval. 5min experimental occlusion. Some dogs exposed 6h/d, 4d (consecutive), filtered air on day 4.	CAPs increased the ST-segment elevation and remained elevated 24h after exposure. This increase was seen in precordial leads V_4 and V_5. Multivariate regression analyses showed that the mass concentration of Si was significantly associated with the peak ST-segment elevation and integrated ST-segment change. Univariate regression analyses showed Pb to also be significantly associated with these measures. CAPs had no effect on peak heart rate during occlusion or the maximum occlusion-induced increase in heart rate.
Reference: Wellenius GA et al. (2004, 087874) **Species:** Rat **Gender:** Male **Strain:** Sprague-Dawley **Age:** Adult **Weight:** ~ 250 g **Use:** Rat Model for Acute Myocardial Infarction (AMI): Left-ventricular MI induced by thermocoagulation. Animals allowed to recover for at least 12h after surgery.	CAPs; CO **Particle Size:** CAPs: $PM_{2.5}$	**Route:** Whole-body Inhalation Chambers **Dose/Concentration:** CO: 35ppm; CAPs (median concentration): 350.5 μg.m^3; CAPs+CO: (CAPs median concentration): 318.2 μg/m^3 **Time to Analysis:** 1h exposure to CAPs or CAPs+CO for 1h. Exposure to pollutants was preceded and followed by 1h exposure to FA. Exposure experiments were performed during the period of 07/2000 and 01/2003	CO exposure reduced the ventricular premature beat (VPB) frequency by 60.4% during the exposure time compared to controls. This effect was modified by both infarct type and the number of pre-exposure VPBs, and was mediated through changes in hear rate (HR). Overall, CAPs exposure increased VPB frequency during the exposure period, but this did not reach statistical significance. This effect was modified by the number of pre-exposure VPBs. In rats with a high number of pre-exposure VPB, CAPS exposure significantly decreased VPB frequency (67.1%). Overall, neither CAPs nor CO had any effect on HR, but CAPs increased HR in specific subgroups. No significant interactions were observed between the effects of CO and CAPs.

Study	Pollutant	Exposure	Effects
Reference: Wellenius et al. (2006, 156152) **Species:** Rat **Gender:** Male **Strain:** Sprague-Dawley **Age:** Adult **Weight:** ~250g **Use:** Rat Model for Acute Myocardial Infarction (AMI): Left-ventricular MI induced by thermocoagulation. Animals allowed to recover for at least 12h after surgery.	CAPs: (Boston, MA) **Particle Size:** CAPs: $PM_{2.5}$	**Route:** Whole-body Inhalation Chambers **Dose/Concentration:** CO: 35ppm; CAPs (median concentration): 645.7 $\mu g.m^3$; CAPs + CO: 37.9ppm **Time to Analysis:** CAPs or CAPs + CO exposure for 1h. Exposure to pollutants was preceded and followed by 1h exposure to FA.	Among rats in the CAPs group, the probability of observing supraventricular arrhythmias (SVA) decreased from the baseline to exposure and post-exposure periods. The pattern was significantly different than that observed for the FA group during the exposure period. In the subset with one or more SVA during the baseline period, the change in SVA rate from baseline to exposure period was significantly lower in the CAPs and CO groups only, when compared to the FA group. No significant effects were observed in the group simultaneously exposed to CAPs and CO.
Reference: Wichers et al. (2004, 055636) **Species:** Rat **Gender:** Male **Strain:** SH **Age:** 75d	HP-12 (oil-combustion derived PM obtained from inside wall of a Boston power plant stack burning residual oil (number 6). Water-leachable constituents ($\mu g/mg$): SO_4 (217.3); Zn (11.4); Ni (6.9); Fe (0.0); V (1.3); Cu (0.2); Pb (0.0) 1M HCl-leachable constituents ($\mu g/mg$): SO_4 (220.6); Zn (15.5); Ni (14.8); Fe (15.6); V (32.9); Cu (1.1); Pb (1.7) **Particle Size:** 3.76 μm (MMAD) (GSD 2.16)	**Route:** IT Instillation **Dose/Concentration:** HP-12 (mg/kg): 0.00 (saline control), 0.83 (low), 3.33 (mid), 8.33 (high) **Time to Analysis:** Single-dose Sacrificed 96h or 192h post-IT.	Exposures to mid and high-dose HP-12 induced large decreases in HR, BP, and body temperature. The decreases in HR and BP were most pronounced at night and did not return to pre-IT values until 72h (HR) and 48h (BP) after dosing. ECG abnormalities (rhythm disturbances, bundle branch block) were observed primarily in the high dose group.
Reference: Wold et al. (2006, 097028) **Species:** Rat **Gender:** Female **Strain:** Sprague-Dawley **Use:** L jugular vein and R carotid artery were canulated.	UFPs from either ambient air (UFAAs) or diesel engine exhaust (UFDGs); UFIDs from industrial forklift exhaust and soluble fraction UFID suspension, particle free (SF-UFID) **Particle Size:** UFAAs diameter ◘ 150 nm; UFDGs diameter ◘ 100nm	**Route:** IV Infusion (in vivo study) **Dose/Concentration:** UFDG (50$\mu g/m$) **Time to Analysis:** Infused w/UFAA or UFDG. Monitored continuously for 1h then sacrificed.	Infusion of UFDGs caused ventricular premature beats (VPBs) in 2 out of 3 rats. Ejection fraction increased slightly in rats receiving UFAA and was unchanged in the UFDG and saline groups.
Reference: Wold LE et al. (2006, 097028) **Species:** Rat **Gender:** Female **Strain:** Sprague-Dawley **Use:** Heart Langendorff-perfusion apparatus	UFPs from either ambient air (UFAAs) or diesel engine exhaust (UFDGs); UFIDs from industrial forklift exhaust and soluble fraction UFID suspension, particle free (SF-UFID) **Particle Size (Distribution):** UFAAs diameter; ◘150 nm; UFDGs diameter: ◘ 100nm	**Route:** Lagendorff Heart Perfusion (in vitro) **Dose/Concentration:** UFDG (100 μg /2ml); UFID (12.5 $\mu g/l$ in perfusate); SF-UFID (12.5 $\mu g/l$) **Time to Analysis:** Lagendorff 1: Treated w/UFDG. Lagendorff 2: Treated with UFID & SFUFID. Both experiments were monitored continuously for 1h after injection.	UFDGs caused a marked increase in left-ventricular and end-diastolic pressure (LVEDP) after 30 min of exposure. UFIDs caused a significant decrease in left-ventricular systolic pressure (LVSP) at 30min after the start of infusion. This effect was absent when SF-UFID was studied.
Reference: Yatera et al. (2008, 157162) **Species:** Rabbit **Gender:** Female **Strain:** WHHL **Age:** 42wks **Weight:** 3.2 ± 0.1 kg (avg)	EHC-93 from Ottawa, Canada **Particle Size:** NR	**Route:** IT Instillation **Dose/Concentration:** PM_{10} suspension: 5mg EHC-93 in 1ml saline **Time to Analysis:** Exposed 2x/wk for 4 wks. Acute effects observed at 0.5, 1, 2, 4, 8, 12, and 24h after initial instillation. Chronic effects observed 1x/wk for 4 wks.	Exposure to PM_{10} caused progression of atherosclerotic lesions in thoracic and abdominal aorta. It also decreased circulating monocytes expressing high levels of CD31 and CD49d, and increased expression of CD54 (ICAM-1) and CD106 (VCAM-1) in plaques. Exposure to PM_{10} increased the number of BrdU-labeled (*) monocytes into plaques and into smooth muscle underneath plaques. (*onocytes labeled with BrdU in donor rabbits were transfused to recipient rabbits as whole blood, and the recruitment of BrdU-labeled cells into vessel walls and plaques in recipients was measured by quantitative histological methodology.

Study	Pollutant	Exposure	Effects
Reference: Ying Z et al. (2001, 019011) **Species:** Mice **Gender:** Male **Strain:** ApoE$^{-/-}$ **Age:** 16wks	CAPs: $PM_{2.5}$, New York City (Manhattan), NY; May-Sept 2007 **Particle Size:** NR	**Route:** Inhalation **Dose/Concentration:** 138.4 ± 83.7 $\mu g/m^3$ **Time to Analysis:** 6h/d × 5d/wk × 4m	**Vascular tone:** Significant decrease in PE-induced maximum contraction of aortic rings in CAPs-exposed mice. No difference in sensitivity to PE between groups. Treatment with the soluble guanylate cyclase inhibitor ODQ restored the response to PE in CAPs aortic rings. No significant differences in relaxation induced by ACh. CAPs abolished the relaxation induced by Ca ionophore A23187. CAPs exposure slightly (but significantly) decreased maximum relaxation induced by SNP. **Protein expression:** iNOS mRNA expression was increased in the aortas of CAPs-exposed mice. eNOS and GTPCH levels were unchanged. Distribution of inOS protein expression was limited to plaque in air-exposed mice and was found in the plaque and media for CAPs-exposed mice. **Superoxide production:** Superoxide levels in CAPs-exposed mice were increased in the aorta compared to air-exposed mice. The addition of L-NAME significantly increased superoxide production. Extensive protein nitration in aortas of CAPs mice. NADPH subunits Rac1 and p47 phox mRNA expression was increased in aortas of mice exposed to CAPs. **Atherosclerosis:** Significant increase in plaque area of CAPs-exposed mice. Higher levels of macrophage infiltration, collagen deposition, and lipid composition of plaques from CAPs-expsoed mice.
Reference: Ying Z et al. (2001, 019011) **Species:** Mouse **Gender:** Male **Strain:** ApoE$^{-/-}$ **Age:** 6wks **Weight:** NR	CAPs (New York, NY) (May-Sept. 2007) **Particle Size:** Diameter: $< 2.5\ \mu m$	**Route:** Exposure Chamber **Dose/Concentration:** Ambient $PM_{2.5}$: 23 ± 75.9 $\mu g/m^3$, Chamber $PM_{2.5}$: 138.4 ± 83.7 $\mu g/m^3$ **Time to Analysis:** Fed high-fat chow 10wks. Exposed 6h/d, 4m.	**Vascular:** Constriction responses to KCl were similar between CAPs-exposed and control mice. PE-induced maximum contraction significantly decreased in CAPs mice, which was attenuated by the soluble guanine cyclase inhibitor OQD. CAPs prevented calcium ionophore A23187-induced relaxation. CAPs decreased SNP-induced maximal relaxation. **iNOS, Aortic $O_2^{\cdot-}$:** iNOS mRNA expression increased. Aortic $O_2^{\cdot-}$ increased and was further increased by NOS inhibitor L-NAME. **Atherosclerosis:** The only NADPH oxidase subunits affected were Rac1 and $p47^{phox}$, which increased. Atherosclerotic burden in the thoracic aorta, macrophage infiltration, collagen deposition, and lipid composition in the aorta all increased.
Reference: Yokota S et al. (2004, 096516) **Species:** Rat **Gender:** Male **Strain:** Sprague-Dawley (IGS) **Weight:** 345-498.2g	DEP (obtained from the Japan Automobile Research Institute) **Particle Size:** NR	**Route:** IT Instillation **Dose/Concentration:** Group 1: DEP: 1mg/0.1ml Group 2: DEP: 0.2 ml (10, 12.5 or 25 mg/ml) Group 3: DEP 2.5 or 5mg/0.2ml **Time to Analysis:** DEP pre-treatment 24-72h before ischemia/reperfusion.	**DEP effects on myocardial Ischemia/Reperfusion-induced arrhythmia:** An increased mortality was observed in the DEP group compared to the vehicle-treated group. 46% of the animals in DEP died during the first 3 min reperfusion period. The animals of other groups were intratracheally instilled with DEP at the beginning of ischemia/reperfusion experiment, or were pretreated with polyethylene glycol-conjugated SOD (1000 IU/kg, iv). In these animals, incidences of both arrhythmia and mortality were similar to those in the animals treated with the vehicle. **DEP effects on the biochemical and hematological parameters:** Neutrophil count was elevated by a higher dose (5 mg) of DEP at 24 h after the IT instillation, and oxygen radical production, which was induced by 12-O-tetradecanoylphorbol 13-acetate, was enhanced at 72h.

Study	Pollutant	Exposure	Effects
Reference: Yokota S et al. (2005, 096003) **Species:** Rat **Gender:** Male **Strain:** Sprague-Dawley (IGS) **Weight:** 303-472.2g **Use:** Rats were used after a 7-day acclimation period	DEP from Japan **Particle Size:** NR	**Route:** IT Instillation **Dose/Concentration:** Vehicle: 0.2 mL/animal DEP: 5 mg/animal **Time to Analysis:** Single exposure 0.5, 1, 2, 3, 6, 12, 24, 48h.	At 12 and 24 h post-instillation, circulatory neutrophil counts in the 5 mg DEP group were significantly elevated, and were 2.1-fold (12h) and 2.3 fold (24 h) in vehicle treated animals. 1 mg DEP caused an increase of approximately 0.4-fold in CNC at 6h. 12-O-tetradecanoylphorbol 13-acetate induced oxyradical production (ORP) in the isolated neutrophil was enhanced at 12 and 24 h after instillation with 5 mg DEP. In Serum, a marked elevation of CINC-1 and a slight elevation of MIP-2 were also observed, while TNF α was not detected. GM-CSF was not detected in serum 24 h post-instillation.
Reference: Yokota S et al. (2008, 190109) **Species:** Mouse **Gender:** Male **Strain:** ddy **Age:** NR **Weight:** 39.6-46.0g	DEP (DMSC (dichloromethane soluble-component), RPC (residual particle-component)) **Particle Size:** NR	**Route:** IT Instillation **Dose/Concentration:** 5mg/kg, 10mg/kg **Time to Analysis:** DMSC and RPC extracted from DEP. Mice acclimatized 7d. DEP, DMSC, or RPC instilled. BALF and blood obtained and G-CSF, GM-CSF, IL-6 measured 2, 4, 12, 24h postinstillation.	**Inflammation:** At 5mg/kg DEP increased the total cell and macrophage count. DEP or RPC increased neutrophils at 5 and 10mg/kg. 10mg/kg DEP or RPC increased macrophages at 4h and decreased at 12h. **Hematology:** Compared to 5mg/kg DEP, RPC increased RBC, WBC, and neutrophils. 10mg/kg RPC or DEP caused sustained increases in RBC, WBC, and neutrophils. **Cytokines:** 5mg/kg RPC markedly increased G-CSF and IL-6. Other cytokine increases at this dose were transient. 10mg/kg DEP increased IL-6 at 4h, and DEP or RPC increased G-CSF and IL-6 at 12h. DEP or RPC also increased IL-1β. **Myocardium:** Myocardial MPO activity significantly increased in 5mg/kg RPC at 12 and 24h. Myocardial MIP-2 increased the most in 5mg/kg RPC, while LIX tended to be lowered by RPC.

Table D-2. Respiratory effects: in vitro studies.

Study	Pollutant	Exposure	Effects
Reference: Aam BB and Fonnum F, (2007, 155123) **Species:** Human, Rat **Tissues/Cell Types:** Human-Neutrophil Granulocytes (NG); Rat- Alveolar Macrophages (AM)	DEP: SRM 1975 **Particle Size:** NR	**Route:** Cell Culture **Dose/Concentration:** NG: 8.8 - 280 μg/mL AM: 140, 280 μg/mL Vitamin E = 5μM **Time to Analysis:** 1h	**ROS of NG:** Formation of ROS in NG decreased with increased doses of DEP. Lucigenin chemiluminescence of ROS formation diminished 25% at 8.8 μg/mL DEP and luminol chemiluminescence 32% with 17.5 μg/mL DEP. DCF fluorescence required much higher doses of DEP. Controls without PMA stimulation had highly reduced lucigenin and luminol with DEP dose of 140 μg/mL while DCF increased 116%. **ROS of AM:** 280 μg/mL of DEP decreased ROS level by 19% with DCF. DEP with PMA-unstimulated cells increased 24% with DCF. **Necrosis:** NG cell death was DEP dose-dependent. At 280 μg/mL, cell death increased 5.4% as compared to control. LDH concentration increased 1.6% with 70 μg/mL DEP and 3.9% with 280 μg/mL after 1h.

Study	Pollutant	Exposure	Effects
Reference: Agopyan et al. (2003, 056065) **Species:** Human **Tissues/Cell Types:** BEAS-2B, NHBE, SAEC	PC: synthetic carboxylate-modified particles **Particle Size:** 2, 10 μm	**Route:** Cell Culture **Dose/Concentration:** PC2 = 0.83 g/mL or $3.4x10^9$ particles/mL PC10 = 0.8 g/mL or $3x10^6$ particles/mL **Time to Analysis:** PC2 = 12, 24, 8h PC10 = 2, 6, 12, 24h	**Calcium Imaging:** PC10 induced increase of Ca2+ concentration in all capsaicin-sensitive cells 100%. Similar reaction observed in cells exposed to PC2. However, more than 3-PC2s were required to induce a Ca increase unlike PC10. CPZ (10um) and amiloride could fully block PC-induced response. **cAMP:** Post 6h, a dose-dependent increase in cAMP was observed. Again, CPZ blocked increase by 70-90% depending on cell type: SAEC > NHBE ~ BEAS-2B. **Apoptosis:** PC10 and PC2 induced apoptosis time-dependently. PC2 was slower in induction than PC10. Post 48h, 80-95% cells were apoptotic in all cell types. Noncapaisin-sensitive cells (which did not bind to particles) did not exhibit apoptosis. CPZ reduced apoptosis by 97% BEAS-2B, 96% NHBE and 98% SAEC. Amiloride did not block apoptosis. **Necrosis:** Induction of necrosis by PC2 and PC 10 was negligible. A slight increase from 1% to 2% was observed at 24-48h in NHBE and SAEC. BEAS-2B showed slight decrease from 3% to 4% in same time period.
Reference: Agopyan et al. (2004, 156198) **Species:** Human, Mouse **Tissues/Cell Types:** Human-NHBE, SAEC; Mouse-Wildtype and TRPV1(-/-) Terminal Ganglion Neurons (TG) ~ 257 Wildtype Neurons ~ 187 TRPV (-/-) Neurons	ROFA MSHA: Mt St Helen Ash **Particle Size:** NR	**Route:** Cell Culture **Dose/Concentration:** 100 μg/mL ROFA or MSHA **Time to Analysis:** ROFA/MSHA in NHBE and SAEC = 2, 6, 24, 48 h ROFA/ MSHA in TG = 24 h cAMP measurements with NHBE and SAEC exposed to ROFA/MSHA = 6h	**Calcium Imaging in NHBE and SAEC:** In 100% of reactive cells, ROFA/MSHA induced an increase in Ca2+. Levels remained elevated as long as PM bound to plasma membrane. Washing and disjoining PM from membrane caused Ca2+ to slowly decline to baseline. CPZ (or CPZ and amiloride) reversibly inhibited PM-induced rises in Ca2+. **Calcium Imaging in TRPV1(+/+) and (-/-) mice sensory neurons:** All sensitive neurons in TRPV1(+/+) increased Ca2+ in response to ROFA. No effect of ROFA in TRPV1(-/-). **cAMP:** ROFA and MSHA induced increases in Ca2+ in NHBE and SAEC cells, which was completely blocked by cAMP. **Apoptosis:** ROFA or MSHA induced time-dependent apoptosis, peaking at 24 h. CPZ again inhibited this response. Neurons bound to PM (< 25um) induced apoptosis in TRPV1(+/+). Cells without bound PM or bound with PM (> 25 μm) showed no effect. No apoptosis occurred in the absence of Ca2+. **Necrosis:** Necrosis for any of the cell types was negligible. **PKA:** Inhibition of PKA resulted in 90+% apoptosis in NHBE and SAEC. Again, no apoptosis was observed in a Ca2+ free environment.
Reference: Ahn E-K et al. (2008, 156199) **Species:** Human **Tissues/Cell Types:** A549	DEP: (6 cyl, 11L, turbo-charged, heavy-duty diesel engine, South Korea) Dex: anti-inflammatory (Sigma, St. Louis, MO) **Particle Size:** NR	**Route:** Cell Culture **Dose/Concentrations:** 0, 1, 5, 10, 50 and 100 μg/mL of DEP Some cells pre-treated with 10, 20, 40, 50 pg/mL of Dex. **Time to Analysis:** 24h	**COX-2 Expression:** Cells expressed dose-dependent increases in COX-2 expression after treatment with 10-100 μg/mL of DEP. Treatment of 50 μg/mL for 24 h induced statistically significant COX-2 expression in both mRNA and protein levels. Pre-treatment with Dex significantly reduced expression of COX-2 mRNA and protein. Dex treatment induced dose-dependent suppression of DEP-induced protein levels. **PGE2 Levels:** Levels of the inflammatory mediator, PGE2, increased when were cells exposed to 50 μg/mL of DEP. Pre-treatment with 50 μg /mL Dex completely inhibited DEP-induced release of PGE2.

Study	Pollutant	Exposure	Effects
Reference: Ahsan (2005, 156200) **Species:** Human **Tissues/Cell Types:** Trx-1-transfected Clone of Murine L-929 cells; Control Clone (L-929-Neo1); A549	DEP: provided by Dr. Masaru Sagai, University of Health and Welfare, Aomori, Japan **Particle Size:** NR	**Route:** Cell Culture **Dose/Concentration:** DEP: 50 μg/mL hTrx-1- or L-929-Neo1: 40 μg/mL Pretreatment: rhTrx-1 (10 μg/mL) or DM-rhTrx-1 (NR) **Time to Analysis:** Pretreatment for 1 h. Parameters measured 3h post exposure.	**ROS:** DEP induced significant increases of ROS in L929-Neo1 cells. hTRx-1 cells showed no affect. RT-PCR revealed hTrx-1 mRNA expression in transfected cells but not control L929-Neo1 cells. Endogenous murine Trx-1 mRNA expression increased in control cells, but not in hTrx-1 cells. A549 cells had increased ROS levels but these levels were suppressed with rhTrx-1 pretreatment. Pre-treatment with DM-rhTrx-1 increased ROS levels more. **Akt (antiapoptotic molecule):** Phosphorylated Akt prevents apoptosis. DEP induced phosphorylation of Akt in control cells after 3h and dephosphorylation after 5h. In hTrx-1 cells, Akt remained phosphorylated after 5h. In A549 cells, Akt phosphorylated at 3h and slowly turned off at 12-24 h. Pre-treatment with rhTrx-1 blocked dephosphorylation. This suggests that Trx-1 preserves active form of Akt and thereby protects against cytotoxicity from DEP.
Reference: Alfaro-Moreno et al. (2002, 156204) **Species:** Human, Mouse, Rat **Strain:** Human-A549; Mouse-J7 74A.1, BALB-c **Tissues/Cell Types:** HUVEC, Mouse Fibroblasts, Rat Lung Fibroblasts (RLF)	PM_{10}: Collected from 3 zones in Mexico City: North (industrial), Center (business) and South (residential) **Particle Size:** PM_{10}	**Route:** Cell Culture 15000 cells/cm^2 except: Cytotoxicity: Confluent Cultures 180,000 cells/cm^2. DNA Breakage: 20,000 cells/well. Cytokine Assays: 180,000 cells/cm^2 **Dose/Concentration:** Cytotoxicity: 10, 20, 40, 80, 160 μg/cm^2 Apoptosis: 160 μg/cm^2 DNA Breakage: 2.5, 5, 10, 20, 40 μg/cm^2 Cytokine Assays: 10, 20, 40, 80 μg/cm^2 E-Selectin Expression: 40 μg/cm^2 **Time to Analysis:** Cytotoxicity: 24, 48, 72h; Apoptosis: 24 h; DNA Breakage: 72h; Cytokine Assays: 24 h	**Cytotoxicity:** Cytotoxic effect exhibited dose-dependency after 72h in proliferating cells of J774A.1, BALB-c and RLF cell lines. **Proliferating Cells:** Northern particles induced a statistically larger effect than central or southern particles. J774A.1 was more susceptible while BALB-c was less susceptible. A549 was most resistant to decreased viability during exposure. No significant variation in viability was observed when compared to the control. Particles were not cytotoxic among confluent cell growth for any cell lines when exposed to 20-160 μg/cm^2. **Apoptosis:** Overall, particles induced low rates of cell death via apoptosis. J774A.1 depicted similar levels of apoptosis when exposed to three PM zones, ~15% apoptotic cells measured. BALB-c was not reported. Results for the A549 measured apoptotic cells were: South- 4%, Central- 11% and North- 15%. HUVEC cells indicated an increase in apoptosis with northern particles. **DNA Breakage:** PM_{10} from all zones induced DNA breakage. A dose-dependent relationship was established with $PM_{2.5}$ particles at concentrations of 10 μg/cm^2. The Southern zone required a higher dose of PM (10μg/cm^2) to produce the same effect as other zones (2.5μg/cm^2). **Cytokines:** Particles induced TNF-α and IL-6 secretion in J774A.1 cells dose-dependently. IL-6 increased significantly with central particles. PGE2 secretion in RLF cells induced by exposure to PM showed dose-dependent responses. PM from the central zone induced the most PGE2 secretion. Max secretion was observed at doses of 40 μg/cm^2 from all three PM zones. **E-Selectin Expression:** HUVEC cells showed a 25% increase in E-selectin expression after exposure to 40 μg/cm^2 of PM.

Study	Pollutant	Exposure	Effects
Reference: Amakawa et al. (2003, 156211) **Species:** Mouse, Human **Strain:** Mouse-ICR **Tissues/Cell Types:** AMs **Gender:** Mouse-Male; Human-Male **Age:** Mouse 6-7wks; Human 20-24yrs	DEP (obtained from a 4JB1, Isuzu, 1500 rpm, 4cyl diesel engine) DEPE = DEP Extract (methanol) CB = Charcoal (Sigma) **Particle Size:** DEP- 0.4 μm, CB- 0.7 μm	**Route:** Cell Culture Mouse: $5X10^5$ cells/mL Human: $3X10^5$ cells/mL **Dose/Concentration:** DEP = 1 or 10 μg/mL;DEPE = 1 or 10 μg/mL; CB = 1, 10, 100 μg/mL **Time to Analysis:** Human cells pre-treated with LPS 1 μg/mL. Murine cells pre-treated with SOD 300 IU/mL. Parameters measured 24h post exposure.	**Cells:** For mice, more than 90% of the cells were macrophages and over90% were viable. For humans, 96% of the cells were macrophages, 3% lymphocytes and 1% neutrophils; over95% of the human cells were viable. **DEP Cytotoxicity:** None observed **Cytokines:** DEP (10μg/mL) suppressed release of TNF-α and IL-6 for both mice and humans in a dose-dependent manner. Murine cells pre-treated with LPS or IFN-γ released even less TNF-α and IL-6. IL-10 was unaffected. Human macrophages pre-treated with LPS also released lower levels of TNF-α, IL-6 and IL-8. **ROS:** Pre-treatment of SOD on murine cells partially attenuated the suppressive effect of DEP as well as decreased the production of ROS generated by DEP (10μg/mL). **Carbon:** Carbon particles did not suppress TNF-α or IL-6 release from murine AMs; however,100 μg/mL of CB stimulated TNF-α production. **Methanol:** No cytotoxicity nor cytokine release effects were observed. **DEPE:** DEPE suppressed TNF-α and IL-6 release in a similar way as DEP.
Reference: Amara et al. (2007, 156212) **Species:** Human **Cell Lines:** A549, NCI-H292	DEP = SRM 2975 CSC = cigarette smoke condensates (collected from Kentucky standard cigarettes, 2R4F; University of Kentucky) DC = DEP + CSC CB (Degussa, Frankfurt, Germany) **Particle Size:** CB: 95nm; DEP: NR	**Route:** Cell Culture **Dose/Concentration:** DEP = 5-10 μg/cm^2 CB = 10 μg/cm^2 CSC = 10 μg/cm^2 **Time to Analysis:** 6 or 24h	**Inflammatory Markers:** LDH of A549 was unaffected at either time point with DEP or CB. LDH increased with CSC at concentrations high than 10μg/mL at both time points. DC had no effect. **Proteases:** MMP-1 mRNA expression showed a dose dependent increase with DEP in A549 cells. DEP also increased MMP-1 in NCI-H292 cells. CB and CSC had no effect. MMP-1 mRNA expressions were inhibited by N-acetylcysteine antioxidant. Similar inhibition was observed with NOX4 oxidase. DC induced a similar effect to DEP. MMP-1 protein expression increased post 24 h with DEP. MMP-2, TIMP-1, TIMP-2 mRNA expression was unaffected. **TGF:** TGF-β mRNA expression was unaffected. **ROS:** DEP and DC increased ROS formation after 1h. DEP effect was inhibited by N-acetylcysteine antioxidant pre-treatment. **MAP-Kinase:** DEPinduced MMP-1 expression increased ERK1/2 phosphorylation after 10 min, peaking at 30min, and returning to normal levels at 60min. Treatment with CBPs did not increase ERK1/2 phosphorylation whereas treatment with CSC resulted in phosphorylation. Only inhibitors of ERK1/2 reduced DEP induced MMP-1 activity. P38 and JNK inhibitors had no effect.
Reference: Anseth et al. (2005, 088646) **Species:** Human **Cell Lines:** A549; A549-p0 (lacking mitrochondria)	s-ROFA: soluble portion **Particle Size:** 1.95 ± 0.18 μm	**Route:** Cell Culture ($3X10^5$ cells/mL) **Dose/Concentration:** 100 μg/mL **Time to Analysis:** Experiments conducted by spreading monolayer of Infasurf (calf lung surfactant extract on PBS, PBS+ROFA or conditioned media from A549 AEC. Parameters measured after 16h incubation period.	**Lung Surfactant Gelation:** ROFA alone and A549 conditioned media alone did not significantly alter Infasurf rheology. However, conditioned media from A549 AEC at 16h induced a significant increase in elastic storage and viscous loss moduli. Inhibiting ROS production lowered effect, indicating s-ROFA gelation mediated through ROS. **ROS:** ROS mediated through mitochondria as evidenced by the effect of ROFA-AEC on surfactant gelation in the presence of mitochondria ROS inhibitors as well as A549-p0 cells.

Study	Pollutant	Exposure	Effects
Reference: Auger et al. (2006, 156235) **Species:** Human **Tissue/Cell Type:** Nasal Epithelial Cells	DEP: SRM1650 $PM_{2.5}$: (obtained from a highway in Paris, France) **Particle Size:** DEP: 400 nm (mean diameter); $PM_{2.5}$	**Route:** Cell Culture (2-3.5x10^4 cells/cm^2) **Dose/Concentration:** 10-80 $\mu g/cm^2$ **Time to Analysis:** Cells treated on apical side. Parameters measured 24h following treatment.	**Cytotoxicity (LDH):** No cytotoxicity for DEP or $PM_{2.5}$ (80 $\mu g/cm^2$). **Cytokines:** In non-stimulated ALI cultures, IL-8 was the most abundantly secreted cytokine, followed by GM-CSF, TNF-α, and IL-6 in decreasing levels of production. Amphiregulin was moderately, but consistently, secreted. After treatment, both DEP and $PM_{2.5}$ induced IL-8 and amphiregulin release in a dose-dependent manner through the basolateral surface. $PM_{2.5}$ stimulated IL-6 and GM-CSF release through the apical surface. **ICAM-1 expression:** No effect from DEP or $PM_{2.5}$. **ROS:** DEP and $PM_{2.5}$ both increased ROS production in a dose-dependent manner.
Reference: Bachoual et al. (2007, 155667) **Species:** Mouse **Cell Type:** RAW 264.7	PM_{10} from two Paris, France subway sites: RER and Metro CB (Frankfurt, Germany) TiO_2 (Calais, France) DEP: SRM1650 (NIST) **Particle Size:** CB: 95nm; TiO_2: 150um; DEP: NR; RER PM_{10}: 79% $<0.5\ \mu m$, 20% 0.5-1 μm; Metro PM_{10}: 88% $<0.5\ \mu m$, 11% 0.5-1 μm.	**Route:** Cell Culture (40,000 cells/mL) **Dose/Concentration:** All particles: 0.01, 0.1, 1, 10 $\mu g/cm^2$ **Time to Analysis:** 3, 8, 24h	**Cell Viability:** No effects from any particulate at concentrations up to 10 $\mu g/cm^2$ for 24 h. **Inflammatory Effect:** Exposure of cells to 10$\mu g/cm^2$ of RER or Metro induced time-dependent increase in TNF-α and MIP-2 protein release. This effect was similar to both locations. No effect was observed at low concentrations of PM_{10}. No effect of CB, TiO_2 or DEP was observed. **GM-CSF or KC production:** RER and Metro PM_{10} did not induce any effect at any concentration. **Effect on Protease mRNA Expression:** Exposure of cells to 10 $\mu g/cm^2$ RER or Metro PM_{10} did not modify mRNA expression of MMP-2 or -9 or their inhibitors TIMP-1 and -2. MMP-12 expression significantly increased after exposure to RER or Metro PM_{10} for 8h. **Effects on HO-1 Protein Expression:** Exposure to 10 $\mu g/cm^2$ of RER or Metro PM_{10} for 24 h induced positive cytoplasmic staining for HO-1.
Reference: Batalha et al. (2002, 088109) **Species:** Rat **Gender:** Male **Strain:** SD **Age:** NR **Weight:** 200-250g	CAPs (Harvard Ambient Particle Concentrator) **Particle Size:** Mean: 2.7 μm	**Route:** Inhalation Chamber **Dose/Concentration:** Range: 73.5-733 $\mu g/m^3$ **Time to Analysis:** CAPs exposure 5h/d, 3d (consecutive). SO_2 exposure to induce CB 5h/d, 5d/wk, 6wks. Killed 24h postexposure.	**Histopathology:** CAPs slightly increased the wall thickness of small pulmonary arteries and edema in the adventitia and hyperplasia of the terminal bronchiole and alveolar ducts epithelium. **L/W ratio:** The L/W ratio decreased in CAPs-exposed rats as particle mass, Si, Pb, SO_4^{2-}, EC and OC increased. Univariate analyses showed significant negative correlations between the L/W ratio and Si and SO_4^{2-} in normal rats and Si and OC in CB rats. Multivariate analysis showed only Si to be significant in both groups.

Study	Pollutant	Exposure	Effects
Reference: Baulig et al. (2007, 151733) **Species:** Human **Cell Line:** 16-HBE14o-	WUB: Winter Urban Background Particles (obtained from Vitry-sur-Seine, suburb of Paris, France) SUB: summer Urban Background Particles Vitry-sur-Seine) WC: Winter Curbside Particles, SRM1648 (obtained from Porte-d'Auteuil, ring road of Paris, France) SC: summer Curbside Particles, SRM 1648 (Porte-d'Auteuil) DEP: SRM 1650a (NIST) DPL (control) **Particle Size:** WUB, SUB: $PM_{2.5}$; WC, SC, DEP: NR	**Route:** Cell Culture (20,000 cells/cm^2) **Dose/Concentration:** 10 μg/cm^2 **Time to Analysis:** 18 or 24h	**EGF:** All native $PM_{2.5}$ induced similar AR secretion by bronchial epithelial cells (in decreasing order WC, WUB, SC, SUB), but this release was significantly greater than the release induced by DEP. βcellulin increased with SC, WUB and WC. No data was available forSUB or DEP. **Interleukins:** IL-1α increased significantly with WUB, WC,SC, DEP, DPL (in decreasing order). No data was available for SUB. Exposure to WUB caused IL-1β to increase to induction factor of over 2. IL-11 R α decreased significantly with SUB. **Cytokines:** Exposure to WUB caused G-CSF to increase with an induction factor of over 2.Though not statistically significant, TNF-R1 also increased. **Proteases:** TIMP-2 decreased with WUB but significantly increased with SUB. Overall, SUB downregulated integrins and interleukins seen with other particles while upregulating neurotrophic factors, chemokine receptors and adhesion molecules. MMPs were not measured. **Chemokines:** CCR-3 significantly increased with SUB. GRO-γ and GRO-α increased with WC at both 18 and 24h. DEP had no effect with GRO-α. Removal of metal from particles lowered response of GRO-α.
Reference: Bayram et al. (2006, 088439) **Species:** Human **Cell Type:** A549	DEP: (obtained from a 4JB1-type, light-duty, 4 cyl, 2.74-L Isuzu diesel engine) DEP-FCS: DEP + FCS DEP-NAC: DEP + N-acetylcystine, antioxidant DEP-A: DEP + AEOL10113, catalytic antioxidant DEP-S: DEP + SP600125, inhibitor of JNK DEP-N: DEP + SN50, inhibitor of NF-kB **Particle Size:** DEP: 0.4 μm (mean diameter)	**Route:** Cell Culture **Dose/Concentration:** DEP: 0, 5, 10, 50, 100, 200 μg/mL **Time to Analysis:** 24, 48, 72h	**Cell Growth:** With 10% FCS (as a positive control), A549 cells exhibited time dependent growth. A mixture of FCS and DEP did not affect cell growth for up to 48h. With DEP alone, cell growth was prevented from cell number reduction due to removal of serum at 48 and 72h. A dose of 10μg/mL induced a maximum proliferation effect. **Cell Cycle:** DEP increased the percentage of serum-starved cells in S phase at 48h. DEP decreased the percentage in G0/1 phase and G2/M phase. **Apoptosis:** DEP prevented the increase in apoptotic, serum-starved cells. **Protein Expression:** p21CIP1/WAF1 expression increased at 48h. DEP dose-dependently decreased this expression. **NAC:** NAC alone, at 33 mM, induced an increase in cell numbers. DEP-NAC inhibited cell numbers at 48h. DEP-NAC inhibited cell numbers in S phase; thus, cells in G0/1 phase increased. DEP-NAC induced a further decrease of cells in G2/M phase. **AEOL10113:** DEP-A caused a dose-dependent decrease in cell numbers. **SP600125:** Alone, SP600125 increased cell numbers at 33mM. DEP-S decreased cell numbers.
Reference: Becher et al. (2007, 097125) 2007 #8030} **Species:** Rat **Strain:** Crl/Wky **Cell Type:** Alveolar Macrophages, Alveolar Type II **Gender:** Male **Weight:** 200g	SPM = suspended PM SRM-1648 **Particle Size:** SPM: 6-8 μm	**Route:** Cell Culture (1.5x10^6 cells/well AM; 6x10^6 cells/well Type II) **Dose:** 200μg/mL = 20 μg/cm^2 **Time to Analysis:** 20h	**Cytokines in Macrophages:** SPM increased TNF-α and MIP-2. NADPH inhibitor DPI reduced MIP-2 response, whereas iNOS inhibitor 1400W did not affect either. **Cytokines in Type 2 Cells:** SPM increased IL-6 and MIP-2 significantly. This SPM effect was inhibited by DPI, whereas1400W reduced the IL-6 response significantly. **ROS in Type 2 Cells:** SPM significantly increased ROS formation. DPI largely blocked this SPM effect. **ROS in Macrophages:** No significant increases were observed.

Study	Pollutant	Exposure	Effects
Reference: Becker et al. (2005, 088590) **Species:** Human **Gender:** Male and Female **Age:** 18-35yrs **Cell T ypes:** Alveolar Macrophages, NHBE	PM (Coarse, Fine, Ultrafine): (EPA, Chapel Hill, NC, Chem Vol Cascade) **Particle Size:** PM-C: $PM_{2.5}$; PM-F: PM0.1; PM-UF: $<0.1\mu m$	**Route:** Cell Culture (0.5-1 x 10^5 cells/well NHBE; 2-3 x 10^5/mL AM) **Dose/Concentation:** NH BE: 25, 50, 100, 250 μg/mL of PM; AMs: 50 μg/mL of DEP or 10ng/mL of LPS **Time to Analysis:** 18h for NHBE; overnight for AMs	**Cytokines:** All 3 fractions induced dose-dependent increases in IL-8 secretion with PM-c, PM-F, PM-UF (in order of decreasing effects). TLR-2 antibody blocked these particle induced IL-8 effects. **Inhibitors of Endotoxin effects and TLR-4 activation:** No effects were observed in NHBE, but all 3 fractions repressed the IL-6 release in AMs. **TLR mRNA Expression:** PM did not affect TLR-2 mRNA in NHBEs. PM-C and PM-F induced a slight increase in TLR-4 mRNA in NHBEs while PM-UF induced a substantial increase. PM-C increased TLR-2 mRNA in AMs and decreased TLR-4 mRNA in AMs. **Induction of Hsp70:** PM-C and PM-F induced Hsp70 in NHBE dose-dependently. Hsp70 was not induced in AM following particle stimulator.
Reference: Becker et al. (2005, 088592) **Species:** Human **Gender:** Male **Age:** 18-35yrs **Cell Types:** Alveolar Macrophages, NHBE	PM (Coarse, Fine, Ultrafine): (EPA, Chapel Hill, NC, Chem Vol Cascade) ROFA Fe, Si, Cr Components Oct 2001, Jan 2002, April 2002, July 2002 **Particle Size:** PM-C: 2.5-10 μm; PM-F: <0.1 μm; PM-UF: <0.1 μm	**Route:** Cell Culture (3-5 x 10^5 cells/well NHBE; 2-3X10^5 cells/mL AM) **Dose/Concentration:** NHBE: 11 μg/mL of PM; AM: 50 μg/mL of PM **Time to Analysis:** 18-24h NHBE; 18h AM	**IL-8 Release in NHBE:** PM-C and PM-UF induced effects. No effects from PM-F (all 4 dates). **IL-6 Release in AM:** All 3 fractions induced increase with later dates having generally lower effects. **ROS (DCF):** NHBE, at lower exposures, were observed to be more responsive to PM than AMs. AM exhibited highly variable results over time. **ROS (DHR):** NHBE cells were observed to be more responsive to PM than AMs. AM responsiveness to PM increased over 4 time periods; this was not observed in NHBE. **Seasonal Variability:** Coarse particles were more potent than F and UF regardless of the month, and the potency for PM to induce IL-6/IL-8 production varied significantly. Coarse particles induced a 5-25 fold change in IL-6 release for AMs and a 3-6 fold change in IL-8 release for NHBEs. **Metal Correlation to IL-6/8 induction:** Fe and Si were positively associated with IL-6 release in AMs incubated with the coarse fraction. Cr was positively associated with IL-8 release in NHBE cells incubated with F or UF.
Reference: Beck-Speier et al. (2005, 156262) **Species:** Human, Canine (Beagle) **Cell Types:** Human AMs, Canine AM (CAM)	DEP = SRM 1650a (NIST) EC = Ultrafine Elemental Carbon (spark discharge) P90 = Printex 90 (Carbon Black, Degussa) PG = Printex G (Carbon Black, Degussa) **Particle Size:** (in diameter) DEP: 20-40nm; EC: 5-10nm; P90: 14nm; PG: 51nm	**Route:** Cell Culture (1X10^6 cells/mL AM) **Dose/Concentration:** All particles: 1 (EC only), 3.2, 10, 32, 100 μg/mL **Time to Analysis:** 60min	**Phagocytosis:** All particles were phagocytosed by CAM within 60 min. **Oxidative Potential:** EC showed a very high effect. DEP, P90 and PG had no effect **Formation of Lipid Mediators:** DEP, EC P90 and PG increased arachidonic acid and PGE2/TXB2 in CAM in a dose-dependent manner. Only EC increased LTB4 and 8-isoprostane. **ROS Activation:** All particles increased activity in canine macrophages with EC, P90 and PG increasing activity in a dose-dependent manner. DEP increased activity in canine macrophages. Similar results were observed human alveolar macrophages but only EC and P90 were tested. **Particle Mass vs Particle Surface Area:** PGE2/TXB2 effects were highly correlated with particle surface area.

Study	Pollutant	Exposure	Effects
Reference: Bitterle et al. (2006, 156276) **Species:** Human **Cell Type:** A549	C-UFP = ultrafine carbonaceous particles (obtained from a spark discharge aerosol generator GFG 1000, Palas, Karlsruhe, Germany) **Particle Size:** 90nm (count median mobility diameter)	**Route:** Cell Culture (3 x 10^7 cells) **Dose/Concentration:** 44 ± 4 ng/cm^2; 87 ± 23 ng/cm^2; 230 ± 70 ng/cm^2 (ng/cm^2 = total mass of deposited particles per cm^2 cell monolayer after 6h exposure) **Time to Analysis:** 6h	**Cell Viability:** Exposure to clean air resulted in a 93.7 ± 9.1% viability. Exposure to low, mid nad high doses of C-UFP resulted in a 94.9 ± 9.5% viability. Thus C-UFP had no effect on cell viability. **Interleukins:** Clean air controls induced a 2-3 fold increase in IL-6 and IL-8 production vs submersed control. U-CFP exposures induced a similar effect on IL-8 and IL-6 levels. **Antioxidant enzyme HO-1:** The mid dose increased transcription of HO-1 by 2.7 fold. There was no observed effect at the high dose level which indicates possible cytotoxcity.
Reference: Blanchet et al. (2004, 087982) **Species:** Human **Cell Type:**16HBE	$PM_{2.5}$ (Vitry-sur-Seine, Paris, France) DEP = SRM 1650a CB = Carbon Black (Degussa) TiO_2 (Huntsman) **Particle Size:** CB: 95 nm; TiO_2: 150 nm	**Route:** Cell Culture (45,000 cells/cm^2) **Dose/Concentration:** All particles: 0.1, 1, 10, 30 μg/cm^2 **Time to Analysis:** 6, 18, 24, 30h	**Amphiregulin Expression:** DEP and $PM_{2.5}$ both increased AR mRNA expression from 6 to 30h, with $PM_{2.5}$ inducing higher expression levels than DEP. Both DEP and $PM_{2.5}$ increased AR protein secretion. No observed effect for CB and TiO_2. $PM_{2.5}$ induced protein secretion dose-dependently. **Signal Pathways in AR Secretion:** MAP kinase and tyrosine kinase inhibitors reduced effects of DEP and $PM_{2.5}$ but p38MAP kinase inhibitor did not. **Role of Oxidative Stress:** N-Acetylcysteine blocked AR secretion following $PM_{2.5}$. Antioxidant enzyme catalase had no effect. **Cytokines:** DEP induced a significantly high release of GM-CSF, higher than $PM_{2.5}$. EGFR antibody reduced GM-CSF release at 0.25 μg/mL dose.
Reference: Bonvallot et al. (2001, 156283){, 2001 #7852} **Species:** Human **Cell Type:** 16HBE14o-	DEP: SRM 1650 OE-DEP: dichloromethane extract (2x) of DEP nDEP: native DEP sDEP: nDEP - OE-DEP CB: Carbon Black FR103 (Degussa) BaP: Benzo[a]pyrene CB: 95nm NR **Particle Size:** CB: 95nm; DEP: NR	**Route:** Cell Culture (3 x 10^6 cells) **Dose/Concentration:** DEP, sDEP, nDEP and CB = 10 μg/cm^2 OE-DEP = 15 μg/mL BaP = 0.25, 50 and 250 μg/mL **Time to Analysis:** 24h	**Proinflammatory Response:** At 10μg/cm^2, nDEP induced GM-CSF release by 4.7 fold. OE-DEP increased GM-CSF by 3.7 fold. BaP and sDEP also induced increases of CN-CSF but had smaller effect. CB had no effect. **NF-κB Activation:** nDEP and OE-DEP induced enhanced degradation of IκB at 2-4h and 1h respectively. NF-κB DNA binding was enhanced by OE-DEP (15 μg/mL, peak < 1h) and nDEP (10 μg/cm^2, peak at 2-h with plateau till 4 h). Both OE- and nDEP enhanced NF-kB DNA binding levels were higher than BaP enhanced binding levels. **CYP1A1 mRNA:** The CYP1A1 mRNA level was markedly increased in nDEP and OE-DEP treated cells in comparison with their respective controls. **Radical Scavengers (decreased ROS in situ):** Increases of GM-CSF and NF-κB DNA binding by nDEP and OE-DEP was attenuated by radical scavengers. **MAPK Activation:** Increases by nDEP and OE-DEP of GM-CSF was inhibited by Erk1/2 inhibitor but not by p38 inhibitors. Both nDEP and OE-DEP triggered Erk1/2 and p38 phosphorylation. sDEP affected p38 phosphorylation only.
Reference: Brown et al. (2007, 156300) **Species:** Human, Mouse **Cell Type:** PBMC, A549 (Human); J774A.1 (Mouse)	PM_{10} (London, England) CM from PM_{10}-treated human monocytes **Particle Size:** PM_{10}	**Route:** Cell Culture (1 x 10^8 cells/mL J774A.1; 5X10^6 cells/mL PBMC; 5X10^5 cells/well A549) **Dose/Concentration:** PM_{10}: 75 μl (10 μg/mL); CM: 250 μl; tBHP: 12.5 μm (in J774); TNF: 0, 500pg, 1ng, 10ng , **Time to Analysis:** tBHP:1, 2, 4h; PM: 4h; TNF: 18h	**Cytokines:** PM_{10} induced release TNF-α protein from PBMCs at 10μg/mL for 4h. Further inhibited by verapamil and BAPTA-AM. Calmodulin inhibitor W-7 had no effect. CM increased IL-8 from A549 cells 3 fold. Verapamil, BAPTA-AM and W-7 significantly inhibited IL-8 release induced by CM. **ICAM-1:** A549 cells treated with TNF-α showed dose-dependently effect of TNF-α on ICAM-1 upregulation at 18h. CM also induced upregulation. Verapamil, BAPTA-AM and W-7 fully inhibited CM-induced upregulation.

Study	Pollutant	Exposure	Effects
Reference: Calcabrini et al. (2004, 096865) **Species:** Human **Cell Type:** A549	$PM_{2.5}$ (Rome, Italy) **Particle Size:** $PM_{2.5}$	**Route:** Cell Culture (5 x 10^4 cells/well) **Dose/Concentration:** 30, 60 $\mu g/cm^2$ (aliquot of 0.1 μg/ul) **Time to Analysis:** 5, 24, 48, 72h	**Particle Characterization:** Components measured include C-rich particles, Ca sulfates, silica, silicates, Fe-rich particles, metals. Carbonaceous particles made up majority of PM. **Cell Surface Changes:** PM deposited on the cell surface showed dose and time-dependent increases in microvilli rearrangement and cell shape alterations without affecting apoptotic markers for up to 72 h. **PM internalization:** At 24 h with the low dose, aggregates of PM in cytoplasm or surrounded by membrane was observed. With the high dose, large particle aggregates often close to nuclear envelopes were observed. **Cytoskeleton:** At 72h PM induced dose-dependent alterations from rearrangement/interweaving of microtubules to bundling of microtubules with some shortening/disruption. **Cell Growth:** PM decreased cell growth in a dose and time –dependently manner **ROS:** PM increased ROS at the high dose for 5h but not at 24 h or with the low low dose. **Cytokines:** PM induced TNF-α peaked at 5h at high dose and 48h at low dose, both ND at 72h. PM induced IL-6 starting at 24 h thru 72h in time and dose dependent manner.
Reference: Cao et al. (2007, 156322) **Species:** Human **Cell Type:** HAEC	NIST-DEP: collected using a diesel forklift and hot bag filter system. (NIST, Minneapolis, MN) C-DEP: obtained from a 30-kw (40 hp) four-cylinder Deutz BF4M1008 diesel engine (U.S. EPA) Organic extract fraction of particles NIST- DEP 2% C-DEP 20 % **Particle Size:** NR	**Route:** Cell Culture (5 x 10^5 cells) **Dose/Concentration:** NIST-DEP, C-DEP: 0, 12.5, 25, 50, 100, 200 μg/mL **Time to Analysis:** 1-4h	**Cell Viability:** DEP had no effect. **Stat3:** Both DEPs induced time-dependent phosphorylization of Stat3 in cytoplasm. NIST-DEP induced phosphorylization dose-dependently from 12.5 to 50 μg/mL but stayed level at 100 and 200 μg/mL. p-Stat3 induction was inhibited by antioxidant BHA though it was reactivated with exposure to H2O2. Reaction induced by H2O2 was similar to that of DEP. **pStat3 Nuclear Transport:** NIST-DEP induced cytoplasmic pStat3 to move from cytoplasm into nucleus. **pEGFR Dephosphorylation:** After 4h of NIST-DEP exposure, dephosphorylation was inhibited for up to 90 min.
Reference: Chang C-C et al. (2005, 097776) **Species:** Human **Cell Type:** A540, THP-1	UfCB (Printex 90, Degussa) **Particle Size:** 14nm diameter	**Route:** Cell Culture (7 x 10^5 cells) **Dose/Concentration:** 100 μg/mL **Time to Analysis:** 4h	**ROS in THP-1 and A549**: UFCB increased ROS. NAC pretreatment blocked most of the UFCB-induced ROS production. **VEGF in THP-1:** UFCB increased VEG. NAC decreased the UFCB effects below those of the control. **VEGF in A549:** Produced similar, but less marked, results as with THP-1.
Reference: Chauhan et al. (2004, 096682) **Species:** Mouse **Strain:** BALB/c **Cell Type:** RAW 264.7 (leukemia virus transformed macrophages); J774A.1 (from a tumor); WR19M.1 (leukemia virus transformed macrophages)	EHC-T: total EHC-93 (Env Health Ctr, Ottawa, Canada) EHC-I: insoluble EHC EHC-S: soluble EHC SRM1648: urban particulate St.Louis (NIST) SRM1649: urban dust/organics Washington (NIST) VERP: fine $PM_{2.5}$ (Vermillion, Ohio) Cristobalite: SRM 1879 (NIST) TiO_2: SRM 154b (NIST) **Particle Size:** EHC-93: 0.5 μm (median diameter); Cristobalite, SRM 1648, SRM 1649, TiO_2,: NR; VERP: $PM_{2.5}$	**Route:** Cell Culture (15000 cells/well) **Dose/Concentration:** Particle suspensions: 20, 50, 100 μg/well LPS: 0-5 μg/mL IFN-γ: 0-1000 U/mL **Time to Analysis:** Particles added to culture at 0h, LPS and IFN-γ added at 2h. Parameters measured after 22h incubation period.	**Stimulation with LPS/IFN-γ:** LPS and IFN-γ each induced NO release. Combination of LPS and IFN-γ produced larger effect in all cell lines. L-NMMA, NOS inhibitor, suppressed most of the NO production with 100nmol/L. **Cellular Viability and Cytotoxicity:** Exposure of cells to particulates did not result in overt cytotoxicity or excessive loss of cellular material. There was no correlation between the cytotoxicity of the particles in the surviving cells and the loss of protein mass in monolayers. **Nitrite Production:** EHC-T, EH-93-I, SRM1648 and SRM 1649 produced dose-dependent decreases. Cristobalite only decreased at higher doses. No effect from EHC-S, VERP or TiO_2. **iNOS:** EHC-I, EHC-T, Crisobalite and SRM1648 inhibited iNOs expression. TiO_2 had no effect. EHC sol, SRM 1649 and VERP were not tested.

Study	Pollutant	Exposure	Effects
Reference: Chauhan et al. (2005, 155722) **Species:** Human **Cell Type:** A549	EHC-T: total EHC-93 EHC-I: insoluble EHC EHC-S: soluble EHC Cristobalite (SiO_2): SRM-1879 TiO_2: SRM-154b **Particle Size:** EHC-93: 0.4 μm (median physical diameter); TiO_2, SiO_2: 0.3-0.6 μm	**Route:** Cell Culture (150000 cells/flask) **Dose/Concentration:** All particles: 0, 1, 4, 8 mg/5ml **Time to Analysis:** 24h	**Cellular Viability:** Decreased after exposure to EHC-T, EHC-I and cristobalite. Rate of reduction was not consistent across doses. EHC-S and TiO_2 had no effect on viability. **ET-1:** Release of ET-1 peptide decreased dose-dependently for EHC-T, -S and -I. Fractions of EHC-S and EHC-I were more potent than EHC-T. TiO_2 and Cristobalite also reduced ET-1 secretion although this was not consistent across the dose range. **Cytokines:** Results showed no detectable amounts of GM-CSF, IL-1β or TNF-α in cell culture supernatants. IL-8 increased dose-dependently with EHC-T, EHC-I and cristobalite. **VEGF:** VEGF significantly increased dose-dependently with EHC-T, EHC-S and cristobalite. EHC-S induced a significant decrease in VEGF. **Gene Expression:** mRNA levels for preproET-1 reduced at 24 h for all particle types. EHC-S induced a significant decrease in ET-1 expression at this high dose. ECE-1 mRNA expression increased with EHC-T and EHC-I. Other particles had no effect. ETaR mRNA increased with EHC-T, EHC-S, and TiO_2 in biphasic manner where the highest expression of mRNA was seen at the middle dose levels. EHC-S had no effect. ETbR mRNA increased with a low dose of EHC-T and decreased with a high dose of EHC-T. EHC-S, EHC-I and cristobalite induced an increase of ETbR. TiO_2 induced a significant decrease. **Proteases:** mRNA levels for MMP-2 reacted similarly to preproET-1. mRNA levels for TIMP-2 was significantly induced with EHC-I. EHC-T and EHC-S induced small effects.
Reference: Cheng et al. (2003, 156337) **Species:** Human **Cell Type:** A549	DEP-h: DEP with high sulfur DEP-LS: DEP with low sulfur GEP: gasoline engine exhaust particles Primed cells pretreated with TNF-α **Particle Size:** DEP-h: 15.9nm; DEP-LS: 17.7nm; GEP: 8.3nm	**Route:** In Vitro Cellular Exposure (Exhaust flow-through cell culture with air-cell-interface, exhaust diluted 10-15x with 8x105 cells/mL) **Dose/Concentration:** DEP (total): 1.5-3.5x10^6 particles/cm^3 air; GEP (total): 1-2x10^6 particles/cm^3 air; TNF-γ: 5ml (25 ng/ml) **Time to Analysis:** 60-360 min	**IL-8:** DEP-h induced a 3 fold increase in IL-8 than that of the control. DEP-LS also induced increases. Primed cell cases had higher levels (10x) than unprimed when exposed to DEP-LS. DEP-h induced higher levels of IL-8 than DEP-LS. This response lasted for up to 6h. GEP induced a statistically insignificant increase of IL-8 in unprimed cells. WIth primed cells, GEP induced levels of IL-8 that exceeded those of DEP-h and DEP-LS. This response lasted for 1-2 h.
Reference: Chin et al. 2002 **Species:** Rat, Human **Cell Line/Type:** RAW 264.7 (Rat), MHS Alveolar Macrophage Cell Line (Rat), A549 (Human)	CB: (N339, with benzo[a]pyrene absorbed on surface. Manufactured in Cabot, Boston, MA) BaP Benzo [a] pyrene 1, 6-quinone: BP-1,6-Q (obtained from NCI, Kansas City, MO) **Particle Size:** CB 0.1 μm (mean diameter)	**Route:** Cell Culture **Dose/Concentration:** CB: 1, 2, 4μg/mL BaP: 2μg/mL BP-1,6-Q: 1μM **Time to Analysis:** 1-24h	**HO-1 mRNA Expression:** In RAW264.7, HO-1 mRNA levels increased with 2 and 4 μg/mL at 2h. Increases continued to 8h and declined by 24h. BaP had no effect. BP-1,6-Q increased HO-1 mRNA after 1h and was maintained until 8h. In A549 and MHS, HO-1 mRNA increased after 1h, peaking at 8h in A549 and 4h in MHS. **HO-1 Protein Expression:** An increase of protein was observed from 4-8h in RAW264.7. **AP-1:** Increases in binding activity were observed in RAW 264.7 cells at 2h.

Study	Pollutant	Exposure	Effects
Reference: Churg A et al. (2005, 088281) **Species:** Rat **Strain:** Sprague-Dawley **Weight:** 250g **Cell Type:** Epithelial Cells of Tracheal Explants	EHC93 (Ottawa Urban Air Particles) TiFe = Iron-loaded fine TiO_2 (obtained from Aldrich Chemicals, Milwaukee, WI) 3-4um EHC93 0.12 ± 1.4 μm TiO_2 **Particle Size:** EHC-93: 3-4 μm (MMAD); TiFe: 0.12 ± 1.4 μm (geometric mean diameter)	**Route:** Cell Culture **Dose/Concentration:** EHC-93, TiFe: 500 μg/cm^2 **Time to Analysis:** 1, 24h. Some experiments (referred to as 2h) explants transferred to different dish and incubated for additional hour. Pre-treated with Inhibitors/Chelators for 2h.	**Activation of NF-κB:** Both particle types increased nuclear translocation of NF-κB. TiFe and EHC-93 increased NF-κB 1.5 fold at 1h. TiFe increased NF-κB 3.5 fold at 2h. EHC-93 increased NF-κB more than 2 fold. TiO_2 by itself did not increase NF-κB at any exposure duration. **Activation of NF-κB into tracheal epithelial cells:**No evidence of dust particles was observed (EHC-93 or TiO_2) in the epithelial cell cytoplasm at 2h. No evidence of morphologic cell damage from particles was observed. **Colchicine:** Treatment with colchicine did not prevent NF-kB activation. **Inhibitors/Activators:** Tetramethylthiourea (TMTU) (membrane-permeable active oxygen scavenger), Deferoxamine (redox-inactive metal chelator), PPS (Src inhibitor) AG1478 (epidermal growth factor receptor inhibitor) prevented NF-κB activation in both EHC93 and TiFe exposed-cells. Iron-containing citrate extract of both dusts increased NF-κB activation in both EHC93 and TiFe exposed-cells.
Reference: Courtois et al. (2008, 156369) **Species:** Rat **Strain:** Wistar **Cell Line:** Dissected intrapulmonary arteries from rats used in corresponding in vivo experiments	PM (SRM 1648) (63% inorganic carbon, 4-7% organic carbon, mass fraction > 1%: Si, S, Al, Fe, K, Na) UF carbon black (FW2, P60) **Particle Size:** SRM 1648 mean diameter 0.4 μm; ultrafine carbon black: FW2- 13nm, P60-21nm	**Route:** Cell Culture **Dose/Concentration:** 100, 200 μg/mL **Time to Analysis:** 24h incubation	**NO:** Generally, Ach-induced relaxation in intrapulmonary arteries decreased, Ach-induced cGMP accumulation decreased, and relaxation by SNP or DEA-NO also decreased. UF carbon black did not affect NO responsiveness. **Oxidative Stress, Inflammatory:** Dexamethasone prevented SRM 1648-induced impairment of the Ach relaxation response but antioxidants did not. TNF-α, MIP2, IL-8 increased. ROS was not affected.
Reference: Dagher et al., (2007, 097566) **Species:** Human **Cell Type:** L132 (Normal Lung Epithelial Cells)	LC10, LC50 = $PM_{2.5}$ (collected Jan-Sept in Dunkerque, France) **Particle Size:** cumulative frequency: 0.5 μm: 34%; 1 μm: 64%; 1.5 μm: 79%; 2 μm: 87%; 2.5 μm: 92%; 5 μm: 98%; 10 μm: 100%	**Route:** Cell Culture (3 x 10^6, 1.5 x 10^6, 0.75 x 10^6 cells/20mL) **Dose/Concentration:** LC10: 19 μg/mL; LC50: 75 μg/mL **Time to Analysis:** 24, 48 or 72h	**p65 Protein:** Phosphorylation of p65 increased in PM-exposed L132 cells in dose-dependent manner. **IkBα Protein:** Phosphorylated IkBα protein concentrations increased in cytoplasm with both particle types at all time points. **p65 and p50 DNA:** p65 DNA binding increased at 24h with LC10 and LC50, at 48h with LC10, and at 72h with LC10 and LC50. p50 DNA binding increased at all time points with LC10 and LC50.
Reference: Dai et al. (2003, 087944) **Species:** Rat **Strain:** Sprague-Dawley **Weight:** 250g **Cell Type:** Tracheal Explants	EHC-93 (Environmental Health Center, Ottawa) DEP: SRM 1650a (NIST) **Particle Size:** EHC-93: 3-4 μm (MMAD); DEP 1.55 ± 0.04 μm (CMD)	**Route:** Cell Culture **Dose/Concentration:** ECH, DEP: 500 μg/cm^2 **Time to Analysis:** Exposed for 1h. Parameters measured following a 7d incubation period.	**Hydroxyproline:** EHC93 induced an almost 3 fold increase in explant hydroxyproline. DEP increased tissue hydroxyproline 2.5 fold. **Procollagen:** EHC-93 doubled gene expression of procollagen. Procollagen gene expressioncould be fully inhibited by SN50, TMTU or treatment of the PM with DFX. Treatment of explants with p38 or ERK (inhibitors) had no effect on procollagen expression. DEP induced an increase in procollagen gene expression but this increase was completely prevented by SN50 and MAP kinase inhibitors (SB203580 and PD98059). Neither TMTU or DFX has any effect. **TGFβ1:** Treatment of explant with EHC93 approximately doubled gene expression for TGFβ1. Treatment with SN50, TMTU and fetuin (TGFβ antagonist) blocked increase. DFX, MAP kinase inhibitors (SB203580 and PD98059) had no effect. DEP roughly doubled TGFβ1 expression.SN50 and MAP kinase inhibitors (SB203580 and PD98059) fully blocked this effect. TMTU and DFX had no effect.

Study	Pollutant	Exposure	Effects
Reference: Doherty SP et al. (2007, 096532) **Species:** Rat **Strain:** NR8383 **Cell Types:** AMs	Ratios of: V: Fe; Al: Fe; Mn: Fe V = sodium vanadate (NaVO3) Al = aluminum chloride hexahydrate ($AlCl_3$) Mn = manganese chloride tetrahydrate ($MnCl_2$) Fe = ferric chloride hexahydrate ($FeCl_3$) Ratios based on $PM_{2.5}$ measurements from NYC, LA and Seattle **Particle Size:** Metals from $PM_{2.5}$ samples	**Route:** Cell Culture ($2X10^5$ cells/mL) **Dose/Concentration:** Fe = 16 μmol (equivalent to urban NYC 500 μg $PM_{2.5}$); V and Mn tested in molar rations of 0.02 to 0.4 relative to Fe; Al tested in molar ratios of 0.125 to 8 relative to Fe. **Time to Analysis:** 20h	**IRP:** Addition of V increased IRP activity 5 to 9 fold. Though there was no seeming dose responsivity, IRP activity remained strongly elevated over the range of V:Fe ratios tested. Addition of Mn only resulted in an effect at 0.1 molar ratio (two-fold), not at higher or lower ratios. Al resulted in peak increases of 5 fold at molar ratios 2 while declining to 2 fold at molar ratios 4 and 8. **Cytoxicity:** Al was cytotoxic at molar ratios of 4 and 8. All other Al, V, Mn ratios had no effect. **Mixtures:** The combination of metals tested at NYC PM ratios and V drove all the Fe transport activity. Combinations of V+Mn and V+Al increased activity more than V:Fe alone.
Reference: Doornaert, et al. (2003, 156410) **Species:** Human **Cell Line/Type:** 16HBE14o-; P-HBE	DEP: SRM 1650 (NIST) CB: (Sigma, France) DPC: Dipalmitoyl phosphatidylcholine (positive control) 0.5um **Particle Size:** NR	**Route:** Cell Culture (10^5-$2x10^5$) **Dose/Concentration: D**EP and CB: 1- 100 μg/mL **Time to Analysis:** Parameters measured 24, 48, 72h post exposure. 1-HBE Cell Deadhesion Capacity: 24h, evaluation of detachment performed every 5min for 40min after. Cell Wound Repair Capacity: 24 h, repair evaluated 3.5, 7, 24 h after.	**Cytotoxicity:** DEP was cytotoxic at 100 μg/mL at all time points in a time-dependent manner. CB and DPC cytotoxicity was substantially lower but significant at 72h. **Phagocytosis:** 1-HBE cell levels that were in contact with DEP or CB or have phagocytized those particles increased in a dose-dependent manner. DEP induced greater levels of cell contact and phagocytosis than CB. **F-actin:** Only DEPs were engulfed by F-actin stained cell fragments. **Actin CSK Stiffness:** DEP (5, 20, 100 μg/mL) induced net dose-dependent decrease in cytoskeleton stiffness and a dose-dependent decrease in actin cytoskeleton stiffness. CB produced no significant decrease. **Adhesion Molecules**: DEP induced a concomitant reduction of both CD49 (α3) and CD29 (β1) integrin subunits and a decrease in level of CD44 (HBE cell-cell and cell-matrix adhesion molecule) at both 20 and 100μg/mL. **Proteases:** DEP also induced an isolated decrease in MMP-1 expression without change in tissue inhibitor of TIMP-1 or TIMP-2 at 100 μg/mL. CB produced no change or insignificant results. **1-HBE Cell Deadhesion Capacity:** DEP exposure induced a dose-dependent amplification of cell detachment at 5min of incubation and onward. **Cell Wound Repair Capacity:** DEP inhibited wound repair/wound closure in a dose-dependent manner.
Reference: Dostert et al. (2008, 155753) **Species:** Human **Cell Line/Type:** THP1, monocyte-derived macrophages (MM)	Asbestos Silica DEP CSE: cigarette smoke extract MSU: monosodium urate crystals **Particle Size:** NR	**Route:** Cell Culture **Dose/Concentration:** Asbestos: 0.1, 0.2 mg/mL; Silica: 0.1, 0.2, 0.25, 0.5 mg/mL; DEP: 0.2, 0.25, 0.5 mg/mL; CSE: 5%, 10% in solution mg/mL; MSU: 0.1, 0.2 mg/mL **Time to Analysis:** 1, 3, 6h	**IL-1β:** Increased levels of IL-1β with asbestos and silica were observed in THP1 at 6h. CSE and DEP had no effect. MM also had increased levels with asbestos, silica and MSU at high dose levels only. **Caspase-1:** Asbestos increased caspase-1 activity. **ROS:** Asbestos doses in THP1 exhibited an increase in ROS formation.
Reference: Doyle, et al. (2004, 088404) **Species:** Human **Cell Type:** A549 from non-smoking adults	BD: 1,3-butadiene, known carcinogen Acrolein: photochemical and NO product of BD in atmosphere Acetaldehyde: photochemical and NO product of BD in atmosphere Formaldehyde: photochemical and NO product of BD and ISO in atmosphere ISO: isoprene, 2-methyl analog of BD Methacrolein: photochemical and NO product of ISO in atmosphere Methyl vinyl ketone: photochemical and NO product of ISO in atmosphere **Particle Size:** NR	**Route:** Environmental Irradiation (smog) Chambers **Dose/Concentration:** 50 ppb NO; 200 ppbV ISO, BD **Time to Analysis:** Exposed to gases for 5h. Analysis 9h post exposure.	**Cytotoxicity:** ISO+NO and BD+NO induced small increases of LDH in A549. However, ISO+NO+light and BD+NO+light increased LDH levels 4-6 fold indicating photochemical products of ISO and BD are highly cytotoxic. LDH levels of each combination were equivocal. **IL-8 Protein:** Methacrolein, methyl vinyl ketone and formaldehyde (products of ISO) increased IL-8 protein levels significantly. ISO+NO had no effect. BD photochemical products (acrolein, acetaldehyde and formaldehyde) also increased IL-8 protein, more than doubling the photochemical products induced by ISO. BD+NO had no effect. **IL-8 mRNA:** IL-8 mRNA expression also increased with photochemical products of ISO and BD but did not reach a statistically significant level.

Study	Pollutant	Exposure	Effects
Reference: Duvall, R.M. (2008, 097969) Norris, G.A. Dailey, L.A. 2008 **Species:** Human **Cell Type:** Airway Epithelial Cells	PM-F, -C, -UF Particles collected from: Seattle, WA (PM-S); Salt Lake City, UT (PM-SL); Phoenix, AZ (PM-P); South Bronx, NY (PM-SB); Hunter College, NY (PM-HRterling Forest, NY (PM-SF) **Particle Size:** Coarse: $>2.5\ \mu m$; Fine: $<2.5\ \mu m$; Ultrafine: $<0.1\ \mu m$	**Route:** Cell Culture (100,000 cells/cm^2) **Dose/Concentration:** 5 mg/ml **Time to Analysis:** 1, 24h post exposure	**Particle Characterization:** PM-HR, PM-SL and PM-S contained the highest UF, F, and C concentrations. PM-SB and PM-HR had similar F and C concentrations. Sulfate was highest in PM-F for all sites except in PM-SB and PM-HR. Wood combustion was highest in PM-SL, PM-S, PM-P. Soil dust was highest in PM-SL and PM-S. **IL-8:** PM-UF induced a greater increase in IL-8 than other types of PM except PM-P. PM-UF is associated with vanadium, lead, copper, sulfate. PM-F-HR caused the greatest increase followed by PM-SB. PM-F-SF and PM-F-P was least effective. PM-C also caused an increase in IL-8 levels and was associated with vanadium and elemental carbon. **COX-2:** PM-F-S induced the greatest increase in COX-2 expression. Other PM-F sites induced similar increases. UF PM had no effect. PM-C, associated with elemental carbon, induced increases. **HO-1:** PM-F-SF induced the greatest increase in HO-1. PM-F-SL was the least effective. UF PM had no effect. PM-C, associated with copper, barium and elemental carbon, caused an increase.
Reference: Dybdahl M et al. (2004, 089013) **Species:** Human **Cell Type:** A549	DEP: SRM 1650 (NIST) **Particle Size:** 90nm (MMAD)	**Route:** Cell Culture (10^5 cells/mL) **Dose/Concentration:** 0, 10, 50, 100, 500 μg/mL **Time to Analysis:** 2, 5, or 24h	**Cytokines:** DEP induced dose-dependent increases of IL-1α, IL-6, IL-8 and TNF-α at 24 h. Cytokines increased between 4 and 18 fold at the highest DEP dose as compared to controlled cells. DEP also increased IL-6 mRNA expression levels in a dose and time-dependent manner.IL-6 mRNA levels increased 14 fold at 24 hours, 8 fold at 5 hours, and 2 fold at 2 hours. **Cell Viability:** DEP exposure did not decrease cell viability at any dose tested.
Reference: Dybdahl M et al. (2004, 089013) **Species:** Mouse **Strain:** BALB/CJ **Gender:** Female **Age:** 10wks **Weight:** ~20g	DEP: SRM 1650 (NIST) **Particle Size:** 90nm (MMAD)	**Route:** Nose-only Inhalation **Dose/Concentration:** Single exposure: 20 or 80 mg/m^3; 4 exposures: 5 or 20 mg/ m^3 **Time to Analysis:** Single 90min or four 90min exposures.	**Inflammation:** DEP induced a dose- and time-dependent expression of pro-inflammatory cytokines. A single high dose of DEP induced an increase in IL-6 mRNA levels persisting for at least 22 hours. However, at the same cumulative dose given as 4 doses, the IL-6 level was only increased 1 hour after exposure. **Cytokines:** Only IL-6 was induced after DEP exposure. A single 90 minute inhalation (at 20 mg/m^3) increase IL-6 gene levels dose-dependently in the lung and was significantly higher than control levels at both 1 and 22 hours post exposure. Repeated exposures at 5 mg/m^3 did not affect IL-6 levels. **BAL Cells:** Inhalation of DEP did not decrease viability of BAL cells. 1 hour after the last dose, mice exposed to 20 mg/m^3 exhibited a 3 fold increase in total cell numbers as compared to control. The number of total cells and neutrophils in BAL fluid were still increased 22 hours after exposure to 20 mg/m^3 DEP. BAL cell population was not affected by repeated exposures to 5 mg/m^3 DEP. Lymphocyte numbers were also unchanged by DEP.

Study	Pollutant	Exposure	Effects
Reference: Fritsch, S.Diabate, S. (2006, 156452) **Species:** Mouse **Cell Type:** RAW 264.7	MAFO2: incinerator fly ash (collected by electrostatic precipitation in commercial municipal waste incinerator facility) composition representing 12% of total mass (mg/g): Fe (9.1); Pb (23.3); Zn (75.7); C (7.5) **Particle Size:** 165nm (modal value)	**Route:** Cell Culture (1 x 10^6 cells/well) **Dose/Concentration:** 6.3-188 μg/cm^2 for Toxicity; 2.6, 6.5, 13.2 μg/cm^2 for Arachidonic Acid; 13.2 μg/cm^2 for MAPK Pathway; Other doses noted in Effect of Particles **Time to Analysis:** 1, 2.5, 5, 24 h	**Toxicity:** Viability decreased from 99% to 18% at 62.5-188 μg/cm^2. Lower doses had no effect. **Arachidonic Acid:** At 2.5h, AA level increased 2 fold for 6.5 μg/cm^2 and 6 fold for 13.2 μg/cm^2. No increase was observed after 5h. **MAPKs:** Cells pretreated with PD98059, an inhibitor of MEK-1, inhibited AA liberation due to MAFO2 treatment of 13.2 μg/cm^2 **COX-2:** A time-dependent increase of COX-2 protein expression was exhibited at 2.5 and 5h. **ROS:** A dose-dependent increase in ROS formation was observed at concentrations greater than 31.3 μg/cm^2 after 3 hours. **GSH:** There was an observed increase of production at 20h. Doses greater than 60 μg/cm^2 reduced total glutathione. **HO-1:** There was an observed dose-dependent increase in expression at 4 hours.
Reference: Fujii et al. (2002, 036478) **Species:** Human **Cell Type:** HBEC (from current smokers), AMs, Co-Culture: AMs + HBEC **Age:** HBEC: 48-70yrs	PM_{10}: EHC-93 (Ottawa, Canada) **Particle Size:** PM_{10}	**Route:** Cell Culture (HBEC: 2.5-3 x 10^6 cells/well); (AMs: 1.0 x 10^7 total) **Dose/Concentration:** 100, 500 μg/mL **Time to Analysis:** 2, 8, 24h	**Viability:** Over 90% of HBEC were viable after a 24 hour exposure of up to 500 μg/mL of PM. AMs incubated with and without 100μg/mL saw no significant difference in viability. **Cytokine mRNA:** TNF-α, GM-CSF, IL-1β, IL-6, LIF, OSM and IL-8 mRNA expression increased in co-culture with 100 μg/mL at 2 and 8h. In AMs, TNF-α, IL-1β, IL-6 mRNA expression increased with 100 μg/mL at 2h. IN HBECs, IL-1β and LIF increased with 100 μg/mL at 2h. HBECs added to AMs exposed to PM_{10}, further increase in mRNA of IL-1β, LIF and IL-8. **Cytokine Protein:** In co-culture and AMs, significant increase in protein production of GM-CSF, IL-8, IL-1β, IL-6 and TNF-α in dose-dependent manner. GM-CSF and IL-6 production significantly higher in co-culture then AM or HBEC alone. **Bone Marrow:** Co-culture instillation of supernatants increased circulating band cell counts at 6 and 24 h with 100 μg/mL.
Reference: Fujii, T. Hayashi, S. Hogg J.C. (2001, 156455) 2001 **Species:** Human **Cell Type:** HBEC from current smokers **Age:** 48-70yrs	PM_{10}:EHC93 (Ottawa, Canada)99% $<$ 3.0um **Particle Size:** PM_{10}(99% $<$ 3.0 μm)	**Route:** Cell Culture (2.5-3 x 10^6 cells/dish) **Dose/Concentration:** 10, 100, 500 μg/mL **Time to Analysis:** 2, 8, 24h	**Phagocytosis:** 18.6% of cells engulfed particles when exposed to 100 μg/mL. Over 90% remained viable. **Cytokine mRNA:** LIF mRNA increased dose-dependently at 2h but declined at 8 and 24 h. GM-CSF increased dose-dependently at 8h and peaked at 24 h. IL-1α increased at 2h, increased dose-dependently at 8 h and peaked at 24 h. M-CSF, MCP-1, IL-8 were unaffected. **Cytokine Protein:** LIF, GM-CSF, IL-1β and IL-8 increased dose-dependently. Soluble fraction of 100 μg/mL PM_{10} did not affect cytokine production.

Study	Pollutant	Exposure	Effects
Reference: Garcon, G. Dagher, Z. Zerimech, F. (2006, 096633) **Species:** Human **Cell Type:** L132 (Embryonic Lung Epithelium)	$PM_{2.5}$ (collected in Dunkerque, France for 9mo, Jan-Sept) **Particle Size:** $PM_{2.5}$- 0-0.5um (33.63%), 0.5-1.0um (30.61%), 1.0-1.5um (14.33%), 1.5-2.0um (8.69%), 2.0-2.5um (4.89%), > 2.5um (7.87 %)	**Route:** Cell Culture: 3 x 10^6 cells/20ml (24 h); 1.5 x 10^6 cells/20ml (48h); 0.75 x 10^6 cells/20ml (72h) **Dose/Concentration:** 18.84, 37.68, 56.52, 75.36, 150.72 μg/mL; LC10- 18.84 μg/mL; LC50- 75.36 μg/mL **Time to Analysis:** 24, 48 or 72h	**Cytotoxicity:** PM induced dose-dependent (R2 =.9907) cytotoxic effect in proliferating L132 cells. **LDH:** Increase at 72 h with 56.52 and 75.36 μg/mL. **Oxidative Stress:** A decrease in MDF activity was observed at all exposure levels at 24, 48, and 72 h (72-h < 5 % of control). MDA levels showed increase concentration after 72 h,both LC10 and LC50. LC10 and LC50 saw an increase in SOD activity at 24 h; LC50 saw a decrease in activity after 48 and 72 h. 8-OHdG and PARP exhibited increases at all time points with LC10 and LC50. **Inflammatory Response:** Increases of TNF-α concentration was exhibited at 24 h at LC50, and at 48 h and 72 h at LC10 and LC50. iNOS activity increase at all time points at LC10 and LC50.NO concentration exhibited increases at all time points after exposure to LC10 and LC50.
Reference: Geng H et al. (2005, 096689) **Species:** Rat **Strain:** Wistar **Tissue/Cell Type:** Lung macrophages	BPM: Blowing $PM_{2.5}$ NPM: Non Blowing Normal $PM_{2.5}$ **Particle Size:** $PM_{2.5}$ PM collected from Wuwei City, Gansu Province, China (Blowing days correspond to desert storm days)	**Route:** Cell Culture **Dose/Concentration:** 0, 33, 100, 300 μg/mL **Time to Analysis:** 4h	**NOTE**: Unless otherwise noted, results are identical for BPM and NPM. **Cytotoxicity**: Dosages greater than 150 μg/mL decreased cell viability. **Plasma Membrane Fluidity**: Dose-dependent decrease had no effect on membrane lipid hydrophilic region. **Plasma Membrane Permeability**: LDH enzyme activity and extracellular AP activity increased dose-dependently, indicating increased membrane permeability, but this was only statistically significant at 300 μg/mL dose. NPM may affect some parameters at 100μg/mL. Overall, NPM induced a slightly higher increase than BPM. **Intracellular Ca^{2+}:** A dose-dependent increase was observed. **Lipid Peroxidation (TBA):** An increase was observed only at 300 μg/mL. **Antioxidant (GSH):** A decrease was observed only at 300 μg/mL.
Reference: Geng H et al. (2006, 097026) **Species:** Rat **Strain:** Wistar **Tissue/Cell Type:** Lung macrophages	DPM: dust storm samples NPM: normal PM **Particle Size:** $PM_{2.5}$ PM collected from Baotou City, Inner Mongolia, China in March 2004	**Route:** Cell Culture **Dose/Concentration:** 0, 33, 100, 300 μg/mL **Time to Analysis:** 4h	**NOTE**: Unless otherwise noted, results are identical for BPM and NPM **Cytotoxicity**: MTT reduction assay revealed a significant decrease in cell viability at 150 μg/mL and 300 μg/mL. LDH enzyme activity significantly increased at 150 and 300 μg/mL. **GSH levels:** Significant decreases were seen in cellular GSH levels and increases in TBARS levels in both groups with a 300 μg/mL dose. **Plasma Membrane Activity:** In the plasma membrane, Na,K-ATPase were significantly inhibited. Ca2+Mg2+ -ATPase were unaffected. **Plasma Membrane Lipid Fluidity**: Results indicate that DPM could increase the surface fluidity of membrane lipid. **Intracellular Ca^{2+}:** A dose-dependent increase in free intracellular Ca2+ levels was observed.
Reference: Ghio et al. (2005, 088272) **Species:** Human **Cell/Tissue Type:** BEAS-2B	FAC: ferric ammonium citrate (component of ROFA) $VOSO_4$: vanadyl sulfate (component of ROFA) **Particle Size:** NR	**Route:** Cell Culture **Dose/Concentration:** 100μM FAC - preexposed before metal compounds or oil fly ash 50μM $VOSO_4$ - preexposed before metal compounds or oil fly ash 100μg/mL ROFA **Time to Analysis:** 0-1h, 4h	**IRE DMT1:** FAC increased mRNA and protein expression for –IRE DMT1. $VOSO_4$ decreased mRNA and protein expression for –IRE DMT1. +IRE DMT1 unaffected by any treatment. **Metal transport:** Uptake of iron increased after pre-exposure to FAC and decreased after pre-exposure to $VOSO_4$. Pre-exposure to FAC again increase the uptake of both iron and vandium. $VOSO_4$ induced opposite effect, decreasing Fe uptake. **ROS:** Increased acetaldehyde, indicating increased oxidative stress. ROS decreased with FAC pretreatment. ROS increased with $VOSO_4$ pretreatment.

Study	Pollutant	Exposure	Effects
Reference: Gilmour et al. (2004, 057420) **Species:** Rat **Strain:** Sprague-Dawley **Cell/Tissue Type:** Alveolar macrophage	Coal Fly Ash MU = Montana Ultrafine MF = Montana Fine MC = Montana Coarse KF = West Kentucky Fine KC = West Kentucky Coarse Coal combustion using a laboratory-scale down-fired furnace rated at 50kW. Montana subbituminous coal and western Kentucky bituminous coal **Particle Size:** Coarse: $>2.5\mu$m; Fine: $<2.5\mu$m; Ultrafine: $<0.2\mu$m	**Route:** Cell Culture ($2X10^5$ cells/mL) **Dose/Concentration:** 125 μg/mL or 250 μg/mL **Time to Analysis:** 4 or 24h	**LDH:** Mid and high doses of Montana ultrafine particles showed significant increase after 4 h exposure vs control. Other particle types had no effect. After 24 h, LDH level was not statistically significant between particles tested and control. **Cytokines:** Treatment with Montana ultrafine particles resulted in a significant production increase of TNF-α. MIP-2 showed increases in all the fine and ultrafine treatments, with Montana ultrafine and W. Kentucky fine PM showing the highest increases. IL-6 increased with Montana ultrafine particles although there was some variability and the increases were not statistically significant.
Reference: Gilmour et al. (2005, 087410) **Species:** Human **Cell/Tissue Types:** monocyte derived macrophages, HUVECs, A549 , 16HBE	PM_{10}: Collected from the Marylebone and Bloomsbury monitoring sites in London, UK **Particle Size:** PM_{10}	**Route:** Cell Culture **Dose/Concentration:** 50 μg/mL **Time to Analysis:** 4h, 6h, 20h	**IL-8**: PM_{10} at 50μg/mL induced a significant increase in IL-8mRNA and protein expression in PMM and 16HBE at 6 and 20h. A less substantial increase was also observed in A549. **Procoagulant Activity**: PM_{10} induced a significant decrease in macrophage mediated clotting time in 16HBE. Other cell types were unaffected. **Annexin V Binding**: At 100 μg/mL, PM_{10} induced a significant increase in binding macrophages at 4 and 20h. There was no effect at 50 μg/mL. **Tissue Factor mRNA Expression**: Expression was increased in macrophages at 6 h only. **tPA Expression**: mRNA expression decreased at 6h. Protein expression decreased at 4 h and 20 h in a dose-dependent manner. **TF Expression:** TF mRNA expression increased in a dose-dependent manner at 6 h in HUVECs. Protein levels also increased at 4 h but declined to basal levels by 20h.
Reference: Gilmour et al. (2003, 096959) **Species:** Human **Cell/Tissue Type:** A549	PM_{10}: Collected from the Marylebone and Bloomsbury monitoring sites in London, UK TSA H2O2 NAC Mannitol Provided by Sigma Chemical, Poole, UK or GIBCO-BRL, Paisley, UK **Particle Size:** PM_{10}	**Route:** Cell Culture **Dose/Concentration:** PM_{10}: 100 μg/mL; TSA: 100 ng/mL; H2O2: 200μM; NAC and Mannitol: 5 mM **Time to Analysis:** 24h	**IL-8:** PM_{10}, TSA and H2O2 treatment induced an increase of IL-8. Concomitant exposure of TSA with PM_{10} or H2O2 significantly increased IL-8 release when compared to PM_{10} or H2O2 alone. IL-8 mRNA expression with PM_{10} or H2O2 exposure and TSA coincubation caused significant increases. Silver staining of PCR products indicated that the IL-8 gene promoter was associated with acetylated H4 following TSA, PM_{10} and TNF treatment. **H4:** PM_{10} exposure significantly increased acetylation levels of H4 over controls. Increased acetylated H4 was mediated by PM_{10} in a dose-dependent manner. Treatment with PM_{10} and H2O2 increased HAT activity associated with H4 by 245% and 166% respectively. Significant increases in acetylation of H4 following treatment of cells with TSA, PM_{10} and H2O2 for 24 h was observed. PM_{10}induced HAT activity was significantly decreased in the presence of NAC and mannitol. Nuclear presence of HDAC2 protein was significantly reduced by exposure to both HDAC inhibitor and PM_{10}. There was a decreasing trend in HDAC2 gene expression following TSA and PM_{10} treatment. **NF-kB:** The activation of the transcription factor NF-kB was enhanced following the inhibition of HDAC with TSA and by treatment with

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Reference: Graff DW et al. (2007, 156488) **Species:** Human **Cell/Tissue Type:** HAEC	PM -UF: ultrafine -F: fine -C: coarse Particles collected from Seattle, WA (-S), Salt Lake City, UT (-SL), Phoenix, AZ (-P), South Bronx, NY (-SB), Hunter College, NY (-H), Sterling Forest, NY (-SF) **Particle Size:** UF: <0.1 μm; F: 0.1- 2.5 μm; C: 2.5-10 μm	**Route:** Cell Culture **Dose/Concentration:** 250 μg/mL **Time to Analysis:** 6h, 24h	**Gene Expression:** PM-UF, PM-F, and PM-C both upregulated and downregulated genes in the HAECs though downregulation was far more common for all the three PM fractions. PM-F affected the greatest number of transcripts, followed by the UF and C fractions. **IL-8:** mRNA expression increased, with PM-F-S having the greatest impact. Aluminum, strontium, manganese and potassium were highly associated with expression. Wood combustion was moderately associated. **HOX-1:** mRNA expression increased, with PM-F-SF having the greatest impact. Potassium, manganese, strontium and wood combustion were highly associated with expression. Aluminum and vanadium were moderately associated.
Reference: Gualtieri M et al. (2005, 097841) **Species:** Human **Cell Type:** A549	TD: Tire debris extracted in methanol, constituent of PM_{10} (generated by spinning a new automotive tire against abrasive surface) **Particle Size:** 10-80 μm	**Route:** Cell Culture **Dose/Concentration:** 10, 50, 60, 75 μg/mL **Time to Analysis:** 24, 48, 72h	**Cytotoxic Effect:** Treated cells presented inhibitory effect on reduction of MTT which appeared to be dose and time-dependent. A statistically significant reduction was observed at 48 and 72 h. Trypan blue showed a significant PM lethality as well as a dose-dependent increase in mortality. **DNA Damage:** At 24 and 72 h, DNA damage increase dose dependently in damaged and ghost cells. **Cell Cycle Analysis:** At 24 h, TD extract-treated cells presented a significant increase in the percentage of cells in G1 phase when compared with control. This increase was associated with a decrease in the percentage of cells in S phase. At 48 and 72 h, the increase in percentage of cells in G1 was associated with a decrease in the percentage of cells in both S and G2/M phases. Cells exposed to TD extracts presented changed morphology. Modifications most obvious at 72 h. The highest dost produced increased vacuolization in cytoplasm and apoptotic nuclear images.
Reference: Hetland et al. (2005, 087887) **Species:** Rat **Gender:** Male **Strain:** Crl/Wky **Cell Type:** AMs	PMC = Coarse PMF = Fine -A = Amsterdam -L = Lodz -R = Rome -O = Oslo Coexposures PAH, Fe, Al, Zn, Cu, V **Particle Size:** PMC: 2.5-10 μm; PMF: 0.2-2.5 μm	**Route:** Cell Culture ($1.5x10^6$ cells/well) **Dose/Concentration:** 50, 100 μg/mL PM **Time to Analysis:** 20h	**IL-6:** PMC from all cities exhibited increases in IL-6 release with spring and summer roughly equal and both inducing higher levels than the winter PMC. For the Spring and Summer samples, PMC-L exhibited the highest IL-6 releases (440% and 460% respectively) followed by Rome, A'dam/Oslo, and Oslo/A'dam. For the winter samples, Rome and Amsterdam induced higher IL-6 levels (340% and 300% respectively) than Lodz and Oslo (165% and 160%). The fine fractions did not induce any significant cytokine release. **TNF-α:** PMC from all cities increased TNF-α release with 50μg/mL generally inducing a slightly higher increase than 100 μg/mL. **Constituent Correlation:** Levels of Fe, Al, Zn, Cu and V as well as PAH (total and fractions) showed no correlation with IL-6 release. **Endotoxin Correlation with IL-6 release:** A confirmatory test revealed no correlation.

Study	Pollutant	Exposure	Effects
Reference: Hetland RB et al. (2004, 097535) **Species:** Rat, Human **Cell Type:** Alveolar Macrophages (Rat), A549 (Human) **Strain:** Wky/NHsd (Rat) **Gender:** Male (Rat) **Weight:** 180-230g (Rat)	AMC = Ambient Coarse AMF = Ambient Fine AMUF = Ambient Ultrafine (AM samples taken at a suburban site, without a dominating PM source, near Utrecht, Netherlands) Road PM: PM_{10}, (collected in a road tunnel with predominating road abrasion due to use of studded tires in Trondheim, Norway) **Particle Size:** AMC: 2.5-10 μm; AMUF: <0.1 μm	**Route:** Cell Culture (1 x 10^6 cells/well) **Dose/Concentration:** 0, 100, 200, 400, 600, 800, 1000 μg/mL **Time to Analysis:** 20h (Type 2 cells); 40h (A549 cells)	**IL-8:** All 3 AM fractions showed dose-dependent increases in A549 cells until 600 μg/mL; at that concentration, levels declined. AMC showed the most pronounced decline which correlates with decreased viability. Road PM showed a near linear response until 1000 μg/mL, whereas DEP plateaued at 600 μg/mL in A549. **MIP-2:** AMC and AMUF had no effect on Type 2 cells. DEP induced increases at 200 μg/mL, whereas Road PM induced the strongest increase, peaking at 600 μg/mL in Type 2 cells. **IL-6:** AMC induced increases at 100μg/mL in Type 2, but levels declined below normal at 200 μg/mL. AMUF induced a decline of IL-6 levels. Road PM induced significant increases in Type 2. DEP had a slight effect. AM fractions induced increases in A549 cells, peaking at 600 μg/mL with AMF. DEP and Road PM induced a dose-dependent increase. **Cell Survival:** AMC showed major effects at 200 μg/mL in Type 2. AMUF showed effects at 400 μg/mL. Road PM and DEP showed a gradual decline from 75% to 50% at 800 μg/mL in Type 2. All AM fractions induced a decrease in viability after 600μg/mL in A549 with AMC inducing a larger decrease than AMUF and AMF; AMUF and AMF induced similar levels. Road PM and DEP had no effect on A549. **Apoptosis:** AMC elicited a marked induction of apoptosis 200 μg/mL in Type 2 cells. AMF showed a dose-dependent increase in A549. Other AM fractions showed some slight increases in both cell types. Statistical significance was reached for all particles except for Road PM.
Reference: Holder AL et al. (2008, 093322) **Species:** Human **Cell Type:** 16HBE14o	DEP: generated from a single cylinder diesel engine using , commercial certified #2 diesel fuel Copollutants: NO_x 7 ppm, CO_2 0.1% **Particle Size:** Suspension: 223nm (mean diameter); ALI: 122nm (mean diameter)	**Route:** Suspension (1x10^5 cells/cm^2), Air Liquid Interface (ALI, 1x10^5 cells/cm^2) **Dose/Concentration:** Suspension: 0.13, 0.24, 1.88, 2.5, and 12.5 μg/cm^2; ALI: 1 g/cm^3 (total number of particles: 2.3 x 10^7 particles/cm^2) **Time to Analysis:** Exposure for 6h. Parameters measured 20h postexposure.	**ALI vs Tracheal Bronchial (TB) Deposition:** The TB region deposition is 1.5 nominally x ALI, but particle diameter deposited in the TB was 62 nm (geometric mean diameter) as compared to the particle deposition in the ALI, measuring 260 nm. **Inflammatory Response:** Suspended DEP decreased viability at concentrations of 2.5 μg/cm2or higher. IL-8 release (corrected for viability) increased at concentrations of 1.88 μg/cm^2 or higher in a dose-dependent manner. IL-8 exhibited intermediate levels of secretion between in vitro levels of 0.25 and 1.88 μg/cm^2. No statistically significant results were observed in ALI. Viability for ALI was near 100% (75% uncorrected).
Reference: Huang et al. (2003, 087376) **Species:** Human, Mouse **Cell Type:** BEAS-2B (human), RAW 264.7 (mouse)	PMC: PM coarse PMF: PM fine PMSM: PM submicron Collected between September-December 2000 from 4 ambient monitoring stations in Taiwan that represented background, urban, traffic, and industrial sites **Particle Size:** PMC: 2.5-10 μm; PMF: 1-2.5 μm; PMSM: <1 μm	**Route:** Cell Culture (5X10^5 cells/mL) **Dose/Concentration:** All PM: 50, 70, 100 μg/mL **Time to Analysis:** BEAS-2B: 8h; RAW 264.7: 16h	**Viability:** None of the PM fractions affected cell viability. **IL-8:** Only PMSM induced a significant IL-8 increase in BEAS-2B. IL-8 response was associated with a combination of Mn and Cr (R2 = 0.28). Response was also correlated with nitrate, although significance disappeared when 1 extreme nitrate value was removed. **Lipid Peroxidation:** Only PMSM enhanced lipid peroxidation in BEAS-2B, correlating with both elemental and organic carbon. **TNF-α:** In RAW264.7, PMSM increased TNF production. Polymixin pretreatment significantly reduced TNF levels for all 3 PMs which indicates an endotoxin role in macrophage response. TNF production (after polymixin pretreatment only) was associated with Cr and Fe content.

Study	Pollutant	Exposure	Effects
Reference: Hutchison et al. (2005, 097750) **Species:** Mouse **Cell Line:** J774.1A	PM_{10}: Samples collected for 7d during closure (-C) and reopening of steel plant (-R) PMT: PM total (aqueous sonicate) PMS: PM soluble aqueous PMI: PM insoluble aqueous **Particle Size:** PM_{10}	**Route:** Suspension **Dose/Concentration:** 500 μl (estimated concentrations of 112, 143, 156, 180, 233, 255 μg/1ml water) **Time to Analysis:** 4h	**Particle Characterization:** Reopening of the plant showed a significant increase in the total and acid extractable metal content of PM. Aqueous extractable metal content did not change. Soluble zinc, copper and manganese also increased significantly post reopening. Iron was the most abundant in acid extractable metals and increased greatly at the reopening. **TNF-α:** PMT-R and PMT-C induced a statistically significant increase. Treatment with chelation agent reduced effect to control levels.
Reference: Imrich A et al. (2007, 155859) **Species:** Rat **Gender:** Female **Age:** 12-14wks **Cell Type:** AM	UAP: SRM 1649 (positive control) TiO_2: Particle control CAPs (Boston, MA) All cells primed with LPS Coexposure with NAC, dimethylthiourea (DMTU), H2O2 or catalase **Particle Size:** CAPs: ¤2.5 μm; UAP: $PM_{2.5}$; TiO_2: ~1 μm	**Route:** Cell Culture (2 $X10^5$ cells/well) **Dose/Concentration:** Caps 100μg/mL; UAP: 50 or 100 μg/mL; LPS 250 ng/mL; NAC, DMTU: 2, 10, 20mM; Catalase: 1, 5, 10mM; H2O2 0-50 μm/hr **Time to Analysis:** 18-20h	**TNF:** DMTU at 20mM reduced TNF in LPS-primed cells in control and UAP-treated groups. NAC at 20mM reduced TNF release but this was not statistically significant. Catalase significantly inhibited TNF in control and UAP-treated groups. CAPs (especially the insoluble portion) significantly increased TNF unless co-exposed with NAC, DMTU or catalase. All three reduced levels back to around basal levels. DMTU was particularly effective at diminishing TNF release. H2O2 increased TNF release in CAPs-exposed cells. TiO_2 had no increased ability to induced cytokine release when mixed with H2O2. **Cell Death:** Viability decreased substantially when exposed to H2O2 + CAPs. The soluble fraction of CAPs showed to be more effective with H2O2 than the insoluble portion. TiO_2 had no significant effect. **NO:** Some CAPs induced slight increases when mixed with H2O2. No difference was observed between soluble and insoluble portions of CAPs. **DFO:** DFO at 0.05mM completely inhibited oxidation induced with soluble CAPs + H2O2. Insoluble CAPs + H2O2 was also DFO-sensitive. DFO was ineffective against the insoluble CAPs induction of TNF and MIP-2.
Reference: Ishii et al. (2004, 088103) **Species:** Human **Cell Type:** A549 (collected from 6 lobectomy or pneumonectomy smokers), HBEC	EHC-93:PM_{10} (obtained from Environmental Health Directorate, Ottawa, Ontario, Canada) **Particle Size:** PM_{10}	**Route:** Cell Culture (1x10E7 cells) **Dose/Concentration:** 100 μg/mL **Time to Analysis:** 3, 6, 24h	**Cytokines:** TNF-α, IL-1β, GM-CSF, IL-6, and IL-8 levels were significantly increased in A549 cells. **mRNA Expression:** MCP-1, ICAM-1 and IL-8 mRNA expression increased in untreated AM supernatants at 3h. Only the MCP-1 levels were statistically significant at 3h. Levels declined by 6h. When A549 cells were exposed to PM_{10} exposed AM, levels of RANTES, TNF-α, ICAM-1, IL-1β, and LIF increased. Except for RANTES mRNA, these differences were less in the 6h samples. VEGF increased as well, but this increase was not statistically significant. **TNF-α and IL-1β-neutralizing Antibodies:** IL-1β antibody alone or in combination with TNF-α significantly reduced expression of all eight mRNAs. Combinations for some mRNAs reduced expression by up to 1/2. This effect was not observed when A549 was treated with the control AM. **Transcription Factor Binding Activity:** Binding of AP-1 and Sp1 increased when A549 treated with supernatants from PM_{10}-exposed AM, but not from control AM.

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Reference: Ishii H et al. (2005, 096138) **Species:** Human **Cell Type:** AMs (obtained from 10 smokers who stopped smoking 6wks prior), HBEC	EHC-93: PM_{10} (obtained from Environmental Health Directorate, Ontario, Canada) **Particle Size:** PM_{10}	**Route:** Cell Culture (HBEC: 2.5-3.0x10^6 cells; AM: 1x10^7 cells; co-culture of AM/HBEC: 5x10^6 cells) **Dose/Concentration:** 100 μg/mL **Time to Analysis:** 2, 24h	**mRNA Expression after 2h exposure:** AM or HBEC exhibited no effect. In contrast, co-culture increased expression of MIP-1β, GM-CSF, M-CSF, IL-6, MCP-1 and ICAM-1-mRNA. **mRNA Expression after 24 h exposure:** AMs exhibited no effect. HBEC increased levels of GM-CSf, LIF and ICAM-1. Co-culture, on the other hand, increased expression of MIP-1β, GM-CSF, M-CSF and ICAM-1 mRNA. **Protein Levels:** AM and HBEC both increased GM-CSF, IL-6 and MIP-1β release into the supernatant. Co-culture effect was not additive but synergistic (i.e., higher than expected). MCP-1 levels did not increase significantly. Co-culture appeared to decrease protein levels for both the control and PM values. M-CSF levels increased for co-culture only. **Surface Expression of ICAM-1:** Upon 24h exposure to PM, HBEC exhibited an increase in expression. Expression in AMs were not affected by 2h PM stimulation. **ICAM-1 Inhibitors:** IgG or anti-CD11b antibody was unaffected in co-culture.
Reference: Jalava P et al. (2005, 088648) **Species:** Mouse **Cell Type:** RAW 264.7	UPM: SRM1649a (Washington, DC) DEP: SRM1650 (NIST) EHC-93: Ottawa dust (Environmental Health Center, Ottawa, Canada) HFP-00: Pooled ambient air $PM_{2.5}$ sample from Helsinki, Finland M-UPM: methanol extract of UPM **Particle Size:** SRM 1649a, SRM 1650, EHC-93: NR; HFP-00: $PM_{2.5}$	**Route:** Cell Culture (5 X10^5 cells/mL) **Dose/Concentration:** 150 μg/mL **Time to Analysis:** Methanol treatment of PM samples: 24h; Exposure to ambient PM samples: 2, 4, 8, 16, or 24h.	**TNF-α:** All the PM samples increased TNF-α. **Cell Viability:** SRM1649a exhibited the most cytotoxicity, followed by HFP-00 and EHC-93. Methanol significantly affected cytotoxicity of the EHC-93 sample only. **Cytokines:** TNF-α concentrations in the cell culture medium significantly increased at all time points between 2 and 24 h. The highest increase was seen in EHC-93. IL-6 production also increased at different levels with the highest increase observed in EHC-93. No response was observed for IL-10. **Cell Viability:** Duration of exposures had no significant effect on any of the samples. A 2h exposure time was sufficient to induce the typical reductions in cell viability.
Reference: Jalava PI et al. (2006, 155872) **Species:** Mouse **Cell Type:** RAW 264.7	PM: Collected east of Helsinki, Finland between Aug 23 and Sept 23, 2002 Divided in 12 groups (4 sizes by 3 exposure types): -S: seasonal average -W: wildfire -M: mixed -B: blank **Particle Size:** PM_{10}-2.5; $PM_{2.5-1}$; PM1-0.2; PM0.2	**Route:** Cell Culture (5x10^6 cells/mL) **Dose/Concentration:** 15, 50, 150 and 300 μg/mL **Time to Analysis:** 24h	**Particulate Mass Concentrations in HVCL Size Ranges:** The largest increase of PM concentrations was observed in PM1-0.2. **NO:** All 12 samples increased NO production when compared to corresponding unexposed controls. Peaks were observed at 150 μg/mL, except in PM1-0.2. **Cytokines:** All 12 samples increased TNF-α and IL-6 production. PM_{10}-2.5 and $PM_{2.5}$-1 produced a much larger response than PM1-0.2 and PM0.2. IL-6 production for PM0.2 was not measured. MIP-2 production also increased with similar trends. **Cytotoxicity:** All 12 samples induced dose-dependent decreases in cell viability. PM_{10}-2.5 were the least active inducers of apoptosis while PM0.2 showed the highest activity (4-17% of apoptotic cells).

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Reference: Jalava et al. (2007, 096950) **Species:** Mouse **Cell Type:** RAW 264.7	Urban background PM PM_{10}, $PM_{2.5}$, and PM0.2 collected from 6 European cities during different times of the year from October 2002 to July 2003: -D: Duisburg (Fall) -P: Prague (Winter) -A: Amsterdam (Winter) -HR: Helsinki (spring), -B: Barcelona (spring) -AT: Athens (summer) **Particle Size:** PM_{10}: 2.5-10 μm; $PM_{2.5}$: 0.2-2.5 μm; PM0.2: < 0.2 μm	**Route:** Cell Culture (5 $X10^5$ cells/mL) **Dose/Concentration:** 15, 50, 150, 300 μg/mL **Time to Analysis:** 24h	**PM Characterizations:** The highest mass concentrations of PM_{10} and PM0.2 were measured in Athens. Prague had the highest $PM_{2.5}$ concentrations. **NO:** All PM fractions induced statistically significant NO production in macrophages. $PM_{2.5}$ -P and $PM_{2.5}$ -AT produced significantly larger responses, though all samples at 150 and 200 μg/mL induced statistically significant production. When compared to the other PM0.2 samples, -P and -HR produced significantly larger responses. **Cytokines:** PM_{10} showed average cytokine production to be 7.8 fold and 83 fold for TNF-α, and 4.4 fold and 530 fold for MIP-2 when compared to $PM_{2.5}$ and PM0.2 respectively. PM_{10} induced statistically significant increases in production of TNF-α, MIP-2 and IL-6. $PM_{2.5}$, with exception of Prague, caused significant increases in cytokines. PM0.2-A and -AT showed small yet statistically significant increases in TNF-α. An increase in MIP-2 was observed with -P and -HR. IL-6 increased significantly with PM_{10} and slightly with $PM_{2.5}$. In the PM0.2 range, only the –A and –AT samples caused a small, statistically significant TNF- α production. MIP-2 production was only detected from the –P and –HR samples. PM0.2 effects on IL-6 response were negligible. **Cytotoxicity:** The average cytotoxicity of PM_{10} and $PM_{2.5}$ were roughly equal, but PM0.2 were less cytotoxic with the exception of –P. The dose-response trends for most of the samples were linearly declining, with PM_{10} and $PM_{2.5}$ exhibiting statistically significant declines in viability.
Reference: Jimenez et al. (2002, 156610) **Species:** Human **Cell Type/Line:** A549, THP-1, Mono Mac 6 (DSMZ)	PM_{10}: Collected from London and Edinburgh air particulate monitoring stations. TiO_2 UFTiO2 Both fine and ultra fine TiO_2 fractions obtained from Tioxide Europe (London, UK) and Degussa-Huls (Cheshire, UK) **Particle Size:** PM_{10}, TiO_2: 200nm; UFTiO2: 20nm	**Route:** Cell Culture (110,625 cells/well) **Dose/Concentration:** PM_{10}, TiO_2, UFTiO2: 100 μg/mL; TNF-α: 10ng/mL **Time to Analysis:** 4h	**NF-kB and AP-1 DNA Binding**: NF-kB DNA binding increased in PM_{10} and TNF-α exposed macrophages by 9.5 and 12 fold. NF-kB activity remained unaltered in TiO_2 and UFTiO2 exposed macrophages. **IL-8:** Cells treated with PM_{10} conditioned media increased transcription binding of NF-kB to IL-8 promoter sites. Increases were observed in gene expression after exposure to TNF-α and PM_{10}. TiO_2 or UFTiO2 had no effect. Increases observed in IL-8 production with PM_{10}. **IL-8 Promoter CAT Activity:** PM_{10} media increased CAT expression by 65% over control. No differences observed with TiO_2 or UFTiO2 media. **Neutrophil Chemotaxis:** PM_{10} conditioned media induced a 2.3 fold increase compared to control. **TNF-α and IL-1β Production:** PM_{10} media increased TNF-α and IL-1β production. No increases were observed in TiO_2 and UFTiO2 media.

Study	Pollutant	Exposure	Effects
Reference: Jung et al. (2006, 132421) **Species:** None **Type:** Surrogate Lung Fluid	Soot Particles: Generated using a co-flow, laminar, diffusion flame system CB (Degussa) $PM_{2.5}$: Collected using IMPROVE air pollution samplers **Particle Size:** Soot: 185nm; CB: 25nm, $PM_{2.5}$	**Route:** Surrogate Lung Fluid **Dose/Concentration:** Soot: 0-30mg; CB: 5-10mg; $PM_{2.5}$: 50 or 100 μg/mL **Time to Analysis:** Parameters measured continuously over 2h.	**OH Radical Formation:** Formation occurred with linear dependence on soot mass. Average response was 0.89nmol OH produced per mg of soot. Formation also occurred with soot + hydrogen peroxide. Hydrogen peroxide alone did not form OH radicals. **Fe:** Average Fe concentration in soot particles was 305 ± 172nM. Observed negative correlation between amount of Fe and amount of OH radical formation. DSF inhibited iron-induced increase in OH radical formation. **Carbon Black:** OH radical generation by carbon black was significantly less than soot. OH generation by CB was observed to be linearly proportional to PM mass, but CB was much less efficient at generating the OH radical. **$PM_{2.5}$:** A high variability in the increase of OH radicals was observed with $PM_{2.5}$. Pretreatment with DSF partially blocked OH radical production, but a significant level remained. This may be due to $PM_{2.5}$ containing high levels of Fe and Cu.
Reference: Kafoury and Madden (2005, 156617) **Species:** Mouse **Cell Type:** RAW 264.7	DEP: SRM 1975 (purchased from NIST, Rockville, MD) BAY11-7082, NF-kB inhibitor (coexposure) IL-1ß: obtained from Santa Cruz Biotechnology (Santa Cruz, CA) **Particle Size:** DEP: 0.3 μm (mean diameter)	**Route:** Cell Culture ($3\text{-}4x10^5$ cells) **Dose/Concentration:** DEP 25, 100, or 250 μg/mL; IL-1ß: 100 ng/mL **Time to Analysis:** DEP: 4h pre-treated with BAY11-7082 for 1.5h; IL-1ß: 4h	**TNF-α:** DEP induced a significant release of TNF-a at 100 and 250 μg/mL dose-dependently. Exposure at 25 μg/mL had no effect. IL-1β containing PM samples at 100 μg/mL also resulted in a significant release of TNF-α. **NF-kB Binding Activity:** Treatment of RAW 264.7 with BAY11-7082 significantly inhibited IL-1β-induced TNF-α release. Similar effects observed with DEP-induced TNF-α release. **Apoptosis:** Inhibition of NF-kB binding activity by BAY11-7082 resulted in DEP-induced apoptotic response. Without BAY11-7082, apoptosis was not induced even at the DEP dose of 250/μg/mL for 4h. The control, U937 cells with campothecin, induced apoptosis.
Reference: Karlsson et al. (2006, 156625) **Species:** Human **Cell Type:** A549, Monocytes (isolated from heparinized whole blood)	PM (W1: wood burning in old-type boiler; W2: wood burning in modern boiler; P: wood pellets burning in pellets burner; T1: PM_{10} tire debris with studded tires and ABT pavement; T2a: PM_{10} tire debris with studded tires and ABS pavement; T2b: $PM_{2.5}$ tire debris with studded tires and ABS pavement; T3: PM_{10} tire debris with friction tires and ABS pavement; St: PM_{10} from busy street in Stockholm, Sweden; Su: PM_{10} from platform of subway station in Stockholm) **Particle Size:** W: NR, T1, T2a, T3, St, Su: PM_{10}, T2b: $PM_{2.5}$	**Route:** Suspension (A549) and Cell Culture (Monocytes) **Dose/Concentration:** Suspension: 40 $\mu g/cm^2$; Culture: 100 $\mu g/cm^2$ (1 ml/well) **Time to Analysis:** Suspension: 4h; Culture: 18h	**PM Characterization:** Boiler emitting PM-W1 led to 4 times higher emission of particles when compared to PM-W2 and 8 times higher emissions when compared to PM-P. Total organic carbon concentration and CO was substantially higher in the old-type wood boiler. **Effects with Filter Fibers:** No increase of DNA damage was observed compared to the water control. Filter fibers led to the induction of cytokines in human macrophages. **Genotoxicity:** All particulate samples induced DNA damage in A549 cells. PM-Su exhibited the most genotoxicity and induced 4-5 times more DNA damage than others. **Cytokines on Glass Fiber Filters:** PM-W2 induced a significant increase in IL-8. PM-St induced the highest increases of IL-6, IL-8, and TNF-α. **Cytokines on Teflon Filters:** PM-2a and PM-2b samples caused significant increases of IL-6, IL-8, and TNF-α.

Study	Pollutant	Exposure	Effects
Reference: Katterman ME et al. (2007, 096358) **Species:** Rat **Cell Type/Line:** RLE-6TN (Alveolar Epithelial Cell Line)	PM: Oils: OAAF, Oil Q, OII I II, NF2 PM: Coal Germany and Ohio Diesel particulates: ZODDA (doped with Zn), ZSDDA (doped with Zn and S): S: PMs washed in solution; F: Fresh samples;L: Leached Al_2O3, Fe_2O3, SiO_2, TiO_2,ZnO also tested **Particle Size:** NR	**Route:** Cell Culture (cytotoxicity: 50,000 cells/well; SEM: 25,000 cells) **Dose/Concentration:** Oils 0.2mg/mL; Coals 0.7mg/mL; Diesel 0.01mg/mL; Al203 0.5mg/mL; Fe_2O3 0.7mg/mL; SiO_2 0.7mg/mL; TiO_2 0.7mg/mL; ZnO 0.05mg/mL **Time to Analysis:** 24h	**Metabolic Activity:** For oils comprised of 3/4 fresh and 1/4 leached, metabolism decreased. Coals (fresh and leached) had no effect. ZODDA-F and ZSDDA-F both induced decreases in activity. ZSDDA-L had no effect. **Cellular Morphology:** PM-S had a minimal effect. PM-F induced widespread cell damage. **Constituent Differences between PM-F and PM-L:** In oil samples Cu, Ti and Ca salts were removed upon washing. Fe, Al, Si remained constant. **Grinding Effects:** Coal toxicity increased upon grinding, whereas diesel PM toxicity decreased upon grinding. **Metal Oxide Effects:** Only SiO_2, and ZnO (much higher at lower concentrations than other metal oxides) decreased metabolic activity. Fresh, washed and sonicated samples exhibited similar results. Grinding only affected TiO_2 (increase) and ZnO (decrease).
Reference: Kendall et al. (2004, 156634) **Species:** Human **Tissue Type:** BALF (obtained by bronchoscope from 6 nonsmokers and 3 smokers)	$PM_{2.5}$ sample sites; 2 schools in Bronx, NY, 6 background urban, 6 urban roadside. Sampling occurred 24h/day for 12d. Particle Surface Chemistry: 79-87% carbonaceous material (Ch, COO, C-(O,N)), 10-17% O (O1s), 1.5-4% N (NH_4+, N-C, NO_3 2-), 0.6-1% S, and 0.3-2 % Si. Only NO_3 - higher in roadside samples. NH_4 and NO_3 - correlated with NO and NO_X in air but not NO_2. **Particle Size:** $PM_{2.5}$	**Route:** BALF treatment **Dose/Concentration:** 5-10ml of 0.5M NaCl or BALF **Time to Analysis:** Filters treated with BALF for 4h	**Saline Washing:** Removed particles and decreased NH_4, NO_3,O and S relative to C1. **BALF treatment (XPS):** $PM_{2.5}$ surfaces interacted strongly with BALF within hours of contact. Specific surface components of $PM_{2.5}$ immersed in BALF were desorbed while biomolecules from BALF were adsorbed to particles. N-C on the PM surface increased 3 fold for smokers and 4 fold for nonsmokers (range 1.4-7.4). This is most likely related to protein-like adsorption on PM. Treatment also induced a slight increase in COO and decreases in NH_4, NO_3, O and S. **ToF-SIMS - Organics:** Particle loading and surface hydrocarbons showed a linear correlation. Loss of hydrocarbons from $PM_{2.5}$ surface averaged 55% (10-75) after undergoing saline and BALF washes. In only 3/12 samples BALF removed less hydrocarbon. BALF treatment increased the amino acid and phospholipid content of the $PM_{2.5}$ surface. **ToF-SIMS - Inorganic:** Saline washing appeared to increase Al and Si but with extreme variability; this increase was not statistically significant. Both saline and BALF washing decreased NH_4 and Na levels to a similar extent. BALF washing did not affect Al or Si.
Reference: Kim et al. (2005, 088454) **Species:** Human **Cell Type/Line:** BEAS-2B	Zn2+ **Particle Size:** NA	**Route:** Cell Culture **Dose/Concentration:** 15, 50, 100 μmol **Time to Analysis:** 1-20h	**Cell Viability:** At 50μM for 20h, no apoptosis was induced. **IL-8:** At 12h, IL-8 increased in dose-dependent manner. At 15 or 50μM, Zn2+ increased protein 1.6 and 4.6 fold respectively. IL-8 mRNA expression increased dose-dependently, reaching statistical significance at 2 h and continuing until 4h. **EGFP (adenoviral IL-8 promoter):** Levels increased 2.4 fold with 50μM Zn2+. **Proteases:** With 50μM Zn2+, phosphorylation of MAPKs ERK, JNK and p38 increased by 15min and continued increasing up to 2h. Pre-exposure of inhibitors of MEK, JNK, before Zn2+ exposure caused inhibition of Zn-induced IL-8 mRNA and protein production. Inhibitor of p38 had no effect. Dephosphorylation of ERK and JNK was partially inhibited with exposure to Zn2+.

Study	Pollutant	Exposure	Effects
Reference: Kleinman et al. (2003, 087938) **Species:** Rat **Strain:** Wistar, Fischer 344 **Age:** 22-24m, 10wks **Cell Type/Line:** AMs	UF1: Utrecht 1 Fine (urban freeway) UC1: Utrecht 1 Coarse UF2: Utrecht 2 Fine (urban, freeway, light industrial) UC2: Utrecht 2 Coarse SRM 1650 SRM 1648 **Particle Size:** UF1: 0.2-2.5 μm; UC1: 2.5-10 μm; UF2: 0.2-2.5 μm; UC2: 2.5-10 μm	**Route:** Cell Culture (10^5 cells/well at 10^6 cells/mL) **Dose/Concentration:** 1.2 to 1200 ng/10^6 cells **Time to Analysis:** 4, 18h	**Macrophage PMA-stimulated respiratory burst activity:** SRM 1648 and 1650 induced dose-dependent decreases approaching 0 at 50 - 100 μg/10^5 cells. Large dose-dependent decreases from old rat AMs exposed to fine PM exposure were followed by young rat AMs exposed to fine PM. However, no age-related effects were statistically significant. **Free radical production:** All coarse particles depressed free radical production in a semi-dose-dependent manner, with UC2 exhibiting more potency than UC1. Both fine particles also showed dose-dependent responses but UF1 and UF2 responses were greater than the control at 3 μg/10^6 cells. **PM Characterization:** Ratios between coarse and fine PM were similar for metals tested (Al, Fe, Mn, Zn). Al was higher in coarse samples and Zn higher in fine PM, although large variability was observed. Fe and Mn results were roughly equivalent for all samples.
Reference: Kocbach, A. Namork, E. Schwarze, P.E. 2008 **Species:** Human **Cell Type/Line:** THP-1	PMW: Wood smoke particles Collected from conventional Norwegian wood stove burning birch PMT+: Traffic-derived particles Collected from road tunnel in winter when studded tires were used PMT-: Traffic-derived particles Collected from road tunnel in summer without studded tires DEP: SRM2975 Porphyr: fine grain syenite porphyry (prepared by SINTEF, Trondheim, Norway) Polymyxin B Sulphate (endotoxin inhibitor) **Particle Size:** PMW, PMT, DEP: NR; Porphyr 8 μm (mean)	**Route:** Cell Culture (1,000,000 cells/mL) **Dose/Concentration:** 30-280 μg/mL **Time to Analysis:** 2, 5, 12h	**Particle Characterization:** PMT+ contained a high mineral particle content. PMT- contained carbon aggregates, organic carbon and polycyclic aromatic hydrocarbons (PAH). PMW and DEP contained carbon aggregates. PAH content of PMW was greater than DEP. Porphyr was not included in the analysis. **Cytokines:** PMT± induced releases of TNF-α, IL-1β, and IL-8 with 30 or 70 μg/mL. PMW similarly induced TNF-α and IL-8. DEP induced IL-1β and IL-8. Porphyr induced IL-8 increases. IL-4, IL-6 and IL-10 were unaffected. Overall, the order of effective cytokine induction from most to least effective was PMT±, PMW, DEP, and Porphyr. mRNA expression of TNF-α, IL-1β, IL-8, and IL-10 increased with 140 μg/mL of PMT± and slightly for PMW. **LDH:** PMT ± induced small but statistically significant increases at low doses. DEP increased LDH at 280 μg/mL only. **Polymyxin B Sulphate:** The endotoxin inhibitor significantly inhibited LPS-induced cytokine release by 80-90% and reduced PMT± induction by 50-60%. **Organic Extraction:** PMT+ washed and native particles showed equivocal induction of cytokine release. PMT+ organic extract had no effect. PMT- and PMW organic extracts significantly increased TNF-α and IL-8. Washed particles induced less signficant increases of IL-8. DEP organic extract had no effect.
Reference: Kristovich R et al. (2004, 087963) **Species:** Human **Cell Type/Line:** HUVEC, HPAEC, HPMVEC, HPBMC	CP: carbon particle (carbonaceous negative image of zeolite) CFE: C/Fe particulate (synthesized) CFE+: C-Fe/F-Al-Si particulate (synthesized) CFA: Coal Fly Ash (Coal-fired power plant, NOS) DEP: (exhaust pipe of diesel powered truck) CP, CFE, CFE+ approx 1 μm (resembling zeolite) Particle Characterization (Surface chemistry): CP = 88% C, 1% Si, 10% O, 1% N. CFE = 80% C, 2% Fe, 2% Si, 16% O. CFE+ = 20% C, 6% Al, 3% Si, 50% F, 6% O, 11% N, 4% Na. CFA = 25% C, 3% Fe, 13% Al, 17% Si, 41% O, 1 % N. DEP = 70% C, 3% Fe, 24% O, 1% N, 2% S. **Particle Size:** CP, CFE, CFE+: approximately 1 μm (resembling zeolite); CFA: <2 μm; DEP: 150nm	**Route:** Cell Culture (4x10E6 cells/well) **Dose/Concentration:** CP: 5-50 μg/cm^2; CFE: 2.5-25 μg/cm^2; CFE+: 2.5-25 μg/cm^2; CFA: 10-100 μg/cm^2; DEP: 2.5-25 μg/cm^2 **Time to Analysis:** 4, 8, or 24h	**Cytotoxicity:** CP exhibited no effects. DEP and CFE exhibited intermediate toxicities in the range of 50-70 μg/cm^2. No toxicity was apparent when treated with CFA (up to 200 μg/cm^2) or synthesized C particulates. **Endothelial Activation:** ICAM-1, VCAM-1, and E-selectin were activated dose-dependently by DEP, CFE, and CFE+. No effects observed for CFA or CP. These effects were not the result of endotoxin release. **Individual Variability:** Donors (humans) showed variability in responses especially for CFA. 3/9 had a medium response negated by ND responses in 6/9.

Study	Pollutant	Exposure	Effects
Reference: Kubatova, A. Dronen, L.C. Picklo, M.J. 2006 **Species:** Rat, Human **Cell Type/Line:** RAW 264.7 (rat), BEAS-2B (human)	PMW: Wood Smoke Collected from airtight wood stove burning hardwoods -P: Polar (fraction extracted from 25-50 C) -MP: Mid Polar (fraction extracted from 100-150 C) -NP: Nonpolar (fraction extracted from 200-300 C) -C: P + MP + NP **Particle Size:** NR	**Route:** Cell Culture (RAW 264.7: 10^6 cells/mL; BEAS-2B: 10^5 cells/mL) **Dose/Concentration:** 50, 100, 200 μg/mL **Time to Analysis:** 12h	**GSH:** PMW-MP and PMW-NP induced GSH depletion substantially in a dose dependent manner starting at 50 μg/mL in both cell types. DMSO had no effect. **Cytotoxicity:** PMW-MP and PMW-NP increased cytotoxicity at 200 μg/mL in RAW 264.7. BEAS-2B was unaffected. **Particle Characterization:** PMW-MP contained higher concentrations of oxy-PAHs, disyringyls, syringylguaiacyls and PAHs. oxy-PAHs include 9-fluorenone, 1-phenalenone, 9,10-anthraquinone and hydroxycadalene. PAHs included phenanthrene, fluoranthene and pyrene. **Effects of Individual Components of PMW-MP on GSH:** 1,8-dihydroxy-9-10anthraquinone and 9,10-phenanthraquinone depleted GSH. 9,10-anthraquione, anthrone, 1-hydroxypyrene increased GSH. Phenanthrene, 1-methylpyrene, 9-fluorenone and xanthone had no effect.
Reference: Kubatova et al. (2004, 087986) **Species:** Monkey **Cell Type/Line:** African green monkey kidney cells designated COS-1 (CV-1 cells with origin -defective mutants of SV40), E coli PQ 37 (SOS Chromotest)	DEP: Obtained from diesel bus PMW: Wood smoke particulates obtained from airtight wood stove burning hardwood HSF: Hot pressure fractionation -C: P + MP + NP -P: Polar -MP: Mid Polar -NP: Nonpolar OE: Organic Extraction -HNP: n-hexane nonpolar -MEP: methanol polar **Particle Size:** NR	**Route:** Cell Culture (10,000 cells/180 ul) **Dose/Concentration:** 0, 50, 100, 150, 200, 250, 300 μg/mL **Time to Analysis:** Cytotoxicity: 24h; Chomotest: 2h SOS	**Cytoxicity:** PMW induced cytotoxicity in a dose-dependent manner. PMW-HNP induced low cytotoxicity, followed by PMW-C (intermediate) and PMW-MEP (highest). Levels above 25 μg/mL were cytotoxic. DEP-HNP induced cytotoxicity but was not dose-dependent. Results similar for all 3 fractions (highly variable). All fractions with concentrations higher than 100 μg/mL were cytotoxic. **Extraction Water Temperature Effect:** PMW was cytotoxic at temperatures over 50 C. DEP was cytotoxic at temperatures higher than 200□ C. At 250□, cytotoxicity between DEP and PMW was similar. At 300□ C, PMW cytotoxicity declined and DEP stayed high, resulting in DEP inducing higher cytotoxicity than PMW. **SOS Chromotest:** β-Galactosidase formation increased, peaked at 200□ C with DEP and declined to control at 300□ C. Individual fractions showed linear dose response from 25-200 μg/mL with 150□ C and 200□ C extracts significantly higher.
Reference: Lee et al. (2005, 156682) **Species:** Human **Cell Type/Line:** A549	MEP: Motorcycle Exhaust Particles (Yamaha Cabin engine, 95 octane unleaded gasoline,150 rpm) MEPE: MEP Particle Free MEP 0.5 μm MEPE $<$ 0.2 μm **Particle Size:** MEP 0.5um; MEPE $<$ 0.2 μm	**Route:** Cell Culture ($1x10^5$ cells/well) **Dose/Concentration:** MEP 0.02, 0.2, 0.2, 2, 20 μg/mL; MEPE 20 μg/mL **Time to Analysis:** 24h	**IL-8:** MEP induced IL-8 at concentrations greater than 0.2 μg/mL. Levels increased 2fold at 24 h with 20 μg/mL. MEPE induced similar responses at 20 μg/mL. Induction of IL-8 mRNA expression was dose-dependent with MEP and MEPE. **Cytotoxicity:** Exposure to particles did not affect cytotoxicity. **NFκB:** MEP (20 μg/l) induced time-dependent activation for 2h and continued at same level for up to 6h. Pretreatment of PDTC (1mM) fully inhibited MEP induction. **MAP Kinase:** MEP induced time-dependent activation up to 30 min and stayed elevated for at least 60 min. **ROI:** MEP treatment induced a time-dependent increase in ROI for up to 1 h and then continued the at same level for up to 6h.

Study	Pollutant	Exposure	Effects
Reference: Lee, C.C. Kang, J.J. 2002 **Species:** Mouse **Strain:** Peritnoeal Macrophages, RAW 264.7 **Species:** Rat **Strain:** AMs	MEP Yamaha 2-stroke engine using unleaded gas) MEPE(particle-free MEP) **Particle Size:** 0.5 μm	**Route:** Cell Culture (5 X10^5 cells/mL (Cytotoxicity), 3 X10^5 cells/mL (Apoptosis), 2 X10^6 cells (MMP and ROI), 1 X10^7 cells (GSH) **Dose/Concentraion:** 5, 10, 50, 100, 300, 1000 μg/mL **Time to Analysis:** 6, 12, 18, 24h	**Cytotoxicity:** Viability decreased dose and time-dependently in all cell types at 24h. **Apoptosis:** subG1 significantly and dose-dependently increased at the 300 MEP μg/mL dose in all cell types, indicating increased apoptosis. MEPE induced similar results. Inhibition was successful against MEP-induced apoptosis by calcium chelators EGTA, BAPTA-AM, cyclosporin A and antioxidants NAC, GSH, catalase and SOD. **Ca^{2+}:** MEP and MEPE increased Ca^{2+} at 300 μg/mL. BAPTA-AM completely inhibited induction. **ROI:** MEP increased ROI in a time-dependent manner. Calcium chelators and antioxidants substantially attenuated induction. **GSH:** MEP significantly decreased GSH. **MMP:** Mitochondria membrane potential decreased dose-dependently with MEP 100 μg/mL and 300 μg/mL. Calcium chelators and antioxidants partially inhibited reduction.
Reference: Li et al. (2002, 042080) **Species:** Mouse **Cell Line:** RAW 264.7, THP-1 **Species:** Murine **Cell Line:** RAW 264.7	VACES (Biosampler PM_{10} in Downey, CA -DEP concentrate in water) DEPM (DEP methanol extract) DEPME (DEP methylene chloride extracts) DEPAL (DEPME aliphatic (hexane)) DEPAR (DEPME aromatic (hexane/methlene chloride)) DEPPO (DEPME polar (methylene chloride/methanol)) **Particle Size:** NR	**Route:** Cell Culture (2 X10^6 cells/well Mouse RAW 264.7 and THP-1; 0.67 X10^6 cells/well Murine RAW 264.7) **Dose/Concentration:** 10 - 200 μg/mL JNK Activation and IL-8 Production: THP-1 cells- 0, 10, 25, 50, 100 μg/mL DEPM; THP-1 cells- 0, 10, 25, 50, 100 μg/mL of DEP; RAW264.7 cells- 10 -100 DEP μg/mL Cytotoxicity: 1, 10, 25 (THP-1 cells only), 50, 100, 200 μg/mL GHS/GSSG: 0, 10, 25, 50, 100 μg/mL HO-1 Expression: 0, 25, 50, 100, 200 μg/mL **Time to Analysis:** GHS/GSSG: DEPM, whole DEP (RAW 264.7only) 8h. HO-1, MnSOD Expression: RAW 264.7, THP-1 7h. RAW 264.7 cells exposed to whole DEP 16h. JNK Activation, IL-8 Production: THP-1 cells 30min, 16h. RAW 264.7 cells 90min. Cytotoxicity: RAW264.7, THP-1 18h.	**GSH/GSSG Ratio:** DEPM induced dose-dependent decrease in GSH/GSSG ratios in both cell lines. DEP induced decreases at comparable doses to DEPM. **HO-1 Expression:** Cells exhibited dose-dependent increases in HO-1 expression. **HO-1 Expression in Murine RAW 264.7:** VACES-F consistently induced HO-1 expression over a 9m period, whereas VACES-C was effective in inducing HO-1 during fall and winter. HO-1 induction positively correlated to higher OC and PAHs that were represented in VACES-F, but also seen with a rise in PAHs in VACES-C during winter months. **MnSOD:** At doses of 2.5 μg/mL, DEPM increased MnSOD in THP-1 cells. **JNK Activation:** DEPM dose-dependently increased JNK phosphorylation but did so without a change in the JNK expression level. DEP-exposed mouse RAW264.7 cells exhibited similar increases in JNK phosphorylation but without increasing JNK expression. **IL-8:** Exposure to DEPM elicited dose-dependent increase in IL-8 levels of THP-1 cells.

Study	Pollutant	Exposure	Effects
Reference: Li et al. (2002, 087451) **Species:** Human **Cell Line:** BEAS-2B, NHBE, THP-1 macrophages **Species:** Murine **Cell Line:** RAW 264.7, macrophages	DEPM (DEP methanol extract) DEPME (DEP methylene chloride extracts) DEPAL (DEPME aliphatic (hexane)) DEPAR (DEPME aromatic (hexane/methlene chloride)) DEPPO (DEPME polar (methylene chloride/methanol)) **Particle Size:** 0.05-1 μm	**Route:** Cell Culture (10^6 cells/mL) **Dose/Concentration:** 0, 10, 25, 50, 100 μg/mL **Time to Analysis:** 30, 60, 120min	**ROS:** BEAS-2B cells demonstrated increased HE fluorescence, indicating increased ROS formation. THP-1 cells were unaffected. **GSH/GSSG Ratio:** DEPM dose-dependently decreased GSH/GSSG in THP-1 and BEAS-2B cells. Similar changes occurred with NHBE cells. THP-1 cells maintained a higher ratio of GSH/GSSG than BEAS-2B and NHBE cells. **NAC on GSH/GSSG Ratio:** Exposure to DEPM in the presence of NAC did not affect the GSH/GSSG ratio in BEAS-2B and NHBE cells. In THP-1 cells, NAC prevented a decline in the GSH/GSSG ratio. **MnSOD and HO-1:** THP-1, BEAS-2B and NHBE cells showed constitutive MnSOD expression and dose-dependent expression of HO-1 protein and mRNA. No change occurred in the expression of β-actin. **DEPAL, DEPAR, DEPPO, CoPP on HO-1 Expression:** DEPPO was more potent than DEPAR. DEPAL lacked activity for THP-1 and BEAS-2B cells. The potency of DEPPO was sufficient to affect cellular viability and HO-1. CoPP induction of HO-1 failed in THP-1 cells, but succeeded in BEAS-2B cells. However, it did not protect against the oxidizing effects of DEPM. **JNK:** JNK activation increased in DEP-exposed THP-1 and BEAS-2B cells.JNK isoforms were observed at doses of ≥ 25 μg/mL. In BEAS-2B cells a high rate of cell death diminished this response at 100μg/mL. NHBE also showed increased JNK phosphorylation at doses 50 - 100 μg/mL. **NAC on JNK:** NAC led to inhibition of JNK activation. **IL-8:** THP-1 cells showed dose-dependent increases of IL-8. NHBE cells showed incremental increases followed by rapid decline at 100 μg/mL attributed to apoptosis. BEAS-2B cells responded to 10 μg/mL with increased IL-8, but cellular toxicity and cell death led to a drop in IL-8 production at higher doses. **Cytotoxicity:** Comparing cytotoxicity at 25 μg/mL DEP, BEAS-2B cells had a higher rate of cell death than THP-1 cells. BEAS-2B cells showed a significant rise in cell death at doses larger than 10 μg/mL. In THP-1 cells, it took doses of 25 μg/mL or more before significant increases occurred. In BEAS-2B, cell death began at 2h. In THP-1, increases in cell death prolonged for 8h or longer. NHBE cells also showed increase rates of cytotoxicity compared to macrophages. NAC in THP-1 interfered with a generation of cytotoxicity, but NAC did not have any decreasing effect on cell death in BEAS-2B or NHBE cells.

Study	Pollutant	Exposure	Effects
Reference: Lindbom et al. (2007, 155934) **Species:** Mouse **Cell Line/Type:** RAW 264.7	PM_{10}: -ST: Street -S: Subway -G: Granite -Q: Quartzite (-G and –Q generated by road simulator at Swedish National Road and Transport Research Institute) Bimodal with peaks around 4-5um and 7-8um. **Particle Size:** PM_{10}	**Route:** Cell Culture (130000 cells/cm^2) **Dose/Concentration:** 1, 10 or 100 μg/mL **Time to Analysis:** 18, 24h Analysis of Arachidonic Release (AA): Cells pre-incubated w/ 1 uCI tritium marked for AA and washed exposed to 10, 50, 100 and 250 μg/mL	**Cellular Viability:** Viability was not influenced by any particle types and in all cases exhibited 90% or higher viability, except for the combination of subway particles and NAC where viability dropped to 20%. **Cytokines:** All particles induced TNF-α secretion in a dose-dependent fashion. PM-S was most potent at 1 μg/mL. PM-G and PM-ST induced effects at 10μg/mL. PM-Q induced increase of TNF-α at 100μg/mL.PM-ST induced IL-6 release at 10μg/mL. PM-G, PM-Q, PM-S induced IL-6 secretion at 100μg/mL. DFX inhibited TNF-α in cells exposed to PM-S and PM-ST. DFX induced increase of TNF-α with PM-Q. For all PM types (except PM-ST) DFX inhibited induced IL-6 secretion. **NO:** PM-ST and PM-G induced a significant release of NO, with PM-ST inducing a higher NO release than PM-G. **NAC:** NAC treatment significantly inhibited both TNF-α and IL-6 secretion with all PM particles. **L-NAME:** L-NAME caused a decrease in NO secretion at 100 μg/mL of PM-ST. L-NAME did not have an effect on granite-induced NO secretion at 100μg/mL. **Cytokine Gene Expression:** TNF-α mRNA showed a trend to increase for –ST, but this did not reach significance. IL-6 gene expression increased for PM-Q, PM-ST, PM-S but not for PM-Q. **AA Release:** PM-S exposure at 100 and 250 μg/mL was the only PM to induce AA release. **Lipid Peroxidation:** All particle types induced lipid peroxidation. PM-S and PM-ST induced significantly higher lipid peroxidation as compared to PM-Q and PM-G. **ROS:** All particle types induced ROS formation. PM-S and PM-ST induced significantly higher formation at 10μg/mL. PM-Q and PM-G induced small but significant decreases in absorption at 100μg/mL. Both PM-ST and PM-S had significant dose responses for all concentrations tested. No difference was observed between PM-G and PM-Q. PM-S and PM-ST pretreated with DFX had a lower ability to induce ROS formation. **Endotoxin Content:** Only PM-ST showed positive results for endotoxin content.

Study	Pollutant	Exposure	Effects
Reference: Liu et al. (2005, 088304) 2005 **Species:** Human **Cell Type:** HPAECs	SE: Wood Smoke Extract (Generated using a stainless steel receptacle containing 100g of dry wood dust) **Particle Size:** NA	**Route:** Cell Culture **Dose/Concentration:** 40 μg/mL **Time to Analysis:** 0-4h; Mitochondrial Membrane Destabilization: 0-60min; DNA Defragmentation: 0-6h; Cytotoxicity: 24h	**Viability:** SE exposure reduced cell viability dose-dependently. Reduction reached ~38% of control. **Effect on Oxidative Stress/ Antioxidant Enzymes:** SE caused an increase in ROS levels, in particular O2- and H2O2 in a time-dependent manner. Exposure to SE for up to 4 h caused a decrease in GSH levels in a time-dependent manner. Increased expression of Cu/Zn SOD mRNA and HO-1 mRNA was observed. Catalase or GPx mRNA expression was unaffected. Upregulation of Cu/Zn SOD and HO-1 occurred in a time-dependent manner **Mitochondrial Translocation/ Capsase-Independent Apoptosis/DNA fragmentation:** Exposure for up to 60 min caused an increase in the percentage of annexin V-FITC-pos cells but not PI-pos cells. At 4h, FDA-pos cells was unaffected. SE exposure caused a loss of mitochondrial membrane potential (indicated by the change in JC-1 fluorescence). Cytosolic bax levels increased after exposure for 1 or 2h and returned to basal level at 4 h after exposure. Levels of procaspase-3 and caspase-9 were unaltered by SE exposure after 4h. Procaspase-3 increased and caspase-9 decreased by H2O2 exposure. SE exposure increased levels of AIF and EndoG (exposure up to 4 h). At 6h, increased DNA defragmentation was observed. Pre-treatment with caspase inhibitors (CMK and Z-VAD-FMK) failed to suppress SE-induced apoptosis. **NAC:** Treatment with NAC prevented ROS increase in cells exposed to SE for 60 min. NAC addition prevented the reduction of GSH by SE. NAC decreased nuclear levels of AIF and EndoG and completely reduced DNA-fragmentation. NAC alleviated the SE-induced reduced viability. GSH and DNA fragmentation were unaffected by NAC.
Reference: Long et al. (2005, 087454) **Species:** Human **Cell Types:** Human, Peripheral blood mononuclear cells (PBMCs) differentiated into MDMs (90-95 % CD14+) and T lymphocytes	Synthetic C and C/Fe particles (phenol and paraformaldehyde polymers on a zeolite template) C/Fe analysis Al 1.38 %, Si 0.33 %, Fe 0.46% **Particle Size:** 1 μm	**Route:** Cell Culture (5 x 10^6 cells (2 mL /well) MDM s **Dose/Concentration:** 5 μg/cm^2 **Time to Analysis:** 2-24h	**ROSs release:** Oxidative burst form C/Fe maxes out at 20 min with no effect from C particles. **Cellular particulate actions:** C particulates were present within lysosomes with small clumps forming after 24 h outside of lysosomes with no evidence of organelle lysis and/or agglomeration. C/Fe particulates showed similar initial effects progressing at 24-h total organelle lysis extending to the outer cell membrane. **T cell effects:** No effects from C or C/Fe particles Medium Effect: Particle agglomeration appears to be a direct result of serum present within a cellfree medium **Hydroxyl radical formation:** C/Fe particles showed an order of magnitude of higher hydroxyl formation as compared to C particles

Study	Pollutant	Exposure	Effects
Reference: Ma et al. (2004, 088417) **Species:** Mouse **Cell Line:** JB6P+ (Epidermal Cell Line)	DEP: SRM 1975 **Particle Size:** 0.5 μm	**Route:** Cell Culture **Dose/Concentration:** Non-cytotoxic: 5, 10, 20 μg/mL; Cytotoxic: 0, 10, 20, 40, 80, 100, 160 μg/mL **Time to Analysis:** 24, 48h; NF-κB and AP-1: 12h Phosphorylation of Akt: 5- 120 min. Effect of LY294002 on DEP: Cells pretreated with LY294002 (0 or 10μM) for 30 min and then exposed to DEP for 0-60min.	**Viability:** Below 20 μg/mL, DEP had no effect. At concentrations greater than 20 μg/mL, DEP caused apoptosis. **NF-κB and AP-1:** DEP stimulated NF-κB activity at 5 and 10 μg/mL. At 20 μg/mL, NF-κB activity decreased, but was still greater than the control. DEP had no effect on AP-1 activity. **PI3K/Akt Signaling Pathway:** DEP induced phosphorylation of Akt on both Thr-308 and Ser-473. LY294002 (an inhibitor of P13K) blocked phosphorylation of Akt, p70/p85 s6 kinase and GSK 3b. LY294002 eliminated DEP-mediated phosphorylation of Akt. Inhibition of P13K by expressing p85 also blocked DEP-induced Akt phosphorylation. DEP induced phosphorylation on GSH-3B on Ser-9 without affecting tyrosine phosphorylation and enhanced phosphorylation of p70/p85 S6 kinase on Thr-389. DEP had no effect on phosphorylation of FKHR. **SAPK/JNK Pathway:** DEP slightly activated the pathway. Increased transient activation of MKK4 (a signal component of the SAPK/JNK pathway) and thus enhanced phosphorylation of SAPK/JNK. DEP promoted phosphorylation of c-Jun and ATF-2. DEP did not affect p38 MAPK or ERK phosphorylation. **LY294002:** Treatment with LY294002 (P13K inhibitor) eliminated DEP-induced NF-κB activity. A similar effect was observed with the use of another P13K inhibitor, wortmannin. TDZD-8 (GSK-3B inhibitor), D-JNKI(a JNK inhibitor), SB202190 (inhibitor for p38 MAPK) or PD98059 (inhibitor for MEK1) had little effect on DEP-mediated NF-κB activation.
Reference: Maciejczyk and Chen (2005, 087456) **Species:** Human **Cell Type:** BEAS-2B isolated from normal bronchial epithelium	CAPs: $PM_{2.5}$ Collected via cyclone inlet on side of building in Tuxedo, NY. Weekdays 9-3 March 4 to September 5, 2003 Mass contributions of the Regional Sulfate, Soil, Oil- Combustions and Unknown/other categories to CAPs are: Regional Sulfate- 65%, Soil- 20%, Unknown/Other- 13% and Oil Combustion- 2%. Composition: * Regional Sulfate characterized by high concentrations of S, Si and organic carbon. * Soil characterized by high concentrations of Ca, Fe, Al and Si. * Oil-Combustion characterized by high concentrations of V, Ni and Se. **Particle Size:** $PM_{2.5}$	**Route:** Cell Exposure (subchronic exposures); Cell Culture (NF-kB) ($9x10^4$ cells/well) **Dose/Concentration:** Ambient 13 ± 9 $\mu g/m^3$ CAPS 109 $\pm$ 178 $\mu g/m^3$ (air exposure); 300 μg PM/ml (culture) **Time to Analysis:** 24h	**NF-κB:** NF-κB response most notably correlated with V and Ni - elements associated with oil combustion source category (oil combustion makes up the group that is the smallest percentage of CAP mass).
Reference: Madden, M. C. Dailey, L.A. Stonehuerner, J.G. 2003 **Species**: Human **Cell Type:** NHBE	DEP(SRM 2975) Diesel Exhaust Extracts from a High load (HL ~ 75% engine load) or Load load (LL 0% engine load): Obtained from Caterpillar diesel engine, 4cycl, 4 stroke, model 3304 Particle Characterization: LL extract has greater amount of low-molecular-weight carbonyls (2-5 carbons). HL had more intermediate size carbonyls (6-9 carbons). Largest carbonyls analyzed (11-12 carbons) found in similar ratios in the two types of extract (number of carbons is indicative of differences in boiling points). **Particle Size:** NR	**Route:** Cell Culture **Dose/Concentration:** 0, 10, 50, 100, 250, 500 μg/well **Time to Analysis:** 24h (after 2h of treatment, 0.5 ml of BEGM added to each well and cells incubated for an additional 22h) .	**Cytotoxicity:** -LL, -HL and SRM had no effect on LDH release. **^{51}Cr:** Incubation of cells with -LL or SRM (10 to 500 μg/well) had no effect. 500 μg/well of -HL induced a significant increase in ^{51}Cr release. **IL-8:** -HL induced a 5-fold increase in IL-8 at 500μg/well. A decrease was observed at the highest dose of -LL extract. SRM did not significantly alter IL-8 production. **PGE2:** Production of PGE2 (inflammatory/immune mediator) increased in cells treated with HL extract at 500 μg/well. -LL had no effect. Stimulation with melittin caused -LL extract to have inhibitory effect on PGE2 at 500 μg/well. SRM had no effect.

Study	Pollutant	Exposure	Effects
Reference: Matsuo, M. Shimada, T. Uenishi, R. 2003 **Species:** Human **Cell Type:** NHBE, NHPAE, TIG-1, TIG-7 (normal human lung embryonic fibroblasts)	DEP: prepared at National Institute for Environmental Studies (Tsukuba, Japan) RDEP: residual DEP (after sequential extraction with hexane (NOS), benzene, dichloromethane, methanol, 1N ammonium hydroxide) **Particle Size:** 0.4μm (MMAD)	**Route:** Cell Culture (NHBE: $5x10^4$ cells/cm^2; NHPAE: $3x10^3$ cells/cm^2; TIG-1 and TIG -7: $3x10^3$ cells/cm^2; Apoptosis: $2x10^5$ cells/cm^2; ROS/NO: $2x10^4$ cells/cm^2; Cytoxicity Modulating Agent: $3x10^4$ cells/cm^2; GSH: $3x10^4$ cells/cm^2) **Dose/Concentration:** 25, 50, 100, 200, 300, 400, 500 μg/mL **Time to Analysis:** 1h	**Cytoxicity in NHBE:** Both DEP and RDEP exhibited dose-dependent cytotoxicity at concentrations beginning from 50 μg/mL and higher. RDEP was less cytotoxic than DEP. DEP exposure resulted in necrosis, not apoptosis. **Comparative Cytotoxicity:** The order of LC_{50} values (50% lethal concentration) was: NHBE (118 μg/ml), NHPAE (137 μg/ml), TIG (270 μg/ml). NHBE's susceptibility was higher than the susceptibility of NHPAE and TIG cells. **ROS/NO:** DEP induced dose-dependent ncreases at 25 and 50 μg/mL. **Reduced Glutathione:** DEP induced dose-dependent decreases. At 200 or 300 μg/mL, GSH levels decreased by 55.2 or 97.3%, respectively. **Antioxidant Effects:** Various antioxidants either decreased DEP cytotoxicity (PMC, Ebselen, EUK-8) or had no effect on DEP cytotoxicity (SOD, catalase, GSH, α-tocopherol) **Chelating Agents:** DEP became less cytotoxic when Ion-chelating agents were preincubated for 24h. No effect on DEP cytotoxicity was observed when chelating agents were administered to cells immediately after sonication. **Endocytosis inhibitors:** Decreased DEP toxicity was observed in a dose-dependent manner.
Reference: Matsuzaki, T. Amakawa, K. Yamaguchi, K. 2006 **Species:** Human **Cell Type:** Peripheral neutrophils **Gender:** Male and Female **Age:** 20-40yrs	DEP: generated from a 4JB1-type, 4 cyl Isuzu diesel engine me-DEP: methanol extract of DEP (40 % of DEP by dry weight) **Particle Size:** 0.4 μm	**Route:** Cell Suspensions ($5 X10^5$ cells/mL) **Dose/Concentration:** all me-DEP f-actin: 1, 5, 10 μg/mL CD11b: 5, 10, 30 μg/mL IL-8: 5, 10, 30 μg/mL H2O2: 5, 10, 30, 60 μg/mL MMP-9, LTB-4: 5, 10, 30, 60 μg/mL **Time to Analysis:** f-Actin: 15min CD11b: 2h IL-8: 2 or 24h H2O2: 30min MMP-9, LTB-4: 2 or 24h	**F-Actin:** Treatment with me-DEP showed a dose-dependent increase in the f-actin content of neutrophils and this increase was significantly higher at 5 and 10 μg/mL. **CD-11b:** Treatment increased CD-11b expression two-fold at 30 μg/mL. **IL-8:** Minimal response was observed after 2h. A significant increase was observed (243%) at 24 h with 30μg/mL. **LTB-4:** At 2h, LTB4 increased to 115% and 119% with 30 and 60 μg/mL me-DEP respectively. At 24 h with 60 μg/mL me-DEP, LTB-4 increased to153%. **H2O2:** Exposure to 30 and 60 μg/mL of me-DEP induced large dose-dependent increases of 563% and 1220%, respectively. **MMP-9:** A significant increase at 2 and 24 h were observed. In both exposure periods, 30 μg/mL induced larger increases than 60 μg/mL.
Reference: Molinelli, A.R. Santacana, G.E. Madden, M.C. 2006 **Species:** Human **Cell Type:** NHBE, BEAS-2b (transformed bronchoepithelial cells)	PMH: PM_{10} extracts in hexane PMA = PM_{10} extracts in acetone of residue after hexane extraction -G: Guaynabo(Urban) and -F: Fajardo (Preservation Area), PR, USA 1999 **Particle Size:** PM_{10} PM_{10} extracts collected by Puerto Rico Environmental Quality Board	**Route:** Cell Culture ($3x10^3$ cells/well) **Dose/Concentration:** NHBE exposed to 0-100 μg/mL of PM_{10} BEAS-2B exposed to 10,100, 250 μg/mL of PM_{10} **Time to Analysis:** 48h	**Metal analysis:** Hexane extracts Cu, V, Ni all higher in winter than summer. For hexane extracts within the same season, metal concentrations were higher in the Fajardo extracts. On the other hand, the acetone extracts from Guaynabo generally had higher metal concentrations than Fajardo. **Cytotoxicity NHBE:** The order of most to least toxic for PM extracted with hexane is: winter–G, winter-F, summer -G , summer-F. The order of most to least toxic for PM extracted with acetone is: summer-G, summer-F, winter-g. **Cytotoxicity BEAS-2:** For PM extracted with hexane, the cytotoxicity order is: winter-G, winter-F, summer-G, summer-F. The order for acetone extracted PM is: summer-G , summer-F, winter-F, winter-G. Effects trend similar to metal levels (no analysis). Summer extracts showed linear dose-response curves. Winter extracts exhibited more equivocal results, especially for Fajardo. Results suggest that NHBE cells are more sensitive than the BEAS-2B cells to PM extracts.

Study	Pollutant	Exposure	Effects
Reference: Moller et al. (2002, 036589) **Species:** Canine, Mouse **Cell Type:** Beagle-Dog Alveolar Macrophages (BD-AM), J774A.1 (from cell line BALB/c/NIH)	fTiO2 (origin NR) ufTiO2 (origin NR) ufP-G: carbon black (Printex-G, Degussa, Frankfurt, Germany) ufP90: carbon black (Printex90, Degussa, Frankfurt, Germany) ufEC90: elemental carbon (produced by electrical spark generator under standardized conditions with low impurities) DEP (SRM 1650) UrbD: Urban Dust (SRM 1649a) **Particle Size:** (in diameter) TiO_2: 220nm; ufTiO2: 20nm; ufP-G: 51nm; ufP90: 12nm; ufEC90: 90nm; DEP: 120nm; UrbD: NR	**Route:** Cell Suspension **Dose/Concentration:** 10, 32, 100, 320 μg/mL **Time to Analysis:** 24h	**Cytoskeleton of J774A.1:** At doses of 32 μg/mL or less, the particles did not significantly influence relaxation and stiffness. fTiO2and ufP90 had no effect at any dose. ufTiO2 at 320 μg/mL induced retarded relaxation and significant stiffening. ufEC90 induced dose-dependent retardation of relaxation and increased stiffening. DEP and UrbD induced similar results. **Cytoskeleton of BD-AM:** ufTiO2 and fTiO2 both induced some retarded relaxation and increased stiffening at 100 μg/mL dose. ufTiO2 appears to increase stiffening in a dose-dependent manner. ufEC90 induced dose-dependent acceleration of relaxation due to the carbon content of ufEC90. DEP also induced acceleration of relaxation as well as a decrease in stiffness. **Phagocytosis:** At 24 h, ufTiO2 and fTiO2 significantly reduced phagocytotic ability in J774A.1 but not in BD-AM. All carbonaceous particles induced significant impairment in J774A.1. All ultrafine carbon particles inhibited BD-AMs. **Cell Proliferation:** ufTiO2 significantly inhibited proliferation compared to the control and fTiO2 at 100 μg/mL in J774A.1. ufEC90 and ufP90 inhibited proliferation slightly with ufEC90 inducing slightly greater inhibition than ufP90. UrbD and DEP also significantly reduced proliferating. **Apoptosis:** All particles induced decreased viability at 100 μg/mL in both cell types. With ufTiO2 inducing greater apootosis than fTiO2, ufEC90 than ufP90 and ufEC90 than ufP-G.
Reference: Mutlu GM et al. (2006, 155994) **Species:** Human, Rat **Cell Type:** A549, Primary Alveolar Type II Epithelial Cells	PM_{10} (Collected by baghouse from ambient air in Dusseldorf, Germany) **Particle Size:** PM_{10}	**Route:** Cell Culture **Dose/Concentration:** 0.05, 0.5, 5. 50 μg/cm^2 **Time to Analysis:** 24h	**Na,K-ATPase Plasma Membrane Protein:** PM_{10} induced a decrease of protein in the plasma membrane of A549 cells. Total Na,K-ATPase levels were unaffected. **ROS:** Pretreatment with EUK-134, superoxide dismutase and catalase mimetic, inhibited the decrease of GSH. Furthermore, it attenuated the decrease of NA,K-ATPase in A549 cells. **NA, K-ATPase Activity:** PM_{10} induced a dose-dependent decrease in ouabain-sensitive liberation of 32P from AT32P in primary rat alveolar type II cells. This effect was inhibited with pretreatment with EUK-134.
Reference: Nam, HR.Y. Choi, B.HR. Lee, J.Y. 2004 **Species:** Human **Cell Type:** A549	$PM_{2.5}$ Collected from hospital rooftop, Seoul, South Korea **Particle Size:** $PM_{2.5}$	**Route:** Cell Culture **Dose/Concentration:** 0.5, 1, 10, 25, 50 μg/cm^2 **Time to Analysis:** 6, 24h	**NFκB/IκBα:** 50 μg/cm^2 DEP induced IκBα degradation which peaked at 2h and recovered after 4h. Treatment with increasing amount of $PM_{2.5}$ resulted in a dose-dependent decrease in IκBα. $PM_{2.5}$ increased NFκB in a dose-dependent manner up to 10 μg/cm^2. NFκB induction peaked at 12h. **IL-8:** $PM_{2.5}$ treatment increased protein level more than 3 fold with 100 μg/cm^2 $PM_{2.5}$. mRNA levels also increased. **iNOS Inhibitor:** $PM_{2.5}$ induced IL-8 elevation was completely blocked by iNOS inhibitor. iNOS inhibitor also negated $PM_{2.5}$ induction of NFκB activity. Antioxidants and iNOS inhibitor reduced PM-induced IκBα degradation.
Reference: Nozaki JI et al. (2007, 097862) **Species:** Mouse **Cell Line:** LA-4 (Alveolar Epithelial Cells)	PM: PM Rooftop 5story, urban, Japan PME: dichloromethane extract of PM filtered P90: Printex 90 (carbon black) (Degussa) **Particle Size:** PM: 0.22 μm; PME: 2.5 μm; P90: 14nm	**Route:** Cell Culture (1.4x10^4 cells/cm^2) **Dose/Concentration:** 1.1 μg/cm^2 **Time to Analysis:** 24, 28, 72h	**Cytotoxicity:** P90 had no effect. PM and PME were cytotoxic at similar levels. **Protein Expression:** All particles affected protein expression (no specific protein- 2D gel electrophoresis).

Study	Pollutant	Exposure	Effects
Reference: Obot et al. (2002, 042370) **Species:** Mouse **Cell Line:** BALB/c **Cell Type:** AMs	PM: SRM 1648 PM-100: PM heated to 100 C PM-500: PM heated to 500 C PM-PH: PM acid digestion PMAC: Acetone extraction PMCH: Cyclohexane extraction PMH2O: Water extraction All extract fraction used as residual particles **Particle Size:** NR	**Route:** Cell Culture (5 X10^5 cells/mL) **Dose/Concentration:** PM: 200 μg/mL; PM-100: 188 μg/mL; PM-500: 130 mg/l; PM-PH: 94 μg/mL; PMAC: 173 μg/mL; PMCH: 171 μg/mL; PMH2O: 188 μg/mL Fraction doses adjusted for mass loss during fraction treatment **Time to Analysis:** 4h	**Cytotoxicity:** All 7 fractions had cytotoxic effects. PM had highest cytotoxicity. PM-500, PM-PH, PMAC less toxic than PM. **Apoptosis:** All 7 fractions significantly increased apoptosis. The PM fractions that induced the greatest apoptosis in descending order are: PM, PMH2O, PM-100, PM-500, PMAC, PMCH and PM-PH. PM-induced apoptosis (only PM, PM-500 and PMAC tested) was blocked by poly I or 2F8 antibody (scavenger receptors). **Particle Characterization:** Untreated PM and PM-100 did not have measurable amounts of transition metals on its surface. Measured components include carbon, O2, N, S, Si, Ca, Al, P, Cl. PM-PH mostly contained O2 and Si. PM-500 had increased O2, Si compared to PM and measurable amounts of Na, K., Zn, Co, Pb, Fe. Included increased surface density of S, P, Al. PMCH lacked nonpolar organic compounds.
Reference: Obot et al. (2004, 095938) **Species:** Mouse (7-9wks), Human **Cell Line:** Mouse-BALB/c **Cell Type:** AMs	PM: SRM 1648 (collected by bag-hosue in St. Louis, MO). PM-100: PM heated to 100¤ C PM-500: PM heated to 500¤ C PM-PH: PM acid digestion PMAC: Acetone extraction PMCH: Cyclohexane extraction PMH2O: Water extraction All of the 6 extract fractions from PM1648 $PM_{2.5}$: Collected in Houston, TX **Particle Size:** PM1648: NR; $PM_{2.5}$	**Route:** Cell Culture (5 X10^5 cells/mL) **Dose/Concentration:** PM: 200 μg/mL; PM-100: 188 μg/mL; PM-500: 130 mg/l; PM-PH: 94 μg/mL; PMAC: 173 μg/mL; PMCH: 171 μg/mL; PMH2O: 188 μg/mL Fraction doses adjusted for mass loss during fraction treatment $PM_{2.5}$ = 50, 100, 150, 200 μg/mL **Time to Analysis:** Mouse-4h; Human-24h.	**Human AM Viability:** Only PM, PM-100, PMAC and PMH2O decreased viability. **Human AM Apoptosis:** PM, PM-100 and PMH2O increased apoptosis. PM induced greater apoptosis than PM-100 and PMH2O. **Regression Analysis Mouse vs Human:** Although individual fractions differed somewhat, cell viability and apoptosis of all 7 fractions showed linear regression **Human and Mouse AM Viability with $PM_{2.5}$:** Nearly identical dose-dependent decrease was exhibited starting at 50 μg/mL **Human and Mouse AM Apoptosis with $PM_{2.5}$:** Nearly identical dose-dependent increases were exhibited with human AM responses peaking at 150 μg /mL and declining at 200 μg/mL (no mouse data for 200 μg/mL). **Regression Analysis with $PM_{2.5}$:** Excellent correlation of mouse and human responses for viability and apoptosis was exhibited.
Reference: Okeson et al. (2003, 042292) **Species:** Rat **Cell Type:** RLE-6TN (Type II Alveolar Epithelial Cells)	CG: Coal ash, Germany CU: Coal ash, USA 5C: PM # 5 Oil fly ash coarse 5F: PM #5 Oil fly ash fine 6MSC: PM #6 Oil med sulfur fly ash coarse 6HSC: PM # 6 Oil high sulfur fly ash coarse 6HSF: PM # 6 Oil high sulfur fly ash fine **Particle Size:** CG, CU: NR; 5C, 6MSC, 6HSC $>$ 2.5 μm; 5F, 6HSF $<$ 2.5 μm	**Route:** Cell Culture **Dose/Concentration:** Coal Fly Ash 12.5, 25, 50, 125, 250 μg/mL Oil Fly Ash - 100 μg/mL **Time to Analysis:** 24h	**Oil PM Characterization:** Generally, the fine fractions had higher metal levels than the coarse fractions except for Zn. High sulfur had a higher metal content than med sulfur. Carbon percent weight was stable across all 5 fractions. **Coal Ash Cytotoxicity:** CG treatment exhibited similar cytototoxic results as CU. Cytotoxic effects were exhibited at concentrations of 12.5 μg/mL and above. Effects remained steady at concentrations above 50 μg/mL. **Oil Ash Cytotoxicity:** Cytotoxic effects were induced by all. The order of PM fractions inducing the most cytotoxicity to the least is the following: 5F, 6HSF, 6HSC, 5C, 6MSC. **Correlation of Metal Content and Cytotoxicity:** Fe, V showed a reasonable correlation. Zn had no correlation. **Cell Metabolism:** An inhibitory effect was observed with 100 μg/mL coal ash after 6h. After 12h of exposure, CU, unlike CG, does not continue to inhibit cell metabolism. Oil ash was generally less effective than coal ash. The order of PM fractions inhibiting metabolism the most to the least is the following: 5F, 6HSC, 5C, 6MSC. 6HSF not tested.

Study	Pollutant	Exposure	Effects
Reference: Okeson et al. (2004, 087961) **Species:** Rat **Cell Type:** RLE-6TN (Type II Alveolar Epithelial Cells)	Zn, V, Fe chloride as salts (valence state not reported i.e., Fe II or Fe III) **Particle Size:** NR	**Route:** Cell Culture (50000 cells/well) **Dose/Concentration:** 0.001, 0.01, 0.1, 1.0, 10 mM **Time to Analysis:** 24h	**Cytotoxicity:** All metals cytotoxic at concentrations greater than 0.1 mM. V is 5 times less cytotoxic than Zn, and Fe is 7 times less cytotoxic than Zn with a EC_{50} of 3mM and 4mM, respectively. At 10 mM of each metal, no surviving cells were present. **NCS:** Incubation with NCS (5 or 10 %) decreased toxicity of Zn, especially at 0.1 mM, but had no effect on Fe or V toxicity. **Albumin:** BSA decreased Zn toxicity at equivalent concentrations but to a lesser extent than NCS.
Reference: Osornio-Vargas et al. (2003, 052417) **Species:** Mouse **Cell Line/Type:** J774A.1, L929 (Mesenchymal Cells)	PM_{10} $PM_{2.5}$ -N = Northern (industrial) -SE = Southeastern (lake basin dust) sites, both heavy vehicular traffic, Mexico City, Mexico **Particle Size:** PM_{10}; $PM_{2.5}$	**Route:** Cell Culture (J774A.1: 15000 cells/cm^2; L929: 30,000 cells/well) **Dose/Concentration:** 20, 40, 80 $\mu g/cm^2$ **Time to Analysis:** 24-72h	**PM Characterization:** Elements similar in particle types with elements in PM_{10} more abundant. Northern particles contained more Co, Zn, Ni, Pb. **Endotoxin:** All PM samples had detectable amounts of endotoxin. $PM_{2.5}$ -N had 22 EU/mg. PM_{10}-N had 30 EU/mg. $PM_{2.5}$ -SE had 12 EU/mg. PM_{10}-SE had 59 EU/mg. **Cytotoxicity (J774A.1):** The two northern samples, $PM_{2.5}$ and PM_{10}, both induced similar cytotoxic effects at 40% survival. PM_{10}-SE and $PM_{2.5}$ -SE induced dose-dependent responses. In general, the northern samples had a higher cytotoxic effect than the southern samples. **Apoptosis (J774A.1):** Northern samples induced more apoptosis than did the southeastern samples. There was no difference between PM_{10} and $PM_{2.5}$ induced apoptosis. **TNF-α and IL-6 (J774A.1):** TNF-α and IL-6 induced dose-dependent increases. At 80 $\mu g/cm^2$, PM_{10} -SE induced the most production of IL-6 followed by $PM_{2.5}$ –SE, PM_{10}-N , and $PM_{2.5}$ -N. **J774A.1 Supernatant Toxicity (L929):** Conditioned medium from J774A.1 pre-exposed to each PM type reduced cell viability in L929 cells. This was correlated with TNF-α level in supernatants.
Reference: Pei, X.HR. Nakanishi, Y. Inoue, HR. 2002 **Species:** Human **Cell Type:** A549	B[a]P 1-NP: 1-nitropyrene Both purchased from Aldrich Chemical Co., MI, USA. **Particle Size:** NR	**Route:** Cell Culture **Dose/Concentration:** B[a]P: 10μM; 1-NP: 5 μmol **Time to Analysis:** 4, 8, or 24h	**IL-8:** IL-8 mRNA increased by B[a]P and 1-NP in a time-dependent manner with 1-NP increasing higher levels than B[a]P at half the molarity. **NK-kB:** 1-NP induced NF-kB DNA binding activity within 4h and maintained it for 24h. B[a]P induced similar responses. This binding was negated by treatment with a 50-fold excess of unlabeled NF-kB consensus probe. **IkBα (NF-kB Inhibitor):** After 3 days of treatment with the recombinant adenovirus, A549 cells expressed more than a 50-fold increase in IkBa levels, completely suppressing 1-NP induced NF-kB activation in addition to reducing IL-8 mRNA levels induced by 1-NP by 80%.
Reference: Penn et al. (2005, 088257) **Species:** Human **Cell Type:** BEAS-2B	BDS: Butadiene soot (created on-site by passing BD through a back-flash protected stainless steel two-stage regulator to a stainless steel Bunsen burner) -P1: < 2.5um -P2: 2.5-10um -P3: > 10um BDS-W: solvent washed Graphite Composition: < 2.5um = 92% 2.5 - 10um = 5% > 10um = 3% **Particle Size:** BDS-P1: < 2.5 μm; BDS-P2: 2.5-10 μm; BDS-P3: > 10 μm	**Route:** Cell Culture (1-1.5x10^6 cells) **Dose/Concentration:** 3mg BDS **Time to Analysis:** 5min-72h	**Particle Characterization:** By weight, elemental carbon makes up 94% of BDS, hydrogen 2%, nitrogen and sulfur 1%, and oxygen less than 0.1%. **PAH Components of BDS: 13 prominent PAHs:** acenaphthylene, fluorene, anthracene, cyclopentaphenanthrene, fluoranthene, acephenanthrylene, pyrene, benzofluorenes, acepyrene, chrysene, benzopyrenes, perylene, benzoperylene. **BDS Activity:** At 60-120min, BDS was observed in the cells. At 4h, fluorescence observed in cytoplasmic vesicles and increased during the first 24 h then plateaued for the next 72h. BDS-W appeared in vesicles sooner than BDS.

Study	Pollutant	Exposure	Effects
Reference: Pozzi et al. (2005, 088610) **Species:** Mouse **Cell Type:** RAW 264.7	PM: Collected continuously for 15 days, 8-10m from street, Sept 1999, Rome, Italy -F = Fine particulate -C = Coarse particulate CB (Degussa Huber NG90) **Particle Size:** PM-F: 0.4-2.5 μm; PM-C: 2.5-10 μm; CB: 200-250nm	**Route:** Cell Culture (1.3x10^5 cells/well) **Dose/Concentration:** 30 μg/mL; 14 μg/cm^2 120 μg/mL; 54 μg/cm^2 **Time to Analysis:** 5, 24h	**Cytotoxicity:** For 24h, lower levels of PM-F, PM-C, and CB had no effect on cell viability. Higher levels of PM-C and CB induced a significant release of LDH. **Arachidonic Acid (AA):** Both fractions of PM increased AA release in a dose-dependent manner at 5h. CB increased a release only at the higher concentrations although, in terms of magnitude, the CB-induced release was much less than the ambient PM-induced release. Pretreatment with deferoxamine was not effective in decreasing AA release. **TNF-α:** TNF-α levels increased significantly for both concentrations and time periods for PM. PM-C at 24 h was significantly lower than at 5h for both concentrations. PM-C at 30 μg/mL induced a much greater TNF-α release than PM-F at 5h. **IL-6:** PM-F significantly increased at 5h for both concentrations. Elevated IL-6 levels were exhibited at both PM-C doses at 24 h. At 5h, only the high dose elevated IL-6 levels. CB was devoid of an effect on IL-6. LPS-induced IL-6 response was similar to coarse PM at the high dose, with the response being greater at 24 h than at 5h.
Reference: Prophete et al. (2006, 156888) **Species:** Rat **Cell Type:** NR8383 AMs	Ambient $PM_{2.5}$ NYC: 1st and 26 St, NYC LA: San Gabriel foothills, Claremont, CA SEA: 15th Ave S and S.Charleston, Seattle, WA V, Mn, Al, Fe levels in PM added metals to cells V: Na_3VO_4 Al: $AlCl_3.6H_2O$ Mn: $MnCl_2.4H_2O$ Fe: $FeCl_3.6H_2O$ **Particle Size:** $PM_{2.5}$	**Route:** Cell Culture (2 X10^5 cells/mL) **Dose/Concentration:** Fe(III) 16 μmol V, Mn, and Fe(III) mixtures with V or Mn in molar ratios 0.02, 0.08, 0.2 and 0.4 X Fe(III) Al and Fe(III) mixtures with Al in molar ratios 0.37, 0.75, 2, 7.5 X Fe(III) **Time to Analysis:** 20h	**Particle Characterization:** Fe and metal to F ratios based on ratios observed in $PM_{2.5}$ from LA, SEA and NYC sites. V: Fe ratios remarkably similar among sites. Fe levels fixed at NYC level of 16 μm (highest). **IRP:** Coexposure with 3 metals increased IRP binding activity relative to Fe III alone, by up to 3.5 fold for Al (1.5-3 ratio), 2 fold for Mn (0.08-0.2 ratio) and 7 fold for V (0.2 ratio). IRP activity dropped at higher ratios. A drop in IRP activity at higher ratios may be result of cytotoxicity for Al, but not for V and Mn. **iNOS:** Al induced iNOS expression dose-dependently. There was no observed effect for Mn and V. **Induction of Hypoxia-inducible Factor (HIF-1α):** Only V and Al induced HIF-1α. **Activation of ERK1 and -2:** V and Al induced pERK1, but only V induced pERK2. Mn had no increasing effects, but data indicated a decreasing induction.
Reference: Ramage and Guy (2004, 055640) **Species:** Human **Cell Type:** A549	PM_{10}: Collected in Wolverhampton, UK ufCB: Ultrafine Carbon Particles (Origin not reported) **Particle Size:** PM_{10}, ufCB: $<$100 nm (diameter)	**Route:** Cell Culture **Dose/Concentration:** 80 μg/mL **Time to Analysis:** 0, 0.5, 3, 6, 18h	**CRP:** Treatment with ufCB or PM_{10} produced an increase in CRP expression with similar effects noted after 6h. PM_{10} induced greater increases than ufCB. Both the cytoplasm and nucleus contained CRP. **Hsp70:** PM_{10} and ufCB induced increased levels at all time points with ufCB inducing greater levels than PM_{10}. Hsp70 expression was observed in the cytoplasm and nucleus. **Antioxidants of CRP and Hsp70:** Coincubation of ufCB with Nacystelin and Trolox caused a small reduction in CRP and Hsp 70.
Reference: Rao et al. (2005, 095756) **Species:** Rat **Strain:** Sprague-Dawley **Cell Type:** AMs and cultured lung fibroblasts	DEP: SRM 2975 (NIST) **Particle Size:** 0.5μm	**Route:** Cell Culture **Dose/Concentration:** 200 μg/mL **Time to Analysis:** Measured after 4h incubation.	**mRNA Expression:** No change in IL-1β or iNOS were observed. Data suggests that the lung fibroblasts is the main source of IL-6 and MCP-1 in BAL fluid because of their comparatively high message levels. Due to the extreme variability in results, the cause of an increase on co-culture with AMs and/or DEPs was not assessed.

Study	Pollutant	Exposure	Effects
Reference: Reibman et al. (2003, 156905) **Species:** Human **Cell Type:** HBEC, BEAS-2B	UFPM: Ultrafine PM FPM: Fine PM IPM: Intermediate PM CPM: Coarse PM CB: Carbon black All PM collected 8th floor, 26th St and 1st Ave, New York City, NY **Particle Size:** UFPM: <0.18 μm; FPM: 0.18 - 1.0 μm; IPM: 1.0 - 3.2 μm; CPM: >3.2 μm	**Route:** Cell Culture **Dose/Concentration:** 11 μg/cm^2; 100 μg/mL **Time to Analysis:** 6, 18h	**Cytotoxicity:** After treatment, cells were more than 90% viable. UFPM and FPM caused no gross alterations in cell morphology or adhesion. **MIP3α/CCL20 mRNA (6h):** Stimulation of mRNA released by HBEC upon exposure to UFPM appeared similar to that provided by TNF-α (5 μg/mL) and IL-1β (10 mg/mL). **MIP3β/CCL20 protein in HBEC (18h):** TNF-α and IL-1β induced a dose-dependent increase in MIP3α/CCL20 protein (0-10 ng/mL), whereas Il-4 and IL-13 induced MIP3α/CCL20 protein release that reached maximum levels at 1 ng/mL. No release of MIP1α/CCL3 nor RANTES/CCL-5 was observed upon stimulation with cytokines. **Secretion of MIP3α/CCL20 in response to PM (18h):** All PM fractions less than 2.5 μm resulted in the release of MIP3α/CCL20 protein in HBEC roughly equivalent amounts. CB similar in size to UF/fine PM did not result in the release of MIP3α/CCL20, nor did LPS (0.01-1.0 μg/mL). No release of MIP1α/CCL3 nor RANTES/CCL 5 was observed upon stimulation by PM fractions. **Activation of MAPK (ERK1/2 and p38):** ERK1/2 and p38 was activated by TNF-α, IL-1β, IL-4 and IL-13 within 15 min and was sustained for at least 60 min. Erk1/2 and p38 inhibitors reduced MIP3α/CCL20 release in BEAS-2B cells in response to cytokines.
Reference: Riley et al. (2003, 053237) **Species:** Rat **Cell Type:** RLE-6TN (Type II Alveolar Epithelial Cells)	Zn: ZnCl2 Cu: CuCl2 Fe: FeCl2 V: VCl4 Ni: NiCl2 **Particle Size:** NR	**Route:** Cell Culture **Dose/Concentration:** 0.01, 0.1, 1.0, 10 mM **Time to Analysis:** 2, 4, 24, 72h	**Cytotoxicity (SDH):** All particles were cytotoxic in a dose-dependent manner. Zn and V were cytotoxic at 0.05 mM, Cu at 0.5mM, Ni at 0.8mM and Fe at 2 mM. For Zn, cell death (LDH) had a biphasic response: a slow logslope until approx 0.1mM at which point it rapidly accelerated to a peak at 5mM with a small decline at 10mM. Most of Zn cytotoxicity was not due to apoptosis. LPS did not affect either Zn or Cu cytotoxicity. **Metabolism Inhibition Time Course Response (Cu and Zn only):** At high (1 mM) concentrations, Zn toxicity peaked at 36-48h followed by a 2-fold recovery by 72h. Cu showed a faster, steady decline plateauing after 36h. At low concentrations (0.1 mM), Cu showed a steady slow decline. At 48h, Zn decreased faster to max activity and returned to control by 72h. **IL-6 Secretion:** Zn and Cu both decreased IL-6 secretion. Decreases were very similar for both metals and concentrations when expressed as secretion per viable cell ratio except for Zn at 1.0 mM. **Metal Combinations:** Zn and Cu gave variable results. Zn protected against V cytotoxicity. Zn and Cu had an additive response. Zn did not affect Fe toxicity.

Study	Pollutant	Exposure	Effects
Reference: Riley et al. (2005, 096452) **Species:** Rat, Human **Cell Type:** RLE-6TN, NR8383 Alveolar Macrophages, A549	Fe: FeCl2 Ni: NiCl2 Cu: CuCl2 V: VCl2 **Particle Size:** NR	**Route:** Cell Culture (5 x10^4 cells/well Alveolar Cells; 1.2x10^5 cells/well NR8383) **Dose/Concentration:** AMs: 0.02, 0.05, 0.07, 0.08 mM; RLE-6TN: 0.1, 0.2, 0.6, 1.0, 6.0 mM; A549: 0.5, 0.8, 4.4, 4.8 mM **Time to Analysis:** 2-48h	**Relative Sensitivity of Cell Strains to Metal Chloride:** NR8383 was more sensitive than RLE-6TN and A549 except for V where NR8383 and RLE-6TN were both more sensitive than A549. **Relative sensitivity of Cell Strains to Metal Chloride vs Sulfate:** With the exception of Cr, sulfate was generally more cytotoxic than chloride (note V valence state). **A549 Cytotoxicity Time Course:** Zn cytotoxicity takes 24 h to develop whereas Cu cytotoxicity develops within 2 h. LDH release for Cu, however, develops in 24h. **RLE Cytotoxicity Time Course:** Zn starts at 2 h and develops until 24 h. Cu develops within 2 h and continues until 24 h where it is less toxic than Zn. Both release equivalent amounts of LDH after 24 h. **NR8383 Cytotoxicity Time Course:** Both Zn and Cu exhibit time dependent toxicity beginning as early as 4h. LDH release maximizes at 12 h and either remains steady or declines.
Reference: Ritz, S.A. Wan, J. Diaz-Sanchez, D. 2007 **Species:** Human **Cell Type:** BEAS-2B, NHBEC	DX: Extract of DEP (generated from a light duty four-cylinder diesel engine 4JB1 type Isuzu Automobile) **Particle Size:** $< 1\mu$m (diameter)	**Route:** Cell Culture **Dose/Concentration:** 0, 20, 50, 100 μg/mL **Time to Analysis:** 24h	**NQO1 (Sentinel Phase II Enzyme):** Cells transfected with NQO1 reduced induction of IL-8 by DX exposure. **Sulfurophane:** Increased gene expression of phase II enzymes, particularly NQO1, was observed in both cell types. Gene expression in BEAS-2B was greater than that of NHBEC. Sulfurophane did not upregulate GSTM1 in BEAS-2B but induced a 2-fold increase in NHBEC. Pretreatment also inhibited DX-induction of IL-8 in both cell types. **Cytokines:** DX induced significant increase of IL-8 in both cell types at concentrations of 10 μg/mL or higher. GM-CSF and IL-8 remained unaffected in BEAS-2B. GM-CSF and IL-8 Increased in NHBECs and reached statistical significance at 25 μg/mL.
Reference: Rosas Perez et al. (2007, 097967) **Species:** Mouse **Cell Type:** J774A.1	PM_{10} Collected in Mexico City, Mexico from January-June, 2002 North: Iztacala, manufacturing industry; Center: Merced, heavy traffic; South: Ciudad Universitaria, residential Principal Component Analysis of Air Pollution Data: Group 1:S/K/Ca/Ti/Mn/Fe/Zn/Pb (43% of variance); Group 2: Cl/Cr/Ni/Cu (16%); Group 3: Endotoxins/OC/EC (14%). For all 3 sites: Averages of Group 1 is statistically different among the center, north and south sites with the central site producing the highest values. Group 2 is similar among the sites and, for Group 3, the north had a lower average than the center and south sites. **Particle Size:** PM_{10}	**Route:** Cell Culture (1.5x10^4 cells/cm^2) **Dose/Concentration:** 20, 40 or 80 μg/cm^2 **Time to Analysis:** 72h	**Cytotoxicity:** Responses were dose-dependent; there was no observed site interaction. Cytotoxicity seems to be a result of of the following components: S/K/Ca/Ti/Mn/Fe/Zn/Pb. **IL-6:** Only the center site at 40 μg/cm^2 induced an increase. Induction of higher IL-6 levels seems to be related to high values of S/K/Ca/Ti/Mn/Fe/Zn/Pb and endotoxins/OC/EC. **TNF-α:** Production was induced by all samples in a dose-dependent manner. Similar to IL-6, induction of higherTNF-α levels seems to be a result of high values of S/K/Ca/Ti/Mn/Fe/Zn/Pb and endotoxins/OC/EC. **p53:** Only south PM had effect. Induction of p54 seems to depend on high levels of Cl/Cr/Ni/Cu and low levels of S/K/Ca/Ti/Mn/Fe/Zn/Pb.

Study	Pollutant	Exposure	Effects
Reference: Sakamoto et al. (2007, 096282) **Species:** Human **Age:** 58-82y (Smokers) **Cell Type:** HBEC	PM_{10}: EHC-93 (Obtained from Health Canada, Canata) **Particle Size:** PM_{10}	**Route:** Cell Culture **Dose/Concentration:** 100, 300 and 500 μg/mL **Time to Analysis:** Calcium responses: up to 60min; cytokines: 6 or 24h	**Intracellular [Ca^{2+}]:** [Ca] concentration slowly increased, elevating after 10 and 30 min for 500 and 300 mg/mL, respectively. The response plateaued at 35 min for 500 μg/mL. **Extracellular [Ca^{2+}]:** Starting at 20 min, the removal of extracellular Ca decreased the PM_{10} response significantly. Calcium channel blocker (10μM or 1mM) LaCl3 and (5mM) NiCl2 significantly blocked the PM-induced intracellular Ca. Lacl2 administration (1mM) inhibited the PM-induced Ca^{2+} response in a dose-dependent manner. **Mode of Action:** Intracellular Ca induced by ATP declined more slowly in the cells exposed by PM_{10}. This indicates that PM_{10} blocks Ca clearance via the calcium pumps. **Cytokines:** PM_{10} induced a dose-dependent increase in cytokine mRNA levels and cytokines IL-1β, LIF, IL-8 and GM-CSF. Cytokine expression was unaffected by the reduction of extracellular Ca^{2+} . Preincubation with the calcium chelator reduced responses for IL-1β and IL-8 but not LIF or GM-CSF.
Reference: Salnikow et al. (2004, 087469) **Species:** Human **Cell Line:** 1HAEo-	$FeSO_4$ $FeCl_3$ $NiSO_4$ **Particle Size:** NR	**Route:** Cell Culture **Dose/Concentration:** 0.25mM Fe exposures also contained 60 μg/mL apotransferrin **Time to Analysis:** 24h	**Cytotoxicity:** Both Fe had no effect. $NiCL_2$ caused marginal cytotoxicity (75%). **Hypoxic Stress:** At 20h, $NiSO_4$ (at concentrations of 0.25 or 0.5mM) induced NDRG-1/Cap43 protein production indicating hypoxic stress. DFX and DMOG induced a similar effect. **IL-8:** $NiSO_4$ induced IL-8 time-dependently for up to 48h. At 48h, the increase was 6+ fold. **Coexposure (Ni + Fe) on Fe uptake:** Fe III uptake was greater than Fe II uptake. $NiSO_4$ had no effect. Ni uptake was greater than Fe uptake but was decreased by coexposure to Fe. Coexposure also did not effect hypoxic stress. Coexposure with Fe did reduce Ni-induced IL-8 production.
Reference: Salonen et al. 2004 (2004, 187053) **Species:** Mouse **Cell Type:** RAW 264.7	PM_{10} (urban traffic) Finland (filter sonicated in water and methanol, extracted with methanol) Pooled as winter (W) spring I (SI) or spring II (SII) based on component/time considerations HVLI slit impactor **Particle Size:** PM_{10}: 0.12-10nm	**Route:** ($2x10^6$ cells/well) **Dose/Concentration:** 15,50, 150,500, 1000 μg/mL **Time to Analysis:** 0, 24h	Air quality parameters: Winter and spring I did not differ. SII much lower $PM_{2.5}$ PM_{10} W = 18.6 ± 10.1; SI = 28.0 ± 5.5; SII = 20.3 ± 2.4 $\mu g/m^3$ Metal data equivocal as well as highly variable resuspension rates Total PAHs: W = 303; SI = 233; SII = 204 ng/mg PM_{10} (ppb**nflammation (IL-6, TNF, NO) /Cytotoxicity:** A dose-dependent increase was observed for TNF, IL-6 and NO except for SI. The IL-6 levels, of those particles exposed to SI, decreased at 1000 μg/mL. **TNF, IL-6:** SI = SII > > W > control. **NO production:** W > = SI > = SII **Cell Viability:** W = SI = SII toxic at 500 and 1000 μg/mL **Watersoluble vs Insoluble:** TNF and IL-6 were nearly entirely the result of insoluble components of PM_{10}. Cytotoxicity was driven by both soluble and insoluble components. **Metal Chelation:** The addition of metal chelators did not modify IL-6, TNF or cytotoxicity **LPS inhibitor:** Treatment with the LPS inhibitor eliminated the IL-6 response and, perhaps, slightly reduced the TNF response but not cytotoxicity **Hydroxy radicals:** A dose-dependent induction of hydroxy radicals and induction of hydroxy radical lesions (at 500 and 1000 $\mu g/m^3$) in the calf thymus DNA were observed.

Study	Pollutant	Exposure	Effects
Reference: Samet et al. (2003, 113782) **Species:** Human **Cell Type:** A431 (Epidermoid Cells)	As: NaASO3 V: $VOSO_4$ Zn: $ZnSO_4$ (obtained from Sigma, St. Louis, MO) **Particle Size:** NR	**Route:** Cell Culture **Dose/Concentration:** 500μM **Time to Analysis:** 20, 30 or 90min	**EGFR Dimerization:** Zn, V or As did not induce EGFR dimerization in a cell free system i.e., no direct crosslinking. Zn did not induce dimerization in whole cells either. **Phosphorylation of EGFR:** Zn induced phosphorylation at 3 sites similar to EGF. As and V had no effect. **EGFR Kinase Inhibitor:** While EGF action was blocked, Zn continued to induce phosphorylation and was independent of EGFR kinase activity. **c-Src:** Blocking of c-Src tyrosine kinase (transactivator of phosphorylation) negated all Zn-induced phosphorylation but only had a slight effect on EGF stimulated cells. **ERK1/2 Phosphorylation:** Zn increased levels of ERK1/2. Pretreatment with EFGR kinase inhibitor reduced both Zn and EGF effect. This effect was not blocked by the c-Src blocker.
Reference: Santini et al. (2004, 087879) **Species:** Mouse **Cell Type:** RAW 264.7	DEP: $PM_{2.5}$ Collected adjacent to moderate traffic in Rome, Italy **Particle Size:** $PM_{2.5}$	**Route:** Cell Culture ($2.5 X10^5$ cells/mL) **Dose/Concentration:** 0.01, 0.1, 1.0 μg/mL **Time to Analysis:** 24h	**500 MHz results (no 1 μg/mL):** DEP induced a dose-dependent increase in choline compounds, α- and βgamma- glutamine/glutamate (0.01 $>$ 0.1 μg/mL), lactate, and CH_2, CH_3 moieties of fatty acids. DEP decreased inositol and (phosphoreatinine. **700 MHz results (no 1 μg/mL):** DEP induced similar results, except α-, βgamma-glutamine were dose-dependent. Inositol showed no effect. Taurine slightly increased. Results were confirmed after eliminating biological interferences via perchloric acid. **Growth Curves/Cell Cycle Analyses/Cell Morphology:** DEP had no effect. **Cytokines:** IL-6 levels increased at 0.1 and 1 μg/mL. TNF-α was unaffected.
Reference: Saxena et al. (2003, 096986) **Species:** Mouse **Cell Type:** RAW 264.7	DEP: SRM 1650 CO: Crude Organic Extract of DEP Fractionated into asphaltene (pentane/hexane), saturated hydrocarbon, less polar (aromatic) hydrocarbon, more polar (aromatic) hydrocarbon, resins, residual (resins) **Particle Size:** NR	**Route:** Cell Culture ($2.5 X10^4$ cells/mL) **Dose/Concentration:** DEP, CO 5, 10, 15, 20, 25 μg/mL IFNy 10 ng/mL LPS 1 mg/mL **Time to Analysis:** 1-3d	**Cytotoxicity:** No cytotoxic effects were observed. **NO:** DEP alone induced NO in a dose-dependent manner which peaked after 1d and plateaued for days 2 and 3. IFNy + DEP showed dose- and time-dependency. LPS + DEP showed no effect at 1d, but dose-dependently reduced NO production on day 2 and 3. Addition of BC G eliminated the effect of DEP at 2d but showed a dose-dependent decrease at 3d. **Effectiveness of Particulate Components:** The carbonaceous core of DEP did not affect BC G-stimulated NO production. CO significantly inhibited BC G-stimulated NO production. Study indicated that the extract of aromatic hydrocarbons and resins caused an inhibitory effect in a dose-dependent manner.

Study	Pollutant	Exposure	Effects
Reference: Seagrave et al. (2007, 097549) **Species:** Human **Gender:** Male (3 donors) **Age:** 16, 23yrs **Cell Type:** Differentiated A549	DE: Generated by DE 5500 watt generator using #2 certification oil performed under 5000w load. Emissions diluted to 3 mg/m^3 total particulate matter. **Particle Size:** 0.14 - 0.5 μm	**Route:** Air Liquid Interface **Dose/Concentration:** 8.33 mL/min/well **Time to Analysis:** 3h; 1 or 21h post-exposure	**Particle Deposition:** 140 and 500 nm microspheres demonstrated uniform deposition of approx. 10% **Transepithelial Electric Resistance:** No effect of DE; rather, more effect was observed from air controls **Macromolecular permeability:** DE caused an increase 1-h but returned to control at 21h. **LDH/Cytotoxicity:** DE had a highly variable(donor specific) effect at 1-h and returned to control levels at 21h **Mitochondrial activity (WST):** DE reduced activity at 1-h and possibly increased activity at 21h (high donor-to-donor variability) **MucusLike Substance Excretion:** There was high donor to donor variability; no overall effects were observed. **Alkaline Phosphatase (AP):** DE decreased at 1-h and perhaps increased at 21h **Glutathione:** DE caused a large decrease at 1-h but returned to normal at 21h. **HO-1:** After DE exposure, levels increased but were still lower than air exposed controls **Cytokines:** No differences for IL- 8 or 12, TNFα, GM-GSF, IL-1α, or IFNγ were observed. IL-4 and 6 were decreased upon DE exposure.
Reference: Seagrave et al. (2004, 087470) **Species:** Human **Cell Type:** A549	DPM: SRM2975 (NIST) DPM-O: DPM organic extract (acetone/DCM) CB: Carbon Black (Elftex-12, Cabot) **Particle Size:** CB: 37nm; Stokes diameter 198nm	**Route:** Cell Culture (1x10^5 cells/well) **Dose/Concentration:** 0.03 - 1,000 μg/cm^2 **Time to Analysis:** 0, 18h	**IL-8 release:** DPM increased semi dose-dependently (perhaps steady based on error range) up to 1 μg/cm^2 after which IL-8 declined dose dependently to zero (control = 100%) at 300 and 1000 μg/cm^2. LDH release was steady which indicates no cytotoxicity. **DPM interaction with IL-8:** DPM depletes IL-8 from solution in a dose-dependent manner (cellfree). BSA preincubation reduced the slope of the dose response but not the final result. CB has no effect. DPM-O residuals act identical to DPM. Increasing NaCl concentrations reduced DPMs depletion of IL-8 **Neutrophil responses:** DPM and bound IL-8 together caused a marked aggregation of cells resulting in spindle shapes. DEM or IL-8 alone did not cause this aggregation although DEP did recruit neutrophils

Study	Pollutant	Exposure	Effects
Reference: Seagrave et al. (2003, 054979) **Species:** Human, Rat **Cell Line:** F344/CrlBR (mouse) **Age:** 11 wks (mouse) **Weight:** 250g **Cell Type:** A549, AMs	PM filter collection Collected from diesel or gasoline powered vehicles as follows: BG: Black Smoke Gasoline G30: Normal Emitter gasoline (30F) G: Normal emitter gasoline (72F) HD: High Emitter Diesel D30: current technology diesel (30F) D: current technology diesel (72F) WG: White Smoke Gasoline **Particle Size:** NR	**Route:** Cell Culture ($1x10^5$ cells/well) **Dose/Concentration:** 0.03 – 10,000 $\mu g/cm^2$ **Time to Analysis:** 16-18h	**Cytotoxicity:** LDH activity increased in A549 cells. The types of pollutants that are most toxic, in decreasing order of cytotoxicity, are the following: BG, G30, and G which are significantly different from HD, D30, D, WG which are also significantly different from DS. LDH activity also increased in rat macrophages. G, G30, and BG were the most toxic. HD and D30 were intermediately toxic and D, WG, and DS were the least toxic. In both cell types, gasoline was more cytotoxic than diesel. **Cytokines:** All particle types except DS increased IL-8 levels in A549 though not all increases were statistically significant. Also, many particle samples at high concentrations produced an apparent suppression of IL-8 release. **Alkaline Phosphatase:** G30 and G were more potent than the other particle samples in A549. WG and D30 induced no significant effects. For A549 cells, activity increased at low concentrations and was suppressed at higher concentrations. **Macrophage Peroxide Production:** In rat AMs, peroxide production was often the highest at the lowest concentrations and the lowest production caused by the highest concentrations. D30 followed this trend and induced the highest production as well as the greatest suppression. Using two different statistical methods, D30 $>$6 others which in turn $>$DS. Using the second method D30 and D $>$all other 6. Order of potency between two methods completely different. Authors noted that in vitro potency quite different from in vivo potency (previous paper).
Reference: Seaton. et al. 2005 **Species:** Human **Cell Type:** A549	$PM_{2.5}$ from London PM_{10} from Manchester (positive control) PM from Holland Park, Hampstead and Oxford Circus stations (HP, HR and OC) $PM_{2.5}$ monitoring Holland Park, Hampstead and Oxford Circus PM had a median diameter of 0.4 μm. 80% of the particles had a diameter less than 1 μm. **Particle Size:** $PM_{2.5}$, PM_{10}, Holland Park, Hampstead and Oxford Circus PM had a median diameter of 0.4 μm. 80% of the particles had a diameter less than 1 μm.	**Route:** Cell Culture **Dose/Concentration:** 1 - 100 μg/mL **Time to Analysis:** Cytotoxicity: 24h; IL-8: 8h; Generation of hydroxy radicals: 8h	**Cytotoxicity:** Dust from all three tunnels (Holland Park, Hampstead and Oxford Circus) were able to cause cell death (LDH). The release of LDH indicated a dose-dependent relationship. The highest dose of Holland Park PM induced the $\sim$17% release of LDH, Hampstead triggered $\sim$13% and Oxford Circus $\sim$3% (no different than control). PM_{10} from Manchester caused a 7% LDH release at the highest dose. The negative control (TiO_2) caused no response (2% release at highest dose). **IL-8:** All three tunnel PMs induced a dose-dependent release of IL-8. At the highest dose, all three tunnel dusts induced IL-8 stimulation more so than the control site $PM_{2.5}$. HP induced a 3 fold increase. Also, the highest TiO_2 concentration caused the least IL-8 stimulation. **Hydroxy Radical Generation/ DNA Plasmid assay:** The plasmid assay indicated that the tunnel dusts induce more free radical activity than the Manchester PM_{10} and TiO_2. HP nearly doubled the percentage of DNA damage with intermediate results for HR and OC. Results for PM_{10}, TiO_2 and control were identical

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Reference: Singal et al. 2005 **Species:** Human, Mouse **Cell Type/Line:** A549Luc1 lung adenocarcinoma epithelial cell line (human), MLE15Luc1 and MLE15Luc2 (mouse) All cells contain human cytokine IL-8 controlling firefly luciferase	AE2: Aerosil 200, amorphous silica (Degussa) CI: Carbon iron particles (25 % Fe) **Particle Size:** AE2: 12nm surface area ~ 200 ± 25 m^2/g; CI: ~ 40nm	**Route:** Cell Culture (5x10^5 cells/well) **Dose/Concentration:** 18 μg/mL, 36 μg/mL, 72 μg/mL all in 1mL /well **Time to Analysis:** 24h	**Luc activity:** Luciferase enzyme activity is significantly less in MLE15Luc2 cells than in MLE15Luc1 cells. For both cells, luciferase activity is time- and dose-dependent peaking at 4-8 hours. **Aerosil 200:** AE2 induced dose- and time-dependent Luc response which peaked at 3 h and decreased thereafter in a similar way as TNF. Contrary to TNF, AE2 induced much cytotoxicity starting at 6h. **Effect of proteasomal inhibitors (MG-132):** Inhibitor reduced AE2 Luc activity to near control levels. Similarly, LDH-cytotoxicity was halved **A549 human cell response:** AE2 acted similarly to the MLE response. CI particles showed slightly less activity without peaks. AE2 increased cytotoxicity after 12 h, whereas CI had no effect. Contrary to MLE mouse, MG_132 did not affect Luc activity but PD98059 (selective noncompetitive inhibitor of the MAP pathway) and SN50 (NF- κB inhibitor) reduced AE 2 and CI-induced activity.
Reference: Song et al. (2008, 156093) **Species:** Rat **Cell Type:** RAW 264.7	DEP collected from a 4JB1-type, light-duty (2740 cc), four-cylinder diesel engine operated using standard diesel fuel at speeds of 1500 rpm under a load of 10 torque. **Particle Size:** 0.4 μm (mean diameter)	**Route:** Cell Culture (5 X 10^5 cells seeded on a 24-well plate) **Dose/Concentration:** 50 μg/mL **Time to Analysis:** Nitrate production: 72h.	**Nitrite Production:** 50 μg/mL of DEP induced production when compared to the control. Over the 72-h period, a general trend was not observed, but maximal induction of nitrite occurred at 4 h after stimulation.
Reference: Steerenberg et al. (2006, 088249) **Species:** Rat, Human **Cell Line:** Crl/WKY (rat) **Cell Type:** AMs (rat), alveolar epithelium cells (rat), A549	PMC: PM Coarse PMF: PM fine Ambient air samples collected from Rome, Italy; Oslo, Norway; Lodz, Poland; Amsterdam, the Netherlands; De Zilk, the Netherlands. **Particle Size:** PMC: 2.35 - 8.5 μm; PMF: 0.12 - 2.35 μm	**Route:** Cell Culture **Dose/Concentration:** NR **Time to Analysis:** 20h	Crustal material (metals and endotoxin but not Ti, As, Cd, Zn, V, Ni, Se) were positively associated with on vitro rat macrophage IL-6 and TNFα and in vitro Type 2 (rat alveolar) MP-2 and Il-6. Sea spray (Na and Cl) was also correlated with Macrophage IL-6
Reference: Tal et al. (2006, 108588) **Species:** Human **Cell Type:** HAEC	100 mM Zn(II) or V(IV) stock solutions (NOS) **Particle Size:** NR	**Route:** Cell Culture **Dose/Concentration:** 500 μmol **Time to Analysis:** 5, 20min	**Zn-mediated EGFR phosphorylation:** EGFR kinase activity was required but not EFGR ligand binding. EGFR Kinase inhibition reduced Zn mediated EGFR activation. (authors NOTE: complete reverse of results in B82L and A431 cells). Src Kinase is not required. Zn inhibiting Src kinase was nearly total after 20 min. **EGFR-specific protein tyrase phosphatase (PTP):** Zn inhibited PTPs, similar to V(IV) resulting in a decrease of exogenous EGFR dephosphorylation
Reference: Tamaoki et al. (2004, 157040) **Species:** Human **Cell Type:** HBEC	UFCB: Ultrafine Carbon Black - (Tokai Carbon, Japan) FCB: Fine Carbon Black (Tokai Carbon, Japan) **Particle Size:** UFCB: 11 ± 0.5 nm (mean diameter) FCB: 250 ± 16nm (mean diameter)	**Route:** Cell Culture (10^4 cells/well) **Dose/Concentration:** 6.1, 12.3, 18.4, 24.5, 30.7 μg/cm^2 **Time to Analysis:** Up to 72h	**DNA synthesis/ Protein synthesis:** Synthesis increased by UFCB (30.7) for up to 72 h and flattened after 48 h. FCB had no effect. UFCB also showed a dose-dependent response beginning at 12.3 μg.cm^2 up to 24.5 whereafter the response plateaued. The addition of Cu/Zn Super oxide dismutase (SOD) or a NADPH oxidase inhibitor completely inhibited the UFCB effects. Similarly, two different EGFR tyrosine kinase inhibitors, and a Me inhibitor all reduced UFCB response to control levels. **ERK activation:** UFCB caused phosphorylation of ERK beginning at 2 min, peaking at 5 min and decreasing at 10 min. ERK activation was inhibited by EGFR tyrosine kinase inhibitor Cu/Zn SOD and neutralizing body for HB-EGF but not by PDGF-R kinase inhibitor. **HB (polyclonal heparin binding)-EGF release:** UFCB induced rapid cell surface loss with recovery after 20 min and nearly full recovery at 360 min. Metalloproteinase inhibitor and Cu/Zn SOD both prevented HB-EGF release.

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Reference: Tao and Kobzik (2002, 157044) **Species:** Rat **Cell Type:** RLE-6TN (Alveolar Type II Epithelial Cells), Fetal Lung Fibroblasts (RFL), AMs	UAP: Urban Air Particles = SRM 1649 TiO_2: Titanium dioxide SiO_2 ROFA **Particle Size:** TiO_2: ~1μm; SiO_2: ~1μm; ROFA: NR	**Route:** Cell Cuilture (1x10E5 cells AM 1.4x10E5 cells RLE/RFL) **Dose/Concentration:** 1-50 μg/mL **Time to Analysis:** 24h	**Cytokines:** TNF-α and MIP-2 in RLE was unaffected by any particle samples. TNF-α and MIP-2 in AM significantly increased with 25 μg/mL UAP. TNF-α and MIP-2 in the co-culture of AM + RLE increased with each particle. The order of particles in decreasing order are as follows: SiO_2 at 25μg/mL, UAP at 12.5 μg/mL, ROFA at 25 μg/mL, and TiO_2 at 50 μg/mL. Except for SiO_2, the blocking of effects caused by LPS absorbed on the particles did not affect the cytokine response. For SiO_2, the response was reduced but still above the control. **Co-culture:** Physically separating AM and RLE cells and adding PM completely negated the co-culture's response to PMs. This indicates that cell to cell contact is required for co-culture potentiation of PM effects. **Inhibitors:** Various inhibitors of cell adhesion molecules (heparin, β -1, 2 or 3 integrin) had no effect on UAP-induced cytokine release.
Reference: Veranth et al. (2007, 090346) **Species:** Human **Cell Type:** BEAS-2B, A549, NHBE	Artificial particles and PMs N-Al: nano alumina Al_2O3 M-Al: Micro Al_2O3 N-Ce: nano CeO_2 M-Ce: micro CeO_2 N-Fe: nano Fe_2O3 M-Fe: micro Fe_2O3 N-Ni: nano NiO M-Ni: micro NiO N-Si: nano SiO_2 M-Si: micro SiO_2 N-Ti: nano TiO_2 M-Ti: micro TiO_2 KLN: kaolin MUS: Min-U-Sil (ground crystalline silica) DD: desert rural soil Utah $PM_{2.5}$ JE: Juarez, urban street $PM_{2.5}$ MNC: Mancos, rural Utah $PM_{2.5}$ LPS: lipopolysaccharide V: $VOSO_4$ (soluble) (19 μg/mL) TNFα (0.01 μg/mL) **Particle Size:** Surface mean diameter/ surface N-Al: 6nm (261 m^2/g) M-Al: 210nm (7.7) N-Ce: 14nm (71) M-Ce: 1500nm (0.6) N-Fe: 5nm (221) M-Fe: 100nm (12) N-Ni: 6nm (145) M-Ni: 16nm (57) N-Si: 19nm (127) M-Si: 440nm (5.4) N-Ti: 6nm (242) M-Ti: 410nm (3.5) KLN: 100nm (24.3) MUS (NOS <5 μm) DD: 400nm (6.2) JE (NOS <3 μm) MNC: 200nm (13.0)	**Route:** Cell Culture (35,000 cells/cm^2 BEAS; 2500 cells/cm^2 NHBE; 20,000 cm^2 A549) **Dose/Concentration:** 0.53, 5.3 and 53 μg/cm^2 (= 1, 10, 100 μg/mL) **Time to Analysis:** 24h	**Cell Viability:** Except for Ni and V no cytotoxicity was observed at the highest concentration. **IL-6 secretion in BEAS-2 B cells:** Nano and micro sizes of the same metal showed no differences in response (high experiment to experiment variability). In general, the soil-derived dusts (JE, DD, MNC) were more potent than the metal and ceramic oxide particles. In KGM media, BEAS-2B cells are more responsive to vanadium and other soluble metals and less responsive to LPS, but this relationship is reversed in LHC-9 media. **IL-8 secretion in BEAS/LHC vs NHBE in BEGM cells:** Levels were much higher in NHBE cells than BEAS-2B cells. For BEAS-2B, the nano size Si and both sizes of Ni induced levels statistically greater than the control. For NHBE, only Si and Ni (for both sizes) were statistically greater than control. **IL-6 in NHBE:** The nano and micro sized particles of Al, Ce, Fe and nano sized Si all induced statistically significant increases. Control levels of IL-6 were much higher in NHBE cells than in BEAS-2B cells. Secretion induced by pure oxide particles was small for both the mid and high concentration levels (5.3 and 53 μg/cm^2). **BSA/ Bovine serum addition effect:** In a fixed solution nano-Ni, nano-Ti and KLN all reduced the measured IL-6 by 60+ percent. Addition of BSA or bovine serum dose dependently reduced the action of the particles to near control levels. **PM effects (without added protein) on IL-6 in solution:** Increasing metal concentration did not affect a fixed IL-6 concentrration until the 100 or 316 μg/mL levels.

Study	Pollutant	Exposure	Effects
Reference: Veranth et al. (2007, 090346) **Species:** Human, mouse, rat **Cell Type:** A549, BEAS-2B (types E and U), RAW 264.7, Primary macrophages	S: desert dust (collected from unpaved desert road in Utah, $PM_{2.5}$ enriched) V: vanadium soluble (prepared from $VOSO_4$, Alfa Aesar, Ward Hill, MA)) C: Coal fly ash ($PM_{2.5}$ enriched and derived from commercial power plant burning Utah bituminous coal) D: Diesel PM (tail-pipe particles collected from high emitting black smoker on-road light duty truck) L: Lipopolysaccharide T: Titanium dioxide (Alfa Aesar) K: Kaolin (purchased from Capitol Ceramics, UT) **Particle Size:** BET surface (m^2/g) S: 6.2 ($PM_{2.5}$ enriched) V: NA C: 5.4 ($PM_{2.5}$ enriched) D: NR L: NA T: 3.5 (1-2 μm) K: 24 (< 200 mesh = 74 μm)	**Route:** Cell Culture **Dose/Concentrations:** Maximum concentrations: S = 100 $\mu g/cm^2$ V = 100 $\mu g/cm^2$ C = 100 $\mu g/cm^2$ D = 32 $\mu g/cm^2$ L = 1000 EU/mL T = 100 $\mu g/cm^2$ K = 100 $\mu g/cm^2$ **Time to Analysis:** 24h	**Viability:** Generally, cell viability was greater than 75% of the control post treatment. Vanadium, at the highest concentration, induced less than 50% of control viability whereas kaolin, also at the highest concentration, induced cell death. **Cytokine IL-6:** BEAS-2B E or U in LHC-9 showed a response to S and L. BEAS-2B (U) was in LHC-9 medium with added serum (FBS). This resulted in a doubling of response coupled with at least an 8 fold increase in control levels. BEAS-2B (E) showed response for S and V but not L. A549 showed response to S and K. RAW 264.7 and Rat macrophages showed responses to S(very low) and L. In general, the IL-6 responses in A549 and RAW 264.7 were similar and significantly lower than the responses in rat macrophages or BEAS-2B. **Effect of culture media composition (BEAS-2B):** Varying ratios of LHC-9 and KGM media resulted in a near 10 fold increase in control rate once LHC was 33% or more of the media. Upon Soil Dust (NOS) exposure IL-6 increased linearly with % LHC-9 in culture/exposure media. Addition of calf serum (0.1-10 %) raised control IL-6 levels at least 40 fold. At a steady PM concentration, the addition of serum resulted in a log-linear increase in IL-6 release which blocked any PM effect. **Reversibility of media effect:** Changing media with every passage showed that media effects do not persist once media are changed. **Culture Well Size:** Going from a 6 well to 96 well plate (decreasing well size) increased IL-6 control values about ten fold, while the positive control (TNF) response increased 3 fold. Hence the sensitivity of the test (i.e., positive/control response) declined from 11 fold to 3 fold with increasing well number / decreasing well size. Because cell seeding density and the like were held constant, these changes suggest that edge effects are the cause of the IL-6 changes.
Reference: Veranth et al. (2006, 087479) **Species:** Human **Cell Type:** BEAS-2B	$PM_{2.5}$ samples from 28 samples from 8 locations in Utah, New Mexico and Texas (rural, industrial, road side, military) 2 coal fly ash samples (a product of combustion using Utah bituminous coal and New Mexico bituminous coal) TiO_2 kaolin clay **Particle Size:** $PM_{2.5}$; TiO_2: 1-2 μm	**Route:** Cell Culture (35,000 cells/ cm^2 in KGM media) **Dose/Concentration:** 10, 20, 40, 80 $\mu g/cm^2$ **Time to Analysis:** 24h	**Cell assays:** In sample soils viability declined dose dependently while IL-6 increased dose-dependently. IL-8 was highly variable (peak at 20 μg/l, dose-dependent increase or flat response.) **IL-6 assays for all soil PMs:** Soils ranged across an order of magnitude greater than LPS, coal fly ash, TiO_2 or kaolin samples. One soil even exceeded the pos V control at equal concentrations **Correlation with cell viability:** Correlation was strong for Mn ($p < 0.001$) and weak for EC3, K, Se, and Hg ($0.01 < p < 0.05$). **IL-6 -10 $\mu g/cm^2$:** Correlation was medial for OC-1(Organic Carbon) and P at $0.001 < p < 0.01$. **IL-6 80 $\mu g/cm^2$:** Correlation was strong for OC3, OP (pyrolized Carbon), OC, EC1, TC and intermediate for OC2, OC4, Zn and weak for Ca2+, EC2, Si, Ca, Ca: Al. **IL-8 10 $\mu g/cm^2$:** Correlation was weak for EU (Endotoxin), CO3, Si, and Br. **IL-8 80 $\mu g/cm^2$:** Correlation was medial for CO3, Sr and weak for K+, EC3, Mg, Si. **IL-8 trend (corr over 10-80 range):** Correlation was strong for EC, intermediate for OC4, EC1, EC2, EC3, TC, Ni and weak for OP, OC, Cr, and Sr. IL-6 and Il-8 were not correlated nor were IL-6 and cell viability. Authors noted that weak correlations ($0.01 < p < 0.05$) contained false positives.

Study	Pollutant	Exposure	Effects
Reference: Veranth et al. (2004, 087480) **Species:** Human **Cell Type:** BEAS-2B Veranth et al. 2004	$PM_{2.5}$ enriched soil samples DD: desert dust, unpaved road, Utah WM: West Mesa, sandy grazing site, NM R40: Range 40 gravel soil, TX UN: Uinta, sandy soil, UT **Particle Size:** 0.4 - 3μm	**Route:** Cell Culture (20,000/cm^2) **Dose/Concentration:** 10, 20, 40, 80, 160 μg/cm^2 **Time to Analysis:** 24h	**Elemental Analysis of PM's:** Major differences UN generally lower in major minerals but high Fe content and high EC. High Mn. Low Pb and Zn **Cytotoxicity:** UN and WM were the most cytotoxic at all dose levels, followed by R40 and DD. All particles showed a dose-dependent cytotoxic response. **IL-6 Release:** DD and R40 (up to the 160 μg/cm^2) showed dose-dependent responses and induced an 8-fold increase at the highest concentration levels. WM peaked at 40 μg/cm^2 and UN induced similar responses above 10 μg/cm^2. **IL- 8 release:** DD induced a dose-dependent response. WM peaked at 10 μg/cm^2. Release induced by DD and WM seemed to be limited by toxicity. There was no treatment with R40. **TNF**-α: DD, WM and UN induced release was not detected at the 40 or 80 μg/cm^2 concentrations. **LPS:** LPS was the primary factor in inducing IL-6 release when exposed to LPS-containing mixtures. LPS alone induced lesser responses than treatment to the environmental dust particles. TiLPS induced a less than two-fold increase in IL-6 versus the over seven-fold increase induced by soil dust positive control. LPS treatments were less cytotoxic than DD. Limited IL-6 and IL-8 responses were observed at 2000 EU/mL compared with DD at 80 μg/cm^2 **Endotoxin:** Inverse relationship between endotoxin content and IL-6 release was observed. **Viability vs Physical Modification of Dust Sample (no UN):** Only leaching in a variety of water based vehicles increased viability minimally (generally $<$ 25 %). Heat treatment (150-, 300, 550F) and methanol extraction had no effect **IL-6 release vs Physical Modification of Dust Sample (no UN):** One hour thermal treatment at 150□ F had no effect on IL-6 response. All other treatments reduced IL-6 release (heat 350□, 500□ and extractions).
Reference: Veronesi et al. (2002, 024599) **Species:** Human **Cell Type:** BEAS-2B	Ambient PM - St. Louis: Urban particulates - Ottawa: Urban particulates -MSH: Volcanic dust from Washington state's Mt. St. Helen -Woodstove: Woodstove particles from conventional fireplace burner -CFA: Coal fly ash from western U.S. powerplant -OFA: Oil fly ash from Niagara, NY - A: Total Fractions - B: Soluble Fractions - C: Washed Fractions **Particle Size:** PM $>$ 2.5μm; PM: 2-10 μm; PM $>$ 10 μm	**Route:** Cell Culture **Dose/Concentration:** 50 μg/mL; 30 μg/cm^2 100μg/mL; 60μg/cm^2 **Time to Analysis:** 4, 16h	**Ca:** Calcium increased significantly with all particles types. **IL-6:** At 50 and 100 μg/mL, IL-6 increased with all particle types at 4 and 16h. Overall, fraction -A was the most potent. **Surface charge:** Surface charge correlated strongly with increases in both Ca2+ and IL-6 levels. OFA, however, was unmeasurable due to technical difficulties.

Study	Pollutant	Exposure	Effects
Reference: Vogel et al. (2005, 087891) **Species:** Human **Cell Type:** U937 (ATCC) monocytes (macrophage differentiation)	UDP: SRM 1649 (NIST) UDP-OE: DCM extract of SRM-1649, 0.45 μm filter sUDP: stripped particles UDP DEP: SRM 2975 (NIST) DEP-OE: DCM extract of SRM-2975, 0.45 μm filter sDEP: stripped particles DEP CB95: Carbon Black (Degussa) **Particle Size:** UDP, DEP: NR; CB95: 95nm	**Route:** Cell Culture (2 X10^5 - 2 X10^6 cells/mL) **Dose/Concentration:** DEP, UDP: 2.5, 10 or 40 μg/cm^2 (eq to 12.5, 40, 200 μg/mL) DEP-OE, UDP- OE: 10 μg/cm^2 (particle equivalent) **Time to Analysis:** 24h	**Effect on mRNA expression (COX-2, TNFα, IL-6, IL-8, C/EBPβ, CRP, CYP1a1):** All DEP and UDP induced dose-dependent increases. IL-6 tended to plateau at 10 μg/cm^2. Generally, with the exception of COX-2, UDP effects on genes were stronger than DEP. **Cytotoxicity:** Both DEP and UDP were cytotoxic at 40 μg/cm^2 **Fractionation and mRNA expression:** For COX-2, TNFα, IL-8 mRNA fractions were much more active than parent particles and consequently stripped particles were much less active than parent particles. CB95 had no effect. The reverse effect occurred for IL-6 and CRP mRNA expression. The particles that induced mRNA expression in decreasing order are: sUDP, UDP, UDP-OE. **Inhibition of mRNA expression:** CRP: pretreatment with IgG and wortmannin (Fcγ receptor binding and ingestion dependent inhibitors resp) blocked the effects of DEP, UDP and sDEP and sUDP. Luteolin (AhR inhibitor) had no effect. **COX-2:** Only luteolin inhibited COX-2 expression for DEP, DEP-OE, UDP, and UDP-OE. **CYP1a1:** Luteolin also inhibited OE-DEP and OE-DUP effects (only those two particles tested). **Cholesterol accumulation:** DEP, UDP and UDP-OE and DEP-OE at 10 μg/cm^2 all increased cholesterol accumulation by at least 2 fold
Reference: Wang et al. (2003, 157106) **Species:** Rat **Cell Type:** Lung Myofibroblasts	V_2O_5: (Aldrich Chemical Co., Wisconsin) **Particle Size:** NR	**Route:** Cell Culture (1 X10^5 cells/100 mm dish; 3.2 X10^4 cells/cm^2) **Dose/Concentration:** 400 μm **Time to Analysis:** 0.5, 1, 4, 24h	**H_2O_2 drives STAT-1 activation:** Pretreatment with NAC or catalase reduced V_2O_5–induced STAT activation by more than 90% and completely abolished H_2O_2–induced STAT activation. Within 5 min of V_2O_5 treatment, H_2O_2 was significantly decreased in the supernatants of cultured myofibroblasts and suppression of H_2O_2 levels continued for up to 24h post V_2O_5 treatment. This supports the findings that myofibroblast-generated H_2O_2 is required for V_2O_5–induced STAT activation. **Temporal STAT-1 activation:** H_2O_2 induced rapid activation within minutes whereas activation by V_2O_5 occurred more slowly (beginning 8h post treatment). **p38, ERK, EGFR:** p38 and EGFR are required for H_2O_2- or V_2O_5–induced STAT-1 activation whereas ERK is not required
Reference: Whitekus et al. (2002, 157142) **Species:** Mouse **Cell Line:** RAW 264.7	DEP (light-duty, four-cylinder engine- 4JB1 type, Isuzu Automobile, Japan; standard diesel fuel) (extracts) **Particle Size:** NR	**Route:** Cell Culture **Dose/Concentration:** 50 μg/mL **Time to Analysis:** DEP extracts suspended in methanol and sonicated 20min. Centrifuged 10min. Dried DEP resuspended and stored -20ºC. Cell cultures maintained 37 ºC. Exposed to antioxidants 5h. HO-1 western blot, determination of cellular GSH:GSSG ratios, carbonyl protein content, lipid hydroperoxides performed.	DEP significantly reduced the GSH:GSSG ratio. This effect was prevented by adding thiol antioxidants NAC or BUC. DEP increased lipid peroxide levels, but the addition of all antioxidants decreased these levels. DEP increased carbonyl groups. NAC, BUC, and luteolin reduced HO-1 expression.

Study	Pollutant	Exposure	Effects
Reference: Wilson et al. (2007, 097268) **Species:** Mouse **Cell Type:** J774	CB: Carbon Black, Printex 90 (Degussa) $FeCl_3$ ZnCl2 **Particle Size:** CB: 14 nm	**Route:** Cell Culture (4 $X10^5$ cells/mL at 1mL/well) **Dose/Concentration:** CB 1.9 -31 μg/mL; $FeCl_3$, ZnCl2 0.01 - 100 μmo **Time to Analysis:** 4h	**ROS production in cells:** CB alone increased ROS. Coexposure with ZnCl2 did not affect ROS. **ROS production - cell free:** CB induced a significant increase in ROS. ZnCl2 had no effect. Coexposure CB/Zn also had no effect. **TNFα production (Fe -Zn 0.01-100 μmol):** Coexposure of CB over a range of metals gave no change over CB alone for Fe. For Zn, only at the concentration of 100 μmol was there a small interaction between Zn and CB. Similar results were seen at metal concentrations between 20 -100 μmol. Synergism was observed between Zn and CB and no observed effect of Fe. **Macrophage cytoskeleton:** CB resulted in black vacuoles. Co-treatment of cells with Zn and CB increased the severity of Zn effects. Fe exhibited no synergism. **Apoptosis /necrosis:** No synergism of CB with either Fe or Zn **Phagocytosis:** Only at 31 μmol CB and 50 μmol Zn did a synergistic effect occur; it resulted in a 4-fold reduction
Reference: Wottrich et al. (2004, 094518) **Species:** Human **Cell Type:** A549, THP-1, Mono Mac 6	Fe: hematite α-Fe_2O3 Si60: silicasol (SiO_2, amorphous silica) Si100: silicasol Q: crystalline quartz DQ12 **Particle Size:** Fe: 50 - 90nm; Si60: 60nm; Si100: 80 -110 nm; Q < 5 μm	**Route:** Cell Culture (2 $X10^4$ cells/well. Co-culture: 2 $X10^4$ A549 and 2 $X10^3$ Macrophages) **Dose/Concentration:** A549 light microscopy hematite 100μg/mL (23 $\mu g/cm^2$) TEM hematite 50 μg/mL (16 $\mu g/cm^2$) Cytotoxicity 10, 50, 100 and 200 μg/mL (6.1, 30, 61 and 121 $\mu g/cm^2$) Cytokines 50 and 200 μg/mL **Time to Analysis:** 24 h	**Particle Uptake:** Hematite agglomeration was observed in all 3 cell lines. TEM confirmed cytosol aggregates as well as single particles, which includes particles transported intracellularly to basolateral membrane of epithelial cells. **Cytotoxicity:** LDH increased significantly in A549. In decreasing order, Q , Fe, S60, and S100 (which exhibited levels similar to controls) all induced cytotoxicity. THP-1 cells appeared the most sensitive with Q, Fe, S60, S100, control inducing cytotoxicity in decreasing order. Mono Mac 6 cells were the least sensitive with Fe, S60, Q, S100. **Cytokines:** IL-6 and IL-8 released from A549 cells upon exposure to all particles. No response was observed in Mono Mac 6 or in THP-1 cells. **Co-cultures:** Mix of A549 with either Mono Mac 6 or THP-1 led to a large (ten fold) increase in response to particles. Ten fold increases were observed in IL-6 and IL-8 levels with the Mono Mac 6 co-culture and the THP-1 co-culture, respectively.

Study	Pollutant	Exposure	Effects
Reference: Wu et al. (2007, 098412) **Species:** Human **Cell Line:** B82L **Cell Type:** B82L- par (parental fibroblasts), B82L-wt (wild type EGFR), B82L-K721M (kinase defective EGFR), B82L-c'958 (COOH-terminally truncated EGFR at Tyr-958)	$ZnSO_4$ (Sigma) **Particle Size:** NR	**Route:** Cell Culture **Dose/Concentration:** Zn: 500 μmol EGF: 100ng/mL **Time to Analysis:** 20min	**EGFR mutations:** EGFR-wt has a functional tyrosine kinase domain,intact Src phosphorylation (Tyr 845) and 5 tyrosine autophosphorylation sites. EGFR-c'958 lacks all 5 tyrosine autophosphorylation sites. EGFR-K721M lacks tyrosine kinase (ATP binding). EGFR-Y845F lacks Src autophosphorylation (Tyr 845) and, instead, has a receptor at Tyr 845 that is phosphorylated by nonreceptor Tyrosine kinase Src. **Zn Induced Ras (MAPK signaling protein):** No effect was observed in B82L-par cells. Zn had an effect in -wt, -c'958, and -K721M which confirms the need for EGFR. This indicates that neither tyrosine kinase nor autophosphorylation sites were required for Zn effects. No observed increase for Y845F indicated that EGFR tyrosine 845 (phosphorylated by c-Src) is required for Zn effects. However, it was not required for EGF effects. **Src Kinase requirement:** Using a Src blocker drastically reduced Zn effect but not the EGF effect. Src activation occurred independent of EGFR Tyr-845. **Zn induced association of EGFR with Src:** Zn induced a physical association in all 4 mutants; EGF did not. **Zn induced phosphorylation of EGFR at Tyr-845:** Zn induced phosporylation of EGFR at Tyr-845 in B82L-wt,-c'958 and -K721M. EGF exhibited similar effects. Src blockers significantly reduced phosphorylation induced by Zn but not for EGF. Neither Zn or EGF induced phosphorylation in B82L-Y845F cells.
Reference: Wu, W. Wang, X. Zhang, W. 2003 **Species:** Human **Cell Type:** BEAS-2B	Zinc Ion: Zn^{2+} **Particle Size:** NR	**Route:** Cell Culture **Dose/Concentration:** 10, 25, 50 μmol **Time to Analysis:** 0-8h	**Cytotoxicity:** Exposure to 50 μmol Zn2+ for 8h did not result in significant alterations in cell viability. **PTEN Protein Levels:** 50 μmol Zn2+ for 4 and 8h significantly decreased levels in a dose-dependent manner. Exposure to 50μM vanadyl sulfate (tyrosine phosphatase inhibitor) had minimal effects on PTEN. 100 ng/mL of non-specified EGF receptor ligand for 1-8h did not exhibit any significant effects on PTEN levels. **P13K/Akt:** Zinc induced Akt activation in a dose- and time- dependent fashion. Active Akt levels were the highest at 1 h post exposure to Zn^{2+}, corresponding with the time period when there was a minimal effect on PTEN protein level. When treated with LY294002 (inhibitor of P13K activity), Akt phosphorylation was significantly inhibited. **PTEN mRNA Levels:** Decreased PTEN mRNA expression was observed in cells exposed to 50 μmol Zn2+ for 8h whereas PTEN protein levels declined as early as 4h. **Proteasome-mediated PTEN Degradation:** Use of MG132 (proteasome inhibitor) had no significant effect on Zn2+ induced PTEN mRNA expression. Therefore mRNA expression may not play a critical role in PTEN protein reduction. Instead data suggested that 26 S proteasome played a vital role in Zn2+ induced PTEN degradation. PI3K inhibitor blocked Zn-induced PTEN degradation, but failed to prevent significant Zn-induced down-regulation of PTEN mRNA.

Study	Pollutant	Exposure	Effects
Reference: Wu et al. (2004, 096949) **Species:** Human **Cell Type:** NHBE	Zinc Ion: Zn^{2+} **Particle Size:** NR	**Route:** Cell Culture **Dose/Concentration:** 100 μmol **Time to Analysis:** 2h	**Cell Viability:** After 2 h of exposure, Zn^{2+} induced effects in NHBE cells at 100 and 200 μmol levels (but not 50 μmol). Continuing exposure to 100 μmol Zn2+ for 4 and 6 h did not significantly alter cell viability. Thus, in all subsequent studies, NHBE cells were treated with 100 μmol Zn^{2+}. **Induced EGFR Phosphorylation:** Exposure to 100μM Zn2+ for 1-4 h induced phosphorylation of EGFR in NHBE cells. EGFR kinase inhibitor PD153035 (to determine ifphosphorylation of EGFR was the result of autophosphorylation of activated EGFR tyrosine kinase activity) caused Zn2+ -induced phosphorylation to subside. Zn2+ activity requires tyrosine kinase activity. **EGFR Phosphorylation Pathway:** To test whether Zn exposure results in ligand release, which in turn can activate phosphorylation, NHBE cells were pretreated with LA1 blocking antibody. Results showed significant suppression of Zn2+ induced phosphorylation, therefore Zn2+ phosphorylation might be initiated by the release of EGFR ligands. **HB-EGF, TGF-α , EGF:** To examine the involvement of specific ligands (HB-EGF. TGF-α and EGF) in the phosphorylation pathway, cells were exposed to anti-HB-EGF, anti-TGF-α and anti-EGF. Results showed that anti-HB-EGF reduced Zn2+ induced phosphorylation significantly, anti-TGF-α produced partial inhibition and anti-EGF had no inhibitory effect. Exposure with blocking antibody LA1 was tested to determine if it caused an increase in soluble HB-EGF. HB-EGF mRNA expression was also elevated in cells exposed to Zn^{2+}. Previous studies indicate metalloproteinase (MMP) involvement in cleaving ligand precursors. It was found that MMP-3 inhibitor partially blocks Zn^{2+} induced HB-EGF release. (MMP-2 and MMP-9 did not show similar inhibition patterns) Zn^{2+} exposure increased the release of MMP-3 from HNBE cells.

Study	Pollutant	Exposure	Effects
Reference: Wu et al. (2005, 097350) **Species:** Human **Cell Line:** Subclone S6 **Cell Type:** BEAS-2B	Zinc Ion: Zn^{2+} **Particle Size:** NR	**Route:** Cell Culture **Dose/Concentration:** 50 μmol Zn^{2+} **Time to Analysis:** 4 or 8h; EGFR phosphorylation: 30, 60, 120, 240 min	**Cell viability:** Exposure to 50 μmol Zn2+ for 8 h did not result in significant alterations in cell viability (assessed by LDH release). **P13K/Akt signaling pathway:** To evaluate P13K's on COX-2 Zn2±induced expression, LY-294002 (a P13 inhibitor) and another unnamed P13 inhibitor were used. Exposed cells indicated suppressed levels of Zn2+ induced COX-2. To determine Akt role, ad-DN-Akt(AAA) was used. Infected cells indicated over-expression of Akt and significant reduction of Zn2+ induced GSK-3α/β phosphorylation. Over expression of DN-Akt(AAA) blocked Zn2+ induced COX-2 expression. **PTEN's role in blocking Zn2±induced COX-2 mRNA expression:** PTEN is an antagonist of P13/Akt pathway. Overexpression of wildtype PTEN blocked Zn2+ induced mRNA COX-2 expression, suggesting PTEN inhibits PIP3 signal transduction to Akt. **Analysis of the Src/EGFR signaling pathway:** Zn2+ induced a time-dependent increase in Src and EGFR phosphorylation in cells. Blockage of Src activity via PP2 (Src inhibitor) decreased Zn2+ induced EGFR phosphorylation. The EGFR tyrosine inhibitor completely blocked Zn2+ induced EGFR phosphorylation. EGF (a ligand of EGFR signaling) induced COX-2 expression, suggesting that EGFR regulated Zn2+ -induced COX-2 expression. **p-38 and EGFR kinase activity:** Use of PD-153035 (EGFR inhibitor) and PP2 (Src inhibitor) and SB-203580 (p38 inhibitor) all blocked Zn2+ induced Akt phosphorylation of Src., EGFR and p38. It is thought that p38 is a critical kinase in regulation of Zn2±induced COX-2 protein expression.
Reference: Yacobi et al. (2007, 156166) **Species:** Rat **Cell Type:** L2 (Lung epithelial cells)	PNP: Polystyrene nanoparticles, negatively charged (Molecular Probes, Eugene, OR) PNPA: Amidine modified PNP, positively charged SWCNT: Single-wall carbon nanotubes (Carbon Nanotech, Houston, TX) QDC: Chitosan coated (CdSe/ZnS) Quantum dots, positively charged (made) QDA: Alginate coated QD, negatively charged UAPS: Ultrafine Ambient particulate suspensions (VACES) (48 % organic carbon) **Particle Size:** PNP20: 20nm; PNP100: 100 μm; SWCNT: 0.8-1.2nm (diameter); SWCNT: 100-1000nm; QD: 30nm; UAPS: < 150nm	**Route:** Cell Culture ($1.2x10^6$ cells/cm^2) **Dose/Concentration:** PNP up to 706 μg/mL QD up to 176 μg/mL SWCNT up to 88 μg/ mL UAPS up to 36 μg/mL **Time to Analysis:** on days 4,5 or 6 by replacing monolayer apical fluid with PM in suspension for up to 1440min. Intermediate measurements at 15, 30, 60, 120, 240 and 1440min	**UAPS and Rt (transmonolayer resistance):** Rt declined up to 60 % within 1 h at 36 μg/mL. Rt plateaued (or exhibited a very slight upgradient) for up to 24 h (last measurement). No cytotoxicity was observed. Replacement of apical fluid with fresh media after 2 h of exposure restored Rt to near control values within 24 h. **UAPS and Ieq (short-circuit current):** Peak decline of 30 % after 4 h followed by gradual recovery over 24 h. Replacing media after 2 h exposure returned Ieq to control values within 24 h. **UAPS and apparent permeability:** Permeability measured via C14 mannitol and inulin showed no effect of UAPS **QD and Rt:** QD depressed Rt by nearly 55 % at 4h for positively charged and 30 % for negatively charged QDs. Recovery towards control values started at 4 h and was near complete at 24 h **SWCNT and Rt:** SWCNT depressed Rt by ~ 40 % at 1h (same for 22,44 and 88 μg/mL). Recovery was near complete at 4 h and complete at 24 h. **PNP and Rt:** No statistically significant effects were observed.
Reference: Yun et al. (2005, 088302) **Species:** Human **Cell Type:** A549	DEP: Collected using a 6 cyl 11L, heavy duty (2001 yr) bus engine (South Korea) **Particle Size:** NR	**Route:** Cell Culture (3x10E4 cells/well) **Dose/Concentration:** 1, 10, 100, 250, 500 and 1000 μg/mL from a 1000mg/mL stock; main testing 250 μg/mL **Time to Analysis:** 12h	**NF-κB transcription activation:** DEP induced dose-dependent activity up to 250 μg/mL. After peaking at 250 μg/mL, concentrations above 250 induced dose- dependent declines. Activity peaked at 12-h for 250 μg/mL and declined to control at 24 or 48 h. The mechanism of DEP action was the degradation of IκBα which is an intracellular inhibitor of nuclear translocation of NF-κB. **TAK1 and NIK required for NF-κB activation by DEP:** Dominant negative mutants of TAK1 and NIK reduced DEP induced response to basal level. TAK1 was phosphorylated after DEP exposure and was sustained for at least 90 min.

Study	Pollutant	Exposure	Effects
Reference: Zhang et al. (2007, 156179) **Species:** Human, Rat **Cell Type:** A549, RLE-6TN (Alveolar Type II Epithelial Cells)	$PM_{2.5}$: Collected by baghouse from Dusseldorf, Germany Particle Characterization: Carbon 20%, Hydrogen 1.4%, Nitrogen < 0.5%, Oxygen 14.1%, Sulfur 2.1%, Ash 63.2%. **Particle Size:** $PM_{2.5}$	**Route:** Cell Culture **Dose/Concentration:** 100 $\mu g/cm^2$ **Time to Analysis:** 24h	**Apoptosis:** At 100 μg/mL for 24 h, PM induced a 2.5 fold increase in apoptosis in A549. **Mitochondrial Membrane Potential:** A significant reduction in AEC mitochondrial membrane potential was observed. **Caspase -3 & -9:** Increased activity of both enzymes in both cell types was observed. More specifically, a 2- to 2.5-fold increase of caspase -3 and -9 in A549 and an 8-fold increase of caspase-9 and 4-fold increase of caspase-3 in RLE-6TN were observed. **BIM:** Downregulation of BIM by RNA interference inhibited PM-induced apoptosis. An inhibited decrease in mitochondrial membrane potential and activation of both caspases were observed.
Reference: Zhang et al. (2004, 157183) **Species:** Mice **Cell Line/Type:** C10 (alveolar type II-like epithelial cell line)	DEP: SRM 1650a **Particle Size:** NR	**Route:** Cell Culture **Dose/Concentration:** 5 or 25 μg/mL **Time to Analysis:** 30-360min	**fra expression:** DEP induces fra-1 but not fra-2 expression. mRNA induction peaks around 180 min DEP affects fra-1 mRNA expression at the transcriptional level. **ERK/JNK/p38 MAPK signaling pathways:** 3 inhibitors (PD-98059, SB-202190 or SP-600125) all reduced DEP stimulated fra-1 induction to near control levels. DEP stimulated phosphorylation of the MAPKs which peaks at 60 min but stays elevated at 180 min. **MMP-9 promoter activity:** fra-1 upregulation may play a role in DEP induced increases in MMP-9 promoter activity as fra-1 appears to bind at the -79 TRE sequence of the MMP-9 promoter. **Study 6203 cell lines:** BEAS-2B (A) HR. Bronch. Epith ATCC # CRL-9609 passage 44 BEAS-2B (E) HR. bronch. Epith. U.S. EPA passage 76-87 BEAS-2C (U) HR. bronch. Epith U. Utah sample passage 89-97 A 549 HR. alveolar epith U. Utah sample starts at passage 84 RAW 264.7 M. macrophage ATCC # TIB-71 1 macrophages Rat lung lavage primary cells Study 6203 culture media LHC-9 Lechner and Laveck medium Invitrogen KGM Keratinocyte growth medium Lonza DMEM/F12 50 % Dulbecco's modification of Eagle's medium, 50% Ham's F12 medium Gibco FBS Fetal bovine serum (added to media formulations) Invitrogen

Table D-3. Respiratory effects: in vivo studies.

Reference	Pollutant	Exposure	Effects
Reference: Adamson et al. (2003, 087943) **Species:** Rat **Gender:** Male **Strain:** Sprague-Dawley **Weight:** 150g	PM_{10}: EHC-93W (whole dust) EHC-93S (soluble) EHC-93L (leached) EHC-2KW, -S, -L Measured components Zn, Mg, Pb, Fe, Cu, Al **Particle Size:** EHC-93W, -93S, -93L, -2KW, -2KS, -2KL: PM_{10}	**Route:** IT Instillation **Dose/Concentration:** 5 mg/rat; 33.3 mg/kg **Time to Analysis:** 4h, 1d, 3d, 7d, 14d	**BALF Cells:** The greatest increase in cell numbers was observed with EHC-93W. Activity peaked at 1d with a return to normal levels by 7d. EHC-93L also induced an increase in cell numbers, more so than EHC-93S, but both particles induced statistically significant increases. However, these increases were mostly attributable to an increase in the AM and PMN populations. **BALF Inflammatory/Injury Markers:** Metallo-proteinase (MMP) 2 and 9 both increased, peaking at 1d and 4h respectively. MMP2 activity appears related to the soluble fraction whereas MMP-9 activity appears to be related to the leachable fraction.

Reference	Pollutant	Exposure	Effects
Reference: Ahn et al. (2008, 156199) **Species:** Mouse **Gender:** Male **Strain:** BALB/C1 **Age:** 6wks **Weight:** 19-24g	DEP: Collected using a turbo-charged, intercooler, 6-cylinder, heavy-duty, diesel engine (model year 2000 DPBS: control **Particle Size:** NR	**Route:** Oropharyngeal Aspiration **Dose/Concentration:** 1, 10, 25 mg/kg per day; Those receiving 25 mg/kg DEP also received pre-treatment of Dex (1, 5 mg/kg) 1h prior **Time to Analysis:** 5 consecutive days; 72h post final exposure	**BALF Inflammatory/Injury Markers:** Lung injury was more severe in mice exposed to 25 mg/kg of DEP than when compared to mice exposed to 1 mg/kg DEP. However, lung injury caused by exposure to 25 mg/kg DEP could be completely prevented with pre-treatment of 5mg/kg Dex. Treatment with 1 mg/kg Dex prior to exposure to 25 mg/kg DEP depicted partial reduction in lung injury. **BALF Cells:** Treatment with DEP over a 5d period caused an increase in total number of cells (macrophages, neutrophils and lymphocytes) when compared to control. Total Cells: Control - 5.33 ± 0.44 cells 1mg/kg DEP - 6.26±0.87 cells 10mg/kg DEP - 14.40± 1.90 cells 25 mg/kg DEP - 47.20 ±3.40 cells **COX-2 Expression:** Exposure to DEP lead to a dose-dependent increase in COX-2 levels; specifically, treatment with 25 mg/kg significantly increased COX-2 levels. This effect was completely reduced by treatment with 5mg/kg of Dex.
Reference: Ahsan et al. (2005, 156200) **Species:** Mouse **Gender:** Male and Female **Strains:** hTrx-1-transgenic and C57BL/6 (control) **Age:** 8-8.5wks	DEP: Obtained from Dr. Masaru Sagai (Amori, Japan) **Particle Size:** NR	**Route:** Intratrachael Instillation **Dose/Concentration:** Lung Damage: 0.1 mg/mouse; Survival Analysis: 0.2 mg/mouse; ESR: 0.05 mg/mouse **Time to Analysis:** 24h	**ESR:** hTrx-1 induced 0.05 mg generation of hydroxyl radicals in the lungs (mid thorax ESR spectra) compared to control. **BALF Inflammatory/Injury Markers:** hTrx-1 attenuated cellular damage from 0.1mg DEP. Control mice showed massive edema with neutrophilic infiltration, hemorrhagic alveolar damage and collapsed air spaces. hTrx-1 mice showed mild/moderate edema with clear demarcation of air spaces. **Viability:** After 4, 12 and 24 h, survival was 32, 24 and 12% respectively as compared to 80, 52 and 40% for hTrx-1 mice.
Reference: Andre et al. (2006, 091376) **Species:** Mouse **Gender:** Female **Strain:** BALB/cJ **Age:** 10-12 wks	UFCP: Ultra Fine Carbon Particles (electric spark generator, Model GFG 1000; Palas, Karlsruhe, Germany) Measured Component: UFCP > 96% elemental carbon **Particle Size:** 49nm	**Route:** Whole-body Inhalation **Dose/Concentration:** 380 $\mu g/m^3$ **Time to Analysis:** 4 and 24h; 0 and 24h post-exposure	**BALF Cells:** A small increase in PMN number suggests a minor inflammatory response after 24 h exposure. Number of macrophages did not increase. **BALF Inflammatory/Injury Markers:** Total protein concentration significantly increased post 24 h inhalation. Post 4h, heat shock proteins were induced. Post 24 h, immunomodulatory proteins (osteopontin, galectin-3 and lipocalin-2) significantly increased in alveolar macrophages and septal cells. 236 (1.9%) genes was increased and 307 (2.5%) genes were decreased with upregulated genes being primarily related to the inflammatory process.
Reference: Antonini et al. (2004, 097199) **Species:** Rats **Gender:** Male **Strain:** Sprague-Dawley **Weight:** ~ 250g	ROFA-P: Precipitator -S: Soluble (0.22 μm filter), Components: Fe, Al, Ni, Ca, Mg, Zn -I: insoluble, Components: Fe, Al, Ni, Ca, Mg, Zn, V -T: total ROFA-AH: Air Heater -S: Soluble (0.22 μm filter), Components: Fe, V, Ni, AL -I: Insoluble, Components: Fe, V, Ni, AL -T: Total **Particle Size:** mean diameter: < 3 μm	**Route:** IT Instillation **Dose/Concentration:** 1mg/100g bw in 300 μl saline; 60mg/kg **Time to Analysis:** 24h; Clearance Experiment: two single exposures day 0 and 3 observed at day 6, 8 and 10	**ESR:** Only ROFA-P contained free radicals, primarily in ROFA-P-S. **BALF Cells:** No effects on alveolar macrophages were observed, but all ROFA-P fractions increased lung neutrophils. ROFA-P-S and ROFA-P-I effects combined roughly equaled ROFA-P-T. **BALF Inflammatory/Injury Markers:** ROFA-AH-T and ROFA-AH-I increased LDH. ROFA-P and -AH increased albumin for T and I fractions. **Pulmonary Clearance (Listeria Monocytogenes):** ROFA-P-T and ROFA-P-S significantly slowed bacteria clearance from lungs. ROFA-AH and ROFA-P-I had no effect.

Reference	Pollutant	Exposure	Effects
Reference: Arimoto et al. (2007, 097973) **Species:** Mouse **Strain:** ICR **Gender:** Male **Age:** 6wks **Weight:** 29-33g	DEP (collected using a 4JB1 4-cyl, 2.74L Isuzu diesel engine) DEP-OC: organic chemical extracts LPS DL = DEP + LPS DOL = DEP-OC + LPS **Particle Size:** 0.4μm	**Route:** IT Instillation **Dose/Concentration:** DEP or DEP-OC: 4mg/kg; LPS: 2.5 mg/kg; DL or DOL: NR **Time to Analysis:** 24h	**Cytokines:** DEP-OC or DEP alone did not change levels of MIP-1α, MCP-1 or MIP-2. DL induced significant increases in MIP-1, MIP-2 and MCP-1. **LPS:** LPS and DOL induced increases in MCP-1 though the increase induced by DL was greater. No effect on MIP-1α or MIP-2 was observed.
Reference: Bachoual et al. (2007, 155667) **Species:** Mouse **Strain:** C5B17 **Gender:** Male **Age:** 7wks **Weight:** 22.3 ± 0.73g	RER: PM_{10} Paris, France subway CB TiO_2 DEP **Particle Size:** RER: 79% < 0.5 μm; 20%: 0.5-1 μm CB: 95nm TiO_2: 150 μm DEP: NR	**Route:** IT Instillation **Dose/Concentration:** 5, 50, 100 μg/mouse, 0.22, 2.2, 4.5 mg/kg **Time to Analysis:** 8 or 24h	**BALF Cells:** 100 μg RER and 100 μg DEP increased total cell count and neutrophil influx after 8h and returned to normal by 24 h. Smaller doses of RER and DEP induced no effect. CB induced no effect. **BALF Inflammatory/Injury Markers:** 100 μg RER increased BALF protein after 8 h. No effect was observed after 24 h nor with smaller doses of PM. RER significantly increased MMP-12 mRNA level after 8h and HO-1 total lung mRNA content. No effects on MMP-2 or -9 or TIMP-1 or -2 expression were observed. No effects from CB or DEP were observed. **Cytokines:** 100 μg RER increased BAL, TNF-α and MIP-2 protein content after 8 h.
Reference: Becher et al. (2007, 097125) **Species:** Mouse **Strain:** Crl/Wky (iNOS(-/-)) and C57Bl/6, **Gender:** Male **Age:** 8-14wks **Weight:** 25g	Suspended PM: SRM-1648 **Particle Size:** NR	**Route:** IT Instillation **Dose/Concentration:** 1.6 μg/lung; 64 mg/kg **Time to Analysis:** 20h	**Cytokines:** In both wild and KO strains, all particles caused increases of IL-6, MIP-2 and TNF-α levels. NADPH-oxidase KO mice showed significantly lower levels of IL-6 and MIP-2 responses to SPM comparatively to wildtype. iNOS KO mice showed significantly reduced IL-6, TNF-α, MIP-2 responses to SPM comparatively to wildtype. **Free Radicals:** SPM induced significant increases in free radical formation in alveolar type 2 cells but could be inhibited by DPI.
Reference: Bhattacharyya et al. (2004, 088095) **Species:** Mouse **Strain:** Sprague-Dawley **Weight:** 200-250g	Douglas Fir Wood Smoke (generated by burning wood at 400□C in crucible oven) **Particle Size:** NR	**Route:** Nose-only Inhalation **Dose/Concentration:** 25g/mouse **Time to Analysis:** Various exposure periods (0, 5, 1 0, 15, 20 min). Parameters measured after 24h recovery period.	**Biochemical Parameters:** Lipid peroxidation increased after 20 min of woodsmoke inhalation as did Myeloperoxidase at 20 min. No effects were observed at other times or for total antioxidant status, reduced or oxidized glutathione. **Antioxidant Enzyme Activities:** No effect was observed. **Histology:** Dose-dependent damage progressing from loss of cilia (5 min), degeneration of mucosal epithelium, loss of mucosal epithelium to disrupted mucosal epithelium with submucosal edema and inflammation. Changes persisted for up to 4 days.
Reference: Cao et al. (2007, 097491) **Species:** Rat **Strain:** SH and WKY **Age:** 12wks	$PM_{2.5}$ (Thermo Anderson G-2.5 sampler, Shanghai, China) Components: As, Cd, Cr, Cu, Fe, Ni, Pb, Zn, V, Ba, Se, Mg, Co, Mn **Particle Size:** $PM_{2.5}$	**Route:** IT Instillation **Dose/Concentration:** 1.6, 8.0 and 40 mg/kg **Time to Analysis:** Exposed 1/d for 3d, sacrificed 24h following last exposure	**BALF Inflammatory/Injury Markers:** LDH activity and TBARs increased a in dose-dependent manner. Notably, activity in SH rats was much higher than WKY at the same dose exposed for each dose level. **BALF Cells:** PM decreased macrophages and increased neutrophils and lymphocytes in a dose-dependent manner. For the same exposed dose, WKY rats had a higher percentage than SH but a smaller percentage of neutrophils and lymphocytes. **Cytokines:** PM induced pro-inflammatory cytokine release (IL-1β, TNF-α, CD44, MIP-2, TLR-4, OPN). Again, SH cytokine level was greater than WKY at all dose levels. PM induced anti-inflammatory cytokines CC16 and HO-1 in a similar manner but at much lower rate.

Reference	Pollutant	Exposure	Effects
Reference: Carter et al. (2006, 095936) **Species:** Rat, Mouse, Hamster **Gender:** Female (all) **Strain:** F-344 (rat), B6C3F1 (mouse), Syrian Golden (hamster) **Age:** 7-10wks (all)	CB: Printex 90 **Paritcle Size:** primary size: 17nm; 1.2-1.6 μm (aerosol aerodynamic diameter)	**Route:** Whole-body Inhalation **Dose/Concentration:** 1, 7, 50 mg/m^3 **Time to Analysis:** 6h/day for 5d/wk for 13 wks; 1d, 3m, 11m post-exposure	**Superoxide:** Levels rose in all species at 50mg dose. Hamsters had no increase at 7 and 1mg doses. Mice also increased at 7mg. Rats significantly increased at all dose levels. Rats maintained elevation except for the 50mg dose at 11mo postexposure; it declined but was still higher than control. Mice maintained elevation at 50mg while 7 mg returned to control levels by 3mo postexposure. **H2O2:** At 50mg, increased levels in all species, with the highest in rat, were observed. At 7mg, increased levels in rats and mice were initially seen but levels returned to baseline by 11mo. Hamster levels were not significant. At 1mg, no significant changes were observed. **NO:** Induced similar reactions as H2O2. Rat response continued through the study while mice and hamsters returned to baseline by 11mo postexposure. Rats produced significantly higher levels at all times than other species. **BALF Cells:** CB induced significant increases in neutrophils at 7 and 50mg for all species. Rats had the highest and most prolonged PMN response. Mice and hamsters had very similar reactions. **Cytokines:** TNF-α, MIP-2 and IL-10 increased in a dose-dependent manner in rats and mice. Hamsters increased for IL-10 only. MIP-2 levels were highest in rats. TNF-α level were similarl in all three species at 50mg, but hamsters started with a markedly higher basal level. **Glutathione Peroxidase:** Hamsters were the most responsive with significant increases at all levels. Rats and mice increased at 50mg and continued to increase for up to 11mo. Hamster levels declined with time but continued to be higher than control. **Glutathione Reductase:** Rats increased only at 50mg and remained elevated for up to 11mo. Mice increased at 7 and 50mg and remained elevated for up to 11mo. Hamsters increased at all levels at 11mo, but at 50mg, levels only increased post 1d. **Superoxide Dismutase:** All species reacted in a dose-dependent manner. Rats were the least responsive. Rat SOD activity increased over time while rat and mouse activity decreased at 50mg. Data were consistent with cytokine data. **Summary:** Rats appear to produce proinflammatory responses while mice and hamsters produce antiinflammatory responses.
Reference: Cassee et al. (2005, 087962) **Species:** Rat **Gender:** Male **Strain:** Wistar (SPFHsdCpb: WU) and SH/NHsd **Age:** 7 wks and 8-12wks	CAPs: $PM_{2.5}$ Netherland suburban, industrial and freeway tunnel site collections Wistar rats pre-exposed to ozone SO_4, NO_3 and NH_4 ions: 54 ± 4% suburban, 53 ± 7% industrial and 35 ± 5% freeway site conc of total CAPS mass **Particle Size:** $PM_{2.5}$ ($0.15 < PM < 2.5$ μm)	**Route:** Nose-only Inhalation **Dose/Concentration:** PM 365-3720 μg/m^3 (results from 16 different exposures 2000, 2002); O_3: 1600 μg/m^3 (0.8 ppm) **Time to Analysis:** 8h O_3 pre-exposure; 6h CAPS exposure; 48h post-exposure	**BALF Cells:** Wistar exhibited increased protein, albumin, NAG and decreased ALP activity and macrophage numbers. Wistar showed increased PMNs due to ozone, but was not significantly increased with additional CAPs exposure. SH showed no effect of CAPS except for the increased PMNs. **BALF Inflammatory/Injury Markers:** No effect on AL, LDH, Glutathione, GSSG, GSH, Uric Acid was observed. **Cytokines:** No effect on IL-6, MIP-2 or TNF-α was observed. CAPs induced an increase in CC16 plasma of SH rats. **Hematology:** CAPS induced an increase in RBC, HGB and HCT of Wistar rats and fibrinogen of SH rats. **Histology:** Wistar and SH rats had no obvious lung abnormalities. Small changes include increased macrophages and cellularity of centriacinar septa of ozone-only rats. Both ozone-only and ozone+CAPS showed bronchial epithelium hypertrophy and perivascular influx of PMNs. **BrdU Labeling Index of Terminal Bronchiolar Epithelium:** No CAPs effects were observed.

Reference	Pollutant	Exposure	Effects
Reference: Chang et al. (2005, 097776) **Species:** Mouse **Gender:** Male **Strain:** ICR **Age:** 5wks **Weight:** 25-30g	UFCB: Ultrafine CarbonBlack - Printex 90 (Degussa) **Particle Size:** 14nm	**Route:** IT Instillation **Dose/Concentration:** 200μg/100ul/mouse **Time to Analysis:** Parameters measured 4, 16, 21, 42h post single exposure	**BALF Cells:** Neutrophil number was at control level at 4h, increased after 16h, peaked at 21h and returned to normal at 42h. No effect was observed for the macrophage count. **BALF Inflammatory/Injury Markers:** UfCB increased total protein with peak at 21h. Cytokines: TNF-α increased at 4h and returned to normal at 16h. **VEGF (Vascular Endothelial Growth Factor):** Increased at 4h and peaked at 16h but remained elevated at 21 and 42h. VEGF and total protein in BALF were correlated (R2 = 0.7352). **ROS:** Pretreatment with NAC (ROS inhibitor) decreased induction of BALF VEGF and total protein by UfCB but did not fully block its effect. **Histology:** Thickened alveolar walls in lungs of UfCB-treated mice 16h post-IT was observed.
Reference: Chang et al. (2007, 097475) **Species:** Mouse **Gender:** Male **Strain:** ICR **Age:** 5wks **Weight:** 25-30g	UFCB: Ultrafine Carbon Black - Printex 90 (Degussa) **Particle Size:** 14nm diameter	**Route:** IT Instillation **Dose/Concentration:** 200 μg/mouse; 8mg/kg **Time to Analysis:** Pretreatment with NAC (N-acetylcysteine) ip 320 mg/kg, 2-h before UFCB IT. Parameters measured 24h post exposure.	**BALF Cells:** Increased relative lung weight, total protein (2 fold), total cells (11 fold) and number of neutrophils were observed. BALF AM count was not affected. **BALF Inflammatory/Injury Markers:** Of the 33 identified proteins, the following 6 were confirmed and validated: Cp (ceruluplasmin), albumin, EGFR, LIFR (leukemia inhibitory factor receptor), α2M and β-actin. All were increased following UFCB exposure. The following were also identified: 3 membrane proteins, 3 intracellular proteins, 10 protease inhibitors and 6 antioxidants. UfCB increased LIFR and EGFR in BALF. UfCB significantly reduced EGFR and LIFR in lung homegenate. UfCB did not affect EGFR protein but down-regulated LIFR in A549 cells treated with UfCB. **Antioxidant:** Pretreatment with NAC reduced the intensity of albumin and α2M bands in BALF as well as most other proteins. Statistical analysis showed positive correlation between VEGF and albumin (R2 = 0.796) and VEGF and α2M (R2 = 0.7331) in BALF.
Reference: Cho et al. (2005, 156344) **Species:** Mouse **Gender:** Male **Strains:** DBA/2J, 129P3/J, C57BL/6J, BALB/cJ, A/J, C3H/HeJ, C3H/HeOuJ **Age:** 6-8wks	ROFA: Obtained from Power unit 4, Boston, MA Absent of LPS **Particle Size:** NR	**Route:** IT Instillation **Dose/Concentration:** 6mg/kg bw (150μg in 50μl/ 25g) **Time to Analysis:** 24h; Additional HeJ and OuJ mice: single: 1.5, 3 and 6h (compare TLR-mediated molecular events)	**BALF Cells:** Significant genetic effects on number of macrophages and PMNs after ROFA challenge. For PMNs, DBA/2J, C57BL/6J, BALB/cJ, and 129P3/J all induced increases significantly higher than C3H/HeJ. For macrophages, only the A/J strain induced increases significantly higher than C57BL/6J. Total protein, PMNs and macrophages all increased with HeOuJ inducing increases significantly different from HeJ. **BALF Inflammatory/Injury Markers:** Significant genetic effect on mean total protein concentration was observed. In decreasing order, DBA/2J, 129P3/J and C57BL/6J all induced increases significantly higher than C3H/HeJ. **TLR4 mRNA Expression:** A significant decrease was observed in TLR4 transcript level in HeJ- ROFA exposed mice post 1.5h. Post 6h, TLR4 levels were greater than the control levels. OuJ expression increased beginning 1.5h post exposure. **TLR4 Protein Level:** Protein level of OuJ mice significantly exceeded (~ 2-3 fold) HeJ mice at 1.5, 3 and 6h. **Activation of Downstream Signal Molecules:** Greater activation of MYD88, TRAF6, IRAK-1, NF-KB, MAPK, and AP-1 was observed in OuJ mice than in HeJ mice before the development of ROFA-induced pulmonary injury. **Cytokines:** IL-1β, LT-β, IL-1α, IL-7, IL-13, IL-16 increased in both strains (OuJ and HeJ). Levels of all cytokines above were significantly higher in OuJ than in HeJ.

Reference	Pollutant	Exposure	Effects
Reference: Churg et al. (2003, 087899) **Species:** Human **Gender:** Female (Mexico City); Male, Female (Vancouver) **Strain:** -- **Age:** 66±9yrs (Mexico City); 76±11yrs (Vancouver) **Weight:** NR	PM (Mexico City- high PM region, Vancouver- low PM region) **Particle Size:** Geometric mean size of individual particles in tissue: 0.040-0.067 μm; Aggregates in tissue: 0.34-0.54 μm; Mexico City: 2.5, 10 μm	**Route:** Ambient Air Exposure. Autopsy Tissue. **Dose/Concentration:** 10 - >1000X10^6g dry tissue; Mexico City: PM_{10}: 66 μg/m^3, Vancouver: PM_{10}: 25 μg, $PM_{2.5}$: 15 μg **Time to Analysis:** Lung samples taken from deceased lifelong Mexico City residents and Vancouver residents >20yrs. Subjects were never-smokers, did not work in dust occupations or cook with biomass fuels.	The lungs from Mexico City residents showed increased muscle and fibrous tissue in the membranous bronchioles and respiratory bronchioles compared to the Vancouver residents. Pigmented dust, lumental distortion and carbonaceous aggregates of UFPs were present in the Mexico City lungs.
Reference: Costa et al. (2006, 088438 **Species:** Rat **Gender:** Male **Strain:** Sprague-Dawley **Age:** 60d	ROFA FP&L plant #6 oil, 1% sulfur **Particle Size:** ~1.95 μm	**Route:** ITInstillation vs Nose-only Inhalation (IH) **Dose/Concentration:** IT = 110 μg/rat IH = 12 mg/m^3 **Time to Analysis:** IT = single IH = 6h 24, 48, 96h (histopath 24 and 48 only)	**ROFA distribution:** IH and IT resulted in equivocal distribution (μg/g lung tissue) in 5 different lung lobes. **Airway Hyperactivity:** IT resulted in doubled airway hyperreactivity at 24 h which was sustained for 96h. IH hyperreactivity did not reach statistically significant level. **BALF Inflammatory/Injury Markers:** IH and IT showed very similar responses (R2 = 0.98). Time-dependent increases were observed for protein and LDH. **BALF Cells:** Neutrophils peaked at 24 h and slowly declined at 48 and 96h. **Lung Pathology: IT** showed more alveolitis, bronchial inflammatory and fibrinous fluid infiltrate. IH showed relatively more congestion of small airways and alveolar hemorrhage.
Reference: Courtois et al. (2008, 156369) **Species:** Rat **Gender:** Male **Strain:** Wistar **Age:** 12-14wks **Weight:** NR	PM (SRM 1648; 63% inorganic carbon, 4-7% organic carbon, >1% mass fraction-Si, S, Al, Fe, K, Na) Carbon black (FW, P60) UF, fine TiO_2 **Particle Size:** PM mean diameter: 0.4 μm; Carbon black: FW- 13nm, P60- 21nm; TiO_2 mean diameter: 0.14 μm	**Route:** IT Instillation **Dose/Concentration:** 5mg PM or TiO_2 **Time to Analysis:** I.P injected then instilled. 6-72h recovery period then killed and lungs removed.	Particles were present in lung parenchyma that was removed 12 and 72h postinstillation. The Ach relaxation response significantly decreased in the 12h recovery period group but not in the other groups. Fine TiO_2 did not alter Ach relaxation.
Reference: Dick et al. (2003, 036605) **Species:** Mouse **Gender:** Female **Strain:** CD1 **Age:** 8-10wks **Weight:** 20-25g	CO: PM Coarse FI: PM Fine FU: PM ultrafine PM collected in RTP, NC **Particle Size:** CO: 3.5 - 20 μm; FI: 1.7- 3.5 μm; FU: <1.7 μm	**Route:** IT Instillation **Dose/Concentration:** 10 μg, 50 μg, 100 μg/mouse; 0.5, 2.5, 5.0mg/kg **Time to Analysis:** DMTU 500 mg/kg bw 30 min pre-exposure for some mice. Parameters measured 18h post-exposure.	**Particle Characteristics:** S increased (CO-33.20μg/mg, FI- 49.44μg/mg FU- 122.79μg/mg) with decreasing particle size (mostly in the water-soluble fraction). Fe and Cu higher in coarse and fine fractions (mostly present in the insoluble). CO PM contained more nickel (in both soluble and insoluble) than FI or FU particles. Also, endotoxin levels similar in CO and FI; much lower in FU (0.165 EU/mg). **BALF Cells:** PMN increased with exposure for all 3 fractions except 100 μg FI. **BALF Inflammatory/Injury Markers:** Albumin increased only at 100 μg FI. No differences in NAG or LDH observed. **Cytokines:** IL-6 increased at 100 μg dose for all 3 fractions with similar responses. TNF-α increased a 100 μg dose of fine PM vs control. **Effect of PM After Pre-treatment w/ DMTU:** Systemic administration of DMTU alone depicted a two-fold increase in total antioxidant capacity. **DMTU halved neutrophil response observed with PMs alone:** No fractions were increased over DMTU alone which was at least two-fold saline control. IL-6 concentrations were drastically reduced in the DMTU group for the mice exposed to coarse particles (all fractions were reduced but only coarse had a significant response). TNF-α levels were decreased after treatment with particles and DMTU but treatment with particles and saline (control) produced similar results.

Reference	Pollutant	Exposure	Effects
Reference: Dybdahl et al. (2004, 089013) **Species:** Mouse **Gender:** Female **Strain:** BALB/CJ or trans-genic (MutaMouse) **Age:** 9-10wks **Weight:** ~20g	DEP: SRM 1650 (NIST) **Particle Size:** DEP: NR; Control: PM 0.13 μm diameter	**Route:** Nose-only Inhalation **Dose/Concentration:** I: 20 & 80 mg/m^3 II: 5 & 20 mg/m^3 **Time to Analysis:** I: single exposure 90min; II: 90 min/day for 4d; I & II: parameters measured 1, 3, or 22h post exposure	**Cytokines:** A single 90-min DEP exposure increased IL-6 gene level dose-dependently in the lung. For 80 mg/m^3 DEP, significantly higher IL-6 gene level was observed, both 1 and 22h post exposure. For 20 mg/m^3 DEP, a significantly higher IL-6 level was observed at 1 h post exposure but normalized at 3h. **BALF Cells:** Inhalation of DEP did not decrease viability of BAL cells (see Table1). For mice exposed to 20 mg/m^3 DEP, at 1 h post exposure in BAL fluid there was 3 fold increase in total cell number. **DNA Damage:** Level of 8-oxodG increased post single exposure with 80 mg/m^3 inducing levels significantly higher than controls. Repeated exposures were associated with significantly higher DNA strand breaks.
Reference: Elder et al. (2004, 055642) **Species:** Rat **Gender:** Male **Strain:** Fisher344, SH **Age:** 23m (Fisher), 11-14m (SH)	UFP: argon-filled chamber with electric arc discharge (TSI, Inc., St. Paul, MN) **Particle Size:** 36nm	**Route:** Whole-body Inhalation. Intraperitoneal (ip) for saline and LPS **Dose/Concentration:** UFP: 150 μg/m^3 bw; LPS: 2 mg/kg **Time to Analysis:** Parameters measured post single exposure of 6h, 18h	**BALF Cells:** Neither inhaled UFP nor LPS cause a significant increase in BAL fluid total cells or percentage of neutrophils in either rat strain. No significant exposure-related alteration in total protein concentration was observed. In both rat strains LPS induced a significant increase in the amount of circulating PMNs. When combined with inhaled UFP, PMNs decreased; for F-344 rats, this decrease was significant. **BALF Inflammatory/Injury Markers:** Plasma fibrinogen increased with LPS in both rat strains with the magnitude of change greater in SH rats. UFP alone decreased plasma fibrinogen in SH rats. Combined UFP and LPS response was blunted but significantly higher than controls. Hematocrit was not altered in either rat strain by any treatment. No change in activites of LDH and b-glucuronidase was observed. **TAT complexes:** With all exposure groups averaged, plasma TAT complexes in SH rats were 6.5 times higher than in F-344 rats. LPS caused an overall increase in TAT complexes for F-344 rats which was further augmented by inhaled UFP. UFP alone induced a decreased response. In SH rats, UFP alone exhibited a significantly increased response and LPS exhibited a decreased response. **ROS in BALF:** In F-344 rats, both UFP and LPS have independent and significant effects on DCFD oxidation. Effects were in opposite directions; particles decreased ROS whereas LPS increased ROS.
Reference: Elder et al. (2004, 087354) **Species:** Rat **Gender:** Male **Strain:** F344 **Age:** 21m	Freshly generated vehicle exhaust emissions from I-90 between Rochester and Buffalo, NY **Particle Size:** NR	**Route:** Whole-body Inhalation; (IT Instillation: Influenza) **Dose/Concentration:** Vehicle exhaust: 0.95-3.13 x 10^5 particles/cm^3 Endotoxin: 84 EU Influenza (IV): 10, 000 EID 50 in 250ul **Time to Analysis:** 1x6h, 3x6h or both. Parameters measured 18h post-exposure. 48h prior to on-road exposures, instilled intratracheally with IV. Immediate pre-exposure of priming agent endotoxin. EXPERIMENTS 1: LPS + PM 6 h 2: LPS + PM 6h, 3 x 6h 3: IV + PM 6 h 4: IV + PM 6h, 3x 6 h	No departures from normal baseline cellular or biochemical values were observed, suggesting that on-road exposures were well tolerated by the rats. **BALF Inflammatory/Injury Markers:** Increase in total protein concentration, LDH and B-glucuronidase activities were observed. **Specific results according to groups 1-4 are as follows:** **Experiment 1:** No endpoints revealed significant differences between groups of rats exposed to gas phase only versus the gas-phase/particle mixture. **Experiment 2:** Combination of endotoxin and particles produced greater inflammatory responses than those treated with saline and particles post 1d. After 3 days, no statistically significant changes were noted. **Experiment 3:** Influenza virus significantly increased ROS release in BAL cells. **Experiment 4:** Influenza virus significantly increased both percentage of PMNs in lavage fluid and BAL cell ROS release.

Reference	Pollutant	Exposure	Effects
Reference: Elder et al. (2005, 088194) **Species:** Rat, Mouse, Syrian Golden Hamster **Gender:** Female **Strain:** F-344, B6C3F1, FIB	HSCb: Printex-90 high surface area carbon black, Degussa-Huels (Trostberg, Germany). LSCb: Sterling V, low surface area carbon black, Cabot (Boston, MA) **Particle Size:** HSCb = 14nm, LSCb = 70nm	**Reference:** Whole-body Inhalation **Dose/Concentration:** 0, 1, 7, 50 mg/m^3 HSCb; 50 mg/m^3 LSCb (rats only) **Time to Analysis:** 6 h/d, 5 d/wk for 13wks. Parameters measured 1d, 3mo, 11mo post-exposure	**Body Weight:** Environmental changes pre and post-exposure affected test subjects' life spans, particularly hamsters. Hamsters also experienced significant loss of body weight when exposed to high doses of HSCb. **Effects of Carbon Black:** In rats, lung weight of the high dose HSCb doubled. After 11mo, analysis of all lungs showed no significant difference. Mice had the highest relative lung burdens at the end of exposure time but also cleared particles faster at high doses than rats. However, clearance slowed over the 11mo recovery period, especially in high dose mice. Hamsters showed significant elevations in lung carbon black burden for all exposures at all time points. Hamsters exposed to high dose HSCb exhibited impaired clearance. **BALF Cells:** Presence of PMNs was limited to the mid and high dose groups. Overall maximal response was reached in mice and hamsters, but not in rats with increasing mass dose of HSCb.
Reference: Evans et al. (2006, 097066) **Species:** Rat **Gender:** Male **Strain:** Sprague-Dawley	DEP: collected under dry, outdoor, ambient conditions from tractor exhaust pipe (1985, Japanese ISEKI 1500 cc tractor) burning Esso 2000 diesel and 20/30 mixture of Esso light engine oil. 10% UF, 90% fine Cabosil: amorphous silicon dioxide 16% UF, 84% fine **Particle Size:** DEP: 30nm; Cabosil: 7nm	**Route:** IT Instillation **Dose/Concentration:** 1mg/rat DEP; 1mg/rat Cabosil **Time to Analysis:** Pretreatment with 0.5 unit of bleomycin; IT 3 or 7d after pre-treatment; 1wk post-IT	**Lung permeability:** In bleomycin-treated group, obvious inflammatory status and edema within the lung was observed. This was shown by significant increases in acellular protein and free cells. **Changes in lung:** Body weight ratio, lung surface protein content, free cell counts, and apical surface protein of rat type I cells were only altered by bleomycin treatment and not particle exposure.
Reference: Finnerty et al. (2007, 156434) **Species:** Mouse **Gender:** Male **Strain:** C57BL/61 **Age:** 12 wks **Weight:** 24.3 ±0.3g	Coal Fly Ash (generated at U.S EPA National Risk Management Research Laboratory by burning Montana subbituminous coal under conditions simulating full-scale utility boiler conditions) Transition metals of Coal Fly Ash: Fe, Mg, Ti, Mn, V **Particle Size:** $>PM_{2.5}$	**Route:** IT Instillation **Dose/Concentration:** PM: 200mg PM/mouse; 9.1 mg/kg PM+LPS10: 200mg PM+10mg LPS PM+LPS100: 200mg PM+100mg LPS LPS: 100 μg **Time to Analysis:** Parameters measured 18h post single exposure	**BALF Cells:** No significant differences in platelet concentration or white blood cell count in any groups were observed. The percentage of neutrophils increased significantly with PM+LPS100. PMN rose in PM groups and increased further with LPS treatment. Increases in PM+LPS were groups statistically significant. More leukocytes were present in the alveolar space in PM+LPS10 compared to the PM group. The most severe response was in the PM+LPS100 group. **Cytokines:** Plasma TNF-α and IL-6 significantly increased for the PM+LPS100 group. An additive effect of LPS and PM for IL-6 was observed. For saline and PM groups, pulmonary TNF-α was below detection range. A synergistic effect for TNF-α was observed. A less than additive effect for IL-6 was observed. Pulmonary TNF-α significantly increased in the PM+ LPS100 group. Pulmonary IL-6 significantly increased in both PM+LPS groups.
Reference: Fujimaki et al. (2006, 096601) **Species:** Mouse **Gender:** Male **Strain:** IL-6(-/-) and WT: B6J129Sv (control) **Age:** 5-6wks	DEP: collected from a 4-cylinder, 2.74 L, Isuzu diesel engine **Particle Size:** 0.4 μm	**Route:** Whole-body Inhalation **Dose/Concentration:** 1.0, 3.0 mg/m^3 **Time to Analysis:** 12 h/d for 4wks. Parameters measured 1d post-exposure	**BALF Cells:** Treatment significantly increased BAL cells from WT mice at both dose levels. The increase of macrophages and neutrophils were dose-dependent. An increase in lymphocytes were present in WT mice with the low dose. No significant increase in cells were observed from IL-6 (-/-). **Cytokines:** TNF-α largely increased in IL-6(-/-) mice exposed to 3 mg/m^3 compared to WT mice. IL-6 production increased in WT mice exposed to 3 mg/m^3. CCL3 increased in both WT and IL-6(-/-) at high dose. IL-1β remained at the control level.

Reference	Pollutant	Exposure	Effects
Reference: Gerlofs-Nijland et al. (2005, 088652) **Species:** Rat **Gender:** Male **Strain:** SH/NHsd **Age:** 11-12wks **Weight:** 250-350g	RTD: road tunnel dust (obtained from a Motorway tunnel in Hendrik-Ido-Ambacht, Netherlands) EHC-93 (Ottawa, Canada) **Particle Size:** Coarse: 2.5 - 10 μm; fine: 0.1- 2.5 μm	**Route:**IT Instillation **Dose/Concentration:** 0.3, 1, 3, 10 mg/kg EHC-93 10 mg/kg **Time to Analysis:** Parameters measured 4, 24, 48h post single exposure	**BALF Cells:** PMN significantly increased in RTD (3 and 10 mg/kg dose) and EHC-93 exposed animals at 24 h and decreased by 48h but remained statistically significant. AM numbers decreased for 3 mg/kg RTD group at 4h. **BALF Inflammatory/Injury Markers:** Myeloperoxidase (measured at 24 h in 1, 3, 10 mg/kg RTD groups) was elevated in a dose-dependent manner. RTD induced time-dependent increases in LDH activity at 24 and 48h, although these increases were less than EHC-93 values at the same time points. Alkaline phosphatase increased dose-dependently for RTD at 48h. GSH decreased at 24 h to approximately the same levels in 0.3, 1, and 3 mg/kg RTD dose groups. Uric acid only decreased in 1 mg/kg RTD group at 24 h. **Cytokines:** IL-6 levels were elevated only at 10 mg/kg for RTD and EHC-93 at 4 and 24 h; it remained elevated for EHC-93 at 48h. A dose-dependent increase in TNF-α at 4h for RTD was observed. TNF-α levels remained elevated only for the 10 mg/kg groups at 24 h and returned to control levels by 48h. A dose-dependent increase in MIP-2 for all RTD dose groups were observed and remained elevated through 48h for both PM types (although values were returning to control levels). **Hematology:** No significant changes in plasma for bigET-1 or von Willebrand factor were observed. At the highest dose, fibrinogen levels significantly increased at 24 and 48h for both PM types. **Pulmonary histopathology:** A dose-dependent increase in the number of inflammatory foci at 24 and 48h for 3 and 10 mg/kg RTD groups was observed. The response was even greater for the EHC-93 exposed group at similar time points.

Reference	Pollutant	Exposure	Effects
Reference: Gerlofs-Nijland et al. (2007, 097840) **Species:** Rat **Gender:** Male **Strain:** SH/NHsd **Age:** 13wks **Weight:** 250-350g	PM samples collected from: 1. MOB high traffic density 2. HIA high traffic density 3. ROM high traffic density 4. DOR moderate traffic density 5 MGH low traffic density 6 LYC low traffic density **Particle Size:** Coarse: 2.5 - 10 μm; Fine: 0.1 - 2.5 μm	**Route:** IT Instillation **Dose/Concentration:** 3, 10mg/kg **Time to Analysis:** 24h	**BALF Inflammatory/Injury Markers:** LDH was significantly increased for all doses of coarse PM and for the high dose of fine PM. BALF protein concentration was observed predominantly at the high dose of coarse PM. Location ROM had evidence of attenuated responses with fine PM. Ascorbate concentrations were reduced but were only significant for rats exposed to the highest dose of coarse PM fractions from the locations MOD, HIA, and LYC. **BALF Cells:** Pulmonary inflammation was induced in a significant and dose-dependent manner for both dose levels. Inflammation in the BALF included airway neutrophilia, increased macrophage numbers and mild lymphocytosis. Both coarse and fine PM caused dose-dependent aveolitis. Fine PM from LYC (10mg/kg dose) also caused some bronchiolitis. **Cytokines:** TNF-α concentrations increased for all coarse samples with the exception of DOR and LYC. Fine PM induced similar responses for all sites. MIIP-2 concentrations increased only at certain sites for coarse but not fine PM. **Hematology:** Fibrinogen responses of SH rats increased significantly at the high dose of both fractions of all PM samples, except fine PM from DOR. **Location-related differences:** Coarse PM from MOB, HIA and MGH induced higher LDH responses than other locations. Coarse PM from HIA produced BALF protein concentrations higher than LYC and ROM. MGH induced greater amounts of BALF protein than ROM. Coarse PM from LYC lowered fibrinogen values more than PM from location MOB, HIA, and MGH. Fine PM showed less differences among the various sites. **Particle Correlation:** Fine PM exhibited significant correlation between zinc content and BALF cytotoxicity markers protein and LDH - mainly from HIA. Fine PM also exhibited positive correlations with copper and barium. Coarse PM showed positive correlation with barium and copper, mainly from MOB.
Reference: Gerlofs-Nijland et al. (2009, 190353) **Species:** Rat **Gender:** Male **Strain:** SH **Age:** 12wks **Weight:** 200-300g	PM (Prague, Czech Republic; Duisburg, Germany; Barcelona, Spain) (Prague and Barcelona coarse PM organic extracts) **Particle Size:** Coarse: 2.5-10 μm, Fine: 0.2-2.5 μm	**Route:** IT Instillation **Dose/Concentration:** 7mg PM/kg body weight **Time to Analysis:** DTPA added to some PM samples preinstillation. Instilled with PM. Necropsy 24h postexposure.	Inflammation (LDH, protein, albumin), cytotoxicity (NAG, MPO, TNF-α), and fibrinogen were increased by PM, and were greatest in the coarse PM fraction. Metal-rich PM had greater inflammatory and cytotoxic effects, and increased fibrinogen and vWF and decreased ACE. PAH content influenced greater inflammation (including neutrophils), cytotoxicity, and fibrinogen. Generally, whole PM and coarse PM were more potent than organic extracts and fine PM, respectively.
Reference: Ghio et al. (2005, 088272) **Species:** Rat **Gender:** Male **Strain:** N8 b/b Belgrade rats and N8+ Ib Belgrade controls	Oil Fly Ash (Southern Research Institute, Birmingham, AL) **Particle Size:** 1.95 ± 0.18 μm (MMAD)	**Route:** IT Instillation **Dose/Concentration:** 500μg/rat; 2mg/kg **Time to Analysis:** 24h	**BALF Cells:** Homozygous Belgrade with mutation G185R had higher levels of Fe and V 24 h post-exposure. This may demonstrate a decreased ability to remove Fe and V from the lower respiratory tract than heterozygous +Ib littermates. This also indicates that DMT1 is normally responsible for at least some Fe and V uptake; thus, a defective DMT1 transports less. **BALF Inflammatory/Injury Markers:** Increased protein and LDH concentrations in the homozygous strain were observed when compared to control

Reference	Pollutant	Exposure	Effects
Reference: Ghio et al. (2005, 088275) **Species:** Rat **Gender:** Male **Strain:** Sprague-Dawley **Age:** 60d **Weight:** 250-300g	Ferric ammonium citrate (FAC) Vanadyl sulfate ($VOSO_4$) **Particle Size:** NR	**Route:** IT Instillation **Dose/Concentration:** 0.5 mL 100 μm FAC/rat; 0.5 mL 10 μm $VOSO_4$/rat; 500 μg oil fly ash; 2 mg/kg **Time to Analysis:** Single or double exposure with 24 h rest period. Parameters measured 15, 30, 60 min, 24h post-exposure.	**DMT1 immunohistochemistry and lung injury:** FAC increased and $VOSO_4$ decreased –IRE DMT1 staining. Same exposures had no effect on +IRE DMT1. –IRE DMT1 expression in macrophages, airway and alveolar epithelial cells increased with increased Fe exposure. Vanadium nearly eliminated staining except in alveolar macrophages. Increased metal clearance with pre-exposure to FAC. Less metal clearance with pre-exposure to $VOSO_4$. Pre-exposure to iron diminished lung injury whereas pre-exposure to vandium increased lung injury after oil fly ash instillation. Lung injury measured by concentration of protein and LDH in BAL.
Reference: Gilmour et al. (2007, 096433) **Species:** Mouse **Gender:** Female **Strain:** BALB/c **Age:** 10-12wks **Weight:** 20-22g	PM - CO, FI, UF (obtained from U.S. Seattle (S), Salt Lake City (SL), South Bronx (SB), Sterling Forest (SF)) SB: included 35% sulfate, 22% gasoline, diesel and brake wear. SF: 48% sulfate. SL: 34% wood combustion and 28% sulfate S: 39% wood combustion and 29% sulfate Residual oil combustion and soil dust less than 5% for all sites. **Particle Size:** CO: 2.5-10μm; FI: ◘ 2.5 μm; UF: ◘ 0.1μm	**Route:** Oropharyngeal Aspiration **Dose/Concentration:** 25μg or 100μg PM; 1.25 or 5 mg/kg **Time to Analysis:** 18h	**BALF Inflammatory/Injury Markers:** Seattle CO fractions showed no dose-dependent effect on protein concentration. Results for other locations were distinctly higher with 100 μg dose than 25 μg and saline doses. SL CO high dose induced the most significant increase. LDH response was weakly dose-related. Only SB showed a statistically significant increase for LDH with the high dose UF. **BALF Cells:** PMN increased with the high dose of CO samples from SB, SL, S, but not SF. No significant increases from FI were observed, though the high dose induced increased PMN. UF from SL caused a highly variable response. **Cytokines:** MIP-2 was similar to PMN response. SB CO induced the most significant response. SL UF was highly variable. **Particle Characteristics:** LPS was higher in S (CO, FI, UF) and SL (CO, FI, UF). Zn levels were highest in SB (CO, FI, UF). Fe was higher in all CO and FI samples with SB CO inducing the highest.
Reference: Gilmour et al. (2004, 057420) **Species:** Mouse **Gender:** Female **Strain:** CD1 **Age:** 8-10wks **Weight:** 20-25g	Coal Fly Ash MU: Montana Ultrafine MF: Montana Fine MC: Montana Coarse KF: W.Kentucky Fine KC: W. Kentucky Coarse Particle Characteristics: Montana Sulfur 0.83%, Ash 11.72%. Trace amounts of Ba, P, Sr, V, Nb, Cd, Se, Ga, Cu. Depleted in Si, Al, Fe, Mg, Ti. Kentucky Sulfur 3.11%, Ash 8.07% **Particle Size:** Coarse: >2.5 μm; Fine: <2.5 μm; Ultrafine: <0.2 μm	**Route:** Oropharyngeal Aspiration **Dose/Concentration:** 25ug or 100μg/mouse **Time to Analysis:** 18h	**BALF Cells:** PMN highly increased for MU at both doses. The level was comparable to the positive control. PMN also increased with KF at high dose. Coarse particles caused no significant increase in PMN. Number of macrophages did not change, but NAG increased significantly with MU for both dose levels and with KF and MF at high dose level. **BALF Inflammatory/Injury Markers:** Total protein and LDH was not significantly elevated. Albumin concentration increased significantly after treatment with the fine high dose of both particle types. **Cytokines:** MU particles caused a significant increase in TNF-α. MIP-2 increased in all fine and ultrafine PM-instilled animals with the highest in the MU and KF at both doses. IL-6 was detectable only in the BALF of MU and KF with substantial variability. The IL-6 levels were not significant.

Reference	Pollutant	Exposure	Effects
Reference: Gilmour et al. (2004, 087948) **Species:** Rat **Gender:** Male **Strain:** SH/NQIBR, WKY **Age:** 12wks **Weight:** 280-340g	PM (collected from precipitator unit of an oil burning power plant in Boston) Measured Components of PM: S, Zn, Ni, V, Al, Cu, Pb, Fe, Ca, Na, K, Mg, Endotoxin **Particle Size:** NR	**Route:** IT Instillation **Dose/Concentration:** 0.0, 0.83, 3.3, and 8.3 mg/kg in SH rats; 0.0 or 3.3 mg/kg in WKY and SH rats **Time to Analysis:** 24h	**BALF Inflammatory/Injury Markers:** LDH activity increased in a dose-related manner; this was observed in SH rats after exposure to 0.83, 3.33 and 8.3 mg/kg PM. SH rats showed greater lung permeability following PM exposure than WKY rats. SH rats showed acute lung inflammatory response after exposure to PM when compared to WKY rats. **BALF Cells:** No increase in macrophage number was observed in either rat strain following saline or PM exposure at 24 h. **Cytokines:** MIP2 mRNA expression increased significantly in SH PM exposure group only. No significant differences in TNF-α RNA expression in either WKY, SH rats or control treatment groups were observed. **CD14:** A significant increase in lung CD14 protein was observed only in SH rats exposed to PM. **TLR4:** A significant increase in TLR4 protein in SH rats exposed to PM was observed. **NF-kB:** A significant increase in NF-kB binding protein in the nuclei of SH rats exposed to PM was observed. This effect was not observed in the control of PM-exposed WKY rats.
Reference: Gilmour et al. (2004, 054175) **Species:** Rat **Gender:** Male **Strain:** Wistar **Age:** 12wks	ufCB: Ultrafine carbon black (Printex 90 (Degussa) CB: (Huber 990, HR. Haeffner and Co) **Particle Size:** ufCB: 14nm; CB: 260nm (primary particle diameter)	**Route:** Whole-body Inhalation **Dose/Concentration:** ufCB: 1.66 mg/m^3 fCB: 1.40 mg/m^3 Number concentrations ufCB: 52380 particles/cm^3 fCB: 3800 particles/cm^3 **Time to Analysis:** Exposed for 7 h. Sacrificed 0, 16 or 48h post-exposure.	**BAL Cells:** Total number of cells increased significantly in UfCB-exposed rats at 0 and 16 h. Recruitment of cells did not occur in response to CB exposure. PMNs increased significantly in the BALF of ufCB-exposed rats at 16 h. Leukocytes remained unchanged following CB exposure but increased significantly at 0 and 48 h post exposure to ufCB. **Cytokine mRNA:** A significant increase in BALF MIP-2 mRNA expression was observed at 48 h. No differences in MIP-2 mRNA levels were observed in the whole lung tissue.
Reference: Godleski et al. (2002, 156478) **Species:** Rat **Gender:** Male **Strain:** SD **Age:** NR **Weight:** 200-250g	CAPs (Boston; Harvard Ambient Particle Concentrator) **Particle Size:** 0.27 ± 2.3 μm (diameter)	**Route:** Inhalation **Dose/Concentration:** 73.5-733 μg/m^3 **Time to Analysis:** Exposed 5h/d, 3d (consecutive). BAL 24h postexposure	PMNs significantly increased with CAPs exposure and also in relation to CAPs mass, Br, SO_4^{2-}, EC, OC and Pb. An overall increase in pro-inflammatory mediators and decrease in immune enhancer and evidence of vascular endothelial responses occurred with CAPs exposure.
Reference: Gottipolu et al. (2009, 190360) **Species:** Rat **Gender:** Male **Strain:** Wistar Kyoto (WKY), SH **Age:** 14-16wks **Weight:** NR	DE (30-kW (40hp) 4-cylinder indirect injection Deutz diesel engine) (O_2- 20%, CO- 1.3-4.8ppm, NO- < 2.5-5.9ppm, NO_2- < 0.25-1.2ppm, SO_2- 0.2-0.3ppm, OC/EC- 0.3 ± 0.03) **Particle Size:** Number Median Diameter: Low- 83 ± 2nm, High- 88.2nm; Volume Median Diameter: Low- 207 ± 2nm, High- 225 ± 2nm	**Route:** Exposure Chamber **Dose/Concentration:** Low- 507 ± 4 μg/m^3, High- 2201 ± 14 μg/m^3 **Time to Analysis:** Exposed 4h/d, 5d/wk, 4wks. Necropsied 1d postexposure.	DE increased neutrophils in a concentration-dependent manner, and GGT activity at the high dose. Particle-laden macrophages were found in DE-exposed rats. DE dose-dependently inhibited mitochondrial aconitase activity. DE caused 377 genes to be differentially expressed within WKY rats, most of which were downregulated, but none in SH rats. However, WKY rats had an expression pattern shift that mimicked baseline expression of SH rats without DE. These genes regulated compensatory response, matrix metabolism, mitochondrial function, and oxidative stress response.
Reference: Gunnison and Chen (2005, 087956) **Species:** Mouse **Gender:** Male **Strain:** DK ($ApoE^{-/-}$, $LDLr^{-/-}$) **Age:** 18-20wks	CAPS (Northeastern regional background) Ambient air copollutants measured O_3, NO_2 **Particle Size:** 389 ± 2nm	**Route:** Whole-body Inhalation **Dose/Concentration:** CAPS = 131 ± 99 μg/m^3 including O_3 = 10 ppb and NO_2 = 4.4 ppb **Time to Analysis:** 6h/d, 5d/wk for 4m (5/12/03-9/5/03). Sacrified 3-4d post exposure.	**Microarray Data:** 13 genes in the heart tissue and 47 genes in the lung tissue were identified as possibly affected. Strict standards (1.5 fold response, 10 % false discovery rate) resulted in responses by only 1/13 genes (Rex3 - no known heart physiology) in the heart tissue and 0/47 genes in the lung tissue. Using more liberal response (nonstatistical) standards (1.5 fold only) and comparison of each CAPS animal with all 3 control animals (3x3 array) resulted in possible effects on 7 additional genes in the heart tissue and 37 genes in the lung tissue.

Reference	Pollutant	Exposure	Effects
Reference: Gurgueira et al. (2002, 036535) **Species:** Rat **Gender:** Male **Strain:** Sprague-Dawley **Weight:** 250-300g	CAPs (Harvard Ambient Particle Concentrator) CB (C198 Fischer Scientific, Pittsburg, PA USA) Composed of 85.9± 0.2% Carbon, 13.0± 0.2% O2, 1.17± 0.2% Sulfur ROFA (Boston, MA USA oil-fired power plant) **Particle Size:** CAPs: 1-2.5 μm; CB: $<$2.5 μm; ROFA: $<$2.5 μm	**Route:** Whole-body Inhalation **Dose/Concentration:** 300 ± 60 $\mu g/m^3$ **Time to Analysis:** 1, 3, 5h CAPs Exposure followed by immediate post-exposure analysis. 5h CB, immediate analysis. 30min ROFA, Immediate analysis.	**In situ Chemiluminescence(CL):** Data show a significant increase in lung and heart CL at 5h. Lung CL increased linearly with time of exposure. **Oxidants:** CAPs-initiated oxidative stress was not detectable in those rats allowed to recover in room air after the simulated "peak" in particulate air pollution. Rats breathing particle-free filtered air for 3 days had significantly lower levels of oxidants. Exposure to inert CB did not exert oxidant effects on the heart and lung. **BALF Inflammatory/Injury Markers:** The water content of the lung and heart increased significantly upon exposure to CAPs but not to filtered air and increased as a function of length of exposure. Rats breathing CAPs also showed increases in LDH and CPK as a function of length of exposure. **Antioxidant enzymes:** Data showed an increase in SOD and catalase activities in both the lung and heart. The pattern of increase was tissue specific.
Reference: Hamoir et al. (2003, 096664) **Species:** Rabbit **Strain:** New Zealand **Age:** 12-16 wks **Weight:** 2.8 ± 0.5kg	PSC: Polystyrene particles, Carboxylate modified, 3 types PSA: Polystyrene particles, Amine modified, 1 type **Particle Size:** PSC: 24, 110 or 190nm (PSC24, PSC110, PSC190); PSA: 190nm	**Route:** IT Instillation **Dose/Concentration:** PSC24: 0.04 or 4 mg/rabbit PSC110, PSC190, PSA190: 4mg/rabbit **Time to Analysis:** 0, 30, 60, 90, 120min	**Capillary Filtration Coefficient:** A time-dependent increase correlating to total number of particles/surface area, not particle size, was observed.PSA induced a significant increase in microvascular permeability as compared to PSC. This suggests that the number of particles exposed should be considered an important parameter for measuring air quality rather than total particle surface area.

Reference	Pollutant	Exposure	Effects
Reference: Happo et al. (2007, 096630) **Species:** Mouse **Gender:** Male **Strain:** C57BL/6J **Weight:** 19-30g **Age:** 10-11wks	PMC (Coarse) PMF (Fine) PMUF (Ultrafine) Collected in 6 European cities: Duisburg, Prague, Amsterdam, Helsinki, Barcelona, Athens **Particle Size:** PMC: $PM_{10\text{-}2.5}$; PMF: $PM_{2.5\text{-}0.2}$; PMUF: PM0.2	**Route:** IT Instillation **Dose/Concentration:** PMC: 5.9-29.6 $\mu g/m^3$; PMF: 8.3-25.2 $\mu g/m^3$; PMUF: 2.7-6.7 $\mu g/m^3$ Dose-response: 1, 3, 10mg/kg Time course: 10 mg/kg **Time to Analysis:** 1. Dose-Response study: parameters measured 24h post exposure. 2. Time course study: parameters measured 4, 12, 24 h post single exposure (at 10 mg/kg).	**Total Cell Numbers:** 1. For the dose-response study, all the PMC samples exhibited dose-dependent increases of total cell numbers. The 3 and 10 mg/kg doses of PMC induced statistically significant increases. At 10 mg/kg, only 2/6 samples induced statistically significant increases. No PMUF samples induced effects at any dose. 2. For the time-response study, no increases in cell numbers were shown at 4 h. Though the levels induced by PMC at 24 h were lower than at 12 h, both levels were statistically significant. PMF induced statistically significant increases only at 12 h for 4/6 samples. PMUF induced only 1 significant increase at 12 h; the 24 h time point was not tested. **Total Protein/LDH:** 1. The lower doses of 1 and 3 mg/kg did not induce significant increases in any of the PM samples, except for PMUF-Athens. All 6 samples of PMC, at 10 mg/kg, induced significant increases. At 10 mg/kg, 4/6 PMF samples induced significant increases. 2. At 4 h, none of the samples increased protein concentration. The PMC samples, excluding Prague, induced significantly higher concentrations at 12 h. At 24 h, only 3/6 PMC samples induced significant increases. Only 2 PMF samples induced significant increases at 12 and 24 h. At 12 h, effects induced by PMUF were minimal and inconsistent; the 24 h time point was not tested. **Cytokine:** 1. Only PMC induced dose-dependent responses that reached statistical significance at 10 mg/kg. PMF and PMUF induced minimal and inconsistent responses. **TNF-α levels:** 2. TNF-α levels increased significantly at 4 and 12 h by PMC. At 24 h, TNF-α levels returned to near control levels. PMF, at 4 h, induced statistically significant increases for 3/6 samples and significant increases in 2/6 samples at 12 h. No PMUF samples significantly increased TNF-α levels. **IL-6:** 2. PMC induced the highest IL-6 levels at 4 h. Levels at 12 and 24 h were reduced with 6/6 and 3/6 samples showing statistically significant increases, respectively. PMF showed a similar trend with 4 h inducing the highest levels that were reduced at 12 and 24 h. Of the PMUF samples, only the Helsinki and Duisburg samples induced statistically significant results at 4 and 12 h. Generally, the PMUF responses were negligible when compared to PMC and PMF. **KC production:** 2. All PMC samples induced the highest levels at 4 h. At 12 and 24 h, levels were reduced but 4/6 samples induced statistically significant levels. PMF showed a similar trend- the highest levels were induced at 4 h (in 3/6 samples). PMUF at 4 h showed small, though not significant, increases. At 12 h, only 2 samples showed statistically significant differences from the control; the 24 h time point was not tested.
Reference: Harder et al. (2005, 087371) **Species:** Rat **Gender:** Male **Strain:** Wistar Kyoto **Age:** 14-17wks **Weight:** NR	Carbon UFP **Particle Size:** Diameter: 37.6 ± 0.7nm	**Route:** Exposure Chamber **Dose/Concentration:** 180 $\mu g/m^3$ **Time to Analysis:** Telemeter implanted into peritoneal cavity. 10d recovery. 3d baseline reading. 24h exposure. 3d recovery.	Carbon UFP mildly but significantly elevated HR compared to the control. SDNN was significantly decreased during exposure. UFP induced mild pulmonary inflammation, significantly increased PMN, and increased the total protein and albumin concentrations. Particle-laden macrophages sporadically accumulated in the alveolar region.

Reference	Pollutant	Exposure	Effects
Reference: Harkema et al. (2004, 056842) **Species:** Rat **Gender:** Male **Strain:** F344, BN **Age:** 10-12wks **Weight:** NR	CAPs (Detroit; July-Sept. 2000; Harvard Ambient Fine Particle Concentrator) **Particle Size:** 2.5 μm (diameter)	**Route:** Inhalation Exposure Chamber. IT Instillation. **Dose/Concentration:** 4d concentration: 676 ± 288 $\mu g/m^3$, 5d concentration: 313 ± 119 $\mu g/m^3$, July concentration: 16-185 $\mu g/m^3$, September concentration: 81-755 $\mu g/m^3$; IT Instillation- 200 μL (soluble and insoluble) **Time to Analysis:** F344 rats sensitized to endotoxin, BN rats to OVA. Exposed 10h/d 1, 4, 5d (consecutive). Another group of rats i.t instilled. Both groups killed 24h postexposure.	The retention of PM in the airways was enhanced by allergic sensitization. Recovery of anthropogenic trace elements was greatest for CAPs-exposed rats. Temporal increases in these elements were associated with eosinophil influx, BALF protein content and increased airway mucosubstances. A mild pulmonary neutrophilic inflammation was observed in rats instilled with the insoluble fraction but instillation of total, soluble or insoluble $PM_{2.5}$ in allergic rats did not result in differential effects.
Reference: Elder et al. (2004, 055642) **Species:** Rat **Gender:** Male **Strain:** Fisher344, SH **Age:** 23m (Fisher), 11-14m (SH)	UFP: argon-filled chamber with electric arc discharge (TSI, Inc., St. Paul, MN) **Particle Size:** 36nm	**Route:** Whole-body Inhalation. Intraperitoneal (ip) for saline and LPS **Dose/Concentration:** UFP: 150 $\mu g/m^3$ bw; LPS: 2 mg/kg **Time to Analysis:** Parameters measured post single exposure of 6h, 18h	**BALF Cells:** Neither inhaled UFP nor LPS cause a significant increase in BAL fluid total cells or percentage of neutrophils in either rat strain. No significant exposure-related alteration in total protein concentration was observed. In both rat strains LPS induced a significant increase in the amount of circulating PMNs. When combined with inhaled UFP, PMNs decreased; for F-344 rats, this decrease was significant. **BALF Inflammatory/Injury Markers:** Plasma fibrinogen increased with LPS in both rat strains with the magnitude of change greater in SH rats. UFP alone decreased plasma fibrinogen in SH rats. Combined UFP and LPS response was blunted but significantly higher than controls. Hematocrit was not altered in either rat strain by any treatment. No change in activites of LDH and b-glucuronidase was observed. **TAT complexes:** With all exposure groups averaged, plasma TAT complexes in SH rats were 6.5 times higher than in F-344 rats. LPS caused an overall increase in TAT complexes for F-344 rats which was further augmented by inhaled UFP. UFP alone induced a decreased response. In SH rats, UFP alone exhibited a significantly increased response and LPS exhibited a decreased response. **ROS in BALF:** In F-344 rats, both UFP and LPS have independent and significant effects on DCFD oxidation. Effects were in opposite directions; particles decreased ROS whereas LPS increased ROS.
Reference: Elder et al. (2004, 087354) **Species:** Rat **Gender:** Male **Strain:** F344 **Age:** 21m	Freshly generated vehicle exhaust emissions from I-90 between Rochester and Buffalo, NY **Particle Size:** NR	**Route:** Whole-body Inhalation; (IT Instillation: Influenza) **Dose/Concentration:** Vehicle exhaust: 0.95-3.13 x 10^5 particles/cm^3 Endotoxin: 84 EU Influenza (IV): 10, 000 EID 50 in 250ul **Time to Analysis:** 1x6h, 3x6h or both. Parameters measured 18h post-exposure. 48h prior to on-road exposures, instilled intratracheally with IV. Immediate pre-exposure of priming agent endotoxin. EXPERIMENTS 1: LPS + PM 6 h 2: LPS + PM 6h, 3 x 6h 3: IV + PM 6 h 4: IV + PM 6h, 3x 6 h	No departures from normal baseline cellular or biochemical values were observed, suggesting that on-road exposures were well tolerated by the rats. **BALF Inflammatory/Injury Markers:** Increase in total protein concentration, LDH and B-glucuronidase activities were observed. **Specific results according to groups 1-4 are as follows:** **Experiment 1:** No endpoints revealed significant differences between groups of rats exposed to gas phase only versus the gas-phase/particle mixture. **Experiment 2:** Combination of endotoxin and particles produced greater inflammatory responses than those treated with saline and particles post 1d. After 3 days, no statistically significant changes were noted. **Experiment 3:** Influenza virus significantly increased ROS release in BAL cells. **Experiment 4:** Influenza virus significantly increased both percentage of PMNs in lavage fluid and BAL cell ROS release.

Reference	Pollutant	Exposure	Effects
Reference: Elder et al. (2005, 088194) **Species:** Rat, Mouse, Syrian Golden Hamster **Gender:** Female **Strain:** F-344, B6C3F1, FIB	HSCb: Printex-90 high surface area carbon black, Degussa-Huels (Trostberg, Germany). LSCb: Sterling V, low surface area carbon black, Cabot (Boston, MA) **Particle Size:** HSCb = 14nm, LSCb = 70nm	**Reference:** Whole-body Inhalation **Dose/Concentration:** 0, 1, 7, 50 mg/m^3 HSCb; 50 mg/m^3 LSCb (rats only) **Time to Analysis:** 6 h/d, 5 d/wk for 13wks. Parameters measured 1d, 3mo, 11mo post-exposure	**Body Weight:** Environmental changes pre and post-exposure affected test subjects' life spans, particularly hamsters. Hamsters also experienced significant loss of body weight when exposed to high doses of HSCb. **Effects of Carbon Black:** In rats, lung weight of the high dose HSCb doubled. After 11mo, analysis of all lungs showed no significant difference. Mice had the highest relative lung burdens at the end of exposure time but also cleared particles faster at high doses than rats. However, clearance slowed over the 11mo recovery period, especially in high dose mice. Hamsters showed significant elevations in lung carbon black burden for all exposures at all time points. Hamsters exposed to high dose HSCb exhibited impaired clearance. **BALF Cells:** Presence of PMNs was limited to the mid and high dose groups. Overall maximal response was reached in mice and hamsters, but not in rats with increasing mass dose of HSCb.
Reference: Evans et al. (2006, 097066) **Species:** Rat **Gender:** Male **Strain:** Sprague-Dawley	DEP: collected under dry, outdoor, ambient conditions from tractor exhaust pipe (1985, Japanese ISEKI 1500 cc tractor) burning Esso 2000 diesel and 20/30 mixture of Esso light engine oil. 10% UF, 90% fine Cabosil: amorphous silicon dioxide 16% UF, 84% fine **Particle Size:** DEP: 30nm; Cabosil: 7nm	**Route:** IT Instillation **Dose/Concentration:** 1mg/rat DEP; 1mg/rat Cabosil **Time to Analysis:** Pretreatment with 0.5 unit of bleomycin; IT 3 or 7d after pre-treatment; 1wk post-IT	**Lung permeability:** In bleomycin-treated group, obvious inflammatory status and edema within the lung was observed. This was shown by significant increases in acellular protein and free cells. **Changes in lung:** Body weight ratio, lung surface protein content, free cell counts, and apical surface protein of rat type I cells were only altered by bleomycin treatment and not particle exposure.
Reference: Finnerty et al. (2007, 156434) **Species:** Mouse **Gender:** Male **Strain:** C57BL/61 **Age:** 12 wks **Weight:** 24.3 ±0.3g	Coal Fly Ash (generated at U.S EPA National Risk Management Research Laboratory by burning Montana subbituminous coal under conditions simulating full-scale utility boiler conditions) Transition metals of Coal Fly Ash: Fe, Mg, Ti, Mn, V **Particle Size:** $>PM_{2.5}$	**Route:** IT Instillation **Dose/Concentration:** PM: 200mg PM/mouse; 9.1 mg/kg PM+LPS10: 200mg PM+10mg LPS PM+LPS100: 200mg PM+100mg LPS LPS: 100 μg **Time to Analysis:** Parameters measured 18h post single exposure	**BALF Cells:** No significant differences in platelet concentration or white blood cell count in any groups were observed. The percentage of neutrophils increased significantly with PM+LPS100. PMN rose in PM groups and increased further with LPS treatment. Increases in PM+LPS were groups statistically significant. More leukocytes were present in the alveolar space in PM+LPS10 compared to the PM group. The most severe response was in the PM+LPS100 group. **Cytokines:** Plasma TNF-α and IL-6 significantly increased for the PM+LPS100 group. An additive effect of LPS and PM for IL-6 was observed. For saline and PM groups, pulmonary TNF-α was below detection range. A synergistic effect for TNF-α was observed. A less than additive effect for IL-6 was observed. Pulmonary TNF-α significantly increased in the PM+ LPS100 group. Pulmonary IL-6 significantly increased in both PM+LPS groups.
Reference: Fujimaki et al. (2006, 096601) **Species:** Mouse **Gender:** Male **Strain:** IL-6(-/-) and WT: B6J129Sv (control) **Age:** 5-6wks	DEP: collected from a 4-cylinder, 2.74 L, Isuzu diesel engine **Particle Size:** 0.4 μm	**Route:** Whole-body Inhalation **Dose/Concentration:** 1.0, 3.0 mg/m^3 **Time to Analysis:** 12 h/d for 4wks. Parameters measured 1d post-exposure	**BALF Cells:** Treatment significantly increased BAL cells from WT mice at both dose levels. The increase of macrophages and neutrophils were dose-dependent. An increase in lymphocytes were present in WT mice with the low dose. No significant increase in cells were observed from IL-6 (-/-). **Cytokines:** TNF-α largely increased in IL-6(-/-) mice exposed to 3 mg/m^3 compared to WT mice. IL-6 production increased in WT mice exposed to 3 mg/m^3. CCL3 increased in both WT and IL-6(-/-) at high dose. IL-1β remained at the control level.

Reference	Pollutant	Exposure	Effects
Reference: Gerlofs-Nijland et al. (2005, 088652) **Species:** Rat **Gender:** Male **Strain:** SH/NHsd **Age:** 11-12wks **Weight:** 250-350g	RTD: road tunnel dust (obtained from a Motorway tunnel in Hendrik-Ido-Ambacht, Netherlands) EHC-93 (Ottawa, Canada) **Particle Size:** Coarse: 2.5 - 10 μm; fine: 0.1- 2.5 μm	**Route:**IT Instillation **Dose/Concentration:** 0.3, 1, 3, 10 mg/kg EHC-93 10 mg/kg **Time to Analysis:** Parameters measured 4, 24, 48h post single exposure	**BALF Cells:** PMN significantly increased in RTD (3 and 10 mg/kg dose) and EHC-93 exposed animals at 24 h and decreased by 48h but remained statistically significant. AM numbers decreased for 3 mg/kg RTD group at 4h. **BALF Inflammatory/Injury Markers:** Myeloperoxidase (measured at 24 h in 1, 3, 10 mg/kg RTD groups) was elevated in a dose-dependent manner. RTD induced time-dependent increases in LDH activity at 24 and 48h, although these increases were less than EHC-93 values at the same time points. Alkaline phosphatase increased dose-dependently for RTD at 48h. GSH decreased at 24 h to approximately the same levels in 0.3, 1, and 3 mg/kg RTD dose groups. Uric acid only decreased in 1 mg/kg RTD group at 24 h. **Cytokines:** IL-6 levels were elevated only at 10 mg/kg for RTD and EHC-93 at 4 and 24 h; it remained elevated for EHC-93 at 48h. A dose-dependent increase in TNF-α at 4h for RTD was observed. TNF-α levels remained elevated only for the 10 mg/kg groups at 24 h and returned to control levels by 48h. A dose-dependent increase in MIP-2 for all RTD dose groups were observed and remained elevated through 48h for both PM types (although values were returning to control levels). **Hematology:** No significant changes in plasma for bigET-1 or von Willebrand factor were observed. At the highest dose, fibrinogen levels significantly increased at 24 and 48h for both PM types. **Pulmonary histopathology:** A dose-dependent increase in the number of inflammatory foci at 24 and 48h for 3 and 10 mg/kg RTD groups was observed. The response was even greater for the EHC-93 exposed group at similar time points.

Reference	Pollutant	Exposure	Effects
Reference: Gerlofs-Nijland et al. (2007, 097840) **Species:** Rat **Gender:** Male **Strain:** SH/NHsd **Age:** 13wks **Weight:** 250-350g	PM samples collected from: 1. MOB high traffic density 2. HIA high traffic density 3. ROM high traffic density 4. DOR moderate traffic density 5 MGH low traffic density 6 LYC low traffic density **Particle Size:** Coarse: 2.5 - 10 μm; Fine: 0.1 - 2.5 μm	**Route:** IT Instillation **Dose/Concentration:** 3, 10mg/kg **Time to Analysis:** 24h	**BALF Inflammatory/Injury Markers:** LDH was significantly increased for all doses of coarse PM and for the high dose of fine PM. BALF protein concentration was observed predominantly at the high dose of coarse PM. Location ROM had evidence of attenuated responses with fine PM. Ascorbate concentrations were reduced but were only significant for rats exposed to the highest dose of coarse PM fractions from the locations MOD, HIA, and LYC. **BALF Cells:** Pulmonary inflammation was induced in a significant and dose-dependent manner for both dose levels. Inflammation in the BALF included airway neutrophilia, increased macrophage numbers and mild lymphocytosis. Both coarse and fine PM caused dose-dependent aveolitis. Fine PM from LYC (10mg/kg dose) also caused some bronchiolitis. **Cytokines:** TNF-α concentrations increased for all coarse samples with the exception of DOR and LYC. Fine PM induced similar responses for all sites. MIIP-2 concentrations increased only at certain sites for coarse but not fine PM. **Hematology:** Fibrinogen responses of SH rats increased significantly at the high dose of both fractions of all PM samples, except fine PM from DOR. **Location-related differences:** Coarse PM from MOB, HIA and MGH induced higher LDH responses than other locations. Coarse PM from HIA produced BALF protein concentrations higher than LYC and ROM. MGH induced greater amounts of BALF protein than ROM. Coarse PM from LYC lowered fibrinogen values more than PM from location MOB, HIA, and MGH. Fine PM showed less differences among the various sites. **Particle Correlation:** Fine PM exhibited significant correlation between zinc content and BALF cytotoxicity markers protein and LDH - mainly from HIA. Fine PM also exhibited positive correlations with copper and barium. Coarse PM showed positive correlation with barium and copper, mainly from MOB.
Reference: Gerlofs-Nijland et al. (2009, 190353) **Species:** Rat **Gender:** Male **Strain:** SH **Age:** 12wks **Weight:** 200-300g	PM (Prague, Czech Republic; Duisburg, Germany; Barcelona, Spain) (Prague and Barcelona coarse PM organic extracts) **Particle Size:** Coarse: 2.5-10 μm, Fine: 0.2-2.5 μm	**Route:** IT Instillation **Dose/Concentration:** 7mg PM/kg body weight **Time to Analysis:** DTPA added to some PM samples preinstillation. Instilled with PM. Necropsy 24h postexposure.	Inflammation (LDH, protein, albumin), cytotoxicity (NAG, MPO, TNF-α), and fibrinogen were increased by PM, and were greatest in the coarse PM fraction. Metal-rich PM had greater inflammatory and cytotoxic effects, and increased fibrinogen and vWF and decreased ACE. PAH content influenced greater inflammation (including neutrophils), cytotoxicity, and fibrinogen. Generally, whole PM and coarse PM were more potent than organic extracts and fine PM, respectively.
Reference: Ghio et al. (2005, 088272) **Species:** Rat **Gender:** Male **Strain:** N8 b/b Belgrade rats and N8+ Ib Belgrade controls	Oil Fly Ash (Southern Research Institute, Birmingham, AL) **Particle Size:** 1.95 ± 0.18 μm (MMAD)	**Route:** IT Instillation **Dose/Concentration:** 500μg/rat; 2mg/kg **Time to Analysis:** 24h	**BALF Cells:** Homozygous Belgrade with mutation G185R had higher levels of Fe and V 24 h post-exposure. This may demonstrate a decreased ability to remove Fe and V from the lower respiratory tract than heterozygous +Ib littermates. This also indicates that DMT1 is normally responsible for at least some Fe and V uptake; thus, a defective DMT1 transports less. **BALF Inflammatory/Injury Markers:** Increased protein and LDH concentrations in the homozygous strain were observed when compared to control

Reference	Pollutant	Exposure	Effects
Reference: Ghio et al. (2005, 088275) **Species:** Rat **Gender:** Male **Strain:** Sprague-Dawley **Age:** 60d **Weight:** 250-300g	Ferric ammonium citrate (FAC) Vanadyl sulfate ($VOSO_4$) **Particle Size:** NR	**Route:** IT Instillation **Dose/Concentration:** 0.5 mL 100 μm FAC/rat; 0.5 mL 10 μm $VOSO_4$/rat; 500 μg oil fly ash; 2 mg/kg **Time to Analysis:** Single or double exposure with 24 h rest period. Parameters measured 15, 30, 60 min, 24h post-exposure.	**DMT1 immunohistochemistry and lung injury:** FAC increased and $VOSO_4$ decreased –IRE DMT1 staining. Same exposures had no effect on +IRE DMT1. –IRE DMT1 expression in macrophages, airway and alveolar epithelial cells increased with increased Fe exposure. Vanadium nearly eliminated staining except in alveolar macrophages. Increased metal clearance with pre-exposure to FAC. Less metal clearance with pre-exposure to $VOSO_4$. Pre-exposure to iron diminished lung injury whereas pre-exposure to vandium increased lung injury after oil fly ash instillation. Lung injury measured by concentration of protein and LDH in BAL.
Reference: Gilmour et al. (2007, 096433) **Species:** Mouse **Gender:** Female **Strain:** BALB/c **Age:** 10-12wks **Weight:** 20-22g	PM - CO, FI, UF (obtained from U.S. Seattle (S), Salt Lake City (SL), South Bronx (SB), Sterling Forest (SF)) SB: included 35% sulfate, 22% gasoline, diesel and brake wear. SF: 48% sulfate. SL: 34% wood combustion and 28% sulfate S: 39% wood combustion and 29% sulfate Residual oil combustion and soil dust less than 5% for all sites. **Particle Size:** CO: 2.5-10μm; FI: ◘ 2.5 μm; UF: ◘ 0.1μm	**Route:** Oropharyngeal Aspiration **Dose/Concentration:** 25μg or 100μg PM; 1.25 or 5 mg/kg **Time to Analysis:** 18h	**BALF Inflammatory/Injury Markers:** Seattle CO fractions showed no dose-dependent effect on protein concentration. Results for other locations were distinctly higher with 100 μg dose than 25 μg and saline doses. SL CO high dose induced the most significant increase. LDH response was weakly dose-related. Only SB showed a statistically significant increase for LDH with the high dose UF. **BALF Cells:** PMN increased with the high dose of CO samples from SB, SL, S, but not SF. No significant increases from FI were observed, though the high dose induced increased PMN. UF from SL caused a highly variable response. **Cytokines:** MIP-2 was similar to PMN response. SB CO induced the most significant response. SL UF was highly variable. **Particle Characteristics:** LPS was higher in S (CO, FI, UF) and SL (CO, FI, UF). Zn levels were highest in SB (CO, FI, UF). Fe was higher in all CO and FI samples with SB CO inducing the highest.
Reference: Gilmour et al. (2004, 057420) **Species:** Mouse **Gender:** Female **Strain:** CD1 **Age:** 8-10wks **Weight:** 20-25g	Coal Fly Ash MU: Montana Ultrafine MF: Montana Fine MC: Montana Coarse KF: W.Kentucky Fine KC: W. Kentucky Coarse Particle Characteristics: Montana Sulfur 0.83%, Ash 11.72%. Trace amounts of Ba, P, Sr, V, Nb, Cd, Se, Ga, Cu. Depleted in Si, Al, Fe, Mg, Ti. Kentucky Sulfur 3.11%, Ash 8.07% **Particle Size:** Coarse: >2.5 μm; Fine: <2.5 μm; Ultrafine: <0.2 μm	**Route:** Oropharyngeal Aspiration **Dose/Concentration:** 25ug or 100μg/mouse **Time to Analysis:** 18h	**BALF Cells:** PMN highly increased for MU at both doses. The level was comparable to the positive control. PMN also increased with KF at high dose. Coarse particles caused no significant increase in PMN. Number of macrophages did not change, but NAG increased significantly with MU for both dose levels and with KF and MF at high dose level. **BALF Inflammatory/Injury Markers:** Total protein and LDH was not significantly elevated. Albumin concentration increased significantly after treatment with the fine high dose of both particle types. **Cytokines:** MU particles caused a significant increase in TNF-α. MIP-2 increased in all fine and ultrafine PM-instilled animals with the highest in the MU and KF at both doses. IL-6 was detectable only in the BALF of MU and KF with substantial variability. The IL-6 levels were not significant.

Reference	Pollutant	Exposure	Effects
Reference: Gilmour et al. (2004, 087948) **Species:** Rat **Gender:** Male **Strain:** SH/NQIBR, WKY **Age:** 12wks **Weight:** 280-340g	PM (collected from precipitator unit of an oil burning power plant in Boston) Measured Components of PM: S, Zn, Ni, V, Al, Cu, Pb, Fe, Ca, Na, K, Mg, Endotoxin **Particle Size:** NR	**Route:** IT Instillation **Dose/Concentration:** 0.0, 0.83, 3.3, and 8.3 mg/kg in SH rats; 0.0 or 3.3 mg/kg in WKY and SH rats **Time to Analysis:** 24h	**BALF Inflammatory/Injury Markers:** LDH activity increased in a dose-related manner; this was observed in SH rats after exposure to 0.83, 3.33 and 8.3 mg/kg PM. SH rats showed greater lung permeability following PM exposure than WKY rats. SH rats showed acute lung inflammatory response after exposure to PM when compared to WKY rats. **BALF Cells:** No increase in macrophage number was observed in either rat strain following saline or PM exposure at 24 h. **Cytokines:** MIP2 mRNA expression increased significantly in SH PM exposure group only. No significant differences in TNF-α RNA expression in either WKY, SH rats or control treatment groups were observed. **CD14:** A significant increase in lung CD14 protein was observed only in SH rats exposed to PM. **TLR4:** A significant increase in TLR4 protein in SH rats exposed to PM was observed. **NF-kB:** A significant increase in NF-kB binding protein in the nuclei of SH rats exposed to PM was observed. This effect was not observed in the control of PM-exposed WKY rats.
Reference: Gilmour et al. (2004, 054175) **Species:** Rat **Gender:** Male **Strain:** Wistar **Age:** 12wks	ufCB: Ultrafine carbon black (Printex 90 (Degussa) CB: (Huber 990, HR. Haeffner and Co) **Particle Size:** ufCB: 14nm; CB: 260nm (primary particle diameter)	**Route:** Whole-body Inhalation **Dose/Concentration:** ufCB: 1.66 mg/m^3 fCB: 1.40 mg/m^3 Number concentrations ufCB: 52380 particles/cm^3 fCB: 3800 particles/cm^3 **Time to Analysis:** Exposed for 7 h. Sacrificed 0, 16 or 48h post-exposure.	**BAL Cells:** Total number of cells increased significantly in UfCB-exposed rats at 0 and 16 h. Recruitment of cells did not occur in response to CB exposure. PMNs increased significantly in the BALF of ufCB-exposed rats at 16 h. Leukocytes remained unchanged following CB exposure but increased significantly at 0 and 48 h post exposure to ufCB. **Cytokine mRNA:** A significant increase in BALF MIP-2 mRNA expression was observed at 48 h. No differences in MIP-2 mRNA levels were observed in the whole lung tissue.
Reference: Godleski et al. (2002, 156478) **Species:** Rat **Gender:** Male **Strain:** SD **Age:** NR **Weight:** 200-250g	CAPs (Boston; Harvard Ambient Particle Concentrator) **Particle Size:** 0.27 ± 2.3 μm (diameter)	**Route:** Inhalation **Dose/Concentration:** 73.5-733 μg/m^3 **Time to Analysis:** Exposed 5h/d, 3d (consecutive). BAL 24h postexposure	PMNs significantly increased with CAPs exposure and also in relation to CAPs mass, Br, SO_4^{2-}, EC, OC and Pb. An overall increase in pro-inflammatory mediators and decrease in immune enhancer and evidence of vascular endothelial responses occurred with CAPs exposure.
Reference: Gottipolu et al. (2009, 190360) **Species:** Rat **Gender:** Male **Strain:** Wistar Kyoto (WKY), SH **Age:** 14-16wks **Weight:** NR	DE (30-kW (40hp) 4-cylinder indirect injection Deutz diesel engine) (O_2- 20%, CO- 1.3-4.8ppm, NO- < 2.5-5.9ppm, NO_2- < 0.25-1.2ppm, SO_2- 0.2-0.3ppm, OC/EC- 0.3 ± 0.03) **Particle Size:** Number Median Diameter: Low- 83 ± 2nm, High- 88.2nm; Volume Median Diameter: Low- 207 ± 2nm, High- 225 ± 2nm	**Route:** Exposure Chamber **Dose/Concentration:** Low- 507 ± 4 μg/m^3, High- 2201 ± 14 μg/m^3 **Time to Analysis:** Exposed 4h/d, 5d/wk, 4wks. Necropsied 1d postexposure.	DE increased neutrophils in a concentration-dependent manner, and GGT activity at the high dose. Particle-laden macrophages were found in DE-exposed rats. DE dose-dependently inhibited mitochondrial aconitase activity. DE caused 377 genes to be differentially expressed within WKY rats, most of which were downregulated, but none in SH rats. However, WKY rats had an expression pattern shift that mimicked baseline expression of SH rats without DE. These genes regulated compensatory response, matrix metabolism, mitochondrial function, and oxidative stress response.
Reference: Gunnison and Chen (2005, 087956) **Species:** Mouse **Gender:** Male **Strain:** DK ($ApoE^{-/-}$, $LDLr^{-/-}$) **Age:** 18-20wks	CAPS (Northeastern regional background) Ambient air copollutants measured O_3, NO_2 **Particle Size:** 389 ± 2nm	**Route:** Whole-body Inhalation **Dose/Concentration:** CAPS = 131 ± 99 μg/m^3 including O_3 = 10 ppb and NO_2 = 4.4 ppb **Time to Analysis:** 6h/d, 5d/wk for 4m (5/12/03-9/5/03). Sacrified 3-4d post exposure.	**Microarray Data:** 13 genes in the heart tissue and 47 genes in the lung tissue were identified as possibly affected. Strict standards (1.5 fold response, 10 % false discovery rate) resulted in responses by only 1/13 genes (Rex3 - no known heart physiology) in the heart tissue and 0/47 genes in the lung tissue. Using more liberal response (nonstatistical) standards (1.5 fold only) and comparison of each CAPS animal with all 3 control animals (3x3 array) resulted in possible effects on 7 additional genes in the heart tissue and 37 genes in the lung tissue.

Reference	Pollutant	Exposure	Effects
Reference: Gurgueira et al. (2002, 036535) **Species:** Rat **Gender:** Male **Strain:** Sprague-Dawley **Weight:** 250-300g	CAPs (Harvard Ambient Particle Concentrator) CB (C198 Fischer Scientific, Pittsburg, PA USA) Composed of 85.9± 0.2% Carbon, 13.0± 0.2% O2, 1.17± 0.2% Sulfur ROFA (Boston, MA USA oil-fired power plant) **Particle Size:** CAPs: 1-2.5 μm; CB: $<$2.5 μm; ROFA: $<$2.5 μm	**Route:** Whole-body Inhalation **Dose/Concentration:** 300 ± 60 $\mu g/m^3$ **Time to Analysis:** 1, 3, 5h CAPs Exposure followed by immediate post-exposure analysis. 5h CB, immediate analysis. 30min ROFA, Immediate analysis.	**In situ Chemiluminescence(CL):** Data show a significant increase in lung and heart CL at 5h. Lung CL increased linearly with time of exposure. **Oxidants:** CAPs-initiated oxidative stress was not detectable in those rats allowed to recover in room air after the simulated "peak" in particulate air pollution. Rats breathing particle-free filtered air for 3 days had significantly lower levels of oxidants. Exposure to inert CB did not exert oxidant effects on the heart and lung. **BALF Inflammatory/Injury Markers:** The water content of the lung and heart increased significantly upon exposure to CAPs but not to filtered air and increased as a function of length of exposure. Rats breathing CAPs also showed increases in LDH and CPK as a function of length of exposure. **Antioxidant enzymes:** Data showed an increase in SOD and catalase activities in both the lung and heart. The pattern of increase was tissue specific.
Reference: Hamoir et al. (2003, 096664) **Species:** Rabbit **Strain:** New Zealand **Age:** 12-16 wks **Weight:** 2.8 ± 0.5kg	PSC: Polystyrene particles, Carboxylate modified, 3 types PSA: Polystyrene particles, Amine modified, 1 type **Particle Size:** PSC: 24, 110 or 190nm (PSC24, PSC110, PSC190); PSA: 190nm	**Route:** IT Instillation **Dose/Concentration:** PSC24: 0.04 or 4 mg/rabbit PSC110, PSC190, PSA190: 4mg/rabbit **Time to Analysis:** 0, 30, 60, 90, 120min	**Capillary Filtration Coefficient:** A time-dependent increase correlating to total number of particles/surface area, not particle size, was observed.PSA induced a significant increase in microvascular permeability as compared to PSC. This suggests that the number of particles exposed should be considered an important parameter for measuring air quality rather than total particle surface area.

Reference	Pollutant	Exposure	Effects
Reference: Happo et al. (2007, 096630) **Species:** Mouse **Gender:** Male **Strain:** C57BL/6J **Weight:** 19-30g **Age:** 10-11wks	PMC (Coarse) PMF (Fine) PMUF (Ultrafine) Collected in 6 European cities: Duisburg, Prague, Amsterdam, Helsinki, Barcelona, Athens **Particle Size:** PMC: $PM_{10\text{-}2.5}$; PMF: $PM_{2.5\text{-}0.2}$; PMUF: PM0.2	**Route:** IT Instillation **Dose/Concentration:** PMC: 5.9-29.6 $\mu g/m^3$; PMF: 8.3-25.2 $\mu g/m^3$; PMUF: 2.7-6.7 $\mu g/m^3$ Dose-response: 1, 3, 10mg/kg Time course: 10 mg/kg **Time to Analysis:** 1. Dose-Response study: parameters measured 24h post exposure. 2. Time course study: parameters measured 4, 12, 24 h post single exposure (at 10 mg/kg).	**Total Cell Numbers:** 1. For the dose-response study, all the PMC samples exhibited dose-dependent increases of total cell numbers. The 3 and 10 mg/kg doses of PMC induced statistically significant increases. At 10 mg/kg, only 2/6 samples induced statistically significant increases. No PMUF samples induced effects at any dose. 2. For the time-response study, no increases in cell numbers were shown at 4 h. Though the levels induced by PMC at 24 h were lower than at 12 h, both levels were statistically significant. PMF induced statistically significant increases only at 12 h for 4/6 samples. PMUF induced only 1 significant increase at 12 h; the 24 h time point was not tested. **Total Protein/LDH:** 1. The lower doses of 1 and 3 mg/kg did not induce significant increases in any of the PM samples, except for PMUF-Athens. All 6 samples of PMC, at 10 mg/kg, induced significant increases. At 10 mg/kg, 4/6 PMF samples induced significant increases. 2. At 4 h, none of the samples increased protein concentration. The PMC samples, excluding Prague, induced significantly higher concentrations at 12 h. At 24 h, only 3/6 PMC samples induced significant increases. Only 2 PMF samples induced significant increases at 12 and 24 h. At 12 h, effects induced by PMUF were minimal and inconsistent; the 24 h time point was not tested. **Cytokine:** 1. Only PMC induced dose-dependent responses that reached statistical significance at 10 mg/kg. PMF and PMUF induced minimal and inconsistent responses. **TNF-α levels:** 2. TNF-α levels increased significantly at 4 and 12 h by PMC. At 24 h, TNF-α levels returned to near control levels. PMF, at 4 h, induced statistically significant increases for 3/6 samples and significant increases in 2/6 samples at 12 h. No PMUF samples significantly increased TNF-α levels. **IL-6:** 2. PMC induced the highest IL-6 levels at 4 h. Levels at 12 and 24 h were reduced with 6/6 and 3/6 samples showing statistically significant increases, respectively. PMF showed a similar trend with 4 h inducing the highest levels that were reduced at 12 and 24 h. Of the PMUF samples, only the Helsinki and Duisburg samples induced statistically significant results at 4 and 12 h. Generally, the PMUF responses were negligible when compared to PMC and PMF. **KC production:** 2. All PMC samples induced the highest levels at 4 h. At 12 and 24 h, levels were reduced but 4/6 samples induced statistically significant levels. PMF showed a similar trend- the highest levels were induced at 4 h (in 3/6 samples). PMUF at 4 h showed small, though not significant, increases. At 12 h, only 2 samples showed statistically significant differences from the control; the 24 h time point was not tested.
Reference: Harder et al. (2005, 087371) **Species:** Rat **Gender:** Male **Strain:** Wistar Kyoto **Age:** 14-17wks **Weight:** NR	Carbon UFP **Particle Size:** Diameter: 37.6 ± 0.7nm	**Route:** Exposure Chamber **Dose/Concentration:** 180 $\mu g/m^3$ **Time to Analysis:** Telemeter implanted into peritoneal cavity. 10d recovery. 3d baseline reading. 24h exposure. 3d recovery.	Carbon UFP mildly but significantly elevated HR compared to the control. SDNN was significantly decreased during exposure. UFP induced mild pulmonary inflammation, significantly increased PMN, and increased the total protein and albumin concentrations. Particle-laden macrophages sporadically accumulated in the alveolar region.

Reference	Pollutant	Exposure	Effects
Reference: Harkema et al. (2004, 056842) **Species:** Rat **Gender:** Male **Strain:** F344, BN **Age:** 10-12wks **Weight:** NR	CAPs (Detroit; July-Sept. 2000; Harvard Ambient Fine Particle Concentrator) **Particle Size:** 2.5 μm (diameter)	**Route:** Inhalation Exposure Chamber. IT Instillation. **Dose/Concentration:** 4d concentration: 676 ± 288 μg/m^3, 5d concentration: 313 ± 119 μg/m^3, July concentration: 16-185 μg/m^3, September concentration: 81-755 μg/m^3; IT Instillation- 200 μL (soluble and insoluble) **Time to Analysis:** F344 rats sensitized to endotoxin, BN rats to OVA. Exposed 10h/d 1, 4, 5d (consecutive). Another group of rats i.t instilled. Both groups killed 24h postexposure.	The retention of PM in the airways was enhanced by allergic sensitization. Recovery of anthropogenic trace elements was greatest for CAPs-exposed rats. Temporal increases in these elements were associated with eosinophil influx, BALF protein content and increased airway mucosubstances. A mild pulmonary neutrophilic inflammation was observed in rats instilled with the insoluble fraction but instillation of total, soluble or insoluble $PM_{2.5}$ in allergic rats did not result in differential effects.
Reference: Hiramatsu et al. (2003, 155846) **Species:** Mouse **Gender:** Female **Strain:** BALB/c and C57BL/6 **Age:** 8wks **Weight:** 17-22g	DE: generated by 2369-cc diesel engine (Isuzu) at 1050 rpm and 80% load with commercial light oil **Particle Size:** NR	**Route:** Whole-body Inhalation **Dose/Concentration:** DEP: 100μg/m^3 or 3 mg/m^3; SO_2 < 0.01ppm; NO_2 2.2 ± 0.3 or 15 ± 1.5 ppm; CO 3.5 ± 0.1 or 9.5 ± 0.6 ppm **Time to Analysis:** 7h/d, 5d/wk for 4 or 12wks, Immediate	**BALF Cells:** Alveolar macrophages (AMs) increased dose-dependently at 30 and 90d. High DE exposure resulted in bronchus-associated lymphoid tissue (BALT) around DEP-AMs; this was less conspicuous in C57BL/6 than in BALB/c mice. B- and T-cell populations were found in the BALT with no significant differences observed between the strains. Lympohocytes and neutrophils increased time- and dose-dependently with agreater increase in BALB/c than C57BL/6 observed. No eosinophils or basophils were observed. Mac-1-positive cells exposed to high DE levels increased in both strains at 1 month (33.8%) and 3 months (20.3%) vs. low dose group (5.3 and 7% respectively). **Cytokines:** At 30d, TNF-α, IL-12p40, IL-4 and IL-10 mRNA increased, IL1b and iNOS decreased. IFNγ increased in BALB/c but decreased in C57BL/c. IL-6 mRNA was not affected. At 90d, IL-4 and IL-10 mRNA similarly increased in C57BL/6 mice exposed to low DE level but decreased at high DE level.
Reference: Hollingsworth et al. (2004, 097816) **Species:** Mouse **Gender:** Male **Strains:** C57BL//6$^{TLR+/+}$, C57BL//6$^{TLR-/-}$ **Age:** 8-9 wks	ROFA **Particle Size:** NR	**Route:** Oropharyngeal Aspiration **Dose/Concentratin:** 50 ul of 1μg/mL suspension per mouse **Time to Analysis:** Parameters measured post single exposure of 6 and 24h.	**TLR4-Knockout vs Wild type:** There were no observed differences. **Methacholine sensitivity:** No ROFA effect was observed in wild type or knockout mice. **BALF Cells:** ROFA increased total cell number. Total number of neutrophils with lavage fluid increased 24 h post-exposure in both strains.
Reference: Hutchison et al. (2005, 097750) **Species:** Rat **Gender:** Male **Strain:** Wistar **Age:** 3m **Weight:** 250-300g	PM_{10} United Kingdom samples collected before (-B), during closure (-C) and reopening of steel plant (-R) PMT = PM total (aqueous sonicate) PMS = PM aqueous supernatant PMI = PM insoluble pellet **Particle Size:** PM_{10}	**Route:** IT Instillation **Dose/Concentration:** 112 to 180 μg PM in 500 ul; 0.44-0.72 mg/kg **Time to Analysis:** 18h	**BALF Cells:** PMT-R neutrophil cell number and percentage were significantly higher than PMT-C or control. PMS-R and PMI-R were also higher than their respective controls. The neutrophil cell numbers induced by PMI-R were greater than PMI-C and the control. Total cell count unchanged. **BALF Inflammatory/Injury Markers:** Only albumin increased after PMT-R. Upon exposure, total protein and LDH did not increase. **Cytokine mRNA expression:** Only PMT-R increased IL-1ß mRNA expression. No effects on TNF-α and TGFß expression levels were observed. IL-6, MIP2, and GM-CSF mRNA was not detected in BAL cell extracts from either the control or treated groups.
Reference: Inoue et al. (2006, 097815) **Species:** Mouse **Gender:** Male **Strains:** C3H/HeJ (TLR-4 point mutant) and C3H/HeN (Control) **Age:** 6 wks	DEP (derived from 4 cyl, 2.74l light duty diesel engine) **Particle Size:** NR	**Route:** IT Instillation **Dose/Concentration:** 12mg/kg of bw **Time to Analysis:** 24h	**Airway Inflammation:** DEP induced an increase in total cells ($P < 0.01$), neutrophils ($P < 0.01$) and mononuclear cells in BALF. TLR4 knockout mice (C3H/HeJ) showed a much lower response. **Cytokines:** DEP induced a massive increase in MIP-1x, IL-1β and KC. However, levels of MIP-1x were significantly less in the knockout than the wild type while levels of IL-1β and KC were significantly higher in knockouts than the wild type. **Hematology:** DEP increased plasma fibrinogen in both strains but with a greater increase in the knockout mice than the wild type.

Reference	Pollutant	Exposure	Effects
Reference: Inoue et al. (2006) **Species:** Mouse **Gender:** Male **Strain:** NC/Nga **Age:** 10wks	DEP (derived from 4 cyl, 2.74l light duty diesel) **Particle Size:** NR	**Route:** IT Instillation **Dose/Concentration:** 100 μg/mouse **Time to Analysis:** 1/wk for 6wks. Parameters measured 24h after last administration	**BALF Cells:** DEP significantly increased total cells, neutrophils and mononuclear cells but did not induce an effect on eosinophils. **Cytokines:** DEP increased IL-4, KC and MIP-1. The increase in IL-5 was not statistically significant.
Reference: Ishihara et al. (2003, 096404) **Species:** Rat **Gender:** Male **Strain:** Wistar **Age:** 5wks	DE (from 2 engines, produced on site) ·L = low level DE ·M = medium level ·MG = DE w/o particulates ·HR = high level Measured Components: NO_2, SO_4, SO_2, CO, CO_2, NO_X, NO, HTHC, HCHO, O2 **Particle Size:** L: 0.33 -0.50 μm M: 0.35 - 0.40 μm HR: 0.42 - 0.45 μm	**Route:** Whole-body Inhalation **Dose/Concentration:** L: 0.18 - 0.21 mg/m^3 M: 0.92- 1.18 mg/m^3 MG: 0.01 mg/m^3 HR: 2.57 - 2.94 mg/m^3 **Time to Analysis:** 16h/d, 6d/wk, for 6, 12, 18 & 24 m. Parameters measured immediately following last exposure.	**Morbidity and Mortality:** Weight gain in HR group was less than other groups at 18 and 24 mo. This indicates a significant difference between the HR and C group. Mortality during the study was frequent. C group experienced an 8% mortality rate, L group 12%, M group 15%, MG group 12% and HR group 23%. **BALF Inflammatory/Injury Markers:** Significant differences were seen among groups with respect to number of total cells and percentages of cell differential, total protein, fucose, sialic acid, phospholipid and prostoglandin E2. Total protein increase was observed in both M and HR dose groups with the HR group increasing time-dependently. **BALF Cells:** The HR group showed a significant increase in total cell count from 6 to 18mo. The percentage of PMN increased at 6mo in M, MG and HR group. M group lymphocytes significantly increased at 6, 12, and 24mo of exposure. Macrophages decreased at 6mo for the M and HR groups. **Mucus and Surfactant:** The HR group showed a significant increase from 12 to 18mo.
Reference: Jones, HR.A. Hamacher, K. Clark, J.C. 2005 **Species:** Rabbit **Strain:** New Zealand **Weight:** 2.5· 3.5kg	ASP: Amorphous silica particles (Hypersil) MCSP: Microcrystalline silica particles **Particle Size:** ASP: 5 μm; MCSP: 5 μm	**Route:** Intrapulmonary Instillation (Right upper lobe of lung) **Dose/Concentration:** 50mg in 0.5mL saline **Time to Analysis:** Parameters measured at varying times from 6h to 91d post treatment.	**MCSP:** At 6h, neutrophils increased. Macrophages increased 3 fold. At 60h, neutrophils were pyknotic and the lungs displayed a thickened interstitium containing silica particles. At 5d, collagen deposition appeared. AT 8d, fibroblastic activity and necrosis were observed. At 15d, aggregation of silica particles and necrotic debris were apparent. AT 8 wks, fibroblasts were still present. At 13 wks, active scarring and raised neutrophil macrophage counts were still present. **ASP:** At 15h, neurophils increased. Macrophages tripled and remained increased for 3wks. At 4d, macrophages bore particles. At 13d, neutrophils decreased significantly. By 25d, silica spheres were gradually removed from lungs. **PET Scanning:** 18F-fluoroproline showed increased activity beginning at 14d and peaking at 41-54d (left lung control vs right lung challenged). **Microautoradiography:** 3h-proline at 13 weeks showed radiolabel localization to fibroblasts in the challenged lung

Reference	Pollutant	Exposure	Effects
Reference: Kato and Kagawa (2003, 089563) **Species:** Rat **Gender:** Male **Strain:** Jcl Wistar **Age:** 5wks	Roadside air (Prefectural Tokyo-Danishi-Yokohama highway, Yokohama-Haneda Airport Metropolitan expressway and Satsukibashi-Mizuecho city road, Japan) **Particle Size:** NR	**Route:** Whole-body Inhalation **Dose/Concentration:** Exposed group: 55.7 ppb NO_2, 62.7 $\mu g/m^3$ PM; Control group: 5.1 ppb NO_2, 14.3 $\mu g/m^3$ PM **Time to Analysis:** Exposed for 24, 48, 60 wks. Parameters measured immediately following exposure.	**Respiratory tissue:** Post 24wks, the lung surface was light gray with some BC particle deposits. Post 48-60wks, however, the surface was scattered with particle deposits in addition to its light gray color. **Airway changes:** After 60 wks, no remarkable changes seen in the epithelium. The structure of the airways remained normal. **Cells:** No proliferation or ectopic growth of goblet cells were noted. Mast cells increased in epithelial intercellular space. No mast cell degranulation was observed. Lysosomes increased in cilitated cells post 48wks. Clara cells were unaffected. **Lymph nodes:** Deposition of carbon particles were noted in the trachea and bronchiole-associated lymph nodes post 24wks. **Alveolar changes:** No changes in morphology of broncho-alveolar junctions were noted. Anthracosis observed within alveolar walls and pleura post 24wks and became progressively marked with increased exposure. No change in the number of alveolar holes between exposure and control groups were observed.
Reference: Kato, T. Yashiro, T. Murata, Y. 2003 **Species:** Rat **Gender:** Male **Strain:** Sprague-Dawley **Age:** 7wks **Weight:** 190-220g	Polystyrene latex suspension of latex beads (Japan Synthetic Rubber Co.), uncoated or coated with lecithin **Particle Size:** 240nm	**Route:** IT Instillation with nebulizer **Dose/Concentration:** 5ml of 0.2% suspension administered over 20 min at flow rate of 0.25ml/min **Time to Analysis:** Exposed for 20min. Parameters measured 30min following treatment.	**Alveolar Macrophages:** Following treatment, AMs appeared undamaged. AMs ingested more uncoated than coated beads, but both were ingested. Ingestion of beads differed as coated beads were engulfed individually while uncoated beads were engulfed individually or in aggregates. **Epithelial Cells:** Type I cells incorporated coated beads within a layer of cytoplasm. Type II cells incorporated beads in lamellar bodies. Uncoated beads were not incorporated. **Other:** Neither type of beads were incorporated into endothelial cells, fibroblasts or interstitium of alveolar wall **Monocytes:** Only the coated beads were incorporated by the monocytes. They were found inside and outside phagosomes and lysosomes of monocytes. PMNs did not incorporate any beads.
Reference: Kleinman et al. (2003, 053535) **Species:** Rat **Gender:** NR **Strain:** F344n-NIA **Age:** 22-24m	O_3 CCL: O_3 + Ammonium bisulfate (ABS) + Elemental Carbon (EC) CCH: O_3 + ABS + EC Purified Air (control) **Particle Size:** CCL: 0.30 ± 2.5 μm CCH: 0.29 ± 2.3 μm	**Route:** Nose-only Inhalation **Dose/Concentration:** O_3: 0.2ppm CCL: 50 $\mu g/m^3$ EC + 70 $\mu g/m^3$ ABS + 0.2 ppm O_3 CCH: 100 $\mu g/m^3$ EC + 140 $\mu g/m^3$ ABS + 0.2 ppm O_3 **Time to Analysis:** 4h/d, 3 consecutive d/wk for 4wks	**DNA Replication:** O_3 caused a slight effect of 20-40% increase. CCL and CCH caused between 250 - 340% increase for interstitial and epithelial cells. CCL induced greater reactions than the high dose. **BALF Inflammatory/Injury Markers:** Total protein, mucus glycoprotein and albumin were somewhat elevated in all exposure groups but only reached statistically significance for CCL and protein (very high variability). CCL and CCH both depressed Fc receptor side binding.No effect for O_3 was observed. **BALF Cells:** CCL and CCH induced macrophage respiratory burst activity. The effect induced by O_3 was not significant.
Reference: Kleinman and Phalen (2006, 088596) **Species:** Rat **Gender:** Male **Strain:** Sprague-Dawley **Age:** 6 wks **Weight:** 200g	LO3: Low O_3 HO3: High O_3 LS: Low H_2SO_4 HS: High H_2SO_4 LOLS: Low O_3 + Low H_2SO_4 LOHS: Low O_3 + High H_2SO_4 HOLS: High O_3 + low H_2SO_4 HOHS: High O_3 + high H_2SO_4 **Particle Size:** LS = 0.23 μm±2.3 HS = 0.28 μm±2.1 LOLS = 0.23 μm ±2.3 LOHS = 0.28 μm ± 2.1 HOLS = 0.23 μm ± 2.3 HOHS = 0.28 μm ± 2.1	**Route:** Nose-only Inhalation **Dose/Concentration:** LO3 = 0.30ppm HO3 = 0.61ppm LS = 0.48mg/m^3 HS = 1.00mg/m^3 LOLS = 0.31ppm + 0.41mg/m^3 LOHS = 0.31ppm +1.04mg/m^3 HOLS = 0.60ppm + 0.52mg/m^3 HOHS = 0.60ppm + 0.86mg/m^3 **Time to Analysis:** Exposed for 4h. Parameters measured 42h post-exposure.	**Inflammatory Lesions in Lung Parenchyma:** Neither Type 1 or 2 lung lesions were affected by sulfuric acid alone. HO3 doubled Type 1 lesions and increased Type 2 lesions 25-fold. Additions of H_2SO_4 to O_3 appeared to have a dose-dependent protective effect for both types of lesions. **DNA Synthesis in Nasal, Tracheal and Lung Tissue:** Increased DNA synthesis was observed at all high O_3 exposures but was not affected by coexposure to H_2SO_4. **Macrophage FcR binding:** No effects were observed (no data for LO3 and HO3). **Macrophage Phagocytosis:** All levels of exposure (no data for LO3 and HO3) decreased phagocytosis.

Reference	Pollutant	Exposure	Effects
Reference: Kodavanti et al. (2005, 087946) **Species:** Rat **Gender:** Male **Strain:** WKY and SH/NCrlBR **Age:** 11-14wks	CAPs (EPA, NC) Measured components included Al, Be, Ba, Co, Cu, Zn, Pb, Mn, Ni, Ag, Ti, As. **Particle Size:** 1d: 1.07-1.19 μm; 2d: 1.27-1.48 μm	**Route:** Whole-body Inhalation **Dose/Concentration:** 1d study: 1138-1765 $\mu g/m^3$ 2d study: 144-2758 $\mu g/m^3$ **Time to Analysis:** 4hr (SH only); 4hr/day, 2d (WKY and SH) Post-exposure: 1d: 3h except study #4, 18-20h; 2d: 18-20h	**Breathing Parameters:** In a paired analysis of control SH and treated SH, treated SH showed an increase in expiratory and inspiratory time due to CAPs. The treated and control groups of WKY rats did not show significant differences. **BALF Cells:** In the 2d study, WKY rats showed decreases in total cells; this decrease was associated with decreased macrophages.WKY showed an increase in neutrophils. **BALF Inflammatory/Injury Markers:** Total protein and albumin in WKY rats decreased whereas SH rats maintained the same approximate level. LDH activity lowered slightly in both strains. **Cell Membrane Integrity:** SH rats showed increased GGT (membrane bound enzyme) activity and plasma fibrinogen for 5/7 exposures but these increases did not appear to be dose-dependent. **Cytokines:** Levels were undetermined in SH rats. WKY showed slight increases in IL-6, TNF-α, and MIP-2 but these increases were not statistically significant.
Reference: Kooter et al. (2006, 097547) **Species:** Rat **Gender:** Male **Strain:** SH **Age:** 12-14wks	CAP-F = fine (Site I) CAP-UF = fine + ultrafine (Site II) (Netherlands) Some measured Components: Ammonium, nitrate, sulphate ions: 56± 16% CAP-F mass, 17 ± 6% CAP-UF mass **Particle Size:** 0.15 < CAP-F < 2.5 0.65-0.75 μm CAP-UF < 2.5 0.58-1.41 μm	**Route:** Nose-only Inhalation **Dose/Concentration:** CAP-F 399- 3613 $\mu g/m^3$ CAP-UF 269-556 $\mu g/m^3$ **Time to Analysis:** 6h/d for 2d consecutive, 18h	**BALF Inflammatory/Injury Markers:** Based on unchanged levels of LDH and ALP, no cytotoxicity was noted. No significant change in the levels of total cells were observed. MDA (malondialdehyde) decreased with CAP-UF. Ho-1 increased with CAP-UF and CAP-F. **Cytokines:** CC16 decreased at 457$\mu g/m^3$ of CAP-F and increased at 3613 $\mu g/m^3$ of CAP-F. **Hematology:** WBC and lymphocytes decreased with both CAP-F and CAP-UF. MPV and MPC (mean platelet volume and component) increased with CAP-UF. **BALF Cells:** A decrease in absolute neutrophils as well as percentages of reticulocytes and percentages of neutrophils were observed with CAP-F. Increased percentages of lymphocytes were observed with CAP-F. **Pathology.** No changes were observed.
Reference: Kumar et al. (2004, 096655) **Species:** Rat **Gender:** Male **Strain:** Wistar **Weight:** 150±20g	Fly Ash (Obra Thermal power Station, India) **Particle Size:** PM < 5 μm (90%)	**Route:** Whole-body Inhalation **Dose/Concentration:** 14.4±1.77 mg/m^3 (fluid bed generator) **Time to Analysis:** 4h/d for 28d. Parameters measured immediately following last exposure.	**Lung Weight:** Lung body weight increased 25.58% relative to controls. Total body weight slightly decreased in the treated group. **BALF Inflammatory/Injury Markers:** LDH, GGT, ALP and lavagable protein increased by 140, 450, 160 and 50%, respectively. **BALF Cells:** Only eosinophils(%) increased 95% over controls.Congestion and focal infiltration of monocytes in alveolar area was seen. Fly ash laden macrophages in alveoli combined with hypertrophy of epithelial lining cells was observed.
Reference: Lei et al. (2004, 087999) **Species:** Rat **Gender:** Male **Strain:** Sprague-Dawley **Weight:** 318 ± 8g	CAPs (Yaipei, Taiwan) **Particle Size:** PM: 0.01- 2.5 μm	**Reference:** Nose-only Inhalation **Dose/Concentration:** 371 ± 208 $\mu g/m^3$ **Time to Analysis:** 6h/d for 3d, 5h post-exposure pulmonary function. 2d post-exposure for BALF collection Pulmonary hypertension induced 2wks pre-exposure	**Respiratory Effects:** Decreased respiratory frequency and increased tidal volume for both experimental and control groups were observed. However, only the experimental group levels were statistically significant. There was an increase in airway responsiveness (Penh/methacholine) for CAPs group when compared to the control. **BALF Cells:** A massive increase in total cell number and percent neutrophils was observed. There were no changes in percent macrophages, lymphocytes and eosinophils. **BALF Inflammatory/Injury Markers:** Total protein and LDH increased in the CAPs group. **Cytokines:** TNF-α and IL-6 were not affected.

Reference	Pollutant	Exposure	Effects
Reference: Lei et al. (2004, 087884) **Species:** Rat **Gender:** Male **Strain:** Sprague-Dawley **Weight:** 300-350g	CAPs from Asian dust storm (Taiwan) Measured Components: Si, Al, S, Ca, K, Mg, Fe, As, Ni, W, V, organic carbon, elemental carbon, SO_2, NO_2, nitrate, sulfate **Particle Size:** PM: 0.01- 2.5 μm	**Route:** Nose-only Inhalation **Dose/Concentration:** 315.6 $\mu g/m^3$ (Low) or 684.5 $\mu g/m^3$ (High) **Time to Analysis:** Low: Exposed for 6h. Sacrified 36h post-exposure High: Exposed for 4.5h. Sacrified 36h post-exposure Pulmonary hypertension induced 2wk pre-exposure	**Hematology:** PM induced a dose-dependent increase in WBCs. No change was seen in RBCs. Platelet results were highly variable. **BALF Cells:** PM induced dose-dependent increases in total cells and percentage of neutrophils. No change in macrophages, lymphocytes or eosinphils occurred. Basophils were highly variable. **BALF Inflammatory/Injury Markers:** Dose-dependent increases were observed for total protein and LDH. **Cytokines:** IL-6 increased dose-dependently. (control: 33.5 ± 7.5, LOW 165.1 ± 117.2, 273.6 ± 62.8 pg/mL)
Reference: Li et al. (2007, 155929) **Species:** Mouse **Gender:** Female **Strain:** BALB/c, C57BL/6 **Age:** 9wks **Weight:** NR	DEP (2369-cc diesel engine manufactured by Isuzu Motor, operated at 1050 rpm, 80% load, commercial light oil) **Particle Size:** NR	**Route:** Exposure Chamber **Dose/Concentration:** DEP: 103.1 ± 9.2 $\mu g/m^3$, CO: 3.5 ± 0.1ppm, NO_2: 2.2 ± 0.3ppm, SO_2: < 0.01ppm **Time to Analysis:** Protocol 1: Exposed 7h/d, 5d/wk. Sacrificed at day 0, week 1, 4, 8. Protocol 2: DE alone or DE + NAC 7h/d, 1-5d.	**Airway hyperresponsiveness:** Penh values increased in BALB/c mice compared to the control at day 0, but no significant changes occurred after this time. Penh values increased in C57BL/6 mice at 1wk compared to the control but returned to control levels at 8wks. **BALF:** Compared to the other strain, the total number of cells and macrophages increased significantly at 1wk in C57BL/6 mice and at 8wks in BALB/c mice. Neutrophils, lymphocytes, MCP-1, IL-12, IL-10, IL-4, IL-13 increased significantly for both strains. No eosinophils were found. IL-1β and IFN-γ increased significantly in BALB/c mice compared to C57BL/6 mice. **HO-1 mRNA and protein:** HO-1 mRNA was more marked in BALB/c mice at 1wk and C57BL/6 mice at 4 and 8wks. HO-1 protein percentage changes from the control were greater in BALB/c mice at 1wk and C57BL/c mice at 8wks. **NAC:** NAC inhibited the increased Penh values, total number of cells and macrophages in C57BL/6 mice at 1wk and neutrophils and lymphocytes in both strains.
Reference: Liu et al. (2008, 156709) **Species:** Mouse **Gender:** Female **Strain:** BALB/c **Age:** 11wks **Weight:** NR	DEP (5500-watt single-cylinder diesel engine generator (Yanmar, Model YDG 5500E), 406 cc displacement air-cooled engine, Number 2 Diesel Certification Fuel, 40 weight motor oil) **Particle Size:** ~0.1 μm (MMAD)	**Route:** Intranasal Exposure **Dose/Concentration:** Average particle concentration: 1.28mg/m^3 **Time to Analysis:** Four groups: saline + air control, saline + DEP, *A. fumigatus* + air, *A.fumigatus* + DEP. A. fumigatus exposure every 4d for 6 doses. DEP exposure 5h/d for 3wks concurrent with *A. fumigatus* exposure.	*A.fumigatus* + DEP increased IgE, the mean BAL eosinophil percentage, goblet cell hyperplasia, and eosinophilic and mononuclear cell inflammatory infiltrate around the airways and blood vessels compared to the *A. fumigatus* or DEP treatments. *A.fumigatus* + DEP also caused methylation at the IFN-γ promoter sites CpG^{-53}, CpG^{-45}, and CpG^{-205}.
Reference: Lopes et al. (2009, 190430) **Species:** Mouse **Gender:** Male **Strain:** BALB/c **Age:** 6-8wks **Weight:** NR	PM (high density traffic; winter 2004; São Paulo, Brazil) (NO_2, CO, SO_2) **Particle Size:** Diameter: 10 μm	**Route:** Open-Top Exposure Chamber **Dose/Concentration:** 33.86 ± 2.09 $\mu g/m^3$ **Time to Analysis:** Some rats pretreated with papain. Exposed to UAP or filtered air 24h/d, 7d/wk, 2m.	The papain + UAP treatment increased Lm values, collagen fibers, and decreased the density of elastin fibers over the papain + filtered air treatment. The papin + UAP treatment increased 8-isoprotane more than any other group.

Reference	Pollutant	Exposure	Effects
Reference: Mangum et al. (2004, 097326) **Species:** Rat **Gender:** Female **Strain:** CDF (F344)/CrlBR **Age:** 7wks	TiO_2 pigment grade (DuPont) **Particle Size:** NR	**Route:** Whole-body Inhalation **Dose/Concentration:** 10, 50 or 250mg/m^3 **Time to Analysis:** 6h/d x 5d/wk for 13wks. Parameters measured 0, 4, 13, 26, 52 wks post-exposure.	**OPN (osteopontin) Expression:** At 0 wks, OPN mRNA expression exhibited a dose-dependent increase. Low dose induced a 2-fold increase while the high dose induced an almost 100 -fold increase. At 4 wks, the mid-dose and high-dose elevated OPN mRNA levels. At 13wks, the high dose elevated OPN mRNA levels.No significant elevation with mid dose level was observed. At 26wks, the mid and high dose induced elevated OPN mRNA levels. At 52wks, rats in the low, mid and high dose groups all indicated elevated levels of OPN mRNA. Specifically, the low, mid and high doses induced a 3-fold increase, 7-fold increase and 400-fold increase, respectively. **OPN Protein in BALF:** Data was not reported at 0 and 4 wks. At 13 wks, protein increased 9-fold ($\sim$800 pg/mL OPN) at mid dose and 100 -fold ($\sim$8000 pg/mL OPn) at high dose. At 26 wks, the mid and high dose groups remained elevated. At 52 wks, protein increased by 2.5 fold in low dose, 7-fold in mid dose and 166-fold in high dose group. **Histopathology:** At 52wks, slight OPN immunoreactivity was observed in control and low dose group (immunostaining mostly limited to intraalveolar MACS).Trichrome-stained lung sections from control and low dose groups showed no increase in collagen. Rats exposed to mid or high dose groups showed areas of lesions.
Reference: Martin et al. (2007, 096366) **Species:** Mouse **Gender:** Male **Strain:** BALB/c **Age:** 1-2 mo	UAP-BA: Urban Air particles (Buenos Airs, Argentina) **Particle Size:** $<2.5\mu$m	**Route:** Intranasal Installation **Dose/Concentration:** 0.17 mg/kg bw **Time to Analysis:** 3xday, 3d/wk, 2d apart (1, 4, 7d). Parameters measured 1h post-exposure.	**Particle Characteristics:** 3 types, ultrafines $<$0.2um (inorganics ND), bunched agglomerates of ultrafines and $<$40um with aluminum silicates, ions and trace metals. **Morphometry:** Induced focal inflammatory lesions. Accumulation of refractile material in upper and lower respiratory tract. PM in phagocytes of bronchiolar lumen and alveolar space. No evidence of fibrosis and/or collagen changes. **BALF Cells:** Increased amount of phagocytes in alveolar area, reducing airspace percentage (control 52.9% $\pm$ 1.39, UAP-BA 24.7% $\pm$ 2.87). Increased number of PAS positive cells.
Reference: Mauad et al. (2008, 156743) **Species:** Mouse **Gender:** Male, Female **Strain:** BALB/c **Age:** 10d **Weight:** Parental: 21.4$\pm$4.0 – 26.3$\pm$2.8g; 15d-old offspring: 7.8$\pm$1.1 – 9.0$\pm$1.0g; 90d-old offspring: 20.3$\pm$2.3 – 27.4$\pm$1.8g	PM (busy traffic street São Paulo, Brazil; Aug. 2005-April 2006) (NO_2, SO_2, CO) **Particle Size:** 2.5, 10 μm (diameter	**Route:** Open-Top Chamber **Dose/Concentration:** $PM_{2.5}$: filtered chamber- 2.9$\pm$3.0 μg/m^3, nonfiltered chamber- 16.9$\pm$8.3 μg/m^3; Outdoor concentration: PM_{10}- 36.3$\pm$15.8 μg/m^3, CO- 1.7$\pm$0.7ppm, NO- 89.4$\pm$31.9 μg/m^3, SO_2- 8.1$\pm$4.8 μg/m^3 **Time to Analysis:** Nonfiltered exposure 24h/d 4m. Mated at 120d exposure. After birth, 30 females and offspring transferred to filtered or nonfiltered chamber. Killed 15 or 90d of age.	Mild foci of macrophage accumulations containing black dots of carbon pigment occurred in the alveolar areas on 90d-old mice. Surface-to-volume ratio decreased from 15 to 90d of age and was higher in mice exposed to air pollution. PM exposure reduced inspiratory and expiratory volumes at higher levels of transpulmonary pressure.
Reference: McDonald et al. (2004, 087459) **Species:** Mouse **Strain:** C57BL/6 **Age:** 8-10wks	DEE: high load, No 2, No cat (620: 1 dilution) DEE-ER (Control): Emissions Reduced (high load, low sulfur ECD1) (same dilution) (Yanmar diesel generator, 406 cc, 5500 watt load) **Particle Size:** DEE: 110nm; DEE-ER: NR	**Route:** IT Installation **Dose/Concentration:** DEE PM: 236 μg/m^3 DEE-ER PM: 7 μg/m^3 **Time to Analysis:** DEE: 6h/d for 7d. DEE-ER: 6h/d for 7d. RSV administered post-exposure for some: single, 4d. Those not infected with RSV sacrificed immediately upon last exposure.	**Differences in Exposure Conditions:** CO, PM, elemental carbon, organic carbon, nitrate, alkyne, c2-c212 alkenes, phenanthrenes, total particle PAHs, total Oxy-PAHs, benzene, pyrene, benzo(ayrene, zinc were reduced by 90-100% in the emissions reduction case. Most other components were reduced by around 60%. **DEE vs. DEE-ER Effects:** DEE increased viral retention and lung histopathology. DEE-ER increases were not statistically significant. **Cytokines:** DEE increased TNF-α, IL-6, IFN-γ and HO-1. DEE-ER responses were not statistically significant (significantly higher variability in DEE-ER controls vs. DEE controls).

Reference	Pollutant	Exposure	Effects
Reference: McQueen et al. (2007, 096266) **Species:** Rat **Gender:** Male **Strain:** Wistar **Weight:** 228-500g	DEP: SRM 2975 (NIST) **Particle Size:** NR	**Route:** IT Instillation **Dose/Concentration:** 0.5 mL/rat of 1 mg/mL; 1-2.2 mg/kg **Time to Analysis:** Single exposure, sacrificed 6h post-exposure. Pre-exposure: Vagotomy (sectioning of vagus nerve) or Atropine 1mg/kg i.p. administered 30 min prior, 2 and 4h post.	**BALF Cells:** A 9-fold increase in neutrophils with high individual variability in response was observed. Bilateral vagotomy prior to DEP reduced neutrophil increase to 3 fold. Vagotomy with saline instillation had no effect. Atropine reduced neutrophils to levels similar to saline response. No differences were observed between DEP response in anesthetized when compared to conscious animals. Macrophages, eosinophils and lymphocytes remain unchanged. **Respiratory Response:** RMV increased post DEP. Vagatomy reduced response by one-third. Atropine pre-treatment did not have effect. **Cardiovascular Response:** Blood pressure and heart rate were unaffected. Average arterial O2 increased after DEP, but not when compared for each animal. CO_2 and pH were not affected
Reference: Medeiron et al. (2004, 096012) **Species:** Mouse **Gender:** Male **Strain:** BALB/c **Age:** 60d **Weight:** 20-30g	CP: Carbon particles PSA: ROFA (solid waste incinerator hospital Sao Paulo, Brazil) PSB: electric precipitator, steel plant, Brazil) PSA/PSB Characteristics: Generally, PSB had greater component concentrations than PSA: Br (100+x), Cr (3x), Fe (10+x), Mn (2x), Rb (60+x), Se (7x), Zn (4x). PMA > PMB: Ce (3x), Co (10+x), La (100x), Sb (15x), V (50x). **Particle Size:** CP: 1.7 ± 2.5 μm (78% < 2.5 μm) PMA: 1.2 ±2.2 μm(98 % < 2.5 μm) PMB: 1.2 ± 2.2 μm (98% < 2.5 μm)	**Reference:** Intranasal Instillation **Dose/Concentration:** CP: 10 μg/mouse; 0.5mg/kg PSA: 0.1, 1 or 10 μg/mouse; 0.005, 0.05, 0.5 mg/kg PSB: 0.1, 1 or 10 μg/mouse; 0.005, 0.05, 0.5 mg/kg **Time to Analysis:** Single, 24h	**Hematology:** PSA and PSB decreased leukocyte count (all 3 doses) and platelet count (2 high doses). No effect on hemoglobin, erythrocytes and reticulocytes was observed. Fibrinogen levels increased for both PSB and PSA with PSB seeing a higher increase. None of the effects were dose-dependent. **Bone Marrow:** Erythroblasts increased for PSA at all dose levels and PSB at mid and high dose levels (high variability). **BALF Cells:** No change in BAL cell count was seen. Quantitative cellular counts increased for perivascular area for both groups at all dose levels. Inflammatory cells in alveolar septum area only increased for PSA.
Reference: Mutlu et al. (2006, 155994) **Species:** Mouse **Strain:** C57BL/6 **Age:** 6-8wks **Weight:** 20-25g	PM_{10} Collected by baghouse from Dusseldorf, Germany **Particle Size:** NR	**Route:** IT Instillation **Dose/Concentration:** 100 ng/mouse; 1 μg/mouse; 10 μg/mouse; 100 μg/mouse **Time to Analysis:** 1-7d	**Alveolar Fluid Clearance:** At 100 μg/mouse, decreased clearance peaked at 24 h and recovered at 7d. **Histology:** Evidence of mild lung injury at doses of 100 μg/mouse or more was seen. **BALF Cells:** Significant increase in total cell number was observed. Neutrophils increased but this was not statistically significant. **Wet/Dry Ratio:** Exposure did not induce any effects. **Na, K-ATPase:** At 100μg/mouse, decreased activity of Na, K-ATPase in basolateral membranes was observed.
Reference: Nadziejko, et al. (2002, 087460) **Species:** Rat **Gender:** Male **Strain:** SHR (spontaneously hypertensive rats) **Age:** 16wks	CAPs: produced at Tuxedo, NY laboratory using centrifugal aerosol concentrator FA: Fine Particle Sulfuric Acid Aerosol UFA: Ultra-Fine Particle Sulfuric Acid Aerosol **Particle Size:** CAPs: $PM_{2.5}$; FA: 160nm; UFA: 50-75nm	**Route:** Nose-only Inhalation (implanted blood pressure transmitters) **Dose/Concentration:** CAPS 80, 66 $\mu g/m^3$; avg 73 $\mu g/m^3$ FA 299, 280, 119, 203 $\mu g/m^3$; avg 225 $\mu g/m^3$ UFA 140, 565, 416, 750 $\mu g/m^3$; avg 468 $\mu g/m^3$ **Time to Analysis:** 10 exposures of 4h each, each exposure at least 1 wk apart. (2 exposures to CAPs, 4 to FA and 4 to UFA)	**Respiratory rate:** CAPs decreased the respiratory rate as did FA at all dose levels. However, the FA-induced respiratory rate was not statistically significant unless the data was combined. UFA increased this rate significantly. **Heart Rate:** CAPS depressed the heart rate significantly during exposures (data only significant when combined) but returned to normal post-exposure. FA induced a decrease as well with continuation 7h post-exposure. UFA increased heart rate. **Diastolic Blood Pressure:** Caps and FA induced a decrease but this was not statistically significant. UFA induced a slight increase.

Reference	Pollutant	Exposure	Effects
Reference: Nemmar et al. (2007, 156800) **Species:** Rat **Gender:** Male **Strain:** Wistar Kyoto **Age:** 16wks **Weight:** 424 ± 8g	DEP: SRM 2975 **Particle Size:** $<1\mu m$	**Route:** Intravenous Injection **Dose/Concentration:** 0.02, 0.1 or 0.5 mg/kg **Time to Analysis:** single, 24h	**Cardiovascular:** DEP depressed blood pressure at all doses approximately equally. DEP depressed heart rate at all doses equally. **Hematology:** No effect on number of platelets, granulocytes, monocytes, lymphocytes or RBC s. Tail bleeding time (associated with platelet activity) decreased at doses of 0.02 and 0.5mg/kg. **BALF Cells:** Marked cellular influx at all dose levels Was observed. Macrophages increased at the high dose, but this was not statistically significant. PMN increased significantly at all dose levels. **Wet/Dry Ratio:** All dose levels induced increases.
Reference: Nemmar et al. (2003, 087931) **Species:** Hamster **Gender:** Male and Female **Weight:** 100-110g	PS: Polystyrene particles PSC: Polystyrene particles, Carboxylate modified PSA: Polystyrene particles, Amine modified **Particle Size:** PS, PSC, PSA-60: 60nm; PSA-400: 400nm	**Route:** IT Instillation **Dose/Concentration:** 5, 50 or 500 μg/animal; 0.05, 0.5, 5mg/kg **Time to Analysis:** Single, 10min post-exposure Rose Bengal administered to induce thrombosis, immediate study thereafter	**Thrombosis:** Only PSA-60 at 50 and 500 levels enhanced thrombus formation but not in a dose-dependent manner. At 500 μg, PSA-60 showed evidence of pulmonary thrombosis. No effect with PSA-400 was seen. **BALF Cells:** Both PSA-60 and PSA-400 (PSA-60 > PSA-400) induced a massive influx of PMNs. PSA-60 effect may exhibit some dose-dependency. **BALF Inflammatory/Injury Markers:** Small increases in total protein were seen at 500 μg level for both PSA-60 and PSA-400. LDH was increased at all PSA-60 levels but not for 500ug PSA-400. Histamine increased for all PSA-60 levels and PSA-400 but due to high variability only the effect at 500 μg PSA-60 was statistically significant. **Hematology:** PSA-60 and PSA-400 had an effect on platelet closure time at very low concentrations: 3 and 9 μg/l respectively and plateaued thereafter. .
Reference: Nemmar et al. (2003, 097487) **Species:** Hamster **Gender:** NR **Weight:** 100-110g	DEP: SRM 1650 **Particle Size:** NR	**Route:** IT Instillation **Dose/Concentration:** 50 μg/animal **Time to Analysis:** Single exposure, parameters measured 1, 3, 6 or 24h post exposure.	**BALF Cells:** DEP led to a significant PMN flux at 1 h (13% of total cell number), 6 h (22%) and 24 h (37%). **Thrombosis:** DEP induced a significant increase in the cumulative mass of in vivo generated thrombus when compared to control subjects. **Hematology:** No decrease in platelet count was observed. Consistent (non time-dependent) decrease in closure time signified increased platelet activation for DEP-exposed groups. **Histamine:** Concentrations in BALF were consistently elevated starting at 1 h. Plasma histamine did not increase until 6 h. **Pretreatment with histamine receptor antagonist:** A major decrease in DEP induced PMN infiltration was seen. Thrombogenicity was decreased after 6h as was closure time shortening. No effect on histamine in BALF or plasma was observed.
Reference: Nurkiewicz et al. (2009, 191961) **Species:** Rat **Gender:** Male **Strain:** SD **Age:** 7-8wks **Weight:** NR	Fine TiO_2 (Sigma-Aldrich-(titanium (IV)) oxide, 224227, St. Louis, MO) (~ 99% rutile) TiO_2 nanoparticles (DeGussa-Aeroxide TiO_2 P25, Parsippany, NJ) (80% anatase, 20% rutile) **Particle Size:** Fine TiO_2-Primary size: $<5\mu m$, MMAD: 402nm, CMD: 710nm; Nano-TiO_2- Primary size: 21nm, MMAD: 138nm, CMD: 100nm	**Route:** Aerosol Inhalation **Dose/Concentration:** 1.5-16mg/m^3 **Time to Analysis:** Acclimated 5d. Exposed 240-720min. Anesthetized 24h postexposure. Intravital microscopy, NO measurement, microvascular oxidative stress measurement, nitrotyrosine staining.	**Arteriolar Dilation:** Nano-TiO_2 significantly impaired endothelium-dependent arteriolar dilation. Equivalent levels of arteriolar dysfunction were found in fine and nano-TiO_2. Arteriolar dilation in response to abluminal microiontophretic application of SNP was not different from the controls or between the exposure groups. Arteriolar dilation was partially restored by radical scavenging with TEMPOL and catalase, NADPH oxidase with apocynin, and MPO inhibition with ABAH. **Microcirculation:** ROS increased in both groups. Nano-TiO_2 significantly increased the area of tissue containing nitrotyrosine in the lung and spinotrapezius microcirculation. **NO:** Fine and nano-TiO_2 significantly and dose-dependently decreased stimulated NO production in isolated microvessels. NO production was increased by radical scavenging with TEMPOL and catalase or NADPH oxidase with apocynin, and was largest in the fine TiO_2 group.

Reference	Pollutant	Exposure	Effects
Reference: Pereira et al. (2007, 156019) **Species:** Rat **Gender:** Male **Strain:** Wistar **Age:** 3m	Ambient Particles (Porto Allegre, Brazil) **Particle Size:** $<10\mu$m	**Route:** Whole-body Inhalation **Dose/Concentration:** P-6: 34, 22 or 225 $\mu g/m^3$ P-20: 139 or 112 $\mu g/m^3$ P-I: 99 $\mu g/m^3$ **Time to Analysis:** P-6: single/continuous for 6h P-20: single/continuous for 20h P-I: intermittent (5h) periods per day for 4d consecutively Parameters measured 0 or 24 h post-exposure	**BALF Inflammatory/Injury Markers:** An increase in lipid peroxidation was statistically significant only for the 20 h continuously exposed group.Leukocytes also increased at P-20. No change at P-6. Total protein remained unaffected at all dose levels. **Wet to Dry Ratio (0h):** No effect was observed.
Reference: Pinkerton et al. (2004, 087465) **Species:** Rat **Gender:** Female (pregnant), Offspring- NR **Strain:** SD **Age:** 10d (pups), Pregnant females- 10-14d of gestation **Weight:** NR	PM (Fe and soot from combustion of acetylene and ethylene in a laminar diffusion flame system) **Particle Size:** Median diameter: 72-74nm; size range: 10-50nm	**Route:** Inhalation **Dose/Concentration:** Mean mass concentration: 243 ± 34 $\mu g/m^3$; Average Fe concentration: 96 $\mu g/m^3$ **Time to Analysis:** Exposed 10d postnatal age, 6h/d, 3d (consecutive). Bromodeoxyuridine injected 2h before necropsy.	A significant reduction of cell proliferation occurred only within the proximal alveolar region of exposed animals compared to controls. There were no significant differences between the groups for alveolar formation and separation within the proximal alveolar region.
Reference: Pinkerton et al. (2002, 087645) **Species:** Rat **Gender:** Male, Female **Strain:** SD **Age:** 11-13wks (adult male), 10-12d (neonatal) **Weight:** NR	PM (Fe, Soot) (ethylene, iron pentacarbonyl, acetylene combined; Fe_2O_3; soot: 60% EC, 40% OC)) (CO, NO_x) **Particle Size:** Fe (diameter)- 40nm; Soot (primary particles, diameter)- 20-40nm	**Route:** Whole-body Exposure **Dose/Concentration:** Adult males: Fe- 57, 90 $\mu g/m^3$, Soot- 250 $\mu g/m^3$, Fe+Soot- Fe: 45 $\mu g/m^3$, Total PM: 250 $\mu g/m^3$; Neonates: Fe+Soot- Low: Fe- 30 $\mu g/m^3$, Total PM: 250 $\mu g/m^3$, High: Fe- 100 $\mu g/m^3$, Total PM: 250 $\mu g/m^3$ **Time to Analysis:** Adult males exposed to Fe, soot, Fe+Soot, or filtered air. Exposed 6h/d, 3d (consecutive). BAL performed within 2h postexposure, lung tissue evaluations 24h postexposure. Pregnant dams housed from GD 3 to weaning at 21d-old. Neonatal rats exposed to Fe+Soot 10-12d-old and 23-25d-old.	**Fe:** Only the high dose had significant effects. This dose increased total protein in the lavage fluid, decreased total antioxidant power, induced GST activity, and induced a non-significant, increasing trend of GSH and GSSG. IL-1β, intracellular ferritin, and NF-κB increased. **Fe+Soot, Soot:** Fe+Soot significantly reduced the total antioxidant power in BALF and supernatant from lung tissue homogenate. Fe+Soot significantly increased GSSG, IL-1β, NF-κB, CYP1A1, and CYP2E1. CYP2B1 increased but was not significant. Soot alone was not significant for anything. **Neonates:** The high-dose significantly decreased cell viability, increased LDH activity, and increased IL-1β and ferritin. Both doses significantly increased GSSG, GRR, and GST, and decreased total antioxidant power.
Reference: Pires-Neto et al. (2006, 096734) **Species:** Mouse **Gender:** Male **Strain:** Swiss **Age:** 6d	Ambient Air: $PM_{2.5}$, NO_2 and CB (Sao Paulo, Brazil) **Particle Size:** $PM_{2.5}$	**Route:** Whole-body Inhalation **Dose/Concentration:** $PM_{2.5}$: 46.49 $\mu g/m^3$ Control: 18.62 $\mu g/m^3$ NO_2: 59.52 $\mu g/m^3$ Control: 37.08 $\mu g/m^3$ CB: 12.52 $\mu g/m^3$ Control: 0 $\mu g/m^3$ **Time to Analysis:** 24h/d, 7d/wk for 5mo (weaned at 21d into exposure - mothers removed)	**Nasal Cavity:** Increased total mucus and acidic mucus at proximal and medial areas of cavity. Nonsecretory epithelium declined. No significant changes in amount of neutral mucus, volume proportion of neutral mucus, volume proportion of total mucus, thickness of epithelium, volume proportion of nonsecretory epithelium or ratio between neutral and acidic mucus were observed. **Types of Acidic Mucus Cells:** Proximal and medium cells increased. Effects on distal cells were equivocal.
Reference: Pourazar et al. (2005, 088305) **Species:** Human **Gender:** Male and Female (nonatopic & nonsmokers) **Age:** 21-28yrs	DEP: generated from idling Volvo diesel engine DEP 300 $\mu g/m^3$ comprised of: NO_2 1.6ppm NO 4.5ppm CO 7.5ppm Hydrocarbons 4.3ppm Formaldehyde 0.26mg/m^3 Suspended particulates 4.3x10^6/cm^3 **Particle Size:** $<10\mu$m	**Route:** Whole-body exposure chamber **Dose/Concentration:** DEP 300 $\mu g/m^3$ **Time to Analysis:** Single exposure for 1h. Parameters measured 6h post exposure.	**Transcription Factors:** Exposure induced increased cytoplasmic and nuclear immunoreactivity of phosphorylated p38 MAPK in bronchial epithelium. Increased nuclear translocation of phosphorylated p38 and JNK MAPK as well as increased nuclear phosphorylated tyrosine immunoreactivity were observed. No change in total or nuclear c-fos immunoreactivity was seen. Exposure induced increased nuclear translocation of phosphorylated JNK significantly associated with phosphorylation of nuclear c-jun and also resulted in an increase in nuclear p65. **Cytokines:** Expression of IL-8 was positively associated with nuclear phosphorylated p38 post-exposure.

Reference	Pollutant	Exposure	Effects
Reference: Pradhan et al. (2005, 096128) **Species:** Rat **Gender:** Female **Strain:** Wistar Albino **Weight:** 120-180g	RSPM: Respirable Suspended PM (Lucknow, India) Quartz dust (positive control) **Particle Size:** $< 5\mu$m	**Route:** IT Instillation **Dose/Concentration:** 2.5, 5.0, or 10.0mg/0.05ml; 20, 42, 83 mg/kg **Time to Analysis:** Sacrificed 15d post single exposure.	**Relative Lung Weight:** A dose-dependent increase in total lung weight of RSPM-instilled animals was observed. **BALF Cells:** Exposure induced a dose-dependent increase in total cells dose-dependent with the low and mid dose levels. PMNs increased massively at all dose levels with RSPM inducing less of an increase than Quartz. Exposure at low dose levels resulted in an influx of inflammatory cells (predominantly macrophages into lumen of alveolar ducts and alveoli). Reaction at the high dose was more intense than that seen in mid dose-exposed lungs. **BALF Inflammatory/Injury Markers:** A significant dose-dependent increase in LDH and NO was observed, but the Quartz-induced increase was greater than the RSPM-induced increase. An increase in protein was significant at the mid dose level for RSPM and significant at the high dose level for both RSPM and Quartz. **Lung biochemistry:** An increase in lipid peroxidation was dose-dependent. Superoxide dismutase (SOD) enzyme levels showed a dose-dependent decrease.
Reference: Ramos et al. (2009, 190116) **Species:** Guinea Pig **Gender:** NR **Strain:** -- **Age:** NR **Weight:** 330-370g	WS (Pine wood) (CO($<$ 80ppm), CO_2(0.35%), O_2(20.1%), $PM_{2.5}$, PM_{10}) **Particle Size:** $PM_{2.5}$, PM_{10}	**Route:** Whole-body Inhalation Chamber **Dose/Concentration:** WS: 60g, $PM_{2.5}$: 363 ± 23 $\mu g/m^3$, PM_{10}: 502 ± 34 $\mu g/m^3$ **Time to Analysis:** Exposed 3h, 5d/wk for 1, 2, 3, 4, 6, 7m.	WS significantly decreased body weight between 4 and 7m exposure. The concentration of blood carboxyhemoglobin increased. Recovered BAL cells were higher in WS-exposed pigs. Macrophages and neutrophils increased. Inflammation in the lungs was seen. Pulmonary arterial hypertension and emphysematous lesions were observed. Macrophage and lung tissue homogenate elastolysis increased. Collagenolysis increased. Generally, MMP-2, MMP-9, and MMP-1 increased. BAL macrophage apoptosis increased with time.
Reference: Rao et al. (2005, 095756) **Species:** Rat **Strain:** Sprague-Dawley **Weight:** 175g	DEP: SRM 2975 **Particle Size:** 0.5μm	**Route:** IT Instillation **Dose/Concentration:** 5, 35, 50mg/kg bw **Time to Analysis:** Sacrificed 1, 7, 30d post single exposure. Cytokines measured after 24h incubation (in vitro).	**BALF Inflammatory/Injury Markers:** Increased albumin at 1 and 30d at all dose levels. Increased LDH except at low dose at 7d. **BALF Cells:** Macrophages unaffected. Increased PMNs at 1d for all dose levels, sustained elevation at 7d for mid and high dose and at 30d for all dose levels. **Cytokines:** The high dose induced a significant increase of mRNA expression for IL-1β, iNOS, MCP-1, and MIP-2 in BAL cells. MCP-1 mRNA sustained high levels at 7d for mid and high dose and at 30d for all dose levels. mRNA expression of IL-6, IL-10, TGF-β1, TNF-α were unaffected. However, IL-6 and MCP-1 proteins increased significantly in BALF at 1d for mid and high dose, returning to basal levels at 7d. MIP-2 increased for all dose levels at all time points. NO level unaffected.

Reference	Pollutant	Exposure	Effects
Reference: Reed et al. (2006, 156043) **Species:** Rat, Mouse **Gender:** Male and Female **Strain:** CDF (F344)/CrlBR (rat), SH (rat), A/J (mouse), and C57BL/6 (mouse) **Age:** 6-12wks	HWS (burned mix of hardwood in noncertified wood stove using a Pineridge model 27000, Heating and Energy Systems, Inc. Clackamas, OR) Measured Components: EC, OM, NO_3, SO_4, NH_4, metals **Particle Size:** ~0.25μm	**Route:** Whole-body Inhalation **Dose/Concentration:** Low: 30 $\mu g/m^3$ Mid-low: 100 $\mu g/m^3$ Mid-high: 300 $\mu g/m^3$ High: 1000 $\mu g/m^3$ **Time to Analysis:** 6hr/d, 7d/wk for 1wk or 6m. Immediate post-exposure analysis.	**Organ weights:** Liver declined in rats of both genders at 1 wk and female rats at 6m. Lung volume increased and lung weight decreased in female rats at 6m. Spleen weight increased in female mice and rats at 1wk. Thymus weight decreased in male rats at 1wk. **Clinical Chemistry:** Cholesterol decreased at the high dose for male rats at 1wk and 6m and increased at mid-low and mid-high doses for female rats at 6m. ALP decreased for rats of both genders at 1wk and 6m for mid-low, mid-high and high dose levels (14-38%). AST decreased by 24% in male rats at 1wk with high dose. No effect on females. Creatinine serum levels decreased in males at 1wk at mid-high and high dose by 13%. No effect observed at 6m. BUN/Cre ratio decreased in females at 1wk (25%) and both genders at 6m at mid-high and high dose (18-19%). **Hematology:** Hemoglobin and hematocrit increased in 6mo male rats. Bilirubin increased in female rats at 6m at high dose. Platelets increased for male and female rats at 1wk (21%, 19% respectively). No effect observed at 6m. WBC increased in males at 1wk. **Cells:** Eosinphils decreased and lymphocytes increased in males at 6m. Neutrophils decreased at 6m in both genders. Minimal increases in alveolar macrophages and sparse brown-appearing macrophages in all species. **Bacterial Clearance:** Mice instilled with bacteria were mostly unaffected by exposure, except for a decline in histopathology summary score after 6m. **Tumorigenesis:** No values for exposed groups differed significantly from controls. There was no evidence of progressive exposure related trend.
Reference: Reed et al. (2004, 055625) **Species:** Rat, Mouse **Gender:** Male and Female **Strain:** CDF (F344)/CrlBR (rat), A/J (mouse) **Age:** 12wks	DE: generated from two 2000 model 5.9 L Cummins ISM turbo diesel engines Co-exposure to 8 gas and 8 solid exhaust components measured **Particle Size:** 0.10 - 0.15μm	**Route:** Whole-body Inhalation **Dose/Concentration:** Low: 30 $\mu g/m^3$ Mid-low: 100 $\mu g/m^3$ Mid-high: 300 $\mu g/m^3$ High: 1000 $\mu g/m^3$ **Time to Analysis:** 6h/d, 7d/wk for 1wk or 6m. Analyzed 1d post-exposure.	**Organ weights:** Kidney weight increased after 6m for both males and female rats at the high dose. Kidney and liver weight increased for female mice at all dose levels at 6m. Lung weight increased at high dose at 6m for female mice and male rats. Spleen weight decreased in male mice at the low and mid-high levels. **Clinical Chemistry:** There was a massive decrease in cholesterol (24%) for rats of both genders after 1wk and a smaller decrease for male rats at 6m. GGT significantly increased at 6m for male and female rats at the mid-high and high dose. ALP increased in male rats at 1 wk by 10%. AST decreased at mid-high (15%) and high dose in female rats at 6m. BUN and BUN/Creatine declined (19%, 17%) in female rats at mid-high and high doses after 6m. BUN increased by 21% at mid-low, mid-high and high doses in male rats at 1wk. **Hematology:** WBC decreased in high females after 6m. Factor VII (blood clotting) decreased in MH and HR males after 1wk and male and female HR after 6m. Thrombin-antithrombin complex declined massively but only in males after 1wk. **Cells:** Minimal increases in alveolar macrophages and PM within the macrophages were seen. **Cytokines:** TNF-α decreased in female rats after 6m. **Tumorigenesis:** No significant effect was observed.

Reference	Pollutant	Exposure	Effects
Reference: Reed et al. (2008, 156903) **Species:** Mouse **Gender:** Male, Female **Strain:** C57BL/6, A/J, BALB/c **Age:** NR **Weight:** NR	GEE (2 1996 General Motors 4.3-L V-6 engines; unleaded gasoline) **Particle Size:** 150nm (MMAD)	**Route:** Whole-body Inhalation **Dose/Concentration:** Control: 2.5±2.9, Low-exposure: 6.6±3.7, Mid-exposure: 30.3±11.8, High-exposure: 59.1±28.3, High filtered exposure: 2.3±2.6 μg/m^3 **Time to Analysis:** Exposed 6h/d, 7d/wk, 3d-6m.	**Body and organ weight and histopathology in A/J:** Kidney weight decreased, but no effects pertaining to weight were significant. No visible inflammatory changes were seen. **Lung Damage in A/J:** No significant effect was seen, but hypomethylation was seen in females at 1wk, and methylation was reduced in all exposed female groups. **Bacteria in lungs of C57BL/6:** Exposure did not affect the clearance of bacteria from the lung. **Respiratory allergic response in BALB/c:** Exposure had little effect, but serum total IgE increased significantly for the high-exposure group. Increasing trends were seen in OVA-specific serum IgE and IgG1, as well as neutrophils and eosinophils.
Reference: Reed et al. (2008, 156903) **Species:** Rat **Gender:** Male, Female **Strain:** CDF (F344)/CrlBR, SH **Age:** NR **Weight:** NR	GEE (2 1996 General Motors 4.3-L V-6 engines; unleaded gasoline)	**Route:** Whole-body Inhalation **Dose/Concentration:** Control: 2.5±2.9 μg/m^3, Low-exposure: 6.6±3.7 μg/m^3, Mid-exposure: 30.3±11.8 μg/m^3, High-exposure: 59.1±28.3 μg/m^3, High filtered exposure: 2.3±2.6 μg/m^3 **Particle Size:** 150nm (MMAD) **Time to Analysis:** Exposed 6h/d, 7d/wk, 3d-6m.	**Body and organ weight and histopathology in F344:** There were no significant effects pertaining to weight, but heart weight increased and seminal vesicle weight decreased. No visible inflammatory changes were seen. **Serum chemistry, hematology, clotting factors in F344:** Serum alanine aminotransferase, aspartate aminotransferase and phosphorous decreased in females at 6m in the mid- and high-exposure groups. Hematocrit, red blood cell count and hemoglobin increased at 1wk and 6m in males and females in the mid- and high-exposure groups. Plasma fibrinogen increased in males at 1wk in the mid-exposure group. **Lung DNA Damage in F344:** No significant effect was seen, but hypermethylation occurred in male rats at 6m in the mid- and high-exposure groups. **BALF in F344:** Generally, in the high-exposure group, increases were seen in LDH and MIP-2, and decreases were seen in hydrogen peroxide produced by unstimulated macrophages and superoxide by both stimulated and unstimulated macrophages. **SH:** HR or ECG parameters were not affected. In the high-exposure group, lipid peroxides increased in males and TAT decreased in males and females.
Reference: Reed et al. (2008, 156903) **Species:** Mouse **Gender:** Male, Female (only BALB/c) **Strain:** C57BL/6, A/J, BALB/c **Age:** NR **Weight:** NR	GEE (two 1996 General Motors 4.3-L V-6 engines; regular, unleaded, non-oxygenated, non-reformulated gasoline blended to US average consumption for summer 2001 and winter 2001-2002- Chevron-Phillips) **Particle Size:** MMAD: 150nm	**Route:** Inhalation Exposure Chamber **Dose/Concentration:** PM: Low- 6.6±3.7 μg/m^3, Medium- 30.3±11.8 μg/m^3, High- 59.1±28.3 μg/m^3 **Time to Analysis:** A/J- 2wk quarantine period in chamber. Exposed 6h/d, 7d/wk, 3d-6m. C57BL/6- 1wk exposure. Instillation of *P. aeruginosa*. Killed 18h postinstillation. BALB/c- Conditioned to exposure chambers and mated. Pregnant females exposed GD 1 and throughout gestation. Offspring exposures continued until 4wks-old. Half of offspring sensitized to OVA. Tested for airway reactivity by methacholine challenge 48h postinstillation and euthanized.	The kidney weight of female A/J mice decreased at 6m and was strongly related to PM by the removal of emission PM. PM-containing macrophages increased by 6m. Hypomethylation occurred in females at 1wk. The clearance of *P. aeruginosa* was unaffected by exposure. Serum total IgE significantly and dose-dependently increased. OVA-specific IgE and IgG1 gave slight exposure-related evidence but were not significant.

Reference	Pollutant	Exposure	Effects
Reference: Reed et al. (2008, 156903) **Species:** Rat **Gender:** Male, Female **Strain:** CDF (F344)/CrlBR, SHR **Age:** NR **Weight:** NR	GEE (two 1996 General Motors 4.3-L V-6 engines; regular, unleaded, non-oxygenated, non-reformulated Chevron-Phillips gasoline, U.S. average consumption for summer 2001 and winter 2001-2002) **Particle Size:** MMAD: 150nm	**Route:** Inhalation Exposure Chamber **Dose/Concentration:** PM: Low- 6.6±3.7 μg/m^3, Medium- 30.3±11.8 μg/m^3, High- 59.1±28.3 μg/m^3 **Time to Analysis:** 2wk quarantine period in chamber. Exposed 6h/d, 7d/wk, 3d-6m. SHR-surgery to implant telemeter in peritoneal cavity. 4wks recovery. ECG data obtained every 15min beginning 3d preexposure, 7d exposure, 4d postexposure.	**Organ Weight:** At 6m exposure, the heart weights of male and female rats increased and male rats' seminal vesicle weight decreased. **Histopathology:** PM-containing macrophages increased by 6m. **Serum Chemistry:** Serum alanine aminotransferase, aspartate aminotransferase, and phosphorus decreased in medium and high-exposure females. **Hematology, Clotting Factors:** Hematocrit, red blood cell count, and hemoglobin dose-dependently increased for both genders at both time points. Plasma fibrinogen increased at 1wk in males. **Lung DNA Damage:** Hypermethylation occurred in medium- and high-exposure male rats at 6m. **BAL:** For both genders in the high-exposure group, LDH and MIP-2 significantly increased at 6m. ROS decreased at 1wk and 6m. Generally, the production of hydrogen peroxide and superoxide decreased in the high-exposure group and medium- and high-exposure groups, respectively. **CV effects in SHR:** Lipid peroxides were significantly increased in males in the high exposure group. TAT complexes decreased in females in the high exposure group. **Removal of Emission PM:** The removal of emission PM strongly linked PM to increased seminal vesicle weight, red blood cell counts, LDH, lipid peroxides, and methylation.
Reference: Rengasamy et al. (2003, 156907) **Species:** Rat **Gender:** Male **Strain:** Sprague-Dawley **Weight:** ~200g	DEP: SRM1650 CB Elftex-12 furnace black, Cabot, Boston, MA **Particle Size:** NR	**Route:** IT Instillation **Dose/Concentration:** 5, 15, or 35 μg/kg bw **Time to Analysis:** single; 1, 3, 5, 7d post exposure	**CYP1A1:** DEP at all doses significantly increased CYP1A1 protein, was maximal at 1d, and normalized at 5d. CB had no effect. **CYP2B1:** DEP and CB at 15 and 35 mg/kg inhibited activity at 1d. Protein level significantly decreased at 1 d with 5, 15 and 35 mg/kg DEP and at 15 and 35 mg/kg CB. A time dependent decrease was shown at 35mg/kg for both DEP and CB.
Reference: Renwick et al. (2004, 056067) **Species:** Rat **Gender:** Male **Strain:** Wistar **Weight:** 370-470g	FCB: Fine Carbon Black (Huber 990) UCB: Ultrafine Carbon Black (Printex 90, Degussa) FTO: Fine Titanium Dioxide (Tioxide) UTO: Ultrafine Titanium dioxide (Degussa) **Particle Size:** FCB: 260nm; UCB: 14nm; FTO: 250nm; UTO: 29nm	**Route:** IT Instillation **Dose/Concentration:** 125 or 500 μg/rat **Time to Analysis:** Single, 24h	**BALF Cells:** UTO and UCB induced a large dose-dependent increase in percent neutrophils (only statistically significant at 500 μg for UTO). **BALF Inflammatory/Injury Markers:** UTO and UCB also increased total protein content only at the 500 μg dose. UCB induced LDH release at 125 and 500 μg, UTO and CB at 500 μg. UTO and UCB induced large dose-dependent increases in GGT activity (only statistically significant at 500 μg for UTO). **Phagocytosis:** All 4 particles decreased but only at the 500 μg level. **Chemotaxis:** Only UTO and UCB at 500 μg/l increased chemotactic migration.
Reference: Rhoden et al. (2004, 087969) **Species:** Rat **Gender:** Male **Strain:** Sprague-Dawley **Weight:** 250-300g	CAPs (Boston, MA) **Particle Size:** CAPS: 0.1-2.5 μm	**Route:** Whole-body Inhalation **Dose/Concentration:** 1060 ± 300 μg/m^3 **Time to Analysis:** Single exposure for 5h. Analyzed 24 h post exposure. (CAPS-NAC = CAPS with 50mg/kg bw NAS (N-acetylcysteine) pretreatment)	**Particle Characteristics:** Major components did not appear to show any correlation to total particle mass. Included Na, Mg, Al, Si, S, Cl, K, Ca, Ti, V, Cr, Mn, Fe, Ni, Cu, Zn, Br, Ba, Pb. Metals Al, Si and Fe (somewhat less for Pb, Cu, K) correlated with TBARS. **Oxidative Stress:** CAPS increased TBARS and oxidized protein by 2+ fold. NAS fully prevented the increase in TBARS and partially prevented an increase in protein carbonyl. Tissue Damage: Wet/dry ratio increased with CAPS but significantly decreased with NAC. **BALF Cells:** CAPS increased PMN 4 fold. NAS treatment reduced this increase to control levels. **BALF Inflammatory/Injury Markers:** LDH and total protein not affected. Histology confirms slight inflammation with CAPS and no inflammation with CAPs-NAC.

Reference	Pollutant	Exposure	Effects
Reference: Rhoden et al. (2008, 190475) **Species:** Rat **Gender:** Male **Strain:** SD **Age:** NR **Weight:** 300g	Urban Air Particles (UAP) (SRM 1649) **Particle Size:** NR	**Route:** IT Instillation **Dose/Concentration:** 1mg in 100 μL saline **Time to Analysis:** Some rats pre-treated with MnTBAP 2h prior to UAP exposure. Instilled with UAP. CL analysis 15min postexposure. Anesthetized 4h postexposure for BAL measurements.	UAP significantly increased the total cell number, PMN, MPO activity, and protein levels. MnTBAP prevented UAP-induced lung inflammation. UAP increased oxidants in lung CL, which was prevented by MnTBAP.
Reference: Rivero et al. (2005, 088653) **Species:** Rat **Gender:** Male **Strain:** Wistar **Age:** 3m **Weight:** 250g	Ambient Air (Sao Paulo, Brazil) **Particle Size:** $<2.5\mu$m	**Route:** IT Instillation **Dose/Concentration:** 100 or 500 μg/rat; 0.4 or 2mg/kg **Time to Analysis:** single, 24h	**Hematology:** Reticulocytes increased at both doses. At the high dose, hematocrit, percent segmented, percent neutrophils increased and percent lymphocytes decreased (relative to control or 100 - very high variability). Fibrinogen decreased at low dose but not at high dose. **Histopathology:** At both doses, acute alveolar inflammation was observed and was more pronounced in the 500 μg group. **Lung Morphometry:** Lumen wall ratio values show a dose-dependent increase in peribronchial as well as intra-acinar pulmonary arterioles. No effect in myocardial arterioles were observed. **Tissue Damage:** Increase in the heart wet/dry ratio at high dose was observed. Lung wet/dry ratios were unaffected.
Reference: Roberts, E.S. Charboneau, L. Espina, V. 2004 **Species:** Rat **Gender:** Male **Strain:** Sprague-Dawley **Age:** 60-90d **Weight:** 300-350g	ROFA: SRI (cyclone power plant) **Particle Size:** NR	**Route:** IT Instillation **Dose/Concentration:** 0.5 mg/rat; 1.67mg/kg **Time to Analysis:** Single, 6 and 24 h	**Technology:** Laser capture microdissection of airway cells were used to analyze results. **Protein: pERK1/2:** ERK1/2 ratio increased by 60% at 6h and 80% at 24 h. NFkB activity increased at 6h but was not statistically significant.
Reference: Saber et al. (2005, 097865) **Species:** Mouse **Gender:** Female **Strain:** TNF(-/-) (B6, 129S-Tnftm1Gk1), C57/BL **Age:** 9-10wks	DEP: SRM 2975 CB: Printex 90 (Degussa) **Particle Size:** DEP: 215nm; CB: 90nm	**Route:** Nose-only Inhalation **Dose/Concentration:** DEP: 20 mg/m^3; CB: 20 mg/m^3 **Time to Analysis:** 90min/d for 4d consecutively, 1h	**BALF Cells:** Neutrophils increased significantly to 15% when compared to control (4%) with DEP exposure. No response difference was observed between TNF (+/+) and TNF(-/-). CB did not induce any changes in neutrophil numbers. **Cytokines:** IL-6 increased 2-3 fold in DEP and CB exposure in both normal and knockout mice. IL-1β was unaffected. **mRNA:** In TNF (+/+) mice, DEP and CB increased expression of TNF mRNA 2- fold. IL-6 mRNA expression was high in DEP-exposed knockout mice when compared to normal mice. **DNA:** DNA strand breaks increased in both strains. Knockout mice showed a higher response to CB and DEP exposure. For normal mice, only CB induced a statistically significant effect.
Reference: Schins et al. (2004, 054173) **Species:** Rat **Gender:** Female **Strain:** Wistar **Weight:** 350-550g	Soluble fractions PMC: $PM_{10-2.5}$ PMF: $PM_{2.5}$ -B: Borken, Germany (rural) -D: Duisburg, Germany (industrialized) **Particle Size:** $PM_{10-2.5}$, $PM_{2.5}$	**Route:** IT Instillation **Dose/Concentration:** 0.32 ± 0.01 mg/rat; 0.91± 0.58 mg/kg **Time to Analysis:** single, 18h	**Radical Formation:** Formation of hydroxl radicals increased with exposure. Relative intensity is as follows: PMC-D, PMF-D, PMC-B, PMF-B, and control. **Cytokines:** TNF-α and IL-8 increased with PMC from both sites. PMF induced a slight increase in IL-8 but did not induce an increase in TNF-α. **BALF cells:** Both PMC showed a massive increase in neutrophils. PMC-B induced the greatest increase followed by PMC-D. Both PMF did not induce a significant increase. **BALF Inflammatory/Injury Markers:** PMC from both sites induced markedly higher endotoxin concentration vs PMF as follows in decreasing order: PMC-B, PMC-D, PMF-B, PMF-D, control. Glutathione decreased only for PMC-B. LDH and total protein were unaffected.

Reference	Pollutant	Exposure	Effects
Reference: Seagrave et al. (2005, 088000) **Species:** Rat **Gender:** Male **Strain:** F344/DCrl BR **Age:** 11 ± 1 wk	PM from 3 sources: NT: New Technology bus, Detroit Diesel 50G, exhaust oxidation catalyst, 216 miles, 2002 model - in use NE: Normal emitter bus, Detroit Diesel 50G, no catalyst, 134259 miles, 1997 model - in use HE: High Emitter bus, Cummins L10G, no catalyst, > 250, 000 miles, 1992, retired Fuel composition very similar for 3 vehicles: methane (96-96.8%), ethane (1.6-1.9%), carbon dioxide (0.9-1.1%), nitrogen (0.6-0.8%), traces of other gases **Particle Size:** NR	**Route:** IT Instillation **Dose/Concentration:** 0.25 - 2.2 mg/rat in 0.5mL saline **Time to Analysis:** Sacrificed 24h post single instillation.	**Engine Specific Emission data:** HE had significantly higher PM and SVOC recovered emission rates than NE and NT. **Organic mass in PM:** The following PM sources are listed in decreasing order of percent of total mass: HE, NE, NT. **Total PAH:** The following PM sources are listed in decreasing order of total mass: HE, NT, Control, NE. **Nitro PAH:** The following PM sources are also listed in decreasing order of total mass: NE, HE, Control, NT. Authors note confounding technical issues (mostly technique related) with mostly mild effects. **BALF Inflammatory/Injury Markers:** LDH showed dose-dependent increases withHE inducing higher increases than NT and NE. Total protein exhibited dose-dependent increases with HE, NT and the positive control SRM2975 inducing higher levels than NE. **Potency Factors Cytotoxicity and Inflammation:** HE was significantly more potent than NT and NE, with NT also showing significant potency. **Lung Toxicity:** The results were highly variable but the general toxicity levels in increasing order is the following: NE, NT, HE, Normal gasoline, diesels, and high gasolines, though individual factors may differ greatly.
Reference: Seagrave et al. (2006, 091291) **Species:** Rat **Gender:** Male **Strain:** F344/Crl BR, **Age:** 11 ± 1 wk	$PM_{2.5}$ sources: BHM: Birmingham, Alabama; urban JST: Jefferson Street, Atlanta, Georgia; urban PNS: Pensacola, Florida; urban/residential CTR: Centreville, Alabama; rural "smoke" = downwind of forest fires/burns (NR) **Particle Size:** $PM_{2.5}$	**Route:** IT Instillation **Dose/Concentration:** 0.75, 1.5, 3 mg/rat **Time to Analysis:** Sacrificed 24h post single instillation.	**BALF Total cells and PMN:** In general, the winter samples induced greater increases in potency than the summer samples except for PNS. For the winter samples, the samples that induced the greatest increases, in descending order, are: JST, BHM, CTR, PNS and Smoke. For the summer, the samples that induced increases, in descending order, are: BHM, JST, PNS, and CTR. **BALF macrophages:** For the winter, the BHM and JST samples significantly increased potency whereas the PNS sample induced significantly negative potency. For the summer, only the BHM sample significantly induced potency. **BALF lymphocytes:** Only the JST-W and BHM-W significantly increased potency. The BHM-S, CTR-S and PNS-S also significantly increased potency. **Histopathological Inflammation:** All the winter and summer samples, excepting PNS, significantly induced potency. **Lung weight/body weight ratio:** In general, for all end points, JST-S was significantly less potent than JST-W. The summer samples of BHM and CTR were also generally more potent than their winter counterparts.

Reference	Pollutant	Exposure	Effects
Reference: Seagrave et al. (2005, 088000) **Species:** Rat **Gender:** Male, Female **Strain:** CDF(F-344)/CrlBR **Age:** 10-12wks	DE: (Two 6 cyl Cummins ISB turb0) HWS = hardwood smoke (mixed black/white oak, uncertified conventional wood stove) DE: EC = 557 OC = 269 μg/m^3 NO = 45 ppm NO_2 = 4 ppm CO = 30 PPM THV = 2 ppm HWS: EC = 43 OC = 908 μg/m^3 NO or NO_2 = 0 ppm CO = 13 ppm THV = 3 ppm **Particle Size:** DE: 0.14 ± 1.8 μm	**Route:** Whole-body Inhalation **Dose/Concentration:** 30, 100, 300, 1000 μg/m^3 TPM HWS: 0.36 ± 2.1 μm (MMAD + GSD **Time to Analysis:** 6h/d, 7d/wk for 6m. 1d post-exposure	**Particle Characteristics:** Major differences K: HWS > > DE; Ca DE > > HWS; Zn: DE > > HWS. **BALF Inflammatory/Injury Markers:** LDH was unaffected by DE. Exposure to HWS induced an increase for males only at 100 and 300 but not at 1000 μg/m^3. Protein was unaffected by DE. HWS exposure showed male-only effects at 100 and 300 μg/m^3 but not at 1000. AP was unaffected by DE or HWS except for slight decline induced by HWS at 1000 μg/m^3 for both genders. **Other:** β-glucose was unaffected by DE. HWS-exposed females showed decreased β-glucose at 100 and 300 but not at 1000μg/m^3. **BALF GSH to (GSH+GSSG):** No effects for DE were observed. HWS significantly decreased the ratio in both males and females at 1000 μg/m^3. The effect for females was greater than the male effect. **BALF Cells:** No effects were observed except for an increase in macrophages at 30 μg/m^3 for HWS males exposed to HWS. **Cytokines:** IL-1β was unaffected by DE or HWS. MIP-2 decreased for both genders at 1000 HWS. TNF-α decreased in females with DE exposure. No TNF-α effects for HWS were observed.
Reference: Seagrave et al. (2008, 191990) **Species:** Rat **Gender:** Male **Strain:** SD **Age:** 10-12wks **Weight:** 250-300g	GEE (2 1996 General Motors 4.3-L V6 gasoline engines; conventional Chevron Phillips gasoline, U.S. average composition) (CO, NO, NO_2, SO_2, THC) ($PM_{2.5}$ composition-EC, OC, SO_4, NH_4, NO_3) Simulated downwind coal emission atmospheres (SDCAs) (fly ash, gas-phase pollutants, sulfate aerosols, NO, NO_2, SO_2) Paved Road Dust (RD) (Los Angeles, CA; New York City, NY; Atlanta, GA) **Particle Size:** GEE: MMAD-150nm, RD: 2.6±1.7 μm, SDCA: 0.1-1.0 μm	**Route:** Nose-only Inhalation **Dose/Concentration:** GEE: 60 μg/m^3, SDCAs: 317-1072 μg/m^3, RD: 306-954 μg/m^3; GEE: CO-104ppm, NO- 16.7ppm, NO_2- 1.1ppm, SO_2-1.0ppm, THC- 12ppm; SDCAs: CO- < 1ppm, NO-0.19-0.62ppm, NO_2- 0.10-0.37ppm, SO_2- 0.07-0.24ppm, THC- < 1ppm **Time to Analysis:** Quarantined 2wks. 6h exposure then ip injected. Cannula ligated into trachea and connected to rodent ventilator. Thorax and abdomen opened. Killed after measurements taken.	GEE produced CL in the lungs, heart, and liver. RD produced a significant effect in the heart at the low dose. SDCAs had no effect on CL. RD significantly increased the heart's oxidative stress, as demonstrated by the TBARS levels. GEE did not affect the amount of macrophages or PMN. SDCAs increased macrophages. The RD low dose increased macrophages and PMN. SDCAs increased P_{enh} values and tidal volumes.
Reference: Singh et al. (2004, 087472) **Species:** Mouse **Gender:** Female **Strain:** CD-1 **Age:** 6-8wks	A-DEP (4cyl light duty 2.7l Isuzu diesel at 6 kg/m) DEP: SRM 2975 **Particle Size:** A-DEP > 50 μm	**Route:** Oropharyngeal Aspiration **Dose/Concentration:** 25 or 100μg/mouse **Time to Analysis:** single, 4h (18h post-exposure measurements taken but NR due to similar results)	**Particle Characteristics:** DEP had 60% EC vs 9% in A-DEP. A-DEP had 50% OC vs 5% in DEP. Phenanthrene and Fluoranthene fractions were much more prevalent in PAH from DEP than A-DEP. **BALF Inflammatory/Injury Markers:** Microalbumin increased for both pollutants except DEP induced increases only at 100 μg. Endotoxin increased microalbumin. NAG increased with 100 μg A-DEP. **BALF Cells:** PMNs significantly increased dose-dependently with DEP and remained elevated at 18h. Endotoxin induced the greatest increases of PMNs. Macrophages increased with A-DEP and were unaffected by DEP. **Cytokines:** Endotoxin induced massive responses for IL-6, MIP-2 and TNF-α but no response from IL-5. A-DEP increased all 4 cytokines but only at the 100μg dose level. Similarly, DEP only increased IL-6 at the 100μg dose level.

Reference	Pollutant	Exposure	Effects
Reference: Smith et al. (2003, 042107) **Species:** Rat **Gender:** Male **Strain:** Sprague-Dawley **Age:** 11-12wks	CAPs (Fresno, CA) **Particle Size:** $<2.5\ \mu m$	**Route:** Whole-body Inhalation **Dose/Concentration:** 6 exp in 2 sets of 3: Fall1 = 847 $\mu g/m^3$ Fall2 = 260 $\mu g/m^3$ Fall3 = 369 $\mu g/m^3$ Winter1 = 815 $\mu g/m^3$ Winter2 = 190 $\mu g/m^3$ Winter3 = 371 $\mu g/m^3$ **Time to Analysis:** 4h/d for 3 consecutive days. Parameters measured immediately following last exposure.	**Particle Characteristics:** Nitrate showed the highest variability near 10 fold, followed by Si, S and EC. OC concentration was relatively consistent. Metals otherwise appeared proportionate to the concentrations. **BALF Cells:** Total cells increased at wk1. Percent of macrophages reduced in wk2 with CAPs. Number of neutrophils increased with CAPs, but only achieved statistical significance during wk1 of the fall and winter. Lymphocytes increased but were not statistically significant. **BAL cell permeability**: Upon CAPs exposure, the proportion of nonviable cells were increased up to 242% when compared to controls. The fall of wk2 induced the highest significant increases followed by fall wk1, fall wk3, and winter wk3.
Reference: Smith et al. (2006, 110864) **Species:** Rat **Gender:** Male **Strain:** Sprague-Dawley **Age:** 8wks **Weight:** 260-270g	CFA: Coal Fly Ash (400 MW, Wasatch Plateau, Utah) (aerodynamic separation) **Particle Size:** 0.4-2.5 μm	**Route:** Nose-only Inhalation **Dose/Concentration:** 1400 $\mu g/m^3$ $PM_{2.5}$ including 600 $\mu g/m^3$ PM1 **Time to Analysis:** 4h/d for 3 consecutive days. Parameters measured 18 or 36h post-exposure.	**BALF Cells:** Percent and total number of neutrophils in BALF and blood increased significantly at both 18 and 36h. Percent of macrophages decreased slightly while number of macrophages increased in bronchiole-alveolar duct regions at both time periods. **Cytokines:** MIP-2 and transferrin increased at 18h. IL-1β increased at 36h. **Hematology:** Plasma protein increased at 18h. Lymphocyte and hematocrit percentage decreased at 36h. **Other:** Gamma glutamyl transferase decreased at 36h. Lung antioxidant increased at 18h.
Reference: Song et al. (2008, 156093) **Species:** Mouse, **Gender:** Ffemale, **Strain:** BALB/c, **Age:** 5-6wks	DEP collected from a 4JB1-type, light-duty (2740 cc), four-cylinder diesel engine operated using standard diesel fuel at speeds of 1500 rpm under a load of 10 torque. **Particle Size:** 0.4 μm (mean diameter)	**Route:** Intranasal Instillation (days 1-5), Whole-body Exposure Chamber (days 6-8) **Dose/Concentration:** 0.6mg/mL in 50 μL of saline (days 1-5), 6mg/m^3 for 1h/d for 3d (days 6-8). **Time to Analysis:** Enhanced Pause (Penh), measured on day 9. BAL and lung tissues collected on day 10.	**Airway Hyper-Responsiveness:** Intranasal exposure plus aerosolized DEP caused a significant increase in methacholine-induced Penh over the control. **BAL Fluid Analysis:** There was no significant increase in IFN-γ in the BAL fluid following DEP treatment but there was a significant increase in IL-4 levels compared to the control. (IL-4 increase could indicate that DEP modulates Th-2 cytokines in the mouse model). DEP also induced an increase in total neutrophils and lymphocytes in the BAL when compared to the control. The nitrite concentration in BAL (indicating NO generation) was significantly greater in the DEP exposed group than the control. **Histological Assessment:** Peribronchial and perivascular infiltrates were more common in the group exposed to DEP than the control. **Ym1 and Ym2 Expression:** (see explanation in comments section) Ym1 and Ym2 transcripts were upregulated in response to DEP exposure in mice.
Reference: Steerenberg et al. (2006, 088249) **Species:** Rat **Strain:** Crl/WKY	Ambient air samples PMC, PMF: -I: Rome, Italy -N: Oslo, Norway -PL: Lodz, Poland -NL: Amsterdam, Netherlands Measured Components: Li, Be, B, Na, Mg, Al, K, Ca, Ti, V, Cr, Mn, Fe, Co, Ni, Cu, Zn, As, Se, Sr, Mo, Cd, Sn, Sb, Ba, Ce, Nd, Sm, Au, Hg, Tl, Pb, Bi, U, Si, Endotoxins, Cl, NO^-, SO_4 **Particle Size:** PMC: 2.35-8.5 μm; PMF: 0.12-2.35 μm	**Route:** IT Instillation **Dose/Concentration:** 1 and 2.5mg/animal **Time to Analysis:** Parameters measured 24h post single exposure	**Particle Characteristics:** Concentrations of metals were highest in Rome. Amsterdam was noted for high Mg and V. Lodz was noted for high Pb, Zn, PAH. More of PMC was composed of Fe, Mn, Al, Cr, Cu. More of PMF, on the other hand, was composed of Zn, Pb, Ni, V. **BALF Inflammatory/Injury Markers:** CC16 decreased substantially. Crustal material (endotoxin, Na, Cl and metals but not Ti, As, Cd, Zn, V, Ni, Se) was positively associated with short term CC16. Albumin increased. **Cytokines:** MIP-2 increased dose-dependently. TNF-α also increased. **BALF Cells:** PMNs increased.

Reference	Pollutant	Exposure	Effects
Reference: Stinn et al. (2005, 088307) **Species:** Rat **Gender:** Male and Female **Strain:** Crl: (WIU BR **Age:** 40d	DE (generated from 1.6 L VW diesel under USFTP 72) CO: 10, 37 ppm CO_2: 2170, 6540 ppm NO: 7.0, 22.8 ppm NO_X: 8.6, 28.3 ppm SO_2: 0.83, 3.09 ppm NH_4: ND Measured Major Components: NO, SO_2, 1-nitropyrene, Zi. 50% by DE weight is elemental carbon. **Particle Size:** 3mg/m^3: 0.19 μm (MMAD)	**Route:** Nose-only Inhalation **Dose/Concentration:** 3 and 10 mg/m^3 10 mg/m^3: 0.21 μm (MMAD) **Time to Analysis:** 6h/d, 7d/wk for 24m; 6 m post-exposure	**Body Weight:** Mean weight increased substantially during the first few weeks in all groups. Food consumption decreased in 1-24mo but was recovered in 24-30mo. Body weight decreased at 23mo in all categories, but recovered except in high dose males at 30mo. **Organ Weight:** Absolute weight of lungs, larynx and trachea increased from 0 to 12 to 24mo and stayed elevated at 30 mo: Low < Hi, male ~ female. **Pulmonary Parameters:** Respiratory frequency, tidal volume, and minute volume were unaffected in any group measured between 3 and 24mo. Elemental carbon increased dose-dependently in exposure groups. No male/female difference was observed, but increases were greater at 24 mo than at 18 mo. **BALF Cells:** PMNs and lymphocytes showed dose and time-dependent effects at 18 and 24 mo (no data at 30 mo). Lymphocytes increased 50 fold in high dose males at 24mo. Peripheral monocytes and neutrophils increased 3 fold in DE groups at the end of the study. Particle-filled macrophages in alveolar lumen and interstitium increased at 12, 24, 30mo in both genders at all dose levels. **BALF Inflammatory/Injury Markers:** LDH increased in dose and time-dependent manner. **Hematology:** Erythrocytes were unaffected (12, 24, 30) except in high dose females at 24 and 30 mo. Hemoglobin and hematocrit increased dose-dependently with no gender differences. Leukocytes increased in a dose- and time-dependent manner. **Nasal Cavity Histopathology:** All effects were resolved at 30 mo. Nasal cavity hyperplasia increased at the high dose at 12 and 24mo in both genders. Squamous metaplasia of respiratory epithelium increased in high dose females (12, 24mo). **Larynx Histopathology:** No effects were observed. **Lung Histopathology:** Alveolar region hyperplasia of alveolar epithelium increased at 12, 24, 30mo in both genders at all dose levels except for 12mo low dose males and females. Above lung histopathology was not time-dependent, though perhaps some small dose-dependence was observed.The following histopathology findings showed strong dose- and time-dependent increases that occurred in both genders (24-30 mo): goblet cell hyperplasia of bronchial epithelia, cuboidal/columnar hyperplasia of alveolar epithelium, chronic active inflammation and septal fibrosis. **Tumorigenicity:** Lung tumors were more prevalent in females than males and appeared to be dose-dependent. The major 3 types of tumors are the following:: bronchio-alveolar adenoma, bronchiolo-alveolar adenoma and benign keratinizing cystic cell tumors. Enhanced effects in females versus males may be the result of enhanced metabolism (body volume versus body weight) and increased repiratory volume/bw for females.

Reference	Pollutant	Exposure	Effects
Reference: Sureshkumar et al. (2005, 088306) **Species:** Mouse **Gender:** Male **Strain:** Swiss **Age:** 10-12wks **Weight:** 20-25g	GE: Gasoline Exhaust (Honda generator EBK 1200, four stroke one cyl) Including: SO_2 = 0.11 mg/m^3 NO_X = 0.49 mg/m^3 CO = 18.7 ppm **Particle Size:** GE >4 μm = 34.1 % 3-4 μm = 15.8 % 2-3 μm = 15.8 % 1.5-2 μm = 10.6 % 0.5-1.5 μm = 5.3 % <0.5 μm = 18.4 %	**Route:** Nose-only Inhalation **Dose/Concentration:** 0.635 mg/m^3 **Time to Analysis:** 15min/d 7, 14 or 21d. Parameters measured less than 1h post-exposure	**Cytokines:** GE caused time-dependent increases in TNF-α and IL-6. IL-10 and IL-1β were unaffected. **BALF Inflammatory/Injury Markers:** γ-GGT, ALP and LDH increased after 2wks of GE exposure and stayed stable at 21d. Total protein slightly increased on 14 and 21d, though these increases were not statistically significant. **BALF Cells:** Neutrophils (%) increased at 7, 14 and 21d (stable). Total cell count, macrophages and eosinophils were unaffected. Leukocytes and lymphocytes increased, though not significantly. **Histopathology:** Minor changes at 7d, mild edema in alveolar region at 14d and sloughing of epithelial cells in bronchiolar region and focal accumulation of inflammatory cells in alveolar region at 21d were observed in a time-dependent manner.
Reference: Tesfaigzi et al. (2002, 025575) **Species:** Rat **Gender:** NR **Strain:** Brown-Norway **Age:** 7-8wks **Weight:** 310-330g	WS (wood stove- Vogelzang Boxwood Stove, Model BX-42E, wood- *Pinus edulis*) (CO, NO, NO_x, total hydrocarbon) **Particle Size:** Smaller size fraction: 0.405-0.496 μm, larger size fraction: 6.7-11.7 μm	**Route:** Whole-body Exposure **Dose/Concentration:** Target concentration (low, high exposure): 1, 10mg/m^3; CO- 15-106.4ppm, NO- 2.2-18.9ppm, NO_x- 2.4-19.7ppm, total hydrocarbon- 3.5-13.8ppm **Time to Analysis:** Exposed 3h/d, 5d/wk, 4 or 12wks.	**Respiratory:** Total pulmonary resistance increased for exposure groups and was significant for the low-exposure group. In exposed groups, forced expiratory flows and quasistatic compliance were lower and dynamic lung compliance higher, the latter being significant for the high-exposure group. For the high-exposure group, vital capacity slightly decreased, residual volume slightly increased, and CO-diffusing capacity had a slight, significant decrease. **Histopathology:** WS caused minimal to mild chronic inflammation in the epiglottis of the larynx. PAS-positive cells increased in the 30d high-exposure group. AMs increased with time and concentration. Particle-laden macrophages were seen after 90d. AB- and PAS-positive epithelial cells increased for the 90d low-exposure group. **BALF:** Macrophages decreased significantly in the high-exposure group. Particle-laden macrophages increased with concentration. Lymphocytes and neutrophils slightly increased in the high-exposure group. **Cytokines:** LDH increased slightly and protein levels decreased slightly in the high-exposure group. Cytokines were below detectable levels.
Reference: Tin-Tin-Win-Shwe et al. (2006, 088415) **Species:** Mouse **Gender:** Male **Strain:** BALB/c **Age:** 7wks	CB14: Printex 90 (Degussa) CB90: Flammruss 101 (Degussa) **Particle Size:** CB14: 14nm CB95: 95 nm	**Route:** IT Instillation **Dose/Concentration:** 25, 125, 625 μg/mouse; approx. 1, 5, 25mg/kg **Time to Analysis:** 1/wk for 4wks. mRNA expression in lungs and mediastinal lymph nodes measured 4h post exposure	**Body weight, thymus, spleens, splenic cell count:** No effects were observed. **BALF Cells:** Increased total cell numbers were observed for 125, 625μg CB14 (dose-dependent) and 625μg CB95. Total cell count was twice as high for CB14 at 125 and 625 μg compared to CB95. AM numbers exhibited a dose-dependent response for both CB14 and CB95 for all doses except 125μg. Lymphocyte numbers increased at 125 and 625μg for CB14 and 625μg for CB95. PMN numbers increased at 125 and 625μg for CB14 and CB95, but the response was greater with CB14. PMN numbers were proportional to dose surface area for both PM sizes. **BALF Cytokines:** CB14 and CB95 induced dose-dependent increases in IL-1β. TNF-α increased at 125 and 625μg dose in CB14 with the 125 dose inducing a slightly greater increase. CB14 and CB95 induced CCL-3 increases 125 and 625μg. **Chemokine mRNA in lung and lymph nodes:** CCL-3 mRNA increased for CB14 but not CB95 4h following the last exposure. CCL-2 was unchanged. **Mediastinal lymph nodes:** The number of CB-laden phagocytes increased in a dose-dependent manner for CB14 and CB95. CB14 had higher numbers at all doses compared to CB95.

Reference	Pollutant	Exposure	Effects
Reference: Tong et al. (2006, 097699) **Species:** Mouse **Gender:** Male **Strain:** KP600 CD-1 **Weight:** 22-26g	$PM_{2.5}$ (collected from stacked filter air sampler in Shanghai, China) Fe: FeSO4 Zn: ZnSO4 PMF: $PM_{2.5}$ + FeSO4 PMFZ: $PM_{2.5}$ + FeSO4 + ZnSO4 Major Measured Components: Fe 26ppm, Zn 9ppm, S 61ppm **Particle Size:** $PM_{2.5}$	**Route:** IT Instillation **Dose/Concentration:** PM: 25mg/mL, 1mg/mouse Fe: 15mg/mL, 0.6 mg/mouse Zn: 15mg/mL, 0.6 mg/mouse PMF: PM 25mg/mL + Fe 15mg/mL, 1.6mg/mouse PMFZ: PM 25 mg/mL + Fe 15mg/mL, 1.6mg/mouse **Time to Analysis:** Instilled twice at 0 and 24h. Parameters measured 24h following last exposure (at 48h).	**Synchrotron X-ray imaging (in vivo):** PMFZ showed the greatest increase in alveolar changes. Fe induced more hemorrhagic changes, whereas Zn induced more nonuniformity of lung texture. This suggests that Zn induces PBMC in a dose-dependent manner which releases IL-1, IL-6, TNF-α, and IFN-γ. **Histopathology:** PMFZ induced the most severe changes including serious inflammation/pus in bronchia and bronchial epidermal cell hyperplasia. For Fe or PMF hemorrhagic changes predominated but were less severe than PMFZ.
Reference: Upadhyay et al. (2008, 159345) **Species:** Rat **Gender:** Male **Strain:** SHR **Age:** 6m **Weight:** NR	Ultrafine Carbon Particles (UFCP) **Particle Size:** Size- 31 ± 0.3nm, MMAD- 46nm, Surface area concentration- 0.139 m^2(particle)/m^3(air), Mass specific surface area- 807m^2/g	**Route:** Whole-body Exposure **Dose/Concentration:** 172 $\mu g/m^3$ **Time to Analysis:** Acclimatized 2d. 1d baseline. 24h exposure. 4d recovery. Sacrificed 1st or 3rd day of recovery.	**Cardiophysiology:** The mean arterial BP and HR increased but returned to baseline levels by the 4th recovery day. SDNN and HRV decreased. RMSSD and LF/HF decreased but were not significant. **Pulmonary Inflammation:** UFCP did not cause pulmonary inflammation. **Pulmonary and Cardiac Tissue:** HO-1, ET-1, ETA, ETB, TF, PAI-1 significantly increased in the lung on the 3rd recovery day. HO-1 was repressed in the heart, but the other markers had slight, nonsignificant increases. **Systemic Responses:** Neutrophil and lymphocyte cell differentials significantly increased on the 1st recovery day. Other blood parameters were unaffected. The plasma renin concentration increased on the first 2 recovery days. Ang I and II concentrations increased on the 1st recovery day but was not significant.
Reference: Wallenborn et al. (2007, 156144) **Species:** Rat **Gender:** Male **Strain:** WKY, SHRWKY, and stroke-prone SH (SHRSP) **Age:** 12-15wks	PM: precipator unit power plant residual oil combustion **Particle Size:** PM: 3.76μm (bulk) ± 2.15	**Route:** IT Instillation **Dose/Concentration:** WKY vs SHRSP: 1.11, 3.33, 8.33 mg/kg SH vs SHRSP: 3.33, 8.33 mg/kg **Time to Analysis:** WKY vs SHRSP: single exposure, parameters measured 24h post-exposure. SH vs SHRSP: single exposure, parameters measured 24h post-exposure. Note: 4h post-exposure study done on WKY vs SHRSP but not published.	**BALF Cells:** A dose-dependent increase in total cells and neutrophils was observed. Equal response for all 3 strains except for SH, for both concentrations was observed. **BALF inflammation/Injury Markers:** LDH exhibited a dose-dependent increase in equal response for all 3 strains. WKY had higher baseline levels of NAG activity but, upon PM exposure, SHRSP induced higher increases than WKY. GGT exhibited a dose-dependent response for all 3 strains. SHRSP showed the highest increase followed by WKY and SH. Protein levels increased at the high dose level with SHRSP exhibiting the highest increases followed by SH and WKY. Albumin levels were inconsistent between experiments. **Oxidative Stress - Lung:** (WKY vs SHRSP only): SOD decreased following increased exposure levels with SHRSP levels generally higher than WKY. Ferritin levels declined only in SHRSP. **Oxidative Stress - Cardiac:** SOD increased in the SHRSP vs WKY experiment only. Only SHRSP at 8.33 mg/kg showed a significant increase when compared to the control. **GPx:** No action but SHRSP levels were similar to SHR and, in the WKY vs SHRSP experiment, SHRSP exhibited higher activity level than WKY. **Ferritin:** Equivocal results were observed. Levels decreased at the high dose for WKY and SHRSP but increased at medium doses for SH and SHRSP. **ICDH:** Levels increased for WKY and decreased for SHRSP.

Reference	Pollutant	Exposure	Effects
Reference: Wallenborn et al. (2008, 191171) **Species:** Rat **Gender:** Male **Strain:** Wistar Kyoto (WKY) **Age:** 13wks **Weight:** NR	Zinc Sulfate ($ZnSO_4$, aerosolized) **Particle Size:** NR	**Route:** Nose-only Inhalation **Dose/Concentration:** 9.0 ± 2.1 μg zinc/m^3, 35 ± 8.1 μg zinc/m^3, 123.2 ± 29.6 μg zinc/m^3 **Time to Analysis:** Exposed 5h/d, 3d/wk, 16wks. Half of the rats used for plasma/serum analysis, other half for isolation of cardiac mitochondria.	A trend toward increased BALF protein was seen. Cardiac mitochondrial ferritin had a small, significant increase. Mitochondrial succinate dehydrogenase and glutathione peroxidase had small, significant decreases. Subchronic exposure to 100 μg/m^3 caused expression changes of cardiac genes involved with cell signaling events, ion channels regulation, and coagulation. No pulmonary-related effects were seen.
Reference: Wegesser and Last (2008, 190506) **Species:** Mouse **Gender:** Male **Strain:** BALB/c **Age:** 8-10wks	Ambient $PM_{2.5}$ -PM_{10} Collected from San Joaquin Valley, CA **Particle Size:** $PM_{10-2.5}$	**Route:** IT Instillation **Dose/Concentration:** 25-50 μg/mouse **Time to Analysis:** 3, 6, 18, 24, 48, 72h post IT instillation.	**BALF Cells:** Increased amount of viable cells found in PM-exposed mice with dose-response relationship between dose of PM and number of total cells recovered in BALF. At 6h, increased numbers of macrophages at both 25 and 50 μg/mouse. Increased percentage of neutrophils observed with 50 μg/mouse PM only. Furthermore, both macrophages and neutrophils increased with longer time period from instillation, peaking at 24 h. MIP-2: At 50 μg/mouse, MIP-2 concentrations increased, peaking at 3h, though not statistically significant and returned to basal levels by 6h. Positive correlation observed between MIP-2 concentration and increased neutrophil counts. No correlation found between MIP-2 and macrophages.
Reference: Whitekus et al. (2002, 157142) **Species:** Mouse **Gender:** Female **Strain:** BALB/c **Age:** 6-8wks **Weight:** NR	DEP (light-duty, four-cylinder engine- 4JB1 type, Isuzu Automobile, Japan; standard diesel fuel) (extracts) **Particle Size:** 0.5-4 μm	**Route:** Inhalation **Dose/Concentration:** 200, 600, 2000 μg/m^3 **Time to Analysis:** Exposed 1h/d 10d. Animals receiving OVA had 20min OVA exposure after DEP exposure.	DEP+OVA dose-dependently increased IgE and IgG1, being more effective than the OVA-alone treatment. This effect was significantly suppressed by thiol antioxidants NAC or BUC. DEP+OVA increased carbonyl protein and lipid peroxide over OVA. NAC or BUC suppressed lipid peroxide and protein oxidation. No general markers for inflammation were observed.
Reference: Wichers et al. (2004, 055636) **Species:** Rat **Gender:** Male **Strain:** SH **Age:** 75d	PM (HP-12): inside wall of stack of Boston, MA power plant burning # 6 oil. **Particle Size:** PM: 3.76 μm ± 2.15	**Route:** IT Instillation **Dose/Concentration:** 0.83, 3.33 or 8.33 mg/kg **Time to Analysis:** single, 6h for Whole-body plethysmographs (WBP) and repeated daily for 4-7d, 96 or 192h post-exposure non-WBP animals: single, 24, 96, 192h post-exposure	**Tidal Volume:** A dose-dependent decrease in tidal volume (45 % at high dose) was sustained for 1d with very slow recovery over 7d. **Breathing frequency:** Dose-dependent increase (100 % at high dose) with recovery at 7d was observed. **Minute ventilation:** Small dose-dependent increases were observed with a return to normal ventilation in 2d. **Penh (enhanced pause):** Equivocal results in all groups were observed (due to major control variation). **BALF Cells:** Dose-dependent increases in total cells at 24 h, with declined, but still elevated, levels at 192h. Neutrophils increased significantly (10 fold) at 24 h in the mid and high dose groupsand showed declined, but still elevated, levels at 192h. Macrophages slowly increased in a dose-dependent manner at 192h. **BALF Inflammatory/Injury Markers:** Protein and albumin increased at 24 h, returned to relative basal level at 192h at the mid and high dose levels. NAG exhibited dose-dependent increases at 24h and sustained these levels through 192h.

Reference	Pollutant	Exposure	Effects
Reference: Wichers LB et al. (2006, 103806) **Species:** Rat **Gender:** Male **Strain:** SH **Age:** 71-73d **Weight:** 255-278g	PM (HP-12): inside wall of stack of Boston, MA power plant burning # 6 oil. **Particle Size:** 1.95 μm ± 3.49	**Route:** Whole-body Inhalation **Dose/Concentration:** 13 mg/m^3 **Time to Analysis:** Phase I: 1st day, filtered air, 2nd day, 6h of PM Phase II: 1st Da, y filtered air, 4 days of 6h PM each Immediate post-exposure	**Body/ Lung weight:** No effects on Phase I rats were observed. HP-12 exposure increased body weight, left lung, right intercostal, and right diaphragmatic lobes in Phase II rats. However, results appeared due to normal growth apptern in juvenile rats over 4d. **Lung lobe to Body Weight Ratio:** No effects at 1 or 4d were observed. **Deposition calculations:** V and Co were used to estimate deposition rates (good correlation between two metals at R2 = 0.94). Total HP-12 deposition using Co was 26 and 99μg (for 1 day and 4 day experiments) and using V was 31 and 116μg. Modeling information estimated HP-12 deposition at 43% in conducting airways and 57% in alveolar region. **Breathing parameters:** No changes were observed for 1 or 4d studies except for a possible decrease in frequency for the 1d study.
Reference: Witten et al. (2005, 087485) **Species:** Rat **Gender:** Female **Strain:** F344 **Age:** 8wks **Weight:** ~175g	DEP (heavy-duty Cummins N14 research engine operated at 75% throttle) **Particle Size:** 7.234-294.27nm	**Route:** Nose-only Inhalation **Dose/Concentration:** Low- 35.3±4.9 μg/m^3, High- 632.9±47.61 μg/m^3 **Time to Analysis:** Exposed 4h/d, 5d/wk, 3wks. Pretreated with saline or capsaicin.	There were no differences for substance P. The low-exposure group had significantly less NK1. DEP reduced NEP activity. Plasma extraversion dose-dependently increased and was greatest in capsaicin animals. Respiratory permeability dose-dependently increased. IL-1β was significantly higher for the low-exposure group. IL-12 was significantly lower in the capsaicin high-expsoure group. TNF-α increased in the high-exposure group and capsaicin low-exposure group. High exposure induced particle-laden AMs in the lungs, perivascular cuffing consisting of mononuclear cells, alveolar edema and increased mast cell number. Neutrophil and eosinophil influx was not seen.
Reference: Wong et al. (2003, 097707) **Species:** Rat **Gender:** Female **Strain:** F344/NH **Age:** ~4wks **Weight:** ~175g	DEP (Cummins N14 research engine at 75% throttle) (EC-34.93-601.67 μg/m^3, OC-1.90-11.25 μg/m^3, Sulfates 0.94-17.96 μg/m^3, Na- 4.07-4.78 ng/m^3, Mg- 0.60-0.86 ng/m^3, Ca- 5.05-10.66 ng/m^3, Fe- 3.17-6.44, Cr- 0.68-1.31 ng/m^3, Mn- 0.11-0.22 ng/m^3, Pb- 0.97-1.24 ng/m^3) **Particle Size:** 7.5-294.3nm	**Route:** Nose-only Inhalation **Dose/Concentration:** Low- 35.3±4.9 μg/m^3, High- 669.3±47.6 μg/m^3 **Time to Analysis:** Exposed 4h/d, 5d/wk, 3wks. Pretreated with saline or capsaicin.	DEP dose-dependently increased plasma extraversion, which was further increased by capsaicin. In the high-exposure group, particle-laden AMs (which were reduced by capsaicin), inflammatory cell margination, perivascular cuffing with subsequent mononuclear cell migration and dispersal, increased mast cells, and decreased substance P were all seen. NK-1R was downregulated in the low-exposure group and upregulated in the capsaicin-pretreated high-exposure group. NEP decreased significantly for both groups.
Reference: Wu, W. Wang, X. Zhang, W. 2003 **Species:** Rat **Gender:** Male **Strain:** Sprague-Dawley **Age:** 60d	Zn2+ **Particle Size:** NA	**Route:** IT Instillation **Dose/Concentration:** 50 μm/rat **Time to Analysis:** Single, 24 h	**Cells:** Decreased number of airway epithelial cells shown with PTEN protein immunostaining. Macrophages were unaffected.

Reference	Pollutant	Exposure	Effects
Route: Yamamoto et al. (2006, 096671) **Species:** Mouse **Gender:** Male **Strain:** BALB/c **Age:** 7wks **Weight:** 23g	CB14: Printex 90 (Degussa) CB95: Flammruss 101 (Degussa) LTA: Lipoteichoic acid 14CL: CB14 + LTA 95CL: CB95 + LTA CB14 measured Components: C 96.79%, HR 0.19%, NO.13%, S 0.11%, Ash 0.05%, O 2.74% CB95 measured Components: C 97.98%, HR 0.15%, N 0.28%, S 0.46%, Ash 0%, O 1.14% **Particle Size:** CB14: 14nm CB95: 90 nm	**Route:** IT Instillation **Dose/Concentration:** CB14: 0, 25, 125, 625 μg/mouse CB95: 0, 25, 125, 625 μg/mouse LTA: 10 or 50 μg/mouse 14CL: 125 μg CB14 + 10 or 50 μg LTA 95CL: 125 μg CB95 + 10 or 50 μg LTA **Time to Analysis:** Single, 4 and 24h	**BALF Cells:** CB95 induced dose-dependent increases of PMN. CB14 induced an increase in PMNs but the increases were not dose-dependent. LTA massively increased PMN. LTA induced dose-dependent increases in total cells, especially at high dose at 24 h. LTA had massive synergistic effect with CB14 and CB95 for total cells and PMNs. Total cell count and PMN levels were highest in 14CL with levels at 24h higher than at 4h. Macrophage data were inconsistent. **Cytokines:** CB95 induced dose-dependent increases in IL-6, TNF-α, CCL2 and CCL3. CB14 induced dose-dependent increase in CCL2 and CCL3. Exposure induced increases of IL-6 at the high dose only. Slight effect on TNF-α was observed. LTA induced dose-dependent increases of IL-6, TNF-α and CCL3. 14CL massively induced IL-6 and CCL2. No combination of CB and LTA affected TNF-α or CCL3. **mRNA Expression:** LTA, 14CL and 95CL increased TLR2 mRNA expression with 95CL and 14CL inducing higher increases than LTA. No effect on TLR4 mRNA expression was observed.
Reference: Yanagisawa et al. (2003, 087487) **Species:** Mouse **Gender:** Male **Strain:** ICR **Age:** 6wks **Weight:** 29-33g	DEP: (4JB1 light duty 4cyc 2, 74 liter Isuzu engine) LPS DEP-OC: organic compounds DL: DEP + LPS DOL: DEP-OC + LPS **Particle Size:** 0.4 μm	**Route:** IT Instillation **Dose/Concentration:** DEP/DEP-OC: 125 μg/mouse LPS: 75 μg/mouse **Time to Analysis:** Single, 24 h	**BALF Cells:** DEP and DEP-OC increased neutrophils but the increases were not statistically significant. LPS increased neutrophils significantly. DL and DOL massively increased neutrophils at greater levels than LPS alone. Macrophages were unaffected. **Pulmonary Edema:** LPS, DEP and DEP-OC increased edema. DL further increased this effect. DOL had no effect compared to LPS alone. **Histology:** DL elevated neutrophil inflammation interstitial edema and alveolar hemorrhages. DOL induced neutrophilic inflammation without the alveolar hemorrhages. **Cytokines:** LPS increased IL-1β, MIP-1α, MCP-1 and KC. DEP and DEP-OC had no effect. DL induced further increases. DOL decreased cytokines compared to LPS alone. DEP-OC increased IL-1β and MIP-1α mRNA expression slightly. DEP had no effect. LPS significantly increased IL-1β and MIP-1α mRNA expression. DL increased expressions while DOL did not. **mRNA Expression of TLRs:** DEP-OC, DL, DOL and LPS increased TLR2. DEP had no effect. All particles increased TLR4 mRNA expression.

Reference	Pollutant	Exposure	Effects
Reference: Yokohira et al. (2007, 097976) **Species:** Rat **Gender:** Male **Strain:** F344/ DuCrj **Age:** 10wks	DQ-12: Quartz dust (Douche Montan) HT: Hydrotalcite (Kyoward 500, PL-1686, KYOWA) POF: Potassium Octatitanate fiber (TISMO, Otsuka) PdO: Palladium Oxide CB: Carbon Black (Mitsubishi Kasei) **Particle Size:** DQ12 $< 7\mu$m HT: 7.8 $\pm 1.5\ \mu$m POF: $<$ 50um length; $< 2\ \mu$m width PdO: 0.54 $\pm$ 1.11 μm CB: 28nm	**Route:** IT Instillation **Dose/Concentration:** 4mg/rat in 0.2ml saline **Time to Analysis:** Single, 1 and 28d	**Lung weight/body weight ratio:** DQ-12, HT and POF induced increases after 1d. After 28 days, all samples induced increases in lung weight. **BALF Cells:** Neutrophils increased significantly in walls and alveolar spaces in all groups on 1d except at HT. AT 28d, this increase was maintained only in walls with severe and moderate elevations, except for DQ-12. **Histopathology:** DQ-12 caused pulmonary edema both at 1 and 28d. PdO and CB induced edema at 28d. Fibrosis was observed after 28d with the most significant increase, in decreasing order, induced by DQ-12,PdO, POF, HT, CB, and the control. Histiocyte infiltration was observed after 1d for DQ-12, POF and PdO. At 28d, infiltration was observed for DQ-12, HT, POF and PdO. Restructuring of alveolar walls and microgranulation was observed for all 5 particles but only at 28d with DQ 12, PdO, HT, POF, CB and control. **Immunohistochemistry:** BrdU: At 1d all 5 particles elevated in both area and number. Activity declined after 28d but was still higher than the control. **iNOS:** At 1d DQ-12, POF and PdO induced increases. At 28d, DQ-12 and HT induced increases. **MMP-3:** DQ-12 induced increases at both 1 and 28d and PdO at 28d. **Toxicity scoring:** The levels of toxicity are, in decreasing order, as follows: DQ-12, HT/PdO/POF, and CB.
Reference: Zhao et al. (2006, 100996) **Species:** Rat **Strain:** Sprague-Dawley	DEP: SRM 2975 DEPE: SRM 1975 **Particle Size:** NR	**Route:** IT Instillation **Dose/Concentration:** 35mg/kg bw **Time to Analysis:** AG (amino guanidine) group pre-treated with 100 mg/kg bw. Single, 1d. AG group coexposed 30 pre and 3, 6, 9h post DEP/DEPE	**iNOS expression in AMs:** Both DEP and DEPE increased 12 and 6 fold respectively. NO and peroxynitrite levels increased accordingly. AG had no effect on iNOS expression but AG attenuated NO for both DEP and DEPE but peroxynitrite only for DEPE. DEP induced much higher levels of oxidants than DEPE. Unlike DEPE, DEP was unaffected by AG. **Role of iNOS in Lung Injury:** DEP and DEPE induced inflammation (PMN), cellular toxicity (LDH) and lung injury (protein). AG significantly attenuated the DEPE response but no effect was observed on the DEP responses. **Cytokines:** IL-12 levels were induced by both DEPE andDEP, with DEPE inducing higher increases than DEP, and both were significantly attenuated by AG. DEP and DEPE induced similar increases in IL-10 levels. AG increased DEP effect 3 fold and attenuated DEPE to control. **CYP enzymes:** DEP and DEPE induced increases in CYP1A1 level and activity.AG attenuated CYP1A1 activity for both DEP and DEPE. CYP2B1 level and activity were slightly decreased by DEP and DEPE. AG had no effect. **Cytosol phase II enzymes:** DEPE had no effect; AG treatment increased catalase activity. DEP reduced catalase and GST activities. AG had no effect. Neither DEP, DEPE nor AG affected QR quinone reductase.

Reference	Pollutant	Exposure	Effects
Reference: Zhou et al. (2003, 087940) **Species:** Rat **Gender:** Male **Strain:** Sprague-Dawley **Age:** 10-12wks	UFe: Ultrafine Fe particles **Particle Size:** 72nm	**Route:** Whole-body Inhalation **Dose/Concentration:** 57 or 90 μg/m^3 **Time to Analysis:** 6h/d for 3d, parameters measured within 2h post-exposure.	**BALF Inflammatory/Injury Markers:** At the high dose, total protein increased. No significant changes were observed in LDH. **BALF Cells:** No significant changes observed in total cell number, cell viability or cell differentials. **Intracellular ferritin:** The high dose induced increases. No significant differences were observed between the low dose and control. **Oxidative stress:** Antioxidant level by FRAP value decreased at the high dose. GST (glutathione-S-tranferase) activity increased at the high dose. No effect on intracellular GSH and GSSG (glutathione disulfide) was observed. **Cytokines:** Only at thehigh dose was an increase in IL-1β observed. No effect on TNF-alph or NFkB-DNA binding activity was observed.

Table D-4. Effects related to immunity and allergy.

Study	Pollutant	Exposure	Effects
Reference: Apicella C et al. (2006, 096586) **Species:** Mouse **Strain:** BALB/c **Cell Line:** 112D5 hybridoma Primary Macrophages: Peritoneal	Poly OVA (Ovalbumin on polystyrene beads) Soluble OVA **Particle Size:** NR	**Route:** Cell Culture **Dose/Concentration:** PolyOVA and Soluble OVA: 0.2,1.0 or 5.0 μg/mL **Time to Analysis:** 48 h	**IL-6:** Stimulation with PolyOVA higher than stimulation with soluble OVA **TNFα:** Stimulation with PolyOVA higher than stimulation with soluble OVA. **IL-10:** No modifications in levels after PolyOVA or soluble OVA stimulation. **Viability of Peritoneal macrophages:** Stimulation with PolyOVA led to 33% decrease in viability. Stimulation with soluble OVA led to 24% in viability. **Effects of PolyOVA stimulated macrophages:** Culture supernatants from PolyOVA stimulated macrophages had a percentage increase of asymmetric IgG; however, the addition of rmIL-6 at identical concentrations did not induce a significant increase. It also decreased the proliferation of 112D5 hybridoma.
Reference: Arantes-Costa et al. (2008, 187137) **Species:** Mouse **Gender:** Male **Strain:** BALB/c **Age:** 6wks **Weight:** NR	ROFA (solid waste incinerator powered by combustible oil; São Paulo, Brazil) **Particle Size:** NR	**Route:** Intranasally Instilled **Dose/Concentration:** 60 μg ROFA in 50 μL saline **Time to Analysis:** OVA sensitized days 1 and 14. OVA-challenged days 22, 24, 26, and 28. ROFA exposed 1-3h after OVA challenge or saline. Pulmonary responsiveness measured day 30 then sacrificed. Lungs removed, fixed for 48h.	ROFA increased pulmonary responsiveness and decreased ciliated cells in nonsensitized mice, which were both further amplified in the presence of OVA. ROFA did not affect eosinophils, macrophages, chronic inflammation, or neutral or acidic mucus.

Study	Pollutant	Exposure	Effects
Reference: Araujo et al. (2008, 156222) **Species:** Mouse **Gender:** Male **Strain:** ApoE$^{-/-}$, C57BL/CJ **Age:** 6wks **Weight:** NR	CAPs (Los Angeles, California freeway; Nov-Dec 2005) **Particle Size:** Fine particles (FP): <2.5 μm; UFP: <0.18 μm	**Route:** Whole-body Exposure **Dose/Concentration:** FP: $\sim$440 μg/m^3, UFP: $\sim$110 μm; PM number concentration: FP: 4.56 X 10^5 particles/cm^3, UFP: 5.59 X 10^5 particles/cm^3 **Time to Analysis:** Exposed 5h/d, 3d/wk for total of 75h. Killed 24 to 48h postexposure.	**Composition:** UFP had a higher particle number, surface area, PAH content and fractional carbon composition than FP. **Atherosclerosis:** UFP had significantly greater aortic atherosclerotic lesions than FP and the control. The lesions were comprised of macrophage infiltration with intracellular lipid accumulation. Plaques were thicker and more extensive in the UFP group. **HDL:** FP had increased plasma total cholesterol. Plasma HDL from both groups had decreased protective effects. The anti-inflammatory effect was lower in the UFP group. **Oxidative Stress:** Lipid peroxidation increased in the FP group. The UFP group had increases in hepatic malondialdehyde, Nef2, catalase, glutathione S-transferase Ya, NAD(P-quinone oxidoreductase and superoxide dismutase 2.
Reference: Archer et al. (2004, 088097) **Species:** Mouse **Strain:** BALB/c DO11.10+/+ transgenic - ova specific receptor for OVA peptide 323-339 **Age:** 4 wks	PM = SRM 1648 (NIST) Titanium dioxide (TiO_2) as a control particle **Particle Size:** SRM1648: avg 1.4 μm TiO_2: avg 0.3 μg (sic)	**Route:** Intranasal instillation **Dose/Concentration:** 500 μg PM/30 μl sterile saline (ultrasonic suspension) initial 0-750 μg range finding **Time to Analysis:** Ova challenge at 68h, Meth-acholine aerosolization/AR at 72h	**Airway responsiveness (WBP):** AR induced by Ova/Mch challenge was significantly and dose-dependently increased at doses of SRM1648 ≥500ug . TiO_2/Ova exposure was not significantly different from saline. PM associated endotoxin did not contribute to enhanced AR. **Lung inflammation/pathology:** No increases in BAL macrophages or eosinophils and no histological alterations after PM exposure. Both TiO_2 and PM increased pulmonary neutrophils, indicating particles alone were responsible for this increase and that the inflammatory response could occur independently of AR.
Reference: Barrett et al. (2006, 155677) **Species:** Mouse **Gender:** Male **Strain:** BALB/c **Age:** 8-10wks **n:** Groups of 15-16 incl. controls	HWS (black/white oak) CO Total Vapor Hydrocarbon (TVH) **Particle Size:** 0.25 ± 3.3, 0.35 ± 2.5, 0.35 ± 2.0, 0.36 ± 2.1 μm (MMAD±GSD)	**Route:** Whole-body Inhalation **Dose/Concentration:** HWS: 30, 100 300, 1000 μg/m^3 CO: 0.7, 1.6, 4.0, 13ppm TVH: 0.3, 0.6, 1.3, 3.1ppm **Time to Analysis:** Pretreatment: ip 10 μg OVA and 2mg aluminum hydroxide post-OVA. OVA aerosol challenge on day 14, followed by 3d of HWS. Pre-OVA received aerosol OVA challenge on day 14, then 3d of HWS on days 26-28 and an immediate (second) OVA challengeHWS 6 h/d for 3d. Sacrificed 18h post-exposure.	**Allergic Inflammation:** A statistically significant increase in eosinophils was observed at 300 μg/m^3 HWS following OVA challenge as compared to OVA alone. No changes in macrophages, neurophils and lymphocytes were observed. Post-OVA HWS did not significantly alter BAL cytokine or serum antibody levels, but linear trend analyses indicated decreases in IL-2, IL-4, and IFN□ in the absence of OVA, as well as a statistically significant upward trend in OVA-specific IgE when HWS exposure followed OVA challenge. HWS exposure pre-OVA (prior to second OVA challenge) resulted in a decrease in IL-13 (statistically significant at the high dose but no evidence of an exposure-dependent response), an increase in OVA IgG1 (trend significant) and no change in IL-2, IL-4, IL-5, IFN□, OVA IgE, total IgE or OVA IgG2a.
Reference: Burchiel et al. (2005, 088090) **Species:** Mouse **Gender:** Female **Strain:** A/J **Age:** 12-14wks	HWS (black/white oak) HWS particle Mass BC Organic Carbon (OC) CO Total Vapor Hydrocarbon 29 other minor components PAH and metals **Particle Size:** 0.3±3, 0.4 ±2, 0.4±2, 0.4 ±2 μm (MMAD±GSD)	**Route:** Inhalation Chambers **Dose/Concentration: HWS:** 30, 100, 300, 1000 μg/m^3 BC: 3, 12, 25, 43 μg/m^3 OC: 40, 107, 281, 908 μg/m^3 CO: 1, 2, 4, 13 ppm TVH: ND, 1, 1, 3 ppm **Time to Analysis:** 6 h/d for 6m	**Proliferative responses:** HWS increased splenic T cell proliferation at 100 μg/m^3 with a dose dependent decrease at 300 and 1000 μg/m^3 exposures ($p<0.05$) HWS exposure did not affect T (CD3), helper T cell (Th, CD4), cytotoxic T cell (CTL, CD8), macrophage (Mac-1), natural killer cell (NK, CD16) cell markers or B cell proliferative response to LPS.

Study	Pollutant	Exposure	Effects
Reference: Burchiel et al. (2004, 055557) **Species:** Mouse **Strain:** AJ **Age:** 10-12wks	DE generated alternatively from two 2000 Cummins ISB Turbo Diesel 5.9 L engines using no 2 (chevron) oil and 15w/40 oil (Rotella T, Shell) run according to USEPA Dynamometer Schedule for Heavy-Duty Diesel Engines 18 PAHs quantified at exposure levels (text mentions 65) **Particle Size:** NR	**Route:** Inhalation Chambers **Dose/Concentration:** 30, 100, 300, 1000 mg/m^3 diesel PM **Time to Analysis:** 6 h/d, 7 d/wk for 6m	**Proliferative responses:** DE depressed splenic T cell proliferation at all exposure levels but was not dose-dependent and most pronounced at the 30 μg/m^3 level. ($p < 0.05$ at all levels) Splenic B cell proliferation was increased at the 30 μg/m^3 level, but not at the other exposure levels. Little, if any, PAH was found in DE, and the majority of PAH tested in vitro enhanced T cell proliferation (below), so PAH is likely not responsible for the immunosuppressive effect of DE on murine spleen cell responses.
Reference: Burchiel et al. (2004, 055557) **Species:** Mouse **Strain:** AJ **Age:** 10-12 weeks **Cell Type:** spleen cells **Use:** In vitro	Benzo(ayrene (BaP) Benzo[a]pyrene-r-7,t-8-dihydrodiol-t-9,10 epoxide(±) ((antiPDE) Benzo[a]pyrene-trans-7,8-dihydrodiol (±) (BP-7,8-diol) Benzo[a]pyrene-1,6-dione (1,6-BPQ) Benzo[a]pyrene-3,6-dione (3,6-BPQ) Benzo[a]pyrene-6,12-dione (6,12 BPQ) **Particle Size:**NR	**Route:** Cell Culture **Dose/Concentration:** 1 x 10 6 cells/mL in 100 μl aliquots 0.01, 0.1 and 1 μm **Time to Analysis:** 72h	BaP at the highest concentration was found to double splenic T cell proliferation. The BPQs all also increased T cell proliferation at much lower concentrations but not in a dose dependent manner. Splenic B cell was increased by Bp-(7,8)-diol, and inhibited by BPDE and 3,6 BPQ but only at the highest level. Authors concluded that due to low level of PAH in DE and absence of BPQs these compounds are not responsible for immunosuppressive effects of DE.
Reference: Chan (2006, 090193) **Species:** Mouse **Strain:** DO11.10, BALB/c, *Nrf 2 -/-* **Cell Types:** Primary bone marrow dendritic cells and dendritic cell line (BC1), T cells (BMDC)	DEP: DE particles DEP methanol extract: **Particle Size:** PM > 50 as characterized by Singh et al. 2004	**Route:** Cell Culture **Dose/Concentration:** DEPs 10 μg/mL LPS 5ng/mL **Time to Analysis:** 24h, stored at -20ºC until analyzed	**Dendritic cell maturation:** Organic DEP chemicals interfered in the expression of several DC maturation markers. Both DEP and DEP extracts were found to inhibit CD86 expression and IL-12 production in LPS-exposed DCs, and intact particles were not as effective as DEP extract. DEP extract treatment of BC1 cells reduced their ability to stimulate co-cultured antigen-specific T cells, leading to decreased IFN- γ and increased IL-10 without affecting IL-4 or IL-13. DEP extract also induced oxidative stress and interfered with DC activation by several other Toll-like receptor agonists as well as the NF-kB cascade. Inhibition of IL-12 production by DEP extract was shown to be mediated by pro-oxidative chemicals that engage the Nrf2 pathway. Taken together the inhibition of both IL-12 and IFN- γ indicates a suppression of the Th1 pathway and provides a novel explanation for the adjuvant effect of DEPs on allergic inflammation

Study	Pollutant	Exposure	Effects
Reference: Ciencewicki et al. (2007, 096557) **Species:** Mouse **Gender:** Female **Strain:** BALB/c **Age:** 10-12wks **Weight:** 17-20g	DE: generated from a 30-kW (40 hp), 4-cylinder Deutz BF4M1008 diesel engine Influeza A/Bangkok/1/79 (H3N2 serotype) from Dr. Melinda Beck of the University of North Carolina, Chapel Hill O_2, CO, NO_2, NO, SO_2 O_2: 20.9- 20.5% (Lo, Hi) CO: 0.9-5.4 ppm NO_2: 0.25-1.13 ppm NO: 2.5-10.8 ppm SO_2: 0.06-0.32 ppm H3N2: NR **Particle Size:** NR	**Route:** Inhalation (oropharyngeal aspiration of virus) **Dose/Concentration:** DE: 529 or 2070 $\mu g/m^3$ **Time to Analysis:** 4 h/d for 5d. Virus exposure immediately after last DE exposure. Analyzed 18h post infection	**DE exposure on susceptibility to Influenza infection:** Mice exposed to 0.5 mg/m^3 had significantly greater levels of HA mRNA compared to air-exposed mice. HA levels not significantly altered in mice exposed to 2.0 mg/m^3. **DE exposure on the Influenza-induced inflammatory response:** f IL-6 mRNA levels were significantly greater when exposed to 0.5 mg/m^3 of DE prior to infection compared to air exposure. Significantly increased amount of IL-6 protein observed in exposed mice. Exposure to DE in absence of influenza infection had no significant effect on IL-6 mRNA or protein levels. **DE exposure on pulmonary injury:** Infection with the influenza virus increases levels of PMN in BAL fluid. Exposure to either dose of DE prior to infection showed no significant effect on PMN levels Exposure to DE alone had no effect on PMNs in BAL fluid. Neither exposure to DE nor infection with influenza significantly increased BAL fluid protein levels when compared to non-infected, air-exposed. **Other markers of injury,** NAG and MIA were not statistically affected by DE or influenza exposure. **DE exposure on the influenza induced interferon response:** No significant change in TFN-α mRNA levels at either dose of DE, although mice exposed to 0.5 mg/m^3 of DE prior to infection had significantly greater levels of IFN-B MRNA compared to air controls. No effect on any of the IFNs observed in uninfected mice exposed to DE. **DE exposure on surfactant protein expression:** Influenza virus infection alone significantly increased expression of SP-A in air-exposed. Exposure to 0.5 mg/m^3 of DE prior to infection had significant decreases in levels of SP-A mRNA in the lungs, this effect was not observed in 2.0 mg/m^3 DE exposed. Decrease seen in expression of SP-A protein in lungs of mice exposed to 0.5 mg/m^3 DE prior to infection. Levels of SP-D mRNA and protein were significantly decreased in lungs of mice exposed to 0.5 mg/m^3 of DE prior to infection compared with mice exposed to air or 2.0 mg/m^3 DE prior to infection. Exposure to 0.5 mg/m^3 of DE prior to infection with influenza decreased levels of SP-D, especially in airways. Mice exposed to 2.0 mg/m^3 DE prior to infection showed no significant difference.
Reference: Day et al. (2008, 190204) **Species:** Mouse **Gender:** Male **Strain:** BALB/c **Age:** 8-10wks **Weight:** NR	GEE (General Motors 1996 model 4.3-L V6 engine; regular unleaded fuel) (CO, NO, NO_2, SO_2, NH_3) **Particle Size:** NR	**Route:** Whole-body Exposure Chamber **Dose/Concentration:** Low(L)- 6.6 ± 3.7 PM/m^3, Medium(M)- 30.3 ± 11.8 PM/m^3, High(H)- 59.1 ± 28.3 PM/m^3, High-Filtered(HF)- **Time to Analysis:** Pre-OVA protocol: OVA or saline sensitized 7d. OVA challenge day 14. GEE or air exposed 6h/d on days 26-28. Immediately after exposure on day 28 challenged with OVA. Tested for MCh-induced changes 24h postexposure then sacrificed. Post-OVA protocol: OVA or saline sensitized 7d. OVA challenge day 14. GEE or air exposed days 15-17. Tested for MCh-induced changes 24h postexposure then sacrificed.	**Post-OVA:** In nonsensitized mice, neutrophils dose-dependently decreased, IL-4 decreased in the M group, IL-5 decreased in the HF group, and IFN-¤ decreased at all exposures. In OVA-sensitized mice, IL-13 dose-dependently decreased. **Pre-OVA:** In nonsensitized mice, neutrophils and IgE decreased in the H group. IL-2 increased in the HF group and was dose-dependent. Eosinophils dose-dependently decreased. OVA-specific IgE increased in the H group, and OVA-specific IgG_{2a} dose-dependently increased. In OVA-sensitized mice, OVA-specific IgG_1 increased in the M group. Airway hyperresponsiveness was lower in the M and HF groups.

Study	Pollutant	Exposure	Effects
Reference: de Haar et al. (2005, 097872) **Species:** Mouse **Gender:** Female **Strain:** BALB/cANNCrl **Age:** 6-8wks **Weight:** NR	CBP: Carbon black particles in phosphate buffered saline, 1: 10 & 1: 100 dilutions (Brunschwich Chemicals, Amsterdam, The Netherlands) OVA: Ovalbumin (igma-Aldrich, Zwijndrecht, The Netherlands) **Particle Size:** CBP: 30-50nm	**Route:** Intranasal Droplet **Dose/Concentration:** CBP± OVA 200, 20, 2 μg (3.3, 0.33, 0.033 mg/ml) OVA only: 20 μg (0.5 mg/ml) **Time to Analysis:** Droplet applied on days 0, 1, 2. Sacrificed on day 4 or challenged with OVA droplet on days 25, 26, & 27. Sacrificed on day 28	**Acute airway damage and inflammation:** Only day 4 had LDH increased in the 200 μg CBP+OVA group. The 200 μg CBP+OVA group induced significantly higher numbers of BAL cells compared to OVA control. Total protein and TNF-a levels were increased only in 200 μg CBP+OVA group. RAS, parameter for phagocytosis, 200 μg and 20 μg CBP+OVA had higher levels than OVA controls. **Adjuvant activity on PBLN:** Total lymphocytes in PBLN significantly increased 4-5 fold in the 200 μg CBP+OVA exposed. 20 μg and 2 μg exposures did not increase the number of PBLN cells compared to OVA control. All CBP+OVA concentrations induced higher levels of IL-4, IL-5, IL-10, and IL-13, with 200 μg concentration having 10-200 times higher levels. IFN-y cytokine was increased in the 200 μg dose. **IgE Production:** In CBP+OVA, IgE were significantly increased. **PBLN and Lung Lymphocytes after OVA challenge:** PBLN cell numbers increased in OVA and CBP+OVA sensitized mice. CD4 and CD8 populations increased in both groups. PBLN levels in CBP+OVA and challenged with PBS were higher than mice treated with OVA and challenged with PBS, both groups cytokine production was low, only IL-5 levels were significant in the CBP+OVA/PBS group. Higher lung lymphocyte numbers were caused by higher numbers of CD4 and CD19. Production of IL-5 and IL-10 was four to five times higher than in OVA treated mice. **OVA challenge induces asthma like airway inflammation in CBP+OVA sensitized mice:** Total number of cells in BAL increased 10 fold in CBP+OVA mice challenged with OVA. Eosinophils exhibited highest increase in CBP+ OVA/OVA group. Perivascular and peribronchial infiltrates and globlet cell hyperplasia in CBP+OVA/OVA was confirmed by histological examination. Antigen specific inflammation induced in CBP+OVA mice.

Study	Pollutant	Exposure	Effects
Reference: de Haar (2006, 144746) **Species:** Mouse **Gender:** Female **Strain:** BALB/cANNCr **Age:** 6-8wks **Weight:** NR	CBP: fine (F) and ultrafine (UF) carbon black particles (Ken Donaldson Group) TiO_2: fine and ultrafine titanium oxide particles (Ken Donaldson Group) OVA: Ovalbumin (Sigma-Aldrich, Zwijndrecht, the Netherlands) **Particle Size:** F CBP: 260.0nm UF CBP: 14.0nm F TiO_2: 250.0nm UF TiO_2: 29.0nm	**Route:** Intranasal Droplet **Dose/Concentration:** CBP: 200 μg (3.3 mg/mL) TiO_2: 200 μg (3.3 mg/mL) OVA: 10 μg CBP+OVA: 200 +10 μg (see note in 3008) **Time to Analysis:** Days 0,1,2: Exposed to OVA or CBP+OVA. Sacrificed on day 8 & analyzed after 2h, or continued to second group. Second group: days 25, 26, 27 given OVA challenge day 28: sacrificed , analyzed 24h post sacrifice	**Ultrafine particles induce lung inflammation:** UF TiO_2 and CBP induced a local inflammatory response in the airways and showed higher levels of LDH and total protein as compared to mice exposed to the F particles. Cytokine levels were much higher in groups exposed to ultrafine particles. Histologic analysis of the airways showed that exposure to ultrafine TiO_2 or CBP leads to peribronchial and perivascular inflammatory infiltrates (mostly neutrophils). Exposure to OVA alone, or combined with fine TiO_2 and fine CBP had no effects on lung histology. **Ultrafine stimulate local immune responses:** TiO_2 and CBP particles stimulated the local immune response against co administered OVA antigen. Fine TiO_2 particles induced a low but significant increase in PBLN cell number. Both types of ultrafine particles elicited higher levels of Th-2 associated cytokines, with UF CBP stimulating a greater response. IFNγ production was low, but significantly higher than OVA exposures. **Ultrafine TiO_2 increase ovalbumin-specific IgE and IgG1 levels:** Levels of OVA specific IgE were significantly increased in animals exposed to the UF TiO_2+ OVA compared to F TiO_2 or OVA-only. . Average IgE level in mice exposed to ultrafine CBP+OVA was not a significant increase. OVA-specific IgG2a not detected in any groups. **Ultrafine particles stimulate allergic airway sensitization against ovalbumin:** At day 28, the PBLN cell numbers were significantly higher in both ultrafine and combination with OVA. Production of OVA specific IL-4, IL-5, IL-10 and IL-13 by PBLN cells was significantly increased in both ultrafine TiO_2 and CBP. IFNγ levels were significantly increased in ultrafine CBP+OVA treated animals. F TiO_2 had low, but significant, increases in IL-4 and IFNγ compared to OVA only. Allergic airway inflammation and Influxes of eosinophils, neutrophils and lymphocytes were only found in both groups exposed to ultrafine particles.
Reference: de Haar (2008, 187128) **Species:** Mouse **Gender:** Female **Strain:** BALB/c, CD80/CD86-deficient, DO11.10 **Age:** 6-8wks **Weight:** NR	Ultrafine Carbon Black (UFCB) (Brunschwich Chemicals; Amsterdam, The Netherlands) **Particle Size:** Diameter: 30-50nm	**Route:** Intranasal Exposure. IP Injection. Tail Injected. **Dose/Concentration:** 20 μg/mL **Time to Analysis:** Exposed days 1, 2, 3. Kept in supine position until recovery. OVA challenge days 25, 26, 27. Spleens and lymph nodes from DO11.10 mice pooled and $CD4^+$ T-cells isolated. Solution injected into tail veins of BALB/c mice day 0. CTLA4-Ig ip injected days 0, 2. PBLN cell suspensions plated, restimulated with OVA 4d.	UFCB+OVA induced proliferation of $CD4^+$ T-cells, increased cytokine production. UFCB+OVA did not induce any effects in CD80/CD86-deficient mice. UFCB-induced airway inflammation is dose-dependent.
Reference: de Haar et al. (2008, 187128) **Species:** Mouse **Cell Line:** Myeloid dendritic cells (mDCs)	Ultrafine Carbon Black (UFCB) (Brunschwich Chemicals; Amsterdam, The Netherlands) **Particle Size:** Diameter: 30-50nm	**Route:** Cell Culture **Dose/Concentration:** 25 μg/mL **Time to Analysis:** mDCs cultured from bone marrow. Exposed 18h. Cells isolated, stained for flow cytometry.	UFCB+OVA increased mDCs in the peribronchial lymph nodes, and their expressions of CD80, CD86, and MHC-11.

Study	Pollutant	Exposure	Effects
Reference: Dong et al. (2005, 088079) **Species:** Rat **Gender:** Male **Strain:** Brown-Norway (BN/CrlBR) **Age:** NR **Weight:** 200-225g	DEP: SRM 2975 (NIST, Gaithersburg, MD) OVA: Ovalbumin (Sigma ,St Louis, MO) **Particle Size:** 0.5 μm (MMAD)	**Route:** Inhalation **Dose/Concentration:** DEP: 20.6 ± 2.7 mg/m^3 OVA 40.5 ± 6.3mg/m^3 **Time to Analysis:** 4 h/d for 5 days + OVA 30 min/d 1 x wk on days 8,15 & 29. Sacrificed on days 9 or 30.	**Lung Inflammation/Injury:** Both the BAL proteins and inflammatory cell counts for DEP exposure alone were not different from those of the air exposed control, suggesting that DEP exposure did not cause lung injury at 9 or 30 days post-exposure. OVA exposure caused significant increases in neutrophils, lymphocytes, eosinophils, albumin and LDH activity in the lung after two exposures. DEP did show a strong effect on OVA-induced inflammatory responses. **Alveolar Macrophage (AM) function:** OVA exposure resulted in an increase in NO levels in the acellular BAL fluid and AM conditioned media. This increase was significantly attenuated in rats exposed to DEP. DEP exposure had no significant effect on the production of IL-10 or IL-12 by AM recovered from rats 9 and 30 days post exposure. In contrast, OVA sensitization elevated both IL-10 and IL-12 secretion by AM at both time points. **Lymphocyte population and cytokine production:** DEP exposure was found to increase the numbers of total lymphocytes, T cells and their CD4+ and CD8+ subsets in LDLN. OVA exposure also significantly increased these cell counts on days 9 and 30. DEP+OVA exposure showed a significant reduction in total lymphocytes, T cells, CD4+ and CD8+ subsets on day 30. Levels of IL-4 and IFN-γ in lymphocyte conditioned media were below detection limit of the ELISA kits. **Intracellular GSH levels in AM and Lymphocytes:** DEP exposure alone slightly decrease GSH levels in AM, but markedly reduced GSH concentration in lymphocytes on days 9 and 30. OVA exposure significantly decreased intracellular GSH in both cell types. Combined exposure showed AM and lymphocytes to have depleted intracellular GSH. **OVA specific IgE and IgG levels in serum:** In all samples collected on day 9, both serum IgG and IgE levels were under the detection limits. On day 30, no measureable IgE levels were found. The OVA exposure, however, resulted in elevated IgE levels, and was enhanced in rats preexposed to DEP. IgE and IgG levels for DEP+OVA was twO times higher than OVA alone indicating that DEP has an adjuvant effect on the production of IgG and IgE. **Effects of DEP and OVA on Lung iNOS expression**: AM from various exposure groups did not stain for iNOS. 1 rat at day 9 from the combined DEP+OVA group showed a slightly positive iNOS staining. On day 30, 2 of 5 rats from combined exposure group and 1 from the OVA group showed a positive airway staining.

Study	Pollutant	Exposure	Effects
Reference: Dong et al. (2005, 088083) **Species:** Rat **Gender:** Male **Strain:** Brown-Norway (BN/CrlBR) **Age:** NR **Weight:** 200-225g	DEP: SRM 2975 Diesel Exhaust Particles (NIST) OVA: Ovalbumin (Sigma Chemical Company, St Louis, MO) **Particle Size:** 0.5μm (MMAD)	**Route:** Nose-only Directed Flow Inhalation **Dose/Concentration:** DEP 22.7 ± 2.5 mg/m^3 OVA 42.3 ± 5.7mg/m^3 **Time to Analysis:** Day 1, 8, 15: OVA exposure 30min/day Days 24-28: DEP exposure 4h/day Day 29: OVA challenge Day 30: Whole-body plethysmography Day 31: Sacrifice	**Effect of DEP on OVA induced allergic responses:** DEP exposure had a synergistic effect with OVA on inducing airway hyper-responsiveness (AHR) in rats. DEP alone had no effect on IgG production. Levels of OVA-specific IgG and IgE increased in OVA+DEP exposure. This indicates that DEP pre-exposure augments the immune response of rats to OVA in the production of allergen specific IgG and IgE. **Effect of DEP on OVA induced cell differentiation:** Neither DEP, OVA nor the combination induced elevated levels of LDH activity or albumin content, indicating that the exposure protocols did not cause significant lung injury. DEP alone induced moderate but significant increase of neutrophil numbers. OVA exposure induced a greater infiltration of neutrophils than DEP, and infiltration of eosinophils and lymphocytes. OVA-induced eosinophil count markedly increased with DEP exposure. Total lymphocytes, T cells, and their CD4+ and CD8+ subsets in LDLN from rats sensitized and challenged by OVA were significantly higher than those of air-exposed non sensitized rats. DEP+OVA exposure resulted in substantial increase in T cells compared to OVA alone. **Effect of DEP on OVA-induced oxidant generation and GSH depletion:** Exposure to DEP or OVA alone had no effect on ROS production by AM. Substantial elevation seen in ROS for the DEP+OVA exposed group. Both OVA and DEP exposures resulted in an increased presence of NO in the acellular BAL fluid and in AM conditioned media; OVA+DEP exposure further increased these levels. The ATII cells from OVA exposed rats exhibited a higher percentage of cells that produce NO and superoxide than air exposed, non sensitized rats. DEP and OVA exposure resulted in a significant increase in the percentage of cells that produce NO and superoxide over the control. **iNOS expression:** Immunohistological analysis in lung tissues showed no AM staining in any group. Airway epithelium was found to be positive in all 5 rats from the DEP+OVA group and 3 of 5 rats from single exposure of DEP or OVA and 2 of 5 in air only exposed rats. iNOS expression was significantly higher in ATII cells isolated from rats exposed to combined DEP and OVA . **GSH levels in AM and lymphocytes:** Levels were slightly lowered by DEP or OVA exposure, though not statistically significant. DEP+OVA showed a significant reduction in GSH levels.

Study	Pollutant	Exposure	Effects
Reference: Drela et al. (2006, 096352) **Species:** Mouse **Gender:** Male **Strain:** BALB/c **Age:** 6 wks **Weight:** NR	ASM: Air suspended PM from Upper Silesia (Poland) 1μg of ASM: Pb (1.136 ng) Cu (0.004 μg) Co (0.072 ng) Mn (0.406 ng) Fe (0.016 μg) Cd (0.154 ng) Cr (0.418 ng) Ni (0.238 ng) **Particle Size:** 0.3-10 μm	**Route:** Intraperitoneal Injection **Dose/Concentration:** 170mg ASM/kg of body weight **Time to Analysis:** One time exposure, sacrificed 72h post exposure	**CD28 expression on thymocytes at different stages of development:** ASM exposure accelerated thymocyte maturation but did not alter the expression of CD28 on peripheral CD4 and CD8 T cells isolated from lymph nodes. A slight but not statistically significant decrease in the expression of CD28 on spleen T cells from ASM animals was observed. **Distribution of CD28(low) and CD28(high):** Acute exposure to ASM resulted in the increase of CD28(low) and decrease of CD28 (high) thymocyte percentages in the total thymocyte population. The percentages of CD28 low and high thymocytes did not differ between intact and PBS controls. Acute ASM exposure resulted in the increase of the percentage of CD28(low) and the decrease of CD28(high) thymocytes in the CD3 low subset. The percentage of CD28 low and high positive thymocytes did not differ in CD3 high thymocyte subset. **Natural regulatory CD4+ CD25+ T cells in the thymus:** The development of thymic natural regulatory cells was unaffected by ASM. **Proliferation of splenocytes and lymph node lymphocytes:** Decreased proliferative responses were evident in splenocytes from ASM-exposed animals when cells were stimulated with low but not high levels of anti-CD3 mAb.In contrast, lymph node lymphocytes from ASM treated mice had increased proliferative responses independent of anti-CD3 concentration. Both CD4+ and CD8+ T cells from ASM treated mice proliferated more vigorously than from controls. Almost all CD8+ T cells from ASM mice were induced to proliferate.
Reference: Dybing et al. (2004, 097545) **Species:** Mouse **Gender:** NR **Strain:** BALB/cA **Age:** NR **Weight:** NR **Assay:** PLN, ELISA	UP: Urban ambient particles collected in 5 different sites (Amsterdam, Lodz, Oslo, Rome, Dutch seaside) during four-week periods in spring, summer, winter seasons from March 2001 to March 2004. DEP as reference std: SRM 1650 (NIST) OVA: Ovalbumin (Sigma Chemical, St. Louis, MO) **Particle Size:** UP: PM_{10} and $PM_{2.5}$	**Route:** Injection in hind foot pad **Dose/Concentration:** UP: 100 μg- 200 μg DEP: 50 μg OVA: 50 μg **Time to Analysis:** PLN assay: Day 0: 1 exposure to OVA alone, OVA w/particles, particles alone. Day 6: Lymph nodes harvested Day 21: 1 OVA w/o particles exposure Day 26: Antibody assay	**Allergy screening:** All samples were immunostimulatory in the popliteal lymph node assay; activity was weak in the absence of OVA but statistically significant when injected with OVA, indicating an adjuvant effect. Particle adjuvancy was further demonstrated via significant enhancement of OVA-specific antibody responses. All ambient particle fractions from all seasons increased IgG1. Except for a few coarse samples, all fractions significantly increased IgE. All fine fractions and some coarse fractions significantly increased IgG2a, indicating that most particles could exert both Th1 and Th2 adjuvancy. In general, fine particles demonstrated stronger adjuvant activity than coarse in a pair-wise comparison of coarse and fine particles from the same location.
Reference: Dybing, et al. (2004, 097545) **Species** Rat **Cell Lines:** Primary rat type 2 cells, rat alveolar macrophages **Use:** Inflammatory screening	UP: Urban ambient particles collected in 5 different sites (Amsterdam, Lodz, Oslo, Rome, Dutch seaside) during four-week periods in spring, summer, winter seasons from March 2001 to March 2004. DEP: SRM 2975 (NIST) OVA: Ovalbumin (Sigma Chemical, St. Louis, MO) **Particle Size:** PM_{10} and $PM_{2.5}$	**Route:** Cell Culture **Dose/Concentration:** 0 – 50 μg/ml **Time to Analysis:** 20h	**Inflammation screening:** The coarse fractions were more potent than the fine fractions. Among the samples, the overall effects of the coarse fractions on the cells were dependent on the site of collection. High MIP-2 levels were found using particles from some spring collections. Coarse particles collected in summer demonstrated the highest potency, and samples collected during winter proved to be less potent but seasonal variation was not obvious for all sites. Only minor responses were observed using fine fractions from urban sites.

Study	Pollutant	Exposure	Effects
Reference: Farraj et al. (2006, 141730) **Species:** Mouse **Gender:** Male **Strain:** BALB/c **Age:** 6 weeks **Weight:** NR	DEP: SRM 2975 NIST OVA: Ovalbumin Anti-p75: Rabbit anti-mouse p75 neruotrophin receptor polyclonal antibody (Chemicon, Temecula, CA) Anti-trkA: anti-mouse trkA NGF receptor antibody (Santa Cruz, Santa Cruz, CA) **Particle Size:** DEP: 1.47 μm (MMAD), 2.75 GSD	**Route:** Nose-only Inhalation **Dose/Concentration:** DEP: 1.78 to 2.18 mg/m^3 Anti-p75: 50 μl Anti-trkA: 50 μl OVA injection: 20μg MCH: 0, 16, 32, 64 mg/ml **Time to Analysis:** On day 0: ip injection of 20 μg OVA Day 14: intranasal instilation of 50 ul anti-p75 or anti-trkA Day 14, 1-h after 1st exposure: challenged with OVA aerosol for 1h followed by a h exposure to DEP 24 h after DEP exposure: MCH challenge	**Airways responsiveness:** No significant differences in avg baseline Penh values of any treatment groups. Vehicle sensitized mice: exposure to DEP, anti-p75 or anti-trkA had no effect on MCH-induced Penh values. OVA-sensitized DEP-exposed: seen increase of Penh values. Administration of anti-p75 or anti-trkA to OVA sensitized mice reversed DEP induced Penh increases. **Lung function in ventilated mice:** Compared to vehicle sensitized mice, central airway resistance (Rn) increased 62% in OVA sensitized mice was not a significant increase. OVA-sensitized DEP-exposed mice, anti-p75 decreased central airway resistance (R_n) and anti-trkA did not significantly alter R_n. though R_n response for anti-p75 was significantly less than anti-trkA response, Constant phase model parameter of tissue elastance not significantly affected by any treatments or by increasing MCH dose, indicating development of significant regional ventilation inhomogeneity during bronchoconstriction. **Airway pathology:** OVA-sensitized mice had small increases in intraepithelial mucus compared to vehicle-sensitized mice. DEP exposure did not enhance severity of OVA-induced airway pathology. Anti-p75 or anti-trkA administration did not influence airway morphology. **BAL cells:** Vehicle-sensitized DEP-exposed mice had significantly enhanced macrophage numbers by 92% compared to air-exposed, vehicle-sensitized mice. Anti-p75 or Anti-trkA administration significantly suppressed DEP-induced macrophage increase to levels similar to air-exposed, vehicle-sensitized group. DEP co exposure significantly decreased number of macrophages in OVA-sensitized mice to control levels. Anti-trkA or anti-p75 had no effect in OVA-sensitized, DEP-exposed. Eosinophil number greater in OVA-sensitized DEP-exposed mice than in vehicle-sensitized air-exposed mice. No significant effects of DEP exposure on neutrophils from vehicle- or OVA-sensitized mice. **Cytokines:** IL4: OVA-sensitized DEP-exposed had five-fold increase over vehicle-sensitized, air-exposed mice and anti-trkA or anti-p75 significantly inhibited the DEP-induced increase. IL5, IL13: OVA-sensitized DEP-exposed had no significant change. Anti-p75 or anti-trkA administration had no significant effect. **Serum IgE**: OVA sensitized mice had a 10 fold increase in IgE levels for air and DEP exposed mice. Anti-p75, anti-trkA treatment did not cause significant effects on IgE levels.

Study	Pollutant	Exposure	Effects
Reference: Farraj et al. (2006, 189604) **Species:** Mouse **Gender:** Male **Strains:** C57/Bl6 **Age:** 6wks	DEP: SRM 2975 collected from diesel-powered industrial forklift filter (NIST) OVA: Ovalbumin Anti-p75: Rabbit anti-mouse p75 neurotrophin receptor polyclonal antibody **Particle Size:** 1.47 (MMAD), 2.75 (GSD)	**Route:** Nose-only Inhalation **Dose/Concentration:** DEP: 0.87 mg/m^3 MCH: 0, 16, 32, 64 mg/ml OVA: 20 μg ip Anti-p75: 50 μl **Time to Analysis:** Day 0: OVA in gel vehicle, ip Day 14: anti-p75 exposure, intranasal instillation 1h post anti-p75 exposure, OVA aerosol challenge for1h 1h post OVA challenge: DEP exposure for5h 48h post DEP exposure: MCH challenge	**Airway responsiveness:** No significant differences in average Penh values among any vehicle control groups. No significant differences in treatment groups in OVA-sensitized mice at baseline 0, 16, or 32 mg/mL of MCH. At 64 mg/mL MCH, OVA-sensitized, DEP-exposed mice had a 22% increase in Penh compared to vehicle mice, and a 68% increase compared to vehicle-sensitized, air-exposed mice. Instillation of anti-p75 inhibited the DEP induced increased Penh. **BAL cells:** DEP exposure in vehicle-sensitized mice significantly increased macrophages by 161% compared to air-exposed, vehicle-sensitized mice, while OVA-sensitized mice had 69% increase. Anti-p75 administration significantly suppressed DEP-induced macrophage increase in vehicle-sensitized mice. No significant effects of DEP exposure or anti-p75 treatment in OVA-allergic mice. OVA-sensitized air-exposed mice had a several hundred fold increase in the number of eosinophils. No significant effects of DEP exposure or anti-p75 treatment on eosinophils from OVA-sensitized mice. OVA-exposure or DEP-exposure had no significant effects on neutrophil or lymphocyte number . **BAL cytokines:** No significant effects of DEP alone or with OVA on IL-4, IL-5, or IL-13. **Serum IgE:** OVA sensitization in the presence or absence of DEP or anti-p75 caused at least a 3 fold increase in IgE levels. No significant effects of DEP or anti-p75 treatment on IgE levels.
Reference: Finkelman et al. (2004, 096572) **Species:** Mouse **Gender:** Female **Strains:** BALB/c and C57BL/6 **Age:** 2-4 m	DEP: 4JB1 type; Isuzu Automobile, Tokyo, Japan **Particle Size:** 2 μm MMAD	**Route:** Two groups: Group1: 1 ip injection of 2 mg of DEP. Group 2: daily ip injections of 2mg of DEP **Dose/Concentration:** 2 mg **Time to Analysis:** 2-96h	**Serum Cytokines:** Mice in group 1 demonstrated an increase in serum IL-6 production but no increase in IL-4 or IL-2 production. IFN-¤ levels were decreased in group 2. TNF production was not affected. **Spleen Cytokines:** When injected before LPS, DEP had little effect on the LPS-induced TNF-α and IL-6 response, but resulted in a minor suppression of INF-¤ and IL-10. DEP LPS-induced increase in INF-¤ mRNA responses in spleen cells. DEP caused a dose related suppression of LPS stimulated INF-¤. DEP had little or no effect on the percentage of NK or NKT cells in the spleenand inhibited LPS-induced IFN-¤ production by NK and NKT. DEP failed to inhibit the IFN-¤ response by anti-CD3 mAb-activated NKT cells. Oxidant activity was not responsible for DEP inhibition of LPS-induced IFN-¤ production.
Reference: Fujimaki and Kurokawa (2004, 189576) **Species:** Mouse **Gender:** Male **Strains:** BALB/c **Age:** 4 wks **Cell Types:** Cervical lymph-node (CLN) cells	DE±particles: Comparison of exposure to DE including particles andexposure to particle-filtered DE All mice were injected with sugi basic protein (SBP), a cedar pollen allergen before exposure to DE **Particle Size:** 0.4 μm MMAD	**Route:** Whole-body Exposure **Dose/Concentration:** Exposure to: 0, 1.0 mg/m^3 or 1.0mg/m^3 DE gas only (0.04 mg/m^3 PM) **Time to Analysis:** Exposure for 12h daily for 5wks. Days 14 and 35 challenge with SBP intranasaly. Evaluation is 24 and 48h after final SBP injection.	**CLN response:** Exposure to DE or DE gas did not affect B1 lymphocyte subpopulations of CLN. Culture supernatants of CLN cells from DE exposed/SBP immunized mice showed significant increase in MCP-1 at 24 and 48 h. Exposure to DE or DE gas significantly increased the amount of TARC and MIP-1α in CLN cells from SBP-immunized mice at 48 h. **Composition of DE and DE gas:** DE: 12.09 ± 0.15 NOx, 1.99±0.02 NO_2, 10.02±0.12 NO, 0.18±0.002SO2 and 1769.2±13.2 CO_2 (all in ppm). DE gas: 11.93 ± 0.13 NOx, 2.93±0.06 NO_2, 8.91±0.09 NO, 0.11±0.003SO2 and 1838.8±15.3 CO_2 (all in ppm)

Study	Pollutant	Exposure	Effects
Reference: Fujimaki et al. (2005, 156456) **Species:** Mouse **Gender:** Male **Strains:** C57BL/6 **Age:** 4 wks **Cell Types** CLN (lymph node) and Plasma cells	DE generated by 4 cyl 2.74 l Isuzu diesel DE gas = DE filtered to remove particles Composition of Disel Exhaust: DE DEP: 1.01 mg/m^3 1796ppm CO_2 12.09ppm NO_x 0.18 ppm SO_2 Composition of filtered DE Gas: DEP: 0.04 mg/m^3 1839ppm CO_2 11.93ppm NO_x 0.11 ppm SO_2 Sugi Basic Protein (SBP)- allergen **Particle Size** Average diameter of DEP 0.4 μm	**Route:** Whole-body Exposure Chamber **Dose/Concentration:** 1.0mg DEP/m^3 or 1.0mg DEP/m^3 DE gas **Time to Analysis:** Exposure for 12h daily for 5wks. All mice were injected IP with 100 μg SBP before exposure to gas or DE and again received 50 μg SBP intranasally on days 14 and 35. Evaluation is 1d after final SBP-immunization (mice are euthanized and CLN and blood samples are collected)	**CLN:** Exposure to DE and gas led to a decrease in total number of CLN cells and percentage of CD4+ and TCR-B levelsCell proliferation response to SBP was higher in gas-exposed mice than in the control group. The production of MCP-1 increased in CLN cells when stimulated with SBP (in vitro) but the difference was not significant at 24 and 48h. SBP-stimulated cells in gas-exposed mice showed greatly enhanced MIP-1α production at 24 and 48 h. Exposure to gas increased the amount of TARC in the culture supernatants of CLN cells. **PLASMA:** Exposure to DE or gas significantly decreased the anti-SBP IgG1 antibody titers and increased increased the anti-SBP IgG2a antibody titers in mouse plasma.
Reference: Fujimoto et al. (2005, 096556) **Species:** Mouse **Gender:** Female 1st day of pregnancy) **Strains:** Slc: IRC **Cell Types** Fetal Cells/ RNA Spleen Cells	DEP: generated by a 2369-cc diesel engine operated at 1050 rpm and 80% load with commercial light oil **Particle Size:** 0.4 μm MMAD	**Route:** Whole-body Inhalation Chambers **Dose/Concentration:** 0.3, 1.0 and 3.0 mg DEP/m^3(Groups 1,2,3) **Time to Analysis:** Exposure began at 2d postcoitum and was continued until 13d postcoitum. Exposure time was 12h daily for 7d/wk. Pregnant females were sacrificed 14d postcoitum.	**mRNA Expression in Placentas**: In groups exposed to DE, the expression of CYP1A1 mRNA decreased to undetectable levels during placental absorption and INF-¤ was increased. Levels of CYP1A1 mRNA in normal placentas from DE-exposed mice were unchanged. mRNA levels of inflammatory cytokines IL-2, IL-5, IL-12α, IL-12B and GM-CSF increased in placentas of mice exposed to DE.
Reference: Gao et al. (2004, 087950) **Species:** Human **Cell Line:** Lung fibroblasts infected with *Mycoplasma fermentans*	ROFA: collected near a power plant in FL burning low sulfur # 6 oil. (PM from Dusseldorf, volcanic ash for Mt. St. Helens, PM from Utah used to compare against ROFA in one experiment) **Particle Size:** NR	**Route:** Cell Culture **Dose/Concentration:** Cells were seeded into 6-well plates (3-4.5X10^5 cells/3mL/well) or 24-well plates (0.6-1 X10^5 cells/1.0 mL/well) 3, 10, 20, 40, 50 μg/ml **Time to Analysis:** 24, 48h	**Cytokines:** ROFA exposure in combination with *Mycoplasma fermentans* infection synergistically amplifies the induction of IL-6 production in human lung fibroblasts (HLF). PM from the other souces has little synergistic effect on IL-6 release. Exposing HLF cells to,*M. fermentans* derived macrophage activating lipopeptide-2 (MALP-2) and ROFA has the same synergistic effect as *M. fermentans* infection and ROFA. MALP-2 and ROFA extract have a similar synergistic effect that requires more time to appear. ROFA contains high levels of V, Ni, Fe and Cu. Exposure of HLF to $NiSO_4$ alone and $NiSO_4$ with MALP-2 produced 10 and 50 fold increases, respectively, in IL-6 production. Exposure of HLF to $CuSO_4$, $VOSO_4$ and Na_3VO_4, with and without the presence of MALP-2, did not produce as dramatic results as seen with Ni. The action of $NiSO_4$ and MALP-2 on IL-6 production was found to be dose dependent.

Study	Pollutant	Exposure	Effects
Reference: Gavett et al. (2003, 053153) **Species:** Mouse **Gender:** Female **Strain:** BALB/c **Age:** 7wks	$PM_{2.5}$ from the German cities of Hettstedt or Zerbst PM Composition: samples from Hettstedt have several-fold higher levels of zinc, magnesium, lead, copper and cadmium than samples from Zerbst. **Particle Size:** $PM_{2.5}$	**Route:** Oropharyngeal Aspirations **Dose/Concentration:** 50-100μg PM in 50 μL saline **Time to Analysis:** Mice were exposed to one dose of 100 μg BAL 18h after exposure. Sensitization Model: Mice were exposed to 50 μg PM 2h before being sensitized with 10 μg OVA, repeated two days later. On day 14- all mice were challenged with20 μg OVA. Parameters measured on days 2 and 7 after FINAL exposure to OVA. Challenge Model: Mice were sensitized IP with 20 μg OVA or adjuvant only. 14d later mice were exposed to 100 μg $PM_{2.5}$ followed 2h later by 20 μg OVA. Parameters measured on days 2 and 7 after FINAL exposure to OVA.	**BAL Analysis**: Hettstedt PM significantly increased BAL protein and NAG levels. Zerbst PM did not. Mice exposed to Zerbst had lower levels of LDH than control groups. Hettstedt exposed mice had increased levels of IL-1B, IL-6 and MIP-2 in comparison to control and to mice exposed to Zerbst PM. $PM_{2.5}$ at a dose of 100 μg was not found to be toxic, therefore used for subsequent studies. **Airway Responsiveness (PenH)**:: In allergic mice tested immediately after exposure, Hettstedt PM increased PenH 190% compared to baseline, Zerbst increased PenH by 120% and the Control increased by 44%.:.: Two days after OVA challenge, no differences in non-allergic mice from either group. In allergic mice, Hettstedt PM still caused a significant response to Mch responsiveness, Zerbst none. No effects on day seven. **IgE Levels**: Serum collected on day 2 showed antigen-specific IgE was increased by Hettstedt $PM_{2.5}$ in both the sensitization and challenge phases when compared to the control and exposure to Zerbst. Day 7 serum indicated no effect. **BAL Cell Counts**: In non-allergic mice both Hettstedt and Zerbst PM increased BAL neutrophil numbers (3-fold; not statistically significant) and in allergic mice, only Hettstedt PM significantly increased neutrophil count. Eosinophil numbers were increased only in allergic mice exposed to Hettstedt PM. Lymphocyte numbers were not different among groups. **BAL Lung Injury**:: At 2 days after both Hettstedt and Zerbst PM administered in allergic mice caused significant increases in protein, LDH and NAG compared to the non-allergic groups. Both PMs caused an increase in LDH in allergic mice compared to the allergic control, but only Hettstedt caused an increase NAG in allergic mice compared to control. At 7 days no effect. **BAL Cytokines**: Allergic mice had increased levels of IL-4, IL-5 and IL-13 compared to non-allergic mice (at 2 days after). IL-5 was significantly increased by exposure to either PM in allergic mice compared to non-allergic mice. Exposure to either PM caused an increase in TNF-α and IFN-◘ (by 6-8 fold) in allergic mice, there was also an increase in these inflammatory cytokines in the non-allergic group but was not statistically significant. No significant effects were observed in animals that underwent the sensitization protocol alone for any measurement or endpoint.
Reference: Gowdy et al. (2008, 097226) **Species:** Mouse **Gender:** Female **Strain:** BALB/c **Age:** ~12-14wks **Weight:** 17-20g	DEP (30kW (40hp) 4-cylinder Deutz BF4M1008 diesel engine, steady state, 20% full load) (Low dose: 21% O_2, 0.4wt ratio OC/EC; High dose: 20.7% O_2, 0.4wt ratio OC/EC) (CO, NO_x, SO_2) **Particle Size:** Diameter: ~240nm	**Route:** Inhalation Exposure Chamber **Dose/Concentration:** Low- 514 ± 3 $\mu g/m^3$, High-2026 ± 38 $\mu g/m^3$ **Time to Analysis:** Exposed 4h/d, 1 or 5d (consecutive). Necropsied immediately or 18h postexposure.	**Inflammation:** Neutrophils and lung injury dose-dependently increased. ICAM-1 increased immediately after both exposures and after 18h postexposure in the low dose. **Cytokines:** After 1d exposure, IFN-◘ and TNF-α increased immediately at both doses and the high dose, respectively. Immediately after 5d exposure TNF-α and IFN-◘ increased at both concentrations and IL-6 increased at the low dose. At 18h postexposure IL-6 and IFN-◘ increased at both doses, TNF-α and IL-13 increased at the low dose, and MIP-2 dose-dependently increased. **CCSP, Surfactants:** CCSP decreased. SP-A and SP-D decreases were only significant after 5d exposure, 18h postexposure.

Study	Pollutant	Exposure	Effects
Reference: Hamada et al. (2007, 091235) **Species:** Mouse **Gender:** Female (Pregnant close to partruition) **Strain:** BALB/c **Use:** Pregnant mice were exposed and offspring were analyzed	ROFA ROFA was obtained from a precipitator until of a local power plant. Composition of ROFA (in μg/mL): 341.2 Ni, 323.4 V, 232.2 Zn, 18.3 Co, 15.8 Mn, 8.4 Ca, 6.7 Cu, 6.1 Sr, 5.0 mg, 0.9 Sb, and 0.6 Cd. **Particle Size:** NR	**Route:** Nebulized ROFA lechate **Dose/Concentration:** 50mg/mL dilution **Time to Analysis:** Pregnant mice exposed to nebulized ROFA leachate for 30 min/day at days 14, 16 and 18 of pregnancy. Newborns received a single injection (ip) of OVA(5 μg) + alum (1mg0 at day followed by exposure to: 1. aerosolized OVA days 12, 13 and 14 (2-week old protocol) OR 2. aerosolized OVA days 32, 33 and 34 (5wk old protocol) Analysis 48h after final allergen exposure	**Susceptibility to Asthma**:.Exposure of motherto PBS aerosols during pregnancy did not result in prominent asthma features in young. The offspring of the ROFA mothers revealed increasing AHRand elevated numbers of eosinophils in the BAL fluid. Similar results were seen in both the 2-week and 5-week old groups. **IgE Levels**: Histopathology revealed prominent inflammation in the lungs of the ROFA neonates and increased allergen-specific IgE and IgG1 levels in the 5-week group. **Maternal Influence**: Breast milk was not shown to be responsible for the increased susceptibility to allergy seen in offspring. **IL-4 and IFN-**: IL-4 and IFN- levels in maternal mice showed no difference between PBS exposed or ROFA exposed mice. Cultured spleen cells from mice born of ROFA-exposed mothers showed either increased or similar levels of IL-4 and decreased production of IFN- causing an increase in the ratio of IL-4/IFN- indicating greater susceptibility to develop Th2- allergic response. **Eosinophils:** Exposure of mothers to Ni levels similar to those found in ROFA had no appreciable effect on BAL eosinophil.
Reference: Hao et al. (2003, 096565) **Species:** Mouse **Gender:** Female **Strain:** BALB/c **Age:** 6-7 weeks	DEP DEP collected from a 4-cylinder diesel engine under a 10-torque load. **Particle Size:** NR	**Route:** Nebulization **Dose/Concentration:** 2 mg/m^3 DEP **Time to Analysis:** Mild Sensitization- Mice receive IP OVA alum and are challenge with aerosolized OVA with and without DEPs. Mice sacrificed d19. Postchallenge Model- DEPs are delivered to mice sensitized by IP OVA and alum. Mice sacrificed d23. Transgenic Mice: Mice exposed to nebulized saline or DEPs for 1-h daily for 3 days. Mice sacrificed d5.	**Mild Sensitization:** Exposure of previously OVA sensitized mice to aerosolized DEP and OVA did not affect OVA-specific IgE production, BAL eosinophilia or methacholine-induced AHR. Aerosolized particles induced inflammation and increased MBP deposition and MBP positive eosinophils in the mucosa. **IL-5 Transgenic:** Exposure to aerosolized DEP did not change BAL cytokine levels, but did increase AHR and BAL cell count. **Classic Sensitization, Post-Challenge:** Did not lead to a discernable increase in OVA-induced AHR. DEP treatment was associated with increased airway inflammation and mucin production in larger and intermediary airways.
Reference: Harkema et al. (2004, 056842) **Species:** Rat **Gender:** Male **Strain:** F344, BN **Age:** 10-12wks **Weight:** NR	CAPs (Detroit; July-Sept. 2000; Harvard Ambient Fine Particle Concentrator) **Particle Size:** Diameter: 2.5 μm	**Route:** Inhalation Exposure Chamber. IT Instillation. **Dose/Concentration:** 4d concentration: 676 ± 288 μg/m^3, 5d concentration: 313 ± 119 μg/m^3, July concentration: 16-185 μg/m^3, September concentration: 81-755 μg/m^3; IT Instillation- 200 μL (soluble and insoluble) **Time to Analysis:** F344 rats sensitized to endotoxin, BN rats to OVA. Exposed 10h/d 1, 4, 5d (consecutive). Another group of rats IT instilled. Both groups killed 24h postexposure.	The retention of PM in the airways was enhanced by allergic sensitization. Recovery of anthropogenic trace elements was greatest for CAPs-exposed rats. Temporal increases in these elements were associated with eosinophil influx, BALF protein content and increased airway mucosubstances. A mild pulmonary neutrophilic inflammation was observed in rats instilled with the insoluble fraction but instillation of total, soluble or insoluble $PM_{2.5}$ in allergic rats did not result in differential effects.

Study	Pollutant	Exposure	Effects
Reference: Harrod et al. (2003, 097046) **Species:** Mouse **Gender:** NR **Strains:** C57BL/6 **Age:** 8-10wks **Use:** Infected with RSV	DEE: Diesel Engine Emissions generated from a 5.9-liter turbo diesel engine fueled by Number 2 fuel. DEE Composition: NOx: 2.0-43.3ppm CO: 0.94-29.0ppm SO_2: 8.3-364.9ppb **Particle Size:** for high & low level: 0.1-0.2 μm (MMAD)	**Route:** DEE: Whole-body Inhalation RSV: IT administration **Dose/Concentration:** DEE: 38.8 $\mu g/m^3$ (low level) or 10027 $\mu g/m^3$ (high level) RSV: 100 μl (10^6pfu) **Time to Analysis:** 6 h/d for 7d. After the final 6h exposure period mice were infected with RSV. Parameters measured 4d post infection	**Viral Gene Expression:** For air+RSV, RSV-F gene expression was not apparent but RSV-G gene expression was detectable at very low levels. In DEE+RSV (for high and low levels), RSV-F and –G were markedly elevated. ß-Actin mRNA levels not changed in DEE-exposed compared to air-treated. DEE+RSV for high and low levels show 10- to 20- fold induction of RSV-G mRNA levels as compared to air+RSV. **Cell numbers in BALF:** Uninfected low-level DEE did not induce statistically significant increase in cell numbers as compared to air+RSV. High level DEE+RSV caused increase as compared to air+RSV. Uninfected high-level DEE had increase as compared to uninfected air group. For all groups, alveolar macrophages were predominant cell type and no substantial changes in infiltrating cell populations by exposure to DEE were noted. **Lung inflammation & Airway Epithelial Morphology**: Lung sections from air- or DEE-exposed, uninfected did not exhibit any observable change. Low level DEE + RSV had increased inflammatory cell infiltration in peribronchial regions and loss of normal cuboidal appearance of Clara cells as compared to air+RSV. High level DEE+RSV had more apparent lung-inflammation, especially surrounding bronchi and bronchioles, and increased appearance of pseudostratified, columnar epithelial cell morphology and apparent airway epithelial cell sloughing as compared to low level DEE+RSV, indicating dose-related increase in lung histopathology to RSV infection by prior DEE exposure. **Inflammatory Mediators:** TNF-α and IFN-◘ were significantly increased in RSV-infected mice exposed to low or high level DEE and not increased in RSV-infected mice exposed to air. TNF-α levels elevated to similar levels for low and high level DEE+RSV. IFN-◘ exhibited more dose-related increase with higher levels in high level DEE+RSV versus low level DEE+RSV. **Mucous Cell Metaplasia:** DEE exposure in uninfected was not altered. Mucous metaplasia was increased in epithelium of RSV-infected mice when exposed to DEE in a dose-dependent manner. Following high level DEE+RSV, muscous staining of airway epithelial cells in more distal airways was occasionally observed. **CCSP Production in Airway Epithelium:** DEE alone did not have an effect CCSP-producing cells, or Clara cells, decreased in Low DEE + RSV iand further decreased in high level DEE+RSV in large and terminal airways. **Surfactant Protein B:** proSP-B staining post RSV alone shows now discernible decrease when compared to uninfected. Staining levels in alveolar lung regions decreased when exposed to low level DEE+RSV, and further decreased in high level DEE+ RSV. Staining in airway epithelium following high level DEE+RSV diminished when compared to RSV alone or low level DEE+RSV. **SP-A:** In alveolar type II cells and airway epithelial cells for untreated and air +RSV, no discernible changes in levels. Prior exposure to low or high level DEE decreased SP-A staining in alveolar type II cells and airways epithelial cells during RSV infection.

Study	Pollutant	Exposure	Effects
Reference: Harrod et al. (2005, 088144) **Species:** Mouse **Gender:** Male **Strain:** C57B1/6 **Age:** 10-12wks **Weight:** NR	DEE (2 2000 model 5.9-1 Cummins ISB turbo diesel engines, No. 2 certification diesel fuel) **Particle Size:** NR	**Route:** Exposure Chamber **Dose/Concentration:** Low- 30 μg/m^3 PM, Mid-Low- 100 μg/m^3 PM, Mid-High- 300 μg/m^3 PM, High- 1000 μg/m^3 PM **Time to Analysis:** Exposed 6h/d, 7d/wk, 1wk or 6m. 1wk exposure repeated on separate occasion. Immediately after exposure, mice anethetized, IT instilled with *Pseudomonas aeruginosa*.	**Bacterial Clearance:** Lung bacterial clearance was decreased at all levels after 1wk exposure and was concentration-dependent 18h postinfection. Bacterial clearance was not affected at 6m and bacterial counts were higher. **Inflammation, Particle Deposition:** Lung inflammation and histopathology were increased in all exposure groups postinfection. All exposure groups possessed particle-laden macrophages. Higher doses had a concentration-dependent increase. **Ciliated, Clara Cells, TTF-1:** Generally, ciliated cells decreased with exposure dose, were more discernible in inflamed airways, and higher doses caused effects in small distal airways. Clara cells decreased equally at all exposures and were most notable in the distal airway epithelium. TTF-1 decreased postinfection.
Reference: Heidenfelder et al. (2009, 190026) **Species:** Rat **Gender:** Male **Strain:** Brown-Norway **Age:** 10-12wks **Weight:** NR	CAPs (Grand Rapids, MI; July) **Particle Size:** Diameter: 0.1-2.5 μm	**Route:** Whole-body Inhalation Chamber **Dose/Concentration:** CAPs: 493 ± 391 μg/m^3; OC: 244 ± 144 μg/m^3, EC: 10 ± 4 μg/m^3, Sulfate: 79 ± 131 μg/m^3, Nitrate: 39 ± 67 μg/m^3, Ammonium: 39 ± 59 μg/m^3, Urban dust (Fe, Al, Ca, Si): 18 ± 6 μg/m^3 **Time to Analysis:** Sensitized to OVA 3d. Challenged with OVA or saline 2wks later for 3d. Exposed to CAPs 8h/d, 13d. OVA or saline challenge 9d after first challenge. Sacrificed 24h after last CAPs exposure.	CAPs enhanced the effects of OVA by causing differential expression in genes primarily involved in inflammation and airway remodeling. CAPs exposure alone had no effect on gene expression. CAPs + OVA also increased IgE, mucin glycoprotein, and BALF total protein, and caused a more severe bronchopneumonia, increased mucus cell metaplasia/hyperplasia and mucosubstances.
Reference: Hiramatsu et al. (2003, 155846) **Species:** Mouse **Gender:** Female **Strains:** BALB/c and C57BL/6 **Age:** 8wks **Weight:** 17-22g	DE -DE (generated by diesel engine and diluted with filtered clean air) **Particle Size:** NR	**Route:** Exposure Chamber **Dose/Concentration:** Low dose –0.1mg/m^3 Highdose - 3mg/m^3 **Time to Analysis:** 1 or 3 months (7 h/d , 5 d/wk)	**Lung Histopathology:** DEP-laden macrophages accumulated in the alveoli and peribronchial tissues in a dose- and duration-dependent manner in both strains. Lymphocytes and neutrophils increased in both strains, but were greatest in the BALB/c mice. **BALF and Mac-1 Positive Cells:** BALT formation in DEP-laden AMs was seen at the high dose group and was greater in the BALB/c mice. Mac-1 positive cells, a marker for phagocytic activation of the AMs, was observed in the high dose groups of both strains at 1 and 3 months, and in the low dose group at 1 month in BALB/c mice. **Cytokine and iNOS mRNA expression:** 1 month of exposure increased TNF-α, IL-12p40, IL-4 and IL-10 mRNA in a dose-dependent manner. IL-1B and iNOS decreased in a dose-dependent manner. IFN-¤ mRNA expression increased in BALB/c mice and decreased in C57BL/6 mice. Similar results were seen at 3 months, except IL-4 and IFN-¤ mRNA expression decreased in the BALB/c mice. In C57BL/6 mice, IL-4 and IL-10 mRNA increased at the low dose but decreased at the high dose. NF-¤B activation occurred after 1 week and 1 month DE exposure and was more prevalent in BALB/c mice.

Study	Pollutant	Exposure	Effects
Reference: Hiramatsu, (2005, 088285) **Species:** Mouse **Gender:** Female **Strains:** BALB/c **Age:** 8wks **Weight:** 17-22g	DE (generated by diesel engine and diluted with filtered clean air.) Mycobacterial Infection - *M.tuberculosis* (ATCC35812) Kurono strain **Particle Size:** NR	**Route:** Exposure Chamber **Dose/Concentration:** Low DE dose – 0.1mg/m^3 High DE dose - 3mg/m^3 Mycobaterial infection: 5mL (nebulized) of a 10^6 colony-forming units (CFU) suspension **Time to Analysis:** 1, 2 or 6m (7h/d, 5d/wk). 6 mice from each group infected on last day of DE exposure. CFU evaluation 7wks postinfection.	**Histopathological observations:** DEP-laden AMs and DEPs in the alveoli and peribronchial tissues increased in a time-dependent manner. DE-exposed mice had a greater number of mycobacterial lesions, which were disseminated. Lesions in the control mice had clear borders and consisted of epithelial cells and lymphocytes. Tubercle bacilli and DEPs coexisted in AMs. BALT was seen around DEPs in the 2 and 6-month exposure groups. Inflammation cells increased in a time-dependent manner with respect to DE exposure. **Granulomatous lesions in lungs:** 6-month DE-exposed mice had a significantly higher amount of gross lesions than the 6-month control mice. **Mycobacterial burden:** CFU in lungs were increased in DE-exposed animals but only the 6 month exposure resulted in statistically significant increases (a ~4-fold increase over control). CFU in spleen were not significantly altered by DE exposure. **Cytokines and iNOS mRNA expression:** Infected DE-exposed mice had time-dependent increases of TNF-α, IL-1B, IL-12p40, IFN-□ and iNOS mRNAs compared to the infected control mice. IL-12 mRNA expression decreased in infected 6-month DE-exposed mice.
Reference: Ichinose, T. et al. (2003, 041525) **Species:** Mouse **Gender:** NR **Strains:** BALB/cAnN, ICR, and C3H/HeN **Age:** 6wks **Weight:** NR	DE: DE generated by 3059cc 4-cylinder diesel engine Der f: Crude extract of *D. farinae* **Particle Size:** DEP - Mass median aerodynamic diameter: 0.4 μm	**Route:** Exposure Chamber; IT Instillation **Dose/Concentration:** 1. Air 2. DE only: 3.0mg/m^3 3. Air + Der f: 1mg Der f 4. DE 3.0mg/m^3 + 1mg Der f **Time to Analysis:** DE: 8wks (12h/d, 7d/wk) Der f: 2wk intervals, 6wks Analyzed 3d after last instillation	**Light microscopic observations:** DE exposure caused the proliferation of nonciliated cells and epithelial cell hypertrophy. Soot-containing macrophages were found in the alveolar tissue spaces. Accumulated lymphocytes were present in the peribronchiolar lymphoid tissue. Inflammatory cells and soot-containing macrophages were found in the submucosal layer and the vessel instertitium of mice treated with DE+Der f in all strains. DE+Der f treated C3H/He mice had desquamated goblet cells. **Eosinophil infiltration:** DE treated C3H/He mice had a slight eosinophile infiltration in the submucosal layer. DE+Der f treated mice in all strains had a slight to moderate eosinophile infiltration. **Lymphocyte accumulation:** Lymphocytes significantly increased in all strains under the DE treatment as compared to the air+saline treatment, and further increased under the DE+Der f treatment. **Goblet cell proliferation:** Little proliferation was seen in all strains under the DE treatment. DE+Der f caused a significant increase in proliferation compared to air+Der f in ICR mice, but a significant decrease in C3H/He mice. **Local cytokine and chemokine expression in lung tissue supernatant:** DE+saline significantly increased MIP-1α in all strains. MCP-1 also increased but not significantly. DE+Der f increased IL-5, RANTES, eotaxin, MCP-1 and MIP-1α in all strains as compared with air+saline and air+Der f. IL-5 decreased in C3H/He mice treated with DE+Der f compared to air+Der f. IL-3 decreased in ICR and C3H/He mice compared to air+saline. **Der f-specific immunoglobulin production in plasma:** Increased production of IgG1 was statistically significant in ICR and C3H/He mice treated with DE+Der f as compared to air+Der f. IgE was low in all strains.

Study	Pollutant	Exposure	Effects
Reference: Ichinose et al. (2004, 180367) **Species:** Mouse **Gender:** NR **Strains:** BALB/c, ICR and C3H/He **Age:** 5wks **Weight:** NR	DEP: 2740cc 4-cylinder engine *D. farinae*: crude extract **Particle Size:** DEP - Mass median aerodynmaic diamter of 0.4 μm	**Route:** IT Instillation **Dose/Concentration:** 1. *D. farinae*: 1 μm in PBS 2. *D. farinae* + DEP: 1 μg in PBS + 50 μg mg DEP **Time to Analysis:** 4 times at 2wk intervals. Mice examined 3wks after last instillation	**Histological changes:** Mice in all three strains treated with DEP+*D. farinae* had a significant recruitment of eosinophils, more proliferation of goblet cells, and more eotaxin positive macrophages in the alveoli than mice treated with *D. farinae* alone. **Local cytokine expression in lung tissue supernatant:** DEP+*D. farinae* induced significant elevation of IL-5 in ICR and C3H/He mice as compared to *D. farinae* alone. Production levels of IL-4 and RANTES did not correlate with the manifestations of allergic airway inflammation induced by the *D. farinae* treatment with or without DEP. **Cytokine expression in plasma:** IL-5 in C3H/He mice treated with DEP+*D. farinae* was significantly higher than *D. farinae* alone. RANTES was unaffected by the DEP treatment in all strains. ***D. farinae*-specific immunoglobulin production in plasma:** The adjuvant effect of DEP on IgG1 production was observed in all three strains, with C3H/H3 being statistically significant. The production levels of IgG1 correlated with the manifestations of eosinophilic airway inflammation by both treatments. No adjuvant effect on IgE production was observed.
Reference: Inoue et al. (2006, 097815) **Species:** Mouse **Gender:** Male **Strain:** ICR **Age:** 6wks **Weight:** 29-33g	PM-OC: Urban PM, collected for 1 month during early summer, 2001 in Urawa city Saitama, Japan LPS **Particle Size:** $<$ 2.0mm, 40mg/m^3	**Route:** IT Instillation **Dose/Concentration:** Vehicle group: PBS PM-OC group: 4mg/kg of PM-OC LPS group: 2.5mg/kg of LPS PM-OC+LPS group: combined adminstration of PM-OC +LPS **Time to Analysis:** 24h after IT administration	**Effects of PM-OC on LPS related lung inflammation**: PM-OC alone did not significantly increase the infiltration of neutrophils, but LPS challenge showed a marked increase in the number of neutrophils compared with vehicle. Administration of LPS combined with PM-OC significantly increased the infiltration of neutrophils compared with LPS administration alone. **Effects of PM-OC on histological changes in the lung:** Combined treatment with PM-OC and LPS resulted in enhanced neutrophilic inflammation. **Effects of PM-OC on pulmonary edema related to LPS**: LPS group compared with vehicle group had a significant increase in lung water. The combined administration of PM-OC and LPS resulted in further increase in the lung water compared with LPS administration alone, however it was not statistically significant. **Effects of PM-OC on protein expression IL-1B, MIP-1α, MCP-1 and KC:** The concentrations of these molecules were below the detection limits in the PM-OC group. LPS treatment significantly increased the protein levels of these molecules compared with the vehicle treatment. In the PM-OC + LPS group all concentrations, particularly KC, were smaller than in the LPS group.

Study	Pollutant	Exposure	Effects
Reference: Inoue et al.(2006, 096720) **Species:** Mouse **Gender:** Male **Strain:** ICR **Age:** 6wks **Weight:** 29-33g	Carbon black (14 nm PrinteX 90; 56nm PrinteX 25; Degussa, Dusseldorf, Germany) **Particle Size:** 14nm - 300 m^2/g 56nm - 45m^2/g	**Route:** IT Instillation **Dose/Concentration:** Vehicle group: PBS at pH7.4 LPS group: 2.5mg/kg of LPS in vehicle Nanoparticle groups: 4 mg/kg carbon black nanoparticles (14nm or 56 nm) in vehicle LPS + nanoparticle group: combined adminstration of carbon black and LPS in vehicle **Time to Analysis:** 24h after IT administration	**Effects of nanoparticles**: Nanoparticles alone increased number of total cells and neutrophils, but not statistically significant. LPS exposure significantly increased numbers for both groups. Nanoparticles and/or LPS enhance pulmonary edema. **Histological changes**: Treatment with LPS+14 nm nanoparticles marketly enhanced neutrophil sequestration into the lung parenchyma compared to LPS alone. LPS+56 nm nanoparticles did not. **Proinflammatory cytokine proteins**: IL-1B level significantly greater for both LPS+ nanoparticles groups. TNF-α was not significantly altered among the experimental groups. **Chemokine proteins**: Challenge with 14nm nanoparticles alone elevated the levels of all chemokines without significance except for KC. LPS alone and with both nanoparticle groups caused significant increases in all chemokines. . **Formations of 8-OHdG in lung:** LPS plus nanparticles resulted in intensive expression 8-OHdG, strongest in LPS+14nm nanoparticle **Plasma coagulatory changes**: PT - no change for any group. APTT - some change with LPS and LPS + nanoparticle groups, fibrinogen level significantly elevated after LPS and for LPS+14nm nanoparticle. APC decrease with LPS (significant) and LPS + nanoparticle groups. vWF increase with LPS (significant) and LPS+14 nm (significant).
Reference: Inoue et al. (2004, 087984) **Species:** Mouse **Gender:** Male **Strain:** ICR **Age:** 6wks **Weight:** 29-33g	DEPs [4JB-1 type light-duty, four-cylinder, 2.74 litre Isuzu diesel engine (Isuzu Automobile Co., Tokyo Japan)] Washed DEP and DEP-OC - extracted with dichloromethane **Particle Size:** NR	**Route:** IT instillation **Dose/Concentration:** Vehicle group: PBS Washed DEP group: 4mg/kg of DEP DEP-OC group: 4mg/kg of DEP-OC LPS group: 2.5mg/kg of LPS Washed DEP+LPS group: combined adminstration of washed DEP +LPS DEP-OC+ LPS group: combined admininstration of DEP-OC + LPS **Time to Analysis:** 4h	**COX-1 mRNA:** Slightly elevated in both washed DEP and DEP-OC groups, but slightly decreased in other groups compared to vehicle group. **COX-2 mRNA:** Slightly increased with DEP-OC, increased with LPS, washed DEP + LPS and DEP-OC + LPS groups compared to vehicle. COX-2 in the DEP-OC + LPS decreased when compared to the LPS only group. **Pulmonary Oedema:** Washed DEP + LPS group showed a synergistic enhancement of pulmonary oedema and local expression of proinflammatory chemokines (MCP-1, MIP-1α, KC, IL-1B).
Reference: Inoue et al. (2006, 190142) **Species:** Mouse **Gender:** Male **Strain:** ICR **Age:** 6-7wks **Weight:** 29-33g	Carbon black (14 nm PrinteX 90; 56nm PrinteX 25; Degussa, Dusseldorf, Germany) **Particle Size:** 14 nm - 300 m^2/g 56nm - 45 m^2/g	**Route:** IT instillation **Dose/Concentration:** Vehicle group: PBS Ovalbumin (OVA) group: 1mg OVA Nanoparticle groups: 50mg carbon black nanoparticles (14nm or 56nm) OVA + nanoparticle group: combined adminstration of nanoparticles and OVA **Time to Analysis:** Vehicle group - weekly for 6wks OVA group - biweekly for 6wks Nanoparticle groups - weekly for 6wks OVA+Nanoparticle group (same protocol as OVA and Nanoparticle) studied 24h after last administration	**Nanoparticles:** Exposure to carbon nanoparticles resulted in the lung expression of TARC, GM-CSF and MIP-1α. The levels wre higher in the 14nm group compared to the 56nm group. **OVA:** In the presence of OVA, nanoparticles enhanced levels of TARC, GM-CSF, MIP-1α, IL-2 and IL-10, with the effects seen more prominently in the 14 nm particles + OVA group.

Study	Pollutant	Exposure	Effects
Reference: Inoue et al. (2005, 188444) **Species:** Mouse **Gender:** Male **Strain:** ICR **Age:** 6-7wks **Weight:** 29-33g	Carbon black (14 nm PrinteX 90; 56nm PrinteX 25; Degussa, Dusseldorf, Germany) **Particle Size:** 14 nm - 300 m^2/g 56nm - 45 m^2/g	**Route:** IT Instillation **Dose/Concentration:** Vehicle group: PBS Ovalbumin (OVA) group: 1mg OVA Nanoparticle groups: 50mg carbon black nanoparticles (14nm or 56 nm) OVA + nanoparticle group: combined adminstration of nanoparticles and OVA **Time to Analysis:** Vehicle group - weekly for 6 wks OVA group - biweekly for 6 wks Nanoparticle groups - weekly for 6 wks OVA+Nanoparticle group: same protocol as OVA and Nanoparticle studied 24h after last adminstration	**Nanoparticles + OVA:** Nanoparticles given with OVA enhanced airway inflammation, characterized by increased eosinophils, neutrophils, mononuclear cells and goblet cells. In addition, nanoparticles + OVA significantly increased local expression of IL-4, IL-5, eotaxin, IL-13, RANTES, MCP-1 and IL-6. The formation of 8-OHdG was enhanced by nanoparticles + OVA. **14nm nanoparticles:** All these effects were more prominent when 14nm nanoparticles were used. The 14nm nanoparticle + OVA group significantly raised levels of total IgE and antigen specific production of IgG1 and IgE.
Reference: Inoue et al. (2006, 190142) **Species:** Mouse **Gender:** Male **Strain:** ICR **Age:** 6wks **Weight:** 29-33g	Whole DE (generated by 4-cylinder, 3.059l, Isuzu diesel engine, Isuzu automobile, Tokyo, Japan) LPS **Particle Size:** Peak particle size 110nm	**Route:** Whole-body Exposure Chamber **Dose/Concentration:** 0.3mgsoot/m^3 1.0mgsoot/m^3 3.0mg soot/m^3 LPS: 125 mg/kg **Time to Analysis:** LPS prior to 12h exposure to exhaust	BAL fluid, total cells, neutrophils, protein and gene levels (MCP-1 and KC) ubcreased compared to control with LPS, but were smaller with LPS + DE. Results are suggestive that short-term exposure to DE does not exacerbate LPS related lung inflammation.
Reference: Inoue et al. (2007, 096702) **Species:** Mouse **Gender:** Male **Strain:** ICR **Age:** 6wks **Weight:** 29-33 \g **Cell Type** Splenocytes	DEPs [4JB-1 type light-duty, four-cylinder, 2.74 litre Isuzu diesel engine (Isuzu Automobile Co., Tokyo Japan)] LPS **Particle Size:** $PM_{2.5}$	**Route:** Cell Culture **Dose/Concentration:** Splenocytes resuspended to cell density of 1X106 mL-1 and 1000 mL applied into each of 12-well plate DEP: 100 mg mL-1 LPS: 1 mg mL-1 LPS(1mg mL-1) + DEP (1, 10 or 100 mg mL-1) **Time to Analysis:** 72h	**Cell viability:** No effect. **Mononuclear cell response:** Incubation with DEP alone inhibited basal cytokine production. LPS significantly increased protein levels of IFN-g, IL-2, and IL-10 compared to control. DEP suppressed the LPS-enhanced protein levels in a dose-dependent manner and moderately elevated the IL-13 level.
Reference: Inoue et al. (2007, 096702) **Species:** Mouse **Gender:** Male **Strain:** ICR **Age:** 6-7wks **Weight:** 20-30g	Carbon nanoparticles (Dusseldorf, Germany) OVA (Sigma Chemical, St. Louis, MO) **Particle Size:** CB14 (PrinteX 90) = 14nm CB56 (PrinteX 25) = 56nm	**Route:** IT Instillation **Dose/Concentration:** 50 μg and/or 1 μg OVA in PBS **Time to Analysis:** 1X/wk for 6wks; sacrifice 24h after last exposure	**Lung Responsiveness:** Respiratory system resistance, Newtonian resistance and tissue dampening were significantly higher in the nanoparticle + OVA groups. Elastance and tissue elastance were higher in these groups but not significantly so. Compliance was significantly lower in the nanoparticle + OVA groups compared to the control. **Lung mRNA level for Muc5ac**: levels were significantly higher in nanoparticle + OVA groups compared to the control.
Reference: Inoue et. al. (2007, 096702) **Species:** Mouse **Gender:** Male **Strain:** ICR **Age:** 6-7wks **Weight:** 29-34g	DEP-OC collected from 4JB1 type, light duty, 4 cylinder, 2.74 liter Isuzu diesel engine, Isuzu Automobile Company, Tokoyo, Japan) OVA **Particle Size:** DEP = 0.4 μm	**Route:** IT Instillation **Dose/Concentration:** 50 μg and/or 1 μg OVA in PBS **Time to Analysis:** DEP or DEP-OC w/ or w/o OVA initially; OVA or vehicle every 2wk for 6wks; DEP components or vehicle 1X/wk for 6wks; sacrifice 24h after last instillation	Total respiratory system resistance, elastance, Newtonian resistance, tissue damping, tissue elastance displayed general positive trends and were significantly higher in OVA and OVA + DEP-OC groups. Compliance displayed a general negative trend and was significantly lower in the washed DEP + OVA group.
Reference: Ito et al. (2006, 088391) **Species:** Rat **Cell Line:** L2 cells of alveolar epithelial cell type II origin	DEP - generated from 2982-cc common rail direct injection diesel engine with oxidation catalyst and exahust gas recirculation system. **Particle Size:** $PM_{2.5}$	**Route:** Cell Culture **Dose/Concentration:** 1X106 1,10 or 30mg/mL **Time to Analysis:** 3h	**ICAM-1 and LDL receptor mRNA**: Up-regulation in a dose-dependent manner. Statistically significant at 30 mg/mL compared to control. **HO-1 andPAF receptor mRNA**: Up-regulation in dose-dependent manner and statistically significant at all doses compared to control. **Correlation between HO-1 and ICAM-1, LDL, and PAF:** Significant correlation between HO-1 and each of these.

Study	Pollutant	Exposure	Effects
Reference: Jang et al. (2005, 155313) **Species:** Mouse **Gender:** Female **Strains:** BALB/c **Age:** 5-6wks	DEP -generated from 4JB1 type, light duty, four-cylinder diesel engine (Isuzu Automobile, Co,Tokyo Japan) Ozone - (generated with Sander Model 50 ozonizers,Sander, Eltze Germany) OVA **Particle Size:** NR	**Route:** Whole-body Exposure Chamber **Dose/Concentration:** DEP: 2,000 μg/μL (sic) Ozone: 2 ppm (ave 1.98 ± 0.08 ppm) OVA sensitization: 10 mg OVA challenge: **Time to Analysis:** OVA sensitization DEP, Ozone and OVA Challenge on d21- 23 Exposed to ozone for 3h and DEP for 1h AH and BAL measured 1d after last challenge	**Airway responsiveness**: OVA + ozone + DEP exposure group had significantly higher methacholine-induce Ptnh than sham group or OVA group. **Total cells, proportion of eosinophils and neutrophils**: the OVA + ozone + DEP group was significantly higher than than OVA group and OVA+ Ozone group **IL-4:** OVA + ozone, OVA + DEP and OVA + ozone + DEP IL-4 level increased compare to OVA group. **IFN-¤**: levels significantly decreased in OVA + DEP and OVA + ozone + DEP compared to OVA + ozone
Reference: Jaspers et al. (2005, 088115) **Species:** Human **Cell Lines:** A549 cells, primary human bronchial and nasal epithelial ells	DE_{as}: aqueous-trapped solution of DE (emissions from Caterpiller diesel engine, model 3304) Influenza: A/Bangkok/1/79 (H3N2 serotype) **Particle Size:** NR	**Route:** Cell Culture. DE_{as} and virus added to apical side of cells **Dose/Concentration:** Influenza: 3 x 10^5 cells infected with 320 hemagglutination units (HAU) DE_{as}: For A549 cells: 6.25, 12.5, 25 μg/cm^2. For bronchial and nasal cells: 22 or 44 μg/cm^2. **Time to Analysis:** 2h incubation with DE_{as} then virus added. HA RNA levels analyzed at 0, 15, 30, 60 or 120min post infection. IFN and MxA responses: analyzed 24h post infection. Fluorescence: some cells treated with GSH-ET 30 min before DE_{as} exposure. Measured 2 h post-influenza infection.	**A549 cells increased susceptibility:** DE_{as} enhances HA RNA levels in A549 cells in a dose-dependent manner. 25 mg/cm^2 significantly enhanced levels in A549 cells compared to the influenza-infected controls. Viral protein levels were increased in A549 cells. Exposure to DE_{as} increased the number of influenza-infected epithelial cells in A549 cells. **Human nasal and bronchial cells susceptibility:** Exposure to DE_{as} increased HA RNA levels in the nasal and bronchial cells. Statistically significant at 22mg/cm^2 for nasal cells and approaching significance at 44 mg/cm^2 for bronchial cells. Exposure of both types to 44 mg/cm^2 enhanced viral protein levels. **Influenza induced IFN response in A549:** Exposure to DE_{as} does not suppress but enhances IFNß mRNA levels. Treatment enhanced influenza-induced nuclear levels of both phospho-STAT-1 and ISFG3g. ISRE-promoter activity was enhanced, but not significantly. Treatment enhanced myxovirus resistance protein (MxA) mRNA levels. This data suggest that DEas exposure enhances influenza virus replication without suppressing production of IFNß or IFNß-inducible genes. **Influenza induced IFN response in human nasal and bronchial cells:** Exposure to DE_{as} increased IFNß and MxA levels. **Oxidative stress in A549:** DE_{as} exposure dose-dependently increases oxidative stress in A549 cells within 2-h post-exposure. Add the antioxidant GSH-ET and it reverses the effect. Pretreatment with GSH-ET A549 cells reversed the effects of DE_{as} on the number of influenza-infected cells, and reduced HA RNA levels. **Oxidative stress in Human bronchial cells:** The results were the same as A549 cells pretreated with GSH-ET. Or Pretreatment with GSH-ET also reversed effects of DE_{as} on HA RNA levels.

Study	Pollutant	Exposure	Effects
Reference: Kaan and Hegele (2003, 095753) **Species:** Guinea pig **Gender:** Female **Strain:** Cam Hartley **Age:** 22-29d **Weight:** 250-300g **Cell Types** Alveolar macrophages (AM) obtained by bronchoalveolar lavage	PM_{10} - EHC-93 obtained (Environmental Health Canada, Ottawa, ON, Canada) RSV - Human RSV (long strain/lot18D) (American Tissue Culture Collection, Bethesda, MD) **Particle Size:** PM_{10} (0.35μm MMAD)	**Route:** Cell Culture **Dose/Concentration:** PM_{10}: 500 μl/well (100 μg/ml MEM) RSV exposure:: 1 ml/well ($6x10^6$pfu/ml MEM) Groups: PM_{10}+RSV RSV+PM_{10} RSV only PM_{10} only negative control **Time to Analysis:** PM_{10} - 60min RSV - 90min Parameters measured 24h post treatment	**Interaction on phagocytic ability of AM:** Not affected by sequential exposure to RSV and PM_{10}. More than 95% of AM exposed to PM_{10} engulfed PM. AM exposed to PM_{10} showed significant increase in mean side scatter in comparison to negative control and RSV-infected AM. No significant difference between AM exposed only to PM_{10} and AM exposed to both agents. No significant side mean side scatter difference between AM exposed to PM only and to both agents. **Interaction on RSV Immunopositivity:** PM_{10} exposure inhibits. All RSV-treated groups showed significantly greater proportion of RSV-immunopositive cells compared with negative control. PM_{10}+RSV showed significantly smaller proportion of RSV-immunopositive cells compared with RSV group. RSV+PM_{10} group similar to RSV group. Proportion of RSV-immunopositive AM was influenced by the sequence of exposure to RSV and PM_{10}. **Interaction on RSV Replication:** PM exposure suppressed RSV replication. AM exposed to both agents produced 3 to 9 fold less RSV progeny compared with RSV alone group. Quantity of RSV progency was not significantly affected by the sequence of exposure RSV and PM_{10}. Negative control and PM_{10} only did not propagate progeny. **Interaction of RSV Yield:** RSV alone group produced the highest RSV yield, those exposed to both agents, independent of sequence, showed a 5-fold decrease. **Cytokine production:** RSV infection stimulated all three cytokines measure (IL-6, IL-8 and TNF-a) compared to negative control. IL-6: PM_{10} significantly reduced RSV-induced IL-6 production. IL-6 was affected by the sequence of exposure to PM_{10} and RSV (PM_{10}+RSV vs. RSV+ PM_{10}, $p < 3x10\text{-}6$). IL-8: PM_{10} significantly decreases RSV-induced IL-8 production and baseline. No affect on sequence of exposure. TNF-a: production was increased when exposed to RSV, PM_{10} or a combination of both agents. No differences among treatments.
Reference: Kleinman et al. (2005, 087880) **Species:** Mouse **Gender:** Male **Strains:** BALB/c **Age:** 8-19wks **Weight:** NR	CAPS: Concentrated fine (F) and ultrafine (UF) Ambient Particles using VACES system (performed a 2 sites in Los Angeles, CA, on 50-m downwind and another 150-m downwind from a complex of three roadways, State Road CA60, Interstate 10, and Interstate 5.) F CAPS in 2001 and 2002, UF CAPS in 2002 only OVA: Ovalbumin (Sigma, St. Louis, MO) **Particle Size:** UF: d_p ◘ 150 nm F: d_p ◘ 2.5 μm	**Route:** CAPS: inhalation via Whole-body chamber exposure OVA sensitization: nasal instillation OVA challenge: inhalation **Dose/Concentration:** UF at 50 m: 433 mg/m^3 –UF at 150 m: 283 mg/m^3 F at 50m or 150 m: average 400 μg /m^3 OVA sensitization: 50 μg/5 μl OVA challenge: 30 mg/m^3 **Time to Analysis:** CAPS: 4 h/d, 5 d/wk for 2wks Sensitization: On morning of each exposure 1st Challenge: week after 10d of treatment 2nd Challenge: one week following 1st challenge Sacrificed: 24h after 2nd challenge	There were significantly higher concentrations of IL-5, IgE, IgG1 and eosinophils in mice exposed to either CAPS compared to air. Mice exposed to CAPS at 50-m downwind showed higher levels of IL-5, IgG1, and eosinophils than those exposed to CAPS 150-m downwind.

Study	Pollutant	Exposure	Effects
Reference: Kleinman et al. (2007, 097082) **Species:** Mouse **Gender:** NR **Strains:** BALB/c **Age:** 6-8wks	CAPS - concentrated fine (F) and ultrafine (UF) using VACES system - performed a 2 sites in Los Angeles, CA, on 50-m downwind and another 150-m downwind from State Road CA60 and Interstate 5. Fall 2001-summer 2004 OVA **Particle Size:** F: $PM_{2.5}$; UF: $PM_{0.15}$	**Route:** Whole-body Chamber **Dose/Concentration:** 50m - F: 394 ± 94 mg/m^3 50m - UF: 297 ± 189 mg/m^3 150m - F: 387 ± 68 mg/m^3 150m - UF: 213 ± 95 mg/m^3 OVA - 50 mg in 5 mL saline **Time to Analysis:** 3 2wk exposures (4 h/d, 5d/wk for 2 wks. OVA the morning of each exposure	**50m Site**: higher levels and statistically significant concentration curves of IL-5 and IgG1 in F-CAP mice at the 50m site. **150m Site:** in no cases were responses greater than the 50m or control groups. **F vs. UF:** The study was not able to differentiate between the effects of F PM and UF PM exposures.
Reference: Klein-Patel et al. (2006, 097092) **Species:** Cattle and Human **Cell Types** Bovine tracheal epithelial cells (BTE) and Human A549 cells	ROFA (U.S. EPA NHEERL, Research Triangle Park, NC) V_2O_5, VOSO4, SiO_2 TiO_2, $Fe_2(SO_4)_3$ NiSO4LPS Recombinant human TNF-α and IL-1B **Particle Size:** MMAD- 1.95mm	**Route:** Cell Culture **Dose/Concentration:** 2x10^6 ROFA: 0, 2.5, 5, 10, 15, 20 μg/cm^2 LPS: 100ng/mL V2O5: 0, 0.15, 0.3, 0.61, 1.25, 2.5, 5, 10, 20 μg/cm^2 NiSO4, $Fe2(SO_4)_3$, TiO_2, SiO_2: 0, 1.23, 2.5, 5, 10, 20 μg/cm^2 VOSO4: 0, 0.145, 0.29, 0.58, 1.16, 2.32 μg/cm^2 **Time to Analysis:** LPS: 0, 6, or 18h ROFA: 0, 2,4,6h V2O5: 0, 0.25, 0.5, 1, 2, 4, 6, 8h NiSO4, Fe2(SO4)3, TiO_2, SiO_2: 6h VOSO4: 6h	**ROFA in BTE:** ROFA and ROFA leachate inhibition of LPS-induced TAP gene expression increases with exposure time and dose. Washed particles of ROFA at doses 2.5 to 10 mg/cm^2 significantly increased inducible TAP expression. **Soluble Metals in BTE:** V_2O_5 inhibition of LPS and IL-1B induced TAP gene expression increases with exposure time and dose. $NiSO_4$ exhibits non-significant dose dependent suppression of inducible TAP gene expression. $Fe2(SO_4)_3$, TiO_2 and SiO_2 were found to have no effect. **A549:** Results with ROFA and V_2O_5 in BTE were replicated using the A549 cell line and IL_1B to induce hBD2 gene expression. **Cellular Viability:** Was not significantly affected in ROFA doses below 20 μg/cm^2 and V_2O_5/$VOSO_4$ doses below 2.5 μg/cm^2.
Reference: Koike and Kobayashi (2005, 088303) **Species:** Rat **Gender:** Male **Strains:** Wistar **Age:** 8-10 wks **Weight:** 280-350g **Cell Types** AM - aveolar macrophages PBM - peripheral blood monocytes T-cells: antigen sensitized	Whole DEP: Diesel Exhaust Particles collected in the dilution tunnel of a diesel inhalation facility. (Ratio of organic extract to residual particles in the whole DEP was 3: 1.) Organic extract of DEP Residual particles of DEP OVA: Ovalbumin (Sigma, St. Louis, MO) **Particle Size:** NR	**Route:** Cell Culture **Dose/Concentration:** Whole DEP: 10, 30, 100 μg/mL Organic extract of DEP: 7.5, 22.5, 75 μg/mL Residual particles: 2.5, 7.5, 25 μg/mL **Time to Analysis:** 24h post exposure	**Ia antigen and costimulatory molecules:** Most control AM did not express these molecules. Whole DEP did not cause any increase in expression level. 20% of control PBM expressed Ia and 10% B7; expression of these molecules was significantly increased by whole DEP. Organic extract significantly increased the expression of Ia and B7 molecules on PBM similar to whole DEP. Residuals caused no effect. **PBM exposure to 100/75/25 μg/mL of DEP/organic extract/residual particles:** Results showed that the DEP-increased expression of Ia and B7 in PBM by DEP was caused by the organic extract fraction. Organic extract-induced expression of Ia antigen in PBM was reduced by treatment with NAC. **AP activity of PBM:** After exposure to organic extract, T cell proliferation was significantly increased by the addition of control PBM in a cell number-dependent manner. AP activity of PBM was increased over control by exposure to 3 mg/mL organic extract, although higher concentrations suppressed the activity of PBM. **Ctyokine production:** Organic extract treatment of PBM decrease IFN□ production from T-cells stimulated by PBM. No significant effect on IL-4 observed. **HO-1 protein level:** levels in PBM was significantly increased by exposure to whole DEP or organic extract. Levels induced by organic extract was diminished by NAC treatment.

Study	Pollutant	Exposure	Effects
Reference: Last et al. (2004, 097334) **Species:** Mouse **Gender:** NR **Strains:** BALB/c **Age:** 6wks **Weight:** 16-20 g	PM - aerosol of soot and iron oxide OVA (ovalbumin, grade V, 98% pure,) **Particle Size:** $PM_{0.1}$ - $PM_{2.5}$	**Route:** PM -Exposure Chamber OVA - Intraperitoneal Injections; Aerosol Exposure **Dose/Concentration:** PM – 235 – 256 $\mu g/m^3$ OVA - 10 μg/0.1mL injection OVA aerosol – 10mL of 10mg/mL (1%) solution **Time to Analysis:** PM: 4h/d, 3 d/wk OVA: 2 ip injections days 1 and 15. Aerosol on day 28 after first ip; 60 min 3x/wk	**2wk PM exposure/4wk OVA aerosol treatment:** The OVA alone group had significantly more airway collagen than the PM alone group. Histology showed significantly more collagen in the treatment than the air alone group. There was a significantly greater amount of goblet cells than the OVA alone group. **4wk OVA aerosol/2wk PM treatment:** The treatment had significantly more goblet cells than the PM alone group. **6wk concurrent PM and OVA treatment:** Significantly more cells were observed in the OVA alone group over the treatment. The treatment had significantly more lymphocytes and significantly less macrophates than groups exposed to PM before or after OVA. Histology showed significantly more collagen in the treatment than the air or PM alone groups. The treatment had significantly more goblet cells than the OVA alone group.
Reference: Li et al. (2007, 093156) **Species:** Mouse **Gender:** Female **Strain:** BALB/c, C57BL/6 **Age:** 9wks **Weight:** NR	DEP (2369-cc diesel engine manufactured by Isuzu Motor, operated at 1050 rpm, 80% load, commercial light oil) **Particle Size:** NR	**Route:** Exposure Chamber **Dose/Concentration:** DEP: 103.1 ± 9.2 $\mu g/m^3$, CO: 3.5 ± 0.1ppm, NO_2: 2.2 ± 0.3ppm, SO_2: < 0.01ppm **Time to Analysis:** Protocol 1: Exposed 7h/d, 5d/wk. Sacrificed at day 0, week 1, 4, 8. Protocol 2: DE alone or DE + NAC 7h/d, 1-5d.	**Airway hyperresponsiveness:** Penh values increased in BALB/c mice compared to the control at day 0, but no significant changes occurred after this time. Penh values increased in C57BL/6 mice at 1wk compared to the control but returned to control levels at 8wks. **BALF:** Compared to the other strain, the total number of cells and macrophages increased significantly at 1wk in C57BL/6 mice and at 8wks in BALB/c mice. Neutrophils, lymphocytes, MCP-1, IL-12, IL-10, IL-4, IL-13 increased significantly for both strains. No eosinophils were found. IL-1B and IFN-□ increased significantly in BALB/c mice compared to C57BL/6 mice. **HO-1 mRNA and protein:** HO-1 mRNA was more marked in BALB/c mice at 1wk and C57BL/6 mice at 4 and 8wks. HO-1 protein percentage changes from the control were greater in BALB/c mice at 1wk and C57BL/c mice at 8wks. **NAC:** NAC inhibited the increased Penh values, total number of cells and macrophages in C57BL/6 mice at 1wk and neutrophils and lymphocytes in both strains.
Reference: Li et al. (2009, 190424) **Species:** Mouse **Gender:** Female **Strain:** BALB/c **Age:** 6-8wks **Weight:** NR	CAPs (downtown Los Angeles, CA from major freeway, traffic mainly passenger cars and diesel trucks; Jan. 2007 or Sept. 2006) Ultrafine carbon black (UFCB; used as control) **Particle Size:** Diameter: Fine- < 2.5 μm, UF- < 0.15 μm	**Route:** Intranasal Instillation **Dose/Concentration:** 0.5 μg PM in 50 μL suspension **Time to Analysis:** Day 1 exposed to PM or saline. Day 2 exposed to PM + OVA or OVA or saline alone. Repeated on days 4, 7, 9. Different experiment: NAC ip injected 4h pre-instillation on days 1, 2, 4, 7, 9. All animals rested and OVA aerosol challenged 30min on days 21, 22. Sacrificed day 23.	UFP alone had no effect on the lung. UFP + OVA significantly increased eosinophils, and OVA-specific IgG1 and IgE. The induction of eosinophils and IgG1 were inhibited by NAC. Generally, UFP + OVA mice had greater signs of inflammation than the other groups as determined by pulmonary histopathology and airway morphometry. UFP had a greater PAH content than fine particles. UFP significantly increased IL-5, IL-13, TNF-α, IL-6, KC, MCP-1, and MIP-1α.
Reference: Li et al. (2009, 190424) **Species:** Mouse **Cell Line:** Macrophage RAW 264.7	CAPs (downtown Los Angeles, CA from major freeway, traffic mainly passenger cars and diesel trucks; Jan. 2007 or Sept. 2006) Ultrafine carbon black (UFCB; used as control) **Particle Size:** Diameter: Fine- < 2.5 μm, UF- < 0.15 μm	**Route:** Cell Culture **Dose/Concentration:** 1, 5, 8.3, 10 μg/mL **Time to Analysis:** Particle suspensions reconstituted with Dulbecco's Modified Eagle's Medium powder. Further replenished with 10% fetal calf serum and 1:200 dilution of penicillin/streptomycin/amphotericin B. Incubated 16h. 100 μg of lysate protein for HO-1 immunoblotting.	UFP induced greater HO-1 expression than fine particles. The higher PAH content of UFP correlated with HO-1 expression.

Study	Pollutant	Exposure	Effects
Reference: Liu et al. (2008, 156709) **Species:** Mouse **Gender:** Female **Strain:** BALB/c **Age:** 11wks **Weight:** NR	DEP (5500-watt single-cylinder diesel engine generator (Yanmar, Model YDG 5500E), 406 cc displacement air-cooled engine, Number 2 Diesel Certification Fuel, 40 weight motor oil) **Particle Size:** MMAD: ~0.1 μm	**Route:** Intranasal Exposure **Dose/Concentration:** Average particle concentration: 1.28mg/m^3 **Time to Analysis:** Four groups: saline + air control, saline + DEP, *A. fumigatus* + air, *A.fumigatus* + DEP. A. fumigatus exposure every 4d for 6 doses. DEP exposure 5h/d for 3wks concurrent with *A. fumigatus* exposure.	*A.fumigatus* + DEP increased IgE, the mean BAL eosinophil percentage, goblet cell hyperplasia, and eosinophilic and mononuclear cell inflammatory infiltrate around the airways and blood vessels compared to the *A. fumigatus* or DEP treatments. *A.fumigatus* + DEP also caused methylation at the IFN-◘ promoter sites CpG^{-53}, CpG^{-45}, and CpG^{-205}.
Reference: Liu et al. (2007, 093093) **Species:** Mouse **Gender:** Female **Strain:** BALB/c **Age:** 11wks	DEP: 5500-watt single-cylinder diesel engine. **Particle Size:** NR	**Route:** Inhalation Exposure **Dose/Concentration:** Average particle concentration 1.28 mg/m^3. **Time to Analysis:** 1. Aerosol vehicle (saline) + air 2. Aerosol vehicle (saline) + DEP 3. *A. fumigatus* + air 4. *A. fumigatus* + DEP *A. fumigatus*: 62.5 μg aerosolized protein extract in 50 μL PBS; 6 total doses, every 4d. DEP exposure 5h/d 3wks concurrent with *A. fumigatus* .	**IgE Production:** IgE production increased withthe *A.fumigatus* treatmen and increased further with the *A.fumigatus* and DEP treatment. **Histopathology:** *A. fumigatus* with DEP caused an increase in goblet cell hyperplasia and eosinophil and mononuclear cell infiltrate around the airways and blood vessels as compared to the control and DEP treatments. **Gene Methylation:** Greater methylation at the CpG^{-53} site of the IFN-◘ promoter occurred under the *A. fumigatus* + DEP treatment compared to the *A. fumigatus* or DEP treatments. The DEP treatment did not induce methylation. Methylation correlated with increased IgE and hypomethylation with decreased IgE. Hypomethylation occurred in the IL-4 promoter under the *A. fumigatus* + DEP treatment.
Reference: Lundborg et al. (2007, 096040) **Species:** Rat **Gender:** Male **Strains:** Sprague-Dawley **Age:** NR **Weight:** 300-400g	Carbon-Black Particles (93% C) Toluene- DEPs (97% C) 10-fold Cr, Mn, N; 50-100 fold Al, Cd, Cu, Fe, Mg, Pb, Zn more in DEP aggregates **Particle Size:** Carbon aggregates--mean diameter = 0.17 ± 0.08um Diesel Particles- mean diameter: 0.69 ± 0.46μm mean size primary particles: 0.044 ± 0.01 μm	**Route:** Cell Culture{Citation} **Dose/Concentration:** 2 X 10^6 AM A 2mL suspension was prepared with 20 μg/mL of carbon or diesel particles added. surface area: 159 ± 4m^2/g **Time to Analysis:** 6 different experiments. AM pre-exposed to carbon or washed DEP. Loaded with particles. Incubated with *S. pneumoniae*, ATCC strain or clinical isolates.	**Effect of time on survival of *S. pneumoniae* when incubated with carbon loaded AM:** Loading AM with carbon significantly increased the bacterial survival. Bactera opsonization decreased bacterial survival. **Effect of carbon load in AM on survival of *S. pneumoniae*:** Bacterial survival increased in a dose-dependent manner as the carbon particle load of AM increased. **Survival of *S. pneumoniae* after incubation with carbon or washed diesel loaded AM:** Bacterial survival increased in carbon loaded AM compared to the control. No difference existed with the washed diesel particles. **Survival of the ATCC strain and clinical isolates of *S. pneumoniae* when incubated with carbon loaded AM or control AM:** Carbon significantly increased the CFU of opsonized and unopsonized bacteria for the ATCC strain and clinical isolates. **Ability of carbon or washed diesel loaded AM, incubated with the ATCC strain of *S. pneumoniae*, to induce LPO of lung surfactant:** A 97% increase in the surfactant LPO occurred after incubation with washed diesel loaded AM compared to contol AM. The effect of washed diesel particles was significantly greater than that of carbon particles. **LPO by carbon loaded AM incubated with the ATCC strain or clinical isolates in the presence of absence of surfactant:** LPO induced by AM increased when incubated with carbon loaded AM compared to control AM.

Study	Pollutant	Exposure	Effects
Reference: Matsumoto et al. (2006, 189213) **Species:** Mouse **Gender:** Female **Strains:** BALB/c **Age:** 6wks **Weight:** 15-20g	DE DE collected from a 2369 cm^3 diesel engine operated at 1050 rpm and 80% load with commercial light oil. Engine exhaust passed through a particulate air filter and charcoal filer. Diluted DE introduced into the exposure chamber. Composition of the DE: 3.5±0.1ppm CO, 2.2±0.3ppm $NO_{2,}$ < 0.01ppm SO_2 and 103.1 ±9.2μg/m^3 DEP. **Particle Size:** NR	**Route:** Whole-body exposure chambers after prior sensitization with OVA through ip injection **Dose/Concentration:** 100 μg/m^3 DE **Time to Analysis:** Mice were initially sensitized w/ OVA (20ug absorbed to 2 mg alum diluted with 0.5 mL saline) via ip injection on day 0, 6 and 7. Two weeks later the mice were challenged with OVA (0.1mg in 0.1mL saline) intranasally on day 21. DE for 1d or 1,2, 3, 4 or 8wks (at 7h/d for 5d/wk).	**Airway Hyper-Responsiveness:** Airway Reactivity (evaluated by an increase in PenH)- Exposure to DE significantly increased airway reactivity to methacholine after 1 week in both 24 and 48 mg/mL Mch and after 4 weeks in the 48 mg/mL. Airway Sensitivity (evaluated by PC200)- DE exposure caused an increase in airway sensitivity after 1 week of exposure, 4 weeks and 8 weeks of exposure did not result in a significant increase. **BAL Cell Counts:** The total cell count was increased after 1 week of DE exposure. This increase was mostly due to an increase in eosinophils. After 1 week the total cell count dropped drastically even after continuous exposure to DE. DE did not effect the number of CD3, CD4, CD8 or NK1 cells at any point in time. **Cytokine/Chemokine mRNA Levels:** DE exposure on day 1 caused an increase in mRNA levels of IL-4, IL-5 and IL-13 when compared to the control mice but longer periods of DE exposure failed to cause an increase. Protein levels of IL-4 were significantly elevated at compared to control at day 1, but did not persist with time. mRNA levels of MDC were increased at 1 week of exposure (compared to control) but also decreased at time periods after. mRNA levels of RANTES were increased at 2 and 3 weeks after exposure and remained elevated at 4 weeks but not significantly. The level of RANTES protein increased as the weeks went along, but increased significantly only at 8 weeks. Lung **Histopathology:** OVA sensitization caused an increase peribronchial and perivascular infiltration of inflammatory cells which peaked at 1 week after exposure and decreased afterward. DE exposure did not cause/show any additional signs of inflammation.
Reference: Morishita et al. (2004, 087979) **Species:** Rat **Gender:** Male **Strain:** Brown-Norway **Age:** 10-12wks	Concentrated Air Particles (CAPs) CAPs were generated from ambient air in an urban Detriot community. The sampling site was chosen because it has a high percentage of pediatric asthma and is located near a lot of industry. **Particle Size:** 0.1-2.5 μm	**Route:** Whole-body Inhalation Chambers **Dose/Concentration:** Air chamber received CAPs at a flow rate of 50 L/min and at a pressure of 0.94-0.95 atm. July 676 μg/m^3 September 313 μg/m^3 **Time to Analysis:** First rats were sensitized (days 1-3) and challenged (days 14-16) with saline (control) or ovalbumin by intranasal instillation (5% in saline, 150 μL/nasal passage). 4 days after the last intranasal challenge, rats began exposure in the chambers. Exposures were 10 h long. There were two separate exposure periods in July and September. The July exposure was for 4 consecutive days. The September exposure was for 5 consecutive days.	**Recovery of Trace Elements in Animal Lung Tissues:** July Exposure- Anthropogenic trace elements were below limit of detection in pulmonary tissue of animals exposed to July CAPs. September Exposure- Several elements were recovered from pulmonary tissue during the Sept. exposure. La concentrations were increased in both control/CAPs exposure and in the Ova/CAPs exposure groups. V concentration was increased in Ova/CAPs exposed animals but not in rats exposed to just CAPs. S content was only significant in animals exposed to Ova/CAPs compared to the non-exposed control. **Particle Characterization:** July PM had an average mass concentration twice as high as the September mass concentration. S concentration was four-folds higher in July PM. In the September PM- the concentration of La was 12.5 fold higher than in July PM, V was 2.7 fold higher than in July PM and Mn was 1.5 fold higher than in July PM. **BALF Analysis:** Eosinophil concentration was not significantly different when comparing rats exposed to CAPs only in either July or September (this was explained by the elapsed time between exposure and BALF collection). However OVA and CAP exposure in the September group led to elevated eosinophil levels. Similarly, the protein content was only significantly increased in the September OVA/CAP exposed rats, compared to the control group.

Study	Pollutant	Exposure	Effects
Reference: Nygaard et al. (2005, 088655) **Species:** Mouse **Gender:** Female **Strain:** BALB/c **Age:** 6-7 weeks	Coarse and fine ambient air particles collected in Rome (spring), Oslo (1-summer, fine only, 2- following spring, fine and coarse), Lodz (summer) and Amsterdam (spring). These represent areas with high population and dominance of traffic. DEP (Standard reference material 1650a) used as a positive control. **Particle Size:** Fine PM0.1 - 2.5 μm; Coarse PM 2.5 - 10 μm	**Route:** Subcutaneous Injection into mouse footpads. **Dose/Concentration:** 100 μg of particle **Time to Analysis:** Animals were in eight groups: 1. Control- Hank's Balanced Salt Solution 2. OVA- 50ug 3. OVA (50ug)+ Amsterdam Coarse PM (100 μg) 4. OVA (50ug)+ Amsterdam Fine PM (100 μg) 5. OVA (50ug)+ Lodz Coarse PM (100 μg) 6. OVA (50ug)+ Lodz Fine PM (100 μg) 7. 5. OVA (50ug)+ Oslo Coarse PM (100 μg) 8. OVA (50ug)+ Oslo Fine PM (100 μg) Analysis 5d after injection	**Cell Numbers and Cell Phenotypes in the Lymph Node**: The overall number of B lymphocytes, lymph node cells, PLN cells, and the expression of MHC class II, CD86 and CD23 on B lymphocytes were increased by coexposure of OVA+ the particles compared to the OVA or particle groups alone. The OVA + particle groups displayed a significant decrese in T lymphocytes. Particles only significantly increased the number of lymph node cells and MHC Class II expression. There were no differences observed between coarse and fine PM fractions. **Cytokine Production by Lymph Node (ex vivo culture of popliteal lymph node cells):** The OVA + particle (DEP and Oslo1 only) significantly increased IL-4 and IL-10 levels. No change was observed in IFN-¤. The particle groups only increased IL-4 and IL-10. All coarse and fine particle fractions co-exposed with OVA significantly increased IL-4 and IL-10 compared to OVA alone. There was no significant difference between coarse and fine particles. IFN-¤ levels were not significantly affected by most of the groups, but the fine fractions of PM consistently produced higher levels of IFN-¤. **Lymph Node Histology:** OVA + particle groups resulted in significantly enlarged lymph nodes and the formation of germinal centers.
Reference: Nygaard et al. (2005, 087980) **Species:** Mouse **Gender:** Female **Strains:** BALB/c **Age:** 6-8wks	Polysterene Particles (PSP **Particle Size):** PSP diameter: 0.1 μm	**Route:** Subcutaneous Injection into footpads. **Dose/Concentration:** 40 μg PSP (5.94 X 10^{10} particles) per injection suspended in HBSS. One injection per footpad **Time to Analysis:** 1. HBSS 2. OVA (10 μg per injection) 3. PSP (40 μg per injection) 4. OVA (10 μg per injection) + PSP (40 μg per injection). Antibody experiments: reinjected with 10 μg OVA on day 21. Killed on day 26. Popliteal lymph node cell experiments-- animals injected. Killed 1 to 21d post-injection.	**OVA-specific IgE, IgG1 and IgG2a Antibodies:** Analysis at day 26 indicated IgE, IgG1 levels were significantly higher in mice exposed to OVA+PSP compared to mice injected with HBSS, OVA or PSP. No significant difference was observed for IgG2a levels. **Number of Particle Containing Cells:** There was no significant difference between PSP alone and OVA+PSP. Throughout days 0 -21 the number of particle-containing cells in the PSP or OVA+ PSP groups were significantly greater than the HBSS group. **Total Cell Numbers, B and T Lymphocytes and MHC class II Expression:** The total cell number and B lymphocytes significantly increased by coexposure to OVA+ PSP when compared to the other groups. Both OVA and OVA+PSP increased T lymphocytes on Days 1, 3 and 5. MCH class II expression was significantly higher in the OVA+PSP group on days 5, 7 and 21 than other groups. **Cell Types and Surface Markers:** The number of CD40+ B Lymphocytes showed a slight but significant decrease with OVA+PSP and OVA compared to HBSS and PSP. CD86+, CD23+ and CD69+ B lymphocytes were significantly higher in OVA+PSP group than other groups. PSP alone did not affect CD86+ or CD23+ levels. **Cytokine Production:** IL-4 and IL-10 were significantly higher in the OVA+PSP group when compared to the other groups. OVA alone caused a slight increase compared to PSP. PSP did not alter IL-4 or IL-10 levels.

Study	Pollutant	Exposure	Effects
Reference: Nygaard et al. (2004, 058558) **Species:** Mouse **Gender:** Female **Strain:** BALB/c **Age:** 6-7wks **Weight:** NR	CB (carbon black/DEP) Polystrene Particles (PSP) **Particle Size:** PSP diameter: 0.0588, 0.202, 1.053, 4.64 or 11.14 μm	**Route:** Single subcutaneous injection into footpad **Dose/Concentration:** 10 μg OVA + 40 μg (low dose) or 200 μg (high dose) of particles **Time to Analysis:** Mice killed 5d after OVA injection; PLN excised	**OVA Specific IgE and Ig2a:** OVA with CB, DEP or PSP of diameters 0.0588 and 0.202 μm increased IgE compared to OVA alone, as well as the 1.053, 4.64 and 11.14 μm PSP. OVA with 0.0588 μm PSP or CB significantly increased IgG2a compared to OVA alone. **Primary Cellular Response:** All OVA and PSP groups (except the low dose of 11.14 μm PSP) had more total lymph node cell numbers than the OVA alone group. The low and high does groups of 0.202 μm PSP had the greatest amount of cell proliferation and lymphoblasts. The OVA and 0.202 PSP treatment produced the greatest amounts of B lymphocytes, IL-4, IL-10 and IFN-**◘**. IL-2 in the PLN cells was significantly lower in both dosage groups of OVA and 0.202 PSP than the OVA control. **Particle mass, size, number and surface area:** Total particle surface area explained 64% of the variance in the IgE levels. 60-80% variance of the PLN cellular paramters (except CD23) were explained by total particle surface area, number and diameter.
Reference: Reed et al. (2008, 156903) **Species:** Mouse **Gender:** Male, Female (only BALB/c) **Strain:** C57BL/6, A/J, BALB/c **Age:** NR **Weight:** NR	GEE (two 1996 General Motors 4.3-L V-6 engines; regular, unleaded, non-oxygenated, non-reformulated gasoline blended to US average consumption for summer 2001 and winter 2001-2002-Chevron-Phillips) **Particle Size:** MMAD: 150nm	**Route:** Inhalation Exposure Chamber **Dose/Concentration:** PM: Low- 6.6 ± 3.7 μg/m^3, Medium- 30.3 ± 11.8 μg/m^3, High- 59.1 ± 28.3 μg/m^3 **Time to Analysis:** A/J- 2wk quarantine period in chamber. Exposed 6h/d, 7d/wk, 3d-6m. C57BL/6- 1wk exposure. Instillation of *P. aeruginosa*. Killed 18h postinstillation. BALB/c- Conditioned to exposure chambers and mated. Pregnant females exposed GD 1 and throughout gestation. Offspring exposures continued until 4wks-old. Half of offspring sensitized to OVA. Tested for airway reactivity by methacholine challenge 48h postinstillation and euthanized.	The kidney weight of female A/J mice decreased at 6m and was strongly related to PM by the removal of emission PM. PM-containing macrophages increased by 6m. Hypomethylation occurred in females at 1wk. The clearance of *P. aeruginosa* was unaffected by exposure. Serum total IgE significantly and dose-dependently increased. OVA-specific IgE and IgG1 gave slight exposure-related evidence but were not significant.
Reference: Reed et al. (2008, 156903) **Species:** Rat **Gender:** Male, Female **Strain:** CDF (F344)/CrlBR, SHR **Age:** NR **Weight:** NR	GEE (two 1996 General Motors 4.3-L V-6 engines; regular, unleaded, non-oxygenated, non-reformulated Chevron-Phillips gasoline, U.S. average consumption for summer 2001 and winter 2001-2002) **Particle Size:** MMAD: 150nm	**Route:** Inhalation Exposure Chamber **Dose/Concentration:** PM: Low- 6.6 ± 3.7 μg/m^3, Medium- 30.3 ± 11.8 μg/m^3, High- 59.1 ± 28.3 μg/m^3 **Time to Analysis:** 2wk quarantine period in chamber. Exposed 6h/d, 7d/wk, 3d-6m. SHR- surgery to implant telemeter in peritoneal cavity. 4wks recovery. ECG data obtained every 15min beginning 3d preexposure, 7d exposure, 4d postexposure.	**Organ Weight:** At 6m exposure, the heart weights of male and female rats increased and male rats' seminal vesicle weight decreased. **Histopathology:** PM-containing macrophages increased by 6m. **Serum Chemistry:** Serum alanine aminotransferase, aspartate aminotransferase, and phosphorus decreased in medium and high-exposure females. **Hematology, Clotting Factors:** Hematocrit, red blood cell count, and hemoglobin dose-dependently increased for both genders at both time points. Plasma fibrinogen increased at 1wk in males. **Lung DNA Damage:** Hypermethylation occurred in medium- and high-exposure male rats at 6m. **BAL:** For both genders in the high-exposure group, LDH and MIP-2 significantly increased at 6m. ROS decreased at 1wk and 6m. Generally, the production of hydrogen peroxide and superoxide decreased in the high-exposure group and medium- and high-exposure groups, respectively. **CV effects in SHR:** Lipid peroxides were significantly increased in males in the high exposure group. TAT complexes decreased in females in the high exposure group. **Removal of Emission PM:** The removal of emission PM strongly linked PM to increased seminal vesicle weight, red blood cell counts, LDH, lipid peroxides, and methylation.

Study	Pollutant	Exposure	Effects
Reference: Roberts et al. (2007, 097623) **Species:** Rat **Gender:** Male **Strain:** Sprague-Dawley **Age:** 10wks **Weight:** 250-300g	R-Total = ROFA (Residual oily fish ash) Sample R-Soluble = Soluble fraction of ROFA R-Chelex = R-Soluble + Chelex (insoluble resin) **Particle Size:** Mean diameter- 2.2 μm	**Route:** IT Instillation **Dose/Concentration:** 10mg/kg of body weight (2.5-3mg) **Time to Analysis:** Pre-exposure to ROFA samples on Day 0. Inoculation with 5 x 10^4L. Monocytogenes or saline on day 3. Sacrifice on days 6, 8, 10.	**Uninfected groups:** Compared to the contols, the R-total and R-soluble groups had increased LDH, PMNs, lymphocytes and AMs. The R-total group had a slight, but significant increase in IL-6 and the R-soluble group had a decrease in IL-2. **Infected groups:** The R-soluble group had increased levels of LDH (which also increased for the R-total group), albumin, BAL cells, NK cells, PMA-stimulated and zyomason-stimulated CL compared to all other groups at various time points. NO_X was significantly elevated in the R-soluble group at early time points, but in later time points R-soluble and R-total AMs produced less NO_X than the controls. IL-10 and IL-6 increased in the R-soluble group, while IL-12, IL-4 and IL-2 decreased. IL-12 also decreased in the R-total group.
Reference: Saxena et al.(2003, 054395) **Species:** Mouse **Gender:** Female **Strain:** C57B1/6J **Age:** 18-30wks **Weight:** NR	DEPs (standard) **Particle Size:** NR	**Route:** Intrapulmonary Instillation **Dose/Concentration:** 100 μg/mouse **Time to Analysis:** Pre-exposure to 2.5 x 10^4 bacillus Calmette-Guerin bacteria (BC G) with or without coadministration of DEP. Sacrifice 5wks later.	The BC G + DEP group had four times the BC G lung load than BC G alone. The load was significantly greater in other organs in the BC G + DEP group. Interstitial lymphocytes, T, B and NK cells were increased in the BC G + DEP group over the DEP-alone group. DEP caused no release of NO by AMs, but inhibited the release of NO in response to IFN-□. Except for CD8 cells, no increase in IFN-□ was seen in the BC G + DEP group.
Reference: Schneider et al. (2005, 088368) **Species:** Mouse **Strain:** BALB/c **Cell Line:** RAW 264.7 macrophage cells	SRM 1648 (greater than 63% inorganic carbon; 4-7% organic carbon; Si, S, Fe, Al, K greater than 1% by weight; Mg, Pb, Na, Zn, Cl, Ti, Cu, As, Cr, Ba, Br, Mn less than 1%) Titanium dioxide **Particle Size:** TiO_2 = 0.3 μm average, 1.0 μm max SRM 1648 = 0.4 μm mean diameter	**Route:** Cell Culture (625,000 cells/cm^2 in 96 well plate) **Dose/Concentration:** 62.5 μg/cm^2 **Time to Analysis:** Particulate introduction at 0, 7.8, 15.6, 31.2, and 62.5 μg/cm^2. Measurements made at 1, 3, 6, and 12h after particulate introduction.	No significant toxicity was exhibited by SRM 1648. The rate of dye oxidation was significantly higher in SRM 1648-exposed cells. SRM 1648 significantly increased reduced glutathione compared to the control at the 12-h time point. SRM 1648 increased GSH and concurrently caused significant PGE_2 production compared to the no ester control at the 6-h and 12-h time points.
Reference: Schober et al. (2006, 097321) **Species:** Human **Gender:** Male and Female **Age:** 21-39yrs, treatment group; 23-32yrs, control group **Tissue Type:** Whole blood samples	PM – organic extracts of airborne sample $AERex^{1d}$ – urban aerosol 1 day sample (total air volume - 1270m^3) $AERex^{5d}$ – urban aerosol 5 day sample (total air volume - 6230m^3) rBet v 1 (birch pollen allergen 1a, Biomay, Vienna, Austria) **Particle Size:** NR	**Route:** Cell Culture **Dose/Concentration:** 100 μL heparinized whole blood **Time to Analysis:** Blood stimulated with PBS/IL-3 for 10min. Incubated with rBet v 1 alone or with AERex for 20min. Ice bath 5min. Incubated with antibody reagent 20min.	Nine organic compound classes were identified in $AERex^{1d}$ and $AERex^{5d}$, with $AERex^{1d}$ having 20 times more PAHs. Basophil activation increased in all treatment groups up to 90%, with $AERex^{1d}$ being the most pronounced. 5-50 fold lower concentrations of $AERex^{1d}$ were needed to achieve the maximal effect on basophil activation. AERex-induced enhancement of CD63 upregulation of rBet v 1 in sensitized basophils occurred in a dose-dependent manner.The AERex-alone treatment did not affect CD63 expression.

Study	Pollutant	Exposure	Effects
Reference: Shwe et al. (2005, 111553) **Species:** Mouse **Gender:** Male **Strains:** BALB/c **Age:** 8 wks **Weight:** NR	CB = carbon black particles (Degussa, Germany) CB14: C: 96.79% H: 0.19% N: 0.13% S: 0.11% Ash: 0.05% Others including O: 2.74% CB95: C: 97.98% H: 0.15% N: 0.28% S: 0.46% Others including O: 1.14% **Particle Size:** CB14 = 14nm primary particle size (pps) CB95 = 95nm (pps)	**Route:** IT instillation **Dose/Concentration:** 25, 125, or 625μg in 1 mL saline solution **Time to Analysis:** CB14 or CB95 instillation 1/wk for 4wks Sacrifice 24 or 4h after last instillation	**BAL cells**: In CB14, the total number of BAL cells increased significantly and dose-dependently. In CB95, only the 625μg dose showed a significant increase. **Cytokine and chemokine:** For CB14 and CB95, 125 or 625μg showed a significant IL-1B increase in a dose-dependent manner. For CB14, only the 625 μg dose showed a significant IL-6 increase. No difference was observed in the CB95 group. For CB14, only larger doses showed a significant TNFα increase. For CB95, no significant differences were observed. **In BAL fluid:** CCL-2 production was significantly increased for the 625μg dose in both the CB14 and CB95 groups. CCL-3 production was significantly increased for the larger doses in both the CB14 and CB95 groups. **Splenic Lymphocytes:** No significant differences were detected among the CB14 dosages, except for $CD8^+$. No significant differences were observed among the various groups for CB95. **Deposition in lymph nodes:** For all dosages, greater deposition of CB14 than CB95 was observed. **Chemokine mRNA expression in lungs and lymph nodes:** At 125μg, significant increases of CLL-3 mRNA expression was observed for CB14; for CB95, no differences were detected.

Study	Pollutant	Exposure	Effects
Reference: Sigaud et al. (2007, 096100) **Species:** Mouse **Gender:** Male **Strains:** BALB/c **Age:** 8-10wks **Weight:** NR	CAPs: Concentrated Ambient Particles (Collected from ambient Boston air on Teflon filters.) TiO_2 IFN¤ *S. pneumoniae* (ATCC 6303, American Type Culture Collection, Manassas, VA) **Particle Size:** CAPs: $< 2.5\ \mu m$	**Route:** IFN¤ priming: aerosol Particle exposure and infection: Intranasal Instillation **Dose/Concentration:** CAPS or TiO_2: 50 μg/50 μL PBS *S. pneumoniae:* 10^5CFU/25μl saline **Time to Analysis:** Primed for 15min One time particle exposure 3h post priming with lung RNA analyzed 3, 6, 24h after exposure Sacrificed 24h after exposure OR one time infection Sacrificed 24h infection	**Inflammation:** Saline-primed and unprimed mice exposed to CAPs produced a significant increase in PMNs in the lung (100% more than mice exposed to TiO_2.) Groups primed with IFN¤ then exposed to CAPs produced a strong inflammatory response, a 2.5 increase in PMNs when compared to the increase caused by PBS+ CAPs exposure. **Cytokine Levels:** IFN¤ primed and CAPs exposed groups **Inflammation+ S. pneumo Infection:** Saline-primed and unprimed mice exposed to CAPs produced a significant increase in PMNs in the lung (100% more than mice exposed to TiO_2.) Groups primed with IFNγ then exposed to CAPs produced a strong inflammatory response, a 2.5 increase in PMNs when compared to the increase caused by PBS+CAPs exposure. **Cytokine Levels:** IFNγ primed and CAPs exposed groups showed a 1.5-fold increase over the control. **PMNs:** Treatment with CAPs enhanced inflammation, causing a 2-fold increase in PMN numbers as compared to the infected control. IFNγ+CAPs+*S.* pneumo produced a 3.5 fold increase compared to the infected control and a 1.6-fold increase compared to PBS+CAPs+*S.pneumo.* Despite increased numbers of PMNs in the IFNγ+CAPs groups, the lungs were unable to clear the *S. pneumo* infection. **Bacterial Load:** Control groups showed efficient clearance of bacteria after infection. Unprimed, CAPs-treated, infected groups did not show a decrease in bacterial numbers. IFNγ+CAPs showed a 2.5-fold increase in bacterial numbers. **Histopathology:** Indicated moderate pneumonia in PBS+CAPs and severe pneumonia in IFNγ+CAPs. The other groups did not indicate areas of pneumonia. **Bacterial Uptake AM and PMN Cells:** In all the treated groups, the bacterial content in AMs showed a decrease, with a more marked decrease in the IFNγ+CAPs group, but these decreases were not statistically significant. Groups exposed to CAPs showed a statistically significant decrease in bacterial uptake by PMNs. **ROS Levels in AM and PMN Cells:** Intracellular ROS significantly increased in AM cells in the IFNγ+CAPs group, approximately 50% greater than controls. In PMNs, iROS increased 100% in the IFNγ+CAPs groups as compared to the controls.

Study	Pollutant	Exposure	Effects
Reference: Steerenberg et al. (2004, 087474) **Species:** Rat **Gender:** Male **Strain:** Wistar **Age:** 6-8wks	Ozone (positive control) DEP:SRM1650a (NIST, Gaithersburg, MD EHC-93: ambient PM (Ottawa, Canada) *L. mono: Listeria monocytogenes* (strain L242/73 type 4B) **Particle Size:** Ozone: DEP, EHC-93: NR	**Route:** Ozone: Whole-body inhalation chamber DEP/EHC-93: intranasal droplet: **Dose/Concentration:** Ozone: 2mg/m^3 DEP/EHC-93: 50μg (1.0 mg/ml) *L. mono:* 0.2 or 0.3 ml (5x10^6 PFU/ml) *I have emailed author regarding correct dose **Time to Analysis:** Ozone 24 h/day for 7 days (-7d to -1dR DEP/EHC-93: 1/day for 7 days (-7d to -1d) All rats infected on day 0. Sacrificed on days 3, 4, or 5	**Body weight:** Growth declined for ozone exposed group while DEP or EHC-93 groups grew progressively. Exposure to *L. mono* caused all groups to decline in weight. **Bacterial Count in the Lung:** The number of bacteria in the lung of those rats exposed to ozone was significantly greater than those exposed to saline. No differences in bacteria number were found for rats exposed to saline, EHC-93 or DEP at any time. **Bacterial Count in the Spleen:** The ozone exposed group exhibited statistically significant increases in bacteria numbers when compared to the saline-treated group. No differences in bacteria number were found for rats exposed to saline, EHC-93 or DEP at any time. Exposure to ozone decreases the defense of the respiratory tract against *L. mono* infection; however, DEP and EHC-93 did not appear to affect the host defense system in regards to clearing/fighting *L. mono.*
Reference: Steerenberg et al. (2005, 088649) **Species:** Mouse **Gender:** Male **Strain:** BALB/cByJ.ico **Age:** 6-8wks	PM: collected from Rome, Oslo, Lodz, Amsterdam and De Zilk during the spring, summer and winter. Rome, Oslo, Lodz and Amsterdam represent areas with high population and dominance of traffic. De Zilk, selected as a negative control site, has low traffic emissions and natural allergerns. EHC-93: used as a positive control (Gift of Dr. R. Vincent, Ottawa, Canada) OVA: Ovalbumin (Sigma) **Particle Size:** Coarse PM: 2.5 - 10.0 μm (MMAD); Fine PM: 0.1 - 2.5 μm (MMAD); Ultrafine: $<$0.1 μm (MMAD); EHC-93: NR	**Route:** Intranasal Exposure OVA challenge: aerosol **Dose/Concentration:** PM: 450 μg PM (at 0, 3, or 9 mg/ml) OVA sensitization: 50 μg (0.4 mg/ml) . OVA challenge: 20 μg (0.4 mg/ml) EHC-93 was administered at 0 - 900 μg to evaluate any dose-response relationship. **Time to Analysis:** Sensitization and PM exposure on days 0, 14 Challenged on days 35, 38, 41 for 20min/day Sacrificed on day 42	**Effects of Coarse and Fine Particles:** Immunoglobulins: 6/13 of the coarse and 9/13 fine PM samples induced an increase in IgE and IgG1when compared to the control. IgG2a levels were increased in 3/13 of the coarse and 5/13 of the fine PM. Particles from De Zilk induced all three immunoglobulins, except the fine PM did not induce IgG2a. De Zilk was intended as a negative control (see Table 3). Analysis among the sites comparing the subclasses of antibodies indicated a rank as follows: Lodz $>$ Rome $\geq$ Oslo. **Histopathology:** 9/13 of the coarse PM samples and 5/13 of the fine PM samples induced an inflammatory response. **Bronchoalveolar Cells:** Lodz (spring/ summer) coarse and fine PM induced a significant increase in eosinophils, neutrophils and monocytes. The coarse and fine PM from Rome (spring) induced an increase in neutrophils and the coarse PM induced an increase in eosinophils. Also both Lodz and Rome from the coarse PM from the spring induced an increase in macrophages. Other PM samples did not have an effect on BAL cell counts. **Cytokine Production:** None of the samples produced a significant effect on IL-4 levels. IFN□ levels were significantly decreased in mice exposed to the fine PM fraction (in 8/13 of the samples) when compared to control. Coarse particle exposure did not appear to affect IFN□ levels. TNFα levels were significantly increased (in 2 of the 13 samples) when exposed to coarse PM; fine PM showed similar responses compared to the OVA only group. IL-5 was significantly increased in 4/13 of the coarse and fine PM samples. **Analysis of PM Components:** Samples from Lodz, Oslo and Rome (all spring) were evaluated and the water-soluble coarse PM fraction showed increased immunoglobulin and pathological responses and the water-insoluble fine PM fraction from Lodz (Spring) showed increased reactivity. Leukocytes and cytokines showed no major differences.

Study	Pollutant	Exposure	Effects
Reference: Steerenberg et al. (2004, 087981) **Species:** Mouse **Gender:** Male **Strain:** BALB/cByJ.ico **Age:** 6-8 wks **Treatment:** 1. C.D2-Vil6: $Nramp1^{S}$ and $Nramp1^{R}$ deficient 2. B6.129P2: Nos2tmLau: iNOS deficient 3. BALB/cIL4 (tm2Nnt): deficient in IL-4 4. BALB/c (wild type) pretreated with N-Acetylcysteine (NAC)	Coexposure to EHC-93 and ovalbumin EHC-93: (Gift of Dr. R. Vincent, Ottawa, Canada) **Particle Size:** EHC-93- has a standard reference size.	**Route:** Sensitization, Challenge: Intranasal NAC: IP injection **Dose/Concentration:** OVA: 200 μg (0.4 mg/ml) EHC-93: 150 μg (3 mg/ml) NAC: 320 mg/kg **Time to Analysis:** OVA-only or OVA + EHC-93 sensitization on days 0 and 14. Some mice received NAC before intranasal exposure on days 0 and 14 OVA challenge on days 35, 38 and 41 Sacrificed on day 42	**Natural-Resistance-Associated Macrophage Protein 1 (Nramp1):** When exposed to only ovalbumin, $Nramp1^{S}$ evoked less of an antibody responses (IgE, IgG1 and IgG2a) compared to $Nramp1^{R}$However when coexposed to OVA and EHC-93, the level of increased production of antibodies was similar in both groups. After coexposure, the wild-type showed increased histopathological lesions, whereas the macrophage-stimulation-deficient types showed only a slight increase (not significant). IL-4, IFN-¤, TNF-α and IL-5 levels were similar in wild-type and the Nramp1 strains. **Pretreatment with NAC:** IgG2a concentration was increased further in the group pretreated with NAC. The wild-type mice and the NAC pretreated mice showed similar histopathological lesion patterns. IL-4 levels were similar in wild-type and the NAC pretreated mice. (IFN-¤, TNF-α and IL-5 levels not reported) **Inducible Nitric Oxide Synthase (iNOS):** The wild-type and the iNOS-deficient mice had similar levels of increased IgE antibody production. The IgG1 and IgG2a antibody response was twice as great in the iNOS-deficient mice compared to the wild type. The wild-type and the iNOS-deficient mice showed similar histopathological lesions. No differences in BAL cells or cytokines were observed between the wild-type and iNOS-deficient mice. **IL-4:** The IL-4-deficient mice did not produce an increase in IgE or IgG1 antibodies, as was seen in the wild-type mice. The IgG2a antibody response in the IL-4-deficient mice was similar to the wild type response resulting in adjuvant activity for the IgG2a antibodies. Overall the histological response of the wild-type mice was greater compared to the IL-4 deficient mice. There was no real difference between the two strains observed in the BAL cells, except IL-5 was significantly lower in the IL-4-deficient mice.

Study	Pollutant	Exposure	Effects
Reference: Stevens et al. (2008, 155363) **Species:** Mouse **Gender:** Female **Strain:** BALB/c **Age:** 10-12 wks **Weight:** 17-20 g	DE: generated using a 30 kW 4-cylinder Deutz BF4M1008 diesel engine connected to a 22.3 kW Saylor Bell air compressor. The engine was operated on diesel fuel purchased from a service station in Research Triangle Park, NC. The engine was operated at a steady-state, approx. 20% of engine's full load. High composition: O_2: 4.3 ± 0.07 ppm NO: 9.2 ± 0.30 ppm NO_2: 1.1 ± 0.05 ppm SO_2: 0.2 ± 0.10 ppm Low composition: O_2, NO, NO_2, SO_2 below detection limits **Particle Size:** NR	**Route:** Whole-body Inhalation Chambers (flow rate = 142 L/min) OVA immunization and challenge: intranasal **Dose/Concentration:** High = 2000μg/m^3 Low = 500μg/m^3 **Time to Analysis:** DE exposure for 4h/d on days 0-4. OVA immunization 40min after DE exposure on days 0-2 Challenged on days 18 and 28. Sacrificed 4h after last exposure of day 4 for gene set analysis or 18, 48, or 96h after the last challenge	**IgE Antibody Production:** In the absence OVA, IgE antibodies were not detected. 18, 48 and 96 hours following OVA, mice exposed to low and high doses of DE had an increase in antibodies over time. Mice exposed to high dose had an increase (non-significant) to the OVA exposed control at the 48 h time mark **BAL Cell Counts:** Cell counts at 18 and 96 hours after OVA treatment did not differ among treatment groups. At 48 hours the number of eosinophils, neutrophils and lymphocytes were significantly increased in mice exposed to both high and low concentrations of DE. With DE exposure alone, only neutrophils were statistically increased in the high DE concentration. This indicates the combination exposure of DE and an antigen is essential to promote the development of allergic lung disease. **BAL Cytokine Production:** IL-6 production showed a dose-dependent and time-dependent increase, but was significantly increase in the high dose group at 96h. The high dose group saw a non significant increase in IL-10 levels over time. The greatest increase in IL-10 for the low dose group occurred18 hours after OVA stimulation. **Pulmonary Inflammation and Lung Injury:** No differences among the groups were observed for macrophage, lymphocyte, neutrophil and eosinophil counts. Protein and LDH levels were not found to be increased in the BALF of any group. **Gene Set Analysis:** Pair wise comparisons revealed significant gene set difference between the high DE and control groups. Comparison of the high DE/OVA versus air/OVA showed significant changes in 23 gene sets, including genes involved in oxidative stress responses. The high DE/saline versus the air/saline showed significantly altered pathways. Altered pathways include those for cell adhesion, cell cycle control, apoptosis, growth and differentiation, and cytokine signaling. The results show that relatively short exposures to DE cause mild increases in immunologic sensitization to allergen.

Study	Pollutant	Exposure	Effects
Reference: Takizawa et al. (2003, 157039) **Species:** Human **Cell Lines:** Normal Small Airway Epithelial Cells and Bronchial Epithelial Cells (BET-1A)	Suspended DEP: collected using a 2,300-cc Isuzu diesel engine using standard diesel fuel at 1,050 rpm under a load of 6 torque. DE exposure in vitro (air explosure): collected using a 2,300-ml Isuzu diesel engine at 1,050 rpm. Composition: Fine particles: 1 mg/m^3 CO: 10.6ppm NO_2: 7.3ppm SO_2: 3.3ppm **Particle Size:** NR	**Route:** Cell Culture **Dose/Concentration:** Suspended DEP: varying doses from 0-50 μg/ml IL-13: varying doses from 0-25 ng/ml DE exposure in vitro (air exposure): 100μg/m^3 **Time to Analysis:** Cells were exposed to varying concentrations of suspended DEP for up to 24h. NF-$_k$B: analyzed at 6h after suspended DEP exposure Air exposure at 0, 2, 4, 8 or 14h	Preliminary experiments indicated that DEP at 0.1- 50 μg/mL had no significant cytotoxicity to BET-1A cells and human bronchial epithelial cells (as analyzed by LDH levels). **Eotaxin Production:** (Eotaxin is a cc chemokine that plays a role in eosinophil accumulation in a variety of allergic disorders) Epithelial and BET-1A cells treated with suspended DEP or IL- showed a dose-dependent stimulatory effect on eotaxin release or production. Simultaneous exposure to 25ng/mL IL-13 and DEPdepicted an additive effect for both cell types. **Eotaxin mRNA:** At 25 μg/mL, suspended DEP showed a time-dependent effect on eotaxin mRNA levels up to 12 h in both cell types. Extracted RNA from human bronchial epithelial cells exposed to varying doses of DEP showed a dose-dependent effect for both cell types (up to 25 μg/mL DEP) on eotaxin mRNA levels after 12h of exposure. IL-13 also induced a dose-dependent increase on eotaxin mRNA levels in cells in both cell types. Combination of IL-13 and DEP showed an additive effect on mRNA levels in BET-1A cells. DE exposure in vitro also showed a time-dependent stimulatory effect on eotaxin production in BET-1A cells. **NF-□B / STAT6 Activation:** (it has been suggested that NF-◘B plays a role in the transcriptional regulation of eotaxin gene expression) Cells exposed to 1-25 μg/mL DEP for 6 h increased NF-◘B. BET-1A cells treated with suspended DEP failed to activate STAT6 **Effect of NAC and PDTC on Eotaxin mRNA Levels:** (NAC and PDTC are antioxidant reagents with inhibitory effects on NF-◘B activation) in BET-1A, both NAC and PDTC showed a dose-dependent inhibitory effect on DEP-induced eotaxin production. Both reagents also blocked DEP-induced eotaxin mRNA levels in BET-1A cells. NAC and PDTC did not suppress eotaxin production or eotaxin mRNA levels in IL-13 stimulated BET-1A cells. In addition pre-treatment with NAC attenuated NF-◘B activation induced by DEP but had no effect on STAT6 induction by IL-13. These findings suggest that DEP stimulated eotaxin gene expression via NF-kB dependent, but STAT6-independent, pathways.

Study	Pollutant	Exposure	Effects
Reference: Tesfaigzi et al. (2005, 156116) **Species:** Rat **Gender:** Male **Strain:** Brown-Norway **Age:** 6wks	PM: Wood smoke generated from a conventional wood stove that has a 0.5m3 firebox and a sliding gate air intake damper. The stove was operated over a 3-phase burn cycle that spanned 6 h. Fire was started (initiated exposure) with unprinted / unbleached newspaper and a mix of black and white oak. Wood smoke components: organic material, small amounts of elemental carbon and metals and associated analytes. **Particle Size:** 0.36 μm (MMAD)	**Route:** Whole-body Inhalation **Dose/Concentration:** PM: 1000 μg/m^3 **Time to Analysis:** Exposed to wood smoke or filtered air 6h/day for 70 consecutive days OVA IP injection immunization on days 2, 9 OVA aerosol exposure 2h/day on days 67-70 following daily exposure to wood smoke or filtered air Sacrificed day 70	**Body Weight and Respiratory Function:** No difference in clinical signs or body weight was observed when comparing the two rat groups. The woodsmoke exposed group had a 45% lower dynamic lung compliance when compared to those exposed to the filtered air groupbefore the methacholine challenge. Challenging the rats with methacholine caused a decrease in dynamic lung compliance in both groups, but the decrease was greater in the air-exposed group. At the highest dose of methacholine, the dynamic lung compliance in controls was similar to the baseline value of the smoke-exposed group. No significant differences in total pulmonary resistance were observed. Wood smoke exposed rats had a 10% increase in functional residual capacity than the air-exposed group. **BAL Cell Counts and Cytokines:** There was no difference in lymphocyte, eosinophil or neutrophils in the BALF of either group. There was an increase, though not statistically significant, in macrophages the wood smoke exposed group when compared to the filtered air group. In the BALF, IFN-¤ and and IL-1ß levels were significantly decreased, IL-4 and GRO-α levels were increased in rats exposed to wood smoke compared to filtered air. Serum IgE levels experienced a reduction trend in the wood smoke group, but it did not reach significance. Both groups showed mild signs of inflammation. The average eosinophils present in stained tissue was 21% higher in the wood smoke exposed group compared to the air exposed.
Reference: Tomita et al. (2006, 097827) **Species:** Mouse **Gender:** Female **Strain:** C57BL/6J; AHR-deficient; mEH-deficient; ARNT floxed (loxP sequences inserted in Arnt gene); Tcell-specific ARNT-deficient **Age:** 7wks **Weight:** 20g	DEP: two independent preparations fractionated into 13 different fractions based on acidic and basic functionality (one from light-duty, 4-cylinder diesel engine using standard diesel fueld from Dr. Kei Miwa and other generated from A4JB-type, Isuzu automobile, Japan) Individual PAH tested (Osaka, Japan): BbF = benzo[b]fluoranthene BeP = Benzo[e]pyrene IDP = Indeno[1,2,3-cd]pyrene BpPe = Benzo[ghi]perylene BaP = Benzo[a]pyrene BkF = Benzo[k]fluoranthene Per = Perylene DBA = Dibenzo[a,h]anthracene **Particle Size:** NR	**Route:** Intraperitoneal Injection **Dose/Concentration:** DEP, fractionated DEP or PAH compounds: 0.5 μg - 10 mg/kg bw in 50 ul of olive oil **Time to Analysis:** Single exposure, sacrificed 3d post exposure.	**Effect on thymus:** DEP treatment (10 mg/kg of body weight) caused severe atrophy of the thymus while the spleen and lymph nodes appeared normal. Three days following DEP treatment showed a marked reduction in thymus size. The total number of thymocytes was reduced by more than 70% mostly due to a massive reduction in DP cells (CD4+CD8+). DEP induced no significant alterations in the cell numbers of CD4/CD8 ratios in the spleen and lymph nodes. **DEP Extracts:** Only the WAC (carbonic acid fraction) and BE (weak basic fraction) did not produce a significant reduction in thymocyte numbers in vivo. Among the active fractions, 7 produced a marked selective loss of immature DP thymocytes, similar to the crude extract of DEP. **PAH effects:** Thymic involution was severely induced by the N and various other fractions. 7 out of the 8 PAH compounds were significantly effective in decreasing the number of thymocytes upon in vivo exposure. Only BpPe did not have an effect. **AHR/ARNT and mEH deficient mice (BaP and DEP only):** In the absence of AHR, BaP treatment did not result in a loss of thymocytes. Like DEP, BaP produced severe thymic involution in mEH-deficient mice. DEP-mediated thymic involution was significantly enhanced in mEH-deficient mice.

Study	Pollutant	Exposure	Effects
Reference: Verstraelen et al. (2005, 096872) **Species:** Human **Tissue/Cell Types:** Monocyte-derived dendritic cells (Mo-DC) Cord blood samples of seven women were collected from umbilical vessels of placentas of normal, full-term infants.	DEP- SRM 2975 **Particle Size:** NR	**Route:** Cell Culture **Dose/Concentration:** DEP in varying concentrations: 0.2, 2, 20, 200, 2000 ng/mL LPS 100 ng/mL **Time to Analysis:** Cells were incubated for 24h and analyzed via flow cytometry.	**Biological Markers:** Exposure to DEP alone did not alter expression levels of HLA-DR, CD86 or CD83. Treatment with LPS alone caused a non-significant increase in all three markers when compared to the control. Treatment with DEP+LPS caused a significant increase in the expression of CD83 and a non-significant increased expression of HLA-DR and CD86. DEP+ LPS induced a bell-shape dose-response curve on the expression of all three markers, with a dose of 20 ng/mL DEP + 100 ng/mL LPS causing the largest increase in upregulation. When only the results of the LPS-responsive donors (5 out of 7 blood cord samples) were included, the effects described above become more pronounced.
Reference: Walczak-Drzewiecka et al. (2003, 096784) **Species:** Mouse **Cell Line:** C1.MC/C57.1 (C57) Mast Cells	Metal and Transition Metal Ions: Sr^{2+}, Ni^{2+}, Cd^{2+}, Al^{3+}, Pb^{2+} **Particle Size:** NR	**Route:** Cell culture, **Dose/Concentration:** 0.1- 5 μmol **Time to Analysis:** 10min – 4h incubation before analysis	**BHex Mediator Release in Mast Cells:** Incubation with $SrCl_2$, NiSO4, $CdCl_2$ or $AlCl_3$ resulted in a 2-5% release of B-hexoaminidase in mast cells. Incubation with a mixture of all these compounds induced a greater (11%) release in B-hexoaminidase, indicating there might be a additive effect. **Cell Viability:** Incubation of cells at concentrations and incubation time employed did not result in decrease in cell viability. **Antigen-Mediated Mediator Release in Mast Cells**: Al^{3+} and Ni^{2+} enhanced antigen-mediated release. 10-7 M $AlCl_3$ released 23% of B-hexoaminidase compared to antigen alone, which induced 11% release of B-hexoaminidase. Cd^{2+}, Sr^{2+} and Pb^{2+} enhanced antigen-mediated release to a lesser extent. Ni^{2+}, Al^{3+}, Sr^{2+} and Cd^{2+} depicted a dose-dependent relationship with antigen-mediated B-hexoaminidase release. **Antigen-Induced Protein Phosphorylation**: Addition of the antigen induced the anticipated phosphorylation of multiple proteins in C57 mast cells The presence of Ni^{2+} and Pb^{2+} mediated an increase in phosphorylation of several of the proteins and Al^{3+} mediated a decrease in phosphorylation of multiple proteins (specifically the 56 and 37 kD bands). **Antigen-Mediated Cytokine Secretion (IL-4):** At certain concentrations all tested metal and transition metal ions were able to induce IL-4 secretion or enhance antigen-induced IL-4 secretion in mast cells, but no dose-dependent relationship was established.
Reference: Wan and Yu (2006, 157104) **Species:** Human **Cell Lines:** Human, B cell lymphocytes PMBC (>98.5% B cells-CD19+CD20+; <1 % Tcells (CD3+)) Human lymphocyte cell lines -- DG75 NQO1 wild type	DEP from 4 cyl Isuzu diesel methanol extracts (Previously published) **Particle Size:** NR	**Route:** Cell Culture, PMBC = 1 x 10^6 cell DG 75 = 3 x 10^6 cells IgE PMBC 1 x 10^6/mL B-cells 0.5x 10^6/mL **Dose /Concentration:** 2.5, 5, 10, 20 μg DEPX/ plate (text refers to 20 μg/mL) IgE DEPX 100 ng/mL sulfurophane at 0 - 30 μmol **Time to Analysis:** 6h mRNA; 16 h protein assay. IgE 14d.	**Induction of NQO1 by DEPX**: In PBMCs and DG75DEPX dose-dependently inudced NQO1 mRNA expression NQO1 ARE was increased NAC inhibited NQO1 gene expressiondose dependently. p38 MAPK and P13K inhibition partially blocked NQO1 mRNA and ARE induction by DEPX. **Induction of phase II enzymes:** DEPX induced IgE potentiation was reduced dose dependently by induced phase II enzymes.

Study	Pollutant	Exposure	Effects
Reference: Whitekus et al. (2002, 157142) **Species:** Mouse **Cell Line:** RAW 264.7	DEP (light-duty, four-cylinder engine-4JB1 type, Isuzu Automobile, Japan; standard diesel fuel) (extracts) **Particle Size:** NR	**Route:** Cell Culture **Dose/Concentration:** 50 μg/mL **Time to Analysis:** DEP extracts suspended in methanol and sonicated 20min. Centrifuged 10min. Dried DEP resuspended and stored -20°C. Cell cultures maintained 37 °C. Exposed to antioxidants 5h. HO-1 western blot, determination of cellular GSH:GSSG ratios, carbonyl protein content, lipid hydroperoxides performed.	DEP significantly reduced the GSH:GSSG ratio. This effect was prevented by adding thiol antioxidants NAC or BUC. DEP increased lipid peroxide levels, but the addition of all antioxidants decreased these levels. DEP increased carbonyl groups. NAC, BUC, and luteolin reduced HO-1 expression.
Reference: Whitekus et al. (2002, 157142) **Species:** Mouse **Gender:** Female **Strain:** BALB/c **Age:** 6-8wks **Weight:** NR	DEP (light-duty, four-cylinder engine-4JB1 type, Isuzu Automobile, Japan; standard diesel fuel) (extracts) **Particle Size:** 0.5-4 μm	**Route:** Inhalation **Dose/Concentration:** 200, 600, 2000 μg/m^3 **Time to Analysis:** Exposed 1h/d 10d. Animals receiving OVA had 20min OVA exposure after DEP exposure.	DEP+OVA dose-dependently increased IgE and IgG1, being more effective than the OVA-alone treatment. This effect was significantly suppressed by thiol antioxidants NAC or BUC. DEP+OVA increased carbonyl protein and lipid peroxide over OVA. NAC or BUC suppressed lipid peroxide and protein oxidation. No general markers for inflammation were observed.
Reference: Witten et al. (2005, 087485) **Species:** Rat **Gender:** Female **Strain:** F344 **Age:** 8wks **Weight:** ~175g	DEP (heavy-duty Cummins N14 research engine operated at 75% throttle) **Particle Size:** 7.234-294.27nm	**Route:** Nose-only Inhalation **Dose/Concentration:** Low- 35.3±4.9 μg/m^3, High- 632.9±47.61 μg/m^3 **Time to Analysis:** Exposed 4h/d, 5d/wk, 3wks. Pretreated with saline or capsaicin.	There were no differences for substance P. The low-exposure group had significantly less NK1. DEP reduced NEP activity. Plasma extraversion dose-dependently increased and was greatest in capsaicin animals. Respiratory permeability dose-dependently increased. IL-1B was significantly higher for the low-exposure group. IL-12 was significantly lower in the capsaicin high-expsoure group. TNF-α increased in the high-exposure group and capsaicin low-exposure group. High exposure induced particle-laden AMs in the lungs, perivascular cuffing consisting of mononuclear cells, alveolar edema and increased mast cell number. Neutrophil and eosinophil influx was not seen.
Reference: Wong et al. (2003, 097707) **Species:** Rat **Gender:** Female **Strain:** F344/NH **Age:** ~4wks **Weight:** ~175g	DEP (Cummins N14 research engine at 75% throttle) (EC- 34.93-601.67 μg/m^3, OC- 1.90-11.25 μg/m^3, Sulfates 0.94-17.96 μg/m^3, Na- 4.07-4.78 ng/m^3, Mg- 0.60-0.86 ng/m^3, Ca- 5.05-10.66 ng/m^3, Fe- 3.17-6.44, Cr- 0.68-1.31 ng/m^3, Mn- 0.11-0.22 ng/m^3, Pb- 0.97-1.24 ng/m^3) **Particle Size:** 7.5-294.3nm	**Route:** Nose-only Inhalation **Dose/Concentration:** Low- 35.3±4.9 μg/m^3, High- 669.3±47.6 μg/m^3 **Time to Analysis:** Exposed 4h/d, 5d/wk, 3wks. Pretreated with saline or capsaicin.	DEP dose-dependently increased plasma extraversion, which was further increased by capsaicin. In the high-exposure group, particle-laden AMs (which were reduced by capsaicin), inflammatory cell margination, perivascular cuffing with subsequent mononuclear cell migration and dispersal, increased mast cells, and decreased substance P were all seen. NK-1R was downregulated in the low-exposure group and upregulated in the capsaicin-pretreated high-exposure group. NEP decreased significantly for both groups.

Study	Pollutant	Exposure	Effects
Reference: Yanagisawa et al. (2006, 096458) **Species:** Mouse **Gender:** Male **Strain:** ICR **Age:** 5wks **Weight:** 25-28g	Washed DEP (carbonaceous core), DEP-OC(extracted organic chemicals) and Whole DEP Particles collected from: 4JB1-Type, four-cylinder, 2.74 L, Isuzu diesel engine, while operated on standard diesel fuel at 200g under a load of 10 torques. **Particle Size:** MMAD- 0.4 μm	**Route:** IT Administration **Dose/Concentration:** 50μg/0.1L **Time to Analysis:** 1. Control- 0.1mL PBS 2. DEP-OC- 50 μg 3. Washed DEP- 50ug 4. Whole DEP- 50ug DEP-OC + 50ug Washed DEP5. OVA- 1 μg = 6. DEP-OC- 1 μg + OVA 7. Washed DEP- 50 μg + OVA 8. Whole DEP- 50 μg DEP-OC + 50 μg Washed DEP + OVA All groups received OVA or PBS every 2 wks for 6 wks and the PM component or PBS once a week for 6 wks.	**BAL Analysis**: DEP-OC + OVA caused a significant increase in PMN infiltration in the BAL compared to the control Exposure to Whole DEP+ OVA caused PMN count to rise further **Macrophages:** OVA alone DEP-OC +OVA, Washed DEP + OVA and Whole DEP + OVAall caused a significant increase in macrophages compared to the control. **Lung Histology**: Exposure to OVA, Washed DEP, DEP-OC and Whole DEP caused a slight increase in PMNs, mononuclear cells and goblet cell proliferation. Treatment with all three DEP groups + OVA caused a significant increase in mononuclear cells, PMNs and goblet cell proliferation. Whole DEP + OVA had the greatest impact. **Th1 and Th2 Cytokine Expression**: Washed DEP+OVA caused a significant increase in IFN-¤ compared to control, whereas Whole DEP+OVA caused a significant decrease compared to control. No significant differences in IL-2 and IL-4 levels were seen among groups. DEP-OC+ OVA and Whole DEP+ OVA caused significant increases in IL-5 compared to control and compared to OVA Whole DEP+OVA caused significant increase in IL-13 compared to control **Eotaxin and MIP-1α Expression**: OVA increased eotaxin levels and DEP-OC+OVA caused a more significant increase in eotaxin. Whole DEP alone caused a significant increase in MIP-1α and Whole DEP+OVA caused an even greater increase in MIP-1α. **IgG1 Levels:** Exposure to DEP-OC+OVA caused an increase in IgG1 and exposure to Whole DEP+OVA caused greater elevation in IgG1 levels.
Reference: Yang et al. (2003, 087886) **Species:** Mouse **Gender:** Female **Strain:** B6C3F1 **Age:** 6-8wks	DEP- SRM 1650 **Particle Size:** MMAD- 0.5mm	**Route:** IT Aspiration **Dose/Concentration:** 1, 5, or 15 mg DEP/kg body weight **Time to Analysis:** Mice exposed to 1, 5, or 15 mg DEP/kg of body weight for either 3 times in 2 wks or 6 times in 4 wks.	**Toxicity of DEP Exposure**: DEP did not have a significant effect on body, liver or spleen weight. The highest dose of DEP caused an increase in lung weight and lung weight relative to body weight. None of the hematological parameters were significantly different in the mice exposed for 2wks; in the 4 wk group there was a significant decrease in platelet counts in mice exposed to 15mg/kg. DEP **Exposure on Spleen IgM AFC:** DEP exposure for 2-weeks induced a dose-dependent decrease in spleen AFC in response to sRBC immunization. Mice exposed to 15 μg/kg depicted a 35% reduction in total spleen activity. In the group exposed to DEP for 4 weeks, the decrease in AFC was not significantly different than the control. **DEP Exposure on Spleen Cell Number/Lymphocyte Counts:** Exposure for 2 or 4 weeks did not affect total number of nucleated splenocytes. DEP caused a 30% reduction in total T cells. The number of B cells were not significantly affected. **DEP Exposure on Spleen T-Cell Function:** (evaluated in 2 week exposure group only) DEP induced a dose-dependent decrease in spleen cell proliferation to ConA. DEP did not affect spleen cell proliferation in response to anti-CD3 mAb. Production of IL-2 in response to ConA was reduced in a dose-dependent manner by DEP exposure. IFN-¤ production was decreased by exposure to DEP. IL-4 production was not measuredable.

Study	Pollutant	Exposure	Effects
Reference: Yin et al. (2005, 088133) **Species:** Rat **Gender:** Male **Strain:** Brown-Norway (BN/CrlBR) **Age:** NR **Weight:** 200-250g	DEP = SRM 2975 (NIST) *Listeria* **Particle Size:** NR	**Route:** Nose-only inhalation (DEP), IIT instillation (*Listeria*) **Dose/Concentration:** 100,000 CFU (*Listeria*); 21.2 + 2.3 mg/m^3 (DEP) **Time to Analysis:** DEP exposure for 4 h/d for 5d; infection with Listeria 7d postexposure; sacrifice 3 and 7d postinfection	**Lung deposit:** Estimated mean lung deposit of DEP = 406 + 29 μg/rat DEP prolonged growth of bacteria in lung **Alveolar Macrophage (AM) response:** DEP significantly inhibited *Listeria*-induced IL-1B secretion at d7 and TNF-α snf IL-12 at both d3 and d7. IL-10 production was enhanced at d7. **T-Lymphocyte response:** DEP significantly reduced the development of T cells in response to *Listeria* infection. These lymphocytes displayed increased production of IL-6 at d7, but significantly diminished levels of IL-10, IL-2 and IFN-□.
Reference: Yin et al. (2004, 097685) **Species:** Rat **Gender:** Male **Strain:** Brown-Norway (BN/CrlBR) **Age:** NR **Weight:** 200-250g	DEP = SRM 2975 (NIST) *Listeria* **Particle Size:** NR	**Route:** Inhalation Exposure (DEP) IT instillation (*Listeria*) **Dose/Concentration:** 20.62 + 1.31 mg/m^3 (DEP). 100,000 CFU *Listeria* **Time to Analysis:** DEP exposure for 4 h/d for 5d; inoculation with bacteria 2h postexposure; sacrifice 3, 7, 10d postinfection	**Lung deposit:** Estimated mean lung deposit of DEP = 389 + 25 μg/rat **Pulmonary responses and bacterial clearance:** DEP significantly augmented *Listeria*-induced neutrophil infiltration, lung CFU and recoverable alveolar macrophages (AM) at all times post-infection. LDH activity was increased 3d post-infection. Bacterial count in DEP exposed rats remained significantly higher through d7. **Cytokine production by AM:** DEP exposure significantly lowered *Listeria*-induced production of IL-1B, TNF-α and IL-12. Production of IL-10 was strongly augmented. **T-lymphocyte responses:** DEP moderately but not significantly lowered the total number of lymphocytes, CD4$^+$ cells and lymphocyte IL-10 production. *Listeria*-induced T-cell development was strongly inhibited, as were the development of CD8$^+$ cells, IL-12 production and IFN-□ secretion. DEP and *Listeria* exposure showed and increased production of IL-6 at d3 and d7 post-exposure.
Reference: Yin et al. 2007 **Species:** Rat **Gender:** Male **Strain:** Brown-Norway (BN/CrlBR) **Age:** NR **Weight:** 225-250g	DEP = SRM 2975 eDEP = organic DEP extract wDEP = washed DEP CB = carbon black **Particle Size:** DEP: median diameter-19.4 μm, surface area- 91 μ^2/g; CB: 0.1-0.6 μm	**Route:** IT Instillation of *Listeria*. Treatment after AM cell isolation **Dose/Concentration:** DEP: 10, 50, 100 μg/mL; CB: 50 μg/mL **Time to Analysis:** Sacrifice postinfection or no infection. AM isolated. DEP or CB treatments for 1, 4, 16, 24h.	**AM Phagocytosis:** None of the DEP or CB treatments were cytotoxic or affected the number of adherent cells. 10-100 μg/mL. DEP significantly decreased AM phagocytosis in a concentration- and time-dependent manner, with increased concentration and time decreasing activity. **Bacterial Activity:** The inhibition of AM bactericidal activity by DEP was time- and concentration-dependent. eDEP and wDEP inhibitied the AM bactericidal activity but were less effective than DEP. The CB treatment was not significant. **Cytokine Secretion by AM:** DEP and eDEP concentration-dependently decreased TNF-α, IL-1B and IL-12, but increased IL-10. wDEP and CB did not show a significant effect. **Cytokine Secretion by Lymphocytes:** DEP and cDEP concentration-dependently decreased IL-2 and IFN-□. wDEP and CB had little effect, except high concentrations of wDEP decreased IFN-□.

Study	Pollutant	Exposure	Effects
Reference: Yin et al. (2004, 087983) **Species:** Rat **Gender:** Male **Strain:** Brown-Norway (BN/CrlBR) **Age:** NR **Weight:** 225-250g	DEP = SRM2975 eDEP = organic DEP extract wDEP = washed DEP CB = carbon black **Particle Size:** DEP- NR, CB- 0.1-0.6 μm	**Route:** IT Instillation of *Listeria*. Treatment after AM cell isolation **Dose/Concentration:** 50μg/mL (DEP or CB) **Time to Analysis:** Killed 7d postinstillation. AM isolated then incubated. DEP treatments for up to 24h at 37 degrees.	**DEP-induced ROS production:** ROS was induced by DEP or eDEP and inhibited by eDEP with ANF or NAC. eDEP induction of ROS was time-dependent. wDEP or CB did not induce ROS. **DEP-induced HO-1 expression:** DEP- or eDEP-induced HO-1 expression was inhibited by ANF, NAC or SB203580. wDEP or CB did not induce ROS. DEP or eDEP exposure resulted in a 2.5- to 3-fold induction of HO-1 expression in uninfected AM. **eDEP-modulated cytokine production:** eDEP exposure resulted in a time-dependent increase in LPS-stimulated IL-10 or TNF-α production, and both were inhibitied by ANF or NAC. wDEP did not affect either. SOD pretreatment attenuated eDEP-upregulated HO-1 expression, inhibited IL-10, and reversed eDEP inhibition of IL-12. Znpp decreased IL-10.
Reference: Yin et al. (2003, 096127) **Species:** Rat **Gender:** Male **Strain:** Brown-Norway (BN/CrlBR) **Age:** NR **Weight:** 200-250g	DEP = SRM 1650a *L. monocytogenes* **Particle Size:** NR	**Route:** Nose-only Inhalation (DEP). IT Instillation (*Listeria*) **Dose/Concentration:** 50 or 100 mg/m^3 (DEP); 100,000 bacteria per 500uL sterile saline (Listeria) **Time to Analysis:** DEP exposure for 4h. Bacterial inoculation. Sacrificed 3, 7d postexposure.	**Lymphocyte population:** DEP-alone exposure increased total lymphocytes, T cells and T-cell subsets. Elevated cell counts in the combined exposure were DEP dose-dependent, with the 100 mg/m^3 treatment having significant increases in the cell number and CD^{8+}/CD^{4+} ratio. **IL-2:** DEP exposure in noninfected rats at both doses increased IL-2 in the 24h culture and decreased IL-2 in the 48h culture. The increase in IL-2 at 3 days postinfection was not significant. DEP exposure increased IL-2Rα in response to ConA stimulation. DEP-treated infected rats had increases in ConA-inducible CD^{4+}/IL-2Rα$^+$ and CD^{8+}/ IL-2Rα$^+$. **IL-6:** IL-6 production was dose-dependent in DEP-treated uninfected rats and infected rats. The combined exposure produced less IL-6 than the DEP-alone or *Listeria*-alone treatments. **IFN-□:** DEP decreased IFN-□ at 3 days postexposure, but increased at 7 days postexposure in a dose-dependent manner. Uninfected DEP-treated rats did not substantially respond to HKLM. HKLM-induced IFN-□ production is strongly inhibited at all tested DEP doses.
Reference: Zelikoff et al. (2003, 039009) **Species:** Rat **Gender:** Male **Strain:** Fischer 344 **Age:** 7-8mo **Weight:** NR	CAPS = concentrated ambient $PM_{2.5}$ from New York City *S.pneumoniae* **Particle Size:** MMAD- 0.4 μm	**Route:** Nose-only Inhalation (CAPS) IT Instillation (*S.pneumoniae*) **Dose/Concentration:** CAPS: Study 1- 60-600 μg/m^3, Mean- 345 μg/m^3; Study 2- 65-150 μg/m^3, Mean-107 μg/m^3; (*S.pneumoniae* 2-4 x 10^7) **Time to Analysis:** Study 1: Uninfected rats exposed to air or CAPS for 3h. Sacrificed 3, 24, or 72h postexposure or IT instilled 4, 24, 72, 120h and sacrificed 4, 24, 72h postinfection Study 2: Infection with bacteria. Exposed 48h later to CAPS or filtered air for 5h. Sacrifice 9, 18, 24, 72, 120h postexposure.	**Study 1:** CAPS did not effect cell numbers, viability, profiles, lavageable LDH activity, total protein, or total circulating WBC counts. Exposure to CAPs prior to infection significantly increased PMN and decreased lymphocytes. WBC levels returned to contol levels by 4h postinfection. CAPS had no effect on circulating monocyte values. CAPS significantly increased bacterial burdens at 24h, but thereafter the burden decreased to below control levels. **Study 2:** In CAPS exposed rats, PMN decreased, Pam increased, and the cytokines TNF-α, IL-1Β and IL-6 decreased. Lymphocytes and monocytes were unaffected. Bacterial burdens in CAP-exposed rats were about 10% greater than air contols at 9h and $>$300% greater at 18h. CAPS significantly increased the percent of affected lung area and severity of infection.

Study	Pollutant	Exposure	Effects
Reference: Zelikoff et al. (2002, 037797) **Species:** Rat **Gender:** Male **Strain:** Fischer 344 **Age:** 7-9m **Weight:** NR	Ambient NYC PM Single transition metals of Fe, Mn, Ni *Streptococcus pneumoniae* **Particle Size:** NYC PM: $PM_{2.5}$ Fe^{2+}, Mn^{2+}, Ni^{2+}: 0.4 μm (MMAD)	**Route:** NYC PM, single transition metals: Nose-only inhalation *S. pneumoniae:* IT instillation **Dose/Concentration:** 15-20 x 10^6 (*S.pneumoniae*); Single metals/NYC PM: 65-90μg/m^3 **Time to Analysis:** Infection/no infection followed by 5h exposure to NYC PM or single transition metal. Sacrifice 4, 5, 9, 18, 24, and 120h after exposure	CAPs exposure to infected rats significantly increased pulmonary bacterial burdens of *S. pneumo* in a time-dependent manner. At 9h, 18h, 24h, and 5d after CAPs exposure, bacterial burdens were 10%, 300%, 70% and 30% above controls. Uninfected rats exposed to the single transition metals showed significant alterations in PMNs and lymphocytes values at 1h postexposure. Exposure to Fe of uninfected rats significantly increased superoxide anion production by pulmonary macrophages. Uninfected rats exposed to inhaled Fe significantly reduced B-lymphocyte proliferation at 48 h, but did not affect T-lymphocyte production. Inhaled Ni, for the uninfected, significantly decreased T-lymphocyte production at 18h, and did not affect B-lymphocyte production. Inhalation of Fe by infected rats facilitated an increase in bacterial numbers while Ni inhibited bacterial clearance. Inhaled Fe by infected also significantly decreased PMNs and lymphocyte numbers by 35% and increased pulmonary macrophage numbers by 29% when compared to the air exposed group. Results demonstrated that inhalation of Fe altered innate and adaptive immunity in uninfected hosts, and both Fe and Ni reduced pulmonary bacterial clearance in previously infected rats.
Reference: Zhong et al. (2006, 093264) **Species:** Mouse **Gender:** Male **Strain:** BALB/c **Age:** 6-8wks **Weight:** NR **Cell Line:** J774A.1	CAPs: Concentrated Air Particles (Boston, MA) Urban air particles (UAP) SRM1649 (Washington, DC) Titanium Oxide (TiO_2) (Baker Chemicals, Phillipsburg, NJ) Carbon Black (CB) (Sigma, St. Louis, MO) **Particle Size:** UAP = NR; TiO_2/CB = NR; CAPs: ◘$PM_{2.5}$ *Streptococcus pneumoniae:* strain ATCC6303	**Route:** Cell Culture **Dose/Concentration:** NR, 100 μg/mL **Time to Analysis:** Pre-treated CAPs cells exposed to CAPs for 1h. All cells, IFNγ-primed AMs, unprimed AMs, and J774A.1, exposed to bacteria for 1h. Binding measured 15h after bacteria exposure. Ingestion measured 2h after bacteria exposure. Rate and number of killed bacteria measured 2h after bacteria exposure.	**Binding, internalization and killing of bacteria:** CAPS significantly increased binding of bacteria by IFNγ-primed AMs, normal AMs and J774A.1. CAPS decreased internalization and absolute number of bacteria killed by macrophages of all types. The rate of killing of internalized bacteria was similar in the presence or absence of CAPs; however, CAPs did cause a decrease in the absolute number of bacteria killed by all three types of macrophages, due to the decrease in internalization. **Effects of other particles:** TiO_2 and CB had no effect on J774 binding or internalization of *S. pneumo.* TiO_2 and CB's effects on primed and unprimed AMs were not reported. Testing with UAPs, however, showed effects similar to those observed with CAPs. **Soluble components:** The soluble fraction of CAPs, especially iron, is responsible for decreased internalization.

Table D-5. Effects of the central nervous system.

Reference	Pollutant	Exposure	Results
Reference: Calderón-Garcidueñas et al. (2003, 156316) **Species:** Dog **Gender:** Male, Female **Strain:** Mixed breed **Age:** 7d-10yrs **Weight:** 349±116g – 20kg	Urban Air (Mexico City-high PM region, Tlaxcala- low PM region) (PM, Pb, volatile organic compounds, formaldehyde, acetaldehyde, mutagenic PM, alkane hydrocarbons, Ni, V, Mn, Cr, peroxyacetyl NO_4^{2-}, LPS, endotoxins) **Particle Size:** PM: 2.5, 10 μm	**Route:** Ambient Air Exposure **Dose/Concentration:** Mexico City: PM_{10}: 78 $\mu g/m^3$, $PM_{2.5}$: 21.6 $\mu g/m^3$, Pb in TSP: <0.4 $\mu g/m^3$ **Time to Analysis:** Dogs raised in house or outdoor-indoor kennel. Lifetime exposure.	Mexico City dogs had significantly greater apurinic and apyrimidic sites in the olfactory bulb and hippocampus. Histopathological changes in the respiratory and olfactory epithelium were greatest in Mexico City dogs. Mexico City dogs also had greater immunoreactivity than the controls for NF¤B, iNOS, cyclooxygenase-2, glial fibrillatory acidic protein, ApoE, amyloid precursor product and B-amyloid.
Reference: Campbell et al. (2005, 087217) **Species:** Mouse **Strain:** BALB/c **Age:** 7wks	CAPs from Los Angeles, lacking reactive organic and H2O soluble gases, O_3, NO_x, SO_x **Particle Size:** F+UF: <2.5 μm; UF: <0.18 μm	**Route:** Whole-body Exposure **Dose/Concentration:** 20-fold concentration of near highway ambient air, avg UF concentration: 282.5 $\mu g/m^3$, avg F concentration: 441.7 $\mu g/m^3$ **Time to Analysis:** 4h/d, 5d/wk for 2wks	Mice were challenged with OVA prior to exposure and 1 and 2 weeks following exposure, and then brains were assayed. F+UF and UF exposure increased NF¤B DNA binding in brain. TNFα increased with F+UF. IL-1α increased with UF and F+UF. This suggests a possible link between PM exposure and neurodegenerative disease processes.
Reference: Che et al. (2007, 096460) **Species:** Rat **Strain:** Sprague-Dawley **Gender:** Male and Female **Age:** 9wks **Weight:** 190-220g	Gasoline exhaust (collected from 1996 Guangzhou passenger car with Dongfeng Gasoline Series 155 kw engine and no exhaust catalytic converter fuelled with 90-octane lead-free gasoline from China Petroleum). **Particle Size:** NR	**Route:** IT Instillation **Dose/Concentration:** 5.6, 16.7, or 50.0 L/kg, final volume 0.3 mL/rat **Time to Analysis:** 1/wk for 4wks. Parameters measured 24h post last instillation.	A dose-dependent increase was observed in brain DNA damage starting at 5.6 L/kg. Increase in lipid peroxidation and carbonyl protein was also observed at 50 L/kg. Decrease in brain SOD occurred at all exposures. GPx activity was unchanged with exposure. This suggests an association between gasoline exhaust and oxidative damage to the brain.
Reference: Kleinman et al. (2008, 190074) **Species:** Mouse **Gender:** Male **Strain:** $ApoE^{-/-}$ **Age:** 6wks **Weight:** NR	CAPs (Los Angeles, CA) (OC, EC = ~50%; sulfate, nitrate ~11%) **Particle Size:** NR	**Route:** Whole-body Exposure Chamber **Dose/Concentration:** High dose: Mass concentration- 114.2 $\mu g/m^3$, Concentrated factor- 15.4±3.2; Low dose: Mass concentration: 30.4 $\mu g/m^3$, Concentrated factor- 4.1±0.9 **Time to Analysis:** Exposed 5h/d, 3d/wk, 6wks. Killed 24h postexposure.	Activated AP-1 dose-dependently increased. Activated NF-¤B significantly increased with the high CAPs dose. GFAP (which represented activated astrocytes) and activated JNK significantly increased with the low CAPs dose.
Reference: Liu et al. (2005, 088650) **Species:** Rat **Strain:** Wistar **Gender:** Male **Age:** 8wks	CAPs from Taiyuan, China **Particle Size:** <2.5 μm	**Route:** IT Instillation **Dose/Concentration:** 0, 1.5, 7.5, or 37.5 mg/kg, final volume 0.2 mL/rat **Time to Analysis:** Assayed 24h following instillation	In the brain, SOD and CAT activity were significantly decreased at the 2 highest doses; GSH levels were significantly decreased at the highest dose. This suggests an association between PM exposure and oxidative damage mediated by prooxidant/antioxidant imbalance or high levels of free radicals.
Reference: Sirivelu et al. (2006, 111151) **Species:** Rat **Gender:** Male **Strain:** Brown Norway **Age:** 12-13wks	CAPs from Grand Rapids, MI **Particle Size:** <2.5 μm	**Route:** Whole-body Exposure **Dose/Concentration:** 500 $\mu g/m^3$ **Time to Analysis:** 8h, assayed at 24-h PE	**PVN:** CAPs alone or with OVA increased NE. **MPA:** CAPs increased Da when treated with OVA while no changes in NE, 5-HIAA and DOPAC were observed. **Arcuate nucleus:** OVA sensitization increased NE levels. **OB:** CAPs and OVA increased NE levels, but no changes in Da, DOPAC, or 5-HIAA were observed. **Other areas:** No differences in other areas of hypothalamus, substantia nigra, or cortex were observed. CAPs alone or with OVA increased serum corticosterone. These results suggest that CAPs can cause region-specific modulation of neurotransmitters in brain and that the stress axis may be activated causing aggravation of allergic airway disease.

Reference	Pollutant	Exposure	Results
Reference: Veronesi et al. (2001, 015977) **Species:** Mouse **Strain:** ApoE$^{-/-}$ or C57Bl/6 **Age:** Young adults	CAPs from Tuxedo, NY **Particle Size:** $< 2.5\ \mu m$	**Route:** Whole-body Inhalation **Dose/Concentration:** Varied daily, $PM_{2.5}$ concentrated 10-fold, producing an average of 113 $\mu g/m^3$. **Time to Analysis:** 6h/d, 5d/wk for 4m	CAPs-exposed ApoE$^{-/-}$ mice had an 29% reduction in TH-stained neurons and a 8% increase in GFAP staining compared to air-exposed ApoE$^{-/-}$. No differences were see in C57 mice. The results suggest that ApoE$^{-/-}$ mice, characterized by increased brain oxidative stress, are susceptible to PM-induced neurodegeneration.
Reference: Veronesi et al. (2005, 087481) **Species:** Mouse **Gender:** NR **Strain:** ApoE$^{-/-}$, C57B1/6 **Age:** NR **Weight:** NR	CAPs (Tuxedo, NY) **Particle Size:** 2.5 μm	**Route:** Whole-body Exposure **Dose/Concentration:** Average: 110 $\mu g/m^3$ **Time to Analysis:** Exposed 6h/d, 5d/wk, 4m.	Dopaminergic neurons significantly decreased in CAPs-exposed mice compared to the controls. CAPs caused significantly more astrocytes (as measured by GFAP staining) in the nucleus compacta compared to the controls.
Reference: Win-Shwe et al. (2008, 190146) **Species:** Mouse **Gender:** Male **Strain:** BALB/c **Age:** 7wks **Weight:** NR	DEP (Nanoparticle-rich – NPDE; 81-diesel engine, steady-state condition, 5h/d, 2000rpm, 0 Nm) (CO, CO_2, NO, NO_2, SO_2) **Particle Size:** Diameter: 26.21 ± 1.50nm	**Route:** Whole-body Exposure Chamber **Dose/Concentration:** $148.86 \pm 8.44\ \mu g/m^3$ **Time to Analysis:** Exposed to NPDE or filtered air 5h/d, 5d/wk, 4wks. Some mice ip injected with lipoteichoic acid (LTA) 1X/wk, 4wks. Morris water maze behavioral test: 3d acquisition, 2d probe trial.	Mice in the LTA+NPDE group had significantly longer mean escape latencies, indicating impaired acquisition of spatial learning. NPDE directly increased NR1 and TNF-α. LTA+NPDE increased NR2A, NR2B, and IL-1B, however LTA was primarily responsible for the increases.
Reference: Zanchi et al. (2008, 157173) **Species:** Rat **Gender:** Male **Strain:** Wistar **Age:** 45d	ROFA from Universidade de São Paulo, Brazil **Particle Size:** $1.2 \pm 2.24\ \mu m$ (MMAD)	**Route:** Intranasal Instillation **Dose/Concentration:** 20μg/ 10μl saline **Time to Analysis:** 30d	Exposed rats had increased lipid peroxidation in striatum and cerebellum. This could be reversed with N-acetylcysteine treatment. ROFA treatment altered motor activity shown by decreased general exploration and peripheral walking, and was not prevented by NAC. Results suggests that chronic ROFA induces behavioral changes and brain oxidative stress.

Table D-6. Reproductive and developmental effects.

Reference	Pollutant	Exposure	Effects
Reference: Fedulov et al. (2008, 097482) **Species:** Mouse **Gender:** Female (pregnant), Offspring: NR **Strain:** BALB/c **Age:** NR **Weight:** NR	DEP Carbon black (CB) TiO_2 **Particle Size:** NR	**Route:** Intranasal Insulfation **Dose/Concentration:** DEP, TiO_2: 50 μg in 50 μL, 50 μg/mouse; CB: 250 μg in 50 μL **Time to Analysis:** Particle samples baked 3h. Protocol 1a: Pregnant mice treated with DEP or TiO_2. Analyzed 19 or 48h later. Protocol 1b: Pregnant mice DEP, TiO_2 or CB treated day 14 of pregnancy. 4d-old offspring i.p. injected with OVA+alum. 12-14d-old exposed aerosolized OVA.	DEP increased BAL PMN counts in normal and pregnant mice. In pregnant mice, DEP and TiO_2 increased IL-1B, TNF-α, IL-6 and KC compared to nonpregnant controls. Offspring of DEP, CB or TiO_2 exposed mice had increased AHR and airway inflammation. TiO_2 exclusively altered the expression of 80 genes in pregnant mice.
Reference: Fujimoto et al. (2005, 096556) **Species:** Mouse **Strain:** Slc:ICR **Gender:** Females (pregnant mice and fetuses) **Age:** NR (pregnant females), 14d of gestation (fetuses)	DE: generated by 2369 cc diesel engine at 1050 rpm at 80% load with commercial light oil **Particle Size:** 0.4 μm (MMAD)	**Route:** Inhalation Chamber **Dose/Concentration:** 0.3, 1.0 or 3.0 mg DEP/m^3 **Time to Analysis:** 12h/d, 7d/wk from 2d post coitum to 13dpc. Sacrificed 14dpc. mRNA expression examined in female fetuses.	Significant increase in absorbed placentas were observed in the 0.3 and 3.0 concentration. A decrease in absorbed placentas was observed for the 1.0 concentration. Increased inflammatory cytokine mRNA in placentas from exposed offspring were observed. An increased number of absorbed placentas in DE-exposed offspring were seen.

Reference	Pollutant	Exposure	Effects
Reference: Hougaard et al. (2008, 156570) **Species:** Mouse **Strain:** C57Bl/6 **Gender:** Pregnant females, male and female offspring **Age:** 12 & 16 wks (female offspring), 13 & 17 wks (male offspring)	DEP(SRM2975) **Particle Size:** 90 m^2/g (SA)	**Route:** Whole-body Inhalation **Dose/Concentration:** 20 mg DEP/m^3 **Time to Analysis:** Exposed 1h/d from gestation days 7-19. Mice separated for behavioral testing on PND 22 (day of delivery is PND 0). Behavioral testing at 12, 16 wks for female offspring and 13, 17 wks for male offspring.	Body wt of exposed Unchanged at birth. Body wt decreased At weaning. T4 Unchanged dams & Pups @ weaning. At 2 months, exposed Female pups required Less time to locate Platform in spatial Reversal task of Morris Water maze.
Reference: Hougaard et al. (2008, 156570) **Species:** Mouse **Gender:** Female (pregnant), Offspring- male, female **Strain:** C57BL/6 **Age:** NR **Weight:** NR	DEP (SRM 2975) **Particle Size:** Surface area: 90mg^2/g, Density: 2.1g/cm^3, MMAD: 240nm	**Route:** Inhalation Chamber **Dose/Concentration:** 19.1 ± 1.13mg DEP/m^3 **Time to Analysis:** Pregnant dams exposed GD 7-19, 1h/d. GD 20 named PND 0 for pups. Weights recorded, 1 pup from each group sacrificed PND 2. Weights recorded PND 9. PND 22 1 male and female removed from each group for behavioral testing. Dams and remaining offspring sacrificed PND 23 or 24.	DEP females gained more weight during gestation. Generally, DEP pups weighed less. No significant DNA damage was measured, but DEP caused slightly higher IL-6, MCP-1, and MIP-2. Plasma thyroxin levels as well as learning and memory were similar amongst the groups.
Reference: Huang et al. (2008, 156574) **Species:** Rat **Gender:** Male (adults), male and female (fetuses) **Strain:** Wistar **Age:** 8 wks (male adults), 20d of gestation (fetuses)	ME: Motorcycle Exhaust (generated from 1992 Yamaha cabin motocycle with two-stroke 50 cc engine). **Particle Size:** NR	**Route:** Nose-only Inhalation **Dose/Concentration:** 1: 10 and 1: 50 dilutions **Time to Analysis:** 2h/d (1h in morning and 1h in afternoon), for 5 consecutive days/wk, for 4 wks (1:50, 1:10 dilutions) and 2 wks (1:10 dilution). Male mated with untreated females. Pregnant females sacrificed on 20d of gestation. Male and female fetuses observed.	After exposure, decreased body weight and testicular spermatid number were observed. 1: 10 ME exposure for 4 weeks (no recovery) decreased testicular weight and increased the inflammatory cytokine mRNA. Glutathione system and LipidPeroxidation were not affected.
Reference: Lichtenfels et al. (2007, 097041) **Species:** Mouse **Gender:** Male and Female **Strain:** Swiss **Age:** NR	Ambient air in São Paulo, Brazil **Particle Size:** NR	**Route:** Ambient Air Exposure **Dose/Concentration:** NA **Time to Analysis:** Males housed in open-top chambers for 24h/d, everyday for 4m, beginning 10d after birth. Males mated to non-exposed females immediately following exposure. Males sacrified immediately following mating. Pregnant females remain in chamber and sacrificed on 19d of pregnancy.	Decreased testicular, epididymal sperm counts, decreased number of germ cells, and decreased elongated spermatids were observed. Decreased SSR, and a sex ratio shift (fewer males) also occurred after exposure.
Reference: Mauad et al. (2008, 156743) **Species:** Mouse **Gender:** Male, Female **Strain:** BALB/c **Age:** 10d **Weight:** Parental: 21.4 ± 4.0 – 26.3 ± 2.8g; 15d-old offspring: 7.8 ± 1.1 – 9.0 ± 1.0g; 90d-old offspring: 20.3 ± 2.3 – 27.4 ± 1.8g	PM (busy traffic street São Paulo, Brazil; Aug. 2005-April 2006) (NO_2, SO_2, CO) **Particle Size:** Diameter: 2.5, 10 μm	**Route:** Ambient Air Exposure **Dose/Concentration:** $PM_{2.5}$: filtered chamber- 2.9 ± 3.0 $\mu g/m^3$, nonfiltered chamber- 16.9 ± 8.3 $\mu g/m^3$; Outdoor concentration: PM_{10}- 36.3 ± 15.8 $\mu g/m^3$, CO- 1.7 ± 0.7ppm, NO- 89.4 ± 31.9 $\mu g/m^3$, SO_2- 8.1 ± 4.8 $\mu g/m^3$ **Time to Analysis:** Nonfiltered exposure 24h/d 4m. Mated at 120d exposure. After birth, 30 females and offspring transferred to filtered or nonfiltered chamber. Killed 15 or 90d of age.	Mild foci of macrophage accumulations containing black dots of carbon pigment occurred in the alveolar areas on 90d-old mice. Surface-to-volume ratio decreased from 15 to 90d of age and was higher in mice exposed to air pollution. PM exposure reduced inspiratory and expiratory volumes at higher levels of transpulmonary pressure.

Reference	Pollutant	Exposure	Effects
Reference: Mohallem et al. (2005, 088657) **Species:** Mouse **Strain:** BALB/c **Gender:** Female **Age:** 10wks, 10d	Filtered or ambient air in downtown Sao Paulo situated at crossroads with high traffic density (predominant source of air pollution is automotive). Component concentrations in ambient air: CO: 2.2 ± 1.0ppm; NO_2: 107.8 ± 42.3 $\mu g/m^3$; PM_{10}: 35.5 ± 12.8 $\mu g/m^3$; SO_2: 11.2 ± 5.3 $\mu g/m^3$ Component concentrations in filtered air: NO_2: 19.5 ± 2.8 $\mu g/m^3$; PM_{10}: 24.1 ± 8.1 $\mu g/m^3$ **Particle Size:** NR	**Route:** Whole-body Inhalation **Dose/Concentration:** 50 L/m (polluted chamber) **Time to Analysis:** Exposed for 24 h/7days/wk for 4 mo. Newborns mated after reaching reproductive age of 12 wks. All pregnant females sacrificed between 19th and 20th day of pregnancy.	No effects in adult exposed animals. Increased implantation Failure of neonatal Exposed-dams. Sex ratio, # of Pregnancies, Resorbtions, Fetal deaths, And fetal placenta Weights unchanged After neonatal Ambient air exposure.
Reference: Mori et al. (2007, 096564) **Species:** Mouse **Strain:** C57/BL **Gender:** Male **Age:** 6wks	DEP: generated by 4-cylinder diesel engine **Particle Size:** NR	**Route:** Dorsal Subcutaneous Instillation **Dose/Concentration:** 0.2 ml (of 1.1mg/ml or 0.37 mg/ml) **Time to Analysis:** 2x/wk for 10 wks. Parameters measured 1wk post last instillation.	cDNA library screen after Sub-cut. Injection Found identified activated Clones related to Prostanoids & Arachadonic Acid (Platg2c2c, Acsl6) & sperm Production (Stk35). Route of exposure. Unconventional.
Reference: Ono et al. (2007, 156007)) **Species:** Mouse **Strain:** ICR **Gender:** Pregnant females, male offspring **Age:** NR (pregnant females), 12 wks (offspring)	DE: generated from 4-cyl diesel Isuzu engine at 1500 rpm using standard diesel fuel. **Particle Size:** NR	**Route:** Inhalation (further details NR) **Dose/Concentration:** **Time to Analysis:** Exposed from 2d post coitum to 16dpc. Parameters for male offspring measured on days 8, 16, 21, 35, 84 and sacrificed at 84d.	PND 8 and 16 male repro accessory gl weight decreased. PND 21decreased serum T; PND 84 increased serum T. FSHr, sTAR mRNA Decreased PND 35 & 84. Rel. testis and epididymal wt unchanged. Sertoli cell degeneration
Reference: Ono et al. (2007, 156007)) **Species:** Mouse **Strain:** ICR **Gender:** Male offspring, Pregnant females **Age:** 12wks (male offspring)	DE: generated from 4JB-2type, light duty 3060 cc 4-cyl Isuzu diesel engine under 1500 rpm **Particle Size:** NR	**Route:** Whole-body Inhalation **Dose/Concentration:** 1.0 mg DEP/m^3 **Time to Analysis:** Pregnant females exposed from 2d postcoitum- 16dpc. Without undergoing further exposure, male offspring sacrificed at 12wks.	Dose-dep increase seminif. Tubule degeneration & decreased DSP. 1 mo recovery, DSP recovered at lowest dose.
Reference: Pinkerton et al. (2004, 087465) **Species:** Rat **Gender:** Female (pregnant), Offspring- NR **Strain:** SD **Age:** 10d (pups), Pregnant females- 10-14d of gestation **Weight:** NR	PM (Fe and soot from combustion of acetylene and ethylene in a laminar diffusion flame system) **Particle Size:** Median diameter: 72-74nm; size range: 10-50nm	**Route:** Inhalation Chamber **Dose/Concentration:** Mean mass concentration: 243 ± 34 $\mu g/m^3$; Average Fe concentration: 96 $\mu g/m^3$ **Time to Analysis:** Exposed 10d postnatal age, 6h/d, 3d (consecutive). Bromodeoxyuridine injected 2h before necropsy. .	A significant reduction of cell proliferation occurred only within the proximal alveolar region of exposed animals compared to controls. There were no significant differences between the groups for alveolar formation and separation within the proximal alveolar region

Reference	Pollutant	Exposure	Effects
Reference: Silva et al. (2008, 156981)) **Species:** Mouse **Strain:** Swiss **Gender:** Females (pregnant mice) **Age:** 1st, 2nd, 3rd wk of pregnancy (females), GD19 (fetuses)	Ambient air Sao Paulo, Brazil **Particle Size:**	**Route:** Ambient Air Exposure **Dose/Concentration:** **Time to Analysis:** 1st week 2nd week 3rd week or combo of exposure during preg.	Decreased fetal wt with exposure in 1st week of preg. Decreased placental Wt with exposure in any of 3 weeks.
Reference: Somers et al. (2002, 078100) **Species:** Mouse **Strain:** Swiss Webster **Gender:** Male and Female **Age:** 6-8wks (adult male and females), 5d (pups)	Ambient air at 2 sites in Canada (polluted industrial area 1km downwind from two integrated steel mills & rural location 30km away) **Particle Size:** NR	**Route:** Ambient Air Exposure **Dose/Concentration:** Ambient air **Time to Analysis:** Exposed 24h/d, 7d/wk for 10 wks from September 10, 1999- November 21, 1999. Exposed to clean air for 6wks post-treatment. Paired with mice within exposure group. 5d old pups measured.	ESTR germ Line mutations followed . Heritable mutation rate increased 1.5 to 2 fold in urban vs. rural site. Increased freq is paternal line dependent.
Reference: Somers et al. (2004, 078098) **Species:** Mouse **Gender:** NR **Strain:** Sentinal Lab **Age:** NR **Weight:** NR	PM (rural or urban-industrial) **Particle Size:** >0.1 μm	**Route:** Ambient Air Exposure **Dose/Concentration:** Mean TSP: Rural- 16.2 ± 8.3 – 31.7 ± 13.2 $\mu g/m^3$, Urban-Industrial- 38.9 ± 10.5 – 115.3 ± 25.3 $\mu g/m^3$ **Time to Analysis:** Exposed 10wks. Bred 9wks postexposure.	The offspring of urban-industrial mice inherited paternal ESTR mutations 1.9-2.1 times more than rural or HEPA-filtered offspring. Maternal ESTR mutations were not significant.
Reference: Sugamata et al. (2006, 157025) **Species:** Mouse **Strain:** ICR **Gender:** Pregnant Females, male and female offspring **Age:** 11wks (offspring), NR (pregnant females)	DE (origins NR) **Particle Size:** NR	**Route:** Inhalation (more specific information NR) **Dose/Concentration:** 0.3 mg DEP/m^3 **Time to Analysis:** Pregnant females exposed from 2d post coitum to 16dpc. Offspring sacrificed 11wks after birth.	Exposed pups increased caspase 3 positive cells & decreased purkinjie cell # (cerebellum). Similar to human Autism brain phenotype.
Reference: Tozuka et al. (2004, 090864) **Species:** Rat **Strain:** Fischer 344 **Gender:** Pregnant females, male and female fetuses **Age:** Gestation day 20 (fetuses), NR (pregnant females)	DE: generated by diesel engine (309 cc Model NFAD-50) **Particle Size:** NR	**Route:** Whole-body Inhalation **Dose/Concentration:** 1.73mg/m^3 **Time to Analysis:** Exposed 6h/d from GD 7-20 with no exposure on Saturdays or Sundays (4 non-exposure days total). Fetuses and maternal blood collected on GD20. PAHs: Exposed 6h/d from GD 7-14 with no exposure on Saturdays or Sundays. Breast milk collected PND14.	Gest & lactational Exposure to DE's And PAHs. 7 milk PAHs increased in DE exposed dams. DE exposure Can lead to PAH Pup exposure through Breast milk.
Reference: Tsukue et al. (2004, 096643) **Species:** Mouse **Strain:** Slc: ICR **Gender:** Pregnant females, female fetuses **Age:** Gestation day 14 (fetuses)	DE: generated by 2369 cc Isuzu diesel engine operating at 1050 rpm with 80% load and using commercial light oil. **Particle Size:** NR	**Route:** Whole-body Inhalation **Dose/Concentration:** 0.1 mg DEP/m^3 (at 1:8 dilution with clean air) **Time to Analysis:** Exposed for 8h/d from 2d postcoitum to 13dpc (with no exposure on days 4, 5, 11, 12). Sacrificed 14dpc. Only female fetuses studied.	SF-1 & MIS mRNA no change. Other steroidogenic Genes unchanged. BMP-15, oocyte Differentiation mRNA decreased.

Reference	Pollutant	Exposure	Effects
Reference: Tsukue et al. (2002, 030593) **Species:** Mouse **Strain:** C57/BL **Gender:** Females, male and female offspring **Age:** 6wks, 70d post natal (offspring)	Filtered air DE: generated by light-duty, 4-cyl Isuzu diesel engine at 1500 rpm. **Particle Size:** NR	**Route:** Whole-body Inhalation **Dose/Concentration:** 0.3, 1.0 or 3.0 mg DEP/m^3 **Time to Analysis:** Exposed 12h/d, 7d/wk for 4 mo. Some females sacrified immediately following exposure. Remainder mated with unexposed males. Parameters measured in offspring at postnatal day 70.	DE-exposed females had Decreased uterine wt at 4 months. Offspring decreased Body weight @ 6 & 8 weeks of age. Decreased rate Of good nest Construction (3 mg/m^3). AGD decreased In males (30 & 70 days old). Organ weight Decreased in Females. Female Crown to rump Length decreased. Relative weights Not taken.
Reference: Ueng et al. (2004, 096199) **Species:** Mouse **Gender:** Female **Strain:** Wistar **Age:** 21d **Cell Line:** MCF-7	ME: generated from a Yamaha Cabin motorcycle 2-stroke 50-cc engine and variable venture carburetor **Particle Size:** NR	**Route:** Intraperiotenial Instillation. Cell Culture **Dose/Concentration:** IP: 1, 10, 50 μg/ml Cell Culture: 0.01, 0.1, 1, 10, 50, 100 μg/ml **Time to Analysis:** IP: 1/d for 3d and sacrificed on 24d. Cell Culture: 3, 24, 30, 48h and 2d.	10 mg/kg +E2 induced anti-estrogenic uterine effects & Antiestrogenic with in vitro (MCF-7 cells) E2 screen.
Reference: Veras et al. (2008, 190493) **Species:** Mouse **Gender:** Male, Female **Strain:** BALB/c **Age:** 20d, newborns **Weight:** NR	PM (downtown São Paulo, Brazil near crossroads with high traffic density, 67% $PM_{2.5}$ comprises air pollution) **Particle Size:** Diameter: 2.5 μm	**Route:** Open-Top Chamber **Dose/Concentration:** $PM_{2.5}$- 27.5 $\mu g/m^3$; NO_2- 101 $\mu g/m^3$; CO- 1.81 $\mu g/m^3$; SO_2- 7.66ppm **Time to Analysis:** 20d-old mice maintained in filtered or nonfiltered chamber until 60d-old. Offspring maintained in respective chambers until 21d-old. Offspring mate at 60d-old. Females euthanized 18th GD.	Fetal weight and maternal blood space volume and surfaces declined in the groups exposed to nonfiltered air. Fetal capillary surfaces were greater in nonfiltered air groups. There was a significant gestational effect on maternal:fetal surface ratios with values declining significantly in groups exposed during pregnancy to nonfiltered air. The total oxygen diffusive conductance of the intervascular barrier increased significantly during pregestational exposure to nonfiltered air. Mass-specific conductance increased during pregestational and gestational periods of exposure to nonfiltered air.
Reference: Veras et al. (2009, 190496) **Species:** Mouse **Gender:** Male, Female **Strain:** BALB/c **Age:** 20d **Weight:** NR	PM (São Paulo, Brazil; near crossroads with high traffic density) (Al, Ca, Cu, Fe, K, Na, Ni, P, Pb, S, Si, Ti, V, Zn, C) **Particle Size:** Diameter: 2.5 μm	**Route:** Open-Top Chamber **Dose/Concentration:** Mean: Non-filtered- 27.5 $\mu g/m^3$, Filtered- 6.5 $\mu g/m^3$ **Time to Analysis:** 20d-old mice maintained in filtered or non-filtered chamber. Allowed to mate at 60d. 2 generation model.	Ambient air pollution extended the estrus cycle, which reduced the number of cycles. Antral follicles decreased. Mating time increased and fertility and pregnancy indices decreased. The mean post-implantation loss rate increased, which was influenced by both pre- and post-gestational exposure. Fetal weight decreased and was also influenced by pre- and post-gestational exposure, which exhibited a significant interaction.
Reference: Watanabe (2005, 087985) **Species:** Rat **Gender:** Female (pregnant), Offspring- male **Strain:** F344/DuCrj **Age:** 7d of gestation – parturition (females), 96d (offspring) **Weight:** 240-262g (offspring)	DE (309cc engine, Model NFAD50, Yanmar Diesel Co., Osaka, Japan, 1800rpm, 45% load) (PM, NO_2) **Particle Size:** 90% <0.5 μm	**Route:** Inhalation Chamber **Dose/Concentration:** High dose total group: PM- 1.71 $\mu g/m^3$, NO_2- 0.79ppm; Low dose total group: PM- 0.17 $\mu g/m^3$, NO_2- 0.10ppm **Time to Analysis:** Pregnant rats exposed gestational day 7 to delivery 6h/d. 5 groups: high dose total DE, high dose PM, NO_2 filtered, low dose total DE, low dose PM, NO_2 filtered, clean air control. Offspring sacrificed day 96 after birth.	All groups had significantly less daily sperm production than the control. PM and NO_2 in DE decreased spermatogenia but was not significant, however the high dose PM filtered group achieved significance. Pachytene cells, spermatids, and Sertoli cells were lower in all groups compared to the control.

Reference	Pollutant	Exposure	Effects
Reference: Yauk et al. (2008, 157164) **Species:** Mouse **Strain:** C57BL/6 x CBA F1 hybrid **Gender:** Male **Age:** 7-9wks	Hepa-Filtered air (PM removed) and ambient air at 2 sites: -2km from two integrated steel mills -1km from major highway on Hamilton Harbor Components: Metals 3.6 ± 0.7 μg/m^3 TSP 9.4+17 μg/m^3 **Particle Size:** NR	**Route:** Ambient Air Exposure **Dose/Concentration:** Ambient air **Time to Analysis:** Parameters measured 3, 10wks, or 10 + 6wks recovery following exposure.	10+6 weeks exposure induced increased ESTR mutations in sperm DNA of exposed v filtered. No testicular DNA adducts seen in exposed males. @ 3wks DNA increased adducts seen in lungs of exposed males, not in filtered males. Mutations PM dependent Gas phase independent.
Reference: Yokota et al. (2009, 190518) **Species:** Mouse **Gender:** Female (pregnant), Male (offspring) **Strain:** ICR **Age:** NR **Weight:** NR	DE (2369-cc diesel engine, Isuzu Motors, Ltd., Tokyo, Japan; 1050rpm, 80% load, commercial light oil) **Particle Size:** NR	**Route:** Inhalation Chamber. Pre-natal Exposure **Dose/Concentration:** DE: 1.0mg/m^3; CO: 2.67ppm, NO_2: 0.23ppm, SO_2: $<$0.01ppm **Time to Analysis:** Pregnant mice exposed 8h for 5d from GD 2-17. Mothers and pups kept in clean room. Pups weaned on PND 21 then transported to Tokyo University of Science. 2wk acclimation. Exposed 12h light/dark cycle. Activity monitor with infrared ray sensor measured spontaneous motor activity (SMA), 10min intervals 2d. After behavioral test, mice decapitated.	Prenatal DE exposure decreased SMA in the male offspring. DE decreased locomotor activity during the light phase. Dopamine levels in the striatum and nucleus accumbens did not change, but HVA concentrations decreased in DE-exposed mice.
Reference: Yoshida et al. (2006, 156170) **Species:** Mouse **Strain:** ICR, C57Bl/6J or DDY **Gender:** Pregnant Females, Male fetuses **Age:** 14d of gestation (fetuses), 2-13d of gestation (pregnant females)	DE(generated from a 4-cyl., 2300 cc diesel Isuzu engine at 1050 rpm and 80% load). **Particle Size:** NR	**Route:** Whole-body Inhalation **Dose/Concentration:** 0.1 mg DEP/m^3 **Time to Analysis:** Exposure on 2-13d of gestation. Parameters measured on 14d of gestation.	Responses to exposure showed strain-related variations with ICR as the most sensitive followed by C57 and ddY as the least sensitive. MIS mRNA expression, a factor in male gonadal differentiation, was significantly decreased in the ICR and C57 strains. Ad4BP/SF-1 expression was significantly decreased in the ICR strain only.
Reference: Yoshida et al. (2006, 097015) **Species:** Mouse **Strain:** ICR **Gender:** Pregnant females and male offspring **Age:** 2-16d postcoitum (pregnant females), 28d (male offspring)	DE: generated by 4Jb1-type, light duty 4-cylinder Isuzu diesel engine using standard diesel fuel at 1500 rpm. **Particle Size:** NR	**Route:** Whole-body Inhalation **Dose/Concentration:** 0.3, 1.0 or 3.0 mg DEP/m^3 **Time to Analysis:** Pregnant females exposed 12h/d, 7d/wk from 2-16d postcoitum. Offspring sacrificed on postnatal day 28.	NOAEL 0.3 mg DEP/m^3 DE exposure induced increased repro gland weight (two higher doses) male mice. mRNA decreases aromatase & 3 μ-hD (3.0 mg DEP/m^3) Sex ratio no change. 2 higher doses induced sig increased repro organ wt. Male pup wt Increased at PND 28. 1.0mg DEP/m^3 pup increased serum T. Serum T positively correlated with DSP, testis wt, steroid enzyme mRNA.
Reference: Yoshida et al. 2004 (2004, 097760) **Species:** Mouse **Gender:** Female (pregnant), Offspring- male **Strain:** ICR **Age:** 4, 6wks **Weight:** NR	DE **Particle Size:** NR	**Route:** Inhalation Chamber **Dose/Concentration:** 6wk-old males, embryos: 0.3, 1.0, 3.0mg DEP/m^3, Pregnant mice: 0.1, 3.0mg DEP/m^3 **Time to Analysis:** 6wk-old males: Exposed 12h/d, 6m. 1m clean air exposure. Pregnant mice: Exposed 2-13p.c. 8h/d. Male embryos: Exposed 2-16p.c. Examined at 4wks-old.	**6wk-old males:** In the seminiferous tubules, DE dose-dependently caused degenerative and necrotic changes, desquamation of the seminiferous epithelium, and loss of spermatozoa. Spermatogenesis was still inhibited after a 1m clean air exposure. **Pregnant mice:** Ad4BP/5F-1 and MIS mRNA significantly and dose-dependently decreased in male fetuses exposed to DE. **4wk-old male newborns:** Tissue weight of the testis and accessory reproductive glands were significantly greater in DE-exposed mice. Blood testosterone concentration was 8X higher than the control at 1.0mg DEP/m^3. No significant differences occurred for testosterone synthetase mRNA.

D.1. Carcinogenisis, Mutagenesis, Genotoxicity

Table D-7. Mutagenic/genotoxic effects in bacterial cultures.

Reference	Pollutant	Exposure	Effects
Reference: Binkova et al. (2007, 156273) **Species:** *Salmonella* (±S9 (rat liver)) **Cell Line:** Calf thymus DNA	PM (Prague, Košice, Sofia, Czech Republic; summer, winter) (organic extracts) **Particle Size:** Diameter: < 10 μm	**Route:** Cell Culture **Dose/Concentration:** 100 μg EOM/mL **Time to Analysis:** PM collected 24h daily 3m, extracted. 24h incubation BaP, c-PAH, EOM, with or without S9. ^{32}P-Postlabeling 4h. Autoradiography 1-24h.	DNA adducts in EOM treatments were greater with S9 than without. Positive correlations were found between the amount of DNA adducts and the PAH content (notably BaP) in the EOM treatment.
Reference: Brits et al. 2007 **Species:** *S. typhimuriam* **Strain:** TA98±S9 (Ames); TA104 recN2-4 and TA104pr1 (Vitotox) **Cell Line:** Human whole blood (Comet, MN assays)	PM (Flanders, Belgium; urban, rural, industrial sites) (organic extracts) **Particle Size:** Diameter: 10 μm	**Route:** Cell Culture **Dose/Concentration:** 2.5, 5, 10, 20m^3 air equivalents/mL **Time to Analysis:** Air samples extracted. Ames assay 48h. Vivotox test. Comet assay 24h. MN assay.	**Ames:** S9 induced mutagenicity of all extracts from all areas in a dose-dependent manner. Without S9, only extracts from the urban and industrial areas were mutagenic at the highest dose. **Vitotox:** Extracts were toxic at the highest dose. **Comet:** Significant DNA damage in the extracts was seen and enhanced by S9. **MN:** A dose-response relationship was seen in the urban extracts for increased micronucleated binuclear cells.
Reference: Brown et al. (2005, 095919) **Species:** *S. typhimuriam* **Strain:**TA98 **Cell Line:** Rat hepatoma H4IIE	PM (New Zealand, summer, winter) (extracts) **Particle Size:** Diameter: 10 μm	**Route:** Cell Culture **Dose/Concentration:** 9.7-20.8 μg/m^3 (summer), 21.8-61 μg/m^3 (winter) **Time to Analysis:** Air samples collected 15d, extracted. Ames test: Bacteria growth 12h, incubated 24h. Hepatoma bioassay: 24h incubation 2x. EROD assay.	Generally, the mutagenic rate was positively correlated to PM_{10}, as well as PAH and BaP. PM_{10} levels were higher and more mutagenic in winter than summer.
Reference: Bunger et al. (2006, 156303) **Species:** *Salmonella typhimuriam* **Strain:** TA98, TA 100	DEP (diesel fuel (DF), low-sulfur diesel fuel (LSDF), rapeseed oil methyl ester (RME), and soybean oil methyl ester (SME)) (SOF-soluble organic fractions) **Particle Size:** Total particulate matter (no OCC) (gh^{-1}): Mean DF- 4.0 ± 0.2; 2.8 ± 0.5; 1.8 ± 0.0; 3.4 ± 0.2; 1.2 ± 0.1	**Route:** Cell Culture **Dose/Concentration:** Log 2 dilutions of extracts: 1.0, 0.5, 0.25, 0.125 **Time to Analysis:** SOF extracted 12h. Plates incubated 48h.	**No OCC:** Without oxidation catalytic converter (OCC), DF extract produced the highest number of revertant colonies at all load modes in both TA98 and TA100 ± S9. RME, SME, and LSDF extracts caused lower or no mutagenic effects, seen especially at partial load modes and idle motion. **OCC:** With OCC, all extracts reduced the number of revertant colonies in TA98 and TA100 ±S9 at partial load modes B, C, and D. At load mode A (rated power), there was an increase of the number of revertant colonies in all assays -S9, significant for extracts from RME (TA98, TA100) and SME (TA98). S9 lowered frequency of mutations. At load mode E (idling), number of revertant colonies of DF extracts increased ±S9.
Reference: Bunger et al. (2007, 156305) **Species:** *Salmonella typhimuriam* **Strain:** TA98, TA 100	Diesel engine emissions (DEE)—rapeseed oil (RSO) and rapeseed methyl ester (RME, biodiesel), natural gas derived synthetic fuel (GTL), and diesel fuel (DF) (SOF- soluble organic fractions) **Particle Size:** NR	**Route:** Cell Culture **Dose/Concentraion:** Log 2 dilutions of extracts: 1.0, 0.5, 0.25, 0.125 **Time to Analysis:** SOF extracted 12h. Plates incubated 48h.	Compared to DF, RSO significantly increased mutagenic effects of particle extracts (i.e., revertants) by 9.7-59 in TA98 and by 5.4-22.3 in TA100. (mRSO, RSO with lowered viscosity and fuel preheating in tank, produced highest number of revertant colonies in both strains ±S9.) RSO fuels condensates had 13.5 times stronger mutagenicity than DF. RME extracts had moderate but significantly higher mutagenic response in TA98 +S9 and TA100 –S9. Effects of GTL did not differ significantly from DF.
Reference: de Kok et al. (2005, 088656) **Species:** *S. typhimurium* **Strain:** TA98 (with and without rat liver S9) **Cell Line:** *Salmon testis* DNA	TSP (Total suspended particulate , Maastricht, The Netherlands; PM_{10} and $PM_{2.5}$ from 6 urban locations with different traffic intensities.) (organic extracts) **Particle Size:** NR	**Route:** Cell Culture **Dose/Concentration:** Mutagenicity assay: 2.5, 9, or 18m^3 sampled air in 100 μL DMSO; DNA adduct assay: 5 μL DMSO containing PM_{10} or TSP from equivalent 50m^3 sampled air. $PM_{2.5}$ concentration equivalent to 35m^3 sampled air. **Time to Analysis:** Mutagenicity assay: Cells incubated 1h with extracts. DNA adduct assay: DNA incubated 4h with extracts.	Overall, the direct mutagenicity and DNA reactivity of $PM_{2.5}$ extracts were higher compared to PM_{10} and TSP. S9 generally reduced mutagenic activity in TA98 but increased reactivity to *Salmon testis* DNA. Total PAH and total carcinogenic PAH levels correlated with the mutagenicity of TSP and the S9-mediated mutagenicity of $PM_{2.5}$. Neither transition metal composition nor radical generating capacity of PM correlated with mutagenic potential. Total PAH and carcinogenic PAH levels from PM_{10} and $PM_{2.5}$ correlated with direct and S9-mediated DNA adducts; for TSP these levels correlated with direct DNA reactivity only.

Reference	Pollutant	Exposure	Effects
Reference: DeMarini et al (2004, 066329) **Species:** *Salmonella* **Strain:** TA98, TA98NR, TA98/1, 8-DNP6, YG1021, YG1024, TA100	A-DEP and forklift DEP (SRM 2975) DEP (EOM) **Particle Size:** Mean Diameter: 0.4 μm	**Route:** Cell Culture **Dose/Concentration:** 0, 0.25, 0.5, 1.0, 2.0 EOM μg/plate **Time to Analysis:** DEPs sonicated 20min. Centrifuged 10min. Organic material extracted and concentrated. Ames assay. Incubated 3d.	A-DEPs were more mutagenic in both TA98 and TA100 than SRM 2975. There was 22x more PAH-related and 8–45x more nitroarene-related activity.
Reference: El Assouli et al. (2007, 186914) **Species:** *S. typhimuriam* **Strain:** TA98 (±S9)	PM (Jeddah, Saudi Arabia; 11 sites, urban, winter) (organic extracts) **Particle Size:** Diameter: 10 μm	**Route:** Cell Culture **Dose/Concentration:** 2.5, 50, 100 μg/plate; EOM range: 6-40 μg/m^3 **Time to Analysis:** 24h air samples, extracted. Refluxed 18-24h. GC-MS. Comet assay. 48h incubation. Ames assay.	PAHs varied from 0.83 to 0.18 ng/m^3. Only 2 locations of heavy petrol driven cars showed strong genotoxic responses. A correlation existed between DNA damage and the amount of pollutants and PAHs. Toxicity and mutagenicity occurred only in the presence of S9. Only 3 of the 11 sites exhibited moderate mutagenic activities.
Reference: Endo et al. (2003, 097260) **Species:** *S. typhimuriam* **Strain:** YG1024 (±S9)	PM (Tokyo, Japan; winter) (organic extracts) **Particle Size:** Diameter: $>12.1 - 0.06>$ μm; Bimodal mass concentration: 1-2 μm	**Route:** Cell Culture **Dose/Concentration:** 2.5, 5, 10 μL; 0.30 – 22.76 μg/m^3 **Time to Analysis:** Air samples collected, extracted. 90min pre-incubation. 48h incubation.	Mutagenicity tests showed dose-response relationships that were higher without S9 and increased with decreasing size.
Reference: Erdinger et al. (2005, 156423) **Species:** *S. typhimurium* **Strain:** TA98. TA100, TA98NR	PM (Baden-Württemberg, Germany; urban, 8 locations, glass fiber filters) (organic extracts) **Particle Size:** NR	**Route:** Cell Culture **Dose/Concentration:** 0.25, 2.5, 5, 12.5, 25 m^3/plate **Time to Analysis:** Standard Ames test protocol followed.	Extracts were mutagenic in all strains evaluated. No significant difference in response with or without metabolic activation. Activity in TA98NR suggests that the mutagenicity correlates with concentrations of air pollutants such as NOx.
Reference: Iba et al. (2006, 156582) **Species:** *S. typhimuriam* **Strain:** TA98, TA100 (±S9 (rat liver))	PM (wood smoke (WS) (New Jersey) and cigarette smoke (CS) (Tobacco Research and Health Institute, University of Kentucky)) (organic extracts) **Particle Size:** 10 μl aliquots of organic extracts	**Route:** Cell Culture **Dose/Concentration:** 62.5, 12.5 μg TPM equivalent/plate **Time to Analysis:** Incubation, shaking 25min. Agar added. 48h incubation. Rat lung explants incubated 18h. 12h incubation with treatments.	WS and CS were equally mutagenetic to TA98, but CS was 3-fold more mutagenetic to TA100 than WS. CS induced CYP1A1 in the explants, but WS did not.
Reference: Liu et al. (2005, 097019) **Species:** *S. typhimurium* **Strain:** YG1024, YG1029 **Cell Line:** Chinese hamster lung V79 cells	DEP extract (DP), gasoline engine exhaust particulate extract (GP), diesel exhaust SVOC extract (DSVOC), gasoline engine SVOC extract (GSVOC), NIST SRM 1650a **Particle Size:** Gasoline PM: 0.554mg extract (mg PM)$^{-1}$; Diesel PM: 0.363mg extract (mg PM)$^{-1}$	**Route:** Cell Culture **Dose/Concentration:** 1.48, 4.44, 13.3, 40, 120, 360, 1080 μg/plate **Time to Analysis:** 30min preincubation. 48h (YG1029). 66h (YG1024). Overnight preincubation 20h.	**Mutations:** All samples induced mutations in both strains. The increase was highly significant and dose-dependent. Response with S9 was generally greater than without S9. PM extract was more mutagenic than SVOC extract. **DP, GP, and GSVOC:** Dose-response was seen for DNA damage and micronuclei induction. GP, GSVOC and SRM 1650a were stronger inducers of micronuclei than DP.
Reference: Matsumoto et al. (2007, 187020) **Species:** *S. typhimuriam* **Strain:** TA98, TA100 (±S9)	APM (airborne particulate matter) APE (airborne particulate extracts) (Hokkaido, Japan; residential) **Particle Size:** NR	**Route:** Cell Culture **Dose/Concentration:** Crude APE: 979mg/m^3 air (CALUX BaP Equivalent (BaPEq)), 21mg/m^3 air (CALUX TCDD Equivalent (TCDDEq)); Cleaned APE: 7.87mg/m^3 air (CALUX BaPEq), 0.614mg/m^3 air (CALUX TCDDEq) **Time to Analysis:** Air samples collected, extracted. Preincubation with *S. typhimuriam*. 3, 24h exposure in CALUX assay. RNA extracted from mice 6d after last application.	Most of the CALUX BaPEq for crude APE was derived from PAH-like compounds, as suggested by the CALUX BaPEq of cleaned APE accounting for 0.80% of CALUX BaPEq for crude APE. CALUX TCDDEq showed TCDD and similar compounds to have a low contribution. The TA100 strain was more mutagenic to APE, with and without S9. S9 increased mutagenecity in both strains.

Reference	Pollutant	Exposure	Effects
Reference: Pastorkova et al. (2004, 087431) **Species:** *S. typhimuriam* **Strain:** TA98, YG1041 (±S9)	PM (EOM) (Plzeň, Prague, Ústí, Zďár – Czech Republic) **Particle Size:** Diameter: 10 μm	**Route:** Cell Culture **Dose/Concentration:** TA98 (4 doses): 20-200 μg/plate, YG1041 (4 doses): 4-20 μg/plate **Time to Analysis:** Collected 24h every 18th day, Oct-Mar, 1999-2003. Extracted. Ames assay. 70h incubation.	Significant dose-response effects in mutagenic potency of EOM occurred. Prague, one of the most polluted cities, had the highest mutagenicity values. Increasing time-trends were observed in the TA98 ± S9 mutagencity and PAH concentrations.
Reference: Rivedal et al. (2003, 097684) **Species:** *S. typhimurim* **Strain:** TA100, TA98, TA100NR, TA98NR, TA98/1,8-DNP6	DEP (SRM 1650)(organic extracts) (fractionated into PAH, nitro-PAH, dinitro-PAH, aliphatics, polar fraction) **Particle Size:** NR	**Route:** Cell Culture **Dose/Concentration:** Ames: 300, 600 DEP/plate; Gap junction: 100, 200 μg/mL DEP **Time to Analysis:** Extracted 16h. Fractionated. Ames assay. Gap junction intracellular communication: exposed 1-6h. Western blot.	TA100 was the most mutagenic without S9 activation. GJIC was dose- and time-dependently inhibited. The polar fraction was the most potent inhibitor. Nitro-PAH and dinitro-PAH were the most responsive fractions in the Ames assay.
Reference: Seagrave et al. (2003, 054979) **Species:** *Salmonella* **Strain:** TA98, TA100	Compressed natural gas (CNG) emissions (heavy-duty vehicles): High emitter (HE), Normal emitter (NE), New technology (NT) **Particle Size:** NR	**Route:** Cell Culture **Dose/Concentration:** PM (mg/mi)- NE- 7.0, NT- 5.0, HE- 406; Recovered PM (mg/mi)- NE-1.26, NT- 0.71, HE- 57.1; Recovered SVOC- NE- 58, NT- 26.4, HE- 227.5 **Time to Analysis:** Samples collected in filters 7x/d over several days. Recovered PM, recovered SVOC extracts combined. Ames assay.	All three CNG emissions were mutagenic in both strains. Mutagenicity was reduced by S9 in TA100 but not in TA98. Activity ranking in both strains was HE > NE > NT.
Reference: Sharma et al. (2007, 156975) **Species:** *S typhimurium* **Strain:** TA98, YG1041, YG5161 **Cell Line:** Human A549 lung epithelial cells	PM (airborne, 4 sites: an oven hall and receiving hall in a waste incineration plant; heavy-traffic street; background; Mar-June 2005) **Particle Size:** Diameter: 2.5 μm	**Route:** Cell Culture **Dose/Concentration:** 0.25mg/ml **Time to Analysis:** Samples taken over 7-16d. A549 cells incubated 24h. Comet and microsuspension assays performed.	**DNA damage:** Samples from all four sites induced DNA damage in the comet assay with the street samples more damaging than the oven hall sample. **Mutations:** Microsuspension assay was used to assess mutagenic activity. No mutagenic activity was observed for any of the non-polar fractions from any sample sites. The moderately polar fractions were all mutagenic, except for the oven hall sample, only when S9 was added. Comparatively, the polar and crude fractions were mutagenic without metabolic activation, suggesting a direct mutagenic effect.
Reference: Song et al. (2007, 155306) **Species:** *S. typhimurium* **Strain:** TA98, TA100 **Cell Line:** Rat fibrocytes L-929 cells	PM (soluble organic fraction (SOF) extracts from diesel engines using fuels blended with ethanol by volume: E0 - base diesel fuel; E5 - 5%; E10 - 10%; E15 - 15%; E20 - 20%) **Particle Size:** Density (g/cm3): E0- 0.8379; E5- 0.8349; E10- 0.8324; E15- 0.8301; E20- 0.8279	**Route:** Cell Culture **Dose/Concentration:** Ames Assay: 0.025, 0.05, 0.1mg/plate; Comet Assay: 0.125, 0.25, 0.5, 1.0mg/mL **Time to Analysis:** Samples extracted 24h. Ames and comet assays performed	All PM extracts induced higher mutational response in TA98 (3- to 5-fold increase over spontaneous) than in TA100 (2-to 3-fold increase). The highest brake specific revertants (BSR) ±S9 in both strains occurred with E20 and lowest BSR was in E5 (except in TA98 –S9). E0 and E20 caused more significant DNA damage (similar in effect) than the other extracts. Damage was dose-dependent but variable with increasing ethanol volume.
Reference: Zhang et al. (2007, 157186) **Species:** *S. typhimurium* **Strain:** TA98, TA100 **Cell Line:** Human lung adenocarcinoma A549 cells	Gasoline engine exhaust (GEE) Methanol engine exhaust (MEE) **Particle Size:** NR	**Route:** Cell Culture **Dose/Concentration:** MTT Assay- 0.05-0.8 GEE or MEE L/ml; MN Assay- 0.025, 0.05, 0.1, 0.2 GEE or MEE L/ml; Comet Assay- 0.025, 0.05, 0.1, 0.2, 0.4 GEE or MEE L/ml; Ames Assay- GEE: 0.625, 1.25, 2.5, 5.0, 10, 20 L/plate; MEE: 0.3125, 0.625, 1.25, 2.5, 5.0, 10, 20 L/plate **Time to Analysis:** Organic extracts from GEE and MEE. MTT assay- 24h incubation, followed by 2 or 24h incubation, followed by 4h incubation. MN assay- 24h incubation. Comet assay. Ames assay- 72h incubation.	**Mutagenicity:** GEE was mutagenic in TA98 but not TA100, –S9 at 10 and 20 L/plate and +S9 at ≥1.25 L/plate. Mutagenicity was higher with S9 than without at 0.625-10 L/plate and a dose-response was reported. MEE had no effect in either strain. **MN:** GEE significantly and dose-dependently induced MN. MEE had no significant effect at any dose. **DNA damage:** GEE significantly induced DNA damage at all doses compared to controls. MEE had no effect at any dose.

Reference	Pollutant	Exposure	Effects
Reference: Zhao et al. (2004, 100972) **Species:** Rat **Gender:** Male **Strain:** Sprague-Dawley **Age:** NR **Weight:** ~200g **Cell Line:** *S. typhimurium* YG1024 (±S9)	DEP (SRM 2975) DEPE (SRM 1975) Carbon black (CB) (Elftex-12 furnace black, Cabot, Boston, MA) **Particle Size:** NR	**Route:** IT Instilled. Cell Culture. **Dose/Concentration:** DEP or CB: 35mg/kg body weight; S9: 25, 50, 100, 200 μg/plate; Cytosolic protein: 20, 40, 80, 160 μg/plate; Microcosmal protein: 5, 10, 20, 40 μg/plate **Time to Analysis:** Rats instilled. Sacrificed 1, 3, 7d postexposure. S9, cytosolic, microcosmal fractions prepared from lung homogenates. Ames assay: 72h incubation.	DEP and CB-exposed lung S9 time-dependently decreased 2-aminoanthracene (2-AA) mutagenicity. Metyrapone and α-napthoflavone inhibited the S9-activation of 2-AA in DEP and CB exposed rats. Lung S9 increased the mutagenicity of DEPE but not of DEP or CB. Liver S9 reduced DEPE dose-dependently. CYP2B1 and CYP1A1 activated DEPE, with CYP2B1 being more effective.
Reference: Zhao et al. (2006, 100996) **Species:** *S. typhimuriam* **Strain:** YGL024 (±S9)	DEP (SRM 2975) DEPE (SRM 1975) Aminoguanidine (AG) **Particle Size:** NR	**Route:** Cell Culture **Dose/Concentration:** NR **Time to Analysis:** Lung S9 obtained from rats used in in vivo experiment. Ames test. Modified microsuspension assay. All assays in duplicate plates. Repeated 3x.	AG significantly lowered 2-aminoanthracene mutagenic activity of DEP or DEPE-exposed lung samples, with DEP being lowered the most.

Table D-8. Mutagenicity and genotoxicity data summary: in vitro studies.

Reference	Particle	Exposure	Effects
Reference: Abou Chakra et al. (2007, 098819) **Species:** Human **Gender:** Male, Female **Age:** 6-13yrs and Adults **Participant Characteristics:** Non-smokers **Cell Line:** HeLa S3 cells	PM (3 French metropolitan cities: Urban $PM_{2.5}$ and PM_{10} from "Residential Sector," "Proximity Sector," "Industrial Sector") (organic extracts) **Particle Size:** Diameter: 2.5, 10 μm	**Route:** Cell Culture **Dose/Concentration:** 261 $PM_{2.5}$; 76 PM_{10} samples **Time to Analysis:** Cells incubated with 200; μL organic extract and 20 μL aphidicoline for 24h.	Seasonal variation was observed with genotoxic effects being greater in winter. $PM_{2.5}$ was more active than PM_{10} extracts. Samples from the "Proximity Sector" (downtown area with heavy traffic) exhibited the strongest genotoxic responses.
Reference: Arrieta et al. (2003, 096210) **Species:** Rat **Cell Line:** Hepatoma (H4IIE) **Species:** Mouse **Cell Line:** Hepatoma H1I1.1c2	PM (El Paso, Texas; Juarez, Chihuahua, Mexico; Sunland Park, New Mexico) (organic extracts) **Particle Size:** Diameter: 10 μm	**Route:** Cell Culture **Dose/Concentration:** EROD test: 0.03, 0.17, 0.34, 0.50, 0.68, 4.96, 9.93 extract equivalents (m^3 air); Luciferase: 0.17, 0.51, 1.26, 5.01 extract equivalents (m^3 air) **Time to Analysis:** Extracts incubated 24h. EROD, luciferase activity, PAH content determined.	EROD activity declined at higher extract amounts, but luciferase activity was not inhibited. Cytotoxicity occurred only at extract equivalents to 0.47 m^3 air. PAH concentration increased with PM mass.
Reference: Bao et al. (2007, 097258) **Cell Line:** Human-hamster hybrid (A_L)	DEP (organic extracts) (SRM 2975) **Particle Size:** NR	**Route:** Cell Culture **Dose/Concentration:** 10, 20, 50, 100 μg/mL **Time to Analysis:** Phagocytosis inhibitors: Exposed 24h with or without cytochalasin B or ammonium chloride. Cytotoxicity: 24, 48h incubation. Mutations: Exposed 24h. 5-7d culture. Incubated additional 7-8d.	The nucleus of DEP-treated cells was condensed and shrunken compared to controls. DEPs accumulated in cells, disrupting the mitochondrial cristae, and were lodged in large cytoplasmic vacuoles. DEP produced minimal toxicity. CD59 locus mutations dose-dependently increased but decreased when simultaneously treated with cytochalasin B or ammonium chloride.
Reference: Carvalho-Oliveria et al. (2005, 077898) **Species:** *T. pallida*; *A. cepa*	PM (Sao Paulo, Brazil; spring, bus strike and non-strike days) (organic extracts) **Particle Size:** Diameter: 2.5 μm	**Route:** Cell Culture **Dose/Concentration:** Strike day: 47.32 $\mu g/m^3$; Non-strike day: 43.01 $\mu g/m^3$ **Time to Analysis:** Air samples from 2d: bus strike day, bus non-strike day. T. pallida kept in lab 24h. Exposed 8h. 24h recovery. Fixed 24h. *A. cepa* roots induced 5d. Exposed 30h. Fixed 24h.	Element concentrations, sulfur and BTEX decreased on the strike day. Micronuclei decreased *in T. pallida* during the strike. Toxicity measured in *A. cepa* was not significant, but higher on strike days.

Reference	Particle	Exposure	Effects
Reference: Dybdahl et al. (2004, 089013) **Species:** Human **Cell Line:** Lung epithelial A549	DEP (SRM 1650) **Particle Size:** NR	**Route:** Cell Culture **Dose/Concentration:** 10, 50, 100, 500 μg DEP/mL **Time to Analysis:** DEP suspended, sonicated. A549 cells diluted. Fresh medium added after 24h. After 48h medium removed, DEP added. 2, 5, 24h incubation.	DEP induced dose-dependent increases of IL-1α, IL-6, IL-8, TNF-α. The cytokines increased 4-18-fold at the highest dose. Cell viability did not decrease. Comet tail length increased at 100 and 500 μg/mL for 2, 5, 24h.
Reference: Gabelova et al. 2006 **Species:** Human **Cell Line:** Hepatoma Hep G2	PM (winter, summer) (organic extracts) **Particle Size:** Diameter: 10 μm	**Route:** Cell Culture **Dose/Concentration:** **5**, 10, 20, 50, 100, 150 μg/mL **Time to Analysis:** 3m sampling periods, winter, summer. Cells grown 48h. Exposed 2h. Single cell gel electrophoresis or cultivated, harvested, processed 2, 4, 16, 24h.	PM, c-PAHs and genotoxicity were higher in winter air samples than summer. EOM samples generally had significant dose-dependent increases in DNA migration. Repair-specific DNA endonucleases did not increase DNA migration. 8-oxodG was below the steady-state level in EOM samples.
Reference: Gabelova et al. 2007 **Species:** Human **Cell Line:** Hepatoma Hep G2	PM (PRG-SM, PRG-LB, Košice, Sofia; winter, summer) (organic extracts) **Particle Size:** Diameter: 10 μm	**Route:** Cell Culture **Dose/Concentration:** 5-150 μg/mL **Time to Analysis:** Air samples collected 24h intervals, 3m sampling period. Cells grown 48h. Exposed 2h. 24, 48h preliminary experiments. Single cell gel electrophoresis.	Cell viability significantly decreased in the 24, 48h exposure groups compared to the 2h exposure group. DNA migration significantly dose-dependently increased at most concentrations. In general, oxidative DNA damage did not significantly increase.
Reference: Gabelova et al. 2007 **Species:** Human **Cell Line:** Hepatoma Hep G2 cell line	PM_{10} (Prague (Czech Republic), Ko˘sice (Slovak Republic) and Sofia (Bulgaria); urban, winter, summer) (organic extracts) **Particle Size:** Diameter: 10 μm	**Route:** Cell Culture **Dose/Concentration:** 5 to 150 μg/ml EOM from 50 μg/ml stock solution **Time to Analysis:** 24h DNA adduct formation. 2h Comet assay. Oxidative DNA damage measured by Fpg-sensitive sites.	Total DNA adducts ranged from ~60 to 200 adducts per 10^8 nucleotides. Extracts also produced approximately the same levels of strand breaks. Results suggested that the genotoxic potential of ambient air was at least 6-fold greater in the winter compared to summer. No substantial difference was reported for oxidative DNA damage induced by summer vs. winter samples.
Reference: Gong et al. (2007, 091155) **Species:** Human **Cell Line:** Microvascular endothelial (HMEC)	DEP (aggregates, exhaust $4JB_1$-type LD,274 1,4-cylinder Isuzu diesel engine, 10 torque load, cyclone impactor, dilution tunnel constant volume sampler) **Particle Size:** Diameter: < 1 μm	**Route:** Cell Culture **Dose/Concentration:** 5, 15, 25 μg/mL **Time to Analysis:** Cells treated with DEP, ox-PAPC (oxidized 1-palmitoyl-2-arachidonyl-*sn*-glycero-3-phosphorylchlorine), DEP+ox-PAPC. Analytical tests performed.	HO-1 expression was dose-dependent and greatest with the DEP+ox-PAPC treatment. DEP significantly dose-dependently upregulated or downregulated a number of genes and was shown to have a synergistic effect with co-treatment of ox-PAPC. The most varying genes were significantly enriched for EpRE, inflammatory response, UPR, immune response, cell adhesion, lipid metabolism, apoptosis and protein folding genes.
Reference: Greenwell et al. (2003, 097478) **Species:** Rat **Cell Line:** Epithelial fluid; icosahedral bacteriophage ¤X174-RF DNA	PM (South Wales, UK) (urban, industrial) **Particle Size:** Coarse diameter: 10-2.5 μm, Fine diameter: 2.5-0.1 μm	**Route:** Cell Culture **Dose/Concentration:** Urban mean: 18.7 ± 4.7mg/day; Industrial mean: 22.6 ± 2.5mg/day **Time to Analysis:** 24h air samples 4-11d. Substrates vortexed 1h, suspended 4h, centrifuged 1h. Oxidation assay.	Industrial PM was more bioreactive than urban PM. Coarse fractions had greater oxidative potential and bioreactivity than fine fractions.
Reference: Gu et al. 2005 **Species:** Hamster **Strain:** Chinese **Cell Line:** Lung fibroblast (V79)	DPM (1980 model General Motors 5.7-L V-8 engine) **Particle Size:** NR	**Route:** Cell Culture **Dose/Concentration:** 25, 50, 100, 150 μg/mL; 10 μg DPM in 10 μg in DPPC/mL; 10 μg DPM in 10 μg DMSO/mL **Time to Analysis:** Chromosomal aberration: 24h incubation. Treated 24h. Incubated again 24h. MN assay: 24h treatment. Gene mutation: 24h treatment. Cells replated. 7d expression times. Staining at 8, 10d.	DPM significantly and dose-dependently increased aberrant cells at 25-100 μg/mL. DPM increased MN formation dose-dependently. Mutant frequencies were not significant and showed no dose-dependent trends. DPM was toxic to cells at the highest concentration.
Reference: Gualtieri et al. (2005, 097841) **Species:** Human **Cell Line:** Alveolar lung (A549)	TD (Tire debris, generated by rotating new vehicle wheel against a steel brush, significant component of PM_{10}) (organic extracts) **Particle Size:** Diameter: 10-80 μm	**Route:** Cell Culture **Dose/Concentration:** 50, 60, 75 μg/mL **Time to Analysis:** Particles extracted 6h. Cells subcultured every 3-4d. After 24h, TD treatments 24, 48, 72h.	A time- and dose-dependent inhibitory effect on the reduction of MTT was seen. Mortality increased dose-dependently and was significantly greater than the controls. DNA strand breaks increased significantly in a dose-dependent manner. A significant cell cycle block in the G1 phase with a consequent decrease in the cell number in the S and G2/M phases was seen. Exposed cells had a modified morphology.

Reference	Particle	Exposure	Effects
Reference: Gutierrez-Castillo et al. (2006, 089030) **Species:** Human **Cell Line:** A549 type II alveolar epithelial cells	$PM_{2.5}$ and PM_{10} (4 monitoring stations in Mexico City: (1) downtown high auto traffic, (2) two industrial areas with high levels of auto traffic and low vegetation, (3) medium-traffic residential area) (winter, spring , 4 sampling days in each period) (aqueous and organic extracts) **Particle Size:** Diameter: 2.5 or 10 μm	**Route:** Cell Culture **Dose/Concentration:** 0.05, 0.07, 0.1m^3/ml equivalents $PM_{2.5}$; 0.82, 1.25, 1.63m^3/ml equivalents PM_{10} **Time to Analysis:** Cells treated 48h with water-soluble or organic-soluble PM extracts.	Higher amounts of water-soluble metals were found in samples collected during winter. Water-soluble extracts increased DNA damage 1.7-fold over the background. Similar results were observed with organic extracts. In general, $PM_{2.5}$ extracts had greater genotoxic potential than PM_{10} extracts, and water soluble fractions form both particle sizes were more genotoxic than the corresponding organic extracts.
Reference: Izawa H et al. (2007, 190387) **Cell Line:** NA	DEPE (4JB-1 Isuzu 4-cylinder direct-injection 2740cc diesel engine; 1500rpm, 10kg/m load) **Particle Size:** NR	**Route:** Cell Culture **Dose/Concentration:** DEP: Ah-1 experiment- 111, 55.5, 27.8, 13.9, 6.9, 3.5, 1.7 μg/mL; Foods, polyphenols experiment- 27.8 μg/mL **Time to Analysis:** DEPE incubated 2h for dioxin toxicity measurement. Absorbance at 405nm measured. Food, polyphenol inhibitory effects: food extract or polyphenol solution added to cytosol solution, shaken 5min. DEPE added, shaken 5min. 2h incubation. Absorbance at 405nm measured.	The dioxin toxicity equivalent was 6,479 ± 58ng DEQ/g of DEP. The absorbance showed a sigmoid curve and dose-dependently increased from 6.9 to 27.8 μg DEP/mL. The *Ginkgo biloba* extract significantly inhibited AhR activation significantly more than the other foods, and was followed by green tea, onions, and garlic. Quercetin and myricetin dose-dependently inhibited AhR activation. Ginkgolides A and B had weak inhibitory effects and resveratol was the weakest.
Reference: Jacobsen et al. (2008, 156597) **Species:** Mouse **Cell Line:** FE1-Muta™ lung epithelial cells	DEP (SRM 1650b) Carbon black (CB) (Printex 90) **Particle Size:** DEP: 18-30nm; CB: 14nm; Agglomerates in suspensions: DEP Peaks- 249nm, CB Peaks-476nm	**Route:** Cell Culture **Dose/Concentration:** 37.5, 75 μg/mL **Time to Analysis:** 8 repeated 72h incubations.	**Mutagenicity:** The 75 μg/mL dose was significantly increased compared to the 37.5 μg/mL dose. Linear regression showed a significant increasing trend by increasing exposure. There was no change in the total cell numbers. **ROS:** ROS production increased in DEP-treated cells after 3h of exposure. CB-treated cells showed a dose-dependent increase.
Reference: Karlsson et al. 2004 **Species:** Human **Cell Line:** Fibroblasts; calf thymus DNA with human liver microsomes or rat liver S9	PM (urban dust particles, SRM 1649) (extracted with DCM, acetone, DMSO, water) (Fe 3% w/w, Ti 0.32% w/w, V 0.04% w/w, Mn 0.03% w/w, Cu 0.025% w/w) **Particle Size:** Mean diameter: < 10 μm	**Route:** Cell Culture **Dose/Concentration:** 0.1, 1.0, 10, 100 μg/cm^2 **Time to Analysis:** Particles extracted. Fibroblasts exposed 24h. Comet assay. Calf thymus incubated 2h with microsomes or S9. ^{32}P-labelled.	DNA damage increased dose-dependently, and a significant amount of DNA-damaged cells had particle interactions. DNA damage induced by the insoluble particle core significantly increased after each extraction. Native particles were more genotoxic than those extracted with DMSO, DCM and water, but not with acetone or hexane. DMSO extracts had the most adduct-forming PACs, and water extracts had the most oxidizing substances.
Reference: Karlsson et al. (2005, 086392) **Species:** Human **Cell Line:** Lung epithelial A549 type II	PM (subway station, urban street) Subway particles: O_2, Fe (Fe from Fe_3O_4) Street particles: Fe from Fe_2O_3 **Particle Size:** Diameter: 10 μm	**Route:** Cell Culture **Dose/Concentration:** Comet: 5, 10, 20, 40 μg/cm^2; 8-oxodG: 10 μg/cm^2 **Time to Analysis:** Air sampled 24h daily. Cells grown 24h. Exposed 4h.	Both PM types induced concentration-dependent DNA damage, but subway particles were more potent. Subway particles caused more 8-oxodG formation and oxidation of dG, the latter of which was inhibited by deferoxaminemesylate. Oxidation from subway particles was due to nonsoluble, redox active substances, and soluble substances from street particles.
Reference: Karlsson et al. (2006, 156625) **Species:** Human **Cell Line:** Lung epithelium A549; monocytes from heparinized whole blood	PM (wood- old, modern boiler; pellets- pellets burner, electrical ignition; tire-road simulator studded, friction tires; Street- busy street, Stockholm; Subway- platform near street) **Particle Size:** Diameter: 2.5, 10 μm	**Route:** Cell Culture **Dose/Concentration:** 40 μg/cm^2 **Time to Analysis:** Samples collected. Blank filter and Teflon filters used. Cells grown 24h. Comet assay. Monocytes incubated 10d. Macrophages incubated 18h.	All particles tested caused DNA damage, but there was no significant difference between the size fractions. Subway particles were the most genotoxic. The urban street particles were the most potent inducers of the cytokines. On the Teflon filters, PM_{10} was somewhat more potent than $PM_{2.5}$.
Reference: Kubátová et al. (2004, 087986) **Species:** Monkey **Cell Line:** African green kidney COS-1 (CV-1 cells with origin-defective SV40 mutants); (±S9)	PM (DE from diesel bus, wood smoke (WS) from chimney, hardwood smoke) (organic extracts) **Particle Size:** NR	**Route:** Cell Culture **Dose/Concentration:** 25, 50, 100, 200 μg/mL; 50mg of each material used for all experiments **Time to Analysis:** DE and WS collected. Extracted. 24h cytotoxicity. 2h SOS chromotest.	WS had significantly increased cytotoxicity in fractions of 25-250°C, and DE in nonpolar fractions of 250 and 300°C and polar fractions of 50°C. The cytotoxicity of DE PM nonpolar fractions corresponded to increased concentrations of PAHs. WS was not genotoxic and DE was genotoxic in midpolarity fractions (50-250°C). Genotoxic response was not increased after S9 activation.

Reference	Particle	Exposure	Effects
Reference: Landvik et al. (2007, 096722) **Species:** Mouse **Cell Line:** Hepatoma Hepa1c1c7 cells	DEP extracts (DEPE in the paper) **Particle Size:** NR	**Route:** Cell Culture **Dose/Concentration:** 10, 20, 30, 50, 70 μg/mL **Time to Analysis:** Cells exposed 24h. DNA fragmentation assay.	50 and 70 μg/mL DEPE did not induce DNA fragmentation but did cleave caspase 3 to a minor extent.
Reference: Mehta et al. (2008, 190440) **Species:** Human **Cell Line:** Lung adenocarcinoma (A549)	PM (SRM 1949a) **Particle Size:** Diameter: ¤ 0.18 μm	**Route:** Cell Culture **Dose/Concentration:** 0, 50, 100, 200, 400 μg/mL **Time to Analysis:** Cell culture and cell viability assay: PM treatment 24h. 10d incubation. Host cell reactivation assay: pGL3-luciferase plasmid UV irradiated 20min. PM treatment 24h. 16h transfection. 24h PM incubation. DNA repair synthesis assay: PM treatment 24h. Proteinase K treatment 30min. supf mutagenesis assay: PM treatment 24h. PM culture 60h. DNA extracted. Overnight incubation of transformed bacteria.	PM reduced colony-forming ability and repair synthesis capacity was proportional to the PM concentration. PM dose-dependently decreased HCR capacity and decreased more than TSP. PM induced cyclobutane dimmers and pyrimidine < 6-4 > pyrimidones mutations in UV-irradiated supf.
Reference: Meng et al. 2007 **Species:** Rat **Gender:** Male **Strain:** Wistar **Age:** NR **Weight:** Mean: 230g; Range: 200-250g **Cell Line:** AMs from treated rats	PM (Baotou, Wuwei, China) (normal weather, dust storms, Mar 1-31) (organic extracts, water soluble fractions) **Particle Size:** 2.5 μm	**Route:** Cell Culture **Dose/Concentration:** AM: 0, 33.3, 100, 300 μg/mL; Water-soluble: 0, 75, 150, 300 μg/mL; Organic extracts: 0, 25, 50, 100 μg/mL; Mass concentration normal day: 68.49 ± 28.83 μg/m^3; Mass concentration dust storm day: 221.83 ± 69.89 μg/m^3 **Time to Analysis:** Samples collected 24h after 5pm. Extracted. Rats instilled 24h. Killed, lavaged. Cultures 4h.	OC, NH_4^+, NO_3^- were higher in normal weather $PM_{2.5}$. SO_4^{2-}, Ca^{2+} were higher in dust storm $PM_{2.5}$. Fe, Al, Ca, Mg were 5x higher in dust storm $PM_{2.5}$. Cell viability reduced in a concentration-dependent manner, with normal weather being slightly more cytotoxic. DNA damage was dose-dependently induced, with normal weather and organic extracts showing the greatest damage.
Reference: Motta et al. 2004 **Species:** Hamster **Strain:** Chinese **Cell Line:** Epithelial liver, ovary	PM (Catania, Sicily; spring) (organic extracts) **Particle Size:** NR	**Route:** Cell Culture **Dose/Concentration:** 0.60, 1.21, 2.42, 4.85, 9.70, 19.40 μg/mL; 0.78, 1.56, 2.12, 6.25, 12.50, 25.00 μg/mL **Time to Analysis:** 2, 3h air sampling. Extracts 4h. 24h treatment.	The treatment was only slightly cytotoxic at the highest dose. DNA damage and aberrant cells generally increased with dose. No effect was seen in the Chinese hamster ovary cells without metabolic activation.
Reference: Oh and Chung (2006, 088296) **Cell Line:** A549 (Comet), CHO-K1 (CBMN), H4IIE (EROD-microbiassay)	Crude extract (CE) DEP and fractions of CE of DEP (organic extracts: F1 - organic bases, F2 - organic acids, F3 – aliphatic, F4 – aromatic, F5 - slightly polar, F6 --moderately polar, F7 - high polar) **Particle Size:** Diameter: < 2.5 μm, 87.71%, 2.5-10 μm, 3.87%, > 10 μm, 8.42%	**Route:** Cell Culture **Dose/Concentration:** 100 μg/mL **Time to Analysis:** DEP generated, extracted. Comet assay- 24h incubation, CE, DEP exposed 24h. MN assay- cultured 24h, 4h treatment, growth medium incubation 20h. EROD-microbioassay- 48h.	**DNA damage:** CE significantly increased the amount of DNA damage in A549 cells with and without SKF-525A, a CYP450 inhibitor, and in CHO-K1 cells. It significantly increased MN formation ±S9 compared to controls. **Organic Extracts:** Organic base (F1) and neutral (F3-F7) fractions of CE of DEP significantly induced DNA damage without SKF-525A compared to controls. Adding SKF-525A completely inhibited damage caused by F3, F4, F6 and F7 but kept the effect of F1 similar to that without SKF and only partially inhibited that of F5. F2 did not induce DNA damage with or without SKF. All fractions except F6 induced MN formation ±S9.
Reference: Poma et al. (2006, 096903) **Species:** Mouse **Cell Line:** Macrophage RAW 264.7	PM (L'Aquila, Italy; urban) Carbon black (CB) **Particle Size:** Diameter: 2.1-0.43 μm	**Route:** Cell Culture **Dose/Concentration:** 1, 3, 10 μg/cm^2 **Time to Analysis:** Air samples collected weekly basis Jan-Mar 2004. Cells cultured 48h. Treatment 48h. MN assay: 44h incubation, 28h incubation.	PM and CB dose-dependently reduced cell proliferation and induced micronuclei. PM and CB also reduced cellular metabolism of the macrophages and induced significant amounts of apoptosis. PM produced more micronuclei than equally-weighted CB.
Reference: Poma et al. 2006 **Species:** Mouse **Cell Line:** RAW 264.7 macrophage	PM (urban, winter) **Particle Size:** Diameter: 2.5 μm	**Route:** Cell Culture **Dose/Concentration:** 2.2, 6.6, 22 μg/mL **Time to Analysis:** Cells treated with particulates 44h. 28h incubation after cytochalasin B. Micronuclei frequency determined.	Extracts produced a dose-dependent increase in micronuclei. Fine carbon black particles were consistently less genotoxic at similar test concentrations. Results indicate that the chemicals adsorbed onto the particles were the primary contributors to the observed genotoxic effects.

Reference	Particle	Exposure	Effects
Reference: Roubicek et al. (2007, 156929) **Species:** Human **Cell Line:** Lung adenocarcinomaA549 cell line	PM (Mexico City from an industrial area with high–traffic and a medium-traffic residential area) (aqueous or organic extracts) **Particle Size:** Diameter: 10 μm	**Route:** Cell Culture **Dose/Concentration:** 1.25, 1.63, 2.5m^3/ml equivalents of PM_{10} **Time to Analysis:** Cells treated 24h followed by 48h incubation with cytochalasin B. Micronuclei frequency determined.	Water and organic extracts induced a significant dose-dependent increase in the micronuclei frequency. After doses of PM from different regions were normalized for mass differences, the genotoxic potency was higher for samples from the industrial area.
Reference: Salonen et al. (2004, 187053) **Species:** Mouse **Cell Line:** Macrophage RAW 264.7	PM (Vallila, Finland; busy traffic site; sping, winter) **Particle Size:** Diameter: $<$ 10 μm	**Route:** Cell Culture **Dose/Concentration:** 15, 50, 150, 500, 1000 μg/mL of RPMI **Time to Analysis:** Air collected 2-7d periods. Extracted. Exposed 24h.	PAHs decreased from winter to spring. TNF-α dose-dependently increased and was higher in spring samples. IL-6 generally increased in spring but not in winter. NO dose-dependently increased and was higher in winter. Cell viability generally decreased but there were no consistent potency differences between the samples. Generally, proinflammatory activity, cytotoxicity and IL-6 were associated with the insoluble PM fractions. Polymyxin B inhibited IL-6 and TNF-α. ·OH and 8-hydroxy-2′-deoxyguanosine dose-dependently increased and were higher in the spring and winter, respectively.
Reference: Seaton et al. 2005 **Species:** Human **Cell Line:** Alveolar epithelial A549	PM (3 busy London underground (LU) stations and cabs) (LU dust in $PM_{2.5}$ samples: iron oxide 64-71%, chromium 0.1-0.2%, manganese 0.5-1%, copper $<$ 0.1-0.9%; respirable dust samples: 1-2%) **Particle Size:** Diameter: $<$ 2.5 μm, 10 μm, Median diameter: 0.4 μm	**Route:** Cell Culture **Dose/Concentration:** Stations: 270-480 $\mu g/m^3$ $PM_{2.5}$, 14000-29000 particles/cm^3; Cabs: 130-200 $\mu g/m^3$ $PM_{2.5}$, 17000-23000 particles/cm^3; Assays: 1, 10, 50, 100 μg/mL **Time to Analysis:** 3d measurements of LU and cabs. Air collected for lab tests. Exposed 8, 24h.	PM_{10} caused less LDH release, IL-8 stimulation and free radical activity than LU dust particles that contained $PM_{2.5}$. Chelation had little effect on PM_{10} soluble components.
Reference: Sevastyanova et al. (2007, 156969) **Species:** Human **Cell Line:** HepG2 cell line, embryonic lung diploid fibroblasts (HEL), or acute monocytic leukemia cells (THP-1)	PM_{10} (Prague, Czech Republic; Ko˘sice; Slovak Republic; Sofia, Bulgaria) (urban, summer, winter) (organic extracts) **Particle Size:** Diameter: 10 μm	**Route:** Cell Culture **Dose/Concentration:** 10-100 μg/ml **Time to Analysis:** Samples collected 24h daily 3m. HepG2 and THP-1 cells treated for 24h.	DNA adducts were observed in all cell types evaluated. Highest adduct levels were observed in HepG2 cells, followed by HEL and THP-1 cells. A correlation between DNA adduct levels and carcinogenic PAH content was observed in HepG2 cells at 50 μg/ml.
Reference: Shi et al. (2003, 088248) **Species:** Human **Cell Line:** Epithelial lung A549	PM (Düsseldorf, Germany, July-Dec.) **Particle Size:** Fine diameter: $<$ 2.5 μm; Coarse diameter: 10-2.5 μm	**Route:** Cell Culture **Dose/Concentration:** Fine: 0.57-2.49mg; Coarse: 0.66-1.89mg; Concentration: 0.57mg/mL **Time to Analysis:** Weekly samplings July-Dec 1999. Electron spin response 8-hydrodeoxyguanosine induction, measurement.	Coarse and fine particles generated ·OH, but coarse particles had significantly higher ·OH formation as well as 8-hydroxy-2′-deoxyguanosine formation. 8-hydroxy-2′-deoxyguanosine and ·OH had a significant correlation.
Reference: Skarek et al. (2007, 096814) **Species:** Rat **Cell Line:** Modified hepatoma H4IIE.luc; SOS: *E. coli* PQ37 (±S9)	PM (urban: Ústí nad Laben, Karviná; background: Červenohorské sedlo, Košetice – Czech Republic; July) (organic extracts, TSP) GP (gas phase) **Particle Size:** Diameter: $<$ 2.5 μm	**Route:** Cell Culture **Dose/Concentration:** SOS: 8, 4, 2, 1m^3 ml^{-1}; Dioxin: TSP+GP- 8, 1.33, 0.22, 0.04m^3 ml^{-1}, $PM_{2.5}$+GP: 4, 0.66, 0.11, 0.02m^3 ml^{-1} **Time to Analysis:** 24h samples July 2002. Extracted. SOS chromotest: 22h incubation. Dioxin toxicity test: 24h exposure.	The urban areas had a much greater level of carcinogenic PAHs and overall number of PAHs than the background areas. Significant genotoxic activity was only detected at TSP+GP without S9 from urban areas. $PM_{2.5}$+GP had lower dioxin activity at the urban areas, but similar levels of toxicity were seen for both treatments in the background areas.
Reference: Song et al. (2007, 155306) **Species:** *S. typhimurium* **Strain:** TA98, TA100 **Cell Line:** Rat fibrocytes L-929 cells	PM (soluble organic fraction (SOF) extracts from diesel engines using fuels blended with ethanol by volume: E0 - base diesel fuel; E5 - 5%; E10 - 10%; E15 - 15%; E20 - 20%) **Particle Size:** Density (g/cm^3): E0-0.8379; E5- 0.8349; E10- 0.8324; E15- 0.8301; E20- 0.8279	**Route:** Cell Culture **Dose/Concentration:** Ames Assay: 0.025, 0.05, 0.1mg/plate; Comet Assay: 0.125, 0.25, 0.5, 1.0mg/mL **Time to Analysis:** Samples extracted 24h. Ames and comet assays performed	All PM extracts induced higher mutational response in TA98 (3- to 5-fold increase over spontaneous) than in TA100 (2-to 3-fold increase). The highest brake specific revertants (BSR) ±S9 in both strains occurred with E20 and lowest BSR was in E5 (except in TA98 – S9). E0 and E20 caused more significant DNA damage (similar in effect) than the other extracts. Damage was dose-dependent but variable with increasing ethanol volume.

Reference	Particle	Exposure	Effects
Reference: Ueng et al. (2005, 097054) **Species:** Human **Cell Line:** Lung epithelium CL5 (cancerous), bronchial epithelial BEAS-2B (noncancerous), WI-38 normal lung fibroblast	MEP (Yamaha cabin motorcycle 2-strok 50-cc engine) **Particle Size:** NR	**Route:** Cell Culture **Dose/Concentration:** 1, 10, 100, 200 μg/mL **Time to Analysis:** Exhaust collected, extracted. cDNA microarray analysis. RT-PCR: 2h. ELISA: 12h incubation. Centrifuged 24h post-treatment. Bioactivity: 12h incubation. Centrifuged 24h post-treatment. Medium replaced 48h post-incubation. Fibroblasts determined 96h post-incubation. Time response studies: 3-48h treatment. Concentration response studies: 6h treatment.	**Drug metabolism array study:** MEP increased CYP1A1, CYP3A7 and UGT2B. **Cytokine array study:** MEP increased fibroblast growth factor (FGF)-6, FGF-9, IL-1α, IL-22 and vascular endothelial growth factor (VEGF)-D mRNA. **Oncogene, tumor suppressor, estrogen signaling pathway:** MEP increased fra-1, c-src, SHC, p21, COX7RP, and decreased p53 and Rb expression. **RT-PCR:** MEP increased CYP1A1, CYP1B1, IL-6, IL-11, IL-1α, FGF-6, FGF-9, VEGF-D, fra-1 and p21. **Concentration and time responses:** Concentration and time-dependent increases occurred for FGF-9, IL-1α, IL-6, IL-11, but decreased time-dependently after 6h exposure. **BEAS-2B Cells:** MEP had concentration-dependent increases on CYP1A1 and CYP1B1 but did not affect anything else. **Peroxide, MEP+NAC, WI-38 Cells:** MEP increased peroxide production. The MEP+NAC treatment reduced MEP-elevated levels of IL-1α, IL-6, FGF-9, VEGF-D to control levels. Fibroblasts increased in WI-38 cells.
Reference: Umbuzeiro et al. (2008, 190491) **Species:** *Salmonella typhimurium* **Strain:** TA98, YG1041 (+/- S9)	PM (urban; São Paulo, Brazil-Cerqueira César street station, Ibirapuera park station) (winter- June 17, 18; average temperature: 16ºC) (EOM) **Particle Size:** NR	**Route:** Cell Culture **Dose/Concentration:** Cerqueira César: UPM- 156 μg/m^3, EOM- 57.7mg/total UPM; Ibirapuera Park: UPM- 32 μg/m^3, EOM- 41.7mg/total UPM; Salmonella assay- 0.5, 1, 5, 10, 50, 100 UPM equiv/plate (μg) **Time to Analysis:** Tests performed 20d after collection date. Organic extraction 20h. PAH fractionation. Salmonella/microsome assay.	The TSP and EOM were similar for both sites. The PAH fraction had very low mutagenicity for the Cerqueira César sample in the YG1041 strain and no mutagenicity for the Ibirapuera sample. Nitro-PAH and oxy-PAH had similar mutagenetic activities from both samples. S9 decreased mutagenicity in nitro-PAH but was increased in oxy-PAH. DNA adduct levels were dose-dependent and not different between the two sites.
Reference: Upadhyay et al. (2003, 097370) **Species:** Human **Cell Line:** Alveolar epithelial A549	PM (Dusseldorf, Germany) (Particles contain carbon (19.70%), hydrogen(1.4%),nitrogen (< .05%), oxygen(14.12%), sulfur (2.09%), ash (63.24%)) (Ionizable metals concentrations (ppm): Co(103), Cu(48),Cr(104),Fe(14,521), Mn(21.3), Ni(1,519),Ti(131), V(2,767) **Particle Size:** NR	**Route:** Cell Culture **Dose/Concentration:** 1, 5, 25, 100 μg/cm^2; 10, 25, 50, 100 μg/cm^2 **Time to Analysis:** Air collected. Cells grown 24h. PM treatments 1, 4, 8, 12, 24h.	PM induced dose- and time-dependent reductions in ds-DNA due to the formation of DNA-SB. The soluble component caused higher DNA damage. Apoptosis and DNA fragmentation increased dose-dependently. □□m decreased dose-dependently in control cells, but not in cells with Bcl-xl overexpression. PM caused activation of caspase 9. Pretreatment with iron chelators or a free radical scavenger reduced PM-induced DNA-SB formation, DNA fragmentation, caspase 9 activation, and weakened □□m reductions.
Reference: Valavanidis et al. (2005, 096432) **Cell Line:** NR	PM (TSP: high volume pumps, Athens; DEP: 2.0L engine GM Astra; GEP: 1.6L passenger vehicle Ford; Wood smoke soot: domestic fireplace exhaust chimney; PM_{10}: high volume sampling system, Athens; $PM_{2.5}$: high volume cascade impactor (Anderson) system **Particle Size:** Diameter: > 10.2 - < 0.41 μm	**Route:** Incubation **Dose/Concentration:** 20, 40mg/5mL **Time to Analysis:** PM collected 30min-1h or 24h basis. Incubated with H_2O_2 and 2′-deoxyguanosine (dG). Stored 3-7d at -20°C. Fenton reaction. EPR analysis.	PM generated ·OH by a Fenton reaction, which is increased by the addition of EDTA but inhibited by deferoxamine. PM dose-dependently induced dG hydroxylation and 8-hydroxy-2′-deoxyguanosine formation. Transition metals Ni, V, Co, Cr that are capable of redox cycling electron producing ROS were found in the PM samples.
Reference: Xu and Zhang (2004, 097231) **Species:** Human **Cell Line:** Lung epithelial A549	PM (Taiyuan, Beijing; Nov-Feb) (Taiyuan: coal-fume pollution; Beijing: coal-fume and vehicle exhaust) **Particle Size:** Diameter: 2.5 μm	**Route:** Cell Culture **Dose/Concentration:** 5, 50, 200 μg/mL **Time to Analysis:** Air samples collected. Cells incubated 12-24h. Comet assay.	Taiyuan had a significantly higher daily $PM_{2.5}$ average than Beijing. It was shown that the smaller the particulate diameter, the higher the concentration of BaP and Pb. A dose- and time-response relationship was seen in DNA fragmentation.

Table D-9. Mutagenicity and genotoxicity data summary: in vivo studies.

Reference	Particle	Exposure	Effects
Reference: Abou Chakra et al. (2007, 098819) **Species:** Human **Gender:** Male, Female **Age:** 6-13yrs and Adults **Participant Characteristics:** Non-smokers **Cell Line:** HeLa S3 cells	PM (3 French metropolitan cities: Urban $PM_{2.5}$ and PM_{10} from "Residential Sector," "Proximity Sector," "Industrial Sector") (organic extracts) **Particle Size:** Diameter: 2.5, 10 μm	**Route:** Cell Culture **Dose/Concentration:** 261 $PM_{2.5}$; 76 PM_{10} samples **Time to Analysis:** Cells incubated with 200; μL organic extract and 20 μL aphidicoline for 24h.	Seasonal variation was observed with genotoxic effects being greater in winter. $PM_{2.5}$ was more active than PM_{10} extracts. Samples from the "Proximity Sector" (downtown area with heavy traffic) exhibited the strongest genotoxic responses.
Reference: Arrieta et al. (2003, 096210) **Species:** Rat **Cell Line:** Hepatoma (H4IIE) **Species:** Mouse **Cell Line:** Hepatoma H1I1.1c2	PM (El Paso, Texas; Juarez, Chihuahua, Mexico; Sunland Park, New Mexico) (organic extracts) **Particle Size:** Diameter: 10 μm	**Route:** Cell Culture **Dose/Concentration:** EROD test: 0.03, 0.17, 0.34, 0.50, 0.68, 4.96, 9.93 extract equivalents (m^3 air); Luciferase: 0.17, 0.51, 1.26, 5.01 extract equivalents (m^3 air) **Time to Analysis:** Extracts incubated 24h. EROD, luciferase activity, PAH content determined.	EROD activity declined at higher extract amounts, but luciferase activity was not inhibited. Cytotoxicity occurred only at extract equivalents to 0.47 m^3 air. PAH concentration increased with PM mass.
Reference: Bao et al. (2007, 097258) **Cell Line:** Human-hamster hybrid (A_L)	DEP (organic extracts) (SRM 2975) **Particle Size:** NR	**Route:** Cell Culture **Dose/Concentration:** 10, 20, 50, 100 μg/mL **Time to Analysis:** Phagocytosis inhibitors: Exposed 24h with or without cytochalasin B or ammonium chloride. Cytotoxicity: 24, 48h incubation. Mutations: Exposed 24h. 5-7d culture. Incubated additional 7-8d.	The nucleus of DEP-treated cells was condensed and shrunken compared to controls. DEPs accumulated in cells, disrupting the mitochondrial cristae, and were lodged in large cytoplasmic vacuoles. DEP produced minimal toxicity. CD59 locus mutations dose-dependently increased but decreased when simultaneously treated with cytochalasin B or ammonium chloride.
Reference: Carvalho-Oliveria et al. (2005, 077898) **Species:** *T. pallida*; *A. cepa*	PM (Sao Paulo, Brazil; spring, bus strike and non-strike days) (organic extracts) **Particle Size:** Diameter: 2.5 μm	**Route:** Cell Culture **Dose/Concentration:** Strike day: 47.32 $\mu g/m^3$; Non-strike day: 43.01 $\mu g/m^3$ **Time to Analysis:** Air samples from 2d: bus strike day, bus non-strike day. T. pallida kept in lab 24h. Exposed 8h. 24h recovery. Fixed 24h. *A. cepa* roots induced 5d. Exposed 30h. Fixed 24h.	Element concentrations, sulfur and BTEX decreased on the strike day. Micronuclei decreased *in T. pallida* during the strike. Toxicity measured in *A. cepa* was not significant, but higher on strike days.
Reference: Dybdahl et al. (2004, 089013) **Species:** Human **Cell Line:** Lung epithelial A549	DEP (SRM 1650) **Particle Size:** NR	**Route:** Cell Culture **Dose/Concentration:** 10, 50, 100, 500 μg DEP/mL **Time to Analysis:** DEP suspended, sonicated. A549 cells diluted. Fresh medium added after 24h. After 48h medium removed, DEP added. 2, 5, 24h incubation.	DEP induced dose-dependent increases of IL-1α, IL-6, IL-8, TNF-α. The cytokines increased 4-18-fold at the highest dose. Cell viability did not decrease. Comet tail length increased at 100 and 500 μg/mL for 2, 5, 24h.
Reference: Gabelova et al. 2006 **Species:** Human **Cell Line:** Hepatoma Hep G2	PM (winter, summer) (organic extracts) **Particle Size:** Diameter: 10 μm	**Route:** Cell Culture **Dose/Concentration: 5**, 10, 20, 50, 100, 150 μg/mL **Time to Analysis:** 3m sampling periods, winter, summer. Cells grown 48h. Exposed 2h. Single cell gel electrophoresis or cultivated, harvested, processed 2, 4, 16, 24h.	PM, c-PAHs and genotoxicity were higher in winter air samples than summer. EOM samples generally had significant dose-dependent increases in DNA migration. Repair-specific DNA endonucleases did not increase DNA migration. 8-oxodG was below the steady-state level in EOM samples.
Reference: Gabelova et al. 2007 **Species:** Human **Cell Line:** Hepatoma Hep G2	PM (PRG-SM, PRG-LB, Košice, Sofia; winter, summer) (organic extracts) **Particle Size:** Diameter: 10 μm	**Route:** Cell Culture **Dose/Concentration:** 5-150 μg/mL **Time to Analysis:** Air samples collected 24h intervals, 3m sampling period. Cells grown 48h. Exposed 2h. 24, 48h preliminary experiments. Single cell gel electrophoresis.	Cell viability significantly decreased in the 24, 48h exposure groups compared to the 2h exposure group. DNA migration significantly dose-dependently increased at most concentrations. In general, oxidative DNA damage did not significantly increase.

Reference	Particle	Exposure	Effects
Reference: Gabelova et al. 2007 **Species:** Human **Cell Line:** Hepatoma Hep G2 cell line	PM_{10} (Prague (Czech Republic), Ko˘sice (Slovak Republic) and Sofia (Bulgaria); urban, winter, summer) (organic extracts) **Particle Size:** Diameter: 10 μm	**Route:** Cell Culture **Dose/Concentration:** 5 to 150 μg/ml EOM from 50 μg/ml stock solution **Time to Analysis:** 24h DNA adduct formation. 2h Comet assay. Oxidative DNA damage measured by Fpg-sensitive sites.	Total DNA adducts ranged from ~60 to 200 adducts per 10^8 nucleotides. Extracts also produced approximately the same levels of strand breaks. Results suggested that the genotoxic potential of ambient air was at least 6-fold greater in the winter compared to summer. No substantial difference was reported for oxidative DNA damage induced by summer vs. winter samples.
Reference: Gong et al. (2007, 091155) **Species:** Human **Cell Line:** Microvascular endothelial (HMEC)	DEP (aggregates, exhaust 4JB_1-type LD,274 1,4-cylinder Isuzu diesel engine, 10 torque load, cyclone impactor, dilution tunnel constant volume sampler) **Particle Size:** Diameter: < 1 μm	**Route:** Cell Culture **Dose/Concentration:** 5, 15, 25 μg/mL **Time to Analysis:** Cells treated with DEP, ox-PAPC (oxidized 1-palmitoyl-2-arachidonyl-*sn*-glycero-3-phosphorylchlorine), DEP+ox-PAPC. Analytical tests performed.	HO-1 expression was dose-dependent and greatest with the DEP+ox-PAPC treatment. DEP significantly dose-dependently upregulated or downregulated a number of genes and was shown to have a synergistic effect with co-treatment of ox-PAPC. The most varying genes were significantly enriched for EpRE, inflammatory response, UPR, immune response, cell adhesion, lipid metabolism, apoptosis and protein folding genes.
Reference: Greenwell et al. (2003, 097478) **Species:** Rat **Cell Line:** Epithelial fluid; icosahedral bacteriophage φX174-RF DNA	PM (South Wales, UK) (urban, industrial) **Particle Size:** Coarse diameter: 10-2.5 μm, Fine diameter: 2.5-0.1 μm	**Route:** Cell Culture **Dose/Concentration:** Urban mean: 18.7 ± 4.7mg/day; Industrial mean: 22.6 ± 2.5mg/day **Time to Analysis:** 24h air samples 4-11d. Substrates vortexed 1h, suspended 4h, centrifuged 1h. Oxidation assay.	Industrial PM was more bioreactive than urban PM. Coarse fractions had greater oxidative potential and bioreactivity than fine fractions.
Reference: Gu et al. 2005 **Species:** Hamster **Strain:** Chinese **Cell Line:** Lung fibroblast (V79)	DPM (1980 model General Motors 5.7-L V-8 engine) **Particle Size:** NR	**Route:** Cell Culture **Dose/Concentration:** 25, 50, 100, 150 μg/mL; 10 μg DPM in 10 μg in DPPC/mL; 10 μg DPM in 10 μg DMSO/mL **Time to Analysis:** Chromosomal aberration: 24h incubation. Treated 24h. Incubated again 24h. MN assay: 24h treatment. Gene mutation: 24h treatment. Cells replated. 7d expression times. Staining at 8, 10d.	DPM significantly and dose-dependently increased aberrant cells at 25-100 μg/mL. DPM increased MN formation dose-dependently. Mutant frequencies were not significant and showed no dose-dependent trends. DPM was toxic to cells at the highest concentration.
Reference: Gualtieri et al. (2005, 097841) **Species:** Human **Cell Line:** Alveolar lung (A549)	TD (Tire debris, generated by rotating new vehicle wheel against a steel brush, significant component of PM_{10}) (organic extracts) **Particle Size:** Diameter: 10-80 μm	**Route:** Cell Culture **Dose/Concentration:** 50, 60, 75 μg/mL **Time to Analysis:** Particles extracted 6h. Cells subcultured every 3-4d. After 24h, TD treatments 24, 48, 72h.	A time- and dose-dependent inhibitory effect on the reduction of MTT was seen. Mortality increased dose-dependently and was significantly greater than the controls. DNA strand breaks increased significantly in a dose-dependent manner. A significant cell cycle block in the G1 phase with a consequent decrease in the cell number in the S and G2/M phases was seen. Exposed cells had a modified morphology.
Reference: Gutierrez-Castillo et al. (2006, 089030) **Species:** Human **Cell Line:** A549 type II alveolar epithelial cells	$PM_{2.5}$ and PM_{10} (4 monitoring stations in Mexico City: (1) downtown high auto traffic, (2) two industrial areas with high levels of auto traffic and low vegetation, (3) medium-traffic residential area) (winter, spring , 4 sampling days in each period) (aqueous and organic extracts) **Particle Size:** Diameter: 2.5 or 10 μm	**Route:** Cell Culture **Dose/Concentration:** 0.05, 0.07, 0.1m^3/ml equivalents $PM_{2.5}$; 0.82, 1.25, 1.63m^3/ml equivalents PM_{10} **Time to Analysis:** Cells treated 48h with water-soluble or organic-soluble PM extracts.	Higher amounts of water-soluble metals were found in samples collected during winter. Water-soluble extracts increased DNA damage 1.7-fold over the background. Similar results were observed with organic extracts. In general, $PM_{2.5}$ extracts had greater genotoxic potential than PM_{10} extracts, and water soluble fractions form both particle sizes were more genotoxic than the corresponding organic extracts.
Reference: Izawa H et al. (2007, 190387) **Cell Line:** NA	DEPE (4JB-1 Isuzu 4-cylinder direct-injection 2740cc diesel engine; 1500rpm, 10kg/m load) **Particle Size:** NR	**Route:** Cell Culture **Dose/Concentration:** DEP: Ah-1 experiment- 111, 55.5, 27.8, 13.9, 6.9, 3.5, 1.7 μg/mL; Foods, polyphenols experiment- 27.8 μg/mL **Time to Analysis:** DEPE incubated 2h for dioxin toxicity measurement. Absorbance at 405nm measured. Food, polyphenol inhibitory effects: food extract or polyphenol solution added to cytosol solution, shaken 5min. DEPE added, shaken 5min. 2h incubation. Absorbance at 405nm measured.	The dioxin toxicity equivalent was 6,479±58ng DEQ/g of DEP. The absorbance showed a sigmoid curve and dose-dependently increased from 6.9 to 27.8 μg DEP/mL. The *Ginkgo biloba* extract significantly inhibited AhR activation significantly more than the other foods, and was followed by green tea, onions, and garlic. Quercetin and myricetin dose-dependently inhibited AhR activation. Ginkgolides A and B had weak inhibitory effects and resveratol was the weakest.

Reference	Particle	Exposure	Effects
Reference: Jacobsen et al. (2008, 156597) **Species:** Mouse **Cell Line:** FE1-Muta™ lung epithelial cells	DEP (SRM 1650b) Carbon black (CB) (Printex 90) **Particle Size:** DEP: 18-30nm; CB: 14nm; Agglomerates in suspensions: DEP Peaks- 249nm, CB Peaks- 476nm	**Route:** Cell Culture **Dose/Concentration:** 37.5, 75 μg/mL **Time to Analysis:** 8 repeated 72h incubations.	**Mutagenicity:** The 75 μg/mL dose was significantly increased compared to the 37.5 μg/mL dose. Linear regression showed a significant increasing trend by increasing exposure. There was no change in the total cell numbers. **ROS:** ROS production increased in DEP-treated cells after 3h of exposure. CB-treated cells showed a dose-dependent increase.
Reference: Karlsson et al. 2004 **Species:** Human **Cell Line:** Fibroblasts; calf thymus DNA with human liver microsomes or rat liver S9	PM (urban dust particles, SRM 1649) (extracted with DCM, acetone, DMSO, water) (Fe 3% w/w, Ti 0.32% w/w, V 0.04% w/w, Mn 0.03% w/w, Cu 0.025% w/w) **Particle Size:** Mean diameter: < 10 μm	**Route:** Cell Culture **Dose/Concentration:** 0.1, 1.0, 10, 100 μg/cm^2 **Time to Analysis:** Particles extracted. Fibroblasts exposed 24h. Comet assay. Calf thymus incubated 2h with microsomes or S9. ^{32}P-labelled.	DNA damage increased dose-dependently, and a significant amount of DNA-damaged cells had particle interactions. DNA damage induced by the insoluble particle core significantly increased after each extraction. Native particles were more genotoxic than those extracted with DMSO, DCM and water, but not with acetone or hexane. DMSO extracts had the most adduct-forming PACs, and water extracts had the most oxidizing substances.
Reference: Karlsson et al. (2005, 086392) **Species:** Human **Cell Line:** Lung epithelial A549 type II	PM (subway station, urban street) Subway particles: O_2, Fe (Fe from Fe_3O_4) Street particles: Fe from Fe_2O_3 **Particle Size:** Diameter: 10 μm	**Route:** Cell Culture **Dose/Concentration:** Comet: 5, 10, 20, 40 μg/cm^2; 8-oxodG: 10 μg/cm^2 **Time to Analysis:** Air sampled 24h daily. Cells grown 24h. Exposed 4h.	Both PM types induced concentration-dependent DNA damage, but subway particles were more potent. Subway particles caused more 8-oxodG formation and oxidation of dG, the latter of which was inhibited by deferoxaminemesylate. Oxidation from subway particles was due to nonsoluble, redox active substances, and soluble substances from street particles.
Reference: Karlsson et al. (2006, 156625) **Species:** Human **Cell Line:** Lung epithelium A549; monocytes from heparinized whole blood	PM (wood- old, modern boiler; pellets-pellets burner, electrical ignition; tire-road simulator studded, friction tires; Street- busy street, Stockholm; Subway- platform near street) **Particle Size:** Diameter: 2.5, 10 μm	**Route:** Cell Culture **Dose/Concentration:** 40 μg/cm^2 **Time to Analysis:** Samples collected. Blank filter and Teflon filters used. Cells grown 24h. Comet assay. Monocytes incubated 10d. Macrophages incubated 18h.	All particles tested caused DNA damage, but there was no significant difference between the size fractions. Subway particles were the most genotoxic. The urban street particles were the most potent inducers of the cytokines. On the Teflon filters, PM_{10} was somewhat more potent than $PM_{2.5}$.
Reference: Kubátová et al. (2004, 087986) **Species:** Monkey **Cell Line:** African green kidney COS-1 (CV-1 cells with origin-defective SV40 mutants); (±S9)	PM (DE from diesel bus, wood smoke (WS) from chimney, hardwood smoke) (organic extracts) **Particle Size:** NR	**Route:** Cell Culture **Dose/Concentration:** 25, 50, 100, 200 μg/mL; 50mg of each material used for all experiments **Time to Analysis:** DE and WS collected. Extracted. 24h cytotoxicity. 2h SOS chromotest.	WS had significantly increased cytotoxicity in fractions of 25-250°C, and DE in nonpolar fractions of 250 and 300°C and polar fractions of 50°C. The cytotoxicity of DE PM nonpolar fractions corresponded to increased concentrations of PAHs. WS was not genotoxic and DE was genotoxic in midpolarity fractions (50-250°C). Genotoxic response was not increased after S9 activation.
Reference: Landvik et al. (2007, 096722) **Species:** Mouse **Cell Line:** Hepatoma Hepa1c1c7 cells	DEP extracts (DEPE in the paper) **Particle Size:** NR	**Route:** Cell Culture **Dose/Concentration:** 10, 20, 30, 50, 70 μg/mL **Time to Analysis:** Cells exposed 24h. DNA fragmentation assay.	50 and 70 μg/mL DEPE did not induce DNA fragmentation but did cleave caspase 3 to a minor extent.
Reference: Mehta et al. (2008, 190440) **Species:** Human **Cell Line:** Lung adenocarcinoma (A549)	PM (SRM 1949a) **Particle Size:** Diameter: ◘ 0.18 μm	**Route:** Cell Culture **Dose/Concentration:** 0, 50, 100, 200, 400 μg/mL **Time to Analysis:** Cell culture and cell viability assay: PM treatment 24h. 10d incubation. Host cell reactivation assay: pGL3-luciferase plasmid UV irradiated 20min. PM treatment 24h. 16h transfection. 24h PM incubation. DNA repair synthesis assay: PM treatment 24h. Proteinase K treatment 30min. supf mutagenesis assay: PM treatment 24h. PM culture 60h. DNA extracted. Overnight incubation of transformed bacteria.	PM reduced colony-forming ability and repair synthesis capacity was proportional to the PM concentration. PM dose-dependently decreased HCR capacity and decreased more than TSP. PM induced cyclobutane dimmers and pyrimidine $<$ 6-4 $>$ pyrimidones mutations in UV-irradiated supf.

Reference	Particle	Exposure	Effects
Reference: Meng et al. 2007 **Species:** Rat **Gender:** Male **Strain:** Wistar **Age:** NR **Weight:** Mean: 230g; Range: 200-250g **Cell Line:** AMs from treated rats	PM (Baotou, Wuwei, China) (normal weather, dust storms, Mar 1-31) (organic extracts, water soluble fractions) **Particle Size:** 2.5 μm	**Route:** Cell Culture **Dose/Concentration:** AM: 0, 33.3, 100, 300 μg/mL; Water-soluble: 0, 75, 150, 300 μg/mL; Organic extracts: 0, 25, 50, 100 μg/mL; Mass concentration normal day: 68.49 ± 28.83 μg/m^3; Mass concentration dust storm day: 221.83 ± 69.89 μg/m^3 **Time to Analysis:** Samples collected 24h after 5pm. Extracted. Rats instilled 24h. Killed, lavaged. Cultures 4h.	OC, NH_4^+, NO_3^- were higher in normal weather $PM_{2.5}$. SO_4^{2-}, Ca^{2+} were higher in dust storm $PM_{2.5}$. Fe, Al, Ca, Mg were 5x higher in dust storm $PM_{2.5}$. Cell viability reduced in a concentration-dependent manner, with normal weather being slightly more cytotoxic. DNA damage was dose-dependently induced, with normal weather and organic extracts showing the greatest damage.
ference: Motta et al. 2004 **Species:** Hamster **Strain:** Chinese **Cell Line:** Epithelial liver, ovary	PM (Catania, Sicily; spring) (organic extracts) **Particle Size:** NR	**Route:** Cell Culture **Dose/Concentration:** 0.60, 1.21, 2.42, 4.85, 9.70, 19.40 μg/mL; 0.78, 1.56, 2.12, 6.25, 12.50, 25.00 μg/mL **Time to Analysis:** 2, 3h air sampling. Extracts 4h. 24h treatment.	The treatment was only slightly cytotoxic at the highest dose. DNA damage and aberrant cells generally increased with dose. No effect was seen in the Chinese hamster ovary cells without metabolic activation.
Reference: Oh and Chung (2006, 088296) **Cell Line:** A549 (Comet), CHO-K1 (CBMN), H4IIE (EROD-microbiassay)	Crude extract (CE) DEP and fractions of CE of DEP (organic extracts: F1 - organic bases, F2 - organic acids, F3 – aliphatic, F4 – aromatic, F5 - slightly polar, F6 --moderately polar, F7 - high polar) **Particle Size:** Diameter: < 2.5 μm, 87.71%, 2.5-10 μm, 3.87%, > 10 μm, 8.42%	**Route:** Cell Culture **Dose/Concentration:** 100 μg/mL **Time to Analysis:** DEP generated, extracted. Comet assay- 24h incubation, CE, DEP exposed 24h. MN assay- cultured 24h, 4h treatment, growth medium incubation 20h. EROD-microbioassay- 48h.	**DNA damage:** CE significantly increased the amount of DNA damage in A549 cells with and without SKF-525A, a CYP450 inhibitor, and in CHO-K1 cells. It significantly increased MN formation ±S9 compared to controls. **Organic Extracts:** Organic base (F1) and neutral (F3-F7) fractions of CE of DEP significantly induced DNA damage without SKF-525A compared to controls. Adding SKF-525A completely inhibited damage caused by F3, F4, F6 and F7 but kept the effect of F1 similar to that without SKF and only partially inhibited that of F5. F2 did not induce DNA damage with or without SKF. All fractions except F6 induced MN formation ±S9.
Reference: Poma et al. (2006, 096903) **Species:** Mouse **Cell Line:** Macrophage RAW 264.7	PM (L'Aquila, Italy; urban) Carbon black (CB) **Particle Size:** Diameter: 2.1-0.43 μm	**Route:** Cell Culture **Dose/Concentration:** 1, 3, 10 μg/cm^2 **Time to Analysis:** Air samples collected weekly basis Jan-Mar 2004. Cells cultured 48h. Treatment 48h. MN assay: 44h incubation, 28h incubation.	PM and CB dose-dependently reduced cell proliferation and induced micronuclei. PM and CB also reduced cellular metabolism of the macrophages and induced significant amounts of apoptosis. PM produced more micronuclei than equally-weighted CB.
Reference: Poma et al. 2006 **Species:** Mouse **Cell Line:** RAW 264.7 macrophage	PM (urban, winter) **Particle Size:** Diameter: 2.5 μm	**Route:** Cell Culture **Dose/Concentration:** 2.2, 6.6, 22 μg/mL **Time to Analysis:** Cells treated with particulates 44h. 28h incubation after cytochalasin B. Micronuclei frequency determined.	Extracts produced a dose-dependent increase in micronuclei. Fine carbon black particles were consistently less genotoxic at similar test concentrations. Results indicate that the chemicals adsorbed onto the particles were the primary contributors to the observed genotoxic effects.
Reference: Roubicek et al. (2007, 156929) **Species:** Human **Cell Line:** Lung adenocarcinomaA549 cell line	PM (Mexico City from an industrial area with high–traffic and a medium-traffic residential area) (aqueous or organic extracts) **Particle Size:** Diameter: 10 μm	**Route:** Cell Culture **Dose/Concentration:** 1.25, 1.63, 2.5m^3/ml equivalents of PM_{10} **Time to Analysis:** Cells treated 24h followed by 48h incubation with cytochalasin B. Micronuclei frequency determined.	Water and organic extracts induced a significant dose-dependent increase in the micronuclei frequency. After doses of PM from different regions were normalized for mass differences, the genotoxic potency was higher for samples from the industrial area.
Reference: Salonen et al. (2004, 187053) **Species:** Mouse **Cell Line:** Macrophage RAW 264.7	PM (Vallila, Finland; busy traffic site; sping, winter) **Particle Size:** Diameter: < 10 μm	**Route:** Cell Culture **Dose/Concentration:** 15, 50, 150, 500, 1000 μg/mL of RPMI **Time to Analysis:** Air collected 2-7d periods. Extracted. Exposed 24h.	PAHs decreased from winter to spring. TNF-α dose-dependently increased and was higher in spring samples. IL-6 generally increased in spring but not in winter. NO dose-dependently increased and was higher in winter. Cell viability generally decreased but there were no consistent potency differences between the samples. Generally, proinflammatory activity, cytotoxicity and IL-6 were associated with the insoluble PM fractions. Polymyxin B inhibited IL-6 and TNF-α. ·OH and 8-hydroxy-2'-deoxyguanosine dose-dependently increased and were higher in the spring and winter, respectively.

Reference	Particle	Exposure	Effects
Reference: Seaton et al. 2005 **Species:** Human **Cell Line:** Alveolar epithelial A549	PM (3 busy London underground (LU) stations and cabs) (LU dust in $PM_{2.5}$ samples: iron oxide 64-71%, chromium 0.1-0.2%, manganese 0.5-1%, copper < 0.1-0.9%; respirable dust samples: 1-2%) **Particle Size:** Diameter: < 2.5 μm, 10 μm, Median diameter: 0.4 μm	**Route:** Cell Culture **Dose/Concentration:** Stations: 270-480 $\mu g/m^3$ $PM_{2.5}$, 14000-29000 particles/cm^3; Cabs: 130-200 $\mu g/m^3$ $PM_{2.5}$, 17000-23000 particles/cm^3; Assays: 1, 10, 50, 100 μg/mL **Time to Analysis:** 3d measurements of LU and cabs. Air collected for lab tests. Exposed 8, 24h.	PM_{10} caused less LDH release, IL-8 stimulation and free radical activity than LU dust particles that contained $PM_{2.5}$. Chelation had little effect on PM_{10} soluble components.
Reference: Sevastyanova et al. (2007, 156969) **Species:** Human **Cell Line:** HepG2 cell line, embryonic lung diploid fibroblasts (HEL), or acute monocytic leukemia cells (THP-1)	PM_{10} (Prague, Czech Republic; Ko˘sice; Slovak Republic; Sofia, Bulgaria) (urban, summer, winter) (organic extracts) **Particle Size:** Diameter: 10 μm	**Route:** Cell Culture **Dose/Concentration:** 10-100 μg/ml **Time to Analysis:** Samples collected 24h daily 3m. HepG2 and THP-1 cells treated for 24h.	DNA adducts were observed in all cell types evaluated. Highest adduct levels were observed in HepG2 cells, followed by HEL and THP-1 cells. A correlation between DNA adduct levels and carcinogenic PAH content was observed in HepG2 cells at 50 μg/ml.
Reference: Shi et al. (2003, 088248) **Species:** Human **Cell Line:** Epithelial lung A549	PM (Düsseldorf, Germany, July-Dec.) **Particle Size:** Fine diameter: < 2.5 μm; Coarse diameter: 10-2.5 μm	**Route:** Cell Culture **Dose/Concentration:** Fine: 0.57-2.49mg; Coarse: 0.66-1.89mg; Concentration: 0.57mg/mL **Time to Analysis:** Weekly samplings July-Dec 1999. Electron spin response 8-hydrodeoxyguanosine induction, measurement.	Coarse and fine particles generated ·OH, but coarse particles had significantly higher ·OH formation as well as 8-hydroxy-2′-deoxyguanosine formation. 8-hydroxy-2′-deoxyguanosine and ·OH had a significant correlation.
Reference: Skarek et al. (2007, 096814) **Species:** Rat **Cell Line:** Modified hepatoma H4IIE.luc; SOS: *E. coli* PQ37 (±S9)	PM (urban: Ústí nad Laben, Karviná; background: Červenohorské sedlo, Košetice – Czech Republic; July) (organic extracts, TSP) GP (gas phase) **Particle Size:** Diameter: < 2.5 μm	**Route:** Cell Culture **Dose/Concentration:** SOS: 8, 4, 2, 1 m^3 ml^{-1}; Dioxin: TSP+GP- 8, 1.33, 0.22, 0.04 m^3 ml^{-1}, $PM_{2.5}$+GP: 4, 0.66, 0.11, 0.02 m^3 ml^{-1} **Time to Analysis:** 24h samples July 2002. Extracted. SOS chromotest: 22h incubation. Dioxin toxicity test: 24h exposure.	The urban areas had a much greater level of carcinogenic PAHs and overall number of PAHs than the background areas. Significant genotoxic activity was only detected at TSP+GP without S9 from urban areas. $PM_{2.5}$+GP had lower dioxin activity at the urban areas, but similar levels of toxicity were seen for both treatments in the background areas.
Reference: Song et al. (2007, 155306) **Species:** *S. typhimurium* **Strain:** TA98, TA100 **Cell Line:** Rat fibrocytes L-929 cells	PM (soluble organic fraction (SOF) extracts from diesel engines using fuels blended with ethanol by volume: E0 - base diesel fuel; E5 - 5%; E10 - 10%; E15 - 15%; E20 - 20%) **Particle Size:** Density (g/cm^3): E0- 0.8379; E5- 0.8349; E10- 0.8324; E15- 0.8301; E20- 0.8279	**Route:** Cell Culture **Dose/Concentration:** Ames Assay: 0.025, 0.05, 0.1mg/plate; Comet Assay: 0.125, 0.25, 0.5, 1.0mg/mL **Time to Analysis:** Samples extracted 24h. Ames and comet assays performed	All PM extracts induced higher mutational response in TA98 (3- to 5-fold increase over spontaneous) than in TA100 (2-to 3-fold increase). The highest brake specific revertants (BSR) ±S9 in both strains occurred with E20 and lowest BSR was in E5 (except in TA98 – S9). E0 and E20 caused more significant DNA damage (similar in effect) than the other extracts. Damage was dose-dependent but variable with increasing ethanol volume.
Reference: Ueng et al. (2005, 097054) **Species:** Human **Cell Line:** Lung epithelium CL5 (cancerous), bronchial epithelial BEAS-2B (noncancerous), WI-38 normal lung fibroblast	MEP (Yamaha cabin motorcycle 2-strok 50-cc engine) **Particle Size:** NR	**Route:** Cell Culture **Dose/Concentration:** 1, 10, 100, 200 μg/mL **Time to Analysis:** Exhaust collected, extracted. cDNA microarray analysis. RT-PCR: 2h. ELISA: 12h incubation. Centrifuged 24h post-treatment. Bioactivity: 12h incubation. Centrifuged 24h post-treatment. Medium replaced 48h post-incubation. Fibroblasts determined 96h post-incubation. Time response studies: 3-48h treatment. Concentration response studies: 6h treatment.	**Drug metabolism array study:** MEP increased CYP1A1, CYP3A7 and UGT2B. **Cytokine array study:** MEP increased fibroblast growth factor (FGF)-6, FGF-9, IL-1α, IL-22 and vascular endothelial growth factor (VEGF)-D mRNA. **Oncogene, tumor suppressor, estrogen signaling pathway:** MEP increased fra-1, c-src, SHC, p21, COX7RP, and decreased p53 and Rb expression. **RT-PCR:** MEP increased CYP1A1, CYP1B1, IL-6, IL-11, IL-1α, FGF-6, FGF-9, VEGF-D, fra-1 and p21. **Concentration and time responses:** Concentration and time-dependent increases occurred for FGF-9, IL-1α, IL-6, IL-11, but decreased time-dependently after 6h exposure. **BEAS-2B Cells:** MEP had concentration-dependent increases on CYP1A1 and CYP1B1 but did not affect anything else. **Peroxide, MEP+NAC, WI-38 Cells:** MEP increased peroxide production. The MEP+NAC treatment reduced MEP-elevated levels of IL-1α, IL-6, FGF-9, VEGF-D to control levels. Fibroblasts increased in WI-38 cells.

Reference	Particle	Exposure	Effects
Reference: Umbuzeiro et al. (2008, 190491) **Species:** *Salmonella typhimurium* **Strain:** TA98, YG1041 (+/- S9)	PM (urban; São Paulo, Brazil-Cerqueira César street station, Ibirapuera park station) (winter- June 17, 18; average temperature: 16ºC) (EOM) **Particle Size:** NR	**Route:** Cell Culture **Dose/Concentration:** Cerqueira César: UPM- 156 $\mu g/m^3$, EOM- 57.7mg/total UPM; Ibirapuera Park: UPM- 32 $\mu g/m^3$, EOM- 41.7mg/total UPM; Salmonella assay- 0.5, 1, 5, 10, 50, 100 UPM equiv/plate (μg) **Time to Analysis:** Tests performed 20d after collection date. Organic extraction 20h. PAH fractionation. Salmonella/microsome assay.	The TSP and EOM were similar for both sites. The PAH fraction had very low mutagenicity for the Cerqueira César sample in the YG1041 strain and no mutagenicity for the Ibirapuera sample. Nitro-PAH and oxy-PAH had similar mutagenetic activities from both samples. S9 decreased mutagenicity in nitro-PAH but was increased in oxy-PAH. DNA adduct levels were dose-dependent and not different between the two sites.
Reference: Upadhyay et al. (2003, 097370) **Species:** Human **Cell Line:** Alveolar epithelial A549	PM (Dusseldorf, Germany) (Particles contain carbon (19.70%), hydrogen(1.4%),nitrogen (< .05%), oxygen(14.12%), sulfur (2.09%), ash (63.24%)) (Ionizable metals concentrations (ppm): Co(103), Cu(48),Cr(104),Fe(14,521), Mn(21.3), Ni(1,519),Ti(131), V(2,767) **Particle Size:** NR	**Route:** Cell Culture **Dose/Concentration:** 1, 5, 25, 100 $\mu g/cm^2$; 10, 25, 50, 100 $\mu g/cm^2$ **Time to Analysis:** Air collected. Cells grown 24h. PM treatments 1, 4, 8, 12, 24h.	PM induced dose- and time-dependent reductions in ds-DNA due to the formation of DNA-SB. The soluble component caused higher DNA damage. Apoptosis and DNA fragmentation increased dose-dependently. $\Delta\Psi m$ decreased dose-dependently in control cells, but not in cells with Bcl-xl overexpression. PM caused activation of caspase 9. Pretreatment with iron chelators or a free radical scavenger reduced PM-induced DNA-SB formation, DNA fragmentation, caspase 9 activation, and weakened $\Delta\Psi m$ reductions.
Reference: Valavanidis et al. (2005, 096432) **Cell Line:** NR	PM (TSP: high volume pumps, Athens; DEP: 2.0L engine GM Astra; GEP: 1.6L passenger vehicle Ford; Wood smoke soot: domestic fireplace exhaust chimney; PM_{10}: high volume sampling system, Athens; $PM_{2.5}$: high volume cascade impactor (Anderson) system **Particle Size:** Diameter: > 10.2 - < 0.41 μm	**Route:** Incubation **Dose/Concentration:** 20, 40mg/5mL **Time to Analysis:** PM collected 30min-1h or 24h basis. Incubated with H_2O_2 and 2′-deoxyguanosine (dG). Stored 3-7d at -20°C. Fenton reaction. EPR analysis.	PM generated ·OH by a Fenton reaction, which is increased by the addition of EDTA but inhibited by deferoxamine. PM dose-dependently induced dG hydroxylation and 8-hydroxy-2′-deoxyguanosine formation. Transition metals Ni, V, Co, Cr that are capable of redox cycling electron producing ROS were found in the PM samples.
Reference: Xu and Zhang (2004, 097231) **Species:** Human **Cell Line:** Lung epithelial A549	PM (Taiyuan, Beijing; Nov-Feb) (Taiyuan: coal-fume pollution; Beijing: coal-fume and vehicle exhaust) **Particle Size:** Diameter: 2.5 μm	**Route:** Cell Culture **Dose/Concentration:** 5, 50, 200 $\mu g/mL$ **Time to Analysis:** Air samples collected. Cells incubated 12-24h. Comet assay.	Taiyuan had a significantly higher daily $PM_{2.5}$ average than Beijing. It was shown that the smaller the particulate diameter, the higher the concentration of BaP and Pb. A dose- and time-response relationship was seen in DNA fragmentation.

Annex D References

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Annex E. Epidemiologic Studies

E.1. Short-Term Exposure and Cardiovascular Outcomes

E.1.1. Cardiovascular Morbidity Studies

Table E-1 Short-term exposure - cardiovascular morbidity outcomes - PM_{10}

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Baccarelli et al. (2007, 091310) **Period of Study:** Jan 1995 – Aug 2005 **Location:** Lombardia region, Italy	**Outcome:** Fasting and postmethionine-load total homocysteine (tHcy) **Age Groups:** 11-84 yrs **Study Design:** Cross-sectional / Panel **N:** 1,213 participants **Statistical Analyses:** Generalized additive models **Covariates:** age, sex, BMI, smoking, alcohol, hormone use, temperature, day of the year, and long-term trends **Season:** Adjusted for long-term trends to account for season **Dose-response Investigated?** No **Statistical Package:** R v2.2.1 **Lags Considered:** 1d, 7d moving avg.	**Pollutant:** PM_{10} (some TSP measures used to predict PM_{10}) **Averaging Time:** 24 h **Mean (SD):** NR **Percentiles:** 25th: 20.1 50th: 34.1 75th: 52.6 **Max:** 390.0 **Monitoring Stations:** 53 **Copollutant:** CO, NO_2, SO_2, O_3	**PM Increment:** IQR **Percent Change: [Lower CI, Upper CI]:** Homocysteine, fasting: 0.4 (-2.4, 3.3) Homocysteine, postmethionine-load: 1.1 (-1.5, 3.7) **Percent Change: per 25.7m^3 increase in 7-day moving avg of PM_{10}** Homocysteine, fasting: 1.0 (-1.9, 3.9) Homocysteine, postmethionine-load: 2.0 (-0.6, 4.7) **Percent Change: on fasting homocysteine per IQR increase in 24-h PM_{10} levels** Among smokers: 6.2 (0.0, 12.7) Among non-smokers: -1.6 (-5.5, 2.5) **Percent Change: on postmethionine-load homocysteine per IQR increase in 24-h PM_{10} levels:** Among smokers: 6.0 (0.5, 11.8) Among non-smokers: -0.1 (-3.6, 3.5)
Reference: Baccarelli et al. (2007, 090733) **Period of Study:** Jan 1995 – Aug 2005 **Location:** Lombardia region Italy	**Outcome:** Prothrombin time (PT) Activated partial thromboplastin time (APTT) Fibrinogen Functional antithrombin Functional protein C Protein C, antigen Functional protein S Free protein S **Age Groups:** 11-84 yrs **Study Design:** Cross-sectional / Panel **N:** 1,218 participants **Statistical Analyses:** Generalized additive models **Covariates:** Age, sex, BMI, smoking, alcohol, hormone use, temperature, day of the year, and long-term trends **Season:** Adjusted for long-term trends to account for season	**Pollutant:** PM_{10} (some TSP measures used to predict PM_{10}) **Averaging Time:** Hourly concentrations used to calculate lags of same day, 7-day, 30-day, and h 0-6 **Mean (SD):** NR **Percentiles:** Sep-Nov: 2 5th: 33.1 50th: 51.2 75th: 76.5 Max: 148.9 Dec-Feb: 25th: 47.9 50th: 68.5 75th: 95.3 Max: 238.3 Mar-May: 25th: 30.0 50th: 64.1 75th: 64.8 Max: 158.5	**PM Increment:** SD **Effect Estimate [Lower CI, Upper CI]:** Estimated changes in endpoint **PT (international normalized ratio):** At time of blood sample: -0.06 (-0.12, 0.00) Avg levels 7 days prior: -0.03 (-0.10, 0.04) Avg levels 30 days prior: -0.08 (-0.14, -0.01) (Hourly moving Avgs presented in Fig 2) **APTT (ratio to reference plasma):** At time of blood sample: 0.02 (-0.04, 0.08) Avg levels 7 days prior: 0.00 (-0.07, 0.06) Avg levels 30 days prior: 0.01 (-0.06, 0.08) **Fibrinogen:** At time of blood sample: 0.01 (-0.05, 0.07) Avg levels 7 days prior: -0.03 (-0.09, 0.04)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
	Dose-response Investigated? No **Statistical Package:** R software v2.2.1	Jun-Aug: 25th: 28.0 50th: 44.3 75th: 61.3 Max: 94.7 **Monitoring Stations:** 53 sites **Copollutant:** CO, NO_2 , SO_2, O_3	Avg levels 30 days prior: -0.02 (-0.09, 0.05) **Functional antithrombin:** At time of blood sample: -0.02 (-0.09, 0.04) Avg levels 7 days prior: -0.06 (-0.13, 0.01) Avg levels 30 days prior: -0.06 (-0.13, 0.02) **Functional protein C:** At time of blood sample: 0.00 (-0.06, 6.1) Avg levels 7 days prior: -0.06 (-0.12, 0.01) Avg levels 30 days prior: -0.06 (-0.14, 0.01) **Protein C, antigen:** At time of blood sample: 0.00 (-0.06, 6.0) Avg levels 7 days prior: -0.04 (-0.10, 0.03) Avg levels 30 days prior: -0.06 (-0.14, 0.01) **Functional protein S:** At time of blood sample: 0.04 (-0.03, 0.10) Avg levels 7 days prior: -0.03 (-0.11, 0.06) Avg levels 30 days prior: -0.14 (-0.23, -0.05) **Free protein S:** At time of blood sample: 0.05 (-0.01, 0.10) Avg levels 7 days prior: 0.01 (-0.05, 0.07) Avg levels 30 days prior: -0.01 (-0.08, 0.06)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Barclay et al. (2009, 179935) **Period of Study:** Jan 2003 – May 2005 **Location:** Aberdeen, Scotland	**Outcome:** Haematological outcomes, Heart Rhythm outcomes, & Heart Rate Variability outcomes **Age Groups:** 70.4 (8.9) **Study Design:** panel **N:** 132 patients w/ chronic heart failure **Statistical Analyses:** Linear & Mixed Effects Regression Model **Covariates:** age, temperature, humidity, pressure **Dose-response Investigated?** No **Statistical Package:** NR **Lags Considered:** lags 0-2d	**Pollutant:** PM_{10} **Averaging Time:** daily **Mean (SD):** 20.25 **Min:** 7.375 **Max:** 68.3 **Monitoring Stations:** 1 **Copollutant:** $PM_{2.5}$, PNC, NO_2 **Co-pollutant Correlation** NO_2 city: 0.294 NO city: 0.112 NO_2 personal: 0.055 PNC DEOM: 0.241 $PM_{2.5}$ total: 0.476* $PM_{2.5}$ traffic: 0.882* PNC total: 0.125 PNC traffic: 0.190 *correlations based on 3-day average concentrations	**PM Increment:** NR **Beta (Lower CI, Upper CI):** Haemoglobin: 0.136 (-0.274, 0.546) Mean corpuscular haemoglobin: 0.030 (-0.232, 0.291) Platelets: 0.096 (-0.923, 1.115) Haematocrit: 0.131 (-0.289, 0.551) White blood cells: 0.034 (-1.175, 1.244) C reactive protein: -4.872 (-12.094, 2.351) IL-6: 2.207 (-4.995, 9.410) von Willebrand factor: 0.660 (-2.651, 3.970) E-selectin: -0.536 (-2.528, 1.457) Fibrinogen: -0.432 (-2.470, 1.607) Factor VII: 0.990 (-1.265, 3.245) d-dimer: -1.225 (-4.505, 2.055) All arrhythmias: -3.447 (-11.521, 4.627) Ventricular ectopic beats: -2.110 (-12.135, 7.915) Ventricular couplets: -1.561 (-10.811, 7.689) Ventricular runs: -0.709 (-6.677, 5.259) Supraventricular ectopic beats: 0.033 (-9.242, 9.308) Supraventricular couplets: 0.006 (-8.618, 8.629) Supraventricular runs: 3.710 (-2.847, 10.266) Average HR: 0.321 (-0.197, 0.838) 24h SDNN: 1.040 (-0.415, 2.494) 24h SDANN: 1.195 (-0.473, 2.863) 24h RMSSD: 0.321 (-0.197, 0.838) 24h PNN: 2.837 (-3.791, 9.465) 24h LF power: 0583 (-3.622, 4.787) 24h LF normalized: -3.137 (-5.540, -0.733)* 24h HF power: 0.872 (-4.649, 6.392) 24h HF normalized: -2.223 (-4.952, 0.505) 24h LF/HF ratio: -0.296 (-3.832, 3.240) *$p < 0.05$ **Notes:** LF = low frequency HF = high frequency
Reference: Briet et al. (2007, 093049) **Period of Study:** NR **Location:** Paris, France	**Outcome:** Endothelial Function **Age Groups:** 20-40 yrs **Study Design:** panel **N:** 40 white male nonsmokers **Statistical Analyses:** Multiple Robust Regrssion **Covariates:** R53R/R53H genotype, diet, subject factor, visit, temperature **Dose-response Investigated?** No **Statistical Package:** NCSS **Lags Considered:** 0-5d	**Pollutant:** PM_{10} **Averaging Time:** 24h **5 d Mean (SD):** 43 (10) **Monitoring Stations:** NR **Co-pollutant:** $PM_{2.5}$, SO_2, NO, NO_2, CO **Co-pollutant Correlation** n/a	**PM Increment:** 1 SD **Beta (Lower CI, Upper CI), *P*, R^2:** Flow-mediated brachial artery dilation: 0.07 (-0.62, 0.76), NS, 0.03 Reactive hyperemia: 15.91 (7.74, 24.0), < 0.001, 0.16

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Choi et al (2007, 093196) **Period of Study:** 2001-2003 **Location:** Incheon, South Korea	**Outcome:** Blood pressure **Study Design:** Cross-sectional **N:** 10459 subjects with a hospital health examination **Statistical Analyses:** Linear regression **Covariates: Season:** Effect modification by season	**Pollutant:** PM_{10} **Averaging Time:** Measured hourly and calculated 24-h means **Percentiles:** Warm season: Median: 36.7 Cold season: Median: 45.7 **Monitoring Stations:** 9 stations **Copollutant:** NO_2, SO_2	**PM Increment:** 10 $\mu g/m^3$ **Effect Estimate [Lower CI, Upper CI]:** Estimate (p-value) for the relationship between systolic blood pressure (SBP) and diastolic blood pressure (DBP) and an increase in PM_{10} on lag day 1 SBP: Warm season: 0.0798 ($p < 0.001$) DBP: Warm season: 0.0240 ($p < 0.001$) **Note:** No evidence of associations between PM_{10} and BP during the cold season
Reference: Chuang et al. (2007, 091063) **Period of Study:** Between Apr-Jun 2004 or 2005 **Location:** Taipei, Taiwan	**Outcome:** High-sensitivity C-reactive protein (hs-CRP) Fibrinogen, plasminogen activator fibrinogen inhibitor-1 (PAI-1), tissue-type plasminogen activator (tPA), 8-hydroxy-2'-deoxyguanosine (8-OHdG), and log-transformed HRV indices (SDNN = standard deviation of NN intervals, r-MSSD = square root of the mean of the sum of the squares of differences between adjacent NN intervals, LF = low frequency [0.04-0.15Hz], and HF = high frequency [0.15-0.40Hz]) **Age Groups:** 18-25 yrs **Study Design:** Panel (cross-sectional) **N:** 76 students **Statistical Analyses:** linear mixed-effects models **Covariates:** Age, sex, BMI, weekday, temperature of previous day, relative humidity **Season:** Only 1 season of data collection **Dose-response Investigated?** No **Statistical Package:** NR	**Pollutant:** PM_{10} **Averaging Time:** Hourly data used to calculate averages over 1-3 day periods **Mean (SD):** 1-day avg: 49.2 (18.0) 2-day avg: 55.3 (18.6) 3-day avg: 54.9 (18.2) **Range (Min, Max):** 1-day avg: 29.5, 83.4 2-day avg: 25.5, 85.1 3-day avg: 22.2, 87.2 **Monitoring Stations:** 2 sites (each pollutant measured at one site only) **Copollutant:** $PM_{2.5}$, Sulfate, Nitrate, OC, EC, NO_2, CO, SO_2, O_3	**PM Increment:** IQR (1-d avg: 32.7 2-day avg: 34.5 3-day avg: 26.0) **Effect Estimate [Lower CI, Upper CI]:** % change in health endpoint per increase in IQR of PM_{10} (1-3 day averaging period single pollutant models) **hs-CRP:** 1-d: 135.8 (1.8, 269.7) 2-d: 108.2 (-10.9, 227.3) 3-d: 109.6 (2.5, 216.7) **8-OHdG:** 1-d: -9.2 (-21.5, 3.2) 2-d: -6.1 (-17.0, 4.8) 3-d: -5.6 (-13.8, 2.6) **PAI-1:** 1-d: 30.0 (12.4, 47.7) 2-d: 19.1 (3.6, 34.7) 3-d: 21.2 (9.7, 32.8) **tPA:** 1-d: 16.0 (-4.1, 36.2) 2-d: 10.4 (-6.3, 27.2) 3-d: 8.8 (-2.8, 20.5) **Fibrinogen:** 1-d: 5.3 (1.5, 15.2) 2-d: 1.5 (-4.4, 7.5) 3-d: 3.3 (-1.1, 7.7) **Heart Rate Variability** **SDNN:** 1-d: -4.9 (-7.8, -2.1) 2-d: -4.0 (-6.6, -1.4) 3-d: -4.1 (-6.1, -2.2) **r-MSSD:** 1-d: -4.8 (-12.3, 2.7) 2-d: -2.2 (-9.0, 4.7) 3-d: -4.0 (-9.0, 0.9) **LF:** 1-d: -6.1 (-10.1, -2.1) 2-d: -3.0 (-7.2, 1.2) 3-d: -4.3 (-7.0, -1.6) **HF:** 1-d: -5.5 (-13.0, 2.1) 2-d: -2.7 (-9.5, 4.1) 3-d: -2.0 (-7.2, 3.2)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Ebelt et al. (2005, 056907) **Period of Study:** Summer of 1998 **Location:** Vancouver, Canada	**Outcome:** CVD **Age Groups:** range from 54-86 yrs mean age = 74 years **Study Design:** extended analysis of a repeated-measures panel study **N:** 16 persons with COPD **Statistical Analyses:** Earlier analysis expanded by developing mixed-effect regression models and by evaluating additional exposure indicators **Dose-response Investigated?** No **Statistical Package:** SAS V8	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (SD):** Ambient PM_{10}: 17 ± 6 Exposure to ambient PM_{10}: 10.3 ± 4.6 **Range (Min, Max):** Ambient $PM_{10-2.5}$: 7 - 36 Exposure to ambient $PM_{10-2.5}$: 1.5 - 23.8 **Monitoring Stations:** 5 **Copollutant (correlation):** Ambient concentrations and exposure to ambient PM were highly correlated for each respective metric: r ≥ 0.71 $PM_{10-2.5}$: r ≥ 0.72 $PM_{2.5}$: r ≥ 0.92	**Note:** Total personal fine particle exposure (T) were dominated by exposures to non ambient particles which were not correlated with ambient fine particle exposure (A) or ambient concentrations (C). Results for each of these metrics are listed. **Effect estimates and 95% CI for IQR range increases in exposure** Increment: C^{10}: IQR = 7 $\mu g/m^3$ SBP (mm Hg): -2.2 (-4.78-0.38) DBP (mm Hg): -0.78 (-2.65-1.09) Ln-SVE (bph): 0.16 (-0.07-0.40) HR (bpm): 1.02 (-0.79-2.82) SDNN (ms): -2.14 (-6.94-2.65) R-MSSD (ms): -2.24 (-4.27-0.21) Increment: A^{10}: IQR = 6.5 $\mu g/m^3$ SBP (mm Hg): -2.81 (-5.67-0.05) DBP (mm Hg): -0.59 (-2.79-1.62) Ln-SVE (bph): 0.27 (0.03-0.52) HR (bpm): 0.86 (-1.61-3.33) SDNN (ms): -3.91 (-9.73-1.91) R-MSSD (ms): -0.81 (-4.94-3.31)
Reference: Folino et al. (2009, 191902) **Period of Study:** Jun 2006 – May 2007 **Location:** Padua, Italy	**Outcome:** HRV & Inflammatory Markers **Age Groups:** 45-65 yrs **Study Design:** panel **N:** 39 patients w/ myocardial infarction **Statistical Analyses:** Linear Regression Model, ANOVA **Covariates:** temperature, relative humidity, atmospheric pressure, beta-blocker, aspirin, or nitrate consumption, smoking habit **Dose-response Investigated?** No **Statistical Package:** Stata **Lags Considered:** NR	**Pollutant:** PM_{10} **Averaging Time:** 24h **Mean (SD):** Summer: 46.4 (16.1) Winter: 73.0 (30.9) Spring: 38.3 (15.4) **Monitoring Stations:** NR **Copollutant:** $PM_{2.5}$, $PM_{0.25}$ **Co-pollutant Correlation** NR	**PM Increment:** 1 $\mu g/m^3$ **Beta (SE), p-value:** SDNN: 0.115 (0.093), 0.218 SDANN: 0.138 (0.103), 0.182 RMSSD: 0.049 (0.034), 0.146 pH: 0.002 (0.001), 0.033 LTB4: 0.427 (0.0279), 0.126 eNO: 0.000 (0.002), 0.851 PTX3: -0.003 (0.001), 0.033 C-reactive protein: -0.006 (0.004), 0.161 CC16: -0.002 (0.002), 0.280 IL-8: 0.000 (0.003), 0.895
Reference: Forbes et al. (2009, 190351) **Period of Study:** 1994, 1998, 2003 **Location:** England	**Outcome:** Inflammation Markers **Age Groups:** 16+ yrs **Study Design:** cross-sectional **N:** 25,000 white adults w/ fibrinogen measurements & 17,000 white adults w/ C-reactive protein measurements **Statistical Analyses:** Multilevel Linear Regression Models **Covariates:** age, sex, BMI, social class, region, cigarette smoking **Dose-response Investigated?** No **Statistical Package:** Stata **Lags Considered:** NR	**Pollutant:** PM_{10} **Averaging Time:** yearly 1994 **Median:** 19.5 **Range:** 12.5-36.1 **IQR:** 3.7 1998 **Median:** 17.9 **Range:** 12.6-27.0 **IQR:** 2.7 2003 **Median:** 16.2 **Range:** 11.0-22.7 **IQR:** 2.6 **Monitoring Stations:** NR **Copollutant:** NO_2, SO_2, O_3 **Co-pollutant Correlation** n/a	**PM Increment:** 1 $\mu g/m^3$ **Percent Change (Lower CI, Upper CI):** Fibrinogen 1994 Crude: -0.068 (-0.367, 0.231) 1994 Adjusted: 0.080 (-0.164, 0.326) 1998 Crude: -0.592 (-0.902, -0.280) 1998 Adjusted: -0.388 (-0.727, -0.047) 2003 Crude: -0.339 (-0.696, 0.019) 2003 Adjusted: -0.069 (-0.458, 0.322) Combined: -0.077 (-0.254, 0.100) C-reactive protein 1998 Crude: -0.914 (-2.206, 0.395) 1998 Adjusted: -0.266 (-1.782, 1.274) 2003 Crude: 0.286 (-1.327, 1.925) 2003 Adjusted: 0.661(-1.068, 2.421) Combined: 0.140 (-1.003, 1.296)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Kaufman (1987, 190960) **Period of Study:** Nov 2004 - 2005 **Location:** Isfahan, Iran	**Outcome:** Inflammation **Age Groups:** 10-18 yrs **Study Design:** panel **N:** 374 children **Statistical Analyses:** Linear Regression, Logistic Regression **Covariates:** age, gender, BMI, waist circumference, healthy eating index, physical activity level **Dose-response Investigated?** No **Statistical Package:** SPSS **Lags Considered:** 0-7d avg	**Pollutant:** PM_{10} **Averaging Time:** 24h **Mean (SD):** 122.08 (33.63) **0th:** 11.00 **25th:** 86.50 **50th:** 153.0 **75th:** 191.00 **Monitoring Stations:** 3 **Copollutant:** O_3, SO_2, NO_2, CO **Co-pollutant Correlation** NR	**PM Increment:** NR **Beta (SE):** CRP: 1.5 (0.2) Ox-LDL: 1.4 (0.1) MDA: 1.3 (0.1) CDE: 1.1 (0.1) HOMA-IR: 1.1 (0.3)
Reference: Liao et al. (2004, 056590) **Period of Study:** 1996-1998 **Location:** ARIC study cohort (Washington County, MD Forsyth County, NC and selected suburbs of Minneapolis, MN). The 4th quarter of the ARIC cohort was sampled exclusively from black residents of Jackson, MS.	**Outcome:** 5-min HR, HRV indices (HF, LF, SDNN) **Study Design:** Cross-sectional **Statistical Analyses:** Linear regression	**Pollutant:** PM_{10} **Averaging Time:** 24-h **Mean (SD):** 24.3 (11.5) **Copollutant:** O_3 CO SO_2 NO_2	**PM Increment:** SD **Effect Estimate [Lower CI, Upper CI]:** Estimate (SE) HF: -0.06 ms^2 (0.018) SDNN: -1.03 ms (0.31) H: 0.32 beats/min (0.158)
Reference: Liao et al. (2005, 088677) **Period of Study:** 1987-1989 baseline health exam **Location:** 3 centers in the US (Forsyth County, NC suburbs of Minneapolis, MN black residents of Jackson, MS)	**Outcome:** Fibrinogen, factor VIII coagulant activity (VIII-C), von Willebrand factor (vWF), white blood cell count (WBC), and serum albumin **Age Groups:** 45-64 yrs **Study Design:** Cross-sectional **N:** 10,208 participants (7705 for PM) **Statistical Analyses:** Multiple linear regression **Covariates:** Age, sex, ethnicity-center, education, smoking, drinking status, BMI, history of chronic respiratory disease, humidity, season, cloud cover, and temperature **Dose-response Investigated?** Yes, examined higher-ordered terms for each pollutant **Statistical Package:** SAS v8.2	**Pollutant:** PM_{10} **Averaging Time:** 24-h averages (1, 2, and 3 days prior to the exam) **Mean (SD):** 29.9 (29.9) **Mean (SD) within Quartiles:** Q1-3: 24.0 (6.96) Q4: 47.3 (10.11) **Copollutant:** CO, SO_2, NO_2, O_3	**PM Increment:** 1 SD (12.8 $\mu g/m^3$) **Effect Estimate:** Adjusted regression coefficient (SE): Fibrinogen (mg/dl): 0.163 (0.755) Factor VIII-C (%): Non-linear association: β (PM_{10}) = -5.30, $p < 0.01$ β $(PM_{10})^2$ = 0.80, $p < 0.05$ vWF (%): Diabetics: 3.93 (1.80) Nondiabetics: -0.54 (0.58) Albumin (g/dl): CVD: -0.006 (0.003) Non-CVD: 0.029 (0.017) $p < 0.05$

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Liao et al. (2007, 180272) **Period of Study:** 1999-2004 **Location:** 24 US states	**Outcome:** Ectopy **Age Groups:** women 50-79 yrs **Study Design:** panel **N:** 57,422 **Statistical Analyses:** logistic regression & random effects modeling **Covariates:** age, race, center, education, history of CVD/chronic lung disease, rel. humidity, temperature, smoking **Dose-response Investigated?** No **Statistical Package:** SAS, Stata **Lags Considered:** lags 0-365d ‡Monitors used in model for spatial interpolation of daily PM values.	**Pollutant:** PM_{10} **Averaging Time:** daily **Mean (SD)*:** All: 27.5 (12.1) No Ectopy: 27.5 (12.1) Any Ectopy: 27.5 (11.9) **5th, 95th percentile*:** All: 12.2, 48.9 No Ectopy: 12.3, 48.8 Any Ectopy: 11.8, 49.3 **Monitoring Stations:** NR‡ **Copollutant:** $PM_{2.5}$ **Co-pollutant Correlation** NR *Lag 1	**PM Increment:** 10 $\mu g/m^3$ **Percent Change (Lower CI, Upper CI):** All Ventricular Ectopy Lag 0: 1.01 (0.95, 1.07) Lag 1: 1.02 (0.96, 1.09) Lag 2: 0.99 (0.93, 1.06) Current Smoker Ventricular Ectopy Lag 0: 1.21 (0.96, 1.53) Lag 1: 1.32 (1.07, 1.65) Lag 2: 1.22 (0.95, 1.56) Nonsmoker Ventricular Ectopy Lag 0: 1 (0.93, 1.06) Lag 1: 1.01 (0.94, 1.07) Lag 2: 0.98 (0.92, 1.05) All Supraventricular Ectopy Lag 0: 1 (0.95, 1.06) Lag 1: 1 (0.95, 1.05) Lag 2: 0.99 (0.94, 1.04) All Ventricular or Supraventricular Ectopy Lag 0: 1 (0.95, 1.04) Lag 1: 1 (0.96, 1.04) Lag 2: 0.98 (0.94, 1.02)
Reference: Liu et al. (2007, 156705) **Period of Study:** May 24, 2005–Jul 8, 2005 **Location:** Windsor, Ontario, Canada	**Outcome:** Heart rate, blood pressure, brachial arterial diameter, flow-mediated vasodilatation (FMD), plasma cytokines, and thiobarbituric acid reactive substances (TBARS) **Age Groups:** 18-65 yrs **Study Design:** Panel **N:** 24 nonsmoking subjects with type I or II diabetes over a 7 week period (2-14 visits for subjects) 170 total vascular measurements and 134 total blood samples collected **Statistical Analyses:** Mixed effects regression models **Covariates:** (time-dependent covariates) Daily temperature, relative humidity, blood glucose level, also checked for confounding by ambient air pollutant concentrations (controlled for ambient $PM_{2.5}$) **Season:** No adjustment since testing was completed within a 7 week period during early summer **Dose-response Investigated?** No **Statistical Package:** S-Plus	**Pollutant:** PM_{10} (personal) **Averaging Time:** Real-time monitor measured exposure during 24-h period prior to clinic measures **Median (5th-95th percentile):** 0-24 hrs: 25.5 (9.8-133.0) 0-6hrs: 15.3 (5.3-83.2) 7-12hrs: 17.0 (7.1-186.3) 13-18hrs: 28.5 (11.4-167.0) 19-24 hrs: 30.5 (10.1-148.2) **Monitoring Stations:** Personal monitoring **Copollutant (correlation):** Ambient $PM_{2.5}$ (r = 0.34)	**PM Increment:** 10 $\mu g/m^3$ **Effect Estimate [Lower CI, Upper CI]:** $^{**}p < 0.05$ $^{*}p < 0.10$. Regression coefficients (SE) **End-diastolic basal diameter (μm):** All subjects (n = 24): -2.52 (3.27) subjects not taking vasoactive meds (n = 17): -3.93 (3.66) subjects w/BMI ≤ 29kg/m^2 (n = 14): 8.85 (5.85) **End-systolic basal diameter (μm):** All subjects (n = 24): -9.02 (3.58)** subjects not taking vasoactive meds (n = 17): -10.59 (4.36)** subjects w/BMI ≤29kg/m^2 (n = 14): 3.85 (5.49) **End-diastolic FMD (%):** All subjects (n = 24): 0.20 (0.08)** subjects not taking vasoactive meds (n = 17): 0.23 (0.09)** subjects w/BMI ≤29kg/m^2 (n = 14): 0.12 (0.05)** **End-systolic FMD (%):** All subjects (n = 24): 0.38 (0.18)** subjects not taking vasoactive meds (n = 17): 0.51 (0.22)** subjects w/BMI ≤ 29kg/m^2 (n = 14): 0.18 (0.10)* **Flow (cm/s):** All subjects (n = 24): -0.16 (0.19) subjects not taking vasoactive meds (n = 17): -0.48 (0.21)** subjects w/BMI ≤ 29kg/m^2 (n = 14): -0.39 (0.23)* **Heart rate (bpm):** All subjects (n = 24): 0.01 (0.11) subjects not taking vasoactive meds (n = 17): -0.06 (0.12) subjects w/BMI ≤ 29kg/m^2 (n = 14): 0.15 (0.12) **Diastolic blood pressure (mm Hg):** All subjects (n = 24): 0.19 (0.16) subjects not taking vasoactive meds (n = 17): 0.40 (0.18)** subjects w/BMI ≤ 29kg/m^2 (n = 14): 0.27

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			(0.21) **Systolic blood pressure (mm Hg):** All subjects (n = 24): 0.17 (0.19) subjects not taking vasoactive meds (n = 17): 0.43 (0.24)* subjects w/ BMI ≤ 29kg/m^2 (n = 14): 0.38 (0.24) **CRP (µg/mL):** All subjects (n = 24): 0.11 (0.07) subjects not taking vasoactive meds (n = 17): 0.10 (0.09) subjects w/ BMI ≤ 29kg/m^2 (n = 14): 0.02 (0.03) **ET-1 (pg/mL):** All subjects (n = 24): 0.00 (0.00) subjects not taking vasoactive meds (n = 17): 0.00 (0.00) subjects w/BMI ≤ 29kg/m^2 (n = 14): 0.00 (0.01) **IL-6 (pg/mL):** All subjects (n = 24): 0.00 (0.05) subjects not taking vasoactive meds (n = 17): 0.01 (0.05) subjects w/BMI ≤ 29kg/m^2 (n = 14): -0.00 (0.03) **TNF-α (pg/mL):** All subjects (n = 24): 0.03 (0.05) subjects not taking vasoactive meds (n = 17): 0.02 (0.05) subjects w/ BMI ≤ 29kg/m^2 (n = 14): 0.03 (0.08) **TBARS (pmol/mL)** All subjects (n = 24): 16.12 (4.00)** subjects not taking vasoactive meds (n = 17): 8.10 (9.18) subjects w/ BMI ≤ 29kg/m^2 (n = 14): -0.28 (6.60) regression coefficients (SE) among subjects not taking vasoactive medications, with lag time **End-diastolic basal diameter (µm):** 0-6 h: 29.91 (10.64)** 7-12 h: 0.72 (3.95) 13-18 h: -3.62 (2.80) 19-24 h: -0.57 (1.7) **End-systolic basal diameter (µm):** 0-6 h: 28.88 (11.22)** 7-12 h: -0.78 (4.58) 13-18 h: -7.70 (3.30)** 19-24 h: -2.87 (2.05) **End-diastolic FMD (%):** 0-6 h: -0.12 (0.10) 7-12 h: 0.04 (0.05) 13-18 h: 0.11 (0.03)** 19-24 h: 0.12 (0.04)** **End-systolic FMD (%):** 0-6 h: 0.36 (0.08)** 7-12 h: 0.48 (0.32) 13-18 h: 0.19 (0.06)** 19-24 h: 0.34 (0.13)** **Flow (cm/s):** 0-6 h: -0.34 (0.22) 7-12 h: -0.26 (0.27 13-18 h: -0.27 (0.15)* 19-24 h: -0.30 (0.11)** **Heart rate (bpm):** 0-6 h: 0.31 (0.13)** 7-12 h: 0.26 (0.12)** 13-18 h: 0.01 (0.09) 19-24 h: -0.08 (0.05)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			Diastolic blood pressure (mm Hg): 0-6 h: -0.29 (0.12)** 7-12 h: 0.24 (0.12)** 13-18 h: 0.46 (0.17)** 19-24 h: 0.18 (0.14) **Systolic blood pressure (mm Hg):** 0-6 h: -0.65 (0.18)** 7-12 h: 0.17 (0.19) 13-18 h: 0.86 (0.24)** 19-24 h: 0.11 (0.10) **CRP (µg/mL):** 0-6 h: 0.15 (0.13) 7-12 h: 0.15 (0.13) 13-18 h: 0.03 (0.06) 19-24 h: 0.04 (0.03) **ET-1 (pg/mL):** 0-6 h: 0.02 (0.00)**: 7-12 h: -0.00 (0.00) 13-18 h: -0.00 (0.00) 19-24 h: 0.00 (0.00) **IL-6 (pg/mL):** 0-6 h: 0.03 (0.06) 7-12 h: 0.00 (0.06) 13-18 h: 0.02 (0.03) 19-24 h: 0.00 (0.02) **TNF-α (pg/mL):** 0-6 h: 0.01 (0.07) 7-12 h: 0.09 (0.04)** 13-18 h: 0.01 (0.04) 19-24 h: -0.00 (0.03) **TBARS (pmol/mL):** 0-6 h: -4.44 (6.72) 7-12 h: 11.94 (5.08)** 13-18 h: 5.06 (4.03) 19-24 h: 1.06 (4.64) **Note:** Adding ambient $PM_{2.5}$ data as a covariate in the model yielded similar regression coefficients for personal PM_{10}
Reference: Lipsett et al. (2006, 088753) **Period of Study:** February–May 2000 **Location:** Coachella Valley, CA	**Outcome:** HRV parameters: SDNN, SDANN, r-MSSD, LF, HF, total power, triangular index (TRII). **Study Design:** Panel study **N:** 19 non-smoking adults with coronary artery disease **Statistical Analysis:** Mixed linear regression models with random effects parameters	**Pollutant:** PM_{10} **Averaging Time:** 2 h **Mean (range):** Indio: 23.2 (6.3-90.4) Palm Springs: 14 (4.7-52) **Monitoring Stations:** 2 **Copollutant:** O_3	**PM Increment:** SE*1000 **Effect Estimate (change in HRV per unit increase in PM concentration):** SDNN: -0.71 msec (SE = 0.268) **Notes:** Weekly ambulatory 24 h ECG recordings (once per week for up to 12 weeks), using Holter monitors, were made. Subjects' residences were within 5 miles of one of two PM monitoring sites. Regressed HRV parameters against 18:00–20:00 mean particulate pollution.

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Ljungman et al. (2008, 180266) **Period of Study:** Aug 2001 – Dec 2006 **Location:** Gothenburg & Stockholm, Sweden	**Outcome:** Ventricular Arrhythmia **Age Groups:** 28-85 yrs **Study Design:** case-crossover **N:** 88 patients w/ implantable cardioverter defibrillators **Statistical Analyses:** conditional logistic regression **Covariates:** temperature, humidity, pressure, ischemic heart disease, ejection fraction, heart disease, diabetes, use of beta-blockers, age, BMI, location at time of arrhythmia, distance from air pollution monitor **Dose-response Investigated?** No **Statistical Package:** Stata, S-plus **Lags Considered:** lags 2-24h	**Pollutant:** PM_{10} **Averaging Time:** hourly Gothenburg, Stockholm **Median:** 2h: 18.95, 14.62 24h: 19.92, 15.23 **Min:** 2h: 0.00, 0.33 24h: 2.13, 3.96 **Max:** 2h: 203.75, 159.79 24h: 78.01, 90.50 **IQR:** 2h: 14.16, 11.59 24h: 11.49, 9.59 **Monitoring Stations:** 2 **Copollutant:** $PM_{2.5}$, NO_2 **Co-pollutant Correlation** 2h NO_2: 0.36 24h NO_2: 0.29	**PM Increment:** Interquartile Range **Odds Ratio (Lower CI, Upper CI):** 2h: 1.31 (1.00, 1.72) 24h: 1.24 (0.87, 1.76) **Notes:** OR of ventricular arrhythmia for an IQR increase of air pollutants in different subgroups (figure 2)
Reference: Ljungman et al. (2009, 191983) **Period of Study:** May 2003 – July 2004 **Location:** Athens, Greece Helsinki, Finland Ausburg, Germany Barcelona, Spain Rome, Italy Stokholm, Sweeden	**Outcome:** Interleukin-6 Response **Age Groups:** 35-80 yrs **Study Design:** panel **N:** 955 male myocardial infarction survivors **Statistical Analyses:** Additive Mixed Models **Covariates:** age, sex, BMI, city, HDL/total cholesterol, smoking, alcohol intake, HbA1c, NT-proBNP, history of MI, heart failure, or diabetes, phlegm **Dose-response Investigated?** No **Statistical Package:** NR **Lags Considered:** 1d	**Pollutant:** PM_{10} **Averaging Time:** 24h **Mean:** 31.6 **25th:** 21.1 **75th:** 38.4 **Monitoring Stations:** NR **Copollutant:** CO, NO_2, PNC, $PM_{2.5}$ **Co-pollutant Correlation** $PM_{2.5}$: 0.81	**PM Increment:** Interquartile Range (17.4 $\mu g/m^3$) **Change of IL-6 (Lower CI, Upper CI), p-value:** 0.0 (-1.3, 1.3), 1.0
Reference: Mar et al. (2005, 087566) **Period of Study:** 1999–2001 **Location:** Seattle, WA	**Outcome:** Change in arterial O_2 saturation, heart rate, and blood pressure (SBP and DBP) **Age Groups:** > 75 years **Study Design:** Panel study **N:** 88 elderly subjects **Statistical Analysis:** GEE	**Pollutant:** PM_{10} **Averaging Time:** 24-hs **Mean (SD):** Indoor: 12.6 (7.8) Outdoor: 14.5 (7.0)	**PM Increment:** 10 $\mu g/m^3$ **Unit change in measure(95% CI): Among all subjects:** Each increase in outdoor same day PM_{10} was associated with: SBP: -0.10 mmHg (95% CI: -1.37, 1.18) DBP: -0.03 mmHg (95% CI: -0.79, 0.73) HR: -0.48 beats/min (95% CI: -1.03, 0.06) **Each increase in indoor same day $PM_{2.5}$ was associated with:** SBP: 0.92 mmHg (95% CI: -0.95, 2.78) DBP: 0.63 mmHg (95% CI: -0.29, 1.56) HR: 0.02 beats/min (95% CI: -0.54, 0.58) **Notes:** Results by health status presented in Fig 1. Used 2 sessions that each were 10 consecutive days of measurement. Used personal, indoor, and outdoor measures of $PM_{2.5}$

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Metzger et al. (2007, 092856) **Period of Study:** January 1993–December 2002 **Location:** Atlanta, GA	**Outcome:** Days with any event recorded by the ICD, days with ICD shocks/defibrillation and days with either cardiac pacing or defibrillation **Study Design:** Repeated measures **N:** 884 subjects **Statistical Analysis:** Logistic regression with GEE to account for residual autocorrelation within subjects	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (SD):** 28.0 (12.2) **Median:** 26.4 **Copollutant:** O_3, NO_2, CO, SO_2. Aug1998-Dec2002: Oxygenated hydrocarbons	**PM Increment: OR (95% CI):** Outcome = Any event recorded by ICD OR = 1.00 (95% CI: 0.97, 1.03)
Reference: Min et al. (2008, 191901) **Period of Study:** Dec 2003-Jan 2004 **Location:** Taein Isalnd, South Korea	**Outcome:** Heart Rate Variability **Age Mean (SD):** 44.3 (21.9) **Study Design:** Panel **N:** 1.349 participants **Statistical Analyses:** Linear Regression **Covariates:** age, sex, BMI, smoking **Dose-response Investigated?** No **Statistical Package:** SAS, R **Lags Considered:** 0-72h	**Pollutant:** PM_{10} **Averaging Time:** 1 h **Mean (SD):** 33.244 (19.017) **Percentiles:** 25th: 18.000 50th: 26.000 75th: 41.000 **Range:** 187.000. 16.000 **Monitoring Stations:** 1 **Copollutant:** NO_2, SO_2	**PM Increment:** 1 SD (19 $\mu g/m^3$) **Percent Change: [Lower CI, Upper CI]:** SDNN 6-h avg: -4.34 (-7.99, -0.55)** 9-h avg: -5.48 (-9.61, -1.17)**h^ 12-h avg: -6.23 (-10.47, -1.79)*** 24h-avg: -4.73 (-9.73, 0.56)- 48-h avg: -1.25 (-5.59, 3.29) 72-h avg: -0.85 (-5.35, 3.86) LF 6-h avg: -10.32 (-18.05, -1.86)** 9-h avg: -13.79 (-22.26, -4.39)*** 12-h avg: -14.48 (-23.18, -4.80)*** 24h-avg: -13.15 (-23.36, -1.57)** 48-h avg: -0.10 (-9.99, 10.87) 72-h avg: -7.61 (-17.04, 2.88) HF 6-h avg: -1.07 (-10.43, 9.28) 9-h avg: -3.28 (-13.72, 8.43) 12-h avg: -4.06 (-14.77, 8.00) 24h-avg: -1.22 (-13.96, 13.41) 48-h avg: -3.55 (-14.01, 8.18) 72-h avg: -3.88 (-14.64, 8.23) **Notes:** Percent Change in HRV for air pollution children, adults, and the elderly (figure 2) Percent Change in HRV for PM_{10} exposure in all ages (figure 3)
Reference: Peters et al. (2009, 191992) **Period of Study:** May 2003 – July 2004 **Location:** Helsinki, Finland Ausburg, Germany Barcelona, Spain Rome, Italy Stokholm, Sweeden	**Outcome:** Plasma Fibrinogen **Age Groups:** 37-81 **Study Design:** panel **N:** 854 adults **Statistical Analyses:** Additive Mixed Models **Covariates:** age, sex, BMI, city, HDL/total cholesterol, smoking, HbA1c, NT-proBNP, history of arrhythmia, asthma, arthrosis, stroke, bronchitis, season, apparent temperature, relative humidity, weekday, hour of visit **Dose-response Investigated?** No **Statistical Package:** NR **Lags Considered:** 0-5d avg	**Pollutant:** PM_{10} **Averaging Time:** 24h **Mean (SD):** 30.3 **Min:** 0 **Max:** 194 **Monitoring Stations:** NR **Copollutant:** $PM_{2.5}$, $PM_{10-2.5}$ **Co-pollutant Correlation** NR	**PM Increment:** 13.5 $\mu g/m^3$ **Change (Lower CI, Upper CI):** Genotype 1 1 rs2070006: 1.22 (0.47, 1.96) rs2070011: 1.16 (0.41, 1.90) rs1800790: 0.27 (-0.36, 0.91) rs2227399: 0.27 (-0.36, 0.91) rs6056: 0.19 (-0.45, 0.83) rs4220: 0.19 (-0.45, 0.83) Haplotype in cluster 2: 0.09 (-0.53, 0.76) rs1800791: 0.18 (0.21, 1.40) Genotype 1 2 rs2070006: 0.5 (-0.19, 2.15) rs2070011: 0.42 (-0.28, 1.13) rs1800790: 1.28 (0.54, 2.01) rs2227399: 1.28 (0.55, 2.02) rs6056: 1.26 (0.49, 2.04) rs4220: 1.27 (0.49, 2.04) Haplotype in cluster 2: 1.17 (0.35, 1.99) rs1800791: 0.40 (-0.48, 1.28) Genotype 2 2 rs2070006: 0.11 (-1.94, 2.15) rs2070011: 0.08 (-2.08, 2.24) rs1800790: 2.15 (0.71, 3.60) s2227399: 2.18 (0.73, 3.63) s6056: 2.24 (0.72, 3.77) s4220: 2.25 (0.73, 3.78) Haplotype in cluster 2: 2.16 (0.61, 3.71) rs1800791: -0.13 (-1.84, 1.58)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Rosenlund et al. (2007, 114679) **Period of Study:** 1985-1996 **Location:** Stockholm County	**Outcome:** Myocardial Infarction **Age Groups:** 15-79 yrs **Study Design:** case-control **N:** 24,387 first event of myocardial infarction cases and 276,926 population based controls **Statistical Analyses:** Logistic Regression **Covariates:** age, sex, calendar year, SES **Dose-response Investigated?** No **Statistical Package:** Stata **Lags Considered:** 5yr	**Pollutant:** PM_{10} **Averaging Time:** 5yrs Cases **Median:** 2.4 **5th-95th:** 0.3-6.2 Controls **Median:** 2.2 **5th-95th:** 0.3-6.0 **Monitoring Stations:** NR **Copollutant:** NO_2, CO **Co-pollutant Correlation** HNR	**PM Increment:** 5th to 95th percentile (5μg/m^3) **Odds Ratio (Lower CI, Upper CI):** All Subjects Controls: 1.0 All Cases: 1.04 (1.00, 1.09) Nonfatal Cases: 0.98 (0.963, 1.03) Fatal Cases: 1.16 (1.09, 1.24) In-hospital death: 1.05 (0.95, 1.17) Out-of-hospital death: 1.23 (1.14, 1.33) Subjects who did not move b/t population censuses Controls: 1.0 All Cases: 1.11 (1.02, 1.21) Nonfatal Cases: 1.05 (0.96, 1.15) Fatal Cases: 1.56 (1.28, 1.91) In-hospital death: 1.58 (1.13, 2.19) Out-of-hospital death: 1.56 (1.22, 1.98)
Reference: Ruckerl et al. (2007, 156931) **Period of Study:** May 2003–Jul 2004 **Location:** Athens, Augsburg, Barcelona, Helsinki, Rome, and Stockholm	**Outcome:** Interleukin-6 (IL-6), fibrinogen, C-reactive protein (CRP) **Age Groups:** 35-80 yrs **Study Design:** Repeated measures / longitudinal **N:** 1003 MI survivors **Statistical Analyses:** Mixed-effect models **Covariates:** City-specific confounders (age, sex, BMI) long-term time trend and apparent temperature RH, time of day, day of week included if adjustment improved model fit **Season:** Long-term time trend **Dose-response Investigated?** Used p-splines to allow for nonparametric exposure-response functions **Statistical Package:** SAS v9.1	**Pollutant:** PM_{10} **Averaging Time:** Hourly and 24-h (lag 0-4, mean of lags 0-4, mean of lags 0-1, mean of lags2-3, means of lags 0-3) **Mean (SD):** Presented by city only **Percentiles:** NR **Range (Min, Max):** NR **Monitoring Stations:** Central monitoring sites in each city **Copollutant:** SO_2 O_3 NO NO_2	**PM Increment:** IQR **Effect Estimate [Lower CI, Upper CI]:** % change in mean blood markers per increase in IQR increase of air pollutant. **IL-6:** Lag (IQR): % change in GM (95%CI) Lag 0 (17.4): -0.34 (-1.66, 0.99) Lag 1 (17.4): -0.69 (-1.95, 0.58) Lag 2 (17.4): -1.59 (-3.99, 0.88) 5-d avg (13.5): -0.87 (-2.28, 0.55) **Fibrinogen:** Lag (IQR): % change in AM (95%CI) Lag 0 (17.4): 0.06 (-0.43, 0.55) Lag 1 (17.4): 0.14 (-0.35, 0.63) Lag 2 (17.4): 0.24 (-0.24, 0.72) 5-d avg (13.5): 0.60 (0.10, 1.09) **CRP:** Lag (IQR): % change in GM (95%CI) Lag 0 (17.4): -0.71 (-2.75, 1.37) Lag 1 (17.4): -0.63 (-2.61, 1.39) Lag 2 (17.4): -1.42 (-4.23, 1.47) 5-d avg (13.5): -1.35 (-3.45, 0.79)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Ruckerl et al. (2006, 088754) **Period of Study:** Oct 2000–Apr 2001 **Location:** Erfurt, Germany	**Outcome:** C-reactive protein (CRP) serum amyloid A (SAA) E-selectin vWF intercellular adhesion molecule-1 (ICAM-1) fibrinogen Factor VII prothrombin fragment 1+2 D-dimer **Age Groups:** 50+ yrs **Study Design:** Panel (12 repeated measures at 2-wk intervals) **N:** 57 male subjects with coronary disease **Statistical Analyses:** Fixed effects linear and logistic regression models **Covariates:** Models adjusted for different factors based on health endpoint CRP: RH, temperature, trend, ID ICAM-1: temperature, trend, ID vWF: air pressure, RH, temperature, trend, ID FVII: air pressure, RH, temperature, trend, ID, weekday **Season:** Time trend as covariate **Dose-response Investigated?** Sensitivity analyses examined nonlinear exposure-response functions **Statistical Package:** SAS v8.2 and S-Plus v6.0	**Pollutant:** PM_{10} **Averaging Time:** 24-h **Mean (SD):** 20.0 (13.0) **Percentiles:** 25th: 10.8 50th: 15.6 75th: 26.0 **Range (Min, Max):** 5.4, 74.5 **Monitoring Stations:** 1 site **Copollutant:** UFPs (ultrafine particles) AP (accumulation mode particles) $PM_{2.5}$ PM_{10} OC (organic carbon) EC (elemental carbon) NO_2 CO	**PM Increment:** IQR (15.2 5-d avg: 12.8) **Effect Estimate [Lower CI, Upper CI]:** Effects of air pollution on blood markers presented as OR (95%CI) for an increase in the blood marker above the 90th percentile per increase in IQR air pollutant. **CRP:** Time before draw: 0 to 23 h: 1.2 (0.8, 1.9) 24 to 47 h: 2.0 (1.1, 3.6) 48 to 71 h: 2.2 (1.2, 3.8) 5-d mean: 2.0 (1.2, 3.7) **ICAM-1:** Time before draw: 0 to 23 h: 1.3 (0.9, 1.8) 24 to 47 h: 3.1 (2.0, 4.8) 48 to 71 h: 3.4 (2.2, 5.2) 5-d mean: 3.4 (2.2, 5.3) Effects of air pollution on blood markers presented as % change from the mean/GM in the blood marker per increase in IQR air pollutant. **vWF:** Time before draw: 0 to 23 h: 4.0 (-0.6, 8.5) 24 to 47 h: 6.0 (0.6, 11.5) 48 to 71 h: 1.1 (-4.9, 7.0) 5-d mean: 6.1 (-0.6, 12.8) **FVII:** Time before draw: 0 to 23 h: -6.6 (-10.4 to -2.5) 24 to 47 h: -8.4 (-12.3 to -4.3) 48 to 71 h: -5.9 (-9.6, -2.0) 5-d mean: -8.0 (-12.4, -3.4) **Note:** summary of results presented in figures. SAA results indicate increases in association with PM (not as strong and consistent as with CRP) no association observed between E-selectin and PM an increase in prothrombin fragment 1+2 was consistently observed, particularly with lag 4 fibrinogen results revealed few significant associations, potentially due to chance D-dimer results revealed null associations in linear and logistic analyses

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Ruckerl et al. (2007, 091379) **Period of Study:** Oct 2000–Apr 2001 **Location:** Erfurt, Germany	**Outcome:** Soluble CD40 ligand (sCD40L), platelets, leukocytes, erythrocytes, hemoglobin **Age Groups:** 50+ yrs **Study Design:** Panel (12 repeated measures at 2-wk intervals) **N:** 57 male subjects with coronary disease **Statistical Analyses:** Fixed effects linear regression models **Covariates:** Long-term time trend, week-day of the visit, temperature, RH, baro-metric pressure **Season:** Time trend as covariate **Dose-response Investigated?** No **Statistical Package:** SAS v8.2 and S-Plus v6.0	**Pollutant:** PM_{10} **Averaging Time:** 24-h **Mean (SD):** 20.0 (13.0) **Percentiles:** 25: 10.8 50: 15.6 75: 26.0 **Range (Min, Max):** 5.4, 74.5 **Monitoring Stations:** 1 site **Copollutant:** UFPs (ultrafine particles), AP (accumulation mode particles), $PM_{2.5}$, PM_{10}, NO	**PM Increment:** IQR (15.2 5-d avg: 12.8) **Effect Estimate [Lower CI, Upper CI]:** Effects of air pollution on blood markers presented as % change from the mean/GM in the blood marker per increase in IQR air pollutant. **sCD40L, % change GM (pg/mL):** lag0: 1.6 (-3.5, 7.0) lag1: 1.1 (-5.4, 7.9) lag2: -3.5 (-8.9, 2.2) lag3: -1.4 (-6.0, 3.4) 5-d mean: -1.2 (-7.8, 5.8) **Platelets, % change mean (10^3/µl):** lag0: -0.4 (-1.9, 1.0) lag1: 0.4 (-1.4, 2.3) lag2: 0.5 (-1.4, 2.3) lag3: -0.1 (-1.6, 1.4) 5-d mean: 0.0 (2.1, 0.0) **Leukocytes, % change in mean (10^3/µl):** lag0: -1.1 (-2.8, 0.7) lag1: -0.5 (-2.6, 1.5) lag2: 0.1 (-2.1, 2.4) lag3: -0.7 (-2.6, 1.2) 5-d mean: -1.1 (-3.6, 1.4) **Erythrocytes, % change mean (10^6/µl):** lag0: 0.0 (-0.4, 0.5) lag1: -0.4 (-1.0, 0.1) lag2: -0.7 (-1.2, -0.2) lag3: -0.4 (-0.8, 0.0) 5-d mean: -0.6 (-1.2, -0.1) **Hemoglobin, % change mean (g/dl):** lag0: -0.1 (-0.7, 0.6) lag1: -0.4 (-1.2, 0.3) lag2: -0.7 (-1.3, 0.0) lag3: -0.3 (-0.9, 0.2) 5-d mean: -0.7 (-1.5, 0.1)
Reference: Steinvil et al. (2008, 188893) **Period of Study:** 2003-2006 **Location:** Tel-Aviv, Israel	**Outcome:** Inflammation **Age Groups:** Mean (SD): 46 (12) years **Study Design:** panel **N:** 3659 **Statistical Analyses:** Linear Regression **Covariates:** age, waist circumference, BMI, HDL, OLDL, triglycerides, diastolic & systolic BP, alcohol consumption, sports intensity, medications, smoking status, family history of CHD, temperature, humidity, precipitation, season, & year **Dose-response Investigated?** No **Statistical Package:** SPSS **Lags Considered:** 0-7d	**Pollutant:** PM_{10} **Averaging Time:** 24h **Mean (SD):** 64 (100.8) **25th:** 33.1 **50th:** 43.0 **75th:** 60.7 **Monitoring Stations:** NR **Copollutant:** SO_2, NO_2, O_3, CO **Co-pollutant Correlation** SO_2: 0.043 NO_2: 0.082 O_3: -0.113 CO: 0.075	**PM Increment:** Interquartile Range (27.6 $\mu g/m^3$) **hs-CRP Relative % Change (Lower CI, Upper CI):** Men: Lag 0: -1 (-2, 1) Lag 1: 0 (-1, 1); Lag 2: -1 (-2, 1) Lag 3: -1 (-2, 0) Lag 4: 0 (-1, 1) Lag 5: 0 (-1, 2) Lag 6: 1 (0, 2) Lag 7: 1 (0, 1) 0-7 avg: -2 (-5, 1) Women: Lag 0: 0 (-2, 2) Lag 1: 0 (-1, 2) Lag 2: 1 (0, 2) Lag 3: 0 (-1, 1) Lag 4: 0 (-1, 2) Lag 5: 0 (-1, 2) Lag 6: -1 (-3, 1) Lag 7: 0 (-2, 1) 0-7 avg: 1 (-2, 4) **Fibrinogen Absolute % Change (Lower CI, Upper CI):** Men: Lag0: 0.7(0.0,1.5); Lag1: 0.4(-0.2, 0.9); Lag2: -0.1(-0.9, 0.6) Lag3: -0.1(-0.7, 0.6); Lag4: 0.0(-0.7, 0.7); Lag5: 0.1(-0.7, 1.0) Lag6: 0.6(-0.1, 1.3); Lag7: 0.4(0.0, 0.8);

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			0-7 avg: -0.4(-1.9, 1.0) Women: Lag0: 0.3(-0.6, 1.2); Lag1: -0.1(-0.8, 0.7); Lag2: -0.3(-0.9, 0.3) Lag3: -0.1(-0.7, 0.5); Lag4: 0.2(-0.4, 0.9); Lag5: 0.2(-0.7, 1.2) Lag6: -0.3(-1.4, 0.8); Lag7: 0.7(-0.1, 1.5); 0-7 avg: 0.0(-1.5, 1.5) **WBC Absolute Change (Lower CI, Upper CI):** Men: Lag0: 2 (-22, 27) Lag1: 3 (-14, 19) Lag2: 1 (-22, 24) Lag3: -7 (-28, 14) Lag4: -22 (-44, -1) Lag5: -20 (-46, 7) Lag6: -5 (-27, 16) Lag7: -4(-16, 9) 0-7avg: -11(-58, 36) Women: Lag 0: 20 (-6, 46)
Reference: Su et al. (2006, 157022) **Period of Study:** February–April 2002 **Location:** Taipei, Taiwan	**Outcome:** Total cholesterol, HDL, tryglycerides, LDL, hs-CRP, IL-6, TNF-α, tPA, PAI-1, and fibrinogen **Age Groups:** 40-75 years **Study Design:** Panel study **N:** 49 subjects (31 males and 18 females) with coronary heart disease or multiple risk factors for CHD **Statistical Analysis:** Linear mixed effects regression	**Pollutant:** PM_{10} **Averaging Time:** 1 h (High pollution day = PM_{10} from 08: 00 to 18: 00 >100) **Copollutant:** O_3	**PM Increment:** High vs. Low pollution days **Effect Estimate [Lower CI, Upper CI]: CHD patients (n = 23):** P-value for paired t-test comparing health endpoint means on high and low pollution days hs-CRP: p = 0.568 IL-6: p = 0.856 TNF-α: p = 0.246 PAI-1: p = 0.008 tPA: p = 0.322 Fibrinogen: p = 0.189 P-value for health endpoint in mixed-effects models PAI-1: p = 0.010 tPA: p = 0.329 Fibrinogen: p = 0.747 **Patients with multiple CHD risk factors (n = 26):** P-value for paired t-test comparing health endpoint means on high and low pollution days hs-CRP: p = 0.475 IL-6: p = 0.561 TNF-α: p = 0.572 PAI-1: p = 0.098 tPA: p = 0.260 Fibrinogen: p = 0.087 P-value for health endpoint in mixed-effects models PAI-1: p = 0.891 tPA: p = 0.789 Fibrinogen: p = 0.923 **Notes:** Subjects had paired fasting blood samples taken during high and low air pollution days.

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Vedal et al., (2004, 055630) **Period of Study:** 1997-2000 **Location:** Vancouver, British Columbia	**Outcome:** Implantable cardioverter defibrillator (ICD) discharge **Age Groups:** All **Study Design:** Time series (Retrospective, longitudinal panel study) **N:** 50 ICD patients with 1+ discharges (40,328 person-days and 257 arrhythmia event days) **Statistical Analyses:** Multiple logistic regression with GEE **Covariates:** Temperature, relative humidity, barometric pressure, rainfall, wind direction and speed **Season:** Summer (May-Sep) and winter (Oct-Apr) **Dose-response Investigated:** No **Statistical Package:** NR **Lags Considered:** -3 days	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (min-max):** 12.9 (3.8-49.3) SD = 5.6 **Monitoring Stations:** 8 **Copollutant (correlation):** O_3: r = 0.11 SO_2: r = 0.70 NO_2: r = 0.49 CO: r = 0.43 **Other variables:** Temp: r = 0.43 Humidity: r = -0.35 Baro Pressure: r = 0.26 Rain: r = -0.63 Wind: r = -0.53	**PM Increment:** 5.6 $\mu g/m^3$ (SD) Percent Change [CI]: Values NR Notes: The author states that significant negative associations were found for ICD discharge with same-day lag, and also for 3-day lag with more arrhythmia-prone patients. All other non-significant percent change estimates are shown in Fig 3 and 4.
Reference: Vedal et al. (2004, 055630) **Period of Study:** 1997–2000 **Location:** Vancouver, British Columbia, Canada	**Outcome:** ICD discharges (arrhythmias) **N:** 150 patients w/ICD, 4 yrs **Statistical Analysis:** Logistic regression, GEE **Covariates:** Temporal trends, temperature, relative humidity, wind speed, rain **Season:** Summer, Winter **Dose-response Investigated?** No **Lags Considered:** 0.1.2.3d	**Pollutant:** PM_{10} **Mean:** 12.9 (SD = 5.6) **Copollutant):** O_3, SO_2, NO_2, CO	**Increment:** 1 SD Effect Estimates, e.g., % change in the rate of arrhythmia, were presented in Figure 3. No association with PM_{10} was observed while SO_2 was associated with an increase in the rate of arrhythmia among 16 patients with at least 2 discharges per year.

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Whitsel et al. (2009, 191980) **Period of Study:** 1993-2004 **Location:** US	**Outcome:** Heart Rate Variability **Age Groups:** 50-79 yrs **Study Design:** panel **N:** 4,295 women **Statistical Analyses:** Random Effects Model **Covariates:** temperature, humidity **Dose-response Investigated?** No **Statistical Package:** SUDAAN **Lags Considered:** 0	**Pollutant:** PM_{10} **Averaging Time:** 24h Amsterdam **Mean:** 20.0 **Min:** 3.8 **25th:** 10.4 **50th:** 16.9 **75th:** 23.9 **Max:** 82.2 Erfurt **Mean:** 23.1 **Min:** 4.5 **25th:** 10.5 **50th:** 16.3 **75th:** 27.4 **Max:** 118.1 Helsinki **Mean:** 12.7 **Min:** 3.1 **25th:** 8.1 **50th:** 10.6 **75th:** 16.0 **Max:** 39.8 **Monitoring Stations: 3** **Copollutant:** NR **Co-pollutant Correlation** n/a	**PM Increment:** 10 $\mu g/m^3$ **Beta (Lower CI, Upper CI):** Supine Position, Amsterdam Lag 0: -0.06 (-0.95, 0.84) Lag 1: 0.18 (-0.74, 1.10) Lag 2: 0.93 (0.01, 1.85) 5d avg: 0.49 (-0.74, 1.72) Supine Position, Erfurt Lag 0: -0.36 (-0.83, 0.11) Lag 1: -0.40 (-0.91, 0.11) Lag 2: -0.68 (-1.20, -0.17) 5d avg: -0.68 (-1.44, 0.09) Supine Position, Helsinki Lag 0: -0.44 (-2.27, 1.40) Lag 1: -0.17 (-1.69, 1.3.5) Lag 2: -1.14 (-2.51, 0.23) 5d avg: -0.59 (-3.08, 1.90) Supine Position, Pooled Lag 0: -0.30 (-0.71, 0.11) Lag 1: -0.25 (-0.68, 0.18) Lag 2: -0.26 (-1.22, 0.70)* 5d avg: -0.36 (-0.99, 0.27) Standing Position, Amsterdam Lag 0: -0.44 (-1.6, 0.72) Lag 1: -0.61 (-1.8, 0.59) Lag 2: 0.32 (-0.88, 1.51) 5d avg: -0.55 (-2.15, 1.04) Standing Position, Erfurt Lag 0: -0.59 (-1.24, 0.06) Lag 1: -0.70 (-1.42, 0.03) Lag 2: -0.65 (-1.37, 0.07) 5d avg: -0.68 (-1.74, 0.39) Standing Position, Helsinki Lag 0: 1.17 (-1.46, 3.80) Lag 1: 0.01 (-2.17, 2.19) Lag 2: -0.63 (-2.60, 1.34) 5d avg: -1.96 (-5.51, 1.60) Standing Position, Pooled Lag 0: -0.48 (-1.03, 0.07) Lag 1: -0.62 (-1.21, -0.03) Lag 2: -0.41 (-1.00, 0.17) 5d avg: -0.72 (-1.57, 0.14) *$p < 0.1$
Reference: Yeatts et al. (2007, 091266) **Period of Study:** 12 wk period b/t Sept 2003 – July 2004 **Location:** Chapel Hill, NC	**Outcome:** Heart Rate Variability **Age Groups:** 21-50 yrs **Study Design:** panel **N:** 12 asthmatics **Statistical Analyses:** Linear Mixed Model **Covariates:** temperature, humidity, pressure **Dose-response Investigated?** No **Statistical Package:** SAS **Lags Considered:** 1 day	**Pollutant:** PM_{10} **Averaging Time:** 24h **Mean (SD):** 17.5 (7.8) **Min:** 1.4 **Max:** 45.6 **Monitoring Stations:** 1 **Copollutant:** $PM_{2.5}$, $PM_{10-2.5}$ **Co-pollutant Correlation** $PM_{2.5}$ = 0.90* $PM_{10-2.5}$ = 0.73* *$p < 0.01$	**PM Increment:** 1 $\mu g/m^3$ **Beta, SE, p-value (Lower CI, Upper CI):** NR

[1]All units expressed in $\mu g/m^3$ unless otherwise specified.

Table E-2. Short-term exposure - cardiovascular morbidity studies: $PM_{10-2.5}$.

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Chuang et al. (2007, 091063) **Period of Study:** Nov 2002 – Mar 2003 **Location:** Taipei, Taiwan	**Outcome:** Heart Rate Variability **Age Groups:** 52-76 yrs **Study Design:** panel **N:** 10 CHD & 16 Hypertensive Patients **Statistical Analyses:** Linear Mixed Effects Model **Covariates:** age, sex, BMI, time of day, temperature, humidity, pressure, HRV **Dose-response Investigated?** No **Statistical Package:** S-PLUS **Lags Considered:** 1-4h ma	**Pollutant:** $PM_{10-2.5}$ **Averaging Time:** 1h among CHD, among hypertensive **Mean (SD):** 16.4 (10.7), 14.0 (11.1) **IQR:** 14.8, 11.9 **Min:** 0.7, 0.3 **Max:** 59.6, 66.5 **Monitoring Stations:** 1 personal monitor each **Copollutant:** $PM_{1.0-2.5}$, $PM_{0.3-1.0}$ **Co-pollutant Correlation** NR	**PM Increment:** Interquartile range **Percent Change (Lower CI, Upper CI):** Cardiac Patients- SDNN 1h moving: -1.73 (-3.53, 0.08) 2h moving: -1.97 (-4.43, 0.49) 3h moving: -1.70 (-4.39, 0.89) 4h moving: -1.75 (-5.42, 1.92) Cardiac Patients- r-MSSD 1h moving: -4.39 (-9.54, 0.03) 2h moving: -4.36 (-8.99, 0.27) 3h moving: -4.20 (-9.02, 0.61) 4h moving: -2.70 (-9.24, 3.84) Cardiac Patients- LF 1h moving: -1.85 (-4.33, 0.62) 2h moving: -3.87 (-8.22, 0.47 3h moving: -2.98 (-6.65, 0.69) 4h moving: -3.11 (-8.22, 1.99) Cardiac Patients- HF 1h moving: -4.46 (-9.23, 0.32) 2h moving: -4.41 (-9.55, 0.72) 3h moving: -3.80 (-9.12, 1.53) 4h moving: -3.39 (-10.62, 3.84) Cardiac Patients- LF: HF ratio 1h moving: 8.45 (-3.48, 20.38) 2h moving: 1.66 (-15.22, 18.55) 3h moving: 11.69 (-7.27, 30.64) 4h moving: 8.18 (-17.22, 33.57) Hypertensive Patients- SDNN 1h moving: -2.64 (-3.93, 0.55 2h moving: -3.51 (-7.87, 0.85) 3h moving: -2.74 (-6.22, 0.74) 4h moving: -2.49 (-6.13, 1.15) Hypertensive Patients- r-MSSD 1h moving: -2.53 (-5.10, 0.04) 2h moving: -5.42 (-10.92, 0.09) 3h moving: -3.15 (-6.32, 0.03) 4h moving: -4.23 (-8.88, 0.42) Hypertensive Patients- LF 1h moving: -4.38 (-8.78, 0.03) 2h moving: -5.23 (-10.95, 0.05) 3h moving: -3.34 (-1.72, 0.04) 4h moving: -2.96 (-6.63, 0.71) Hypertensive Patients- HF 1h moving: -4.92 (-9.94, 0.10) 2h moving: -6.07 (-12.28, 0.13) 3h moving: -1.94 (-5.44, 1.55) 4h moving: -2.78 (-6.78, 1.21) Hypertensive Patients- LF: HF ratio 1h moving: 5.94 (-3.27, 15.15) 2h moving: 10.70 (-2.19, 23.59) 3h moving: -1.51 (-17.02, 14.00) 4h moving: 3.41 (-16.91, 23.74) *$p < 0.05$

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Ebelt et al. (2005, 056907) **Period of Study:** Summer of 1998 **Location:** Vancouver, Canada	**Outcome:** CVD **Age Groups:** range from 54-86 yrs mean age = 74 years **Study Design:** extended analysis of a repeated-measures panel study **N:** 16 persons with COPD **Statistical Analyses:** Earlier analysis expanded by developing mixed-effect regression models and by evaluating additional exposure indicators **Dose-response Investigated?** No **Statistical Package:** SAS V8	**Pollutant:** $PM_{10-2.5}$ **Averaging Time:** 24 h **Mean (SD):** Ambient PM_{10}-2.5: 5.6 (3.0) Exposure to ambient PM_{10}-2.5: 2.4 (1.7) **Range (Min, Max):** Ambient $PM_{10-2.5}$: (-1.2-11.9) Exposure to ambient $PM_{10-2.5}$: (-0.4-7.2) **Monitoring Stations:** 5 **Copollutant (correlation):** Ambient concentrations and exposure to ambient PM were highly correlated for each respective metric: $r \geq 0.71$	**Note:** Total personal fine particle exposure (T) were dominated by exposures to non ambient particles which were not correlated with ambient fine particle exposure (A) or ambient concentrations (C). Results for each of these metrics are listed. **PM Increment:** Increment: $C^{10-2.5}$: IQR = 4.5 $\mu g/m^3$ SBP (mm Hg): -2.12 (-5.07-0.82) DBP (mm Hg): -0.92 (-3.37-0.36) Ln-SVE (bph): 0.06 (-0.24-0.36) HR (bpm): 1.09 (-0.69-2.86) SDNN (ms): 2.64 (-2.85-8.13) R-MSSD (ms): -0.33 (-4.49-3.82) Increment: $A^{10-2.5}$: IQR = 2.4 $\mu g/m^3$ SBP (mm Hg): -2.55 (-6.15-1.05) DBP (mm Hg): -0.75 (-3.50-2.01) Ln-SVE (bph): 0.26 (-0.07-0.58) HR (bpm): 1.04 (-0.95-3.03) SDNN (ms): 0.68 (-3.07-4.42) R-MSSD (ms): 1.10 (-3.08-5.28)
Reference: Lipsett et al. (2006, 088753) **Period of Study:** February–May 2000 **Location:** Coachella Valley, CA	**Outcome:** HRV parameters, specifically SDNN, SDANN, r-MSSD, LF, HF, total power, triangular index (TRII). **Study Design:** Panel study **N:** 19 non-smoking adults with coronary artery disease **Statistical Analysis:** Mixed linear regression models with random effects parameters	**Pollutant:** PM_{10}-2.5 **Averaging Time:** 2 h **Monitoring Stations:** 2 **Copollutant:** O_3	**PM Increment:** SE*1000 **Effect Estimate (change in HRV per unit increase in PM concentration):** SDNN: -0.72 msec (SE = 0.296) **Notes:** $PM_{10-2.5}$ calculated by subtracting $PM_{2.5}$ concentration from PM_{10} concentration. Weekly ambulatory 24 h ECG recordings (once per week for up to 12 weeks), using Holter monitors, were made. Subjects' residences were within 5 miles of one of two PM monitoring sites. Regressed HRV parameters against 18:00–20:00 mean particulate pollution
Reference: Metzger et al. (2007, 092856) **Period of Study:** August 1998–December 2002 **Location:** Atlanta, GA	**Outcome:** Days with any event recorded by the ICD, days with ICD shocks/defibrillation and days with either cardiac pacing or defibrillation **Study Design:** Repeated measures **N:** 884 subjects between 1993 and 2002 **Statistical Analysis:** Logistic regression with GEE to account for residual autocorrelation within subjects	**Pollutant:** PM_{10} -2.5 (n/cm^3) **Averaging Time:** 24-hs **Mean (SD):** 9.6 (5.4) **Median:** 8.7 **Copollutant:** O_3, NO_2, CO, SO_2, oxygenated hydrocarbons	**PM Increment: OR (95% CI):** OR = 1.03 (95% CI: 1.00, 1.07)
Reference: Pekkanen et al. (2002, 035050) **Period of Study:** Winter 1998 to 1999 **Location:** Helsinki, Finland	**Outcome:** ST Segment Depression (> 0.1mV) **Study Design:** Panel of ULTRA Study participants **N:** 45 subjects, 342 biweekly submaximal exercise tests, 72 exercise induced ST Segment Depressions **Statistical Analysis:** Logistic regression / GAM	**Pollutant:** PM_{10} -2.5 (n/cm^3) **Averaging Time:** 24 h **Median:** 4.8 **IQR:** 5.5 **Monitoring Stations:** 1 **Copollutant:** NO_2, CO, $PM_{2.5}$, PM1, ACP, ultrafine	**PM Increment:** IQR **Effect Estimate(s):** PM_{10}-2.5: OR = 1.99 (0.70, 5.67), lag 2 **Notes:** The effect was strongest for ACP and $PM_{2.5}$, which in two pollutant models appeared independent. Increases in NO_2 and CO were also associated with increased risk of ST segment depression, but not with coarse particles.
Reference: Timonen et al. (2006, 088747) **Period of Study:** 1998–1999 **Location:** Amsterdam, Netherlands Erfurt, Germany Helsinki, Finland	**Outcome:** HRV measurements: [LF, HF, LFHFR, NN interval, SDNN, r-MSSD] **Study Design:** Panel study **N:** 131 elderly subjects with stable coronary heart disease **Statistical Analysis:** Linear mixed models	**Pollutant:** PM_{10}-2.5 **Means:** Amsterdam: 15.3 Erfurt: 3.7 Helsinki: 6.7 **Copollutant:** NO_2, CO	**PM Increment:** 10 $\mu g/m^3$ **Effect Estimate:** SDNN 0.69ms (95% CI: -1.24, 2.63) HF: 2.9% (95% CI: -7.3, 13.1) LFHFR: -3.3 (95% CI: -12.7, 6.1) **Notes:** Followed for 6 months with biweekly clinic visits 2 day lag. ULTRA Study

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Yeatts et al. (2007, 091266) **Period of Study:** 12 wk period b/t Sept 2003 – July 2004 **Location:** Chapel Hill, NC	**Outcome:** Heart Rate Variability **Age Groups:** 21-50 yrs **Study Design:** panel **N:** 12 asthmatics **Statistical Analyses:** Linear Mixed Model **Covariates:** temperature, humidity, pressure **Dose-response Investigated?** No **Statistical Package:** SAS **Lags Considered:** 1 day	**Pollutant:** $PM_{10-2.5}$ **Averaging Time:** 24h **Mean (SD):** 5.3 (2.8) **Min:** 0 **Max:** 14.6 **Monitoring Stations:** 1 **Copollutant:** $PM_{2.5}$, PM_{10} **Co-pollutant Correlation** $PM_{2.5}$ = 0.46* PM_{10} = NR *$p < 0.01$	**PM Increment:** 1 $\mu g/m^3$. **Beta, SE (Lower CI, Upper CI), p-value** HRV Max Heart Rate: -1.95, 0.88 (-3.67, -0.23), 0.03 ASDNN5: -0.77, 0.37 (-1.580, -0.04), 0.05 SDANN5: -3.76, 1.53 (-6.76, -0.76), 0.02 SDNN24HR(mesc): -3.36, 1.38 (-6.06, -0.65), 0.02 rMSSD: -0.75, 0.53 (-1.79, 0.28), 0.16 pNN50_24hour: -0.50, 0.27 (-1.03, 0.03), 0.07 pNN50_7min: -1.88, 0.55 (-2.95, -0.81), 0.07 Low-frequency power: -0.19, 0.42 (-1.01, 0.63), 0.65 Percent low frequency: 0.57, 1.08 (-1.55, 2.69), 0.60 High-frequency power: -0.46, 0.17 (-0.79, -0.14), 0.01 Percent high frequency: -2.14, 0.94 (-3.98, -0.30), 0.03 Blood Lipids Triglycerides: 4.78, 2.02 (0.81, 8.74), 0.02 VLDL: 1.15, 0.44 (0.29, 2.02), 0.01 Total cholesterol: 0.78, 0.54 (-0.28, 1.84), 0.15 Hematologic Factors Circulating eosinophils: 0.16, 0.06 (0.04, 0.28), 0.01 Platelets: -1.71, 1.11 (-3.89, 0.47), 0.13 Circulating Proteins Plasminogen: -0.01, 0.01 (-0.02, 0.00), 0.08 Fibrenogen: -0.04, 0.02 (-0.08, 0.00), 0.07 Von Willibrand factor: -1.23, 0.66 (-2.53, 0.06), 0.07 Factor VII: -0.90, 0.85 (-2.58, 0.77), 0.29

[1]All units expressed in $\mu g/m^3$ unless otherwise specified.

Table E-3. Short-term exposure - cardiovascular morbidity studies: $PM_{2.5}$ (including PM components/sources).

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Adar et al. (2007, 001458) **Period of Study:** Mar–Jun 2002 **Location:** St. Louis, Missouri	**Outcome:** Heart rate variability: heart rate, standard deviation of all normal-to-normal intervals (SDNN), square root of the mean squared difference between adjacent normal-to-normal intervals (rMSSD), percentage of adjacent normal-to-normal intervals that differed by more than 50 ms (pNN50), high frequency power (HF in the range of 0.15-0.4Hz), low frequency power (LF, in the range of 0.04-0.15Hz), and the ratio of LF/HF **Age Groups:** ≥ 60 yrs **Study Design:** Panel (4 planned repeated measures surrounding bus trips with a total of 158 person-trips 35 participating in all 4 trips)	**Pollutant:** $PM_{2.5}$ ($\mu g/m^3$) **Averaging Time:** Measurements collected over 48 h period surrounding the bus trip (during which health endpoints were measured) used to calculate 5-, 30-, 60-minute, 4-h, 24-h moving averages **Median (IQR):** All: 7.7 (6.8) Facility: 6.8 (5.1) Bus: 17.2 (10.3) Activity: 8.2 (16.1) Lunch: 11.2 (5.9) **Monitoring Stations:** 2 portable carts **Copollutant:** $PM_{2.5}$ BC Fine particle counts coarse particle counts **Correlation notes:** 24-h mean $PM_{2.5}$, BC, and fine particle count concentrations	**PM Increment:** IQR **Effect Estimate [Lower CI, Upper CI]:** % change (95%CI) in HRV per IQR in the 24-h moving avg of the microenvironmental pollutant (IQr = 4.5 $\mu g/m^3$) **Single-pollutant models:** SDNN: -5.5 (-6.3, -4.8) rMSSD: -9.1 (-9.8, -8.4) pNN50 + 1: -12.2 (-13.3, -11.1). LF: -10.8 (-12.3, -9.3) HF: -15.1 (-16.7, -13.7) LF/HF: 5.1 (3.9, 6.4) H: 1.0 (0.9, 1.2) **Two-pollutant models (with particle number count coarse):** SDNN: -5.7 (-6.5, -4.9) rMSSD: -9.4(-10.1, -8.6) pNN50+1: -13.1(-14.3, -11.9). LF: -

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
	N: 44 participants	ranged from 0.80 to 0.98	10.7(-12.4, -9.1)
	Statistical Analyses: Generalized additive models	r = 0.76 to 0.97 when limited to time spent on the bus	HF: -14.9(-16.5, -13.3); LF/HF: 4.9 (3.6, 6.2)′ H: 0.9 (0.7,1.1)
	Covariates: Subject, weekday, time, apparent temperature, trip type, activity, medications, and autoregressive terms	r = 0.55 to 0.86 when comparing bus concentrations to 24-h moving averages	Independent short- and medium-term associations with HRV across all time periods
	Season: Limited data collection period	r = -0.003 to 0.51 when comparing 5-min averages and 24-h moving averages	% change per IQR (95%CI)
	Dose-response Investigated? No	Poor correlations found between coarse particle count concentrations and all fine particulate measures during all times periods	IQR 5-min means = 6.8 μg/m^3 and 23: 55-h means = 4.2 μg/m^3
	Statistical Package: SAS v8.02, R v2.0.1		SDNN: 5-min mean: -0.5 (-0.8, -0.1)
			23: 55-h mean: -4.6 (-5.3, -4.0)
			rMSSD: 5-min mean: -0.9 (-1.3, -0.5)
			23: 55-h mean: -7.5 (-8.1 to -6.8)
			pNN50 + 1
			5-min mean: -1.1 (-1.7 to -0.5)
			23: 55-h mean: -9.9 (-10.9 to -8.9). LF
			5-min mean: 0.4 (-0.5, 1.2)
			23: 55-h mean: -10.0 (-11.4 to -8.6)
			HF
			5-min mean: -1.5 (-2.3 to -0.6)
			23: 55-h mean: -12.9 (-14.2 to -11.5)
			LF/HF
			5-min mean: 1.9 (1.3, 2.4)
			23: 55-h mean: 3.2 (2.1, 4.3)
			H: 5-min mean: 0.1 (0.1, 0.2)
			23: 55-h mean: 0.8 (0.7, 0.9)
			Independent associations of short-term averages (5-min means) of PM with HRV by bus and nonbus periods
			IQR for bus = 10 μg/m^3) and nonbus = 5.6 μg/m^3)
			% change (95%CI)
			p-value of interaction
			SDNN
			Bus: -5.0 (-6.3 to -3.7)
			Nonbus: -0.5 (-0.9 to -0.2)
			p-value for interaction: $<$ 0.0001. rMSSD
			Bus: -4.8 (-6.2 to -3.5)
			Nonbus: -0.7 (-1.1 to -0.4. p-value for interaction: $<$ 0.0001
			pNN50 + 1
			Bus: -6.3 (-8.4 to -4.2)
			Nonbus: -0.8 (-1.4 to -0.3)
			p-value for interaction: $<$ 0.0001
			LF: Bus: -7.0 (-9.8 to -4.1) Nonbus: 0.6 (-0.1, 1.4)
			p-value for interaction: $<$ 0.0001. HF: Bus: -10.7 (-13.5 to -7.9)′ Nonbus: -0.7 (-1.5, 0.04) p-value for interaction: $<$ 0.0001. LF/HF: Bus: 3.9 (1.7, 6.0)

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			Nonbus: 1.4 (0.8, 1.9) p-value for interaction: 0.39. H: Bus: 0.7 (0.5, 1.0) Nonbus: -0.01 (-0.08, 0.1) p-value for interaction: $<$ 0.0001 **Note:** Exposure to health associations by all lag periods presented in Figure 2 (magnitude of associations increased with averaging period, with the largest associations consistently found for 24-h moving averages)
Reference: Adar et al. (2007, 001458) **Period of Study:** March–Jun 2002 **Location:** St. Louis, Missouri	**Outcome:** Heart rate variability: heart rate, standard deviation of all normal-to-normal intervals (SDNN), square root of the mean squared difference between adjacent normal-to-normal intervals (rMSSD), percentage of adjacent normal-to-normal intervals that differed by more than 50 ms (pNN50), high frequency power (HF in the range of 0.15-0.4Hz), low frequency power (LF, in the range of 0.04-0.15Hz), and the ratio of LF/HF **Age Groups:** ≥ 60 yrs **Study Design:** Panel (4 planned repeated measures with a total of 158 person-trips 35 participating in all 4 trips) **N:** 44 participants **Statistical Analyses:** Generalized additive models **Covariates:** Subject, weekday, time, apparent temperature, trip type, activity, medications, and autoregressive terms **Season:** Limited data collection period **Dose-response Investigated?** No **Statistical Package:** SAS v8.02, R v2.0.1	**Pollutant:** BC (ng/m^3) **Averaging Time:** Measurements collected over 48 h period surrounding the bus trip (during which health endpoints were measured) used to calculate 5-, 30-, 60-minute, 4-h, 24-h moving averages **Median (IQR):** All: 330 (337) Facility: 285 (270) Bus: 2911 (2464) Activity: 482 (1168) Lunch: 434 (276) **Monitoring Stations:** 2 portable carts **Copollutant:** $PM_{2.5}$ BC Fine particle counts Coarse particle counts **Correlation notes:** 24-h mean $PM_{2.5}$, BC, and fine particle count concentrations ranged from 0.80 to 0.98 r = 0.76 to 0.97 when limited to time spent on the bus r = 0.55 to 0.86 when comparing bus concentrations to 24-h moving averages r = -0.003 to 0.51 when comparing 5-min averages and 24-h moving averages Poor correlations found between coarse particle count concentrations and all fine particulate measures during all times periods	**PM Increment:** IQR **Effect Estimate [Lower CI, Upper CI]:** % change (95%CI) in HRV per IQR in the 24-h moving avg of the microenvironmental pollutant (IQr = 459 ng/m^3) Single-pollutant models SDNN: -5.3 (-6.5 to -4.1) rMSSD: -10.7 (-11.9 to -9.5) pNN50 + 1: -13.2 (-15.0 to -11.4) LF: -11.3 (-13.7 to -8.8) HF: -18.8 (-21.1 to -16.5) LF/HF: 9.3 (7.2, 11.4) H: 1.0 (0.8, 1.3) Independent short- and medium-term associations with HRV across all time periods % change per IQR (95%CI) IQR 5-min means = 337 ng/m^3 and 23: 55-h means = 490 ng/m^3) SDNN: 5-min mean: -0.3 (-0.5 to -0.1) 23: 55-h mean: -4.7 (-5.9 to -3.5) rMSSD: 5-min mean: -0.3 (-0.5 to -0.1) 23: 55-h mean: -9.3 (-10.5 to -8.1) pNN50 + 1: 5-min mean: -0.3 (-0.6 to -0.1) 23: 55-h mean: -10.5 (-12.3 to -8.7) LF: 5-min mean: -0.5 (-0.9 to -0.1) 23: 55-h mean: -9.8 (-12.4 to -7.2) HF: 5-min mean: -0.9 (-1.2 to -0.5) 23: 55-h mean: -15.4 (-17.8 to -12.9) LF/HF: 5-min mean: 0.3 (0.1, 0.6) 23: 55-h mean: 6.5 (4.5, 8.6) H: 5-min mean: 0.1 (0.1, 0.2) 23: 55-h mean: 0.4 (0.2, 0.7) Independent associations of short-term averages (5-min means) of PM with HRV by bus and nonbus periods IQR for bus = 2.6 µg/m^3) and nonbus = 0.27 µg/m^3) % change (95%CI) p-value of interaction SDNN: Bus: -4.6 (-6.1 to -3.0)' Nonbus: -0.1 (-0.3, 0.1) p-value for interaction: $<$ 0.0001

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			rMSSD: Bus: -2.6 (-4.2 to -0.9): Nonbus: -0.3 (-0.5 to -0.1) p-value for interaction: 0.64 pNN50 + 1: Bus: -2.0 (-4.5, 0.5): Nonbus: -0.5 (-0.8 to -0.1) p-value for interaction: 0.34 LF: Bus: -6.0 (-9.3 to -2.5): Nonbus: -0.2 (-0.7, 0.3) p-value for interaction: 0.028 HF: Bus: -5.8 (-9.1 to -2.3) Nonbus: -0.9 (-1.4 to -0.4) p-value for interaction: 0.50 LF/HF: Bus: -0.8 (-3.1, 1.7) Nonbus: 0.8 (0.5, 1.1) p-value for interaction: $<$ 0.0001 H: Bus: -0.5 (-0.8 to -0.2) Nonbus: 0.3 (0.26, 0.34) p-value for interaction: $<$ 0.0001 **Note:** Exposure to health associations by all lag periods presented in Figure 2 (magnitude of associations increased with averaging period, with the largest associations consistently found for 24-h moving averages)
Reference: Auchincloss et al. (2008, 156234) **Period of Study:** Jul 2000–Aug 2002 **Location:** 6 US communities (Baltimore City and Baltimore County, Maryland Chicago, Illinois Forsyth County, North Carolina Los Angeles, California Northern Manhattan and the Bronx, New York and St. Paul, Minnesota) part of MESA (Multi-ethnic Study of Atherosclerosis)	**Outcome:** Blood pressure: systolic (SBP), diastolic (DBP), mean arterial (MAP), pulse pressure (PP) Avg of 2nd and 3rd BP measurement used for analyses **Age Groups:** 45-84 years **Study Design:** Cross-sectional (Multi-Ethnic Study of Atherosclerosis baseline examination) **N:** 5,112 persons (free of clinically apparent cardiovascular disease) **Statistical Analyses:** Linear regression secondary analyses used log binomial models to fit a binary hypertension outcome **Covariates:** Age, sex, race/ ethnicity, per capita family income, education, BMI, diabetes status, cigarette smoking status, exposure to ETS, high alcohol use, physical activity, BP medication use, meteorology variables, and copollutants examined site as a potential confounder and effect modifier heterogeneity of effects also examined by traffic-related exposures, age, sex, type 2 diabetes, hypertensive status, cigarette use, by levels of SO_2 and CO, and for weather variables **Season:** Adjusted for temperature and barometric pressure to adjust for seasonality (because seasons vary by the study sites) Also performed sensitivity analyses adjusting for season to examine the potential for residual confounding not accounted for by weather variables	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 5 exposure metrics constructed: prior day, avg of prior 2 days, prior 7 days, prior 30 days, and prior 60 days **Mean (SD):** Prior day: 17.0 (10.5) Prior 2 days: 16.8 (9.3) Prior 7 days: 17.0 (6.9) Prior 30 days: 16.8 (5.0) Prior 60 days: 16.7 (4.4) **Percentiles:** NR **Range (Min, Max):** NR **Monitoring Stations:** Used monitor nearest the participant's residence to calculate exposure metrics **Copollutant:** SO_2 NO_2 CO Traffic-related exposures (straight-line distance to a highway total road length around a residence) **Correlations with $PM_{2.5}$ averaged over prior 30 days:** O_3 Cool: r = -0.67 Moderate: r = -0.30 Warm: r = 0.23 CO Cool: r = 0.20 Moderate: r = 0.71 Warm: r = 0.23 SO_2 Cool: r = 0.36 Moderate: r = -0.17	**PM Increment:** 10 $\mu g/m^3$ (approx. equivalent to difference between 90th and 10th percentile for prior 30 day mean) **Effect Estimate [Lower CI, Upper CI]:** Adjusted mean difference (95% CI) in PP and SBP (mmHg) per 10 $\mu g/m^3$ increase in $PM_{2.5}$ (averaged for the prior 30 days) **Pulse Pressure** ($PM_{2.5}$ averaged for prior 30 days) Adjustment variables: Person-level Covariates: 1.04 (0.25, 1.84), p = 0.010 Person-level cov., weather: 1.12 (0.28, 1.97), p = 0.009 Person-level cov., weather, gaseous copollutants: 2.66 (1.61, 3.71), p = 0.000 Person-level cov., study site: 0.93 (-0.04, 1.90), p = 0.060 Person-level cov., study site, weather: 1.11 (0.01, 2.22), p = 0.049 Person-level cov., study site, weather, gaseous copollutants: 1.34 (0.10, 2.59) , p = 0.035 **Systolic Blood Pressure** Adjustment variables: Person-level Covariates: 0.66 (-0.41, 1.74) , p = 0.226 Person-level cov., weather: 0.99 (-0.15, 2.13) , p = 0.089 Person-level cov., weather, gaseous

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
	Dose-response Investigated? Assessed nonlinear relationships–no evidence of strong threshold/nonlinear effects for $PM_{2.5}$ **Statistical Package:** NR	Warm: r = -0.11 NO_2 Cool: r = 0.55 Moderate: r = 0.66 Warm: 0.32	copollutants: 2.8 (1.38, 4.22) , p = 0.000 Person-level cov., study site: 0.86 (-0.45, 2.17) , p = 0.200 Person-level cov., study site, weather: 1.32 (-0.18, 2.82) , p = 0.085 Person-level cov., study site, weather, gaseous copollutants: 1.52 (-0.16, 3.21) , p = 0.077 **Additional results:** Associations became stronger with longer averaging periods up to 30 days. For example: Adjusted (personal covariates and weather) mean differences in PP: Prior day: -0.38 (-0.76, 0.00) Prior 2 days: -0.22 (-0.65, 0.21) Prior 7 days: 0.52 (-0.08, 1.11) Prior 30 days: 1.12 (0.28, 1.97) Prior 60 days: 1.08 (0.11, 2.05) (Pattern held for additional adjustments and for SBP results therefore, only results for 30-day mean differences were presented) **Additional results (not presented):** None of DBP results were statistically significant results for MAP were similar to SBP, though weaker and generally not significant **Effect modification:** associations between $PM_{2.5}$ and BP were stronger for persons taking medications, with hypertension, during warmer weather, in the presence of high NO_2, residing ◘ 300m from a highway, and surrounded by a high density of roads (Fig 1) associations were not modified by age, sex, diabetes, cigarette smoking, study site, high levels of CO or SO_2, season , nor residence ◘ 400m fro a highway **Note:** supplementary material available on-line shows results for DBP and MAP, among others
Reference: Baccarelli et al. (2009, 188183) **Period of Study:** Nov 2000 – Jun 2005 **Location:** Boston, Mass	**Outcome:** Heart rate variability **Age Groups:** elderly **Study Design:** Panel **N:** 549 men **Statistical Analyses:** Mixed-effects model **Covariates:** age, past/current CHD, BMI, mean arterial pressure, fasting blood glucose, smoking, alcohol consumption, use of beta-blockers, CA channel blockers, angiotensin-converting enzyme inhibitors, room temperature, season, apparent temperature **Season:** no **Dose-response Investigated?** No **Statistical Package:** SAS	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 48 h moving average **Geometric Mean (95%CI):** All Visits: 10.5 (10.0, 10.9) Visits w/ Genotype Data: 10.4 (9.9, 11.0) Visits w/o Genotype Data: 10.5 (9.8, 11.4) **Monitoring Stations:** 1 **Copollutant:** NR **Correlation:** n/a	**PM Increment:** 10 $\mu g/m^3$ **Percent Change [Lower CI, Upper CI], P:** All Subjects w/ Genotype Data SDNN: -6.0 (-13.5, 2.0), 0.14 HF: -17.1 (-32.3, 1.6), 0.07 LF: -8.2 (-22.1, 8.2), 0.31 All Subjects SDNN: -7.1 (-13.2, -0.6), 0.03 HF: -18.7 (-31.1, -4.0), 0.01 LF: -11.8 (-23.2, -1.3), 0.08

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Barclay et al. (2007, 192229) **Period of Study:** Jan 2003 – May 2005 **Location:** Aberdeen, Scotland	**Outcome:** Haematological outcomes, Heart Rhythm outcomes, & Heart Rate Variability outcomes **Age Groups:** 70.4 (8.9) **Study Design:** panel **N:** 132 patients w/ chronic heart failure **Statistical Analyses:** Linear & Mixed Effects Regression Model **Covariates:** age, temperature, humidity, pressure **Dose-response Investigated?** No **Statistical Package:** NR **Lags Considered:** lags 0-2d	**Pollutant:** $PM_{2.5}$ **Averaging Time:** daily **Mean:** 7.454 **Min:** 1.092 **Max:** 21.97 **Monitoring Stations:** 0 **Copollutant:** PM_{10}, PNC, NO_2 **Co-pollutant Correlation** NO_2 city: 0.164 NO city: 0.048 PM_{10} city: 0.476* NO_2 personal: 0.169 PNC DEOM: 0.115 $PM_{2.5}$ traffic: 0.522* PNC total: 0.367* PNC traffic: 0.234 *correlations based on 3-day average concentrations **Notes:** $PM_{2.5}$ values model predicted	**PM Increment:** NR **Beta (Lower CI, Upper CI):** Haemoglobin: -0.509 (-1.560, 0.542) Mean corpuscular haemoglobin: 0.188 (-0.481, 0.857) Platelets: 3.022 (0.403, 5.642) Haematocrit: -0.813 (-1.892, 0.267) White blood cells: -1.652 (-4.727, 1.424) C reactive protein: 4.924 (-13.022, 22.869) IL-6: -5.980 (-23.649, 11.690) von Willebrand factor: 1.363 (-6.561, 9.287) E-selectin: 2.136 (-2.946, 7.217) Fibrinogen: -5.579 (-10.403, -0.755)* Factor VII: 3.747 (-1.959, 9.452) d-dimer: 5.211 (-2.974, 13.397) All arrhythmias: -7.082 (-28.789, 14.626) Ventricular ectopic beats: -12.203 (-39.021, 14.615) Ventricular couplets: -1.255 (-25.678, 23.168) Ventricular runs: -2.548 (-17.448, 12.351) Supraventricular ectopic beats: 4.898 (-19.772, 29.568) Supraventricular couplets: 6.138 (-16.242, 28.518) Supraventricular runs: -0.545 (-17.577, 16.487) Average HR: 0.617 (-0.782, 2.016) 24h SDNN: 3.645 (-0.227, 7.517) 24h SDANN: 4.437 (0.030, 8.844)* 24h RMSSD: 0.617 (-0.782, 2.016) 24h PNN 50%: 11.247 (-6.228, 28.722) 24h LF power: 4.439 (-6.823, 15.701) 24h LF normalized: -5.659 (-11.815, 0.497) 24h HF power: 3.800 (-10.863, 18.464) 24h HF normalized: -6.597 (-13.724, 0.531) 24h LF/HF ratio: 1.033 (-8.355, 10.414) *$p < 0.05$ **Notes:** Estimates also available for $PM_{2.5}$ traffic LF= low frequency HF= high frequency
Reference: Briet et al. (2007, 093049) **Period of Study:** NR **Location:** Paris, France	**Outcome:** Endothelial Function **Age Groups:** 20-40 yrs **Study Design:** panel **N:** 40 white male nonsmokers **Statistical Analyses:** Multiple Robust Regrssion **Covariates:** R53R/R53H genotype, diet, subject factor, visit, temperature **Dose-response Investigated?** No **Statistical Package:** NCSS **Lags Considered:** 0-5d	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24h **5 d Mean (SD):** 28 (6) **Monitoring Stations:** NR **Co-pollutant:** PM_{10}, SO_2, NO, NO_2, CO **Co-pollutant Correlation** n/a	**PM Increment:** 1 SD **Beta (Lower CI, Upper CI), *P*, R^2:** Flow-mediated brachial artery dilation: -0.32 (-1.10, 0.46), NS, 0.04 Reactive hyperemia: 15.68 (7.11, 23.30), < 0.0001, 0.24 Changes in Endothelial function b/t visits: 1.98 (0.67, 3.259), 0.004, 0.44

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Cárdenas et al. (2008, 191900) **Period of Study:** NR **Location:** Mexico City, Mexico	**Outcome:** Heart Rate Variability **Age Groups:** 20-40 yrs **Study Design:** panel **N:** 54 subjects **Statistical Analyses:** Linear GEE models **Covariates:** localization, supine position, gender, age, humidity, heart rate, orthostatic position, head-up tilt test result **Dose-response Investigated?** No **Statistical Package:** NR **Lags Considered:** NR	**Pollutant:** $PM_{2.5}$ **Averaging Time:** NR **25th, 50th, 75th percentile:** Indoor: 14.8, 28.3, 47.9 Outdoor: 6.4, 10.8, 16.8 **Monitoring Stations:** NR **Co-pollutant:** NR **Co-pollutant Correlation** n/a	**PM Increment:** NR **Mean Difference (Lower CI, Upper CI), lag:** Ln low frequency Indoors: -0.028 (-0.0423, -0.0138) Outdoors: -0.194 (-0.4509, 0.0627) Ln high frequency Indoors: -0.019 (-0.0338, -0.0044) Outdoors: -0.298 (-0.5553, -0.0401) Ln LF/HF ratio Indoors: -0.017 (-0.0330, -0.0007) Outdoors: -0.278 (-0.5540, 0.0030)
Reference: Cavallari et al. (2007, 157425) **Period of Study:** 1999-2006 **Location:** Massachusetts	**Outcome:** Heart Rate Variability **Age Groups:** 22-63 **Study Design:** panel **N:** 36 males **Statistical Analyses:** Mixed Effects Regression Model **Covariates:** age, smoking, heart rate at work **Dose-response Investigated?** No **Statistical Package:** SAS **Lags Considered:** lags 0-14h	**Pollutant:** $PM_{2.5}$ **Averaging Time:** hourly **Mean (SD):** 1.12 (0.76) **Min:** 0.12 **Max:** 3.99 **Monitoring Stations:** NR **Copollutant:** NR **Co-pollutant Correlation** n/a	**PM** 1 mg **Increment:** $/m^3$ **Beta (Lower CI, Upper CI):** Model 1 Lag1h: -1.44 (-7.75, 4.87) Lag2h: -5.33 (-10.97, 0.31)* Lag3h: -6.86 (-11.91, -1.81)‡ Lag4h: -2.17 (-9.33, 4.99) Lag5h: -4.73 (-11.99, 2.53) Lag6h: -3.52 (-9.89, 2.84) Lag7h: -1.59 (-7.53, 4.35) Lag8h: -0.72 (-7.63, 6.20) Lag9h: -5.55 (-10.65, -0.45)‡ Lag10h: -3.66 (-8.85, 1.53) Lag11h: -8.60 (-17.45, 0.24)* Lag12h: -5.98 (-14.67, 2.70) Lag13h: -8.27 (-17.00, 0.46)* Lag14h: -4.19 (-12.71, 4.33) Model 2 Lag1h: 4.10 (-0.39, 8.60)* Lag2h: -3.21, (-8.78, 2.37) Lag3h: -6.45 (-11.59, -1.31)‡ Lag4h: -0.01 (-6.96, 6.94) Lag5h: -2.03 (-8.27, 4.22) Lag6h: -1.99 (-8.46, 4.48) Lag7h: -0.34 (-6.22, 5.54) Lag8h: 0.72 (-6.35, 7.78) Lag9h: -5.26 (-10.62, 0.11)* Lag10h: -3.68 (-9.17, 1.80) Lag11h: -9.41 (-18.60, -0.23)‡ Lag12h: -6.45 (-15.62, 2.72) Lag13h: -7.33 (-16.55, 1.89) Lag14h: -4.75 (-13.81, 4.32) *$p < 0.05$, ‡$p < 0.10$ Notes: Model 1 adjusted for smoking status & age only. Model 2 adjusted for smoking status, age, & heart rate during work.

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Chahine et al. (2007, 156327) **Period of Study:** Jan 2000 – Jun 2005 **Location:** Boston, MA	**Outcome:** Heart Rate Variability **Age Groups:** mean 72.8(6.6) yrs **Study Design:** panel **N:** 539 white males **Statistical Analyses:** Mixed Effects Model **Covariates:** age, BMI, mean arterial pressure, fasting blood glucose, smoking, alcohol consumption, use of beta-blockers, calcium channel blockers, ACE inhibitors, room temperature, season, outdoor temperature **Dose-response Investigated?** No **Statistical Package:** SAS **Lags Considered:** 0-2d ma	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 1h **Mean (SD):** 11.7 (7.8) **Monitoring Stations:** 1 **Copollutant:** $PM_{1.0}$ **Co-pollutant Correlation** n/a	**PM Increment:** 10 $\mu g/m^3$ **Percent Change (Lower CI, Upper CI), p-value:** log10 SDNN Total: -6.8 (-12.9, -0.2), 0.0436 GSTM1 wildtype: -2.0 (-11.3, 8.3), 0.6908 GSTM1 null: -10.5 (-18.2, -2.2), 0.0150 HMOX-1 < 25 repeats: 7.4 (-8.7, 26.2), 0.3891 HMOX-1 ≥25 repeats: -8.5 (-14.8, -1.8), 0.0137 log10 HF Total: -17.3 (-30.0, -2.3), 0.0263 GSTM1 wildtype: -4.0 (-24.8, 22.6), 0.7442 GSTM1 null: -24.2 (-39.2, -5.5), 0.0139 HMOX-1 < 25 repeats: 8.9 (-27.1, 62.8), 0.6759 HMOX-1 ≥25 repeats: -20.1 (-32.9, -5.0), 0.0115 log10 LF Total: -11.2 (-22.8, 2.2), 0.0986 GSTM1 wildtype: -0.6 (-19.0, 22.0), 0.9545 GSTM1 null: -17.0 (-31.0, -0.2), 0.0478 HMOX-1 < 25 repeats: 14.0 (-18.6, 59.5), 0.4465 HMOX-1 ≥25 repeats: -14.0 (-25.7, -0.5), 0.0430
Reference: Chen and Schwartz (2008, 190106) **Period of Study:** 1989-1991 **Location:** US	**Outcome:** White Blood Cell count **Age Groups:** 20-89 yrs **Study Design:** panel **N:** 2,978 participants **Statistical Analyses:** Mixed Effects Models **Covariates:** age, sex, race, SES, smoking, alcohol consumption, MS abnormalities, indoor air pollutants, exercise **Dose-response Investigated?** No **Statistical Package:** Stata **Lags Considered:** NR	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24h **Mean (SD):** 36.8 (13.0) **Median(range) for** **Q_1:** 23.1(14.6-27.8) **Q_2:** 31.2 (27.9-34.3) **Q_3:** 38.8 (34.3-43.3) **Q_4:** 53.7 (43.3-78.5) **Monitoring Stations:** NR **Copollutant:** NR **Co-pollutant Correlation** n/a	**PM Increment:** quartile, 1yr avg (36.8 $\mu g/m^3$) **Average WBC count(SE) by PM quartile:** **Q_1:** 6760 (79) **Q_2:** 6942 (99) **Q_3:** 6895 (84) **Q_4:** 7109 (61) **Beta(Lower CI, Upper CI), p-value:** Crude: 239 (58, 420), 0.01 Model 1: 145 (10, 281), 0.035 Model 2: 141 (6, 277), 0.041 Model 3: 138 (2, 273), 0.046 Model 1: age, sex, race, SES, smoking, alcohol consumption, MS abnormalities. Model 2: Model 1 plus indoor air pollutants, exercise. Model 3: clean areas (Q1) vs. .other more polluted areas

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Chuang et al. (2007, 091063) **Period of Study:** Between Apr-Jun 2004 or 2005 **Location:** Taipei, Taiwan	**Outcome:** High-sensitivity C-reactive protein (hs-CRP) Fibrinogen, plasminogen activator fibrinogen inhibitor-1 (PAI-1), tissue-type plasminogen activator (tPA), 8-hydroxy-2′-deoxyguanosine (8-OHdG), and log-transformed HRV indices (SDNN = standard deviation of NN intervals, r-MSSD = square root of the mean of the sum of the squares of differences between adjacent NN intervals, LF = low frequency [0.04-0.15Hz], and HF = high frequency[0.15-0.40Hz]) **Age Groups:** 18-25 yrs **Study Design:** Panel (cross-sectional) **N:** 76 students **Statistical Analyses:** linear mixed-effects models **Covariates:** Age, sex, BMI, weekday, temperature of previous day, relative humidity **Season:** Only 1 season of data collection **Dose-response Investigated?** No **Statistical Package:** NR	**Pollutant:** PM_{10}, nitrate, sulfate **Averaging Time:** Hourly data used to calculate averages over 1-3 day periods **Mean (SD):** 1-day avg: 31.8 (10.6) 2-day avg: 36.4 (12.6) 3-day avg: 36.5 (12.6) **Range (Min, Max):** 1-day avg: 16.2, 50.1 2-day avg: 15.0, 53.4 3-day avg: 12.7, 59.5 **Monitoring Stations:** 2 sites (each pollutant measured at one site only) **Copollutant:** PM_{10} Sulfate Nitrate OC EC NO_2 CO SO_2 O_3	**$PM_{2.5}$ Increment:** IQR (1-d avg: 20.4 2-day avg: 25.2 3-day avg: 20.0) **Effect Estimate [Lower CI, Upper CI]:** % change in health endpoint per increase in IQR of $PM_{2.5}$ (1-3 day averaging period single pollutant models) hs-CRP: 1-d: 90.2 (-10.2, 190.1) 2-d: 99.1 (-26.1, 224.3) 3-d: 100.4 (-2.9, 203.7) 8-OHdG: 1-d: -5.0 (-14.3, 4.4) 2-d: -5.5 (-15.6, 4.6) 3-d: -5.6 (-13.8, 2.6) PAI-1: 1-d: 20.4 (17.3, 33.5) 2-d: 16.2 (1.9, 30.5) 3-d: 20.0 (18.5, 31.5) tPA: 1-d: 12.0 (-2.4, 26.3) 2-d: 12.0 (-2.9, 26.9); 3-d: 12.0 (-2.7, 26.6) Fibrinogen: 1-d: 2.6 (-2.7, 7.8) 2-d: 1.5 (-4.1, 7.1); 3-d: 3.6 (-0.8, 8.1) **Heart Rate Variability** SDNN: 1-d: -4.0 (-6.1 to -1.9) 2-d: -2.5 (-4.6 to -0.4) 3-d: -3.0 (-5.0 to -1.1) r-MSSD: 1-d: -3.0 (-8.7, 2.7) 2-d: -2.0 (-8.4, 4.4); 3-d: -3.6 (-8.8, 1.6) LF: 1-d: -3.1 (-6.1 to -0.1) 2-d: -3.2 (-4.6, 0.1); 3-d: -3.4 (-6.1 to -0.6) HF: 1-d: -3.7 (-9.4, 2.1) 2-d: -2.1 (-8.4, 4.3); 3-d: -4.0 (-9.3, 1.2)

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Chuang et al. (2007, 091063) **Period of Study:** Between Apr-Jun 2004 or 2005 **Location:** Taipei, Taiwan	**Outcome:** High-sensitivity C-reactive protein (hs-CRP) Fibrinogen, plasminogen activator fibrinogen inhibitor-1 (PAI-1), tissue-type plasminogen activator (tPA), 8-hydroxy-2′-deoxyguanosine (8-OHdG), and log-transformed HRV indices (SDNN = standard deviation of NN intervals, r-MSSD = square root of the mean of the sum of the squares of differences between adjacent NN intervals, LF = low frequency [0.04-0.15Hz], and HF = high frequency[0.15-0.40Hz]) **Age Groups:** 18-25 yrs **Study Design:** Panel (cross-sectional) **N:** 76 students **Statistical Analyses:** Linear mixed-effects models **Covariates:** Age, sex, BMI, weekday, temperature of previous day, relative humidity **Season:** Only 1 season of data collection **Dose-response Investigated?** No **Statistical Package:** NR	**Pollutant:** Nitrate **Averaging Time:** Hourly data used to calculate averages over 1-3 day periods **Mean (SD):** 1-day avg: 4.5 (2.7) 2-day avg: 4.7 (2.4) 3-day avg: 4.4 (2.2) **Range (Min, Max):** 1-day avg: 0.7, 10.6 2-day avg: 0.7, 8.9 3-day avg: 0.8, 7.5 **Monitoring Stations:** 2 sites (each pollutant measured at one site only) **Copollutant:** PM_{10} Sulfate $PM_{2.5}$ OC EC NO_2 CO SO_2 O_3	**Nitrate Increment:** IQR (1-d avg: 2.5 2-day avg: 4.0 3-day avg: 3.4) **Effect Estimate [Lower CI, Upper CI]:** % change in health endpoint per increase in IQR of nitrate (1-3 day averaging period single pollutant models) hs-CRP: 1-d: -2.1 (-21.9, 17.8) 2-d: -11.6 (-58.6, 35.5) 3-d: -18.7 (-69.9, 32.5) 8-OHdG: 1-d: 9.0 (4.0, 14.1) 2-d: 15.1 (5.9, 24.3) 3-d: 15.0 (4.9, 25.0) PAI-1: 1-d: 4.0 (-2.5, 10.4) 2-d: 11.6 (0.1, 23.1) 3-d: 16.9 (4.3, 29.4) tPA: 1-d: 2.0 (-6.2, 10.3) 2-d: 12.9 (-1.6, 27.5) 3-d: 10.0 (-5.8, 25.8) Fibrinogen: 1-d: 1.6 (-1.3, 4.5) 2-d: 1.3 (-3.9, 6.5) 3-d: 1.0 (-4.6, 6.6) **Heart Rate Variability** SDNN: 1-d: -1.5 (-2.6 to -0.3) 2-d: -2.6 (-4.7 to -0.5) 3-d: -3.0 (-5.3 to -0.7) r-MSSD: 1-d: -5.5 (-8.7 to -2.2) 2-d: -7.1 (-14.0 to -0.2) 3-d: -8.1 (-14.5 to -1.8) LF: 1-d: -1.0 (-1.6 to -0.5) 2-d: -2.0 (-5.6, 1.6) 3-d: -2.0 (-5.2, 1.2) HF: 1-d: -2.0 (-5.3, 14[potential typo, possibly 1.4]) 2-d: -4.9 (-10.9, 0.9) 3-d: -6.9 (-13.4 to -0.3)

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Chuang et al. (2007, 091063) **Period of Study:** Between Apr-Jun 2004 or 2005 **Location:** Taipei, Taiwan	**Outcome:** High-sensitivity C-reactive protein (hs-CRP) Fibrinogen, plasminogen activator fibrinogen inhibitor-1 (PAI-1), tissue-type plasminogen activator (tPA), 8-hydroxy-2′-deoxyguanosine (8-OHdG), and log-transformed HRV indices (SDNN = standard deviation of NN intervals, r-MSSD = square root of the mean of the sum of the squares of differences between adjacent NN intervals, LF = low frequency [0.04-0.15Hz], and HF = high frequency[0.15-0.40Hz]) **Age Groups:** 18-25 yrs **Study Design:** Panel (cross-sectional) **N:** 76 students **Statistical Analyses:** Linear mixed-effects models **Covariates:** Age, sex, BMI, weekday, temperature of previous day, relative humidity **Season:** Only 1 season of data collection **Dose-response Investigated?** No **Statistical Package:** NR	**Pollutant:** Sulfate **Averaging Time:** Hourly data used to calculate averages over 1-3 day periods **Mean (SD):** 1-day avg: 4.1 (3.6) 2-day avg: 4.1 (3.7) 3-day avg: 3.9 (3.5) **Range (Min, Max):** 1-day avg: 0.4, 10.9 2-day avg: 0.4, 11.9 3-day avg: 0.4, 11.5 **Monitoring Stations:** 2 sites (each pollutant measured at one site only) **Copollutant:** PM_{10} $PM_{2.5}$ Nitrate OC EC NO_2 CO SO_2 O_3	**Sulfate Increment:** IQR (1-d avg: 3.9 2-day avg: 4.3 3-day avg: 3.8) **Effect Estimate [Lower CI, Upper CI]:** % change in health endpoint per increase in IQR of sulfate (1-3 day averaging period single pollutant models) hs-CRP: 1-d: 80.0 (9.8, 150.2) 2-d: 87.1 (14.9, 159.4) 3-d: 71.1 (13.0, 129.2) 8-OHdG: 1-d: 1.0 (0.3, 1.3) 2-d: -0.4 (-5.4, 4.7) 3-d: -0.3 (-4.3, 3.7) PAI-1: 1-d: 12.0 (5.4, 18.7) 2-d: 13.3 (6.6, 19.9) 3-d: 11.2 (5.7, 16.6) tPA: 1-d: 2.0 (-4.6, 8.7) 2-d: 3.8 (-2.8, 10.3) 3-d: 3.0 (-2.3, 8.2) Fibrinogen: 1-d: 2.9 (0.2, 5.5) 2-d: 2.8 (0.1, 5.5) 3-d: 2.2 (0.4, 4.7) **Heart Rate Variability** SDNN: 1-d: -3.1 (-4.1 to -2.1) 2-d: -4.1 (-5.2 to -3.1) 3-d: -2.0 (-2.9 to -1.2) r-MSSD: 1-d: -5.0 (-8.0 to -2.0) 2-d: -6.0 (-8.9 to -2.9) 3-d: -5.7 (-8.2 to -3.2) LF: 1-d: -3.4 (-4.9 to -1.8) 2-d: -3.0 (-4.5 to -1.5) 3-d: -3.0 (-4.3 to -1.7) HF: 1-d: -3.5 (-6.5 to -0.4) 2-d: -3.9 (-7.0 to -0.8) 3-d: -3.0 (-5.5 to -0.5)
Reference: Chuang et al. (2007, 098629) **Period of Study:** NR **Location:** Boston, MA	**Outcome:** ST Segment Depression **Age Groups:** 43-75 yrs **Study Design:** panel **N:** 48 coronary artery disease patients **Statistical Analyses:** Linear & Mixed Logistic Regression models **Covariates:** participant, day of week, order of visit, visit date, hour of day, hourly temperature **Dose-response Investigated?** No **Statistical Package:** R **Lags Considered:** lags 1-72h	**Pollutant:** $PM_{2.5}$ **Averaging Time:** hourly **25th, 50th, 75th percentile:** 12h avg: 6.18, 8.91, 13.18 24h avg: 6.38, 9.20, 13.31 **Max:** 12h avg: 37.13 24h avg: 40.38 **Monitoring Stations:** 1 **Co-pollutant:** BC, CO, O_3, NO_2, SO_2 **Co-pollutant Correlation** BC: 0.56 O_3: 0.20 NO_2: 0.38 SO_2: 0.25	**PM Increment:** Interquartile Increase **Change (Lower CI, Upper CI):** 12-hour mean $PM_{2.5}$: -0.022 (-0.032, -0.012) $PM_{2.5}$ + NO_2: -0.023 (-0.034, -0.012) $PM_{2.5}$ + SO_2: -0.009 (-0.02, 0.001) $PM_{2.5}$ + BC: -0.011 (-0.023, 0.001) 24-hour mean $PM_{2.5}$: -0.026 (-0.037, -0.015) $PM_{2.5}$ + NO_2: -0.017 (-0.029, 0.004) $PM_{2.5}$ + SO_2: -0.014 (-0.025, -0.002) $PM_{2.5}$ + BC: -0.012 (-0.026, 0.003) **Relative Risk (Lower CI, Upper CI):** 12-hour mean $PM_{2.5}$: 1.02 (0.86, 1.21) $PM_{2.5}$ + NO_2: 0.99 (0.82, 1.21) $PM_{2.5}$ + SO_2: 0.87 (0.71, 1.05) $PM_{2.5}$ + BC: 0.92 (0.74, 1.14) 24-hour mean $PM_{2.5}$: 1.22 (0.99, 1.50) $PM_{2.5}$ + NO_2: 1.00 (0.80, 1.25) $PM_{2.5}$ + SO_2: 1.04 (0.83, 1.30) $PM_{2.5}$ + BC: 0.87 (0.65, 1.17) **Mean (Lower CI, Upper CI):** 12-hour mean Myocardial Infarction: -0.042 (-0.057, -0.026) No Myocardial Infarction: -0.012 (-0.023, 0.00)

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			p- for interaction: 0.002 Visit 1: -0.102 (-0.12, -0.085) Visits 2-4: 0.006 (-0.005, 0.017) p- for interaction: < 0.001 Diabetic: -0.097 (-0.119, -0.074) Non-diabetic: -0.009 (-0.019, 0.002) p- for interaction: < 0.001 Diurnal daytime pattern: -0.032 (-0.043, -0.021) Diurnal nighttime pattern: -0.006 (-0.018, 0.006) p- for interaction: < 0.001 24-hour mean Myocardial Infarction: -0.027 (-0.043, -0.012) No Myocardial Infarction: -0.025 (-0.038, 0.011) p- for interaction: 0.787 Visit 1: -0.127 (-0.148, -0.105) Visits 2-4: 0.001 (-0.011, 0.013) p- for interaction: < 0.001 Diabetic: -0.118 (-0.144, -0.091) Non-diabetic: -0.13 (-0.024, -0.002) p- for interaction: < 0.001 Diurnal daytime pattern: -0.031 (-0.043, -0.020) Diurnal nighttime pattern: -0.018 (-0.030, -0.005) p- for interaction: 0.233 **Notes:** The effects of PM on half-hour St segment levels (figure 1)
Reference: Dales et al. (2007, 155743) **Period of Study:** NR **Location:** Ottawa, Canada	**Outcome:** Vascular Reactivity **Age Groups:** 18-50 yrs **Study Design:** panel **N:** 39 volunteers **Statistical Analyses:** Mixed Effects Model **Covariates:** temperature, humidity, wind speed, time of day testing was done, site **Dose-response Investigated?** No **Statistical Package:** S-PLUS **Lags Considered:** NR	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 2h **Mean (SD):** Downtown: 40 (20) Tunney's Pasture: 10 (10) p-value 0.000 **Monitoring Stations:** NR **Copollutant:** $PM_{1.0}$ **Co-pollutant Correlation** n/a	**PM Increment:** Interquartile Range (27.02 $\mu g/m^3$) **Beta (SE), p-value:** Flow mediated vasodilation (%): -0.016 (0.0072) p = 0.03 Heart Rate (beats/min): 0.081 (0.135) p = 0.55 Diastolic blood pressure (mmHg): 0.088 (0.088) p = 0.32 Systolic blood pressure (mmHg): -0.108 (0.006) p = 0.48

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: de Hartog et al. (2009, 191904) **Period of Study:** 1998-1999 **Location:** Amsterdam, Netherlands Erfurt, Germany and Helsinki, Finland	**Outcome:** Heart Rate Variability **Age Groups:** 50+ **Study Design:** panel **N:** 122 coronary heart disease patients **Statistical Analyses:** Linear Regression **Covariates:** time trend, temperature, humidity, pressure **Dose-response Investigated?** No **Statistical Package:** SAS **Lags Considered:** lags 0-3d	**Pollutant:** $PM_{2.5}$ **Averaging Time:** daily **p25, p50, p75. p95:** Amsterdam: 10.4, 16.7, 23.9, 47.0 Erfurt: 10.8, 16.3, 26.7, 62.3 Helsinki: 8.3, 10.6, 15.9, 25.8 **Monitoring Stations:** NR **Copollutant:** PM $_{<0.1}$, $PM_{0.1\text{-}1.0}$, NO_2, SO_2 **Co-pollutant Correlation** NR **Note:** Correlations are provided for source-specific $PM_{2.5}$ & elements	**PM Increment:** 1 $\mu g/m^3$ **Beta (Lower CI, Upper CI):** SDNN Local traffic: -0.12 (-0.36, 0.12) Long-range transport: -0.04 (-0.14, 0.06) Oil combustion: -0.29 (-1.04, 0.45) Industry: 0.03 (-0.12, 0.19) Crustal: 0.11 (-0.35, 0.56) Salt: -0.19 (-1.92, 1.55) HF Local traffic: 0.43 (-0.91, 1.79) Long-range transport: 0.19 (-0.38, 0.77) Oil combustion: 1.05 (-2.70, 4.94) Industry: 0.62 (-0.34, 1.59) Crustal: 1.57 (-1.28, 4.50) Salt: -1.43 (-9.86, 7.78) SDNN ABS: -0.52 (-1.39, 0.31) S: -0.51 (-1.36, 0.33) V: -0.66 (-1.73, 0.41) Zn: 0.12 (-0.55, 0.79) Ca: 0.27 (-0.58, 1.11) Cl: 0.14 (-0.39, 0.67) Fe: 0.15 (-1.00, 1.30) Cu: -0.08 (-0.74, 0.57) SDNN ABS: 2.91 (-2.54, 8.67) S: 0.25 (-4.42, 5.14) V: 0.73 (-4.74, 6.53) Zn: 3.85 (-0.26, 8.13) Ca: 3.39 (-1.80, 8.86) Cl: 1.13 (-1.48, 3.81) Fe: 6.69 (0.11, 13.69) Cu: 3.00 (-0.85, 7.00) Notes: Estimates provided are for all subjects at lag 1, estimates are also available at lags 0, 2, and 3, as well as for subjects w/o beta-blockers at lags 0-3.

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: DeMeo et al. (2004, 087346) **Period of Study:** July through August, 1999 **Location:** Boston, MA	**Outcome:** Oxygen Saturation **Age Groups**: 60.4 to 89.2 years **Study Design:** Cross-sectional study **N:** 28 adult participants **Statistical Analyses:** GLM, Natural Spline Smoothing, Regression Analysis, Random-effects model **Covariates:** Mean temperature, Dew point temperature, Barometric pressure, Medication use **Season**: Summer **Dose-response Investigated?** No **Statistical Package**: S-PLUS, SAS **Lags Considered:** Hourly lags between 2 and 7 h	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 6 h, 12 h, 24 h, 48 h	**PM Increment:** IQR (13.42 $\mu g/m^3$) increase 6 h: 13.42 $\mu g/m^3$ 12 h: 10.81 $\mu g/m^3$ 24 h: 10.26$\mu g/m^3$ 48: 10.57 $\mu g/m^3$ Overall: 0.172% (-0.313, 0.031) decrease 6-h: -0.769% (-1.21 to -0.327) decrease B-blocker users: -0.062% (-0.248, 0.123) Rest: 6 h: -0.173 (-0.345 to -0.001) 12 h: -0.160 (-0.308 to -0.012) 24 h: -0.169 (-0.316 to -0.022) 48 h: -0.153 (-0.304, 0.002) Exercise: 6 h: -0.005 (-0.215, 0.205) 12 h: -0.014 (-0.196, 0.168) 24 h: 0.001 (-0.180, 0.182) 48 h: -0.011 (-0.196, 0.174) Post exercise Rest: 6 h: -0.173 (-0.332 to -0.014) 12 h: -0.128 (-0.266, 0.010) 4 h: -0.113 (-0.250, 0.023) 48 h: -0.157 (-295 to -0.019) Paced breathing: 6 h: -0.142 (-0.292, 0.007) 12 h: -0.139 (-0.269 to -0.010) 24 h: -0.121 (-0.248, 0.007) 48 h: -0.082 (0.211, 0.047) Summary over protocol 6 h: -0.131 (-0.247 to -0.015) 12 h: -0.120 (-0.221, 0.020) 24 h: -0.112 (-0.212 to -0.013) **Notes:** Figure of the Variation in Oxygen Saturation during the first rest period versus individual hourly lag measurements for $PM_{2.5}$

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Diez-Roux et al. (2006, 156400) **Period of Study:** Baseline data collected June 2000–Aug 2002 **Location:** USA (6 field centers: Baltimore, MD Chicago, IL Forsyth Co, NC Los Angeles, CA New York, NY St. Paul, MN	**Outcome:** C-reactive protein (CRP) assessed continuously and as a dichotomous variable (cutpoint, 3 mg/L) interleukin-6 (IL-6) **Age Groups:** 45-84 yrs **Study Design:** Cross-sectional **N:** 5634 persons **Statistical Analyses:** Linear regression & logistic regression **Covariates:** Age, sex, race/ethnicity, general health status, BMI, diabetes, cigarette status, secondhand smoke, physical activity, arthritis flare in last 2 weeks, medications, infections in last 2 weeks (also ran models including site, copollutants, and weather) **Season:** Examined seasonal patterns in the residuals of fully adjusted models stratified by season **Dose-response Investigated?** No **Statistical Package:** NR	**Pollutant:** $PM_{2.5}$ **Averaging Time:** Prior day, prior 2 days, prior week, prior 30 days, and prior 60 days **Mean (SD):** Presented in Fig 1 by site **Percentiles:** Presented in Fig 1 by site **Range:** NR **Monitoring Stations:** NR Long-term exposure to PM estimated based on residential history reported retrospectively all addresses geocoded ambient AP obtained from US EPA **Copollutant:** SO_2 NO_2 CO O_3	**PM Increment:** 10 $\mu g/m^3$ **Effect Estimate [Lower CI, Upper CI]:** Adjusted (all personal-level covariates) relative difference in CRP (mg/L) per 10 $\mu g/m^3$ increase in $PM_{2.5}$ Prior day: 0.99 (0.96, 1.01) Prior 2 days: 0.99 (0.96, 1.01) Prior 7 days: 1.00 (0.96, 1.04) Prior 30 days: 1.03 (0.98, 1.10) Prior 60 days: 1.04 (0.97, 1.11) Odds Ratios of CRP of ≥ 3 mg/L per 10 $\mu g/m^3$ increase in $PM_{2.5}$ (adjusted for all personal-level covariates) Prior day: 0.98 (0.92, 1.04) Prior 2 days: 0.99 (0.93, 1.06) Prior 7 days: 1.05 (0.96, 1.15) Prior 30 days: 1.12 (0.98, 1.29) Prior 60 days: 1.12 (0.96, 1.32)
Reference: Dubowsky et al. (2006, 088750) **Period of Study:** March–Jun 2002 **Location:** St. Louis, Missouri	**Outcome:** White blood cells (WBC), C-reactive protein (CRP), interleukin-6 (IL-6) **Age Groups:** ≥ 60 yrs **Study Design:** Panel (4 planned repeated measures n = 35 participated in 4 trips) **N:** 44 participants **Statistical Analyses:** Linear mixed models **Covariates:** Sex, obesity, diabetes, smoking history, time-varying parameters (apparent temperature, h, day, trip, residence, mold, pollen, illness, and juice intake), medication and vitamin consumption (day of blood draw) **Season:** Limited data collection period **Dose-response Investigated?** No **Statistical Package:** SAS v8.02	**Pollutant:** $PM_{2.5}$ (ambient) **Averaging Time:** Hourly data used to calculate avg concentrations over 1-7 days preceding the blood draw (ambient $PM_{2.5}$) microenvironmental $PM_{2.5}$ measures were averaged over the 1-2 days preceding the blood draw **Mean (SD) (1-day):** 16 (6.0) **Percentiles (1-day):** 0: 6.5 25th: 12 75th: 22 100th: 28 **Monitoring Stations:** 1 ambient monitor **Copollutant:** $PM_{2.5}$ (ambient) BC (ambient) $PM_{2.5}$ (microenvironment) CO NO_2 SO_2 O_3	**PM Increment:** 6.1 $\mu g/m^3$ (5-d mean) **Effect Estimate [Lower CI, Upper CI]:** **Note:** Most results presented in figures. Selected result in abstract text: % change in WBC per increase in IQR (5.4 $\mu g/m^3$) of $PM_{2.5}$ averaged over the previous week: 5.5 (0.1, 11) Associations (% changes and 95%CI) between 5-day mean ambient concentrations and markers of inflammation per increase (IQR) in pollutant. CRP: All participants: 14 (-5.4, 37) Among those with all 3 conditions (diabetes, obesity, and hypertension): 81 (21, 172) Among those with at least 2 of the conditions: 11 (-7.3, 33) IL-6: All participants: -2.1 (-13, 11) Among those with all 3 conditions (diabetes, obesity, and hypertension): 23 (-5.3, 59) Among those with at least 2 of the conditions: -3.1 (-14, 9.7) WBC ($x10^9$/L): All participants: 3.4 (-1.8, 8.9) Among those with all 3 conditions (diabetes, obesity, and hypertension): 0.4 (-8.8, 11) Among those with at least 2 of the conditions: 3.6 (-1.7, 9.1)

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Dubowsky et al. (2006, 088750) **Period of Study:** March–Jun 2002 **Location:** St. Louis, Missouri	**Outcome:** White blood cells (WBC), C-reactive protein (CRP), interleukin-6 (IL-6) **Age Groups:** ≥ 60 yrs **Study Design:** Panel (4 planned repeated measures n = 35 participated in 4 trips) **N:** 44 participants **Statistical Analyses:** Linear mixed models **Covariates:** Sex, obesity, diabetes, smoking history, time-varying parameters (apparent temperature, h, day, trip, residence, mold, pollen, illness, and juice intake), medication and vitamin consumption (day of blood draw) **Season:** Limited data collection period **Dose-response Investigated?** No **Statistical Package:** SAS v8.02	**Pollutant:** BC (ng/m^3) (ambient) **Averaging Time:** Hourly data used to calculate avg concentrations over 1-7 days preceding the blood draw (ambient PM) microenvironmental $PM_{2.5}$ measures were averaged over the 1-2 days preceding the blood draw **Mean (SD) (1-day):** 900 (280) **Percentiles (1-day):** 0: 290 25th: 730 75th: 1,100 100th: 1,400 **Monitoring Stations:** 1 ambient monitor **Copollutant:** $PM_{2.5}$ (ambient) BC (ambient) $PM_{2.5}$ (microenvironment) CO; NO_2 SO_2 O_3	**PM Increment:** 230 ng/m^3 (5-d mean) **Effect Estimate [Lower CI, Upper CI]:** **Note:** Most results presented in figures. **Associations (% changes and 95%CI) between 5-day mean ambient concentrations and markers of inflammation per increase (IQR) in pollutant.** CRP: All participants: 13 (-0.34, 28) Among those with all 3 conditions (diabetes, obesity, and hypertension): 49 (16, 90) Among those with at least 2 of the conditions: 9.0 (-3.8, 24) IL-6: All participants: -0.8 (-8.9, 8.0) Among those with all 3 conditions (diabetes, obesity, and hypertension): 15 (-2.2, 35) Among those with at least 2 of the conditions: -2.7 (-11, 6.2) WBC ($x10^9/L$): All participants: 1.3 (-2.1, 4.8) Among those with all 3 conditions (diabetes, obesity, and hypertension): 0.05 (-5.9, 6.3) Among those with at least 2 of the conditions: 1.5 (-2.0, 5.1)
Reference: Ebelt et al. (2005, 056907) **Period of Study:** Summer of 1998 **Location:** Vancouver, Canada	**Outcome:** CVD **Age Groups:** range from 54-86 yrs mean age = 74 years **Study Design:** extended analysis of a repeated-measures panel study **N:** 16 persons with COPD **Statistical Analyses:** Earlier analysis expanded by developing mixed-effect regression models and by evaluating additional exposure indicators **Dose-response Investigated?** No **Statistical Package:** SAS V8	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean (SD):** Ambient $PM_{2.5}$: 11.4 ± 4.6 Exposure to ambient $PM_{2.5}$: 7.9 ± 3.7 **Range (Min, Max):** Ambient $PM_{2.5}$: 4.2 - 28.7 Exposure to ambient $PM_{2.5}$: 0.9 - 21.3 **Monitoring Stations:** 5 **Copollutant (correlation):** Ambient concentrations and exposure to ambient PM were highly correlated for each respective metric: r ≥ 0.71	**Note:** Total personal fine particle exposure (T) were dominated by exposures to non ambient particles which were not correlated with ambient fine particle exposure (A) or ambient concentrations (C). Results for each of these metrics are listed. **PM Increment:** Increment: $C^{2.5}$: IQR = 5.8 SBP (mm Hg): -1.70 (-3.48-0.08) DBP (mm Hg): -0.58 (-2.02-0.85) Ln-SVE (bph): 0.20 (0.00-0.40) HR (bpm): 0.93 (-0.90-2.75) SDNN (ms): -4.37 (-9.40-0.65) R-MSSD (ms): -2.79 (-6.16-0.57) Increment: NS_$C^{2.5}$: IQR = 4.2 SBP (mm Hg): -1.52 (-2.94- -0.09) DBP (mm Hg): -0.77 (-1.87-0.32) Ln-SVE (bph): 0.19 (-0.01-0.38) HR (bpm): 1.03 (-0.43-2.48) SDNN (ms): -3.83 (-7.77-0.11) R-MSSD (ms): -2.90 (-5.55- -0.25) Increment: S_$C^{2.5}$: IQR = 1.5 SBP (mm Hg): -1.10 (-3.48-1.28) DBP (mm Hg): 0.76 (-1.15-2.68) Ln-SVE (bph): 0.09 (-0.05-0.23) HR (bpm): -0.42 (-2.28-1.44) SDNN (ms): -3.14 (-9.73-3.45) R-MSSD (ms): 0.24 (-5.14-5.63) Increment: $A^{2.5}$: IQR = 4.4 SBP (mm Hg): -1.90 (-3.66- -0.14) DBP (mm Hg): -0.33 (-1.72-1.06) Ln-SVE (bph): 0.20 (0.02-0.37) HR (bpm): 0.57 (-1.34-2.47) SDNN (ms): -3.91 (-8.79-0.97) R-MSSD (ms): -1.05 (-4.79-2.17) Increment: NS_$A^{2.5}$: IQR = 3.4

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			SBP (mm Hg): -1.70 (-3.27- -0.14) DBP (mm Hg): -0.51 (-1.71-0.70) Ln-SVE (bph): 0.20 (0.02-0.37) HR (bpm): 0.69 (-0.96-2.35) SDNN (ms): -4.18 (-8.51-0.15) R-MSSD (ms): -1.40 (-4.40-1.60) Increment: $S\_T^{2.5}$: IQR = 0.9 SBP (mm Hg): -1.55 (-3.35-0.26) DBP (mm Hg): 0.49 (-0.91-1.90) Ln-SVE (bph): 0.08 (-0.14-0.19) HR (bpm): -0.24 (-1.75-1.26) SDNN (ms): -0.68 (-4.74-3.38) R-MSSD (ms): 0.91 (-3.51-5.33) Increment: $T^{2.5}$: IQR = 10.1 SBP (mm Hg): -1.26 (-2.60-0.08) DBP (mm Hg): 0.34 (-1.26-1.94) Ln-SVE (bph): 0.01 (-0.10-0.11) HR (bpm): -0.23 (-1.09-0.63) SDNN (ms): -2.11 (-4.90-0.68) R-MSSD (ms): -0.83 (-3.60-1.94) Increment: $N^{2.5}$: IQR = 8.9 SBP (mm Hg): -0.81 (-2.15-0.53) DBP (mm Hg): 0.40 (-1.19-1.98) Ln-SVE (bph): -0.04 (-0.18-0.10) HR (bpm): -0.35 (-0.85-0.14) SDNN (ms): -1.10 (-3.10-0.90) R-MSSD (ms): -0.54 (-2.54-1.46)
Reference: Fan et al. (2008, 191979) **Period of Study:** Feb – May 2005 **Location:** Paterson, New Jersey	**Outcome:** Cardiopulmonary Health (FEV, FVC, PEF, SDNN, HR) **Age Groups:** 61.2 (13.7) **Study Design:** panel **N:** 11 **Statistical Analyses:** Mixed Effects models, Linear Regression models **Covariates:** temperature, humidity **Dose-response Investigated?** No **Statistical Package:** SAS **Lags Considered:** 0	**Pollutant:** $PM_{2.5}$ **Averaging Time:** daily **Mean (SD):** | $\Delta PM_{2.5}$ avg Morning: 35.2 (25.9) Afternoon: 24.1 (22.1) $\Delta PM_{2.5}$ peak Morning: 71.3 (56.1) Afternoon: 64.3 (43.5) **Range:** $\Delta PM_{2.5}$ avg Morning: 1.1 - 87 Afternoon: 1.2 - 98 $\Delta PM_{2.5}$ peak Morning: 4.0 - 278 Afternoon: 3.0 - 150 **Monitoring Stations:** NR **Copollutant:** NR **Co-pollutant Correlation** n/a	**PM Increment:** 10$\mu g/m^3$ **Beta (SE), p-value:** **ΔSDNN** Morning, $\Delta PM_{2.5}$ avg 15min: -14.5 (6.9), 0.06 2h: -18.9 (4.2), 0.0002 4h: -2.5 (8.6), 0.78 Morning, $\Delta PM_{2.5}$ peak 15min: -9.2 (11.2), 0.43 2h: -5.1 (13.8), 0.72 4h: -7.4 (12.0), 0.55 Afternoon, $\Delta PM_{2.5}$ avg 15min: -2.4 (7.6), 0.77 2h: -20.2 (10.8), 0.10 4h: -0.7 (11.2), 0.95 Afternoon, $\Delta PM_{2.5}$ peak 15min: 0.6 (8.9), 0.95 2h: 19.2 (14.6), 0.23 4h: -6.8 (14.1), 0.64 **Δ HR** Morning, $\Delta PM_{2.5}$ avg 15min: 1.2 (3.1), 0.71 2h: -5.5 (2.9), 0.08 4h: -3.1 (4.6), 0.51 Morning, $\Delta PM_{2.5}$ peak 15min: 0.8 (4.4), 0.86 2h: -7.2 (4.2), 0.11 4h: -7.1 (6.3), 0.28 Afternoon, $\Delta PM_{2.5}$ avg 15min: -2.0 (4.0), 0.62 2h: 0.9 (5.4), 0.87 4h: 8.2 (5.2), 0.14 Afternoon, $\Delta PM_{2.5}$ peak 15min: -5.6 (5.3), 0.31 2h: 3.1 (8.1), 0.71 4h: 11.1 (8.1), 0.20 **ΔFEV_1** Morning, $\Delta PM_{2.5}$ avg: 0.02 (0.04), 0.68 Morning, $\Delta PM_{2.5}$ peak: -0.13 (0.08), 0.16 **Δ FVC** Morning, $\Delta PM_{2.5}$ avg: -0.10 (0.09), 0.31

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			Morning, $\Delta PM_{2.5}$ peak: -0.12 (0.17), 0.51 **Δ PEF** Morning, $\Delta PM_{2.5}$ avg: -0.54 (0.62), 0.42 Morning, $\Delta PM_{2.5}$ peak: -1.46 (1.12), 0.24 **Notes:** Estimates relative to increases in the average and peak $PM_{2.5}$ concentrations
Reference: Folino et al. (2009, 191902) **Period of Study:** Jun 2006 – May 2007 **Location:** Padua, Italy	**Outcome:** HRV & Inflammatory Markers **Age Groups:** 45-65 yrs **Study Design:** panel **N:** 39 patients w/ myocardial infarction **Statistical Analyses:** Linear Regression Model, ANOVA **Covariates:** temperature, relative humidity, atmospheric pressure, beta-blocker, aspirin, or nitrate consumption, smoking habit **Dose-response Investigated?** No **Statistical Package:** Stata **Lags Considered:** NR	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24h **Mean (SD):** Summer: 33.9 (12.7) Winter: 62.1 (27.9) Spring: 30.8 (14.0) **Monitoring Stations:** NR **Copollutant:** PM_{10}, $PM_{0.25}$ **Co-pollutant Correlation** NR	**PM Increment:** 1 $\mu g/m^3$ **Beta (SE), p-value:** SDNN: 0.109 (0.115), 0.345 SDANN: 0.127 (0.126), 0.314 RMSSD: 0.045 (0.040), 0.256 pH: 0.002 (0.001), 0.041 LTB4: 0.590 (0.324), 0.069 eNO: -0.002 (0.003), 0.503 PTX3: -0.004 (0.002), 0.013 C-reactive protein: -0.008 (0.005), 0.115 CC16: -0.002 (0.002), 0.410 IL-8: 0.000 (0.003), 0.989
Reference: Folino et al. (2009, 191902) **Period of Study:** Jun 2006 – May 2007 **Location:** Padua, Italy	**Outcome:** HRV & Inflammatory Markers **Age Groups:** 45-65 yrs **Study Design:** panel **N:** 39 patients w/ myocardial infarction **Statistical Analyses:** Linear Regression Model, ANOVA **Covariates:** temperature, relative humidity, atmospheric pressure, beta-blocker, aspirin, or nitrate consumption, smoking habit **Dose-response Investigated?** No **Statistical Package:** Stata **Lags Considered:** NR	**Pollutant:** $PM_{0.25}$ **Averaging Time:** 24h **Mean (SD):** Summer: 17.6 (7.5) Winter: 30.5 (17.4) Spring: 18.8 (10.8) **Monitoring Stations:** NR **Copollutant:** PM_{10}, $PM_{2.5}$ **Co-pollutant Correlation** NR	**PM Increment:** 1 $\mu g/m^3$ **Beta (SE), p-value:** SDNN: 0.214 (0.204), 0.295 SDANN: 0.214 (0.214), 0.316 RMSSD: 0.081 (0.077), 0.291 pH: 0.005 (0.002), 0.004 LTB4: 0.835 (0.533), 0.117 eNO: -0.006 (0.005), 0.182 PTX3: -0.006 (0.003), 0.071 C-reactive protein: -0.011 (0.007), 0.104 CC16: 0.001 (0.004), 0.890 IL-8: -0.004 (0.006), 0.527
Reference: Goldberg et al. (2008, 180380) **Period of Study:** Jul 2002 – Oct 2003 **Location:** Montreal, Canada	**Outcome:** Oxygen saturation & pulse rate **Age Groups:** 50-85 yrs **Study Design:** panel **N:** 31 **Statistical Analyses:** Mixed Random Effects Model **Covariates:** body temperature, consumption of salt, intake of fluids, being ill the day before, ambient temperature, relative humidity, barometric pressure **Dose-response Investigated?** No **Statistical Package:** Splus **Lags Considered:** lags 1d & 0-2d avg	**Pollutant:** $PM_{2.5}$ **Averaging Time:** daily **IQR:** 7.3 **Monitoring Stations:** 8 **Co-pollutant:** CO, NO_2, SO_2, O_3 **Co-pollutant Correlation** CO: 0.72 NO_2: 0.62	**PM Increment:** Interquartile Range (7.3 $\mu g/m^3$) **Mean Difference (Lower CI, Upper CI), lag:** Oxygen Saturation Unadjusted: -0.087 (-0.143, -0.031), lag 0 Unadjusted: -0.058 (-0.114, -0.002), lag 1 Unadjusted: -0.083 (-0.155, -0.010), lag 0-2d avg Adjusted: -0.056 (-0.117, 0.005), lag 0 Adjusted: -0.019 (-0.079, 0.041), lag 1 Adjusted: -0.039 (-0.118, 0.039), lag 0-2d avg Pulse Rate Unadjusted: 0.226 (-0.037, 0.489), lag 0 Unadjusted: 0.288 (0.022, 0.554), lag 1 Unadjusted: 0.420 (0.067, 0.772), lag 0-2d avg Adjusted: 0.158 (-0.136, 0.451), lag 0 Adjusted: 0.246 (-0.040, 0.531), lag 1 Adjusted: 0.353 (-0.034, 0.740), lag 0-2d avg

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Goldberg et al. (2008, 180380) **Period of Study:** Jul 2002 – Oct 2003 **Location:** Montreal, Canada	**Outcome:** Shortness of Breath & General health **Age Groups:** 50-85 yrs **Study Design:** panel **N:** 31 **Statistical Analyses:** Mixed Random Effects Model **Covariates:** body temperature, consumption of salt, intake of fluids, being ill the day before, ambient temperature, relative humidity, barometric pressure **Dose-response Investigated?** No **Statistical Package:** Splus **Lags Considered:** lags 0-4d & 0-2d avg	**Pollutant:** $PM_{2.5}$ **Averaging Time:** daily **Mean:** 9.5 **Median:** 7.0 **Min:** 0.8 **Max:** 50.2 **IQR:** 7.3 **Monitoring Stations:** 8 **Co-pollutant:** CO, NO_2, SO_2, O_3 **Co-pollutant Correlation** CO: 0.66 NO_2: 0.54 O_3: 0.32 SO_2: 0.50	**PM Increment:** Interquartile Range (7.3 $\mu g/m^3$) **Mean Difference (Lower CI, Upper CI), lag:** General Health Unadjusted: -0.317 (-0.699, 0.064), lag 0 Unadjusted: -0.284 (-0.670, 0.103), lag 1 Unadjusted: -0.048 (-0.427, 0.332), lag 2 Unadjusted: -0.241 (-0.620, 0.139), lag 3 Unadjusted: -0.010 (-0.390, 0.370), lag 4 Unadjusted: -0.482 (-1.053, 0.090), lag 0-2d avg Adjusted: -0.125 (-0.545, 0.295), lag 0 Adjusted: -0.167 (-0.568, 0.234), lag 1 Adjusted: -0.081 (-0.464, 0.302), lag 2 Adjusted: -0.222 (-0.602, 0.157), lag 3 Adjusted: 0.016 (-0.364, 0.396), lag 4 Adjusted: -0.281 (-0.886, 0.325), lag 0-2d avg Shortness of breath at night Unadjusted: -0.421 (-0.847, 0.006), lag 0 Unadjusted: -0.278 (-0.711, 0.155), lag 1 Unadjusted: -0.100 (-0.526, 0.327), lag 2 Unadjusted: -0.220 (-0.645, 0.206), lag 3 Unadjusted: -0.206 (-0.632, 0.220), lag 4 Unadjusted: -0.555 (-1.172, 0.063), lag 0-2d avg Adjusted: -0.171 (-0.639, 0.297), lag 0 Adjusted: -0.130 (-0.579, 0.319), lag 1 Adjusted: -0.127 (-0.553, 0.299), lag 2 Adjusted: -0.192 (-0.616, 0.231), lag 3 Adjusted: -0.171 (-0.594, 0.253), lag 4 Adjusted: -0.301 (-0.952, 0.350), lag 0-2d avg
Reference: Ibald-Mulli et al. (2004, 087415) **Period of Study:** winter 1998-1999 **Location:** Helsinki, Finland Erfurt, Germany Amsterdam, the Netherlands	**Outcome:** Blood Pressure & Heart Rate **Age Groups:** 40-84 **Study Design:** panel **N:** 131 adults w/ CHD **Statistical Analyses:** Linear Regression **Covariates:** trend, day of week, temperature, barometric pressure, relative humidity, medication use **Dose-response Investigated?** No **Statistical Package:** SAS **Lags Considered:** 0-2, 5d avg	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24h **Mean (SD):** Downtown: 40 (20) Tunney's Pasture: 10 (10) p-value 0.000 **Monitoring Stations:** NR **Copollutant:** $PM_{1.0}$ **Co-pollutant Correlation** n/a	**PM Increment:** Interquartile Range (27.02 $\mu g/m^3$) **Beta (SE), p-value:** Flow mediated vasodilation (%): -0.016 (0.0072) p=0.03 Heart Rate (beats/min): 0.081 (0.135) p=0.55 Diastolic blood pressure (mmHg): 0.088 (0.088) p=0.32 Systolic blood pressure (mmHg): -0.108 (0.006) p=0.48
Reference: Langrish et al. (2009, 191908) **Period of Study:** August 2008 **Location:** Beijing, China	**Outcome:** Cardiovascular Effects **Age Groups:** median 28 yrs **Study Design:** panel **N:** 15 **Statistical Analyses:** NR **Covariates:** NR **Dose-response Investigated?** No **Statistical Package:** NR **Lags Considered:** NR	**Pollutant:** $PM_{2.5}$ **Averaging Time:** NR **Mean:** W/o mask: 86 W/ mask: 140 **Monitoring Stations:** NR **Co-pollutant:** CO, SO_2, NO_2 **Co-pollutant Correlation:** n/a	**PM Increment:** NR **Mean (Lower CI, Upper CI):** W/o Mask (Day) SBP: 100 (104, 116) DBP: 73 (69, 76) MAP: 85 (81, 88) Heart Rate: 79 (74, 84) Avg NN interval: 829 (789, 869) pNN50: 15.9 (10.7, 21.0) RMSSD: 35.1 (29.2, 41.0) SDNN: 61.2 (54.9, 67.5) Triangular index: 12.9 (11.9, 13.9) LF power: 816 (628, 1004) HF power: 460 (325, 595) LFn: 62.8 (56.7, 68.9) HFn: 29.2 (25.5, 32.8) HF/LF ratio: 0.738 (0.507, 0.970) W/ Mask (Day) SBP: 109 (104, 114) DBP: 73 (70-76) MAP: 85 (81, 89)

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			Heart Rate: 78 (73, 82) Avg NN interval: 850 (805, 896) pNN50: 17.9 (14.2, 21.6) RMSSD: 37.1 (32.2, 42.0) SDNN: 65.5 (59.0, 72.2)* Triangular index: 13.8 (13.0, 14.5) LF power: 919 (717, 1122)* HF power: 485 (400, 569) LFn: 64.5 (60.6, 68.4) HFn: 30.0 (27.0, 33.1) HF/LF ratio: 0.680 (0.519, 0.842) W/o Mask (During Walk) SBP: 121 (115, 127) DBP: 81 (75-87) MAP: 94 (89, 99) Heart Rate: 88 (82, 94) Avg NN interval: 594 (562, 627) pNN50: 3.3 (0.8, 5.7) RMSSD: 17.2 (13.4, 21.0) SDNN: 45.8 (36.8, 54.8) Triangular index: 10.7 (9.1, 12.4) LF power: 313 (170, 455) HF power: 76.5 (33.6, 120.0) LFn: 68.2 (60.9, 75.5) HFn: 16.1 (11.9, 20.3) HF/LF ratio: 0.259 (0.173, 0.344) W/ Mask (During Walk) SBP: 114 (108, 120) DBP: 79 (74, 83) MAP: 90 (86, 94) Heart Rate: 91 (85, 97) Avg NN interval: 613 (571, 655) pNN50: 2.1 (-0.1, -4.4) RMSSD: 20.0 (15.5, 24.6) SDNN: 54.8 (42.5, 67.0) Triangular index: 11.4 (9.4, 13.3) W/ Mask (During Walk) LF power: 414 (233, 595) HF power: 116.8 (52.6, 181.0) LFn: 67.9 (61.9, 73.9) HFn: 16.0 (12.5, 19.4) HF/LF ratio: 0.247 (0.180, 0.314) **Mean (SD):** W/o Mask (After Walk) Headache: 2.53 (5.55) Dizziness: 1.07 (2.22) Tiredness: 8.47 (12.14) Sickness: 1.07 (2.22) Cough: 1.80 (4.80) Difficulty Breathing: 0.67 (0.90) Eye irritation: 1.40 (3.60) Throat irritation: 1.47 (4.07) Nose irritation: 1.53 (3.78) Unpleasant Smell: 0.93 (1.22) Bad taste: 0.73 (0.96) Difficulty walking: 12.53 (13.24) Perception of Pollution: 19.80 (18.37) W/ Mask (After Walk) Headache: 0.73 (1.03) Dizziness: 0.80 (1.57 Tiredness: 7.40 (9.37) Sickness: 0.87 (1.51) Cough: 1.00 (1.73) Difficulty Breathing: 3.80 (8.10) Eye irritation: 1.67 (3.27) Throat irritation: 1.07 (2.63) Nose irritation: 1.07 (1.91) Unpleasant Smell: 0.60 (0.91) Bad taste: 0.60 (1.18) Difficulty walking: 15.13 (11.51) Perception of Pollution: 11.60 (10.44) *$p < 0.05$ **Notes:** Estimates also available for 24

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			hours, night, before walk, and 24 hours after walk.
Reference: Lanki et al. (2006, 088412) **Period of Study:** Autumn 1998–spring 1999 **Location:** Helsinki, Finland	**Outcome:** ST segment depressions (2 endpoints: $>$0.1mV regardless of the direction of the ST slope and $>$0.1mV with horizontal or downward slope [stricter criteria]) **Age Groups:** Mean = 68.2 (6.5) yrs **Study Design:** Panel **N:** 45 elderly nonsmoking persons with stable coronary heart disease 342 total exercise tests for analyses **Statistical Analyses:** Generalized additive models with penalized splines (logistic regression) principal components analysis and linear regression of 13 measured elements used to apportion $PM_{2.5}$ mass between different sources **Covariates:** Subject, linear terms for time trend, temperature, relative humidity, penalized spline for change in heart rate during the exercise test **Season:** NR **Dose-response Investigated?** No **Statistical Package:** S-plus 2000 and R	**Pollutant:** $PM_{2.5}$ (Analyses conducted for source specific $PM_{2.5}$) **Averaging Time:** Daily filter samples **Mean:** Crustal: 0.6 Long-range transported: 6.4 Oil combustion: 1.6 Salt: 0.9 Local traffic: 2.9 Total: 12.8 **Percentiles:** Crustal 25: 0.0 50: 0.4 75: 1.1; Max: 5.3 Long-range transported 25: 2.2 50: 5.5 75: 9.8; Max: 26.5 Oil combustion 25: 0.6 50: 1.3 75: 2.3; Max: 12.2 Salt 25: 0.3 50: 0.8 75: 1.2; Max: 5.9 Local traffic 25: 1.7 50: 2.5 75: 3.4; Max: 12.0 Total 25: 8.3 50: 10.6 75: 15.9; Max: 39.8 **Monitoring Stations:** 1 monitor **Copollutant (correlation):** Correlations with $PM_{2.5}$: Crustal: r = -0.01 Long-range transported: r = 0.82 Oil combustion: r = 0.35 Salt: r = 0.19 Local traffic: r = 0.26	**PM Increment:** 1 $\mu g/m^3$ **Effect Estimate [Lower CI, Upper CI]:** Adjusted ORs between daily source-specific $PM_{2.5}$ concentrations and ST segment depressions. ST segment depression defined as $>$0.1 mV (n = 62) **Crustal** Lag0: 0.80 (0.47, 1.36) Lag1: 0.66 (0.40, 1.10) Lag2: 1.18 (0.68, 2.06) Lag3: 1.87 (0.85, 4.09) **Long-range transport** Lag0: 0.94 (0.84, 1.05) Lag1: 1.00 (0.92, 1.08) Lag2: 1.11 (1.02, 1.20) Lag3: 1.06 (0.95, 1.18) **Oil combustion** Lag0: 0.87 (0.57, 1.32) Lag1: 1.04 (0.75, 1.45) Lag2: 1.10 (0.83, 1.46) Lag3: 1.12 (0.79, 1.58) **Salt** Lag0: 1.03 (0.57, 1.85) Lag1: 0.72 (0.37, 1.40) Lag2: 0.66 (0.31, 1.40) Lag3: 1.55 (0.83, 2.89) **Local traffic** Lag0: 0.91 (0.69, 1.21) Lag1: 1.22 (0.88, 1.69) Lag2: 1.53 (1.19, 1.97) Lag3: 0.98 (0.78, 1.23) ST segment depression defined as $>$0.1 mV with horizontal or downward slope (n = 46) **Crustal** Lag0: 0.76 (0.42, 1.35) Lag1: 0.41 (0.22, 0.79) Lag2: 1.17 (0.65, 2.09) Lag3: 1.60 (0.72, 3.59) **Long-range transport** Lag0: 0.98 (0.86, 1.10) Lag1: 1.03 (0.95, 1.12) Lag2: 1.11 (1.02, 1.21) Lag3: 1.02 (0.95, 1.10) **Oil combustion** Lag0: 0.95 (0.61, 1.49) Lag1: 1.13 (0.76, 1.68) Lag2: 1.33 (0.98, 1.80) Lag3: 1.29 (0.90, 1.86) **Salt** Lag0: 1.15 (0.56, 2.38) Lag1: 0.90 (0.44, 1.81) Lag2: 1.39 (0.63, 3.08) Lag3: 1.93 (1.00, 3.72) **Local traffic** Lag0: 0.89 (0.64, 1.23) Lag1: 1.21 (0.86, 1.71) Lag2: 1.37 (1.03, 1.83) Lag3: 1.03 (0.80, 1.32) Adjusted ORs for the association of indicator elements of $PM_{2.5}$ sources and ST segment depressions in multipollutant models (models include all 5 indicator elements). ST segment depression defined as $>$0.1 mV (n = 62) **Si (Crustal)**

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			Lag0: 0.73 (0.39, 1.38) Lag1: 0.48 (0.25, 0.93) Lag2: 0.78 (0.35, 1.71) Lag3: 1.95 (0.69, 5.48) **S (Long-range transport)** Lag0: 0.70 (0.25, 1.95) Lag1: 0.58 (0.23, 1.47) Lag2: 1.08 (0.44, 2.63) Lag3: 1.60 (0.73, 3.48) **Ni (Oil combustion)** Lag0: 0.78 (0.30, 2.04) Lag1: 1.20 (0.58, 2.46) Lag2: 1.15 (0.61, 2.18) Lag3: 1.02 (0.41, 2.54) **Cl (Salt)** Lag0: 1.03 (0.79, 1.34) Lag1: 0.88 (0.56, 1.38) Lag2: 1.02 (0.62, 1.69) Lag3: 1.27 (0.85, 1.91) **ABS (Local traffic)** Lag0: 0.92 (0.36, 2.37) Lag1: 1.83 (0.73, 4.59) Lag2: 4.46 (1.69, 11.79) Lag3: 0.92 (0.40, 2.12) ST segment depression defined as >0.1 mV with horizontal or downward slope (n = 46) **Si (Crustal)** Lag0: 0.67 (0.33, 1.36) Lag1: 0.34 (0.15, 0.81) Lag2: 0.81 (0.33, 2.00) Lag3: 1.90 (0.64, 5.65) **S (Long-range transport)** Lag0: 0.84 (0.29, 2.47) Lag1: 0.89 (0.34, 2.32) Lag2: 1.36 (0.54, 3.45) Lag3: 1.12 (0.53, 2.40) **Ni (Oil combustion)** Lag0: 1.10 (0.36, 3.37) Lag1: 1.16 (0.45, 2.96) Lag2: 1.64 (0.84, 3.20) Lag3: 1.63 (0.64, 4.14) **Cl (Salt)** Lag0: 1.13 (0.80, 1.62) Lag1: 0.99 (0.58, 1.68) Lag2: 1.55 (0.87, 2.76) Lag3: 1.45 (0.94, 2.25) **ABS (Local traffic)** Lag0: 0.74 (0.25, 2.23) Lag1: 1.76 (0.62, 5.00) Lag2: 4.86 (1.55, 15.26) Lag3: 0.97 (0.39, 2.41)

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Lanki et al. (2008, 191984) **Period of Study:** Jan 1999 – Apr 1999 **Location:** Helsinki, Finland	**Outcome:** ST Segment Depressions >0.1 mV **Age Groups:** 50+ **Study Design:** panel **N:** 41 elderly people w/ CHD **Statistical Analyses:** Logistic Regression Model **Covariates:** long-term time trend, temperature, humidity, change in heart rate following exercise test **Dose-response Investigated?** No **Statistical Package:** R **Lags Considered:** lags 0-24h	**Pollutant:** $PM_{2.5}$ **Averaging Time:** hourly **25th, 50th, 75th, Max:** Personal $PM_{2.5}$ 1h: 6.9, 11.2, 15.8, 41.5 4h: 5.9, 10.0, 14.6, 41.3 8h: 5.0, 7.9, 13.0, 34.9 12h: 5.2, 7.8, 12.1, 28.8 22h: 6.6, 9.3, 13.0, 30.2 Outdoor $PM_{2.5}$ 1h: 8.9, 12.9, 17.8, 42.9 4h: 8.8, 12.5, 17.6, 40.8 8h: 8.3, 12.1, 17.2, 39.2 12h: 8.3, 11.9, 17.0, 37.0 24h: 9.0, 12.5, 17.7, 30.5 **Monitoring Stations:** 1 **Co-pollutant:** $PM_{<0.1}$ **Co-pollutant Correlation** Personal & Outdoor $PM_{2.5}$ 1h & 1h: 0.70 4h & 4h: 0.54 8h & 8h: 0.60 12h & 12h: 0.50 22h & 24h: 0.80 **Notes:** 1-22h pollutant averaging times. Correlations also available for personal-personal and outdoor-outdoor.	**PM Increment:** 10 $\mu g/m^3$ **Odds Ratio (Lower CI, Upper CI):** Personal $PM_{2.5}$ 1h avg: 3.26 (1.07, 9.99)* 4h avg: 2.42 (0.75, 7.83) 8h avg: 1.57 (0.49, 5.09) 12h avg: 1.96 (0.44, 8.64) 22h avg: 2.06 (0.30, 14.10) Outdoor $PM_{2.5}$ 1h avg: 1.77 (0.87, 3.58) 4h avg: 2.47 (1.05, 5.85)* 8h avg: 1.83 (0.80, 4.20) 12h avg: 1.90 (0.77, 4.65) 24h avg: 1.60 (0.59, 4.39) *$p<0.05$
Reference: Liao et al. (2007, 180272) **Period of Study:** 1999-2004 **Location:** 24 US states	**Outcome:** Ectopy **Age Groups:** women 50-79 yrs **Study Design:** panel **N:** 57,422 **Statistical Analyses:** logistic regression & random effects modeling **Covariates:** age, race, center, education, history of CVD/chronic lung disease, rel. humidity, temperature, smoking **Dose-response Investigated?** No **Statistical Package:** SAS, Stata **Lags Considered:** lags 0-365d	**Pollutant:** $PM_{2.5}$ **Averaging Time:** daily **Mean (SD)*:** All: 13.8 (79) No Ectopy: 13.8 (7.9) Any Ectopy: 13.8 (7.6) **5th, 95th percentile*:** All: 5, 29.1 No Ectopy: 5, 29.2 Any Ectopy: 5.06, 28.5 **Monitoring Stations:** NR‡ **Copollutant:** PM_{10} **Co-pollutant Correlation** NR *Lag 1 ‡Monitors used in model for spatial interpolation of daily PM values.	**PM Increment:** 10 $\mu g/m^3$ **Percent Change (Lower CI, Upper CI):** All Ventricular Ectopy Lag 0: 1.01 (0.91, 1.13) Lag 1: 1.07 (0.96, 1.20) Lag 2: 1.09 (0.98, 1.21) Current Smoker Ventricular Ectopy Lag 0: 1.52 (1.04, 2.24) Lag 1: 2 (1.32, 3.03) Lag 2: 1.59 (0.99, 2.55) Nonsmoker Ventricular Ectopy Lag 0: 0.99 (0.89, 1.11) Lag 1: 1.05 (0.94, 1.17) Lag 2: 1.08 (0.97, 1.21) All Supraventricular Ectopy Lag 0: 1.04 (0.96, 1.13) Lag 1: 1.01 (0.93, 1.10) Lag 2: 0.96 (0.87, 1.05) All Ventricular or Supraventricular Ectopy Lag 0: 1.03 (0.96, 1.11) Lag 1: 1.04 (0.97, 1.11) Lag 2: 1 (0.94, 1.07)
Reference: Lipsett et al. (2006, 088753) **Period of Study:** February–May 2000 **Location:** Coachella Valley, CA	**Outcome:** HRV parameters, specifically SDNN, SDANN, r-MSSD, LF, HF, total power, triangular index (TRII). **Study Design:** Panel study **N:** 19 non-smoking adults with coronary artery disease **Statistical Analysis:** Mixed linear regression models with random effects parameters	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 2 h **Mean (range)** Indio: 23.2 (6.3-90.4) Palm Springs: 14 (4.7-52) **Monitoring Stations:** 2 **Copollutant:** O_3	**PM Increment:** SE*100 **Effect Estimate (change in HRV per unit increase in PM concentration):** SDNN: -0.37 msec (SE = 1.01) **Notes:** Weekly ambulatory 24 h ECG recordings (once per week for up to 12 weeks), using Holter monitors, were made. Subjects' residences were within 5 miles of one of two PM monitoring sites. Decreased HRV was associated with $PM_{2.5}$, but these effects were not statistically significant. Regressed HRV parameters against 18: 00–20: 00 mean particulate pollution.

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Ljungman et al. (2008, 180266) **Period of Study:** Aug 2001 – Dec 2006 **Location:** Stockholm, Sweden	**Outcome:** Ventricular Arrhythmia **Age Groups:** 28-85 yrs **Study Design:** case-crossover **N:** 88 patients w/ implantable cardioverter defibrillators **Statistical Analyses:** conditional logistic regression **Covariates:** temperature, humidity, pressure, ischemic heart disease, ejection fraction, heart disease, diabetes, use of beta-blockers, age, BMI, location at time of arrhythmia, distance from air pollution monitor **Dose-response Investigated?** No **Statistical Package:** Stata, S-plus **Lags Considered:** lags 2-24h	**Pollutant:** $PM_{2.5}$ **Averaging Time:** hourly **Median:** 2h: 9.17 24h: 9.49 **Min:** 2h: 0.15 24h: 2.97 **Max:** 2h: 99.25 24h: 47.07 **IQR:** 2h: 6.69 24h: 5.27 **Monitoring Stations:** 1 **Copollutant:** PM_{10}, NO_2 **Co-pollutant Correlation** NR	**PM Increment:** Interquartile Range **Odds Ratio (Lower CI, Upper CI):** 2h: 1.23 (0.84, 1.80) 24h: 1.28 (0.90, 1.84) **Notes:** OR of ventricular arrhythmia for an IQR increase of air pollutants in different subgroups (figure 2)
Reference: Ljungman et al. (2009, **191983**) **Period of Study:** May 2003 – July 2004 **Location:** Athens, Greece Helsinki, Finland Ausburg, Germany Barcelona, Spain Rome, Italy Stokholm, Sweeden	**Outcome:** Interleukin-6 Response **Age Groups:** 35-80 yrs **Study Design:** panel **N:** 955 male myocardial infarction survivors **Statistical Analyses:** Additive Mixed Models **Covariates:** age, sex, BMI, city, HDL/total cholesterol, smoking, alcohol intake, HbA1c, NT-proBNP, history of MI, heart failure, or diabetes, phlegm **Dose-response Investigated?** No **Statistical Package:** NR **Lags Considered:** 1d	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24h **Mean:** 17.7 **25th:** 10.9 **75th:** 21.9 **Monitoring Stations:** NR **Copollutant:** CO, NO_2, PNC, $PM_{2.5}$ **Co-pollutant Correlation** PM_{10}: 0.81	**PM Increment:** Interquartile Range (11.0 $\mu g/m^3$) **Change of IL-6 (Lower CI, Upper CI), p-value:** 0.6 (-0.8, 2.0), 0.40
Reference: Luttman-Gibson et al. (2006, 089794) **Period of Study:** June–December 2000 **Location:** Steubenville, OH	**Outcome:** Heart rate variability **Age Groups:** **Study Design:** Panel study **N:** 32 participants **Statistical Analysis:** Linear mixed models	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 1 h 24 h **Mean (IQR)** $PM_{2.5}$: 20.0 (15.2) Sulfate: 6.9 (5.1) EC: 1.1 (0.6) **Copollutant:** NO_2, SO_2, O_3	**PM Increment:** IQR **Percent change (95% CI):** Each 13.4 $\mu g/m^3$ increase in 24 hour mean $PM_{2.5}$ concentration was associated with: SDNN: -4.0% (95% CI: -7.0% to -0.9%) r-MSSD: -6.5% (95% CI: -12.1% to -0.6%) HF: -11.4% (95% CI: -21.5% to -0.1%) Each 5.1 $\mu g/m^3$ increase in sulfates on the previous day was associated with: SDNN: -3.3% (95% CI: -6.0% to -0.5%) r-MSSD: -5.6% (95% CI: -10.7%, 0.2%) HF: -10.3% (95% CI: -19.5% to -0.1%) **Notes:** The authors conclude that increases in both traffic related particles and sulfates may adversely effect autonomic function.

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Mar et al. (2005, 087566) **Period of Study:** 1999–2001 **Location:** Seattle, WA	**Outcome:** Change in arterial O_2 saturation, heart rate, and blood pressure (SBP and DBP) **Age Groups:** > 75 years **Study Design:** Panel study **N:** 88 elderly subjects **Statistical Analysis:** GEE	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean (SD):** Personal: 9.3(8.4) Indoor: 7.4 (4.8) Outdoor: 9.0 (4.6)	**PM Increment:** 10 µg/m^3 **Unit change in measure (95% CI):** Among all subjects: Each increase in outdoor same day $PM_{2.5}$ was associated with: SBP: -0.81 mmHg (95% CI: -2.34, 0.73) DBP: -0.46 mmHg (95% CI: -1.49, 0.57) H: -0.75 beats/min (95% CI: -1.42 to -0.07) Each increase in indoor same day $PM_{2.5}$ was associated with: SBP: 0.92 mmHg (95% CI: -2.04, 3.87) DBP: 0.38 mmHg (95% CI: -1.43, 2.20) H: 0.22 beats/min (95% CI: -0.71, 1.16) Each increase in personal same day $PM_{2.5}$ was associated with: SBP: 0.37 mmHg (95% CI: -0.93, 1.67) DBP: -0.20 mmHg (95% CI: -0.85, 0.46) H: 0.44 beats/min (95% CI: 0.04, 0.84) **Notes:** Results by health status presented in Figure 1 Used 2 sessions that each were 10 consecutive days of measurements Used personal, indoor, and outdoor measures of $PM_{2.5}$
Reference: Metzger et al. (2007, 092856) **Period of Study:** August 1998–December 2002 **Location:** Atlanta, GA	**Outcome:** Days with any event recorded by the ICD, days with ICD shocks/defibrillation and days with either cardiac pacing or defibrillation **Study Design:** Repeated measures **N:** 884 subjects between 1993 and 2002 **Statistical Analysis:** Logistic regression with GEE to account for residual autocorrelation within subjects	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean (SD):** $PM_{2.5}$: 17.8 (8.6) $PM_{2.5}$ sulfates: 5.0 (3.4) $PM_{2.5}$ EC: 1.7 (1.2) $PM_{2.5}$ OC: 4.4 (2.4) $PM_{2.5}$ water-soluble metals: 0.029 (0.024) **Percentiles:** $PM_{2.5}$: Median: 16.2 $PM_{2.5}$ sulfates: Median: 4.1 $PM_{2.5}$ EC: Median: 1.4 $PM_{2.5}$ OC: Median: 3.9 $PM_{2.5}$ water-soluble metals: Median: 0.022 **Copollutant:** O_3 NO_2 CO SO_2 oxygenated hydrocarbons	**PM Increment: OR (95% CI):** Outcome = Any event recorded by ICD $PM_{2.5}$ OR = 1.00 (95% CI: 0.95, 1.04) $PM_{2.5}$ EC OR = 1.01 (95% CI: 0.98, 1.05) $PM_{2.5}$ OC OR = 1.01 (95% CI: 0.98, 1.03) $PM_{2.5}$ Sulfates OR = 0.99 (95% CI: 0.93, 1.06) $PM_{2.5}$ Water soluble metals OR = 0.95 (95% CI: 0.90, 1.00
Reference: O'Neill et al. (2007, 091362) **Period of Study:** May 1998–Dec 2002 **Location:** Boston, MA	**Outcome:** Soluble intercellular adhesion molecule 1 (ICAM-1) vascular cell adhesion molecule 1 (VCAM-1) von Willebrand factor (vWF) **Age Groups:** Mean (SD): 56.6 (10.6) **Study Design:** Cross-sectional **N:** 92 participants (type 2 diabetic patients) **Statistical Analyses:** linear regression **Covariates:** Apparent temperature, season, age, race, sex, glycosylated hemoglobin, cholesterol, smoking history, BMI **Dose-response Investigated?** No	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h (lagged moving averages of days 0 to 1, 2, 3, 4, and 5) **Mean (SD):** 11.4 (5.9) descriptive statistics represent entire study period **Percentiles:** IQR range: 7.6 **Range (Min, Max):** 0.07, 33.7) **Monitoring Stations:** 1 site **Copollutant:** $PM_{2.5}$ BC SO_4^{2-}	**PM Increment:** IQR (specific to lag period) **Effect Estimate [Lower CI, Upper CI]:** % change per IQR of $PM_{2.5}$ **ICAM-1** - All subjects Lag 0: 2.87 (-4.63, 10.95) 2 dma: 2.25 (-5.15, 10.22) 3 dma: 1.48 (-5.63, 9.11) 4 dma: 1.80 (-4.98, 9.07) 5 dma: 1.51 (-5.30, 8.80) 6 dma: 2.12 (-4.23, 8.89) **Subjects not known to be taking statins** Lag 0: 5.47 (-3.74, 15.57) 2 dma: 5.70 (-3.70, 16.01) 3 dma: 4.57 (-4.31, 14.27) 4 dma: 4.57 (-4.27, 14.23) 5 dma: 3.80 (-4.84, 13.22) 6 dma: 3.79 (-4.49, 12.80)

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
	Statistical Package: NR		**Subjects who report smoking in the past (but not within 6 months)** Lag 0: 0.9 (-9.56, 12.66) 2 dma: 0.40 (-12.08, 14.65) 3 dma: 1.34 (-9.23, 13.14) 4 dma: 2.29 (-6.84, 12.30) 5 dma: 1.09 (-8.30, 11.44) 6 dma: 3.08 (-6.30, 13.40); **Subjects who did not report smoking in the past** Lag 0: 0.46 (-8.23, 9.97) 2 dma: 1.37 (-7.96, 11.65) 3 dma: -0.96 (-10.01, 9.00) 4 dma: -1.34 (-10.35, 8.58) 5 dma: -0.87 (-10.17, 9.40) 6 dma: -1.78 (-10.64, 7.94) **VCAM-1** - All subjects Lag 0: 6.88 (-2.88, 17.62) 2 dma: 8.18 (-1.43, 18.72) 3 dma: 6.92 (-1.66, 16.25) 4 dma: 6.46 (-1.16, 14.66) 5 dma: 8.57 (0.05, 17.80) 6 dma: 11.76 (3.48, 20.70) **Subjects not known to be taking statins** Lag 0: 10.26 (-0.64, 22.35) 2 dma: 15.02 (3.76, 27.49) 3 dma: 14.59 (3.94, 26.34) 4 dma: 15.15 (4.54, 26.84) 5 dma: 16.16 (5.77, 27.58) 6 dma: 17.66 (7.77, 28.45) **Subjects who report smoking in the past (but not within 6 months)** Lag 0: 13.2 (-1.30, 29.72) 2 dma: 18.4 (0.69, 39.33) 3 dma: 15.7 (1.19, 32.30) 4 dma: 13.1 (0.88, 26.78) 5 dma: 13.2 (0.49, 27.58) 6 dma: 16.2 (3.76, 30.10) **Subjects who did not report smoking in the past** Lag 0: -3.12 (-12.41, 7.17) 2 dma: -0.34 (-10.57, 11.05) 3 dma: -1.09 (-11.15, 10.12) 4 dma: -0.81 (-10.91, 10.43) 5 dma: 2.07 (-8.59, 13.96) 6 dma: 4.89 (-5.56, 16.50) **vWF** - All subjects Lag 0: 15.16 (-9.79, 47.01) 2 dma: 12.57 (-9.19, 39.55) 3 dma: 25.14 (-9.87, 73.74) 4 dma: 23.42 (-9.47, 68.25) 5 dma: 17.92 (-10.22, 54.87) 6 dma: 20.48 (-8.82, 59.22) **Subjects not known to be taking statins** Lag 0: 7.40 (-19.82, 43.88) 2 dma: 7.10 (-19.09, 41.76) 3 dma: 10.78 (-17.92, 49.52) 4 dma: 11.61 (-16.64, 49.42) 5 dma: 9.15 (-20.32, 49.53) 6 dma: 7.91 (-20.70, 46.85) **Subjects who report smoking in the past (but not within 6 months)** Lag 0: 19.23 (-24.29, 87.77) 2 dma: 19.92 (-29.65,104.41) 3 dma: 29.54 (-17.24, 102.76) 4 dma: 41.98 (-6.95, 116.63) 5 dma: 44.05 (-1.23, 110.07) 6 dma: 50.39 (9.35, 106.82) **Subjects who did not report smoking in the past**

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			Lag 0: -14.21 (-53.20, 57.24) 2 dma: -20.66 (-63.14, 70.77) 3 dma: -28.89 (-68.43, 60.19) 4 dma: -23.51 (-55.11, 30.34) 5 dma: -29.18 (-60.08, 25.66) 6 dma: -30.68 (-55.95, 9.08)
Reference: O'Neill et al. (2007, 091362) **Period of Study:** May 1998–Dec 2002 **Location:** Boston, MA	**Outcome:** Soluble intercellular adhesion molecule 1 (ICAM-1) vascular cell adhesion molecule 1 (VCAM-1) von Willebrand factor (vWF) **Age Groups:** Mean (SD): 56.6 (10.6) **Study Design:** Cross-sectional **N:** 92 participants (type 2 diabetic patients) **Statistical Analyses:** Linear regression **Covariates:** Apparent temperature, season, age, race, sex, glycosylated hemoglobin, cholesterol, smoking history, BMI **Dose-response Investigated?** No **Statistical Package:** NR	**Pollutant:** BC **Averaging Time:** 24 h (lagged moving averages of days 0 to 1, 2, 3, 4, and 5) **Mean (SD):** 1.1 (0.8) descriptive statistics represent entire study period **Percentiles:** IQR range: 0.8 **Range (Min, Max):** 0.2, 5.8 **Monitoring Stations:** 1 site **Copollutant:** $PM_{2.5}$ BC SO_4^{2-}	**PM Increment:** IQR (specific to lag period) **Effect Estimate [Lower CI, Upper CI]:** % change per IQR of BC **ICAM-1**- -All subjects Lag 0: 5.09 (-2.37, 13.11) 2 dma: 3.97 (-10.24, 20.42) 3 dma: 5.10 (-10.17, 22.96) 4 dma: 8.38 (-6.46, 25.56) 5 dma: 10.09 (-7.36, 30.83) 6 dma: 10.58 (-5.34, 29.18) **Subjects not known to be taking statins** Lag 0: 5.77 (-3.92, 16.44) 2 dma: 2.39 (-7.65, 13.52) 3 dma: 0.84 (-8.16, 10.73) 4 dma: 1.67 (-6.71, 10.80) 5 dma: 1.55 (-6.46, 10.24) 6 dma: 2.20 (-6.47, 11.68) **Subjects who report smoking in the past (but not within 6 months)** Lag 0: 5.84 (0.87, 11.05) 2 dma: 5.08 (-2.34, 13.07) 3 dma: 4.44 (-2.70, 12.11) 4 dma: 5.02 (-1.78, 12.29) 5 dma: 5.89 (-2.14, 14.58) 6 dma: 6.73 (-1.54, 15.70) **Subjects who did not report smoking in the past** Lag 0: 6.04 (0.87, 11.48) 2 dma: 6.54 (-1.64, 15.39) 3 dma: 5.86 (-1.90, 14.22) 4 dma: 6.11 (-1.18, 13.94) 5 dma: 6.89 (-1.42, 15.89) 6 dma: 7.86 (-1.35, 17.94) **VCAM-1** - All subjects Lag 0: 9.26 (2.98, 15.91) 2 dma: 10.18 (1.93, 19.10) 3 dma: 15.45 (2.70, 29.78) 4 dma: 17.97 (3.63, 34.30) 5 dma: 23.83 (8.41, 41.44) 6 dma: 27.51 (11.96, 45.21) **Subjects not known to be taking statins** Lag 0: 9.19 (3.23, 15.49) 2 dma: 14.64 (5.02, 25.14) 3 dma: 14.39 (5.30, 24.28) 4 dma: 14.19 (5.71, 23.36) 5 dma: 19.11 (9.44, 29.65) 6 dma: 22.60 (11.79, 34.45) **Subjects who report smoking in the past (but not within 6 months)** Lag 0: 12.4 (2.77, 22.92) 2 dma: 28.5 (8.38, 52.24) 3 dma: 25.14 (3.50, 51.30) 4 dma: 23.1 (2.70, 47.58) 5 dma: 32.0 (7.29, 62.30) 6 dma: 31.8 (9.74, 58.26) **Subjects who did not report smoking in the past** Lag 0: 5.15 (-5.63, 17.17) 2 dma: 2.09 (-9.07 ,14.61) 3 dma: 3.90 (-6.38, 15.31) 4 dma: 4.92 (-4.63, 15.43) 5 dma: 7.89 (-1.31, 17.95)

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			6 dma: 10.97 (0.98, 21.96) **vWF**- All subjects Lag 0: 7.96 (-4.34, 21.84) 2 dma: 14.87 (-2.85, 35.82) 3 dma: 15.34 (-3.22, 37.45) 4 dma: 15.47 (-7.60, 44.31) 5 dma: 19.50 (-8.89, 56.74) 6 dma: 20.53 (-9.80, 61.05) **Subjects not known to be taking statins** Lag 0: 3.23 (-8.91, 17.00) 2 dma: 9.82 (-8.39, 31.66) 3 dma: 17.79 (-16.03, 65.21) 4 dma: 13.14 (-18.71, 57.47) 5 dma: 16.14 (-20.43, 69.52) 6 dma: 13.25 (-22.09, 64.62) **Subjects who report smoking in the past (but not within 6 months)** Lag 0: 7.63 (-17.01, 39.58) 2 dma: 37.64 (-7.18, 104.10) 3 dma: 75.41 (6.16, 189.85) 4 dma: 72.05 (-3.34, 206.22) 5 dma: 73.14 (6.94, 180.32) 6 dma: 71.23 (14.00, 157.19) **Subjects who did not report smoking in the past** Lag 0: 10.22 (-23.14, 58.04) 2 dma: 17.07 (-18.86, 68.91) 3 dma: 6.56 (-42.75, 98.36) 4 dma: -9.20 (-65.79, 140.99) 5 dma: -23.86 (-71.05, 100.29) 6 dma: -48.69 (-77.75, 18.29)
Reference: O'Neill et al. (2005, 088423) **Period of Study:** Baseline period: May 1998–January 2000 Time trial: 2000–2002 **Location:** Boston, MA	**Outcome:** Changes in vascular reactivity, specifically percent change in brachial artery diameter (flow-mediated and nitroglycerin-mediated) **N:** 270 patients with diabetes or at risk of diabetes, who participated in non-air pollution related studies at the Joselyn Diabetes Center in Boston **Statistical Analysis:** Linear regression	**Pollutant:** $PM_{2.5}$ **Mean (SD):** 11.5 (6.4) **Range:** 1.1–40.0 **Monitoring Stations:** 1 **Copollutant:** Sulfates BC Ultrafine particle counts	**PM Increment:** IQR (value not given) **Percent change (95% CI):** $PM_{2.5}$ 6-day moving avg Nitroglycerin-mediated reactivity: -7.6% (95% CI: 12.8% to -2.1%) **Notes:** $PM_{2.5}$ was positively associated with nitroglycerin-mediated reactivity an association was also reported with ultrafine particles. Effect estimates were larger in type II than type I diabetes. BC and sulfate increases were associated with decreased flow-mediated reactivity among those with diabetes. Although the largest associations were with the 6-day moving avg, similar patterns and quantitatively similar results appear in the other lags.
Reference: O'Neill et al. (2007, 091362) **Period of Study:** May 1998–Dec 2002 **Location:** Boston, MA	**Outcome:** soluble intercellular adhesion molecule 1 (ICAM-1) vascular cell adhesion molecule 1 (VCAM-1) von Willebrand factor (vWF) **Mean Age:** 56.6 (10.6) **Study Design:** Cross-sectional **N:** 92 participants (type 2 diabetic patients) **Statistical Analyses:** Linear regression **Covariates:** Apparent temperature, season, age, race, sex, glycosylated hemoglobin, cholesterol, smoking history, BMI **Dose-response Investigated?** No **Statistical Package:** NR	**Pollutant:** SO42– **Averaging Time:** 24 h (lagged moving averages of days 0 to 1, 2, 3, 4, and 5) **Mean (SD):** 3.0 (2.0) descriptive statistics represent entire study period **Percentiles:** IQR range: 2.2 **Range (Min, Max):** 0.5, 9.6) **Monitoring Stations:** 1 site **Copollutant:** $PM_{2.5}$, BC, SO_4^{2-}	**PM Increment:** IQR (specific to lag period) **Effect Estimate [Lower CI, Upper CI]:** % change per IQR of $PM_{2.5}$ **ICAM-1** All subjects Lag 0: 5.30 (-2.60, 13.83) 2 dma: 4.02 (-3.26, 11.85) 3 dma: 4.03 (-5.34, 14.34) 4 dma: -0.79 (-7.30, 6.18) 5 dma: 1.06 (-7.10, 9.93) 6 dma: 3.15 (-5.66, 12.78) **Subjects not known to be taking statins** Lag 0: 10.14 (0.44, 20.77) 2 dma: 9.39 (-1.28, 21.20) 3 dma: 10.93 (-2.23, 25.85) 4 dma: -0.24 (-9.66, 10.16) 5 dma: 4.03 (-8.66, 18.47) 6 dma: 5.66 (-7.52, 20.72) **Subjects who report smoking in the**

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			past (but not within 6 months) Lag 0: -4.00 (-24.79, 22.52) 2 dma: -4.82 (-18.01, 10.48) 3 dma: -7.19 (-23.66, 12.83) 4 dma: -9.8 (-27.96, 12.97) 5 dma: -10.4 (-29.92, 14.44) 6 dma: -6.8 (-25.72, 17.03) **Subjects who did not report smoking in the past** Lag 0: 6.67 (-4.34, 18.94) 2 dma: 5.65 (-4.67, 17.10) 3 dma: 10.21 (-5.83, 28.99) 4 dma: 0.80 (-9.94, 12.83) 5 dma: 2.80 (-10.85, 18.54) 6 dma: 5.15 (-7.78, 19.89) **VCAM-1** All subjects Lag 0: -0.04 (-3.75, 3.80) 2 dma: 0.94 (-4.79, 7.01) 3 dma: -0.87 (-3.50, 1.82) 4 dma: 0.13 (-2.02, 2.34) 5 dma: -0.47 (-2.67, 1.78) 6 dma: -0.46 (-1.99, 1.09) **Subjects not known to be taking statins** Lag 0: -1.34 (-11.23, 9.66) 2 dma: -0.19 (-11.13, 12.09) 3 dma: -2.84 (-13.90, 9.64) 4 dma: 4.28 (-6.18, 15.90) 5 dma: -0.26 (-13.44, 14.93) 6 dma: -3.44 (-16.51, 11.67) **Subjects who report smoking in the past (but not within 6 months)** Lag 0: 0.07 (-23.40, 30.73) 2 dma: -5.62 (-20.77, 12.43) 3 dma: -26.92 (-33.31 to -19.91) 4 dma: -3.06 (-28.01,30.56) 5 dma: -6.42 (-30.75, 26.47) 6 dma: -6.46 (-28.55, 22.47) **Subjects who did not report smoking in the past** Lag 0: -3.28 (-12.66, 7.12) 2 dma: -3.17 (-11.75, 6.23) 3 dma: -9.67 (-22.07, 4.70) 4 dma: -5.51 (-14.28, 4.15) 5 dma: -12.17 (-22.05 to -1.05) 6 dma: -11.77 (-20.95 to -1.52) **vWF** (sulfate measures not available)
Reference: Park et al. (2008, 156845) **Period of Study:** Jan 1995–Jun 2005 **Location:** Greater Boston area, MA	**Outcome:** Total homocysteine (tHcy) **Mean Age:** 73.6 ± 6.9 yrs **Study Design:** Cross-sectional and longitudinal analyses performed **N:** 960 men **Statistical Analyses:** Generalized additive models (also hierarchical mixed-effects regression models to assess repeated measures of tHcy) **Covariates:** Model 1: season, age, long-term trend, apparent temperature Model 2: further adjustment for BMI, systolic blood pressure, smoking status, pack years of cigarettes, alcohol consumption Model 3: further adjustment for serum creatinine, plasma folate, vitamin B6, and vitamin B12 **Dose-response Investigated?** Modeled continuous covariates as penalized splines to determine if association with tHcy was	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h (moving averages up to 7 days prior to blood collection) **Mean (SD):** 12.0 (6.6) **Median:** 10.6 **Range (Min, Max):** 2.0, 62.0 **Monitoring Stations:** 1 site **Copollutant:** $PM_{2.5}$ BC (r = 0.51) OC (r = 0.51) SO_4^{2-} (r = 0.85)	**PM Increment:** IQR **Effect Estimate [Lower CI, Upper CI]:** Estimated % change in tHcy per IQR increase in pollutant. Lag model Concurrent day. IQR: 7.66 Model 1: 1.32 (-0.83, 3.52) Model 2: 1.55 (-0.77, 3.91) Model 3: 1.57 (-0.38, 3.56) 1-day previous. IQR: 6.91 Model 1: -1.43 (-3.51, 0.69) Model 2: -1.41 (-3.53, 0.76) Model 3: -1.28 (-3.12, 0.60) 2-day moving avg. IQR: 6.47 Model 1: 0.04 (-2.13, 2.26) Model 2: -0.07 (-2.26, 2.17) Model 3: 0.25 (-1.69, 2.22) 3-day moving avg. IQR: 5.83 Model 1: -0.64 (-2.92, 1.69) Model 2: -0.74 (-3.04, 1.61) Model 3: -0.59 (-2.63, 1.49)

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
	linear **Statistical Package:** R software		4-day moving avg. IQR: 5.21 Model 1: -0.63 (-2.94, 1.72) Model 2: -0.86 (-3.19, 1.52) Model 3: -0.73 (-2.78, 1.37) 5-day moving avg. IQR: 4.68 Model 1: -0.51 (-2.79, 1.83) Model 2: -0.82 (-3.13, 1.54) Model 3: -0.84 (-2.85, 1.22) 6-day moving avg. IQR: 4.50 Model 1: -0.91 (-3.32, 1.56) Model 2: -1.32 (-3.76, 1.17) Model 3: -1.44 (-3.58, 0.74) 7-day moving avg. IQR: 4.20 Model 1: -0.84 (-3.27, 1.64) Model 2: -1.19 (-3.64, 1.33) Model 3: -1.69 (-3.84, 0.51) Stratified analyses: No significant difference in effect of $PM_{2.5}$ among those with high and low levels of vitamins
Reference: Park et al. (2008, 156845) **Period of Study:** Jan 1995–Jun 2005 **Location:** Greater Boston area, MA	**Outcome:** Total homocysteine (tHcy) **Mean Age:** 73.6 ± 6.9 yrs **Study Design:** cross-sectional and longitudinal analyses performed **N:** 960 men **Statistical Analyses:** Generalized additive models (also hierarchical mixed-effects regression models to assess repeated measures of tHcy) **Covariates:** Model 1: season, age, long-term trend, apparent temperature Model 2: further adjustment for BMI, systolic blood pressure, smoking status, pack years of cigarettes, alcohol consumption Model 3: further adjustment for serum creatinine, plasma folate, vitamin B6, and vitamin B12 **Dose-response Investigated?** Modeled continuous covariates as penalized splines to determine if association with tHcy was linear **Statistical Package:** R software	**Pollutant:** BC **Averaging Time:** 24 h (moving averages up to 7 days prior to blood collection) **Mean (SD):** 0.99 (0.56) **Median:** 0.87 **Range (Min, Max):** 0.07, 3.7 **Monitoring Stations:** 1 site **Copollutant** **(correlation):** $PM_{2.5}$ (r = 0.51) BC OC (r = 0.0.51) SO_4^{2-} (r = 0.50)	**PM Increment:** IQR **Effect Estimate [Lower CI, Upper CI]:** Estimated % change in tHcy per IQR increase in pollutant. Lag model Concurrent day. IQR: 0.66 Model 1: 2.64 (-0.12, 5.48) Model 2: 2.62 (-0.17, 5.48) Model 3: 3.13 (0.76, 5.55) 1-day previous. IQR: 0.66 Model 1: 1.46 (-0.98, 3.96) Model 2: 1.32 (-1.14, 3.85) Model 3: 0.95 (-1.12, 3.05) 2-day moving avg. IQR: 0.60 Model 1: 2.75 (-0.18, 5.76) Model 2: 2.63 (-0.33, 5.67) Model 3: 2.59 (0.10, 5.14) 3-day moving avg. IQR: 0.57 Model 1: 2.95 (-0.44, 6.46) Model 2: 2.97 (-0.46, 6.51) Model 3: 3.12 (0.21, 6.11) 4-day moving avg. IQR: 0.52 Model 1: 3.94 (0.24, 7.78) Model 2: 3.76 (0.02, 7.64) Model 3: 3.00 (-0.13, 6.22) 5-day moving avg. IQR: 0.49 Model 1: 3.26 (-0.60, 7.27) Model 2: 2.64 (-1.23, 6.67) Model 3: 2.38 (-0.89, 5.77) 6-day moving avg IQR: 0.44 Model 1: 1.63 (-1.99, 5.38) Model 2: 1.03 (-2.62, 4.80) Model 3: 0.93 (-2.15, 4.11) 7-day moving avg. IQR: 0.44 Model 1: 1.38 (-2.45, 5.36) Model 2: 0.69 (-3.16, 4.70) Model 3: 0.45 (-2.81, 3.83) % change in tHcy per IQR increase in BC, 24-h avg Among those with low folate: 5.31 (2.26, 8.42) Among those with low B12: 5.06 (2.03, 8.17) nearly null associations among those with high levels

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Park et al. (2008, 156845) **Period of Study:** Jan 1995–Jun 2005 **Location:** Greater Boston area, MA	**Outcome:** Total homocysteine (tHcy) **Mean Age:** 73.6 ± 6.9 yrs **Study Design:** Cross-sectional and longitudinal analyses performed **N:** 960 men **Statistical Analyses:** Generalized additive models (also hierarchical mixed-effects regression models to assess repeated measures of tHcy) **Covariates:** Model 1: season, age, long-term trend, apparent temperature Model 2: further adjustment for BMI, systolic blood pressure, smoking status, pack years of cigarettes, alcohol consumption Model 3: further adjustment for serum creatinine, plasma folate, vitamin B6, and vitamin B12 **Dose-response Investigated?** Modeled continuous covariates as penalized splines to determine if association with tHcy was linear **Statistical Package:** R software	**Pollutant:** OC **Averaging Time:** 24 h (moving averages up to 7 days prior to blood collection) **Mean (SD):** 3.5 (1.8) **Median:** 3.1 **Range (Min, Max):** 0.29, 11.8 **Monitoring Stations:** 1 site **Copollutant (correlation):** $PM_{2.5}$ (r = 0.51) BC (r = 0.51) OC SO_4^{2-} (r = 0.41)	**PM Increment:** IQR **Effect Estimate [Lower CI, Upper CI]:** Estimated % change in tHcy per IQR increase in pollutant. Lag model Concurrent day. IQR: NA Model 1: NA Model 2: NA Model 3: NA 1-day previous. IQR: 2.00 Model 1: 2.12 (-0.98, 5.31) Model 2: 1.69 (-1.51, 5.00) Model 3: 1.87 (-0.81, 4.62) 2-day moving avg. IQR: 1.93 Model 1: -0.39 (-3.67, 3.01) Model 2: -0.88 (-4.26, 2.61) Model 3: 1.05 (-1.86, 4.06) 3-day moving avg. IQR: 1.68 Model 1: 0.53 (-2.66, 3.83) Model 2: 0.14 (-3.15, 3.54) Model 3: 1.32 (-1.44, 4.16) 4-day moving avg. IQR: 1.64 Model 1: 1.57 (-1.89, 5.15) Model 2: 1.42 (-2.14, 5.12) Model 3: 1.89 (-1.15, 5.03) 5-day moving avg, IQR: 1.60 Model 1: 2.27 (-1.49, 6.16) Model 2: 2.11 (-1.77, 6.15) Model 3: 2.12 (-1.29, 5.65) 6-day moving avg. IQR: 1.43 Model 1: 2.83 (-0.74, 6.52) Model 2: 2.78 (-0.90, 6.60) Model 3: 2.53 (-0.59, 5.74) 7-day moving avg. IQR: 1.23 Model 1: 2.75 (-0.41, 6.02) Model 2: 2.55 (-0.71, 5.92) Model 3: 2.55 (-0.21, 5.39) % change in tHcy per IQR increase in OC, 7-d avg. Among those with low B12: 5.23 (1.59, 9.01) nearly null associations among those with high levels

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Park et al. (2005, 057331) **Period of Study:** November 2000–October 2003 **Location:** Greater Boston area, MA	**Outcome:** Change in HRV (SDNN, HF, LF, LFHFR) **Mean age:** 72.7 years **Study Design:** Cross-sectional **N:** 497 adult males living in the Greater Boston, MA area	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 4 h 24 h 48 h **Mean (SD):** 11.4 (8.0) **Range:** 6.45–62.9 **Copollutant:** O_3, Particle number count, BC, NO_2, SO_2, CO	**PM Increment:** 8 µg/m^3 **Percent change (95% CI):** 48h mean $PM_{2.5}$: 20.8% decrease in HF (95% CI: 4.6%, 34.2%) 18.6% increase in LFHFR (4.1%, 35.2%). **Notes:** Subjects were monitored during a 4-min rest period between 8 a.m. and 1 p.m. Modifying effects of hypertension, IHD, diabetes, and use of cardiac/anti-hypertensive medications also examined. Linear regression analyses. This subject group is from the VA Normative Aging Study. The 4-h averaging period was most strongly associated with HRV indices. The PM effect was robust in models including O_3. The HRV change per IQR increase in $PM_{2.5}$ were larger in subjects with hypertension (n = 335) IHD (n = 142), and diabetes (n = 72). In addition, those who did not use calcium-channel blockers had a greater decline in LF associated with each IQR increase in $PM_{2.5}$ than did those who did use calcium channel blockers. IQR increases in 48h mean BC concentration were also associated with adverse changes in HRV, suggesting traffic pollution may be particularly toxic.
Reference: Park et al. (2006, 091245) **Period of Study:** November 2000–December 2004 **Location:** Greater Boston area, MA	**Outcome:** Change in HF **Study Design:** Cross-sectional **N: Statistical Analysis:** Linear regression models	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 48 h **Mean (SD):** $PM_{2.5}$: 11.7 (7.8) Sulfates: 3.3 (3.3) BC: 0.92 (0.46) **Copollutant:** O_3	**PM Increment:** 10 µg/m^3 **Percent change (95% CI):** Wild-type HFE genotype: 31.7% (95% CI: 10.3, 48.1) Among those with either of the two HFE variants, there was no association between 48h $PM_{2.5}$ and HF (shown in a graph, ~10% non-significant increase). **Notes:** Normative Aging Study. Examining association between PM and HF among those with and without the wild-type HFE genotype.
Reference: Pekkanen et al. (2002, 035050) **Period of Study:** Winter 1998 to 1999 **Location:** Helsinki, Finland	**Outcome:** ST Segment Depression (>0.1mV) **Study Design:** Panel of ULTRA Study participants **N:** 45 Subjects, n = 342 biweekly submaximal exercise tests, 72 exercise induced ST Segment Depressions **Statistical Analysis:** Logistic regression / GAM	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h Median: 10.6 IQR: 7.9 **Pollutant:** PM_1 Median: 7.0 IQR: 5.6 **Pollutant:** ACP (100 to 1000nm) (n/cm^3) **Median:** 1200 IQR: 760 **Copollutant:** NO_2, CO, $PM_{10-2.5}$, ultrafine	**PM Increment:** IQR **Effect Estimate(s):** ACP: OR = 3.29 (1.57, 6.92), lag 2 PM1: OR = 4.56 (1.73, 12.03), lag 2 $PM_{2.5}$: OR = 2.84 (1.42, 5.66), lag 2 **Notes:** The effect was strongest for ACP and $PM_{2.5}$, which in two pollutant models appeared independent. Increases in NO_2 and CO were also associated with increased risk of ST segment depression, but not with coarse particles.

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Park et al. (2008, 156845) **Period of Study:** Jan 1995–Jun 2005 **Location:** Greater Boston area, MA	**Outcome:** Total homocysteine (tHcy) **Mean Age:** 73.6 ± 6.9 yrs **Study Design:** Cross-sectional and longitudinal analyses performed N: 960 men **Statistical Analyses:** Generalized additive models (also hierarchical mixed-effects regression models to assess repeated measures of tHcy) **Covariates:** Model 1: season, age, long-term trend, apparent temperature Model 2: further adjustment for BMI, systolic blood pressure, smoking status, pack years of cigarettes, alcohol consumption Model 3: further adjustment for serum creatinine, plasma folate, vitamin B6, and vitamin B12 **Dose-response Investigated?** Modeled continuous covariates as penalized splines to determine if association with tHcy was linear **Statistical Package:** R software	**Pollutant:** SO42 **Averaging Time:** 24 h (moving averages up to 7 days prior to blood collection) **Mean (SD):** 3.2 (3.0) **Median:** 2.4 **Range (Min, Max):** 0.39, 29.0 **Monitoring Stations:** 1 site **Copollutant (correlation):** $PM_{2.5}$ (r = 0.85) BC (r = 0.50) OC (r = 0.41) SO_4^{2-}	**PM Increment:** IQR **Effect Estimate [Lower CI, Upper CI]:** Estimated % change in tHcy per IQR increase in pollutant. Lag model Concurrent day: IQR: NA Model 1: NA Model 2: NA Model 3: NA 1-day previous: IQR: 2.61 Model 1: 0.91 (-0.77, 2.62) Model 2: 0.99 (-0.94, 2.95) Model 3: 0.91 (-0.72, 2.57) 2-day moving avg: IQR: 2.10 Model 1: -0.25 (-2.07, 1.60) Model 2: -0.29 (-2.35, 1.82) Model 3: 0.05 (-1.74, 1.86) 3-day moving avg: IQR: 1.73 Model 1: -0.15 (-1.97, 1.69) Model 2: -0.17 (-2.23, 1.93) Model 3: -0.01 (-1.78, 1.80) 4-day moving avg: IQR: 1.64 Model 1: -0.69 (-2.74, 1.41) Model 2: -0.60 (-2.95, 1.81) Model 3: -0.58 (-2.63, 1.51) 5-day moving avg: IQR: 1.60 Model 1: -1.14 (-3.53, 1.30) Model 2: -0.90 (-3.64, 1.92) Model 3: -1.09 (-3.48, 1.36) 6-day moving avg; ' IQR: 1.40 Model 1: 0.00 (-2.39, 2.44) Model 2: 0.36 (-2.36, 3.16) Model 3: 0.41 (-2.01, 2.89) 7-day moving avg IQR: 1.30 Model 1: -0.16 (-2.51, 2.24) Model 2: 0.30 (-2.37, 3.04) Model 3: 0.07 (-2.25, 2.43) Stratified analyses: No significant difference in effect of SO42– among those with high and low levels of vitamins
Reference: Peters et al. (2005, 095747) Also Peters et al, 2005 (2005, 156859) **Period of Study:** February 1999–July 2001 **Location:** Augsburg, Germany	**Outcome:** Myocardial infarction **Study Design:** Case-crossover **N:** 691 myocardial infarction patients **Statistical Analysis:** Conditional logistic regression **Dose-response Investigated?** No	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 1 h: Median = 14.5 IQR: 9.1 24-h: Median = 14.9 IQR: 7.7 **Copollutant:** NO_2, SO_2, CO	**Effect Estimate:** 2-h lag: OR = 0.93 95% CI: 0,83, 1.04 24-h mean, 2-day lag: OR = 1.18 95% CI: 1.03, 1.34 **Notes:** Examined triggering for MI at various lags before MI onset (up to 6 h before MI, up to 5 days before MI). $PM_{2.5}$ levels 2 days before MI onset were associated with increased risk of MI, but not on the concurrent day, or lags 1, 3, 4, or 5. These findings are consistent with the prior Boston MI study for a 1- to 2-day lagged effect of $PM_{2.5}$.

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Pope et al. (2004, 055238) **Period of Study:** Winter 1999–2000 (in Wasatch Front, UT). Summer 2000 (in Hawthorne, UT). Winter 2000–2001 (in Bountiful, UT and Lindon, UT) **Location:** Utah: Wasatch Front, Hawthorne, Bountiful, and Lindon	**Outcome:** Change in autonomic function (measured by changes in HRV), C-reactive protein (CRP), blood cell counts, platelets, and blood viscosity associated with short-term changes in $PM_{2.5}$ **Age Groups:** Elderly (specific age range not given) **Study Design:** Panel study **N:** 88 elderly subjects **Statistical Analysis:** Linear regression **Season:** Winter, summer **Dose-response Investigated?** No	**Pollutant:** $PM_{2.5}$ (TEOM) **Averaging Time:** 24 h **Mean (SD):** 18.9 (13.4) **Copollutant:** None	**PM Increment:** 100 $\mu g/m^3$ **Effect Estimate:** Each 100 $\mu g/m^3$ increase associated with: -35 (SE = 8) msec decline in SDNN 0.81 (SE 0.17) mg/dL increase in CRP 0.31 (SE 9.34) k/μL increase in platelets 0.07 (SE 0.21) cP increase in blood viscosity **Notes:** The study observed small but statistically significant adverse associations between daily mean $PM_{2.5}$ and HRV and C-reactive protein (CRP). The authors point out, however, that most of the variability in the temporal deviation of these physiological endpoints was not explained by $PM_{2.5}$. These observations therefore suggest that $PM_{2.5}$ may be one of multiple factors that influence HRV and CRP.
Reference: Pope et al. (2006, 091246) **Period of Study:** 1994 - 2004 **Location:** Wasatch Front, Utah	**Outcome:** Acute ischemic heart disease **Study Design:** Case-crossover study (time-stratified control selection) **N: Statistical Analysis:** Conditional logistic regression	**Pollutant:** $PM_{2.5}$ (FRM) **Averaging Time:** 24 h **Mean (SD):** Site 1: 10.1 Site 2: 10.8 Site 3: 11.3 **Monitoring Stations:** 3 **Copollutant:** PM_{10} (FRM) measured at 4 monitoring sites	**PM Increment:** 10 $\mu g/m^3$ **Effect Estimate:** For same-day increase in $PM_{2.5}$: OR = 1.045 95% CI: 1.011, 1.080 **Notes:** Case-crossover study (time-stratified control selection) triggering of acute ischemic heart disease by ambient $PM_{2.5}$ concentrations on the same and previous 3 days. $PM_{2.5}$ measured at 3 sites and estimated for missing days. Effect estimates were larger for those with angiographically demonstrated coronary artery disease.
Reference: Pope et al. (2004, 055238) **Period of Study:** 1999-2001 **Location:** Wasatch Front, Utah	**Outcome:** Heart rate variability (HRV) C-reactive protein (CRP) blood cell counts, whole blood viscosity **Age Groups:** 54-89 yrs **Study Design:** Panel study **N:** 88 participants **Statistical Analyses:** Linear regression **Covariates:** Subject-specific fixed effects interactive spline smooths for temp, RH (partial control for H) **Season:** Temperature as covariate **Dose-response Investigated?** Yes, also assessed PM by including cubic smoothing splines with 3 df **Statistical Package:** SAS	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean (SD):** 23.7 (20.2) **Range (Min, Max):** 1.7, 74.0 **Monitoring Stations:** NR **Copollutant:** None	**PM Increment:** 100 $\mu g/m^3$ **Effect Estimate [Lower CI, Upper CI]: Regression coefficients (SE) for associations with concurrent day pollutant:** Mean H: -4.49 (1.73) SDNN: -34.94 (8.32) SDANN: -18.98 (8.67) r-MSSD: -42.25 (10.90) CRP: 0.81 (0.18) Whole blood viscosity: 0.07 (0.21) WBC: -0.07 (0.38) Granulocytes: 0.02 (0.37) Lymphocytes: -0.07 (0.14) Monocytes: 0.12 (0.04) Basophils: -0.01 (0.01) Eosinophils: -0.01 (0.02) RBC: 0.03 (0.06) Platelets: 0.31 (9.34)
Reference: Rich et al. (2005, 079620) **Period of Study:** July 1995–July 2002 **Location:** Eastern Massachusetts, USA	**Outcome:** Confirmed ventricular arrhythmias **Study Design:** Case-crossover (time-stratified control selection) **N:** 203 patients with implantable cardioverter defibrillators **Statistical Analysis:** Conditional logistic regression	**Pollutant:** $PM_{2.5}$ (TEOM) **Averaging Time:** 1-h avg 24-h avg **Median (IQR):** 1-h avg: Median = 9.2 $\mu g/m^3$ 24-h avg: Median = 9.8 $\mu g/m^3$ IQr = 7.8 **Copollutant:** O_3, BC, CO, NO_2, SO_2	**PM Increment:** 7.8 $\mu g/m^3$ **Effect Estimate:** For mean $PM_{2.5}$ in the 24 h before ventricular arrhythmia: OR = 1.19 95% CI: 1.02, 1.38 **Notes:** 794 ventricular arrhythmias among 84 subjects. **Lag h:** 0-2, 0-6, 0-23, 0-47

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Rich et al. (2006, 088427) **Period of Study:** July 1995–July 2002 **Location:** Eastern Massachusetts, USA	**Outcome:** Confirmed episodes of paroxysmal atrial fibrillation **Study Design:** Case-crossover (time-stratified control selection) **N:** 203 patients with implantable cardioverter defibrillators **Statistical Analysis:** Conditional logistic regression	**Pollutant:** $PM_{2.5}$ (TEOM) **Averaging Time:** 1 h avg 24-h avg **Median (IQR):** 1-h avg: Median = 9.2 $\mu g/m^3$ 24-h avg: Median = 9.8 $\mu g/m^3$ IQr = 7.8 **Copollutant:** O_3, BC, CO, NO_2, SO_2	**PM Increment:** 9.4 $\mu g/m^3$ **Effect Estimate:** 0-h lag: OR 1.41 (0.82, 2.42) **Notes:** 91 paroxysmal atrial fibrillation (PAF) episodes among 29 subjects. **Lag h:** 0, 0 - 23 Positive, but not significant increases in the relative odds of PAF associated with $PM_{2.5}$ concentrations in the same h and 24-h before PAF episode onset. Authors note reduced statistical power for $PM_{2.5}$ analyses due to missing data.
Reference: Rich et al. (2006, 088427) **Period of Study:** July 1995–July 2002 **Location:** Eastern Massachusetts, USA	**Outcome:** Confirmed episodes of paroxysmal atrial fibrillation **Study Design:** Case-crossover (time-stratified control selection) **N:** 203 patients with implantable cardioverter defibrillators **Statistical Analysis:** Conditional logistic regression	**Pollutant:** BC **Averaging Time:** 1-h avg 24-h avg **Median (IQR):** IQR: 0.91$\mu g/m^3$ **Copollutant:** O_3, $PM_{2.5}$, CO, NO_2, SO_2	**PM Increment:** 0.91$\mu g/m^3$ (IQR) **Effect Estimate:** 0- to 23-h lag period: OR 1.46 (95% CI: 0.67, 3.17) **Notes:** 91 paroxysmal atrial fibrillation (PAF) episodes among 29 subjects. **Lag h:** 0, 0-23 Positive, but not significant increases in the relative odds of PAF associated with BC concentrations in the same h and 24 h before PAF episode onset. Authors note reduced statistical power for BC analyses due to missing data.
Reference: Rich et al. (2006, 089814) **Period of Study:** May 2001–December 2002 **Location:** St. Louis, MO metropolitan area	**Outcome:** Confirmed ventricular arrhythmia **Study Design:** Case-crossover design (time-stratified control selection) **Dose-response Investigated?** No	**Pollutant:** $PM_{2.5}$ (CAMM) **Averaging Time:** 24 h **Median (IQR):** 16.2 $\mu g/m^3$ (IQr = 9.7) **Copollutant:** NO_2, SO_2, CO, O_3, EC, OC	**PM Increment:** 9.7 $\mu g/m^3$ (IQR) **Effect Estimate:** OR ($PM_{2.5}$) = 0.95 (95% CI: 0.72, 1.27) OR (SO_2) = OR = 1.24 (95% CI: 1.07, 1.44) **Notes:** 139 confirmed ventricular arrhythmia episodes among 56 subjects. Lags: 0-2h, 0-6h, 0-11h, 0-23h, 0-47h Authors did not find increased relative odds of VA associated with each IQR increase in 24-h mean $PM_{2.5}$, but did find non-significantly increased relative odds of VA associated with 24-h EC. Shorter and longer lag times' relative odds estimates provided no evidence of immediate ventricular arrhythmic effects of air pollution.
Reference: Rich et al. (2004, 055631) **Period of Study:** February–December 2000 **Location:** Vancouver, British Columbia, Canada	**Outcome:** ICD discharges (as a proxy for VT/VF) **Age Groups:** 15-85 years **Study Design:** Case-crossover design (ambidirectional control selection ± 7 days) **N:** 34 patients with implantable cardioverter defibrillators **Statistical Analysis:** Conditional logistic regression **Dose-response Investigated?** No	**Pollutant:** $PM_{2.5}$ (Partisol) **Averaging Time:** 1 h **Mean (SD), IQR:** Mean: : 8.2 $\mu g/m^3$ (SD = 10.7) IQr = 5.2 **Copollutant:** O_3, EC, OC, SO_4^{2-}, CO, NO_2, SO_2, PM_{10} PM_{10}: Mean: : 13.3 $\mu g/m^3$ (SD = 4.9) IQr = 7.4	**PM Increment: Effect Estimate:** Odds ratios were less than 1.0 at all lags (0, 1, 2, 3) for $PM_{2.5}$. No consistent association between any of the air pollutants and implantable cardioverter defibrillators discharges. **Notes:** Same study as Vedal et al. (2004, 055630), except Rich (2004) used data from a shorter time period so as to estimate relative odds of ICD discharge associated with acute increases in more pollutants than Vedal (2004, 055630).

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Rich et al. (2008, 156910) **Period of Study:** NR **Location:** New Jersey	**Outcome:** Pulmonary Artery and Right Ventricular Pressures **Age Groups:** 25-68 **Study Design:** panel **N:** 11 subjects **Statistical Analyses:** Repeated Measures **Covariates:** long-term trends, calendar month, weekday, apparent temperature **Dose-response Investigated?** No **Statistical Package:** SAS **Lags Considered:** 0-6d	**Pollutant:** $PM_{2.5}$ Averaging Time: 24h **Mean (SD):** NR **Monitoring Stations:** NR **Copollutant:** NR **Co-pollutant Correlation** n/a	**PM Increment:** 11.62μg/m^3 **Change (Lower CI, Upper CI), p-value:** ePAD: 0.19 (0.05, 0.33), 0.01 RV diastolic pressure: 0.23 (0.11, 0.34), $<$0.001 RV systolic pressure: 0.12 (-0.07, 0.31), 0.23 MPAP: 0.12 (-0.05, 0.28), 0.16/
Reference: Riediker et al. (2004, 091261) **Period of Study:** Fall 2001 **Location:** Wake County, North Carolina	**Outcome:** Heart rate variability (measured 10 h after shift): mean cycle length of normal R-R intervals (MCL), the standard deviation of normal R-R intervals (SDNN), and percentage of normal R-R interval differences greater than 50 msec (PNN50), low frequency (0.04-0.15Hz), high frequency (0.15-0.40Hz), the ratio of low to high frequency. **Blood analysis (measured 15 h after shift):** Uric acid, blood urea nitrogen, gamma glutamyl transpeptidase, white blood cell count, red blood cell count, hematocrit, hemoglobin, mean red blood cell volume (MCV), neutronphils (count and %), lymphocytes (count and %), C-reactive protein, plasminogen, plasminogen activator inhibitor type 1, von Willebrand factor (vWF), endovthzelin-1, protein C, and interleukin-6 **Age Groups:** 23-30 yrs **Study Design:** Panel **N:** 9 healthy male troopers, repeated measures (36 person-days) **Statistical Analyses:** Mixed effects regression models (principal factor analysis for classification of exposure) **Covariates:** Potential confounders: temperature, relative humidity, number of law-enforcement activities during the shift and the avg speed during the shift controlling had no effect on effect estimates for "crustal" and "speed-change" factors however, confounder inclusion in the "speed change" and blood urea nitrogen and vWF reduced the effect estimate and the CI included zero **Season:** Only 1 season included **Dose-response Investigated?** No **Statistical Package:** S-Plus 6.1	**Pollutant:** In-vehicle $PM_{2.5}$ components identified with factor analysis (crustal material, wear of steel automotive components, gasoline combustion, speed-changing traffic with engine emissions and brake wear **Averaging Time:** Exposure assessed during 3pm to 12am work shifts **Mean:** $PM_{2.5}$mass = 23.0 μg/m^3 **Monitoring Stations:** Per vehicle **Copollutant (correlation):** Correlation to $PM_{2.5}$Mass Benzene: r = 0.50 Aldehydes: r = 0.34 CO: r = 0.52 Aluminum: r = 0.58 Silicon: r = 0.66 Sulfur: r = 0.58 Calcium: r = 0.37 Titanium: r = 0.41 Chromium: r = 0.51 Iron: r = 0.71 Copper: r = 0.16 Selenium: r = 0.38 Tungsten: r = 0.37 PM_2.Lightscatter: r = 0.71	**PM Increment:** 1 SD change in source factor **Effect Estimate:** % change in the health outcome per 1 SD change in the "speed change" factor MCL: 7% HRV: 16% supraventricular ectopic beats: 39% % Neutrophils: 7% % lymphocytes: -10% red blood cell volume MCV: 1% vWF: 9% blood urea nitrogen: 7% protein C: -11% % change in the health outcome per 1 SD change in the "crustal" factor MCL: 3% serum uric acid concentrations: 5% **Note:** Results (including CIs) are reported in figures 2 & 3.

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Riojas-Rodriguez et al. (2006, 156913) **Period of Study:** December 2001–April 2002 **Location:** Mexico City metropolitan area	**Outcome:** Heart rate variability (5-minute periods) **Study Design:** Panel study **N:** 30 patients from the outpatient clinic of the National Institute of Cardiology of Mexico, where each subject had existing ischemic heart disease. **Statistical Analysis:** Mixed models	**Pollutant:** $PM_{2.5}$ (nephelometry) **Averaging Time:** 5 minutes **Mean (SD), Range:** 46.8 $\mu g/m^3$ (SD = 1.82) **Range:** 0–483 $\mu g/m^3$ **Copollutant:** CO	**PM Increment:** 10 $\mu g/m^3$ **Effect Estimate:** Each 20 $\mu g/m^3$ increase in 5 minute $PM_{2.5}$ was associated with a: -0.008 decrease in the ln(HF)(95% CI: -0.015, 0.0004 **Notes:** Population of subjects with known ischemic heart disease (25 men and 5 women who had at least 1 prior MI [not in last 6 months]) Each 10 $\mu g/m^3$ increase in 5 minute mean $PM_{2.5}$ was associated with non-significantly decreased HF, and with similar, but smaller changes in LF and VLF.
Reference: Romieu et al. (2005, 086297) **Period of Study:** 2000–2001 **Location:** Mexico City, Mexico	**Outcome:** Heart rate variability (HF, LF, VLF, PNN50, SDNN, r-MSSD) **Age Groups:** > 60 years of age **Study Design:** Double blind randomized controlled trial **N:** 50 elderly residents of a Mexico City nursing home	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Copollutant:** O_3, NO_2, SO_2, PM_{10}	**PM Increment:** 8 $\mu g/m^3$ **Effect Estimate:** In the group receiving the fish oil supplement, each 8 $\mu g/m^3$ change in 24 h mean total exposure $PM_{2.5}$ was associated with a: a) 54% reduction (95% CI: -72% to -24%) in HF (log transformed) in the pre-supplementation phase b) 7% reduction (95% CI: -20%, 7%) in the supplementation phase. Changes in other HRV parameters were also smaller in the supplementation phase. In the group receiving soy oil supplementation, the % reduction in HF was also smaller in the supplementation phase, but the differences were smaller and not statistically significant. **Notes:** Study of the effect of omega-3-fatty acid supplementation (2 g/day of fish oil versus 2 g/day of soy oil) to mitigate the effect of ambient $PM_{2.5}$ on HRV. Subjects had no cardiac arrhythmias, cardiac pacemakers, allergies to omega-3 fatty acids or fish, treatment with oral anticoagulants, or history of bleeding diathesis. $PM_{2.5}$ was measured and estimated indoors, outdoors, and with regards to total exposure (the same as Holguin et al. (2003)).

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Romieu et al. (2008, 156922) **Period of Study:** Sep 2001–Apr 2002 **Location:** Mexico City, Mexico	**Outcome:** Copper/zinc superoxide dismutase activity (Cu/Zn SOD) lipoperoxidation (LPO) reduced glutathione (GSH) **Age Groups:** 60-96 yrs **Study Design:** Intervention (randomly assigned fish oil or soy oil) **N:** 52 participants **Statistical Analyses:** Linear mixed models **Covariates:** Time **Dose-response Investigated?** Assessed possible nonlinearity using generalized additive mixed models with p-splines **Statistical Package:** STATA v8.2 and SAS v9.1	**Pollutant:** $PM_{2.5}$ (indoor) **Averaging Time:** 24 h (same day) **Mean (SD):** 38.7 (14.7) **Percentiles:** 25th: 30.62 50th: 35.11 75th: 41.10 **Range (Min, Max):** 14.8, 70.9 **Monitoring Stations:** Indoor measured inside nursing home **Copollutant:** O_3	**PM Increment:** 10 $\mu g/m^3$ **Effect Estimate [Lower CI, Upper CI]:** **Regression coefficient (SE** **p-value):** Cu/Zn SOD: -0.05 (0.02 0.001) LPO (square root transformed): 0.08 (0.09 0.381) GSH (log-transformed quadratic term for PM): -0.05 (0.01 0.002) Regression coefficient (SE p-value) by supplementation groups (same transformations as above): Cu/Zn SOD Soy Oil: -0.06 (0.02, $<$ 0.001) Fish Oil: * 0.04 (0.02, 0.009) LPO Soy Oil: -0.02 (0.14, 0.904) Fish Oil: * 0.16 (0.07, 0.024) GSH Soy Oil: -0.03 (0.04, 0.406) Fish Oil: -0.09 (0.04, 0.017) *Quadratic term for PM
Reference: Ruckerl et al. (2007, 156931) **Period of Study:** May 2003–Jul 2004 **Location:** Athens, Augsburg, Barcelona, Helsinki, Rome, and Stockholm	**Outcome:** Interleukin-6 (IL-6), fibrinogen, C-reactive protein (CRP) **Age Groups:** 35-80 yrs **Study Design:** Repeated measures / longitudinal **N:** 1003 MI survivors **Statistical Analyses:** Mixed-effect models **Covariates:** City-specific confounders (age, sex, BMI) long-term time trend and apparent temperature RH, time of day, day of week included if adjustment improved model fit **Season:** Long-term time trend **Dose-response Investigated?** Used p-splines to allow for nonparametric exposure-response functions **Statistical Package:** SAS v9.1	**Pollutant:** $PM_{2.5}$ **Averaging Time:** Hourly and 24-h (lag 0-4, mean of lags 0-4, mean of lags 0-1, mean of lags2-3, means of lags 0-3) **Mean (SD):** Presented by city only **Monitoring Stations:** Central monitoring sites in each city **Copollutant:** SO_2 O_3 NO NO_2	**PM Increment:** IQR **Effect Estimate [Lower CI, Upper CI]:** % change in mean blood markers per increase in IQR of air pollutant. IL-6 Lag (IQR): % change in GM (95%CI) Lag 0 (11.0): 0.46 (-0.89, 1.83) Lag 1 (11.0): -0.39 (-1.69, 0.93) Lag 2 (11.0): -0.23 (-1.53, 1.07) 5-d avg (8.6): 0.05 (-1.37, 1.50) Fibrinogen Lag (IQR): % change in AM (95%CI) Lag 0 (11.0): 0.05 (-0.48, 0.58) Lag 1 (11.0): 0.17 (-0.35, 0.69) Lag 2 (11.0): 0.20 (-0.32, 0.71) 5-d avg (8.6): 0.38 (-0.21, 0.96) CRP Lag (IQR): % change in GM (95%CI) Lag 0 (11.0): 0.11 (-1.95, 2.21) Lag 1 (11.0): -0.06 (-1.98, 1.90) Lag 2 (11.0): 0.11 (-1.80, 2.06) 5-d avg (8.6): -0.13 (-2.15, 1.92)

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Ruckerl et al. (2006, 088754) **Period of Study:** Oct 2000–Apr 2001 **Location:** Erfurt, Germany	**Outcome:** C-reactive protein (CRP) serum amyloid A (SAA) E-selectin von Willebrand Factor (vWF) intercellular adhesion molecule-1 (ICAM-1) fibrinogen Factor VII prothrombin fragment 1+2 D-dimer **Age Groups:** 50+ **Study Design:** Panel (12 repeated measures at 2-wk intervals) **N:** 57 male subjects with coronary disease **Statistical Analyses:** Fixed effects linear and logistic regression models **Covariates:** Models adjusted for different factors based on health endpoint CRP: RH, temperature, trend, ID ICAM-1: temperature, trend, ID vWF: air pressure, RH, temperature, trend, ID FVII: air pressure, RH, temperature, trend, ID, weekday **Season:** Time trend as covariate **Dose-response Investigated?** Sensitivity analyses examined nonlinear exposure-response functions **Statistical Package:** SAS v8.2 and S-Plus v6.0	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean (SD):** 20.0 (15.0) **Percentiles:** 2th5: 9.7 50th: 14.9 75th: 26.1 **Range (Min, Max):** 2.6, 83.7 **Monitoring Stations:** 1 site **Copollutant:** UFPs (ultrafine particles) AP (accumulation mode particles) $PM_{2.5}$ PM_{10} OC (organic carbon) EC (elemental carbon) NO_2 CO	**PM Increment:** IQR (16.4 5-d avg: 12.2) **Effect Estimate [Lower CI, Upper CI]:** Effects of air pollution on blood markers presented as OR (95%CI) for an increase in the blood marker above the 90th percentile per increase in IQR air pollutant. **CRP** Time before draw: 0 to 23 h: 1.1 (0.7, 1.8) 24 to 47 h: 1.5 (0.9, 2.5) 48 to 71 h: 1.2 (0.8, 1.9) 5-d mean: 1.4 (0.9, 2.3) **ICAM-1** Time before draw: 0 to 23 h: 0.7 (0.4, 0.9) 24 to 47 h: 1.3 (0.8, 1.8) 48 to 71 h: 1.8 (1.2, 2.7) 5-d mean: 1.1 (0.8, 1.5) Effects of air pollution on blood markers presented as % change from the mean/GM in the blood marker per increase in IQR air pollutant. **vWF** Time before draw: 0 to 23 h: 3.9 (-0.3, 8.1) 24 to 47 h: 3.1 (-1.6, 7.8) 48 to 71 h: 3.6 (-1.1, 8.3) 5-d mean: 5.6 (0.5, 10.8) **FVII** Time before draw: 0 to 23 h: -2.5 (-6.2, 1.4) 24 to 47 h: -2.8 (-6.1, 0.6) 48 to 71 h: -2.3 (-5.0, 0.6) 5-d mean: -3.5 (-6.4 to -0.4) **Note:** Summary of results presented in figures. SAA results indicate increase in association with PM (not as strong and consistent as with CRP) no association observed between E-selectin and PM an increase in prothrombin fragment 1+2 was consistently observed, particularly with lag 4 fibrinogen results revealed few significant associations, potentially due to chance D-dimer results revealed null associations in linear and logistic analyses

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Ruckerl et al. (2006, 088754) **Period of Study:** Oct 2000–Apr 2001 **Location:** Erfurt, Germany	**Outcome:** C-reactive protein (CRP) serum amyloid A (SAA) E-selectin von Willebrand Factor (vWF) intercellular adhesion molecule-1 (ICAM-1) fibrinogen Factor VII prothrombin fragment 1+2 D-dimer **Age Groups:** 50+ yrs **Study Design:** Panel (12 repeated measures at 2-wk intervals) **N:** 57 male subjects with coronary disease **Statistical Analyses:** Fixed effects linear and logistic regression models **Covariates:** Models adjusted for different factors based on health endpoint CRP: RH, temperature, trend, ID ICAM-1: temperature, trend, ID vWF: air pressure, RH, temperature, trend, ID FVII: air pressure, RH, temperature, trend, ID, weekday **Season:** Time trend as covariate **Dose-response Investigated?** Sensitivity analyses examined nonlinear exposure-response functions **Statistical Package:** SAS v8.2 and S-Plus v6.0	**Pollutant:** EC **Averaging Time:** 24 h **Mean (SD):** 2.6 (2.4) **Percentiles:** 25th: 1.0 50th: 1.8 75th: 3.2 **Range (Min, Max):** 0.2, 12.4 **Monitoring Stations:** 1 site **Copollutant:** UFPs (ultrafine particles) AP (accumulation mode particles) $PM_{2.5}$ PM_{10} OC (organic carbon) EC (elemental carbon) NO_2 CO	**PM Increment:** IQR (2.3 5-d avg: 1.8) **Effect Estimate [Lower CI, Upper CI]:** Effects of air pollution on blood markers presented as OR (95%CI) for an increase in the blood marker above the 90th percentile per increase in IQR air pollutant. **CRP** Time before draw: 0 to 23 h: 1.2 (0.7, 2.0) 24 to 47 h: 1.3 (0.7, 2.4) 48 to 71 h: 1.6 (0.9, 2.7) 5-d mean: 1.2 (0.7, 2.1) **ICAM-1** Time before draw: 0 to 23 h: 1.0 (0.7, 1.6) 24 to 47 h: 2.6 (1.7, 3.8) 48 to 71 h: 4.0 (2.5, 6.1) 5-d mean: 2.2 (1.4, 3.3) Effects of air pollution on blood markers presented as % change from the mean/GM in the blood marker per increase in IQR air pollutant. **vWF** Time before draw: 0 to 23 h: 5.0 (0.0, 10.1) 24 to 47 h: 7.6 (1.4, 13.7) 48 to 71 h: 1.1 (-5.2, 7.4) 5-d mean: 5.7 (-0.5, 12.0) **FVII** Time before draw: 0 to 23 h: -5.7 (-10.5 to -0.7) 24 to 47 h: -6.9 (-11.2 to -2.3) 48 to 71 h: -4.2 (-8.4, 0.2) 5-d mean: -6.0 (-10.5 to -1.2) **Note:** Summary of results presented in figures. SAA results indicate increase in association with PM (not as strong and consistent as with CRP) no association observed between E-selectin and PM an increase in prothrombin fragment 1+2 was consistently observed, particularly with lag 4 fibrinogen results revealed few significant associations, potentially due to chance D-dimer results revealed null associations in linear and logistic analyses

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Ruckerl et al. (2006, 088754) **Period of Study:** Oct 2000–Apr 2001 **Location:** Erfurt, Germany	**Outcome (ICD9 and ICD10):** C-reactive protein (CRP) serum amyloid A (SAA) E-selectin von Willebrand Factor (vWF) intercellular adhesion molecule-1 (ICAM-1) fibrinogen Factor VII prothrombin fragment 1+2 D-dimer **Age Groups:** 50+ yrs **Study Design:** Panel (12 repeated measures at 2-wk intervals) **N:** 57 male subjects with coronary disease **Statistical Analyses:** Fixed effects linear and logistic regression models **Covariates:** Models adjusted for different factors based on health endpoint CRP: RH, temperature, trend, ID ICAM-1: temperature, trend, ID vWF: air pressure, RH, temperature, trend, ID FVII: air pressure, RH, temperature, trend, ID, weekday **Season:** Time trend as covariate **Dose-response Investigated?** Sensitivity analyses examined nonlinear exposure-response functions **Statistical Package:** SAS v8.2 and S-Plus v6.0	**Pollutant:** OC **Averaging Time:** 24 h **Mean (SD):** 1.5 (0.6) **Percentiles:** 25th: 1.1 50th: 1.4 75th: 1.8 **Range (Min, Max):** 0.3, 3.4 **Monitoring Stations:** 1 site **Copollutant:** UFPs (ultrafine particles) AP (accumulation mode particles) $PM_{2.5}$ PM_{10} OC (organic carbon) EC (elemental carbon) NO_2 CO	**PM Increment:** IQR (0.7 5-d avg: 0.5) **Effect Estimate [Lower CI, Upper CI]:** Effects of air pollution on blood markers presented as OR (95%CI) for an increase in the blood marker above the 90th percentile per increase in IQR air pollutant. **CRP** Time before draw: 0 to 23 h: 1.2 (0.7, 1.9) 24 to 47 h: 1.3 (0.8, 2.1) 48 to 71 h: 1.4 (0.8, 2.4) 5-d mean: 1.2 (0.7, 1.8) **ICAM-1** Time before draw: 0 to 23 h: 0.9 (0.6, 1.3) 24 to 47 h: 2.0 (1.3, 3.2) 48 to 71 h: 3.0 (1.8, 4.8) 5-d mean: 1.3 (0.8, 2.0) Effects of air pollution on blood markers presented as % change from the mean/GM in the blood marker per increase in IQR air pollutant. **vWF** Time before draw: 0 to 23 h: 5.5 (0.2, 10.8) 24 to 47 h: 8.0 (2.1, 13.9) 48 to 71 h: 3.5 (-2.6, 9.6) 5-d mean: 7.4 (2.0, 12.8) **FVII** Time before draw: 0 to 23 h: -6.1 (-10.6 to -1.4) 24 to 47 h: -7.2 (-11.4 to -2.8) 48 to 71 h: -3.8 (-8.2, 0.9) 5-d mean: -5.6 (-9.8 to -1.1) **Note:** Summary of results presented in figures. SAA results indicate increase in association with PM (not as strong and consistent as with CRP) no association observed between E-selectin and PM an increase in prothrombin fragment 1+2 was consistently observed, particularly with lag 4 fibrinogen results revealed few significant associations, potentially due to chance D-dimer results revealed null associations in linear and logistic analyses

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Ruckerl et al. (2007, 091379) **Period of Study:** Oct 2000–Apr 2001 **Location:** Erfurt, Germany	**Outcome:** Soluble CD40 ligand (sCD40L), platelets, leukocytes, erythrocytes, hemoglobin **Age Groups:** 50+ yrs **Study Design:** Panel (12 repeated measures at 2-wk intervals) **N:** 57 male subjects with coronary disease **Statistical Analyses:** Fixed effects linear regression models **Covariates:** Long-term time trend, weekday of the visit, temperature, RH, barometric pressure **Season:** Time trend as covariate **Dose-response Investigated?** No **Statistical Package:** SAS v8.2 and S-Plus v6.0	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean (SD):** 20.0 (15.0) **Percentiles:** 25th: 9.7 50th: 14.9 75th: 26.1 **Range (Min, Max):** 2.6, 83.7 **Monitoring Stations:** 1 site **Copollutants:** UFPs (ultrafine particles) AP (accumulation mode particles) $PM_{2.5}$ PM_{10} NO	**PM Increment:** IQR (16.4 5-d avg: 12.2) **Effect Estimate [Lower CI, Upper CI]:** Effects of air pollution on blood markers presented as % change from the mean/GM in the blood marker per increase in IQR air pollutant. **sCD40L, % change GM (pg/mL)** lag0: 1.5 (-4.0, 7.3) Lag1: 0.2 (-5.4, 6.2) Lag2: -2.6 (-8.0, 3.1) Lag3: 0.5 (-3.9, 5.0) 5-d mean: 0.2 (-5.4, 6.2) **Platelets, % change mean (10^3/µl)** Lag0: -0.6 (-1.9, 0.7) Lag1: 0.1 (-1.3, 1.5) Lag2: 0.5 (-0.9, 1.9) Lag3: 0.2 (-1.1, 1.5) 5-d mean: -0.4 (-1.9, 1.2) **Leukocytes, % change in mean (10^3/µl)** Lag0: -1.6 (-3.2, 0.0) Lag1: -0.4 (-2.2, 1.4) Lag2: -0.2 (-2.1, 1.7) Lag3: -0.8 (-2.4, 0.7) 5-d mean: -1.6 (-3.5, 0.3) **Erythrocytes, % change mean (10^6/µl)** Lag0: -0.1 (-0.5, 0.3) Lag1: -0.3 (-0.7, 0.2) Lag2: -0.4 (-0.8, 0.0) Lag3: -0.2 (-0.5, 0.1) 5-d mean: -0.4 (-0.8, 0.0) **Hemoglobin, % change mean (g/dl)** Lag0: 0.0 (-0.6, 0.5) Lag1: -0.2 (-0.8, 0.3) Lag2: -0.5 (-1.1, 0.0) Lag3: -0.2 (-0.7, 0.2) 5-d mean: -0.5 (-1.0, 0.1)
Reference: Sarnat et al. (2006, 090489) **Period of Study:** summer and Autumn 2000 **Location:** Steubenville, OH	**Outcome:** Supraventricular ectopy (SVE) or ventricular ectopy (VE) **N:** 32 nonsmoking older adults **Statistical Analysis:** Logistic mixed effects regression **Season:** Summer, Autumn **Dose-response Investigated?** No	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 5 days **Median (IQR):** $PM_{2.5}$: Median: 19.0 $\mu g/m^3$ IQr = 10.0 Sulfate: Median: 6.1. IQR: 4.2 EC: Median: 0.9. IQR: 0.5 **Copollutants:** O_3, NO_2, SO_2	**PM Increment:** IQR **Effect Estimate:** $PM_{2.5}$: SVE: OR = 1.42 (95% CI: 0.99, 2.04) VE: OR = 1.02 (95% CI: 0.63-1.65) Sulfate: SVE: OR = 1.70 (95% CI: 1.12, 2.57) VE: OR = 1.08 (95% CI: 0.65, 1.80) EC: SVE: OR = 1.15 (95% CI: 0.73, 1.81) VE: OR = 1.00 (95% CI: 0.57, 1.75) **Notes:** Longitudinal study of 32 nonsmoking older adults who had ECG measurements made every week for 24 weeks. PM measured within 1 mile of subjects' residences, and central site pollutant measurements were also made.

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Schneider et al. (2008, 191985) **Period of Study:** Nov 2004 – Dec 2005 **Location:** Chapel Hill, NC	**Outcome:** Endothelial Function Parameters **Age Groups:** 48-80 yrs **Study Design:** panel **N:** 22 diabetics **Statistical Analyses:** Mixed Models **Covariates:** season, day of the week, temperature, relative humidity, barometric pressure **Dose-response Investigated?** No **Statistical Package:** SAS **Lags Considered:** 0-4d & 5d ma	**Pollutant:** $PM_{2.5}$ **Averaging Time:** daily **Mean (SD):** 13.6 (7.0) **Min:** 2.0 **Max:** 38.9 **Monitoring Stations:** 2 **Copollutant:** NR	**PM Increment:** 10$\mu g/m^3$ **Percent Change: (Lower CI, Upper CI), lag:** FMD: ^I -17.3 (-34.6, 0.0), lag 0 -4.4 (-24.6, 15.8), lag 1 -18.6 (-44.8, 7.6), lag 2 1.6 (-23.6, 26.9), lag 3 18.4 (-3.5, 40.3), lag 4 -19.4 (-62.6, 23.8), 5d ma NTGMD: 2.5 (-9.0, 13.9), lag 0 -13.6 (-24.5, -2.6), lag 1* -10.2 (-23.5, 3.0), lag 2 -8.0 (-22.4, 6.4), lag 3 3.6 (-7.9, 15.0), lag 4 -19.4 (-44.3, 5.5), 5d ma LAEI: 0.4 (-4.2, 5.0), lag 0 -0.3 (-6.0, 5.4), lag 1 2.5 (-4.3, 9.4), lag 2 -7.3 (-13.5, -1.1), lag 3* -2.3 (-8.0, 3.3), lag 4 -4.6 (-15.3, 6.1), 5d ma SAEI: -3.0 (-13.0, 7.0), lag 0 -17.0 (-27.5, -6.4), lag 1** -9.7 (-23.5, 4.2), lag 2 -15.1 (-29.3, -0.9)*, lag 3 -2.1 (-14.0, 9.7), lag 4 -25.4 (-45.4, -5.3), 5d ma* SVR: -1.6 (-3.7, 0.4), lag 0 1.6 (-0.9, 4.1), lag 1 3.5 (0.5, 6.5), lag 2 2.4 (-0.5, 5.3), lag 3 3.2 (0.7, 5.6), lag 4* 4.5 (-0.3, 9.2), 5d ma *$p < 0.05$, ** $p < 0.01$ **Notes:** Percent change (95%CI) per 10$\mu g/m^3$ $PM_{2.5}$ by GSTM1 genotype (figure 3)

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Schwartz et al. (2005, 074317) **Period of Study:** 12 weeks during the summer of 1999 **Location:** Boston, MA	**Outcome:** Heart rate variability (HRV), ((SDNN, r-MSSD, PNN50, LFHFR) **Age Groups:** 61–89 years **Study Design:** Panel study **N:** 28 elderly subjects **Statistical Analysis:** Mixed models. To examine heterogeneity of effects, hierarchical modeling was used. **Season:** Summer **Dose-response Investigated?** No	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 1 h 24 h **Median:** 24-hs: 10 μg/m^3 **Monitoring Stations:** 1 **Copollutant:** BC, O_3, CO, SO_2, NO_2	**PM Increment:** IQR (not given) **Effect Estimate:** 24 h: 2.6 ms decrease in SDNN (95% CI: 0.8 to -6.0) 10.1 ms decrease in r-MSSD (95% CI: -2.8 to -16.9). 1 h: 3.4 ms decrease in SDNN (95% CI: 0.6 to -7.3) 7.4 ms decrease in r-MSSD (95% CI: 1.6 to -15.5). **Notes:** Various log-transformed HRV parameters were measured for 30 minutes once a week. The random effects model indicated that the negative effect of BC on HRV was not restricted to a few subjects. Same study population as Gold et al. (2005). Boston Elders Study For each pollutant/averaging time, similarly sized changes were observed for PNN50 (%) and LFHFR.
Reference: Schwartz et al. (2005, 074317) **Period of Study:** 12 weeks during the summer of 1999 **Location:** Boston, MA	**Outcome:** Heart rate variability (HRV), ((SDNN, r-MSSD, PNN50, LFHFR) **Age Groups:** 61–89 years **Study Design:** Panel study **N:** 28 elderly subjects **Statistical Analysis:** Mixed models. To examine heterogeneity of effects, hierarchical modeling was used. **Season: S**ummer **Dose-response Investigated?** No	**Pollutant:** BC **Averaging Time:** 24 h **Median:** 1.0 μg/m^3 **Monitoring Stations:** 1 **Copollutant:** $PM_{2.5}$, O_3, CO, SO_2, NO_2	**PM Increment:** IQR **Effect Estimate:** 5.1 ms decrease in SDNN (-1.5 to -8.6) 10.1 ms decrease in r-MSSD (-2.4 to -17.2). **Notes:** Various log-transformed HRV parameters were measured for 30 minutes once a week. The random effects model indicated that the negative effect of BC on HRV was not restricted to a few subjects. Same study population as Gold et al. (2005). Boston Elders Study. Subjects with a prior MI experienced greater declines in BC associated HRV. For each pollutant/averaging time, similarly sized changes were observed for PNN50 (%) and LFHFR.
Reference: Schwartz et al. (2005, 074317) **Period of Study:** 2000 **Location:** Boston, Massachusetts	**Outcome:** HF (high frequency component of heart rate variability) **Study Design:** Cross-sectional **N:** 497 subjects **Statistical Analysis:** Linear regression, controlling for covariates	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 48 h **Mean (SD):** 11.4 μg/m^3 (8.0) **Copollutant:** None	**PM Increment:** 10 μg/m^3 **Effect Estimate:** 34% decrease in HF (95% CI: -9% to -52%) in subjects without the GSTM1 allele. In subjects with the allele, no effect was noted. Similar findings for obese subjects and those with high neutrophil counts. **Notes:** Study population: Normative Aging Study. Effects of $PM_{2.5}$ appear to be mediated by ROS.

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Sorensen et al. (2005, 089428) **Period of Study:** Nov 1999–Aug 2000 **Location:** Copenhagen, Denmark	**Outcome:** 7-Hydro-8-Oxo-2′-Deoxyguanosine (8-oxodG) (measured in lymphocytes and urine) **Age Groups:** 20-33 yrs **Study Design:** Panel (repeated measures) **N:** 49 students living and studying in central Copenhagen 50 students examined each season (66 subjects total 32 participated in each season total of 98 measurements) **Statistical Analyses:** Mixed models repeated measures **Covariates:** $PM_{2.5}$, season, subject (random factor) **Dose-response Investigated?** No **Statistical Package:** SAS v8e	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 48 h **Mean (SD):** Autumn: 20.7 Summer: 12.6 **Percentiles:** IQR Autumn: 13.1-27.7 IQR summer: 9.4-24.3 **Range (Min, Max):** NR **Monitoring Stations:** NA (personal assessment) **Copollutant (correlation):** Spearman correlations with $PM_{2.5}$ mass: chromium (r = 0.22) copper (r = 0.33) iron (r = 0.29) vanadium ($p > 0.5$) nickel ($p > 0.5$) platinum ($p > 0.5$)	**PM Increment:** see below **Effect Estimate [Lower CI, Upper CI]:** Association between 8-oxodG in lymphocytes and personal exposure to transition metals in $PM_{2.5}$. % increase in 8-oxodG per increase in metal concentration indicated Vanadium: 1.9% per 1 µg/L (0.6, 3.3) Chromium: 2.2% per 1 µg/L (0.8, 3.5) Platinum: 6.1% per 1 ng/L (-0.6, 13.2) Nickel: 0.8% per 10 µg/L (-2.1, 3.7) Copper: -0.8% per 10 µg/L (-2.7, 1.0) Iron: 0.6% per 10 µg/L (-1.4, 2.6) **Note:** $PM_{2.5}$ mass was independently associated with 8-oxodG in 5 of 6 transition metal models ($p < 0.02$ in models with vanadium, chromium, nickel, copper, and iron p = 0.07 in platinum model). No transition metals were associated with 8-oxodG measured in urine
Reference: Sorensen et al. (2003, 042700) **Period of Study:** Nov 1999–Aug 2000 **Location:** Copenhagen, Denmark	**Outcome:** RBC count, hemoglobin, platelet count, fibrinogen, PLAAS (2-aminoadipic semialdehyde in plasma proteins), HBGGS (α-glutamyl semialdehyde in hemoglobin), HBAAS (2-aminoadipic semialdehyde in hemoglobin), MDA (malondialdehyde) **Age Groups:** 20-33 yrs **Study Design:** Panel (repeated measures) **N:** 50 students living and studying in central Copenhagen 50 students examined each season (68 subjects total 31 participated in each season total of 195 measurements) **Statistical Analyses:** Mixed model repeated-measures analysis **Covariates: S**eason, avg outdoor temperature, and sex **Season:** Repeated measures 4 times (once per season) **Dose-response Investigated?** No **Statistical Package:** SAS v8e	**Pollutant:** $PM_{2.5}$ (personal) **Averaging Time:** 48 h **Median:** 16.1 µg/m^3 **Percentiles:** Q25-Q75: 10.0-24.5 **Copollutant:** Urban background $PM_{2.5}$ Personal $PM_{2.5}$	**PM Increment:** 1 µg/m^3 **Effect Estimate [Lower CI, Upper CI]:** **Relationship between exposure and biomarkers** Estimate (p-value): Platelet count (x 10^8/g protein): 0.0008 (0.37) Fibrinogen (nmol/g protein): 0.0006 (0.69) PLAAS (pmol/mg protein): 0.0016 (0.061) HBGGS (pmol/mg protein): 0.0001 (0.94) HBAAS (pmol/mg protein): 0.0006 (0.64) **Increase (95%CI) in biomarkers per 10 µg/m^3 increase in $PM_{2.5}$** RBC Men: 0% (-1.6, 1.6) Women: 2.3% (0.5, 4.1) Hemoglobin Men: 0.0% (-1.7, 1.5) Women: 2.6% (0.8, 4.5)

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Sorensen et al. (2003, 042700) **Period of Study:** Nov 1999–Aug 2000 **Location:** Copenhagen, Denmark	**Outcome:** RBC count, hemoglobin, platelet count, fibrinogen, PLAAS (2-aminoadipic semialdehyde in plasma proteins), HBGGS (¤-glutamyl semialdehyde in hemoglobin), HBAAS (2-aminoadipic semialdehyde in hemoglobin), MDA (malondialdehyde) **Age Groups:** 20-33 yrs **Study Design:** Panel (repeated measures) **N:** 50 students living and studying in central Copenhagen 50 students examined each season (68 subjects total 31 participated in each season total of 195 measurements) **Statistical Analyses:** Mixed model repeated-measures analysis **Covariates: S**eason, avg outdoor temperature, and sex **Season:** Repeated measures 4 times (once per season) **Dose-response Investigated?** No **Statistical Package:** SAS v8e	**Pollutant:** Personal exposure to black carbon (10^{-6}/m) **Averaging Time:** 48 h **Median:** 8.1 **Percentiles:** Q25-Q75: 5.0-13.2 **Copollutant:** Urban background $PM_{2.5}$ Personal $PM_{2.5}$	**PM Increment:** 10^{-6}/m **Effect Estimate [Lower CI, Upper CI]:** **Relationship between exposure and biomarkers** Estimate (p-value): RBC count (x 10^9/g protein): 0.0003 (0.75) Hemoglobin (µmol/g protein): 0.0004 (0.65) Platelet count (x 10^6/g protein): 0.0009 (0.51) Fibrinogen (nmol/g protein): -0.0027 (0.29) PLAAS (pmol/mg protein): 0.0041 (0.0009) HBGGS (pmol/mg protein): 0.0024 (0.25) HBAAS (pmol/mg protein): 0.0022 (0.20) MDA (pmol/mg protein): 0.0018 (0.30)
Reference: Sorensen et al. (2003, 042700) **Period of Study:** Nov 1999–Aug 2000 **Location:** Copenhagen, Denmark	**Outcome:** RBC count, hemoglobin, platelet count, fibrinogen, PLAAS (2-aminoadipic semialdehyde in plasma proteins), HBGGS (¤-glutamyl semialdehyde in hemoglobin), HBAAS (2-aminoadipic semialdehyde in hemoglobin), MDA (malondialdehyde) **Age Groups:** 20-33 yrs **Study Design:** Panel (repeated measures) **N:** 50 students living and studying in central Copenhagen 50 students examined each season (68 subjects total 31 participated in each season total of 195 measurements) **Statistical Analyses:** Mixed model repeated-measures analysis **Covariates: S**eason, avg outdoor temperature, and sex **Season:** Repeated measures 4 times (once per season) **Dose-response Investigated?** No **Statistical Package:** SAS v8e	**Pollutant:** $PM_{2.5}$ (urban background concentration) **Averaging Time:** 48 h **Median:** 9.2 µg/m^3 **Percentiles:** Q25-Q75: 5.3-14.8 **Copollutant:** Urban background $PM_{2.5}$ Personal carbon black	**PM Increment:** 1 µg/m^3 **Effect Estimate [Lower CI, Upper CI]:** **Relationship between exposure and biomarkers** Estimate (p-value): RBC count (x 10^9/g protein): 0.0008 (0.36) Hemoglobin (µmol/g protein): 0.0005 (0.53) Platelet count (x 10^6/g protein): -0.0008 (0.49) Fibrinogen (nmol/g protein): 0.0004 (0.84) PLAAS (pmol/mg protein): 0.0004 (0.76) HBGGS (pmol/mg protein): -0.0020 (0.39) HBAAS (pmol/mg protein): -0.0021 (0.29) MDA (pmol/mg protein): 0.0012 (0.52)

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Sullivan et al. (2007, 100083) **Period of Study:** February 2000–March 2002 **Location:** Seattle, Washington, USA	**Outcome:** Blood CRP, fibrinogen, D-dimer **Age Groups:** > 55 years of age **Study Design:** Panel study **N:** 47 elderly subjects	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Median (IQR):** 7.7 $\mu g/m^3$ (6.4) **Monitoring Stations:** 1 **Copollutant:** Indoor $PM_{2.5}$	**PM Increment:** 10 $\mu g/m^3$ **Effect Estimate:** Among those with CVD, $PM_{2.5}$ 1 day earlier: CRP: 1.25 (95% CI: 0.97, 1.58) Fibrinogen: 1.01 (95% CI: 0.97, 1.05) D-dimer: 1.04 (95% CI: 0.93, 1.15) With COPD: CRP: 0.69 (95% CI: 0.34, 1.42) Fibrinogen: 1.05 (95% CI: 0.97, 1.13) D-dimer: 1.10 (95% CI: 0.95, 1.28) Healthy: CRP: 1.01 (95% CI: 0.85, 1.19) Fibrinogen: 0.88 (95% CI: 0.81, 0.95) D-dimer: 1.10 (95% CI: 0.75, 1.58) **Notes:** Out of 47 subjects, n = 23 with CVD and n = 24 (n = 16 COPD and 8 healthy) without CVD. Blood markers were measured on 2-3 morning over a 5-10 day period, and outdoor $PM_{2.5}$ was measured at a central monitoring site. These findings are not consistent with and effect of fine PM on markers of inflammation and thrombosis in the elderly.
Reference: Sullivan et al. (2005, 109418) **Period of Study:** February 2000–March 2002 **Location:** Seattle, Washington, USA	**Outcome:** Heart rate variability (H, LF, HF, r-MSSD, SDNN) **Study Design:** Panel study **N:** 34 elderly subjects with (n = 21) and without (n = 13) CVD. **Statistical Analysis:** Linear mixed effects regression	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 1 h **Median (IQR):** 10.7 (7.6) **Copollutant:** CO, NO_2	**PM Increment:** 10 $\mu g/m^3$ **Effect Estimate:** 1 h: With CVD: HF: (3% increase, 95% CI: -19, 32) Without CVD: HF(5% decrease, 95% CI: -34, 36) Similarly, no association was found for 4-h or 24-h mean $PM_{2.5}$ concentrations. **Notes:** 285 daily 20 minute HRV measures were made in the homes of study subjects over a 10-day period.
Reference: Sullivan et al. (2005, 109418) **Period of Study:** February 2000–March 2002 **Location:** Seattle area, WA	**Outcome (ICD9 and ICD10):** High-sensitivity C-reactive protein (hs-CRP) fibrinogen D-dimer endothelin-1 (ET-1) interleukin-6 (IL-6 interleukin-6 receptor (IL-6r) tumor necrosis factor-□ (TNF-8- □) tumor necrosis factor-receptors (p55, p75) monocyte chemoattractant protein-1 (MCP-1) **Age Groups:** ≥ 55 yrs **Study Design:** Panel (repeated measures) **N:** 47 participants with (23) and without (10 COPD and 8 healthy) CVD **Statistical Analyses:** Mixed models **Covariates:** Age, gender, medication use, meteorological variables (temperature and RH) **Dose-response Investigated?** No	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h (0-day and 1-day lags) **Mean (SD):** NR **Percentiles:** For all subject-days: 25th: 5.2 50th: 7.7 75th: 11.5 90th: 19.9 **Range (Min, Max):** 1.3, 33.9 **Monitoring Stations:** NA, measured at participant's residence **Copollutant:** None	**PM Increment:** 10 $\mu g/m^3$ **Effect Estimate [Lower CI, Upper CI]:** Multiplicative change in mean outcome associated with 10 $\mu g/m^3$ increase in PM **Among those with different disease status.** **CRP Fold-rise (95%CI)** CV 0-d lag: 1.21 (0.86, 1.70) CV 1-d lag: 1.25 (0.97, 1.58); COPD 0-d lag: 0.93 (0.48, 1.80) COPD 1-d lag: 0.69 (0.33, 1.46) Healthy 0-d lag: 0.98 (0.88, 1.08) Healthy 1-d lag: 1.01 (0.84 1.21)

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
	Statistical Package: SAS v8.02		**Fibrinogen Fold-rise (95%CI)** CV 0-d lag: 1.02 (0.98, 1.06) CV 1-d lag: 1.0 (0.97, 1.03); COPD 0-d lag: 1.0 (0.91, 1.09) COPD 1-d lag: 1.08 (0.99, 1.17) Healthy 0-d lag: 0.94 (0.87, 1.01) Healthy 1-d lag: 0.99 (0.88, 1.17) **D-dimer Fold-rise (95%CI)** CV 0-d lag: 1.02 (0.88, 1.17) CV 1-d lag: 1.03 (0.93, 1.15); COPD 0-d lag: 1.04 (0.93, 1.16) COPD 1-d lag: 1.09 (0.94, 1.27) Healthy 0-d lag: 0.95 (0.79, 1.14) Healthy 1-d lag: 0.97 (0.71, 1.31) **Among those with cardiovascular disease** **MCP-1 Fold-rise (95%CI)** 0-d lag: 1.3 (1.1, 1.7) 1-d lag: 1.0 (0.9, 1.3) **ET-1 Fold-rise (95%CI)** 0-d lag: 1.1 (0.8, 1.2) 1-d lag: 1.1 (0.9, 1.2) **Note:** TNF-α and IL-6 measures were below the limit of detection of assays
Reference: Timonen et al. (2006, 088747) **Period of Study:** 1998–1999 **Location:** Amsterdam, Netherlands Erfurt, Germany Helsinki, Finland	**Outcome:** Heart variability (HRV) measurements: [LF, HF, LFHFR, NN interval, SDNN, r-MSSD] **Study Design:** Panel study **N:** 131 elderly subjects with stable coronary heart disease **Statistical Analysis:** Linear mixed models	**Pollutant:** $PM_{2.5}$ **Means:** Amsterdam: 20.0 Erfurt: 23.3 Helsinki: 12.7 **Copollutant:** NO_2, CO	**PM Increment:** 10 $\mu g/m^3$ **Effect Estimate:** SDNN -0.33ms (95% CI: -1.05, 0.38) HF: -0.3% (95% CI: -10.6, 5.4) LFHFR: -1.4 (95% CI: -5.9, 8.7) **Notes:** Followed for 6 months with biweekly clinic visits 2-day lag. ULTRA Study

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Vallejo et al. (2006, 157081) **Period of Study:** April–August 2002 **Location:** Mexico City metropolitan area	**Outcome:** Heart rate variability measures (SDNN, pNN50) **Age Groups:** Mean age 27 yrs **Study Design:** Panel study **N:** 40 young healthy participants (non-smokers, no meds or history of CVD, respiratory, neurological, or endocrine disease) **Statistical Analysis:** Linear mixed effects models	**Pollutant:** $PM_{2.5}$ (pDR nephelometric method-DataRAM) **Copollutant:** None	**PM Increment:** 30 $\mu g/m^3$ **Effect Estimate: pNN50:** 0 h lag: -0.01% (95% CI: -0.03, 0.01) 1 h: -0.01% (95% CI: -0.04, 0.02) 2 h: -0.05% (95% CI: -0.09, 0.00) 3 h: -0.07% (95% CI: -0.13 to -0.02) 4 h: -0.08% (95% CI: -0.14 to -0.01) 5 h: -0.06% (95% CI: -0.13, 0.02) 6 h: -0.05% (95% CI: -0.13, 0.04) **SDNN:** 0 h: 0.00% (95% CI: 0.00, 0.01) 1 h: 0.00% (95% CI: -0.01, 0.01) 2 h: 0.00% (95% CI: -0.02, 0.01) 3 h: -0.01% (95% CI: -0.02, 0.00) 4 h: -0.01% (95% CI: -0.02, 0.01) 5 h: -0.01% (95% CI: -0.02, 0.01) 6 h: 0.00% (95% CI: -0.02, 0.02) **Notes:** Subjects underwent 13 h of ECG monitoring and personal $PM_{2.5}$ measurement. HRV measures were regressed against different lags of $PM_{2.5}$ concentration.
Reference: Van Hee et al. (2009, 192110) **Period of Study:** Jul 2000-Aug 2002 **Location:** Baltimore, Maryland Chicago, Illinois Winston-Salem, North Carolina St. Paul Minnesota New York, New York Los Angeles, California	**Outcome:** Left Ventricular Mass Index and Ejection Fraction **Age Groups:** 45-84 yrs **Study Design:** cross-sectional **N:** 3,827 participants **Statistical Analyses:** Linear Regression Models **Covariates:** age, race, income, sex, education, medication use, LDL, HDL, physical activity, alcohol consumption, smoking, diabetes, systolic BP, diastolic BP **Dose-response Investigated?** No **Statistical Package:** Stata **Lags Considered:** NR	**Pollutant:** $PM_{2.5}$ Averaging Time: NR **Mean (SD):** figure only **Monitoring Stations:** n/a Interpolation used **Copollutant:** NR **Co-pollutant Correlation** n/a	**PM Increment:** 10 $\mu g/m^3$ **Difference (Lower CI, Upper CI), p-value:** Left Ventricular Mass Index Unadjusted: -6.0 (-7.8, -4.2), <0.0001 All covariates except center, BP: -6.1 (-7.8, -4.4), <0.0001 All covariates except BP: 3.7 (-6.0, 13.4), 0.46 Full model: 4.6 (-4.7, 13.9), 0.33 Full model plus center/race interaction: 3.8 (-6.1, 13.7), 0.45 Left Ventricular Ejection Fraction Unadjusted: 3.0 (2.2, 3.8), <0.0001 All covariates except center, BP: 1.4 (0.5, 2.2), 0.001 All covariates except BP: -1.1(-5.8, 3.7), 0.66 Full model: -1.3 (-6.0, 3.5), 0.60 Full model plus center/race interaction: -3.0 (-8.0, 2.0), 0.24

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Wellenius et al. (2007, 092830) **Period of Study:** February 2002–March 2003 **Location:** Boston, Massachusetts, USA	**Outcome:** Circulating levels of B-type natriuretic peptide (BNP measured in whole blood at 0, 6, 12 weeks) **Study Design:** Panel study **N:** 28 subjects (each with chronic stable HF and impaired systolic function) **Statistical Analysis:** Linear mixed effects models	**Pollutant:** $PM_{2.5}$ **Copollutant:** NO_2, SO_2, O_3, CO, BC	**PM Increment:** 10 $\mu g/m^3$ **Effect Estimate:** Same day $PM_{2.5}$: 0.8% increase in BNP (95% CI: -16.4, 21.5) **Notes:** The study found no association between any pollutant and measures of BNP at any lag. Further, the within subject coefficient of variation was large suggesting the magnitude of effected air pollutant health effects are small in relation to within subject variability in BNP.
Reference: Wellenius et al. (2007, 092830) **Period of Study:** February 2002–March 2003 **Location:** Boston, Massachusetts	**Outcome (ICD9 and ICD10):** B-type natriuretic peptide (BNP) (natural-log transformed) **Age Groups:** 33-88 yrs **Study Design:** Panel (blood collected at 0, 6, and 12 weeks) **N:** 28 patients with chronic stable heart failure and impaired systolic function **Statistical Analyses:** Linear mixed-effects models **Covariates:** Temperature, dew point, mean dew point over the past 3 days, calendar month of blood draw, measurement occasion, treatment assignment, measurement occasion by treatment assignment interaction **Season:** Adjusted for calendar month **Dose-response Investigated?** No **Statistical Package:** SAS v9.1	**Pollutant:** $PM_{2.5}$ **Averaging Time:** Daily (assessed lags of 0-3 days) **Mean (SD):** 10.9 (8.4) **Percentiles:** 50th: 8.0 $\mu g/m^3$ **Range (Min, Max):** 0.7-50.9 $\mu g/m^3$ **Monitoring Stations:** 1 monitor **Copollutant (correlation):** CO (r = 0.35) NO_2 (r = 0.31) SO_2 (r = 0.18) O_3 (r = 0.35) BC(r = 0.68)	**PM Increment:** IQr = 8.1 $\mu g/m^3$ **Effect Estimate [Lower CI, Upper CI]:** % change in BNP per IQR increase in $PM_{2.5}$ Lag0: 1.5 (-18.7, 19.2) Lag1: 2.1 (-20.0, 30.3) Lag2: 1.3 (12.3, 17.1) Lag3: 5.6 (-16.8, 34.0) **Note:** No significant associations observed between any pollutant and BNP levels at any lags (presented in Fig 2)
Reference: Wheeler et al. (2006, 088453) **Period of Study:** Fall 1999 and spring 2000 **Location:** Atlanta, GA	**Outcome:** Heart rate variability **Age Groups:** 49–76 years **N:** 18 subjects with COPD and 12 subjects with a recent MI **Statistical Analysis:** Linear-mixed effect model **Season:** Fall and spring	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 1 h 4 h 24 h **Mean:** 24-hs: 17.8 $\mu g/m^3$ **Copollutant:** O_3, CO, SO_2, NO_2	**PM Increment:** 11.65 $\mu g/m^3$ (IQR) in 4 h $PM_{2.5}$ **Effect Estimate:** Among COPD patients: 8.3% increase in SDNN (95% CI: 1.7, 15.3) Among MI patients: 2.9% decrease in SDNN (95% CI: -7.8, 2.3) Results for 1h and 24 h averaging times were similar. **Notes:** Data was collected on 7 days in the Fall of 1999 or spring of 2000. Effects were modified by medication use, baseline pulmonary function, and health status.

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Yeatts et al. (2007, 091266)	**Outcome:** Heart Rate Variability	**Pollutant:** $PM_{2.5}$	**PM Increment:** 1 $\mu g/m^3$
Period of Study: 12 wk period b/t Sept 2003 – July 2004	**Age Groups:** 21-50 yrs	Averaging Time: 24h	**Beta, SE (Lower CI, Upper CI), p-value:**
Location: Chapel Hill, NC	**Study Design:** panel	**Mean (SD):** 12.5 (6.0)	HRV
	N: 12 asthmatics	**Min:** 0.6	Max Heart Rate: 0.40, 0.43 (-0.45, 1.24), 0.36
	Statistical Analyses: Linear Mixed Model	**Max:** 37.1	ASDNN5: -0.07, 0.15 (-0.37, 0.22), 0.63
	Covariates: temperature, humidity, pressure	**Monitoring Stations:** 1	SDANN5: 1.66, 0.65 (0.39, 2.93), 0.02
	Dose-response Investigated? No	**Copollutant:** $PM_{10-2.5}$, PM_{10}	SDNN24HR(mesc): 1.16, 0.58 (0.02, 2.29), 0.06
	Statistical Package: SAS	**Co-pollutant Correlation**	rMSSD: 0.53, 0.20 (0.14, 0.91), 0.01
	Lags Considered: 1 day	$PM_{10-2.5}$ = 0.46*	pNN50_24hour: -0.06, 0.11 (-0.27, 0.15), 0.58
		PM_{10} = NR	pNN50_7min: 0.47, 0.42 (-0.35, 1.29), 0.27
		*$p < 0.01$	Low-frequency power: -0.23, 0.14 (-0.51, 0.05), 0.11
			Percent low frequency: -0.78, 0.41 (-1.59, 0.03), 0.07
			High-frequency power: 0.14, 0.07 (-0.01, 0.28), 0.07
			Percent high frequency: 0.64, 0.36 (-0.07, 1.34), 0.09
			Blood Lipids
			Triglycerides: -0.63, 0.84 (-2.29, 1.02), 0.46
			VLDL: -0.17, 0.22 (-0.61, 0.26), 0.44
			Total cholesterol: -0.06, 0.22 (-0.49, 0.36), 0.77
			Hematologic Factor
			Circulating eosinophils: -0.02, 0.00 (-0.02, -0.02), 0.27
			Platelets: -0.01, 0.45 (-0.88, 0.86), 0.98
			Circulating Proteins
			Plasminogen: 0.00, 0.00 (-0.01, 0.00), 0.82
			Fibrenogen: 0.00, 0.01 (-0.01, 0.02), 0.59
			Von Willibrand factor: -0.31, 0.29 (-0.87, 0.25), 0.28
			Factor VII: -0.65, 0.33 (-1.29, -0.01), 0.05

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Yue et al. (2007, 097968) **Period of Study:** October 2000–April 2001 **Location:** Erfurt, Germany	**Outcome:** QT interval and T-wave amplitude for ECG recordings, and vWF, CRP from blood samples **Study Design:** Panel study **N:** 56 patients (male CAD patients with 12 clinical visits) **Statistical Analysis:** Linear and logistic regression models **Dose-response Investigated?** No	**Pollutant:** $PM_{2.5}$, Particle Number Concentration (PNC) (n/cm^3) **Averaging Time: Mean:** Mass concentrations of PNC (0.1-2.84 n/cm^3) **Monitoring Stations:** 1 **Copollutant:** None	**PM Increment:** . IQR **Effect Estimate:** Each IQR increase in 0-23 h mean traffic particle concentration was associated with: QT interval: 0.6% (95% CI: -0.3, 1.4) T wave amplitude: -1.6% (95% CI: -3.3, 0.1) vWF: 3.2% (95% CI: -0.5, 7.0) CRP: (OR = 1.5 95% CI 1.0–2.3) Each IQR increase in 0-23 h mean combustion-generated particle concentration was associated with: QT interval: 0.1%(-0.3, 0.6) T wave amplitude: -0.2% (-1.2, 0.7) vWF: 2.8% (0.8, 4.8) CRP (OR = 1.0 0.8, 1.2) **Notes:** Five sources of particles were identified (airborne soil, local traffic-related ultrafine particles, combustion-generated aerosols, diesel traffic-related particles, and secondary aerosols).

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Yue et al. (2007, 097968) **Period of Study:** Oct 12, 2000–Apr 27, 2001 **Location:** Erfurt, Germany	**Outcome:** QT interval, T wave amplitude, von Willebrand factor (vWF), C-reactive protein (CRP above 90th percentile compared to below) **Age Groups:** > 50 yrs **Study Design:** Panel (12 visits 625 observations for repolarization parameters and 578 observations for inflammatory markers) **N:** 57 male coronary artery disease patients **Statistical Analyses:** Linear and logistic fixed-effects regression models (generalized additive models) **Covariates:** Trend, weekday, and meteorological variables (temperature, relative humidity, barometric pressure) **Dose-response Investigated?** No **Statistical Package:** SAS v9.1 and S-Plus v6.0	**Pollutant:** Five particle source factors (airborne soil, local traffic-related ultrafine particles, combustion-generated aerosols, diesel traffic-related particles, and secondary aerosols) see below for size fractions (factor scores) **Averaging Time:** Used daily factor scores in analyses **Mean (SD):** Factor 1: particles from airborne soil (1.0-2.8 μm): 2390 (1696) Factor 2: ultrafine particles from local traffic (0.01-0.1 μm): 9931 (5858) Factor 3: secondary aerosols from local fuel combustion (0.1-0.5 μm): 3770 (6129) Factor 4: particles from traffic (0.01-0.5 μm): 6865 (5689) Factor 5: secondary aerosols from multiple sources (0.2-1.0 μm): 4732 (3890) **Median:** Factor 1: 2053 Factor 2: 8531 Factor 3: 1348 Factor 4: 5045 Factor 5: 3752 **IQR (5-day avg):** Factor 1: 1110 Factor 2: 5749 Factor 3: 4124 Factor 4: 5000 Factor 5: 3393 **Range (Min, Max):** Factor 1: 284, 12960 Factor 2: 866, 26632 Factor 3: 139, 39097 Factor 4: 283, 27605 Factor 5: 67, 20129 **Monitoring Stations:** 1 monitor **Copollutant:** NA	**PM Increment:** IQR **Effect Estimate [Lower CI, Upper CI]: QT interval, % change (95%CI)** **Factor 1:** 0-5h: -0.1 (-0.6, 0.6) 6-11h: -0.5 (-1.1, 0.2) 12-17h: 0.1 (-0.4, 0.4) 18-23h: -0.2 (-0.7, 0.2) 0-23h: -0.2 (-0.9, 0.4) 1d: -0.1 (-0.7, 0.6) 2d: -0.3 (-0.9, 0.4) 3d: -0.7 (-1.4, 0.1) 4d: -0.2 (-0.9, 0.5) 0-4d avg: -0.7 (-1.8, 0.3) **Factor 2:** 0-5h: 0.2 (-0.4, 0.8) 6-11h: 0.8 (-0.0, 1.7) 12-17h: 0.6 (-0.2, 1.4) 18-23h: 0.5 (-0.4, 1.4) 0-23h: 0.9 (-0.1, 2.0) 1d: 1.5 (0.3, 2.7) 2d: -0.4 (-1.7, 1.0) 3d: 0.5 (-0.9, 1.9) 4d: 0.1 (-1.2, 1.4) 0-4d avg: 1.6 (-0.1, 3.3) **Factor 3:** 0-5h: 0.1 (-0.3, 0.5) 6-11h: 0.2 (-0.3, 0.6) 12-17h: 0.2 (-0.3, 0.6) 18-23h: 0.1 (-0.3, 0.4) 0-23h: 0.1 (-0.3, 0.6) 1d: 0.1 (-0.3, 0.4) 2d: -0.1 (-0.4, 0.3) 3d: -0.2 (-0.5, 0.2) 4d: -0.1 (-0.5, 0.2) 0-4d avg: -0.1 (-0.7, 0.6) **Factor 4:** 0-5h: 0.2 (-0.4, 0.8) 6-11h: 0.8 (0.0, 1.6) 12-17h: 0.5 (-0.2, 1.3) 18-23h: 0.5 (-0.2, 1.2) 0-23h: 0.6 (-0.3, 1.4) 1d: -0.4 (-1.5, 0.7) 2d: -0.9 (-2.0, 0.1) 3d: -0.5 (-1.4, 0.5) 4d: -0.5 (-1.3, 0.2) 0-4d avg: -0.3 (-1.7, 1.1) **Factor 5**: n0-5h: 1.0 (-0.1, 2.1) 6-11h: 0.9 (-0.2, 2.0) 12-17h: 0.3 (-0.7, 1.4) 18-23h: -0.1 (-1.2, 1.0) 0-23h: 0.7 (-0.6, 1.9) 1d: 0.1 (-1.1, 1.3) 2d: -0.2 (-1.5, 1.1) 3d: -0.6 (-1.9, 0.8)

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Zanobetti et al. (2004, 087489) **Period of Study:** 1999 to 2001 **Location:** Boston, Massachusetts, USA	**Outcome:** Blood pressure (systolic blood pressure, diastolic blood pressure, mean arterial blood pressure) **Age Groups:** Elderly **Study Design:** Panel study **N:** 62 elderly subjects with n = 631 repeated visits for cardiac rehabilitation **Statistical Analysis:** Linear mixed effects models	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Median (10th–90th percentile)** Median: 8.8 $\mu g/m^3$ 10th-90th: 13.4 **Monitoring Stations:** 1 **Copollutant:** SO_2, O_3, CO, NO_2, BC 120-h avg Median: 0.651 10th-90th: 0.376	**PM Increment:** .10.4 $\mu g/m^3$ for 5 day mean, 13.9 $\mu g/m^3$ for 2-day mean **Effect Estimate:** Each 10.4 $\mu g/m^3$ increase in 5 day mean $PM_{2.5}$ concentration was associated with: Systolic BP: 2.8mmHg (95% CI: 0.1, 5.5) Diastolic BP: 2.7mmHg (95% CI: 1.2, 4.3) Mean arterial BP: 2.7mmHg (95% CI: 1.0, 4.5) Each 13.9 $\mu g/m^3$ increase in 2-day mean $PM_{2.5}$, during exercise in person with H.70bpm Diastolic: 7.0mmHg (95% CI: 2.3, 12.1) Mean arterial BP: 4.7mmHg (95% CI: 0.5, 9.1)

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Zeka et al. (2006, 157177) **Period of Study:** Nov 2000–Dec 2004 **Location:** Greater Boston area (Massachusetts)	**Outcome:** White blood cells (WBC), C-reactive protein (CRP), sediment rate, fibrinogen **Age Groups:** Mean age (SD) = 73.0 (6.7) **Study Design:** Cross-sectional **N:** 710 subjects **Statistical Analyses:** Linear regression **Covariates:** Age, BMI, season (also assessed potential for confounding by temperature, RH, barometric pressure, hypertensive or cardiac medications, hypertension, smoking, alcohol, and fasting glucose levels) **Dose-response Investigated?** No	**Pollutant:** BC **Averaging Time:** Hourly (PN, BC, $PM_{2.5}$) and 24-h ($SO4^{2-}$) measurements used to create 48-h, 1-wk, and 4-wk moving averages **Mean (SD):** 0.77 (0.63) **Percentiles:** 50th: 0.61 75th: 1.00 90th: 1.51 **Monitoring Stations:** 2 sites **Units:** ng/m^3 **Copollutant (correlation):** $PM_{2.5}$ (r = 0.52) BC PN (r = 0.30) $SO4^{2-}$ (r = 0.30)	**PM Increment:** 1 SD increase **Effect Estimate [Lower CI, Upper CI]:** % increase (95%CI) in biomarker per 1 SD increase in pollutant. **Fibrinogen** 48 h: 0.84 (-0.63, 2.31) 1 wk: 0.60 (-0.95, 2.15) 4 wk: 1.78 (0.19, 3.36) **CRP** 48 h: 4.51 (-2.03, 11.06) 1 wk: 1.07 (-5.55, 7.68) 4 wk: 5.41 (-1.00, 11.81) **Sediment rate** 48 h: -4.56 (-25.55, 16.43) 1 wk: 1.98 (-18.15, 22.11) 4 wk: 21.65 (1.48, 41.82) **WBC count** 48 h: -0.63 (-2.45, 1.19) 1 wk: -0.13 (-1.87, 1.60) 4 wk: -0.55 (-2.36, 1.26) **Note:** No statistically significant difference was reported for any category of effect modifiers (age, obesity, medications, homozygous for the deletion of GSTM1-null, hypertension) however, results suggested almost all the effect of BC on sediment rate was among the younger group (< 78 yrs) there was a 4-fold difference for the association between BC and CRP in the presence of obesity also evidence for effect modification by obesity of the association between BC and sediment rate there was a suggestive greater effect of BC on CRP among GSTM1-null subjects (9.73% [1.48, 17.98]) vs. GSTM1-present subjects (-2.97% [-14.05, 8.10] for concentrations 4-wk prior) a stronger effect of BC on sediment rate was seen among non-users of statins (36.01% [13.88, 58.13]) vs users (-12.29% [39.13, 14.55])

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Zeka et al. (2006, 157177) **Period of Study:** Nov 2000–Dec 2004 **Location:** Greater Boston area (Massachusetts)	**Outcome:** White blood cells (WBC), C-reactive protein (CRP), sediment rate, fibrinogen **Age Groups:** Mean age (SD) = 73.0 (6.7) **Study Design:** Cross-sectional **N:** 710 subjects **Statistical Analyses:** Linear regression **Covariates:** Age, BMI, season (also assessed potential for confounding by temperature, RH, barometric pressure, hypertensive or cardiac medications, hypertension, smoking, alcohol, and fasting glucose levels) **Dose-response Investigated?** No	**Pollutant:** SO42– **Averaging Time:** Hourly (PN, BC, $PM_{2.5}$) and 24-h (SO42–) measurements used to create 48-h, 1-wk, and 4-wk moving averages **Mean (SD):** 2.29 (1.62) **Percentiles:** 50th: 1.84 75th: 2.81 90th: 4.10 **Monitoring Stations:** 2 sites **Copollutant (correlation):** $PM_{2.5}$ (r = 0.50) BC (r = 0.30) PN (r = -0.15) SO42–	**PM Increment:** 1 SD increase **Effect Estimate [Lower CI, Upper CI]:** % increase (95%CI) in biomarker per 1 SD increase in pollutant. **Fibrinogen:** 48 h: 0.60 (-1.23, 2.42) 1 wk: 0.03 (-1.93, 1.99) 4 wk: 1.12 (-0.52, 2.77) **CRP:** 48 h: 1.57 (-7.13, 10.27) 1 wk: 0.21 (-8.27, 8.69) 4 wk: 5.29 (-1.91, 12.49) **Sediment rate:** 48 h: 4.05 (-23.26, 31.36) 1 wk: -5.87 (-32.39, 20.64) 4 wk: -1.60 (-25.24, 22.04) **WBC count:** 48 h: -0.12 (-2.35, 2.11) 1 wk: -0.48 (-2.87, 1.90) 4 wk: 0.75 (-1.30, 2.80) **Note:** No statistically significant difference was reported for any category of effect modifiers (age, obesity, medications, homozygous for the deletion of GSTM1-null, hypertension)
Reference: Zeka et al. (2006, 157177) **Period of Study:** Nov 2000–Dec 2004 **Location:** Greater Boston area (Massachusetts)	**Outcome (ICD9 and ICD10):** White blood cells (WBC), C-reactive protein (CRP), sediment rate, fibrinogen **Age Groups:** Mean age (SD) = 73.0 (6.7) **Study Design:** Cross-sectional **N:** 710 subjects **Statistical Analyses:** Linear regression **Covariates:** Age, BMI, season (also assessed potential for confounding by temperature, RH, barometric pressure, hypertensive or cardiac medications, hypertension, smoking, alcohol, and fasting glucose levels) **Dose-response Investigated?** No **Statistical Package:** NR	**Pollutant:** $PM_{2.5}$ **Averaging Time:** Hourly (PN, BC, $PM_{2.5}$) and 24-h (SO42–) measurements used to create 48-h, 1-wk, and 4-wk moving averages **Mean (SD):** 11.16 (7.95) **Percentiles:** 50th: 9.39 75th: 14.57 90th: 21.48 **Monitoring Stations:** 2 sites **Copollutant (correlation):** $PM_{2.5}$ BC (r = 0.52) PN (r = -0.02) SO42– (r = 0.50)	**PM Increment:** 1 SD increase **Effect Estimate [Lower CI, Upper CI]:** % increase (95%CI) in biomarker per 1 SD increase in pollutant. **Fibrinogen:** 48 h: -0.18 (-1.93, 1.57) 1 wk: -1.39 (-3.46, 0.67) 4 wk: 1.14 (-0.60, 2.88) **CRP:** 48 h: -4.88 (-13.29, 3.53) 1 wk: -1.37 (-10.44, 7.71) 4 wk: 4.36 (-3.25, 11.96) **Sediment rate:** 48 h: -16.91 (-43.66, 9.84) 1 wk: -18.89 (-47.48, 9.70) 4 wk: 24.93 (0.68, 49.18) **WBC count:** 48 h: -3.18 (-5.39 to -0.97) 1 wk: -0.51 (-3.02, 2.00) 4 wk: -0.03 (-2.17, 2.10) **Note:** No statistically significant difference was reported for any category of effect modifiers (age, obesity, medications, homozygous for the deletion of GSTM1-null, hypertension)
Reference: Zhang et al. (2009, 191970) **Period of Study:** 1999-2003 **Location:** US	**Outcome:** Myocardial Ischemia **Age Groups:** 52-90 **Study Design:** panel **N:** 55,529 **Statistical Analyses:** Logistic & Linear Regression **Covariates:** age, race/ethnicity, education, exam site, BMI, current	**Pollutant:** $PM_{2.5}$ **Averaging Time:** NR **Mean (SD):** Lag 0: 14.1 (8) Lag 1: 13.8 (8) Lag 2: 13.8 (8) Lag 3: 13.8 (8)	**PM Increment:** 10$\mu g/m^3$ **Odds Ratio (Lower CI, Upper CI), lag:** Minnesota Codes* MC4: 1.04 (0.97, 1.10), lag 0-2 MC4: 1.04 (0.98, 1.11), lag 3-5 MC5: 1.05 (1.00, 1.09), lag 0-2 MC5: 1.04 (1.00, 1.08), lag 3-5

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
	smoking status, history of CHD, diabetes, hypertension, SBP, chronic lung disease, or hypercholesterolemia, day of week, time of day, temperature, dew point, pressure, season	Lag 4: 13.9 (8)	MC 4 or 5: 1.04 (1.00, 1.09), lag 0-2
		Lag 5: 14.1 (8)	MC 4 or 5: 1.03 (0.99, 1.07), lag 3-5
		Lag 0-2: 13.9 (7)	**Change (Lower CI, Upper CI), lag:**
	Dose-response Investigated? No	**Monitoring Stations:** NR[‡]	ST-segment amplitude
	Statistical Package: SAS	**Co-pollutant:** NR	Lead I: -0.07 (-0.36, 0.21), lag 0-2
	Lags Considered: 0-5d	[‡]Monitors used in model for spatial interpolation of daily PM values.	Lead I: 0.18 (-0.10, 0.46), lag 3-5
			Lead II: -0.12 (-0.47, 0.23), lag 0-2
			Lead II: 0.16 (-0.18, 0.50), lag 3-5
			Lead aVL: -0.01 (-0.25, 0.23), lag 0-2
			Lead aVL: 0.11 (-0.12, 0.34), lag 3-5
			Lead V1: -0.02 (-0.39, 0.35), lag 0-2
			Lead V1: -0.22 (-0.58, 0.14), lag 3-5
			Lead V2: 0.07 (-0.57, 0.70), lag 0-2
			Lead V2: -0.01 (-0.61, 0.62), lag 3-5
			Lead V3: -0.11 (-0.68, 0.47), lag 0-2
			Lead V3: -0.02 (-0.58, 0.54), lag 3-5
			Lead V4: -0.0.3 (-0.51, 0.45), lag 0-2
			Lead V4: 0.24 (-0.23, 0.71), lag 3-5
			Lead V5: -0.01 (-0.41, 0.39), lag 0-2
			Lead V5: 0.35 (-0.04, 0.74), lag 3-5
			Lead V6: 0.02 (-0.30, 0.33), lag 0-2
			Lead V6: 0.35 (0.04, 0.65), lag 3-5
			T-wave amplitude
			Lead I: -1.60 (-3.07, -0.13), lag 0-2
			Lead I: -0.31 (-1.73, 1.11), lag 3-5
			Lead II: -0.54 (-1.99, 0.92), lag 0-2
			Lead II: 0.71 (-0.70, 2.13), lag 3-5
			Lead aVL: -1.21 (-2.50, 0.10), lag 0-2
			Lead aVL: -0.55 (-1.18, 0.71), lag 3-5
			Lead V1: 1.45 (-0.16, 3.06), lag 0-2
			Lead V1: 0.03 (-1.53, 1.59), lag 3-5
			Lead V2: -0.18 (-2.96, 2.60), lag 0-2
			Lead V2: 0.57 (-2.12, 3.27), lag 3-5
			Lead V3: -2.33 (-5.15, 0.49), lag 0-2
			Lead V3: -0.13 (-2.87, 2.60), lag 3-5
			Lead V4: -2.03 (-4.69, 0.63), lag 0-2
			Lead V4: 0.64 (-1.94, 3.22), lag 3-5
			Lead V5: -1.92 (-4.22, 0.38), lag 0-2
			Lead V5: 0.55 (-1.69, 2.78), lag 3-5
			Lead V6: -0.63 (-2.36, 1.10), lag 0-2
			Lead V6: 0.82 (-0.86, 2.49), lag 3-5
			QRS/T angles and heart rate (change)
			QRS/T angle – spatial (°): 0.19 (-0.21, 0.59), lag 0-2
			QRS/T angle – spatial (°): -0.20 (-0.59, 0.19), lag 3-5

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			QRS/T angle – frontal (°): 0.13 (-0.24, 0.50), lag 0-2 QRS/T angle – frontal (°): 0.35 (-0.01, 0.71), lag 3-5 Heart Rate (beats/min): 0.16 (0.02, 0.30), lag 0-2 Heart Rate (beats/min): 0.04 (-0.10, 0.18), lag 3-5 *Any ST abnormality (MC 4.1-4.4) Any T abnormality (MC 5.1-5.4)

[1]All units expressed in $\mu g/m^3$ unless otherwise specified.

Table E-4. Short-term exposure – cardiovascular morbidity studies - other size fractions.

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Adar et al. (2007, 001458) **Period of Study:** March–June 2002 **Location:** St. Louis, Missouri	**Outcome:** Heart rate variability: heart rate, standard deviation of all normal-to-normal intervals (SDNN), square root of the mean squared difference between adjacent normal-to-normal intervals (rMSSD), percentage of adjacent normal-to-normal intervals that differed by more than 50 ms (pNN50), high frequency power (HF in the range of 0.15-0.4Hz), low frequency power (LF, in the range of 0.04-0.15Hz), and the ratio of LF/HF **Age Groups:** ≥ 60 yrs **Study Design:** Panel (4 planned repeated measures with a total of 158 person-trips 35 participating in all 4 trips) **N:** 44 participants **Statistical Analyses:** Generalized additive models **Covariates:** Subject, weekday, time, apparent temperature, trip type, activity, medications, and autoregressive terms **Season:** Limited data collection period **Dose-response Investigated?** No **Statistical Package:** SAS v8.02, R v2.0.1	**Pollutant:** Particle count fine (PC fine) (particles/cm^3) **Averaging Time:** Measurements collected over 48 h period surrounding the bus trip (during which health endpoints were measured) used to calculate 5-, 30-, 60-minute, 4-h, 24-h moving averages **Median (IQR):** All: 42 (57) Facility: 36 (45) Bus: 105 (96) Activity: 50 (133) Lunch: 69 (48) **Monitoring Stations:** 2 portable carts **Copollutant:** $PM_{2.5}$ BC Fine particle counts Coarse particle counts **Correlation notes:** 24-h mean $PM_{2.5}$, BC, and fine particle count concentrations ranged from 0.80 to 0.98 r = 0.76 to 0.97 when limited to time spent on the bus r = 0.55 to 0.86 when comparing bus concentrations to 24-h moving averages r = -0.003 to 0.51 when comparing 5-min averages and 24-h moving averages. Poor correlations found between coarse particle count concentrations and all fine particulate measures during all times periods	**PM Increment:** IQR **Effect Estimate [Lower CI, Upper CI]:** % change (95%CI) in HRV per IQR in the 24-h moving avg of the microenvironmental pollutant (IQr = 39 pt/cm^3) **Single-pollutant models** SDNN: -5.1 (-5.8 to -4.4) rMSSD: -8.0 (-8.7 to -7.2) pNN50 + 1: -10.2 (-11.3 to -9.0) LF: -9.9 (-11.4 to -8.4) HF: -13.7 (-15.1 to -12.2) LF/HF: 4.3 (3.1, 5.5) H: 0.9 (0.8, 1.1) **Note:** Exposure to health associations by all lag periods presented in Figure 2 (magnitude of associations increased with averaging period, with the largest associations consistently found for 24-h moving averages)
Reference: Adar et al. (2007, 001458) **Period of Study:** March–June 2002 **Location:** St. Louis, Missouri	**Outcome:** Heart rate variability: heart rate, standard deviation of all normal-to-normal intervals (SDNN), square root of the mean squared difference between adjacent normal-to-normal intervals (rMSSD), percentage of adjacent normal-to-normal intervals that differed by more than 50 ms (pNN50), high frequency power (HF in the range of 0.15-0.4Hz), low frequency power (LF, in the range of	**Pollutant:** Particle count coarse (PT coarse) (pt/cm^3) **Averaging Time:** Measurements collected over 48-h period surrounding the bus trip (during which health endpoints were measured) used to calculate 5-, 30-, 60-minute, 4-h, and 24-h moving averages **Median (IQR):** All: 0.02 (0.11) Facility: 0.01 (0.04)	**PM Increment:** IQR **Effect Estimate [Lower CI, Upper CI]:** % change (95%CI) in HRV per IQR in the 24-h moving avg of the microenvironmental pollutant (IQr = 0.066 pt/cm^3) **Single-pollutant models** SDNN: 2.4 (1.3, 3.6) rMSSD: 3.9 (2.6, 5.1)

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
	0.04-0.15Hz), and the ratio of LF/HF **Age Groups:** ≥ 60 yrs **Study Design:** Panel (4 planned repeated measures with a total of 158 person-trips 35 participating in all 4 trips) **N:** 44 participants **Statistical Analyses:** Generalized additive models **Covariates:** Subject, weekday, time, apparent temperature, trip type, activity, medications, and autoregressive terms **Season:** Limited data collection period **Dose-response Investigated?** No **Statistical Package:** SAS v8.02, R v2.0.1	Bus: 0.16 (0.13) Activity: 0.29 (0.26) Lunch: 0.16 (0.36) **Monitoring Stations:** 2 portable carts **Copollutant:** $PM_{2.5}$ BC Fine particle counts Coarse particle counts **Correlation notes:** 24-h mean $PM_{2.5}$, BC, and fine particle count concentrations ranged from 0.80 to 0.98 r = 0.76 to 0.97 when limited to time spent on the bus r = 0.55 to 0.86 when comparing bus concentrations to 24-h moving averages r = -0.003 to 0.51 when comparing 5-min averages and 24-h moving averages. Poor correlations found between coarse particle count concentrations and all fine particulate measures during all times periods	pNN50 + 1: 2.9 (1.0, 4.9) LF: 6.4 (3.7, 9.1) HF: 10.2 (7.4, 13.1) LF/HF: -3.3 (-5.0 to -1.6) H: -1.1 (-1.3 to -0.8) **Two-pollutant models (with $PM_{2.5}$):** SDNN: -0.7 (-1.9, 0.6) rMSSD: -1.3 (-2.6 to -0.05) pNN50 + 1: -4.3 (-6.3 to -2.4) LF: 0.2 (-2.5, 3.0) HF: 1.3 (-1.5, 4.1) LF/HF: -0.9 (-2.7, 1.0) H: -0.6 (-0.9 to -0.4) **Note:** Exposure to health associations by all lag periods presented in Figure 2 (magnitude of associations increased with averaging period, with the largest associations consistently found for 24-h moving averages)
Reference: Delfino et al. (2008, 156390) **Period of Study:** 2005-2006 **Location:** Los Angeles, California, air basin	**Outcome:** C-reactive protein (CRP) fibrinogen, tumor necrosis factor-□ (TNF-□) and its soluble receptor-II (TNF-RII) interleukin-6 (IL-6) and its soluble receptor (IL-6sR) fibrin D-dimer soluble platelet selectin (sP-selectin) soluble vascular cell adhesion molecule-1 (sVCAM-1) intracellular adhesion molecule-1 (sICAM-1) and myeloperoxidase (MPO) erythrocyte lysates for glutathione peroxidase-1 (GPx-1) copper-zinc superoxide dismutase (cu, Zn-SOD) **Age Groups:** ≥ 65 yrs **Study Design:** Panel (biomarkers measured weekly 12 times) **N:** 29 participants (nonsmoking with history of coronary artery disease) **Statistical Analyses:** Mixed models **Covariates:** temperature (infectious illnesses were excluded by excluding weeks with such observations) **Season:** Collected 6 weeks of data during warm period and 6 weeks of data during cool period **Dose-response Investigated?** No **Statistical Package:** NR	**Pollutant:** PM (multiple size fractions and components) **Averaging Time:** 24-h avg preceding the blood draw (lag 0) and cumulative averages up to 5 days preceding the draw **Outdoor hourly PM:** EC: Mean (SD): 1.61 (0.62) Median: 1.56 IQR: 0.92 Min, Max: 0.24, 3.94 OC: Mean (SD): 5.94 (2.11) Median: 5.58 IQR: 2.79 Min-Max: 2.51, 13.60 BC: Mean (SD): 2.00 (0.77) Median: 1.89 IQR: 0.96 Min-Max: 0.58, 5.11 OC_{pri}: Mean (SD): 3.37 (1.21) Median: 3.21 IQR: 1.63 Min-Max: 0.99, 7.11 Secondary OC: Mean (SD): 2.49 (1.50) Median: 2.10 IQR: 1.86 Min-Max: 0, 8.10 PN (p/cm^3): Mean (SD): 16,043 (5886) Median: 13,968	**PM Increment:** IQR **Effect Estimate [Lower CI, Upper CI]:** **Note:** Nearly all results presented in figures **Results:** The authors found significant positive associations for CRP, IL-6, sTNF-RII, and sP-selectin with outdoor and/or indoor concentrations of quasi-ultrafine PM □ 0.25 µm in diameter, EC, OC_{pri}, BC, PN, CO, and nitrogen dioxide from the current-day and multiday averages. There were consistent positive but largely nonsignificant coefficients for TNF-□, sVCAM-1, and sICAM-1, but not fibrinogen, IL-6sR, or D-dimer. The authors found inverse associations for erythrocyte Cu, Zn-SOD with these pollutants and other PM size fractions (0.25–2.5 and 2.5–10 µm). Inverse associations of GPx-1 and MPO with pollutants were largely nonsignificant. Indoor associations were often stronger for estimated indoor EC, OC_{pri}, and PN of outdoor origin than for uncharacterized indoor measurements. There was no evidence for positive associations with SOA.

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
		IQR: 7,386	
		Min-Max: 6837, 31263	
		Indoor hourly PM EC: Mean (SD): 1.31 (0.52)	
		Median: 1.30	
		IQR: 0.70	
		Min-Max: 0.19, 2.89	
		EC of outdoor origin: Mean (SD): 1.11 (0.39)	
		Median: 1.06	
		IQR: 0.51	
		Min-Max: 0.41, 2.97	
		OC: Mean (SD): 5.69 (1.51)	
		Median: 5.60	
		IQR: 1.96	
		Min-Max: 2.34, 10.79	
		OC_{pri} of outdoor origin: Mean (SD): 2.18 (0.82)	
		Median: 2.15	
		IQR: 1.07	
		Min-Max: 0.32, 5.21	
		Secondary OC of outdoor origin: Mean (SD): 2.08 (1.26)	
		Median: 1.75	
		IQR: 1.45	
		Min-Max: 0, 6.87	
		PN (particles/cm^3): Mean (SD): 14,494 (6770)	
		Median: 12,341	
		IQR: 7,337	
		Min-Max: 1016, 43027	
		PN of outdoor origin (p/cm^3): Mean (SD): 10,108 (3108)	
		Median: 9,580	
		IQR: 3,684	
		Min-Max: 1016, 17700	
		Outdoor PM mass $PM0.2_5$: Mean (SD): 9.47 (2.97)	
		Median: 9.4	
		IQR: 4.2	
		Min-Max: 3.31, 18.75	
		$PM0.2_{5\text{-}2.5}$: Mean (SD): 13.53 (10.67)	
		Median: 11.7	
		IQR: 11.5	
		Min-Max: 1.29, 66.77	
		$PM_{2.5\text{-}10}$: Mean (SD): 10.04 (4.07)	
		Median: 9.9	
		IQR: 5.9	

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
		Min-Max: 1.76, 22.38 **Indoor PM mass** $PM0.2_5$: Mean (SD): 10.45 (6.77) Median: 9.5 IQR: 4.5 Min-Max: 1.42, 69.86 $PM_{0.25\text{-}2.5}$ ($\mu g/m^3$): Mean (SD): 7.36 (4.57) Median: 6.5 IQR: 5.7 Min-Max: 0.77, 30.86 $PM_{2.5\text{-}10}$: Mean (SD): 4.12 (4.76) Median: 2.8 IQR: 3.5 Min-Max: 0.12, 37.63 **Copollutant:** Outdoor hourly gases (NO_2, CO, O_3) and indoor hourly gases (NO_2, CO)	
Reference: Pekkanen et al. (2002, 035050) **Period of Study:** Winter 1998 to 1999 **Location:** Helsinki, Finland	**Outcome**: ST Segment Depression (>0.1mV) **Study Design**: Panel of ULTRA Study participants **N**: 45 Subjects, n = 342 biweekly submaximal exercise tests, 72 exercise induced ST Segment Depressions **Statistical Analysis:** Logistic regression / GAM	**Pollutant:** Ultrafine NC0.01-0.1 μm (n/cm^3) **Averaging Time:** 24 h **Median**: 14,890 **IQR**: 9830 **Monitoring Stations**: 1 **Copollutant:** NO_2, CO, $PM_{2.5}$, PM_{10}-2.5, PM1, ACP	**PM Increment:** IQR **Effect Estimate(s):** NC0.01-0.1: OR = 3.14 (1.56, 6.32), lag 2 **Notes**: The effect was strongest for ACP and $PM_{2.5}$, which in two pollutant models appeared independent. Increases in NO_2 and CO were also associated with increased risk of ST segment depression, but not with coarse particles.
Reference: Peters et al. (2005, 095747) Also Peters et al, 2005 (2005, 156859) **Period of Study:** February 1999–July 2001 **Location:** Augsburg, Germany	**Outcome:** Myocardial infarction **Study Design:** Case-crossover **N:** 691 myocardial infarction patients **Statistical Analysis:** Conditional logistic regression **Dose-response investigated (yes/no)?** No	**Pollutant:** Ultrafine (TNC) (n/cm^3) **Averaging Time:** 1 h: Median = 10,001 IQR: 7919 24 h: Median = 10,934 IQR: 6276 **Copollutant:** NO_2, SO_2, CO	**PM Increment: Effect Estimate:** 2-h lag: OR = 0.95 95% CI: 0.84, 1.06 24-h mean, 2-day lag: OR = 1.04 95% CI: 0.90, 1.20 **Notes:** Examined triggering for MI at various lags before MI onset (up to 6 h before MI, up to 5 days before MI). No statistically significant increases in lagged ultrafine particle concentration were found.
Reference: Ruckerl et al. (2006, 088754) **Period of Study:** Oct 2000–Apr 2001 **Location:** Erfurt, Germany	**Outcome (ICD9 and ICD10):** C-reactive protein (CRP) serum amyloid A (SAA) E-selectin von Willebrand Factor (vWF) intercellular adhesion molecule-1 (ICAM-1) fibrinogen Factor VII prothrombin fragment 1+2 D-dimer **Age Groups:** 50+ yrs **Study Design:** Panel (12 repeated measures at 2-wk intervals) **N:** 57 male subjects with coronary disease	**Pollutant:** AP (n/cm^3) **Averaging Time:** 24 h **Mean (SD):** 1593 (1034) **Percentiles:** 25: 821 50: 1238 75: 2120 **Range (Min, Max):** 328, 4908 **Unit (i.e. $\mu g/m^3$):** n/cm^3 **Monitoring Stations:** 1 site **Copollutant:** UFPs (ultrafine particles) AP (accumulation mode particles) $PM_{2.5}$ PM_{10}	**PM Increment:** IQR (1299 5-d avg: 1127) **Effect Estimate [Lower CI, Upper CI]:** Effects of air pollution on blood markers presented as OR (95%CI) for an increase in the blood marker above the 90th percentile per increase in IQR air pollutant. **CRP** Time before draw: 0 to 23 h: 0.7 (0.5, 1.2) 24 to 47 h: 1.5 (0.9, 2.6) 48 to 71 h: 3.2 (1.7, 6.0) 5-d mean: 1.5 (0.8, 3.0) **ICAM-1** Time before draw: 0 to 23 h: 0.6 (0.4, 0.9) 24 to 47 h: 1.8 (1.2, 2.8) 48 to 71 h: 1.6 (1.0, 2.5)

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
	Statistical Analyses: Fixed effects linear and logistic regression models **Covariates:** Models adjusted for different factors based on health endpoint CRP: RH, temperature, trend, ID ICAM-1: temperature, trend, ID vWF: air pressure, RH, temperature, trend, ID FVII: air pressure, RH, temperature, trend, ID, weekday **Season:** Time trend as covariate **Dose-response Investigated?** Sensitivity analyses examined nonlinear exposure-response functions **Statistical Package:** SAS v8.2 and S-Plus v6.0	OC (organic carbon) EC (elemental carbon) NO_2 CO	5-d mean: 0.9 (0.6, 1.5) Effects of air pollution on blood markers presented as % change from the mean/GM in the blood marker per increase in IQR air pollutant. **vWF** Time before draw: 0 to 23 h: 4.8 (0.2, 9.3) 24 to 47 h: 5.9 (0.4, 11.5) 48 to 71 h: 7.0 (0.7, 13.4) 5-d mean: 13.5 (6.3, 20.6) **FVII** Time before draw: 0 to 23 h: 0.0 (-2.9, 3.0) 24 to 47 h: -2.9 (-6.1, 0.4) 48 to 71 h: -3.6 (-6.8 to -0.3) 5-d mean: -4.1 (-7.9 to -0.3) **Note:** summary of results presented in figures. SAA results indicate increase in association with PM (not as strong and consistent as with CRP) no association observed between E-selectin and PM an increase in prothrombin fragment 1+2 was consistently observed, particularly with lag 4 fibrinogen results revealed few significant associations, potentially due to chance D-dimer results revealed null associations in linear and logistic analyses
Reference: Ruckerl et al. (2006, 088754) **Period of Study:** Oct 2000–Apr 2001 **Location:** Erfurt, Germany	**Outcome:** Soluble CD40 ligand (sCD40L), platelets, leukocytes, erythrocytes, hemoglobin **Age Groups:** 50+ yrs **Study Design**: Panel (12 repeated measures at 2-wk intervals) **N:** 57 male subjects with coronary disease **Statistical Analyses:** Fixed effects linear regression models **Covariates:** Long-term time trend, weekday of the visit, temperature, RH, barometric pressure **Season:** Time trend as covariate **Dose-response Investigated?** No **Statistical Package:** SAS v8.2 and S-Plus v6.0	**Pollutant:** AP (n/cm^3) **Averaging Time:** 24 h **Mean (SD):** 1593 (1034) **Percentiles:** 25th: 821 50th: 1238 75th: 2120 **Range (Min, Max):** 328, 4908 **Monitoring Stations:** 1 site **Copollutant:** UFPs (ultrafine particles) AP (accumulation mode particles) $PM_{2.5}$ PM_{10} NO	**PM Increment:** IQR (1299 5-d avg: 1127) **Effect Estimate [Lower CI, Upper CI]:** Effects of air pollution on blood markers presented as % change from the mean/GM in the blood marker per increase in IQR air pollutant. **sCD40L, % change GM (pg/mL)** lag0: 6.9 (0.5, 13.8) lag1: -1.1 (-8.0, 6.4) lag2: -4.9 (-11.9, 2.7) lag3: -3.8 (-10.3, 3.2) 5-d mean: -1.3 (-9.9, 8.1) **Platelets, % change mean (10^3/µl)** lag0: -1.0 (-2.5, 0.5) lag1: -0.4 (-2.1, 1.6) lag2: 0.8 (-1.0, 2.4) lag3: 0.0 (-1.8, 1.7) 5-d mean: -0.9 (-3.0, 1.3) **Leukocytes, % change in mean (10^3/µl)** lag0: -1.9 (-3.8 to -0.1) lag1: -0.6 (-2.9, 1.6) lag2: -0.6 (-3.2, 2.0)

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			lag3: -2.3 (-4.6, 0.1) 5-d mean: -2.7 (-5.5, 0.1) **Erythrocytes, % change mean (10^6/µl)** lag0: -0.1 (-0.5, 0.3) lag1: -0.4 (-0.9, 0.2) lag2: -0.4 (-0.9, 0.2) lag3: -0.4 (-0.6, 0.3) 5-d mean: -0.4 (-1.0, 0.2) **Hemoglobin, % change mean (g/dl)** lag0: -0.2 (-0.7, 0.4) lag1: -0.3 (-1.0, 0.4) lag2: -0.1 (-0.9, 0.7) lag3: -0.1 (-0.8, 0.6) 5-d mean: -0.2 (-1.1, 0.6)
Reference: Ruckerl et al. (2007, 156931) **Period of Study:** May 2003–Jul 2004 **Location:** Athens, Augsburg, Barcelona, Helsinki, Rome, and Stockholm	**Outcome:** Interleukin-6 (IL-6), fibrinogen, C-reactive protein (CRP) **Age Groups:** 35-80 yrs **Study Design:** Repeated measures / longitudinal **N:** 1003 MI survivors **Statistical Analyses:** Mixed-effect models **Covariates:** City-specific confounders (age, sex, BMI) long-term time trend and apparent temperature RH, time of day, day of week included if adjustment improved model fit **Season:** Long-term time trend **Dose-response Investigated?** Used p-splines to allow for nonparametric exposure-response functions **Statistical Package:** SAS v9.1	**Pollutant:** UFP (n/cm^3) **Averaging Time:** Hourly and 24 h (lag 0-4, mean of lags 0-4, mean of lags 0-1, mean of lags2-3, means of lags 0-3) **Mean (SD):** Presented by city only **Percentiles:** NR **Range (Min, Max):** NR **Monitoring Stations:** Central monitoring sites in each city **Copollutant:** SO_2 O_3 NO NO_2	**PM Increment:** IQR **Effect Estimate [Lower CI, Upper CI]:** % change in mean blood markers per increase in IQR of air pollutant. IL-6 Lag (IQR): % change in GM (95%CI) Lag 0 (11852): 1.88 (-0.16, 3.97) Lag 1 (11852): -0.67 (-2.56, 1.25) Lag 2 (11852): -2.12 (-4.03 to -0.17) 5-d avg (11003): -0.93 (-3.37, 1.56) Fibrinogen Lag (IQR): % change in AM (95%CI) Lag 0 (11852): 0.40 (-0.40, 1.19) Lag 1 (11852): 0.11 (-0.69, 0.91) Lag 2 (11852): 0.09 (-0.71, 0.90) 5-d avg (11003): 0.50 (-2.20, 3.20) CRP Lag (IQR): % change in GM (95%CI) Lag 0 (11852): 1.33 (-3.05, 5.90) Lag 1 (11852): -1.52 (-4.39, 1.45) Lag 2 (11852): -1.63 (-6.70, 3.71) 5-d avg (11003): -0.08 (-3.78, 3.75)
Reference: Pekkanen et al. (2002, 035050) **Period of Study:** Winter 1998 to 1999 **Location:** Helsinki, Finland	**Outcome:** ST Segment Depression (>0.1mV) **Age Groups: Study Design:** Panel of ULTRA Study participants **N:** 45 Subjects, n = 342 biweekly submaximal exercise tests, 72 exercise induced ST Segment Depressions **Statistical Analysis:** Logistic regression / GAM	**Pollutant:** Ultrafine NC0.01-0.1 μm (n/cm^3) **Averaging Time:** 24 h **Median:** 14,890 **IQR:** 9830 **Monitoring Stations:** 1 **Copollutant:** NO_2, CO, $PM_{2.5}$, $PM_{10-2.5}$, PM1, ACP	**PM Increment:** IQR **Effect Estimate(s):** NC0.01-0.1: OR = 3.14 (1.56, 6.32), lag 2 **Notes:** The effect was strongest for ACP and $PM_{2.5}$, which in two pollutant models appeared independent. Increases in NO_2 and CO were also associated with increased risk of ST segment depression, but not with coarse particles.

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Peters et al. (2005, 095747) Also Peters et al, 2005 (2005, 156859) **Period of Study:** February 1999–July 2001 **Location:** Augsburg, Germany	**Outcome:** Myocardial infarction **Study Design:** Case-crossover **N:** 691 myocardial infarction patients **Statistical Analysis:** Conditional logistic regression **Dose-response Investigated?** No	**Pollutant:** Ultrafine (TNC) (n/cm^3) **Averaging Time:** 1 h: Median = 10,001 IQR: 7919 24-h: Median = 10,934 IQR: 6276 **Copollutant:** NO_2, SO_2, CO	**PM Increment: Effect Estimate:** 2 h lag: OR = 0.95 95% CI: 0.84, 1.06 24-h mean, 2-day lag: OR = 1.04 95% CI: 0.90, 1.20 **Notes:** Examined triggering for MI at various lags before MI onset (up to 6 h before MI, up to 5 days before MI). No statistically significant increases in lagged ultrafine particle concentration were found.
Reference: Ruckerl et al. (2007, 091379) **Period of Study:** Oct 2000–Apr 2001 **Location:** Erfurt, Germany	**Outcome (ICD9 and ICD10):** Soluble CD40 ligand (sCD40L), platelets, leukocytes, erythrocytes, hemoglobin **Age Groups:** 50+ yrs **Study Design**: Panel (12 repeated measures at 2-wk intervals) **N:** 57 male subjects with coronary disease **Statistical Analyses:** Fixed effects linear regression models **Covariates:** Long-term time trend, weekday of the visit, temperature, RH, barometric pressure **Season:** Time trend as covariate **Dose-response Investigated?** No **Statistical Package:** SAS v8.2 and S-Plus v6.0	**Pollutant:** UFP **Averaging Time:** 24 h **Mean (SD):** 12,602 (6455) **Percentiles:** 25th: 7326 50th: 11,444 75th: 17,332 **Range (Min, Max):** 328, 4908 **Monitoring Stations:** 1 site **Copollutant:** AP $PM_{2.5}$ PM_{10} NO	**PM Increment:** IQR (10,005 5-d avg: 6,821) **Effect Estimate [Lower CI, Upper CI]:** **sCD40L, % change GM (pg/mL)** lag 0: 7.1 (0.1, 14.5) lag 1: 0.3 (-6.6, 8.6) lag 2: 0.6 (-5.9, 8.6) lag 3: -8.5 (-15.8, -0.5) 5-d mean: -0.7 (-7.6, 6.8) **Platelets, % change mean (10^3/µl)** lag 0: -1.8 (-3.4, -0.2) lag 1: -1.1 (-2.9, 0.6) lag 2: 1.0 (-2.9, 0.8) lag 3: -2.4(4.5, -0.3) 5-d mean: -2.2 (-4.0, -0.3) **Leukocytes, [10^3/µl]** lag 0: -2.4 (-4.5, -0.2) lag 1: -2.1 (-4.4, 0.2) lag 2: -0.2 (-2.4, 2.8) lag 3: -1.5 (-4.4, 1.4) 5-d mean: -1.6 (-4.1, 0.8)

[1]All units expressed in $\mu g/m^3$ unless otherwise specified.

E.1.2. Cardiovascular Emergency Department Visits and Hospital Admissions

Table E-5. Short-term exposure–cardiovascular–ED/HA PM_{10}

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Anderson et al. (2003, 054820) **Period of Study:** 1992-1994 **Location:** London, United Kingdom	**Outcome:** Ischemic Heart Disease **Age Groups:** 0-15, 15-64, 65-74, 75+ **Study Design:** Time series **N:** NR **Statistical Analyses:** NR **Covariates:** NR **Dose-response Investigated?** No **Statistical Package:** NR	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (min-max):** NR **Monitoring Stations:** NR **Copollutant:** NR	**PM Increment:** 10th–90th percentile **% Change in Daily IHD Admissions by Age [CI]:** 0-15 yrs: NR 15-64 yrs: 2.6 [0.3,5] 65-74 yrs: 2.5 [0.1,4.9] 75+ yrs: 2.2 [0.2,4.6] **Notes:** RRs are presented in graph form showing little change with increasing age (PM increment of 10 $\mu g/m^3$). This article is primarily a systematic literature review of other studies.
Reference: Andersen et al. (2008, 189651) **Period of Study:** May 2001 - December 2004 **Location:** Los Angeles and San Diego counties, California	**Outcome (ICD-10):** CVD, including angina pectoris (I20), myocardial infarction (I21–22), other acute ischemic heart diseases (I24), chronic ischemic heart disease (I25), pulmonary embolism (I26), cardiac arrest (I46), cardiac arrhythmias (I48–48), and heart failure (I50). **Age Groups:** >65 yrs (CVD and RD), 5–18 years (asthma) **Study Design:** Time series **N:** NR **Statistical Analyses:** Poisson GAM **Covariates:** Temperature, dew-point temperature, long-term trend, seasonality, influenza, day of the week, public holidays. **Season:** NR **Dose-response Investigated:** No **Statistical Package:** R (gam procedure, mgcv package) **Lags Considered:** Lag 0 -5 days, 4-day pollutant avg (lag 0 -3) for CVD.	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (SD** **median** **IQR** **99th percentile):** 24 (14 21 16–29 72) **Monitoring Stations:** 1 **Copollutant (correlation):** NCtot: r = 0.39 NC100: r = 0.28 NCa12: r = 0.02 NCa23: r = -0.12 NCa57: r = 0.45 NC_{a212}: r = 0.63 $PM_{2.5}$: r = 0.80 CO: r = 0.37 NO_2: r = 0.35 NO_X: r = 0.32 NO_X curbside: r = 0.18 O_3: r = -0.21 **Other variables:** Temperature: r = 0.12 Relative humidity: r = 0.05	**PM Increment:** 13 $\mu g/m^3$ (IQR) **Relative risk (RR) Estimate [CI]:** CVD hospital admissions (4-day avg, lag 0 -3), age 65+: One-pollutant model: 1.03 [1.01–1.05] Adj for NCtot: 1.04 [1.02–1.06] Adj for NC_{a212}: 1.05 [1.01–1.09] RD hospital admissions (5 day avg, lag 0 -4), age 65+: One-pollutant model: 1.06 [1.02–1.09] Adj for NCtot: 1.05 [1.01–1.10] Adj for NC_{a212}: 1.04 [0.98–1.11] Asthma hospital admissions (6-day avg lag 0–5), age 5 - 18: One-pollutant model: 1.02 [0.93–1.12] Adj for NCtot: 1.01 [0.91–1.12] Adj for NC_{a212}: 0.94 [0.81–1.09] Estimates for individual day lags reported only in figure form (see notes): **Notes:** Figure 2: Relative risks and 95% confidence intervals per IQR in single day concentration (0- to 5-day lag). Summary of Figure 2: CVD: Positive, marginally or statistically significant associations at Lag 0–Lag 2.

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Anderson et al. (2007, 156214) **Period of Study:** 1/99-12-04 **Location:** Copenhagen, Denmark	**Outcome (ICD10):** Hospital Admission, CVD, including angina pectoris (I20), myocardial infarction (I21 – 22), other acute ischemic heart diseases (I24), chronic ischaemic heart disease (I25), pulmonary embolism (I26), cardiac arrest (I46), cardiac arrhythmias (I48 – 48), and heart failure (I50). **Age Groups Analyzed:** Age > 65 **Study Design:** Time series N: 2192 days, 9 Hospitals **Statistical Analyses:** Principal Component Analysis and Constrained Physical Receptor Model (COPREM), Poisson regression, GAM, **Covariates:** Season, day of the wk, public holidays, influenza epidemics and meteorology **Season:** All year **Dose-response Investigated?** No **Statistical package:** R, gam/mgcv package **Lags Considered:** 0-6 days	**Pollutant:** Source specific PM_{10} components **Averaging Time:** 24-h **Mean (SD): Percentiles:** 25th: 16 50th (Median): NR 75th: 30 **Monitoring Stations:** 1 **Copollutant (correlation):** PM_{10}: Biomass r = 0.53 Secondary r = 0.73 Oil r = 0.57 Crustal r = 0.37 Sea salt r = 0.04 Vehicle r = 0.02 Notes: Correlations between source specific PM_{10} components presented in paper	**PM Increment:** IQR **RR Estimate** **Respiratory disease (age > 65)** Single pollutant model: PM_{10}: 1.027 (1.013, 1.042), IQR = 14 PM_{10} (other 5 sources): 1.045 (1.016, 1.074), IQR = 13 Biomass: 1.040 (0.009, 1.072), IQR = 5.4 Secondary: 1.050 (1.021, 1.081), IQR = 6.1 Oil: 1.035 (1.006, 1.065), IQR = 2.8 Crustal: 1.054 (1.028, 1.081), IQR = 1.8 Sea salt: 0.98 (0.947, 1.017), IQR = 2.2 Vehicle: 0.989 (0.949, 1.032), IQR = 0.6 **Notes:** 2 pollutant model results for PM_{10} with source specific components and gases also presented in manuscript.
Reference: Baccarelli et al. (2007, 091310) **Period of Study:** Jan 1995–Aug 2005 **Location:** Lombardia region, Italy	**Outcome (ICD9 and ICD10):** Fasting and postmethionine-load total homocysteine (tHcy) **Age Groups:** 11-84 yrs **Study Design:** Cross-sectional/Panel **N:** 1,213 participants **Statistical Analyses:** Generalized additive models **Covariates:** age, sex, BMI, smoking, alcohol, hormone use, temperature, day of the year, and long-term trends **Season:** Adjusted for long-term trends to account for season **Dose-response Investigated?** No **Statistical Package:** R software v2.2.1	**Pollutant:** PM_{10} (some TSP measures used to predict PM_{10}) **Averaging Time:** Hourly concentrations used to calculate 24-h moving averages and 7-day moving averages **Mean (SD):** NR **Percentiles:** 25th: 20.1 50th: 34.1 75th: 52.6 **Range (Min, Max):** Max: 390.0 **Monitoring Stations:** 53 sites **Copollutant:** CO, NO_2 SO_2 O_3	**PM Increment:** IQR **Effect Estimate [Lower CI, Upper CI]:** Estimates (%) per 32.5 $\mu g/m^3$ increase in 24-h moving avg of PM_{10} Homocysteine, fasting: 0.4 (-2.4, 3.3) Homocysteine, postmethionine-load: (-1.5, 3.7) Estimates (%) per 25.7m^3 increase in 7-day moving avg of PM_{10} Homocysteine, fasting: 1.0 (-1.9, 3.9) Homocysteine, postmethionine-load: 2.0 (-0.6, 4.7) Estimates of effect (%) on fasting homocysteine per IQR increase in 24-h PM_{10} levels Among smokers: 6.2 (0.0, 12.7) Among non-smokers: -1.6 (-5.5, 2.5) Estimates of effect (%) on postmethionine-load homocysteine per IQR increase in 24-h PM_{10} levels Among smokers: 6.0 (0.5, 11.8) Among non-smokers: -0.1 (-3.6, 3.5)

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Ballester et al. (2006, 088746) **Period of Study:** 1995 - 1999 **Location:** 5 Spanish cities: Granada, Huelva, Madrid, Seville, Zaragoza	**Outcome (ICD-9):** All cardiovascular disease (390–459), including all heart diseases (410–414, 427, 428) **Age Groups:** All ages **Study Design:** Time series **N:** NR **Statistical Analyses:** Poisson GAMs **Covariates:** daily temp, barometric pressure relative humidity daily influenza incidence, day of the week, holidays, unusual events (ex. medical strikes), seasonal variation, trend **Dose-response Investigated:** No **Statistical Package:** S-Plus GAM function **Lags Considered:** lag 0 -3 days, lag 0-1 avg	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (10-90th percentile):** overall mean NR. City specific means Granada: 43.2 (24.8, 62.6) Huelva: 38.6 (23.1, 57.3) Madrid: 35.7 (21.4, 54.4) Seville: 41.9 (27.3, 57.6) Zaragoza: 32.8 (17.3, 50.3) **Monitoring Stations:** At least three stations/city (15+) **Copollutant (correlation):** Summary of the correlation coefficients between each pair of pollutants within cities: BS: r = 0.48 TSP: N/A NO_2: from r = 0.13 to r = 0.62 (median r = 0.40) SO_2: from r = 0.20 to r = 0.51 (median r = 0.46) CO: from r = 0.34 to r = 0.45 (median r = 0.37) O_3: from r = -0.07 to r = 0.16 (median r = 0.11)	**PM Increment:** 10 $\mu g/m^3$ **Relative risk [CI]:** Relative risks are expressed only in the form of figures (see notes). **Percentage change in risk [CI]:** All cardiovascular diseases (avg of lags 0 -1): 0.91% [0.35, 1.47] Heart disease (avg of lags 0 -1) 1.56% [0.82, 2.31] **Notes: Relative risks for the single pollutant models are expressed in Figure 2.** Figure 2: Time sequence of the combined association between PM_{10} and hospital admissions for all CVD (A) and heart disease (B). Summary of results: Significant, positive association of PM_{10} with both overall CVD and heart disease hospitalizations at Lag 0 and Lag 1. **Relative risks for two pollutant models are expressed in Figure 3:** Figure 3: Combined estimates of the association between hospital admissions for heart diseases and air pollutants (avg of lags 0–1 adjusted for CO, NO_2, O_3, or SO_2) Summary of results: Significant, positive association remains after adjusting for pollutants.

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Bell et al. (2008, 091268) **Period of Study:** 1995 - 2002 **Location:** Taipei, Taiwan	**Outcome (ICD-9):** Hospital admissions for ischemic heart disease (410 , 411, 414), cerebrovascular disease (430–437). **Age Groups:** All **Study Design:** Time series **N:** 6,909 hospital admissions for ischaemic heart diseases, 11,466 for cerebrovascular disease. **Statistical Analyses:** Poisson regression **Covariates:** Day of the week, time, apparent temperature, long-term trends, seasonality **Season:** All **Dose-response Investigated:** No **Statistical Package:** NR **Lags Considered:** lags 0-3 days, avg of lags 0-3	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (range** **IQR):** 49.1 (12.7–215.5 27.6) **Monitoring Stations:** Taipei area: 13 monitors Taipei City: 5 monitors Monitors with correlations of 0.75 + for PM_{10}: 12 monitors **Copollutant:** NR	**PM Increment:** 28 $\mu g/m^3$ (near IQR) **Percentage increase estimate [95% CI]: Ischemic heart disease:** Taipei area (13 monitors): L0: 1.91 (-1.25, 5.17) L1: 0.39 (-2.73, 3.61) L2: 1.80 (-1.33, 5.04) L3: 2.01 (-1.14, 5.26) L03: 2.91 (-1.52, 7.55) Taipei City (5 monitors): L0: 2.08 (-1.04, 5.30) L1: 0.43 (-2.64, 3.60) L2: 2.17 (-0.92, 5.36) L3: 2.16 (-0.94, 5.36) L03: 3.40 (-1.19, 8.20) Monitors with > = 0.75 between monitor correlations (12 monitors): L0: 1.82 (-1.29, 5.03) L1: 0.35 (-2.72, 3.52) L2: 1.93 (-1.15, 5.10) L3: 1.93 (-1.16, 5.12) L03: 2.86 (-1.63, 7.54) **Cerebrovascular disease:** Taipei area (13 monitors): L0: -1.41 (-3.80, 1.04) L1: -1.95 (4.31, 0.48) L2: 0.77 (-1.62, 3.23) L3: 2.64 (0.21, 5.12) L03: 0.01 (-3.33, 3.47) Taipei City (5 monitors): L0: -1.27 (-3.64, 1.16) L1: -2.13 (-4.47, 0.27) L2: 0.85 (-1.52, 3.28) L3: 2.52 (0.13, 4.97) L03: -0.07 (-3.53, 3.51) Monitors with > = 0.75 between monitor correlations (12 monitors): L0: -1.34 (-3.70, 1.07) L1: -1.98 (-4.31, 0.40) L2: 0.80 (-1.56, 3.22) L3: 2.61 (0.22, 5.05) L03: -0.02 (-3.40, 3.49)

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Chan et al. (2007, 147787) **Period of Study:** Apr 1997 – Dec 2002 **Location:** Boston, MA	**Outcome:** Cerebrovascular Emergency Admissions **Age Groups:** 50+ yrs **Study Design:** time series **Statistical Analyses:** GAM Poisosn Regression **Covariates:** year, month, day of week, temperature, dew point **Dose-response Investigated?** No **Statistical Package:** NR **Lags Considered:** 0-3d	**Pollutant:** PM_{10} Averaging Time: 24h **Mean (SD):** 50.2 (22.1) **Min:** 16.0 **Max:** 325.4 **IQR:** 25.4 **Monitoring Stations:** 16 **Copollutant:** O_3, CO, SO_2, NO_2, $PM_{2.5}$ **Co-pollutant Correlation** O_3: 0.43 CO: 0.47 SO_2: 0.59 NO_2: 0.64 $PM_{2.5}$: 0.61	**PM Increment:** Interquartile Range (25.4 $\mu g/m^3$) **Percent Change (Lower CI, Upper CI), p-value:** Cerebrovascular Disease Lag 0: 1.001 (0.969, 1.033) Lag 1: 0.999 (0.9787, 1.020) Lag 2: 1.023 (0.989, 1.057) Lag 3: 1.030 (1.011, 1.049) Lag 3 + O_3: 1.018 (0.987, 1.049) Lag 3 + CO: 1.019 (0.988, 1.050) Lag 3 + O_3 + CO: 1.015 (0.985, 1.045) Stroke Lag 0: 0.969 (0.897, 1.041) Lag 1: 0.992 (0.918, 1.066) Lag 2: 1.004 (0.993, 1.015) Lag 3: 1.009 (0.988, 1.030) Ischaemic stroke Lag 0: 0.984 (0.932, 1.036) Lag 1: 0.993 (0.939, 1.047) Lag 2: 0.989 (0.927, 1.041) Lag 3: 1.042 (0.981, 1.103) Haemorrhagic stroke Lag 0: 0.966 (0.884, 1.048) Lag 1: 0.990 (0.908, 1.072) Lag 2: 1.002 (0.920, 1.084) Lag 3: 0.974 (0.902, 1.046)
Reference: Chan et al. (2008, 093297) **Period of Study:** 1995 - 2002 **Location:** Taipei Metropolitan area, Taiwan	**Outcome (ICD-9):** Emergency visits for ischaemic heart diseases (410–411, 414), cerebrovascular diseases (430–437), and COPD (493, 496) **Age Groups:** All **Study Design:** Time series **N:** NR **Statistical Analyses:** Poisson regression models **Covariates:** Year, month, day of week, temperature, dew point temperature, $PM_{2.5}$, NO_2 **Season:** All **Dose-response Investigated:** No **Statistical Package:** SAS version 8.0 **Lags Considered:** 0- to 7-day lags	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (SD):** High dust events: Pre-dust periods: 45.5 (17.6) Asian dust events: 122.7 (24.4) Low dust events: Pre-dust periods: 59.4 (31.0) Asian dust events: 61.1 (17.8) **Monitoring Stations:** 1 **Copollutant:** NR	**PM Increment:** 25.4 $\mu g/m^3$ (IQR) **OR [95% CI]:** In environmental conditions without dust storms (results only shown for best-fitting model) Lag 3 days: 1.023 (1.003, 1.041)

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Chang et al. (2007, 147621) **Period of Study:** 1997-2001 **Location:** Taipei, Taiwan	**Outcome:** CVD HA **Age Groups:** NR **Study Design:** case-crossover **Statistical Analyses:** Conditional Logistic Regression **Covariates:** temperature, humidity **Dose-response Investigated?** No **Statistical Package:** SAS **Lags Considered:** 0-2d	**Pollutant:** PM_{10} Averaging Time: 24h **Mean:** 48.32 **Min:** 14.44 **25th:** 32.65 **50th:** 42.80 **75th:** 57.16 **Max:** 234.91 **Monitoring Stations:** 6 **Copollutant:** O_3, CO, SO_2, NO_2 **Co-pollutant Correlation** NR	**PM Increment:** Interquartile Range (24.51 $\mu g/m^3$) **Odds Ratio (Lower CI, Upper CI):** ≥20°C PM_{10}: 1.085 (1.061, 1.110) PM_{10} + SO_2: 1.131 (1.103, 1.161) PM_{10} + NO_2: 10.977 (0.950, 1.006) PM_{10} + CO: 1.025 (0.999, 1.052) PM_{10} + O_3: 1.064 (1.039, 1.090) < 20°C PM_{10}: 1.142 (1.105, 1.180) PM_{10} + SO_2: 1.235 (1.184, 1.288) PM_{10} + NO_2: 1.148 (1.103, 1.194) PM_{10} + CO: 1.165 (1.121, 1.212) PM_{10} + O_3: 1.142 (1.105, 1.180)
Reference: D'Ippoliti et al. (2003, 074311) **Period of Study:** Jan 1995 – Jun 1997 **Location:** Rome, Italy	**Outcome:** Myocardial Infarction HA **Age Groups:** 18+ yrs **Study Design:** case-crossover **Statistical Analyses:** Conditional Logistic Regression **Covariates:** temperature, humidity **Dose-response Investigated?** No **Statistical Package:** NR **Lags Considered:** 0-4d	**Pollutant:** TSP **Averaging Time:** 24h **Mean (SD):** 66.9 (19.7) **25th:** 54.7 **50th:** 66.4 **75th:** 78.4 **IQR:** 23.7 **Monitoring Stations:** 3 **Copollutant:** CO, SO_2, NO_2 **Co-pollutant Correlation** CO: 0.35 SO_2: 0.29 NO_2: 0.38	**PM Increment:** Quartiles **Odds Ratio (Lower CI, Upper CI):** Lag 0-2d avg QI: 1.0 (ref) QII: 1.048 (0.957, 1.148) QIII: 1.105 (1.007, 1.214) QIV: 1.132 (1.023, 1.253) Various Lags Lag 0: 1.023 (1.004, 1.042) Lag 1: 1.015 (0.996, 1.034) Lag 2: 1.017 (0.999, 1.035) Lag 3: 0.989 (0.974, 1.003) Lag 4: 1.001 (0.987, 1.016)
Reference: Fung et al., (2005, 093262) **Period of Study:** Nov 1, 1995–Dec 31, 2000 **Location:** London, Ontario	**Outcome (ICD-9):** Cardiovascular diseases (410-414, 427-428) **Age Groups:** < 65 yrs, 65+ yrs **Study Design:** Time series **N:** 12,947 CVD admissions **Statistical Analyses:** GAM with locally weighted regression smoothers (LOESS) **Covariates:** Maximum and minimum temp, humidity, day of the week, seasonal cycles, secular trends **Season:** NR **Dose-response Investigated?** No **Statistical Package:** S-Plus **Lags Considered:** Current to 3-day mean	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (min-max):** 38.0 (5-248) SD = 23.5 **Monitoring Stations:** 4 **Copollutant (correlation):** NO_2: r = 0.30 SO_2: r = 0.24 CO: r = 0.21 O_3: r = 0.53 COH: r = 0.29	**PM Increment:** 26 $\mu g/m^3$ **% Change in Daily Admission [CI]:** Age < 65 Current day mean: 2.6 [-2.3,7.7] 2-day mean: -1.2 [-7.2,5.1] 3-day mean: -3 [-9.6,4] Age 65+ Current day mean: 0.9 [-2.3,4.2] 2-day mean: -0.9 [-4.8,3.2] 3-day mean: -0.1 [-4.4,4.5]

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Hanigan et al (2008, 156518) **Period of Study:** 1996–2005 (April–November of each year) **Location:** Darwin, Australia	**Outcome:** Daily emergency hospital admissions for total cardiovascular (ICD-9: 390–459 ICD-10: I00–I99), ischemic heart disease (ICD-9: 410–414 ICD-10: I20–I25). **Age Groups:** All **Study Design:** Time series **N:** 8,279 hospital admissions **Statistical Analyses:** Poisson generalized linear models **Covariates:** Indigenous status, time in days, temperature, relative humidity, day of the week, influenza epidemics, change between ICD editions, holidays, yearly population **Season:** April–November (corresponding to the dry season) **Dose-response Investigated?** No **Statistical Package:** R version 2.3.1 **Lags Considered:** 0-3	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (SD range):** 21.2 (8.2 55.2) **Monitoring Stations:** N/A (see notes) **Copollutant:** NR	**PM Increment:** 10 $\mu g/m^3$ **Percent change [95% CI]:** Overall CVD: Lag 0 (indigenous): -3.78 [-13.4, 6.91] Lag 0 (non-indigenous): -3.43 [-9.00, 2.49] All unstratified associations either negative or zero and not statistically significant. All other results of stratified analysis (by indigenous status) reported in a figure (see notes). **Notes:** Figure 3: Associations between hospitalizations for non-indigenous and indigenous people with estimated ambient PM_{10}. Summary: Confidence intervals were wide, but indigenous people generally had stronger associations with PM_{10} than non-indigenous people. Daily PM_{10} exposure levels were estimated for the population of the city from visibility data using a previously validated models.
Reference: Hanigan et al (2008, 156518) **Period of Study:** 1996–2005 (April–November of each year) **Location:** Darwin, Australia	**Outcome:** Cardiorespiratory Disease HA (ICD 9: 390-519 ICD 10: I00-99 & J00-99) **Age Groups:** NR **Study Design:** time series **N:** 8279 events **Statistical Analyses:** poisson regression **Covariates:** indigenous status, time in days, temperature, relative humidity, day of the week, influenza epidemics, change between ICD editions, holidays, yearly population **Dose-response Investigated?** No **Statistical Package:** R **Lags Considered:** lags 0-3	**Pollutant:** PM_{10} **Averaging Time:** 24h **Mean (SD):** 21.2 (8.2) **Range:** 55.2 **Monitoring Stations:** 2 (monitored & modeled) **Copollutant:** NR **Co-pollutant Correlation** n/a	**PM Increment:** 10 $\mu g/m^3$ **Percent Change (Lower CI, Upper CI), lag:** Tot. Cardiovascular, Indigenous: -3.43 (-9.00, 2.49), lag 0 Tot Cardiovascular, Non-Indigenous: -3.78 (-13.4, 6.91), lag 0 *figure 3. percent change in hospital admissions per 10$\mu g/m^3$ increase in PM_{10}
Reference: Henrotin et al. (2007, 093270) **Period of Study:** March 1994–December 2004 **Location:** Dijon, France	**Outcome:** Ischemic and hemorrhagic strokes **Age Groups:** All **Study Design:** Bi-directional case-crossover **N:** 1487 (ischemic) and 220 (hemorrhagic) stroke patients **Statistical Analyses:** Conditional logistic regression **Covariates:** Temperature, relative humidity, influenza epidemics, holidays **Season:** NR **Dose-response Investigated?** Yes **Statistical Package:** STATA software v. 8.2 **Lags Considered:** 0-3 days	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (min-max):** 21.1 (2-103) SD = 11.3 **Monitoring Stations:** 1 **Copollutant:** NR	**PM Increment:** 10 $\mu g/m^3$ **OR Estimate [CI]:** Ischemic stroke Same-day lag: 1.009 [0.930,1.094] 1-day lag: 1.011 [0.998,1.094] 2-day lag: 0.960 [0.889,1.036] 3-day lag: 0.990 [0.919,1.066] Hemorrhagic stroke Same-day lag: 0.901 [0.730,1.111] 1-day lag: 1.014 [0.828,1.241] 2-day lag: 1.100 [0.903,1.339] 3-day lag: 0.991 [0.881,1.212] **Notes:** Ischemic stroke ORs were also categorized into male and female, yielding similar results (none were significant for any lag days).

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Issever et al. (2005, 097736) **Period of Study:** 1 Jan, 1997–31 Dec, 2001 **Location:** Istanbul, Turkey	**Outcome:** Acute coronary syndrome (ACS) **Age Groups:** All **Study Design:** Time series **N:** 2889 ACS admissions **Statistical Analyses:** Multiple stepwise regression, Pearson correlation **Covariates:** Humidity, temperature, pressure **Season:** NR **Dose-response Investigated?** No	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean:** NR **Monitoring Stations:** 1 **Copollutant (correlation):** ACS: r = 0.37 (p = 0.003) ACS controlled for temp: r = 0.29 (p = 0.02)	**PM Increment:** NR **RR Estimate [CI]:** NR **Notes:** This study focused more on the seasonal change in acute coronary syndrome admissions.
Reference: Jalaludin et al. (2006, 189416) **Period of Study:** 1 Jan, 1997–31 Dec, 2001 **Location:** Sydney, Australia	**Outcome (ICD-9):** Cardiovascular disease (390-459), cardiac disease (390-429), ischemic heart disease (410-413) and cerebrovascular disease or stroke (430-438) **Age Groups:** 65+ yrs **Study Design:** Time series **N:** NR **Statistical Analyses:** GAM, GLM **Covariates:** Temperature, humidity **Season:** Warm (Nov-Apr) and cool (May-Oct) **Dose-response Investigated?** No **Statistical Package:** S-Plus **Lags Considered:** 0-3	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (min-max):** 16.8 (3.8-103.9) SD = 7.2 **Monitoring Stations:** 14 **Copollutant (correlation):** Warm BSP: r = 0.82 $PM_{2.5}$: r = 0.89 O_3: r = 0.59 NO_2: r = 0.44; CO: r = 0.31 SO_2: r = 0.37 Cool BSP: r = 0.75 $PM_{2.5}$: r = 0.88 O_3: r = 0.22 NO_2: r = 0.67 CO: r = 0.48 SO_2: r = 0.46 **Other variables:** Warm Temp: r = 0.36 Rel humidity: r = -0.25 Cool Temp: r = 0.13 Rel humidity: r = 0.05	**PM Increment:** 7.8 $\mu g/m^3$ (IQR) **Percent Change Estimate [CI]:** All CVD Same-day lag: 0.72 [-0.14,1.60] Avg 0-1 day lag: 0.25 [-0.61,1.12] Cool (same-day lag): 1.34 [0.08,2.61] Warm (same-day lag): 0.33 [-0.83,1.50] Cardiac disease Same-day lag: 1.15 [0.14,2.18] Avg 0-1 day lag: 0.97 [-0.07,2.02] Cool (same-day lag): 1.35 [-0.16,2.89] Warm (same-day lag): 1.12 [-0.23,2.48] Ischemic heart disease Same-day lag: 0.59 [-0.95,2.17] Avg 0-1 day lag: 0.61 [-0.95,2.20] Cool (same-day lag): 0.33 [-2.00,2.72] Warm (same-day lag): 0.79 [-1.23,2.85] Stroke Same-day lag: -1.66 [-3.48,0.20] Avg 0-1 day lag: -2.05 [-3.88,-0.20] Cool (same-day lag): 0.46 [-2.17,3.17] Warm (same-day lag): -3.49 [-5.97,-0.95] **Notes:** All other lag-day ORs were provided, yet none were significant. Percent change in ED attendance was also reported graphically (Fig 1-5).

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Johnston et al. (2007, 155882) **Period of Study:** 2000, 2004, 2005 (April–November of each year) **Location:** Darwin, Australia	**Outcome (ICD-10):** All cardiovascular conditions (I00–I99), including ischemic heart disease (I20–I25). **Age Groups:** All **Study Design:** Case-crossover **N:** 2466 emergency admissions **Statistical Analyses:** Conditional logistic regression **Covariates:** Weekly influenza rates, temperature, humidity, days with rainfall > 5mm, public holidays, school holiday periods (for respiratory conditions only) **Season:** April–November (dry season) **Dose-response Investigated?** No **Statistical Package:** NR **Lags Considered:** 0–3	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Median (IQR, 10th–90th percentile, range):** 17.4 (13.6–22.3 10.3–27.7 1.1–70.0) **Monitoring Stations:** 1 **Copollutant:** NR	**PM Increment:** 10 $\mu g/m^3$ **OR Estimate [95% CI]: All respiratory conditions: Ischemic heart disease:** Lag 0: 0.82 [0.68–0.98] Lag 0 (non-indigenous): 0.75 [0.61–0.93] Lag 3 (indigenous): 1.71 [1.14–2.55] **Notes:** **Figure 5:** OR and 95% CI for hospital admissions for cardiovascular conditions. Summary: Negative associations in overall study population and in non-indigenous people. Positive associations in Indigenous people at Lag 1, Lag 2, and Lag 3. **Figure 6:** OR and 95% CI for hospital admissions for ischaemic heart disease. Summary: Negative associations in overall study population and non-indigenous people. Positive association in indigenous people.
Reference: Koken et al. (2003, 049466) **Period of Study:** July and August, 1993-1997 **Location:** Denver, Colorado	**Outcome (ICD-9):** Acute myocardial infarction (410.00-410.92), pulmonary heart disease (416.0-416.9), cardiac dysrhythmias (427.0-427.9), congestive heart failure (428.0) **Age Groups:** 65+ yrs **Study Design:** Time series **N:** 298 days **Statistical Analyses:** GLM, GEE **Covariates:** Maximum temp and dew point temp **Season:** NR **Dose-response Investigated:** Yes **Statistical Package:** SAS (PROC GENMOD) **Lags Considered:** 0-4 days	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (min-max):** 24.2 (7.0-51.6) SD = 6.25 **Monitoring Stations:** 3 **Copollutant (correlation):** NO_2: r = 0.56 SO_2: r = 0.36 O_3: r = 0.03 CO: r = 0.25 **Other variables:** Max temp: r = 0.38 Dew point temp: r = -0.24	**PM Increment:** 8.0 $\mu g/m^3$ (IQR) **Percent Change Estimate [CI]:** No PM data reported

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Lanki et al., (2006, 089788) **Period of Study:** 1992-2000 **Location:** Augsburg, Barcelona, Helsinki, Rome, and Stockholm	**Outcome (ICD-9):** Acute myocardial infarction (410 ICD-10: I21, I22) **Age Groups:** 35+ yrs, <75 yrs, 75+ yrs **Study Design:** Time series **N:** 26,854 hospitalizations **Statistical Analyses:** GAM **Covariates:** Temperature, barometric pressure **Season:** Warm (April-September) and cold (October-March) **Dose-response Investigated:** No **Statistical Package:** R package mgcv 0.9-5 **Lags Considered:** 0-3 days	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Median:** Augsburg: 43.5 Barcelona: 57.4 Helsinki: 21.0 Rome: 48.5 Stockholm: 12.5 **Copollutant (correlation):** Augsburg PNC: r = 0.53 CO: r = 0.56 NO_2: r = 0.64 O_3: r = 0.43 Barcelona: PNC: r = 0.38 CO: r = 0.44 NO_2: r = 0.48 O_3: r = 0.01 Helsinki: PNC: r = 0.45 CO: r = 0.21 NO_2: r = 0.40 O_3: r = 0.40 Rome: PNC: r = 0.32 CO: r = 0.41 NO_2: r = 0.29 O_3: r = 0.59 Stockholm: PNC: r = 0.06 CO: r = 0.41 NO_2: r = 0.29 O_3: r = 0.59	**PM Increment:** 10 $\mu g/m^3$ **Pooled Rate Ratio [CI]:** All 5 cities (35+ yrs) Same-day lag: 1.003 [0.995,1.011] 1-day lag: 1.001 [0.990,1.011] 2-day lag: 1.002 [0.994,1.010] 3-day lag: 1.002 [0.991,1.013] 3 cities with hospital discharge register (35+ yrs) Same-day lag: 1.003 [0.994,1.012] 1-day lag: 0.997 [0.988,1.006] 2-day lag: 1.003 [0.995,1.012] 3-day lag: 1.003 [0.986,1.020] Warm season (35+ yrs) Same-day lag: 1.006 [0.990,1.022] 1-day lag: 1.000 [0.985,1.016] 2-day lag: 1.005 [0.990,1.020] 3-day lag: 1.010 [0.995,1.025] Cold season (35+ yrs) Same-day lag: 1.001 [0.991,1.012] 1-day lag: 0.998 [0.987,1.009] 2-day lag: 1.001 [0.991,1.012] 3-day lag: 0.991 [0.981,1.002] Age >75 Non-fatal Same-day lag: 1.012 [0.995,1.029] 1-day lag: 1.000 [0.983,1.017] 2-day lag: 0.999 [0.982,1.017] 3-day lag: 1.001 [0.984,1.018] Fatal Same-day lag: 1.009 [0.985,1.034] 1-day lag: 0.998 [0.974,1.023] 2-day lag: 1.003 [0.978,1.028] 3-day lag: 1.018 [0.975,1.063] **Notes:** Pooled rate ratios were also provided for groups <75 yielding similar results to the overall 3-city data.

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Lee et al. (2003, 095552) **Period of Study:** 1 Dec, 1997–31 Dec, 1999 **Location:** Seoul, Korea	**Outcome (ICD-10):** Angina pectoris (I20), acute/subsequent myocardial infarction (I21-I23), other acute ischemic heart diseases (I24) **Age Groups:** All ages, 64+ yrs **Study Design:** Time series **N:** 822 days **Statistical Analyses:** GAM with LOESS, Pearson correlation **Covariates:** Temperature, relative humidity, day of the week **Season:** Summer (Jun-Aug) and winter **Dose-response Investigated:** Yes **Statistical Package:** NR **Lags Considered:** 0-6 days	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (SD):** 64.0 (31.8) **Monitoring Stations:** 27 **Copollutant (correlation):** All year SO_2: r = 0.59 NO_2: r = 0.74 O_3: r = 0.11 CO: r = 0.60 Temp: r = -0.07 Humidity: r = 0.02 Summer SO_2: r = 0.61 NO_2: r = 0.73 O_3: r = 0.64 CO: r = 0.55 Temp: r = -0.01 Humidity: r = -0.11	**PM Increment:** 40.4 $\mu g/m^3$ (IQR) **RR Estimate [CI]: All year** All ages: 0.99 [0.96,1.01] 64+ yrs: 1.05 [1.01,1.10] **Summer** All ages: 1.03 [0.97,1.09] 64+ yrs: 1.09 [1.00,1.19] **Two-pollutant model** CO (1 ppm IQI): 1.04 [0.98,1.11] O_3 (21.7 ppb IQI): 1.07 [1.03,1.11] NO_2 (14.6 ppb IQI): 1.09 [1.02,1.16] SO_2 (4.4 ppb): 0.98 [0.94,1.03]
Reference: Lee et al. (2008, 192076) **Period of Study:** 1996-2005 **Location:** Taipei, Taiwan	**Outcome:** Congestive Heart Failure HA (ICD 9: 428) **Age Groups:** NR **Study Design:** case-crossover **N:** 18593 events **Statistical Analyses:** conditional logistic regression **Covariates:** temperature, humidity **Dose-response Investigated?** No **Statistical Package:** SAS **Lags Considered:** lags 0-2	**Pollutant:** PM_{10} **Averaging Time:** 24h **Mean:** 49.94 **Min:** 11.33 **25th:** 33.37 **50th:** 45.05 **75th:** 60.82 **Max:** 234.92 **Monitoring Stations:** 6 **Copollutant:** SO_2, CO, NO_2, O_3 **Co-pollutant Correlation** SO_2: 0.52 CO: 0.67 NO_2: 0.35 O_3: 0.39	**PM Increment:** Interquartile Range (27.45 $\mu g/m^3$) **Odds Ratio (Lower CI, Upper CI):** W/ Hypertension: 1.23 (1.15, 1.32) W/o Hypertension: 1.20 (1.15, 1.25) W/ Diabetes: 1.20 (1.12, 1.40) W/o Diabetes: 1.21 (1.15, 1.26) W/ Dysrhythmia: 1.17 (1.08, 1.27) W/o Dysrhythmia: 1.22 (1.17, 1.27) W/ COPD: 1.21 (1.07, 1.36) W/o COPD: 1.21 (1.16, 1.25)

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Larrieu et al. (2007, 093031) **Period of Study:** 1998 - 2003 **Location:** 8 French urban area: Bordeaux, Le Havre, Lille, Lyon, Marseille, Paris, Rouen, and Toulouse	**Outcome (ICD-10):** Hospital admissions for cardiovascular disease (I00–I99), cardiac disease (I00–I52), ischemic heart disease (I20–I25), and stroke (cerebrovascular disease: I60–64 and transient ischemic attack: G45–G46). **Age Groups:** All, and 65 + **Study Design:** Time series **N: Statistical Analyses:** generalized additive Poisson regression **Covariates:** Temperature, holidays, influenza epidemic periods, long-term trend, season, day of the week, **Season:** NR **Dose-response Investigated:** No **Statistical Package:** R 2.2.1 **Lags Considered:** 0 -1 day lag (mean)	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean:** Bordeaux: 21.0 Le Havre: 21.7 Lille: 22.1 Lyon: 24.6 Marseille: 28.9 Paris: 23.1 Rouen: 21.2 Toulouse: 21.8 **Monitoring Stations:** 32 **Copollutant:** NR	**PM Increment:** 10 $\mu g/m^3$ **ERR [95% CI]:** CVD: All ages: 0.7 [0.1, 1.2] 65+ years: 1.1 [0.5, 1.7] Cardiac diseases: All ages: 0.8 [0.2, 1.4] 65+ years: 1.5 [0.7, 2.2] Ischemic heart diseases: All ages: 1.9 [0.8, 3.0] 65+ years: 2.9 [1.5, 4.3] Strokes: All ages: 0.2 [-1.6, 1.9] 65+ years: 0.8 [-0.9, 2.5]
Reference: Le Tertre et al. (2002, 023746) **Period of Study:** 1990-1997 **Location:** Barcelona, Birmingham, London, Milan, the Netherlands, Paris, Rome, and Stockholm	**Outcome (ICD-9):** Cardiac diseases (390-429), ischemic heart disease (410-413), and stroke (430-438) **Age Groups:** < 65 yrs, 65+ yrs **Study Design:** Time series **N:** NR **Statistical Analyses:** GAM **Covariates:** Long term trend, season, days of the week, holidays, influenza epidemics, temperature, and humidity **Season:** NR **Dose-response Investigated:** No **Statistical Package:** S-Plus **Lags Considered:** 0-3 days	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (SD):** Barcelona: 55.7 (18.4) Birmingham: 24.8 (13.1) London: 28.4 (12.3) Milan: 51.5 (22.7) Netherlands: 39.5 (19.9) Paris: 22.7 (10.8) Rome: 52.5 (12.9) Stockholm: 15.5 (7.2) **Monitoring Stations:** 1-12 **Copollutant:** NR	**PM Increment:** 10 $\mu g/m^3$ **Pooled Percent Increase [CI]:** Cardiac (all ages) Fixed: 0.5 [0.3,0.7] Random: 0.5 [0.2,0.8] Cardiac (over 65) Fixed: 0.7 [0.4,1.0] Random: 0.7 [0.4,1.0] IHD (< 65) Fixed: 0.3 [-0.1,0.6] Random: 0.3 [-0.2,0.7] IHD (over 65) Fixed: 0.6 [0.3,0.8]; Random: 0.8 [0.3,1.2] Stroke (over 65) Fixed: 0.0 [-0.3,0.3]; Random: 0.0 [-0.3,0.3] Deaths: Cardiac: 0.5 [0.2,0.8]; Cardiac (65+): 0.7 [0.4,1.0] IHD (65+): 0.8 [0.3,1.2] **Notes:** Estimated percentage increases are also provided by city for cardiac admissions and ischemic heart disease in Fig 1-3.

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Mann et al. (2002, 036723) **Period of Study:** 1988-1995 **Location:** South Coast Air Basin, California	**Outcome (ICD-9):** Ischemic heart disease (410-414), secondary congestive heart failure (sCHF) (428), and secondary arrhythmia (sARR) (426, 427) **Age Groups:** All, 40-59 yrs, >60 yrs **Study Design:** Time series **N:** 54,863 IHD admissions **Statistical Analyses:** GAM **Covariates:** Temperature, day of the week, relative humidity **Season:** NR **Dose-response Investigated:** No **Statistical Package:** S-Plus **Lags Considered:** 0-5 days	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (min-max):** 43.7 (0.22-251) SD = 27.7 **Monitoring Stations:** 20 **Copollutant (correlation):** Region 1: CO: r = 0.28 O_3: r = 0.20 NO_2: r = 0.36 Region 2: CO: r = 0.15 O_3: r = 0.57 NO_2: r = 0.53 Region 3: CO: r = 0.36 O_3: r = 0.30 NO_2: r = 0.46 Region 4: CO: r = 0.27 O_3: r = 0.33 NO_2: r = 0.50 Region 5: CO: r = 0.40 O_3: r = 0.43 NO_2: r = 0.53 Region 6: CO: r = 0.33 O_3: r = 0.20 NO_2: r = 0.42 Region 7: CO: r = 0.28 O_3: r = 0.48 NO_2: r = 0.60	**PM Increment:** 10 $\mu g/m^3$ **Percent Change in IHD Admissions [CI]:** Secondary ARR Same-day lag: 0.59 [-0.71,1.91] 1-day lag: 0.46 [-0.86,1.80] 2-day lag: -0.04 [-1.37,1.31] Secondary CHF Same-day lag: -0.62 [-1.77,0.55] 1-day lag: -0.45 [-1.60,0.71] 2-day lag: -0.36 [-1.52,0.82] No secondary diagnosis Same-day lag: -0.25 [-1.23,0.75] 1-day lag: 0.04 [-0.97,1.06] 2-day lag: 0.18 [-0.82,1.20] All IHD admissions: 0.19 [-0.576,0.955] MI admissions: -0.10 [-1.33,1.12] Other acute IHD admissions: 0.36 [-0.87,1.60]

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Metzger et al. (2004, 044222) **Period of Study:** August 1993–August 2000 **Location:** Atlanta Metropolitan area (Georgia)	**Outcome (ICD-9):** Emergency visits for ischemic heart disease (410–414), cardiac dysrhythmias (427), cardiac arrest (427.5), congestive heart failure (428), peripheral vascular and cerebrovascular disease (433-437, 440, 443-444, 451–453), atherosclerosis (440), and stroke (436). **Age Groups:** All **Study Design:** Time series **N:** 4,407,535 emergency department visits **Statistical Analyses:** Poisson generalized linear modeling **Covariates:** Day of the week, hospital entry and exit indicator variables, federally observed holidays, temporal trends, temperature, dew point temperature **Season:** All **Dose-response Investigated:** No **Statistical Package:** SAS **Lags Considered:** 3-day moving avg, lags 0 -7	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Median (10% - 90% range):** 26.3 (13.2, 44.7) **Monitoring Stations:** NR **Copollutant (correlation):** O_3: r = 0.59 NO_2: r = 0.49 CO: r = 0.47 SO_2: r = 0.20 $PM_{2.5}$: r = 0.84 $PM_{10-2.5}$: r = 0.59 UFP: r = -0.13 $PM_{2.5}$ water-sol metals: r = 0.74 $PM_{2.5}$ sulfates: r = 0.74 $PM_{2.5}$ acidity: r = 0.68 $PM_{2.5}$ organic carbon: r = 0.69 $PM_{2.5}$ elemental carbon: r = 0.56 oxygenated hydrocarbon: r = 0.58 **Other variables:** Temperature: r = 0.58 Dew point: r = 0.44	**PM Increment:** 10 $\mu g/m^3$ (approximately 1 SD) **RR [95% CI]:** For 3-day moving avg: All CVD: 1.009 [0.998, 1.019] Dysrhythmia: 1.008 [0.989, 1.029] Congestive heart failure: 0.992 [0.968–1.016] Ischemic heart disease: 1.011 [0.992–1.030] Peripheral vascular and cerebrovascular disease: 1.020 [0.999–1.043] **Notes:** Results for Lags 0–7 expressed in figures Figure 1: RR (95% CI) for single-day lag models for the association of ER visits for CVD with daily ambient PM_{10}. Summary: Statistically significant association at Lag 0. Positive but not statistically significant association at Lag 1. Negative, statistically significant association at Lag 7, and negative associations at Lag 2 through Lag 6.
Reference: Middleton et al. (2008, 156760) **Period of Study:** 1995–1998, 2000 - 2004 **Location:** Nicosia, Cyprus	**Outcome:** Hospital admissions for all cardiovascular disease (ICD-10: I00–I52). **Age Groups:** All, also stratified by age (< 15 vs. > 15 years) **Study Design:** Time series **Statistical Analyses:** Generalized additive Poisson models **Covariates:** Seasonality, day of the week, long- and short-term trend, temperature, relative humidity **Dose-response Investigated:** No **Statistical Package:** STATA SE 9.0, R 2.2.0 **Lags Considered:** Lag 0 -2 days	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (SD** **median** **5% - 95%** **range):** Cold: 57.6 (52.5 50.8 20.0–103.0 5.0–1370.6) Warm: 53.4 (50.5 30.7 32.0–77.6 18.4–933.5) **Monitoring Stations:** 2 **Copollutant:** NR	**PM Increment:** 10 $\mu g/m^3$, and across quartiles of increasing levels of PM_{10} **Percentage increase estimate [CI]: All age/sex groups (Lag 0):** All admissions: 0.85 (0.55, 1.15) Cardiovascular: 1.18 (-0.01, 2.37) **Nicosia residents (Lag 0):** Cardiovascular: 0.73 (-0.62, 2.09) **Males (Lag 0):** All admissions: 0.96 (0.54, 1.39) Cardiovascular: 1.27 (-0.15, 2.72) **Females (Lag 0):** All admissions: 0.74 (0.31, 1.18) Cardiovascular: 0.99 (-1.11, 3.14) **Aged < 15 years (Lag 0):** All admissions: 0.47 (-0.13, 1.08) **Aged > 15 years (Lag 0):** All admissions: 0.98 (0.63, 1.33)

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Peel et al. (2007, 090442) **Period of Study:** 1 Jan, 1993–31 Aug, 2000 **Location:** Atlanta, GA	**Outcome (ICD-9):** Ischemic heart disease (410-414), dysrhythmia (427), congestive heart failure (428), peripheral vascular and cerebrovascular disease (433-437, 440, 443, 444, 451-453) **Age Groups:** All **Study Design:** Case-crossover **N:** 4,407,535 ED visits **Statistical Analyses:** Conditional logistic regression **Covariates:** Avg temp and dew point temp **Season:** NR **Dose-response Investigated:** No **Statistical Package:** SAS v. 9.1 **Lags Considered:** 0-2 days	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (SD):** Daily levels: 27.9 (12.3) Diff in case and control day avgs: 9.1 (7.5) **Monitoring Stations:** 1 **Copollutant:** NR	**PM Increment:** 10 $\mu g/m^3$ **OR Estimate [CI]:** All CVD: 1.010 [1.000,1.020] IHD: 1.009 [0.991,1.027] Dysrhythmia: 1.011 [0.991, 1.031] Peripheral/Cerebrovascular disease: 1.017 [0.996,1.039] CHF: 1.001 [0.978,1.024] With comorbid hypertension IHD: 1.003 [0.973,1.034] Dysrhythmia: 1.037 [0.988,1.089] Peripheral/Cerebrovascular disease: 1.024 [0.990,1.060] CHF: 1.041 [0.999,1.084] No comorbid hypertension IHD: 1.013 [0.991,1.036] Dysrhythmia: 1.006 [0.985,1.028] Peripheral/Cerebrovascular disease: 1.013 [0.987,1.040] CHF: 0.982 [0.955,1.010] With comorbid diabetes IHD: 1.022 [0.979,1.067] Dysrhythmia: 1.049 [0.968,1.137] Peripheral/Cerebrovascular disease: 1.016 [0.965,1.069] CHF: 1.029 [0.982,1.078] No comorbid diabetes IHD: 1.006 [0.987,1.026] Dysrhythmia: 1.009 [0.989,1.029] Peripheral/Cerebrovascular disease: 1.018 [0.995,1.042] CHF: 0.992 [0.966,1.019] With comorbid COPD IHD: 0.981 [0.921,1.044] Dysrhythmia: 0.984 [0.889,1.088] Peripheral/Cerebrovascular disease: 1.086 [0.998,1.181] CHF: 1.010 [0.954,1.069] No comorbid COPD IHD: 1.012 [0.993,1.031] Dysrhythmia: 1.012 [0.992,1.032] Peripheral/Cerebrovascular disease: 1.013 [0.991,1.035] CHF: 0.999 [0.974,1.025]

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Pope et al., (2006, 091246) **Period of Study:** 1994 - 2004 **Location:** Wasatch Front area, Utah	**Outcome:** Myocardial infarction or unstable angina (ICD codes not reported) **Age Groups:** All **Study Design:** Case-crossover **N:** 12,865 patients who underwent coronary arteriography **Statistical Analyses:** Conditional logistic regression **Covariates:** Temperature and dew point temperature **Season:** NR **Dose-response Investigated:** No **Statistical Package:** NR **Lags Considered:** 0- to 3-day lag, 2- to 4-day lagged moving averages	**Pollutant:** PM_{10} Averaging Time: 24 h **Mean (SD** **maximum):** Ogden: 28.5 (16.5 163) SLC Hawthorne: 27.7 (17.4 162) Provo/Orem, Lindom: 32.7 (21.1 240) SLC AMC: 35.9 (20.4 161) SLC North: 45.1 (25.1 199) **Monitoring Stations:** 5 **Copollutant:** NR	**PM Increment:** 10 $\mu g/m^3$ **Percent increase in risk [95% CI]:** Results summarized in figure (see notes). **Notes:** Figure 1: Percent increase in risk (and 95% CI) of acute coronary events associated with 10 $\mu g/m^3$ of PM_{10} for different lag structures. Summary of Figure 1: Positive, statistically significant or marginally significant associations between association seen for Lag 0, Lag 1 and 2-, 3-, and 4-day moving averages. Non-statistically significant associations
Reference: Santos et al. (2008, 192004) **Period of Study:** Jan 1998 – Aug 1999 **Location:** Sao Paulo, Bazil	**Outcome:** Cardiac Arrhythmia ER Visits (ICD 10: I45-I49) **Age Groups:** 17+ yrs **Study Design:** time series **N:** 3251 ER visits **Statistical Analyses:** Poisson **Covariates:** temperature, humidity, seasonality **Dose-response Investigated?** Yes **Statistical Package:** S-Plus **Lags Considered:** lags 0-13	**Pollutant:** PM_{10} **Averaging Time:** 24h **Mean (SD):** 48.64 (20.34) **Min:** 18.68 **Max:** 137.76 **Monitoring Stations:** 14 **Copollutant:** SO_2, CO, NO_2, O_3 **Co-pollutant Correlation** SO_2: 0.675* CO: 0.580* NO_2: 0.781* O_3: 0.438* *$p < 0.01$	**PM Increment:** Interquartile Range (22.2 $\mu g/m^3$) **Percent Increase (Lower CI, Upper CI):** PM_{10} + NO_2,CO: -5.6 (-12.7, 2.1) PM_{10} + CO: -1.1 (-7.0, 5.1) PM_{10} + NO_2: -2.4 (-9.4, 5.1) Figure 1. PM_{10} effects, reported as percent increase, on arrhythmia ER visits caused by interquartile range increases, lags 0-6. Figure 2. Relative risks and 95% CI for arrhythmia ER visits according to the division of air pollutant daily concentrations in quintiles.

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Tolbert et al. (2007, 090316) **Period of Study:** 1993 - 2004 **Location:** Atlanta Metropolitan area, Georgia	**Outcome (ICD-9):** Combined CVD group, including: Ischemic heart disease (410–414), cardiac dysrhythmias (427), congestive heart failure (428), and peripheral vascular and cardiovascular disease (433–437, 440, 443–445, and 451–453). **Age Groups:** All **Study Design:** Time series **N:** 10,234,490 ER visits (283,360 and 1,072,429 visits included in the CVD and RD groups, respectively) **Statistical Analyses:** Poisson generalized linear models **Covariates:** Long-term temporal trends, season (for RD outcome), temperature, dew point, days of week, federal holidays, hospital entry and exit **Season:** All **Dose-response Investigated:** No **Statistical Package:** SAS version 9.1 **Lags Considered:** 3-day moving avg(lag 0 -2)	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (median** **IQR, range, 10th–90th percentiles):** 26.6 (24.8 17.5–33.8 0.5–98.4 12.3–42.8) **Monitoring Stations:** NR **Copollutant (correlation):** O_3: r = 0.59 NO_2: r = 0.53 CO: r = 0.51 SO_2: r = 0.21 Coarse PM: r = 0.67 $PM_{2.5}$: r = 0.84 $PM_{2.5}$ SO_4: r = 0.69 $PM_{2.5}$ EC: r = 0.61 $PM_{2.5}$ OC: r = 0.65 $PM_{2.5}$ TC: r = 0.67 $PM_{2.5}$ water-sol metals: r = 0.73 OHC: r = 0.53	**PM Increment:** 16.30 $\mu g/m^3$ (IQR) **Risk ratio [95% CI]:** Single pollutant models: CVD: 1.008 (0.997–1.020)
Reference: Tsai et al. (2003, 080133) **Period of Study:** 1997-2000 **Location:** Kaohsiung, Taiwan	**Outcome (ICD-9):** Cerebrovascular diseases (430-438), subarachnoid hemorrhagic stroke (430), primary intracerebral hemorrhage (431-432), ischemic stroke (433-435), and others (436-438) **Age Groups:** All **Study Design:** Case-crossover **N:** 23,179 admissions **Statistical Analyses:** Conditional logistic regression **Covariates:** Temperature and humidity **Season:** NR **Dose-response Investigated:** No **Statistical Package:** SAS **Lags Considered:** Cumulative 0-2 days	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (min-max):** 78.82 (20.50-217.33) **Monitoring Stations:** 6 **Copollutant:** NR	**PM Increment:** 66.33 $\mu g/m^3$ (IQR) **OR Estimate [CI]: Two-pollutant model (all stroke admissions)** Primary intracerebral hemorrhage (PIH) Adj for SO_2: 1.55 [1.31,1.83] Adj for NO_2: 1.28 [1.01,1.61]; Adj for CO: 1.45 [1.20,1.74] Adj for O_3: 1.56 [1.27,1.91] Ischemic stroke (IS) Adj for SO_2: 1.46 [1.32,1.61] Adj for NO_2: 1.16 [1.01,1.34] Adj for CO: 1.35 [1.21,1.51] Adj for O_3: 1.51 [1.34,1.71] **Single-pollutant model** Temp > 20°C PIH: 1.54 [1.31,1.81] IS: 1.46 [1.32,1.61] Temp < 20°C PIH: 0.82 [0.48,1.40] IS: 0.97 [0.65,1.44]

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Ulirsch et al. (2007, 091332) **Period of Study:** November 1994–March 2000 **Location:** Pocatello, Idaho and Chubbuck, Idaho	**Outcome (ICD-9):** CVD (390-429). **Age Groups:** 65 + **Study Design:** Time series **N:** 39,347 admissions/visits **Statistical Analyses:** Log-linear generalized linear models **Covariates:** Time, temperature, relative humidity, influenza, day of the week **Season:** All, and separate analyses were performed for the all-age group for cool months (October–March) vs. warm months (April–September). **Dose-response Investigated:** No **Statistical Package:** S-plus version 6.1 **Lags Considered:** 0- to 4-day lags, and mean of days 0 -4	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (range** **10th - 90th percentiles):** 24.2 (3.0–183.0 10.5–40.7) **Monitoring Stations:** 4 **Copollutant (correlation):** NO_2: r = 0.47 **Other variables:** Correlation for PM_{10} between monitors: r = 0.42–0.87	**PM Increment:** 50 $\mu g/m^3$, and 24.3 $\mu g/m^3$ (mean increase in PM_{10}) **Mean percent of change (% change in the mean number of daily admissions and visits) [95% CI]:** **For 24.3 $\mu g/m^3$ increase in PM_{10}:** All-age RD/CVD: 3.7 [1.3, 6.3] All-age CVD (Lag 0): -0.02 [-5.9, 6.3] All-age CVD (Lag 1): 1.9 [-4.1, 8.4] All-age CVD (Lag 2): -3.1 [-9.1, 3.4] All-age CVD (Lag 3): 0.5 [-5.6, 6.9] All-age CVD (Lag 4): -1.7 [-4.3, 0.9] Lag 0–4 days: -0.5 [-8.0, 7.6] **For 50 $\mu g/m^3$ increase in PM_{10}** (single pollutant models, CIs not given): All-age respiratory disease: 8.4 All-age RD/CVD: 7.9 18-64 years RD: 7.2 All-age CVD (Lag 3): 1.0 All-age CVD (Lag 4): -3.6 All-age CVD (Lag 0 -4): -1.1 **Notes:** Included urgent care visits as well as emergency department visits and hospital admissions.
Reference: Yang et al. (2007, 092847) **Period of Study:** 1996 - 2005 **Location:** Taipei, Taiwan	**Outcome:** Congestive Heart Failure HA (ICD 9: 428) **Age Groups:** NR **Study Design:** case-crossover **N:** 24,240 events **Statistical Analyses:** Poisson **Covariates:** temperature, humidity **Dose-response Investigated?** No **Statistical Package:** SAS **Lags Considered:** lags 0-3	**Pollutant:** PM_{10} **Averaging Time:** 24h **Mean:** 49.47 **Min:** 14.42 **25th:** 33.08 **50th:** 44.71 **75th:** 60.10 **Max:** 234.91 **Monitoring Stations:** 6 **Copollutant:** NR **Co-pollutant Correlation** n/a	**PM Increment:** Interquartile Range (27.02 $\mu g/m^3$) **Odds Ratio (Lower CI, Upper CI):** Temp ≥20°C PM_{10}: 1.15 (1.10-1.21)* PM_{10} + SO_2: 1.23 (1.17, 1.30)* PM_{10} + NO_2: 1.03 (0.97, 1.10) PM_{10} + CO_2: 1.09 (1.03, 1.15)* PM_{10} + O_3: 1.10 (1.04, 1.15)* Temp < 20°C PM_{10}: 0.99 (0.93, 1.05) PM_{10} + SO_2: 0.96 (0.89, 1.03) PM_{10} + NO_2: 0.97 (0.90, 1.04) PM_{10} + CO_2: 0.96 (0.90, 1.03) PM_{10} + O_3: 1.00 (0.94, 1.05) *p < 0.05

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Yang et al. (2007, 092847) **Period of Study:** 1996 - 2001 **Location:** Taipei, Taiwan	**Outcome:** Congestive Heart Failure HA **Age Groups:** NR **Study Design:** case-crossover **N:** NR **Statistical Analyses:** Poisson **Covariates:** NR **Dose-response Investigated?** No **Statistical Package:** SAS **Lags Considered:** lags 0-3	**Pollutant:** PM_{10} **Averaging Time:** 24h **Mean (SD):** Index days 111.68 (38.32) Comparison days 55.43 (24.66) **Monitoring Stations:** 7 **Copollutant:** NR **Co-pollutant Correlation** n/a	**PM Increment:** Index ($>125\ \mu g/m^3$) vs. Comparison ($\leq 125\ \mu g/m^3$) **Relative Risk (Lower CI, Upper CI), lag:** 0.915 (0.805, 1.041), lag 0 1.114 (0.993, 1.250), lag 1 0.983 (0.873, 1.106), lag 2 0.974 (0.870, 1.090), lag 3
Reference: Villeneuve et al. (2006, 090191) **Period of Study:** April, 1992 –March, 2002 **Location:** Edmonton, Canada	**Outcome (ICD-9):** Stroke (430-438), including ischemic stroke (434-436), hemorrhagic stroke (430,432), and transient ischemic attacks (TIA) (435). **Age Groups:** 65+ yrs **Study Design:** Case-crossover N: 12,422 visits **Statistical Analyses:** Conditional logistic regression **Covariates:** Temperature and relative humidity Season: summer (Apr-Sep), winter (Oct-Mar) **Dose-response Investigated:** No **Statistical Package:** SAS (PHREG) **Lags Considered:** 0-, 1-, and 3-day	**Pollutant:** PM_{10} **Averaging Time:** 24 h Mean (SD): All year: 24.2 (14.8) Summer: 25.9 (16.4) Winter: 22.6 (12.9) **Monitoring Stations:** 3 **Copollutant (correlation):** All year SO_2: r = 0.19 NO_2: r = 0.34; CO: r = 0.30 O_3-mean: r = 0.07; O_3-max: r = 0.22 $PM_{2.5}$: r = 0.79 Summer SO_2: r = 0.18 NO_2: r = 0.57; CO: r = 0.38 O_3-mean: r = 0.20; O_3-max: r = 0.40 $PM_{2.5}$: r = 0.85 Winter SO_2: r = 0.27 NO_2: r = 0.48; CO: r = 0.53 O_3-mean: r = -0.26; O_3-max: r = -0.09 $PM_{2.5}$: r = 0.70	**PM Increment:** $\mu g/m^3$ (IQR) All year: 16.0 Summer: 17.5 Winter: 16.0 Adjusted OR Estimate [CI]: Acute ischemic stroke All year Same-day lag: 0.98 [0.94,1.03] 1-day lag: 1.00 [0.96,1.05] 3-day lag: 0.99 [.93,1.05] summer Same-day lag: 0.93 [0.87,1.00] 1-day lag: 1.01 [0.94,1.08] 3-day lag: 0.96 [0.88,1.04] Winter Same-day lag: 1.04 [0.97,1.11] 1-day lag: 1.00 [0.94,1.06]; 3-day lag: 1.05 [0.95,1.15] Hemorrhagic stroke All year Same-day lag: 1.01 [0.90,1.12] 1-day lag: 1.03 [0.93,1.15] 3-day lag: 1.13 [0.98,1.30] summer Same-day lag: 1.02 [0.88,1.20] 1-day lag: 1.07 [0.91,1.26] 3-day lag: 1.20 [0.98,1.46] Winter Same-day lag: 1.05 [0.90,1.22] 1-day lag: 1.04 [0.91,1.19] 3-day lag: 1.11 [0.90,1.37] Transient cerebral ischemic attack All year Same-day lag: 0.96 [0.90,1.02]

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			1-day lag: 0.99 [0.94,1.05]
			3-day lag: 0.94 [0.87,1.01]
			summer
			Same-day lag: 0.97 [0.89,1.09]
			1-day lag: 0.99 [0.91,1.08]
			3-day lag: 0.94 [0.84,1.04]
			Winter
			Same-day lag: 0.95 [0.87,1.04]
			1-day lag: 0.99 [0.92,1.07]
			3-day lag: 0.93 [0.83,1.05]
			Notes: Adjusted ORs are provided for an IQR increase in the 3-day mean in Fig 1-4 for single and two-pollutant models.

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: von Klot et al. (2005, 088070) **Period of Study:** 1992-2001 **Location:** Augsburg, Germany Barcelona, Spain Helsinki, Finland Rome, Italy Stockholm, Sweden	**Outcome (ICD-9):** Acute myocardial infarction (410 ICD-10: I21-I22), angina pectoris (411, 413 ICD-10: I20, I24), dysrhythmia (427 ICD-10: I46.0, 46.9, I47-I49, R00.1, R00.8), heart failure (428 ICD-10: 150) **Age Groups:** 35+ yrs **Study Design:** Cohort N: 22,006 MI survivors **Statistical Analyses:** GAM, Spearman correlation **Covariates:** Temperature, dew point temp, avg barometric pressure, relative humidity **Season:** NR **Dose-response Investigated:** No **Statistical Package:** R **Lags Considered:** 0-3 days	**Pollutant:** PM_{10} **Averaging Time:** 24 h Mean (5th–95th percentile): Augsburg: 44.7 (16.8-81.4) Barcelona: 52.2 (25.3-89.2) Helsinki: 25.3 (9.5-57.6) Rome: 51.1 (23.3-89.4) Stockholm: 14.6 (6.4-30.0) **Monitoring Stations:** NR **Copollutant (correlation):** Augsburg PNC: r = 0.52 CO: r = 0.57; NO_2: r = 0.64 O_3: r = -0.32 Barcelona PNC: r = 0.29 CO: r = 0.39; NO_2: r = 0.36 O_3: r = -0.14 Helsinki PNC: r = 0.46 CO: r = 0.21; NO_2: r = 0.40 O_3: r = 0.02 Rome PNC: r = 0.33 CO: r = 0.31; NO_2: r = 0.48 O_3: r = -0.22 Stockholm PNC: r = 0.06 CO: r = 0.38; NO_2: r = 0.29 O_3: r = 0.15	**PM Increment:** 10 $\mu g/m^3$ Pooled RR Estimate [CI]: All cardiac admissions: 1.021 [1.005,1.048] Myocardial infarction: 1.026 [0.995,1.058] Angina pectoris: 1.008 [0.986,1.032] Notes: Rate ratios for 0-3 day lags are provided in graphical form (Fig 1). Same-day levels were significantly associated with cardiac readmissions.

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Wellenius et al. (2005, 087483) **Period of Study:** 1 Jan, 1987–30 Nov, 1999 **Location:** Pittsburgh, Pennsylvania	**Outcome (ICD-9):** Congestive heart failure (428.0-428.1) **Age Groups:** 65+ yrs **Study Design:** Case-crossover N: 55,019 patients **Statistical Analyses:** Conditional logistic regression, Pearson's pairwise correlation **Covariates:** Temperature, barometric pressure, dew point **Season:** NR **Dose-response Investigated:** No **Statistical Package:** SAS **Lags Considered:** 0-3 days	**Pollutant:** PM_{10} **Averaging Time:** 24 h Mean (5th–95th percentile): 31.06 (8.89-70.49) SD = 20.10 **Monitoring Stations:** 17 **Copollutant (correlation):** CO: r = 0.57 NO_2: r = 0.64 O_3: r = 0.29 SO_2: r = 0.51	**PM Increment:** 24 $\mu g/m^3$ (IQR) Percent Increase [CI]: Single-pollutant: 3.07 [1.59,4.57] Adj. for CO: -1.10 [-3.02,0.86] Adj. for NO_2: 0.52 [-1.46,2.53] Adj. for O_3: 2.80 [1.29,4.33] Adj. for SO_2: 2.18 [0.37,4.02] Percent Increase (with 10 $\mu g/m^3$ increment) 1.27 [0.66,1.88]
Reference: Wellenius et al. (2005, 088685) **Period of Study:** 1 Jan, 1986–30 Nov, 1999 **Location:** Birmingham, Chicago, Cleveland, Detroit, Minneapolis, New Haven, Pittsburgh, Salt Lake City, Seattle	Outcome (ICD-NR): Ischemic stroke and hemorrhagic stroke **Age Groups:** 65+ yrs **Study Design:** Case-crossover (time-stratified) N: 115,503 hospital admissions **Statistical Analyses:** Conditional logistic regression **Covariates:** Temperature and humidity **Season:** NR **Dose-response Investigated:** No **Statistical Package:** SAS (v.9) and R-statistical package **Lags Considered:** 0-2 lags	**Pollutant:** PM_{10} **Averaging Time:** 24 h Mean (SD): 32.69 (19.75) **Monitoring Stations:** NR (data obtained from the US EPA) **Copollutant (correlation):** CO: r = 0.43 NO_2: r = 0.53 SO_2: r = 0.39 Other variables: Temp: r = 0.22	**PM Increment:** 22.96 $\mu g/m^3$ (IQR) Percent Increase [CI]: Ischemic (same-day lag): 1.03 [0.04,2.04] Hemorrhagic: -0.58 [-5.48,4.58] Notes: Percent increase in rate for ischemic and hemorrhagic stroke are provided for each city in graphical form (Fig A and B).
Reference: Wellenius et al.,(2006, 088748) **Period of Study:** 1 Jan, 1986–30 Nov, 1999 **Location:** Birmingham, Chicago, Cleveland, Detroit, Minneapolis, New Haven, Pittsburgh, Salt Lake City, Seattle	**Outcome (ICD-9):** Congestive heart failure (428) **Age Groups:** 65+ yrs **Study Design:** Case-crossover (time-stratified) N: 292,918 admissions **Statistical Analyses:** Conditional logistic regression **Covariates:** Temperature and barometric pressure **Season:** NR **Dose-response Investigated:** No **Statistical Package:** SAS (v.9) and R-statistical package **Lags Considered:** 0-3 days	**Pollutant:** PM_{10} **Averaging Time:** 24 h Median: Overall: 28.3 Birmingham: 33.0 Chicago: 31.5 Cleveland: 34.5 Detroit: 29.5 Minneapolis: 24.0 New Haven: 22. Seattle: 25.8 **Monitoring Stations:** NR (data obtained from the US EPA) Copollutant: NR	**PM Increment:** 10 $\mu g/m^3$ Percent Increase [CI]: Same-day lag: 0.72 [0.35,1.10] p-value = 0.0002 Notes: City-specific percent increases are graphed in Fig 1 for same-day lag showing a significant association in Chicago, Detroit, Seattle, and the summary values. Percent increase in admission rate s are provided for lag 0-3 days in Fig 2 where same-day lag showed a significant association.

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Yang et al. (2004, 094376) **Period of Study:** 1997-2000 **Location:** Kaohsiung, Taiwan	**Outcome (ICD-9):** Cardiovascular diseases (410-429) **Age Groups:** All **Study Design:** Case-crossover N: 29,661 admissions **Statistical Analyses:** Conditional logistic regression **Covariates:** Temperature and humidity **Season:** NR **Dose-response Investigated:** No **Statistical Package:** SAS **Lags Considered:** Cumulative 0-2 days	**Pollutant:** PM_{10} **Averaging Time:** 24 h Median (min-max): 78.82 (20.50-217.33) **Monitoring Stations:** 6 Copollutant: NR	**PM Increment:** 66.33 $\mu g/m^3$ (IQR) OR Estimate [CI]: Temp > 25ºC: 1.439 [1.316,1.573] Temp < 25ºC: 1.568 [1.433,1.715] Adj for SO_2 Temp > 25ºC: 1.460 [1.333,1.599] Temp < 25ºC: 1.543 [1.404,1.696] Adj for NO_2 Temp > 25ºC: 1.306 [1.154,1.478] Temp < 25ºC: 0.912 [0.809,1.028] Adj for CO Temp > 25ºC: 1.260 [1.144,1.388] Temp < 25ºC: 1.259 [1.128,1.406] Adj for O_3 Temp > 25ºC: 1.086 [0.967,1.220] Temp < 25ºC: 1.703 [1.541,1.883]
Reference: Yang et al (2008, 157160) **Period of Study:** 1996 - 2004 **Location:** Taipei, Taiwan	**Outcome (ICD-9):** Congestive heart failure (428) **Age Groups:** All **Study Design:** Case-crossover N: 24,240 CHF hospital admissions **Statistical Analyses:** Conditional logistic regression **Covariates:** temperature, humidity **Season:** All **Dose-response Investigated:** No **Statistical Package:** SAS **Lags Considered:** Cumulative lag 0-2 days	**Pollutant:** PM_{10} **Averaging Time:** 24 h Mean (median, range, IQR): 49.47 (44.71, 14.42–234.91, 33.08–44.71) **Monitoring Stations:** 6 Copollutant: NR	**PM Increment:** 27.02 $\mu g/m^3$ (IQR) OR [95% CI]: Single pollutant models: > 20 °C: 1.15 [1.10–1.21] < 20 °C: 0.99 [0.93–1.05] Adjusted for SO_2: ≥ 20 °C: 1.23 [1.17–1.30] < 20 °C: 0.96 [0.89–1.03] Adjusted for NO_2: ≥ 20 °C: 1.03 [0.97–1.10] < 20 °C: 0.97 [0.90–1.04] Adjusted for CO: ≥ 20 °C: 1.09 [1.03–1.15] < 20 °C: 0.96 [0.90–1.03] Adjusted for O_3: ≥ 20 °C: 1.10 [1.04–1.15] < 20 °C: 1.00 [0.94–1.05]

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Zanobetti and Schwartz (2002, 034821) **Period of Study:** 1988-1994 **Location:** Cook county (Chicago), Illinois Wayne county (Detroit), Michigan Allegheny county (Pittsburgh), Pennsylvania and King county (Seattle), Washington	**Outcome (ICD-9):** Cardiovascular disease (390-429) with/without diabetes (250) **Age Groups:** 65-74 and 75+ yrs with diabetes, 65-74 and 75+ yrs without diabetes **Study Design:** Time series N: NR **Statistical Analyses:** GAM, meta-regression **Covariates:** Temperature, prior day's temperature, relative humidity, barometric pressure, day of the week **Season:** NR **Dose-response Investigated:** No	**Pollutant:** PM_{10} **Averaging Time:** 24 h Median (25-75th percentile): Chicago: 33 (23-46) Detroit: 32 (21-49) Pittsburgh: 30 (19-47) Seattle: 27 (18-39) **Monitoring Stations:** NR (obtained from USEPA Aerometric Information Retrieval System) Copollutant: NR	**PM Increment:** 10 $\mu g/m^3$ Percent Change [CI]: All four cities < 75 (w/ diabetes): 1.6 [1.2,2.0] 75+ (w/ diabetes): 2.0 [1.6,2.4] < 75 (w/o diabetes): 0.9 [0.6,1.1] 75+ (w/o diabetes): 1.3 [1.0,1.5] Chicago < 75 (w/ diabetes): 1.9 [1.1,2.7] 75+ (w/ diabetes): 2.0 [1.1,3.0] < 75 (w/o diabetes): 0.7 [0.2,1.2] 75+ (w/o diabetes): 1.2 [0.8,1.7] Detroit < 75 (w/ diabetes): 1.3 [0.5,2.2] 75+ (w/ diabetes): 2.1 [1.0,3.1] < 75 (w/o diabetes): 1.2 [0.7,1.7] 75+ (w/o diabetes): 1.2 [0.7,1.6] Pittsburgh < 75 (w/ diabetes): 1.8 [0.9,2.7] 75+ (w/ diabetes): 0.9 [-0.2,2.0] < 75 (w/o diabetes): 0.6 [0.1,1.2] 75+ (w/o diabetes): 1.6 [1.2,2.1] Seattle < 75 (w/ diabetes): 1.9 [0.1,3.7] 75+ (w/ diabetes): 2.7 [0.7,4.8] < 75 (w/o diabetes): 0.8 [0.0,1.6] 75+ (w/o diabetes): 0.9 [0.2,1.6] Notes: Overall percent increases were also provided for each city, yielding similar results.

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Zanobetti and Schwartz (2005, 088069) **Period of Study:** 1985-1999 **Location:** 21 U.S. cities (Birmingham, Alabama Boulder, Colorado Canton, Ohio Chicago, Illinois Cincinnati, Ohio Cleveland, Ohio Colorado Springs, Colorado Detroit, Michigan Honolulu, Hawaii Houston, Texas Minneapolis-St. Paul, Minnesota Nashville, Tennessee New Haven, Connecticut Pittsburgh, Pennsylvania Provo-Orem, Utah Salt Lake City, Utah Seattle, Washington Steubenville, Ohio Youngstown, Ohio)	**Outcome (ICD-9):** Myocardial infarction (410) **Age Groups:** > 65 yrs **Study Design:** Case-crossover N: 302,453 admissions **Statistical Analyses:** Conditional logistic regression **Covariates:** Temperature **Season:** NR **Dose-response Investigated:** Yes **Statistical Package:** SAS (PROC PHREG) **Lags Considered:** 0-2 days	**Pollutant:** PM_{10} **Averaging Time:** 24 h Median: Ranged from 15.5-34.1Avg across all cities = 27 **Monitoring Stations:** 1+ (data obtained from USEPA's Aerometric Information Retrieval System) Copollutant: NR	**PM Increment:** 10 $\mu g/m^3$ Percent Increase [CI]: MI only: 0.65 [0.3,1] Previous COPD admission: 1.3 [-0.1,2.8] Secondary pneumonia diagnosis: 1.4 [-0.8,3.6] Notes: Figure 1 presents percent change in MI per lag day, showing same-day lag to be significant. Figure 2 shows percent change with/without other co-morbidities.

[1]All units expressed in $\mu g/m^3$ unless otherwise specified.

Table E-6. Short-term exposure–cardiovascular–ED/HA - $PM_{10-2.5}$.

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Halonen et al. (2009, 180379) **Period of Study:** 1998-2004 **Location:** Helsinki, Finland	**Outcome:** Cardiovascular Hospitalizations & Mortality (ICD 10: I00-99) **Age Groups:** 65+ yrs **Study Design:** time series **N:** NR **Statistical Analyses:** Poisson, GAM **Covariates:** temperature, humidity, influenza epidemics, high pollen episodes, holidays **Dose-response Investigated?** No **Statistical Package:** R **Lags Considered:** lags 0-3 & 5d (0-4) mean	**Pollutant:** $PM_{10-2.5}$ **Averaging Time:** daily **Mean (SD):** NR **Min:** 0.0 **25th percentile:** 4.9 **50th percentile:** 7.5 **75th percentile:** 12.1 **Max:** 101.4 **Monitoring Stations:** NR **Copollutant:** $PM_{<0.03}$, $PM_{0.03-0.1}$, $PM_{<0.1}$, $PM_{<0.10.29}$, $PM_{2.5}$, CO, NO_2 **Co-pollutant Correlation** $PM_{<0.03}$: 0.14 $PM_{0.03-0.1}$: 0.28 $PM_{<0.1}$: 0.24 $PM_{<0.10.29}$: 0.20 $PM_{2.5}$: 0.25	**PM Increment:** Interquartile Range **Percent Change (Lower CI, Upper CI):** All Cardiovascular Morality Lag 0: -0.01 (-1.52, 1.53) Lag 1: -0.26 (-1.69, 1.18) Lag 2: -0.61 (-2.03, 0.83) Lag 3: -0.57 (-1.98, 0.85) 5-d mean: -0.70 (-2.56, 1.20) Coronary Heart Disease HA Lag 0: 1.12 (-0.28, 2.55) Lag 1: -0.38 (-1.68, 0.94) Lag 2: 0.01 (-1.33, 1.37) Lag 3: -0.53 (-1.82, 0.78) 5-d mean: 0.23 (-0.29, 0.75) Stroke HA Lag 0: -1.33 (-3.26, 0.63) Lag 1: -1.90 (-3.82, 0.07) ‡ Lag 2: -1.09 (-3.04, 0.89) Lag 3: -0.51 (-2.40, 1.43) 5-d mean: -2.21 (-4.75, 0.39) Arrhythmia HA Lag 0: 0.57 (-1.33, 2.49) Lag 1: -0.65 (-2.55, 1.29) Lag 2: 0.02 (-1.93, 2.00) Lag 3: -1.34 (-3.26, 0.62) 5-d mean: -1.11 (-3.68, 1.53) *$p < 0.05$, ‡$p < 0.10$

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Host et al. (2008, 155852) **Period of Study:** 2000 - 2003 **Location:** Six French cities: Le Havre, Lille, Marseille, Paris, Rouen, and Toulouse	**Outcome (ICD-10):** Daily hospitalizations for all cardiovascular (I00–I99), cardiac (I00–I52), and ischemic heart diseases (I20–I25). **Age Groups:** For cardiovascular diseases: All ages, and restricted to ≥ 65 years Study Design: Time series N: NR (Total population of cities: approximately 10 million) **Statistical Analyses:** Poisson regression **Covariates:** Seasons, days of the week, holidays, influenza epidemics, pollen counts, temperature, and temporal trends **Dose-response Investigated:** No **Statistical Package:** MGCV package in R software (R 2.1.1) **Lags Considered:** Avg of 0-1 days	**Pollutant:** $PM_{10-2.5}$ **Averaging Time:** 24 h Mean $\mu g/m^3$ (5th -95th percentile): Le Havre: 7.3 (2.5–14.0) Lille: 7.9 (2.2–13.7) Marseille: 11.0 (4.5–21.0) Paris: 8.3 (3.2–15.9) Rouen: 7.0 (3.0–12.5) Toulouse: 7.7 (3.0–15.0) **Monitoring Stations:** 13 total: 1 in Toulouse 4 in Paris 2 each in other cities **Copollutant (correlation):** $PM_{2.5}$: Overall: $r > 0.6$ Ranged between r = 0.28 and r = 0.73 across the six cities.	**PM Increment:** 10 $\mu g/m^3$, and an 18.8 $\mu g/m^3$ increase (corresponding to an increase in pollutant levels between the lowest of the 5th percentiles and the highest of the 95th percentiles of the cities' distributions) ERR (excess relative risk) Estimate [CI]: For all cardiovascular diseases (10 $\mu g/m^3$ increase): All ages: 0.5% [-1.2, 2.3] ≥ 65 years: 1.0% [-1.0, 3.0] For all cardiovascular diseases (18 $\mu g/m^3$ increase): All ages: 1.0% [-2.3, 4.3] ≥ 65 years: 1.9% [-2.0, 5.9] For cardiac diseases (10 $\mu g/m^3$ increase): All ages: 0.1% [-1.9, 2.1] ≥ 65 years: 1.6% [-0.8, 4.1] For cardiac diseases (18.8 $\mu g/m^3$ increase): All ages: 0.1% [-3.6, 4.0] ≥ 65 years: 3.1% [-1.5, 7.9] For ischemic heart diseases (10 $\mu g/m^3$ increase): All ages: 2.8% [-0.8, 6.6] ≥ 65 years: 6.4% [1.6, 11.4] For ischemic heart diseases (18 $\mu g/m^3$ increase): All ages: 5.4% [-1.5, 12.8] ≥ 65 years: 12.4 [3.1, 22.6]
Reference: Metzger et al. (2004, 044222) **Period of Study:** August 1998–August 2000 **Location:** Atlanta Metropolitan area (Georgia)	**Outcome (ICD-9):** Emergency visits for ischemic heart disease (410–414), cardiac dysrhythmias (427), cardiac arrest (427.5), congestive heart failure (428), peripheral vascular and cerebrovascular disease (433-437, 440, 443-444, 451–453), atherosclerosis (440), and stroke (436). **Age Groups:** All **Study Design:** Time series N: 4,407,535 emergency department visits between 1993–2000 (data not reported for 1998 - 2000) **Statistical Analyses:** Poisson generalized linear modeling **Covariates:** Day of the week, hospital entry and exit indicator variables, federally observed holidays, temporal trends, temperature, dew point temperature **Season:** All **Dose-response Investigated:** No **Statistical Package:** SAS **Lags Considered:** 3-day moving avg, lags 0 -7	**Pollutant:** $PM_{10-2.5}$ **Averaging Time:** 24 h Median $\mu g/m^3$ (10% - 90% range): 9.1 (4.4, 16.2) **Monitoring Stations:** 1 **Copollutant (correlation):** PM_{10}: r = 0.59 O_3: r = 0.35 NO_2: r = 0.46 CO: r = 0.32 SO_2: r = 0.21 $PM_{2.5}$: r = 0.43 UFP: r = 0.13 $PM_{2.5}$ water soluble metals: r = 0.47 $PM_{2.5}$ sulfates: r = 0.26 $PM_{2.5}$ acidity: r = 0.23 $PM_{2.5}$ organic carbon: r = 0.51 $PM_{2.5}$ elemental carbon: r = 0.48 $PM_{2.5}$ oxygenated hydrocarbon: r = 0.31 Other variables: Temperature: r = 0.20 Dew point: r = 0.00	**PM Increment:** 5 $\mu g/m^3$ (approximately 1 SD) RR [95% CI]: For 3 day moving avg: All CVD: 1.012 [0.985, 1.040] Dysrhythmia: 1.021 [0.974, 1.070] Congestive heart failure: 1.020 [0.964–1.079] Ischemic heart disease: 0.994 [0.946–1.045] Peripheral vascular and cerebrovascular disease: 1.022 [0.972–1.074]] Results for Lags 0–7 expressed in figures (see notes). Notes: Figure 1: RR (95% CI) for single-day lag models for the association of ER visits for CVD with daily ambient $PM_{10-2.5}$. Summary of Figure 1 results: Positive association at Lag 0.

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Peng et al. (2008, 156850) **Period of Study:** January 1, 1999–December 31, 2005 **Location:** 108 U.S. counties in the following states: Alabama, Arizona, California, Colorado, Connecticut, District of Columbia, Florida, Georgia, Idaho, Illinois, Indiana, Kentucky, Louisiana, Maine, Maryland, Massachusetts, Michigan, Minnesota, Missouri, Nevada, New Hampshire, New Jersey, New Mexico, New York, North Carolina, Ohio, Oklahoma, Oregon, Pennsylvania, Rhode Island, South Carolina, Tennessee, Texas, Utah, Virginia, Washington, West Virginia, Wisconsin	**Outcome (ICD-9):** Emergency hospitalizations for: Cardiovascular disease, including heart failure (428), heart rhythm disturbances (426–427), cerebrovascular events (430–438), ischemic heart disease (410–414, 429), and peripheral vascular disease (440–448). **Age Groups:** 65 + years, 65–74, 75+ **Study Design:** Time series N: approximately 12 million Medicare enrollees (3.7 million CVD and 1.4 million RD admissions) **Statistical Analyses:** Two-stage Bayesian hierarchical models: Overdispersed Poisson models for county-specific data. Bayesian hierarchical models to obtain national avg estimate **Covariates:** Day of the week, age-specific intercept, temperature, dew point temperature, calendar time, indicator for age of 75 years or older. Some models were adjusted for $PM_{2.5}$. **Dose-response Investigated:** No **Statistical Package:** R version 2.6.2 **Lags Considered:** 0-2 days	**Pollutant:** $PM_{10-2.5}$ **Averaging Time:** 24 h Mean $\mu g/m^3$ (IQR): All counties assessed: 9.8 (6.9–15.0) Counties in Eastern US: 9.1 (6.6–13.1) Counties in Western US: 15.4 (10.3–21.8) **Monitoring Stations:** At least 1 pair of co-located monitors (physically located in the same place) for PM_{10} and $PM_{2.5}$ per county **Copollutant (correlation):** $PM_{2.5}$: r = 0.12 PM_{10}: r = 0.75 Other variables: Median within-county correlations between monitors: r = 0.60	**PM Increment:** 10 $\mu g/m^3$ Percentage change [95% CI]: CVD: Lag 0 (unadjusted for $PM_{2.5}$): 0.36 [0.05, 0.68] Lag 0 (adjusted for $PM_{2.5}$): 0.25 [-0.11, 0.60] Notes: Effect estimates for $PM_{10-2.5}$ (0–2 day lags) are showing in Figures 2–5. Figure 2: Percentage change in emergency hospital admissions for CVD per 10 $\mu g/m^3$ increase in PM (single pollutant model and model adjusted for $PM_{2.5}$ concentration) Figure 4: Percentage change in emergency hospital admissions rate for CVD and RD per a 10 $\mu g/m^3$ increase in $PM_{10-2.5}$ (0–2 day lags, Eastern vs. Western USA) Figure 5: County-specific log relative risks of emergency hospital admissions for CVD per 10 $\mu g/m^3$ increase in $PM_{10-2.5}$ at Lag 0 (unadjusted for $PM_{2.5}$ and plotted vs percentage of urbanicity) No significant associations between $PM_{10-2.5}$ and cause-specific cardiovascular disease.
Reference: Tolbert et al. (2007, 090316) **Period of Study:** August 1998–December 2004 **Location:** Atlanta Metropolitan area, Georgia	**Outcome (ICD-9):** Combined CVD group, including: Ischemic heart disease (410–414), cardiac dysrhythmias (427), congestive heart failure (428), and peripheral vascular and cardiovascular disease (433–437, 440, 443–445, and 451–453) **Age Groups:** All **Study Design:** Time series N: NR for 1998–2004. For 1993–2004: 10,234,490 ER visits (283,360 visits). **Statistical Analyses:** Poisson generalized linear models **Covariates:** Long-term temporal trends, temperature, dew point, days of week, federal holidays, hospital entry and exit **Season:** All **Dose-response Investigated:** No **Statistical Package:** SAS version 9.1 **Lags Considered:** 3-day moving avg (lag 0-2)	**Pollutant:** $PM_{10-2.5}$ **Averaging Time:** 24 h Mean ($\mu g/m^3$) (median IQR, range, 10th–90th percentiles): 9.0 (8.2 5.6–11.5 0.5–50.3 3.6–15.1) **Monitoring Stations:** 1 **Copollutant (correlation):** PM_{10}: r = 0.67 O_3: r = 0.36 NO_2: r = 0.48 CO: r = 0.38SO2: r = 0.16 $PM_{2.5}$: r = 0.47 $PM_{2.5}$ SO_4: r = 0.32 $PM_{2.5}$ EC: r = 0.49 $PM_{2.5}$ OC: r = 0.49 $PM_{2.5}$ TC: r = 0.51 $PM_{2.5}$ water-sol metals: r = 0.50 OHC: r = 0.41	**PM Increment:** 5.89 $\mu g/m^3$ (IQR) Risk ratio [95% CI]: CVD: 1.004 (0.990–1.019)

[1]All units expressed in $\mu g/m^3$ unless otherwise specified.

Table E-7. Short-term exposure - cardiovascular–ED/HA – $PM_{2.5}$ (including PM components/sources)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Andersen et al. (2008, 189651) **Period of Study:** May 2001 - December 2004 **Location:** Los Angeles and San Diego counties, California	**Outcome (ICD-10):** CVD, including angina pectoris (I20), myocardial infarction (I21–22), other acute ischemic heart diseases (I24), chronic ischaemic heart disease (I25), pulmonary embolism (I26), cardiac arrest (I46), cardiac arrhythmias (I48–48), and heart failure (I50). RD, including chronic bronchitis (J41–42), emphysema (J43), other chronic obstructive pul-monary disease (J44), asthma (J45), and status asthmaticus (J46). Pediatric hospital admissions for asthma (J45) and status asthmaticus (J46). **Age Groups:** > 65 yrs (CVD and RD), 5–18 years (asthma) **Study Design:** Time series **N:** NR **Statistical Analyses:** Poisson GAM **Covariates:** Temperature, dew-point temperature, long-term trend, seasonality, influenza, day of the week, public holidays, school holidays (only for 5–18 year olds), pollen (only for pediatric asthma outcome) **Season:** NR **Dose-response Investigated:** No **Statistical Package:** R statistical software (gam procedure, mgcv package) **Lags Considered:** Lag 0-5 days, 4-day pollutant avg (lag 0-3) for CVD, 5-day avg (lag 0-4) for RD, and a 6-day avg (lag 0-5) for asthma.	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h Mean µg/m^3 (SD median IQR 99th percentile): 10 (5 9 7–12 28) **Monitoring Stations:** 1 **Copollutant (correlation):** NCtot: r = 0.40 NC100: r = 0.29 NCa12: r = 0.07 Nca23: r = -0.25 NCa57: r = 0.51 NC_{a212}: r = 0.82 PM_{10}: r = 0.80 CO: r = 0.46 NO_2: r = 0.42 NO_x: r = 0.40 NO_x curbside: r = 0.28 O_3: r = -0.20 Other variables: Temperature: r = -0.01 Relative humidity: r = 0.21	**PM Increment:** 5 µg/m^3 (IQR) Relative risk (RR) Estimate [CI]: CVD hospital admissions (4 day avg, lag 0 -3), age 65+: One-pollutant model: 1.03 [1.01–1.06] Adj for NCtot: 1.03 [1.01–1.06] RD hospital admissions (5 day avg, lag 0 - 4), age 65+: One-pollutant model: 1.00 [0.95–1.00] Adj for NCtot: 1.00 [0.95–1.06] Asthma hospital admissions (6 day avg lag 0–5), age 5 - 18: One-pollutant model: 1.15 [1.00–1.32] Adj for NCtot: 1.13 [0.98–1.32] Estimates for individual day lags reported only in figure form (see notes): Notes: Figure 2: Relative risks and 95% confidence intervals per IQR in single day concentration (0–5 day lag). Summary: CVD: Marginally significant association at Lag 0. RD: No statistically or marginally significant associations. Positive associations at Lag 4–5.Asthma: Wide confidence intervals make interpretation difficult. Positive associations at Lag 1, 2, 3.

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Ballester et al. (2006, 088746) **Period of Study:** 1995 - 1999 **Location:** 6 Spanish cities: Barcelona, Bilbao, Pamplona, Valencia, Vigo, Zaragoza	**Outcome (ICD-9):** The number of daily emergency admissions with primary diagnosis for all cardiovascular disease (390–459) and heart diseases (410–414, 427, 428) **Age Groups:** All ages **Study Design:** Time series N: NR **Statistical Analyses:** Poisson GAMs **Covariates:** daily temperature, barometric pressure, and relative humidity daily influenza incidence, day of the week, holidays, unusual events (ex. medical strikes), seasonal variation, trend of the series **Season:** NR **Dose-response Investigated:** No **Statistical Package:** S-Plus GAM function **Lags Considered:** 0-3 days, 0- to 1-day avg	**Pollutant:** Black smoke (BS) **Averaging Time:** 24 h Mean $\mu g/m^3$ (10-90th percentile): Overall mean NR. City specific means Barcelona: 35.0 (19.4, 53.0) Bilbao: 18.5 (8.8, 31.0) Pamplona: 7.4 (2.3, 13.0) Valencia: 40.3 (20.3, 66.4) Vigo: 79.4 (43.9, 122.3) Zaragoza: 40.4 (23.8, 61.3) **Monitoring Stations:** NR (at least three stations per city) **Copollutant (correlation):** Summary of the correlation coefficients between each pair of pollutants within cities: PM_{10}: r = 0.48 TSP: from r = 0.16 to r = 0.69 (median r = 0.43) NO_2: from r = 0.23 to r = 0.69 (median r = 0.48) SO_2: from r = 0.09 to r = 0.59 (median r = 0.24) CO: from r = 0.62 to r = 0.69 (median r = 0.69) O_3: from r = -0.43 to r = -0.06 (median r = -0.16)	**PM Increment:** 10 $\mu g/m^3$ Relative risk [CI]: Relative risks are expressed only in the form of figures (see notes). Percentage change in risk [CI]: All cardiovascular diseases (avg of lags 0 -1) 0.24% [-0.18, 0.67] Heart disease (avg of lags 0 -1) 0.71% [0.13, 1.29] Notes: Relative risks for the single pollutant models are expressed in Figure 2. Figure 2: Time sequence of the combined association between BS and hospital admissions for all CVD (A) and heart disease (B). Summary: Significant, positive association of TSP with both overall CVD and heart disease hospitalizations at Lag 0. Relative risks for two pollutant models are expressed in Figure 3: Combined estimates of the association between hospital admissions for heart diseases and air pollutants (avg of lags 0–1 adjusted for CO, NO_2, O_3, or SO_2). Summary: Significant, positive association remains after adjusting for NO_2, O_3, and SO_2. Association remains positive but becomes marginally significant after adjusting for CO.

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Ballester et al. (2006, 088746) **Period of Study:** 1993 - 1999 **Location:** 7 Spanish cities: Barcelona, Bilbao, Cartagena, Castellon, Gijon, Oviedo, Valencia	**Outcome (ICD-9):** The number of daily emergency admissions with primary diagnosis for all cardiovascular disease (390–459) and heart diseases (410–414, 427, 428) **Age Groups:** All ages **Study Design:** Time series **N:** NR **Statistical Analyses:** Poisson GAMs **Covariates:** daily temperature, barometric pressure, and relative humidity daily influenza incidence, day of the week, holidays, unusual events (ex. medical strikes), seasonal variation, trend of the series **Season:** NR **Dose-response Investigated:** No **Statistical Package:** S-Plus GAM function **Lags Considered:** 0-3 days, 0- to 1-day avg	**Pollutant:** TSP **Averaging Time:** 24 h Mean $\mu g/m^3$ (10-90th percentile): overall mean NR. City specific means Barcelona: 51.8 (29.4, 78.8) Bilbao: 58.3 (30.3, 92.3) Cartagena: 54.9 (32.5, 79.9) Castellon: 60.4 (32.0, 92.1) Gijon: 77.4 (47.4, 118.3) Oviedo: 76.0 (48.3, 111.8) Valencia: 61.0 (44.1, 80.7) **Monitoring Stations:** NR (at least three stations per city) **Copollutant (correlation):** Summary of the correlation coefficients between each pair of pollutants within cities: BS: from r = 0.16 to r = 0.69 (median r = 0.43) PM_{10}: NA NO_2: from r = -0.13 to r = 0.65 (median r = 0.48) SO_2: from r = 0.06 to r = 0.69 (median r = 0.31) CO: from r = 0.06 to r = 0.59 (median r = 0.47) O_3: from r = -0.27 to r = 0.07 (median r = -0.03)	**PM Increment:** 10 $\mu g/m^3$ Relative risk [CI]: Relative risks are expressed only in the form of figures (see notes). Percentage change in risk [CI]: All cardiovascular diseases: 0.07% [-0.23, 0.36] Heart disease 0.45% [0.04, 0.86] Notes: Relative risks for the single pollutant models are expressed in Figure 2. Figure 2: Time sequence of the combined association between TSP and hospital admissions for all CVD (A) and heart disease (B). Summary of results: Positive, marginally significant association of TSP with overall CVD at Lag 0. Positive, statistically significant relation between TSP and heart disease hospitalizations at Lag 0. Relative risks for two pollutant models are expressed in Figure 3: Figure 3: Combined estimates of the association between hospital admissions for heart diseases and air pollutants (avg of lags 0–1 adjusted for CO, NO_2, O_3, or SO_2). Summary of results: Small positive significant or marginally significant associations between TSP and general CVD and heart disease hospitalizations remain constant after adjustment for CO, NO_2, O_3, or SO_2.

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Bell et al. (2008, 091268) **Period of Study:** 1995 - 2002 **Location:** Taipei, Taiwan	**Outcome (ICD-9):** Hospital admissions for ischemic heart disease (410, 411, 414), cerebrovascular disease (430–437), asthma (493), and pneumonia (486). **Age Groups:** All **Study Design:** Time series **N:** 6,909 hospital admissions for ischaemic heart diseases, 11,466 for cerebrovascular disease, 19,966 for pneumonia, and 10,231 for asthma **Statistical Analyses:** Poisson regression **Covariates:** Day of the week, time, apparent temperature, long-term trends, seasonality **Season:** All **Dose-response Investigated:** No **Statistical Package:** NR **Lags Considered:** lags 0-3 days, mean of lags 0-3	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h Mean **μg/m^3** (range IQR): 31.6 (0.50–355.0 20.2) **Monitoring Stations:** 2 **Copollutant (correlation):** NR	**PM Increment:** 20 μg/m^3 (near IQR) Percentage increase estimate [95% CI]: Ischemic heart disease: L0: 3.48 (-0.39, 7.51) L1: 3.55 (-0.30, 7.56) L2: 3.32 (-0.50, 7.29) L3: 2.80 (-1.04, 6.79) L03: 8.38 (2.28, 14.84) Cerebrovascular disease: L0: -2.22 (-50.2, 0.67) L1: -1.30 (-4.08, 1.55) L2: 0.24 (-2.49, 3.040 L3: 1.21 (-1.41, 3.90) L03: -1.45 (-5.58, 2.87) Asthma: L0: 0.46 (-2.41, 3.42) L1: -1.36 (-4.33, 1.71) L2: -0.83 (-3.67, 2.10) L3: -0.78 (-3.63, 2.16) L03: -1.75 (-6.21, 2.92) Pneumonia: L0: 0.06 (-2.74, 2.94) L1: 0.34 (-2.446, 3.20) L2: -0.59 (-3.38, 2.29) L3: -0.44 (-3.22, 2.41) L03: -0.61 (-4.87, 3.85)
Reference: Bell et al. (2008, 091268) **Period of Study:** 1999 - 2005 **Location:** 202 US counties	**Outcome (ICD-9):** Heart failure (428), heart rhythm disturbances (426–427), cerebrovascular events (430–438), ischemic heart disease (410–414, 429), peripheral vascular disease (440–449), COPD (490–492), respiratory tract infections (464 - 466, 480 - 487) **Age Groups:** 65+ **Study Design:** Time series **N:** NR **Statistical Analyses:** Two-stage Bayesian hierarchical model to find national avg First stage: Poisson regression (county-specific) **Covariates:** day of the week, temperature, dew point temperature, temporal trends, indicator for persons 75+ years, population size **Season:** All, June–August (Summer), September–November (Fall), December–February (Winter), March–May (Spring) **Dose-response Investigated:** No **Statistical Package:** NR **Lags Considered:** 0–2 day lags	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h Mean (μg/m^3): Descriptive information presented in Figure S2 (boxplots): **IQR:** 8.7 μg/m^3 **Monitoring Stations:** NR **Copollutant (correlation):** NR	**PM Increment:** 10 μg/m^3 **Percent increase [95% PI]:** **Cardiovascular admissions:** Lag 0 (all seasons): 0.80 [0.59–1.01] Lag 0 (winter, national): 1.49 [1.09–1.89] Lag 0 (winter, northeast): 2.01 [1.39–2.63] Lag 0 (winter, southeast): 1.06 [-0.07–2.21] Lag 0 (winter, northwest): 0.85 [-4.11–6.07] Lag 0 (winter, southwest): 0.76 [-0.25–1.79] Lag 0 (spring, national): 0.91 [0.47–1.35] Lag 0 (spring, northeast) 0.95 [0.32–1.58] Lag 0 (spring, southeast): 0.75 [-0.26–1.78] Lag 0 (spring, northwest): -0.07 [-12.40–13.98] Lag 0 (spring, southwest): 1.78 [-0.87–4.51] Lag 0 (summer, national): 0.18 [-0.23–0.58] Lag 0 (summer, northeast): 0.55 [0.08–

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			1.02] Lag 0 (summer, southeast): -0.67 [-1.60–0.26] Lag 0 (summer, northwest): -1.55 [-15.22–14.31] Lag 0 (summer, southwest): -1.20 [-4.90–2.65] Lag 0 (autumn, national): 0.68 [0.29–1.07] Lag 0 (autumn, northeast): 1.03 [0.48–1.58] Lag 0 (autumn, southeast): 0.17 [-0.72–1.07] Lag 0 (autumn, northwest): -0.67 [-6.96–6.05] Lag 0 (autumn, southwest): 0.30 [-0.98–1.59] Lag 1 (all seasons): 0.07 [-0.12–0.26] Lag 1 (winter): 0.56 [0.16–0.96] Lag 1 (spring): -0.10 [-0.58–0.39] Lag 1 (summer): -0.16 [-0.54–0.22] Lag 1 (autumn): 0.04 [-0.28–0.35] Lag2 (all seasons): [0.06 [-0.12–0.23] Lag 2 (winter): 0.27 [-0.12–0.65] Lag 2 (spring): 0.19 [-0.23–0.60] Lag 2 (summer): -0.12 [-0.50–0.26] Lag 2 (autumn): 0.02 [-0.30–0.34] **Respiratory admissions:** Lag 0 (all seasons): 0.22 [-0.12–0.56] Lag 0 (winter, national): 1.05 [0.29–1.82] Lag 0 (winter, northeast): 1.76 [0.60–2.93] Lag 0 (winter, southeast): 0.59 [-1.35–2.58] Lag 0 (winter, northwest): -0.07 [-6.74–7.08] Lag 0 (winter, southwest): 0.03 [-1.25–1.34] Lag 0 (spring, national): 0.31 [-0.47–1.11] Lag 0 (spring, northeast): 0.34 [-0.66–1.34] Lag 0 (spring, southeast): -0.06 [-1.77–1.68] Lag 0 (spring, northwest): -8.52 [-25.62–12.51] Lag 0 (spring, southwest): 1.87 [-2.00–5.90] Lag 0 (summer, national): -0.62 [-1.33–0.09] Lag 0 (summer, northeast): -0.8 [-1.65–0.07] Lag 0 (summer, southeast): -0.15 [-1.88–1.61]

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			Lag 0 (summer, northwest): 0.25 [-21.46–27.96]
			Lag 0 (summer, southwest): 0.64 [-5.38–7.04]
			Lag 0 (autumn, national): 0.02 [-0.63–0.67]
			Lag 0 (autumn, northeast): -0.01 [-0.87–0.85]
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			Lag 0 (autumn, southeast): -0.58 [-2.06–0.91]
			Lag 0 (autumn, northwest): -1.38 [-11.84–10.32]
			Lag 0 (autumn, southwest): 1.77 [-0.73–4.33]
			Lag 1 (all seasons): 0.05 [-0.29–0.39]
			Lag 1 (winter): 0.50 [-0.27–1.27]
			Lag 1 (spring): -0.24 [-1.01–0.53]
			Lag 1 (summer): 0.28 [-0.39–0.95]
			Lag 1 (autumn): 0.15 [-0.49–0.79
			Lag 2 (all seasons): 0.41 [0.09–0.74]
			Lag 2 (winter, national): 0.72 [0.01–1.43]
			Lag 2 (winter, northeast): 0.79 [-0.21–1.80]
			Lag 2 (winter, southeast): 0.4 [-1.45, 2.27]
			Lag 2 (winter, northwest): -0.06 [-6.52–6.85]
			Lag 2 (winter, southwest): 1.2 [-0.10–2.52]
			Lag 2 (spring, national): 0.35 [-0.29–0.99]
			Lag 2 (spring, northeast): 0.04 [-0.88–0.97]
			Lag 2 (spring, southeast): 0.75 [-0.82–2.34]
			Lag 2 (spring, northwest): 2.29 [-14.26–22.03]
			Lag 2 (spring, southwest): 1.05 [-2.18–4.39]
			Lag 2 (summer, national): 0.57 [-0.07–1.23]
			Lag 2 (summer, northeast): 0.77 [-0.01–1.56]
			|Lag 2 (summer, southeast): -0.52 [-2.07–1.06]
			Lag 2 (summer, northwest): 0.74 [-18.73–24.86]
			Lag 2 (summer, southwest): 2.41 [-2.61–7.69]
			Lag 2 (autumn, national): 0.39 [-0.22–1.01]
			Lag 2 (autumn, northeast): 0.12 [-0.82–

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			1.07] Lag 2 (autumn, southeast): 0.14 [-1.29–1.59] Lag 2 (autumn, northwest): -0.74 [-10.08–9.58] Lag 2 (autumn, southwest): 0.97[-1.36–3.36]
Reference: Bell et al. (2009, 191007) **Period of Study:** 1999-2005 **Location:** 168 US Counties	**Outcome:** CVD hospital admissions **Study Design:** Retrospective Cohort **Covariates:** socio-economic conditions, long term temperature **Statistical Analysis:** Bayesian hierarchical model **Age Groups:** ≥65	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24h **Mean (SD) Unit:** NR **Range (Min, Max):** NR **Copollutant (correlation):** NR	**Increment:** 20% of the population acquiring air conditioning **Percent Change (95% CI) in community-specific PM health effect estimates for CVD hospital admissions** Any AC, including window units Yearly health effect: -4.3 (-72.7-4.2) Summer health effect: -148 (-327-31.1) Winter health effect: -80.0 (-182-22.0) Central AC Yearly health effect: -42.5(-63.4--21.6) Summer health effect: -79.5 (-143--15.7) Winter health effect: -41.9 (-124-40.0)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Bell et al. (2009, 191997)	**Outcome:** Cardiovascular HA	**Pollutant:** $PM_{2.5}$	**PM Increment:** Interquartile Range in the fraction of $PM_{2.5}$
Period of Study: 1999-2005	**Age Groups:** 65+	**Averaging Time:** daily	**Percent Increase in PM Health Effect (Lower CI, Upper CI), lag**
Location: US	**Study Design:** time series	**Mean:**	EC: 25.8 (4.4, 47.2), lag 0
	N: NR	EC: 0.715	EC + Ni: 14.0 (-7.6, 35.5), lag 0
	Statistical Analyses: Bayesian Hierarchical Regression	Ni: 0.002	EC + V: 14.9 (-7.8, 37.6), lag 0
	Covariates: time trend, day of week, seasonality, dew point, temperature	V: 0.003	EC+ V, HS education: 15.0 (3.3, 26.8), lag 0
	Statistical Package: NR	**Min:**	EC+ V, median income: 15.8 (4.1, 27.5), lag 0
	Lags Considered: 0-2	EC: 0.309	EC+ V, racial composition: 14.2 (2.8, 25.6), lag 0
		Ni: 0.003	EC+ V, percent living in urban area: 14.7 (3.1, 26.3), lag 0
		V: 0.001	EC+ V, population: 13.6 (2.2, 25.0), lag 0
		Max:	EC + Ni, V: 11.9 (-10.4, 43.2), lag 0
		EC: 1.73	Ni: 19.0 (9.9, 28.2), lag 0
		Ni: 0.021	Ni + EC: 17.3 (7.7, 26.9), lag 0
		V: 0.010	Ni + V: 15.5 (4.1, 26.9), lag 0
		Interquartile Range:	Ni + EC, V: 14.9 (3.4, 26.4), lag 0
		EC: 0.245	V: 27.5 (10.6, 44.4), lag 0
		Ni: 0.001	V + EC: 23.1 (4.9, 41.4), lag 0
		V: 0.001	V+ Ni: 10.9 (-9.6, 31.5), lag 0
		Interquartile Range of Percents:	V + EC, Ni: 8.1 (-13.3, 29.5), lag 0
		EC: 1.7	EC: 11.8 (-69.2, 92.8), lag 1
		Ni: 0.01	EC: 21.0 (-46.6, 88.6), lag 2
		V: 0.01	Ni: 20.6 (-15.5, 56.7), lag 1
		Monitoring Stations: NR	Ni: -2.3 (-32.5, 27.9), lag 2
		Copollutant: Al, NH_4+, As, Ca, Cl, Cu, EC, OMC, Fe, Pb, Mg. Ni, NO_3-, K, Si, $Na+$, $SO_4=$, Ti, V, Zn	V: 34.0 (-31.2, 99.1), lag 1
		Co-pollutant Correlation	V: 8.0 (-46.8, 62.7), lag 2
		Ni, V: 0.48	Percent HS education: -17.4 (-46.8, 11.9), lag 0
		V, EC: 0.33	Median income: 21.3 (-20.0, 62.5), lag 0
		Ni, EC: 0.30	Percent black: 26.9 (-15.8, 69.6), lag 0
		Note: Pollutant concentrations available for all fractions of $PM_{2.5}$	Percent living in urban area: 34.4 (-29.0, 97.8), lag0
			Population: -4.3 (-13.3, 4.8), lag 0
			Notes: Interquartile ranges in percent HS education, median income, percent black, percent living in urban area, and population are 5.2 %, $9,223, 17.3%, 11.0%, and 549,283 respectively.

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Chan et al. (2007, 147787) **Period of Study:** Apr 1997 – Dec 2002 **Location:** Boston, MA	**Outcome:** Cerebrovascular Emergency Admissions **Age Groups:** 50+ yrs **Study Design:** time series **Statistical Analyses:** GAM Poisosn Regression **Covariates:** year, month, day of week, temperature, dew point **Dose-response Investigated?** No **Statistical Package:** NR **Lags Considered:** 0-3d	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24h **Mean (SD):** 31.5 (16.0) **Min:** 15.6 **Max:** 200.6 **IQR:** 19.7 **Monitoring Stations:** 16 **Copollutant:** O_3, CO, SO_2, NO_2, PM_{10} **Co-pollutant Correlation** O_3: 0.33 CO: 0.44 SO_2: 0.51 NO_2: 0.50 PM_{10}: 0.61	**PM Increment:** Interquartile Range (19.7 $\mu g/m^3$) **Percent Change (Lower CI, Upper CI), p-value:** Cerebrovascular Disease Lag 0: 1.006 (0.993, 1.019) Lag 1: 1.002 (0.990, 1.014) Lag 2: 1.015 (0.978, 1.052) Lag 3: 1.021 (1.005, 1.037) Lag 3 + O_3: 1.009 (0.987, 1.031) Lag 3 + CO: 1.014 (0.993, 1.035) Lag 3 + O_3 + CO: 1.009 (0.987, 1.031) Stroke Lag 0: 0.931 (0.831, 1.031) Lag 1: 0.936 (0.845, 1.027) Lag 2: 0.931 (0.820, 1.042) Lag 3: 0.991 (0.969, 1.013) Ischaemic stroke Lag 0: 0.981 (0.907, 1.055) Lag 1: 0.994 (0.920, 1.078) Lag 2: 0.960 (0.885, 1.035) Lag 3: 1.059 (0.984, 1.134) Haemorrhagic stroke Lag 0: 0.870 (0.740, 1.010) Lag 1: 0.882 (0.761, 1.003) Lag 2: 0.909 (0.810, 1.008) Lag 3: 0.921 (0.830, 1.012)
Reference: Chan et al. (2008, 093297) **Period of Study:** 1995 - 2002 **Location:** Taipei Metropolitan area, Taiwan	**Outcome (ICD-9):** Emergency visits for ischaemic heart diseases (410–411, 414), cerebrovascular diseases (430-437), and COPD (493, 496) **Age Groups:** All **Study Design:** Time series **N:** NR **Statistical Analyses:** Poisson regression **Covariates:** Year, month, day of week, temperature, dewpoint temperature, PM_{10}, NO_2 **Season:** All **Dose-response Investigated:** No **Statistical Package:** SAS version 8.0 **Lags Considered:** 0- to 7-day lags	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean $\mu g/m^3$ (SD):** NR **Monitoring Stations:** 1 **Copollutant (correlation):** NR	**PM Increment:** 19.7 $\mu g/m^3$ (IQR) OR [95% CI]: In environmental conditions without dust storms (results only given for best-fitting model) Lag 6 days: 1.024 (1.004, 1.044)
Reference: Delfino et al, (2008, 156390) **Period of Study:** 10/1/2003-11/15/2003 **Location:** Southern California	**Outcome:** Cardiovascular hospital admissions **Study Design:** Time series **Statistical Analysis:** Poisson regression with GEE **Age Groups:** All	**Pollutant:** $PM_{2.5}$ **Averaging Time:** Hourly **Mean (SD) Unit by county:** Los Angeles Before Fires: 27.2 (12.4) $\mu g/m^3$ During Fires: 54.1 (21.0) $\mu g/m^3$	**Increment:** 10 $\mu g/m^3$ **Relative Rate (Min CI, Max CI)** All Cardiovascular All Periods: 0.996 (0.989-1.003) Pre-Wildfire: 0.992 (0.976-1.009) Wildfire: 1.008 (0.999-1.018), p = 0.104

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
		After Fires: 15.9 (5.5) $\mu g/m^3$	Post-Wildfire: 0.991 (0.964-1.019), p = 0.955
		Orange	Ischaemic Heart Disease
		Before Fires: 23.2 (9.6) $\mu g/m^3$	All Periods: 0.991 (0.980-1.003)
		During Fires: 64.3 (26.5) $\mu g/m^3$	Pre-Wildfire: 0.990 (0.963-1.017)
		After Fires: 15.5 (10.2) $\mu g/m^3$	Wildfire: 0.117 (0.990-1.024), p = 0.313
		Riverside	Post-Wildfire: 0.989 (0.950-1.030), p = 0.976
		Before Fires: 32.7 (14.7) $\mu g/m^3$	Congestive Heart Failure
		During Fires: 42.1 (25.5) $\mu g/m^3$	All Periods: 0.989 (0.974-1.004)
		After Fires: 16.9 (10.2) $\mu g/m^3$	Pre-Wildfire: 0.978 (0.942-1.015)
		San Bernadino	Wildfire: 1.016 (0.933-1.039), p = 0.096
		Before Fires: 35.7 (16.6) $\mu g/m^3$	Post-Wildfire: 0.969 (0.914-1.027), p = 0.791
		During Fires: 45.3 (28.7) $\mu g/m^3$	Cardiac Dysrhythmia
		After Fires: 18.5 (8.3) $\mu g/m^3$	All Periods: 0.980 (0.962-0.998)
		San Diego	Pre-Wildfire: 0.979 (0.935-1.025)
		Before Fires: 18.5 (6.7) $\mu g/m^3$	Wildfire: 0.989 (0.961-1.017), p = 0.721
		During Fires: 76.1 (66.6) $\mu g/m^3$	Post-Wildfire: 0.976 (0.912-1.044), p = 0.934
		After Fires: 14.2 (7.2) $\mu g/m^3$	Cerebrovascular Disease and Stroke
		Ventura	All Periods: 1.019 (1.004-1.035)
		Before Fires: 18.4 (8.3) $\mu g/m^3$	Pre-Wildfire: 1.015 (0.980-1.052)
		During Fires: 50.1 (50.5) $\mu g/m^3$	Wildfire: 1.016 (0.997-1.036), p = 0.971
		After Fires: 12.9 (4.3) $\mu g/m^3$	Post-Wildfire: 1.044 (0.987-1.104), p = 0.379
		Copollutant (correlation): NR	**Relative Rate (Min CI, Max CI) in relation to pre-wildfire period (1)**
			All Cardiovascular: Wildfire, unadjusted for $PM_{2.5}$: 0.958 (0.920-0.997)
			Wildfire, adjusted for $PM_{2.5}$: 0.947 (0.902-0.994)
			Post-wildfire, unadjusted for $PM_{2.5}$: 1.061 (1.006-1.119)
			Post-wildfire, adjusted for $PM_{2.5}$: 1.053 (0.994-1.114)
			Ischaemic Heart Disease: Wildfire, unadjusted for $PM_{2.5}$: 0.913 (0.852-0.978)
			Wildfire, adjusted for $PM_{2.5}$: 0.905 (0.832-0.985)
			Post-wildfire, unadjusted for $PM_{2.5}$: 1.029 (0.943-1.123)
			Post-wildfire, adjusted for $PM_{2.5}$: 1.029 (0.936-1.131)
			Congestive Heart Failure: Wildfire, unadjusted for $PM_{2.5}$: 0.981 (0.817-0.972)
			Wildfire, adjusted for $PM_{2.5}$: 0.911 (0.819-1.014)
			Post-wildfire, unadjusted for $PM_{2.5}$: 1.113 (0.997-1.242)
			Post-wildfire, adjusted for $PM_{2.5}$: 1.105

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			(0.982-1.244) Cardiac Dysrhythmia: Wildfire, unadjusted for $PM_{2.5}$: 0.968 (0.874-1.072) Wildfire, adjusted for $PM_{2.5}$: 0.964 (0.851-1.093) Post-wildfire, unadjusted for $PM_{2.5}$: 1.089 (0.949-1.251) Post-wildfire, adjusted for $PM_{2.5}$: 1.057 (0.914-1.223) Cerebrovascular Disease and Stroke: Wildfire, unadjusted for $PM_{2.5}$: 1.066 (0.981-1.159) Wildfire, adjusted for $PM_{2.5}$: 1.017 (0.922-1.123) Post-wildfire, unadjusted for $PM_{2.5}$: 1.013 (0.907-1.132) Post-wildfire, adjusted for $PM_{2.5}$: 1.013 (0.902-1.138)
Reference: Dominici et al. (2006, 088398) **Period of Study:** 1999 - 2002 **Location:** 204 US counties, located in: Alabama, Alaska, Arizona, Arkansas, California, Colorado, Connecticut, Delaware, District of Columbia, Florida, Georgia, Hawaii, Idaho, Illinois, Indiana, Iowa, Kansas, Kentucky, Louisiana, Maine, Maryland, Massachusetts, Michigan, Minnesota, Mississippi, Missouri, Nevada, New Hampshire, New Jersey, New Mexico, New York, North Carolina, Ohio, Oklahoma, Oregon, Pennsylvania, Rhode Island, South Carolina, Tennessee, Texas, Utah, Virginia, Washington, West Virginia, Wisconsin	Outcome (ICD-9: Daily counts of hospital admissions for primary diagnosis of heart failure (428), heart rhythm disturbances (426–427), cerebrovascular events (430–438), ischemic heart disease (410–414, 429), peripheral vascular disease (440–448), chronic obstructive pulmonary disease (490–492), and respiratory tract infections (464–466, 480–487). **Age Groups:** > 65 years **Study Design:** Time series N: 11.5 million Medicare enrollees **Statistical Analyses:** Bayesian 2-stage hierarchical models. First stage: Poisson regression (county-specific) Second stage: Bayesian hierarchical models, to produce a national avg estimate **Covariates:** Day of the week, seasonality, temperature, dew point temperature, long-term trends **Season:** NR **Dose-response Investigated**: No **Statistical Package:** R statistical software version 2.2.0 **Lags Considered:** 0-2 days, avg of days 0-2	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h Mean ($\mu g/m^3$) (IQR): 13.4 (11.3–15.2) **Monitoring Stations:** NR **Copollutant (correlation):** NR Other variables: Median of pairwise correlations among $PM_{2.5}$ monitors within the same county for 2000: r = 0.91 (IQR: 0.81-0.95)	**PM Increment:** 10 $\mu g/m^3$ (Results in figures see notes) Percent increase in risk [95% PI]: Cerebrovascular disease (Lag 0): Age 65+: 0.81 [0.30, 1.32] Age 65-74: 0.91 [0.01, 1.82] Age 75+: 0.80 [0.21, 1.38] Peripheral vascular disease (Lag 0): Age 65+: 0.86 [-0.06, 1.79] Age 65-74: 1.21 [-0.26, 2.67] Age 75+: 0.86 [-0.39, 2.11] Ischemic heart disease (Lag 2): Age 65+: 0.44 [0.02, 0.86] Age 65-74: 0.37 [-0.22, 0.96] Age 75+: 0.52 [-0.01, 1.04] Heart rhythm disturbances (Lag 0): Age 65+: 0.57 [-0.01, 1.15] Age 65-74: 0.46 [-0.63, 1.54] Age 75+: 0.72 [0.02, 1.42] Heart failure (Lag 0): Age 65+: 1.28 [0.78, 1.78] Age 65-74: 1.21 [0.35, 2.07] Age 75+: 1.36 [0.78, 1.94] COPD (Lag 0): Age 65 +: 0.91 [0.91, 1.64] Age 65–74: 0.42 [-0.64, 1.48] Age 75+: 1.47 [0.54, 2.40] Respiratory tract infection: Age 65+: 0.92 [0.41, 1.43] Age 65–74: 0.93 [0.04, 1.82] Age 75+: 0.92 [0.32, 1.53] Annual reduction in admissions attributable to a 10 $\mu g/m^3$ reduction in daily $PM_{2.5}$ level (95% PI): Cerebrovascular disease: Annual number of admissions: 226,641

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			Annual reduction in admissions: 1836 [680, 2992] Peripheral vascular disease: Annual number of admissions: 70,061 Annual reduction in admissions: 602 [-42, 1254] Ischemic heart disease: Annual number of admissions: 346,082 Annual reduction in admissions: 1523 [69, 2976] Heart rhythm disturbances: Annual number of admissions: 169,627 Annual reduction in admissions: 967 [-17, 1951] Heart failure: Annual number of admissions: 246,598 Annual reduction in admissions: 3156 [1923, 4389] COPD: Annual number of admissions: 108,812 Annual reduction in admissions: 990 [196, 1785] Respiratory tract infections: Annual number of admissions: 226,620 Annual reduction in admissions: 2085 [929, 3241] Notes: Figure 2: Point estimates and 95% posterior intervals of the % change in admissions rates per 10 $\mu g/m^3$ (national avg relative rates) for single lag (0, 1, and 2 days) and distributed lag models for 0 to 2 days (total) for all outcomes. Summary: Positive significant or marginally significant associations between $PM_{2.5}$ and cerebrovascular disease at Lag 0 peripheral vascular disease at Lags 0 and 2 ischemic heart disease at Lag 2 heart rhythm disturbances at Lag 0 heart failure at Lag 0, Lag 2, and Lags 0 - 2 COPD at Lag 0, Lag 1, and Lags 0–2 and respiratory tract infections at Lag 2 and Lags 0–2. Figure 3: Point estimates and 95% posterior intervals of the % change in admission rates per 10 $\mu g/m^3$ (regional relative rates). Summary: For cardiovascular diseases, all estimates in the Midwestern, Northeastern, and Southern regions were positive, while estimates in the other regions (South, West, Central, Northwest) were close to 0. For respiratory disease, there were larger effects in the Central, Southeastern, Southern, and Western regions than in the other regions. Figure 4: Point estimates and 95% posterior intervals of the % change in admission per 10 $\mu g/m^3$ (Eastern vs. Western regions): Summary: All estimates for cardiovascular outcomes were

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			positive in the US Eastern region but not in the US Western region. The estimates for respiratory tract infections were larger in the Western region than in the Eastern region. The estimates for CCPD were positive in the both regions.
Reference: Halonen et al. (2009, 180379) **Period of Study:** 1998-2004 **Location:** Helsinki, Finland	**Outcome:** Cardiovascular Hospitalizations & Mortality (ICD 10: I00-99) **Age Groups:** 65 + yrs **Study Design:** time series **N:** NR **Statistical Analyses:** Poisson, GAM **Covariates:** temperature, humidity, influenza epidemics, high pollen episodes, holidays **Dose-response Investigated?** No **Statistical Package:** R **Lags Considered:** lags 0-3 & 5d (0-4) mean	**Pollutant:** $PM_{2.5}$ **Averaging Time:** daily **Mean (SD):** NR **Min:** 1.1 **25th percentile:** 5.5 **50th percentile:** 9.5 **75th percentile:** 11.7 **Max:** 69.5 **Monitoring Stations:** NR **Copollutant:** $PM_{<0.03}$, $PM_{0.03-0.1}$, $PM_{<0.1}$, $PM_{<0.10.29}$, $PM_{2.5-10}$, CO, NO_2 **Co-pollutant Correlation** $PM_{<0.03}$: 0.14 $PM_{0.03-0.1}$: 0.48 $PM_{<0.1}$: 0.35 $PM_{<0.10.29}$: 0.88 $PM_{2.5-10}$: 0.25	**PM Increment:** Interquartile Range **Percent Change (Lower CI, Upper CI):** All Cardiovascular Morality Lag 0: 0.73 (-0.66, 2.13) Lag 1: 0.74 (-0.63, 2.13) Lag 2: 0.74 (-0.62, 2.11) Lag 3: 0.06 (-1.29, 1.43) 5-d mean: 0.87 (-0.94, 2.70) Coronary Heart Disease HA Lag 0: -0.17 (-1.5.0, 1.18) Lag 1: -0.03 (-1.31, 1.26) Lag 2: -0.63 (-1.87, 0.62) Lag 3: 0.48 (-0.78, 1.76) 5-d mean: 0.80 (-0.94, 2.58) Stroke HA Lag 0: -0.99 (-2.78, 0.84) Lag 1: 0.02 (-1.74, 1.82) Lag 2: -1.38 (-3.13, 0.40) Lag 3: -0.17 (-1.92, 1.61) 5-d mean: -0.78 (-3.10, 1.60) Arrhythmia HA Lag 0: 0.82 (-1.03, 2.68) Lag 1: 0.18 (-1.58, 1.97) Lag 2: -0.09 (-1.82, 1.67) Lag 3: -0.48 (-2.22, 1.29) 5-d mean: 0.16 (-2.16, 2.54) *$p < 0.05$, ‡$p < 0.10$

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: (Host et al., 2008, 155852) **Period of Study:** 2000 - 2003 **Location:** Six French cities: Le Havre, Lille, Marseille, Paris, Rouen, and Toulouse	**Outcome (ICD-10):** Daily hospitalizations for all cardiovascular (I00–I99), cardiac (I00–I52), and ischemic heart diseases (I20–I25), all respiratory diseases (J00–J99), respiratory infections (J10–J22). **Age Groups:** For cardiovascular diseases: All ages, and restricted to ≥ 65 years. For all respiratory diseases: 0–14 years, 15–64 years, and ≥ 65 years. For respiratory infections: All ages **Study Design:** Time series N: NR (Total population of cities: approximately 10 million) **Statistical Analyses:** Poisson regression **Covariates:** Seasons, days of the week, holidays, influenza epidemics, pollen counts, temperature, and temporal trends **Season:** NR **Dose-response Investigated:** No **Statistical Package:** MGCV package in R software (R 2.1.1) **Lags Considered:** Avg of 0-1 days	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h Mean (5th -95th percentile): Le Havre: 13.8 (6.0–30.5) Lille: 15.9 (6.9–26.3) Marseille: 18.8 (8.0–33.0) Paris: 14.7 (6.5–28.8) Rouen: 14.4 (7.5–28.0) Toulouse: 13.8 (6.0–25.0) **Monitoring Stations:** 13 total: 1 in Toulouse 4 in Paris 2 each in other cities **Copollutant (correlation):** $PM_{10-2.5}$: Overall: $r > 0.6$ Ranged between $r = 0.28$ and $r = 0.73$ across the six cities.	**PM Increment:** 10 $\mu g/m^3$ increase, and a 27 $\mu g/m^3$ increase (corresponding to the difference between the lowest of the 5th percentiles and the highest of the 95th percentiles of the cities' distributions) ERR (excess relative risk) Estimate [CI]: For all cardiovascular diseases (10 $\mu g/m^3$ increase): All ages: 0.9% [0.1, 1.8] ≥ 65 years: 1.9% [0.9, 3.0] For all cardiovascular diseases (27 $\mu g/m^3$ increase): All ages: 2.5% [0.2, 4.9] ≥ 65 years: 5.3% [2.6, 8.2] For ischemic heart diseases (27 $\mu g/m^3$ increase): All ages: 5.2% [-0.6, 11.3] ≥ 65 years: 12.7% [6.3, 19.5] For cardiac diseases (10 $\mu g/m^3$ increase): All ages: 0.9% [-0.1, 2.0] ≥ 65 years: 2.4% [1.2, 3.7] For cardiac diseases (27 $\mu g/m^3$ increase): All ages: 2.5% [-0.3, 5.4] ≥ 65 years: 6.8% [3.3, 10.3] For ischemic heart diseases (10 $\mu g/m^3$ increase): All ages: 1.9 % [-0.2, 4.0] ≥ 65 years: 4.5% [2.3, 6.8] For all respiratory diseases (10 $\mu g/m^3$ increase): 0–14 years: 0.4% [-1.2, 2.0] 15–64 years: 0.8% [-0.7, 2.3]; ≥ 65 years: 0.5% [-2.0, 3.0] For all respiratory diseases (27 $\mu g/m^3$ increase): 0–14 years: 1.1% [-3.1, 5.5] 15–64 years: 2.2% [-1.8, 6.4]; ≥ 65 years: 1.3% [-5.3, 8.2] For respiratory infections (10 $\mu g/m^3$ increase): All ages: 2.5% [0.1, 4.8] For respiratory infections (27 $\mu g/m^3$ increase): All ages: 7.0% [0.7, 13.6]

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Jalaludin et al. (2006, 189416) **Period of Study:** 1 Jan, 1997–31 Dec, 2001 **Location:** Sydney, Australia	**Outcome (ICD-9):** Cardiovascular disease (390-459), cardiac disease (390-429), ischemic heart disease (410-413) and cerebrovascular disease or stroke (430-438) **Age Groups:** 65+ yrs **Study Design:** Time series N: NR **Statistical Analyses:** GAM, GLM **Covariates:** Temperature, humidity **Season:** Warm (Nov-Apr) and cool (May-Oct) **Dose-response Investigated:** No **Statistical Package:** S-Plus **Lags Considered:** 0-3 days	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean (min-max):** 9.5 (2.4-82.1) SD = 5.1 **Monitoring Stations:** 14 **Copollutant (correlation):** Warm BSP: r = 0.93 PM_{10}: r = 0.89 O_3: r = 0.57 NO_2: r = 0.45 CO: r = 0.35 SO_2: r = 0.27 Cool BSP: r = 0.90 PM_{10}: r = 0.88 O_3: r = 0.05 NO_2: r = 0.68 CO: r = 0.60 SO_2: r = 0.46 Other variables: Warm Temp: r = 0.24 Rel humidity: r = -0.15 Cool Temp: r = -0.04 Rel humidity: r = 0.20	**PM Increment:** 4.8 $\mu g/m^3$ (IQR) Percent Change Estimate [CI]: All CVD Same-day lag: 1.26 [0.56,1.96] Avg 0-1 day lag: 0.85 [0.18,1.52] Cool (same-day lag): 2.23 [0.98,3.50] Warm (same-day lag): 0.73 [-0.05,1.52] Cardiac disease Same-day lag: 1.55 [0.74,2.38] Avg 0-1 day lag: 1.33 [0.54,2.13] Cool (same-day lag): 2.37 [0.87,3.89] Warm (same-day lag): 1.13 [0.22,2.04] Ischemic heart disease Same-day lag: 1.17 [-0.08,2.44] Avg 0-1 day lag: 1.24 [0.04,2.45] Cool (same-day lag): 0.57 [-1.74,2.94] Warm (same-day lag): 1.31 [-0.04,2.68] Stroke Same-day lag: -0.89 [-2.41,0.65] Avg 0-1 day lag: -1.08 [-2.54,0.41] Cool (same-day lag): 1.45 [-1.17,4.15] Warm (same-day lag): -2.19 [-4.00,-0.36] Notes: All other lag-day ORs were provided, yet none were significant. Percent change in ED attendance was also reported graphically (Fig 1-5).

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Jalaludin et al. (2006, 189416) **Period of Study:** 1 Jan, 1997–31 Dec, 2001 **Location:** Sydney, Australia	**Outcome (ICD-9):** Cardiovascular disease (390-459), cardiac disease (390-429), ischemic heart disease (410-413) and cerebrovascular disease or stroke (430-438) **Age Groups:** 65+ yrs **Study Design:** Time series N: NR **Statistical Analyses:** GAM, GLM **Covariates:** Temperature, humidity **Season:** Warm (Nov-Apr) and cool (May-Oct) **Dose-response Investigated:** No **Statistical Package:** S-Plus **Lags Considered:** 0-3 days	**Pollutant:** $B_{S,P}$ **Averaging Time:** 24 h Mean/ 104/m (min-max): 0.26 (0.04-3.37) SD = 0.22 **Monitoring Stations:** 14 **Copollutant (correlation):** Warm $PM_{2.5}$: r = 0.93 PM_{10}: r = 0.82 O_3: r = 0.48 NO_2: r = 0.35 CO: r = 0.33 SO_2: r = 0.21; Cool $PM_{2.5}$: r = 0.90 PM_{10}: r = 0.75 O_3: r = -0.08 NO_2: r = 0.59 CO: r = 0.62 SO_2: r = 0.48 Other variables: Warm Temp: r = 0.23 Rel humidity: r = -0.04 Cool Temp: r = -0.09 Rel humidity: r = 0.36	**PM Increment:** 0.18/ 10^4/m (IQR) Percent Change Estimate [CI]: All CVD Same-day lag: 1.05 [0.44,1.66] Avg 0-1 day lag: 0.79 [0.20,1.38]; Cool (same-day lag): 2.38 [1.15,3.62] Warm (same-day lag): 0.45 [-0.18,1.09] Cardiac disease Same-day lag: 1.34 [0.63,2.05] Avg 0-1 day lag: 1.13 [0.44,1.82]; Cool (same-day lag): 2.50 [1.04,3.98] Warm (same-day lag): 0.80 [0.07,1.54] Ischemic heart disease Same-day lag: 0.91 [-0.17,2.02] Avg 0-1 day lag: 0.90 [-0.14,1.95]; Cool (same-day lag): 0.52 [-1.74,2.83] Warm (same-day lag): 0.93 [-0.15,2.03] Stroke Same-day lag: -0.93 [-2.27,0.42] Avg 0-1 day lag: -0.82 [-2.11,0.49]; Cool (same-day lag): 1.38 [-1.19,4.01]; Warm (same-day lag): -1.85 [-3.31,-0.36] Notes: All other lag-day ORs were provided, yet none were significant. Percent change in ED attendance was also reported graphically (Fig 1-5).
Reference: Lisabeth et al. (2008, 155939) **Period of Study:** 2001 - 2005 **Location:** Nueces County, Texas	**Outcome:** Ischemic stroke and transient ischemic attacks (ICD codes not reported). **Age Groups:** 45+ years **Study Design:** Time series **N:** 3,508 stroke/TIAs (2,350 strokes, and 1,158 TIAs) **Statistical Analyses:** Poisson regression **Covariates:** Temperature, day of week, temporal trends **Season:** All, but looked at potential effect modification by season (Summer: June–September Non-summer: October-May) **Dose-response Investigated:** No **Statistical Package:** S-plus 7.0 **Lags Considered:** Lags 0–5 days, and averaged lag effect (0–5 days)	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h Median $\mu g/m^3$ (IQR): 7.0 (4.8–10.0) **Monitoring Stations:** 6 **Copollutant (correlation):** NR	**PM Increment:** 5.1 $\mu g/m^3$ (IQR) RR Estimate [CI]: Lag 0: 1.03 (0.99, 1.07) Lag 1: 1.03 (1.00–1.07) All other lags and avg (lag 0–5) were not statistically or marginally significant. Adjusted for O_3: Lag 0: 1.03 (0.99, 1.07) Lag 1: 1.03 (0.99–1.06) All other lags and avg (lag 0–5) were not statistically or marginally significant. Notes: Figure 3: % change in stroke/TIA risk associated with an IQR increase in $PM_{2.5}$

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Metzger et al. (2004, 044222) **Period of Study:** August 1998–August 2000 **Location:** Atlanta Metropolitan area (Georgia)	**Outcome (ICD-9):** Emergency visits for ischemic heart disease (410–414), cardiac dysrhythmias (427), cardiac arrest (427.5), congestive heart failure (428), peripheral vascular and cerebrovascular disease (433-437, 440, 443-444, 451–453), atherosclerosis (440), and stroke (436). **Age Groups:** All **Study Design:** Time series **N:** 4,407,535 emergency department visits for 1993–2000 (data not reported for 1998-2000) **Statistical Analyses:** Poisson generalized linear modeling **Covariates:** Day of the week, hospital entry and exit indicator variables, federally observed holidays, temporal trends, temperature, dew point temperature **Season:** All **Dose-response Investigated:** No **Statistical Package:** SAS **Lags Considered:** 3-day moving avg, lags 0 -7	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h Median $\mu g/m^3$ (10%-90% range): $PM_{2.5}$: 17.8 (8.9, 32.3) $PM_{2.5}$ water soluble metals: 0.021 (0.006–0.061) $PM_{2.5}$ acidity: 4.5 (1.9–1.07) $PM_{2.5}$ organic carbon: 0.010 (-0.001–0.045) $PM_{2.5}$ elemental carbon: 4.1 (2.2–7.1) **Monitoring Stations:** 1 **Copollutant (correlation):** PM_{10}: r = 0.84 O_3: r = 0.65 NO_2: r = 0.46 CO: r = 0.44 SO_2: r = 0.17 $PM_{10-2.5}$: r = .43 UFP: r = -0.16 $PM_{2.5}$ water-sol metals: r = 0.70 $PM_{2.5}$ sulfates: r = 0.77 $PM_{2.5}$ acidity: r = 0.58 $PM_{2.5}$ organic carbon: r = 0.51 $PM_{2.5}$ elemental carbon: r = 0.48 oxygenated hydrocarbon: r = 31 Other variables: Temperature: r = 0.20 Dew point: r = 0.00	**PM Increment:** Approximately 1 SD increase: $PM_{2.5}$: 10 $\mu g/m^3$ $PM_{2.5}$ water-sol metals: 0.03 $\mu g/m^3$ $PM_{2.5}$ sulfates: 5 $\mu g/m^3$ $PM_{2.5}$ acidity: 0.02 $\mu equ/m^3$ $PM_{2.5}$ organic carbon: 2 $\mu g/m^3$ $PM_{2.5}$ elemental carbon: 1 $\mu g/m^3$ RR [95% CI]: $PM_{2.5}$ (3-day moving avg): All CVD: 1.033 [1.010, 1.056] Dysrhythmia: 1.015 [0.976, 1.055] Congestive heart failure: 1.055 [1.006–1.105] Ischemic heart disease: 1.023 [0.983–1.064] Peripheral vascular and cerebrovascular disease: 1.050 [1.008–1.093] $PM_{2.5}$ water soluble metals (3-day moving avg): All CVD: 1.027[0.998, 1.056] Dysrhythmia: 1.031 [0.982, 1.082] Congestive heart failure: 1.040 [0.981–1.103] Ischemic heart disease: 1.000 [0.951–1.051] Peripheral vascular and cerebrovascular disease: 1.043 [0.991–1.098] $PM_{2.5}$ sulfates (3-day moving avg): All CVD: 1.003 [0.968, 1.039] Dysrhythmia: 0.986 [0.926, 1.048] Congestive heart failure: 1.009 [0.938–1.085] Ischemic heart disease: 0.997 [0.936–1.062] Peripheral vascular and cerebrovascular disease: 1.025 [0.964–1.090] $PM_{2.5}$ acidity (3-day moving avg): All CVD: 0.994 [0.966, 1.022] Dysrhythmia: 0.991 [0.942, 1.043] Congestive heart failure: 0.989 [0.930–1.052] Ischemic heart disease: 0.992 [0.944–1.043] Peripheral vascular and cerebrovascular disease: 1.004 [0.955–1.056] $PM_{2.5}$ organic carbon (3-day moving avg): All CVD: 1.026 [1.006, 1.046] Dysrhythmia: 1.008 [0.975, 1.044] Congestive heart failure: 1.048 [1.007–1.091] Ischemic heart disease: 1.028 [0.994–1.064] Peripheral vascular and cerebrovascular disease: 1.026 [0.990–1.062] hydrocarbons simultaneously. $PM_{2.5}$ organic carbon (3-day moving avg): All CVD: 1.020 [1.005, 1.036] Dysrhythmia: 1.011 [0.985, 1.037] Congestive heart failure: 1.035 [1.003–1.068] Ischemic heart disease: 1.019 [0.992–1.046] Peripheral vascular and cerebrovascular

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Peng et al. (2008, 156850) **Period of Study:** January 1, 1999–December 31, 2005 **Location:** 108 U.S. counties in the following states: Alabama, Arizona, California, Colorado, Connecticut, District of Columbia, Florida, Georgia, Idaho, Illinois, Indiana, Kentucky, Louisiana, Maine, Maryland, Massachusetts, Michigan, Minnesota, Missouri, Nevada, New Hampshire, New Jersey, New Mexico, New York, North Carolina, Ohio, Oklahoma, Oregon, Pennsylvania, Rhode Island, South Carolina, Tennessee, Texas, Utah, Virginia, Washington, West Virginia, Wisconsin	**Outcome (ICD-9):** Emergency hospitalizations for: Cardiovascular disease, including heart failure (428), heart rhythm disturbances (426–427), cerebrovascular events (430–438), ischemic heart disease (410–414, 429), and peripheral vascular disease (440–448). Respiratory disease, including COPD (490–492) and respiratory tract infections (464–466, 480-487) **Age Groups:** 65 + years, 65–74, ,75 + **Study Design:** Time series **N:** ~12 million Medicare enrollees (3.7 million CVD and 1.4 million RD admissions) **Statistical Analyses:** Two-stage Bayesian hierarchical models: Overdispersed Poisson models for county-specific data Bayesian hierarchical models to obtain national avg estimate **Covariates:** Day of the week, age-specific intercept, temperature, dew point temperature, calendar time, indicator for age of 75 years or older. Some models were adjusted for PM_{10}-2.5. **Season:** NR **Dose-response Investigated:** No **Statistical Package:** R version 2.6.2 **Lags Considered:** 0-2 days	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h Mean $\mu g/m^3$ (IQR): All counties assessed: 13.5 (11.1–15.8) Counties in Eastern US: 13.8 (12.3–15.8) Counties in Western US: 11.1 (10.1–14.3) **Monitoring Stations:** At least 1 pair of co-located monitors (physically located in the same place) for PM_{10} and $PM_{2.5}$ per county Other variables: Median within-county correlations between monitors: r = 0.92	**PM Increment:** 10 $\mu g/m^3$ Percentage change [95% CI]: CVD and RD (unadjusted for PM_{10}-25): Lag 0: 0.71 [0.45, 0.96] Lag 2: 0.44 [0.06, 0.82] Most values NR (see note) Notes: Effect estimates for PM_{10}-2.5 (0–2 day lags) are showing in Figures 2–5. Figure 2: Percentage change in emergency hospital admissions for CVD per 10 $\mu g/m^3$ increase in $PM_{2.5}$ (single pollutant model and model adjusted for PM_{10}-2.5 concentration) Figure 3: Percentage change in emergency hospital admissions for RD per 10 $\mu g/m^3$ increase in $PM_{2.5}$ (single pollutant model and model adjusted for PM_{10}-2.5 concentration) No significant associations between $PM_{2.5}$ and cause-specific cardiovascular disease.
Reference: Peters et al. (2005, 156859) **Period of Study:** February 1999–July 31, 2001 **Location:** Germany: City of Augsburg, County Augsburg, and County Aichach-Friedlberg	**Outcome:** Transmural or nontransmural acute MI **Age Groups:** NR **Study Design:** Case-crossover and time series **N:** 851 MI survivors **Statistical Analyses:** Conditional logistic regression for case-crossover element. Poisson regression for time series element. **Covariates:** Case-crossover: Season, temperature, day of the week, time series: trend, season, influenza, weather, and day of the week **Season:** All **Dose-response Investigated:** No **Statistical Package:** SAS, version 8.2 Poisson: R, version 1.7.1 **Lags Considered:** Lags 0–6 h, 0–5 days Poisson: Single lagged days, 5-day, 15-day, 30-day, and 45-day moving averages	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 1 h and 24 h Mean $\mu g/m^3$ (range IQR median IQR): 1-h avg: 16.3 (-6.9–355.2 10.7–19.8 14.5) 24-h avg: 16.3 (6.1–58.5 11.6–19.3 14.9) **Monitoring Stations:** 1 **Copollutant (correlation):** 24-h avg: TNC: r = 0.37 TSP: r = 0.89 PM_{10}: r = 0.92 CO: r = 0.57 NO_2: r = 0.67 NO: r = 0.59 SO_2: r = 0.58 O_3: r = -0.24 1hr avg:	**PM Increment:** 1-h avg: 9.1 $\mu g/m^3$ (IQR) 24-h avg: 7.7 $\mu g/m^3$ (IQR) OR [95% CI]: Case-Crossover (control selection method (unidirectional with three control periods): **1-h averages:** Lag 0: 0.98 (0.88, 1.10) Lag 1: 0.97 (0.87, 1.09) Lag 2: 0.93 (0.83, 1.04) Lag 3: 0.98 (0.88, 1.09) Lag 4: 0.96 (0.86, 1.07) Lag 5: 0.94 (0.84, 1.05) Lag 6: 0.90 (0.80, 1.01). **24-h averages:** Lag 0: 0.95 (0.83, 1.080) Lag 1: 1.10 (0.96, 1.25) Lag 2: 1.18 (1.03, 1.34) Lag 3: 1.07 (0.94, 1.22) Lag 4: 0.94 (0.83, 1.07) Lag 5: 0.90 (0.79, 1.02) Case-Crossover (control selection method: bidirectional with 16 control periods): 24-h averages: Lag 0: 1.03 (0.94, 1.12) Lag 1: 1.07 (0.98, 1.16) Lag 2: 1.08 (0.99, 1.17) Lag 3: 1.01 (0.92, 1.10) Lag 4: 0.96 (0.88, 1.04)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
		TNC: r = 0.42	Lag 5: 0.93 (0.85, 1.02)
		CO: r = 0.52	Lag 0 -4 (IQR = 5.8): 1.03 (0.94, 1.14)
		NO_2: r = 0.58	Unidirectional: Model 1 (unadjusted): 1.175 (1.033, 1.337)
		NO: r = 0.50	Model 2 (adjusted for day of week using indicator variables): 1.179 (1.035, 1.343)
		SO_2: r = 0.48	Model 3 (adjusted for temperature-quadratic, linear air pressure): 1.170 (1.028, 1.333)
		O_3: r = -0.35	Model 4 (adjusted for temperature-quadratic, linear air pressure, day of week): 1.176 (1.031, 1.341)
		Other variables:	Model 5 (temperature-quadratic, air pressure-quadratic, relative humidity-quadratic, day of week using indicator variables): 1.170 (1.026, 1.336)
		24-h avg: Temperature: r = 0.05	Model 6 (temperature–penalized spline, 4.4 df, linear air pressure, day of week using indicator variables): 1.175 (1.030, 1.340
		1-h avg: Temperature: r = -0.01	Model 7 (temperature–penalized spline, 4.4 df, linear air pressure, relative humidity–penalized spline, 7.8 df, day of week using indicator variables: 1.177 (1.030, 1.344)
			Bidirectional (16 control periods): Model 1 (unadjusted): 1.077 (0.988, 1.174)
			Model 2 (adjusted for day of the week using indicator variables): 1.078 (0.988, 1.175)
			Model 3 (adjusted for temperature-quadratic, linear air pressure): 1.060 (0.970, 1.160)
			Model 4 (adjusted for temperature-quadratic, linear air pressure, day of the week): 1.060 (0.969, 1.160)
			Model 5
			--------Continued on next page--------
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			(temperature-quadratic, air pressure-quadratic, relative humidity-quadratic, day of the week using indicator variables): 1.065 (0.973, 1.166)
			Model 6 (temperature–penalized spline, 4.4 df, linear air pressure, day of the week using indicator variables): 1.068 (0.976, 1.168)
			Model 7 (temperature–penalized spline, 4.4 df, linear air pressure, relative humidity–penalized spline, 7.8 df, day of the week using indicator variables: 1.077 (0.983, 1.179)
			Bidirectional (4 control periods): Model 1 (unadjusted): NR
			Model 2 (adjusted for day of the week by design): 1.049 (0.964, 1.141)
			Model 3 (adjusted for temperature-quadratic, linear air pressure): NR
			Model 4 (adjusted for temperature-quadratic, linear air pressure, day of the week): 1.032 (0.944, 1.128)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			Model 5 (temperature-quadratic, air pressure-quadratic, relative humidity-quadratic, day of the week by design): 1.033 (0.945, 1.130) Model 6 (temperature–penalized spline, 4.4 df, linear air pressure, day of the week by design): 1.036 (0.947, 1.132) Model 7 (temperature–penalized spline, 4.4 df, linear air pressure, relative humidity–penalized spline, 7.8 df, day of the week by design): 1.039 (0.950, 1.136) Stratified: Model 1 (unadjusted): NR Model 2 (adjusted for day of week by design): 1.059 (0.972, 1.154) Model 3 (adjusted for temperature-quadratic, linear air pressure): NR Model 4 (adjusted for temperature-quadratic, linear air pressure, day of week): 1.047 (0.957, 1.145) Model 5 (temperature-quadratic, air pressure-quadratic, relative humidity-quadratic, day of week by design): 1.045 (0.954, 1.144) Model 6 (temperature–penalized spline, 4.4 df, linear air pressure, day of week by design): 1.054 (0.964, 1.153odel 7 (temperature–penalized spline, 4.4 df, linear air pressure, relative humidity–penalized spline, 7.8 df, day of week by design): 1.056 (0.965, 1.156) RR (95% CI): Time series (24 h avg): Lag 0: 0.97 (0.89, 1.07) Lag 1: 1.04 (0.96, 1.13) Lag 2: 1.07 (0.98, 1.15) Lag 3: 1.03 (0.95, 1.11) Lag 4: 0.98 (0.90, 1.07) Lag 5: 0.98 (0.90, 1.06) Lag 0–4: 1.03 (0.94, 1.12) Lag 0–14: 1.03 (0.95, 1.13) Lag 0–29: 1.09 (1.01, 1.18) Lag 0–44: 1.08 (1.00, 1.17) **Time series (OR [95% CI]):** Model 1 (unadjusted): 1.059 (0.981, 1.142) Model 2 (adjusted for day of week using indicator variables): 1.056 (0.979, 1.140) Model 3 (adjusted for temperature-quadratic, linear air pressure): 1.062 (0.982, 1.148) Model 4 (adjusted for temperature-quadratic, linear air pressure, day of week): 1.059 (0.979, 1.146) Model 5 (temperature-quadratic, air pressure-quadratic, relative humidity-quadratic, day of week using indicator variables): 1.063 (0.981, 1.151) Model 6 (temperature–penalized spline, 4.4 df, linear air pressure, day of week using indicator variables): 1.065 (0.985, 1.153)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			Model 7 (temperature–penalized spline, 4.4 df, linear air pressure, relative humidity–penalized spline, 7.8 df, day of week using indicator variables: 1.069 (0.988, 1.157)
Reference: Pope et al.(2006, 091246) **Period of Study:** 1994-2004 **Location:** Wasatch Front area, Utah	**Outcome:** Myocardial infarction or unstable angina (ICD codes not reported) **Age Groups:** All, < 65, 65 + **Study Design:** Case-crossover **N:** 12,865 patients who underwent coronary arteriography **Statistical Analyses:** Conditional logistic regression **Covariates:** Temperature and dew point temperature **Season:** NR **Dose-response Investigated:** No **Statistical Package:** NR **Lags Considered:** 0- to 3-day lag, 2- to 4-day lagged moving averages	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h Mean ($\mu g/m^3$) (SD maximum): Ogden: 10.8 (10.6 108) SLC Hawthorne: 11.3 (11.9 94) Provo/Orem, Lindom: 10.1 (9.8 82) **Monitoring Stations:** 3 **Copollutant (correlation):** NR	**PM Increment:** 10 $\mu g/m^3$ Percent increase in risk [95% CI]: Same-day increase in $PM_{2.5}$ (Lag 0): Index MI and unstable angina: 4.81 [0.98–8.79] Subsequent MI: 3.23 [-3.87, 10.85] All acute coronary events: 4.46 [1.07–7.97] All acute coronary events excluding observations using imputed $PM_{2.5}$ data: 4.24 [0.33–8.31] Stable presentation: -2.57 [-5.39, 0.34] Remaining results summarized in figures (see notes). Notes: Figure 1: Percent increase in risk (and 95% CI) of acute coronary events associated with 10 $\mu g/m^3$ of $PM_{2.5}$ for different lag structures. Summary of Figure 1: Positive, statistically significant association seen for Lag 0, Lag 1 and 2, 3, and 4 day moving averages. Positive but non-statistically significant associations seen for Lags 2 and 3. Figure 2: Percent increase in risk (and 95% CI) of acute coronary events associated with 10 $\mu g/m^3$ of $PM_{2.5}$ stratified by various characteristics.

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Pope et al. (2008, 191969) **Period of Study:** 1994-2006 **Location:** Ogden, Salt Lake City, & Provo/Orem, Utah	**Outcome:** Heart Failure Hospitalizations **Age Groups:** NR **Study Design:** case-crossover **N:** 2,618 **Statistical Analyses:** Conditional Logistic Regression **Covariates:** age, sex, length of stay, temperature, pressure, clearing index, day of the week, seasonality, and long-term trends **Season:** Adjusted for long-term trends to account for season **Dose-response Investigated?** No **Statistical Package:** NR **Lags Considered:** 0-28d moving avg.	**Pollutant:** $PM_{2.5}$ **Averaging Time:** NR **Mean (SD):** Ogden: 10.6(9.9) SLC, Hawthorne: 11.1 (11.2) Provo/Orem, Lindon: 10.1 (9.3) **Max:** Ogden: 108 SLC, Hawthorne: 94 Provo/Orem, Lindon: 82 **Monitoring Stations:** NR **Copollutant:** PM_{10}	**PM Increment:** 10μg/m^3 **Percent Increase: (Lower CI, Upper CI):** All HF Admissions All: 13.1 (1.3, 26.2)* Men: 13.4 (-1.7, 30.7)‡ Women: 12.7 (-5.1, 33.9) Age < 65 yrs: 3.5 (-13.5, 23.8) Age ≥65 yrs: 19.6 (4.0, 37.5)* Length of stay 0-2 days: 24.4 (-0.8, 56.0) ‡ Length of stay 3-7 days: 10.8 (-4.6, 28.7) Length of stay 8+ days: 6.5 (-15.9, 34.8) First HF Admissions: 2.1 (-11.3, 17.5) Subsequent HF Admits: 32.4 (10.7, 58.4) † All HF Admissions All: 32.4 (10.7, 58.4) † Men: 29.2 (2.7, 62.6)* Women: 41.5 (5.4, 89.9)* Age < 65 yrs: -3.1 (-26.5, 27.8) Age ≥65 yrs: 64.1 (28.6, 109) † Length of stay 0-2 days: 68.9 (12.5, 154)* Length of stay 3-7 days: 35.7 (5.9, 73.9)* Length of stay 8+ days: 2.6 (-28.5, 47.1) *p < 0.05, † p < 0.01, ‡p < 0.10

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Sarnat et al. (2008, 097972) **Period of Study:** November 1998–December 2002 **Location:** Atlanta (Georgia) metropolitan area	**Outcome (ICD-9):** Cardiovascular disease ED visits: Ischemic heart disease (410–414), cardiac dysrhythmias (427), congestive heart failure (428), and peripheral vascular and cerebrovascular disease (433–437, 440, 443–444, 451-453) **Age Groups:** All **Study Design:** Time series **N:** >4.5 million emergency department visits **Statistical Analyses:** Poisson generalized linear models **Covariates:** Day of the week, holidays, hospital, long-term trends, temperature, dew point temperature **Season:** All, warm season (April 15–October 14), and cool season (October 15–April 14). **Dose-response Investigated:** No **Statistical Package:** NR **Lags Considered:** 0-day lag	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h Mean ($\mu g/m^3$) (median 10th-90th percentile): Total $PM_{2.5}$: Cool season: 15.8 (14.3 7.5–25.5). Warm season: 18.2 (17.0 9.1–29.0) $PM_{2.5}$ elemental carbon: Cool: 1.7 (1.4 0.6–3.3). Warm: 1.4 (1.3 0.6–2.5) $PM_{2.5}$ Zn (ng/m^3): Cool: 15.7 (11.7 4.6–30.2) Warm: 10.9 (8.5 3.3–20.2) $PM_{2.5}$ K (ng/m^3): Cool: 63.0 (53.9 24.3–114.2) Warm: 52.7 (43.3 23.2–93.5) $PM_{2.5}$ Si (ng/m^3): Cool: 67.7 (54.1 24.3–123.5). Warm: 110.9 (89.0 32.9–186.3) $PM_{2.5}$ SO_4^{2-}: Cool: 3.4 (0.6 1.5–5.8). Warm: 6.0 (5.2 2.3–10.8) $PM_{2.5}$ NO_{3-}: Cool: 1.4 (1.2 0.5–2.6). Warm: 0.7 (2.9 0.3–1.2) $PM_{2.5}$ Se (ng/m^3): Cool: 1.4 (1.1 0.4–3.0). Warm: 1.2 (0.9 0.4–2.7) $PM_{2.5}$ OC: Cool: 4.6 (3.9 1.9–8.0) Warm: 4.0 (3.7 2.1–6.4) **Monitoring Stations:** 1 **Copollutants:** NR	**PM Increment:** IQR (specific values not given) Risk ratio [95% CI]: CVD (Lag 0): All seasons: Total $PM_{2.5}$: 1.022 [1.007, 1.038] $PM_{2.5}$ elemental carbon: 1.02 [1.013–1.037] $PM_{2.5}$ zinc: 1.013 [1.005–1.022] $PM_{2.5}$ potassium: 1.030 [1.018–1.042] $PM_{2.5}$ silicon: 1.008 [1.00–1.016] $PM_{2.5}$ sulfate: 1.007 [0.994–1.019] $PM_{2.5}$ nitrate: 1.002 [0.990–1.014] $PM_{2.5}$ selenium: 1.002 [0.991–1.012] $PM_{2.5}$ organic carbon: 1.024 [1.013–1.035] Cool season: Total $PM_{2.5}$: 1.028 [1.012–1.044] $PM_{2.5}$ EC: 1.029 [1.015–1.044] $PM_{2.5}$ Zinc: 1.012 [1.002–1.022] $PM_{2.5}$ K: 1.037 [1.021–1.054] $PM_{2.5}$ Si: 1.022 [1.002–1.043] $PM_{2.5}$ sulfate: 1.014 [0.991–1.037] $PM_{2.5}$ nitrate: 1.006 [0.993–1.019] $PM_{2.5}$ Se: 1.012 [0.997–1.027] $PM_{2.5}$ organic carbon: 1.027 [1.013–1.040] Warm season: Total $PM_{2.5}$: 1.006 [0.990–1.022] $PM_{2.5}$ EC: 1.021 [1.000–1.043] $PM_{2.5}$ Zinc: 1.017 [1.002–1.033] $PM_{2.5}$ K: 1.024 [1.007–1.041] $PM_{2.5}$ Si: 1.005 [0.996–1.014] $PM_{2.5}$ sulfate: 1.001 [0.988–1.015] $PM_{2.5}$ nitrate: 1.000 [0.969–1.033] $PM_{2.5}$ Se: 0.996 [0.981–1.011] $PM_{2.5}$ organic carbon: 1.027 [1.004–1.051]

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Schreuder et al. (2006, 097959) **Period of Study:** Sept 1995 – May 2002 **Location:** Spokane, Washington	**Outcome:** Cardiac HA **Age Groups:** NR **Study Design:** time series **Statistical Analyses:** GAM Poisosn Regression **Covariates:** season, temperature, relative humidity, day of week **Dose-response Investigated?** No **Statistical Package:** S-Plus **Lags Considered:** 0-1d	**Pollutant:** $PM_{2.5}$ (ng/m^3) **Averaging Time:** 24h **Arithmetic Mean:** 10,580 **Geometric Mean:** 8,790 **Min:** 930 **Max:** 43,230 **IQR:** entire period: 7.7 $\mu g/m^3$ heating season: 10.1$\mu g/m^3$ non-heating season: 5.5$\mu g/m^3$ **Monitoring Stations:** NR **Copollutant:** NR **Co-pollutant Correlation** NR	**PM Increment:** Interquartile Range **Relative Risk (Lower CI, Upper CI):** Entire Period, Lag 0: 1.008 (0.985, 1.032) Entire Period, Lag 1: 1.000 (0.978, 1.023) Heating Season, Lag 1: 1.015 (0.968, 1.063) Non-Heating Season, Lag 1: 0.995 (0.969, 1.021)
Reference: Sullivan et al. (2005, 109418) **Period of Study:** 1988-1994 **Location:** King County, Washington	**Outcome:** Acute MI **Age Groups:** All, < 50, 50–59, 70+ **Study Design:** Case-crossover **N:** 5793 cases of acute MI (5793 case days and 20,134 referent exposure days from these case individuals) **Statistical Analyses:** Conditional logistic regression **Covariates:** Relative humidity, temperature, season, day of week **Season:** All, and also conducted stratified analysis by season of event (heating season: November–February nonheating season: March-October) **Dose-response Investigated:** No **Statistical Package:** SAS version 8.0 and SPSS version 10 **Lags Considered:** Lag 1 and Lag 2 for 24-h avg	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 1 h, 2 h, 4 h, and 24 h Summary of $PM_{2.5}$ 1 h before MI onset: Mean ($\mu g/m^3$) (median IQR, 90th percentile range): 12.8 (8.6 5.3–15.9 27.3 2.0–147) **Monitoring Stations:** 3 **Copollutant (correlation):** 1-h avg: PM_{10}: r = 0.78 CO: r = 0.47 SO_2: r = 0.16	**PM Increment:** 10 $\mu g/m^3$ **Odds ratio [95% CI]:** **1-h Averaging Time:** 1.01 [0.98, 1.05] **2-h Averaging Time:** 1.01 [0.97, 1.05] **4-h Averaging Time:** 1.02 [0.98, 1.04] **24-h Averaging Time:** 1.02 [0.98, 1.07] Association between $PM_{2.5}$ (24 h) lagged 1 or 2 days non-significant (data not shown) Season (1-h avg): Heating: 1.01 [0.98–1.05] Nonheating: 0.99 [0.91–1.09] Age (1-h avg): < 50 years: 1.04 [0.95, 1.14] 50–60 years: 0.99 [0.94, 1.05] 70+ years: 1.03 [0.98, 1.08] Age (24-h avg): < 50 years: 1.07 [0.98, 1.19] 50–69 years: 0.99 [0.93, 1.06] 70+ years: 1.04 [0.99, 1.11] Sex (1-h avg): Men: 1.02 [0.98, 1.06] Women: 1.00 [0.95, 1.06] Sex (24-h avg): Men: 1.03 [0.99, 1.08] Women: 1.00 [0.94, 1.07] Race (1-h avg): White: 1.01 [0.97, 1.04] Nonwhite: 1.06 [0.97, 1.17] Race (24-h avg): White: 1.01 [0.97, 1.06] Nonwhite: 1.10 [0.99, 1.23] Smoking status (1-h avg): Current: 0.99 [0.93, 1.06] Nonsmoker: 1.03 [0.97, 1.08] Smoking status (24-h avg): Current: 0.99 [0.95, 1.14]

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			Nonsmoker: 1.03 [0.98, 1.09] Survivor of MI * (1-h avg): Yes: 1.02 [0.98, 1.06] No: 0.96 [0.86, 1.08] Survivor of MI * (24-h avg): Yes: 1.03 [0.98, 1.07] No: 0.97 [0.85, 1.10] Previous congestive heart failure (1 h avg): Yes: 1.06 [0.97, 1.16] No: 1.00 [0.97, 1.04] Previous congestive heart failure (24-h avg): Yes: 1.08 [0.97, 1.2] No: 1.00 [0.97, 1.04] Previous MI (1-h avg): Yes: 1.03 [0.97, 1.1] No: 1.01 [0.96, 1.06] Previous MI (24-h avg): Yes: 1.04 [0.97, 1.17] No: 1.02 [0.98, 1.08] Hypertension (1-h avg): Yes: 1.02 [0.97, 1.07] No: 1.01 [0.96, 1.06] Hypertension (24-h avg): Yes: 1.02 [0.97, 1.07] No: 1.02 [0.97, 1.08] Diabetes mellitus (1-h avg): Yes: 1.06 [0.98, 1.14] No: 1.01 [0.97, 1.05] Diabetes mellitus (24-h avg): Yes: 1.04 [0.95, 1.14] No: 1.01 [0.97, 1.06] *Compares those who survive hospitalization (yes) with those who died in hospital from complications of MI.

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Symons et al. (2006, 091258) **Period of Study:** Apr–Dec, 2002 **Location:** Baltimore, Maryland	**Outcome:** Congestive heart failure **Age Groups:** All **Study Design:** Case-crossover **N:** 125 patients **Statistical Analyses:** Conditional logistic regression **Covariates:** Temperature and humidity **Season:** NR **Dose-response Investigated:** Yes **Statistical Package:** SAS and S-Plus **Lags Considered:** 0-3 days (single and cumulative)	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 8 & 24 h **Mean (min-max):** 8 h 17.0 (0.1-111.9) SD = 12.7 24 h 16.0 (3.5-69.2) SD = 10.0 **Monitoring Stations:** 8 **Copollutant (correlation):** NR	**PM Increment:** 9.2 $\mu g/m^3$ (IQR) RR Estimate [CI]: 8 h (participant's onset period) Same-day lag: 0.87 [0.69,1.09] 1-day lag: 0.96 [0.78,1.18] 2-day lag: 1.09 [0.91,1.30] 3-day lag: 0.99 [0.79,1.23] Cumulative 1-day lag: 0.89 [0.67,1.16] Cumulative 2-day lag: 0.99 [0.74,1.33] Cumulative 3-day lag: 0.98 [0.70,1.36] 24 h avg Same-day lag: 0.81 [0.65,1.01] 1-day lag: 0.90 [0.74,1.11] 2-day lag: 0.85 [0.68,1.07] 3-day lag: 0.86 [0.70,1.05] Cumulative 1-day lag: 0.82 [0.64,1.04] Cumulative 2-day lag: 0.76 [0.57,1.01] Cumulative 3-day lag: 0.70 [0.51,0.97] Notes: β coefficients presented in Fig 5

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Tolbert et al. (2007, 090316) **Period of Study:** August 1998–December 2004 **Location:** Atlanta Metropolitan area, Georgia	**Outcome (ICD-9):** Combined CVD group, including: Ischemic heart disease (410–414), cardiac dysrhythmias (427), congestive heart failure (428), and peripheral vascular and cardiovascular disease (433–437, 440, 443–445, and 451–453) **Age Groups:** All **Study Design:** Time series **N:** NR for 1998–2004. For 1993–2004: 10,234,490 ER visits (283,360 and 1,072,429 visits included in the CVD and RD groups, respectively) **Statistical Analyses:** Poisson generalized linear models **Covariates:** long-term temporal trends, season (for RD outcome), temperature, dew point, days of week, federal holidays, hospital entry and exit **Season:** All **Dose-response Investigated:** No **Statistical Package:** SAS version 9.1 **Lags Considered:** 3-day moving avg(lag 0 -2)	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h Mean (**$\mu g/m^3$**) (median IQR, range, 10^{th} –90^{th} percentiles): $PM_{2.5}$: 17.1 (15.6 11.0–21.9 0.8–65.8 7.9–28.8). $PM_{2.5}$ sulfate: 4.9 (3.9 2.4–6.2 0.5–21.9 1.7–9.5). $PM_{2.5}$ organic carbon: 4.4 (3.8 2.7–5.3 0.4–25.9 2.1–7.2). $PM_{2.5}$ elemental carbon: 1.6 (1.3 0.9–2.0 0.1–11.9 0.6–3.0). $PM_{2.5}$ water-soluble metals: 0.030 (0.023 0.014–0.039 0.003–0.202 0.009–0.059) **Monitoring Stations:** 1 **Copollutant (correlation):** Between $PM_{2.5}$ and: PM_{10}: r = 0.84 O_3: r = 0.62 NO_2: r = 0.47 CO: r = 0.47 SO_2: r = 0.17 PM_{10}-2.5: r = 0.47 $PM_{2.5}$ SO_4: r = 0.76 $PM_{2.5}$ EC: r = 0.65 $PM_{2.5}$ OC: r = 0.70 $PM_{2.5}$ TC: r = 0.71 $PM_{2.5}$ water-sol metals: r = 0.69 OHC: r = 0.50 Between $PM_{2.5}$ SO_4 and: PM_{10}: r = 0.69 O_3: r = 0.56 NO_2: r = 0.14 CO: r = 0.14 SO_2: r = 0.09 PM_{10}-2.5: r = 0.32 $PM_{2.5}$: r = 0.76 $PM_{2.5}$ EC: r = 0.32 $PM_{2.5}$ OC: r = 0.33 $PM_{2.5}$ TC: r = 0.34 $PM_{2.5}$ water-sol metals: r = 0.65 OHC: r = 0.47 Between $PM_{2.5}$ elemental carbon and: PM_{10}: r = 0.61 O_3: r = 0.40	**PM Increment:** $PM_{2.5}$: 10.96 $\mu g/m^3$ (IQR) $PM_{2.5}$ sulfate: 3.82 $\mu g/m^3$ (IQR) $PM_{2.5}$ total carbon: 3.63 $\mu g/m^3$ (IQR) $PM_{2.5}$ organic carbon: 2.61 $\mu g/m^3$ (IQR) $PM_{2.5}$ elemental carbon: 1.15 $\mu g/m^3$ (IQR) $PM_{2.5}$ water-soluble metals: 0.03 $\mu g/m^3$ (IQR) Risk ratio [95% CI] (single pollutant models): $PM_{2.5}$: CVD: 1.005 [0.993–1.017] $PM_{2.5}$ sulfate: CVD: 0.999 [0.987–1.011] $PM_{2.5}$ total carbon: CVD: 1.016 [1.005–1.026] $PM_{2.5}$ organic carbon: CVD: 1.015 [1.005–1.026] $PM_{2.5}$ elemental carbon: CVD: 1.015 [1.005–1.025] $PM_{2.5}$ water-soluble metals: CVD: 1.009 [0.997–1.021] Notes: Results of selected multi-pollutant models for cardiovascular disease are presented in Figure 1. Figure 1: $PM_{2.5}$ total carbon adjusted for CO, NO_2, or NO_2 + CO Summary of results: $PM_{2.5}$ total carbon continued to have a positive, statistically significant association with CVD after adjustment for NO_2 but not after adjustment

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Villeneuve et al. (2006, 090191)	**Outcome (ICD-9):** Stroke (430-438), including ischemic stroke (434-436), hemorrhagic stroke (430,432), and transient ischemic attacks (TIA) (435).	**Pollutant:** $PM_{2.5}$	**PM Increment:** $\mu g/m^3$ (IQR)
Period of Study: 1 Apr, 1992–31 Mar, 2002	**Age Groups:** 65+ yrs	**Averaging Time:** 24 h	All year: 6.3
Location: Edmonton, Canada	**Study Design:** Case-crossover	Mean $\mu g/m^3$ (SD):	Summer: 6.5
	N: 12,422 visits	All year: 8.5 (6.2)	Winter: 6.0
	Statistical Analyses: Conditional logistic regression	Summer: 8.7 (7.1)	Adjusted OR Estimate [CI]:
	Covariates: Temperature and relative humidity	Winter: 8.3 (5.2)	Acute ischemic stroke
	Season: Summer (Apr-Sep), winter (Oct-Mar)	**Monitoring Stations:** 3	All year: Same-day lag: 1.00 [0.96,1.04]
	Dose-response Investigated: No	**Copollutant (correlation):**	1-day lag: 1.00 [0.96,1.05]
	Statistical Package: SAS (PHREG)	All year	3-day lag: 1.01 [0.96,1.06]
	Lags Considered: 0, 1, and 3-day	SO_2: r = 0.22	Summer: Same-day lag: 0.96 [0.90,1.03]
		NO_2: r = 0.41	1-day lag: 1.01 [0.94,1.07]
		CO: r = 0.43	3-day lag: 0.98 [0.89 [1.07]
		O_3-mean: r = -0.07	Winter: Same-day lag: 1.04 [0.99,1.10]
		O_3-max: r = 0.07	1-day lag: 1.01 [0.96,1.07]
		PM_{10}: r = 0.79	3-day lag: 1.05 [0.98,1.13]
		Summer	Hemorrhagic stroke
		SO_2: r = 0.20	All year: Same-day lag: 0.99 [0.90,1.08]
		NO_2: r = 0.52	1-day lag: 1.07 [0.98,1.16]
		CO: r = 0.42	3-day lag: 1.05 [0.93,1.19]
		O_3-mean: r = 0.11	Summer: Same-day lag: 0.99 [0.86,1.15]
		O_3-max: r = 0.34	1-day lag: 1.12 [0.97,1.30]
		PM_{10}: r = 0.85	3-day lag: 1.08 [0.88,1.31]
		Winter	Winter: Same-day lag: 1.04 [0.92,1.18]
		SO_2: r = 0.28	1-day lag: 1.08 [0.97,1.20]
		NO_2: r = 0.57	3-day lag: 1.11 [0.94,1.31]
		CO: r = 0.71	Transient cerebral ischemic attack
		O_3-mean: r = -0.45	All year: Same-day lag: 0.98 [0.93,1.03]
		O_3-max: r = -0.35	1-day lag: 0.99 [0.95,1.04]
		PM_{10}: r = 0.70	3-day lag: 0.96 [0.90,1.03]
			Summer: Same-day lag: 1.00 [0.92,1.08]
			1-day lag: 1.03 [0.95,1.12]
			3-day lag: 0.98 [0.88,1.09]
			Winter: Same-day lag: 0.97 [0.90,1.05]
			1-day lag: 0.97 [0.91,1.04]
			3-day lag: 0.94 [0.86,1.03]
			Notes: Adjusted ORs are provided for an IQR increase in the 3-day mean in Fig 1-4 for single and two-pollutant models.

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Zanobetti and Schwartz (2006, 090195) **Period of Study:** 1995-1999 **Location:** Boston Metropolitan area	**Outcome (ICD-9):** Myocardial infarction (410) or pneumonia (480–487) **Age Groups:** 65 + years **Study Design:** Case-crossover **N:** 15,578 patients admitted for MI and 25,857 admitted for pneumonia **Statistical Analyses:** conditional logistic regression **Covariates:** temperature, day of the week. **Season:** All, and also tested for interaction by warm (April–September) vs. cold season **Dose-response Investigated:** No **Statistical Package:** SAS version 8.2 (PROC PHREG) **Lags Considered:** lag 0 , and mean of lags 0 -1	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h Median ($\mu g/m^3$) (IQR 5th-95th percentile): 11.1 (7.23-16.14 3.87–26.31) **Monitoring Stations:** 1 **Copollutant (correlation):** BC: r = 0.66 NO_2: r = 0.55 CO: r = 0.52 O_3: r = 0.20 PM non-traffic: r = 0.74	**PM Increment:** Difference between the 90th and 10th percentile for $PM_{2.5}$ Myocardial infarction cohort (Lag 0): 17.17 $\mu g/m^3$ Myocardial infarction cohort (Lag 0-1): 16.32 $\mu g/m^3$ Pneumonia cohort (Lag 0): 17.14 $\mu g/m^3$ Pneumonia cohort (Lag 0): 16.32 $\mu g/m^3$ Percentage (%) increase in risk [95% CI]: Myocardial infarction cohort: Lag 0: 8.50 (1.89–14.43) Lag 0–1: 8.65 (1.22–15.38) Pneumonia cohort: Lag 0: 6.48 (1.13–11.43) Lag 0–1: 5.56 (-0.45, 11.27) Notes: Assessed for effect modification by season. Results are reported in Figure 2. Summary of results: $PM_{2.5}$ is associated with pneumonia hospitalization in the cold season but not the hot season. $PM_{2.5}$ is associated with MI hospitalization in the hot season but not the cold season.
Reference: Zanobetti and Schwartz (2006, 090195) **Period of Study:** 1995–1999 **Location:** Boston Metropolitan area	**Outcome (ICD-9):** Myocardial infarction (410) or pneumonia (480–487) **Age Groups:** 65 + years Study Design: Case-crossover **N:** 15,578 patients admitted for MI and 25,857 admitted for pneumonia **Statistical Analyses:** conditional logistic regression **Covariates:** temperature, day of the week. **Season:** All, and also assessed for interaction by hot (April–September) vs. cold season **Dose-response Investigated:** No **Statistical Package:** SAS Software Release 8.2 **Lags Considered:** lag 0 , and mean of lags 0 -1	**Pollutant:** BC **Averaging Time:** 24 h Median ($\mu g/m^3$) (IQR 5th–95th percentiles): 1.15 (0.74–1.72 0.42–2.83) **Monitoring Stations:** 1 **Copollutant (correlation):** $PM_{2.5}$: r = 0.66 NO_2: r = 0.70 CO: r = 0.82 O_3: r = -0.25 PM non-traffic: r = -0.01	**PM Increment:** Difference between the 90th and 10th percentile for BC Myocardial infarction cohort (Lag 0): 2.01 $\mu g/m^3$ Myocardial infarction cohort (Lag 0-1): 1.69 $\mu g/m^3$ Pneumonia cohort (Lag 0): 2.05 $\mu g/m^3$ Pneumonia cohort (Lag 0 -1): 1.69 $\mu g/m^3$ Percentage (%) increase in risk [95% CI]: Myocardial infarction cohort: Lag 0: 6.98 (-0.27–13.76) Lag 0–1: 8.34 (0.21–15.82) Pneumonia cohort: Lag 0: 10.76 (4.54–15.89) Lag 0–1: 11.71 (4.79, 17.36) Notes: Assessed for effect modification by season. Results are reported in Figure 2. Summary of results: PM2.BC is associated with pneumonia hospitalization in the cold season but not the hot season. BC had a stronger positive association with MI hospitalization in the cold season, but the confidence interval was wide.

[1]All units expressed in $\mu g/m^3$ unless otherwise specified.

Table E-8. Short-term exposure-cardiovascular–ED/HA-other size fractions.

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Andersen et al, (2008, 189651)	**Outcome (ICD-10):** CVD, including angina pectoris (I20), myocardial infarction (I21–22), other acute ischemic heart diseases	**Pollutant:** Total number concentration of ultrafine and accumulation mode particles	**PM Increment:** IQR increase in pollutant level: Nctot: 3907 particles/cm^3 (IQR)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Period of Study: May 2001-December 2004 **Location:** Los Angeles and San Diego counties, California	(I24), chronic ischaemic heart disease (I25), pulmonary embolism (I26), cardiac arrest (I46), cardiac arrhythmias (I48–48), and heart failure (I50). RD, including chronic bronchitis (J41–42), emphysema (J43), other chronic obstructive pulmonary disease (J44), asthma (J45), and status asthmaticus (J46). Pediatric hospital admissions for asthma (J45) and status asthmaticus (J46). **Age Groups:** > 65 yrs (CVD and RD), 5–18 years (asthma) **Study Design:** Time series **N:** NR **Statistical Analyses:** Poisson GAM **Covariates:** temperature, dew-point temperature, long-term trend, seasonality, influenza, day of the week, public holidays, school holidays (only for 5–18 year olds), pollen (only for pediatric asthma outcome) **Season:** NR **Dose-response Investigated:** No **Statistical Package:** R statistical software (gam procedure, mgcv package) **Lags Considered:** Lag 0 -5 days, 4-day pollutant avg (lag 0 -3) for CVD, 5-day avg (lag 0–4) for RD, and a 6-day avg (lag 0–5) for asthma.	(NCtot) (particles/cm^3) **Averaging Time:** 24 h Mean (SD median IQR 99th percentile: NCtot*: 8116 (3502 7358 5738–9645, 19,895) NCa12: 493 (315 463 308–650 1463) Nca23: 2253 (1364 2057 1280–3066 6096) NCa57: 5104 (2687 4562 3248–6274 14,410) NC100: 6847 (2864 6243 4959–8218 16189) NC_{a212}: 392 (441 89 246–584 2248) *NC, number concentration tot, total (all particles 6–700 in diameter) a12, size mode with mean diameter of 12 nm a23, size mode with median diameter of 23 nm a57, size mode with median diameter of 57 nm a212 size mode with median diameter of 212 nm NC100 = a12+a23+0.797*a57+0.084 *a212. **Monitoring Stations:** 1 **Copollutant (correlation):** Correlation of NCtot with: PM_{10}: r = 0.39 $PM_{2.5}$: r = 0.40 NO_2: r = 0.68	Nca12: 342 particles/cm^3 (IQR) Nca23: 1786 particles/cm^3 (IQR) Nca57: 3026 particles/cm^3 (IQR) NC100: 3259 particles/cm^3 (IQR) Nca212: 495 particles/cm^3 (IQR) Relative risk (RR) Estimate [CI]: CVD hospital admissions (4 day avg, lag 0 -3), age 65+ One-pollutant model (NCtot): 1.00 [0.99–1.02] Adj for PM_{10}: 0.98 [0.96–1.01] Adj for $PM_{2.5}$: 0.99 [0.95–1.03] Adj for CO: 0.99 [0.97–1.02] Adj for NO_2: 1.01 [0.98–1.03] Adj for O_3: 1.01 [0.96–1.06] One-pollutant model (NC100): 1.00 [0.98–1.02] One pollutant model (Nca12): 0.99 [.97–1.01] Adj for other size fractions: 0.99 [0.97–1.02] One pollutant model (Nca23): 0.99 [0.96–1.01] Adj for other size fractions: 0.99 [0.96–1.02] One pollutant model (NCa57): 1.01 [0.98–1.02] Adj for other size fractions: 0.99 [0.97–1.02] One pollutant model (Nca212): 1.02 [1.00–1.04] Adj for other size fractions: 1.02 [1.00–1.05] Adj for PM_{10}: 0.98 [0.95–1.01] RD hospital admissions (5 day avg, lag 0 -4), age 65+: One-pollutant model: 1.04 [1.00–1.07] Adj for PM_{10}: 1.00 [0.96–1.05] Adj for $PM_{2.5}$: 0.97 [0.89–1.05] Adj for CO: 1.03 [0.98–1.07] Adj for NO_2: 1.00 [0.95–1.05] Adj for O_3: 0.95 [0.87–1.04] One pollutant model (NC100): 1.03 [0.99–1.07] One pollutant model (Nca12): 1.01 [0.98–1.05] Adj for other size fractions: 1.01 [0.97–1.05] One pollutant model (Nca23): 0.99 [0.94–1.03] Adj for other size fractions: 0.98 [0.94–1.03] One pollutant model (Nca57): 1.04 [1.00–

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
		NO_X: r = 0.66 NC100: r = 0.98 NCa12: r = 0.31 NCa23: r = 0.57 NCa57: r = 0.87 NC_{a212}: r = 0.29 CO: r = 0.54 NO_X curbside: r = 0.36 O_3: r = -0.12 Other variables: Temperature: r = -0.06 Relative humidity: r = -0.04	1.08] Adj for other size fractions: 1.02 [0.97–1.06] One pollutant model (Nca212): 1.04 [1.01–1.08] Adj for other size fractions: 1.03 [0.99–1.07] Adj for PM_{10}: 1.01 [0.96–1.07] Asthma hospital admissions (6 day avg lag 0–5), age 5-18: One-pollutant model: 1.07 [0.98–1.17] Adj for PM_{10}: 1.03 [0.92–1.15] Adj for $PM_{2.5}$: 1.04 [0.85–1.28] Adj for CO: 1.09 [0.99–1.21] Adj for NO_2: 1.07 [0.96–1.19] Adj for O_3: 1.08 [0.87–1.35] One pollutant model (NC100): 1.06 [0.97–1.16] One pollutant model (Nca212): 1.08 [0.99–1.18] Adj for other size fractions: 1.07 [0.97–1.19] One pollutant model (Nca23): 1.09 [0.98–1.21] Adj for other size fractions: 1.08 [0.97–1.21] One pollutant model (Nca57): 1.02 [0.94–1.12] Adj for other size fractions: 0.93 [0.83–1.04] One pollutant model (Nca212): 1.08 [1.00–1.17] Adj for other size fractions: 1.12 [1.02–1.23] Adj for PM_{10}: 1.10 [0.96–1.13] Notes: Figure 2: Relative risks and 95% confidence intervals per IQR in single day concentration (0–5 day lag). Summary of Figure 2: CVD: Positive, marginally or statistically significant associations at Lag 2 (Nctot, Nca57, Nca212), Lag 3 (Nca212), and Lag 1 (Nca212). RD: Positive, statistically or marginally significant associations at Lag 4 (Nctot, Nca57, NCa212) and Lag 5 (Nctot, Nca57, Nca212), and to a lesser extent Lag 2 (Nctot, Nca212) and Lag 3 (Nctot, Nca212). Asthma: Wide confidence intervals make interpretation difficult. Positive, significant association for Nca212 at Lag 1.

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Lanki et al. (2006, 089788) **Period of Study:** 1992-2000 **Location:** Augsburg, Barcelona, Helsinki, Rome, and Stockholm	**Outcome (ICD-9):** Acute myocardial infarction (410 ICD-10: I21, I22) **Age Groups:** 35+ yrs, < 75 yrs, 75+ yrs **Study Design:** Time series **N:** 26,854 hospitalizations **Statistical Analyses:** GAM **Covariates:** Temperature, barometric pressure **Season:** Warm (Apr-Sep) and cold (Oct-Mar) **Dose-response Investigated:** No **Statistical Package:** R package mgcv 0.9-5 **Lags Considered:** 0-3 days	**Pollutant: UFP** (PNC) **Averaging Time:** 24 h Median particles/cm^3: Augsburg: 12,400 Barcelona: 76,300 Helsinki: 13,600 Rome: 46,000 Stockholm: 11,800 **Copollutant (correlation):** Augsburg PM_{10}: r = 0.53 CO: r = 0.63 NO_2: r = 0.65 O_3: r = 0.26 Barcelona: PM_{10}: r = 0.38 CO: r = 0.80 NO_2: r = 0.49 O_3: r = -0.35 Helsinki: PM_{10}: r = 0.45 CO: r = 0.48 NO_2: r = 0.82 O_3: r = 0.01 Rome: PM_{10}: r = 0.32 CO: r = 0.83 NO_2: r = 0.68 O_3: r = 0.03 Stockholm: PM_{10}: r = 0.06 CO: r = 0.56 NO_2: r = 0.83 O_3: r = -0.01	**PM Increment:** 10,000 particles/cm^3 Pooled Rate Ratio [CI]: All 5 cities (35+ yrs) Same-day lag: 1.005 [0.996,1.015] 1-day lag: 0.997 [0.982,1.012] 2-day lag: 0.999 [0.990,1.008] 3-day lag: 0.998 [0.979,1.017] 3 cities with hospital discharge register (35+ yrs) Same-day lag: 1.013 [1.000,1.026] 1-day lag: 0.995 [0.953,1.039] 2-day lag: 1.001 [0.989,1.014] 3-day lag: 1.009 [0.974,1.046] Warm season (35+ yrs) Same-day lag: 1.009 [0.972,1.048] 1-day lag: 1.023 [0.988,1.060]; 2-day lag: 1.050 [1.016,1.085] 3-day lag: 1.022 [0.987,1.058] Cold season (35+ yrs) Same-day lag: 1.014 [1.001,1.028] 1-day lag: 1.001 [0.956,1.048] 2-day lag: 1.001 [0.989,1.014] 3-day lag: 1.009 [0.971,1.049] Age > 75Non-fatal Same-day lag: 1.032 [1.008,1.056] 1-day lag: 1.009 [0.985,1.032] 2-day lag: 0.989 [0.966,1.013] 3-day lag: 1.009 [0.969,1.051] Fatal Same-day lag: 1.016 [0.978,1.055] 1-day lag: 1.001 [0.966,1.038] 2-day lag: 1.005 [0.969,1.041] 3-day lag: 0.984 [0.948,1.021] Notes: Rate ratios for PNC are given for 0-5 lag days in graph form (Fig 1) for each city. Pooled rate ratios were also provided for groups < 75 yielding similar results to the overall 3-city data.

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Metzger et al. (2004, 044222) **Period of Study:** August 1998–August 2000 **Location:** Atlanta Metropolitan area (Georgia)	**Outcome (ICD-9):** Emergency visits for ischemic heart disease (410–414), cardiac dysrhythmias (427), cardiac arrest (427.5), congestive heart failure (428), peripheral vascular and cerebrovascular disease (433-437, 440, 443-444, 451–453), atherosclerosis (440), and stroke (436). **Age Groups:** All **Study Design:** Time series **N:** 4,407,535 emergency department visits between 1993–2000 (data not reported for 1998-2000) **Statistical Analyses:** Poisson generalized linear modeling **Covariates:** Day of the week, hospital entry and exit indicator variables, federally observed holidays, temporal trends, temperature, dew point temperature **Season:** All **Dose-response Investigated:** No **Statistical Package:** SAS **Lags Considered:** 3-day moving avg, lags 0-7	**Pollutant:** UFP (10–100 nm particle count) (no/cm^3) **Averaging Time:** 24 h Median (10%-90% range): 25,900 (11,500-74,600) **Monitoring Stations:** 1 **Copollutant (correlation):** PM_{10}: r = -0.13 O_3: r = -0.13 NO_2: r = 0.26 CO: r = 0.10 SO_2: r = 0.24 $PM_{2.5}$: r = -0.16 $PM_{2.5}$ water soluble metals: r = -0.27 $PM_{2.5}$ sulfates: r = -0.31; $PM_{2.5}$ acidity: r = -0.39; $PM_{2.5}$ organic carbon: r = 0.08; $PM_{2.5}$ elemental carbon: r = 0.08; $PM_{2.5}$ oxygenated hydrocarbon: r = 0.05 Other variables: Temperature: r = -0.33 Dew point: r = -0.41	**PM Increment:** 30,000 no/cm^3 (approximately 1 SD)3 RR [95% CI]: For 3 day moving avg: All CVD: 0.985 [0.965, 1.005] Dysrhythmia: 0.972 [0.937, 1.008] Congestive heart failure: 0.983 [0.943–1.025] Ischemic heart disease: 0.989 [0.953–1.026] Peripheral vascular and cerebrovascular disease: 0.998 [0.960–1.039] Results for Lags 0–7 expressed in figures (see notes). Notes: Figure 1: RR (95% CI) for single-day lag models for the association of ER visits for CVD with daily ambient UFP. Summary of Figure 1 results: Null or negative associations.

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: von Klot et al. (2005, 088070) **Period of Study:** 1992-2001 **Location:** Augsburg, Germany Barcelona, Spain Helsinki, Finland Rome, Italy Stockholm, Sweden	**Outcome (ICD-9):** Acute myocardial infarction (410 ICD-10: I21-I22), angina pectoris (411, 413 ICD-10: I20, I24), dysrhythmia (427 ICD-10: I46.0, 46.9, I47-I49, R00.1, R00.8), heart failure (428 ICD-10: 150) **Age Groups:** 35+ yrs **Study Design:** Cohort **N:** 22,006 MI survivors **Statistical Analyses:** GAM, Spearman correlation **Covariates:** Temperature, dew point temp, avg barometric pressure, relative humidity **Season:** NR **Dose-response Investigated:** No **Statistical Package:** R-software with "mgcv" package **Lags Considered:** 0-3 days	**Pollutant:** UFP (PNC) **Averaging Time:** 24 h Mean particle/cm^3 (5th–95th percentile): Augsburg: Barcelona: Helsinki: Rome: Stockholm: **Monitoring Stations:** NR **Copollutant (correlation):** Augsburg PM_{10}: r = 0.52 CO: r = 0.63 NO_2: r = 0.64 O_3: r =-0.32Barcelona PM_{10}: r = 0.29 CO: r = 0.71; NO_2: r = 0.44 O_3: r =-0.55 Helsinki PM_{10}: r = 0.46 CO: r = 0.47; NO_2: r = 0.83 O_3: r =-0.16 Rome PM_{10}: r = 0.33 CO: r = 0.80; NO_2: r = 0.71 O_3: r =-0.47 Stockholm PM_{10}: r = 0.06 CO: r = 0.54; NO_2: r = 0.80 O_3: r =-0.17	**PM Increment:** 10,000 particles/cm^3 Pooled RR Estimate [CI]: All cardiac admissions: 1.026 [1.005,1.048] Myocardial infarction: 1.039 [0.998.1.082] Angina pectoris: 1.020 [0.992,1.048]

[1]All units expressed in $\mu g/m^3$ unless otherwise specified.

E.2. Short-Term Exposure and Respiratory Outcomes

E.2.1. Respiratory Morbidity Studies

Table E-9. Short-term exposure–respiratory morbidity outcomes -PM_{10}

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Aekplakorn, et al. (2003, 089908) **Period of Study**: 107 days, from October 1, 1997 to January 15, 1998 **Location**: Mae Mo district, Lampang Province, North Thailand	**Outcome**: Upper respiratory symptoms, lower respiratory symptoms, cough **Age Groups**: 6-14 years old **Study Design**: Logistic regression **N:** 98 asthmatic school children, 98 non-asthmatic school children **Statistical Analyses:** GEE, stratified analysis, PROC GENMOD **Covariates:** Temperature and relative humidity **Season**: winter **Dose-response Investigated?** No **Statistical Package**: SAS v 8.1	**Pollutant**: PM_{10} **Averaging Time:** daily Mean (SD): Sob Pad station: 31.92 Sob Mo station: 33.64 Hua Fai station: 37.45 **Range (Min, Max):** Sob Pad: 6.63, 153.25 Sob Mo: 4.23, 121.80 Hua Fai: 6.98, 113.30 **Monitoring Stations**: 3 **Copollutant:** $PM_{2.5}$, SO_2	**PM Increment:** 10 $\mu g/m^3$ Odds Ratios [Lower CI, Upper CI] lag: Asthmatics: URS: 1.03 (0.99, 1.07) lag 0 LRS: 1.04 (0.99, 1.09) lag 0 Cough: 1.04 (1.00, 1.07) lag 0 Non-Asthmatics: URS: 1.04 (0.99, 1.08) lag 0 LRS: 1.0 (0.93, 1.07) lag 0 Cough: 0.99 (0.94, 1.05) lag 0 PM_{10} + SO_2 Asthmatics: URS: 1.03 (0.99, 1.07) lag 0 LRS: 1.03 (0.98, 1.09) lag 0 Cough: 1.04 (1.00, 1.08) lag 0 Non-Asthmatics: URS: 1.04 (0.99, 1.08) lag 0 LRS: 1.0 (0.93, 1.07) lag 0 Cough: 0.99 (0.95, 1.05) lag 0

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Andersen et al. (2008, 189651) **Period of Study:** Dec 12, 1998–Dec 19, 2004 **Location:** Copenhagen	**Outcome:** Daily symptoms (prospective daily recording of symptoms via diary) **Age Groups:** 0-3 yrs **Study Design:** Panel study of children with genetic susceptibility to asthma (mothers had asthma) **N:** 205 children (living within a 15km radius of the central monitor during the first 3 yrs of life) born between Aug 2, 1998 and Dec 12, 2001 **Statistical Analyses:** logistic regression model (GEE) **Covariates:** temperature, season, gender, age, exposure to smoking, and paternal history of asthma Effect modification: gender, medication use, and paternal history of asthma **Statistical Package:** SAS v9.1 **Lag:** 0,1,2,3,4,2-4	**Pollutant:** PM_{10} Mean: 25.1 SD: 16.7 Percentiles: 25th: 15.7 75th: 30.2 IQR: 14.5 **Copollutant (correlation):** $PM_{2.5}$ (r = 0.79) Number concentration of ultrafine particles, UFP (r = 0.37) NO_2 (r = 0.43) NO_X (r = 0.40) CO (r = 0.45) O_3 (r = -0.32) Temp (r = 0.25)	**PM Increment:** IQR (14.5 $\mu g/m^3$) increase Odds Ratios (95%CI) for incident wheezing symptoms Age 0-1 L0: 1.05 (0.88, 1.25) L1: 1.00 (0.82, 1.22) L2: 1.01 (0.83, 1.23) L3: 1.20 (0.98, 1.46) L4: 1.23 (1.02, 1.48) L2-4: 1.21 (0.99, 1.48) Age 1-2 L0: 1.00 (0.86, 1.15) L1: 1.02 (0.87, 1.19) L2: 1.05 (0.93, 1.19) L3: 0.96 (0.84, 1.09) L4: 1.04 (0.90, 1.21) L2-4: 1.03 (0.88, 1.22) Age 2-3 L0: 0.87 (0.72, 1.06) L1: 0.95 (0.78, 1.15) L2: 0.99 (0.82, 1.17) L3: 1.03 (0.84, 1.25) L4: 0.89 (0.74, 1.09) L2-4: 0.94 (0.74, 1.19) Age 0-3 L0: 0.97 (0.87, 1.08) L1: 0.99 (0.89, 1.10) L2: 1.01 (0.92, 1.12) L3: 1.03 (0.93, 1.14) L4: 1.04 (0.94, 1.15) L2-4: 1.04 (0.92, 1.17) Two pollutant models (lag 2-4) 1-pollutant model: 1.21 (0.99, 1.48) 2-pollutant (adj for NO_2): 1.13 (0.88, 1.45) 2-pollutant (adj for NO_X): 1.16 (0.90, 1.48) 2-pollutant (adj for CO): 1.23 (0.96, 1.57) 110 children living within 5km radius from monitor (sensitivity analysis): Age 0-1: 1.32 (0.95, 1.82) Age 1-2: 1.20 (0.87, 1.67) Age 2-3: 0.78 (0.52, 1.16) Age 0-3: 1.11 (0.88, 1.39)
Reference: Boezen et al. (2005, 087396) **Period of Study:** Two consecutive	**Outcome:** FEV_1, airway hyperresponsiveness (AHR), serum total IgE and daily data on lower respiratory symptoms (LRS), upper respiratory	**Pollutant:** PM_{10} **Averaging Time:** 24 h	**PM Increment:** 10 $\mu g/m^3$ Effect Estimate [Lower CI, Upper CI]: AHR-/IgE-

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
winters (winter 1993-winter 1995) **Location:** rural (Meppel, Nunspeet) and urban (Amsterdam) areas in the Netherlands	symptoms (URS), cough and morning and evening peak expiratory flow **Age Groups:** 50-70 years **Study Design:** Case-control study **N:** 327 patients **Statistical Analyses:** Logistic regression **Covariates:** daily minimum temperature, linear, quadratic and cubic time trend, weekend/holidays, and influenza incidence for the rural and urban areas and two winters separately **Season**: winter **Dose-response Investigated?** No **Lags Considered:** 0, 1, 2, and 5-day mean	Mean (SD): Winter 93/94 Urban: 41.5 Winter 93/94 Rural: 44.1 Winter 94/95 Urban: 31.1 Winter 94/95 Rural: 26.6 **Percentiles:** 50th(Median): Winter 93/94 Urban: 34.6 Winter 93/94 Rural: 30.4 Winter 94/95 Urban: 28.9 Winter 94/95 Rural: 23.7 **Range (Min, Max):** 93/94 Urban: (12.1-112.7) 93/94 Rural: (7.9-242.2) 94/95 Urban: (8.8-89.9) 94/95 Rural: (7.1-96.9) Copollutant: SO_2 NO_2 Black Smoke	Upper Respiratory Symptoms Lag 0: OR = 0.99 (0.97-1.01) Lag 1: OR = 1.01 (0.99-1.03) Lag 2: OR = 1.00 (0.96-1.02) 5-day mean: OR = 1.00 (0.96-1.04) Cough Lag 0: OR = 1.00 (0.99-1.02) Lag 1: OR = 0.99 (0.98-1.01) Lag 2: OR = 1.00 (0.98-1.01) 5-day mean: OR = 0.98 (0.95-1.01) $>$10% fall in morning peak expiratory flow Lag 1: OR = 1.01 (0.98-1.04) Lag 2: OR = 0.97 (0.94-1.00) 5-day mean: OR = 0.97 (0.92-1.02) AHR-/IgE+ Upper Respiratory Symptoms Lag 0: OR = 1.01 (0.99-1.03) Lag 1: OR = 1.02 (1.00-1.04) Lag 2: OR = 1.01 (0.99-1.03) 5-day mean: OR = 1.08 (1.04-1.11) Cough Lag 0: OR = 1.01 (0.99-1.03) Lag 1: OR = 0.99 (0.98-1.01) Lag 2: OR = 1.00 (0.98-1.02) 5-day mean: OR = 1.01 (0.97-1.05) $>$10% fall in morning peak expiratory flow Lag 1: OR = 0.99 (0.97-1.02) Lag 2: OR = 0.99 (0.97-1.02) 5-day mean: OR = 0.97 (0.93-1.01) AHR+/IgE- Upper Respiratory Symptoms Lag 0: OR = 0.99 (0.95-1.03) Lag 1: OR = 1.01 (0.97-1.05) Lag 2: OR = 0.99 (0.96-1.03) 5-day mean: OR = 0.98 (0.91-1.06) Cough Lag 0: OR = 1.00 (0.97-1.02) Lag 1: OR = 1.01 (0.98-1.03) Lag 2: OR = 0.99 (0.96-1.02) 5-day mean: OR = 1.02 (0.96-1.08) $>$10% fall in morning peak expiratory flow Lag 1: OR = 0.99 (0.95-1.03) Lag 2: OR = 0.99 (0.95-1.03)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			5-day mean: OR = 0.99 (0.93-1.06)
			AHR+/IgE+
			Upper Respiratory Symptoms
			Lag 0: OR = 1.01 (0.98-1.04)
			Lag 1: OR = 1.03 (1.00-1.05)
			Lag 2: OR = 1.02 (0.99-1.05)
			5-day mean: OR = 1.06 (1.00-1.11)
			Cough
			Lag 0: OR = 1.03 (1.01-1.06)
			Lag 1: OR = 1.00 (0.98-1.02)
			Lag 2: OR = 0.99 (0.97-1.01)
			5-day mean: OR = 0.99 (0.95-1.04)
			Lag 2: OR = 0.99 (0.96-1.03)
			5-day mean: OR = 0.99 (0.92-1.05)
			> 10% fall in morning peak expiratory flow
			Lag 1: OR = 1.04 (1.00-1.07)
			Lag 2: OR = 1.03 (0.99-1.06)
			5-day mean: OR = 1.05 (0.99-1.11)
Reference: Boezen et al. (1999, 040410)	**Outcome:** Respiratory symptoms	**Pollutant:** PM_{10}	Increment: 100 $\mu g/m^3$
Periods of Study: 3 Winters (1992-1995)	Lower respiratory symptoms (wheeze, attacks of wheezing, shortness of breath)	**Averaging Time:** 24-h avg	Odds Ratio (Lower CI, Upper CI)
Location: Urban and rural areas of the Netherlands	Upper respiratory symptoms (sore throat, runny or blocked nose)	**Mean (SD):**	lag: OR for respiratory symptoms and exposure to PM_{10} in children with BHR and high serum total IgE
	Bronchial hyperresponsiveness (BHR)	Winter 1992-93	Lower Respiratory Symptoms
	Study Design: Time-series	Urban: 54.8	1.32 (1.07, 1.63)
	Statistical Analyses: Logistic regression (PROC model)	Rural: 44.7	0
	Age Groups: 7-11	Winter 1993-94	1.36 (1.13, 1.64)
		Urban: 41.5 3	1
		Rural: 44.1	1.36 (1.13, 1.65)
		Winter 1994-95	2
		Urban: 31.1	2.39 (1.71, 3.35)
		Rural: 26.6	0-5 avg.
		Range (Min, Max):	Upper Respiratory Symptoms
		Winter 1992-93	1.13 (0.97, 1.32)
		Urban: (4.7, 145.6)	0
		Rural: (4.8, 103.8)	1.00 (0.87, 1.16)
		Winter 1993-94	1
		Urban: (12.1, 112.7)	0.96 (0.84, 1.11)
		Rural: (7.9, 242.2)	2
		Winter 1994-95	0.91 (0.70, 1.18)
		Urban: (8.8, 89.9)	0-5 avg
		Rural: (7.1, 96.9)	> 10% morning peak expiratory flow (PEF) decrease
		Copollutants:	1.10 (0.92, 1.33)
		BS	0
		SO_2	1.08 (0.90, 1.28)
		NO_2	

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			1
			1.03 (0.87, 1.23)
			2
			1.10 (0.83, 1.46)
			0-5 avg
			> 10% evening peak expiratory flow (PEF) increase
			1.37 (1.16, 1.63)
			0
			1.09 (0.92, 1.29)
			1
			1.16 (0.98. 1.36)
			2
			1.35 (1.04, 1.77)
			0-5 avg.
			OR for respiratory symptoms and exposure to PM_{10} in children without BHR and low serum total IgE
			Lower Respiratory Symptoms
			1.08 (0.75, 1.57)
			0
			1.04 (0.70, 1.53)
			1
			0.98 (0.69, 1.39)
			2
			1.15 (0.61, 2.15)
			0-5 avg.
			Upper Respiratory Symptoms
			1.12 (0.99, 1.28)
			0
			1.01 (0.89, 1.15)
			1
			1.01 (0.89, 1.15)
			2
			0.93 (0.67, 1.28)
			0-5 avg
			> 10% morning PEF decrease
			1.07 (0.93, 1.23)
			0
			0.86 (0.75, 0.99)
			1
			0.97 (0.85, 1.11)
			2
			0.99 (0.79, 1.23)
			0-5 avg
			> 10% evening PEF decrease

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			1.13 (0.98, 1.30)
			0
			1.05 (0.91, 1.21)
			1
			0.99 (0.87, 1.14)
			2
			0.94 (0.75, 1.17)
			0-5 avg
			OR for respiratory symptoms and exposure to PM_{10} in children with BHR and low serum total IgE
			Lower Respiratory Symptoms
			0.77 (0.48, 1.24)
			0
			1.34 (0.94, 1.93)
			1
			1.24 (0.86, 1.81)
			2
			1.92 (0.84, 4.41)
			0-5 avg
			Upper Respiratory Symptoms
			1.13 (0.92, 1.40)
			0
			0.98 (0.79, 1.22)
			1
			0.97 (0.79, 1.20)
			2
			0.83 (0.54, 1.25)
			0-5 avg
			> 10% morning PEF decrease
			1.04 (0.78, 1.38)
			0
			0.86 (0.66, 1.12)
			1
			0.91 (0.71, 1.17)
			2
			0.78 (0.51, 1.20)
			0-5 avg
			> 10% evening PEF decrease
			1.07 (0.82, 1.41)
			0
			0.98 (0.76, 1.26)
			1
			0.93 (0.73, 1.19)
			2
			0.83 (0.55, 1.26)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			0-5 avg
			OR for respiratory symptoms and exposure to PM_{10} in children without BHR and high serum total IgE
			Lower Respiratory Symptoms
			1.04 (0.80, 1.35)
			0
			1.21 (0.98, 1.51)
			1
			1.18 (0.96, 1.45)
			2
			1.35 (0.89, 2.04)
			0-5 avg
			Upper Respiratory Symptoms
			1.01 (0.85, 1.20)
			0
			0.95 (0.81, 1.12)
			1
			0.93 (0.80, 1.09)
			2
			0.93 (0.69, 1.25)
			0-5 avg
			> 10% morning PEF decrease
			0.97 (0.80, 1.17)
			0
			1.09 (0.91, 1.30)
			1
			1.02 (0.85, 1.21)
			2
			0.95 (0.71, 1.28)
			0-5 avg
			> 10% evening PEF decrease
			1.02 (0.85, 1.22)
			0
			1.06 (0.90, 1.25)
			1
			1.08 (0.93, 1.27)
			2
			1.04 (0.80, 1.34)
			0-5 avg.
Reference: Chattopadhyay et al. (2007, 147471) **Period of Study:** NR **Location:** Three different points in Kolkata, India: North, South, and Central	**Outcome:** pulmonary function tests (respiratory impairments) **Age Groups:** All ages **Study Design:** Cross-sectional **N:** 505 people studied for PFT total population of Kolkata not given	**Pollutant:** PM_{10} **Averaging Time:** 8 h Mean (SD): North Kolkata: 535.9 Central Kolkata: 1114.5 South Kolkata: 909.2	**PM Increment:** NR Respiratory impairments (SD): North Kolkata Male (n = 137) Restrictive: 4 (2.92) Obstructive: 5 (3.64)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
	Statistical Analyses: Frequencies	**Monitoring Stations:** 1	Combined Res. And Obs.: 6 (4.37)
	Covariates: Meteorologic data (i.e. temperature, wind direction, wind speed, and humidity)	Copollutant:	Total: 15 (10.95)
		PM < 10-3.3	Female (n = 152)
	Dose-response Investigated? No	PM < 3.3-0.4	Restrictive: 3 (1.97)
			Obstructive: 5 (3.28)
			Combined Res. And Obs.: n/a
			Total: 8 (5.26)
			Total (n = 289)
			Restrictive: 7 (2.42)
			Obstructive: 10 (3.46)
			Combined Res. And Obs.: 6 (2.07)
			Total: 23 (7.96)
			Central Kolkata
			Male (n = 44)
			Restrictive: 6 (13.63)
			Obstructive: 1 (2.27)
			Combined Res. And Obs.: 1 (2.27)
			Total: 8 (18.18)
			Female (n = 50)
			Restrictive: 3 (6.00)
			Obstructive: 2 (4.00)
			Combined Res. And Obs.: n/a
			Total: 5 (10.00)
			Total (n = 94)
			Restrictive: 9 (9.57)
			Obstructive: 3 (3.19)
			Combined Res. And Obs.: 1 (1.06)
			Total: 13 (13.82)
			South Kolkata
			Male (n = 52)
			Restrictive: 1 (1.92)
			Obstructive: 2 (3.84)
			Combined Res. And Obs.: 3 (5.76)
			Total: 6 (11.53)
			Female (n = 70)
			Restrictive: 2 (2.85)
			Obstructive: 1 (1.42)
			Combined Res. And Obs.: n/a
			Total: 3 (4.28)
			Total (n = 122)
			Restrictive: 3 (2.45)
			Obstructive: 3 (2.45)
			Combined Res. And Obs.: 3 (2.45)
			Total: 9 (7.37)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Dales et al. (2006, 090744) **Period of Study:** 1/1/1986-12/31/2000 **Location:** 11 Canadian Cities: Calgary, Edmonton, Halifax, London, Hamilton, Ottawa, St. John, Toronto, Vancouver, Windsor, Winnipeg	**Health Outcome:** Respiratory Illness: Asphyxia (799) Respiratory failure (799.1) Dyspnea and respiratory abnormalities (786) Respiratory distress syndrome (769) Unspecified birth asphyxia in live-born infant (768.9) Other respiratory problems after birth (770.8) Pneumonia (486) **Study Design:** Time-series **Statistical Analyses:** Poisson **Age Groups:** 0-27 days	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Copollutants (correlation):** O_3: r = -0.29 to 0.41 NO_2: r = -0.26 to 0.69 SO_2: r = -0.09 to 0.61 CO: r = -0.13 to 0.71	Increment: 10 $\mu g/m^3$ % Increase (Lower CI, Upper CI) Lag In respiratory illness and exposure to PM_{10} in neonates PM_{10} alone: 2.13 (-0.50, 4.76) Multipollutant model PM_{10}: 1.45 (-1.90, 4.80) PM_{10}, O_3: 2.67 (0.98, 4.39) PM_{10}, NO_2: 2.48 (1.18, 3.80) PM_{10}, SO_2: 1.41 (0.35, 2.47) PM_{10}, CO: 1.30 (0.13, 2.49)
Reference: de Hartog et al. (2003, 001061) **Period of Study:** winter of 1998-1999 (in Amsterdam, from November 2, 1998 to June 18, 1999 in Erfurt, from October 12, 1998 to April 4, 1999 and in Helsinki, from November 2, 1998 to April 30, 1999.) **Location:** Amsterdam, the Netherlands Erfurt, Germany and Helsinki, Finland	**Outcome:** Respiratory symptoms **Age Groups:** ≥ 50 yrs **Study Design:** panel **N:** 131 subjects with history of coronary heart disease **Statistical Analyses:** Logistic regression **Covariates:** ambient temperature, relative humidity, atmospheric pressure, incidence of influenza-like illness **Season:** Winter **Dose-response Investigated?** No **Statistical Package:** S-PLUS 2000 **Lags Considered:** 0, 1, 2, 3, and 5-day avg	**Pollutant:** PM_{10} **Averaging Time:** 24 h Mean (SD): Amsterdam, the Netherlands: 36.5 Erfurt, Germany: 27.1 Helsinki, Finland: 19.6 **Range (Min, Max):** Amsterdam, the Netherlands: (13.6-112.0) Erfurt, Germany: (5.2-104.2) Helsinki, Finland: (6.4-67.4) **Monitoring Stations:** 1 Copollutant: $PM_{2.5}$ $NC_{0.01-0.1}$ CO NO_2 SO_2	'There was a tendency toward positive associations between avoidance of activities and both particulate air pollution (PM_{10}) and gases, but none of the associations were statistically significant....In both incidence analyses and prevalence analyses, odds ratios for PM_{10} were generally similar to the corresponding odds ratios for $PM_{2.5}$, but were somewhat less significant.'

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Delfino et al. (1998, 051406) **Period of Study:** August 1–October 30, 1995 **Location:** Alpine, CA	**Outcome:** asthma symptom severity **Age Groups:** 9-17 **Study Design:** Panel Study **N:** 24 non-smoking pediatric asthmatics **Statistical Analyses:** GEE **Covariates:** day of week, temperature, humidity, wind speed **Statistical Package:** SAS **Lags Considered:** 0-5, 0, 0-4	**Pollutant:** PM_{10} **Averaging Time:** 24 h Mean (SD): 31 (8) 90th: 42 **Range (Min, Max):** 16, 54 **Copollutant (correlation):** O_3 (r = 0.32)	**PM Increment:** 42 $\mu g/m^3$ (90th percentile increase) Asthma symptoms: Everyone: 1.47 (0.90, 2.39) lag 0 Everyone: 1.73 (1.03, 2.89) lag 0-4 Less symptomatic: 2.47 (1.23-4.95) lag 0 Less symptomatic: 4.03 (1.22, 13.33) lag 0-4 More symptomatic: 1.50 (0.80, 2.80) lag 0 More symptomatic: 1.95 (1.12, 3.43) lag 0-4 PM_{10} + O_3 Asthma symptoms: 1.31 (0.84, 2.06) lag 0 1.65 (1.03, 2.66) lag 0-4 Less symptomatic: 2.08 (1.12-3.83) lag 0 Less symptomatic: 3.35 (1.06, 10.51) lag 0-4 More symptomatic: 1.40 (0.77, 2.53) lag 0 More symptomatic: 1.87 (1.11, 3.13) lag 0-4

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Delfino et al. (2002, 093740) **Period of Study:** March 1 through April 30, 1996 **Location:** Alpine, California (a semi-rural area)	**Outcome:** Asthma symptoms that interfere with daily activities **Age Groups:** 9-19 yrs **Study Design:** Daily panel study **N:** 22 asthmatic children **Statistical Analyses:** GEE **Covariates:** temperature, relative humidity, day-of-week trends, linear time trend across the 61 days, and upper or lower respiratory infection **Season:** "early spring season" of March through April **Dose-response Investigated?** Yes **Statistical Package:** SAS, version 8 **Lags Considered:** 0, 1, 2, 3, 4, 5, 3-day mov avg	**Pollutant:** PM_{10} **Averaging Time:** 1 h max Mean (SD): 38(15) Percentiles: 90th: 63 **Range (Min, Max):** (12-69) **Averaging Time:** 8 h max Mean (SD): 28(12) Percentiles: 90th: 46 **Range (Min, Max):** (8-57) **Averaging Time:** 24 h Mean (SD): 20(9) Percentiles: 90th: 32 **Range (Min, Max):** (7-42) **Copollutant (correlation):** 1 h max PM_{10}: 8 h max PM_{10}: r = 0.93 24 h PM_{10}: r = 0.84 1 h max O_3: r = 0.68 8 h max O_3: r = 0.95 1 h max NO_2: r = 0.49 8 h max NO_2: r = 0.55 8 h max PM_{10}: 1 h max PM_{10}: r = 0.93 24 h PM_{10}: r = 0.95 1 h max O_3: r = 0.72 8 h max O_3: r = 0.65 1 h max NO_2: r = 0.48 8 h max NO_2: r = 0.55 24 h PM_{10}: 1 h max PM_{10}: r = 0.84 8 h max PM_{10}: r = 0.95 1 h max O_3: r = 0.74 8 h max O_3: r = 0.71 1 h max NO_2: r = 0.37 8 h max NO_2: r = 0.44	**PM Increment:** 90th percentile increase Effect Estimate [Lower CI, Upper CI]: ORs for risk of asthma symptoms in those who report a respiratory infection compared to those who do not have a respiratory infection 1 h max PM_{10} lag 0: 4.88 (1.31-18.2) 8 h max PM_{10} lag 0: 6.78 (1.38-33.3) 24 h mean PM_{10} lag 0: 4.68 (0.71-30.7) 3-day mov avg 1 h max PM_{10}: 11.1 (1.10-112) 3-day mov avg 8 h max PM_{10}: 10.1 (1.42-72.0) 3-day mov avg 24 h PM_{10}: 2.67 (0.60-11.8) Effect modification by anti-inflammatory medication use on the relationship of asthma symptoms in children 1 h max PM_{10} lag 0: 1.41 (0.87-2.30) On medication: 0.96 (0.25-3.69) Not on medication: 1.92 (1.22-3.02) 8 h max PM_{10} lag 0: 1.19 (0.74-1.94) On medication: 0.75 (0.18-3.04) Not on medication: 1.68 (0.91-3.09) 24 h mean PM_{10} lag 0: 1.08 (0.73-1.61) On medication: 0.80 (0.24-2.69) Not on medication: 1.35 (0.82-2.22) 3-day mov avg 1 h max PM_{10}: 1.45 (0.76-2.76) On medication: 1.01 (0.14-7.02) Not on medication: 1.92 (0.99-3.71) 3-day mov avg 8 h max PM_{10}: 1.32 (0.76-2.29) On medication: 0.82 (0.17-3.94) Not on medication: 1.89 (1.10-3.24) 3-day mov avg 24 h PM_{10}: 1.22 (0.84-1.77) On medication: 0.75 (0.26-2.14) Not on medication: 1.75 (1.15-2.68) Dose-response results are found in Figure 2 and not quantitatively reported elsewhere.

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Delfino et al. (2003, 090941) **Period of Study:** November 1999 to January 2000 **Location:** Huntington Park, Los Angeles	**Outcome:** Asthma severity scale Peak Expiratory Flow Rate (PEF) **Age Groups:** Ages 10 to 16 **Study Design:** Longitudinal study panel **N:** 22 children **Statistical Analyses:** Regression analysis (GEE, GLM) multivariate regression models **Covariates:** Day of the week, Maximum Temperature, Respiratory Infections **Season:** Winter **Dose-response Investigated?** No **Statistical Package:** SAS **Lags Considered:** 0, 1	**Pollutant:** PM_{10} Mean (SD): 59.9 (24.7) **Range (Min, Max):** 20-126 IQR: 37 90th: 86.0 **Monitoring Stations:** 1 **Copollutant (correlation):** 8-h max NO_2 = 0.38 8-h max O_3 = -0.16 8-h max CO = 0.50 8-h max SO_2 = 0.73	**PM Increment:** IQR 37.0 $\mu g/m^3$ OR Estimate [Lower CI, Upper CI] lag: Lag 0 Symptom Scores > 1: 1.45 (1.11, 1.90) Symptom Scores > 2: NR Lag 1 Symptom Scores > 1: 1.07 (0.64, 1.77) Symptom Scores > 2: NR
Reference: Delfino et al. (2004, 056897) **Period of Study:** September–October 1999 April–June 2000 **Location:** Alpine, California	**Outcome:** FEV_1 **Age Groups:** 9-19 years old **Study Design:** Panel study **N:** 24 children **Statistical Analyses:** GLM Akaike's information criterion and Bayesian information criterion **Covariates:** Day of week, Personal temperature and relative humidity, time of FEV_1 maneuver (morning, afternoon, or evening), Season (fall 1999 or spring 2000) As-needed medication use Presence or absence of upper or lower respiratory infections **Season: S**pring, Fall **Dose-response Investigated?** No **Statistical Package:** SAS **Lags Considered:** Lag 0-4	**Pollutant:** PM_{10} **Averaging Time:** 4 h, 8 h, 12 h, 24-h Personal Monitor 1-h max personal PM last 24-h Mean (SD): 151.0 (12.03) 90th: 292.4 **Range (Min, Max):** (9.1, 996.8) Mean personal PM last 24-h Mean (SD): 37.9 (19.9) 90th: 65.1 **Range (Min, Max):** (3.9, 113.8) Central outdoor stationary-site PM 1-h Maximum TEOM PM_{10} last 24-h Mean (SD): 54.4 (13.8) 90th: 71.0 **Range (Min, Max):** (24.4, 95.4) Mean TEOM PM_{10} last 24-h Mean (SD): 29.7 (8.6) 90th: 40.9 **Range (Min, Max):** (12.9, 50.7) 24-h mean PM_{10} Mean (SD): 23.6 (9.1) 90th: 34.6 **Range (Min, Max):** (3.2, 48.0) **Copollutant (correlation):** 8-h max personal PM 8-h max O_3 = 0.03 8-h Max NO_2 = 0.26 24-h Mean Personal	Results presented graphically: Percent predicted FEV_1 was inversely associated with personal exposure to fine particles. - Inverse associations of FEV_1 with stationary-site indoor, outdoor and central-site gravimetric $PM_{2.5}$ and PM_{10}, and with hourly TEOM PM_{10}

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
		PM = 0.94	
		8-h Max TEOM PM_{10} = 0.38	
		24-h Mean TEOM PM_{10} = 0.40	
		24-h Central HI PM_{10} = 0.37	
		24-h Central HI $PM_{2.5}$ = 0.38	
		24-h Outdoor HI PM_{10} = 0.32	
		24-h Outdoor HI $PM_{2.5}$ = 0.39	
		24-h Indoor HI PM_{10} = 0.23	
		24-h Indoor HI $PM_{2.5}$ = 0.37	
		24-h mean personal PM	
		8-h max O_3 = 0.01	
		8-h Max NO_2 = 0.27	
		8-h Max Personal PM = 0.94	
		8-h Max TEOM PM_{10} = 0.36	
		24-h Mean TEOM PM_{10} = 0.39	
		24-h Central HI PM_{10} = 0.36	
		24-h Central HI $PM_{2.5}$ = 0.43	
		24-h Outdoor HI PM_{10} = 0.34	
		24-h Outdoor HI $PM_{2.5}$ = 0.44	
		24-h Indoor HI PM_{10} = 0.29	
		24-h Indoor HI $PM_{2.5}$ = 0.46	
		24-h Mean TEOM PM_{10}	
		8-h max O_3 = 0.41	
		8-h Max NO_2 = 0.58	
		8-h Max Personal PM = 0.40	
		24-h Mean Personal PM = 0.39	
		8-h Max TEOM PM_{10} = 0.92	
		24-h Central HI PM_{10} = 0.86	
		24-h Central HI $PM_{2.5}$ = 0.78	
		24-h Outdoor HI PM_{10} = 0.79	
		24-h Outdoor HI $PM_{2.5}$ = 0.78	
		24-h Indoor HI PM_{10} = 0.36	
		24-h Indoor HI $PM_{2.5}$ = 0.59	

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Delfino et al. (2006, 090745) **Period of Study:** Region 1: August to Mid December 2003. Region 2: July through November 2004 **Location:** Region 1: Riverside, CA. Region 2: Whittier, CA	**Outcome:** Fractional Concentration of Nitric Oxide in exhaled air (FENO) **Age Groups:** 9 through 18 **Study Design:** Longitudinal Panel Study **N:** 45 children **Statistical Analyses:** Linear mixed-effects models Two-stage hierarchical model Empirical Variograms Fourth-order polynomial distributed lag mixed-effects model **Covariates:** Personal temperature, Personal Rel. Humid., 10-day exposure run, Respiratory infections, Region of study, Sex, Cumulative daily use of as-needed B-agonist inhalers **Dose-response Investigated?** No **Lags Considered:** Lag 0, Lag 1, 2-day moving avg	**Pollutant:** PM_{10} Central Site **Averaging Time:** 24- h Riverside **Mean (SD):** 70.82 (29.36) 50th(Median): 65.96 **Range (Min, Max):** (30.75, 154.05) $\mu g/m^3$ Whittier **Mean (SD):** 35.73 (16.6) 50th(Median): 34.65 **Range (Min, Max):** (5.86, 105.46) $\mu g/m^3$ **Monitoring Stations:** 48 personal nephelometers, 2 central sites	**PM Increment:** IQR increase (Riverside: 28.41 $\mu g/m^3$, Whittier 21.87 $\mu g/m^3$) Coefficient [Lower CI, Upper CI] lag: Lag = 2-day moving avg Stratified by Medication Use Not Taking Anti-Inflamm. Medication Central 0.76 (-1.54, 3.07) Taking Anti-Inflamm. Medication Central 0.53 (-0.83, 1.90) Inhaled Corticosteroids Central 1.28 (-0.01, 2.58) Antileukotrienes +- inhaled corticosteroids Central -2.10 (-5.33, 1.12) Notes: Figure of Estimated lag effect of hourly personal $PM_{2.5}$ on FENO. Figure of the Estimated lag effect of hourly personal $PM_{2.5}$ on FENO by use of medications. Figure of One- and two-pollutant models for change in FENO using 2-day Moving Averages personal and central-site pollutant measurements.

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Desqueyroux et al. (2002, 026052) **Period of Study:** Nov 1995-Nov 1996 **Location:** Paris, France	**Outcome:** Asthma attacks **Age Groups:** Adults. **Study Design:** Panel study **N:** 60 moderate to severe adult asthmatics **Statistical Analyses:** Marginal logistic regression **Covariates:** FEV_1, smoking, allergy, oral steroid treatment, mean daily temperature, relative humidity, pollen counts, season, holiday period **Season:** winter, summer **Dose-response Investigated?** No **Statistical Package:** SAS **Lags Considered:** 1, 2, 3, 4, 5, 3-5	**Pollutant:** PM_{10} **Averaging Time:** 24 h Mean (SD): Summer: 23 (9) Winter: 28 (14) **Range (Min, Max):** Summer: 6, 63 Winter: 9, 84 **Monitoring Stations:** 7 **Copollutant:** SO_2 , NO_2, O_3	**PM Increment:** 10 $\mu g/m^3$ OR Estimate [Lower CI, Upper CI] lag: 0.87 [0.71, 1.06] lag 1 0.93 [0.80, 1.08] lag 2 1.11 [0.98, 1.26] lag 3 1.17 [1.03, 1.33] lag 4 1.16 [1.01, 1.34] lag 5 1.21 [1.01, 1.34] lag 3-5 vs seasons alone: Winter: 1.41 [1.16, 1.71] lag 3-5 summer: 1.03 [0.72, 1.47] lag 3-5 vs link to explanatory factors: No link: [1.71 [1.20, 2.43] lag 3-5 Link: 1.27 [1.06, 1.52] lag 3-5 vs occurrence of infection: Without infection: 1.52 [1.16, 2.00] lag 3-5 With infection: 1.30 [1.03, 1.65] lag 3-5 vs baseline pulmonary function: FEV_1 >/ = 68% predicted: 1.38 [1.06, 1.79] lag 3-5 FEV < 68% predicted: 1.45 [1.11, 1.90] lag 3-5 vs smoking habits: Nonsmokers: 1.53 [1.18, 1.98] lag 3-5 Current & ex-smokers: 1.18 [0.90, 1.54] lag 3-5 vs allergy: Non-allergic: 1.29 [0.94, 1.77] lag 3-5 Allergic: 1.49 [1.17, 1.90] lag 3-5 vs regular oral steroid treatment: No: 1.41 [1.15, 1.73] lag 3-5 Yes: 1.41 [0.88, 2.25] lag 3-5 Multipollutant model: PM_{10} + NO_2: 1.43 [1.16, 1.76] Lag 3-5 PM_{10} + SO_2: 1.51 [1.20, 1.90] Lag 3-5 PM_{10} + O_3: 1.09 [0.71, 1.67] Lag 3-5
Reference: Diette et al. (2007, 156399) **Period of Study:** 9/2001-12/2003 **Location:** East Baltimore, MD	**Outcome:** Asthma in the last 12 months (493.x) **Age Groups:** 2 to 6 years old **Study Design:** Prospective cohort **N:** 150 with asthma 150 without asthma **Statistical Analyses:** Student's two-tailed t-test Kruskal-Wallis test Pearson's chi square Fisher's exact test **Covariates:** Season of collection **Dose-response Investigated?** No **Statistical Package:** STATASE 8.0	**Pollutant:** PM_{10} **Averaging Time:** 72 50th(Median): 43.7 IQR: (29-70)	Notes: "Pollutant concentrations in the homes of asthmatic and control children who lived in the same home for their whole life were not different compared with those who had moved at least once."

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Ebelt et al. (2005, 056907) **Period of Study:** summer of 1998 **Location:** Vancouver, Canada	**Outcome:** spirometry **Age Groups:** range from 54-86 yrs mean age = 74 years **Study Design:** extended analysis of a repeated-measures panel study **N:** 16 persons with COPD **Statistical Analyses:** Earlier analysis expanded by developing mixed-effect regression models and by evaluating additional exposure indicators **Dose-response Investigated?** No **Statistical Package:** SAS V8	**Pollutant:** PM_{10} **Averaging Time:** 24 h Mean (SD): Ambient PM_{10}: 17 (6) Exposure to ambient PM_{10}: 10.3 (4.6) **Range (Min, Max):** Ambient PM_{10}: (7-36) Exposure to ambient PM_{10}: (1.5-23.8) **Monitoring Stations:** 5 **Copollutant (correlation):** Ambient $PM_{10-2.5}$: r = 0.69 Ambient $PM_{2.5}$: r = 0.78 Exposure to Ambient PM_{10}: r = 0.71	**PM Increment:** Ambient PM_{10}: 7 (IQR) Exposure to ambient PM_{10}: 6.5 (IQR) Notes: Effect estimates are presented in Figure 2 and Electronic Appendix Table 1 (only available with electronic version of article) and not provided quantitatively elsewhere.
Reference: Fischer et al. (2007, 156435) **Period of Study:** 7 weeks (dates not specified) **Location:** Netherlands	**Outcome:** Respiratory Symptoms, Sore throat, Runny nose, Cold, Sick at home **Study Design:** Prospective cohort **N:** 68 **Statistical Analyses:** Linear regression model (PROC mixed) **Age Groups:** 10-11 **Lag:** 1-2 **Statistical Package:** SAS v 6.11	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg Mean (SD): 56 $\mu g/m^3$ IQ (25th, 75th): (21, 187) $\mu g/m^3$ Copollutants: BS NO_2 CO NO	Increment: 10 $\mu g/m^3$ % Increase in eNO and PM_{10} and change in spirometric lung function lag eNO and PM_{10} only 6.5 (0.9, 12.4) 1 7.8 (-11.3, 31.0) 2 FVC mean (SEM) 0.4 (0.5) 1 0.6 (1.6) 2 FEV_1 mean (SEM) -0.3 (0.5) 1 -2.1 (1.9) 2 PEF mean (SEM) -2.8 (3.3) 1 7.1 (12.0) 2 MMEF mean (SEM)-0.5 (1.7) 1 -2.5 (5.9) 2

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Forsberg et al. (1998, 051714) **Period of Study:** 1/3/1994–3/27/1994 **Location:** Urban and rural areas of Umea, Sweden	**Outcome:** Respiratory Symptoms, Shortness of breath Wheeze, Asthma attacks, Recent asthma, Dry cough, Doctor-diagnosed asthma, Recently treated for asthma, Early chest illness **Study Design:** Cohort panel **Statistical Analyses:** Logistic linear regression **Age Groups:** 6-12	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg Mean (SD): Urban: 13.4 $\mu g/m^3$ Rural: 11.5 $\mu g/m^3$ **Range (Min, Max):** Urban: (0, 40.5) $\mu g/m^3$ Rural: (1.6, 29.0) $\mu g/m^3$ Copollutants (correlation): BS: r = 0.73	Increment: 10 $\mu g/m^3$ OR between prevalence of acute respiratory symptoms and PM_{10} exposure for urban and rural children lag Urban children – Cough: 1.031 (0.957, 1.112) 0 0.997 (0.923, 1.077) 1 1.018 (0.940, 1.103): 2 1.094 (0.895, 1.338) 0-6 avg Phlegm: 0.998 (0.899, 1.108) 0 1.035 (0.928, 1.154) 1 1.121 (1.013, 1.240) 2 1.043 (0.822, 1.324) 0-6 avg Upper respiratory symptoms: 1.004 (0.949, 1.063) 0 0.975 (0.922, 1.031) 1 0.951 (0.895, 1.010) 2 0.849 (0.687, 1.050) 0-6 avg Lower respiratory symptoms: 0.984 (0.872, 1.110) 0 0.919 (0.812, 1.039) 1 0.894 (0.771, 1.036) 2 0.800 (0.617, 1.038) 0-6 avg Rural children (control) Cough: 0.997 (0.900, 1.105) 0 1.003 (0.906, 1.112) 1 0.997 (0.891, 1.116) 2 0.855 (0.655, 1.115) 0-6 avg Phlegm: 1.024 (0.880, 1.192) 0 0.995 (0.853, 1.160) 1 1.117 (0.956, 1.305)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Goncalves et al. (2005, 089884) **Period of Study:** Dec-Mar 1992/93. Dec-Mar 1993/94 **Location:** Sao Paulo	**Outcome:** Respiratory morbidity/admissions **Age Groups:** Children < 13 yrs **Study Design:** Time series **Statistical Analyses:** Principal component analysis **Covariates:** Daily mean temperature, daily mean water vapor density, solar radiation **Season:** summer **Dose-response Investigated?** No **Statistical Package:** NR **Lags Considered:** Lag 3	**Pollutant:** PM_{10} **Averaging Time:** 24 h Copollutant: SO_2 , O_3	PCA coefficients: PC1, PC2, PC3: Summer 1992/1993: PM_{10}: 0.69, 0.45, 0.13 Solar Radiation: -0.04, 0.94 to -0.12 Mean Temperature: 0.62, 0.44 to -0.47 Mean Water Vapor Density: 0.73 to -0.46 to -0.26 SO_2: 0.78 to -0.03, 0.33 O_3: 0.18, 0.63, 0.37 Respiratory Mortality: 0.05 to -0.02, 0.81 Variations explained by Principal Component: PC1: 0.29 PC2: 0.27 PC3: 0.17 Summer 1993/1994: PM_{10}: 0.38, 0.80 to -0.23 Solar Radiation: 0.02, 0.09 to -0.97 Mean Temperature: 0.71, 0.40 to -0.37 Mean Water Vapor Density: 0.88, 0.25, 0.09 SO_2: 0.01, 0.92, 0.00 O_3: 0.47 to -0.06 to -0.35 Respiratory Mortality: -0.73, 0.11, 0.08 Variations explained by Principal Component: PC1: 0.31 PC2: 0.25 PC3: 0.18 Notes: Association between respiratory morbidity and air pollution more likely during summer with smaller contrasts in synoptic weather condition (summer 1992/93) but respiratory morbidity more related to weather variables during summer with larger contrasts (summer 1993/94).
Reference: Gordian and Choudhury (2003, 054842) **Period of Study:** 1994-Dec 1996 **Location:** Anchorage, Alaska	**Outcome:** Asthma medication among school children **Age Groups:** Elementary school children (kindergarten-6th grade) **Study Design:** Time series **Statistical Analyses:** Time series regression model **Covariates:** Day of the week, month, time trend, temperature **Season:** All seasons **Dose-response Investigated?** No **Statistical Package:** SAS **Lags Considered:** 1, 2, 7, 14, 21, 28	**Pollutant:** PM_{10} **Averaging Time:** 24 h Mean (SD): 36.11 (30.46) **Range (Min, Max):** 2.96, 210.0 **Monitoring Stations:** 1	Model regression slope coefficient for PM_{10} (estimated SE) lag: 7.25 (2.88) lag 21 RR: 1.075 (1.016, 1.138) Notes: PM_{10} coefficients for other lags were also statistically significant but not reported.

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Harre et al. (1997, 095726) **Period of Study:** 6/1994–8/1994 **Location:** Christchurch, New Zealand	**Outcome:** Respiratory symptoms, Cough, Wheeze, Chest tightness, Shortness of breath, Change in sputum volume, Nose, throat, or eye irritation, PEFR **Study Design:** Prospective cohort **Statistical Analyses:** Poisson, log linear regression **Age Groups:** >55	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Copollutants:** CO SO_2 NO_2	Increment: 35.04 $\mu g/m^3$ Relative Risk (Lower CI, Upper CI) lag: Chest symptoms: 1.38 (1.07, 1.78) 1 Wheeze: 0.97 (0.75, 1.26) 1 Nebulizer Use: 0.71 (0.42, 1.18) 1 Inhaler Use: 0.94 (0.78, 1.13) 1
Reference: Hastings and Jardine (2002, 030344) **Period of Study:** 1997-1998 **Location:** Bosnia (US military camps)	**Outcome:** Weekly rates of upper respiratory disease (URD), reported by the medical treatment facility in each military camp **Age Groups:** US soldiers **Study Design:** Ecologic (at level of military camp) **N:** 5 camps **Statistical Analyses:** 1.Pearson correlations between weekly URD rates and weekly PM_{10} (avg and max) 2.Kruskal Wallace test to compare URD rates in the 4 exposure quartiles 3. Mann Whitney test to compare dichotomized exposure groups (above and below 50th percentile) **Dose-response Investigated?** Yes **Lags Considered:** Weekly rates of URD disease were related to avg weekly PM levels in the same week	**Pollutant:** PM_{10} Mean (SD): PM_{10} avg: 75.5 PM_{10} max: 92.9 Percentiles: PM_{10} max: 25th: 58.57 50th: 74.55 75th: 107.56 PM_{10} avg: 25th: 42.19 50th: 64.17 75th: 81.75 **Range (Min, Max):** PM_{10} avg: 25.0, 338.7 PM_{10} max: 25.0, 338.7 **Monitoring Stations:** at least one in each of the 5 camps	PM max Quartiles (combining all camps): Q1: <58.7 $\mu g/m^3$ Q2: 60.1 to <75.54 $\mu g/m^3$ Q3: 78.56 to <107.56 $\mu g/m^3$ Q4: >107.56 $\mu g/m^3$ For dichotomous analysis cutoff = 74.55 $\mu g/m^3$ PM avg Quartiles (combining all camps): Q1: <42.19 $\mu g/m^3$ Q2: 42.19 to 64.17 $\mu g/m^3$ Q3: 64.17 to 81.75 $\mu g/m^3$ Q4: >81.75 $\mu g/m^3$ For dichotomous analysis cutoff = 64.17 $\mu g/m^3$ Pearson correlation coefficients between URD rate and PM category [p-value]: PM_{10} max: quartiles of PM*URD rates All camps 0.203 [0.041] Blue Factory camp 0.277 [0.095] Comanche 0.165 [0.237] Demi 0.639 [0.123] McGovern 0.535 [0.177] Tuzla Main 0.107 [0.327] PM_{10} max: dichotomous PM*URD rates: All camps 0.283 [0.007] Blue Factory camp 0.038 [0.430] Comanche 0.282 [0.107] Demi 0.927 [0.012] McGovern 0.853 [0.033] Tuzla Main 0.155 [0.258] PM_{10} avg: quartiles of PM*URD rates: All camps 0.149 [0.101] Blue Factory camp 0.301 [0.077] Comanche 0.246 [0.141] Demi 0.437 [0.231] McGovern 0.853 [0.033] Tuzla Main 0.182 [0.222]

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			PM_{10} avg: dichotomous PM*URD rates: All camps 0.060 [0.305]
			Blue Factory camp -0.075 [0.365]
			Comanche 0.143 [0.268]
			Demi N/A*
			McGovern N/A*
			Tuzla Main 0.123 [0.303]
			Kruskal Wallace p-value comparing URD rates across exposure quartiles: PM_{10} max
			All camps 0.047
			Blue Factory camp 0.321
			Comanche 0.556
			Demi 0.165
			McGovern 0.202
			Tuzla Main 0.554
			PM_{10} avg
			All camps 0.672
			Blue Factory camp 0.809
			Comanche 0.658
			Demi 0.564
			McGovern 0.157
			Tuzla Main 0.891
			Mann-Whitney p-value comparing URD rates between upper and lower 50th percentile of PM: PM_{10} max
			All camps 0.034
			Blue Factory camp 0.173
			Comanche 0.314
			Demi 0.083
			McGovern 0.401
			Tuzla Main 0.481
			PM_{10} avg
			All camps 0.824
			Blue Factory camp 0.682
			Comanche 0.508
			Demi N/A*
			McGovern N/A*
			Tuzla Main 0.656
			Notes: * there were no days that fell in the upper 50%ile for PM avg in these camps
			-Rates of URD by PM quartiles for each camp presented in figures. Authors state, "Generally the avg URD rate increased with quartile of maximum exposure…the trend was not as clear for quartiles of PM_{10} avg exposure"
Reference: Hong et al. (2007, 091347) **Period of Study:** March 23-May 3, 2004	**Outcome:** Peak expiratory flow rate (PEFR) **Age Groups:** 3rd to 6th grade (mean	**Pollutant:** PM_{10} **Averaging Time:** 24 h	Effect Estimate: Regression coefficients of morning and daily mean PEFR on PM_{10} and metal components using linear mixed-

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Location: School on the Dukjeok Island near Incheon City, Korea	age = 9.6 yrs) **Study Design:** panel study **N:** 43 schoolchildren **Statistical Analyses:** Mixed linear regression **Covariates:** age, sex, height, weight, asthma history, and passive smoking exposure at home **Dose-response Investigated?** No **Lags Considered:** 0, 1, 2, 3, 4, 5	Mean (SD): 35.30 (23.48) 50th (Median): 29.36 **Range (Min, Max):** (12.24-124.87) PM Component: Fe: mean = 0.208 (0.203) $\mu g/m^3$ Median = 0.112 **Range (Min, Max):** (0.061-0.806) Mn: mean = 0.008 (0.005) $\mu g/m^3$ Median = 0.007 **Range (Min, Max):** (0.000-0.019) Pb: mean = 0.051 (0.031) $\mu g/m^3$ Median = 0.051 **Range (Min, Max):** (0.011-0.155) Zn: mean = 0.021 (0.021) $\mu g/m^3$ Median = 0.013 **Range (Min, Max):** (0.006-0.112) Al: mean = 0.085 (0.100) $\mu g/m^3$ Median = 0.031 **Range (Min, Max):** (0.017-0.344) Copollutant: $PM_{2.5}$	effects regression Lag 1 (PM_{10}) Morning PEFR Crude: ß = -0.00, p = 0.99 Adjusted: ß = -0.04, p = 0.37 Mean PEFR Crude: ß = 0.00, p = 0.93 Adjusted: ß = -0.05, p = 0.12 Lag 1 (logFe) Morning PEFR Crude: ß = -1.26, p = 0.31 Adjusted: ß = -3.24, p = 0.13 Mean PEFR Crude: ß = -1.20, p = 0.20 Adjusted: ß = -2.37, p = 0.15 Lag 1 (logMn) Morning PEFR Crude: ß = -4.40, p < 0.01 Adjusted: ß = -9.82, p < 0.01 Mean PEFR Crude: ß = -4.05, p < 0.01 Adjusted: ß = -8.44, p < 0.01 Lag 1 (logPb) Morning PEFR Crude: ß = -6.79, p < 0.01 Adjusted: ß = -6.83, p < 0.01 Mean PEFR Crude: ß = -6.23, p < 0.01 Adjusted: ß = -6.37, p < 0.01 Lag 1 (logZn) Morning PEFR Crude: ß = -0.55, p = 0.71 Adjusted: ß = -0.98, p = 0.59 Mean PEFR Crude: ß = 1.33, p = 0.24 Adjusted: ß = 1.53, p = 0.28 Lag1 (logAl) Morning PEFR Crude: ß = -0.58, p = 0.57 Adjusted: ß = -2.22, p = 0.25 Mean PEFR Crude: ß = -0.59, p = 0.45 Adjusted: ß = -1.48, p = 0.32 Regression coefficients of morning and daily mean PEFR on metal components of PM_{10} and GSTM1 and GSTT1 genotype

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			using linear mixed-effects regression
			Lag 1 (logPb)
			Morning PEFR: ß = -7.26, p < 0.01
			Mean PEFR: ß = -6.43, p < 0.01
			GSTM1
			Morning PEFR: ß = 21.19, p = 0.23
			Mean PEFR: ß = 20.09, p = 0.25
			Lag 1 (logMn)
			Morning PEFR: ß = -10.31, p < 0.01
			Mean PEFR: ß = -8.66, p < 0.01
			GSTM1
			Morning PEFR: ß = 21.02, p = 0.23
			Mean PEFR: ß = 19.84, p = 0.25
			Lag 1 (logPb)
			Morning PEFR: ß = -7.26, p < 0.01
			Mean PEFR: ß = -6.43, p < 0.01
			GSTT1
			Morning PEFR: ß = 2.07, p = 0.90
			Mean PEFR: ß = -2.39, p < 0.88
			Lag 1 (logMn)
			Morning PEFR: ß = -10.32, p < 0.01
			Mean PEFR: ß = -8.67, p < 0.01
			GSTT1
			Morning PEFR: ß = 2.02, p = 0.90
			Mean PEFR: ß = 2.33, p = 0.88

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Hwang et al. (2006, 088971)	**Outcome:** Allergic rhinitis	**Pollutant:** PM_{10}	Increment: 10 $\mu g/m^3$
Period of Study: 2001	**Study Design:** Cross-sectional	**Averaging Time:** 1-h avg	Odds Ratio (Lower CI, Upper CI)
Location: Taiwan	**Statistical Analyses:** Two-stage hierarchical models	Mean (SD): 55.58 (16.57)	lag:
	Age Groups: 6-15	**Range (Min, Max):**	PM_{10} alone: 1.00 (0.99, 1.02)
		(29.36, 99.58)	NO_X, PM_{10}: 0.99 (0.97, 1.00)
		Copollutants (correlation):	CO, PM_{10}: 1.00 (0.99, 1.01)
		CO: r = 0.27	O_3, PM_{10}: 1.00 (0.99, 1.02)
		NO_X: r = 0.34	Gender
		O_3: r = 0.28	Male: 1.02 (0.99, 1.04)
		SO_2: r = 0.58	Female: 0.99 (0.97, 1.02)
			Parental atopy*
			Yes: 1.00 (0.98, 1.03)
			No: 1.01 (0.99, 1.03)
			Parental education
			< 6 years: 1.05 (0.96, 1.14)
			6-8 years: 1.03 (0.98, 1.07)
			9-11 years: 1.00 (0.98, 1.03)
			12+ years: 0.99 (0.97, 1.02)
			Environmental tobacco smoke
			Yes: 1.01 (0.99, 1.03)
			No: 1.00 (0.98, 1.03)
			Visible mold**
			Yes: 1.02 (0.99, 1.06)
			No: 1.00 (0.98, 1.02)
			* Parental atopy was a measure of genetic predisposition and was defined as the father or the mother of the index child ever having been diagnosed as having asthma, allergic rhinitis, or atopic eczema.
			** Visible mold found in the home.
Reference: Jalaludin et al. (2004, 056595)	**Outcome:** Respiratory symptoms, Wheeze, Dry cough, Wet cough	**Pollutant:** PM_{10}	**Increment:** 10 $\mu g/m^3$
Period of Study: 2/1/1994–12/31/1994	**Study Design:** Longitudinal study panel	**Averaging Time:** 24-h avg	**Odds Ratio (Lower CI, Upper CI)**
Location: Western and southwestern Sydney, Australia	**Statistical Analyses:** Logistic regression model (GEE)	Mean (SD): 22.8 (13.8)	**Lag**
	Age Groups: 9-11	**IQ Range (25th,75th):** (12.00, 122.8)	Wheeze
		Copollutants (correlation): O_3: r = 0.13	1.01 (0.99, 1.03)
		NO_2: r = 0.26	0
			1.01 (0.97, 1.04)
			1
			0.99 (0.96, 1.03)
			2
			1.02 (0.98, 1.06)
			0-2 avg
			1.04 (0.99, 1.10)
			0-5 avg
			Dry Cough
			1.00 (0.98, 1.03)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			0
			1.00 (0.97, 1.03)
			1
			1.00 (0.97, 1.02)
			2
			1.00 (0.97, 1.03)
			0-2 avg
			1.03 (0.98, 1.08)
			0-5 avg
			Wet Cough
			1.01 (0.99, 1.04)
			0
			0.99 (0.97, 1.01)
			1
			1.00 (0.97, 1.03)
			2
			0.99 (0.96, 1.02)
			0-2 avg
			0.99 (0.94, 1.04)
			0-5 avg
			Inhaled B2-agonist Use
			0.99 (0.98, 1.01)
			0
			1.00 (0.98, 1.03)
			1
			0.99 (0.97, 1.01)
			2
			1.00 (0.97, 1.02)
			0-2 avg
			1.02 (0.98, 1.06)
			0-5 avg
			Inhaled Corticosteroid Use
			1.00 (0.99, 1.01)
			0
			1.00 (0.99, 1.02)
			1
			1.00 (0.99, 1.02)
			2
			1.00 (0.98, 1.02)
			0-2 avg
			1.00 (0.97, 1.02)
			0-5 avg
			Doctor Visit for Asthma
			1.11 (1.04, 1.19)
			0

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			1.10 (1.02, 1.19)
			1
			1.15 (1.06, 1.24)
			2
			1.11 (1.03, 1.20)
			0-2 avg
			1.14 (0.98, 1.31)
			0-5 avg
			OR for respiratory symptoms and PM_{10} exposure by different groups
			All children
			Wheeze: 1.01 (0.99, 1.04)
			Dry Cough: 1.00 (0.97, 1.02)
			Wet Cough: 1.01 (0.98, 1.04)
			Inhaled B2-agonist Use: 1.00 (0.98, 1.02)
			Inhaled Corticosteroid Use: 0.99 (0.98, 1.01)
			Doctor Visit for asthma: 1.11 (1.03, 1.19)
			Group 1*
			Wheeze: 1.01 (0.98, 1.04)
			Dry Cough: 0.97 (0.94, 0.99)
			Wet Cough: 1.00 (0.97, 1.03)
			Inhaled B2-agonist use: 1.00 (0.98, 1.02)
			Inhaled Corticosteroid Use: 1.00 (0.98, 1.01)
			Doctor Visit for asthma: 1.09 (0.98, 1.21)
			Group 2**
			Wheeze: 1.01 (0.97, 1.05)
			Dry Cough: 1.02 (0.98, 1.06)
			Wet Cough: 1.01 (0.96, 1.06)
			Inhaled B2-agonist use: 0.99 (0.94, 1.05)
			Inhaled Corticosteroid Use: 0.99 (0.97, 1.01)
			Doctor Visit for asthma: 1.12 (1.02, 1.23)
			Group 3***
			Wheeze: 1.08 (0.90, 1.31)
			Dry Cough: 1.01 (0.91, 1.11)
			Wet Cough: 1.02 (0.94, 1.11)
			Inhaled B2-agonist use: 0.98 (0.84, 1.11)
			Inhaled Corticosteroid Use: 1.27 (1.08, 1.49)
			Doctor Visit for asthma: NR
			*Group 1 consists of children with a history of wheeze in the past 12 months, positive histamine challenge, and doctor diagnosed asthma.
			**Group 2 consists of children with a

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			history of wheeze in the past 12 months and doctor diagnosed asthma. ***Group 3 consists of children only with a history y of wheeze in the past 12 months.
Reference: Jansen, et al. (2005, 082236) **Period of Study:** 1987-2000 **Location:** Seattle, WA	**Outcome:** FENO: fractional exhaled nitrogen oxide, Spirometry, Blood pressure, SaO2: oxygen saturation, Pulse rate **Age Groups:** 60-86-years-old **Study Design:** short-term cross-sectional case series **N:** 16 subjects diagnosed with COPD, asthma, or both **Statistical Analyses:** linear mixed effects model with random intercepts **Covariates:** age, relative humidity, temperature, medication use **Season:** winter 2002-2003 **Dose-response Investigated?** No **Statistical Package:** STATA	**Pollutant:** PM_{10} **Averaging Time:** 24-h **Mean (SD):** Fixed-site Monitor: 18.0 All Subjects (N = 16) Indoor, home: 11.93 Outdoor, home: 13.47 Personal: 23.34 Asthmatic Subjects (N = 7) Indoor, home: 12.54 Outdoor, home: 11.86 Personal: 26.88 COPD Subjects (N = 9) Indoor, home: 11.45 Outdoor, home: 14.76 Personal: 19.91 **Range (Min, Max):** Fixed-site Monitor 2.5, 51 **IQR:** All Subjects Indoor, home: 6.93 Outdoor, home: 9.53 Personal: 20.72 Asthmatic Subjects Indoor, home: 10.19 Outdoor, home: 8.77 Personal: 20.08 COPD Subjects Indoor, home: 4.56 Outdoor, home: 6.14 Personal: 19.94	**PM Increment:** 10 $\mu g/m^3$ Slope [95% CI]: dependence of FENO concentration [ppb] on PM_{10} Asthmatic Subjects Indoor, home: 3.81 [-0.86: 8.50] Outdoor, home: 5.87 [2.87: 8.88]* Personal: 0.66 [-0.56: 1.88] COPD Subjects Indoor, home: 2.19 [-3.48: 7.87] Outdoor, home: 4.45 [-1.11: 10.01] Personal: 0.17 [-1.61: 1.96] Results indicate that FENO may be a more sensitive biomarker of PM exposure than other traditional health endpoints.
Reference: Johnston, et al. (2006, 091386) **Period of Study:** 7 months. (April 7 through November 7, 2004) **Location:** Darwin, Australia	**Outcome:** Asthma symptoms **Age Groups:** all ages **Study Design:** Time-series **N:** 251 people (130 adults, 121 children **Statistical Analyses:** Logistic regression model **Covariates:** minimum air temperature, doctor visits for influenza and the prevalence of asthma symptoms and, the fungal spore count and both onset of asthma symptoms and commencement of reliever medication **Season:** "dry season"-specific months NR, note Southern Hemisphere **Dose-response Investigated?** No **Statistical Package:** STATA8	**Pollutant:** PM_{10} **Averaging Time:** daily **Mean (SD):** 20 (6.4) **Range (Min, Max):** 2.6-43.3 **PM Component:** Vegetation fire smoke (95%) and motor vehicle emissions (5%) **Monitoring Stations:** 1 **Correlation:** $PM_{2.5}$ r = 0.90	**PM Increment:** 10 $\mu g/m^3$ **RR Estimate [Lower CI, Upper CI]** Symptoms attributable to asthma Overall-1.010 (0.98,1.04) Adults–1.027 (0.987,1.068) Children–0.930 (0.966, 1.060) Using preventer- 1.022 (0.985, 1.060) Became symptomatic Overall- 1.240 (1.106,1.39) Adults- 1.277 (1.084,1.504) Children- 1.247 (1.058,1.468) Using preventer–1.317 (1.124,1.543) Used Reliever

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
	Lags Considered: 0-5 days		Overall- 1.010 (0.99, 1.04)
			Adults- 1.026 (0.990, 1.063)
			Children- 1.006 (0.960,1.055)
			Using preventer–1.035 (1.004,1.060)
			Commenced Reliever
			Overall- 1.132 (0.99, 1.29)
			Adults- 1.199 (0.994, 1.446)
			Children- 1.093 (0.906,1.319)
			Using preventer–1.194 (0.996, 1.432)
			Commenced Oral Steroids
			Overall- 1.540 (1.01, 2.34)
			Adults- 1.752 (1.008, 3.045)
			Children- 1.292 (0.682, 2.448)
			Using preventer–1.430 (0.888, 2.304)
			Asthma Attack
			Overall- 1.030 (0.95, 1.12)
			Adults- 1.08 (0.976, 1.202)
			Children- 0.861 (0.710, 1.044)
			Using preventer–1.051 (0.939,1.175)
			Exercise induced asthma
			Overall- 0.980 (0.92, 1.05)
			Adults- 0.988 (0.902, 1.081)
			Children- 0.972 (0.844,1.119)
			Using preventer–1.026 (0.928,1.134)
			Saw a health professional for asthma
			Overall- 1.030 (0.85, 1.26)
			Adults- 1.064 (0.794, 1.424)
			Children- 0.998 (0.749,1.328)
			Using preventer–0.924 (0.731, 1.169)
			Missed school or work due to asthma
			Overall- 1.102 (0.941, 1.290)
			Adults- 1.135 (0.897, 1.435)
			Children- 1.073 (0.862,1.333)
			Using preventer–1.025 (0.857,1.228)
			Mean daily number of asthma symptoms
			Overall- 1.020 (1.001,1.031)
			Adults- 1.027 (1.005,1.049)
			Children- 1.016 (0.986,1.047)
			Using preventer–1.034 (1.011,1.058)
			Mean Daily number of applications of reliever
			Overall- 1.020 (1.00,1.030)
			Adults- 1.032 (1.008, 1.057)
			Children- 1.002 (0.969,1.034)
			Using preventer–1.022 (1.001,1.043)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Just et al. (2002, 035429) **Period of Study:** 4/1/1996–6/30/1996 **Location:** Paris, France	**Outcome:** Incident and prevalent episodes of asthma attacks, nocturnal cough, wheeze, symptoms of irritation, respiratory infections, supplementary use of β2-agonists, Z-transformed peak expiratory flow (PEF), daily PEF variability **Age Groups:** 7-15 years old **Study Design:** Cohort **N:** 82 children **Statistical Analyses:** Linear regression, logistic regression, GEE **Covariates:** Effects of time trend, day of the week, weather, pollen levels **Season:** Spring/summer **Lags Considered:** 0, 0-2 mean, 0-4 mean	**Pollutant:** PM_{10} **Averaging Time:** Daily **Mean (SD):** 23.5 (8.4) **Range (Min, Max):** 9.0, 44.0 **Monitoring Stations:** 5 **Copollutant (correlation):** BS: 0.59 SO_2: 0.70 NO_2: 0.54 O_3: 0.21 temp: 0.04 humid: -0.41	**PM Increment:** 10 $\mu g/m^3$ for binary responses data (results that use odds ratios [ORs]) **Incident episodes of** 1) Asthma a) lag 0: 1.06 (0.61, 1.83) b) 0-2 mean: 1.09 (0.48, 2.49) c) 0-4 mean: 1.07 (0.44, 2.65) 2) Nocturnal cough a) lag 0: 1.10 (0.88, 1.37) b) 0-2 mean: 1.03 (0.77, 1.37) c) 0-4 mean: 1.11 (0.86, 1.42) 3) Respiratory infections a) lag 0: 0.64 (0.35, 1.15) b) 0-2 mean: 0.74 (0.38, 1.43) c) 0-4 mean: 0.99 (0.58, 1.68) **Prevalent episodes of** 1) Asthma a) lag 0: 1.07 (0.72, 1.59) b) 0-2 mean: 1.18 (0.64, 2.17) c) 0-4 mean: 1.16 (0.63, 2.13) 2) Nocturnal cough a) lag 0: 1.05 (0.83, 1.34) b) 0-2 mean: 1.10 (0.81, 1.50) c) 0-4 mean: 1.09 (0.79, 1.52) 3) Respiratory infections a) lag 0: 1.17 (0.68, 2.03) b) 0-2 mean: 1.31 (0.51, 3.36) c) 0-4 mean: 1.71 (0.71, 4.12) 4) Eye irritation a) lag 0: 1.18 (1.01, 1.39) b) 0-2 mean: 1.28 (1.03, 1.59) c) 0-4 mean: 1.42 (1.12, 1.80) **Analysis restricted to days with no steroid use** **Incident episodes of** 1) Eye irritation a) lag 0: 1.07 (0.66, 1.71) b) 0-2 mean: 0.83 (0.45, 1.53) c) 0-4 mean: 0.92 (0.46, 1.83) 2) Throat irritation a) lag 0: 1.33 (0.66, 2.69) b) 0-2 mean: 1.28 (0.58, 2.80) c) 0-4 mean: 1.06 (0.38, 2.95) 3) Nose irritation a) lag 0: 0.74 (0.48, 1.13)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			b) 0-2 mean: 0.76 (0.42, 1.36) c) 0-4 mean: 0.96 (0.53, 1.73) **Prevalent episodes of** 1) Eye irritation a) lag 0: 1.20 (0.88, 1.65) b) 0-2 mean: 1.71 (0.97, 3.01) c) 0-4 mean: 1.97 (1.03, 3.76) 2) Throat irritation a) lag 0: 1.23 (0.83, 1.82) b) 0-2 mean: 1.08 (0.68, 1.73) c) 0-4 mean: 0.91 (0.47, 1.73) 3) Nose irritation a) lag 0: 1.20 (0.91, 1.58) b) 0-2 mean: 1.09 (0.78, 1.52) c) 0-4 mean: 1.09 (0.73, 1.61) **Notes:** The authors noted that incident or prevalent wheeze was not correlated with levels of any type of pollutant also, they state no relationship was observed between PEF variables and levels of PM. The authors also note that in a multipollutant model assessing independent effects of PM and O_3 on prevalent episodes of eye irritation (mean 0-4), the PM parameter decreased and was not significant (p = 0.19).
Reference: Kulkarni et al (2006, 089257) **Period of Study:** 11/2002–12/2003 **Location:** Leicester, United Kingdom	**Outcome:** Lung function by spirometry: FVC, FEV_1, FEV_1: FVC, FEF_{25-75} **Age Groups:** 8-15 **Study Design:** Cross-sectional **N:** 114 children, 64 provided sputum for assessment of carbon content of macrophages. **Statistical Analyses:** Linear regressions, Spearman rank correlations. Mann-Whitney, Chi-square and unpaired t tests were used to compare results between asthmatic and non asthmatic children **Covariates:** BMI, sex, exercise, traffic PM_{10} **Dose-response Investigated?** Yes **Statistical Package:** SPSS	**Pollutant:** Primary PM_{10} ($\mu g/m^3$) concentration was modeled, and was considered a covariate for carbon content of macrophages. Carbon content of alveolar macrophages was the primary variable of interest. **Averaging Time:** 1 yr 50th(Median): Children without asthma, 1.21 Children with asthma, 1.81 **Range (Min, Max):** Children without asthma, 0.10, 2.17 Children with asthma, 0.17, 2.13 **PM Component:** Carbon content in alveolar macrophages **Monitoring Stations:** NR. **Copollutant (correlation):** vs carbon content in macrophages (increment, coefficient [range])–1.0 $\mu g/m^3$, 0.1 [0.01-0.18]	**PM Increment:** 1.0 $\mu g/m^3$ **% Change [Lower CI, Upper CI]:** Single pollutant model: FEV_1: -4.3 [-8.5, 0.2] p = 0.04 R^2 = 0.06 Single pollutant model: FVC: -1.2 [-5.6, 3.2] p = 0.59 R^2 = 0.005 Single pollutant model: FEF_{25-75}: -8.6 [-17.3, 0.1] p = 0.05 R^2 = 0.06 2 pollutant model with Macrophage Carbon: FEV_1: PM_{10} -2.9 [-6.9, 1.2] p = 0.17 (FVC): PM_{10} 0.1 [-4.4, 4.6] p = 0.96 FEF_{25-75}: PM_{10} -5.5 [-14.2, 3.1] p = 0.21

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Kuo, et al. (2002, 036310) **Period of Study:** 1-yr period (year not specified) **Location:** Central Taiwan	**Outcome:** Asthma (yes/no) **Age Groups:** 13-16 years **Study Design:** Cohort **N:** 12926 total children 775 asthmatic children 8 junior high schools **Statistical Analyses:** Pearson correlation coefficients Logistic regression **Covariates:** Gender, age, residential area, level of parental education, number cigarettes smoked by family members, incense burning in the home, frequency of physical activities **Dose-response Investigated?** No **Statistical Package:** SAS 6.12 **Lags Considered:** Monthly averages at each school	**Pollutant:** PM_{10} **Averaging Time:** 1-h **Mean (SD):** School A: 59.7 School B: 65.3 School C: 84.3 School D: 59.2 School E: 75.3 School F: 60.2 School G: 54.1 School H: 69.0 **Monitoring Stations:** 8 (1 for each school)	**PM Increment:** Dichotomized annual avg: $< 65.9\ \mu g/m^3$ $\geq 65.9\ \mu g/m^3$ OR Estimate [Lower CI, Upper CI] lag: Crude (outcome = asthma, yes/no) $< 65.9\ \mu g/m^3$: 1 (ref) $\geq 65.9\ \mu g/m^3$: 0.837 [NR] Adjusted (outcome = asthma, yes/no) $< 65.9\ \mu g/m^3$: 1 (ref) $\geq 65.9\ \mu g/m^3$: 0.947 [0.640, 1.401] **Notes:** asthma prevalence was highest in urban areas and lowest in rural areas Pearson correlation between annual PM levels at each school and asthma prevalence at each school: 0.214 [$p > 0.05$]
Reference: Lagorio et al. (2006, 089800) **Period of Study:** 5/24/1999 to 6/24/1999 and 11/181999 to 12/22/1999 **Location:** Rome, Italy	**Outcome:** Lung function of subjects (FVC and FEV_1) with COPD, Asthma **Age Groups:** COPD 50 to 80 yrs Asthma 18 to 64 yrs **Study Design**: Time series panel **N:** COPD N = 11 Asthma N = 11 **Statistical Analyses:** Non-parametric Spearman correlation GEE **Covariates:** COPD and IHD: daily mean temperature, season variable (spring or winter), relative humidity, day of week Asthma: season variable, temperature, humidity, and β-2-agonist use **Season:** Spring and winter **Dose-response Investigated?** Yes **Statistical Package:** STATA **Lags Considered:** 1–3 days	**Pollutant:** PM_{10} **Averaging Time:** 24-h **Mean (SD):** Overall: 42.8 (21.8) Spring: 36.9 (10.8) Winter: 49.0 (28.1) **Range (Min, Max):** (7.9, 123) **PM Component:** NR **Monitoring Stations**: Two fixed sites: (Villa Ada and Istituto superior di Sanita) **Copollutant (correlation):** NO_2 r = 0.45 O_3 r = -0.36 CO r = 0.55 SO_2 r = 0.21 $PM_{10-2.5}$ r = 0.61 $PM_{2.5}$ r = 0.93	**PM Increment:** 1 $\mu g/m^3$ They observed negative association between ambient PM_{10} and respiratory function (FVC and FEV_1) in the COPD panel. The effect on FVC was seen at lag 24 h, 48 h, and 72 h. The effect on FEV_1 was evident at lag 72 h. There was no statistically significant effect of PM_{10} on FVC and FEV_1 in the asthmatic and IHD panels. **β Coefficient (SE)** **COPD** FVC(%) 24 h -0.66 (0.30) 48-h -0.75 (0.35) 72-h -0.94 (0.47) FEV_1(%) 24 h -0.37 (0.27) 48-h -0.58 (0.31) 72-h -0.87 (0.43) **Asthma** FVC(%) 24 h -0.12 (0.24) 48-h -0.09 (0.29) 72-h -0.08 (0.36) FEV_1(%) 24 h -0.28 (0.28) 48-h -0.40 (0.34) 72-h -0.40 (0.43)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Lee, et al. (2007, 093042) **Period of Study:** 2000-2001 **Location:** South-Western Seoul Metropolitan area, Seoul, South Korea	**Outcome:** PEFR (peak expiratory flow rate), lower respiratory symptoms (cold, cough, wheeze) **Age Groups:** 61-89 years of age (77.8 mean age) **Study Design:** longitudinal panel survey **N:** 61 adults **Statistical Analyses:** Logistic regression model **Covariates:** Temperature (Celsius), relative humidity, age, season **Dose-response Investigated?** No **Statistical Package:** SAS 8.0 **Lags Considered:** 0-4 days	**Pollutant:** PM_{10} **Averaging Time:** 24-h **Mean (SD):** 71.40 (30.69) Percentiles: 25th: 43.47 50th(Median): 74.92 75th: 87.54 **Range (Min, Max):** 26.23, 148.34 **Monitoring Stations:** 2	**PM Increment:** 10 $\mu g/m^3$ **Effect Estimate [Lower CI, Upper CI]** **lag:** PEFR (peak expiratory flow rate) -0.39 (-0.63 to -0.14) 1 day relative odds of a lower respiratory symptom (cold, cough, wheeze) 1.015 (0.900,1.144) 1 day
Reference: Lewis, et al (2005, 081079) **Period of Study:** winter 2001-spring 2002 **Location:** Detroit, Michigan, USA	**Outcome:** Poorer lung function (increased diurnal variability and decreased forced expiratory volume) **Age Groups:** 7-11 years old **Study Design:** longitudinal cohort study **N:** 86 children **Statistical Analyses:** descriptive statistics and bivariate analyses of exposures, multivariable regression models that included interaction terms between exposure measures and CS use or, alternatively, presence of a URI, multivariate analog of linear regression. **Covariates:** sex, home location, annual family income, presence of one or more smokers in household, race, season (entered as dummy variables), and parameters to account for intervention group effect. **Season: W**inter 2001 (February 10–23), spring 2001 (May 5–18), summer 2001 (July 14–27), fall 2001 (September 22–October 5), winter 2002 (January 18–31), and spring 2002 (May 18–31). **Dose-response Investigated?** No **Lags Considered:** 1-2 days 3-5 days	**Pollutant:** PM_{10} **Averaging Time:** 2 weeks **Mean (SD):** Eastside 23.0 (13.5) Southwest 28.2 (16.1) **Range (Min, Max):** 2.9, 70.9 **PM Component:** ("likely" in southwest site) carbon and diesel emissions **Monitoring Stations:** 2 **Copollutant:** $PM_{2.5}$ 0.93 O_3 Daily mean 0.59 O_3 8-h peak 0.57	**PM Increment:** 19.1 $\mu g/m^3$ **Lung function among children reporting use of maintenance CSs** Diurnal variability FEV_1 Lag 1: 1.53 [-0.85, 3.90] Lag 1: 2.94 [-1.07, 6.96] $PM_{10} + O_3$ Lag 2: 5.32 [0.32, 10.33] Lag 2: 13.73 [8.23, 19.23] $PM_{10} + O_3$ Lag 3-5: 1.46 [-2.21,5.13] Lag 3-5: 3.30 [0.58, 6.02] $PM_{10} + O_3$ Lowest daily value FEV_1 Lag 1: -0.28 [-2.34, 1.77] Lag 1: -6.25 [-11.15 to -1.36] $PM_{10} + O_3$ Lag 2: -2.21 [-3.97 to -0.46] Lag 2: -5.97 [-11.06 to -0.87] $PM_{10} + O_3$ Lag 3-5: -2.58 [-7.65, 2.49] Lag 3-5: 1.98 [-0.38, 4.33] $PM_{10} + O_3$ **Lung function among children reporting presence of URI on day of lung function assessment** Diurnal variability FEV_1 Lag 1: 3.51 [-4.52,11.55] Lag 1: 3.21 [-1.28,7.71] $PM_{10} + O_3$ Lag 2: 1.12 [-4.62, 6.86] Lag 2: 5.40 [-0.82, 11.62] $PM_{10} + O_3$ Lag 3-5: 3.90 [0.34, 7.47] Lag 3-5: 6.27 [0.07, 12.47] $PM_{10} + O_3$ Lowest daily value FEV_1 Lag 1: -2.72 [-9.47, 4.03] Lag 1: -13.11 [-21.59 to -4.62] $PM_{10} + O_3$ Lag 2: 0.24 [-5.10, 4.63] Lag 2: -3.32 [-6.83, 0.18] $PM_{10} + O_3$ Lag 3-5: -4.48 [-8.36, 0.60] Lag 3-5: -3.17 [-5.82 to -0.51] $PM_{10} + O_3$

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Mar et al. (2004, 057309)	**Outcome:** Respiratory symptoms	**Pollutant:** PM_{10}	**PM Increment:** 10 $\mu g/m^3$
Period of Study: 1997-1999	**Age Groups:** Adults: Ages 20-51 yrs	**Mean (SD):**	**OR Estimate [Lower CI, Upper CI]**
Location: Spokane, Washington	Children: Ages 7-12 yrs	1997: 24.5 (18.5)	**lag:**
	Study Design: Time-series	1998: 20.6 (12.3)	**Adult Respiratory symptoms:** Wheeze: 1.01[0.93, 1.09]
	N: 25 people	1999: 16.8 (8.0)	lag 0
	Statistical Analyses: Logistic regression	**Monitoring Stations:**	0.98[0.91, 1.06]
	Covariates: Temperature, relative humidity, day of-the-wk	1 station	lag 1
	Statistical Package: STATA 6	**Copollutant (correlation):** PM_{10}	0.99[0.92, 1.06]
	Lags Considered: 0-2 days	PM_1	lag 2
		r = 0.48	Breath: 1.02[0.96, 1.08]
		$PM_{2.5}$	lag 0
		r = 0.61	1.01[0.97, 1.06]
		PM_{10}-2.5	lag 1
		r = 0.93	1.02[0.97, 1.06]
			lag 2
			Cough: 0.96[0.88, 1.05]
			lag 0
			0.97[0.90, 1.04]
			lag 1
			0.98[0.92, 1.05]
			lag 2
			Sputum: 1.01[0.92, 1.12]
			lag 0
			0.99[0.91, 1.08]
			lag 1
			1.00[0.93, 1.08]
			lag 2
			Runny Nose: 0.98[0.93, 1.04]
			lag 0
			0.97[0.93, 1.02]
			lag 1; 0.97[0.94, 1.01]
			lag 2
			Eye Irritation: 0.97[0.87, 1.08]
			lag 0
			0.97[0.88, 1.06]
			lag 1
			0.97[0.91, 1.04]
			lag 2
			Lower Symptoms: 0.96[0.91, 1.02]
			lag 0
			0.95[0.89, 1.00]
			lag 1
			0.95[0.90, 1.00]
			lag 2
			Any Symptoms: 0.97[0.93, 1.02]
			lag 0
			0.96[0.91, 1.00]
			lag 1
			0.95[0.91, 0.99]
			lag 2

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Mar et al. (2005, 087566) **Period of Study:** 1999-2001 **Location:** Seattle, Washington	**Outcome:** Pulmonary function (arterial oxygen saturation) and cardiac function (heart rate and blood pressure) **Study Design:** Time series **N:** 88 **Statistical Analyses:** Linear logistic regression **Age Groups:** > 57	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg	**Increment:** 10 $\mu g/m^3$ **% Increase (Lower CI, Upper CI)** **Lag** Indoor Systolic: 0.92 (-0.95, 2.78) 0 Diastolic: 0.63 (-0.29, 1.56) 0 Outdoor Systolic: -0.10 (-1.37, 1.18) 0 Diastolic: -0.03 (-0.79, 0.73) 0 Nephelometer Systolic: 0.35 (-0.91, 1.61) 0 Diastolic: -0.12 (-0.91, 0.67) 0 **% Increase between heart rate and PM_{10} exposure for people > 57** PM_{10} Indoor: 0.02 (-0.54, 0.58) 0 Outdoor: -0.48 (-1.03, 0.06) 0 Nephelometer: -0.31 (-0.76, 0.14) 0

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: McCormack et al, 2009 **Period of Study:** September 2001- April 2004 **Location:** East Baltimore, Maryland	**Outcome:** Asthma symptoms **Study Design:** Panel **Statistical Analysis:** Chi-square, Student t-test, Negative binomial regression models with GEE, Logistic regression with GEE **Statistical Package:** StataSE **Age Groups:** Asthmatic children aged 2-6	**Pollutant:** $PM_{10-2.5}$, $PM_{2.5}$ **Averaging Time:** 3d **Mean (SD) Unit:** $PM_{10-2.5}$: 17.4 ± 21.2 $\mu g/m^3$ $PM_{2.5}$: 40.3 ± 35.4 $\mu g/m^3$ **Range (Min, Max):** NR **Copollutant (correlation):** NR	**Increment:** 10 $\mu g/m^3$ **Relative Risk (Min CI, Max CI)** **Lag** Bivariate Models, $PM_{10-2.5}$ Cough, wheezing, chest tightness: 1.05 (0.99-1.10), p = 0.08 Slow down: 1.08 (1.03-1.13). p < 0.01 Symptoms with running: 1.03 (0.97-1.09). p = 0.39 Nocturnal symptoms: 1.06 (1.01-1.11), p = 0.03 Limited speech: 1.11 (1.05-1.18), p < 0.01 Rescue medication use: 1.06 (1.02-1.11), p < 0.01 Bivariate Models, $PM_{2.5}$ Cough, wheezing, chest tightness: 1.01 (0.98-1.05), p = 0.41 Slow down: 1.00 (0.97-1.04), p = 0.85 Symptoms with running: 1.04 (1.01-1.07), p = 0.14 Nocturnal symptoms: 1.02 (0.98-1.05), p = 0.37 Limited speech: 1.01 (0.95-1.07), p = 0.33 Rescue medication use: 1.03 (1.00-1.60), p = 0.06 Multivariate Models, $PM_{10-2.5}$ Cough, wheezing, chest tightness: 1.06 (1.01-1.12), p = 0.02 Slow down: 1.08 (1.02-1.14), p = 0.01 Symptoms with running: 1.00 (0.94-1.08), p = 0.81 Nocturnal symptoms: 1.08 (1.01-1.14), p = 0.02 Limited speech: 1.11 (1.03-1.19), p < 0.01 Rescue medication use: 1.06 (1.01-1.10), p = 0.02 Multivariate Models, $PM_{2.5}$ Cough, wheezing, chest tightness: 1.03 (0.99-1.07), p = 0.18 Slow down: 1.04 (1.00-1.09), p = 0.06 Symptoms with running: 1.07 (1.02-1.11), p < 0.01 Nocturnal symptoms: 1.06 (1.01-1.10), p = 0.01 Limited speech: 1.07 (1.00-1.14), p = 0.04 Rescue medication use: 1.04 (1.01-1.08), p = 0.04

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Mortimer et al. (2008, 187280) **Period of Study:** 1989-2000 **Location:** Joaquin Valley, California	**Outcome:** Respiratory Symptoms, Decreased lung function **Study Design:** Time series **Statistical Analyses:** Deletion/Substitution/ Addition algorithm (GEE) Logistic linear regression **Age Groups:** 6-11	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Copollutants (correlation):** CO: r = 0.05 NO_2: r = 0.30 O_3: r = 0.39	**Increment:** NR β (SE): **FVC:** PM_{10} (age 0-3 yrs): 0.0121 (0.0037) FEV_1: PM_{10} (age 0-3 yrs): 0.0102 (0.0034) **PEF:** PM_{10} (Mother smoked during pregnancy): -0.0102 (0.0039)
Reference: Mortimer et al. (2002, 030281) **Period of Study:** June-August 1993 **Location:** Eight urban areas of the US: Bronx and East Harlem, NY Baltimore, MD Washington, DC Detroit, MI Cleveland, OH Chicago, IL and St. Louis, MO.	**Outcome:** peak expiratory flow rate (PEFR) and symptoms **Age Groups:** 4-9 yrs **Study Design:** Cohort study **N:** 846 children with a history of asthma **Statistical Analyses:** Mixed linear models and GEE **Covariates:** day of study, previous 12-h mean temperature, urban area, diary number, rain in the past 24 h **Season:** Summer **Dose-response Investigated?** No **Statistical Package:** SAS **Lags Considered:** 0, 1, 2, 3, 4, 5, 6, 1-5 avg, 1-4 avg, 0-4 avg, 0-3 avg	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (SD):** 53 **Monitoring Stations:** NR **Copollutant (correlation):** 8-h avg ozone: r = 0.51	**PM Increment:** 20 $\mu g/m^3$ **Effect Estimate [Lower CI, Upper CI]:** (RR estimates are odds ratios for incidence of morning asthma symptoms using the avg of lag 1-2) 3 urban areas (DE, CL, CH) Single pollutant: OR = 1.26 (1.00-1.59) Ozone + PM_{10}: OR = 1.25 (0.97-1.61) Ozone + SO_2 + NO_2 + PM_{10}: OR = 1.14 (0.80-1.48)
Reference: Moshammer and Neuberger (2003, 041956) **Period of Study:** 2000-2001 **Location:** Linz, Austria	**Outcome:** Lung Function: FVC, FEV_1, MEF_{25}, MEF_{50}, MEF_{75}, PEF, LQ Signal, PAS Signal **Age Groups:** Ages 7 to 10 **Study Design:** Case-crossover **N:** 161 children 1898–2120 "half-h means" **Statistical Analyses:** Correlations Regression Analysis **Covariates:** Morning, Evening, Night **Season:** Spring, summer, Winter, Fall **Dose-response Investigated?** No	**Pollutant:** PM_{10} **Averaging Time:** 8 h Daily Means **Mean (SD):** 23.13 (20.08) **Range (Min, Max):** (NR, 190.79) **Monitoring Stations:** 1 **Copollutant (correlation):** LQ = 0.751 PAS = 0.406	**Notes:** "Acute effects of 'active particle surface' as measured by diffusion charging were found on pulmonary function (FVC, FEV_1, MEF50) of elementary school children and on asthma-like symptoms of children who had been classified as sensitive."

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Moshammer et al. (2006, 090771) **Period of Study:** 2000-2001 **Location:** Linz, Austria	**Outcome:** Respiratory symptoms and decreased lung function **Age Groups:** Children ages 7-10 **Study Design:** Time-series **N:** 163 children **Statistical Analyses:** GEE model **Covariates:** Sex, age, height, weight **Dose-response Investigated?** NR **Statistical Package:** NR **Lags Considered:** 1	**Pollutant:** PM_{10} **Averaging Time:** 8-h **Mean (SD):** Maximum 24 h: 76.39 **Annual avg:** 19.06 **Percentiles:** 8-h mean 25th: 14.39 8-h mean 50th(Median): 24.85 8-h mean 75th: 38.82 **Monitoring Stations:** 1 station **Copollutant (correlation):** PM_1 r = 0.91 $PM_{2.5}$ r = 0.93 NO_2 r = 0.62	**PM Increment:** 10 $\mu g/m^3$ **% change in Lung Function per 10 $\mu g/m^3$** FEV: 0.11 FVC: 0.06 $FEV_{0.5}$: -0.19 $MEF_{75\%}$: -0.30 $MEF_{50\%}$: -0.36 $MEF_{25\%}$: 0.41 PEF: 0.22 **% change in Lung Function per IQR** FEV: -0.27 FVC: -0.07 $FEV_{0.5}$: -0.47 $MEF_{75\%}$: -0.74 $MEF_{50\%}$: -0.86 $MEF_{25\%}$: 0.98 PEF: -0.54
Reference: Neuberger et al. (2004, 093249) **Period of Study:** Sept 1999-March 2000 **Location:** Vienna, Austria	**Outcome:** Ratio measure: Time to peak tidal expiratory flow divided by total expiration time (i.e., tidal lung function, a surrogate for bronchial obstruction) **Age Groups:** 3.0-5.9 years (preschool children) **Study Design:** Longitudinal prospective cohort **N:** 56 children **Statistical Analyses:** mixed models linear regression, with autoregressive correlation structure **Covariates:** Age, sex, respiratory rate, phase angle, temperature, kindergarten, parental education, observer (also in sensitivity analyses: height, weight, cold/sneeze on same day, heating with fossil fuels, hair cotinine, number of tidal slopes used to measure tidal lung function) **Dose-response Investigated?** No **Statistical Package:** SAS 8.0 **Lags Considered:** 0	**Pollutant:** PM_{10} **Averaging Time:** 24-h **Copollutant (correlation):** $PM_{2.5}$ (r = 0.94) in Vienna	**PM Increment:** Interquartile range (NR) Change in mean associated with an IQR increase in PM (p-value) lag -1.067 (0.241) lag 0

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Neuberger et al. (2004, 093249) **Period of Study:** Oct. 2000-May 2001 **Location:** Linz, Austria	**Outcome:** Forced oscillatory resistance (at zero Hz), FVC, FEV_1, MEF_{25}, MEF_{50}, MEF_{75}, PEF **Age Groups:** 7-10 years **Study Design:** Longitudinal prospective cohort **N:** 164 children **Statistical Analyses:** mixed models linear regression with autoregressive correlation structure **Covariates:** sex, time and individual **Season:** Oct-May **Dose-response Investigated?** No **Statistical Package:** NR **Lags Considered:** 0-7	**Pollutant:** PM_{10} **Averaging Time:** 24-h **Monitoring Stations:** 1	**PM Increment:** 1 $\mu g/m^3$ **Notes:** No significant associations between PM_{10} and the metrics of lung function were reported. The authors state they only reported significant associations, so results are assumed to be null.
Reference: Odajima et al. (2008, 192005) **Period of Study:** April 2003-March 2004 **Location:** Fukuoka, Japan	**Outcome:** Peak Expiratory Flow (PEF) **Study Design:** Panel/Field **Statistical Analysis:** GEE **Statistical Package:** SAS **Covariates:** Age, Sex, Growth Index, Temperature, NO_2, O_3 **Age Groups:** Asthmatic children, 4-11 years old	**Pollutant:** PM_{10} **Averaging Time:** 3h **Mean (SD) Unit:** Warmer Months, 5-8am SPM: 40.7 $\mu g/m^3$ NO_2: 15.2 ppb O_3: 17.7 ppb Warmer Months, 7-10pm SPM: 41.5 $\mu g/m^3$ NO_2: 20.0 ppb O_3: 28.1 ppb Colder Months, 5-8am SPM: 32.6 $\mu g/m^3$ NO_2: 20.5 ppb O_3: 17.5 ppb Colder Months, 7-10pm SPM: 34.7 $\mu g/m^3$ NO_2: 28.0 ppb O_3: 19.4 ppb **Range (Min, Max):** Warmer Months, 5-8am SPM: (11.0, 126.0) NO_2: (1.3, 44.7) O_3: (0.3, 52.3) Warmer Months, 7-10pm SPM: (8.3, 191.3) NO_2: (3.0, 51.3) O_3: (1.3, 71.3) Colder Months, 5-8am SPM: (9.0, 160.0) NO_2: (1.3, 44.0) O_3: (0.6, 48.7)	**Increment:** 10 $\mu g/m^3$ **Relative Risk (Min CI, Max CI)** **Lag** April-September, Morning Sample, Multi-pollutant SPM, 5am-8am: -0.6 (-1.228, 0.028) SPM, 2am-5am: -0.78 (-1.399, -0.161) SPM, 11pm-2am: -0.612 (-1.180, -0.045) SPM, 8pm-11am: -0.732 (-1.318, -0.145) O_3, 5am-8am: -0.575 (-1.569, 0.419) O_3, 2am-5am: -0.052 (-0.997, 0.893) O_3, 11pm-2am: -0.305 (-1.269, 0.658) O_3, 8pm-11am: -0.416 (-1.283, 0.451) NO_2, 5am-8am: -0.3 (-2.246, 1.645) NO_2, 2am-5am: 0.265 (-1.354, 1.885) NO_2, 11pm-2am: -0.187 (-1.447, 1.073) NO_2, 8pm-11am: 0.432 (-0.689, 1.553) Single-Pollutant Model SPM, 5am-8am: -0.67 (-1.236, -0.104) SPM, 2am-5am: -0.761 (-1.328, -0.194) SPM, 11pm-2am: -0.661 (-1.159, -0.163) SPM, 8pm-11am: -0.714 (-1.212, -0.215) Evening Sample, Multi-pollutant Model SPM, 7pm-10pm: -0.449 (-1.071, 0.174) SPM, 4pm-7pm: -0.434 (-1.122, 0.254) SPM 1pm-4pm: -0.415 (-1.015, 0.184) SPM 10am-1pm: -0.522 (-1.199, 0.155) O_3, 7pm-10pm: -0.22 (-1.171, 0.731) O_3, 4pm-7pm: -0.118 (-0.809, 0.574) O_3

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
		Colder Months, 7-10pm	1pm-4pm: -1.086 (-0.888, 0.516)
		SPM: (10.3, 131.0)	O_3
		NO_2: (3.6, 49.0)	10am-1pm: -0.315 (-1.123, 0.493)
		O_3: (1.0, 60.0)	NO_2, 7pm-10pm: 0.296 (-0.806, 1.397)
		Copollutant (correlation):	NO_2, 4pm-7pm: 0.220 (-0.818, 1.258)
		Warmer Months (24h Mean):	NO_2
		O_3	1pm-4pm: 0.438 (-0.568, 1.444)
		r = 0.32	NO_2
		NO_2	10am-1pm: 0.536 (-0.546, 1.617)
		r = 0.30	Single-pollutant Model
		Colder Months (24h Mean):	SPM, 7pm-10pm: -0.449 (-0.956, 0.058)
		O_3	SPM, 4pm-7pm: -0.449 (-1.029, 0.131)
		r = -0.02	SPM
		NO_2	1pm-4pm: -0.414 (-0.943, 0.115)
		r = 0.45	SPM
			10am-1pm: -0.486 (-1.051, 0.079)
			October-March, Morning Sample, Multi-pollutant
			SPM, 5am-8am: 0.290 (-0.279, 0.859)
			SPM, 2am-5am: 0.431 (-0.173, 1.036)
			SPM, 11pm-2am: 0.304 (-0.311, 0.919)
			SPM, 8pm-11am: 0.010 (-0.523, 0.543)
			O_3, 5am-8am: -0.415 (-1.568, 0.738)
			O_3, 2am-5am: -0.046 (-1.245, 1.153)
			O_3, 11pm-2am: 0.004 (-1.265, 1.273)
			O_3, 8pm-11am: -0.470 (-2.017, 1.077)
			NO_2, 5am-8am: -0.319 (-2.269, 1.631)
			NO_2, 2am-5am: 0.262 (-1.777, 2.300)
			NO_2, 11pm-2am: 0.609 (-1.132, 2.350)
			NO_2, 8pm-11am: 0.155 (-1.545, 1.856)
			Single-Pollutant Model
			SPM, 5am-8am: 0.308 (-0.189, 0.805)
			SPM, 2am-5am: 0.485 (-0.026, 0.996)
			SPM, 11pm-2am: 0.486 (-0.049, 1.022)
			SPM, 8pm-11am: 0.100 (-0.414, 0.613)
			Evening Sample, Multi-pollutant Model
			SPM, 7pm-10pm: 0.059 (-0.397, 0.515)
			SPM, 4pm-7pm: 0.360 (-0.093, 0.812)
			SPM
			1pm-4pm: 0.357 (-0.157, 0.871)
			SPM
			10am-1pm: 0.169 (-0.394, 0.731)
			O_3, 7pm-10pm: -0.656 (-2.394, 1.083)
			O_3, 4pm-7pm: 0.046 (-1.140, 1.232)
			O_3

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			1pm-4pm: 0.164 (-1.038, 1.365)
			O_3
			10am-1pm: 0.665 (-0.613, 1.942)
			NO_2, 7pm-10pm: -0.415 (-2.444, 1.613)
			NO_2, 4pm-7pm: -0.144 (-1.490, 1.202)
			NO_2
			1pm-4pm: -0.181 (-1.821, 1.459)
			NO_2
			10am-1pm: 0.194 (-1.503, 1.890)
			Single-pollutant Model
			SPM, 7pm-10pm: 0.071 (-0.388, 0.529)
			SPM, 4pm-7pm: 0.318 (-0.123, 0.758)
			SPM
			1pm-4pm: 0.317 (-0.171, 0.804)
			SPM
			10am-1pm: 0.112 (-0.412, 0.636)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Peacock et al. (2003, 042026)	**Outcome:** Reduced peak expiratory flow rate (PEFR)	**Pollutant:** PM_{10}	**Increment:** 10 $\mu g/m^3$
Period of Study: 11/1/1996–2/14/1997	**Age Groups:** 7-13 years of age	**Averaging Time:** daily	**Odds Ratio (Lower CI, Upper CI)**
Location: Southern England	**Study Design:** Time-series	**Mean (SD):** Rural (nationally validated) 21.2 (11.3)	**Lag**
	N: 179	Rural (locally validated) 18.7 (11.3)	**Change in PEFR**
	Statistical Analyses: GEE, multiple regression	Urban 1 18.4 (9.8)	Community
	Covariates: Day of the week, 24-h mean outside temperature.	Urban 2 22.7 (10.6)	-0.04 (-0.11, 0.03)
	Season: winter	**Percentiles:** 10th	0
	Dose-response Investigated? No	Rural (nationally validated) 11.0	0.03 (-0.04, 0.05)
	Statistical Package: STATA	Rural (locally validated) 9.0	1
	Lags Considered: Same day, lag 1, lag 2, five day moving avg	Urban 1 10.5	-0.01 (-0.07, 0.05)
		Urban 2 12.5	2
		90th	-0.10 (-0.25, 0.05)
		Rural (nationally validated) 33.0	0-4 avg
		Rural (locally validated) 32.5	Local
		Urban 1 32.0	-0.01 (-0.06, 0.03)
		Urban 2 36.0	0
		Range (Min, Max): Rural (nationally validated) 7.0, 82.0	0.04 (0.01, 0.08)
		Rural (locally validated) 6.6, 87.9	1
		Urban 1 4.7, 62.8	0.01 (-0.04, 0.05)
		Urban 2 6.7, 63.7	2
		Monitoring Stations: 3**Copollutants:**	0.04 (-0.05, 0.13)
		NO_2	0-4 avg
		O_3	**20% decrease in PEFR**
		SO_2	All children
		SO_4^{2-}	1.012 (0.992, 1.031)
			0
			1.016 (0.995, 1.036)
			1
			1.013 (1.000, 1.025)
			2
			1.037 (0.992, 1.084)
			0-4 avg
			Wheezy Children Only
			1.016 (0.986, 1.047)
			0
			1.030 (1.001, 1.060)
			1
			1.018 (0.995, 1.041)
			2
			1.114 (1.057, 1.174)
			0-4 avg

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Peled, et al (2005, 156015) **Period of Study:** 5-6 weeks between March-June 1999 and September-December 1999. **Location:** Ashdod, Ashkelon and Sderot, Israel	**Outcome:** Reduced peak expiratory flow (PEF) **Age Groups:** 7-10 years **Study Design:** Nested cohort study **N:** 285 **Statistical Analyses:** Time series analysis, generalized linear model, GEE, one-way ANOVA **Covariates:** seasonal changes, meteorological conditions and personal physiological, clinical and socioeconomic measurements **Season:** spring, autumn **Dose-response Investigated?** No **Statistical Package:** STATA	**Pollutant:** PM_{10} **Averaging Time:** daily Mean: Ashkelon: 67.1 Sderot: 52.9 Ashdod: 31.0 **PM Component:** Local industrial emissions, desert dust, vehicle emissions and emissions from two electric power plants **Monitoring Stations:** 6 **Copollutant:** $PM_{2.5}$	**PM Increment:** 1 $\mu g/m^3$ **β coefficient (SE) [95% CI]** Sderot: PM_{10} MAX: -0.34 (0.41) [-1.16, 0.46] PM_{10} MAX x sin(¤2 day): 0.84 (0.22) [0.405, 1.28] PM_{10} MAX x cos (¤1 day): -1.61 (0.41) [-2.43, 0.79] PM_{10} MAX x sin (¤1 day): 0.44 (0.120) [-0.68-0.21] In Sderot, an interaction between PM_{10} and the sequential day were significantly associated with PEF.
Reference: Pitard, et al (2004, 087433) **Period of Study:** 732 days (July 1998-June 2000) **Location:** City of Rouen, France	**Outcome:** Respiratory drug sales **Age Groups:** 0-14, 15-64, 65-74, over 75 years **Study Design:** Ecological time-series **N:** 106,592 **Statistical Analyses:** Generalized additive model **Covariates:** Days of the weeks, trend, seasonal variations, influenza epidemics, meteorological variables, holidays **Dose-response Investigated?** No **Statistical Package:** S-plus **Lags Considered:** 0 to 10 days	**Pollutant:** PM_{10} **Averaging Time:** daily **Mean (SD):** 16.7 (13.3) **Percentiles:** 25th: 8.00 50th(Median): 13.0 75th: 20 **Range (Min, Max):** 2.00, 126 **Monitoring Stations:** 2 **Copollutant (correlation):** SO_2 (0.39) NO_2 (0.61)	**PM Increment:** 10 $\mu g/m^3$ Percent increase in sales of anti-asthmatics and bronchodilators (Lower CI, Upper CI) lag: 6.2 (2.4, 10.1) lag 10 days Percent increase in sales of cough and cold preparation for children under 15 years of age (Lower CI, Upper CI) lag: 9.2 (5.9, 12.6) 10 days

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Preutthipan et al. (2004, 055598) **Period of Study:** 31 days (school days) from January 14 to February 26, 1999 **Location:** Mae Pra Fatima School, central Bangkok, Thailand	**Outcome:** Decreases in peak expiratory flow rates (PEFR), respiratory symptoms including wheeze, shortness of breath, runny/stuffed nose, sneezing, cough, phlegm, and sore throat **Age Groups:** Third to ninth grade **Study Design:** Time- Series **N:** 133 children (93 asthmatics, 40 nonasthmatics) **Statistical Analyses:** For continuous data, an unpaired t-test or Mann-Whitney U test was used. For categorical data, the chi-square test or Fisher's exact test was used. One-way analysis of covariance (ANCOVA) was used to compare avg daily reported respiratory symptoms, diurnal PEFR variability, and the prevalence of PEFR decrements between groups of days. **Covariates:** Age, sex, weight, height, parents smoking, person smoking in home, daily number of household cigarettes, air-conditioned bedroom, fuel used for cooking (charcoal, gas), distance from home to main road **Dose-response Investigated?** No **Lags Considered:** Up to 5 days	**Pollutant:** PM_{10} **Averaging Time:** daily **Mean (SD):** 111.0 (39) **Range (Min, Max):** 46, 201 **Monitoring Stations:** 1 **Copollutant:** SO_2 CO O_3	**PM Increment:** Authors classified exposure according to High and Low PM_{10} days: High = >120 $\mu g/m^3$ Low = <120 $\mu g/m^3$ Daily reported respiratory symptoms and diurnal PEFR variability as classified by concurrent days with high vs. low PM_{10} Mean % reporting (SEM) Asthmatics: High PM_{10} Wheeze/shortness of breath = 21.3 (1.4) Runny/stuffed nose or sneezing = 42.3 (1.8) Cough = 59.9 (1.9) Phlegm = 60.5 (2.3) Sore throat = 23.7 (1.5) Any respiratory symptoms = 72.2 (3.2) Diurnal PEFR variability = 3.0 (0.4) Asthmatics: Low PM_{10} Wheeze/shortness of breath = 19.3 (1.3) Runny/stuffed nose or sneezing = 35.8 (1.6) Cough = 59.1 (1.6) Phlegm = 58.6 (2.0) Sore throat = 21.0 (1.4) Any respiratory symptoms = 63.8 (2.8) Diurnal PEFR variability = 2.8 (0.3) Nonasthmatics: High PM_{10} Wheeze/shortness of breath = 11.7 (1.4) Runny/stuffed nose or sneezing = 40.9 (2.5) Cough = 50.4 (2.6) Phlegm = 50.2 (2.5) Sore throat = 27.1 (1.7) Any respiratory symptoms = 67.8 (3.7) Diurnal PEFR variability = 2.4 (0.4) Nonasthmatics: Low PM_{10} Wheeze/shortness of breath = 9.3 (1.2) Runny/stuffed nose or sneezing = 33.1 (2.2) Cough = 54.0 (2.2) Phlegm = 49.9 (2.2) Sore throat = 23.9 (1.5) Any respiratory symptoms = 56.4 (3.2) Diurnal PEFR variability = 2.1 (0.4) **Notes:** None of the daily reported respiratory symptoms had significant direct correlations with daily PM_{10} levels, according to the authors.

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Rabinovitch et al. (2004, 096753) **Periods of Study:** 11/15/1999–3/15/2000 11/13/2000–3/23/2001 11/15/2001–3/22/2002 **Location:** Denver, Colorado	**Outcome:** Respiratory symptoms, Asthma symptoms (cough and wheeze), Upper respiratory symptoms **Study Design:** Time-series panel **Statistical Analyses:** Logistic linear regression **Age Groups:** 6-12	**Pollutants:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** 28.1 (13.2) **Range (Min, Max):** (6.0, 102.0) **Copollutant:** CO NO_2 SO_2 O_3	**Increment:** 1 $\mu g/m^3$ β (SE) AM: -0.010 (0.008) PM: -0.011 (0.010) **Odds Ratio (Lower CI, Upper CI)** **Lag** 1.016 (0.911, 1.133) 0-3 avg. OR for respiratory symptoms and PM_{10} exposure for children age 6-12 Asthma exacerbation: 1.00 (0.75, 1.25) 0-3 avg Medication: 0.85 (0.75, 0.95) 0-3 avg Previous night's symptoms: 1.10 (1.00, 1.20) 0-3 avg Current day's symptoms: 1.00 (0.90, 1.10) 0-3 avg **% Increase (Lower CI, Upper CI)** **Lag** % Increase in FEV_1 or PEF and PM_{10} exposure for children age 6-12 AM FEV_1: -0.01 (-0.02, 0.01) 0-3 avg PM FEV_1: -0.02 (-0.03, 0.02) 0-3 avg AM PEF: -0.025 (-0.035, 0.02) 0-3 avg PM PEF: 0.00 (-0.03, 0.03) 0-3 avg.
Reference: Renzetti et al, 2009 **Period of Study:** 6/1/06-7/31/06 **Location:** Pescara and Ovindoli, Italy	**Outcome:** Airway inflammation and function **Study Design:** Panel **Covariates:** NR **Statistical Analysis:** Student T-test, Pearson's correlation coefficients **Statistical Package:** StatView **Age Groups:** Children, mean age 9.9 years	**Pollutant:** PM_{10} **Averaging Time:** daily **Mean (SD) Unit:** Urban: 56.9 ± 13.1 $\mu g/m^3$ Rural: 13.8 ± 5.6 $\mu g/m^3$ **Copollutant (correlation):** NR	All results are presented in figure format.
Reference: Rojas-Martinez et al. (2007, 091064) **Period of Study:** 1996-1999 **Location:** Mexico City, Mexico	**Outcome:** Lung function: FEV_1, FVC, $FEF_{25-75\%}$ **Age Groups:** Children 8 years old at time of cohort recruitment **Study Design:** school-based "dynamic" cohort study **N:** 3170 children 14,545 observations	**Pollutant:** PM_{10} **Averaging Time:** 24-h, 6-mo **Mean (SD):** 24-h averaging Tlalnepantla: 66.7 (35.6) Xalostoc: 96.7 (49.4) Merced: 79.3 (40.8) Pedregal: 53.4 (31.9)	**PM Increment:** IQR PM_{10}, 6-LC: 36.4 **GIRLS** **One-pollutant model** FVC: -39 [-47: -31] FEV: -29 [-36: -21] $FEF_{25-75\%}$: -17 [-36: 1]

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
	Statistical Analyses: Three-level generalized linear mixed models with unstructured variance-covariance matrix **Covariates:** age, body mass index, height, height by age, weekday spent outdoors, environmental tobacco smoke, previous-day mean air pollutant concentration, time since first test **Dose-response Investigated?** No **Statistical Package:** SAS **Lags Considered:** 0-1 days	Cerro de la Estrella: 69.6 (35.3) 6-mo averaging **Mean:** 75.6 **Percentiles:** 6-mo averaging 25th: 55.8 50th(Median): 67.5 75th: 92.2 **Monitoring Stations:** 5 sites for PM_{10}, 10 for other pollutants **Copollutant:** O_3 NO_2	FEV_1/FVC: 0.12 [0.07: 0.17] **Two-pollutant model** PM_{10}, 6-LC & O_3 FVC: -30 [-39: -22] FEV: -24 [-31: -16] $FEF_{25-75\%}$: -9 [-26: 9] FEV_1/FVC: 0.10 [0.06: 0.15] PM_{10}, 6-LC & NO_2 FVC: -21 [-30: -13] FEV: -17 [-25: -8] $FEF_{25-75\%}$: -23 [-43: -4] FEV_1/FVC: 0.07 [0.02: 0.13] **Multipollutant model** PM_{10}, 6-LC, O_3, & NO_2 FVC: -14 [-23: -5] FEV: -11 [-20: -3] $FEF_{25-75\%}$: -7 [-27: 12] FEV_1/FVC: 0.08 [0.03: 0.13] **BOYS** **One-pollutant model** FVC: -33 [-41: -25] FEV: -27 [-34: -19] $FEF_{25-75\%}$: -18 [-34: -2] FEV_1/FVC: 0.04 [-0.01: 0.09] **Two-pollutant model** PM_{10}, 6-LC & O_3 FVC: -28 [-36: -19] FEV: -22 [-30: -15] $FEF_{25-75\%}$: -10 [-27: 7] FEV_1/FVC: 0.04 [-0.01: 0.09] PM_{10}, 6-LC & NO_2 FVC: -16 [-26: -7] FEV: -19 [-27: -10] $FEF_{25-75\%}$: -26 [-44: -9] FEV_1/FVC: 0.005 [-0.06: 0.05] **Multipollutant model** PM_{10}, 6-LC, O_3, & NO_2 FVC: -12 [-22: -3] FEV: -15 [-23: -6] $FEF_{25-75\%}$: -12 [-30: 6] FEV_1/FVC: -0.002 [-0.06: 0.05] Long-term exposure to O_3, PM_{10}, and NO_2 is associated with decrements in FVC and FEV_1 growth in Mexico City schoolchildren. In a multipollutant model, PM_{10} (-12%), O_3 (-9%), and NO_2 (-41%) each contribute independently and statistically significantly to diminished

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			FVC growth. For FEV_1, however, the multipollutant model indicates that only PM_{10} (-15%) and NO_2 (-25%) each contribute independently and statistically significantly to diminished FEV_1 growth.
Reference: Sahsuvaroglu et al, 2009 **Period of Study:** 1994-1995 **Location:** Hamilton, Canada	**Outcome:** Asthma symptoms **Study Design:** Panel **Covariates:** Neighborhood income, dwelling value, state of housing, deprivation index, smoking **Statistical Analysis:** Logistic regressions **Statistical Package:** SPSS **N:** 6388 **Age Groups:** Children in grades 1 and 8	**Pollutant:** PM_{10} **Averaging Time:** 3 year averages **Average:** All Subjects: 20.90 $\mu g/m^3$ Boys: 20.88 $\mu g/m^3$ Girls: 20.92 $\mu g/m^3$ **Range:** All Subjects: 26.98 Boys: 26.98 Girls: 20.10 **Copollutant (correlation):** NO_xTheissen: 0.083 SO_2Theissen: -0.021 O_3Theissen: -0.251 NO_2Kriged: 0.126 NO_2LUR: 0.072	**Increment:** NR **Odds Ratio (95%CI) for copollutant model PM_{10}Spline and NO_2LUR** All Girls: 1.063 (0.969-1.666) Older Girls: 1.058 (0.918-1.219) **Odds Ratio (95%CI) for copollutant model PM_{10}Spline and NO_2LUR, SO_2Theissen and O_3Theissen** All Girls: 1.045 (0.943-1.158) Older Girls: 1.044 (0.891-1.225) **Regression coefficients (95%CI) between non-allergic asthma and PM_{10}Spline exposure** All Children: 1.043 (0.996-1.092) Younger Children: 1.011 (0.929-1.100) Older Children: 1.073 (1.013-1.136) All Girls: 1.069 (0.999-1.144) All Boys: 1.024 (0.962-1.091) Younger Girls: 1.065 (0.943-1.203) Younger Boys: 0.962 (0.853-1.085) Older Girls: 1.072 (0.984-1.169) Older Boys: 1.075 (0.995-1.160)
Reference: Sanchez-Carrillo et al. (2003, 098428) **Period of Study:** 1996-1997 **Location:** metropolitan Mexico City, Mexico	**Outcome:** Upper respiratory symptom indicator (wet cough, sore throat, hoarseness, nose dryness, and head cold) Lower respiratory symptom indicator (dry cough, lack of air, and chest sounds) and Ocular symptom indicator (eye irritation, eye itch, eye burning, teary eyes, red eyes, and eye infection) **Age Groups:** All ages **Study Design:** Cohort **N:** 151,418 interviews **Statistical Analyses:** Logistic regression models **Covariates:** sex, age, education, cigarette smoking, season, emergency episode mass media report, temperature, and relative humidity **Dose-response Investigated?** Yes **Statistical Package:** NR **Lags Considered:** 1	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (SD):** Northeast: 132 (52) Northwest: 87 (46) Central: 85 (37) Southeast: 79 (35) Southwest: 55 (28) **Range (Min, Max):** Northeast: (34-269) Northwest: (10-275) Central: (9-319) Southeast: (14-225) Southwest: (12-264) **Monitoring Stations**: Up to 32 **Copollutant (correlation):** O_3: r = 0.067 O_3 8: 00-18: 00 h: r = 0.075 SO_2: r = 0.265 NO_2: r = 0.265	**Effect Estimate [Lower CI, Upper CI]:** **PM_{10} quartiles**10.04-52.62 (ref) 52.63-73.58 Upper respiratory indicator: 1.02 (0.99-1.06) Lower respiratory indicator: 1.04 (0.99-1.09) Ocular indicator: 0.99 (0.95-1.03) 73.59-101.91 Upper respiratory indicator: 1.07 (1.03-1.10) Lower respiratory indicator: 1.09 (1.04-1.14) Ocular indicator: 0.89 (0.86-0.92)101.92-318.80 Upper respiratory indicator: 0.93 (0.90-0.97) Lower respiratory indicator: 1.03 (0.98-1.08) Ocular indicator: 0.84 (0.81-0.87) **Northeast** - 2nd quartile Upper respiratory indicator: 0.354 (0.112-1.222) Lower respiratory indicator: 0.215 (0.040-1.160) Ocular indicator: 1.080 (0.915-1.274) 3rd quartile

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			Upper respiratory indicator: 0.118 (0.039-0.356)
			Lower respiratory indicator: 0.126 (0.023-0.690)
			Ocular indicator: 1.228 (0.720-2.095)
			4th quartile
			Upper respiratory indicator: 0.095 (0.034-0.267)
			Lower respiratory indicator: 0.119 (0.026-0.549)
			Ocular indicator: 0.878 (0.619-1.246)
			Northwest - 2nd quartile
			Upper respiratory indicator: 0.990 (0.898-1.090)
			Lower respiratory indicator: 1.246 (1.087-1.429)
			Ocular indicator: 1.218 (0.808-1.834)
			3rd quartile
			Upper respiratory indicator: 1.133 (0.974-1.317)
			Lower respiratory indicator: 1.202 (1.044-1.385)
			Ocular indicator: 0.345 (0.125-0.951)
			4th quartile
			Upper respiratory indicator: 1.019 (0.904-1.149)
			Lower respiratory indicator: 1.344 (1.137-1.589)
			Ocular indicator: 1.949 (1.416-2.683)
			Central - 2nd quartile
			Upper respiratory indicator: 1.088 (1.002-1.183)
			Lower respiratory indicator: 1.046 (0.930-1.176)
			Ocular indicator: 1.220 (1.115-1.335)
			3rd quartile
			Upper respiratory indicator: 1.054 (0.977-1.137)
			Lower respiratory indicator: 1.055 (0.948-1.175)
			Ocular indicator: 1.049 (0.965-1.142)
			4th quartile
			Upper respiratory indicator: 0.899 (0.826-0.979)
			Lower respiratory indicator: 0.952 (0.845-1.073)
			Ocular indicator: 0.875 (0.796-0.963)
			Southeast - 2nd quartile
			Upper respiratory indicator: 0.778 (0.575-1.052)
			Lower respiratory indicator: 1.047 (0.916-1.196)
			Ocular indicator: 0.460 (0.299-0.708)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			3rd quartile Upper respiratory indicator: 1.297 (1.127-1.491) Lower respiratory indicator: 1.391 (1.131-1.711) Ocular indicator: 0.474 (0.314-0.715) 4th quartile Upper respiratory indicator: 0.893 (0.812-0.983) Lower respiratory indicator: 0.937 (0.818-1.073) Ocular indicator: 0.314 (0.182-0.542) **Southwest** - 2nd quartile Upper respiratory indicator: 0.987 (0.913-1.066) Lower respiratory indicator: 2.181 (1.177-4.040) Ocular indicator: 1.026 (0.928-1.135) 3rd quartile Upper respiratory indicator: 0.673 (0.673-1.886) Lower respiratory indicator: 0.899 (0.790-1.024) Ocular indicator: 1.017 (0.862-1.200) 4th quartile Upper respiratory indicator: 0.524 (0.524-1.787) Lower respiratory indicator: 4.346 (0.917-20.606) Ocular indicator: 0.187 (0.090-0.387)
Reference: Schildcrout et al. (2006, 089812) **Period of Study:** November 1993 to September 1995 **Location:** Albuquerque, New Mexico Baltimore, Maryland Boston, Massachusetts Denver, Colorado San Diego, California Seattle, Washington St. Louis, Missouri Toronto, Ontario, Canada	**Outcome:** Asthma Symptoms, Rescue Inhaler Uses **Age Groups:** 5 to 12 year olds **Study Design:** Meta-analysis of CAMP **N:** 990 children **Statistical Analyses:** "Working independence covariance structure" Logistic Regression Poisson Regression "GEE Procedure" **Covariates:** Season, age, race-ethnicity, annual family income, day of the week **Dose-response Investigated?** **Statistical Package:** SAS 8.2 R **Lags Considered:** 0 day lag, 1 day lag, 2 day lag, 3-day moving sum	**Pollutant:** PM_{10} **Averaging Time:** 24-h averages Seattle: Daily Albuquerque: Daily Baltimore: 50% of study days measured Boston: 23% of study days measured Denver: 37% of study days measured San Diego: 24% of study days measured St. Louis: 19% of study days measured Toronto: 47% of study days measured **Percentiles:** 10th: 6.8-14.0 25th: 12.0-22.4 50th(Median): 17.7-32.4 75th: 26.2-42.7 90th: 32.5-53.9 **Monitoring Stations:** 1-12 **Copollutant (correlation):** NO_2 r = 0.26-0.64 SO_2 r = 0.31-0.65 O_3 r = 0.03-0.73	**PM Increment:** 25 $\mu g/m^3$ **One-pollutant Model** Asthma Symptoms: 1.02 [0.94, 1.11] 0 1.01 [0.97, 1.06] 1 1.02 [0.98, 1.07] 2 1.01 [0.98, 1.05] 3-day moving sum Rescue Inhaler Uses: [0.97, 1.05] 0 [0.97, 1.05] 1 1.00 [0.97, 1.03] 2 1.01 [0.98, 1.03] 3-day moving sum **Two-pollutant Model**

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
		CO r = 0.24-0.88	Asthma Symptoms: CO-PM_{10} 1.08 [1.01, 1.15] 0 1.06 [0.99, 1.14] 1 1.08 [1.02, 1.14] 2 1.05 [1.01, 1.08] 3-day moving sum NO_2-PM_{10} 1.06 [0.99, 1.13] 0 1.04 [0.97, 1.11] 1 1.08 [1.02, 1.15] 2 1.04 [1.00, 1.07] 3-day moving sum SO_2-PM_{10} 1.05 [0.98, 1.13]; 0 1.04 [0.96, 1.14] 1 1.05 [0.98, 1.12] 2 1.04 [0.99, 1.08] 3-day moving sum Rescue Inhaler Uses: CO-PM_{10} 1.06 [0.99, 1.13] 0 1.05 [0.99, 1.11] 1; 1.05 [1.01, 1.09] 2 1.03 [1.00, 1.07] 3-day moving sum NO_2-PM_{10} 1.03 [0.97, 1.08] 0 1.03 [0.98, 1.08] 1 1.04 [1.00, 1.09] 2 1.02 [1.00, 1.05]

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			3-day moving sum
			SO_2-PM_{10}
			1.01 [0.95, 1.07]
			0
			1.02 [0.97, 1.07]
			1
			1.03 [0.98, 1.09]
			2
			1.02 [0.98, 1.05]
			3-day moving sum

[1]All units expressed in $\mu g/m^3$ unless otherwise specified.

Table E-10. Short-term exposure - respiratory morbidity outcomes - $PM_{10-2.5}$.

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Aekplakorn et al. (2003, 089908)	**Outcome:** Upper respiratory symptoms, lower respiratory symptoms, cough	**Pollutant:** $PM_{10-2.5}$	**PM Increment:** 10 $\mu g/m^3$
Period of Study: 107 days, from October 1, 1997 to January 15, 1998	**Age Groups**: 6-14 years old	**Averaging Time:** daily	Odds Ratios [Lower CI, Upper CI]
Location: Mae Mo district, Lampang Province, north Thailand	**Study Design:** Logistic regression	**Mean (SD):** NR	lag:
	N: 98 asthmatic school children	**Range (Min, Max):** NR	Asthmatics:
	Statistical Analyses: Generalized Estimating Equations, stratified analysis, PROC GENMOD	**Monitoring Stations**: 3	URS: 1.04 (0.93, 1.17)
	Covariates: Temperature and relative humidity	**Copollutant**: PM_{10}, SO_2	lag 0
	Season: Winter		LRS: 1.09 (0.95, 1.26)
	Dose-response Investigated? No		lag 0
	Statistical Package: SAS v 8.1		Cough: 1.08 (0.96, 1.21)
			lag 0
			Non-Asthmatics:
			URS: 1.05 (0.99, 1.19)
			lag 0
			LRS: 0.90 (0.72, 1.11)
			lag 0
			Cough: 0.95 (0.81, 1.11)
			lag 0
Reference: Bourotte et al. (2007, 150040)	**Outcome:** Peak expiratory flow (PEF)	**Pollutant:** $PM_{2.5}$	**PM Increment:** NR
Period of Study: 13 May 2002, 19 July 2002	**Age Groups**: Avg age 39.8 +/- 12.3	**Averaging Time:** 24 h	Effect [Lower CI, Upper CI]
Location: Sao Paulo, Brazil	**Study Design:** Cross-sectional	**Mean (SD):** 21.7 (12.9) $\mu g/m^3$	lag:
	N: 33 patients	**Range (Min, Max):** (4.13, 62.0)	Morning PEF
	Statistical Analyses: Linear mixed-effects model	**Components:**	Na^+ concurrent day = -0.454 (-1.605, 0.697)
	Covariates: Gender, Age, BMI, Air Pollutants, Ambient temperature, Relative Humidity	Na^+	Na^+ 2-day lag = -0.907 (-2.288, 0.474)
		K^+	Na^+ 3-day lag = -1.361 (-2.972, 0.251)
	Season: Winter	Mg^{2+}	K^+ concurrent day = 1.685 (-0.492, 3.862)
	Dose-response Investigated? No	Ca^{2+}	K^+ 2-day lag = 1.838 (-1.272, 4.984)
	Statistical Package: S-plus	F_{inf}	K^+ 3-day lag = 2.604 (-0.812, 6.025)
	Lags Considered: 2 day lag, 3 day lag	Cl^-	Mg^{2+} concurrent day = 2.265* (-0.427, 4.956)
		NO_3^-	Mg^{2+} 2-day lag = 1.271 (-1.869, 4.410)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
		SO_4^{2-}	Mg^{2+} 3-day lag = 0.939 (-2.425, 4.303)
		Monitoring Stations: 1	Ca^{2+} concurrent day = 5.491* (2.558, 8.424)
			Ca^{2+} 2-day lag = 6.358* (2.251, 10.465)
			Ca^{2+} 3-day lag = 6.069 (1.962, 10.176)
			F_{inf} concurrent day = 1.572 (-0.792, 3.935)
			F_{inf} 2-day lag = 1.630 (-1.679, 4.939)
			F_{inf} 3-day lag = 2.736* (-1.754, 7.226)
			Cl^- concurrent day = -0.951 (-2.238, 0.336)
			Cl^- 2-day lag = -1.871 (-3.242 to -0.4997)
			Cl^- 3-day lag = -2.286* (-3.934 to -0.638)|
			NO_3^- concurrent day = 4.195* (-0.063, 8.452)
			NO_3^- 2-day lag = 6.292* (2.034, 10.55)
			NO_3^- 3-day lag = 7.341* (3.083, 11.60)
			SO_4^{2-} concurrent day = 3.528 (-0.053, 7.110)
			SO_4^{2-} 2-day lag = 4.411* (0.829, 7.991)|
			SO_4^{2-} 3-day lag = 6.175* (2.593, 9.756)
			Evening PEF
			Na^+ concurrent day = -0.680 (-1.831, 0.471)
			Na^+ 2-day lag = -1.90 (-3.316 to -0.494)
			Na^+ 3-day lag = -2.336* (-3.878 to -0.794)
			K^+ concurrent day = 0.613 (-1.564, 2.790)
			K^+ 2-day lag = 0.613 (-2.497, 3.723)
			K^+ 3-day lag = 0.000 (-3.421, 3.421)|
			Mg^{2+} concurrent day = -0.718 (-3.522, 2.085)
			Mg^{2+} 2-day lag = -1.933 (-5.073, 1.206)
			Mg^{2+} 3-day lag = -3.591 (-7.056 to -0.126)
			Ca^{2+} concurrent day = 2.312* (-1.208, 5.832)
			Ca^{2+} 2-day lag = 2.023 (-2.084, 6.130)
			Ca^{2+} 3-day lag = 0.578 (-3.530, 4.685)
			F_{inf} concurrent day = -1.281 (-3.644, 1.083)
			F_{inf} 2-day lag = -2.503 (-5.930, 0.924)
			F_{inf} 3-day lag = -4.540 (-9.149, 0.068)
			Cl^- concurrent day = -0.317 (-1.604, 0.970)
			Cl^- 2-day lag = -1.268 (-2.556, 0.019)
			Cl^- 3-day lag = -1.902 (-3.589 to -0.216)
			NO_3^- concurrent day = 3.146 (-1.112, 7.404)
			NO_3^- 2-day lag = 3.146 (-1.112, 7.404)
			NO_3^- 3-day lag = 1.049 (-3.209, 5.306)
			SO_4^{2-} concurrent day = 1.764 (-1.817, 5.346)
			SO_4^{2-} 2-day lag = 2.646 (-0.935, 6.228)
			SO_4^{2-} 3-day lag = 1.764 (-1.817, 5.346)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Ebelt et al. (2005, 056907) Check Ref: 2006, 1st page: 396 **Period of Study:** Summer of 1998 **Location:** Vancouver, Canada	**Outcome:** spirometry **Age Groups**: range from 54-86 yrs mean age = 74 years **Study Design:** extended analysis of a repeated-measures panel study **N:** 16 persons with COPD **Statistical Analyses:** Earlier analysis expanded by developing mixed-effect regression models and by evaluating additional exposure indicators **Dose-response Investigated?** No **Statistical Package**: SAS V8	**Pollutant:** $PM_{10-2.5}$ **Averaging Time:** 24 h **Mean (SD):** Ambient PM_{10}-2.5: 5.6 (3.0) Exposure to ambient PM_{10}-2.5: 2.4 (1.7) **Range (Min, Max):** Ambient $PM_{10-2.5}$: (-1.2-11.9) Exposure to ambient $PM_{10-2.5}$: (-0.4-7.2) **Monitoring Stations**: 5 **Copollutant (correlation):** Ambient PM_{10}: r = 0.69 Ambient $PM_{2.5}$: r = 0.15 Nonsulfate Ambient $PM_{2.5}$: r = 0.14 Exposure to Ambient $PM_{10-2.5}$: r = 0.73	**PM Increment:** Ambient PM_{10}-2.5: 4.5 (IQR) Exposure to ambient PM_{10}-2.5: 2.4 (IQR) Notes: Effect estimates are presented in Figure 2 and Electronic Appendix Table 1 (only available with electronic version of article) and not provided quantitatively elsewhere.
Reference: Lagorio et al.(2006, 089800) **Period of Study:** 5/24/1999 to 6/24/1999 and 11/181999 to 12/22/1999 **Location:** Rome, Italy	**Outcome:** Lung function of subjects (FVC and FEV_1) with COPD, Asthma **Age Groups**: COPD 50 to 80 yrs Asthma 18 to 64 yrs **Study Design:** Time series **N:** COPD N = 11 Asthma N = 11 **Statistical Analyses:** Non-parametric Spearman correlation GEE **Covariates:** COPD: daily mean temperature, season variable (spring or winter), relative humidity, day of week Asthma: season variable, temperature, humidity, and ¤-2-agonist use **Season:** Spring and winter **Dose-response Investigated?** Yes **Statistical Package**: STATA **Lags Considered:** 1–3 days	**PM Size:** $PM_{10-2.5}$ **Averaging Time:** 24 h **Mean (SD):** Overall: 15.6 (7.2) Spring: 18.7 (7.4) Winter: 12.3 (5.4) **Range (Min, Max):** (3.4, 39.6) **PM Component:** Cd: 0.46 ± 0.40 ng/m^3 Cr: 1.9 ± 1.7 ng/m^3 Fe: 283 ± 167 ng/m^3 Ni: 4.8 ± 6.5 ng/m^3 Pb: 30.6 ± 19.0 ng/m^3 Pt: 5.0 ± 8.6 pg/m^3 V: 1.8 ± 1.4 ng/m^3 Zn: 45.8 ± 33.1 ng/m^3 **Monitoring Stations**: Two fixed sites: (Villa Ada and Istituto superior di Sanita) **Copollutant (correlation):** NO_2 r = 0.51 O_3 r = 0.31 CO r = -0.09 SO_2 r = -0.16 PM_{10} r = 0.61 $PM_{2.5}$ r = 0.34	**PM Increment:** 1 $\mu g/m^3$ They observed no statistically significant effect of $PM_{10-2.5}$ on FVC and FEV_1 on any of the panels (COPD, Asthma). β Coefficient (SE) COPD FVC(%) 24 h -1.32 (1.06)^I 48-h -1.46 (1.31) 72-h -1.38 (1.53) FEV_1(%) 24 h -0.59 (0.95) 48-h -1.01 (1.19) 72-h -0.90 (1.42) Asthma FVC(%) 24 h -0.17 (0.75) 48-h -0.36 (0.91) 72-h -0.24 (1.07) FEV_1(%) 24 h -0.67 (0.89) 48-h -1.19 (1.07) 72-h -0.51 (1.26)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: (Laurent et al., 2008, 156672) **Period of Study:** 12/21/1003-12/31/2004 **Location:** Strasbourg, France	**Outcome:** sales of short acting β-agonists **Study Design:** Case-crossover **Covariates:** NR **Statistical Analysis:** Conditional logistic regression **Age Groups:** 0-39 years	**Pollutant:** PM_{10} **Averaging Time:** NR **Mean (SD) Unit:** 20.8 (10.2) $\mu g/m^3$ **Range (Min, Max):** NR **Copollutant (correlation):** NO_2, O_3, correlations NR	**Increment:** 10$\mu g/m^3$ **Percent Increase in Short Acting β-agonists sold** Per increment increase in ambient PM_{10} at lags 4-7, a 7.5% increase (95% CI: 4-11.2%) was seen in SABA sales. All other results were given in figures 1 and 2
Reference: Tang et al. (2007, 091269) **Period of Study:** Dec 2003 to Feb 2005 **Location:** Sin-Chung City, Taipei County, Taiwan	**Outcome:** Peak expiratory flow rate (PEFR) of asthmatic children **Age Groups**: 6–12 years **Study Design:** Panel study **N:** 30 children **Statistical Analyses:** Linear mixed-effect models were used to estimate the effect of PM exposure on PEFR **Covariates:** Gender, age, BMI, history of respiratory or atopic disease in family, SHS, acute asthmatic exacerbation in past 12 months, ambient temperature and relative humidity, presence of indoor pollutants, and presence of outdoor pollutants, **Dose-response Investigated?** yes **Statistical Package**: S-Plus 2000 **Lags Considered:** 0-2	**Pollutant:** $PM_{10-2.5}$ **Averaging Time:** 1 h **Mean (SD):** Personal: 17.8 (19.6) Ambient: 17.0 (10.6) **Range (Min, Max):** Personal: 0.3–195.7 Ambient: 0.1–80.2 **Monitoring Stations**: 1	**PM Increment:** 15.9 $\mu g/m^3$ RR Estimate [Lower CI, Upper CI] lag: Change in morning PEFR: -20.55 (-45.83, 4.73) lag 0 -39.05 (-104.16 , 26.06) lag 1 -39.56 (-79.56, 0.44) lag 2 -37.15 (-105.01, 30.7) 2-day mean -35.47 (-27.32, 56.38) 3-day mean Change in evening PEFR: -1.68 (-19.13, 15.78) lag 0 1.59 (-14.32, 17.5) lag 1 0.86 (-30.84, 32.57) lag 2 5.97 (-15.57, 27.5) 2-day mean 29.75 (-1.69, 61.18) 3-day mean
Reference: Trenga et al., (2006, 155209) **Period of Study:** 1999-2002 **Location:** Seattle, WA	**Outcome**: Lung function: FEV_1, PEF, MMEF (maximal midexpiratory flow assessed only for children) **Age Groups**: Adults (56-89-years-old) healthy & with COPD asthmatic children 6-13-years-old **Study Design:** adult and pediatric panel study over three years with 1 monitoring period ("session") per year **N:** 57 adults (33 healthy, 24 with COPD) = 692 subject-days = 207 study-days 17 asthmatic children = 319 subject-days = 98 study-days **Statistical Analyses:** mixed effects, longitudinal regression models, with the effects of pollutant decomposed into each subject's a) overall mean b) difference between their session-specific mean and overall mean c) difference between their daily values and session-specific mean **Covariates:** gender, age, ventral site temperature and relative humidity, CO, NO_2 **Season:** NR **Dose-response Investigated?** No **Statistical Package**: SAS **Lags Considered:** 0-1 days	**Pollutant:** $PM_{10-2.5}$ (coarse) **Averaging Time:** 24-h **Percentiles:** Subject-specific exposure PM_{10}-$PM_{2.5}$ Outdoor 25th: 3.3 50th (Median): 4.7 75th: 6.9 Adults Outdoor 25th: 3.3 50th (Median): 5.0 75th: 7.1 **Range (Min, Max):** Subject-specific exposure Children Outdoor (0.0, 25.3) Adults Outdoor (0.0, 25.7) **Monitoring Stations**: 2 also subject-specific local outdoors (i.e., at each home), indoor, and personal	**PM Increment:** 10 $\mu g/m^3$ **Adult** Outdoor Home PM_{10}-$PM_{2.5}$ FEV_1 Overall: Lag 0 -27.9 [-87.5: 31.8] Lag 1 47.1 [-5.1: 99.4] No-COPD: Lag 0 -49.2 [-22.3: 23.9] Lag 1 74.3 [6.8: 141.8] COPD: Lag 0 7.3 [-84.7: 99.4] Lag 1 11.5 [-65.4: 88.3] PEF Overall: Lag 0 5.3 [-5.1: 15.7] Lag 1 -2.5 [-11.6: 6.5] No-COPD: Lag 0 5.1 [-7.7: 17.8] Lag 1 -5.8 [-17.5: 5.9] COPD: Lag 0 5.7 [-10.3: 21.6] Lag 1 1.7 [-11.5: 14.9] **Pediatric** FEV_1 Outdoor Home PM_{10}-$PM_{2.5}$ Overall Lag 0 -7.43 [-69.41: 54.55] Lag 1 -25.61 [-88.16: 36.94] No Anti-inflam. Medication

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
		Copollutant (correlation):	Lag 0 -63.87 [-199.58: 71.84]
		CO	Lag 1 -96.48 [-232.48: 39.52]
		NO_2	Anti-inflam. Medication
		$PM_{2.5}$	Lag 0 6.57 [-96.90: 110.04]
			Lag 1 -8.63 [-217.39: 200.14]
			PEF
			Outdoor Home PM_{10}-$PM_{2.5}$
			Overall
			Lag 0 4.53 [-6.60: 15.67]
			Lag 1 -3.35 [-14.31: 7.62]
			No Anti-inflam. Medication
			Lag 0 2.05 [-22.36: 26.45]
			Lag 1 -6.56 [-30.90: 17.78]
			Anti-inflam. Medication
			Lag 0 5.15 [-7.90: 18.19]
			Lag 1 -2.58 [-15.35: 10.19]
			MMEF
			Outdoor Home PM_{10}-$PM_{2.5}$
			Overall
			Lag 0 -0.01 [-7.29: 7.28]
			Lag 1 -2.07 [-9.25: 5.12]
			No Anti-inflam. Medication
			Lag 0-7.14 [-23.16: 8.87]
			Lag 1 -14.39 [-30.11: 1.32]
			Anti-inflam. Medication
			Lag 0 1.76 [-6.78: 10.30]
			Lag 1 0.89 [-7.56: 9.33]

[1]All units expressed in $\mu g/m^3$ unless otherwise specified.

Table E-11. Short-term exposure - respiratory morbidity outcomes - $PM_{2.5}$ (including components/sources).

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Adamkiewicz et al. (2004, 087925)	**Outcome:** FENO	**Pollutant:** $PM_{2.5}$	**PM Increment:** 17.9 $\mu g/m^3$
Period of Study: August–December 2000	**Age Groups:** ranged 53.5-90.6 years	**Averaging Time:** 1 h	Effect Estimate [Lower CI, Upper CI]:
Location: Steubenville, Ohio	**Study Design:** prospective cohort	**Mean (SD):** 19.5	1-h Single pollutant models: 0.36 (0.58-2.14)
	N: total of 294 breaths from 29 subjects	**Percentiles:** 25th: 7.6	**PM Increment:** 17.7
	Statistical Analyses: Fixed effect models, ANOVA, GLM procedure	75th: 25.5	Effect Estimate [Lower CI, Upper CI]:
	Covariates: Subject, week of study, day of the week, h of the day, ambient barometric pressure, temperature, and relative humidity	**Range (Min, Max):** NR, 105.8	24 h moving avg: 1.45 (0.33-2.57)
		Monitoring Stations: 1	Multipollutant models for $PM_{2.5}$, ambient NO and room NO and estimated change in FENO (ppb) for an IQR in pollutant measure
		Averaging Time: 24 h	
	Dose-response Investigated? No	**Mean (SD):** 19.7	Model 1 1.95 (0.47-3.43)
	Statistical Package: SAS	**Percentiles:** 25th: 9.7	Model 2 1.38 (0.26-2.51)
	Lags Considered: Hourly lags, 0-48 h	75th: 27.4	
		Range (Min, Max): NR, 57.8	

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
		Monitoring Stations: 1	Model 4 1.97 (0.48-3.46)
		Copollutant (correlation): Ambient NO	Notes: Association of FENO with $PM_{2.5}$ at different lags presented in Figure 1 are not presented quantitatively elsewhere.
		Indoor NO	
		NO_2;	
		O_3	
		SO_2	
Reference: Adar et al. (2007, 098635)	**Outcome:** FENO	**Pollutant:** $PM_{2.5}$	**PM Increment:** 9.8 $\mu g/m^3$
Period of Study: March-June 2002	**Age Groups**: 60+	**Averaging Time:** 24 h	Effect Estimate [Lower CI, Upper CI]:
Location: St. Louis, MO	**Study Design:** Panel Study	**Mean (SD):** Pretrip: 14.8	Pre-trip % change: 21.9 (6.7, 39.4)
	N: 44 non-smoking seniors	Post-trip: 16.5	Post-trip % change: -4.7 (-17.1, 9.6)
	Statistical Analyses: mixed models containing random subject effects	**Percentiles:**	
		25th (pretrip): 11.2	
	Covariates: Day of week, trip type, FENO collection device, current illness, use of vitamins, antihistamines, statins, steroids, and asthma medications, temperature, pollen, mold, NO concentration in testing room	75th (pretrip): 20.1	
		25th (post-trip): 11.7	
		75th (post-trip): 21.6	
	Statistical Package: SAS	**Monitoring Stations**: 1	
	Lags Considered: 0	**Copollutant (correlation):** BC	
		CO	
		NO_2	
		SO_2	
		O_3	
Reference: Aekplakorn et al (2003, 089908)	**Outcome:** Upper respiratory symptoms, lower respiratory symptoms, cough	**Pollutant:** $PM_{2.5}$	**PM Increment:** 10 $\mu g/m^3$
Period of Study: 107 days, from October 1, 1997 to January 15, 1998	**Age Groups**: 6-14 years old	**Averaging Time:** daily	**Odds Ratios [Lower CI, Upper CI]**
Location: Mae Mo district, Lampang Province, north Thailand	**Study Design:** Logistic regression	**Mean (SD):**	**lag:** Asthmatics: URS: 1.04 (0.99, 1.09)
	N: 98 asthmatic school children	Sob Pad station: 24.77	lag 0
	Statistical Analyses: Generalized Estimating Equations, stratified analysis, PROC GENMOD	Sob Mo station: 24.89	LRS: 1.05 (0.98, 1.2)
		Hua Fai station: 26.27	lag 0
	Covariates: Temperature and relative humidity	**Range (Min, Max):**	Cough: 1.05 (0.99, 1.10)
		Sob Pad: 4.52, 24.77	lag 0
	Season: Winter	Sob Mo: 3.13, 24.89	Non-Asthmatics: URS: 1.03 (0.96, 1.09)
	Dose-response Investigated? No	Hua Fai: 3.67, 26.27	lag 0
	Statistical Package: SAS v 8.1	**Monitoring Stations**: 3	LRS: 1.02 (0.93, 1.10)
		Copollutant:	lag 0
		PM_{10}	Cough: 1.00 (0.93, 1.07)
		SO_2	lag 0
			PM_{10} + SO_2
			Asthmatics: URS: 1.04 (0.99, 1.10)
			lag 0
			LRS: 1.05 (0.98, 1.10)
			lag 0
			Cough: 1.05 (0.99, 1.11)
			lag 0
			Non-Asthmatics: URS: 1.03 (0.97, 1.09)
			lag 0
			LRS: 1.02 (0.93, 1.11)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			lag 0 Cough: 1.00 (0.93, 1.07) lag 0
Reference: Allen et al (2008, 156208) **Period of Study:** 1999-2002 (additional PM composition data collected Dec 2000 and May 2001) **Location:** Seattle, USA	**Outcome:** daily changes in exhaled nitric oxide (FENO) and 4 lung function measures, midexpiratory flow (MEF), peak expiratory flow (PEF), forced expiratory volume in one second (FEV_1), and forced vital capacity (FVC) **Age Groups:** 6-13 yrs **Study Design:** Panel study **N:** 19 children with asthma **Statistical Analyses:** linear mixed effects model with random intercept to test for within participant associations **Covariates:** Temperature, relative humidity, BMI, age, and, in the case of FENO, ambient NO measured at a centrally located monitoring site models also included a term for within-participant, within-session effects, and a term for participant between-session effects **Effect modification**: Decided a priori to include interaction term for $PM_{2.5}$ exposure and inhaled corticosteroids	**Pollutant:** $PM_{2.5}$ **Mean (SD):** 11.23 (6.48) **Range (Min, Max):** 2.76-40.38 25th: 6.38 75th: 14.73 **Copollutant (correlation):** Ambient LAC* r = 0.83 Ambient LG**r = 0.84 Personal $PM_{2.5}$: r = 0.34 Personal LAC: r = 0.54 Ambient-generated $PM_{2.5}$: r = 0.87 Nonambient-generated $PM_{2.5}$: r = -0.06 * LAC Light-absorbing carbon ** LG: Leroglucosan (a marker of wood smoke)	Health effect estimates presented in graphic form (Fig 1). Summary from text is as follows: Personal LAC, personal $PM_{2.5}$, and ambient-generated $PM_{2.5}$ were associated with ($p < 0.05$) and ambient $PM_{2.5}$ was marginally associated ($p = 0.09$) with increased FENO. Neither of the ambient combustion markers (LAC, LG) nor nonambient-generated $PM_{2.5}$ was associated with FENO changes. All of the ambient concentrations were associated with decrements in PEF and MEF while ambient-generated $PM_{2.5}$ was marginally associated ($p < 0.10$). Only ambient LG was associated with a decrease in FEV_1 and there were no associations between exposure metrics and FVC.
Reference: Barraza-Villarreal et al.(2008, 156254) **Period of Study:** 6/2003–6/2005 **Location:** Mexico City	**Outcome:** Respiratory Symptoms, Coughing, Wheezing, Airway inflammation, Asthma **Study Design:** Prospective cohort **Statistical Analyses:** Bivarate analysis **Age Groups**: 6-14	**Pollutant:** $PM_{2.5}$ **Averaging Time:** Maximum 8-h avg **Mean (SD) unit:** 28.9 (2.8) **Range (Min, Max):** (4.2, 102.8) **Copollutants (correlation):** O_3 NO_2	**Increment:** 17.5 $\mu g/m^3$ **% Increase (Lower CI, Upper CI)** **lag:** **Asthmatic children** Inflammatory Marker: FENO: 1.08 (1.01, 1.16) 0 IL-8: 1.08 (0.98, 1.19) 0 ph_EBC: -0.03 (-0.09, 0.03) 0 Lung Function: FEV_1: -16.0 (-31.0 to -0.13) 0-4 avg FVC: -23.0 (-42.0 to -5.21) 0-4 avg FEV_{25-75}: -11.0 (-42.0, 20.3) 0-4 avg **Nonasthmatic children** Inflammatory Marker: FENO: 0.89 (0.78, 1.01) 0 IL-8: 1.16 (1.00, 1.36) 0; ph_EBC: -0.05 (-0.14, 0.04) 0 Lung Function: FEV_1: -21.0 (-42.3, 0.38)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			0-4 avg FVC: -29.0 (-52.8 to -4.35) 0-4 avg FEV_{25-75}: -20.0 (-69.0, 29.0) 0-4 avg **All children age 6-14** Respiratory Symptom: Cough: 1.11 (1.06, 1.17) Wheezing: 1.06 (0.99, 1.13)
Reference: Bennett et al. (2007, 156268) **Period of Study:** 1992-2005 **Location:** Melbourne, Australia	**Outcome:** Adverse respiratory symptoms (wheeze, shortness of breath on waking, cough in the morning, phlegm in the morning, cough with phlegm in the morning, asthma attack) **Age Groups:** All ages with a mean of 37.2 yrs **Study Design:** cohort study **N:** 1446 persons **Statistical Analyses:** Logistic regression models **Covariates:** Age, gender, current smoking status, medication use (ß2-agonist and inhaled steroid), atopy **Dose-response Investigated?** No **Statistical Package:** STATA statistical software, version 9 (Statcorp, 2005)	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean (SD):** 6.8 **Range (Min, Max):** (1.8-73.3) **Monitoring Stations:** 1	**PM Increment:** 1 $\mu g/m^3$ **Effect Estimate [Lower CI, Upper CI]:** Within-person (longitudinal effects) Wheeze: OR = 1.08 (0.79-1.48) SOB on waking: OR = 1.34 (0.84-2.16) Cough in the morning: OR = 0.74 (0.47-1.15) Phlegm in the morning: OR = 1.55 (0.95-2.53) Cough w/ phlegm morning: OR = 1.28 (0.70-2.33) Asthma attack: OR = 0.91 (0.55-1.49) Between-person (cross-sectional) effects Wheeze: OR = 1.32 (0.82-2.10) SOB on waking: OR = 1.29 (0.46-3.60) Cough in the morning: OR = 0.21 (0.07-0.62) Phlegm in the morning: OR = 0.49 (0.16-1.44) Cough w/ phlegm morning: OR = 0.28 (0.08-0.97) Asthma attack: OR = 0.52 (0.17-1.59)
Reference: Bourotte et al. (2007, 150040) **Period of Study:** 13 May 2002-19 July 2002 **Location:** Sao Paulo, Brazil	**Outcome:** Peak expiratory flow (PEF) **Age Groups:** Avg age 39.8 +/- 12.3 **Study Design:** Cross-sectional **N:** 33 patients **Statistical Analyses:** Linear mixed-effects model **Covariates:** Gender, Age, BMI, Air Pollutants, Ambient temperature, Relative Humidity **Season:** Winter **Dose-response Investigated?** No **Statistical Package:** S-plus **Lags Considered:** 2 day lag, 3 day lag	**Pollutant:** $PM_{2.5}$ (Fine) **Averaging Time:** 24 h **Mean (SD):** 11.9 (5.12) **Range (Min, Max):** (2.82, 26.6) **Components:** K^+ Mg^{2+} Ca^{2+} F_{inf} Cl- NO_3- SO42– **Monitoring Stations:** 1	**PM Increment:** NR **Effect [Lower CI, Upper CI]** **lag:** **Morning PEF** Na^+ concurrent day = -0.409 (-2.485, 1.667) Na^+ 2-day lag = -0.818 (-4.139, 2.503) Na^+ 3-day lag = -0.205 (-4.356, 3.974) K^+ concurrent day = -0.211 (-2.778, 2.357) K^+ 2-day lag = -0.843 (-4.695, 3.008) K^+ 3-day lag = 0.843 (-4.292, 5.978) Mg^{2+} concurrent day = -1.750 (-5.302, 1.802) Mg^{2+} 2-day lag = -5.016 (-10.79, 0.762) Mg^{2+} 3-day lag = -3.850 (-10.15, 2.449) Ca^{2+} concurrent day = 3.192* (-0.599, 6.943) Ca^{2+} 2-day lag = 5.880 (1.105, 10.65)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			Ca^{2+} 3-day lag = 7.560* (2.103, 13.02)
			F_{inf} concurrent day = 2.218* (-0.033, 4.470)
			F_{inf} 2-day lag = 3.697* (1.446, 5.949)
			F_{inf} 3-day lag =4.067* (1.065, 7.069)
			Cl^- concurrent day = -1.010 (-3.469, 1.450)
			Cl^- 2-day lag = -1.615 (-5.714, 2.483)
			Cl^- 3-day lag = -1.615 (-6.534, 3.303)
			NO_3^- concurrent day = 3.144 (0.409, 5.878)
			NO_3^- 2-day lag = 3.593 (0.858, 6.328)
			NO_3^- 3-day lag = 4.491 (1.756, 7.226)
			SO_4^{2-}concurrent day = 2.210 (-0.032, 4.272)
			SO_4^{2-} 2-day lag = 3.180 (1.028, 5.332)
			SO_4^{2-} 3-day lag = 3.180 (1.028, 5.332)
			Evening PEF
			Na^+ concurrent day = -1.636 (-3.712, 0.440)
			Na^+ 2-day lag = -0.205 (-3.256, 3.117)
			Na^+ 3-day lag = -1.023 (-5.174, 3.129)
			K^+ concurrent day = -1.897 (-4.465, 0.670)
			K^+ 2-day lag = -1.686 (-5.966, 2.592)
			K^+ 3-day lag = -1.054 (-6.189, 4.081)
			Mg^{2+} concurrent day = -2.753 (-6.400, 0.894)
			Mg^{2+} 2-day lag = -2.567 (-8.534, 3.401)
			Mg^{2+} 3-day lag = -4.876 (-11.36, 1.612)
			Ca^{2+} concurrent day = 2.184 (-1.567, 5.935)
			Ca^{2+} 2-day lag = 5.040 (0.265, 9.815)
			Ca^{2+} 3-day lag = 5.040 (-0.417, 10.50)
			F_{inf} concurrent day = 1.479 (-0.773, 3.730)
			F_{inf} 2-day lag = 1.819 (-0.403, 4.100)
			F_{inf} 3-day lag = 2.958 (-0.044, 5.960)
			Cl^- concurrent day = -0.404 (-2.863, 2.055)
			Cl^- 2-day lag = 0.000 (-4.099, 4.099)
			Cl^- 3-day lag =0.202 (-4.716, 5.120)
			NO^{3-} concurrent day = 1.796 (-0.939, 4.531)
			NO^{3-} 2-day lag = 2.695 (-0.040, 5.430)
			NO^{3-} 3-day lag = 3.144 (0.409, 5.878)
			SO_4^{2-} concurrent day = 2.120 (-0.032, 4.272)
			SO_4^{2-} 2-day lag = 2.120 (-0.032, 4.272)
			SO_4^{2-} 3-day lag =2.120 (-0.032, 4.272)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: de Hartog et al. (2003, 001061) **Period of Study:** winter of 1998-1999 (in Amsterdam, from November 2, 1998 to June 18, 1999 in Erfurt, from October 12, 1998 to April 4, 1999 and in Helsinki, from November 2, 1998 to April 30, 1999.) **Location:** Amsterdam, the Netherlands Erfurt, Germany and Helsinki, Finland	**Outcome:** Respiratory symptoms **Age Groups:** ≥ 50 yrs **Study Design: C**ohort **N:** 131 subjects with history of coronary heart disease **Statistical Analyses:** Logistic regression **Covariates:** Ambient temperature, relative humidity, atmospheric pressure, incidence of influenza-like illness **Season**: Winter **Dose-response Investigated?** No **Statistical Package**: S-PLUS 2000 **Lags Considered:** 0, 1, 2, 3, and 5-day avg	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean (SD):** Amsterdam, the Netherlands: 20.0 Erfurt, Germany: 23.4 Helsinki, Finland: 12.8 **Range (Min, Max):** Amsterdam, the Netherlands: (3.8-82.2) Erfurt, Germany: (4.5-118.1) Helsinki, Finland: (3.1-39.8) Unit (i.e. $\mu g/m^3$): $\mu g/m^3$ **Monitoring Stations**: 1 **Copollutant:** PM_{10} $NC_{0.01-0.1}$ CO NO_2 SO_2	**PM Increment:** 10 $\mu g/m^3$ **Effect Estimate [Lower CI, Upper CI]:** Association of air pollution and incidence of symptoms in three panels of elderly subjects **Lag 0** Chest pain w/ physical exertion: 1.04 (0.96-1.13) Shortness of breath: 1.04 (0.96-1.12) Awakened, breathing problems: NA Avoidance of activities: 1.04 (0.96-1.14) Phlegm: 1.03 (0.93-1.13) **Lag 1** Chest pain w/ physical exertion: 1.01 (0.93-1.09) Shortness of breath: 1.06 (0.99-1.14) Awakened, breathing problems: 1.09 (1.00-1.20) Avoidance of activities: 1.03 (0.95-1.12) Phlegm: 1.10 (1.01-1.19) **Lag 2** Chest pain w/ physical exertion: 0.98 (0.90-1.05) Shortness of breath: 1.05 (0.98-1.12) Awakened, breathing problems: 1.04 (0.95-1.14) Avoidance of activities: 1.05 (0.97-1.14) Phlegm: 1.08 (1.00-1.18) **Lag 3** Chest pain w/ physical exertion: 1.00 (0.93-1.08) Shortness of breath: 1.08 (1.01-1.15) Awakened, breathing problems: 0.99 (0.91-1.08) Avoidance of activities: 1.06 (0.98-1.14) Phlegm: 1.10 (1.01-1.19) **5-day** Chest pain w/ physical exertion: 1.02 (0.91-1.13) Shortness of breath: 1.12 (1.02-1.24) Awakened, breathing problems: 1.03 (0.90-1.18) Avoidance of activities: OR = 1.09 (0.97-1.22) Phlegm: OR = 1.16 (1.03-1.32)
Reference: Delfino et al. (2004, 056897) **Period of Study:** September–October 1999 April–June 2000 **Location:** Alpine, California	**Outcome**: FEV_1 **Age Groups:** 9-19 years old **Study Design:** Panel study **N:** 24 children **Statistical Analyses:** GLM	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-h avg 1-h max personal PM last 24 h **Mean (SD):** 151.0 (12.03) 90th: 292.4 **Range (Min, Max):** (9.1, 996.8)	Results presented graphically; -Percent predicted FEV_1 was inversely associated with personal exposure to fine particles. - Inverse associations of FEV_1 with stationary-site indoor, outdoor and central-site gravimetric $PM_{2.5}$ and PM_{10}, and with hourly TEOM PM_{10}

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
	Akaike's information criterion and Bayesian information criterion **Covariates:** Day of week, Personal temperature and relative humidity, time of FEV_1 maneuver (morning, afternoon, or evening), Season (fall 1999 or spring 2000), As-needed medication use, Presence or absence of upper or lower respiratory infections **Season:** Spring, Fall **Dose-response Investigated?** No **Statistical Package:** SAS **Lags Considered:** 0-4	Mean personal PM last 24 h **Mean (SD):** 37.9 (19.9) 90th: 65.1 **Range (Min, Max):** 3.9, 113.8 Home stationary-site PM 24-h Mean indoor $PM_{2.5}$ **Mean (SD):** 12.1 (5.4) 90th: 20.2 **Range (Min, Max):** 2.8, 35.3 24-h Mean outdoor $PM_{2.5}$ **Mean (SD):** 11.0 (5.4) 90th: 18.4 **Range (Min, Max):** 1.8, 31.0 Central outdoor stationary-site PM 24-h Mean $PM_{2.5}$ **Mean (SD):** 10.3 (5.6) 90th: 18.4 **Range (Min, Max):** 1.7, 29.1 **Copollutant (correlation):** 24-h Central HI $PM_{2.5}$ 8-h max O_3 = 0.24 8-h Max NO_2 = 0.73 8-h Max Personal PM = 0.38 24-h Mean Personal PM = 0.43 8-h Max TEOM PM_{10} = 0.71 24-h Mean TEOM PM_{10} = 0.78 24-h Central HI PM_{10} = 0.90 24-h Outdoor HI $PM_{2.5}$ = 0.89 24-h Outdoor HI PM_{10} = 0.72 24-h Indoor HI PM_{10} = 0.40 24-h Indoor HI $PM_{2.5}$ = 0.73	
Reference: Delfino et al. (2006, 090745) **Period of Study:** Region 1: August to Mid December 2003. Region 2: July through November 2004 **Location:** Region 1: Riverside, CA. Region 2: Whittier, CA	**Outcome:** Fractional Concentration of Nitric Oxide in exhaled air (FENO) **Age Groups:** 9 through 18 **Study Design:** Longitudinal Panel Study **N:** 45 children Riverside children 32 Whittier children **Statistical Analyses:** Linear mixed-effects models Two-stage hierarchical model Empirical Variograms Fourth-order polynomial distributed lag mixed-effects model **Covariates:** Personal temperature, Personal Rel. Humid., 10-day exposure run, Respiratory infections, Region of study, Sex, Cumulative daily use of as-	**Pollutant:** $PM_{2.5}$ **Personal Exposure** **Averaging Time:** 24 h **Riverside** **Mean (SD):** 32.78 (21.84) 50th(Median): 28.14 **Range (Min, Max):** 7.27, 98.43 **Whittier** **Mean (SD):** 36.2 (25.46) 50th(Median): 29.07 **Range (Min, Max):** 7.55, 197.05 **Personal Exposure** **Averaging Time:** 1 h **Riverside** **Mean (SD):** 97.94 (70.29) 50th(Median):	**PM Increment:** IQR increase (Riverside: 28.41 $\mu g/m^3$, Whittier 21.87 $\mu g/m^3$) **Coefficient [Lower CI, Upper CI]** **lag:** Mixed-model estimates of the association between personal and central-site air pollutant exposure and FENO Lag 0 Personal 0.42 (-0.15, 0.99) Central 0.03 (-0.68, 0.74) Lag 1 Personal 0.51 (-0.10, 1.12) Central 0.44 (-0.28, 1.16) 2-day MA Personal 1.01 (0.14, 1.88)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
	needed B-agonist inhalers **Dose-response Investigated?** No **Lags Considered:** 0, 1, 2, MA day	83.7 **Range (Min, Max):** 14.9, 431.8 **Whittier** **Mean (SD):** 93.63 (75.19) 50th(Median): 71.95 **Range (Min, Max):** 5.8, 572.9 **Personal Exposure** **Averaging Time:** 8 h **Riverside** **Mean (SD):** 47.21 (30.9) 50th(Median): 38.5 **Range (Min, Max):** 8.9, 132.1 **Whittier** **Mean (SD):** 51.75 (36.88) 50th(Median): 40.15 **Range (Min, Max):** 8.7, 254.1 **Central Site** **Averaging Time:** 24 h **Riverside** **Mean (SD):** 36.63 (23.46) 50th(Median): 29.26 **Range (Min, Max):** (9.52, 87.22) **Whittier** **Mean (SD):** 18 (12.14) 50th(Median): 16.3 **Range (Min, Max):** 2.7, 77.09 **Monitoring Stations**: 48 personal nephelometers 2 central sites **Copollutant (correlation):** **Personal** 24-h personal $PM_{2.5}$ 1.00 24-h personal EC 0.18 24-h personal OC 0.15 24-h personal NO_2 0.33 24-h central $PM_{2.5}$ 0.64 24-h central EC 0.12 24-h central OC 0.21 24-h central NO_2 0.22 **Central** 24-h personal $PM_{2.5}$ 0.64 24-h personal EC 0.00 24-h personal OC -0.11 24-h personal NO_2 0.12 24-h central $PM_{2.5}$ 1.00 24-h central EC 0.55 24-h central OC 0.66 24-h central NO_2 0.25	Central 0.52 (-0.43, 1.47) Stratified by Medication Use Lag = 2-day moving avg Not Taking Anti-Inflamm. Medication Personal 1.11 (-1.39, 3.60) Central 0.44 (-1.65, 2.53) Taking Anti-Inflamm. Medication Personal 1.01 (0.19, 1.84) Central 0.55 (-0.47, 1.57) Inhaled Corticosteroids Personal 1.58 (0.72, 2.43) Central 1.16 (0.11, 2.20) Antileukotrienes +- inhaled corticosteroids Personal -0.89 (-2.73, 0.95) Central -0.75 (-2.83, 1.32) Notes: Figure of Estimated lag effect of hourly personal $PM_{2.5}$ on FENO. Figure of the Estimated lag effect of hourly personal $PM_{2.5}$ on FENO by use of medications. Figure of One- and two-pollutant models for change in FENO using 2-day Moving Averages personal and central-site pollutant measurements.

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Delfino et al. (2006, 090745) **Period of Study:** Region 1: August to Mid December 2003. Region 2: July through November 2004 **Location:** Region 1: Riverside, CA. Region 2: Whittier, CA	**Outcome:** Fractional Concentration of Nitric Oxide in exhaled air (FENO) **Age Groups:** 9 through 18 **Study Design:** Longitudinal Panel Study **N:** 45 children **Statistical Analyses:** Linear mixed-effects models Two-stage hierarchical model Empirical Variograms Fourth-order polynomial distributed lag mixed-effects model **Covariates:** Personal temperature, personal rel. humid., 10-day exposure run, respiratory infections, region of study, sex, cumulative daily use of as-needed B-agonist inhalers **Dose-response Investigated?** No **Lags Considered:** Lag 0, Lag 1, 2-day moving avg	**Pollutant:** $PM_{2.5}$ **PM Component:** Elemental carbon **Personal Exposure** **Averaging Time:** 24 h Riverside **Mean (SD):** 0.42 (0.69) 50th(Median): 0.34 $\mu g/m^3$ **Range (Min, Max):** 0.01, 6.94 Whittier **Mean (SD):** 0.78 (1.42) 50th(Median): 0.47 **Range (Min, Max):** 0, 17.2 **Central Site** **Averaging Time:** 24 h Riverside **Mean (SD):** 1.61 (0.78) 50th(Median): 1.35 **Range (Min, Max):** 0.52, 3.64 Whittier **Mean (SD):** 0.71 (0.43) 50th(Median): 0.63 **Range (Min, Max):** 0.14, 2.95 **Monitoring Stations:** 48 personal nephelometers, 2 central sites **Copollutant (correlation):** **Personal** 24-h personal $PM_{2.5}$ 0.18 24-h personal EC 1.00 24-h personal OC 0.41 24-h personal NO_2 0.0.21 24-h central $PM_{2.5}$ 0.00 24-h central EC 0.04 24-h central OC -0.01 24-h central NO_2 0.23 **Central** 24-h personal $PM_{2.5}$ 0.12 24-h personal EC 0.04 24-h personal OC 0.03 24-h personal NO_2 0.19 24-h central $PM_{2.5}$ 0.55 24-h central EC 1.00 24-h central OC 0.87 24-h central NO_2 0.70	**PM Increment:** IQR increase (Riverside: 28.41 $\mu g/m^3$, Whittier 21.87 $\mu g/m^3$) Coefficient [Lower CI, Upper CI] lag: Mixed-model estimates of the association between personal and central-site air pollutant exposure and FENO Lag 0 Personal 0.29 (0.10, 0.48) Central 0.10 (-0.65, 0.85) Lag 1 Personal -0.01 (-0.23, 0.21) Central 0.99 (0.27, 1.71) 2-day MA Personal 0.72 (0.32, 1.12) Central 1.38 (0.15, 2.61) Stratified by Medication Use Lag = 2-day moving avg Not Taking Anti-Inflamm. Medication Personal 0.84 (0.08, 1.60) Central 1.02 (-2.55, 4.60) Taking Anti-Inflamm. Medication Personal 0.71 (0.28, 1.15) Central 1.42 (0.25, 2.60) Inhaled Corticosteroids Personal 0.67 (0.28, 1.07) Central 1.28 (0.07, 2.49) Antileukotrienes +- inhaled corticosteroids Personal 0.03 (-3.29, 3.35) Central 1.15 (-1.58, 3.88) Notes: Figure of Estimated lag effect of hourly personal $PM_{2.5}$ on FENO. Figure of the Estimated lag effect of hourly personal $PM_{2.5}$ on FENO by use of medications. Figure of One- and two-pollutant models for change in FENO using 2-day Moving Averages personal and central-site pollutant measurements.
Reference: Delfino et al. (2006, 090745) **Period of Study:** Region 1: August to Mid December 2003. Region 2: July	**Outcome:** Fractional Concentration of Nitric Oxide in exhaled air (FENO) **Age Groups:** 9 through 18	**Pollutant:** $PM_{2.5}$ **PM Component:** Organic carbon **Personal Exposure**	**PM Increment:** IQR increase (Riverside: 28.41 $\mu g/m^3$, Whittier 21.87 $\mu g/m^3$) Mixed-model estimates of the association between personal and central-site air

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
through November 2004 **Location:** Region 1: Riverside, CA. Region 2: Whittier, CA	**Study Design:** Longitudinal Panel Study **N:** 45 children **Statistical Analyses:** Linear mixed-effects models Two-stage hierarchical model Empirical Variograms Fourth-order polynomial distributed lag mixed-effects model **Covariates:** Personal temperature, personal rel. humid., 10-day exposure run, respiratory infections, region of study, sex, cumulative daily use of as-needed B-agonist inhalers **Dose-response Investigated?** No **Lags Considered:** Lag 0, Lag 1, 2-day moving avg	**Averaging Time:** 24 h Riverside **Mean (SD):** 5.63 (2.59) 50th(Median): 4.98 **Range (Min, Max):** 1.94, 12.38 Whittier **Mean (SD):** 6.81 (3.45) 50th(Median): 6.43 **Range (Min, Max):** 2.18, 31.5 **Central Site** **Averaging Time:** 24 h Riverside **Mean (SD):** 6.88 (1.86) **Percentiles:** 50th Median: 6.07 **Range (Min, Max):** 4.11, 11.62 Whittier **Mean (SD):** 3.93 (1.49) 50th(Median): 3.76 **Range (Min, Max):** 1.64, 8.82 **Monitoring Stations**: 48 personal nephelometers, 2 central sites **Copollutant (correlation):** **Personal** 24-h personal $PM_{2.5}$ 0.15 24-h personal EC 0.41 24-h personal OC 1.00 24-h personal NO_2 0.20 24-h central $PM_{2.5}$ -0.11 24-h central EC 0.03 24-h central OC -0.02 24-h central NO_2 0.21 **Central** 24-h personal $PM_{2.5}$ 0.21 24-h personal EC -0.01 24-h personal OC -0.02 24-h personal NO_2 0.17 24-h central $PM_{2.5}$ 0.66 24-h central EC 0.87 24-h central OC 1.00 24-h central NO_2 0.62	pollutant exposure and FENO Lag 0 Personal 0.51 (-0.28, 1.30) Central 0.93 (-0.20, 2.06) Lag 1 Personal 0.13 (-0.77, 1.03) Central0.51 (-0.64, 1.66) 2-day MA Personal 0.94 (-0.47, 2.35) Central 1.6 (-0.17, 3.37) Stratified by Medication Use Lag = 2-day moving avg. Not Taking Anti-Inflamm. Medication Personal 0.88 (-1.62, 3.38) Central 0.36 (-4.07, 4.79) Taking Anti-Inflamm. Medication Personal 0.87 (-0.79, 2.53) Central 2.05 (0.24, 3.86) Inhaled Corticosteroids Personal 2.47 (0.30, 4.64) Central 1.96 (0.14, 3.78) Antileukotrienes +- inhaled corticosteroids Personal 0.52 (-1.99, 3.02) Central 1.29 (-2.58, 5.15) Notes: Figure of Estimated lag effect of hourly personal $PM_{2.5}$ on FENO. Figure of the Estimated lag effect of hourly personal $PM_{2.5}$ on FENO by use of medications. Figure of One- and two-pollutant models for change in FENO using 2-day Moving Averages personal and central-site pollutant measurements
Reference: Dubowsky et al (2006, 088750) **Period of Study:** 3/2002-6/2002 **Location:** St. Louis, Missouri	**Outcome:** Chronic inflammation, Diabetes, Obesity, Hypertension, Cardiac Risk **Study Design:** Prospective Cohort	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-h avg **Mean (SD) unit:** 16 (6.0) **Range (Min, Max):** 6.5, 28 **Copollutants:**	**Increment:** 5.4 $\mu g/m^3$ **% Increase (Lower CI, Upper CI)** **Lag** % increase in inflammatory response and exposure to $PM_{2.5}$ in people ≥ 60

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
	Statistical Analyses:	BC	Inflammatory Marker:
	Poisson, LOESS	CO	IL-6: -8 (-16, 8)
	Age Groups:	NO_2	1: -6 (-10, 5)
	≥ 60	SO_2	2: -5 (-11, 6)
		O_3	3: -3 (-9, 6)
			4: -4 (-12, 10)
			5: -5 (-13, 8)
			6: -6 (-14, 9)
			7
			CRP: -2 (-22, 15)
			1: 3 (-8, 17)
			2: 4 (-9, 20)
			3: 9 (-4, 27)
			4: 11 (-5, 35)
			5: 8 (-9, 29)
			6: 5 (-12, 26)
			7
			WBC: 0 (-2, 4)
			1: 1 (-1, 2)
			2: 2 (-1, 3)
			3: 1 (-2, 5)
			4: 3 (-1, 10)
			5: 5 (0, 12)
			6: 8 (0, 14)
			7
			% Increase in inflammatory responses and exposure to ambient $PM_{2.5}$ concentrations in people ≥ 60
			Inflammatory Marker:
			CRP
			All conditions*: 14 (-5.4, 37)
			0-5 avg
			3 conditions met*: 81 (21, 172)
			0-5 avg
			2 conditions met*: 11 (-7.3, 33)
			0-5 avg
			IL-6
			All conditions*: -2.1 (-13, 11)
			0-5 avg
			3 conditions met*: 23 (-5.3, 59)
			0-5 avg
			2 conditions met*: -3.1 (-14, 9.7)
			0-5 avg
			WBC
			All conditions*: 3.4 (-1.8, 8.9)
			0-5 avg

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			3 conditions met*: 0.4 (-8.8, 11) 0-5 avg 2 conditions met*: 3.6 (-1.7, 9.1) 0-5 avg * All conditions met means model is adjusted for sex, obesity, diabetes, smoking history, ambient and microenvironmental apparent temperature, mold, pollen, trip, h, and vitamins. Three conditions met means model is adjusted for three of the variables. Two conditions met means model is adjusted for two of the variables.
Reference: Ebelt et al. (2005, 056907) **Period of Study:** Summer of 1998 **Location:** Vancouver, Canada	**Outcome:** spirometry, **Age Groups**: range from 54-86 yrs mean age = 74 years **Study Design:** extended analysis of a repeated-measures panel study **N:** 16 persons with COPD **Statistical Analyses:** Earlier analysis expanded by developing mixed-effect regression models and by evaluating additional exposure indicators **Dose-response Investigated?** No **Statistical Package**: SAS V8	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean (SD):** Ambient $PM_{2.5}$: 11.4 (4.6) Exposure to ambient $PM_{2.5}$: 7.9 (3.7) Nonsulfate ambient $PM_{2.5}$: 9.3 (3.7) Exposure to nonsulfate ambient $PM_{2.5}$: 6.5 (3.0) Total exposure to $PM_{2.5}$: 18.5 (14.9) Exposure to nonambient $PM_{2.5}$: 10.6 (14.5) **Range (Min, Max):** Ambient $PM_{2.5}$: (4.2-28.7) Exposure to ambient $PM_{2.5}$: (0.9-21.3) Nonsulfate ambient $PM_{2.5}$: (3.3-23.3) Exposure to nonsulfate ambient $PM_{2.5}$: (0.7-16.9) Total exposure to $PM_{2.5}$: (2.2-90.9) Exposure to nonambient $PM_{2.5}$: (-2.6-85.0) **Monitoring Stations**: 5 **Copollutant (correlation):** Ambient PM_{10}: r = 0.78 Ambient $PM_{10-2.5}$: r = 0.15 Ambient Sulfate- 0.82 Nonsufate Ambient $PM_{2.5}$: r = 0.98	**PM Increment:** Ambient $PM_{2.5}$: 5.8 (IQR) Exposure to ambient $PM_{2.5}$: 4.4 (IQR) Nonsulfate ambient $PM_{2.5}$: 4.2 (IQR) Exposure to nonsulfate ambient $PM_{2.5}$: 3.4 (IQR) Total exposure to $PM_{2.5}$: 10.1 (IQR) Exposure to nonambient $PM_{2.5}$: 8.9 (IQR) **Notes:** Effect estimates are presented in Figure 2 and Electronic Appendix Table 1 (only available with electronic version of article) and not provided quantitatively elsewhere.
Reference: Ebelt et al. (2005, 056907) **Period of Study:** Summer of 1998 **Location:** Vancouver, Canada	**Outcome:** spirometry **Age Groups**: Range from 54-86 yrs mean age = 74 years **Study Design:** extended analysis of a repeated-measures panel study **N:** 16 persons with COPD **Statistical Analyses:** Earlier analysis expanded by developing mixed-effect regression models and by evaluating additional exposure indicators **Dose-response Investigated?** No	**Pollutant:** Sulfate (SO_4) **Averaging Time:** 24 h **Mean (SD):** Ambient Sulfate: 2.0 (1.1) Exposure to Ambient Sulfate: 0.2 (4.7) **Range (Min, Max):** Ambient Sulfate: (0.4-5.4) Exposure to ambient Sulfate: (0.2-4.7) **Monitoring Stations**: 5 **Copollutant (correlation):**	**PM Increment:** Ambient Sulfate: 1.5 (IQR) Exposure to Ambient Sulfate: 0.9 (IQR) Notes: Effect estimates are presented in Figure 2 and Electronic Appendix Table 1 (only available with electronic version of article) and not provided quantitatively elsewhere.

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
	Statistical Package: SAS V8	Ambient $PM_{2.5}$: r = 0.82 Nonsulfate Ambient $PM_{2.5}$: r = 0.74 Exposure to Ambient Sulfate: r = 0.82	
Reference: Ferdinands et al. (2008, 156433) **Period of Study:** 8/16/2004–8/31/2004 **Location:** Atlanta, Georgia	**Outcome:** Respiratory Symptoms, airway inflammation **Study Design:** Prospective cohort **Statistical Analyses:** Pearson Correlation Analysis **Age Groups:** 14-18	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-h avg **Mean (SD) unit:** 27.2 (11.9) **Range (Min, Max):** 21.7, 34.7 **Copollutants (correlation):** O_3: r = 0.8-0.9	The study presents results qualitatively not quantitatively.
Reference: Gent et al. (2003, 052885) **Period of Study:** April 1 through September 30, 2001 **Location:** Connecticut Springfield, MA	**Outcome:** Respiratory symptoms including: Wheeze, persistent cough, chest tightness, shortness of breath **Age Groups:** Infants **Study Design:** 1-year prospective cohort study **N:** 1002 infants 17160 observations **Statistical Analyses:** Logistic regression analysis GEEs Tests for linear trend Test for goodness of fit Hosmer-Lemeshow statistic for regression **Covariates:** Temperature **Dose-response Investigated?** No **Statistical Package:** SAS **Lags Considered:** 1-day lag	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean (SD):** 13.1 (7.9) **Percentiles:** 20th: 6.9 40th: 9.0 50th(Median): 10.3 60th: 12.1 80th: 19.0 **Range (Min, Max):** 3.7, 44.2 **Monitoring Stations:** 4 sites **Copollutant (correlation):** Temperature: 0.58	**PM Increment:** 12 $\mu g/m^3$ same day 19 $\mu g/m^3$ previous day **Model 5 (same day)** **Wheeze** < 6.9 = 1.00 6.9–8.9 = 0.95 (0.83, 1.10) 9.0–12.0 = 1.04 (0.89, 1.20) 12.1–18.9 = 1.05 (0.92, 1.20) ≥ 19.0 = 0.93 (0.78, 1.11) **Persistent Cough** < 6.9 = 1.00 6.9–8.9 = 0.95 (0.87, 1.04) 9.0–12.0 = 0.96 (0.87, 1.06) 12.1–18.9 = 1.00 (0.91, 1.09) ≥ 19.0 = 0.95 (0.83, 1.09) **Chest Tightness** < 6.9 = 1.00 6.9–8.9 = 1.01 (0.86, 1.19) 9.0–12.0 = 1.06 (0.89, 1.26) 12.1–18.9 = 1.24 (1.06, 1.45) ≥ 19.0 = 1.05 (0.84, 1.33) **Shortness of Breath** < 6.9 = 1.00 6.9–8.9 = 1.01 (0.87, 1.17) 9.0–12.0 = 1.03 (0.87, 1.22) 12.1–18.9 = 1.07 (0.91, 1.25) ≥ 19.0 = 1.03 (0.83, 1.28) **Bronchodilator** < 6.9 = 1.00 6.9–8.9 = 1.04 (0.99, 1.09) 9.0–12.0 = 1.02 (0.96, 1.08) 12.1–18.9 = 1.04 (0.99, 1.09) ≥ 19.0 = 1.02 (0.97, 1.08) **Model 6 (previous day)** **Wheeze** < 6.9 = 1.00 6.9–8.9 = 1.06 (0.95, 1.20) 9.0–12.0 = 1.09 (0.94, 1.28) 12.1–18.9 = 1.03 (0.89, 1.19) ≥ 19.0 = 1.14 (0.97, 1.34) **Persistent Cough** < 6.9 = 1.00 6.9–8.9 = 1.04 (0.94, 1.14)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			9.0–12.0 = 1.05 (0.94, 1.17)
			12.1–18.9 = 1.03 (0.94, 1.14)
			≥ 19.0 = 1.12 (1.02 1.24)
			Chest Tightness < 6.9 = 1.00
			6.9–8.9 = 1.03 (0.87, 1.23)
			9.0–12.0 = 1.04 (0.85, 1.27)
			12.1–18.9 = 1.00 (0.84, 1.19)
			≥ 19.0 = 1.21 (1.00, 1.46)
			Shortness of Breath < 6.9 = 1.00
			6.9–8.9 = 1.00 (0.84, 1.19)
			9.0–12.0 = 1.09 (0.90, 1.31)
			12.1–18.9 = 1.09 (0.90, 1.31)
			≥ 19.0 = 1.26 (1.02, 1.54)
			Bronchodilator < 6.9 = 1.00
			6.9–8.9 = 0.98 (0.94, 1.03)
			9.0–12.0 = 0.99 (0.95, 1.03)
			12.1–18.9 = 0.97 (0.94, 1.01)
			≥ 19.0 = 0.99 (0.95, 1.04)
			$PM_{2.5}$ + O_3: Medication Users: Same-day
			Wheeze < 6.9 = 1.00
			6.9–8.9 = 0.89 (0.75, 1.29)
			9.0–12.0 = 1.02 (0.87, 1.19)
			12.1–18.9 = 0.94 (0.77, 1.15)
			≥ 19.0 = 0.83 (0.65, 1.06)
			Persistent Cough < 6.9 = 1.00
			6.9–8.9 = 0.95 (0.84, 1.06)
			9.0–12.0 = 0.97 (0.86, 1.10)
			12.1–18.9 = 0.94 (0.77, 1.15)
			≥ 19.0 = 0.83 (0.65, 1.06)
			Chest Tightness < 6.9 = 1.00
			6.9–8.9 = 0.90 (0.74, 1.09)
			9.0–12.0 = 0.97 (0.79, 1.18)
			12.1–18.9 = 0.97 (0.76, 1.25)
			≥ 19.0 = 0.76 (0.54, 1.05)
			Shortness of Breath < 6.9 = 1.00
			6.9–8.9 = 0.95 (0.80, 1.12)
			9.0–12.0 = 1.00 (0.82, 1.21)
			12.1–18.9 = 0.90 (0.73, 1.12)
			≥ 19.0 = 0.87 (0.65, 1.17)
			Bronchodilator < 6.9 = 1.00
			6.9–8.9 = 1.03 (0.98, 1.08)
			9.0–12.0 = 1.01 (0.96, 1.07)
			12.1–18.9 = 1.02 (0.95, 1.08)
			≥ 19.0 = 0.99 (0.91, 1.07)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			Previous Day
			Wheeze < 6.9 = 1.00
			6.9–8.9 = 1.03 (0.89, 1.18)
			9.0–12.0 = 1.05 (0.88, 1.24)
			12.1–18.9 = 0.98 (0.82, 1.17)
			≥ 19.0 = 1.05 (0.85, 1.29)
			Persistent Cough < 6.9 = 1.00
			6.9–8.9 = 0.99 (0.89, 1.11)
			9.0–12.0 = 0.98 (0.86, 1.10)
			12.1–18.9 = 0.95 (0.83, 1.10)
			≥ 19.0 = 1.00 (0.88, 1.15)
			Chest Tightness < 6.9 = 1.00
			6.9–8.9 = 0.89 (0.72, 1.10)
			9.0–12.0 = 0.90 (0.70, 1.16)
			12.1–18.9 = 0.81 (0.63, 1.03)
			≥ 19.0 = 0.91 (0.71, 1.17)
			Shortness of Breath < 6.9 = 1.00
			6.9–8.9 = 0.96 (0.78, 1.18)
			9.0–12.0 = 1.00 (0.81, 1.25)
			12.1–18.9 = 0.96 (0.74, 1.24)
			≥ 19.0 = 1.20 (0.94, 1.52)
			Bronchodilator < 6.9 = 1.00
			6.9–8.9 = 0.99 (0.94, 1.04)
			9.0–12.0 = 0.97 (0.93, 1.02)
			12.1–18.9 = 0.96 (0.91, 1.02)
			≥ 19.0 = 0.97 (0.89, 1.04)
			$PM_{2.5}$ + O_3: Non-users: Same-day
			Wheeze < 6.9 = 1.00
			6.9–8.9 = 0.92 (0.72, 1.17)
			9.0–12.0 = 1.08 (0.85, 1.36)
			12.1–18.9 = 0.94 (0.73, 1.22)
			≥ 19.0 = 1.15 (0.75, 1.75)
			Persistent Cough < 6.9 = 1.00
			6.9–8.9 = 0.96 (0.83, 1.12)
			9.0–12.0 = 1.02 (0.89, 1.18)
			12.1–18.9 = 0.93 (0.78, 1.12)
			≥ 19.0 = 1.07 (0.85, 1.34)
			Chest Tightness < 6.9 = 1.00
			6.9–8.9 = 0.84 (0.54, 1.31)
			9.0–12.0 = 1.09 (0.74, 1.61)
			12.1–18.9 = 0.78 (0.47, 1.30)
			≥ 19.0 = 0.71 (0.36, 1.39)
			Shortness of Breath < 6.9 = 1.00
			6.9–8.9 = 0.61 (0.39, 0.95)
			9.0–12.0 = 1.13 (0.85, 1.50)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			12.1–18.9 = 0.72 (0.42, 1.23) ≥ 19.0 = 1.17 (0.72, 1.90) **Bronchodilator Use:** < 6.9 = 1.00 6.9–8.9 = 0.95 (0.78, 1.15) 9.0–12.0 = 0.95 (0.78, 1.16) 12.1–18.9 = 0.85 (0.69, 1.06) ≥ 19.0 = 0.99 (0.76, 1.30) **Previous-day** **Wheeze** < 6.9 = 1.00 6.9–8.9 = 1.01 (0.78, 1.31) 9.0–12.0 = 1.15 (0.88, 1.51) 12.1–18.9 = 1.08 (0.78, 1.51) ≥ 19.0 = 1.18 (0.71, 1.97) **Persistent Cough** < 6.9 = 1.00 6.9–8.9 = 1.07 (0.94, 1.22) 9.0–12.0 = 1.13 (0.97, 1.32) 12.1–18.9 = 1.03 (0.87, 1.22) ≥ 19.0 = 1.14 (0.88, 1.46) **Chest Tightness** < 6.9 = 1.00 6.9–8.9 = 1.44 (0.90, 2.30) 9.0–12.0 = 1.50 (0.97, 2.33) 12.1–18.9 = 1.56 (0.91, 2.66) ≥ 19.0 = 1.76 (0.83, 3.73) **Shortness of Breath** < 6.9 = 1.00 6.9–8.9 = 0.99 (0.75, 1.30) 9.0–12.0 = 1.30 (0.88, 1.91) 12.1–18.9 = 0.84 (0.57, 1.24) ≥ 19.0 = 1.48 (0.94, 2.34) **Bronchodilator Use** < 6.9 = 1.00 6.9–8.9 = 1.05 (0.85, 1.34) 9.0–12.0 = 1.28 (1.01, 1.62) 12.1–18.9 = 1.05 (0.80, 1.37) ≥ 19.0 = 1.19 (0.83, 1.71) **Notes:** Line graphs of daily levels of ozone and $PM_{2.5}$ and daily temperature with daily prevalence of respiratory symptoms for users of asthma maintenance medication
Reference: Gent et al, (2009, 180399) **Period of Study:** 2000-2003 **Location:** New Haven County CT	**Outcome:** Increased asthma symptoms and medication use **Study Design:** Panel **Covariates:** Season, day of the week, date **Statistical Analysis:** Logistic regression **Statistical Package:** SAS **Age Groups:** Children aged 4-12	**Pollutant:** $PM_{2.5}$ and components **Averaging Time:** Daily **Mean:** (estimated sources, $\mu g/m^3$) Motor Vehicle: 6.6 Road Dust: 2.3 Sulfur: 5.5 Biomass Burning: 0.9 Oil: 0.8 Sea Salt: 0.5	**Odds Ratio and p-value for sources and components of $PM_{2.5}$. Lags are 0, 1 or 2 days, and the mean of days 0-2 (L02).** Source: Motor Vehicle *Elemental Carbon, Increment = 1000 ng/m^3* Wheeze L0: 1.04, p = 0.04 L1: 1.01, p = 0.70

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
		Range (Min, Max): NR	L2: 1.00, p = 0.99
		Copollutant (correlation): NR	L02: 1.07, p = 0.06
			Persistent Cough
			L0: 1.01, p = 0.42
			L1: 1.01, p = 0.38
			L2: 0.99, p = 0.44
			L02: 1.03, p = 0.23
			Shortness of Breath
			L0: 1.06, p = 0.001
			L1: 1.01, p = 0.65
			L2: 1.01, p = 0.63
			L02: 1.12, p = 0.01
			Chest Tightness
			L0: 1.03, p = 0.20
			L1: 1.02, p = 0.24
			L2: 1.01, p = 0.59
			L02: 1.10, p = 0.04
			Inhaler Use
			L0: 1.01, p = 0.15
			L1: 1.00, p = 0.72
			L2: 1.00, p = 0.75
			L02: 1.02, p = 0.40
			Zn, Increment = 10 ng/m^3
			Wheeze
			L0: 1.00, p = 0.69
			L1: 0.99, p = 0.54
			L2: 1.00, p = 0.89
			L02: 1.00, p = 0.98
			Persistent Cough
			L0: 1.00, p = 0.60
			L1: 1.00, p = 0.77
			L2: 0.99, p = 0.24
			L02: 1.00, p = 0.94
			Shortness of Breath
			L0: 1.02, p = 0.001
			L1: 1.00, p = 0.57
			L2: 1.01, p = 0.49
			L02: 1.04, p = 0.06
			Chest Tightness
			L0: 1.00, p = 0.72
			L1: 1.00, p = 0.96
			L2: 1.01, p = 0.38
			L02: 1.03, p = 0.13
			Inhaler Use
			L0: 1.00, p = 0.41

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			L1: 1.00, p = 0.44
			L2: 1.00, p = 0.52
			L02: 1.01, p = 0.53
			Pb, Increment = 5 ng/m^3
			Wheeze
			L0: 1.02, p = 0.31
			L1: 1.00, p = 0.91
			L2: 1.01, p = 0.62
			L02: 1.07, p = 0.13
			Persistent Cough
			L0: 1.02, p = 0.25
			L1: 1.00, p = 0.88
			L2: 1.00, p = 0.87
			L02: 1.05, p = 0.12
			Shortness of Breath
			L0: 1.03, p = 0.11
			L1: 0.98, p = 0.51
			L2: 1.03, p = 0.05
			L02: 1.12, p = 0.01
			Chest Tightness
			L0: 1.02, p = 0.31
			L1: 0.99, p = 0.79
			L2: 1.03, p = 0.13
			L02: 1.10, p = 0.02
			Inhaler Use
			L0: 1.01, p = 0.06
			L1: 0.98, p = 0.11
			L2: 1.02, p = 0.04
			L02: 1.04, p = 0.10
			Cu, Increment = 5 ng/m^3
			Wheeze
			L0: 1.01, p = 0.59
			L1: 0.99, p = 0.55
			L2: 0.99, p = 0.82
			L02: 1.02, p = 0.67
			Persistent Cough
			L0: 1.02, p = 0.13
			L1: 1.02, p = 0.21
			L2: 0.98, p = 0.26
			L02: 1.05, p = 0.04
			Shortness of Breath
			L0: 1.06, p = 0.01
			L1: 1.01, p = 0.74
			L2: 0.96, p = 0.10
			L02: 1.06, p = 0.21

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			Chest Tightness
			L0: 10.3, p = 0.23
			L1: 1.02, p = 0.42
			L2: 0.97, p = 0.17
			L02: 1.04, p = 0.39
			Inhaler Use
			L0: 1.01, p = 0.22
			L1: 0.99, p = 0.37
			L2: 1.00, p = 0.70
			L02: 1.01, p = 0.46
			Se, Increment = 1 ng/m^3
			Wheeze
			L0: 1.00, p = 0.97
			L1: 0.99, p = 0.52
			L2: 1.00, p = 0.91
			L02: 1.02, p = 0.71
			Persistent Cough
			L0: 1.00, p = 0.84
			L1: 0.99, p = 0.32
			L2: 1.00, p = 0.93
			L02: 0.98, p = 0.43
			Shortness of Breath
			L0: 1.02, p = 0.40
			L1: 0.97, p = 0.10
			L2: 1.01, p = 0.55
			L02: 1.02, p = 0.67
			Chest Tightness
			L0: 1.00, p = 0.79
			L1: 0.97, p = 0.13
			L2: 1.01, p = 0.72
			L02: 0.98, p = 0.61
			Inhaler Use
			L0: 0.99, p = 0.20
			L1: 1.01, p = 0.02
			L2: 0.99, p = 0.32
			L02: 0.99, p = 0.75
			Source: Road Dust
			Si, Increment = 100 ng/m^3
			Wheeze
			L0: 1.03, p = 0.03
			L1: 1.00, p = 0.99
			L2: 1.02, p = 0.26
			L02: 1.07, p = 0.04
			Persistent Cough
			L0: 1.02, p = 0.01

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			L1: 1.00, p = 0.78
			L2: 1.01, p = 0.60
			LO2: 1.05, p = 0.02
			Shortness of Breat1.04, p = 0.01h
			LO: 1.04, p = 0.01
			L1: 1.01, p = 0.60
			L2: 1.01, p = 0.63
			LO2: 1.08, p = 0.02
			Chest Tightness
			LO: 1.02, p = 0.20
			L1: 1.02, p = 0.17
			L2: 1.00, p = 0.88
			LO2: 1.06, p = 0.10
			Inhaler Use
			LO: 1.02, p = 0.004
			L1: 0.99, p = 0.18
			L2: 1.01, p = 0.45
			LO2: 1.03, p = 0.09
			Fe, Increment = 100 ng/m^3
			Wheeze
			LO: 1.04, p = 0.02
			L1: 1.00, p = 0.80
			L2: 1.00, p = 0.87
			LO2: 1.07, p = 0.05
			Persistent Cough
			LO: 1.02, p = 0.06
			L1: 1.01, p = 0.52
			L2: 0.99, p = 0.52
			LO2: 1.04, p = 0.04
			Shortness of Breath
			LO: 1.06, p = 0.002
			L1: 1.01, p = 0.65
			L2: 0.98, p = 0.27
			LO2: 1.08, p = 0.04
			Chest Tightness
			LO: 1.01, p = 0.47
			L1: 1.02, p = 0.22
			L2: 0.98, p = 0.35
			LO2: 1.05, p = 0.21
			Inhaler Use
			LO: 1.02, p = 0.004
			L1: 0.99, p = 0.44
			L2: 1.00, p = 0.91
			LO2: 1.03, p = 0.08
			Al, Increment = 50 ng/m^3

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			Wheeze
			L0: 1.02, p = 0.17
			L1: 1.01, p = 0.73
			L2: 1.02, p = 0.30
			L02: 1.07, p = 0.03
			Persistent Cough
			L0: 1.03, p = 0.001
			L1: 1.00, p = 0.96
			L2: 1.00, p = 0.68
			L02: 1.06, p = 0.01
			Shortness of Breath
			L0: 1.05, p = 0.002
			L1: 1.01, p = 0.63
			L2: 1.01, p = 0.59
			L02: 1.09, p = 0.004
			Chest Tightness
			L0: 1.02, p = 0.21
			L1: 1.02, p = 0.18
			L2: 1.00, p = 0.94
			L02: 1.07, p = 0.04
			Inhaler Use
			L0: 1.02, p = 0.02
			L1: 0.99, p = 0.27
			L2: 1.01, p = 0.50
			L02: 1.02, p = 0.11
			Ca, Increment = 50 ng/m^3
			Wheeze
			L0: 1.07, p = 0.02
			L1: 1.00, p = 0.97
			L2: 1.01, p = 0.74
			L02: 1.14, p = 0.04
			Persistent Cough
			L0: 1.05, p = 0.01
			L1: 0.99, p = 0.64
			L2: 1.00, p = 0.90
			L02: 1.09, p = 0.03
			Shortness of Breath
			L0: 1.10, p = 0.002
			L1: 1.02, p = 0.66
			L2: 1.00, p = 0.89
			L02: 1.18, p = 0.01
			Chest Tightness
			L0: 1.04, p = 0.26
			L1: 1.03, p = 0.43
			L2: 1.00, p = 0.93

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			L02: 1.14, p = 0.07
			Inhaler Use
			L0: 1.04, p = 0.01
			L1: 0.97, p = 0.06
			L2: 1.01, p = 0.44
			L02: 1.04, p = 0.17
			Ba, Increment = 10 ng/m^3
			Wheeze
			L0: 0.99, p = 0.57
			L1: 1.00, p = 0.92
			L2: 0.99, p = 0.48
			L02: 0.99, p = 0.81
			Persistent Cough
			L0: 1.00, p = 0.83
			L1: 1.01, p = 0.38
			L2: 0.99, p = 0.32
			L02: 1.00, p = 0.81
			Shortness of Breath
			L0: 1.04, p = 0.02
			L1: 1.00, p = 0.96
			L2: 0.96, p = 0.05
			L02: 1.03, p = 0.38
			Chest Tightness
			L0: 1.01, p = 0.63
			L1: 1.00, p = 0.88
			L2: 0.98, p = 0.30
			L02: 1.02, p = 0.51
			Inhaler Use
			L0: 1.01, p = 0.08
			L1: 0.99, p = 0.19
			L2: 1.00, p = 0.92
			L02: 1.01, p = 0.36
			Ti, Increment = 5 ng/m^3
			Wheeze
			L0: 1.00, p = 0.59
			L1: 0.99, p = 0.49
			L2: 1.01, p = 0.34
			L02: 1.01, p = 0.56
			Persistent Cough
			L0: 1.00, p = 0.57
			L1: 1.00, p = 0.55
			L2: 1.00, p = 0.30
			L02: 1.01, p = 0.29
			Shortness of Breath
			L0: 1.01, p = 0.01

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			L1: 1.00, p = 0.56
			L2: 1.00, p = 0.60
			LO2: 1.03, p = 0.05
			Chest Tightness
			LO: 1.00, p = 0.34
			L1: 1.00, p = 0.55
			L2: 0.99, p = 0.49
			LO2: 1.01, p = 0.52
			Inhaler Use
			LO: 1.00, p = 0.72
			L1: 1.00, p = 0.30
			L2: 1.00, p = 0.67
			LO2: 1.00, p = 0.66
			Source: Sulfur
			S, Increment = 1000 ng/m^3
			Wheeze
			LO: 0.98, p = 0.43
			L1: 0.99, p = 0.62
			L2: 1.02, p = 0.29
			LO2: 1.00, p = 0.99
			Persistent Cough
			LO: 1.00, p = 0.84
			L1: 1.00, p = 0.69
			L2: 1.02, p = 0.21
			LO2: 1.02, p = 0.27
			Shortness of Breath
			LO: 1.01, p = 0.63
			L1: 0.99, p = 0.71
			L2: 1.01, p = 0.55
			LO2: 1.01, p = 0.79
			Chest Tightness
			LO: 0.99, p = 0.80
			L1: 1.01, p = 0.62
			L2: 1.01, p = 0.81
			LO2: 1.02, p = 0.68
			Inhaler Use
			LO: 0.99, p = 0.13
			L1: 1.00, p = 0.81
			L2: 1.02, p = 0.04
			LO2: 1.00, p = 0.81
			P, Increment = 50 ng/m^3
			Wheeze
			LO: 0.98, p = 0.39
			L1: 0.98, p = 0.48
			L2: 1.02, p = 0.38

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			L02: 0.99, p = 0.89
			Persistent Cough
			L0: 1.00, p = 0.75
			L1: 0.99, p = 0.69
			L2: 1.01, p = 0.38
			L02: 1.03, p = 0.30
			Shortness of Breath
			L0: 1.01, p = 0.61
			L1: 0.99, p = 0.71
			L2: 1.01, p = 0.67
			L02: 1.01, p = 0.78
			Chest Tightness
			L0: 1.00, p = 0.88
			L1: 1.01, p = 0.72
			L2: 1.00, p = 0.87
			L02: 1.02, p = 0.67
			Inhaler Use
			L0: 0.98, p = 0.15
			L1: 1.00, p = 0.83
			L2: 1.01, p = 0.11
			L02: 1.00, p = 0.99
			Source: Biomass Burning
			K, Increment = 50 ng/m^3
			Wheeze
			L0: 0.98, p = 0.06
			L1: 0.99, p = 0.43
			L2: 1.00, p = 0.85
			L02: 0.96, p = 0.04
			Persistent Cough
			L0: 1.00, p = 0.64
			L1: 1.00, p = 0.83
			L2: 1.00, p = 0.46
			L02: 1.00, p = 0.86
			Shortness of Breath
			L0: 1.01, p = 0.01
			L1: 0.98, p = 0.09
			L2: 1.00, p = 0.38
			L02: 1.00, p = 0.79
			Chest Tightness
			L0: 1.00, p = 0.02
			L1: 0.99, p = 0.24
			L2: 0.98, p = 0.07
			L02: 0.99, p = 0.67
			Inhaler Use
			L0: 1.00, p = 0.68

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			L1: 0.99, p = 0.05
			L2: 1.00, p = 0.59
			LO2: 0.99, p = 0.28
			Source: Oil
			V, Increment = 10 ng/m^3
			Wheeze
			LO: 0.99, p = 0.73
			L1: 0.96, p = 0.03
			L2: 0.99, p = 0.56
			LO2: 0.93, p = 0.04
			Persistent Cough
			LO: 1.01, p = 0.56
			L1: 0.99, p = 0.24
			L2: 0.98, p = 0.01
			LO2: 0.96, p = 0.05
			Shortness of Breath
			LO: 1.01, p = 0.46
			L1: 0.98, p = 0.24
			L2: 1.00, p = 0.83
			LO2: 0.98, p = 0.58
			Chest Tightness
			LO: 0.99, p = 0.71
			L1: 0.98, p = 0.32
			L2: 0.98, p = 0.23
			LO2: 0.94, p = 0.12
			Inhaler Use
			LO: 0.98, p = 0.12
			L1: 1.00, p = 0.68
			L2: 0.99, p = 0.22
			LO2: 0.96, p = 0.03
			Ni, Increment = 5 ng/m^3
			Wheeze
			LO: 1.01, p = 0.59
			L1: 0.97, p = 0.09
			L2: 1.00, p = 0.76
			LO2: 0.99, p = 0.72
			Persistent Cough
			LO: 1.01, p = 0.21
			L1: 0.99, p = 0.57
			L2: 0.99, p = 0.23
			LO2: 1.00, p = 0.99
			Shortness of Breath
			LO: 1.04, p = 0.05
			L1: 0.98, p = 0.36
			L2: 1.00, p = 0.81

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			L02: 1.04, p = 0.32
			Chest Tightness
			L0: 1.01, p = 0.58
			L1: 1.00, p = 0.89
			L2: 0.98, p = 0.27
			L02: 1.01, p = 0.84
			Inhaler Use
			L0: 1.01, p = 0.48
			L1: 1.00, p = 0.83
			L2: 0.99, p = 0.51
			L02: 1.01, p = 0.48
			Source: Sea Salt
			Na, Increment = 100 ng/m^3
			Wheeze
			L0: 0.98, p = 0.23
			L1: 1.00, p = 0.80
			L2: 1.00, p = 0.88
			L02: 0.97, p = 0.29
			Persistent Cough
			L0: 1.00, p = 0.58
			L1: 0.99, p = 0.19
			L2: 1.00, p = 0.61
			L02: 0.98, p = 0.21
			Shortness of Breath
			L0: 1.00, p = 0.94
			L1: 0.99, p = 0.46
			L2: 1.01, p = 0.63
			L02: 0.99, p = 0.74
			Chest Tightness
			L0: 0.99, p = 0.43
			L1: 0.99, p = 0.75
			L2: 1.00, p = 0.88
			L02: 0.98, p = 0.61
			Inhaler Use
			L0: 0.99, p = 0.35
			L1: 1.00, p = 0.61
			L2: 1.00, p = 0.85
			L02: 0.99, p = 0.37
			Cl, Increment = 10 ng/m^3
			Wheeze
			L0: 1.00, p = 0.89
			L1: 1.00, p = 0.88
			L2: 1.00, p = 0.38
			L02: 1.00, p = 0.81
			Persistent Cough

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			L0: 1.00, p = 0.31
			L1: 1.00, p = 0.31
			L2: 1.00, p = 0.51
			L02: 1.00, p = 0.06
			Shortness of Breath
			L0: 1.00, p = 0.89
			L1: 1.00, p = 0.94
			L2: 1.00, p = 0.70
			L02: 1.00, p = 0.80
			Chest Tightness
			L0: 1.00, p = 0.24
			L1: 1.00, p = 0.28
			L2: 1.00, p = 0.52
			L02: 1.00, p = 0.65
			Inhaler Use
			L0: 1.00, p = 0.69
			L1: 1.00, p = 0.52
			L2: 1.00, p = 0.51
			L02: 1.00, p = 0.83
			Odds Ratio (95%CI) from repeated measures logistic regression models of respiratory symptoms and daily source concentrations of $PM_{2.5}$.
			Lag 0 Model
			Wheeze, p = 0.23
			Motor Vehicle: 1.05 (0.99-1.10)
			Road Dust: 1.10 (1.01-1.19)
			Sulfur: 0.97 (0.94-1.00)
			Biomass Burning: 0.80 (0.66-0.98)
			Oil: 1.02 (0.86-1.20)
			Sea Salt: 0.96 (0.86-1.07)
			Persistent Cough, $p < 0.001$
			Motor Vehicle: 1.02 (0.99-1.04)
			Road Dust: 1.06 (1.01-1.11)
			Sulfur: 1.00 (0.98-1.01)
			Biomass Burning: 0.97 (0.92-1.03)
			Oil: 1.02 (0.95-1.10)
			Sea Salt: 0.99 (0.92-1.07)
			Shortness of Breath, $p < 0.001$
			Motor Vehicle: 1.06 (1.01-1.11)
			Road Dust: 1.12 (1.02-1.22)
			Sulfur: 0.98 (0.94-1.02)
			Biomass Burning: 1.05 (0.95-1.17)
			Oil: 1.07 (0.92-1.26)
			Sea Salt: 1.01 (0.92-1.12)
			Chest Tightness, $p < 0.001$

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			Motor Vehicle: 1.02 (0.97-1.08)
			Road Dust: 1.04 (0.95-1.15)
			Sulfur: 0.99 (0.94-1.03)
			Biomass Burning: 1.06 (0.95-1.18)
			Oil: 0.99 (0.82-1.18)
			Sea Salt: 0.95 (0.84-1.08)
			Inhaler Use, $p < 0.001$
			Motor Vehicle: 1.02 (1.00-1.05)
			Road Dust: 1.06 (1.02-1.11)
			Sulfur: 0.98 (0.97-1.00)
			Biomass Burning: 1.00 (0.96-1.03)
			Oil: 0.98 (0.91-1.05)
			Sea Salt: 0.99 (0.94-1.04)
			Lag 02 Model
			Wheeze, $p = 0.86$
			Motor Vehicle: 1.10 (1.01-1.19)
			Road Dust: 1.26 (1.05-1.51)
			Sulfur: 0.98 (0.92-1.04)
			Biomass Burning: 0.64 (0.46-0.88)
			Oil: 0.80 (0.56-1.08)
			Sea Salt: 0.91 (0.82-1.16)
			Persistent Cough, $p < 0.001$
			Motor Vehicle: 1.03 (0.98-1.09)
			Road Dust: 1.16 (1.02-1.32)
			Sulfur: 1.01 (0.98-1.05)
			Biomass Burning: 0.93 (0.81-1.06)
			Oil: 0.84 (0.71-1.00)
			Sea Salt: 0.88 (0.77-1.01)
			Shortness of Breath, $p = 0.006$
			Motor Vehicle: 1.12 (1.01-1.24)
			Road Dust: 1.28 (1.05-1.55)
			Sulfur: 0.97 (0.90-1.04)
			Biomass Burning: 0.78 (0.52-1.18)
			Oil: 0.94 (0.69-1.29)
			Sea Salt: 1.01 (0.79-1.29)
			Chest Tightness, $p = 0.39$
			Motor Vehicle: 1.08 (0.98-1.20)
			Road Dust: 1.20 (0.97-1.49)
			Sulfur: 1.00 (0.92-1.08)
			Biomass Burning: 0.87 (0.62-1.22)
			Oil: 0.80 (0.58-1.10)
			Sea Salt: 0.95 (0.71-1.27)
			Inhaler Use, $p < 0.001$
			Motor Vehicle: 1.03 (0.98-1.08)
			Road Dust: 1.09 (1.00-1.19)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			Sulfur: 1.00 (0.97-1.03)
			Biomass Burning: 0.95 (0.87-1.04)
			Oil: 0.92 (0.81-1.05)
			Sea Salt: 0.97 (0.88-1.07)
			Odds Ratio (95%CI) from repeated measures logistic regression models of respiratory symptoms and daily source concentrations of $PM_{2.5}$ when copollutants are included.
			Wheeze
			Motor Vehicle
			NO_2: 1.03 (0.98-1.08)
			CO: 1.05 (0.99-1.11)
			SO_2: 1.04 (0.99-1.09)
			O_3: 1.06 (0.97-1.16)
			Road Dust
			NO_2: 1.11 (1.02-1.20)
			CO: 1.10 (1.01-1.19)
			SO_2: 1.10 (1.01-1.19)
			O_3: 1.11 (1.01-1.23)
			Sulfur
			NO_2: 0.96 (0.92-0.99)
			CO: 0.97 (0.94-1.01)
			SO_2: 0.97 (0.93-1.00)
			O_3: 0.95 (0.91-1.00)
			Biomass Burning
			NO_2: 0.79 (0.65-0.98)
			CO: 0.80 (0.66-0.98)
			SO_2: 0.79 (0.64-0.98)
			O_3: 0.74 (0.57-0.97)
			Oil
			NO_2: 1.02 (0.87-1.21)
			CO: 1.02 (0.86-1.20)
			SO_2: 1.01 (0.86-1.19)
			O_3: 0.92 (0.62-1.39)
			Sea Salt
			NO_2: 0.96 (0.85-1.07)
			CO: 0.96 (0.86-1.08)
			SO_2: 0.95 (0.85-1.07)
			O_3: 1.01 (0.72-1.40)
			Inhaler Use
			Motor Vehicle
			NO_2: 1.02 (0.99-1.04)
			CO: 1.02 (0.99-1.05)
			SO_2: 1.02 (0.99-1.04)
			O_3: 1.02 (0.98-1.07)
			Road Dust

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			NO_2: 1.06 (1.02-1.10)
			CO: 1.06 (1.02-1.11)
			SO_2: 1.06 (1.02-1.11)
			O_3: 1.06 (1.00-1.13)
			Sulfur
			NO_2: 0.98 (0.96-1.00)
			CO: 0.98 (0.96-1.00)
			SO_2: 0.98 (0.96-1.00)
			O_3: 0.97 (0.95-1.00)
			Biomass Burning
			NO_2: 1.00 (0.96-1.03)
			CO: 0.99 (0.96-1.03)
			SO_2: 0.99 (0.96-1.03)
			O_3: 0.99 (0.95-1.03)
			Oil
			NO_2: 0.98 (0.91-1.05)
			CO: 0.97 (0.91-1.04)
			SO_2: 0.97 (0.91-1.04)
			O_3: 1.03 (0.88-1.22)
			Sea Salt
			NO_2: 0.99 (0.94-1.04)
			CO: 0.99 (0.94-1.04)
			SO_2: 0.99 (0.94-1.04)
			O_3: 1.01 (0.88-1.15)
Reference: Girardot et al. (2006, 088271) **Period of Study:** 10 August 2002-16 October 2002 17 June 2003-27 August 2003 **Location:** Charlies Bunion Trail (portion of Appalachia Trail)	**Outcome:** Pulmonary function/spirometry–FVC, FEV_1, PEF, FVC/FEV_1, FEF_{25-75} **Age Groups**: 18-82 yrs **Study Design:** Cohort **N:** 354 hikers **Statistical Analyses:** Multiple linear regression **Covariates:** Age, h hiked, mean temperature, sex, smoking status, history of asthma or wheeze symptoms, carriage of backpack, whether reaching summit or not **Season:** Fall 2002, Summer 2003 **Dose-response Investigated?** No **Statistical Package**: SAS	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean:** Trail: 13.9 +/- 8.2 Estimated personal: 15.0 +/- 7.4 **Range (Min, Max):** Trail: 1.6 , 38.4 Estimated personal: 0.21, 41.9 **Copollutant (correlation):** O_3 (r = 0.67, for estimated personal exposure)	**PM Increment:** 1 $\mu g/m^3$ % Change +/- CI p value Univariate: FVC: 0.023 +/ 0.035 0.51 FEV_1: 0.015 +/- 0.029 0.607 PEF: 0.185 +/- 0.091 0.043 FVC/FEV_1: 0.003 +/- 0.023 0.905 $FEF_{25-75\%}$: 0.052 +/- 0.093 0.578 Adjusted: FVC: 0.007 +/ 0.040 0.966 FEV_1: 0.003 +/- 0.033 0.937 PEF: 0.258 +/- 0.103 0.013 FVC/FEV_1: - 0.011 +/- 0.027 0.676

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			$FEF_{25-75\%}$: - 0.041 +/- 0.109
			0.707
			Spirometry result for each quintile +/- CI
			Quintile 1 (6.0 $\mu g/m^3$): FVC (L): Prehike: 4.32 +/- 0.13
			Posthike: 4.33 +/- 0.12
			FEV_1 (L): Prehike: 3.39 +/- 0.10
			Posthike: 3.40 +/- 0.10
			FEV_1/FVC (%): Prehike: 78.66 +/- 0.86
			Posthike: 78.63 +/- 0.81
			$FEF_{25-75\%}$ (L/sec): Prehike: 3.27 +/- 0.14
			Posthike: 3.26 +/- 0.14
			PEF (L/sec): Prehike: 7.91 +/ 0.22
			Posthike: 7.58 +/- 0.22
			Quintile 2 (10.4 $\mu g/m^3$): FVC (L): Prehike: 4.30 +/- 0.11
			Posthike: 4.30 +/- 0.11
			FEV_1 (L): Prehike: 3.42 +/- 0.09
			Posthike: 3.43 +/- 0.09
			FEV_1/FVC (%): Prehike: 79.37 +/- 0.71
			Posthike: 79.55 +/- 0.69
			$FEF_{25-75\%}$ (L/sec): Prehike: 3.39 +/- 0.14
			Posthike: 3.38 +/- 0.14
			PEF (L/sec): Prehike: 8.37 +/ 0.23
			Posthike: 8.26 +/- 0.25
			Quintile 3 (14.8 $\mu g/m^3$): FVC (L): Prehike: 4.34 +/- 0.12
			Posthike: 4.33 +/- 0.12
			FEV_1 (L): Prehike: 3.42 +/- 0.10
			Posthike: 3.40 +/- 0.09
			FEV_1/FVC (%): Prehike: 79.20 +/- 0.81
			Posthike: 78.83 +/- 0.80
			$FEF_{25-75\%}$ (L/sec): Prehike: 3.19 +/- 0.13
			Posthike: 3.21 +/- 0.13
			PEF (L/sec): Prehike: 8.12 +/ 0.25
			Posthike: 7.89 +/- 0.25
			Quintile 4 (17.9 $\mu g/m^3$):
			FVC (L): Prehike: 4.23 +/- 0.11
			Posthike: 4.23 +/- 0.11
			FEV_1 (L): Prehike: 3.36 +/- 0.10
			Posthike: 3.36 +/- 0.10
			FEV_1/FVC (%): Prehike: 79.18 +/- 0.81
			Posthike: 79.26 +/- 0.79
			$FEF_{25-75\%}$ (L/sec): Prehike: 3.34 +/- 0.15
			Posthike: 3.30 +/- 0.15
			PEF (L/sec): Prehike: 7.75 +/ 0.25

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			Posthike: 7.73 +/- 0.26 **Quintile 5 (25.6 μg/m^3):** FVC (L): Prehike: 4.15 +/- 0.11 Posthike: 4.18 +/- 0.12 FEV_1 (L): Prehike: 3.31 +/- 0.09 Posthike: 3.33 +/- 0.10 FEV_1/FVC (%): Prehike: 79.73 +/- 0.66 Posthike: 79.55 +/- 0.64 $FEF_{25-75\%}$ (L/sec): Prehike: 3.22 +/- 0.14 Posthike: 3.24 +/- 0.14 PEF (L/sec): Prehike: 7.72 +/ 0.22 Posthike: 7.77 +/- 0.23 **Overall (15.0 μg/m^3):** FVC (L): Prehike: 4.27 +/- 0.05 Posthike: 4.27 +/- 0.05 FEV_1 (L): Prehike: 3.38 +/- 0.04 Posthike: 3.38 +/- 0.04 FEV_1/FVC (%): Prehike: 79.2 +/- 0.34 Posthike: 79.2 +/- 0.33 $FEF_{25-75\%}$ (L/sec): Prehike: 3.28 +/- 0.06 Posthike: 3.28 +/- 0.06 PEF (L/sec): Prehike: 7.97 +/ 0.11 Posthike: 7.97 +/- 0.11
Reference: Hertz-Picciotta et al. (2007, 135917) **Period of Study:** 1994-2003 **Location:** Teplice and Prachatice, Czech Republic	**Outcome:** Lower respiratory illness–croup (J05, J04), acute bronchitis (J20), acute bronchiolitis (J21) **Age Groups:** Neonates followed for 2 to 4.5 yrs **Study Design:** Cohort **N:** 1133 children **Statistical Analyses:** Generalized linear longitudinal models **Covariates:** District, mother's age, mother's education, mother or adult smoke, child's sex, season, day of the week, fuel for heating and/or cooking, breastfeeding category, number of other children, temperature **Season:** Winter, spring, summer and fall **Dose-response Investigated?** No **Statistical Package:** SUDAAN version 8 **Lags Considered:** 1-3, 1-7, 1-14, 1-30, 1-45	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean (SD):** PAH: 22.3 (SD–16 for 3-day avg and 11 for 45-day avg)	**PM Increment:** 25 μg/m^3 **RR Estimate [Lower CI, Upper CI]** **lag:** Birth–23 months: 1.30 [1.08, 1.58] lag 1-30 2–4.5 yrs: 1.23 [0.94, 1.62] lag 1-30 RR Estimate for categories of exposure [Lower CI, Upper CI] lag: Crude RR: Birth–23 months: $>$ 50 μg/m^3: 2.26 [1.81, 2.82] lag 1-30 25-50 μg/m^3: 1.48 [1.32, 1.65] lag 1-30 $<$ 25 μg/m^3: Reference 2–4.5 yrs: $>$ 50 μg/m^3: 3.66 [2.07, 6.48] lag 1-30 25-50 μg/m^3: 1.60 [1.41, 1.82] lag 1-30 $<$ 25 μg/m^3: Reference
Reference: Hertz- Picciotta et al. (2007, 135917) **Period of Study:** 1994-2003 **Location:** Teplice and Prachatice, Czech Republic	**Outcome:** Lower respiratory illness–croup (J05, J04), acute bronchitis (J20), acute bronchiolitis (J21) **Age Groups:** Neonates followed for 2 to 4.5 yrs	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean (SD):** PAH:	**PAH Increment:** 100 ng/m^3 **RR Estimate [Lower CI, Upper CI]** **lag:** Birth–23 months:

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
	Study Design: Cohort **N:** 1133 children **Statistical Analyses:** Generalized linear longitudinal models **Covariates:** District, mother's age, mother's education, mother or adult smoke, child's sex, season, day of the week, fuel for heating and/or cooking, breastfeeding category, number of other children, temperature **Dose-response Investigated?** No **Statistical Package:** SUDAAN version 8 **Lags Considered:** 1-3, 1-7, 1-14, 1-30, 1-45	52.5 ng/m^3 (SD–57 ng/m^3 for 3-day avg and 46 ng/m^3 for 45-day avg)	1.29 [1.07, 1.54] lag 1-30 2–4.5 yrs: 1.56 [1.22, 2.00] lag 1-30 RR Estimate for categories of exposure [Lower CI, Upper CI] lag: Crude RR: Birth–23 months: $>$ 100 ng/m^3: 2.52 [2.22, 2.87] lag 1-30 40-100 ng/m^3: 1.87 [1.65, 2.13] lag 1-30 $<$ 40 ng/m^3: Reference 2–4.5 yrs: $>$ 100 ng/m^3: 2.26 [1.93, 2.65] lag 1-30 40-100 ng/m^3: 1.40 [1.20, 1.64] lag 1-30 $<$ 40 ng/m^3: Reference
Reference: Hogervorst, et al (2006, 189460) **Period of Study:** 2002 **Location:** Maastricht, the Netherlands (six schools selected)	**Outcome**: Decreased lung function **Age Groups**: 8-13 years old **Study Design:** Multivariate linear regression (enter method) analysis **N:** 342 children **Statistical Analyses:** ANOVA, chi square **Covariates:** Independent variables: Age, height, gender, smoking at home by parents, pets, use of ventilation hoods during cooking, presence of unvented geysers, tapestry in the home, indoor/outdoor time, education level of parents. Dependent variables: lung function indices **Dose-response Investigated?** No	**Pollutant:** $PM_{2.5}$ **Averaging Time:** Daily **Mean (SD):** 19.0 (3.2) **Monitoring Stations**: 6 **Copollutant:** PM_{10} Total Suspended Particles (TSP)	**PM Increment:** 10 μg/m^3 **RR Estimate [Lower CI, Upper CI]** **lag:** FEV: 3.62 [0.50,7.63] lag NR FVC: 1.80 [-2.10, 5.80] lag NR FEF: 5.93 [-2.34, 14.89] lag NR
Reference: Holguin et al, (2007, 099000) **Period of Study:** NR??? **Location:** Ciudad Juarez, Mexico	**Outcome:** FeNO, FEV_1 **Study Design:** Panel **Covariates:** sex, age, body mass index, day of week, season, years of maternal and paternal education, passive smoking **Statistical Analysis:** linear and nonlinear mixed effects models **Age Groups:** 6-12 years	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 48h **Mean (SD) Unit:** 17.5 (8.9) μg/m^3 **Range (Min, Max):** NR **Copollutant (correlation):** NR	**Increment:** NR **Relative Risk (Min CI, Max CI)** **Lag** Results not given in table form, but abstract states that no significant associations with $PM_{2.5}$ were observed.
Reference: Hong et al. (2007, 091347) **Period of Study:** March 23-May3, 2004 **Location:** School on the Dukjeok Island near Incheon City, Korea	**Outcome:** Peak expiratory flow rate (PEFR) **Age Groups**: 3rd to 6th grade (mean age=9.6 yrs) **Study Design: P**anel study **N:** 43 schoolchildren **Statistical Analyses:** Mixed linear regression **Covariates:** age, sex, height, weight, asthma history, and passive smoking exposure at home **Dose-response Investigated?** No **Lags Considered:** 0, 1, 2, 3, 4, 5	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean (SD):** 20.27 (8.23) 50th(Median): 22.07 **Range (Min, Max):** 5.94-36.28 **Copollutant:** PM_{10} Components of PM_{10} (Fe, Mn, Pb, Zn, Al)	**Effect Estimate:** Regression coefficients of morning and daily mean PEFR on $PM_{2.5}$ Lag 1 ($PM_{2.5}$) Morning PEFR Crude: ß= -0.14, p=0.12 Adjusted: ß= -0.54, p,0.01 Mean PEFR Crude: ß= -0.15, p=0.02 Adjusted: ß= -0.54, p,0.01 Regression coefficients of morning and daily mean PEFR on $PM_{2.5}$ and GSTM1

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			and GSTT1 genotype using linear mixed-effects regression Lag 1 ($PM_{2.5}$) Morning PEFR: ß= -0.57, p < 0.01 Mean PEFR: ß= -0.56, p < 0.01 GSTM1 Morning PEFR: ß= 20.04, p=0.25 Mean PEFR: ß= 18.75, p=0.28 GSTT1 Morning PEFR: ß= 2.31, p=0.89 Mean PEFR: ß= 1.75, p=0.91
Reference: Jansen, et al. (2005, 082236) **Period of Study:** 1987-2000 **Location:** Seattle, WA	**Outcome:** FENO: fractional exhaled nitrogen oxide, Spirometry, Blood pressure, SaO2: oxygen saturation, Pulse rate **Age Groups**: 60-86-years-old **Study Design:** Short-term cross-sectional case series **N:** 16 subjects diagnosed with COPD, asthma, or both **Statistical Analyses:** Linear mixed effects model with random intercepts **Covariates:** Age, relative humidity, temperature, medication use **Season: W**inter 2002-2003 **Dose-response Investigated?** No **Statistical Package**: STATA	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean (SD):** **Fixed-Site Monitor:** 14.0 All Subjects (N=16) Indoor, home: 7.29 Outdoor, home: 10.47 Asthmatic Subjects (N=7) Indoor, home: 7.25 Outdoor, home: 8.99 COPD Subjects (N=9) Indoor, home: 7.33 Outdoor, home: 11.66 **Range (Min, Max):** **Fixed-Site Monitor:** 1.3, 44 IQR All Subjects Indoor, home: 4.05 Outdoor, home: 8.87 Asthmatic Subjects Indoor, home: 5.72 Outdoor, home: 7.55 COPD Subjects Indoor, home: (3.18 Outdoor, home: 6.71	**PM Increment:** $PM_{2.5}$: 10 $\mu g/m^3$ Slope [95% CI]: dependence of FENO concentration [ppb] on $PM_{2.5}$ **Asthmatic Subjects** Indoor, home: 3.69 [-0.74: 8.12] Outdoor, home: 4.23 [1.33: 7.13]* **Copd Subjects** Indoor, home: -0.35 [-7.45: 6.75] Outdoor, home: 3.83 [-1.84: 9.49] Results indicate that FENO may be a more sensitive biomarker of PM exposure than other traditional health endpoints.
Reference: Johnston, et al. (2006, 091386) **Period of Study:** 7 months (April 7 through November 7, 2004) **Location:** Darwin, Australia	**Outcome:** Asthma symptoms **Age Groups**: All Ages **Study Design:** Time-series **N: 2**51 people (130 adults, 121 children **Statistical Analyses:** Logistic regression model **Covariates:** Minimum air temperature, doctor visits for influenza and the prevalence of asthma symptoms and, the fungal spore count and both onset of asthma symptoms and commencement of	**Pollutant:** $PM_{2.5}$ **Averaging Time: D**aily **Mean (SD):** 11.1 (5.4) **Range (Min, Max):** 2.2, 36.5 **PM Component:** Vegetation fire smoke (95%) and motor vehicle emissions (5%) **Monitoring Stations**: 1	**PM Increment:** 5 $\mu g/m^3$ **RR Estimate [Lower CI, Upper CI]** **lag:** **Symptoms attributable to asthma** Overall: 1.000 (0.98,1.01) Adults: 1.000 (0.976,1.026) Children: 1.008 (0.980, 1.037) Using preventer: 1.013 (0.990, 1.037) **Became symptomatic**

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
	reliever medication **Season:** "Dry season"- note Southern Hemisphere **Dose-response Investigated?** No **Statistical Package:** STATA8 **Lags Considered:** 0-5 days		Overall: 1.150 (1.07,1.23) Adults: 1.165 (1.058,1.284) Children: 1.148 (1.042,1.264) Using preventer: 1.181 (1.076,1.296) **Used Reliever** Overall: 1.000 (0.98,1.02) Adults: 1.007 (0.980, 1.035) Children: 1.002 (0.972,1.034) Using preventer: 1.020 (1.000,1.042) **Commenced Reliever** Overall: 1.120 (1.03,1.210) Adults: 1.141 (1.021, 1.275) Children: 1.112 (0.994,1.243) Using preventer: 1.129 (1.013,1.257) **Commenced Oral Steroids** Overall: 1.310 (1.03,1.66) Adults: 1.601 (1.192, 2.150) Children: 0.995 (0.625,1.459) Using preventer: 1.350 (1.040,1.752) **Asthma Attack** Overall: 0.980 (0.94,1.04) Adults: 1.026 (0.962, 1.095) Children: 0.832 (0.731, 0.946) Using preventer: 1.002 (0.934,1.075) **Exercise induced asthma** Overall: 0.990 (0.95,1.03) Adults: 0.998 (0.943, 1.056) Children: 0.982 (0.899,1.071) Using preventer: 1.002 (0.942,1.067) **Saw a health professional for asthma** Overall: 1.030 (0.91,1.16) Adults: 1.079 (0.899, 1.296) Children: 1.003 (0.841,1.195) Using preventer: 0.980 (0.847,1.133) **Missed school or work due to asthma** Overall: 1.025 (0.9284,1.131) Adults: 1.077 (0.923, 1.247) Children: 1.000 (0.873,1.458) Using preventer: 1.005 (0.897,1.124) **Mean daily number of asthma symptoms** Overall: 1.003 (0.99,1.01) Adults: 0.998 (0.984, 1.012) Children: 1.004 (0.985,1.023) Using preventer: 1.013 (0.999,1.028) **Mean Daily number of applications of**

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			reliever Overall: 1.002 (0.993,1.010) Adults: 1.001 (0.986, 1.016) Children: 1.000 (0.980,1.021) Using preventer: 1.005 (0.994,1.017)
Reference: Koenig et al. (2003, 156653) **Period of Study:** Winter 2000-2001, Spring 2001 **Location:** Seattle, WA	**Outcome:** Exhaled NO (eNO) **Age Groups:** 6-13 years old **Study Design:** Cohort **N:** 19 children **Statistical Analyses:** Linear mixed-effects regression **Covariates:** Medication use, ambient NO reading for specific individual on specific day of session, mean ambient NO for subject during session, mean ambient NO for subject during all sessions **Season:** Winter, Spring **Dose-response Investigated?** No **Statistical Package:** STATA	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 10 consecutive days **Mean (SD):** Outdoor: 13.3 (1.4) Indoor: 11.1 (4.9) Personal: 13.4 (3.2) Central-site: 10.1 (5.7) **Range (Min, Max):** Outdoor: Max: 40.4 Indoor: Max: 36.3 Personal: Max: 49.4 Central-site: NR **Monitoring Stations:** Outdoor: NR Indoor: NR Personal: NR Central-site: 3 **Copollutant (correlation):** Outdoor PM-central-site NO: 0.50 For NO values < 100 ppb, outdoor PM-central-site NO: 0.04	**PM Increment:** 10 $\mu g/m^3$ Results presented as change in eNO (95% CI) Among ICS* nonuser Personal monitor 4.48 (1.02, 7.93) Outdoor monitor 4.28 (1.38, 7.17) Indoor monitor 4.21 (1.02, 7.41) Central site 3.82 (1.22, 6.43) Among ICS* user Personal monitor -0.09 (-2.39, 2.21) Outdoor monitor 0.74 (-2.28, 3.76) Indoor monitor -1.11 (-5.08, 2.87) Central site 1.28 (-1.23, 3.79) * ICS: Inhaled corticosteroid
Reference: Koenig et al. (2003, 156653) **Period of Study:** Winter 2000-2001, spring 2001 **Location:** Seattle, WA	**Outcome:** Increased exhaled nitric oxide (eNO) **Age Groups:** 6–13 years of age **Study Design:** Combined recursive and predictive model **N:** 19 children with asthma **Statistical Analyses:** Linear mixed effects model **Covariates:** Residence type, air cleaner, avg outdoor temperature, avg daily rainfall **Season:** Winter, Spring **Dose-response Investigated?** No **Statistical Package:** STATA 7.0 for health analyses, SAS 8.0	**Pollutant:** $PM_{2.5}$ **Averaging Time:** Daily **Mean:** Home indoor 9.5 Home outdoor 11.1 Recursive model Eag: 7.0 Recursive model Eig: 2.1 Predictive model Eag: 6.0 Predictive model Eig: 4.0 Combined model Eag: 6.4 Combined model Eig: 3.2 **25th:** Home indoor 5.7 Home outdoor 6.3 Recursive model Eag: 4.2 Recursive model Eig: 0.0 Predictive model Eag: 3.4 Predictive model Eig: 0.9 Combined model Eag: 3.7 Combined model Eig: 0.5 **50th(Median):** Home indoor 7.6 Home outdoor 9.5 Recursive model Eag: 5.9 Recursive model Eig: 1.2 Predictive model Eag: 5.0	**PM Increment:** 10-µg/m^3 RR Estimate [Lower CI, Upper CI] lag: Eag = ambient-generated personal exposure Eig = indoor-generated personal exposure eNO = exhaled nitric oxide Recursive model with 8 children, Eag was marginally associated with increases in eNO [5.6 ppb [–0.6,11.9]. Eig was not associated with eNO (–0.19 ppb). For those combined estimates, only Eag was significantly associated with an increase in eNO: Eag: 5.0 ppb [0.3, 9.7] Eig: 3.3 ppb [1.1, 7.7] Notes: Effects were seen only in children who were not using corticosteroid therapy

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
		Predictive model Eig: 2.2 Combined model Eag: 5.5 Combined model Eig: 1.7 **75th**: Home indoor 10.8 Home outdoor 14.6 Recursive model Eag: 9.2 Recursive model Eig: 2.3 Predictive model Eag: 7.5 Predictive model Eig: 4.9 Combined model Eag: 7.8 Combined model Eig: 4.2 **Range (Min, Max):** Home indoor 2.3, 36.3 Home outdoor 2.8, 40.4 Recursive Eag: 1.8,22.6 Recursive Eig: 0.0,17.2 Predictive Eag: 1.3,22.6 Predictive Eig: 0.0,33.0 Combined Eag: 1.3,22.6 Combined Eig: 0.0,33.0 **Monitoring Stations**: 19 personal environmental monitors	
Reference: Kongtip et al. (2006, 096920) **Period of Study:** September 1–October 31, 2004 **Location:** Dindang district, Bangkok metropolitan, Thailand	**Outcome:** respiratory and other Outcomes reported **Age Groups**: Age range 15 to 55 yrs **Study Design:** panel study **N:** 77 street vendors **Statistical Analyses:** Binary logistic regression **Covariates:** Gender, age, type of fuel used, working duration (months) **Dose-response Investigated?** No	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean (SD):** 70.94 **Percentiles:** 50th(Median): 72.05 **Range (Min, Max):** 23.20-120.00 **Monitoring Stations**: 1 **Copollutant (correlation):** SO_2 NO_2 O_3 VOCs CO	**PM Increment:** 1 $\mu g/m^3$ **Effect Estimate [Lower CI, Upper CI]:** **Model 1** Headache: 1.011 (0.999-1.022) Nose congestion: 1.006 (0.997-1.015) Sore throat: 1.000 (0.991-1.008) Cold: 1.006 (0.995-1.017) Cough: 0.989 (0.980-0.998) Phlegm: 0.998 (0.992-1.003) Chest tightness: 0.995 (0.955-1.036) Fever: 1.008 (0.993-1.024) Eye irritation: 1.022 (1.011-1.033) Dizziness: 1.027 (1.013-1.041) Weakness: 0.996 (0.983-1.008) Upper respiratory symptom: 1.001 (0.994-1.008) Lower respiratory symptom: 0.997 (0.992-1.002) **Model 2** Headache: 1.004 (0.996-1.013) Nose congestion: 1.003 (0.996-1.010) Sore throat: 0.995 (0.989-1.001) Cold: 0.996 (0.988-1.004) Cough: 0.990 (0.983-0.996) Phlegm: 0.995 (0.991-0.999)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			Chest tightness: 0.997 (0.970-1.025) Fever: 1.010 (0.998-1.022) Eye irritation: 1.019 (1.010-1.028) Dizziness: 1.020 (1.009-1.032) Weakness: 1.003 (0.994-1.012) Upper respiratory symptom: 0.995 (0.990-1.000) Lower respiratory symptom: 0.995 (0.991-0.999)
Reference: Lagorio et al. (2006, 089800) **Period of Study:** 5/24/1999 to 6/24/1999 and 11/181999 to 12/22/1999 **Location:** Rome, Italy	**Outcome:** Lung function (FVC and FEV_1) of subjects with COPD, Asthma **Age Groups**: COPD 50 to 80 yrs Asthma 18 to 64 yrs **Study Design:** Time series **N:** COPD = 11 Asthma = 11 **Statistical Analyses:** Non-parametric Spearman correlation GEE **Covariates:** COPD and IHD: daily mean temperature, season variable (spring or winter), relative humidity, day of week Asthma: season variable, temperature, humidity, and β-2-agonist use **Season**: Spring and Winter **Dose-response Investigated?** Yes **Statistical Package**: STATA **Lags Considered:** 1–3 days	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean (SD):** Overall: 27.2 (19.4) Spring: 18.2 (5.0) Winter: 36.7 (24.1) **Range (Min, Max):** 4.5, 100 **PM Component:** Cd: 0.46 ± 0.40 ng/m^3 Cr: 1.9 ± 1.7 ng/m^3 Fe: 283 ± 167 ng/m^3 Ni: 4.8 ± 6.5 ng/m^3 Pb: 30.6 ± 19.0 ng/m^3 Pt: 5.0 ± 8.6 pg/m^3 V: 1.8 ± 1.4 ng/m^3 Zn: 45.8 ± 33.1 ng/m^3 **Monitoring Stations**: 2 fixed sites: (Villa Ada and Istituto superior di Sanita) **Copollutant (correlation):** NO_2 r = 0.43 O_3 r = -0.51 CO r = 0.67 SO_2 r = 0.34 $PM_{10-2.5}$ r = 0.34 PM_{10} r = 0.93	**PM Increment:** 1 μg/m^3 They observed negative association between ambient $PM_{2.5}$ and respiratory function (FVC and FEV_1) in the COPD panel. The effect on FVC was seen at lag 24 h, 48 h, and 72 h. The effect on FEV_1 was evident at lag 72 h. There was no statistically significant effect of $PM_{2.5}$ on FVC and FEV_1 in the asthmatic and IHD panels. **β Coefficient (SE)** **COPD** FVC(%) 24 h -0.80 (0.36) 48-h -0.89 (0.41) 72-h -1.10 (0.55) FEV_1(%) 24 h -0.47 (0.33) 48-h -0.69 (0.37) 72-h -1.06 (0.50) **Asthma** FVC(%) 24 h -0.14 (0.29) 48-h -0.07 (0.33) 72-h -0.06 (0.39) FEV_1(%) 24 h -0.30 (0.34) 48-h -0.36 (0.39) 72-h -0.40 (0.46)
Reference: Lee et al. (2007, 093042) **Period of Study:** 2000-2001 **Location:** South-Western Seoul Metropolitan area, Seoul, South Korea	**Outcome:** PEFR (peak expiratory flow rate), lower respiratory symptoms (cold, cough, wheeze) **Age Groups**: 61-89 years of age (77.8 mean age) **Study Design:** longitudinal panel survey **N:** 61 adults **Statistical Analyses:** SAS MIXED, logistic regression model **Covariates:** Temperature (Celsius), relative humidity, age, **Dose-response Investigated?** No **Statistical Package**: SAS 8.0	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean (SD):** 51.15 (19.94) **Percentiles:** 25th: 33.00 50th(Median): 53.20 75th: 87.54 **Range (Min, Max):** 17.94, 92.71 **Monitoring Stations**: 2	**PM Increment:** 10 μg/m^3 **Effect Estimate [Lower CI, Upper CI]** **lag:** PEFR (peak expiratory flow rate) -0.54 (-0.89,-0.19) 1 day relative odds of a lower respiratory symptom (cold, cough, wheeze) 0.976 (0.849,1.121) 1 day

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
	Lags Considered: 0-4 days		
Reference: Lewis et al. (2005, 081079) **Period of Study:** winter 2001-spring 2002 **Location:** Detroit, Michigan, USA	**Outcome:** Poorer lung function (increased diurnal variability and decreased forced expiratory volume) **Age Groups:** 7-11 years old **Study Design:** Longitudinal cohort study **N:** 86 children **Statistical Analyses:** Descriptive statistics and bivariate analyses of exposures, multivariable regression multivariate analog of linear regression. **Covariates:** Sex, home location, annual family income, presence of one or more smokers in household, race, season (entered as dummy variables), and parameters to account for intervention group effect. **Season:** Winter 2001 (February 10–23), Spring 2001 (May 5–18), Summer 2001 (July 14–27), Fall 2001 (September 22–October 5), Winter 2002 (January 18–31), and Spring 2002 (May 18–31)]. **Dose-response Investigated?** No **Lags Considered:** 1 to 2 days, 3-5 days	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 2 weeks **Mean (SD):** Eastside 15.7 (10.6) Southwest 17.5 (12.2) **Range (Min, Max):** 1.0, 56.1 **Monitoring Stations:** 2 **Copollutant (correlation):** PM_{10} 0.93 O_3 Daily mean 0.57 O_3 8-h peak 0.53	**PM Increment:** 12.5 $\mu g/m^3$ **RR Estimate [Lower CI, Upper CI]** **lag:** Lung function among children reporting use of maintenance CSs **Diurnal variability** FEV_1 Lag 1: 1.61 [-0.5,3.72] Lag 1: 0.99 [-5.64, 7.62] $PM_{2.5}$ + O_3 Lag 2: 2.96 [-1.74,7.66] Lag 2: 4.62 [-4.31, 13.54] $PM_{2.5}$ + O_3 Lag 3-5: 1.37 [-1.49,4.22] Lag 3-5: 2.70 [1.0, 4.40] $PM_{2.5}$ + O_3 **Lowest daily value** FEV_1 Lag 1: -2.23 [-6.99,2.53] Lag 1: 3.36 [-3.92, 10.63] $PM_{2.5}$ + O_3 Lag 2: -0.21 [-4.09,3.68] Lag 2: 0.88 [-8.69, 10.46] $PM_{2.5}$ + O_3 Lag 3-5: -0.76 [-5.00, 3.49] Lag 3-5: -2.78 [-4.87 to -0.70] $PM_{2.5}$ + O_3 Lung function among children reporting presence of URI on day of lung function assessment **Diurnal variability** FEV_1 Lag 1: 4.08 [-1.78, 9.94] Lag 1: 3.99 [-2.76, 10.74] $PM_{2.5}$ + O_3 Lag 2: 7.62 [-0.49, 15.73] Lag 2: 4.10 [-1.41, 9.60] $PM_{2.5}$ + O_3 Lag 3-5: 1.47 [-7.73, 10.67] Lag 3-5: 3.81 [-1.83, 9.45] $PM_{2.5}$ + O_3 **Lowest daily value** FEV_1 Lag 1: -1.21 [5.62,3.21] Lag 1: -0.74 [-4.14, 2.65] $PM_{2.5}$ + O_3 Lag 2: -0.10 [4.36,4.16] Lag 2: -1.67 [-5.09, 1.75] $PM_{2.5}$ + O_3 Lag 3-5: -2.88 [-5.46 to -0.30] Lag 3-5: -2.78 [-4.79 to -0.77] $PM_{2.5}$ + O_3
Reference: Liu et al. (2009, 192003) **Period of Study:** 4wks in 2005 **Location:** Windsor, Ontario, Canada	**Outcome:** Decreased lung function **Study Design:** Panel **Statistical Analysis:** mixed-effects regression models **Statistical Package:** S-PLUS **Age Groups:** Asthmatic children, 9-14 yrs.	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 1, 2 & 3 days **Mean (SD) Unit (1d):** 6.5 $\mu g/m^3$ **Range (Min, Max):** 2.0-19.0 **Copollutant (correlation):** SO_2: 0.56 NO_2: 0.71 O_3: -0.41	**Increment:** 5.4 $\mu g/m^3$ **Percent Change (Min CI, Max CI)** **Lag** FEV1 Same Day: -0.5 (-1.3-0.3) Lag 1 Day: -0.5 (-1.1-0.5) 2-Day Average: -0.6 (-1.5-0.4) 3-Day Average: -1.1 (-3.1-0.9)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			FEF 25%-75%
			Same Day: -1.9 (-3.5--0.3)
			Lag 1 Day: -1.2 (-2.8-0.3)
			2-Day Average: -2.0 (-3.8--0.2)
			3-Day Average: -3.3 (-7.2-0.8)
			FeNO
			Same Day: 5.3 (-3.6-15)
			Lag 1 Day: 1.7 (-6.3-15)
			2-Day Average: 4.3 (-5.4-15.1)
			3-Day Average: -17.3 (-33.5-2.9)
			TBARS
			Same Day: 16.9 (2.2-33.6)
			Lag 1 Day: 14.6 (0.8-30.4)
			2-Day Average: 22.0 (4.8-42.1)
			3-Day Average: 69.1 (20.1-138.2)
			8-Isoprostane
			Same Day: 5.1 (-3.6-14.5)
			Lag 1 Day: -3.8 (-12.1-5.3)
			2-Day Average: 0.1 (-9.8-11.1)
			3-Day Average: 5.8 (-15.8-33.0)/
Reference: Mar et al. (2004, 057309)	**Outcome:** Respiratory Symptoms	**Pollutant:** 2.5	**PM Increment:** 10 μg/m^3
Period of Study: 1997-1999	**Age Groups**: Adults: Ages 20-51 yrs	**Mean (SD):**	**OR Estimate [Lower CI, Upper CI]**
Location: Spokane, Washington	Children: Ages 7-12 yrs	1997: 11.0 (5.9)	**lag:**
	N: 25 people	1998: 10.3 (5.4)	**Adult Respiratory symptoms: Wheeze:** 1.04[0.86, 1.26]
	Statistical Analyses: Logistic regression	1999: 8.1 (3.8)	lag 0
	Covariates: Temperature, relative humidity, day of-the-wk	Unit (i.e. μg/m^3):	1.00[0.83, 1.19]
	Statistical Package: STATA 6	**Monitoring Stations**: 1 station	lag 1
	Lags Considered: 0-2 days	**Copollutant (correlation):**	0.99[0.84, 1.17]
		$PM_{2.5}$	lag 2
		PM_1	**Breath:** 0.97[0.87, 1.08]
		r = 0.92	lag 0
		PM_{10}	0.98[0.87, 1.10]
		r = 0.61	lag 1
		$PM_{10-2.5}$	0.95[0.80, 1.13]
		r = 0.28	lag 2
			Cough: 0.86[0.62, 1.21]
			lag 0
			0.87[0.63, 1.20]
			lag 1
			0.89[0.66, 1.20]
			lag 2
			Sputum: 0.94[0.63, 1.41]
			lag 0
			0.90[0.62, 1.31]

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			lag 1
			0.92[0.66, 1.27]
			lag 2
			Runny Nose: 0.98[0.83, 1.15]
			lag 0
			0.95[0.82, 1.10]
			lag 1
			0.93[0.80, 1.08]
			lag 2
			Eye Irritation: 0.91[0.70, 1.20]
			lag 0
			0.89[0.70, 1.13]
			lag 1
			0.86[0.68, 1.08]
			lag 2
			Lower Symptoms: 0.91[0.73, 1.13]
			lag 0
			0.89[0.72, 1.10]
			lag 1
			0.89[0.72, 1.10]
			lag 2
			Any Symptoms: 0.92[0.80, 1.07]
			lag 0
			0.89[0.76, 1.04]
			lag 1
			0.89[0.75, 1.05]
			lag 2
			Children Respiratory symptoms: Wheeze: 0.55[0.26, 1.19]
			lag 0
			0.53[0.18, 1.58]
			lag 1
			0.55[0.19, 1.64]
			lag 2
			Breath: 1.13[0.86, 1.48]
			lag 0
			1.12[0.86, 1.44]
			lag 1
			1.10[0.82, 1.48]
			lag 2
			Cough: 1.17[0.98, 1.40]
			lag 0
			1.21[1.00, 1.47]
			lag 1
			1.18[0.99, 1.42]

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			lag 2 **Sputum:** 1.06[0.92, 1.22] lag 0 1.10[0.91, 1.34] lag 1 1.09[0.92, 1.30] lag 2 **Runny Nose:** 1.09[0.85, 1.39] lag 0 1.12[0.89, 1.41] lag 1 1.16[0.94, 1.42] lag 2 **Eye Irritation:** 0.93[0.53, 1.64] lag 0 0.75[0.45, 1.27] lag 1 0.77[0.65, 0.91] lag 2 **Lower Symptoms:** 1.18[1.00, 1.38] lag 0 1.21[1.00, 1.46] lag 1 1.17[0.96, 1.43] lag 2 **Any Symptoms:** 1.17[1.03, 1.34] lag 0 1.22[1.04, 1.43] lag 1 1.23[1.07, 1.42] lag 2
Reference: Mar et al. (2005, 087566) **Period of Study:** 1999-2001 **Location:** Seattle, Washington	**Outcome:** Pulmonary function (arterial oxygen saturation) and cardiac function (heart rate and blood pressure) **Study Design:** Time series **Statistical Analyses:** Linear logistic regression **Age Groups:** > 57	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-h avg	**Increment:** 10 $\mu g/m^3$ **% Increase (Lower CI, Upper CI)** **Lag** **Personal:** Systolic: 0.37 (-0.93, 1.67) 0 Diastolic: -0.20 (-0.85, 0.46) 0 **Indoor:** Systolic: 0.92 (-2.04, 3.87) 0 Diastolic: 0.38 (-1.43, 2.20) 0 **Outdoor:** Systolic: -0.81 (-2.34, 0.73) 0 Diastolic: -0.46 (-1.49, 0.57)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			0 % Increase between heart rate and $PM_{2.5}$ exposure for people > 57 **$PM_{2.5}$:** Personal: 0.44 (0.04, 0.84) 0 Indoor: 0.22 (-0.71, 1.16) 0 Outdoor: -0.75 (-1.42 to -0.07) 0
Reference: Mar et al. (2005, 088759) **Period of Study:** 1999-2002 **Location:** Seattle, Washington	**Outcome:** Respiratory Symptoms **Age Groups:** 6-13 years **Study Design:** Time-Series **N:** 19 children **Statistical Analyses:** Polynomial distributed lag model, Poisson regression **Covariates:** Age, ambient NO levels, temperature, relative humidity, modification of use of inhaled corticosteroids **Season:** Winter, Spring **Dose-response Investigated?** No **Statistical Package:** STATA **Lags Considered:** 0-8 h	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-h **Mean (SD):** Results presented in Figure 1. **Monitoring Stations:** 3 Stations	**PM Increment:** 10 $\mu g/m^3$ **Change in FE(NO) (exhaled NO concentration) with air pollution [Lower CI, Upper CI]** **lag:** Medication use: No meds: 6.99[3.43, 10.55] lag 1-h Meds: -0.18[-3.33, 2.97] lag 1-h No meds: 6.30[2.64, 9.97] lag 4-h Meds: -0.77[-4.58, 3.04] lag 4-h No meds: 0.46[-1.18, 2.11] lag 8-h Meds: 0.40[-1.94, 2.74] lag 8-h
Reference: McCreanor et al. (2007, 092841) **Period of Study:** 2003-2005 **Location:** London, England	**Outcome:** Decreased Lung Function **Age Groups:** Adults **Study Design:** Crossover study **N:** 60 adults **Statistical Analyses:** Linear regression **Covariates:** Temperature, relative humidity, age, sex, bod-mass index, and race or ethnic group	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 1 h **Mean (SD):** NR 50th(Median): Oxford St: 28.3 Hyde Park: 11.9 **Range (Min, Max):** Oxford St: (13.9, 76.1) Hyde Park: (3, 55.9)	% changes in FEV and FVC are presented in figures 1-3. Results are not presented quantitatively in text or tables. The authors did not find any significant differences in respiratory symptoms between the two locations. Also, there were no significant differences in sputum eosinophil counts or eosinophil cationic protein levels.
Reference: Moshammer and Neuberger (2003, 041956) **Period of Study:** 2000-2001 **Location:** Linz, Austria	**Outcome:** Lung Function: FVC, FEV_1, MEF_{25}, MEF_{50}, MEF_{75}, PEF, LQ Signal, PAS Signal **Age Groups:** Ages 7 to 10 **Study Design:** Case-crossover **N:** 161 children 1898–2120 "half-h means" **Statistical Analyses:** Correlations Regression Analysis **Covariates:** Morning, evening, night **Season:** Spring, Summer, Winter, Fall	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 8 h means & Daily Means **Mean (SD):** 14.61 (10.83) **Range (Min, Max):** (NR, 119.92) **Monitoring Stations:** 1 **Copollutant (correlation):** LQ = 0.751 PAS = 0.354	**Notes:** "Acute effects of 'active particle surface' as measured by diffusion charging were found on pulmonary function (FVC, FEV_1, MEF50) of elementary school children and on asthma-like symptoms of children who had been classified as sensitive."

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
	Dose-response Investigated? No		
Reference: Moshammer et al. (2006, 090771) **Period of Study:** 2000-2001 **Location:** Linz, Austria	**Outcome:** Respiratory symptoms and decreased lung function **Age Groups:** Children ages 7-10 **Study Design:** Time-series **N:** 163 children **Statistical Analyses:** Generalized estimating equations model **Covariates:** Sex, age, height, weight **Dose-response Investigated?** NR **Statistical Package:** NR **Lags Considered:** 1	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 8 h **Mean (SD):** Maximum 24 h: 76.39 Annual avg: 19.06 **Percentiles:** 8-h mean 25th: 8.64 8-h mean 50th(Median): 15.70 8-h mean 75th: 25.82 **Monitoring Stations:** 1 station **Copollutant (correlation):** PM_1 r = 0.95 PM_{10} r = 0.93 NO_2 r = 0.54	**PM Increment:** 10 $\mu g/m^3$ **% change in Lung Function per 10 $\mu g/m^3$** FEV: 0.23 FVC: 0.08 $FEV_{0.5}$: 0.33 $MEF_{75\%}$: -0.49 $MEF_{50\%}$: -0.58 $MEF_{25\%}$: -0.83 PEF: 0.41 **% change in Lung Function per IQR** FEV: -0.59 FVC: -0.2 $FEV_{0.5}$: 0.85 $MEF_{75\%}$: -1.25 $MEF_{50\%}$: -1.48 MEF_{25}%: -2.14 PEF: -1.06 **Multiple pollutant model** FEV: 0.10 FVC: 0.21 $FEV_{0.5}$: 0.06 $MEF_{75\%}$: -0.15 MEF50%: 0.04 MEF25%: -0.21 PEF: -0.18 **% change in Lung Function per IQR** FEV: 0.27 FVC: 0.54 $FEV_{0.5}$: 0.15 $MEF_{75\%}$: -0.39 $MEF_{50\%}$: 0.11 $MEF_{25\%}$: 0.54 PEF: 0.015: -0.47
Reference: Murata et al. (2007, 156787) **Period of Study:** Nov 2nd- 12th 2004 **Location:** Tokyo, Japan	**Outcome:** Exhaled nitric oxide levels, (eNO), a marker of airway inflammation **Age Groups:** 5-10 years **Study Design:** Cohort/Panel study **N:** 19 schoolchildren* **Statistical Analyses:** Linear regression **Covariates:** None **Season:** November (fall) **Dose-response Investigated?** No **Statistical Package:** SAS **Lags Considered:** Lag h 1-24, 8-h	**Pollutant:** $PM_{2.5}$ **Averaging Time:** Hourly, 24-h **Mean (SD):** 39.0 (16.9) $\mu g/m^3$ (daily mean) **Range (Min, Max):** 10, 120 (range of hourly values) **Monitoring Stations:** 1, on the street where the children lived	**PM Increment:** IQR 110 $\mu g/m^3$ **Mean [Lower CI, Upper CI]** **lag:** 0.145 [0.62, 0.228] ppb eNO 8 h moving avg **Notes:** Associations for lag h 1-24 presented in figures. Authors state "Individual hourly lag models showed a consistent association between the eNO value and $PM_{2.5}$ for exposure in the previous 24 h" "The trend on the graphs strongly suggest that fluctuations in eNO were

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
	moving avg, 7-h moving avg, 6-h moving avg, 24-h moving avg		affected by changes in air pollutants over at least the previous 8-h period" $PM_{2.5}$, black carbon, and NOx were all highly correlated (shown in figures), so effects are difficult to separate Pollutant concentrations peaked in the morning and evening h during traffic peaks
Reference: Neuberger et al. (2004, 093249) **Period of Study:** 6/1999-6/2000 **Location:** Austria (Vienna and a rural area near Linz)	**Outcome:** Questionnaire derived asthma score, and a 1-5 point respiratory health rating by parent **Age Groups**: 7-10 years **Study Design:** Cross-sectional survey **N:** about 2000 children **Statistical Analyses:** mixed models linear regression-used factor analysis to develop the "asthma score" **Covariates:** Pre-existing respiratory conditions, temperature, rainy days, # smokers in household, heavy traffic on residential street, gas stove or heating, molds, sex, age of child, allergies of child, asthma in other family members **Dose-response Investigated?** No **Statistical Package**: NR **Lags Considered:** 4 week avg (preceding interview)	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Copollutant (correlation):** PM_{10} (r = 0.94) in Vienna	**PM Increment:** 10 $\mu g/m^3$ **Change in mean associated unit increase in PM** **(p-value)** **lag** Respiratory Health score Vienna: 0.016 ($p > 0.05$) lag 4 week avg Rural area: 0.022 ($p < 0.05$) lag 4 week avg Asthma score Vienna: 0.006 ($p > 0.05$) lag 4 week avg Rural area: 0.004 ($p > 0.05$) lag 4 week avg
Reference: Neuberger et al. (2004, 093249) **Period of Study:** Sept 1999-March 2000 **Location:** Vienna, Austria	**Outcome:** Ratio measure: Time to peak tidal expiratory flow divided by total expiration time (i.e., tidal lung function, a surrogate for bronchial obstruction) **Age Groups**: 3.0-5.9 years (preschool children) **Study Design:** Longitudinal prospective cohort **N:** 56 children **Statistical Analyses:** mixed models linear regression, with autoregressive correlation structure **Covariates:** Age, sex, respiratory rate, phase angle, temperature, kindergarten, parental education, observer (also in sensitivity analyses: height, weight, cold/sneeze on same day, heating with fossil fuels, hair cotinine, number of tidal slopes used to measure tidal lung function) **Dose-response Investigated?** No **Statistical Package**: SAS 8.0 **Lags Considered:** Lag 0	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **PM Component:** Total carbon Elemental carbon Organic Carbon **Copollutant (correlation):** PM_{10} (r = 0.94) in Vienna	**PM Increment:** Interquartile range (NR) **Change in mean associated with an IQR increase in PM (p-value)** **lag** $PM_{2.5}$ mass: -0.987 (0.091) lag 0 Total carbon: -0.815 (0.041) lag 0 Elemental carbon: -0.657 (0.126) lag 0 Organic carbon: -0.942 (0.025) lag 0
Reference: Neuberger et al. (2004, 093249) **Period of Study:** Oct. 2000-May 2001 **Location:** Linz, Austria	**Outcome**: Forced oscillatory resistance (at zero Hz), FVC, FEV_1, MEF_{25}, MEF_{50}, MEF_{75}, PEF **Age Groups**: 7-10 years **Study Design:** Longitudinal prospective cohort **N:** 164 children **Statistical Analyses:** Mixed models linear regression with autoregressive correlation structure	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Monitoring Stations**: 1	**PM Increment:** 1 $\mu g/m^3$ **Notes:** Authors report increased oscillatory resistance significantly associated with $PM_{2.5}$ (lag 0)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
	Covariates: Sex, time and individual **Season:** October–May **Dose-response Investigated?** No **Statistical Package:** NR **Lags Considered:** Lag 0-7		
Reference: O'Connor et al. (2008, 156818) **Period of Study:** August 1998–July 2001 **Location:** Boston, the Bronx, Chicago, Dallas, New York, Seattle, Tucson	**Outcome:** Pulmonary function and respiratory symptoms **Age Groups:** 5-12 years **Study Design:** Inner-City Asthma Study (ICAS)–Panel/cohort study **N:** 861 children **Statistical Analyses:** Mixed effects models **Lags Considered:** Lag 0-6, 0-4	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-h **Mean (SD):** 14 **Range (Min, Max):** 5-35 (estimated from figure) **Copollutant (correlation):** NO_2 (r = 0.59) SO_2 (r = 0.37) CO (r = 0.44) O_3 (r = -0.02)	**PM Increment:** 13.2 $\mu g/m^3$ 90th-10th percentile **Change in pulmonary function** **lag** FEV_1: -1.47 (-2.00 to -0.94) lag 0-4 PEFR: -1.10 (-1.65 to -0.56) lag 0-4 **$PM_{2.5}$+O_3+NO_2** FEV_1: -0.73 (-1.33 to -0.12) lag 0-4 PEFR: -0.25 (-0.88, 0.38) lag 0-4 **Risk of Respiratory Symptoms** **lag** Wheeze: 0.98 (0.88, 1.09) lag 0-4 Nighttime asthma: 1.11 (0.94, 1.30) lag 0-4 Slow play: 1.01 (0.89, 1.15) lag 0-4 Missed school: 1.33 (1.06, 1.66) lag 0-4 **$PM_{2.5}$+O_3+NO_2** Wheeze: 0.92 (0.81, 1.05) lag 0-4 Nighttime asthma: 1.03 (0.86, 1.23) lag 0-4 Slow play: 0.92 (0.79, 1.06) lag 0-4 Missed school: 1.13 (0.87, 1.45) lag 0-4

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Peacock et al. (2003, 042026) **Period of Study:** November 1, 1996 to 14 February 1997 **Location:** northern Kent, UK	**Outcome:** Reduced peak expiratory flow rate (PEFR) **Age Groups:** 7-13 years of age **Study Design:** Time Series **N:** 179 **Statistical Analyses:** generalized estimating equations **Covariates:** Day of the week, 24-h mean outside temperature. **Season:** Winter **Dose-response Investigated?** No **Statistical Package:** STATA **Lags Considered:** Same day, lag 1, lag 2, five day moving avg	**Pollutant:** Sulfate (SO_4^{2-}) **Averaging Time:** Daily avg **Mean (SD):** Urban 2 24 h avg: 1.3 (1.1) **Percentiles:** 10th: Urban 2 0.5 90th: Urban 2 2.4 **Range (Min, Max):** Urban 2 0.3, 6.7 Unit (i.e. $\mu g/m^3$): $\mu g/m^3$ **Monitoring Stations:** 3	Sulfate (SO_4^{2-}) Increment: 1.3 $\mu g/m^3$ **Odds ratio [Lower CI, Upper CI]** **lag:** 1.090 [0.898, 1.322] 5 days
Reference: Peled, et al. (2005, 156015) **Period of Study:** 5-6 weeks between March-June 1999 and September-December 1999. **Location:** Ashdod, Ashkelon and Sderot, Israel	**Outcome:** Reduced peak expiratory flow (PEF) **Age Groups:** 7-10 years **Study Design:** Nested cohort study **N:** 285 **Statistical Analyses:** Time series analysis Generalized linear model, generalized estimating equations, one-way ANOVA, generalized linear model **Covariates:** Seasonal changes, meteorological conditions and personal physiological, clinical and socioeconomic measurements **Season:** Spring, Autumn **Dose-response Investigated?** No **Statistical Package:** STATA	**Pollutant:** $PM_{2.5}$ **Averaging Time:** Daily **Mean:** Ashkelon: 24.0 Sderot: 29.2 Ashdod: 23.9 **PM Component:** Local industrial emissions, desert dust, vehicle emissions and emissions from two electric power plants **Monitoring Stations:** 6 **Copollutant:** PM_{10}	**PM Increment:** 1 $\mu g/m^3$ β coefficient (SE) [95% CI] Ashkelon: $PM_{2.5}$ MAX: -0.144 (0.12) [-0.38-0.09] Ashdod: $PM_{2.5}$ MAX: -2.74 (0.61) [-3.95-1.53] $PM_{2.5}$ MAX x TMAX: 0.11 (0.02) [0.06-0.16] In Ashdod, $PM_{2.5}$ and an interaction between $PM_{2.5}$ and temperature were significantly associated.

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Penttinen et al. (2006, 087988)	**Outcome:** Decreased lung function and respiratory symptoms	**Pollutant:** $PM_{2.5}$	**PM Increment:** 1.3 $\mu g/m^3$
Period of Study: 11/1996–4/1997	**Age Groups:** Adults, mean age 53 years	**PM Component:** Soil, heavy fuel oil, sea salt	**$PM_{2.5}$, long range: PEF Morning:** 0.37[-0.59, 1.34]
Location: Helsinki, Finland	**Study Design:** Time Series	**Averaging Time:** 24 h	lag 0
	N: 78 people	**Percentiles: 25th:** Long range transport: 2.44	-1.04[-1.88 to -0.19]
	Statistical Analyses: Generalized least squares autoregressive model	Local combustion: 1.75	lag 1
	Covariates: Temperature, relative humidity, day of study, day of study squared, binary dummy variable for weekends	Soil: 0.14	-0.82[-1.81, 0.16]
	Season: Winter, Spring	Heavy fuel oil: -0.13	lag 2
	Dose-response Investigated? NR	Sea Salt: 0.22	0.22[-0.64, 1.08]
	Statistical Package: SAS version 6	Unidentifiable: -1.41	lag 3
	Lags Considered: 0-3	All sources: 6.47	-0.24[-1.12, 0.64]
		50th(Median): Long range transport: 4.15	5 day mean. **PEF Afternoon:** 0.20[-0.67, 1.06]
		Local combustion: 2.41	lag 0
		Soil: 0.64	-0.20[-1.24, 0.83]
		Heavy fuel oil: 0.10	lag 1
		Sea Salt: 0.27	-0.30[-1.14, 0.53]
		Unidentifiable: 0.02	lag 2
		All sources: 8.37	0.45[-0.57, 1.47]
		75th: Long range transport: 7.33	lag 3
		Local combustion: 3.05	0.03[-0.79, 0.85]
		Soil: 1.46	5 day mean. **PEF Evening:** -0.33[-1.30, 0.64]
		Heavy fuel oil: 0.52	lag 0
		Sea Salt: 0.42	-0.29[-1.13, 0.55]
		Unidentifiable: 0.74	lag 1
		All sources: 11.15	-0.41[-1.46, 0.64]
		Range (Min, Max): Long range transport: (-0.89, 28.31)	lag 2
		Local combustion: (0.83, 6.51)	0.39[-0.47, 1.24]
		Soil: (-1.13, 6.43)	lag 3
		Heavy fuel oil: (-0.67, 4.74)	0.07[-0.81, 0.95]
		Sea Salt: (0.09, 0.98)	5 day mean
		Unidentifiable: (-4.40, 4.77)	**$PM_{2.5}$, local combustion: PEF Morning:** -0.73[-1.69, 0.23]
		All sources: (4.11, 33.53)	lag 0
		Monitoring Stations: 1 site	-0.46[-1.24, 0.32]
			lag 1
			-0.43[-1.49, 0.63]
			lag 2
			0.34[-0.47, 1.15]
			lag 3
			-0.25[-1.03, 0.53]
			5 day mean. **PEF Afternoon:** -0.21[-1.07, 0.65]
			lag 0
			-0.81 [-1.77, 0.16]
			lag 1
			-0.83[-1.74, 0.09]
			lag 2
			0.20[-0.80, 1.20]
			lag 3
			-0.87[-1.63 to -0.12]
			5 day mean. **PEF Evening:** -0.51[-1.48,

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Pino et al. (2004, 050220) **Period of Study:** 4/1995–10/1996 **Location:** Santiago, Chile	**Outcome:** Respiratory Symptoms, Wheezing bronchitis **Study Design:** Time-series **Statistical Analyses:** Bayesian hierarchical analysis, cubic spline **Age Groups:** 4 months–2 years old	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-h avg **Mean (SD) unit:** 52.0 (31.6) **Range (5th, 95th):** 17.0, 114.0 **Copollutants (correlation):** SO_2: r = 0.73 NO_2: r = 0.85	**Increment:** 10 $\mu g/m^3$ **% Increase (Lower CI, Upper CI)** **lag:** % increase in wheezing bronchitis and $PM_{2.5}$ exposure for infants 4 months to 2 years old 4.75 (1.25, 8.25) 1 3.85 (0.45, 7.75) 2 2.25 (-1.00, 6.00) 3 1.75 (-2.20, 5.75) 4 4.00 (0.25, 8.00) 5 5.00 (1.00, 8.50) 6 7.00 (3.50, 11.00) 7 8.10 (4.00, 11.25) 8 9.00 (6.00, 12.00) 9 8.75 (5.75, 12.00) 10 1.50 (-3.50, 4.75) 11 0.25 (-3.75, 4.25) 12 0.00 (-4.00, 4.00) 13 1.00 (-3.50, 4.50) 14 1.50 (-3.50, 4.50) 15 OR for wheezing bronchitis and $PM_{2.5}$ exposure in infants 4 months to 2 years old according to family history of asthma Yes to family history of asthma 1.09 (1.00, 1.19) 1 1.10 (1.02, 1.20) 2 1.11 (1.02, 1.22) 3 No to family history of asthma

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			1.04 (1.00, 1.08) 1 1.02 (0.98, 1.06) 2 1.01 (0.96, 1.05) 3
Reference: Rabinovitch et al., (2006, 088031) **Period of Study:** 2001-2003 (two winters 2001-2002 and 2002-2003) **Location:** Denver, CO	**Outcome:** Bronchodilator doser activations (daily) and urinary leukotriene E4 (daily) **Age Groups:** Children 6-13 years old **Study Design: S**chool-based cohort study **N:** 73 children **Statistical Analyses:** Doser activation: Poisson regression with GEE with AR1 working covariance Urinary leukotriene E4: linear mixed model with spatial exponential covariance **Covariates:** Temperature, pressure, humidity, time trend, Friday indicator, upper respiratory infection (URI), height (leukotriene E4 only). **Season:** Winter **Dose-response Investigated?** NR **Statistical Package:** SAS **Lags Considered:** 0-2 days	**Pollutant:** $PM_{2.5}$ **Averaging Time:** Morning (midnight to 11: 00 AM) mean Morning (midnight to 11: 00 AM) maximum 24-h mean **Mean (SD):** 24-h mean, TEOM **Year 1, N: 55 days** 6.5 (3.2) **Year 2, N: 128 days** 8.2 (3.7) 24-h mean, FRM **Year 1, N: 55 days:** 11.8 (7.2) **Year 2, N: 122 days:** 11.2 (5.5) Morning mean, TEOM **Year 1, N: 71 days:** 7.4 (4.7) **Year 2, N: 127 days:** 9.1 (5.0) Morning maximum, TEOM **Year 1, N: 71 days:** 15.5 (9.5) **Year 2, N: 127 days:** 18.4 (9.6) **Percentiles:** 24-h mean, TEOM **Year 1** 25th: 4.4 50th(Median): 6.2 75th: 7.9 **Year 2** 25th: 55 50th(Median): 7.3 75th: 9.9 24-h mean, FRM **Year 1** 25th: 7.8 50th(Median): 10.1 75th: 14.1 **Year 2** 25th: 7.5 50th(Median): 9.3 75th: 13.3 Morning mean, TEOM **Year 1**	**PM Increment:** IQR (over current and previous day) **Doser Activation** **Morning avg $PM_{2.5}$ TEOM** Year 1: Pct Increase: 3.0 [-0.5: 6.6] p = 0.10 Year 2: Pct Increase: 2.7 [1.1: 4.4] p = 0.006 Aggregated years: 2.2 [0.7: 3.6] p = 0.005 **Morning max $PM_{2.5}$ TEOM** Year 1 Pct Increase: 4.0 [0.5: 7.6] p = 0.02 Year 2 Pct Increase: 2.3 [0.7: 4.0] p = 0.009 Aggregated years 2.6 [0.9: 4.2] p= 0.002 **24-h $PM_{2.5}$** **TEOM** Lag 0: 0.4 [-0.7: 1.6] p-value = 0.45 Lag 1: 0.9 [-0.7: 2.4] p-value = 0.27 Lag 2: -0.4 [-1.7: 0.9] p-value = 0.59 Lag 0-2 Avg: 0.6 [-1.0: 2.2] p-value = 0.43 **FRM** Lag 0: 0.2 [-1.2: 1.6] p-value = 0.81 Lag 1: 0.9 [-0.9: 2.6] p-value = 0.31 Lag 2: -0.2 [-2.2: 1.8] p-value = 0.88 Lag 0-2 Avg: 1.2 [-0.6: 2.9] p-value = 0.20 **Morning avg $PM_{2.5}$** **TEOM** URI not adjusted Mild/Moderate Asthmatics: 1.5 [-0.5: 3.4] p = 0.14 Severe Asthmatics: 3.7 [1.6: 5.8] p- = 0.0006 Difference between severity groups, p = 0.12 Aggregated severity group: 2.2 [0.7: 3.6] p= 0.005 URI adjusted Mild/Moderate Asthmatics: 1.0 [-1.9: 3.9]p= 0.50 Severe Asthmatics: 6.0 [1.8: 10.1] p =

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
		25th: 4.0	0.006
		50th(Median): 5.9	Difference between severity groups, p = 0.08
		75th: 9.6	Aggregated severity groups: 2.7 [-0.1: 5.4] p= 0.06
		Year 2 25th: 5.2	**Morning maximum $PM_{2.5}$**
		50th (Median): 8.5	**TEOM**
		75th: 11.6	URI not adjusted
		Morning maximum, TEOM	Mild/Moderate Asthmatics: 1.9 [-0.2: 4.1] p= 0.07
		Year 1 25th: 8	Severe Asthmatics: 3.9 [1.1: 6.8] p = 0.006
		50th (Median): 13	Difference between severity groups, p = 0.29
		75th: 20	Aggregated severity groups: 2.6 [0.9: 4.2] p= 0.002
		Year 2 25th: 11	URI adjusted
		50th (Median): 16	Mild/Moderate Asthmatics: 1.6 [-2.2: 5.4] p = 0.41
		75th: 23	Severe Asthmatics: 8.1 [2.9: 13.4] p = 0.003
		Range (Min, Max): 24-h mean, TEOM	Difference between severity groups, p = 0.03
		Year 1 (2.1, 23.7)	Aggregated severity groups: 3.8 [0.2: 7.4] p = 0.04
		Year 2 (1.7, 20.5)	**Leukotriene E4**
		24-h mean, FRM	**24-h $PM_{2.5}$**
		Year 1 (4.3, 53.5)	**TEOM**
		Year 2 (3.4, 26.3)	Lag 0: 3.3 [-0.7: 7.2] p = 0.09
		Morning mean, TEOM	Lag 1: -1.6[-5.7: 2.5] p = 0.40
		Year 1 (1.4, 22.7)	Lag 2: 1.1 [-2.8: 5.1] p= 0.64
		Year 2 (1.6, 30.2)	Lag 0-2 Avg: 2.3 [-4.0: 8.6] p = 0.45
		Morning maximum, TEOM	**FRM**
		Year 1 (4, 42)	Lag 0: 2.7 [1.1: 6.5] p = 0.12
		Year 2 (4, 46)	Lag 1: -0.8 [-4.9: 3.3] p = 0.65
		Monitoring Stations: 2 (1 TEOM and 1 Federal Reference Monitor [FRM])	Lag 2: -0.8 [-4.9: 3.3] p = 0.71
			Lag 0-2 Avg: 2.6 [-2.3: 7.5] p = 0.27
			Leukotriene E4
			Morning avg $PM_{2.5}$ TEOM
			Height 25%ile: 8.9 [3.0: 14.7] p= 0.004
			Height 50%ile: 5.9 [1.4: 10.4] p = 0.01
			Height 75%ile: 1.9 [-3.4: 7.3] p = 0.47
			Model w/o Height × **Pollutant:** 5.6 [1.0: 10.2] p = 0.02
			Morning maximum $PM_{2.5}$
			TEOM
			Height 25%ile: 8.3 [3.4: 13.2] p = 0.001
			Height 50%ile: 6.1 [2.1: 10.2] p= 0.004
			Height 75%ile: 3.2 [-2.0: 8.4] p= 0.23
			Model w/o Height × **Pollutant:** 6.2 [1.9:

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			10.5] p = 0.006
Reference: Rabinovitch et al. (2004, 096753) **Periods of Study:** 11/15/1999–3/15/2000 11/13/2000–3/23/2001 11/15/2001–3/22/2002 **Location:** Denver, Colorado	**Outcome:** Respiratory symptoms, Asthma symptoms (cough and wheeze), Upper respiratory symptoms **Study Design:** Time-series **Statistical Analyses:** Logistic linear regression, PROC Mixed, PROC Genmod **Age Groups:** 6-12	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-h avg **Mean (SD):** 10.8 (7.1) **Range (Min, Max):** (1.8, 53.5) **Copollutant (correlation):** CO NO_2 SO_2 O_3	**PM Increment:** 1 $\mu g/m^3$ β (SE) AM: -0.003 (0.009) PM: 0.004 (0.011) Odds Ratio (Lower CI, Upper CI) Lag 0.971 (0.843, 1.118) 0-3 avg.
Reference: Ranzi et al. (2004, 089500) **Period of Study:** February-May 1999 **Location:** Emilia-Romagmna, Italy (urban-industrial and rural area)	**Outcome:** respiratory symptoms, PEF measurements, drug consumption and daily activity **Age Groups:** Children, mean age = (7.2-7.9 yrs) **Study Design:** Panel study **N:** 120 children **Statistical Analyses:** Ecological analysis and Panel analysis **Covariates:** Temperature, humidity, gender, medicinal use, symptomatic status of previous day **Dose-response Investigated?** No **Statistical Package:** NR **Lags Considered:** 0, 1, 2, 3, 0-3 mov avg	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean (SD):** Urban = 53.07 Rural = 29.11 **Monitoring Stations:** 3 **Copollutant (correlation):** TSP: r = 0.613 daily air pollution concentrations: r = 0.658	**PM Increment:** 10 $\mu g/m^3$ Effect Estimate: Urban-industrial panel Cough and Phlegm: RR = 1.0044 (1.0011-1.0077)
Reference: Rodriguez et al. (2007, 092842) **Period of Study:** 1996-2003 **Location:** Perth, Australia	**Outcome:** Body temperature, cough, runny/ blocked nose, wheeze/ rattle chest (daily) **Age Groups:** Children 0-5 years old **Study Design:** hospital-based cohort study **N:** 198-263 children **Statistical Analyses:** Logistic regression with GEE and AR (order not specified) working covariance **Covariates:** temperature, humidity **Dose-response Investigated?** No **Statistical Package:** SAS **Lags Considered:** 0-5 days	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 1-h and 24-h **Mean (SD):** 1-h averaging, 20.767 24-h averaging, 8.534 **Range (Min, Max):** 1-h averaging (0.012: 93.433) 24-h averaging (0.004: 39.404) **Monitoring Stations:** 10 total, usually 3-5 sites for each pollutant **Copollutant (correlation):** O_3 NO + CO	**PM Increment:** NR [Lower CI, Upper CI] lag: NR LAG: 0 day **$PM_{2.5}$, 1-h** Body temperature: 1.004 [0.998: 1.011] Cough: 1.006 [1.000: 1.012] Wheeze/rattle chest: 1.004 [0.998: 1.010] Runny/blocked nose: 0.997 [0.983: 1.010] **$PM_{2.5}$, 24-h** Body temperature: 1.005 [0.986: 1.024] Cough: 1.019 [0.999: 1.040] Wheeze/rattle chest: 0.990 [0.969: 1.012] Runny/blocked nose: 0.968 [0.926: 1.013] LAG: 5 days **$PM_{2.5}$, 1-h** Body temperature: 1.005 [0.999: 1.040] Cough: 1.003 [0.995: 1.010] Wheeze/rattle chest: 1.005 [0.998:

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			10.12] Runny/blocked nose: 1.015 [1.000: 1.030] **$PM_{2.5}$, 24-h** Body temperature: 1.020 [0.998: 1.011] Cough: 1.006 [0.984: 1.011] Wheeze/rattle chest: 1.018 [0.997: 1.040] Runny/blocked nose: 1.039 [0.990: 1.089] LAG: 0-5 days **$PM_{2.5}$, 1-h** Body temperature: 1.000 [0.998: 1.002] Cough: 1.001 [0.999: 1.003] Wheeze/rattle chest: 1.002 [1.000: 1.004] Runny/blocked nose: 1.001 [0.997: 1.006] **$PM_{2.5}$, 24-h** Body temperature: 1.000 [0.994: 1.005] Cough: 1.004 [0.997: 1.011] Wheeze/rattle chest: 1.001 [0.995: 1.007] Runny/blocked nose: 0.998 [0.985: 1.011]
Reference: Sakai et al. (2004, 087435) **Period of Study:** November 14, 1999-March 28, 2001 **Location:** Diesel-powered ship from Tokyo, Japan to Showa Station on Ongul Island, Antarctica for 366 days (from February 1, 2000) and then heading back to Japan on February 1, 2001	**Outcome:** circulating leukocyte counts and serum inflammatory cytokine levels **Age Groups**: 24-57 yrs, mean = 36.1 ± 4.7 yrs **Study Design:** cohort **N:** 39 members of 41st Japanese Antarctic Research Expedition (JARE-41) **Statistical Analyses:** ANOVA **Covariates:** Smoking history, occupational pollutant exposure **Dose-response Investigated?** No **Statistical Package**: SPSS 11.5J	**Pollutant:** $PM_{5.0-2.0}$ **Averaging Time:** 24 h Unit (i.e. $\mu g/m^3$): particles/L PM Component: organic and inorganic substances, including microorganisms **Copollutant (correlation):** $PM_{2.0-0.3}$ $PM_{10-5.0}$	Effect Estimate: Multiple regression analysis between inhaled factors in Antarctica Total leukocyte Cigarette smoking = 0.211, $p < 0.001$ Support staff = 0.139, $p = 0.024$ Total PM = 0.168, $p = 0.004$ Segmented PMN Cigarette smoking = 0.015, $p = 0.805$ Support staff = 0.097, $p = 0.119$ Total PM = 0.272, $p < 0.001$ Band-formed PMN Cigarette smoking = 0.035, $p = 0.543$ Support staff = 0.010, $p = 0.864$ Total PM = 0.470, $p < 0.001$ Monocyte Cigarette smoking = 0.081, $p = 0.187$ Support staff = -0.019, $p = 0.759$ Total PM = 0.328, $p < 0.001$ G-CSF Cigarette smoking = 0.131, $p < 0.038$ Support staff = 0.176, $p = 0.005$ Total PM = 0.078, $p = 0.186$ IL-6

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			Cigarette smoking = 0.182, p = 0.004 Support staff = 0.076, p = 0.228 Total PM = 0.158, p = 0.008
Reference: Sakai et al. (2004, 087435) **Period of Study:** November 14, 1999-March 28, 2001 **Location:** Diesel-powered ship from Tokyo, Japan to Showa Station on Ongul Island, Antarctica for 366 days (from February 1, 2000) and then heading back to Japan on February 1, 2001	**Outcome:** circulating leukocyte counts and serum inflammatory cytokine levels **Age Groups:** 24-57 yrs, mean = 36.1 ± 4.7 yrs **Study Design:** cohort **N:** 39 members of 41st Japanese Antarctic Research Expedition (JARE-41) **Statistical Analyses:** ANOVA **Covariates:** Smoking history, occupational pollutant exposure **Dose-response Investigated?** No **Statistical Package:** SPSS 11.5J	**Pollutant:** $PM_{10-5.0}$ **Averaging Time:** 24-h Unit (i.e. $\mu g/m^3$): particles/L **Monitoring Stations:** NR **Copollutant (correlation):** $PM_{2.0-0.3}$ $PM_{10-5.0}$	Effect Estimate: Multiple regression analysis between inhaled factors in Antarctica Total leukocyte Cigarette smoking = 0.211, p < 0.001 Support staff = 0.139, p = 0.024 Total PM = 0.168, p = 0.004 Segmented PMN Cigarette smoking = 0.015, p = 0.805 Support staff = 0.097, p = 0.119 Total PM = 0.272, p < 0.001 Band-formed PMN Cigarette smoking = 0.035, p = 0.543 Support staff = 0.010, p = 0.864 Total PM = 0.470, p < 0.001 Monocyte Cigarette smoking = 0.081, p = 0.187 Support staff = -0.019, p = 0.759 Total PM = 0.328, p < 0.001 G-CSF Cigarette smoking = 0.131, p < 0.038 Support staff = 0.176, p = 0.005 Total PM = 0.078, p = 0.186 IL-6 Cigarette smoking = 0.182, p = 0.004 Support staff = 0.076, p = 0.228 Total PM = 0.158, p = 0.008
Reference: Silkoff et al. (2005, 087471) **Period of Study:** Winter 1999-2000, Winter 2000-2001 **Location:** Denver, CO	**Outcome:** Lung function: FEV_1, PEF **Age Groups:** Adults (> 40 years-old) with COPD, as well as > 10 pack-years tobacco use, FEV_1 < 70%, FEV_1/FVC < 60%, and no other lung disease **Study Design:** COPD patient panel study (2 independent panels one for each winter) **N:** 34 subjects (16 1st winter, 18 second winter) **Statistical Analyses:** mixed effects models with first-order, autoregressive, moving avg variance-covariance binary outcomes (rescue medication use, total symptom score) assessed using Poisson regression with GEE and first-order, auto-regressive variance-covariance **Covariates:** temperature, relative humidity, barometric pressure	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-h **Mean (SD):** Winter 1999-2000: 9.0 (5.2) Winter 2000-2001: 14.3 (9.6) **Percentiles:** Winter 1999-2000 25th 5.4 50th(Median): 7.7 75th: 11.3 Winter 2000-2001 25th 7.6 50th(Median): 11.7 75th: 17.2 **Range (Min, Max):** Winter 1999-2000	**PM Increment:** SD Winter 1999-2000: 5.2 Winter 2000-2001: 9.6 Model results reported graphically only. No quantitative results reported. Direction of slope (+/-) and statistical significance (SIG: yes NS: no) inferred from graphs. Among subjects with severe COPD observed in Winter 1999-2000, statistically significant, but marginal, improvements in PEF associated with morning lag 0 $PM_{2.5}$. There were no statistically significant associations between rescue medication use and symptom score with PM.

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
	analysis run separately for each winter **Season:** Winter **Dose-response Investigated?** No **Statistical Package:** SAS **Lags Considered:** 0-2 days	(1.8, 36.6) Winter 2000-2001 (3.4, 59.6) **Monitoring Stations:** multiple sites **Copollutant (correlation):** CO NO_2 PM_{10}	
Reference: Sivacoumar et al. (2006, 111115) **Period of Study:** 4/1998–5/1998 9/1998–10/1998 **Location:** Pammal, India	**Outcome:** Respiratory symptoms, Decreased pulmonary function **Study Design:** Case-control **Statistical Analyses:** Poisson **Age Groups:** > 18	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-h avg	The study does not present quantitative results of association.
Reference: Slaughter et al. (2003, 086294) **Period of Study:** 1994 **Location:** Seattle, WA	**Outcome:** Asthma attacks, asthma severity, medication use **Age Groups:** 5.1 to 13.1 years old **Study Design:** Cross-sectional study **N:** 133 children **Statistical Analyses:** Ordinal Logistic Regression Poisson Modeling **Covariates:** Temperature, Day of the Week, Seasonality **Dose-response Investigated?** No **Statistical Package:** STATA **Lags Considered:** 1, 2, 3 day lag	**Pollutant:** $PM_{2.5}$ **Averaging Time:** Daily Averages 25th: 5.0 50th(Median): 7.3 [3] 75th: 11.3 **Monitoring Stations:** 3 **Copollutant (correlation):** PM_{10} = 0.75 CO = 0.82	**PM Increment:** 10 $\mu g/m^3$ increase RR Estimate [Lower CI, Upper CI] lag: Inhaler use: 1-day lag: 1.04 (0.98, 1.10) OR Estimate [Lower CI, Upper CI] lag: Asthma Attack: 1-day lag: 1.20 (1.05, 1.37) Previous day: 1.13 (1.03, 1.23) Medication Use Nontransition model: Previous Day: 1.08 (1.01, 1.15) **Notes:** Figures of estimated odds ratios for having a more serious asthma attack for short-term, within-subject increases in $PM_{2.5}$, PM_{10}, and CO. Transition models additionally control for the previous day's severity. Figures of estimated relative risks for having inhaler use for short-term, within-subject increases in $PM_{2.5}$, PM_{10}, and CO. Transition models additionally control for the previous day's severity.
Reference: Strand et al (2006, 089203) **Period of Study:** 2002-2004 **Location:** Denver, Colorado, United States	**Outcome:** Reduced forced expiratory volume (FEV_1) **Age Groups:** 6-12 years old **Study Design:** Mixed model analysis (using the default restricted maximum likelihood (REML) estimators) **N:** 50 children **Statistical Analyses:** least squares regression, SAS "Output Delivery System" (ODS) **Season:** Autumn and Winter **Dose-response Investigated?** Yes **Statistical Package:** SAS	**Pollutant:** $PM_{2.5}$ **Averaging Time:** daily **Mean (SD):** Outdoor: 12.699 (6.426) Indoor: 8.148 (4.348) Sulfate/$PM_{2.5}$/outdoor: 0.079 (0.067) Sulfate/$PM_{2.5}$/indoor: 0.074 (0.060) **Range (Min, Max):** Mean Personal: (0, 3.035) Outdoor: (0, 6.303) Indoor: (0, 2.759) PM Component: elemental carbon, sulfate, nitrate and ETS.	**PM Increment:** 10 $\mu g/m^3$ Effects Estimate: Using the estimated slope for the validation study model [Lower CI, Upper CI] lag: 2.2 percent decrease in FEV_1 per 10 $\mu g/m^3$ increase in ambient $PM_{2.5}$ [0.0, 4.3 decrease] 1 day

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
		Monitoring Stations: 2 fixed monitors and up to 10 personal monitors on a given day. **Copollutant (correlation):** Sulfate (0.63)	
Reference: Tang et al. (2007, 091269) **Period of Study:** Dec 2003 to Feb 2005 **Location:** Sin-Chung City, Taipei County, Taiwan	**Outcome:** Peak expiratory flow rate (PEFR) of asthmatic children **Age Groups:** 6–12 years **Study Design:** Panel study **N:** 30 children **Statistical Analyses:** Linear mixed-effect models were used to estimate the effect of PM exposure on PEFR **Covariates:** Gender, age, BMI, history of respiratory or atopic disease in family, SHS, acute asthmatic exacerbation in past 12 months, ambient temp and relative humidity, presence of indoor pollutants, and presence of outdoor pollutants, **Dose-response Investigated?** yes **Statistical Package:** S-Plus 2000 **Lags Considered:** 0-2	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 1 h **Mean (SD):** Personal: 27.8 (25.3) **Range (Min, Max):** Personal: 1.4–263.4 **Monitoring Stations:** 1	**PM Increment:** 24.5 $\mu g/m^3$ RR Estimate [Lower CI, Upper CI] lag: Change in morning PEFR: -6.00 (-29.85, 17.85) lag 0 -12.52 (-77.93, 52.9) lag 1 -24.87 (-71.49, 21.74) lag 2 -45.67 (-117.09, 25.74) 2-day mean -5.69 (-105.96, 94.59) 3-day mean Change in evening PEFR: 0.50 (-18.82, 19.82) lag 0 16.66 (-7.59, 40.9) lag 1 11.60 (-11.1, 34.31) lag 2 39.97 (7.1, 72.85) 2-day mean -3.32 (-66.14, 59.5) 3-day mean
Reference: Timonen et al. (2004, 087915) **Period of Study:** Oct 1998 to April 1999 **Location:** Amsterdam, Netherlands Erfurt, Germany Helsinki, Finland	**Outcome:** Urinary concentration of Clara cell protein CC16 of subjects with coronary heart disease **Age Groups:** 50+ **Study Design:** Longitudinal cohort study (panel) **N:** 37 (Amsterdam) 47 (Erfurt) 47 (Helsinki) **Statistical Analyses:** The response of interest was log transformed, creatinine adjusted CC16. Mixed-effect model was used to investigate the association between CC16 and air pollutants. **Covariates:** Subjects, long term time trend, temperature (lags 0-3), relative humidity (lags 0-3), barometric pressure (lags 0-3), and weekday of visit. **Dose-response Investigated?** yes **Statistical Package:** S-Plus and SAS **Lags Considered:** 0-3	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean (SD):** Amsterdam: 20.0 $\mu g/m^3$ Erfurt: 23.1 $\mu g/m^3$ Helsinki: 12.7 $\mu g/m^3$ **Range (Min, Max):** Amsterdam: 3.8–82.2 Erfurt: 4.5–118.1 Helsinki: 3.1–39.8 **Monitoring Stations:** 3 **Copollutant (correlation):** Spearman Correlation: $NC_{0.01-0.1}$: Amsterdam -0.15 Erfurt 0.62 Helsinki 0.14 $NC_{0.1-1.0}$: Amsterdam 0.80 Erfurt 0.84 Helsinki 0.80 NO_2: Amsterdam 0.49 Erfurt 0.82 Helsinki 0.35 CO: Amsterdam 0.58 Erfurt 0.77 Helsinki 0.40	**PM Increment:** 10 $\mu g/m^3$ RR Estimate [Lower CI, Upper CI] lag: Pooled estimate; 2.8 (-1.1–6.7) lag 0 2.9 (-0.6–6.5) lag 1 5.0 (-2.4–12.4) lag 2 1.6 (-4.7–7.9) lag 3 9.7 (-6.0–25.4) 5-day mean CC16 was not associated to $PM_{2.5}$ in the pooled analysis but CC16 was significantly associated to $PM_{2.5}$ in Helsinki: 23.3 (6.3–40.3) lag 0 6.4 (-8.2–21.1) lag 1 20.2 (6.9–33.5) lag 2 17.6 (4.3–30.9) lag 3 38.8 (15.8–61.8) 5-day mean
Reference: Trenga et al. (2006, 155209) **Period of Study:** 1999-2002	**Outcome:** Lung function: FEV_1, PEF, MMEF (maximal midexpiratory flow	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-h	**PM Increment:** 10 $\mu g/m^3$ **ADULT Personal $PM_{2.5}$ - FEV_1**

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Location: Seattle, WA	assessed only for children) **Age Groups:** Adults (56-89-years-old) healthy & with COPD asthmatic children 6-13-years-old **Study Design:** adult and pediatric panel study over three years with 1 monitoring period ("session") per year **N:** 57 adults (33 healthy, 24 with COPD) = 692 subject-days = 207 study-days 17 asthmatic children = 319 subject-days = 98 study-days **Statistical Analyses:** mixed effects, longitudinal regression models, with the effects of pollutant decomposed into each subject's a) overall mean b) difference between their session-specific mean and overall mean c) difference between their daily values and session-specific mean **Covariates:** gender, age, ventral site temperature and relative humidity, CO, NO_2 **Dose-response Investigated?** No **Statistical Package:** SAS **Lags Considered:** 0-1 days	Percentiles: Children, Personal 25th: 8.1 50th(Median): 11.3 75th: 16.3 Indoor 25th: 5.7 50th(Median): 7.5 75th: 10.2 Local outdoor 25th: 6.4 50th(Median): 9.6 75th: 14.8 Adults, Personal 25th: 5.9 50th(Median): 8.5 75th: 12.4 Indoor 25th: 5.1 50th(Median): 7.6 75th: 10.8 Local outdoor 25th: 6 50th(Median): 8.6 75th: 13.1 **Range (Min, Max):** Children, Personal 1.0, 49.4 Indoor (2.2, 36.3) Local outdoor (2.8, 40.4) Adults, Personal 1.3, 66.6 Indoor(1.6, 65.3) Local outdoor (0.0, 41.5) **Monitoring Stations:** 2 also subject-specific local outdoors (i.e., at each home), indoor, and personal **Copollutant (correlation):** CO NO_2 $PM_{2.5}$ $PM_{10-2.5}$ (coarse)	Overall: Lag 0 -6.0 [-29.1: 17.2] Lag 1 12.0 [-12.9: 36.9] No-COPD: Lag 0 -4.6 [-31.0: 21.9] Lag 1 19.3 [-8.2: 46.7] COPD: Lag 0 -10.2 [-55.8: 35.4] Lag 1 -19.0 [-74.1: 36.2] PEF: Lag 0 1.5 [-2.2: 5.2] Lag 1 2.1 [-1.9: 6.1] No-COPD: Lag 0 3.4 [-0.9: 7.6] Lag 1 1.9 [-2.5: 6.3] COPD: Lag 0 -4.3 [-11.5: 3.0] Lag 1 2.6 [-6.3: 11.5] **Indoor $PM_{2.5}$ - FEV_1** Overall: Lag 0 -12.8 [-44.5: 19.0] Lag 1 19.4 [-11.3: 50.1] No-COPD: Lag 0 -15.8 [-50.0: 18.4] Lag 1 28.4 [-4.6: 61.3] COPD: Lag 0 2.6 [-71.7: 76.8] Lag 1 -29.7 [-102.9: 43.5] **PEF** Overall: Lag 0 -0.5 [-5.6: 4.6] Lag 1 2.3 [-3.3: 7.8] No-COPD: Lag 0 0.1 [-5.4: 5.6] Lag 1 2.5 [-3.5: 8.4] COPD: Lag 0 -3.2 [-15.1: 8.7] Lag 1 1.1 [-12.0: 14.3] **Outdoor Home $PM_{2.5}$ - FEV_1** Overall: Lag 0 -1.4 [-35.6: 32.7] Lag 1 -2.4 [-37.6: 32.7]. No-COPD: Lag 0 1.5 [-36.1: 39.2] Lag 1 10.7 [-26.9: 48.4] COPD: Lag 0 -8.9 [-62.2: 44.4] Lag 1 -45.2 [-102.6: 12.1] **PEF** Overall: Lag 0 2.3 [-3.3: 7.9] Lag 1 0.4 [-5.6: 6.4] No-COPD: Lag 0 4.0 [-2.2: 10.1] Lag 1 2.0 [-4.4: 8.4] COPD: Lag 0 -1.8 [-10.6: 6.9] Lag 1 -4.8 [-14.6: 4.9] **Central Sites $PM_{2.5}$ - FEV_1** Overall: Lag 0 -35.5 [-70.0: -1.0] Lag 1 -40.4 [-71.1: -9.6]. No-COPD: Lag 0 -32.6 [-69.5: 4.3] Lag 1 -29.0 [-62.5: 4.5] COPD: Lag 0 -43.6 [-95.0: 7.8] Lag 1 -70.8 [-118.4: 23.1] **PEF** Overall: Lag 0 1.5 [-4.2: 7.1] Lag 1 -2.3 [-7.4: 2.9]

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			No-COPD: Lag 0 2.5 [-3.5: 8.6]
			Lag 1 -0.5 [-6.1: 5.0]
			COPD: Lag 0 -1.5 [-9.9: 6.9]
			Lag 1 -7.1 [-15.0: 0.9]
			PEDIATRIC FEV_1 Personal $PM_{2.5}$ Overall: Lag 0 -13.08 [-38.26: 12.10]
			Lag 1 -16.12 [-42.61: 10.37]. No Anti-inflam. Medication: Lag 0 -41.73 [-94.31: 10.84]
			Lag 1 -30.99 [-82.17: 20.19]. Anti-inflam. Medication: Lag 0 -4.61 [-34.49: 25.28]
			Lag 1 -10.87 [-45.01: 23.27]
			Indoor $PM_{2.5}$ Overall: Lag 0 -45.90 [-89.92: 1.88]
			Lag 1 -64.78 [-111.27: 18.28]
			No Anti-inflam. Medication: Lag 0 -75.92 [-145.16: 6.67]
			Lag 1 -65.08 [-136.98: 6.82]. Anti-inflam. Medication: Lag 0 -28.50 [-94.72: 37.71]
			Lag 1 -64.60 -147.23: 18.04]
			Outdoor Home $PM_{2.5}$ Overall: Lag 0 -13.11 [-57.41: 31.19]
			Lag 1 -9.37 [-54.73: 36.00]. No Anti-inflam. Medication: Lag 0 -24.42 [-81.22: 32.38]
			Lag 1 16.52 [-45.76: 78.80]. Anti-inflam. Medication: Lag 0 -3.59 [-75.88: 68.70]
			Lag 1-26.76 [-89.53: 36.01]
			Central Sites $PM_{2.5}$. Overall: Lag 0 -12.32 [-53.21: 28.56]
			Lag 1 5.75 [-33.27: 44.76]. No Anti-inflam. Medication: Lag 0 -33.59 [-89.99: 22.82]
			Lag 1 31.30 [-29.91: 92.51]Anti-inflam. Medication: Lag 0 -2.13 [-71.99: 67.73]
			Lag 1 -3.53 [-67.32: 60.27]
			PEF: Personal $PM_{2.5}$ Overall: Lag 0 0.31 [-4.02: 4.64]
			Lag 1 -2.19 [-6.49: 2.12]
			No Anti-inflam. Medication: Lag 0 0.22 [-8.85: 9.29]
			Lag 1 -10.48 [-18.68: 2.28]
			Anti-inflam. Medication: Lag 0 0.34 [-4.67: 5.35]
			Lag 1 0.74 [-4.21: 5.69]
			Indoor $PM_{2.5}$ Overall: Lag 0 -8.68 [-16.64: -0.72]
			Lag 1 -9.22 [-17.51: -0.93]
			No Anti-inflam. Medication: Lag 0 -13.34 [-25.90: -0.79]
			Lag 1 -17.13 [-29.86: 4.41]. Anti-inflam. Medication: Lag 0 -5.98 [-15.85: 3.89]
			Lag 1 -4.19 [-14.59: 6.20]
			Outdoor Home $PM_{2.5}$ Overall: Lag 0 -

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			6.27 [-14.07: 1.53] Lag 1 -5.64 [-13.73: 2.44]. No Anti-inflam. Medication: Lag 0 -7.52 [-17.56: 2.51] Lag 1 -6.92 [-18.03: 4.19]. Anti-inflam. Medication: Lag 0 -5.22 [-14.77: 4.34] Lag 1 -4.78 [-14.42: 4.86] **Central Sites $PM_{2.5}$** Overall: Lag 0 -5.62 [-12.86: 1.62] Lag 1 -2.45 [-9.34: 4.43]. No Anti-inflam. Medication: Lag 0 -6.32 [-16.31: 3.68] Lag 1 -0.83 [-11.60: 9.95] Anti-inflam. Medication: Lag 0 -5.29 [-13.42: 2.85] Lag 1 -3.04 [-10.76: 4.67] **MMEF - Personal $PM_{2.5}$** Overall: Lag 0 -0.99 [-3.96: 1.98] Lag 1 -1.08 [-4.05: 1.88]. No Anti-inflam. Medication: Lag 0 -3.32 [-9.52: 2.88] Lag 1 -2.49 [-8.23: 3.25]. Anti-inflam. Medication: Lag 0 -0.31 [-3.77: 3.16] Lag 1 -0.59 [-4.06: 2.89] **Indoor $PM_{2.5}$** Overall: Lag 0 -3.29 [-8.52: 1.94] Lag 1 -11.08 [-16.26: 5.90]. No Anti-inflam. Medication: Lag 0-12.65 [-20.74: -4.56]' Lag 1 -13.84 [-21.82: 5.85]/. Anti-inflam. Medication: Lag 0 2.14 [-4.17: 8.45] Lag 1 -9.33 [-15.89: -2.78] **Outdoor Home $PM_{2.5}$** Overall: Lag 0 -4.13 [-9.28: 1.01] Lag 1 -0.73 [-6.02: 4.56] No Anti-inflam. Medication: Lag 0 -8.23 [-14.77: 1.69] Lag 1 -1.19 [-8.45: 6.07] Anti-inflam. Medication: Lag 0 -0.68 [-6.87: 5.50] Lag 1 -0.42 [-6.72: 5.87] **Central Sites $PM_{2.5}$.** Overall: Lag 0 -2.10 [-6.99: 2.79] Lag 1 -0.12 [-4.67: 4.42] No Anti-inflam. Medication: Lag 0 -8.21 [-14.79: 1.62] Lag 1 -0.22 [-7.34: 6.90] Anti-inflam. Medication: Lag 0 0.82 [-4.48: 6.12]. Lag 1 -0.09 [-5.19: 5.01]
Reference: Tang et al. (2007, 091269) **Period of Study:** Dec 2003 to Feb 2005 **Location:** Sin-Chung City, Taipei County, Taiwan	**Outcome:** Peak expiratory flow rate (PEFR) of asthmatic children **Age Groups:** 6–12 years **Study Design:** Panel study **N:** 30 children	**Pollutant:** $PM_{2.5-1}$ **Averaging Time:** 1 h **Mean (SD):** Personal: 6.2 (4.8)	No quantitative effects reported.

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
	Statistical Analyses: Linear mixed-effect models were used to estimate the effect of PM exposure on PEFR **Covariates:** Gender, age, BMI, history of respiratory or atopic disease in family, SHS, acute asthmatic exacerbation in past 12 months, ambient temp and relative humidity, presence of indoor pollutants, and presence of outdoor pollutants, **Dose-response Investigated?** yes **Statistical Package**: S-Plus 2000 **Lags Considered:** 0-2	**Range (Min, Max):** Personal: 0.3–86.8 **Monitoring Stations**: 1	
Reference: Tang et al. (2007, 091269) **Period of Study:** Dec 2003 to Feb 2005 **Location:** Sin-Chung City, Taipei County, Taiwan	**Outcome:** Peak expiratory flow rate (PEFR) of asthmatic children **Age Groups**: 6–12 years **Study Design:** Panel study **N:** 30 children **Statistical Analyses:** Linear mixed-effect models were used to estimate the effect of PM exposure on PEFR **Covariates:** Gender, age, BMI, history of respiratory or atopic disease in family, SHS, acute asthmatic exacerbation in past 12 months, ambient temp and relative humidity, presence of indoor pollutants, and presence of outdoor pollutants, **Dose-response Investigated?** yes **Statistical Package**: S-Plus 2000 **Lags Considered:** 0-2	**Pollutant:** PM_1 **Averaging Time:** 1 h **Mean (SD):** Personal: 34.0 (28.9) Ambient: 31.4 (18.8) **Range (Min, Max):** Personal: 1.8–284.6 Ambient: 0.1–128.4 Unit (i.e. $\mu g/m^3$): $\mu g/m^3$ **Monitoring Stations**: 1	**PM Increment:** 27.6 $\mu g/m^3$ RR Estimate [Lower CI, Upper CI] lag: Change in morning PEFR: -6.44 (-30.18, 17.29) lag 0 -12.26 (-77.6 , 53.09) lag 1 -4.38 (-54.79, 46.03) lag 2 -44.06 (-113.79, 25.67) 2-day mean -6.01 (-101.48, 89.46) 3-day mean Change in evening PEFR: 1.17 (-17.79, 20.13) lag 0 -4.98 (-27.77, 17.81) lag 1 11.30 (-11.55, 34.16) lag 2 41.74 (11.36, 72.13) 2-day mean 28.21 (-19.08, 75.5) 3-day mean
Reference: Timonen et al. (2004, 087915) **Period of Study:** Oct 1998 to April 1999 **Location:** Amsterdam, Netherlands Erfurt, Germany Helsinki, Finland	**Outcome:** Urinary concentration of Clara cell protein CC16 of subjects with coronary heart disease **Age Groups**: 50+ **Study Design:** Longitudinal cohort study (panel) **N:** N=37 (Amsterdam) N=47 (Erfurt) N=47 (Helsinki) **Statistical Analyses:** The response of interest was log transformed, creatinine adjusted CC16. Mixed-effect model was used to investigate the association between CC16 and air pollutants. **Covariates:** Subjects, long term time trend, temperature (lags 0-3), relative humidity (lags 0-3), barometric pressure (lags 0-3), and weekday of visit. **Dose-response Investigated?** yes **Statistical Package**: S-Plus and SAS **Lags Considered:** 0-3	**Pollutant:** $NC_{0.01-0.1}$ **Averaging Time:** 24 h **Mean (SD):** Amsterdam: 17338 /cm^3 Erfurt: 21124 /cm^3 Helsinki: 17041 /cm^3 **Range (Min, Max):** Amsterdam: 5699-37195 Erfurt: 3867-96678 Helsinki: 2305-50306 Unit (i.e. $\mu g/m^3$): 1/cm^3 **Monitoring Stations**: 3 $PM_{2.5}$: Amsterdam -0.15 Erfurt 0.62 Helsinki 0.14 NO_2: Amsterdam 0.49 Erfurt 0.82	**PM Increment:** 10,000 /cm^3 RR Estimate [Lower CI, Upper CI] lag: Pooled estimate; 1.7 (-4.4–7.8) lag 0 -1.8 (-8.3–4.6) lag 1 1.5 (-5.6–8.6) lag 2 2.3 (-4.8–9.3) lag 3 1.8 (-9.4–13.0) 5-day mean There was no association between $NC_{0.01-0.1}$ and CC16 in the pooled analysis.

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
		Helsinki 0.72 CO: Amsterdam 0.22 Erfurt 0.72 Helsinki 0.35	
Reference: Timonen et al. (2004, 087915) **Period of Study:** Oct 1998 to April 1999 **Location:** Amsterdam, Netherlands Erfurt, Germany Helsinki, Finland	**Outcome:** Urinary concentration of Clara cell protein CC16 of subjects with coronary heart disease **Age Groups**: 50+ **Study Design:** Longitudinal cohort study (panel) **N:** N=37 (Amsterdam) N=47 (Erfurt) N=47 (Helsinki) **Statistical Analyses:** The response of interest was log transformed, creatinine adjusted CC16. Mixed-effect model was used to investigate the association between CC16 and air pollutants. **Covariates:** Subjects, long term time trend, temperature (lags 0-3), relative humidity (lags 0-3), barometric pressure (lags 0-3), and weekday of visit. **Dose-response Investigated?** yes **Statistical Package**: S-Plus and SAS **Lags Considered:** 0-3	**Pollutant:** $NC_{10-0.1}$ **Averaging Time:** 24 h **Mean (SD):** Amsterdam: 2131 /cm^3 Erfurt: 1829 /cm^3 Helsinki: 1390 /cm^3 **Range (Min, Max):** Amsterdam: 413-6413 Erfurt: 303-6848 Helsinki: 344-3782 Unit (i.e. $\mu g/m^3$): 1/cm^3 **Monitoring Stations**: 3 **Copollutant (correlation):** Spearman Correlation: $NC_{0.1-0.01}$: Amsterdam 0.16 Erfurt 0.67 Helsinki 0.53 $PM_{2.5}$: Amsterdam 0.80 Erfurt 0.84 Helsinki 0.80 NO_2: Amsterdam 0.67 Erfurt 0.82 Helsinki 0.72 CO: Amsterdam 0.60 Erfurt 0.78 Helsinki 0.51	**PM Increment:** 1000 /cm^3 RR Estimate [Lower CI, Upper CI] lag: Pooled estimate; 4.3 (-1.4–10.0) lag 0 5.1 (-0.6–10.7) lag 1 4.5 (-0.5–9.6) lag 2 1.6 (-3.5–6.7) lag 3 13.1 (-4.3–30.5) 5-day mean CC16 was not associated to NC 0.1-1.0 in the pooled analysis but CC16 was significantly associated to NC 0.1-1.0 in Helsinki: 15.5 (0.001–30.9) lag 0 10.8 (-4.2–25.8) lag 1 10.5 9-4.1–25.1) lag 2 17.4 (3.4–31.4) lag 3 43.2 (17.4–69.0) 5-day mean
Reference: von Klot et al. (2002, 034706) **Period of Study:** September 1996 to March 1997 (winter) **Location:** Erfurt, Germany	**Outcome:** Asthma symptoms (wheezing, shortness of breath at rest, waking up with breathing problems, or coughing without having a cold) and Asthma medication (inhaled short-acting ß2-agonists, inhaled long-acting ß2- agonists, inhaled corticosteroids, cromolyn sodium, theophylline, oral corticosteroids, and N-acetylcysteine) **Age Groups**: Adults, mean=59.0 yrs and range =37-77 yrs **Study Design:** panel study **N:** 53 adult asthmatics **Statistical Analyses:** Logistic regression	**Pollutant:** $MC_{0.5-0.1}$ **Averaging Time:** 10 min intervals **Mean (SD):** 24.8 Percentiles: 25th: 11.4 50th(Median): 19.6 75th: 33.1 **Range (Min, Max):** (2.4-108.3) **Copollutant (correlation):** $PM_{10-2.5}$: r= 0.51	NC Increment: 1 IQR Effect Estimate [Lower CI, Upper CI]: Association between the prevalence of inhaled ß2- agonist use and MC0.1-0.5 Same day, IQR= 21, OR= 0.98 (0.92-1.04) 5-day mean, IQR= 21 OR= 1.11 (1.02-1.20) 14-day mean IQR= 17, OR= 1.01 (0.93-1.10) Association between the prevalence of inhaled corticosteroid use and MC0.1-0.5

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
	models **Covariates:** seasonal variation in medication use or symptom prevalence, meteorological factors (relative humidity, temperature), weekend, Christmas holidays **Season:** winter **Dose-response Investigated?** No **Statistical Package**: NR **Lags Considered:** 0, 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, mov avg calculated from same day and preceding days	$NC_{0.1-0.01}$: r = 0.45 $NC_{0.5-0.1}$: r = 0.95 $NC_{2.5-0.5}$: r = 0.92 $MC_{2.5-0.01}$: r = 1.00 PM_{10}: r = 0.91 NO_2: r = 0.69 CO: r = 0.66 SO_2: r = 0.60	Same day, IQR = 2, OR = 1.09 (1.02-1.17) 5-day mean IQR = 21, OR = 1.28 (1.18-1.39) 14-day mean, IQR = 17, OR = 1.49 (1.38-1.61) Association between the prevalence of wheezing and MC0.1-0.5 Same day, IQR = 21, OR = 1.01 (0.94-1.08) 5-day mean, IQR = 21, OR = 1.08 (0.99-1.17) 14-day mean, IQR = 17, OR = 1.05 (0.96-1.15)
Reference: von Klot et al. (2002, 034706) **Period of Study:** September 1996 to March 1997 (winter) **Location:** Erfurt, Germany	**Outcome:** Asthma symptoms (wheezing, shortness of breath at rest, waking up with breathing problems, or coughing without having a cold) and Asthma medication (inhaled short-acting ß2-agonists, inhaled long-acting ß2- agonists, inhaled corticosteroids, cromolyn sodium, theophylline, oral corticosteroids, and N-acetylcysteine) **Age Groups**: Adults, mean = 59.0 yrs and range = 37-77 yrs **Study Design:** panel study **N:** 53 adult asthmatics **Statistical Analyses:** Logistic regression models **Covariates:** seasonal variation in medication use or symptom prevalence, meteorological factors (relative humidity, temperature), weekend, Christmas holidays **Season:** Winter **Dose-response Investigated?** No **Statistical Package**: NR **Lags Considered:** 0, 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, mov avg calculated from same day and preceding days	**Pollutant:** $MC_{2.5-0.01}$ **Averaging Time:** 10 min intervals **Mean (SD):** 30.3 Percentiles: 25th: 13.5 50th(Median): 24.6 75th: 41.3 **Range (Min, Max):** (3.6-133.8) **Copollutant (correlation):** $PM_{10-2.5}$: r = 0.52 $NC_{0.5-0.1}$: r = 0.45 $NC_{2.5-0.5}$: r = 0.94 $MC_{0.5-0.1}$: r = 1.00 $NC_{0.1-0.01}$: r = 0.45 PM_{10}: r = 0.94 NO_2: r = 0.68 CO: r = 0.65 SO_2: r = 0.62	NC Increment: 1 IQR Effect Estimate [Lower CI, Upper CI]: Association between the prevalence of inhaled ß2- agonist use and MC0.01-2.5 Same day, IQR = 28, OR = 0.96 (0.90-1.04) 5-day mean, IQR = 26 , OR = 1.10 (1.01-1.20) 14-day mean, IQR = 20, OR = 1.03 (0.95-1.12)
Reference: von Klot et al. (2002, 034706) **Period of Study:** September 1996 to March 1997 (winter) **Location:** Erfurt, Germany	**Outcome:** Asthma symptoms (wheezing, shortness of breath at rest, waking up with breathing problems, or coughing without having a cold) and Asthma medication (inhaled short-acting ß2-agonists, inhaled long-acting ß2- agonists, inhaled corticosteroids, cromolyn sodium, theophylline, oral corticosteroids, and N-acetylcysteine) **Age Groups**: Adults, mean = 59.0 yrs and range = 37-77 yrs **Study Design:** panel study **N:** 53 adult asthmatics **Statistical Analyses:** Logistic regression models **Covariates:** seasonal variation in medication use or symptom prevalence, meteorological factors (relative humidity, temperature), weekend, Christmas holidays	**Pollutant:** $NC_{0.1-0.01}$ **Averaging Time:** 10 min intervals **Mean (SD):** 17300 /cm^3 Percentiles: 25th: 9286 50th(Median): 16940 75th: 24484 **Range (Min, Max):** (3272-46195) Unit (i.e. $\mu g/m^3$): 1/cm^3 **Copollutant (correlation):** $PM_{10-2.5}$: r = 0.41 $NC_{0.5-0.1}$: r = 0.55 $NC_{2.5-0.5}$: r = 0.34 $MC_{0.5-0.1}$: r = 0.45	NC Increment: 1 IQR Effect Estimate [Lower CI, Upper CI]: Association between the prevalence of inhaled ß2- agonist use and NC0.01-0.1 Same day, IQR = 15000, OR = 0.97 (0.90-1.04) 5-day mean, IQR = 10000, OR = 1.11 (1.01-1.21) 14-day mean, IQR = 7700, OR = 1.08 (0.96-1.21) Association between two pollutants, jointly in one model, and the Outcomes Inhaled short-acting ß2- agonist use $NC_{0.1-0.01}$ OR = 1.07 (0.97-1.18) $MC_{0.5-0.1}$: OR = 1.07 (0.98-1.18) Inhaled corticosteroid use

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
	Season: winter	$MC_{2.5-0.01}$: r = 0.45	$NC_{0.1-0.01}$ OR = 1.01 (0.87-1.18)
	Dose-response Investigated? No	PM_{10}: r = 0.51	$MC_{0.5-0.1}$: OR = 1.53 (1.39-1.69)
	Statistical Package: NR	NO_2: r = 0.66	Wheezing
	Lags Considered: 0, 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, mov avg calculated from same day and preceding days	CO: r = 0.66	$NC_{0.1-0.01}$ OR = 1.12 (1.01-1.24)
		SO_2: r = 0.36	$MC_{0.5-0.1}$: OR = 1.02 (0.92-1.12)
			Association between the prevalence of inhaled corticosteroid use and NC0.01-0.1
			Same day, IQR = 15000, OR = 1.07 (1.00-1.15)
			5-day mean, IQR = 10000, OR = 1.22 (1.12-1.33)
			14-day mean, IQR = 7700, OR = 1.45 (1.29-1.63)
			Association between the prevalence of wheezing and $NC_{0.1-0.01}$
			Same day, IQR = 15000, OR = 0.94 (0.86-1.01)
			5-day mean, IQR = 10000, OR = 1.13 (1.03-1.24)
			14-day mean, IQR = 7700, OR = 1.27 (1.13-1.43)
			Association between the prevalence of respiratory symptoms and $NC_{0.1-0.01}$
			Attack of shortness of breath and wheezing
			Same day, IQR = 15000, OR = 1.01 (0.91-1.12)
			5-day mean, IQR = 10000, OR = 1.08 (0.96-1.21)
			14-day mean, IQR = 7700, OR = 1.26 (1.08-1.48)
			Walking up with breathing problems
			Same day, IQR = 15000, OR = 1.04 (0.96-1.13)
			5-day mean, IQR = 10000, OR = 1.09 (0.99-1.19)
			14-day mean, IQR = 7700, OR = 1.26 (1.13-1.41)
			Shortness of breath
			Same day, IQR = 15000, OR = 0.98 (0.90-1.06)
			5-day mean, IQR = 10000, OR = 1.09 (0.99-1.19)
			14-day mean, IQR = 7700, OR = 1.24 (1.11-1.40)
			Phlegm
			Same day, IQR = 15000, OR = 1.01 (0.94-1.09)
			5-day mean, IQR = 10000, OR = 1.11 (1.02-1.21)
			14-day mean, IQR = 7700, OR = 1.11 (0.99-1.25)
			Cough
			Same day, IQR = 15000, OR = 1.07 (0.98-1.16)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			5-day mean, IQR = 10000, OR = 1.17 (1.07-1.28) 14-day mean, IQR = 7700, OR = 1.20 (1.06-1.35)
Reference: von Klot et al. (2002, 034706) **Period of Study:** September 1996 to March 1997 (winter) **Location:** Erfurt, Germany	**Outcome:** Asthma symptoms (wheezing, shortness of breath at rest, waking up with breathing problems, or coughing without having a cold) and Asthma medication (inhaled short-acting ß2-agonists, inhaled long-acting ß2- agonists, inhaled corticosteroids, cromolyn sodium, theophylline, oral corticosteroids, and N-acetylcysteine) **Age Groups**: Adults, mean = 59.0 yrs and range = 37-77 yrs **Study Design:** panel study **N:** 53 adult asthmatics **Statistical Analyses:** Logistic regression models **Covariates:** seasonal variation in medication use or symptom prevalence, meteorological factors (relative humidity, temperature), weekend, Christmas holidays **Season:** winter **Dose-response Investigated?** No **Statistical Package**: NR **Lags Considered:** 0, 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, mov avg calculated from same day and preceding days	**Pollutant:** $NC_{0.5-0.1}$ **Averaging Time:** 10 min intervals **Mean (SD):** 2005 /cm^3 Percentiles: 25th: 958 50th(Median): 1610 75th: 2767 **Range (Min, Max):** (291-6700) Unit (i.e. $\mu g/m^3$): 1/cm^3 **Copollutant (correlation):** $PM_{10-2.5}$: r = 0.50 $NC_{0.1-0.01}$: r = 0.55 $NC_{2.5-0.5}$: r = 0.76 $MC_{0.5-0.1}$: r = 0.95 $MC_{2.5-0.01}$: r = 0.93 PM_{10}: r = 0.85 NO_2: r = 0.75 CO: r = 0.79 SO_2: r = 0.51	NC Increment: 1 IQR Effect Estimate [Lower CI, Upper CI]: Association between the prevalence of inhaled ß2- agonist use and $NC_{0.5-0.1}$ Same day, IQR = 1800, OR = 0.99 (0.92-1.05) 5-day mean, IQR = 1500, OR = 1.10 (1.03-1.19) 14-day mean, IQR = 1450, OR = 0.95 (0.86-1.05) Association between the prevalence of inhaled corticosteroid use and $NC_{0.5-0.1}$ Same day, IQR = 1800, OR = 1.06 (0.99-1.14) 5-day mean, IQR = 1500, OR = 1.23 (1.14-1.32) 14-day mean, IQR = 1450, OR = 1.51 (1.37-1.67) Association between the prevalence of wheezing and $NC_{0.5-0.1}$ Same day, IQR = 1800, OR = 1.00 (0.93-1.07) 5-day mean, IQR = 1500, OR = 1.08 (1.00-1.17) 14-day mean, IQR = 1450, OR = 1.11 (1.00-1.24)
Reference: von Klot et al. (2002, 034706) **Period of Study:** September 1996 to March 1997 (winter) **Location:** Erfurt, Germany	**Outcome:** Asthma symptoms (wheezing, shortness of breath at rest, waking up with breathing problems, or coughing without having a cold) and Asthma medication (inhaled short-acting ß2-agonists, inhaled long-acting ß2- agonists, inhaled corticosteroids, cromolyn sodium, theophylline, oral corticosteroids, and N-acetylcysteine) **Age Groups**: Adults, mean = 59.0 yrs and range = 37-77 yrs **Study Design:** panel study **N:** 53 adult asthmatics **Statistical Analyses:** Logistic regression models **Covariates:** seasonal variation in medication use or symptom prevalence, meteorological factors (relative humidity, temperature), weekend, Christmas holidays **Season:** winter **Dose-response Investigated?** No **Statistical Package**: NR **Lags Considered:** 0, 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, mov avg calculated from same day and preceding days	**Pollutant:** $NC_{2.5-0.5}$ **Averaging Time:** 10 min intervals **Mean (SD):** 21.4 /cm^3 Percentiles: 25th: 5.6 50th(Median): 13.0 75th: 31.6 **Range (Min, Max):** (0.9-127.6) Unit (i.e. $\mu g/m^3$): 1/cm^3 **Copollutant (correlation):** $PM_{10-2.5}$: r = 0.48 $NC_{0.1-0.01}$: r = 0.34 $NC_{0.5-0.1}$: r = 0.76 $MC_{0.5-0.1}$: r = 0.92 $MC_{2.5-0.01}$: r = 0.94 PM_{10}: r = 0.88 NO_2: r = 0.54 CO: r = 0.46 SO_2: r = 0.66	NC Increment: 1 IQR Effect Estimate [Lower CI, Upper CI]: Association between the prevalence of inhaled ß2- agonist use and $NC_{2.5-0.5}$ Same day, IQR = 26, OR = 0.99 (0.93-1.05) 5-day mean, IQR = 22, OR = 1.09 (1.01-1.17) 14-day mean, IQR = 17, OR = 1.08 (1.02-1.15) Association between the prevalence of inhaled corticosteroid use and $NC_{2.5-0.5}$ Same day, IQR = 26, OR = 1.13 (1.06-1.21) 5-day mean, IQR = 22, OR = 1.28 (1.19-1.37) 14-day mean, IQR = 17, OR = 1.44 (1.36-1.53) Association between the prevalence of wheezing and $NC_{2.5-0.5}$ Same day, IQR = 26, OR = 1.03 (0.95-1.10) 5-day mean, IQR = 22, OR = 1.05 (0.97-1.13) 14-day mean, IQR = 17, OR = 1.03 (0.96-

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			1.10)
Reference: von Klot et al. (2002, 034706) **Period of Study:** September 1996 to March 1997 (winter) **Location:** Erfurt, Germany	**Outcome:** Asthma symptoms (wheezing, shortness of breath at rest, waking up with breathing problems, or coughing without having a cold) and Asthma medication (inhaled short-acting ß2-agonists, inhaled long-acting ß2- agonists, inhaled corticosteroids, cromolyn sodium, theophylline, oral corticosteroids, and N-acetylcysteine) **Age Groups**: Adults, mean = 59.0 yrs and range = 37-77 yrs **Study Design:** panel study **N:** 53 adult asthmatics **Statistical Analyses:** Logistic regression models **Covariates:** seasonal variation in medication use or symptom prevalence, meteorological factors (relative humidity, temperature), weekend, Christmas holidays **Season**: winter **Dose-response Investigated?** No **Statistical Package**: NR **Lags Considered:** 0, 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, mov avg calculated from same day and preceding days	**Pollutant:** $PM_{10-2.5}$ **Averaging Time:** 24 h **Mean (SD):** 10.3 Percentiles: 25th: 2.9 50th(Median): 6.9 75th: 14.6 **Range (Min, Max):** (-8.7-64.3) **Copollutant (correlation):** $NC_{0.1-0.01}$: r = 0.41 $NC_{0.5-0.1}$: r = 0.50 $NC_{2.5-0.5}$: r = 0.48 $MC_{0.5-0.1}$: r = 0.51 $MC_{2.5-0.01}$: r = 0.52 PM_{10}: r = 0.67 NO_2: r = 0.45 CO: r = 0.42 SO_2: r = 0.28	**PM Increment:** 1 IQR Effect Estimate [Lower CI, Upper CI]: Association between the prevalence of inhaled ß2- agonist use and $PM_{10-2.5}$ Same day, IQR = 12, OR = 1.01 (0.95-1.06) 5-day mean, IQR = 11, OR = 1.01 (0.94-1.09) 14-day mean, IQR = 6.7, OR = 0.92 (0.86-1.00) Association between the prevalence of inhaled corticosteroid use and $PM_{10-2.5}$ Same day, IQR = 12, OR = 1.03 (0.98-1.08) 5-day mean, IQR = 11, OR = 1.12 (1.04-1.20) 14-day mean, IQR = 6.7, OR = 1.27 (1.18-1.37) Association between the prevalence of wheezing and $PM_{10-2.5}$ Same day, IQR = 12, OR = 0.97 (0.91-1.02) 5-day mean, IQR = 11, OR = 1.06 (0.98-1.15) 14-day mean, IQR = 6.7, OR = 1.05 (0.96-1.15)
Reference: Ward et al. (2002, 025839) **Period of Study:** 1997 (two 8-week periods) **Location:** Birmingham and Sandwell, UK	**Outcome:** Change in PEF (peak expiratory flow), self reported respiratory symptoms (same day cough, illness, short of breath, waking up at night with cough or wheeze, wheeze) **Age Groups**: 9 year olds **Study Design:** Time–series panel study **N:** 162 children from 5 schools **Statistical Analyses:** Linear regression (PEF), Logistic regression (respiratory symptoms) **Covariates:** Trend, temperature, schooldays (yes/no) **Season:** Winter (Jan 13-Mar 10) Summer (May 19- July 14) **Dose-response Investigated?** No **Statistical Package**: Nr **Lags Considered:** Lag 0, lag 1, lag 2, lag 3, 7-day moving avg	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-h **Mean (SD):** Winter: 12.7 $\mu g/m^3$ Summer: 12.3 $\mu g/m^3$ **Range (Min, Max):** Winter: 4, 37 Summer: 5, 28 PM Component: Total mass **Monitoring Stations**: 5 stations near the 5 schools **Copollutant (correlation):** Winter: PM_{10}(r = 0.93) NO_2 (r = 0.88) O_3 (r = -0.83) Summer: HNO_3 (r = 0.81)	**PM Increment:** Winter: 12.3 $\mu g/m^3$ Summer: 6.3 $\mu g/m^3$ Mean (PEF l/min) [Lower CI, Upper CI] lag: **Winter morning:** 0.80 [-1.97, 3.67] lag0 0.62 [-2.22, 3.54] lag 1 -0.86 [-4.32, 2.47] lag 2 -2.47 [-5.30, 0.36] lag 3 -4.07 [-10.60, 2.42] 7-day mean **Winter afternoon:** 0.95 [-2.22, 4.23] lag0 -0.99 [-4.69, 2.72] lag 1

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			-1.60 [-5.18, 2.01]
			lag 2
			-3.45 [-6.53 to -0.25]
			lag 3
			1.00 [-11.47, 13.56]
			7-day mean
			Summer morning:
			-1.49 [-3.65, 0.67]
			lag 0
			0.21 [-2.12, 2.55]
			lag1
			2.50 [0.28, 4.72]
			lag2
			3.41 [1.40, 5.44]
			lag3
			3.90 [-2.53, 10.33]
			7-day mean
			Summer afternoon:
			-0.49 [-2.43, 1.45]
			lag 0
			-0.78 [-2.72, 1.16]
			lag 1
			0.57 [-1.35, 2.49]
			lag 2
			0.16 [-1.85, 2.17]
			lag 3
			-0.08 [-5.43, 5.27]
			7-day mean
			Winter morning in atopy/recent wheezing subgroup:
			–0.072 [–0.527, 0.383]
			lag 0
			–0.271 [–0.701, 0.159]
			lag 1
			0.127 [–0.354, 0.608]
			lag 2
			0.055 [–0.391, 0.501]
			lag 3
			Winter morning in no atopy or recent wheezing subgroup:
			0.126 [–0.413 , 0.666]
			lag 0
			0.193 [–0.340 , 0.728] lag 1
			–0.170 [–0.788 , 0.447]; lag2
			–0.314 [–0.846 , 0.216]
			lag 3

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			Winter morning in subgroup with parental atopy/recent wheezing:
			0.187 [–0.008 , 0.382]
			lag 0
			–0.006 [–0.207 , 0.195]
			lag 1
			–0.011 [–0.226 , 0.204]
			lag 2
			–0.037 [–0.228 , 0.154]
			lag 3
			Winter morning in subgroup without parental atopy/recent wheezing:
			0.026 [–0.341 , 0.395]
			lag 0
			0.068 [–0.307 , 0.444]
			lag 1
			–0.099 [–0.535 , 0.335]
			lag 2
			–0.252 [–0.615 , 0.110]
			lag 3
			RR Estimate [Lower CI, Upper CI]
			lag:
			Cough:
			Winter: 0.98 [0.80, 1.18]
			lag 0
			0.95 [0.77, 1.17]
			lag 1
			1.02 [0.83, 1.24]
			lag 2
			1.01 [0.83, 1.23]
			lag 3
			1.31 [0.82, 2.09]
			7-day mean
			Summer: 1.13 [1.04, 1.22]
			lag 0
			1.04 [0.94, 1.13]
			lag 1
			0.94 [0.87, 1.02]
			lag 2
			0.89 [0.82, 0.96]
			lag 3
			0.81 [0.62, 1.06]
			7 day mean
			Illness:
			Winter: 1.17 [1.05, 1.32]
			lag 0

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			1.07 [0.95, 1.23]
			lag 1
			1.16 [1.01, 1.35]
			lag 2
			1.01 [0.90, 1.16]
			lag 3
			1.57 [1.15, 2.13]
			7-day mean
			Summer: 1.02 [0.91, 1.13]
			lag 0
			1.00 [0.89, 1.13]
			lag 1
			0.96 [0.85, 1.07]
			lag 2
			0.97 [0.86, 1.09]
			lag 3
			0.68 [0.41, 1.13]
			7-day mean
			Shortness of breath:
			Winter: 1.07 [0.94, 1.24]
			lag 0
			0.98 [0.84, 1.13]
			lag 1
			0.96 [0.82, 1.13]
			lag2
			0.91 [0.79, 1.07]
			lag 3
			0.82 [0.58, 1.18]
			7-day mean
			Summer: 1.04 [0.90, 1.20]
			lag 0
			1.08 [0.93, 1.25]
			lag 1
			0.97 [0.84, 1.13]
			lag 2
			0.93 [0.81, 1.08]
			lag 3
			1.16 [0.76, 1.77]
			7-day mean
			Wake at night with cough/wheeze:
			Winter: 1.10 [0.96, 1.26]
			lag 0
			1.05 [0.90, 1.22]
			lag 1
			0.98 [0.83, 1.13]; lag 2

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			0.94 [0.81, 1.09]; lag 3
			0.93 [0.66, 1.32]
			7-day mean
			Summer: 0.93 [0.78, 1.10]
			lag 0
			0.81 [0.67, 0.98]
			lag 1
			0.91 [0.77, 1.09]
			lag 2
			0.97 [0.83, 1.13]
			lag 3
			1.04 [0.57, 1.90]
			7-day mean
			Wheeze:
			Winter: 0.98 [0.83, 1.16]
			lag 0
			0.90 [0.75, 1.05]
			lag 1
			1.00 [0.83, 1.20]
			lag 2
			1.13 [0.95, 1.35]
			lag 3
			1.02 [0.68, 1.57]; 7-day mean
			Summer: 1.02 [0.88, 1.19]
			lag 0
			0.98 [0.84, 1.16]
			lag 1
			0.87 [0.74, 1.02]
			lag 2
			0.85 [0.72, 0.99]
			lag 3
			0.96 [0.51, 1.81]
			7-day mean
Reference: Ward et al. (2002, 025839) **Period of Study:** 1997 (two 8-week periods) **Location:** Birmingham and Sandwell, UK	**Outcome:** Change in PEF (peak expiratory flow), self reported respiratory symptoms (same day cough, illness, short of breath, waking up at night with cough or wheeze, wheeze) **Age Groups:** 9 year olds **Study Design:** Time–series panel study **N:** 162 children from 5 schools **Statistical Analyses:** Linear regression (PEF), Logistic regression (respiratory symptoms) **Covariates:** Trend, temperature, schooldays (yes/no)	**Pollutant:** Sulfate **Averaging Time:** 24-h **Mean (SD):** Winter: 2.4 μg/m^3 Summer: 3.8 μg/m^3 **Range (Min, Max):** Winter: 0.8, 14.9 Summer: 1.1, 7.8 PM Component: SO_4 **Monitoring Stations:** 2 stations	**PM Increment:** Winter: 4.8 μg/m^3 Summer: 3.1 μg/m^3 Mean (PEF l/min) [Lower CI, Upper CI] Ia **Winter morning:** -1.75 [-4.00, 0.50] lag0 -0.91 [-3.44, 1.62] lag 1 -0.62 [-3.16, 1.91] lag 2

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
	Season: Winter (Jan 13-Mar 10)		-1.82 [-4.27, 0.64]
	Summer (May 19- July 14)		lag 3
	Dose-response Investigated? No		-3.22 [-8.03, 1.58]
	Statistical Package: Nr		7-day mean
	Lags Considered: Lag 0, lag 1, lag 2, lag 3, 7-day moving avg		**Winter afternoon:**
			0.99 [-1.58, 3.55]
			lag0
			0.79 [-2.42, 4.00]
			lag 1
			-1.89 [-4.99, 1.21]
			lag 2
			-1.73 [-4.69, 1.23]
			lag 3
			-1.96 [-13.35, 9.42]
			7-day mean
			Summer morning:
			-0.72 [-3.27, 1.82]
			lag 0
			-1.69 [-4.28, 0.90]
			lag1
			1.35 [-1.27, 3.97]
			lag2
			3.38 [1.03, 5.72]
			lag3
			2.98 [-4.17, 10.13]
			7-day mean
			Summer afternoon:
			-0.32 [-2.81, 2.17]
			lag 0
			0.84 [-1.63, 3.30]
			lag 1
			-0.08 [-2.61, 2.44]
			lag 2
			-0.25 [-2.69, 2.19]
			lag 3
			-2.20 [-9.51, 5.12]
			7-day mean
			Winter morning in atopy/recent wheezing subgroup:
			0.200 [–0.755, 1.156]
			lag 0
			–0.219 [–1.318, 0.881]
			lag 1
			–0.431 [–1.526, 0.664]; lag 2
			1.200 [0.095, 2.305]

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			lag 3
			Winter morning in no atopy or recent wheezing subgroup:
			–0.613 [–1.714, 0.488]
			lag 0
			–0.174 [–1.423, 1.075]
			lag 1
			0.006 [–1.243, 1.253]
			lag 2
			–1.080 [–2.308, 0.148]
			lag 3
			Winter morning in subgroup with parental atopy/recent wheezing:
			0.457 [0.003 , 0.910]
			lag 0
			0.078 [–0.503, 0.660]
			lag 1
			–0.102 [–0.656, 0.452]
			lag 2
			0.002 [–0.609, 0.613]
			lag 3
			Winter morning in subgroup without parental atopy/recent wheezing:
			–0.622 [–1.379, 0.136]
			lag 0
			–0.272 [–1.147, 0.602]
			lag 1
			–0.138 [–1.005, 0.728]
			lag 2
			–0.496 [–1.359, 0.367]
			lag 3
			RR Estimate [Lower CI, Upper CI]
			lag:
			Cough:
			Winter: 1.01 [0.84, 1.20]
			lag 0
			1.02 [0.85, 1.24]
			lag 1
			0.99 [0.82, 1.20]
			lag 2
			0.86 [0.71, 1.05]
			lag 3
			0.78 [0.53, 1.14]
			7-day mean
			Summer: 1.08 [0.98, 1.20]
			lag 0

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			1.03 [0.93, 1.15]
			lag 1
			0.97 [0.88, 1.07]
			lag 2′ 0.90 [0.82, 0.99]
			lag 3
			0.73 [0.54, 0.97]
			7 day mean
			Illness:
			Winter: 1.06 [0.96, 1.17]
			lag 0
			1.15 [1.03, 1.28]
			lag 1
			1.14 [1.00, 1.28]
			lag 2
			1.04 [0.92, 1.18]
			lag 3
			1.30 [1.00, 1.66]
			7-day mean
			Summer: 0.98 [0.86, 1.11]
			lag 0
			0.97 [0.84, 1.12]
			lag 1
			1.01 [0.88, 1.16]
			lag 2′ 0.95 [0.84, 1.09]
			lag 3
			0.72 [0.46, 1.12]
			7-day mean
			Shortness of breath:
			Winter: 0.96 [0.85, 1.07]
			lag 0: 0.98 [0.86, 1.12]
			lag 1
			0.94 [0.82, 1.07]
			lag2
			0.93 [0.81, 1.08]
			lag 3
			0.80 [0.59, 1.07]
			7-day mean
			Summer: 0.95 [0.80, 1.14]
			lag 0
			1.07 [0.89, 1.28]
			lag 1
			1.04 [0.87, 1.24]
			lag 2
			0.94 [0.80, 1.12]
			lag 3

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			|0.58 [0.33, 1.04]
			7-day mean
			Wake at night with cough/wheeze:
			Winter: 0.97 [0.87, 1.08]
			lag 0
			1.01 [0.89, 1.15]
			lag 1
			1.00 [0.88, 1.14]; lag 2
			0.93 [0.82, 1.07]; lag 3
			0.79 [0.59, 1.05]
			7-day mean
			Summer: 0.95 [0.78, 1.16]
			lag 0
			0.81 [0.67, 0.99]
			lag 1
			0.93 [0.76, 1.13]
			lag 2
			0.87 [0.72, 1.05]
			lag 3
			0.77 [0.41, 1.48]
			7-day mean
			Wheeze:
			Winter: 1.00 [0.87, 1.15]
			lag 0
			0.96 [0.82, 1.13]
			lag 1
			0.88 [0.75, 1.04]
			lag 2
			1.12 [0.95, 1.32]
			lag 3
			0.83 [0.58, 1.20]; 7-day mean
			Summer: 0.97 [0.80, 1.17]
			lag 0
			.09 [0.89, 1.32]
			lag 1
			1.00 [0.82, 1.22]
			lag 2
			0.81 [0.69, 0.97]
			lag 3
			1.30 [0.68, 2.50]
			7-day mean
Reference: Ward et al. (2002, 025839) **Period of Study:** 1997 (two 8-week periods) **Location:** Birmingham and Sandwell, UK	**Outcome:** Change in PEF (peak expiratory flow), self reported respiratory symptoms (same day cough, illness, short of breath, waking up at night with cough or wheeze, wheeze) **Age Groups:** 9 year olds	**Pollutant:** NO_3 **Averaging Time:** 24-h **Mean (SD):** Winter: 3.6 $\mu g/m^3$	**PM Increment:** Winter: 6.7 $\mu g/m^3$ Summer: 3.7 $\mu g/m^3$ Mean (PEF l/min) [Lower CI, Upper CI] lag:

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
	Study Design: Time–series panel study	Summer: 3.5 $\mu g/m^3$	**Winter morning:**
	N: 162 children from 5 schools	**Range (Min, Max):**	-2.08 [-4.02 to -0.15]
	Statistical Analyses: Linear regression (PEF),	Winter: 0.1, 29.9	lag0
		Summer: 0.7, 13.2	-0.64 [-2.87, 1.59]
	Logistic regression (respiratory symptoms)	**Monitoring Stations:**	lag 1
	Covariates: Trend, temperature, schooldays (yes/no)	2 stations	0.71 [-1.69, 3.11]
			lag 2
	Season: Winter (Jan 13-Mar 10)		-1.38 [-3.61, 0.84]
	Summer (May 19- July 14)		lag 3
	Dose-response Investigated? No		-0.92 [-5.32, 3.47]
	Statistical Package: Nr		7-day mean
	Lags Considered: Lag 0, lag 1, lag 2, lag 3, 7-day moving avg		**Winter afternoon:**
			0.24 [-1.89, 2.38]
			lag0
			-0.72 [-3.87, 2.43]
			lag 1
			-1.37 [-5.11, 2.38]
			lag 2
			-2.54 [-5.74, 0.66]
			lag 3
			0.21 [-7.67, 8.11]
			7-day mean
			Summer morning:
			-0.80 [-2.74, 1.15]
			lag 0
			0.68 [-1.31, 2.67]
			lag1
			1.42 [-0.73, 3.58]
			lag2
			2.54 [0.48, 4.59]
			lag3
			1.74 [-2.66, 6.13]
			7-day mean
			Summer afternoon:
			-0.72 [-2.47, 1.03]
			lag 0
			-0.59 [-2.36, 1.18]
			lag 1
			-0.33 [-2.11, 1.45]
			lag 2
			0.66 [-1.26, 2.58]
			lag 3
			0.47 [-3.36, 4.29]
			7-day mean
			Winter morning in atopy/recent

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			wheezing subgroup:
			–0.036 [–0.627 , 0.555]
			lag 0
			0.142 [–0.573 , 0.857]
			lag 1
			0.000 [–0.760, 0.759]
			lag 2
			0.689 [–0.061, 1.439]
			lag 3
			Winter morning in no atopy or recent wheezing subgroup:
			–0.434 [–1.116, 0.248]
			lag 0
			–0.201 [–1.002 , 0.600]
			lag 1
			0.154 [–0.703 , 1.010]
			lag 2
			–0.605 [–1.422 , 0.210]
			lag 3
			Winter morning in subgroup with parental atopy/recent wheezing:
			0.228 [–0.054, 0.511]
			lag 0
			0.476 [0.060, 0.892]
			lag 1
			0.196 [–0.202, 0.594]
			lag 2
			0.083 [–0.321, 0.487]
			lag 3
			Winter morning in subgroup without parental atopy/recent wheezing:
			–0.482 [–0.952, –0.012]
			lag 0
			–0.276 [–0.846, 0.294]
			lag 1
			0.078 [–0.520, 0.675]
			lag 2
			–0.298 [–0.864, 0.268]
			lag 3
			RR Estimate [Lower CI, Upper CI]
			lag:
			Cough: Winter:
			0.92 [0.80, 1.07]
			lag 0
			0.91 [0.77, 1.07]
			lag 1

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			0.99 [0.83, 1.17]
			lag 2
			0.87 [0.73, 1.03]
			lag 3
			0.71 [0.52, 0.97]
			7-day mean
			Summer:
			1.05 [0.97, 1.13]
			lag 0
			1.01 [0.93, 1.10]
			lag 1
			0.95 [0.88, 1.03]
			lag 2
			0.89 [0.83, 0.96]
			lag 3
			0.81 [0.68, 0.97]
			7 day mean
			Illness: Winter:
			1.05 [0.97, 1.14]
			lag 0
			1.11 [1.01, 1.22]
			lag 1
			1.13 [1.01, 1.26]
			lag 2
			1.13 [1.04, 1.26]
			lag 3
			1.13 [0.92, 1.38]
			7-day mean
			Summer:
			0.97 [0.87, 1.09]
			lag 0
			0.98 [0.87, 1.10]
			lag 1
			0.95 [0.85, 1.06]
			lag 2
			0.94 [0.85, 1.05]
			lag 3
			0.74 [0.54, 1.03]
			7-day mean
			Shortness of breath: Winter:
			0.99 [0.90, 1.10]
			lag 0
			1.01 [0.90, 1.13]
			lag 1
			0.93 [0.82, 1.05]

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			lag2
			0.98 [0.86, 1.13]
			lag 3
			0.85 [0.67, 1.08]
			7-day mean
			Summer:
			1.04 [0.90, 1.18]
			lag 0
			1.12 [0.98, 1.28]
			lag 1
			1.04 [0.90, 1.20]
			lag 2
			0.90 [0.79, 1.03]
			lag 3
			1.06 [0.78, 1.43]
			7-day mean
			Wake at night with cough/wheeze: Winter:
			0.98 [0.89, 1.08]
			lag 0
			1.05 [0.94, 1.16]
			lag 1
			0.99 [0.88, 1.12]; lag 2
			0.99 [0.87, 1.12]; lag 3
			0.84 [0.67, 1.05]
			7-day mean
			Summer:
			0.94 [0.80, 1.09]
			lag 0
			0.86 [0.72, 1.01]
			lag 1
			0.94 [0.79, 1.11]
			lag 2
			0.92 [0.79, 1.07]
			lag 3
			0.95 [0.62, 1.47]
			7-day mean
			Wheeze: Winter:
			0.98 [0.87, 1.10]
			lag 0
			1.00 [0.87, 1.14]
			lag 1
			0.89 [0.77, 1.03]
			lag 2
			1.11 [0.95, 1.30]

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			lag 3
			0.80 [0.61, 1.07]
			7-day mean
			Summer:
			1.01 [0.87, 1.17]
			lag 0
			0.96 [0.83, 1.11]
			lag 1
			0.95 [0.82, 1.10]
			lag 2
			0.87 [0.75, 1.01]
			lag 3
			1.04 [0.67, 1.60]
			7-day mean

[1]All units expressed in $\mu g/m^3$ unless otherwise specified.

E.2.2. Respiratory Emergency Department Visits and Hospital Admissions

Table E-12. Short-term exposure–respiratory–ED/HA-PM_{10}

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Andersen et al. (2008, 189651) 1st page: 458 **Period of Study:** May 2001 - December 2004 **Location:** Los Angeles and San Diego counties, California	Hospital Admissions/ED visits **Outcome (ICD-10):** **RD**, including chronic bronchitis (J41 – 42), emphysema (J43), other chronic obstructive pulmonary disease (J44), asthma (J45), and status asthmaticus (J46). **Pediatric hospital admissions for asthma** (J45) and status asthmaticus (J46). **Age Groups Analyzed:** > 65 yrs (RD combined), 5 – 18 years (asthma) **Study Design:** Time series **N:** NR **Statistical Analyses:** Poisson GAM **Covariates:** temperature, dew-point temperature, long-term trend, seasonality, influenza, day of the week, public holidays, school holidays (only for 5 – 18 year olds), pollen (only for pediatric asthma outcome) **Season:** NR **Dose-response Investigated:** No **Statistical package:** R statistical software (gam procedure, mgcv package) **Lags Considered:** Lag 0 -5 days, 5-day average (lag 0 – 4) for RD, and a 6-day average (lag 0 – 5) for asthma.	**Pollutant:** PM_{10} ($\mu g/m^3$) **Averaging Time:** 24 h **Mean (SD** **median** **IQR** **99th percentile:** 24 (14 21 16 – 29 72) **Monitoring Stations:** 1 **Copollutant (correlation):** NCtot: r = 0.39 NC100: r = 0.28 NCa12: r = 0.02 Nca23: r = -0.12 NCa57: r = 0.45 Nca212: r = 0.63 $PM_{2.5}$: r = 0.80 CO: r = 0.37 NO_2: r = 0.35 NO_X: r = 0.32 NO_X curbside: r = 0.18 O_3: r = -0.21 **Other variables:** Temperature: r = 0.12 Relative humidity: r = 0.05	**PM Increment:** 13 $\mu g/m^3$ [3] **Relative risk (RR) Estimate [CI]:** **RD hospital admissions (5 day average, lag 0 -4), age 65+:** One-pollutant model: 1.06 [1.02 – 1.09] Adj for NCtot: 1.05 [1.01 – 1.10] Adj for NCa212: 1.04 [0.98 – 1.11] **Asthma hospital admissions (6 day avg lag 0 – 5), age 5 - 18:** One-pollutant model: 1.02 [0.93 – 1.12] Adj for NCtot: 1.01 [0.91 – 1.12] Adj for NCa212: 0.94 [0.81 – 1.09] Estimates for individual day lags reported only in figure form (see notes): **Notes:** Figure 2: Relative risks and 95% confidence intervals per IQR in single day concentration (0 – 5 day lag). Summary of Figure 2: RD: Positive, statistically or marginally significant associations at Lag 2 – 5. Asthma: Wide confidence intervals make interpretation difficult. Positive associations at Lag 1, 2, 3, and 5.
Reference: Cheng et al. (2007, 093034) **Period of Study:** 1996-2004 **Location:** Kaohsiung, Taiwan	**Outcome (ICD-9: 480-486):** Pneumonia **Age Groups:** NR **Study Design:** Case-crossover **N:** 82,587 pneumonia hospital admissions **Statistical Analyses:** Conditional logistic regression **Covariates:** Temperature and humidity on the same day **Season:** NR **Dose-response Investigated?** No **Statistical Package:** SAS **Lags Considered:** Cumulative lag period up to 2 previous days	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (min-max):** 77.01 (16.7-232) Percentiles: 25%: 42.12 50%: 75.27 75%: 104.65 **Monitoring Stations:** 6 **Copollutant:** NR	**PM Increment:** 62.53 $\mu g/m^3$ (IQR) **OR Estimate [CI]:** Single Pollutant Model: Temp > 25ºC: 1.21 [1.15,1.28] Temp < 25ºC: 1.57 [1.50,1.65] Two-Pollutant Model: Temp > 25ºC Adj. for SO_2: 1.21 [1.14,1.28] Adj. for NO_2: 1.15 [1.07,1.24] Adj. for CO: 1.10 [1.03,1.17] Adj. for O_3: 0.96 [0.89,1.03] Temp < 25ºC Adj. for SO_2: 1.56 [1.48,1.65] Adj. for NO_2: 1.09 [1.02,1.16] Adj. for CO: 1.30 [1.22,1.39] Adj. for O_3: 1.56 [1.48,1.65]

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Chimonas and Gessner (2007, 093261) **Period of Study:** January 1, 1999–June 30, 2003 **Location:** Anchorage, Alaska	**Outcome (ICD-9):** Asthma (493.0-493.9) Lower respiratory illness-LRI (466.1, 466.0, 480-487, 490, 510-511) Inhaled quick-relief medication Steroid medication **Age Groups:** < 20 years old **Study Design:** Time series **N:** 42,667 admissions **Statistical Analyses:** GEE for multivariable modeling **Covariates:** Season, serial correlation, year, weekend, temperature, precipitation, and wind speed **Season:** NR **Dose-response Investigated?** No **Statistical Package:** SPSS (dataset), SAS (analysis) **Lags Considered:** 1 day and 1 week	**Pollutant:** PM_{10} **Averaging Time:** 24-hs and 1 week **Mean (min-max):** Daily: 27.6 (2-421) Weekly: 25.3 (5.0-116.0) **Monitoring Stations:** NR **Copollutant:** Daily $PM_{2.5}$ □ = 0.25 ($p < 0.01$) Weekly $PM_{2.5}$ □ = 0.08 ($p = 0.21$)	**PM Increment:** 10 $\mu g/m^3$ **RR Estimate [CI]:** Same Day Outpatient Asthma: 1.006 [1.001,1.013] Outpatient LRI: 1.001 [0.987,1.015] Inpatient Asthma: 1.003 [0.922,1.091] Inpatient LRI: 1.015 [0.978,1.053] Inhaled Steroid Prescriptions: 1.006 [0.996,1.011] Quick-relief Medication: 1.018 [1.006,1.030] Weekly (median increase) Outpatient Asthma: 1.021 [1.004,1.038] Outpatient LRI: 1.013 [0.978,1.049] Inpatient Asthma: 1.023 [0.948,1.104] Inpatient LRI: 1.025 [0.981,1.072] Inhaled Steroid Prescriptions: 0.989 [0.969,1.010] Quick-relief Medication: 1.057 [1.037,1.077]
Reference: Chiu et al. (2008, 191989) **Period of Study:** 1996-2001 **Location:** Taipei, Taiwan	**Outcome:** Hospital admissions for COPD **Study Design:** Time-series **Covariates:** Temperature, humidity, PM_{10} and O_3 **Statistical Analysis:** Poisson regression **Statistical Package:** SAS **Age Groups:** All	**Pollutant:** PM_{10} **Averaging Time:** 24h **Mean (SD) Unit:** Index Days: 111.68 ± 38.32 $\mu g/m^3$ Comparison Days: 55.43 ± 24.66 $\mu g/m^3$ **Range (Min, Max):** NR **Copollutant (correlation):** NR	All results refer to "dust storm days" and can be found in Table 3
Reference: Chiu et al. (2009, 190249) **Period of Study:** 1996-2004 **Location:** Taipei, Taiwan	**Outcome:** Hospital admissions for pneumonia (ICD-9 480-486) **Study Design:** Time-series **Covariates:** Weather variables, day of the week, seasonality, long-term time trends **Statistical Analysis:** Conditional logistic regression **Statistical Package:** SAS **Age Groups:** All	**Pollutant:** PM_{10} **Averaging Time:** 24h **Mean Unit:** 49.47 $\mu g/m^3$ **Range (Min, Max):** 14.42, 234.91 **Copollutant (correlation):** SO_2: 0.50 NO_2: 0.58 CO: 0.34 O_3: 0.31	**Increment:** IQR **Odds Ratio (95% CI)** Temperature ≥ 23° C: 1.11 (1.08-1.14) Temperature < 23° C: 1.09 (1.07-1.11) Adjusted for SO_2 Temperature ≥ 23° C: 1.10 (1.08-1.13) Temperature < 23° C: 1.19 (1.17-1.22) Adjusted for NO_2 Temperature ≥ 23° C: 0.90 (0.88-0.93) Temperature < 23° C: 1.09 (1.07-1.12) Adjusted for CO Temperature ≥ 23° C: 1.03 (1.00-1.05) Temperature < 23° C: 1.07 (1.05-1.10) Adjusted for O_3 Temperature ≥ 23° C: 1.05 (1.03-1.08) Temperature < 23° C: 1.09 (1.07-1.11)

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Erbas et al. (2005, 073849) **Period of Study:** Jan 2000–Dec 2001 **Location:** Melbourne, Australia	Hospital Admissions **Outcome (ICD-10):** Asthma (J45, J46) **Age Groups:** 1-15 yrs **Study Design:** Time series **N:** 8955 asthma cases **Statistical Analyses:** GAM, GEE (if autocorrelation was present in residuals) **Covariates:** Temp and humidity **Season:** NR **Dose-response Investigated?** No **Statistical Package:** NR **Lags Considered:** 0, 1, 2 days	**Pollutant:** PM_{10} **Averaging Time:** 1 h **Mean (SD):** Western: 2.99 (2.11) 10th percentile: 13.67 90th percentile: 48.00 Inner Melbourne: 4.54 (2.65) 10th percentile: 15.63 90th percentile: 59.73 South/Southeastern: 1.13 (1.18) 10th percentile: 12.00 90th percentile: 36.05 Eastern: 3.61 (2.39) 10th percentile: 16.00 90th percentile: 51.05 Combined: 30.07 (10.55-112.33) SD = 15.27 10th percentile: 16.00 90th percentile: 50.51 **Monitoring Stations:** Data obtained from an air quality simulation model (TAPM) by CSIRO Atmospheric Research **Copollutant:** NR	**PM Increment:** Increase from 10th to 90th percentile **RR Estimate [CI]:** Same day lag Western: NR Inner Melbourne: 1.17 [1.05,1.31] South/Southeastern: 1.14 [0.95,1.33] Eastern: 1.09 [1.01,1.18] **Notes:** All other lags NR

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Farhat et al. (2005, 089461) **Period of Study:** Aug 1996–Aug 1997 **Location:** São Paulo, Brazil	Hospital Admissions and Emergency Room Visits **Outcome (ICD-9):** Lower respiratory tract diseases (466, 480-519) including pneumonia or bronchopneumonia (480-486), asthma (493), bronchiolitis (466) **Age Groups:** < 13 yrs **Study Design:** Time series **N:** 43,635 **Statistical Analyses:** GAM, Poisson regression, Pearson correlation **Covariates:** Time, temperature, humidity, weekday **Season:** NR **Dose-response Investigated?** No **Statistical Package:** S-Plus **Lags Considered:** 0-7 days	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (min-max):** 62.6 (25.5-186.3) SD = 26.6 IQr = 30 N = 396 **Monitoring Stations:** 13 **Copollutant (correlation):** SO_2: r = 0.69 NO_2: r = 0.83 O_3: r = 0.35 CO: r = 0.72 (all $p < 0.05$) **Additional correlations:** Rel humidity: r = -0.55 Min temp: r = -0.44 (both $p < 0.05$)	**PM Increment:** 30 $\mu g/m^3$ (IQR) **RR Estimate [CI]:** Lower respiratory tract disease 5-day moving avg Copollutant model: NO_2: 2.1 [-7.1,11.3] SO_2: 16.5 [10.5,22.6] O_3: 10.1 [5.0,15.2] CO: 14.1 [8.1,20.2] Multipollutant model: 5.2 [-4.6,15.1] Pneumonia or bronchopneumonia 6-day moving avg Copollutant model: NO_2: 14.8 [-3.8,33.4] SO_2: 14.8 [-0.3,30.0]; O_3: 16.2 [1.0,31.3] CO: 17.6 [0.4,34.8] Multipollutant model: 5.23 [-16.2,26.6] Asthma or bronchiolitis 2-day moving avg Copollutant model: NO_2: -11.04 [-50.0,28.0] SO_2: 15.8 [-7.8,39.3] O_3: 11.7 [-10.4, 33.9] CO: 12.4 [-14.8,39.7] Multipollutant model: -15.5 [-61.2,30.2]

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Fung et al. (2006, 089789) **Period of Study:** 6/1/95–3/31/99 **Location:** Vancouver, Canada	Hospital Admission/ED **Outcome:** Respiratory diseases (460-519) **Age Groups:** Age > 65 **Study Design:** Time series **N:** 26,275 individuals admitted **Statistical Analyses:** Poisson regression (spline 12 knots), case-crossover (controls +/7 d days from case date), Dewanji and Moolgavkar (DM) method **Covariates:** Long-term trends, day-of-the-week effect, weather **Season:** All year **Dose-response Investigated?** No **Statistical Package:** SPlus, R **Lags Considered:** 0-7 d	**Pollutant:** PM_{10} **Averaging Time:** 24-h Avg **Mean (SD):** 13.31(6.13) $\mu g/m^3$ **Range (Min, Max):** (3.77, 52.17) **Monitoring Stations:** NR **Copollutant (correlation):** PM_{10}: $PM_{2.5}$ r = 0.80 $PM_{10-2.5}$ r = -0.11 CO r = 0.46 Coh r = 0.61 O_3 r = -0.08 NO_2 r = 0.54 SO_2 r = 0.61	**PM Increment:** : 7.9 $\mu g/m^3$ Rr Estimate (65 + Years) Dm Method: 1.014[0.998,1.029] Lag 0 1.016[0.998,1.034] 3 D Avg 0.988[0.970, 1.006] 5 D Avg 0.983[0.963, 1.004] 7 D Avg Time Series: 1.016[0.999, 1.033] Lag 0 1.015[0.996, 1.035] 3 D Avg 1.009[0.987, 1.032] 5 D Avg 1.009[0.983, 1.036] 7 D Avg Case-Crossover: 1.017[0.998, 1.036] Lag 0 1.015[0.993, 1.037] 3 D Avg 1.008[0.984, 1.033] 5 D Avg 1.003[0.976, 1.031] 7 D Avg
Reference: Fung al. (2005, 093262) **Period of Study:** Nov 1, 1995–Dec 31, 2000 **Location:** London, Ontario	Hospital Admissions **Outcome (ICD-9):** Asthma (493) and all other respiratory diseases (460-519) **Age Groups:** < 65 yrs 65 + yrs **Study Design:** Time series **N:** 5574 respiratory admissions **Statistical Analyses:** GAM with locally weighted regression smoothers (LOESS) **Covariates:** Maximum and minimum temp, humidity, day of the week, seasonal cycles, secular trends **Season:** NR **Dose-response Investigated?** No **Statistical Package:** S-Plus **Lags Considered:** Current to 3-day mean	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (min-max):** 38.0 (5-248) SD = 23.5 **Monitoring Stations:** 4 **Copollutant (correlation):** NO_2: r = 0.30 SO_2: r = 0.24 CO: r = 0.21 O_3: r = 0.53 COH: r = 0.29	**PM Increment:** 26 $\mu g/m^3$ **% Change in Daily Admission [CI]:** Age < 65 Current day mean: -0.9 [-6.8,5.4] 2-day mean: -1.3 [-8.5,6.6] 3-day mean: 1.9 [-6.5,11] Age 65 + Current day mean: 3.3 [-1.7,8.6] 2-day mean: 5 [-1.5,11.9] 3-day mean: 1.2 [-6.1,9.1]

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Galán et al. (2003, 087408)	Hospital Admissions	**Pollutant:** PM_{10}	**PM Increment:** 10 $\mu g/m^3$
Period of Study: 1995-1998	**Outcome (ICD):** Asthma (493)	**Averaging Time:** 24 h	**RR Estimate [CI]:**
Location: Madrid, Spain	**Age Groups:** all ages	**Mean (min-max):** 32.1 (11.2-108.6)	Single-pollutant
	Study Design: Time series	SD = 12.1	Current-day lag: 1.011 (0.980-1.042)
	N: 555,153 at-risk	**Monitoring Stations:** 13	1-day lag: 1.006 (0.976-1.037)
	Statistical Analyses: GAM, autoregressive Poisson regression	**Copollutant (correlation):** SO_2: r = 0.581	2-day lag: 1.008 (0.978-1.038)
	Covariates: temperature, relative humidity, pollen, year, day of the week, public holiday	NO_2: r = 0.717	3-day lag: 1.039 (1.010-1.068)
	Season: NR	O_3: r = -0.188	4-day lag: 1.027 (0.999-1.056)
	Dose-response Investigated? No	**Other variables:** *O.europaea*: r = -0.066	Adjustment for pollen (PM_{10} 3-day lag)
	Statistical Package: S-Plus	*Plantago sp.*: r = -0.202	*O. europaea*: 1.041 (1.011-1.071)
	Lags Considered: 0, 1, 2, 3, and 4-day	Poaceae: r = -0.132	*Plantago sp.*: 1.046 (1.017-1.076)
		Urticaceae: r = -0.104	Poaceae: 1.043 (1.015-1.073)
		Temp: r = -0.122	Urticaceae: 1.038 (1.009-1.068)
		Humidity: r = 0.119	All four: 1.045 (1.016-1.074)
Reference: Hajat et al. (2002, 030358)	Family Practice consultations	**Pollutant:** PM_{10}	**PM Increment:** All Year: 18
Period of Study: 1/1992-12-1994	**Outcome:** Upper Resp Disease (excluding allergic rhinitis) (460-3), (465), (470-5), (478)	**Averaging Time:** 24-H	Warm Season: 15
Location: London, England	**Age Groups:** 0-14,	**Mean (SD):** 28.5 (13.7) $\mu g/m^3$	Cold Season: 20
	15-64, > 65 yrs	Percentiles: 10th: 15.8	**% Change, Single Pollutant Models:** All Year: Ages 0-14: 2.0[-0.2, 4.2] Lag 3
	Study Design: Time series	90th: 46.5	Ages 15-64: 5.7[2.9, 8.6]
	N: 268,718-295,740 registered patients	**Monitoring Stations:** 1	Lag 2
	Statistical Analyses: Poisson regression, GAM, LOESS smoothers, default convergence criteria	**Copollutant:** NR	Ages > 65: 10.2[5.3, 15.3] Lag 2
	Covariates: long term trends, pollen counts, flu, meteorological variables		Warm Season: Ages 0-14: 1.1[-2.4, 4.8] Lag 3
	Season: All year		Ages 15-64: 6.0[2.7, 9.4]
	Dose-response Investigated? No		Lag 2
	Statistical Package: SPLUS		Ages > 65: 0.1[-7.7, 8.5] Lag 2
	Lags Considered: 2-3		Cold Season: Ages 0-14: 2.7[-0.1, 5.5] Lag 3
			Ages 15-64: 3.6[1.0, 6.4]
			Lag 2
			Ages > 65: 18.9[11.7, 26.7] Lag 2
			% Change, 2 Pollutant Models: 0-14 Yrs
			PM_{10} w/ NO_2: 3.8[1.6, 6.1]
			PM_{10} w/ O_3: 1.8[-0.4, 3.9]
			PM_{10} w/ SO_2: 2.0[-0.6, 4.6]
			15-65 Yrs
			PM_{10} w/ NO_2: 2.8[0.7, 4.9]
			PM_{10} w/ O_3: 4.8[2.6, 7.0]
			PM_{10} w/ SO_2: 4.8[2.2, 7.5]
			> 65 Yrs
			PM_{10} w/ NO_2: 4.6[0.5, 8.8]
			PM_{10} w/ O_3: 10.7[5.7, 16.0]
			PM_{10} w/ SO_2: 10.6[4.5, 17.1]

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Hanigan et al (2008, 156518) **Period of Study:** 1996–2005 (April–November of each year) **Location:** Darwin, Australia	**Outcome:** Cardiorespiratory Disease HA (ICD 9: 390-519 ICD 10: I00-99 & J00-99) **Age Groups:** NR **Study Design:** time series **N:** 8279 events **Statistical Analyses:** poisson regression **Covariates:** indigenous status, **Dose-response Investigated?** No **Statistical Package:** R **Lags Considered:** lags 0-3	**Pollutant:** PM_{10} **Averaging Time:** 24h **Mean (SD):** 21.2 (8.2) **Range:** 55.2 **Monitoring Stations:** 2 (monitored & modeled) **Copollutant:** NR **Co-pollutant Correlation** n/a	**PM Increment:** 10 $\mu g/m^3$ **Percent Change (Lower CI, Upper CI), lag:** Total Respiratory: 4.81 (-1.04, 11.01), lag 0 Total Resp., Indigenous: 9.40 (1.04, 18.46), lag 0 Total Resp., Non-Indigenous: 3.14 (-2.99, 9.66), lag Resp. Infection, Indigenous: 15.02 (3.73, 27.54), lag 3 Resp. Infection, Non-Indigenous: 0.67 (-7.55, 9.61), lag 3 Asthma Indigenous: 16.27 (3.55, 40.17), lag 1 Asthma Non-Indigenous: 8.54 (-5.60, 24.80), lag 1 *figure 3. percent change in hospital admissions per 10$\mu g/m^3$ increase in PM_{10}
Reference: Hanigan et al (2008, 156518) **Period of Study:** 1996–2005 (April–November of each year) **Location:** Darwin, Australia	Hospital Admissions/ED visits **Outcome (ICD-9 or ICD-10):** Daily emergency hospital admissions for total respiratory (ICD-9: 460 – 519 ICD-10: J00 – J99), asthma (ICD-9: 493 ICD-10: J45 – J47), COPD (ICD-9: 490 – 492, 494 – 496 ICD-10: J40 – J44, J47, J67), and respiratory infections (ICD-9: 461 – 466, 480 – 487, 514 ICD-10: J00 – J22). **Age Groups Analyzed:** All **Study Design:** Time series **N:** 8,279 hospital admissions **Statistical Analyses:** Poisson generalized linear models **Covariates:** indigenous status, time in days, temperature, relative humidity, day of the week, influenza epidemics, change between ICD editions, holidays, yearly population **Season:** April – November (corresponding to the dry season) **Dose-response Investigated?** No **Statistical package:** R version 2.3.1 **Lags Considered:** lag 0 -3	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (SD** **range):** 21.2 (8.2 55.2) **Monitoring Stations:** N/A (see notes) **Copollutant (correlation):** NR Other variables:	**PM Increment:** 10 $\mu g/m^3$ **Percent change [95% CI]:** Overall respiratory disease: Lag 0: 4.81 [-1.04, 11.01] Lag 0 (indigenous people): 9.40 [1.04, 18.46] Lag 0 (non-indigenous people): 3.14 [-2.99, 9.66] In unstratified analyses, the subgroups of respiratory infections, asthma, and COPD all had positive associations with PM_{10} Lag 0. Asthma: Lag 1 (indigenous people): 16.27 [-3.55 40.17] Lag 1 (non-indigenous people): 8.54 [-5.60, 24.80] Respiratory infections: Lag 3 (indigenous people): 15.02 [3.73, 27.54] Lag 3 (non-indigenous people) 0.67 [-7.55, 9.61] **Notes:** **Figure 3:** Associations between hospitalizations for non-indigenous and indigenous people with estimated ambient PM_{10}. **Summary of Figure 3:** Confidence intervals were wide, but indigenous people generally had stronger associations with PM_{10} than non-indigenous people. Daily PM_{10} exposure levels were estimated for the population of the city from visibility data using a previously validated models.

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Hapcioglu et al. (2006, 093263) **Period of Study:** Jan 1, 1997–Dec 31, 2001 **Location:** Istanbul, Turkey	Hospital Admissions **Outcome (ICD-9):** COPD (ICD: NR) **Age Groups:** NR **Study Design:** Time series **N:** 1586 patients **Statistical Analyses:** Multiple stepwise regression, Pearson correlation **Covariates:** Humidity, temperature, and pressure **Season:** summer, autumn, winter, spring **Dose-response Investigated?** No **Statistical Package:** SPSS **Lags Considered:** NR	**Pollutant:** PM_{10} **Averaging Time:** 1 month **Mean (SD):** NR **Monitoring Stations:** 1 **Copollutant:** NR	**PM Increment:** NR **Correlation with COPD:** r = 0.28 p = 0.03 Adj for temp: r = 0.16 p = 0.23 **Notes:** RRs only provided for season, not PM
Reference: Hwang and Chan (2002, 023222) **Period of Study:** 1998 **Location:** Taiwan	Clinic visits **Outcome:** LRI 466, 480-486 (acute bronchitis, acute bronchiolitis, pneumonia) **Age Groups:** 0-14 yrs, 15-64, 65+ yrs **Study Design:** Cluster analysis of small study areas **N:** 50 communities **Statistical Analyses:** GLM to model temporal patterns, hierarchical model to obtain estimates across 50 communities **Covariates:** day of week, temperature, dew point, summer/Winter **Season:** All **Dose-response Investigated?** Yes **Statistical Package:** NR **Lags Considered:** 0-2	**Pollutant:** PM_{10} **Averaging Time:** 24 H **Mean (SD):** 58.9 $\mu g/m^3$ (14.0) **Range (Min, Max):** 33.3, 83.1 $\mu g/m^3$ **PM Component:** **Monitoring Stations:** 59 **Notes:** Number Of Stations Estimated From Figure. **Copollutant:** NR	**PM Increment:** 10% Increase In PM_{10} (5.9 $\mu g/m^3$) Percent Change: 0-14 0.5% (-0.1, 0.8] Lag0 [-0.3, 0.3] Lag1 0.3 [0.0, 0.6] Lag2 15-64 0.6 [0.2, 0.9] Lag0 0.2 [-0.1, 0.5] Lag1 0.3 [0.0, 0.6] Lag2 65+ 0.8 [0.4, 1.1] Lag0 0.3 [-0.1, 0.6] Lag1 0.5 [0.1, 0.8] Lag2 All Ages 0.5 [0.2, N0.8] Lag0 [-0.3, 0.3] Lag1 0.3 [0.0, 0.6] Lag2

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Jaffe et al. (2003, 041957)	ED visits	**Pollutant:** PM_{10}	**PM Increment:** 50 $\mu g/m^3$
Period of Study: 7/1/91–6/30/96	**Outcome (ICD10):** Asthma (493)	**Averaging Time:** 24-H	**% Change**
Location: Cincinnati, Cleveland, Columbus, Ohio	**Age Groups:** Age 5-34 years	**Mean (SD):** Cincinnati: 43.0(16.4)	Asthma
	Study Design: Time-series	Cleveland: 60.8(28.4)	Cincinnati: -22%[-49,-19] Lag 3
	N: 4,416 recipients	Columbus: 37.4(16.3)	Cleveland: 12%[0,27] Lag 2
	Statistical Analyses: Poisson regression, GAM	**Range (Min, Max):** Cincinnati: (16,90)	Columbus: 32%[-6,-85] Lag 3
	Covariates: City, day of week, wk, yr, minimum temperature, dispersion parameter	Cleveland: (12,183)	**Ar Estimate [Lower Ci, Upper Ci]**
		Columbus: (7,87)	**Lag:**
	Season: June-August only	**Monitoring Stations:** 3	Asthma
	Dose-response Investigated? Yes	**Copollutant (correlation):** Cincinnati: PM_{10}	Cincinnati: PM_{10}: Nr
	Statistical Package: NR	O_3	Cleveland: PM_{10}: 1.32
	Lags Considered: 0-3 days	r = 0.42	Columbus: PM_{10}: 3.62
		NO_2	**Notes:** dose response was investigated by assessing the relationship between odds of ed visit by quintile of PM_{10}. Results are displayed in figure. "no consistent effects for all three cities were observed for PM_{10}." Rate ratios were also reported for each city.
		r = 0.36	
		SO_2	
		r = 0.31	
		Cleveland: PM_{10}	
		O_3	
		r = 0.42	
		NO_2	
		r = 0.34	
		SO_2	
		r = 0.29	
		Columbus: PM_{10}	
		O_3	
		r = 0.51	
		NO_2	
		r = Na	
		SO_2	
		r = 0.42	

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Jalaludin et al. (2004, 056595) **Period of Study:** Feb 1–Dec 31, 1994 **Location:** Sydney, Australia	Doctor Visits **Outcome (ICD- NR):** Respiratory symptoms (wheeze, dry cough, and wet cough), asthma medication use, and doctor visits for asthma **Age Groups:** Primary school children **Study Design:** Longitudinal cohort study **N:** 125 children **Statistical Analyses:** GEE logistic regression models **Covariates:** Temperature, humidity, daily pollen count, daily alternaria count, number of h spend outdoors, season **Season:** Autumn (Feb-Apr), winter (May-Aug), spring/summer (Sep-Dec) **Dose-response Investigated?** No **Statistical Package:** SAS **Lags Considered:** 0-2 days	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (SD):** 22.8 (13.8) **Monitoring Stations:** 4 **Copollutant (correlation):** O_3: r = 0.13 NO_2: r = 0.26 Other variables: Temp: r = 0.04 Humidity: r = -0.29 Total pollen: r = 0.04 Alternaria: r = 0.04	**PM Increment:** IQR ($\mu g/m^3$) Same day: 12.0 1-day lag: 12.02 2-day lag: 12.25 2-day avg: 11.15 5-day avg: 10.23 **OR Estimate [CI]:** Doctor Visits for Asthma Same day: 1.11 [1.04,1.19] 1-day lag: 1.10 [1.02,1.19] 2-day lag: 1.15 [1.06,1.24] 2-day avg: 1.11 [1.03,1.20] 5-day avg: 1.14 [0.98,1.31] **Prevalence of Doctor Visits for Asthma:** Quartile 1: 0.50 (mean PM = 12.4) Quartile 2: 0.38 (mean PM = 17.2) Quartile 3: 0.65 (mean PM = 23.0) Quartile 4: 0.63 (mean PM = 38.3) **Notes:** ORs and prevalence are also provided for wheeze, dry cough, wet cough, inhaled β2-agonist use, and inhaled corticosteroid use. None were statistically significant.

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Johnston et al. (2007, 155882) **Period of Study:** 2000, 2004, 2005 (April–November of each year) **Location:** Darwin, Australia	Hospital Admissions/ED visits **Outcome (ICD-10):** All respiratory conditions (J00 – J99), including asthma (J45 – 46), COPD (J40 – J44), and respiratory infections (J00 – J22). **Age Groups Analyzed:** All **Study Design:** Case-crossover **N:** 2466 emergency admissions **Statistical Analyses:** Conditional logistic regression **Covariates:** weekly influenza rates, temperature, humidity, days with rainfall > 5mm, public holidays, school holiday periods (for respiratory conditions only) **Season:** April – November (dry season) **Dose-response Investigated?** No **Statistical package:** NR **Lags Considered:** 0 – 3 days	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Median (IQR, 10^{th} – 90^{th} percentile, range):** 17.4 (13.6 – 22.3 10.3 – 27.7 1.1 – 70.0) **Monitoring Stations:** 1 **Copollutant (correlation):** NR	**PM Increment:** 10 $\mu g/m^3$ **OR Estimate [95% CI]: All respiratory conditions:** Lag 0: 1.08 [0.98 – 1.18] Lag 0 (indigenous): 1.17 [0.98 – 1.40] **COPD:** Lag 0: 1.21 [1.0 – 1.47] Lag 0 (indigenous): 1.98 [1.10 – 3.59] **Asthma:** Lag 0: 1.14 [0.90 – 1.44] **Asthma + COPD:** Lag 0: 1.19 [1.03 – 1.38] **Notes: Figure 1:** Adjusted OR and 95% CI for hospital admissions for all respiratory conditions per 10 $\mu g/m^3$ rise in PM_{10} for the same day and lags up to 3 days, overall and stratified by indigenous status. **Summary of Figure 1 results:** Marginally significant positive association at Lag 0 in overall study population. Larger marginally significant positive association among indigenous people. **Figure 2**: OR and 95% CI for hospital admissions for COPD. **Summary of Figure 2 results:** Marginally significant positive associations at Lag 0 and Lag 1 in overall study population and among non-indigenous people. Large, statistically significant positive association at Lag 0 for indigenous people, with smaller, non-significant positive associations at Lag 1 and Lag2. **Figure 3:** OR and 95% CI for hospital admissions for asthma. **Summary of Figure 3 results:** Positive, non-significant (sometime marginally significant) associations at Lag 0, Lag 2, and Lag 3 for overall population and indigenous status strata. **Figure 4:** OR and 95% CI for hospital admissions for respiratory infections. **Summary of Figure 4 results:** Negative associations at Lag 2 and Lag 3 in all population strata.

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Kim et al. (2007, 092837) **Period of Study:** 2002 **Location:** Seoul, Korea	Ed Visits **Outcome (ICD10):** Asthma (J45), (J46) **Age Groups:** All Ages **Study Design:** Cass-Crossover **N:** 92,535 Visits **Statistical Analyses:** Conditional Logistic Regression, Relative Effect Modification (Rem) **Covariates:** Time Trend, Season, Daily Mean Temperature, Relative Humidity, Air Pressure. Sep As Modifier Of Air Pollution Asthma Visit Association. **Season:** All Year **Dose-response Investigated?** No **Statistical Package:** Nr **Lags Considered:** 0-2 Days	**Pollutant:** PM_{10} **Averaging Time:** 8-H **Mean (SD):** Daily Concentration: 67.6 (39.0) $\mu g/m^3$ Relevant Exposure Term (Difference Between Concentration On Event Day And Mean Of Concentrations On Control Days): 26.0 (19.7) **Percentiles:** 50th(Median): Daily Concentration: 61.9 Relevant Exposure Term: 21.6 **Range (Min, Max):** Daily Concentration: (4.9, 302.0) Relevant Exposure Term: (0.0, 143.1) **Monitoring Stations:** 3 **Copollutant:** Nr	**PM Increment:** 47.4 $\mu g/m^3$ **Rr Estimate For Asthma (Stratified By Sep):** Individual Level Sep: Quintile 1–1.06[1.02, 1.09] Quintile 2–1.07[1.04, 1.10] Quintile 3–1.06[1.03, 1.10] Quintile 4–1.03[0.99, 1.07] Quintile 5–1.10[1.05, 1.14] Regional Level Sep: Quintile 1–1.04[0.99, 1.10] Quintile 2–1.03[1.00, 1.07] Quintile 3–1.05[1.03, 1.08] Quintile 4–1.06[1.02, 1.10] Quintile 5–1.09[1.06, 1.13] Total-1.06[1.04, 1.08], 3 D Ma **Notes:** Relative Effect Modification (Rem) Estimates Presented In Paper.
Reference: Ko et al. (2007, 091639) **Period of Study:** 1/2000-12/2004 **Location:** Hong Kong, China	Ed Visits **Outcome (ICD-9):** COPD: chronic bronchitis (491), emphysema (492), chronic airway obstruction (496) **Age Groups:** All Ages **Study Design:** Time Series **N:** 15 hospitals, 119,225 admissions **Statistical Analyses:** Poisson regression, gam with stringent convergence criteria, aphea2 protocol. **Covariates:** time trend, season, temperature, humidity, other cyclical factors, day, day of wk, holidays **Season:** All year, interactions with season tested **Dose-response Investigated?** No **Statistical Package:** Splus 4.0 **Lags Considered:** 0-5 days	**Pollutant:** PM_{10} **Averaging Time:** 24-H **Mean (SD):** 50.1(23.9) $\mu g/m^3$ **Percentiles:** 25th: 31.9 50th(Median): 44.5 75th: 64.1 **Range (Min, Max):** (13.6, 172.2) **Monitoring Stations:** 14 Stations **Copollutant (correlation):** PM_{10}: SO_2 r = 0.436 NO_2 r = 0.229 O_3 r = 0.421 $PM_{2.5}$ r = 0.952	**PM Increment:** 10 $\mu g/m^3$ **Rr Estimate** COPD: 1.003[1.000, 1.005] Lag 0 1.005[1.002, 1.007] Lag 1 1.010[1.007, 1.012] Lag 2 1.011[1.008, 1.013] Lag 3 1.008[1.006, 1.011] Lag 4 1.007[1.004, 1.009] Lag 5 1.005[1.002, 1.008] Lag 0-1 1.011[1.008, 1.014] Lag 0-2 1.016[1.013, 1.019] Lag 0-3 1.020[1.017, 1.024] Lag 0-4 1.024[1.021, 1.028] Lag 0-5

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Ko et al. (2007, 091639) **Period of Study:** 1/2000-12/2004 **Location:** Hong Kong, China	Hospital Admission **Outcome (ICD-9):** Asthma (493) **Age Groups:** All, 0-14, 15-56, 65+ **Study Design:** Time series **N:** 69,716 admissions, 15 hospitals **Statistical Analyses:** Poisson regression, with GAM with stringent convergence criteria. **Covariates:** Time trend, season, temperature, humidity, other cyclical factors **Season:** All year, evaluated effect of season in analysis **Dose-response Investigated?** No **Statistical Package:** SPLUS 4.0 **Lags Considered:** 0-5 days	**Pollutant:** PM_{10} **Averaging Time:** 24-h **Mean (SD):** 52.5(27.1) $\mu g/m^3$ **Percentiles: 25th:** 30.9 **50th(Median):** 47.1 **75th:** 68.8 **Range (Min, Max):** (13.4, 198.9) **Monitoring Stations:** 14 stations **Copollutant (correlation):** PM_{10}: SO_2 r = 0.436 NO_2 r = 0.761 O_3 r = 0.600 $PM_{2.5}$ r = 0.956	**PM Increment:** 10.0 $\mu g/m^3$ **RR Estimate:** Asthma (Single-pollutant model): 1.006[1.003, 1.010] lag 0 1.005[1.002, 1.009] lag 1 1.005[1.002, 1.009] lag 2 1.008[1.005, 1.012] lag 3 1.006[1.002, 1.009] lag 4 1.006[0.999, 1.006] lag 5 1.008[1.004, 1.012]; lag 0-1 1.012[1.008, 1.016] lag 0-2 1.015[1.011, 1.019] lag 0-3 1.018[1.013, 1.022] lag 0-4 1.019[1.015, 1.024] lag 0-5 Asthma by age group 0-14: 1.023[1.015, 1.031] lag 0-5 14-65: 1.014[1.006, 1.022] lag 0-5 >65: 1.015[1.009, 1.022] lag 0-4 Asthma–Effect of season: 1.148[1.051, 1.245] lag 0-5
Reference: Kuo et al. (2002, 036310) **Period of Study:** 1 yr **Location:** central Taiwan	Hospital Admissions **Outcome (ICD-NR):** Asthma **Age Groups:** 13-16 yrs **Study Design:** Cohort **N:** 12,926 **Statistical Analyses:** Multiple logistic regression, Pearson correlation **Covariates:** Sex, age, residential area, level of parents' education, number of cigarettes smoked by smokers in the family, incense burning, frequency of physical activity **Season:** NR **Dose-response Investigated?** No **Statistical Package:** SAS **Lags Considered:** NR	**Pollutant:** PM_{10} **Averaging Time:** 1 h **Mean (min-max):** NR Range: (54.1-84.3) **Monitoring Stations:** 8 **Copollutant:** Values NR **Notes:** Author states that a positive correlation was found between NO_2 and PM_{10}	**PM Increment:** NR **OR Estimate:** PM_{10} < 65.9 $\mu g/m^3$–referent PM_{10} > 65.9 $\mu g/m^3$ Crude OR: 0.837 Adj OR: 0.947 95% CI: (0.640,1.401)

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Langley-Turnbaugh et al. (2005, 093269) **Period of Study:** 2000-2001 **Location:** Portland, Bridgeton, and Presque Isle, Maine	Hospital Admissions **Outcome (ICD-9):** Asthma (493xx) **Age Groups:** 0-18 yrs, 19+ yrs **Study Design:** Time series **N:** NR **Statistical Analyses:** NR **Covariates:** NR **Season:** Winter, spring, summer, fall **Dose-response Investigated?** No **Statistical Package:** NR **Lags Considered:** NR **Notes:** Hospital admissions were used to determine seasonality of asthma admissions so that PM components from those time periods could be analyzed	**Pollutant:** PM_{10} **Averaging Time:** NR **Mean (min-max):** NR **Monitoring Stations:** NR **Copollutant:** NR	**PM Increment:** NR **RR Estimate [CI]:** NR **Notes:** Portland filters contained more PM in the winter (Jan) and Bridgeton filters contained more PM in the spring (May) study analyzed metal components of PM_{10} (Mn, Cu, Pb, As, V, Ni, Al) Clinical data shows a strong peak in fall and weaker peaks in Jan and May for asthma admissions
Reference: Lee et al. (2002, 034826) **Period of Study:** 12/1/1997-12/31/1999 **Location:** Seoul, Korea	Hospital Admissions **Outcome (ICD10):** Asthma, J45, J46, **Age Groups:** Children $<$ 15 years **Study Design:** Time-Series **N:** 822 d, 6,436 admissions **Statistical Analyses:** Poisson regression, GAM, LOESS smoothers. **Covariates:** Days of the week, temperature, humidity **Season:** All **Dose-response Investigated?** No **Statistical Package:** NR **Lags Considered:** 0-5, 0-1 moving averages for 1-2, 2-3, and 3-4 days	**Pollutant:** PM_{10} **Averaging Time:** 24-h **Mean (SD):** 64.0 (31.8) $\mu g/m^3$ **Percentiles: 25th:** 40.5 $\mu g/m^3$ **50th(Median):** 59.1 $\mu g/m^3$ **75th:** 80.9 $\mu g/m^3$ **Range (Min, Max):** NR **Monitoring Stations:** 27 **Notes: Copollutant (correlation):** PM_{10}-SO_2: 0.585 PM_{10}-NO_2: 0.738 PM_{10}-O_3: 0.106 PM_{10}-CO: 0.598	**PM Increment:** IQR: 40.4 $\mu g/m^3$ **RR Estimate:** Single **Pollutant:** 1.07 (1.04, 1.11) lag 1 Two pollutant models: +SO_2: 1.05 (1.01, 1.09) lag 1 +NO_2: 1.03 (0.99, 1.07) lag 1 +O_3: 1.06 (1.03, 1.10) lag 1 +CO: 1.04 (1.00, 1.08) lag 1 Three pollutant models: +O_3 + CO: 1.02 (0.98, 1.06), lag 1 Four pollutant models: +O_3 + CO +SO_2: 1.02 (0.98, 1.06), lag 1 Five pollutant model: 1.016 (0.975, 1.059) lag 1 **Notes:** Investigated the association between outdoor air pollution and asthma attacks in children $<$ 15 yrs.

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Lee et al. (2006, 090176) **Period of Study:** 1/1997-12/2002 **Location:** Hong Kong, China	**Hospital Admission** **Outcome:** Asthma (493) **Age Groups:** < 18 years **Study Design**: Time series **N:** 26,663 asthma admissions for asthma and 5821 admissions for influenza **Statistical Analyses:** Poisson regression, GAM **Covariates:** Temperature, atmospheric pressure, relative humidity **Season:** All **Dose-response Investigated?** No **Statistical Package:** SAS 8.02 **Lags Considered:** 0-5 **Notes:** Controls were admissions for influenza ICD9 487	**Pollutant:** PM_{10} **Averaging Time:** 24-hs **Mean (SD):** 56.1 (24.2) **Percentiles: 25th:** 37.3 50th(Median): 51.1 75th: 70.7 **Monitoring Stations:** 10 **Notes: Copollutant (correlation):** PM_{10}-$PM_{2.5}$: 0.90 PM_{10}-SO_2: 0.39 PM_{10}-NO_2: 0.80 PM_{10}-O_3: 0.60	**PM Increment:** IQr = 33.4 **Percent Increase:** Single pollutant model: 4.97 [2.96, 7.03], lag 0 5.71 [3.78, 7.68], lag 1 6.40 [4.51, 8.32], lag 2 7.25 [5.38, 9.16], lag 3 7.45 [5.58, 9.35], lag 4 5.96 [4.11, 7.85], lag 5 Multipollutant model (SO_2, CO, NO_2, O_3) 3.67 [1.52,5.86] lag4
Reference: Lin et al. (2005, 087828) **Period of Study:** 1998-2001 **Location:** Toronto, North York, East York, Etobicoke, Scarborough, and York (Canada)	Hospital Admissions **Outcome (ICD-9):** Respiratory infections including laryngitis, tracheitis, bronchitis, bronchiolitis, pneumonia, and influenza (464, 466, 480-487) **Age Groups:** 0-14 yrs **Study Design:** Bidirectional case-crossover **N:** 6782 respiratory infection hospitalizations **Statistical Analyses:** Conditional logistic regression (Cox proportional hazards model) **Covariates:** Daily mean temp and dew point temp **Season:** NR **Dose-response Investigated?** No **Statistical Package:** SAS 8.2 PHREG procedure **Lags Considered:** 1-7 day averages	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (min-max):** 20.41 (4.00-73.00) SD = 10.14 **Monitoring Stations:** 4 **Copollutant (correlation):** $PM_{2.5}$: r = 0.87 $PM_{10-2.5}$: r = 0.76 CO: r = 0.10 SO_2: r = 0.48 NO_2: r = 0.54 O_3: r = 0.54	**PM Increment:** 12.5 $\mu g/m^3$ **OR Estimate [CI]:** Adjusted for weather 4 day avg: 1.22 [1.10,1.34] 6 day avg: 1.25 [1.11,1.40] Adj for weather and other gaseous pollutants 4 day avg: 1.14 [0.99,1.32] 6 day avg: 1.20 [1.01,1.42] **Notes:** OR's were also categorized into "Boys" and "Girls," yielding similar results
Reference: Lin et al, (2008, 126812) **Period of Study:** 1991-2001 **Location:** New York State, US	**Outcome:** Respiratory hospital admissions (ICD-9 466, 490-493, 496 **Study Design:** Time-series **Covariates:** Demographic characteristics, PM_{10}, meteorological conditions, day of the week, seasonality, long term trends and different lag periods **Statistical Analysis:** GAM and case-crossover design at the regional level and Bayesian hierarchical model at the state level **Age Groups:** Children 0-17 years	**Pollutant:** O_3 (PM_{10} is secondary) **Averaging Time:** 24h **Mean (SD) Unit:** 19.56 (10.92) $\mu g/m^3$ **Range (Min, Max):** 1.0, 90.00 **Copollutant (correlation):** Given in Figure 3	All PM_{10} results are given in Figure 3

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Lin et al. (2002, 026067) **Period of Study:** Jan 1, 1981–Dec 31, 1993 **Location:** Toronto	Hospital Admissions **Outcome (ICD-9):** Asthma (493) **Age Groups:** 6-12 yrs **Study Design:** Uni- and bi-directional case-crossover (UCC, BCC) and time-series (TS) **N:** 7,319 asthma admissions **Statistical Analyses:** Conditional logistic regression, GAM **Covariates:** Maximum and minimum temp, avg relative humidity **Season:** Apr-Sep, Oct-Mar **Dose-response Investigated?** No **Statistical Package:** NR **Lags Considered:** 1-7 day averages	**Pollutant:** PM_{10} **Averaging Time:** 6 days (predicted daily values) **Mean (min-max):** 30.16 (3.03-116.20) SD = 13.61 **Monitoring Stations:** 1 **Copollutant (correlation):** $PM_{2.5}$: r = 0.87 $PM_{10-2.5}$: r = 0.83 CO: r = 0.38 SO_2: r = 0.44 NO_2: r = 0.52 O_3: r = 0.44	**PM Increment:** 14.8 $\mu g/m^3$ **RR Estimate [CI]:** Adj for weather and gaseous pollutants BCC 5 day avg: 0.99 [0.90,1.09] BCC 6 day avg: 1.01 [0.90,1.12] TS 5 day avg: 1.03 [0.95,1.11] TS 6 day avg: 1.02 [0.94,1.11] Boys–adj for weather UCC 1 day avg: 1.10 [1.04,1.17] UCC 2 day avg: 1.10 [1.02,1.17] BCC 1 day avg: 1.04 [0.98,1.09] BCC 2 day avg: 1.01 [0.95,1.08] TS 1 day avg: 1.03 [0.99,1.07] TS 2 day avg: 1.01 [0.96,1.05] Girls–adj for weather UCC 1 day avg: 1.07 [0.99,1.16] UCC 2 day avg: 1.15 [1.04,1.26] BCC 1 day avg: 0.99 [0.92,1.06] BCC 2 day avg: 1.03 [0.95,1.12] TS 1 day avg: 0.99 [0.94,1.04] TS 2 day avg: 1.02 [0.96,1.08] **Notes:** The author also provides RR using UCC, BCC, and TS analysis for female and male groups for days 3-7, yielding similar results
Reference: Linares et al. (2006, 092846) **Period of Study:** Jan 1995-Dec 2000 **Location:** Madrid, Spain	**Outcome:** Respiratory system diseases 460-519, bronchitis 460-496, pneumonia 480-487 **Age Groups:** < 10 years **Study Design:** Time series **N:** ~ 15,000 admissions, 2192 days **Statistical Analyses:** Poisson regression, dummy variables to adjust for season and weather **Covariates:** Temperature, difference in barometric pressure, relative humidity, pollen counts, influenza epidemics **Season:** All **Dose-response Investigated?** Yes **Statistical Package:** S-Plus 2000 **Lags Considered:** 0-13	**Pollutant:** PM_{10} **Averaging Time:** 24-hs **Mean (SD):** 33.4 $\mu g/m^3$, (13.7) **Range (Min, Max):** 6, 109 $\mu g/m^3$ **Monitoring Stations:** 24 **Notes: Copollutant (correlation):** PM_{10}-SO_2: 0.532 PM_{10}-O_3: -0.289 PM_{10}-NO_X: 0.721 PM_{10}-NO_2: 0.711	**PM Increment:** 10 $\mu g/m^3$ **RR Estimate** Bronchitis 1.09 [1.01, 1.16] lag 2 **AR% Estimate** Bronchitis 7.9 [CI NR] lag2 **Notes:** Only statistically significant relative and attributable risks were presented by the authors. The authors conducted multivariate modeling using a linear term to represent PM_{10}. They also report an apparent estimated PM_{10} effect threshold of 60 $\mu g/m^3$, based on examination of a scatter plot of respiratory emergency hospital admissions and PM_{10} levels.

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Luginaah, et al. (2005, 057327) **Period of Study:** Apr 1995-Dec 2000 **Location:** Windsor, Ontario, Canada	**Hospital Admission/ED:** admission **Outcome:** All respiratory: 460-519 **Age Groups:** All, 0-14, 15-64, and > 65 **Study Design:** Times-series, bi-directional case-crossover **N:** 4214 admissions **Statistical Analyses:** Poisson regression, GAM w/ stringent convergence criteria or natural splines, conditional logistic regression **Covariates:** Age, sex Maximum & minimum temperature, change in barometric pressure from previous day **Season:** All **Dose-response Investigated?** No **Statistical Package:** S-Plus **Lags Considered:** 1-3	**Pollutant:** PM_{10} **Averaging Time:** 24 h maximum **Mean (SD):** 50.6 ,(35.5) **Range (Min, Max):** 9, 349 **Monitoring Stations:** 4 **Notes: Copollutant (correlation):** PM_{10}-NO_2: 0.33 PM_{10}-SO_2: 0.22 PM_{10}-CO: 0.21 PM_{10}-O_3: 0.33	**PM Increment:** Interquartile range (75th-25th) 31 $\mu g/m^3$ **RR Estimates (Time Series)** **All Age Groups Females** 0.996 [0.950, 1.044], lag 1 1.015 [0.963, 1.069], lag 2 1.022 [0.968, 1.078], lag 3 **All Age Groups Males** 1.008 [0.965, 1.054], lag 1 1.036 [0.986, 1.089], lag 2 1.027 [0.974, 1.083], lag 3 **RR Estimates (Case Crossover)** **All Age Groups Females** 1.034 [0.974, 1.098], lag 1 1.045 [0.972, 1.124], lag 2 1.054 [0.970, 1.145], lag 3 **All Age Groups Males** 0.997 [0.942, 1.056], lag 1 1.022 [0.953, 1.097], lag 2 1.008 [0.930, 1.092], lag 3 **Notes:** Results, stratified by age group available in manuscript.
Reference: Martins et al. (2002, 035059) **Period of Study:** May 1996-Sep 1998 **Location:** Sao Paulo, Brazil	Hospital Admission/ED: ER visits **Outcome (ICD10):** Chronic lower respiratory disease (CLRD) (40-47) includes chronic bronchitis, emphysema, other COPDs, asthma, bronchiectasia **Age Groups:** > 64 years **Study Design:** Time series **N:** 712 for CLRD 1 hospital **Statistical Analyses:** Poisson regression GAM, LOESS smoothers, no mention of stringent criteria **Covariates:** Day of week, time minimum temperature, relative humidity **Season:** All **Statistical Package:** S-Plus **Lags Considered:** 2-7 3 d ma	**Pollutant:** PM_{10} **Averaging Time:** daily **Mean (SD):** 60.0 $\mu g/m^3$, (26.3) **Range (Min, Max):** 22.8. 186.5 $\mu g/m^3$ **Unit (i.e. $\mu g/m^3$):** $\mu g/m^3$ **PM Component:** None **Monitoring Stations:** 12 **Notes: Copollutant (correlation):** PM_{10}-CO: 0.73 PM_{10}- NO_2: 0.83 PM_{10}-SO_2: 0.72 PM_{10}-O_3: 0.35	**PM Increment:** 1 $\mu g/m^3$ Regression Coefficients (SE): 0.0024 (0.0023), 6 d ma **Notes:** % Increase (SD) for ER visits per 2435 $\mu g/m^3$ (IQR) PM_{10} (lag 6 d ma) presented graphically in text.

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Masjedi et al. (2003, 052100) **Period of Study:** Sep 1997–Feb 1998 **Location:** Tehran, Iran	Hospital Admissions **Outcome (ICD-9):** Acute asthma and COPD exacerbations (ICD: NR) **Age Groups:** NR **Study Design:** Time series **N:** 355 patients **Statistical Analyses:** Multiple stepwise regression, autoregression method (time series), Pearson correlation **Covariates:** NR **Season:** NR **Dose-response Investigated?** No **Statistical Package:** NR **Lags Considered:** 3, 7, and 10 day mean	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (min-max):** 108.41 (14.5-506.60) SD = 59.55 **Monitoring Stations:** 3 **Copollutant:** NR	**PM Increment:** NR **Results:** Time-series analysis Asthma: β = 0.002 p = 0.32 COPD: β = 0.004 p = 0.02 Total Acute Resp Conditions: β = 0.006 p = 0.27 Correlation of 3-day mean Asthma: r = -0.21 β = -0.16 p = 0.08 Correlation of weekly mean Asthma: r = -0.27 β = -0.008 p = 0.12 Correlation of 10-day mean Asthma: r = -0.38 β = -0.066 p = 0.089
Reference: McGowan et al. (2002, 030325) **Period of Study:** Jun 1988–Dec 1998 **Location:** Christchurch, New Zealand	Hospital Admissions **Outcome (ICD-9):** Pneumonia (480-487), acute respiratory infections (460-466), chronic lung diseases (491-492, 494-496), asthma (493) **Age Groups:** < 15 yrs, 15-64, 65+ **Study Design:** Time series **N:** 20,938 admissions **Statistical Analyses:** GAM with log link, Linear Regression Model **Covariates:** Wind speed, relative humidity, temperature **Season:** NR **Dose-response Investigated?** No **Statistical Package:** S-PLUS **Lags Considered:** 0-6 days	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (min-max):** 25.17 (0-283) SD = 25.49 **Monitoring Stations:** 1 **Copollutant:** NR	**PM Increment:** 14.8 $\mu g/m^3$ (IQR) **% Increase [CI]:** Respiratory Admissions (2-day lag) 0-14 yrs: 3.62 [2.34,4.90] 15-64 yrs: 3.39 [1.85,4.93] 65+ yrs: 2.86 [1.23,4.49] All ages: 3.37 [2.34,4.40] Overall Acute respiratory infections: 4.53 [2.82,6.24] Pneumonia/influenza: 5.32 [3.46,7.18] Chronic lung diseases: 3.95 [2.15,5.75] Asthma: 1.86 [0.48,.3.24] Total Respiratory Admissions Same day lag: 2.52 [1.49,3.55] 1-day lag: 2.56 [1.53,3.59] 2-day lag: 3.37 [2.34,4.40] 3-day lag: 3.09 [2.06,4.12] 4-day lag: 3.13 [2.10,4.16] 5-day lag: 3.21 [2.18,4.24]; 6-day lag: 3.09 [2.06,4.12]

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Medina-Ramon et al (2006, 087721) **Period of Study:** 1986-99 **Location:** 36 US Cities	**Outcome:** 490-496, except 493 (COPD), 480-487 (Pneumonia) **Age Groups:** 65 + (US Medicare beneficiaries) **Study Design:** Case crossover **N:** 578,006 COPD admissions 1,384,813 Pneumonia admissions **Statistical Analyses:** Conditional logistic regression, Meta-analysis using REML random effects models **Covariates:** Mean and variance of daily summer apparent temperature index, % 65+ living in poverty,% households with central air-conditioning mortality rate for emphysema among 65+(surrogate for smoking history), % PM_{10} from traffic **Season:** Warm(May –Sepnd Cold(Oct-Apr) **Dose-response Investigated?** No **Statistical Package:** SAS STATA **Lags Considered:** 0-1 days	**Pollutant:** PM_{10} **Averaging Time:** 24 h avg **Mean (SD):** 30.4 $\mu g/m^3$ (5.1) **Monitoring Stations:** at least one per city **Notes:** PM_{10} measurements made every 2, 3 or 6 days depending on the city. **Copollutant:** NR	**PM Increment:** 10 $\mu g/m^3$ **% change [Lower CI, Upper CI]** **lag:** COPD warm season 0.81(0.22,1.41) at lag 0 1.47(0.93,2.01) at lag 1 COPD cold season 0.06(-0.40,0.51) at lag 0 0.10(-0.30,0.49) at lag 1 Pneumonia warm season 0.84 (0.50,1.19) at lag 0 0.79 (0.45,1.13) at lag 1 Pneumonia cold season 0.30 (0.07,0.53) at lag 0 0.14 (-0.17,0.45) at lag 1
Reference: Meng et al., (2007, 093275) **Period of Study:** Nov 2000–Sep 2001 **Location:** Los Angeles and San Diego counties, California	**Outcome (ICD-NR):** Poorly controlled asthma defined as (1) daily or weekly asthma symptoms or (2) at least 1 ED visit or hospitalization due to asthma over the past 12 months **Age Groups:** > 18 yrs **Study Design:** Time series **N:** 1609 asthma patients **Statistical Analyses:** Logistic regression **Covariates:** Age, sex, race/ethnicity, poverty level, insurance status, smoking behavior, employment, asthma medication use, and county **Season:** NR **Dose-response Investigated:** No **Statistical Package:** NR **Lags Considered:** NR	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (25-75th percentile):** NR **Monitoring Stations:** NR **Copollutant (correlation):** $PM_{2.5}$: r = 0.84 O_3: r = -0.72 NO_2: r = 0.83 CO: r = 0.42 Other variables: Traffic: r = 0.14	**PM Increment:** 10 $\mu g/m^3$ **OR Estimate [CI]:** All Adults: 1.08 [0.82,1.43] 18-64 yrs: 1.14 [0.84,1.55] 65+: 0.84 [0.41,1.73] Men: 0.72 [0.42,1.21] Women: 1.38 [0.99,1.94] Exposure above 44.01 $\mu g/m^3$ (annual concentration) All Adults: 1.56 [0.96,2.52] 18-64 yrs: 1.40 [0.81,2.41] 65+: 2.23 [0.60,8.27] Men: 0.80 [0.27,2.41] Women: 2.06 [1.17,3.61] **Notes:** This study focused more on the relation between poorly controlled asthma and traffic density.

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Middleton et al. (2008, 156760) **Period of Study:** 1995 – 1998, 2000 - 2004 **Location:** Nicosia, Cyprus	Hospital Admissions/ED visits **Outcome (ICD-NR):** Hospital admissions for all respiratory disease (ICD-10: J00 – J99). **Age Groups Analyzed:** All, also stratified by age (< 15 vs. > 15 years) **Study Design:** Time series **N: Statistical Analyses:** generalized additive Poisson models **Covariates: seasonality, day of the week, long- and short-term trend, temperature, relative humidity** **Season:** NR **Dose-response Investigated:** No **Statistical package:** STATA SE 9.0, and the MGCV package in the R software (R 2.2.0) **Lags Considered:** lag 0 -2 days	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (SD** **median** **5% - 95%** **range):** **Cold:** 57.6 (52.5 50.8 20.0 – 103.0 5.0 – 1370.6) **Warm:** 53.4 (50.5 30.7 32.0 – 77.6 18.4 – 933.5) **Monitoring Stations:** 2 **Copollutant (correlation):** NR Other variables:	**PM Increment:** 10 $\mu g/m^3$, and across quartiles of increasing levels of PM_{10} **Percentage increase estimate [CI]: All age/sex groups (Lag 0):** All admissions: 0.85 (0.55, 1.15) Respiratory (all): 0.10 (-0.91, 1.11) Respiratory (cold months): -0.33 (-1.47, 0.82) Respiratory (warm months): 1.42 (-0.42, 3.31) CVD + RD: 0.56 (-0.21, 1.34) **Nicosia residents (Lag 0):** Respiratory (all): 0.25 (-0.84, 1.36) Respiratory (cold months): -0.22 (-1.45, 1.02) Respiratory (warm months): 1.80 (-0.22, 3.85) CVD + RD: 0.38 (-0.47, 1.23) **Males (Lag 0):** All admissions: 0.96 (0.54, 1.39) Respiratory (all): -0.06 (-1.37, 1.26) Respiratory (cold months): -0.16 (-1.76, 1.46) Respiratory (warm months): 1.10 (-1.47, 3.74) CVD + RD: 0.63 (-0.34, 1.62) **Females (Lag 0):** All admissions: 0.74 (0.31, 1.18) Respiratory (all): 0.39 (-1.21, 2.02) Respiratory (cold months): -0.26 (-2.18, 1.70) Respiratory (warm months): 3.27 (-0.00, 6.65) CVD + RD: 0.59 (-0.68, 1.87) **Aged < 15 years (Lag 0):** All admissions: 0.47 (-0.13, 1.08) Respiratory (all): -0.35 (-1.77, 1.08) Respiratory (cold months): -0.31 (-2.02, 1.42) Respiratory (warm months): -0.59 (-3.53, 2.45) **Aged > 15 years (Lag 0):** All admissions: 0.98 (0.63, 1.33) Respiratory (all): 0.59 (-0.87, 2.07) Respiratory (cold months): 0.02 (-1.76, 1.83) Respiratory (warm months): 3.89 (1.05, 6.80)

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Moore et al, 2008 **Period of Study:** 1983-2000 **Location:** California's South Coast Air Basin	**Outcome:** Hospital admissions for asthma (ICD-9 493) **Study Design:** Time-series **Covariates:** Income, demographic and residential variables **Statistical Analysis:** HRMSM **Age Groups:** Children ages 0-19 years	**Pollutant:** O_3 (PM_{10} secondary) **Averaging Time:** Quarterly **Mean (SD) Unit:** NR **Range (Min, Max):** NR **Copollutant (correlation):** 1hr O_3: 0.52 8hr O_3: 0.46 24hr NO_2: 0.53 24hr CO: 0.36 24hr SO_2: 0.13	Results given are for O_3
Reference: Nascimento et al. (2006, 093247) **Period of Study:** May 1, 2000–Dec 31, 2001 **Location:** São Jose dos Campos, Brazil	Hospital Admissions **Outcome (ICD-10):** Pneumonia (J12-J18) **Age Groups:** 0-10 yrs **Study Design:** Time series **N:** 1265 admissions **Statistical Analyses:** GAM, Poisson regression **Covariates:** Temperature, humidity **Season:** NR **Dose-response Investigated?** Yes **Statistical Package:** S-Plus, SPSS **Lags Considered:** 0-7 days	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (min-max):** 40.2 (3.4-196.6) SD = 26.9 **Monitoring Stations:** 2 **Copollutant (correlation):** SO_2: r = 0.30 O_3: r = 0.09 Other variables: Admissions: r = 0.21 Temp: r = -0.14 **Notes:** All $p < 0.05$	**PM Increment:** 24.7 $\mu g/m^3$ **Regression coefficients (SE):** Same day: -0.00053 (0.00125) 1-day lag: 0.00029 (0.00057) 2-day lag: 0.00089 (0.00069) 3-day lag: 0.00122 (0.00053)* 4-day lag: 0.00126 (0.00055)* 5-day lag: 0.00098 (0.00071) 6-day lag: 0.00035 (0.00056) 7-day lag: -0.00067 (0.00123) *$p < 0.05$ **Notes:** Percent increase over all lag days is displayed in Fig 2
Reference: Neuberger et al. (2004, 093249) **Period of Study:** 1999-2000 (1 yr period) **Location:** Vienna and Lower Austria	Hospital Admissions **Outcome (ICD-9):** Bronchitis, emphysema, asthma, bronchiectasis, extrinsic allergic alveolitis, and chronic airway obstruction (490-496) **Age Groups:** 3.0-5.9 yrs 7-10 yrs 65+ **Study Design:** Time series **N:** 366 days (admissions NR) **Statistical Analyses:** GAM **Covariates:** SO_2, NO, NO_2, O_3, temperature, humidity, and day of the week **Season:** NR **Dose-response Investigated?** Yes **Statistical Package:** S-Plus 2000 **Lags Considered:** 0-14 days	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Maximum daily mean:** Vienna: 105 Rural area: NR **Monitoring Stations:** NR **Copollutant:** NR	**PM Increment:** 10 $\mu g/m^3$ **Log Relative Rate Estimate (p-value):** Vienna Male: 2 day lag = 4.217 (0.030) Association with tidal lung function: β = -1.067 (p-value = 0.241) **Notes:** Effect parameters with significant coefficients for respiratory health included: male sex, allergy, asthma in family, and traffic for Vienna and age, allergy, asthma in family, and passive smoking for the rural area. Effect parameters with significant coefficients for log asthma score were allergy, asthma in family, and rain for Vienna and allergy, asthma in family, and passive smoking for the rural area.

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Oftedal et al. (2003, 055623) **Period of Study:** 1995-2000 **Location:** Drammen, Norway	**Hospital** Admissions **Outcome:** All Respiratory (460-517) **Age Groups:** All **Study Design:** Time-series **N:** ~4,458 admissions **Statistical Analyses:** Poisson regression, GAM w/ stringent convergence criteria **Covariates:** Temperature, humidity, influenza epidemics, summer and Christmas vacation **Season:** All **Dose-response Investigated?** Yes **Statistical Package:** S-Plus **Lags Considered:** 2-3	**Pollutant:** PM_{10} **Averaging Time:** 24-hs **Mean (SD):** 16.8 $\mu g/m^3$, (10.2) 1994-1997 16.5 $\mu g/m^3$, (10.3) 1998-2000 16.6 , $\mu g/m^3$ (10.2) total period **PM Component:** Benzene, formaldehyde, toluene **Monitoring Stations:** NR **Notes: Copollutant (correlation):** Correlation between pollutants ranged from -0.47–0.78 with the exception of the VOCs studied **Notes:** Benzene, formaldehyde and toluene also evaluated	**PM Increment:** IQr = 11.04 **RR Estimate** 1.035 [0.990, 1.083] 1994-1997 0.992 [0.948, 1.037] 1998-2000 1.021 [0.990, 1.053] 1994-2000 2 Pollutant Model PM_{10} w/ benzene: 1.01 (0.978, 1.043)
Reference: Peel et al. (2005, 056305) **Period of Study:** Jan 1993-Aug 2000 **Location:** Atlanta, Georgia	ED visits **Outcome:** Asthma (493, 786.09) COPD (491, 492, 496) URI (460-466, 477) Pneumonia (480-486) **Age Groups:** All ages. Secondary analyses conducted by age group: 0-1, 2-18, >18 **Study Design:** Time series **N:** 31 hospitals **Statistical Analyses:** Poisson GEE for URI, asthma and all RD Poisson GLM for pneumonia and COPD) **Covariates:** Avg temperature and dew point, pollen counts **Season:** All (secondary analyses of warm season) **Dose-response Investigated?** Yes **Statistical Package:** SAS 8.3, S-Plus 2000 **Lags Considered:** 0-7 d , 3 d ma, 0-13 d unconstrained distributed lag	**Pollutant:** PM_{10} **Averaging Time:** 24 h avg **Mean (SD):** 27.9 (12.3) $\mu g/m^3$ **Percentiles: 10th:** 13.2 **90th:** 44.7 **Monitoring Stations:** "Several" **Copollutant (correlation):** 8 h O_3: r = 0.59 1 h NO_2: r = 0.49 1 h CO: r = 0.47 1 h SO_2: r = 0.20 24-h $PM_{2.5}$: 0.84 24 h PM_{10}-2.5: r = 0.59 24 h UF: r = -0.13 Components: r ranged from 0.42-0.74	**PM Increment:** PM_{10}: 10 $\mu g/m^3$ RR Estimate [Lower CI, Upper CI] All Respiratory Outcomes: 1.013 (1.004–1.021), 3 d ma URI: 1.014 (1.004–1.025) , 3 d ma 1.073 (1.048–1.099) , 14-day dist. lag Asthma: 1.009 (0.996–1.022), 3 d ma 1.099 (1.065–1.135), 14-day dist. lag: Pediatric Asthma 2–18yrs): 1.016 (0.998 –1.034) Pneumonia: 1.011 (0.996–1.027) , 3 d ma 1.087 (1.044–1.132), 14-day dist. lag COPD: 1.018 (0.994–1.043), 3 d ma 1.092 (1.023–1.165), 14-day dist. lag Notes: RRs obtained using AQS 1993-2000, AQS 1998-2000 and ARIES data compared. Infant (0-1 y) and pediatric (2-18 y) asthma was associated more strongly with PM_{10}, $PM_{2.5}$ and OC than adult asthma.

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Ren et al. (2006, 092824) **Period of Study:** Jan 1, 1996–Dec 31, 2001 **Location:** Brisbane, Australia	Hospital Admissions **Outcome (ICD-9):** Respiratory diseases (460-519) excluding influenza (487.0-487.8) **Age Groups:** NR **Study Design:** Time series **N:** NR **Statistical Analyses:** GAM **Covariates:** Day of week, relative humidity, influenza outbreaks **Season:** NR **Dose-response Investigated?** Yes **Statistical Package:** S-Plus **Lags Considered:** 0, 1, and 2 days	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (min-max):** 15.84 (2.5-60) **Monitoring Stations:** 1 **Copollutant:** NR	**PM Increment:** NR **Coefficient Estimates:** Respiratory Hospital Admissions Same day: -0.004296 1-day lag: -0.002474 2-day lag: -0.004229 *all statistically significant Respiratory Emergency Visits Same day: -0.000887 1-day lag: -0.004209 2-day lag: -0.003440 **Notes:** Relative risks were provided in graphical form (Fig 3)
Reference: (Sauerzapf et al., 2009, 180082) **Period of Study:** 3/1/2006-3/2/2007 **Location:** Norfolk, UK	**Outcome:** COPD **Study Design:** Case-Crossover **Covariates:** Environmental factors and Influenza **Statistical Analysis:** Logistic regression **Statistical Package:** SPSS 14 **Age Groups:** > 18 years **N:** 1050 adult COPD admissions	**Pollutant:** PM_{10} **Averaging Time:** 24h **Mean (SD) Unit:** Control: 19.87 (8.51) $\mu g/m^3$ Case: 20.47 (9.27) $\mu g/m^3$ **Range (Min, Max):** Control: 9.77-34.27 Case: 10.04-35.03 **Copollutant (correlation):** NR	**Increment:** 10 $\mu g/m^3$ **Odds Ratio (95% CI)** Lag 0-7, unadjusted: 1.079 (0.980-1.188) Lag 0-8, adjusted: 1.101 (0.988-1.226) Lag 1-8, unadjusted: 1.056 (0.961-1.161) Lag 1-8, adjusted: 1.054 (0.949-1.170)
Reference: Sinclair and Tolsma (2004, 088696) **Period of Study:** 25 Months **Location:** Atlanta, Georgia	**Outpatient Visits** **Outcome:** Asthma (493) URI (460, 461, 462, 463, 464, 465, 466, 477) LRI (466.1, 480, 481, 482, 483, 484, 485, 486). **Age Groups:** < = 18 y, 18+ y (asthma) All ages (URI//LRI) **Study Design:** Times series **N:** 25 months 260,000 to 275,000 health plan members (August 1998–August 2000) **Statistical Analyses:** Poisson GLM **Covariates:** Season, Day of week, Federal Holidays, Study Months **Season:** NR **Dose-response Investigated?:** No **Statistical Package:** SAS **Lags Considered:** Three 3 d moving averages (0-2, 2-5, 6-8)	**Pollutant:** PM_{10} **Averaging Time:** 24 h avg **Mean (SD):** PM_{10} mass–29.03 $\mu g/m^3$ (11.61) **Monitoring Stations:** 1 **Notes: Copollutant:** NR	**PM Increment:** 11.61 (1 SD) **RR Estimate [Lower CI, Upper CI]** **lag:** Child Asthma: 1.049 (S), lag 3-5 d LRI: 1.074 (S), 3-5 d lag **Notes:** Numerical findings for significant results only presented in manuscript. Results for all lags presented graphically for each outcome (asthma, URI, and LRI).

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Slaughter et al. (2005, 073854) **Period of Study:** January 1995 through June 2001 **Location:** Spokane, WA	Hospital Admissions and ED visits **Outcome:** All respiratory (460-519) Asthma (493) COPD (491,492, 494,496) Pneumonia (480-487) Acute URI not including colds and sinusitis (464, 466, 490) **Age Groups:** All, 15+ years for COPD **Study Design:** Time series **N:** 2373 visit records **Statistical Analyses:** Poisson regression, GLM with natural splines. For comparison also used GAM with smoothing splines and default convergence criteria. **Covariates:** Season, temperature, relative humidity, day of week **Season:** All **Dose-response Investigated?:** No **Statistical Package:** SAS, SPLUS **Lags Considered:** 1 -3 d	**Pollutant:** PM_{10} **Averaging Time:** 24 h avg **Range (90% of concentrations):** 7.9-41.9 $\mu g/m^3$ **Monitoring Stations:** 1 **Notes: Copollutant (correlation):** PM_{10} PM_1 r = 0.50 $PM_{2.5}$ r = 0.62 $PM_{10-2.5}$ r = 0.94 CO r = 0.32 Temperature r = 0.11	**PM Increment:** 25 $\mu g/m^3$ **RR Estimate [Lower CI, Upper CI]** **lag:** **ER visits** -- PM_{10} All Respiratory Lag 1: 1.01 [0.99, 1.04] Lag 2: 1.01 [0.98, 1.03] Lag 3: 1.02 [0.99, 1.04] Acute Asthma Lag 1: 1.03 [0.98, 1.07] Lag 2: 1.01 [0.96, 1.05] Lag 3: 1.00 [0.95, 1.04] COPD (adult) Lag 1: 1.00 [0.93, 1.07] Lag 2: 0.99 [0.92, 1.06] Lag 3: 1.02 [0.95, 1.08] **Hospital Admissions** -- PM_{10} All Respiratory Lag 1: 0.99 [0.95, 1.02] Lag 2: 0.99 [0.96, 1.02] Lag 3: 1.00 [0.97, 1.03] Asthma Lag 1: 1.03 [0.95, 1.12] Lag 2: 1.01 [0.94, 1.10] Lag 3: 1.00 [0.92, 1.09] COPD (adult) Lag 1: 0.98 [0.90, 1.07] Lag 2: 1.03 [0.96, 1.11] Lag 3: 1.02 [0.94, 1.09]
Reference: Sun et al. (2006, 090768) **Period of Study:** January 1, 2004 to December 31, 2004 **Location:** Taichung, Taiwan (Central Taiwan)	ED visits **Outcome:** Asthma (493.xx) **Age Groups:** < 55, < 16, 16-55 yrs **Study Design:** Cross-sectional **N:** NR All diagnoses for all patients at 4 medical centers **Statistical Analyses:** Pearson's correlations, multiple correlation coefficients from regression analyses. **Covariates:** Only copollutants considered **Dose-response Investigated?** No **Statistical Package:** SPSS **Lags Considered:** None	**Pollutant:** PM_{10} **Averaging Time:** Monthly avg for 2004 **Mean (SD):** ~ 60.3 $\mu g/m^3$ (NR) (estimated from figure)* **Range (Min, Max):** (~35, 80) **Monitoring Stations:** 11 **Copollutant:** NR	Children ED Visits r = 0.626 P = 0.015 Adult ED Visits r = 0.384 P = 0.109

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Szyszkowicz (2007, 092829) **Period of Study:** 1/4/1992-31/3/2002 **Location:** Edmonton, Canada	**Outcome:** ED visits for asthma (ICD-(493) **Study Design:** Time-series **Covariates:** Temperature, relative humidity **Statistical Analysis:** Poisson regression **Age Groups:** All	**Pollutant:** PM_{10} **Averaging Time:** 24h **Mean (SD) Unit:** 22.6 (13.1) $\mu g/m^3$ **Median, IQR:** 19.4, 15.0 **Copollutant (correlation):** NR	**Increment:** IQR **Percent Relative Risk (95% CI)** *Only statistically significant results are presented in the paper* No lag, ≥ 10 years April to September, All: 3.7 (1.5-6.0) April to September, Female: 4.5 (1.8-7.3) April to September, Male: 3.3 (0.1-6.7) 2d lag, < 10 years Year round, All: 2.7 (0.1-5.4) April to September, All: 6.3 (2.6-10.2) April to September, Male: 7.4 (3.1-11.9) 2d lag, ≥ 10 years April to September, All: 2.4 (0.1-4.7) April to September, Female: 3.9 (1.1-6.7)

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Tecer et al, (2008, 180030) **Period of Study:** 12/2004-10/ **Location:** Zonguldak, Turkey	**Outcome:** ED visits for respiratory problems (ICD-9 470-478, 493) **Study Design:** Bidirectional Case-crossover **Covariates:** Daily meteorological parameters **Statistical Analysis:** Conditional logistic regression **Statistical Package:** Stata **Age Groups:** 0-14 years	**Pollutant:** PM_{10} **Averaging Time:** NR **Mean, Unit:** 53.3 $\mu g/m^3$ **Range (Min, Max):** 12-237.5 **Copollutant (correlation):** $PM_{2.5}/PM_{10}$ Mean: 0.56 Range: 0.17-0.88	**Increment:** 10 $\mu g/m^3$ **Odds Ratio (95% CI)** Asthma Lag 0: 1.14 (1.03-1.26) Lag 1: 0.92 (0.83-1.02) Lag 2: 0.92 (0.81-1.03) Lag 3: 1.01 (0.92-1.11) Lag 4: 1.16 (1.06-1.26) Allergic Rhinitis with Asthma Lag 0: 1.07 (1.01-1.13) Lag 1: 0.96 (0.91-1.02) Lag 2: 0.93 (0.88-0.99) Lag 3: 0.96 (0.90-1.02) Lag 4: 1.08 (1.02-1.14) Allergic Rhinitis Lag 0: 1.06 (0.99-1.13) Lag 1: 1.08 (1.01-1.16) Lag 2: 0.92 (0.87-0.99) Lag 3: 0.97 (0.92-1.03) Lag 4: 1.09 (1.03-1.16) Upper Respiratory Disease Lag 0: 0.88 (0.68-1.14) Lag 1: 1.17 (0.91-1.51) Lag 2: 1.00 (0.76-1.31) Lag 3: 0.95 (0.76-1.19) Lag 4: 1.15 (0.97-1.35) Lower Respiratory Disease Lag 0: 1.01 (0.86-1.19) Lag 1: 1.04 (0.88-1.23) Lag 2: 1.04 (0.92-1.18) Lag 3: 1.23 (1.07-1.41) Lag 4: 0.99 (0.90-1.08)

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Tolbert et al. (2007, 090316)	Hospital Admissions/ED visits	**Pollutant:** PM_{10}	**PM Increment:** 16.30 $\mu g/m^3$ (IQR)
Period of Study: 1993 - 2004	**Outcome (ICD-9):**	**Averaging Time:** 24 h	**Risk ratio [95% CI]:**
Location: Atlanta Metropolitan area, Georgia	**Combined RD group, including:**	**Mean (median**	**Single pollutant models:**
	Asthma (493, 786.07, 786.09), COPD (491, 492, 496), URI (460 – 465, 460.0, 477), pneumonia (480 – 486), and bronchiolitis (466.1, 466.11, and 466.19))	**IQR, range, 10^{th} – 90^{th} percentiles):** 26.6 (24.8 17.5 – 33.8 0.5 – 98.4 12.3 – 42.8)	RD: 1.015 (1.006 – 1.024) **Notes:** Results of selected multi-pollutant models for respiratory disease are presented in Figure 2. **Figure 2:** PM_{10} adjusted for CO, O_3, NO_2, or NO_2/O_3 (non-winter months only) **Summary of results:** PM_{10} remained predictive of RD in non-winter months after adjustment for pollutants.
	Age Groups Analyzed: All **Study Design:** Time series **N:** 10,234,490 ER visits (283,360 and 1,072,429 visits included in the CVD and RD groups, respectively) **Statistical Analyses:** Poisson generalized linear models **Covariates:** long-term temporal trends, season (for RD outcome), temperature, dew point, days of week, federal holidays, hospital entry and exit **Season:** All **Dose-response Investigated:** No **Statistical package:** SAS version 9.1 **Lags Considered:** 3-day moving average(lag 0 -2)	**Monitoring Stations:** NR **Copollutant (correlation):** O_3: r = 0.59 NO_2: r = 0.53 CO: r = 0.51 SO_2: r = 0.21 Coarse PM: r = 0.67 $PM_{2.5}$: r = 0.84 $PM_{2.5}$ SO_4: r = 0.69 $PM_{2.5}$ EC: r = 0.61 $PM_{2.5}$ OC: r = 0.65 $PM_{2.5}$ TC: r = 0.67 $PM_{2.5}$ water-sol metals: r = 0.73 OHC: r = 0.53	
Reference: Tsai et al. (2006, 089768)	**Outcome:** Asthma (493)	**Pollutant:** PM_{10}	**PM Increment:** 62.28 $\mu g/m^3$
Period of Study: 1996 to 2003	**Age Groups:** All (universal health care covers > 96% of the population)	**Averaging Time:** 24 h avg	**OR Estimate [Lower CI, Upper CI]**
Location: Kaohsiung City, Taiwan	**Study Design:** Case crossover	**Mean (SD):** 76.62 $\mu g/m^3$ (NR)	**lag:**
	N: 17,682 admissions 63 hospitals	**Percentiles:** 25th: 41.73 50th(Median): 74.40 75th: 104.01	**Single-pollutant model, 0-2 d cumulative lag** ≥ 25ºC: 1.302 [1.155, 1.467] < 25ºC: 1.556 [1.398, 1.371]
	Statistical Analyses: Conditional Logistic Regression	**Range (Min, Max):** (16.70, 232.00)	**Two-pollutant models, 0-2 d cumulative lag**
	Covariates: Temperature, humidity	**Monitoring Stations:** 6	PM_{10} w/ SO_2
	Season: Warm and cool seasons	**Copollutant:** NR	≥ 25ºC: 1.305 [1.156, 1.473] < 25ºC: 1.540 [1.374, 1.727]
	Dose-response Investigated? No		PM_{10} w/ O_3
	Statistical Package: SAS		≥ 25ºC: 0.985 [0.842, 1.152] < 25ºC: 1.581 [1.402, 1.783]
	Lags Considered: 0-2 d cumulative		PM_{10} w/ NO_2
			≥ 25ºC: 1.237 [1.052, 1.455] < 25ºC: 1.009 [0.875, 1.163]
			PM_{10} w/ CO
			≥ 25ºC: 1.156 [1.012, 1.320] < 25ºC: 1.300 [1.134, 1.490]

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Ulirsch et al. (2007, 091332) **Period of Study:** 11/1994 to 3/2000 **Location:** Pocatello, Idaho Chubbuck, Idaho	**Outcome:** Respiratory Disease (460-499, 509-519) Reactive Airway Disease (786.09) **Age Groups:** All age groups **Study Design:** Time series **N:** 39,347 visits (TS1) 29,513 visits (TS2) **Statistical Analyses:** Poisson regression, GLM. Sensitivity Analyses **Covariates:** Time, Temperature, Relative Humidity Influenza **Season:** Warm/Cool **Dose-response Investigated?** No **Statistical Package:** S-Plus **Lags Considered:** 0 to 4 day lags **Notes:** Time series (TS) 1 includes HA, ED and urgent care visits. TS 2 includes family practice data available after 1997	**Pollutant:** PM_{10} **Averaging Time:** NR **Mean (SD):** TS1: 24.2 $\mu g/m^3$ (NR) 10th: 10.5 90th: 40.7 TS2: 23.2 10th: 10.0 90th: 37.4 **Range (Min, Max):** TS1: (3.0, 183.0) TS2: (3.0, 183.0) **Monitoring Stations:** 4 **Notes: Copollutant (correlation):** PM_{10} w/ NO_2: r = 0.47. PM_{10} with other copollutants weakly correlated.	**PM Increment:** Single Pollutant Models, TS1: 24.4 $\mu g/m^3$ Single Pollutant Models: TS2: 23.2 $\mu g/m^3$ Multipollutant Models: TS1/TS2: 50 $\mu g/m^3$ **Mean Percentage Change, lag 0** TS 1: Single Pollutant All-age (all year): 4.0 [1.4,6.7] 18-64: 3.4 [0.2, 6.7] 0-17: 4.3 [-0.1, 8.9] 65+: 5.6 [-1.4, 13.1] 0-17/65+: 5.5 [1.4, 9.6] All age (Cool season): 4.3 [1.3, 7.5] All age (Warm season): 6.7 [-0.8, 14.8] TS2: Single Pollutant All-age: 3.3 [0.3, 6.3] 18-64: 3.3 [-0.4, 7.0] 0-17: 5.0 [0.1, 10.1] 65+: 6.9 [-0.4, 14.7] Multipollutant (PM_{10} + SO_2) All-age (all year): TS1 10.8 TS2 17.5 18-64: TS1 8.0 TS2 9.1 0-17: TS1 10.8 TS2 32.7 65+: TS1 8.7 TS2 31.3 0-17/65+: TS1 14.2 TS2 25.3 All age (Cool season) TS1 11.9 Multipollutant (PM_{10} + NO_2) All-age (all year) TS1: TS2 16.3 18-64: TS1 9.3 TS2 17.3 0-17: TS1 4.6 TS2 18.7 65+: TS1 12.4 TS2 32.7 0-17/65+: TS1 9.5 32.7 All age (Cool season): TS1 11.1 TS2 16.8 **Notes:** Results from multipollutant model with PM_{10}, SO_2 and NO_2 also available.

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Vegni and Ros (2004, 087448) **Period of Study:** Sep 1, 2001–Sep 31, 2002 **Location:** Milan area, Italy	Hospital Admissions **Outcome (ICD-9):** Respiratory, non-infectious admissions (ICD: NR) **Age Groups:** NR **Study Design:** Time series **N:** 9881 admissions **Statistical Analyses:** Poisson regression **Covariates:** Temperature, wind velocity, relative humidity, week day, holidays **Season:** Spring, summer, autumn, winter **Dose-response Investigated?** No **Statistical Package:** STATA v. 5 **Lags Considered:** 0, 1, and 2-day	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (5th-95th percentile):** Overall: 41.5 (13-98) SD = 28.2 Spring: 29.0 (10-51) SD = 12.6 summer: 24.8 (10-40) SD = 9.9 Autumn: 51.8 (21-114) SD = 27.1 Winter: 64.1 (20-135) SD = 35.7 **Monitoring Stations:** 1 **Copollutant:** NR	**PM Increment:** Increase from 5th–95th percentile Spring: 85 $\mu g/m^3$ summer: 30 $\mu g/m^3$ Autumn: 93 $\mu g/m^3$ Winter: 115 $\mu g/m^3$ **RR Estimate [CI]:** Overall: 1.10 [0.83,1.46] Adjusted: 0.97 [0.67,1.41] **Notes:** 1-day and 2-day lags show similar results, with no association between PM_{10} and daily hospital admissions
Reference: Vigotti et al. (2007, 090711) **Period of Study:** 1/2000–12/2000 **Location:** Pisa, Italy	**ED Visits** **Outcome:** Asthmatic attack (493), dry cough (468), acute bronchitis (466) **Age Groups:** < 10 y 65+ **Study Design:** Time series **N:** 966 Emergency room visits **Statistical Analyses:** Poisson regression, GAM, LOESS smoothers, stringent criteria **Covariates:** temperature, humidity, relative humidity, day of study, rainfall, influenza, day of-the-wk, holidays, time trend **Season:** All year **Dose-response Investigated?** No **Statistical Package:** NR **Lags Considered:** 0-5 d	**Pollutant:** PM_{10} **Averaging Time:** 24-h **Mean (SD):** 35.4 (15.8) $\mu g/m^3$ **Percentiles:** 25th: NR 50th(Median): 31.6 75th: NR **Range (Min, Max):** (9.5, 100.1) **Monitoring Stations:** 2 **Copollutant (correlation):** PM_{10}: NO_2 r = 0.58 CO r = 0.70	**PM Increment:** 10 $\mu g/m^3$ **RR Estimate [Lower CI, Upper CI]** **lag:** < 10 y: 10%[2.3, 18.2] lag 1 65+: 8.5% [1.5, 16.1] lag 2

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Xirasagar et al. (2006, 093267) **Period of Study:** 1998–2001 **Location:** Taiwan	**Hospital Admission/ED:** **Outcome:** Asthma or Asthmatic Bronchitis (493) **Age Groups:** Less than 2 years old, 2 ~ 5 years old, 6 ~ 14 years old **Study Design: N:** N = 27, 275 pediatric hospitalizations **Statistical Analyses:** ARIMA Modeling Spearman's Correlations **Covariates: S**eason, ambient temp., rel. humidity, atmospheric pressure, rainfall, h of sunshine **Season:** Spring: February to April summer: May to July Autumn: August to October Winter: November to January **Dose-response Investigated?** No **Statistical Package:** EViews 4 **Lags Considered:** NR	**Pollutant:** PM_{10} **Averaging Time:** Monthly Means **Mean (SD):** 24.4 $\mu g/m^3$ (NR) **Percentiles:** NR **Range (Min, Max):** NR **PM Component:** NR **Monitoring Stations:** 44 air quality monitoring banks. 23 weather observatories **Notes: Copollutant (correlation):** Less than 2 years old: r = 0.315 2 ~ 5 years old: r = 0.589 6 ~ 14 years old: r = 0.493	**PM Increment:** NR **RR Estimate [Lower CI, Upper CI]** **lag:** NR **AR Estimate [Lower CI, Upper CI]** **lag:** NR **Notes:** Plot of monthly asthma admission rates per 100,000 population by age group Plot of mean monthly concentration trends of criteria air pollutants Mean monthly trends of climatic factors **Other Outcomes Assessed?** NR **Other Exposures Assessed?** Seasonality
Reference: Wong et al., (2002, 023232) **Period of Study:** 1995-1997 (Hong Kong) and 1992-1994 (London) **Location:** Hong Kong and London	Hospital Admissions **Outcome (ICD- NR):** Asthma (493) for ages 15-64 and respiratory disease (460-519) for ages 65+ **Age Groups:** 15-64, 65+ **Study Design:** Time series **N:** NR **Statistical Analyses:** Poisson regression, GAM **Covariates:** Temperature, humidity, and influenza **Season:** Warm (Apr-Sep) and cool (Oct-Mar) **Dose-response Investigated?** Yes **Statistical Package:** S-Plus **Lags Considered:** 0-3 days	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (min-max):** Hong Kong: 51.8 (14.1-163.8) SD = 25.0 London: 28.5 (6.8-99.8) SD = 13.7 **Monitoring Stations:** NR **Copollutant (correlation):** Hong Kong NO_2: r = 0.82 SO_2: r = 0.30 O_3: r = 0.54 London NO_2: r = 0.68 SO_2: r = 0.64 O_3: r = 0.17 **Other variables:** Hong Kong Temp: r = -0.42 Humidity: r = -0.53 London Temp: r = 0.02 Humidity: r = -0.05	**PM Increment:** 10 $\mu g/m^3$ **ER Estimate [CI]:** Single-pollutant excess risk (mean lag 0-1 day) Asthma–Hong Kong: -1.1 [-2.4,0.1] Asthma–London: 1.4 [-0.1,3.0] Respiratory Disease–Hong Kong: 1.0 [0.5,1.5] Respiratory Disease–London: 0.4 [-0.3,1.2] Warm season Asthma–Hong Kong: -1.0 [-2.8, 0.8] Asthma–London: 0.6 [-1.9,3.1] Respiratory Disease–Hong Kong: 0.8 [0.1,1.4] Respiratory Disease–London: 1.8 [0.5,3.1] Cool season Asthma–Hong Kong: -1.2 [-2.8,0.4] Asthma–London: 1.6 [-0.3,3.6] Respiratory Disease–Hong Kong: 1.2 [0.6,1.9] Respiratory Disease–London: -0.5 [-1.5,0.5] **Notes:** RRs are shown graphically in Fig 1 and 2. Exposure response curves are provided in Fig 5 of the article

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Wong et al. (2006, 093266) **Period of Study:** 2000-2002 **Location:** Hong Kong (8 districts)	General Practitioner Visits **Outcome (ICPC-2):** Respiratory diseases/symptoms: upper respiratory tract infections (URTI), lower respiratory infections, influenza, asthma, COPD, allergic rhinitis, cough, and other respiratory diseases **Age Groups:** All ages **Study Design:** Time series **N:** 269,579 visits **Statistical Analyses:** GAM, Poisson regression **Covariates:** Season, day of the week, climate **Season:** NR **Dose-response Investigated?** No **Statistical Package:** S-Plus **Lags Considered:** 0-3 days	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (min-max):** Ranged from 43.4-56.9 (dependent on location) **Monitoring Stations:** 1 per district **Copollutant (correlation):** $PM_{2.5}$: r = 0.94 O_3: r = 0.40 SO_2: r = 0.28	**PM Increment:** 10 $\mu g/m^3$ **RR Estimate [CI]:** Overall URTI 1.020 [1.016,1.025] Overall Non-UTRI 1.025 [1.018,1.032] **Notes:** RRs are also reported for each individual general practitioner yielding similar results
Reference: Yang et al. (2007, 092847) **Period of Study:** 1996-2003 **Location:** Taipei, Taiwan	**Hospital Admission/ED:** **Outcome:** Asthma (493) **Age Groups:** All ages **Study Design:** Case-crossover **N:** 25,602 asthma hospital admissions **Statistical Analyses:** NR **Covariates:** Temperature, humidity, day of-the-wk, seasonality, long term trends **Season:** All year **Dose-response Investigated?** No **Statistical Package:** SAS **Lags Considered:** 0-2	**Pollutant:** PM_{10} **Averaging Time:** NR **Mean (SD):** 48.99 $\mu g/m^3$ **Percentiles: 25th:** 32.64 **50th(Median):** 44.13 **75th:** 59.05 **Range (Min, Max):** (14.44, 234.91) **PM Component:** NR **Monitoring Stations:** 6 Stations **Notes: Copollutant:** NR	**PM Increment:** 26.41 $\mu g/m^3$ **OR Estimate [Lower CI, Upper CI]** **lag:** **Asthma** Single-Pollutant Model: Temperature ＞25° C: 1.046[0.971, 1.128] Temperature ＜25° C: 1.048[1.011, 1.251] Two-Pollutant Model: Adjusted for SO_2: ＞25° C-1.006[0.920, 1.099] ＜25° C–1.088[1.040, 1.138] Adjusted for NO_2: ＞25° C–0.800[0.717, 0.892] ＜25° C–0.982[0.937, 1.029] Adjusted for CO: ＞25° C–0.920[0.844, 1.002] ＜25° C–1.029[0.984, 1.076] Adjusted for O_3: ＞25° C–1.038[0.950, 1.134] ＜25° C–1.042[1.004, 1.081] **AR Estimate [Lower CI, Upper CI]** **lag:** NR **Notes: Other Outcomes Assessed?** NR **Other Exposures Assessed?** SO_2, NO_2, CO, O_3

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Yang et al. (2007, 092847) **Period of Study:** 1996-2003 **Location:** Taipei, Taiwan	Hospital Admission **Outcome:** COPD (490-192), (494), (496) **Age Groups:** All ages **Study Design:** Case-crossover **N:** 46,491COPD admissions, 47 hospitals **Statistical Analyses:** Conditional logistic regression **Covariates:** Weather, day of-the-wk, seasonality, long term trends **Season: Warm/Cool** **Dose-response Investigated?** No **Statistical Package:** SAS **Lags Considered:** 0-2 cumulative	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (SD):** 48.99 $\mu g/m^3$ **25th:** 32.64 **50th(Median):** 44.13 **75th:** 59.05 **Range (Min, Max):** (14.44, 48.99) **Monitoring Stations:** 6 Stations **Notes: Copollutant:** NR	**PM Increment:** 26.41 $\mu g/m^3$ **OR Estimate [Lower CI, Upper CI]** Single-Pollutant Model (0-2 d cum lag): Temperature > 20° C: 1.133[1.098, 1.168] Temperature < 20° C: 1.035[0.994, 1.077] Two-Pollutant Model: PM_{10} w/ SO_2: > 20° C–1.180[1.139, 1.223] < 20° C–1.004[0.954, 1.057] PM_{10} w/ NO_2: > 20° C–1.013[0.973, 1.055] < 20° C–1.074[1.022, 1.129] PM_{10} w/ CO: > 20° C–1.061[1.023, 1.100] < 20° C–1.067[1.016, 1.120] PM_{10} w/ O_3: > 20° C–1.097[1.062, 1.133] < 20° C–1.036[0.996, 1.079]
Reference: Yang et al. (2004, 087488) **Period of Study:** Jun 1, 1995–Mar 31, 1999 **Location:** Vancouver area, British Columbia	Hospital Admissions **Outcome (ICD-9):** Respiratory diseases (460-519), pneumonia only (480-486), asthma only (493) **Age Groups:** 0-3 yrs **Study Design:** Case control, bidirectional case-crossover (BCC), and time series (TS) **N:** 1610 cases **Statistical Analyses:** Chi-square test, Logistic regression, GAM (time-series), GLM with parametric natural cubic splines **Covariates:** Gender, socioeconomic status, weekday, season, study year, influenza epidemic month **Season:** Spring, summer, fall, winter **Dose-response Investigated?** No **Statistical Package:** SAS (Case control and BCC), S-Plus (TS) **Lags Considered:** 0-7 days	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (min-max):** 13.3 (3.8-52.2) SD = 6.1 **Monitoring Stations:** NR (data obtained from Greater Vancouver Regional District Air Quality Dept) **Copollutant (correlation):** $PM_{2.5}$: r = 0.83 $PM_{10-2.5}$: r = 0.83 CO: r = 0.46 O_3: r = -0.08 NO_2: r = 0.54 SO_2: r = 0.61	**PM Increment:** 7.9 $\mu g/m^3$ (IQR) **OR Estimate [CI]:** Values NR **Notes:** Author states that ORs for PM_{10} increased with lag time up to 3 days for both single and multiple-pollutant models.

[1]All units expressed in $\mu g/m^3$ unless otherwise specified.

Table E-13. Short-term exposure–respiratory–ED/HA-$PM_{10-2.5}$

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Chen et al. (2005, 087555) **Period of Study:** Jun 1, 1995–Mar 31, 1999 **Location:** Vancouver area, BC	Hospital Admissions **Outcome (ICD-9):** Acute respiratory infections (460-466), upper respiratory tract infections (470-478), pneumonia and influenza (480-487), COPD and allied conditions (490-496), other respiratory diseases (500-519) **Age Groups:** > 65 yrs **Study Design:** Time series **N:** 12,869 **Statistical Analyses:** GLM **Covariates:** Temp and relative humidity **Season:** NR **Dose-response Investigated?** No **Statistical Package:** S-Plus **Lags Considered:** 1, 2, 3, 4, 5, 6, and 7-day avg	**Pollutant:** $PM_{10-2.5}$ ($\mu g/m^3$) **Averaging Time:** 24 h **Mean (min-max):** 5.6 (0.1-24.6) SD = 3.6 **Monitoring Stations:** 13 **Copollutant (correlation):** $PM_{2.5}$: r = 0.38 PM_{10}: r = 0.83 COH: r = 0.63 CO: r = 0.53 O_3: r = -0.13 NO_2: r = 0.54 SO_2: r = 0.57 Other variables: Mean temp: r = 0.13 Rel humidity: r = -0.27	**PM Increment:** 4.2 $\mu g/m^3$ RR Estimate [CI]: Adj for weather conditions Overall admission 1-day avg: 1.03 [1.00,1.06] 2-day avg: 1.05 [1.02,1.08] 3-day avg: 1.06 [1.02,1.09] Adj for weather conditions and copollutants Overall admission 1-day avg: 1.02 [0.98,1.06] 2-day avg: 1.05 [1.01,1.10] 3-day avg: 1.06 [1.02,1.11] Notes: RR's were also provided for lags 4-7 in Table 3, yielding similar results

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Fung et al. (2006, 089789)	**Hospital Admission/ED:** Hospital Admission	**Pollutant:** $PM_{10-2.5}$ ($\mu g/m^3$)	**PM Increment: :**
Period of Study: 6/1/95–3/31/99	**Outcome:** Respiratory diseases (460-519)	**Averaging Time:** 24-h Avg	4.3 $\mu g/m^3$
Location: Vancouver, Canada	**Age Groups:** Age > 65	**Mean (SD)**	**RR Estimate** (65+ years)
	Study Design: Time series	5.6(3.88) $\mu g/m^3$	DM method:
	N: 26,275 individuals admitted	**Range (Min, Max):**	1.011[0.998,1.024]
	Statistical Analyses: Poisson regression (spline 12 knots), case-crossover (controls +/7 d days from case date), Dewanji and Moolgavkar (DM) method	(-2.9, 27.07)	lag 0
		Monitoring Stations:	1.016[1.0,1.032]
		NR	3 d avg
		Notes: Copollutant (correlation): $PM_{10-2.5}$	1.020[1.001,1.039]
	Covariates: Long-term trends, day-of-the-week effect, weather	PM_{10}	5 d avg
		r = 0.83	1.020[0.998,1.042]
	Season: All year	$PM_{2.5}$	7 d avg
	Dose-response Investigated? No	r = 0.34	Time series:
	Statistical Package: SPlus, R	CO	1.0168[1.003, 1.031]
	Lags Considered: 0-7 d	r = 0.51	lag 0
		CoH	1.020[1.003, 1.037]
		r = 0.61	3 d avg
		O_3	1.019[0.999, 1.039]
		r = -0.11	5 d avg
		NO_2	1.018[0.994, 1.042]
		r = 0.52	7 d avg
		SO_2	Case-crossover:
		r = 0.57	1.019[1.003, 1.034]
			lag 0
			1.019[1.009, 1.038]
			3 d avg
			1.020[0.999, 1.042]
			5 d avg
			1.018[0.994, 1.043]
			7 d avg

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Halonen et al. (2009, 180379) **Period of Study:** 1998-2004 **Location:** Helsinki, Finland	**Outcome:** Hospital Admissions **Age Groups:** 65 + yrs **Study Design:** time series **N:** NR **Statistical Analyses:** Poisson, GAM **Covariates:** temperature, humidity, influenza epidemics, high pollen episodes, holidays **Dose-response Investigated?** No **Statistical Package:** R **Lags Considered:** lags 0-3 & 5d (0-4) mean	**Pollutant:** $PM_{10-2.5}$ **Averaging Time:** daily **Mean (SD):** NR **Min:** 0.0 **25th percentile:** 4.9 **50th percentile:** 7.5 **75th percentile:** 12.1 **Max:** 101.4 **Monitoring Stations:** NR **Copollutant:** $PM_{<0.03}$, $PM_{0.03-0.1}$, $PM_{<0.1}$, $PM_{<0.10.29}$, $PM_{2.5}$, CO, NO_2 **Co-pollutant Correlation** $PM_{<0.03}$: 0.14 $PM_{0.03-0.1}$: 0.28 $PM_{<0.1}$: 0.24 $PM_{<0.10.29}$: 0.20 $PM_{2.5}$: 0.25	**PM Increment:** Interquartile Range **Percent Change (Lower CI, Upper CI):** All Respiratory Mortality Lag 0: -0.66 (-4.16, 2.97) Lag 1: 2.90 (-0.48, 6.39)‡ Lag 2: 0.35 (-3.03, 3.84) Lag 3: -0.38 (-3.67, 3.02) 5-d mean: 0.36 (-4.54, 5.51) Pneumonia HA Lag 0: 0.72 (-1.28, 2.77) Lag 1: 0.55 (-1.34, 2.49) Lag 2: 0.65 (-1.24, 2.58) Lag 3: 0.03 (-1.86, 1.96) 5-d mean: Asthma + COPD HA Lag 0: 2.49 (0.47, 4.56)* Lag 1: 1.37 (-0.66, 3.44) Lag 2: 0.7 (-1.36, 2.80) Lag 3: 1.97 (-0.02, 4.00)‡ 5-d mean: 2.67 (-0.17, 5.58)‡ Other HA Lag 0: 1.38 (-1.24, 4.06) Lag 1: -1.62 (-4.22, 1.05) Lag 2: -1.25 (-3.88, 1.45) Lag 3: 0.04 (-2.52, 2.67) 5-d mean: 0.24 (-3.62, 4.26) *$p < 0.05$, ‡$p < 0.10$
Reference: Host et al. (2007, 155851) **Period of Study:** 2000 - 2003 **Location:** Six French cities: Le Havre, Lille, Marseille, Paris, Rouen, and Toulouse	**Outcome (ICD-10):** Daily hospitalizations for all respiratory diseases (J00–J99), respiratory infections (J10–J22). **Age Groups:** For all respiratory diseases: 0–14 years, 15–64 years, and ≥ 65 years For respiratory infections: All ages **Study Design:** Time series **N:** NR (Total population of cities: approximately 10 million) **Statistical Analyses:** Poisson regression **Covariates:** Seasons, days of the week, holidays, influenza epidemics, pollen counts, temperature, and temporal trends **Dose-response Investigated:** No **Statistical Package:** MGCV package in R software (R 2.1.1) **Lags Considered:** Avg of 0-1 days	**Pollutant:** $PM_{10-2.5}$ **Averaging Time:** 24 h Mean $\mu g/m^3$ (5th -95th percentile): Le Havre: 7.3 (2.5–14.0) Lille: 7.9 (2.2–13.7) Marseille: 11.0 (4.5–21.0) Paris: 8.3 (3.2–15.9) Rouen: 7.0 (3.0–12.5) Toulouse: 7.7 (3.0–15.0) **Monitoring Stations:** 13 total: 1 in Toulouse 4 in Paris 2 each in other cities **Copollutant (correlation):** $PM_{2.5}$: Overall: $r > 0.6$ Ranged between $r = 0.28$ and $r = 0.73$ across the six cities.	**PM Increment:** 10 $\mu g/m^3$, and an 18.8 $\mu g/m^3$ increase (corresponding to an increase in pollutant levels between the lowest of the 5th percentiles and the highest of the 95th percentiles of the cities' distributions) ERR (excess relative risk) Estimate [CI]: For all respiratory diseases (10 $\mu g/m^3$ increase): 0–14 years: 6.2% [0.4, 12.3] 15–64 years: 2.6% [-0.5, 5.8] ≥ 65 years: 1.9% [-1.9, 5.9] For all respiratory diseases (18.8 $\mu g/m^3$ increase): 0–14 years: 12.0 [0.8, 24.3] 15–64 years: 5.0 [-0.9, 11.1] ≥ 65 years: 3.7 [-3.6, 11.4] For respiratory infections (10 $\mu g/m^3$): All ages: 4.4% [0.9, 8.0] For respiratory infections (18 $\mu g/m^3$): All ages: 8.4% [1.7, 15.5]

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Lin et al. (2005, 087828) **Period of Study:** 1998-2001 **Location:** Toronto, North York, East York, Etobicoke, Scarborough, and York (Canada)	Hospital Admissions **Outcome (ICD-9):** Respiratory infections including laryngitis, tracheitis, bronchitis, bronchiolitis, pneumonia, and influenza (464, 466, 480-487) **Age Groups:** 0-14 yrs **Study Design:** Bidirectional case-crossover **N:** 6782 respiratory infection hospitalizations **Statistical Analyses:** Conditional logistic regression (Cox proportional hazards model) **Covariates:** Daily mean temp and dew point temp **Season:** NR **Dose-response Investigated?** No **Statistical Package:** SAS 8.2 PHREG procedure **Lags Considered:** 1-7 day averages	**Pollutant:** $PM_{10-2.5}$ ($\mu g/m^3$) **Averaging Time:** 24 h **Mean (min-max):** 10.86 (0-45.00) SD = 5.37 **Monitoring Stations:** 4 **Copollutant (correlation):** $PM_{2.5}$: r = 0.33 PM_{10}: r = 0.76 CO: r = 0.06 SO_2: r = 0.29 NO_2: r = 0.40 O_3: r = 0.30	**PM Increment:** 6.5 $\mu g/m^3$ OR Estimate [CI]: Adjusted for weather 4 day avg: 1.16 [1.07,1.26] 6 day avg: 1.21 [1.10,1.32] Adj for weather and other gaseous pollutants 4 day avg: 1.13 [1.03,1.23] 6 day avg: 1.17 [1.06,1.29] Notes: OR's were also categorized into "Boys" and "Girls," yielding similar results
Reference: Lin et al. (2002, 026067) **Period of Study:** Jan 1, 1981–Dec 31, 1993 **Location:** Toronto	Hospital Admissions **Outcome (ICD-9):** Asthma (493) **Age Groups:** 6-12 yrs **Study Design:** Uni- and bi-directional case-crossover (UCC, BCC) and time-series (TS) **N:** 7,319 asthma admissions **Statistical Analyses:** Conditional logistic regression, GAM **Covariates:** Maximum and minimum temp, avg relative humidity **Season:** Apr-Sep, Oct-Mar **Dose-response Investigated?** No **Statistical Package:** NR **Lags Considered:** 1-7 day averages	**Pollutant:** $PM_{10-2.5}$ ($\mu g/m^3$) **Averaging Time:** 6 days (predicted daily values) **Mean (min-max):** 12.17 (0-68.00) SD = 7.55 **Monitoring Stations:** 1 **Copollutant (correlation):** $PM_{2.5}$: r = 0.44 PM_{10}: r = 0.83 CO: r = 0.17 SO_2: r = 0.28 NO_2: r = 0.38 O_3: r = 0.56	**PM Increment:** 8.4 $\mu g/m^3$ **RR Estimate [CI]:** Adj for weather and gaseous pollutants BCC 5 day avg: 1.14 [1.01,1.28] BCC 6 day avg: 1.17 [1.03,1.33] TS 5 day avg: 1.14 [1.05,1.23] TS 6 day avg: 1.15 [1.06,1.25] Boys–adj for weather UCC 1 day avg: 1.08 [1.01,1.16] UCC 2 day avg: 1.08 [0.99,1.17] BCC 1 day avg: 1.06 [1.00,1.14] BCC 2 day avg: 1.06 [0.98,1.14] TS 1 day avg: 1.08 [1.03,1.12] TS 2 day avg: 1.07 [1.01,1.13] Girls–adj for weather UCC 1 day avg: 1.07 [0.97,1.18] UCC 2 day avg: 1.16 [1.03,1.31] BCC 1 day avg: 0.98 [0.90,1.07] BCC 2 day avg: 1.05 [0.94,1.16] TS 1 day avg: 1.00 [0.94,1.06] TS 2 day avg: 1.05 [0.98,1.13] **Notes:** The author also provides RR using UCC, BCC, and TS analysis for female and male groups for days 3-7, yielding similar results

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Peel et al. (2005, 056305) **Period of Study:** Jan 1993-Aug 2000 **Location:** Atlanta, Georgia	ED visits **Outcome:** Asthma (493, 786.09) COPD (491, 492, 496) URI (460-466, 477) Pneumonia (480-486) **Age Groups:** All ages. Secondary analyses conducted by age group: 0-1, 2-18, >18 **Study Design:** Time series **N:** 31 hospitals **Statistical Analyses:** Poisson GEE for URI, asthma and all RD Poisson GLM for pneumonia and COPD) **Covariates:** Avg temperature and dew point, pollen counts **Season:** All (secondary analyses of warm season) **Dose-response Investigated?** Yes **Statistical Package:** SAS 8.3 S-Plus 2000 **Lags Considered:** 0-7 d , 3 d ma, 0-13 d unconstrained distributed lag	**Pollutant:** $PM_{10-2.5}$ ($\mu g/m^3$) **Averaging Time:** 24 h avg **Mean (SD):** 9.7 (4.7) Percentiles: 10th: 4.4 90th: 16.2 **Monitoring Stations:** "Several" **Copollutant (correlation):** 24 h PM_{10}: r = 0.59 8 h O_3: r = 0.35 1 h NO_2: r = 0.46 1 h CO: r = 0.32 1 h SO_2: r = 0.21 24 h $PM_{2.5}$: r = 0.43 Components: r ranged from 0.23-0.51	**PM Increment:** 5 RR Estimate [Lower CI, Upper CI] All Respiratory Outcomes: 1.003 [0.982, 1.025] URI: 1.013 [0.987, 1.039] Asthma: 0.998 [0.987, 1.039] Pneumonia: 0.975 [0.940, 1.011] COPD: 0.948 [0.897, 1.003]
Reference: Peng et al. (2008, 156850) **Period of Study:** January 1, 1999–December 31, 2005 **Location:** 108 U.S. counties in the following states: Alabama, Arizona, California, Colorado, Connecticut, District of Columbia, Florida, Georgia, Idaho, Illinois, Indiana, Kentucky, Louisiana, Maine, Maryland, Massachusetts, Michigan, Minnesota, Missouri, Nevada, New Hampshire, New Jersey, New Mexico, New York, North Carolina, Ohio, Oklahoma, Oregon, Pennsylvania, Rhode Island, South Carolina, Tennessee, Texas, Utah, Virginia, Washington, West Virginia, Wisconsin	**Outcome (ICD-9):** Emergency hospitalizations for respiratory disease, including COPD (490–492) and respiratory tract infections (464–466, 480 - 487) **Age Groups:** 65 + years, 65–74, ,75 + **Study Design:** Time series **N:** approximately 12 million Medicare enrollees (1.4 million RD admissions) **Statistical Analyses:** Two-stage Bayesian hierarchical models: Over dispersed Poisson models for county-specific data. Bayesian hierarchical models to obtain national avg estimate **Covariates:** Day of the week, age-specific intercept, temperature, dew point temperature, calendar time, indicator for age of 75 years or older. Some models were adjusted for $PM_{2.5}$. **Dose-response Investigated:** No **Statistical Package:** R version 2.6.2 **Lags Considered:** 0-2 days	**Pollutant:** $PM_{10-2.5}$ **Averaging Time:** 24 h Mean (IQR): All counties assessed: 9.8 (6.9–15.0) Counties in Eastern US: 9.1 (6.6–13.1) Counties in Western US: 15.4 (10.3–21.8) **Monitoring Stations:** At least 1 pair of co-located monitors (physically located in the same place) for PM_{10} and $PM_{2.5}$ per county **Copollutant (correlation):** $PM_{2.5}$: r = 0.12 PM_{10}: r = 0.75 Other variables: Median within-county correlations between monitors: r = 0.60	**PM Increment:** 10 $\mu g/m^3$ Percentage change [95% CI]: Respiratory disease (RD): Lag 0 (unadjusted for $PM_{2.5}$): 0.33 [-0.21, 0.86] Lag 0 (adjusted for $PM_{2.5}$): 0.26 [-0.32, 0.84] Most values NR (see note) Notes: Figure 3: Percentage change in emergency hospital admissions for RD per 10 $\mu g/m^3$ increase in PM (single pollutant model and model adjusted for $PM_{2.5}$ concentration) Figure 4: Percentage change in emergency hospital admissions rate for CVD and RD per a 10 $\mu g/m^3$ increase in $PM_{10-2.5}$ (0–2 day lags, Eastern vs. Western USA)

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Slaughter et al. (2005, 073854)	Hospital Admissions and ED visits	**Pollutant:** $PM_{10-2.5}$ ($\mu g/m^3$)	**PM Increment:** 25 $\mu g/m^3$
Period of Study: January 1995 through June 2001	**Outcome:** All respiratory (460-519)	**Averaging Time:** 24 h avg	RR Estimate [Lower CI, Upper CI]
Location: Spokane, WA	Asthma (493)	**Range (90% of Concentrations):** Reported for $PM_{2.5}$ and PM_{10} only	lag:
Notes	COPD (491,492, 494,496)	**Monitoring Stations:** 1	ER visits:
	Pneumonia (480-487)	**Copollutant (correlation):** $PM_{10-2.5}$	$PM_{10-2.5}$
	Acute URI not including colds and sinusitis (464, 466, 490)	PM_1 r = 0.19	All Respiratory
	Age Groups: All, 15+ years for COPD	$PM_{2.5}$ r = 0.31	Lag 1: 1.01 [0.98, 1.04]
	Study Design: Time series	PM_{10} r = 0.94	Lag 2: 1.01 [0.98, 1.04]
	N: 2373 visit records	CO r = 0.32	Lag 3: 1.02 [0.99, 1.05]
	Statistical Analyses: Poisson regression, GLM with natural splines. For comparison also used GAM with smoothing splines and default convergence criteria.	Temperature r = 0.11	Acute Asthma
			Lag 1: 1.03 [0.98, 1.08]
			Lag 2: 1.01 [0.96, 1.07]
			Lag 3: 0.99 [0.94, 1.05]
	Covariates: Season, temperature, relative humidity, day of week		COPD (adult)
	Season: All		Lag 1: 1.01 [0.93, 1.09]
	Dose-response Investigated?: No		Lag 2: 0.98 [0.90, 1.06]
	Statistical Package: SAS, SPLUS		Lag 3: 1.02 [0.95, 1.10]
	Lags Considered: 1 -3 d		

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Tecer et al. (2008, 180030)	**Outcome:** ED visits for respiratory problems (ICD-9 470-478, 493)	**Pollutant:** $PM_{10-2.5}$	**Increment:** 10 $\mu g/m^3$
Period of Study: 12/2004-10/	**Study Design:** Bidirectional Case-crossover	**Averaging Time:** NR	**Odds Ratio (95% CI)**
Location: Zonguldak, Turkey	**Covariates:** Daily meteorological parameters	**Mean, Unit:** 24.3 $\mu g/m^3$	Asthma
	Statistical Analysis: Conditional logistic regression	**Range (Min, Max):** 4, 195.8	Lag 0: 1.18 (1.01-1.39)
	Statistical Package: Stata	**Copollutant (correlation):**	Lag 1: 0.92 (0.78-1.08)
	Age Groups: 0-14 years	$PM_{2.5}/PM_{10-2.5}$	Lag 2: 0.98 (0.84-1.15)
		Mean: 1.49	Lag 3: 1.11 (0.97-1.27)
		Range: 0.21, 7.53	Lag 4: 1.17 (1.05-1.31)
			Allergic Rhinitis with Asthma
			Lag 0: 0.96 (0.88-1.04)
			Lag 1: 1.08 (0.99-1.18)
			Lag 2: 0.93 (0.86-1.02)
			Lag 3: 0.94 (0.86-1.03)
			Lag 4: 1.10 (1.03-1.18)
			Allergic Rhinitis
			Lag 0: 1.06 (0.95-1.19)
			Lag 1: 1.17 (1.04-1.31)
			Lag 2: 0.92 (0.84-1.02)
			Lag 3: 0.99 (0.91-1.08)
			Lag 4: 1.15 (1.06-1.25)
			Upper Respiratory Disease
			Lag 0: 0.80 (0.54-1.19)
			Lag 1: 1.22 (0.92-1.61)
			Lag 2: 0.97 (0.70-1.33)
			Lag 3: 0.94 (0.66-1.33)
			Lag 4: 1.08 (0.88-1.32)
			Lower Respiratory Disease
			Lag 0: 0.90 (0.71-1.16)
			Lag 1: 1.20 (0.97-1.50)
			Lag 2: 1.00 (0.84-1.19)
			Lag 3: 1.26 (1.08-1.47)
			Lag 4: 1.02 (0.93-1.13)

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Yang et al., (2004, 087488) **Period of Study:** Jun 1, 1995–Mar 31, 1999 **Location:** Vancouver area, British Columbia	Hospital Admissions **Outcome (ICD-9):** Respiratory diseases (460-519), pneumonia only (480-486), asthma only (493) **Age Groups:** 0-3 yrs **Study Design:** Case control, bidirectional case-crossover (BCC), and time series (TS) **N:** 1610 cases **Statistical Analyses:** Chi-square test, Logistic regression, GAM (time-series), GLM with parametric natural cubic splines **Covariates:** Gender, socioeconomic status, weekday, season, study year, influenza epidemic month **Season:** Spring, summer, fall, winter **Dose-response Investigated?** No **Statistical Package:** SAS (Case control and BCC), S-Plus (TS) **Lags Considered:** 0-7 days	**Pollutant:** $PM_{10-2.5}$ ($\mu g/m^3$) **Averaging Time:** 24 h **Mean (min-max):** 5.6 (0-24.6) SD = 3.6 **Monitoring Stations:** NR (data obtained from Greater Vancouver Regional District Air Quality Dept) **Copollutant (correlation):** $PM_{2.5}$: r = 0.39 PM_{10}: r = 0.83 CO: r = 0.33 O_3: r = -0.16 NO_2: r = 0.37 SO_2: r = 0.54	**PM Increment:** 4.2 $\mu g/m^3$ (IQR) OR Estimate [CI]: 3-day lag 1.12 [0.98,1.28] Adj for gaseous pollutants: 1.22 [1.02,1.48] Notes: Author states that ORs for $PM_{10-2.5}$ increased with lag time up to 3 days for both single and multiple-pollutant models. More adjusted ORs and RRs are provided in Fig 1.

[1]All units expressed in $\mu g/m^3$ unless otherwise specified.

Table E-14. Short-term exposure–respiratory–ED/HA-$PM_{2.5}$ (including PM components/sources).

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Andersen et al. (2008, 189651) **Period of Study:** May 2001 - December 2004 **Location:** Copenhagen, Denmark	**Outcome (ICD-10):** RD, including chronic bronchitis (J41–42), emphysema (J43), other chronic obstructive pulmonary disease (J44), asthma (J45), and status asthmaticus (J46). Pediatric hospital admissions for asthma (J45) and status asthmaticus (J46). **Age Groups:** > 5–18 years (asthma) **Study Design:** Time series **N:** NR **Statistical Analyses:** Poisson GAM **Covariates:** Temperature, dew-point temperature, long-term trend, seasonality, influenza, day of the week, public holidays, school holidays (only for 5–18 year olds), pollen (only for pediatric asthma outcome) **Season:** NR **Dose-response Investigated:** No **Statistical Package:** R statistical software (gam procedure, mgcv package) **Lags Considered:** Lag 0-5 days, 4-day pollutant avg (lag 0-3) for CVD, 5-day avg (lag 0-4) for RD, and a 6-day avg (lag 0-5) for asthma.	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h Mean µg/m^3 (SD median IQR 99th percentile): 10 (5 9 7–12 28) **Monitoring Stations:** 1 **Copollutant (correlation):** NCtot: r = 0.40 NC100: r = 0.29 NCa12: r = 0.07 Nca23: r = -0.25 NCa57: r = 0.51 NC_{a212}: r = 0.82 PM_{10}: r = 0.80 CO: r = 0.46 NO_2: r = 0.42 Nox: r = 0.40 No_x curbside: r = 0.28 O_3: r = -0.20 Other variables: Temperature: r = -0.01 Relative humidity: r = 0.21	**PM Increment:** 5 µg/m^3 (IQR) Relative risk (RR) Estimate [CI]: RD hospital admissions (5 day avg, lag 0 -4), age 65+: One-pollutant model: 1.00 [0.95–1.00] Adj for NCtot: 1.00 [0.95–1.06] Asthma hospital admissions (6 day avg lag 0–5), age 5 - 18: One-pollutant model: 1.15 [1.00–1.32] Adj for NCtot: 1.13 [0.98–1.32] Estimates for individual day lags reported only in figure form (see notes): Notes: RD: No statistically or marginally significant associations. Positive associatons at Lag 4–5.Asthma: Wide confidence intervals make interpretation dificult. Positive associations at Lag 1, 2, 3.
Reference: Babin et al. (2007, 188476) **Period of Study:** 10/2001-9/2004 **Location:** Washington, DC	ED Visit/Admissions **Outcome:** Asthma–493 **Age Groups:** 1-17 years,1-4, 5-12, 13-17 **Study Design:** Time-series **N:** NR **Statistical Analyses:** Poisson regression, spline w/ 12 knots to adjust for long term trend **Covariates:** Temperature, mold, pollen, seasonal trends, **Season:** All **Dose-response Investigated?**No **Statistical Package:** STATA **Lags Considered:** 0-4	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-hs **Mean:** "low, never reached code red" **Percentiles:** NR **Range (Min, Max):** NR **Monitoring Stations:** 3 **Copollutant (correlation):** NR	**PM Increment:** 1 $\mu g/m^3$ %Change ED Visits Ages 5-12: -0.2 (-0.6,0.2), lag 0 % Change ED Admissions: Ages 5-12: -0.4 (-1.6,0.8), lag 0 Ages 1-17: 0.2 (-0.6,1.1), lag 0 AR Estimate [Lower CI, Upper CI] lag: NR Notes: No significant interactions between PM and ozone or other covariates were observed.

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Barnett et al. (2005, 087394) **Period of Study:** 1998-2001 **Location:** 5 Australian cities (Brisbane, Canberra, Melbourne, Perth, and Sydney) and 2 New Zealand cities (Auckland, Christchurch)	**Outcome (ICD: NR):** All respiratory admissions (including asthma, pneumonia, and acute bronchitis) **Age Groups:** Children aged < 1 year, 1-4 years, and 5-14 years **Study Design:** Matched case-crossover **N:** ~2.4 million children < 15 years old **Statistical Analyses:** Random effects meta-analysis **Covariates:** Temperature, current minus previous day's temperature, relative humidity, pressure, extremes of hot and cold, day of the week, public holiday, and day after public holiday **Season:** Warm (Nov-Apr) and Cool (May-Oct) **Dose-response Investigated?** No **Statistical Package:** SAS **Lags Considered:** NR	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-hs **Mean (min-max):** Auckland (A): 11.0 (2.1-37.6) Brisbane (B): 9.7 (3.2-122.8) Canberra (Ca): NR Christchurch (Ch): NR Melbourne (M): 8.9 (2.8-43.3) Perth (P): 8.1 (1.7-29.3) Sydney (S): 9.4 (2.4-82.1) **Monitoring Stations:** 1-3 per city **Copollutant:** NR	**PM Increment:** 3.8 $\mu g/m^3$ (IQR) Percent Increase Estimate [CI]: Pneumonia & Acute Bronchitis: Single Pollutant Model < 1 yr (B,M,P,S): 1.7 [0.0,3.4] 1-4 yrs (B,M,P,S): 2.4 [0.1,4.7] Matched Multipollutant Model 1-4 yrs with 1-h SO_2 (B,S): 1.9 [-1.7,5.6] 1-4 yrs with temp (B,M,P,S): 2.3 [-0.4,5.1] Respiratory Admissions: Single Pollutant Model < 1 yr (B,M,P,S): 2.4 [1.0,3.8] 1-4 yrs (B,M,P,S): 1.7 [0.7,2.7] Matched Pollutant Model < 1 yr with 1-h SO_2 (B,S): 3.1 [0.5,5.7] < 1 yr with temp (B,M,P,S): 1.8 [0.2,3.4] 1-4 yrs with PM_{10} (B,M,P,S): 2.9 [0.2,5.6] 1-4 yrs with 1-h SO_2 (B,S): 1.3 [-1.8,4.4] 1-4 yrs with 1-h NO_2 (B,M,P,S): -1.5 [-3.2,0.2] 1-4 yrs with temp (B,M,P,S,): 1.5 [-0.2,3.1]
Reference: Bell et al. (2008, 091268) **Period of Study:** 1995 - 2002 **Location:** Taipei, Taiwan	**Outcome (ICD-9):** Hospital admissions for asthma (493), and pneumonia (486). **Age Groups:** All **Study Design:** Time series **N:** 19,966 hospital admissions for pneumonia, and 10,231 for asthma **Statistical Analyses:** Poisson regression **Covariates:** Day of the week, time, apparent temperature, long-term trends, seasonality **Season:** All **Dose-response Investigated:** No **Statistical Package:** NR **Lags Considered:** lags 0-3 days, mean of lags 0-3	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h Mean (range IQR): 31.6 (0.50–355.0 20.2) **Monitoring Stations:** 2 **Copollutant (correlation):** NR	**PM Increment:** 20 $\mu g/m^3$ (near IQR) Percentage increase estimate [95% CI]: Asthma: L0: 0.46 (-2.41, 3.42) L1: -1.36 (-4.33, 1.71) L2: -0.83 (-3.67, 2.10) L3: -0.78 (-3.63, 2.16) L03: -1.75 (-6.21, 2.92) Pneumonia: L0: 0.06 (-2.74, 2.94) L1: 0.34 (-2.446, 3.20) L2: -0.59 (-3.38, 2.29) L3: -0.44 (-3.22, 2.41) L03: -0.61 (-4.87, 3.85)
Reference: Bell et al. (2008, 091268) **Period of Study:** 1999 - 2005 **Location:** 202 US counties	**Outcome (ICD-9):** COPD (490–492), respiratory tract infections (464 - 466, 480 - 487) **Age Groups:** 65+ **Study Design:** Time series **N:** NR **Statistical Analyses:** Two-stage Bayesian hierarchical model to find national avg First stage: Poisson regression (county-specific)	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h Mean ($\mu g/m^3$): Descriptive information presented in Figure S2 (boxplots): IQR: 8.7 $\mu g/m^3$ **Monitoring Stations:** NR **Copollutant (correlation):** NR	**PM Increment:** 10 $\mu g/m^3$ Percent increase [95% PI]: **Respiratory admissions:** Lag 0 (all seasons): 0.22 [-0.12–0.56] Lag 0 (winter, national): 1.05 [0.29–1.82] Lag 0 (winter, northeast): 1.76 [0.60–2.93] Lag 0 (winter, southeast): 0.59 [-1.35–2.58] Lag 0 (winter, northwest): -0.07 [-6.74–7.08]

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
	Covariates: day of the week, temperature, dew point temperature, temporal trends, indicator for persons 75+ years, population size **Season:** All, June–August (Summer), September–November (Fall), December–February (Winter), March–May (Spring) **Dose-response Investigated:** No **Statistical Package:** NR **Lags Considered:** 0–2 day lags		Lag 0 (winter, southwest): 0.03 [-1.25–1.34] Lag 0 (spring, national): 0.31 [-0.47–1.11] Lag 0 (spring, northeast): 0.34 [-0.66–1.34] Lag 0 (spring, southeast): -0.06 [-1.77–1.68] Lag 0 (spring, northwest): -8.52 [-25.62–12.51] Lag 0 (spring, southwest): 1.87 [-2.00–5.90] Lag 0 (summer, national): -0.62 [-1.33–0.09] Lag 0 (summer, northeast): -0.8 [-1.65–0.07] Lag 0 (summer, southeast): -0.15 [-1.88–1.61] Lag 0 (summer, northwest): 0.25 [-21.46–27.96] Lag 0 (summer, southwest): 0.64 [-5.38–7.04] Lag 0 (autumn, national): 0.02 [-0.63–0.67] Lag 0 (autumn, northeast): -0.01 [-0.87–0.85] Lag 0 (autumn, southeast): -0.58 [-2.06–0.91] Lag 0 (autumn, northwest): -1.38 [-11.84–10.32] Lag 0 (autumn, southwest): 1.77 [-0.73–4.33] Lag 1 (all seasons): 0.05 [-0.29–0.39] Lag 1 (winter): 0.50 [-0.27–1.27] Lag 1 (spring): -0.24 [-1.01–0.53] Lag 1 (summer): 0.28 [-0.39–0.95] Lag 1 (autumn): 0.15 [-0.49–0.79] Lag 2 (all seasons): 0.41 [0.09–0.74] Lag 2 (winter, national): 0.72 [0.01–1.43] Lag 2 (winter, northeast): 0.79 [-0.21–1.80] Lag 2 (winter, southeast): 0.4 [-1.45, 2.27] Lag 2 (winter, northwest): -0.06 [-6.52–6.85] Lag 2 (winter, southwest): 1.2 [-0.10–2.52] Lag 2 (spring, national): 0.35 [-0.29–0.99] Lag 2 (spring, northeast): 0.04 [-0.88–0.97] Lag 2 (spring, southeast): 0.75 [-0.82–2.34] Lag 2 (spring, northwest): 2.29 [-14.26–22.03]

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			Lag 2 (spring, southwest): 1.05 [-2.18–4.39] Lag 2 (summer, national): 0.57 [-0.07–1.23] Lag 2 (summer, northeast): 0.77 [-0.01–1.56] | Lag 2 (summer, southeast): -0.52 [-2.07–1.06] Lag 2 (summer, northwest): 0.74 [-18.73–24.86] Lag 2 (summer, southwest): 2.41 [-2.61–7.69] Lag 2 (autumn, national): 0.39 [-0.22–1.01] Lag 2 (autumn, northeast): 0.12 [-0.82–1.07] Lag 2 (autumn, southeast): 0.14 [-1.29–1.59] Lag 2 (autumn, northwest): -0.74 [-10.08–9.58] Lag 2 (autumn, southwest): 0.97[-1.36–3.36]
Reference: Bell et al. (2009, 191007) **Period of Study:** 1999-2005 **Location:** 168 US Counties	**Outcome:** Respiratory hospital admissions **Study Design:** Retrospective Cohort **Covariates:** socio-economic conditions, long term temperature **Statistical Analysis:** Bayesian hierarchical model **Age Groups:** ≥65	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24h **Mean (SD) Unit:** NR **Range (Min, Max):** NR **Copollutant (correlation):** NR	**Increment:** 20% of the population acquiring air conditioning **Percent Change (95% CI) in community-specific PM health effect estimates for respiratory hospital admissions** Any AC, including window units Yearly health effect: 44.5 (-87.5-176) Summer health effect: -74.8 (-417-267) Winter health effect: -32.5 (-245-180) Central AC Yearly health effect: 27.6 (-46.7-102) Summer health effect: -38.6 (-160-82.6) Winter health effect: 43.8 (-125-213)

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Bell et al. (2009, 191007) **Period of Study:** 1999-2005 **Location:** 168 US Counties	**Outcome:** Respiratory HA **Age Groups:** 65+ **Study Design:** time series **N:** NR **Statistical Analyses:** Bayesian Hierarchical Regression **Covariates:** time trend, day of week, seasonality, dew point, temperature **Statistical Package:** NR **Lags Considered:** 0-2	**Pollutant:** $PM_{2.5}$ **Averaging Time:** daily **Mean:** EC: 0.715 Ni: 0.002 V: 0.003 **Min:** EC: 0.309 Ni: 0.003 V: 0.001 **Max:** EC: 1.73 Ni: 0.021 V: 0.010 **Interquartile Range:** EC: 0.245 Ni: 0.001 V: 0.001 **Interquartile Range of Percents:** EC: 1.7 Ni: 0.01 V: 0.01 **Monitoring Stations:** NR **Copollutant:** Al, NH_4+, As, Ca, Cl, Cu, EC, OMC, Fe, Pb, Mg. Ni, NO_3-, K, Si, $Na+$, $SO_4=$, Ti, V, Zn **Co-pollutant Correlation** Ni, V: 0.48 V, EC: 0.33 Ni, EC: 0.30 **Note:** Pollutant concentrations available for all fractions of $PM_{2.5}$	**PM Increment:** Interquartile Range in the fraction of $PM_{2.5}$ **Percent Increase (Lower CI, Upper CI):** EC: 511 (80.7, 941), lag 0 EC + Ni: 399 (-45.1, 843), lag 0 EC + V: 386 (-74.8, 846), lag 0 EC + Ni, V: 362 (-98.0, 823), lag 0 Ni: 223 (36.9, 410), lag 0 Ni + EC: 176 (-18.7, 370), lag 0 Ni + V: 151 (-78.4, 381), lag 0 Ni + EC, V: 136 (-94.9, 368), lag 0 V: 392 (46.3, 738), lag 0 V + EC: 279 (-93.2, 651), lag 0 V+ Ni: 230 (-193.7, 653), lag 0 V + EC, Ni: 140 (-300, 579), lag 0 EC: -1.5 (80.7, 941), lag 1 EC: 17.5 (-22.3, 57.3), lag 2 Ni: -7.2 (-66.6. 52.1), lag 1 Ni: -4.9 (-22.3, 12.5), lag 2 V: -19.6 (-127, 88.3), lag 1 V: 10.5 (-21.5, 42.4), lag 2 HS education: -77.8 (-390, 234), lag 0 median income: 45.9 (-411, 503), lag0 Percent black: -53.1 (-557, 451), lag 0 Percent living in urban area: -41.9 (-774.7, 691), lag 0 Population: -22.9 (-121, 75.3), lag 0 **Notes:** Interquartile ranges in percent HS education, median income, percent black, percent living in urban area, and population are 5.2 %, $9,223, 17.3%, 11.0%, and 549,283 respectively.

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Chardon et al. (2007, 091308) **Period of Study:** 2000-2003 **Location:** Greater Paris Area, France	Doctors house calls **Outcome (ICPC2):** Asthma (R96), Upper respiratory disease (URD R07, R21, R29, R75, R76, R02) , Lower respiratory disease (LRD, R05, R78) **Age Groups:** all **Study Design:** Time series **N:** 8027 for asthma 52928 for LRD 74845 for URD **Statistical Analyses:** Quasi-Poisson, GAM, parametric penalized spline smoothers. **Covariates:** Lagged and current temperature, humidity, long term trends, seasonality, pollen counts, influenza epidemic, days of the week, holidays, bank holidays **Season:** All **Dose-response Investigated?** No **Statistical Package:** R **Lags Considered:** 0-3 days	**Pollutant:** $PM_{2.5}$ **Averaging Time:** mean of the daily means **Mean (SD):** 14.7(7.34) $\mu g/m^3$ **Percentiles:** 25th: 9.5 50th(Median): 12.9 75th: 18.2 **Range (Min, Max):** (3, 69.6) **Monitoring Stations:** 1- 4 **Copollutant:** PM_{10}: r = 0.95 NO_2: r = 0.68	**PM Increment:** 10 $\mu g/m^3$ % Change, lag 0-3 d avg URD 6.0 (3.1, 9.1) LRD 5.8 (2.8, 8.9) Asthma 4.4 (-1.3, 10.4)
Reference: Chen et al. (2005, 087555) **Period of Study:** Jun 1, 1995–Mar 31, 1999 **Location:** Vancouver area, BC	Hospital Admissions **Outcome (ICD-9):** Acute respiratory infections (460-466), upper respiratory tract infections (470-478), pneumonia and influenza (480-487), COPD and allied conditions (490-496), other respiratory diseases (500-519) **Age Groups:** > 65 yrs **Study Design:** Time series **N:** 12,869 **Statistical Analyses:** GLM **Covariates:** Temp and relative humidity **Season:** NR **Dose-response Investigated?** No **Statistical Package:** S-Plus **Lags Considered:** 1, 2, 3, 4, 5, 6, and 7-day avg	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean (min-max):** 7.7 (2.0-32.0) SD = 3.7 **Monitoring Stations:** 13 **Copollutant (correlation):** PM_{10}: r = 0.83 $PM_{10-2.5}$: r = 0.38 COH: r = 0.39 CO: r = 0.23 O_3: r = -0.01 NO_2: r = 0.36 SO_2: r = 0.42 **Other variables:** Mean temp: r = 0.41 Rel humidity: r = -0.23	**PM Increment:** 4.0 $\mu g/m^3$ (IQR) **RR Estimate [CI]:** Adj for weather conditions Overall admission 1-day avg: 1.02 [0.99,1.05] 2-day avg: 1.02 [0.99,1.06] 3-day avg: 1.02 [0.98,1.05] Adj for weather conditions and copollutants Overall admission 1-day avg: 1.01 [0.98,1.06] 2-day avg: 1.01 [0.98,1.05] 3-day avg: 1.00 [0.96,1.04] **Notes:** RR's were also provided for lags 4-7 in Table 3, yielding similar results

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Chimonas and Gessner (2007, 093261) **Period of Study:** January 1, 1999–June 30, 2003 **Location:** Anchorage, Alaska	**Outcome (ICD-9):** Asthma (493.0-493.9) Lower respiratory illness-LRI (466.1, 466.0, 480-487, 490, 510-511) Inhaled quick-relief medication Steroid medication **Age Groups:** < 20 years old **Study Design:** Time series **N:** 42,667 admissions **Statistical Analyses:** GEE for multivariable modeling **Covariates:** Season, serial correlation, year, weekend, temperature, precipitation, and wind speed **Season:** NR **Dose-response Investigated?** No **Statistical Package:** SPSS (dataset), SAS (analysis) **Lags Considered:** 1 day and 1 week	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-hs and 1 week **Mean (min-max):** Daily: 6.1 (0.5-69.8) Weekly: 5.8 (1.8-45.0) **Monitoring Stations:** NR **Copollutant:** N/A	**PM Increment:** 5 $\mu g/m^3$ RR Estimate [CI]: Same Day Outpatient Asthma: 0.992 [0.964,1.024] Outpatient LRI: 0.952 [0.907,1.001] Inpatient Asthma: 0.936 [0.798,1.098] Inpatient LRI: 0.919 [0.823,1.027] Inhaled Steroid Prescriptions: 0.988 [0.902,1.083] Quick-relief Medication: 0.962 [0.901,1.028] Weekly (median increase) Outpatient Asthma: 0.983 [0.935,1.038] Outpatient LRI: 0.969 [0.874,1.075] Inpatient Asthma: 0.754 [0.513.1.109] Inpatient LRI: 0.943 [0.715,1.245] Inhaled Steroid Prescriptions: 1.018 [0.883,1.175] Quick-relief Medication: 0.978 [0.882,1.087]

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Delfino et al. (2009, 191994) **Period of Study:** 10/1/2003-11/15/2003 **Location:** Southern California	**Outcome:** Respiratory hospital admissions **Study Design:** Time series **Statistical Analysis:** Poisson regression with GEE **Age Groups:** All	**Pollutant:** $PM_{2.5}$ **Averaging Time:** Hourly **Mean (SD) Unit by county:** Los Angeles Before Fires: 27.2 (12.4) $\mu g/m^3$ During Fires: 54.1 (21.0) $\mu g/m^3$ After Fires: 15.9 (5.5) $\mu g/m^3$ Orange Before Fires: 23.2 (9.6) $\mu g/m^3$ During Fires: 64.3 (26.5) $\mu g/m^3$ After Fires: 15.5 (10.2) $\mu g/m^3$ Riverside Before Fires: 32.7 (14.7) $\mu g/m^3$ During Fires: 42.1 (25.5) $\mu g/m^3$ After Fires: 16.9 (10.2) $\mu g/m^3$ San Bernadino Before Fires: 35.7 (16.6) $\mu g/m^3$ During Fires: 45.3 (28.7) $\mu g/m^3$ After Fires: 18.5 (8.3) $\mu g/m^3$ San Diego Before Fires: 18.5 (6.7) $\mu g/m^3$ During Fires: 76.1 (66.6) $\mu g/m^3$ After Fires: 14.2 (7.2) $\mu g/m^3$ Ventura Before Fires: 18.4 (8.3) $\mu g/m^3$ During Fires: 50.1 (50.5) $\mu g/m^3$ After Fires: 12.9 (4.3) $\mu g/m^3$ **Copollutant (correlation):** NR	**Increment:** 10 $\mu g/m^3$ **Relative Rate (Min CI, Max CI)** All Respiratory, All Ages: All Periods: 1.009 (0.999-1.018) Pre-Wildfire: 1.022 (1.004-1.040) Wildfire: 1.028 (1.014-1.041), p = 0.639 Post-Wildfire: 0.999 (0.968-1.031), p = 0.198 All Respiratory, Ages 0-4: All Periods: 0.994 (0.967-1.021) Pre-Wildfire: 0.982 (0.921-1.046) Wildfire: 1.045 (1.010-1.082), p = 0.103 Post-Wildfire: 0.894 (0.807-0.991), p = 0.126 All Respiratory, Ages 5-19: All Periods: 1.014 (0.983-1.046) Pre-Wildfire: 1.026 (0.946-1.113) Wildfire: 1.027 (0.984-1.076), p = 0.990 Post-Wildfire: 0.958 (0.852-1.077), p = 0.354 All Respiratory, Ages 20-64: All Periods: 1.015 (1.002-1.029) Pre-Wildfire: 1.036 (1.007-1.066) Wildfire: 1.024 (1.005-1.044), p = 0.534 Post-Wildfire: 1.007 (0.960-1.056), p = 0.315 All Respiratory, Ages 65-99: All Periods: 1.009 (0.996-1.022) Pre-Wildfire: 1.022 (0.994-1.050) Wildfire: 1.030 (1.011-1.049), p = 0.649 Post-Wildfire: 1.024 (0.967-1.074), p = 0.932 Asthma, All Ages, Male and Female: All Periods: 1.022 (1.001-1.042) Pre-Wildfire: 0.998 (0.949-1.050) Wildfire: 1.048 (1.021-1.076), p = 0.097 Post-Wildfire: 0.986 (0.910-1.068), p = 0.792 Asthma, All Ages, Male: All Periods: 1.010 (0.980-1.040) Pre-Wildfire: 1.021 (0.944-1.106) Wildfire: 1.031 (0.990-1.073), p = 0.848 Post-Wildfire: 1.063 (0.948-1.192), p = 0.553 Asthma, All Ages, Female: All Periods: 1.029 (1.001-1.058) Pre-Wildfire: 0.979 (0.913-1.050) Wildfire: 1.059 (1.022-1.097), p = 0.056 Post-Wildfire: 0.928 (0.829-1.037), p = 0.412 Asthma, Ages 0-4, Males and Females: All Periods: 0.996 (0.947-1.048) Pre-Wildfire: 0.924 (0.824-1.035) Wildfire: 1.083 (1.021-1.149), p = 0.017 Post-Wildfire: 0.924 (0.767-1.113), p = 0.999 Asthma, Ages 0-4, Males: All Periods: 1.018 (0.963-1.076) Pre-Wildfire: 0.942 (0.815-1.089)

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Dominici et al. (2006, 088398) **Period of Study:** 1999 - 2002 **Location:** 204 US counties, located in: Alabama, Alaska, Arizona, Arkansas, California, Colorado, Connecticut, Delaware, District of Columbia, Florida, Georgia, Hawaii, Idaho, Illinois, Indiana, Iowa, Kansas, Kentucky, Louisiana, Maine, Maryland, Massachusetts, Michigan, Minnesota, Mississippi, Missouri, Nevada, New Hampshire, New Jersey, New Mexico, New York, North Carolina, Ohio, Oklahoma, Oregon, Pennsylvania, Rhode Island, South Carolina, Tennessee, Texas, Utah, Virginia, Washington, West Virginia, Wisconsin	Outcome (ICD-9: Daily counts of hospital admissions for primary diagnosis of chronic obstructive pulmonary disease (490–492), and respiratory tract infections (464–466, 480–487). **Age Groups:** > 65 years **Study Design:** Time series **N:** 11.5 million Medicare enrollees **Statistical Analyses:** Bayesian 2-stage hierarchical models. First stage: Poisson regression (county-specific) Second stage: Bayesian hierarchical models, to produce a national avg estimate **Covariates:** Day of the week, seasonality, temperature, dew point temperature, long-term trends **Season:** NR **Dose-response Investigated:** No **Statistical Package:** R statistical software version 2.2.0 **Lags Considered:** 0-2 days, avg of days 0-2	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h Mean ($\mu g/m^3$) (IQR): 13.4 (11.3–15.2) **Monitoring Stations:** NR **Copollutant (correlation):** NR Other variables: Median of pairwise correlations among $PM_{2.5}$ monitors within the same county for 2000: r = 0.91 (IQR: 0.81-0.95)	**PM Increment:** 10 $\mu g/m^3$ (Results in figures see notes) Percent increase in risk [95% PI]: COPD (Lag 0): Age 65 +: 0.91 [0.18, 1.64] Age 65–74: 0.42 [-0.64, 1.48] Age 75+: 1.47 [0.54, 2.40] Respiratory tract infection: Age 65+: 0.92 [0.41, 1.43] Age 65–74: 0.93 [0.04, 1.82] Age 75+: 0.92 [0.32, 1.53] Annual reduction in admissions attributable to a 10 $\mu g/m^3$ reduction in daily $PM_{2.5}$ level (95% PI): Cerebrovascular disease: Annual number of admissions: 226,641 Annual reduction in admissions: 1836 [680, 2992] COPD: Annual number of admissions: 108,812 Annual reduction in admissions: 990 [196, 1785] Respiratory tract infections: Annual number of admissions: 226,620 Annual reduction in admissions: 2085 [929, 3241]
Reference: Dominici et al. (2006, 088398) **Period of Study:** 1999-2002 **Location:** U.S. (mainland)	**Outcome (ICD-9):** Respiratory tract infections (464-466, 480-487) and Chronic Obstructive Pulmonary Disease (490-492) **Age Groups:** All > 65 yrs 65-74 yrs > 75 yrs **Study Design:** Time series **N:** 11.5 million at-risk **Statistical Analyses:** Bayesian 2-stage hierarchical models (day-to-day variation), Poisson regression (county-specific RRs) **Covariates:** Calendar time (seasonality and year), temperature, dew point **Season:** NR **Dose-response Investigated?** No **Statistical Package:** NR **Lags Considered:** 0, 1, 2 days	**Pollutant:** $PM_{2.5}$ **Averaging Time:** daily or every 3 days (depending on county) **Mean:** 13.4 (IQR: 11.3-15.2) **Monitoring Stations:** NR (used data from Air Quality System database) **Copollutant:** NR	**PM Increment:** 10 $\mu g/m^3$ **Percentage Change in Hospital Admission Rates [PI]:** COPD–Same day All > 65: 0.91 [0.18,1.64] 65-74 yrs: 0.42 [-0.64,1.48] > 75: 1.47 [0.54,2.40] Respiratory Tract Infections–2-day lag All > 65: 0.92 [0.41,1.43] 65-74 yrs: 0.93 [0.04,1.82] > 75: 0.92 [0.32,1.53] **Notes:** Other lag data shown in Fig 2-4

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Erbas et al. (2005) **Period of Study:** Jul 1, 1989–Dec 31, 1992 **Location:** Melbourne, Australia	**Outcome (ICD):** COPD (490-492, 494, 496) Asthma (493) **Age Groups:** NR **Study Design:** Time series **N:** NR **Statistical Analyses:** GLM, GAM, Parameter Driven Poisson Regression, Transitional Regression, Seasonal-Trend decomposition based on Loess smoothing for seasonal adjustment **Covariates:** Secular trends, seasonality, relative humidity, dry bulb temp, dew point temp **Season:** NR **Dose-response Investigated?** Yes **Statistical Package:** S-Plus, SAS **Lags Considered:** 0-5 days	**Pollutant:** $PM_{0.1-1}$ (API) **Averaging Time:** 24-hs **Mean (min-max):** NR **Monitoring Stations:** 9 **Copollutant (correlation):** NR	**PM Increment:** Increase from the 10th-90th percentile (value NR) **RR Estimate [CI]:** COPD GAM: 0.95 [0.91,1.00] GLM, PDM, TRM: NR Asthma NR **Notes:** This study was used to demonstrate that conclusions are highly dependent on the type of model used
Reference: Fung et al. (2006, 089789) **Period of Study:** 6/1/95–3/31/99 **Location:** Vancouver, Canada	Hospital Admission/ED: Hospital Admission **Outcome:** Respiratory diseases (460-519) **Age Groups:** Age > 65 **Study Design:** Time series, case crossover **N:** 26,275 individuals admitted **Statistical Analyses:** Poisson regression (spline 12 knots), case-crossover (controls +/7 d days from case date), Dewanji and Moolgavkar (DM) method **Covariates:** Long-term trends, day-of-the-week effect, weather **Season:** All year **Dose-response Investigated?** No **Statistical Package:** SPlus, R **Lags Considered:** 0-7 d	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-h Avg **Mean (SD):** 7.72(3.61) **Range (Min, Max):** (2, 32) **Monitoring Stations:** NR **Copollutant (correlation):** $PM_{2.5}$: PM_{10} r = 0.80 $PM_{10-2.5}$ r = 0.34 CO r = 0.23 CoH r = 0.38 O_3 r = -0.03 NO_2 r = 0.36 SO_2 r = 0.42	**PM Increment: :** 4 $\mu g/m^3$ **RR Estimate (65+ years)** DM method: 1.007[0.994, 1.020] Current 1.007[0.990,1.023] 3 day 0.995[0.979,1.012] 5 day 0.995[0.971,1.020] 7 day Time series: 1.003[0.989, 1.018] Current 1.000[0.982, 1.018] 3 day 0.993[0.972, 1.014] 5 day 0.995[0.971, 1.020] 7 day Case-crossover: 1.002[0.986, 1.019] Current 1.001[0.981, 1.021] 3 day 0.988[0.966, 1.011] 5 day 0.984[0.959, 1.010] 7 day

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Hinwood et al. (2006, 088976) **Period of Study:** 1/1992-12/1998 **Location:** Perth, Australia	Hospital Admission **Outcome (ICD-9):** COPD (490-496.99, except asthma), pneumonia /influenza (480-489.99), asthma **Age Groups:** All ages **Study Design:** Time stratified case-crossover **N:** NR **Statistical Analyses:** Conditional logistic regression **Covariates:** Time trend, season, temperature, humidity, day of wk, holidays **Season:** All year **Dose-response Investigated?** No **Statistical Package:** NR **Lags Considered:** 0-3 days	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-h Avg **Mean (SD):** 9.2 (4.3) **Percentiles:** 10th: 5.0 90th: 14.5 **Monitoring Stations:** 13 **Notes: Copollutant:** NR	**Increment:** 1 $\mu g/m^3$ **Notes:** Odds ratio for $PM_{2.5}$ and all respiratory, COPD, pneumonia and asthma. Authors found an elevation in the odds ratio for lags 2 and 3 reaaching significance in all age groups for lag 3. For each increase of 1 $\mu g/m^3$, the number of hospitalizations increases 0.2% for respiratory disease, 0.5% for pneumonia and 0.3% for asthna. $PM_{2.5}$ concentrations were also significantly associated with asthma for those aged under 15 years with an estimated 0.5% increase in hospitalizations.

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Hirshon et al. (2008, 180375)	**Outcome:** Hospital admissions for asthma	**Pollutant:** $PM_{2.5}$ zinc	**Increment:** NR
Period of Study: 6/2002-11/2002	**Study Design:** Time-series	**Averaging Time:** 24h	**Relative Risk (95% CI), Best fit Model**
Location: Baltimore, Maryland	**Covariates:** Spatial distance from pollution monitor, demographic variation, long term, seasonal and daily trends, weather and other pollutants	**Mean (SD) Unit:** 22.42 (25.14) $\mu g/m^3$	Medium = 8.63-20.76 ng/m^3
		Range (Min, Max): NR	High = > 20.76 ng/m^3
	Statistical Analysis: Overdispersed Poisson regression	**Copollutant (correlation):**	No Lag
		Ni: 0.41	Medium: 1.12 (0.98-1.28)
	Age Groups: 0-17 years	Cr: 0.17	High: 1.09 (0.91-1.30)
		Fe: 0.54	1-day Lag
		Sulfate: 0.01	Medium: 1.23 (1.07-1.41)
		CO: 0.40	High: 1.16 (0.97-1.39)
		$PM_{2.5}$: 0.39	2-day Lag
		O_3: 0.01	Medium: 1.11 (0.94-1.30)
		NO_2: 0.66	High: 1.15 (0.96-1.38)
		Elemental Carbon: 0.48	**Controlling for Time Trends**
			No Lag
			Medium: 1.08 (0.95-1.23)
			High: 0.98 (0.86-1.11)
			1-day Lag
			Medium: 1.13 (1.003-1.28)
			High: 1.03 (0.91-1.16)
			2-day Lag
			Medium: 1.13 ()
			High: 0.98-1.31
			Controlling for Time Trends and Additional Copollutants
			No Lag
			Medium: 1.12 (0.98-1.29)
			High: 1.09 (1.01-1.30)
			1-day Lag
			Medium: 1.20 (1.04-1.38)
			High: 1.12 (0.93-1.35)
			2-day Lag
			Medium: 1.12 (0.95-1.32)
			High: 1.19 (0.98-1.44)

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Host et al. (2007, 155851) **Period of Study:** 2000 - 2003 **Location:** Six French cities: Le Havre, Lille, Marseille, Paris, Rouen, and Toulouse	**Outcome (ICD-10):** Daily hospitalizations for all respiratory diseases (J00–J99), respiratory infections (J10–J22). **Age Groups:** For all respiratory diseases: 0–14 years, 15–64 years, and ≥ 65 years. For respiratory infections: All ages **Study Design:** Time series **N:** NR (Total population of cities: approximately 10 million) **Statistical Analyses:** Poisson regression **Covariates:** Seasons, days of the week, holidays, influenza epidemics, pollen counts, temperature, and temporal trends **Season:** NR **Dose-response Investigated:** No **Statistical Package:** MGCV package in R software (R 2.1.1) **Lags Considered:** Avg of 0-1 days	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h Mean (5th -95th percentile): Le Havre: 13.8 (6.0–30.5) Lille: 15.9 (6.9–26.3) Marseille: 18.8 (8.0–33.0) Paris: 14.7 (6.5–28.8) Rouen: 14.4 (7.5–28.0) Toulouse: 13.8 (6.0–25.0) **Monitoring Stations:** 13 total: 1 in Toulouse 4 in Paris 2 each in other cities **Copollutant (correlation):** $PM_{10-2.5}$: Overall: $r > 0.6$ Ranged between r = 0.28 and r = 0.73 across the six cities.	**PM Increment:** 10 $\mu g/m^3$ increase, and a 27 $\mu g/m^3$ increase (corresponding to the difference between the lowest of the 5th percentiles and the highest of the 95th percentiles of the cities' distributions) ERR (excess relative risk) Estimate [CI]: For all respiratory diseases (27 $\mu g/m^3$ increase): 0–14 years: 1.1% [-3.1, 5.5] 15–64 years: 2.2% [-1.8, 6.4]; ≥ 65 years: 1.3% [-5.3, 8.2] For respiratory infections (10 $\mu g/m^3$ increase): All ages: 2.5% [0.1, 4.8] For respiratory infections (27 $\mu g/m^3$ increase): All ages: 7.0% [0.7, 13.6]
Reference: Ko et al. (2007, 091639) **Period of Study:** 1/2000-12/2004 **Location:** Hong Kong, China	ED Visits **Outcome (ICD-9):** COPD: Chronic bronchitis (491), Emphysema (492), Chronic airway obstruction (496) **Age Groups:** All ages **Study Design:** Time series **N:** 15 hospitals, 119,225 admissions **Statistical Analyses:** Poisson regression, GAM with stringent convergence criteria, APHEA2 protocol. **Covariates:** Time trend, season, temperature, humidity, other cyclical factors, day, day of wk, holidays **Season:** All year, interactions with season tested **Dose-response Investigated?** No **Statistical Package:** SPLUS 4.0 **Lags Considered:** 0-5 days	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-h **Mean (SD):** 35.7 (20.6) **Percentiles:** 25th: 19.4 50th(Median): 31.7 75th: 46.7 **Range (Min, Max):** (6.0, 163.2) **Monitoring Stations:** 14 **Copollutant (correlation):** $PM_{2.5}$: PM_{10} r = 0.952 NO_2 r = 0.441 O_3 r = 0.394 SO_2 r = 0.282	**PM Increment:** PM_{10} **RR Estimate** COPD 1.002[0.998, 1.001] lag 0 1.003[0.999, 1.007] lag 1 1.011[1.007, 1.014] lag 2 1.013[1.010, 1.017] lag 3 1.011[1.008, 1.015] lag 4 1.009[1.006, 1.013] lag 5 1.004[0.999, 1.008]lag 0-1 1.010[1.006, 1.015]lag 0-2 1.018[1.013, 1.022]lag 0-3 1.024[1.019, 1.029]lag 0-4 1.031[1.026, 1.036]lag 0-5 4-Pollutant model: 1.014[1.007, 1.022] lag 0-5 3-Pollutant model: 1.011[1.004, 1.017] lag 0-5

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Ko et al. (2007, 092844)	Hospital Admission	**Pollutant:** $PM_{2.5}$	**PM Increment:** 10.0 $\mu g/m^3$
Period of Study: 1/2000-12/2005	**Outcome (ICD-9):** Asthma (493)	**Averaging Time:** 24-h	**RR Estimate**
Location: Hong Kong, China	**Age Groups:** All, 0-14, 15-56, 65+	**Mean (SD):** 36.4 (21.1)	Asthma (Single-pollutant model): 1.008[1.004, 1.013]
	Study Design: Time series	**Percentiles:**	lag 0
	N: 69,716 admissions, 15 hospitals	25th: 20.0	1.004[1.000, 1.009]
	Statistical Analyses: Poisson regression, with GAM with stringent convergence criteria.	50th(Median): 32.5	lag 1
		75th: 47.7	1.004[1.000, 1.009]
	Covariates: Time trend, season, temperature, humidity, other cyclical factors	**Range (Min, Max):** (6, 163)	lag 2
		Monitoring Stations: 14	1.009[1.005, 1.014]
	Season: All year, evaluated effect of season in analysis	**Copollutant (correlation):** $PM_{2.5}$:	lag 3
		PM_{10}	1.006[1.001, 1.011]
	Dose-response Investigated? No	r = 0.956	lag 4
	Statistical Package: SPLUS 4.0	NO_2	1.002[0.998, 1.007]
	Lags Considered: 0-5 days	r = 0.774	lag 5
		O_3	1.009[1.004, 1.014]
		r = 0.585	lag 0-1
		SO_2	1.012[1.007, 1.018]
		r = 0.482	lag 0-2
			1.017[1.011, 1.022]
			lag 0-3
			1.020[1.014, 1.026]
			lag 0-4
			1.021[1.015, 1.028]
			lag 0-5
			Asthma in Age
			0-14: 1.024[1.013, 1.034]
			lag 0-5
			14-65: 1.018[1.008, 1.029]
			lag 0-5
			>65: 1.021[1.012, 1.030]
			lag 0-4
			Asthma–Cold Season: 1.139[1.043, 1.244] lag 0-5

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Lee et al. (2006, 090176) **Period of Study:** 1/1997-12/2002 **Location:** Hong Kong, China	Hospital Admission **Outcome:** Asthma (493) **Age Groups:** < 18 years **Study Design:** Time series **N:** 26,663 asthma admissions for asthma and 5821 admissions for influenza **Statistical Analyses:** Poisson regression, GAM **Covariates:** Temperature, atmospheric pressure, relative humidity **Season:** All **Dose-response Investigated?** No **Statistical Package:** SAS 8.02 **Lags Considered:** 0-5 **Notes:** Controls were admissions for influenza ICD9 487	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-hs **Mean (SD):** 45.3 $\mu g/m^3$, (16.2) **Percentiles:** 25th: 33.4 50th(Median): 43.0 75th: 54.0 **Range (Min, Max):** NR **Monitoring Stations:** 10 **Copollutant (correlation):** $PM_{2.5}$-PM_{10}: 0.89 $PM_{2.5}$-SO_2: 0.48 $PM_{2.5}$-NO_2: 0.74 $PM_{2.5}$-O_3: 0.47	**PM Increment:** IQr = 20.6 $\mu g/m^3$ Percent increase: Single pollutant model: 5.10 [2.95, 7.30], lag 0 5.00 [2.88, 7.16], lag 1 5.48 [2.75, 6.95], lag 2 4.83 [2.78, 6.93], lag 3 6.59 [4.51, 8.72], lag 4 5.24 [3.18, 7.34], lag 5 Multipollutant model (SO_2, NO_2, CO, O_3) 3.24 [0.93, 5.60], lag 4
Reference: Letz and Quinn (2005, 088752) **Period of Study:** Oct 1, 2001–Aug 24, 2002 **Location:** San Antonio, Texas	Emergency Dept Visits **Outcome (ICD-9):** Asthma or reactive airway disease (493.0-493.9), wheezing (786.07), dyspnea (786.01-786.9), shortness of breath (786.05), bronchitis (490-496), or cough (786.2) **Age Groups:** NR (basic air force trainees) **Study Design:** Historic (retrospective) cohort **N:** 149 ED visits **Statistical Analyses:** Pearson correlation **Covariates:** NR **Season:** NR **Dose-response Investigated?** No **Statistical Package:** SPSS **Lags Considered:** NR	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-h AQI **AQI Range (min-max):** (4-109) **Monitoring Stations:** Data obtained from the Texas Commission on Environmental Quality **Copollutant (correlation):** NR	**PM Increment:** NR Correlation with Outcomes: Same-day All visits: r = 0.082 Proven asthmatic events: r = -0.042 3-day All visits: r = 0.097 Proven asthmatic events: r = 0.011
Reference: Lin et al. (2005, 087828) **Period of Study:** 1998-2001 **Location:** Toronto, North York, East York, Etobicoke, Scarborough, and York (Canada)	Hospital Admissions **Outcome (ICD-9):** Respiratory infections including laryngitis, tracheitis, bronchitis, bronchiolitis, pneumonia, and influenza (464, 466, 480-487) **Age Groups:** 0-14 yrs **Study Design:** Bidirectional case-crossover **N:** 6782 respiratory infection hospitalizations **Statistical Analyses:** Conditional logistic regression (Cox proportional hazards model) **Covariates:** Daily mean temp and dew point temp **Season:** NR **Dose-response Investigated?** No **Statistical Package:** SAS 8.2 PHREG procedure **Lags Considered:** 1-7 day averages	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean (min-max):** 9.59 (0.25-50.50) SD = 7.06 **Monitoring Stations:** 4 **Copollutant (correlation):** $PM_{10-2.5}$: r = 0.33 PM_{10}: r = 0.87 CO: r = 0.10 SO_2: r = 0.47 NO_2: r = 0.48 O_3: r = 0.56	**PM Increment:** 7.8 $\mu g/m^3$ OR Estimate [CI]: Adjusted for weather 4 day avg: 1.11 [1.02,1.22] 6 day avg: 1.11 [1.00,1.24] Adj for weather and other gaseous pollutants 4 day avg: 0.94 [0.81,1.08] 6 day avg: 0.90 [0.76,1.07] **Notes:** OR's were also categorized into "Boys" and "Girls," yielding similar results

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Lin et al. (2002, 026067) **Period of Study:** Jan 1, 1981–Dec 31, 1993 **Location:** Toronto	Hospital Admissions **Outcome (ICD-9):** Asthma (493) **Age Groups:** 6-12 yrs **Study Design:** Uni- and bi-directional case-crossover (UCC, BCC) and time-series (TS) **N:** 7,319 asthma admissions **Statistical Analyses:** Conditional logistic regression, GAM **Covariates:** Maximum and minimum temp, avg relative humidity **Season:** Apr-Sep, Oct-Mar **Dose-response Investigated?** No **Statistical Package:** NR **Lags Considered:** 1-7 day averages	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 6 days (predicted daily values) **Mean (min-max):** 17.99 (1.22-89.59) SD = 8.49 **Monitoring Stations:** 1 **Copollutant (correlation):** PM_{10}: r = 0.87 $PM_{10-2.5}$: r = 0.44 CO: r = 0.45 SO_2: r = 0.46 NO_2: r = 0.50 O_3: r = 0.21	**PM Increment:** 9.3 $\mu g/m^3$ **RR Estimate [CI]:** Adj for weather and gaseous pollutants BCC 5 day avg: 0.94 [0.85,1.03] BCC 6 day avg: 0.92 [0.83,1.02] TS 5 day avg: 0.96 [0.90,1.02] TS 6 day avg: 0.94 [0.88,1.01] Boys–adj for weather UCC 1 day avg: 1.09 [1.04,1.15] UCC 2 day avg: 1.09 [1.02,1.16] BCC 1 day avg: 1.01 [0.97,1.06] BCC 2 day avg: 0.99 [0.93,1.05] TS 1 day avg: 1.00 [0.97,1.04] TS 2 day avg: 0.98 [0.94,1.02] Girls–adj for weather UCC 1 day avg: 1.06 [0.99,1.14] UCC 2 day avg: 1.11 [1.02,1.21] BCC 1 day avg: 0.99 [0.93,1.06] BCC 2 day avg: 1.02 [0.94,1.09] TS 1 day avg: 0.99 [0.95,1.04] TS 2 day avg: 1.00 [0.95,1.06] **Notes:** The author also provides RR using UCC, BCC, and TS analysis for female and male groups for days 3-7, yielding similar results
Reference: Magas et al. (2007, 090714) **Period of Study:** 2001-2003 **Location:** Oklahoma City Metro area, Oklahoma and Cleveland counties	Hospital Admission/ED: Admissions **Outcome:** Asthma 493.01-493.99 **Age Groups:** < 15 yrs **Study Design:** Time series **N:** 1,270 admissions **Statistical Analyses:** Negative binomial regression **Covariates:** Temperature, humidity, pollen count, mold **Season:** All **Dose-response Investigated?** No **Statistical Package:** NR **Lags Considered:** 1	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h avg **Mean (SD):** NR **Range (Min, Max):** NR **Monitoring Stations:** 10 **Copollutant (correlation):** NR	**Notes:** Coefficient for $PM_{2.5}$ was not significant and thus not reported.

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Mohr et al. (2008, 180215) **Period of Study:** Jun 2001 – May 2003 **Location:** St. Louis, MO	**Outcome:** Asthma ER Visits **Age Groups:** 2-17 yrs **Study Design:** Time series **Statistical Analyses:** GEE Poisson models **Covariates:** season, weekend exposure, allergens **Dose-response Investigated:** No **Statistical Package:** SAS **Lags Considered:** 1d	**Pollutant:** $PM_{2.5}$ EC **Averaging Time:** 24 h **Std Dev:** 0.1 **Monitoring Stations:** 1 **Copollutant:** NO_x, SO_2, O_3 **Co-pollutant Correlation** NO_x: 0.68* SO_2: 0.09 O_3: -0 06 *p¤0.05	**PM Increment:** 0.1 $\mu g/m^3$ **Relative Risk Effect (Lower CI, Upper CI):** Weekend Exposure Summer: 1.05 (1.00, 1.11) Fall: 0.99 (0.97, 1.01) Winter: 0.96 (0.92, 1.00) Spring: 0.96 (0.92, 1.00) Weekday Exposure Summer: 1.01 (0.98, 1.03) Fall: 1.00 (0.99, 1.01) Winter: 0.99 (0.96, 1.01) Spring: 0.98 (0.96, 1.01)
Reference: Neuberger et al. (2004, 093249) **Period of Study:** 1999-2000 (1 yr period) **Location:** Vienna and Lower Austria	Hospital Admissions **Outcome (ICD-9):** Bronchitis, emphysema, asthma, bronchiectasis, extrinsic allergic alveolitis, and chronic airway obstruction (490-496) **Age Groups:** 3.0-5.9 yrs 7-10 yrs 65+ **Study Design:** Time series **N:** 366 days (admissions NR) **Statistical Analyses:** GAM **Covariates:** SO_2, NO, NO_2, O_3, temperature, humidity, and day of the week **Season:** NR **Dose-response Investigated?** Yes **Statistical Package:** S-Plus 2000 **Lags Considered:** 0-14 days	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Maximum daily mean:** Vienna: 96.4 Rural area: 48.0 **Monitoring Stations:** NR **Copollutant (correlation):** NR	**PM Increment:** 10 $\mu g/m^3$ Log Relative Rate Estimate (p-value): Vienna Male: 2 day lag = 5.467 (0.019) Female: 3 day lag = 5.596 (0.009) Rural Male: 10 day lag = 9.893 (0.012) Female: 11 day lag = 10.529 (0.011) Association with tidal lung functioN: β = -0.987 (p-value = 0.091) **Notes:** Effect parameters with significant coefficients for respiratory health included: male sex, allergy, asthma in family, and traffic for Vienna and age, allergy, asthma in family, passive smoking, and PM fraction for the rural area. Effect parameters with significant coefficients for log asthma score were allergy, asthma in family, and rain for Vienna and allergy, asthma in family, and passive smoking for the rural area. Cross-correlation coefficients are provided in Fig 1.

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Ostro et al. (2008, 097971) **Period of Study:** 2000-2003 **Location:** Six California Counties	**Outcome:** Respiratory disease (ICD-9 460-519) **Study Design:** Time-Series **Statistical Analysis:** Poisson Regression **Statistical Package:** R **Age Groups:** Children < 19	**Pollutant:** $PM_{2.5}$ and components **Averaging Time:** 24h **Mean (SD) Unit:** 19.4 $\mu g/m^3$ **IQR:** 14.6 $\mu g/m^3$ **Copollutants:** EC, OC, NO_2, SO_4, Cu, Fe, K, Si, Zn	**Increment:** NR **Relative Risk (Min CI, Max CI)** **Lag** Full results are presented graphically in Figures 1 and 2. Excess risks for all-year respiratory hospital admissions in children < 19yrs, 3d lag $PM_{2.5}$: 4.1% (1.8-6.4) EC: 5.4% (0.8-10.3) Fe: 4.7% (2.2-7.2) OC: 3.4% (1.1-5.7) Nitrates: 3.3% (1.1-5.5) Sulfates: 3.0% (0.4-5.7) Excess risks for cool season (Oct - Mar) respiratory hospital admissions in children < 19yrs, 3d lag $PM_{2.5}$: 5.1% (1.6-8.9) EC: 6.8% (-0.2-14.2) Fe: 4.8% (1.7-8.0) K: 4.0% (0.3-7.7)

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Slaughter et al. (2005, 073854) **Period of Study:** January 1995 through June 2001 **Location:** Spokane, WA	Hospital Admissions and ED visits **Outcome:** All respiratory (460-519) Asthma (493) COPD (491,492, 494,496) Pneumonia (480-487) Acute URI not including colds and sinusitis (464, 466, 490) **Age Groups:** All, 15+ years for COPD **Study Design:** Time series **N:** 2373 visit records **Statistical Analyses:** Poisson regression, GLM with natural splines. For comparison also used GAM with smoothing splines and default convergence criteria. **Covariates:** Season, temperature, relative humidity, day of week **Season:** All **Dose-response Investigated?:** No **Statistical Package:** SAS, SPLUS **Lags Considered:** 1 -3 d	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h avg **Range (90% of Concentrations):** 4.2-20.2 $\mu g/m^3$ **Monitoring Stations:** One **Notes: Copollutant (correlation):** $PM_{2.5}$ PM_1 r = 0.95 PM_{10} r = 0.62 $PM_{10-2.5}$ r = 0.31 CO r = 0.62 Temperature r = 0.21	**PM Increment:** 10 $\mu g/m^3$ **RR Estimate [Lower CI, Upper CI]** **lag:** **ER visits:** $PM_{2.5}$ All Respiratory Lag 1: 1.01 [0.98, 1.04] Lag 2: 1.02 [0.99, 1.04] Lag 3: 1.02 [0.99, 1.05] Acute Asthma Lag 1: 1.03 [0.98, 1.09] Lag 2: 1.00 [0.95, 1.05] Lag 3: 1.01 [0.96, 1.06] COPD (adult) Lag 1: 0.96 [0.89, 1.04] Lag 2: 1.01 [0.93, 1.09] Lag 3: 1.00 [0.93, 1.08] **Hospital Admissions:** $PM_{2.5}$ All Respiratory Lag 1: 0.98 [0.94, 1.01] Lag 2: 0.99 [0.96, 1.03] Lag 3: 1.01 [0.98, 1.05] Asthma Lag 1: 1.01 [0.91, 1.11] Lag 2: 1.03 [0.94, 1.13] Lag 3: 1.02 [0.93, 1.13] COPD (adult) Lag 1: 0.99 [0.91, 1.08] Lag 2: 1.06 [0.98, 1.16] Lag 3: 1.03 [0.94, 1.12]

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Tecer et al. (2008, 180030)	**Outcome:** ED visits for respiratory problems (ICD-9 470-478, 493)	**Pollutant:** $PM_{2.5}$	**Increment:** 10 $\mu g/m^3$
Period of Study: 12/2004-10/	**Study Design:** Bidirectional Case-crossover	**Averaging Time:** NR	**Odds Ratio (95% CI)**
Location: Zonguldak, Turkey	**Covariates:** Daily meteorological parameters	**Mean, Unit:** 29.1 $\mu g/m^3$	Asthma
	Statistical Analysis: Conditional logistic regression	**Range (Min, Max):** 4.55, 95.65	Lag 0: 1.15 (0.99-1.34)
	Statistical Package: Stata	**Copollutant (correlation):**	Lag 1: 0.85 (0.70-1.03)
	Age Groups: 0-14 years	$PM_{2.5}/PM_{10}$	Lag 2: 0.87 (0.73-1.04)
		Mean: 0.56	Lag 3: 0.93 (0.79-1.10)
		Range: 0.17-0.88	Lag 4: 1.25 (1.05-1.50)
		$PM_{2.5}/PM_{10-2.5}$	Allergic Rhinitis with Asthma
		Mean: 1.49	Lag 0: 1.21 (1.10-1.33)
		Range: 0.21-7.53	Lag 1: 0.84 (0.75-0.93)
			Lag 2: 0.89 (0.81-0.98)
			Lag 3: 0.99 (0.90-1.09)
			Lag 4: 1.06 (0.95-1.19)
			Allergic Rhinitis
			Lag 0: 1.08 (0.98-1.20)
			Lag 1: 1.03 (0.93-1.13)
			Lag 2: 0.89 (0.80-0.99)
			Lag 3: 0.98 (0.89-1.09)
			Lag 4: 1.18 (1.00-1.24)
			Upper Respiratory Disease
			Lag 0: 0.99 (0.49-2.00)
			Lag 1: 0.52 (0.22-1.20)
			Lag 2: 1.29 (0.75-2.22)
			Lag 3: 1.29 (0.69-2.43)
			Lag 4: 1.47 (0.87-2.50)
			Lower Respiratory Disease
			Lag 0: 1.06 (0.78-1.44)
			Lag 1: 0.85 (0.59-1.22)
			Lag 2: 1.08 (0.72-1.61)
			Lag 3: 1.18 (0.92-1.52)
			Lag 4: 0.72 (0.54-0.96)h

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Tolbert et al. (2007, 090316) **Period of Study:** August 1998–December 2004 **Location:** Atlanta Metropolitan area, Georgia	**Outcome (ICD-9):** Combined RD group, including: Asthma (493, 786.07, 786.09), COPD (491, 492, 496), URI (460–465, 460.0, 477), pneumonia (480–486), and bronchiolitis (466.1, 466.11, and 466.19)) **Age Groups:** All **Study Design:** Time series **N:** NR for 1998–2004. For 1993–2004: 10,234,490 ER visits (283,360 and 1,072,429 visits included in the CVD and RD groups, respectively) **Statistical Analyses:** Poisson generalized linear models **Covariates:** long-term temporal trends, season (for RD outcome), temperature, dew point, days of week, federal holidays, hospital entry and exit **Season:** All **Dose-response Investigated:** No **Statistical Package:** SAS version 9.1 **Lags Considered:** 3-day moving avg(lag 0 -2)	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h Mean (median IQR, range, 10th–90th percentiles): $PM_{2.5}$: 17.1 (15.6 11.0–21.9 0.8–65.8 7.9–28.8) $PM_{2.5}$ sulfate: 4.9 (3.9 2.4–6.2 0.5–21.9 1.7–9.5) $PM_{2.5}$ organic carbon: 4.4 (3.8 2.7–5.3 0.4–25.9 2.1–7.2) $PM_{2.5}$ elemental carbon: 1.6 (1.3 0.9–2.0 0.1–11.9 0.6–3.0) $PM_{2.5}$ water-soluble metals: 0.030 (0.023 0.014–0.039 0.003–0.202 0.009–0.059) **Monitoring Stations:** 1 **Copollutant (correlation):** Between $PM_{2.5}$ and: PM_{10}: r = 0.84 O_3: r = 0.62 NO_2: r = 0.47 CO: r = 0.47 SO_2: r = 0.17 PM_{10}-2.5: r = 0.47; $PM_{2.5}$ SO_4: r = 0.76; $PM_{2.5}$ EC: r = 0.65; $PM_{2.5}$ OC: r = 0.70; $PM_{2.5}$ TC: r = 0.71; $PM_{2.5}$ water-sol metals: r = 0.69 OHC: r = 0.50 Between $PM_{2.5}$ SO_4 and: PM_{10}: r = 0.69 O_3: r = 0.56 NO_2: r = 0.14 CO: r = 0.14 SO_2: r = 0.09 PM_{10}-2.5: r = 0.32; $PM_{2.5}$: r = 0.76; $PM_{2.5}$ EC: r = 0.32; $PM_{2.5}$ OC: r = 0.33; $PM_{2.5}$ TC: r = 0.34; $PM_{2.5}$ water-sol metals:	**PM Increment:** $PM_{2.5}$: 10.96 $\mu g/m^3$ (IQR) $PM_{2.5}$ sulfate: 3.82 $\mu g/m^3$ (IQR) $PM_{2.5}$ total carbon: 3.63 $\mu g/m^3$ (IQR) $PM_{2.5}$ organic carbon: 2.61 $\mu g/m^3$ (IQR) $PM_{2.5}$ elemental carbon: 1.15 $\mu g/m^3$ (IQR) $PM_{2.5}$ water-soluble metals: 0.03 $\mu g/m^3$ (IQR) Risk ratio [95% CI] (single pollutant models): $PM_{2.5}$: RD: 1.005 [0.995–1.015] $PM_{2.5}$ sulfate: RD: 1.007 [0.996–1.018] $PM_{2.5}$ total carbon: RD: 1.001 [0.993–1.008] $PM_{2.5}$ organic carbon: RD: 1.003 [0.995–1.011] $PM_{2.5}$ elemental carbon: RD: 0.996 [0.989–1.004] $PM_{2.5}$ water-soluble metals: RD: 1.005 [0.995–1.015]

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Wong et al. (2006, 093266) **Period of Study:** 2000-2002 **Location:** Hong Kong (8 districts)	General Practitioner Visits **Outcome (ICPC-2):** Respiratory diseases/symptoms: upper respiratory tract infections (URTI), lower respiratory infections, influenza, asthma, COPD, allergic rhinitis, cough, and other respiratory diseases **Age Groups:** All ages **Study Design:** Time series **N:** 269,579 visits **Statistical Analyses:** GAM, Poisson regression **Covariates:** Season, day of the week, climate **Season:** NR **Dose-response Investigated?** No **Statistical Package:** S-Plus **Lags Considered:** 0-3 days	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean (min-max):** 35.7 (9-120) SD = 16.7 **Monitoring Stations:** 1 per district **Copollutant (correlation):** PM_{10}: r = 0.94	**PM Increment:** 10 $\mu g/m^3$ RR Estimate [CI]: Overall URTI 1.021 [1.010,1.032] **Notes:** RRs are also reported for each individual general practitioner yielding similar results
Reference: Yang Q et al. (2004, 087488) **Period of Study:** Jun 1, 1995–Mar 31, 1999 **Location:** Vancouver area, British Columbia	Hospital Admissions **Outcome (ICD-9):** Respiratory diseases (460-519), pneumonia only (480-486), asthma only (493) **Age Groups:** 0-3 yrs **Study Design:** Case control, bidirectional case-crossover (BCC), and time series (TS) **N:** 1610 cases **Statistical Analyses:** Chi-square test, Logistic regression, GAM (time-series), GLM with parametric natural cubic splines **Covariates:** Gender, socioeconomic status, weekday, season, study year, influenza epidemic month **Season:** Spring, summer, fall, winter **Dose-response Investigated?** No **Statistical Package:** SAS (Case control and BCC), S-Plus (TS) **Lags Considered:** 0-7 days	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean (min-max):** 7.7 (2.0-32.0) SD = 3.7 **Monitoring Stations:** NR (data obtained from Greater Vancouver Regional District Air Quality Dept) **Copollutant (correlation):** PM_{10}: r = 0.83 $PM_{10-2.5}$: r = 0.39 CO: r = 0.24 O_3: r = -0.03 NO_2: r = 0.37 SO_2: r = 0.43	**PM Increment:** 4.0 $\mu g/m^3$ (IQR) OR Estimate [CI]: Values NR **Notes:** Author states that no significant association was found between $PM_{2.5}$ and respiratory disease hospitalizations.

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Zanobetti and Schwartz (2006, 090195) **Period of Study:** 1995-1999 **Location:** Boston, MA	Hospital Admission/ED: **Outcome:** Pneumonia (480-487) **Age Groups:** >65 y **Study Design:** Case-crossover, time stratified **N:** 24,857 for Pneumonia **Statistical Analyses:** Condition logistic regression **Covariates:** Season, long term trend, day of-the-wk, mean temperature, relative humidity, barometric pressure, extinction coefficient **Season:** All year **Dose-response Investigated?** No **Statistical Package:** SAS **Lags Considered:** 0-1 **Notes:** Also looked at MI cohort	**Pollutant:** PM non-traffic **Averaging Time:** 24 h **Percentiles (pneumonia cohort):** 5th: -7.3 25th: -3.28 μg/m^3 50th(Median): -0.88 75th: 1.92 95th: 12.11 **PM Component:** BC **Monitoring Stations:** 4-5 monitors **Copollutant (correlation):** PM non-traffic: $PM_{2.5}$ r = 0.74 CO r = -0.01 NO_2 r = 0.14 O_3 r = -0.47 BC r = -0.01	**PM Increment:** PM non-traffic lag 0: 13.44 μg/m^3 PM non-traffic lag 0-1 avg: 10.28 μg/m^3 % change in Pneumonia: PM non-traffic -0.57 [-7.51, 6.36] lag 0 PM non-traffic -0.94 [-7.20, 5.32] mean lag 1
Reference: Zhong et al. (2006, 093264) **Period of Study:** Apr–Oct 2002 **Location:** Cincinnati, Ohio	Hospital Admissions **Outcome (ICD-9):** Asthma (493-493.91) **Age Groups:** 1-18 yrs **Study Design:** Time series **N:** 1254 admissions **Statistical Analyses:** Poisson multiple regression, GAM **Covariates:** Season, temperature, humidity, ozone, day of the week **Season:** NR **Dose-response Investigated?** Yes **Statistical Package:** NR **Lags Considered:** 1-5 days	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean (SD):** Apr: 12.4 (3.8) May: 13.6 (5.8) Jun: 21.6 (9.9) Jul: 25.8 (11.9) Aug: 20.3 (8.7) Sep: 19.5 (11.1) Oct: 12.8 (6.4) **Monitoring Stations:** NR (data obtained from the National Virtual Data System) **Copollutant (correlation):** NR **Notes:** Author states all pairwise correlations were insignificant	**PM Increment:** NR RR Estimate [CI]: NR **Notes:** This study focused primarily on aeroallergens and asthma visits

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Zanobetti and Schwartz (2006, 090195)	**Outcome:** Pneumonia (480-487)	**Pollutant:** $PM_{2.5}$	**PM Increment:** $PM_{2.5}$ lag 0: 17.17 $\mu g/m^3$
Period of Study: 1995-1999	**Age Groups:** > 65 y	**Averaging Time:** 24 h	$PM_{2.5}$ lag 0-1 avg: 16.32 $\mu g/m^3$
Location: Boston, MA	**Study Design:** Case-crossover, time stratified	**Percentiles** (pneumonia cohort)**:**	% change in Pneumonia:
	N: 24,857 for Pneumonia	25th: 7.23 $\mu g/m^3$	6.48[1.13, 11.43]
	Statistical Analyses: Condition logistic regression	50th(Median): 11.10	lag 0
	Covariates: Season, long term trend, day of-the-wk, mean temperature, relative humidity, barometric pressure, extinction coefficient	75th: 16.14	5.56[-0.45, 11.27]
		PM Component: Black Carbon (BC), PM non-traffic	mean lag 1
	Season: All year	**Monitoring Stations:**	
	Dose-response Investigated? No	4-5 monitors	
	Statistical Package: SAS	**Copollutant (correlation):**	
	Lags Considered: 0-1	$PM_{2.5}$:	
	Notes: Also looked at MI cohort	CO	
		r = 0.52	
		NO_2	
		r = 0.55	
		O_3	
		r = 0.20	
		BC	
		r = 0.66	
		PM non-traffic	
		r = 0.74	

[1]All units expressed in $\mu g/m^3$ unless otherwise specified.

Table E-15. Short-term exposure–respiratory–ED/HA-Other Size Fractions

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Andersen et al. (2007, 093201)	**Outcome (ICD10):** Respiratory disease (J41-46)	**Pollutant:** Number concentration (NC) of ultrafine & accumulation mode particles	**PM Increment:** Based on the IQR, specific to metric (see below).
Period of Study: 2001-2004	Asthma (J45, 46)	**Averaging Time:** 24-h	**RR Estimate:**
Location: Copenhagen, Sweden	**Age Groups:** 5-18 and > 65	**Mean particles/cm^3 (SD):** NCtot (total): 8116 (3502)	Single pollutant results, Asthma, (5-18 yrs), lag 0-5:
	Study Design: Time-series	25th: 4959	$PM_{2.5}$: 1.15 [1, 1.32], IQr = 5
	N: 1327 days	50th: 6243	NCtot: 1.07 [0.98, 1.17], IQr = 3907
	~ 1.5 million people at-risk	75th: 8218	NC100: 1.06 [0.97, 1.16], IQr = 3259
	Statistical Analyses: Poisson regression, GAM.	99th: 16189	NCa12: 1.08 [0.99, 1.18], IQr = 342
	Covariates: influenza epidemics, pollen, temperature, dew point, day-of-week, holiday, season.	IQR: 3259	NCa212: 1.08 [1, 1.17], IQr = 495
		NC100 (< 100 nm): 6847 (2864)	NCa23: 1.09 [0.98, 1.21], IQr = 1786
	Season: All	25th: 5738	NCa57: 1.02 [0.94, 1.12], IQr = 3026
	Dose-response Investigated? No	50th (Median): 7358	2-pollutant results:
	Statistical Package: R with *gam* and *mgcv* packages.	75th: 9645	NCa212 w/ PM_{10}: 1.1 [0.96, 1.13], IQr = 495
	Lags Considered: 0-5	99th: 19895	NCtot w/ PM_{10}: 1.03 [0.92, 1.15]
		IQR: 3907	NCtot w/ $PM_{2.5}$: 1.04 [0.85, 1.28]
		Mean particles/cm^3 for four size modes (median diameter (nm) noted):	All RD, (> 65 yrs), lag 0-4, single pollutant results:
		NCa12: 493(315)	$PM_{2.5}$: 1 [0.95, 1.05]
		NCa23: 2253 (1364)	NCtot: 1.04 [1, 1.07] IQr = 3907
		NCa57: 5104 (2687)	NC100: 1.03 [0.99, 1.07], IQr = 3259
		NCa212: 6847 (2864)	NC12: 1.01 [0.98, 1.05], IQr = 342
		Monitoring Stations: 3 (Background, rural Background, urban Curbside, urban)	NC212: 1.04 [1.01, 1.08], IQr = 495
		Notes: NC exposure data available for n = 578 days. Information on distribution of 4 size modes provided in the paper.	NCa23: 0.99 [0.94, 1.03], IQr = 1786
		Copollutant (correlation):	NCa57: 1.04 [1, 1.08], IQr = 3026
		NCtot and PM_{10}: r = 0.39	2-pollutant results:
		NCtot and $PM_{2.5}$: r = 0.40	NCa212 w/ PM_{10}: 1.01 [0.96, 1.07], IQr = 495
		NCtot and NO_2: r = 0.68	NCtot w/ $PM_{2.5}$: 0.97 [0.89, 1.05]
		PM_{10} and $PM_{2.5}$: r = 0.8	NCtot w/ PM_{10}: 1 [0.96, 1.05]
		"Low or no" correlations between 4 size modes	**Notes:** Multipollutant model results also included for models with 4 size modes.
		NCa212 and $PM_{2.5}$: r = 0.8	
		NCa212 and PM_{10}: r = 0.63	
		NCa57 and NO_2: r = 0.57	
		Notes: selected correlations reported in text, all correlations in annex to the manuscript	

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Agarwal et al. (2006, 099086) **Period of Study:** 2000-2003 **Location:** Safdarjung area of Delhi	**Outcome (ICD-NR):** COPD, asthma, emphysema **Age Groups:** NR **Study Design:** Time series **N:** NR **Statistical Analyses:** Kruskal-Wallis one-way analysis, Chi-square, Multivariate linear regression **Covariates:** Temp (min & max), relative humidity at 0830 and 1730 h, wind speed **Season:** I (Jan-Mar), II (Apr-Jun), III (Jul-Sep), IV (Oct-Dec) **Dose-response Investigated?** Yes **Statistical Package:** SPSS **Lags Considered:** NR	**Pollutant:** SPM (Suspended PM) **Averaging Time:** 8 h **Mean $\mu g/m^3$ (SD):** Qtr I: 297.5 (34.6) Qtr II: 398.0 (85.6) Qtr III: 220.0 (78.0) Qtr IV: 399.0 (54.6) **Monitoring Stations:** 2 **Copollutant (correlation):** RSPM: r = 0.771 **Other variables:** RH0830: r = -0.482 RH1730: r = -0.531 COPD: r = 0.474	**PM Increment:** NR **RR Estimate [CI]:** NR **Notes:** This study analyzed seasonal variation of pollutants and health outcomes and correlations among the variables
Reference: Agarwal et al. (2006, 099086) **Period of Study:** 2000-2003 **Location:** Safdarjung area of Delhi	**Outcome (ICD-NR):** COPD, asthma, emphysema **Age Groups:** NR **Study Design:** Time series **N:** NR **Statistical Analyses:** Kruskal-Wallis one-way analysis, Chi-square, Multivariate linear regression **Covariates:** Temp (min & max), relative humidity at 0830 and 1730 h, wind speed **Season:** I (Jan-Mar), II (Apr-Jun), III (Jul-Sep), IV (Oct-Dec) **Dose-response Investigated?** Yes **Statistical Package:** SPSS **Lags Considered:** NR	**Pollutant:** RSPM (Respirable Suspended PM $< 10\ \mu m$) **Averaging Time:** 8 h **Mean $\mu g/m^3$ (SD):** Qtr I: 119.0 (19.8) Qtr II: 132.0 (28.4) Qtr III: 75.0 (23.4) Qtr IV: 168.0 (40.6) **Monitoring Stations:** 2 **Copollutant (correlation):** SPM: r = 0.771 **Other variables:** Temp (min): r = -0.420 COPD: r = 0.353	**PM Increment:** NR **RR Estimate [CI]:** NR **Notes:** This study analyzed seasonal variation of pollutants and health outcomes and correlations among the variables

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Arbex et al. (2007, 091637) **Period of Study:** Mar 2003-Jul 2004 **Location:** Araraquara, Sao Paulo State, Brazil	Hospital Admission **Outcome (ICD10):** Asthma (J15, J45) **Age Groups:** All **Study Design:** Time-series **N:** 493 days, 1 hospital, 640 admissions **Statistical Analyses:** Generalized linear Poisson regression model with natural cubic spline, Mann-Whitney U Test **Covariates:** Temperature and humidity **Season:** All **Dose-response Investigated?** Yes, quintile analysis **Statistical Package:** SPSS V.11 & Splus 4.5 **Lags Considered:** 0-9	**Pollutant:** TSP **Averaging Time:** 24 h **Mean (SD):** 46.8 μg/m^3 (24.4) **Range (Min, Max):** 6.7-137.8 μg/m^3 **Monitoring Stations:** 1 **Notes:** TSP used as a proxy for fine & ultrafine particles since it is composed of 85-95% $PM_{2.5}$. **Copollutant (correlation):** NR	**PM Increment:** 10 μg/m^3 **% Increase** 6.96 [1.4-12.86] 2-d ma 9.090 [3.12-15.40] 3 d ma 10.28 [4.05-16.90] 4-d ma 11.63 [5.46-19.318] 5 d ma 12.61 [5.68-20.00] 6-d ma 12.56 [5.47-20.13] 7-d ma **% Increase by TSP quintile:** 9.25-28.45 μg/m$^{3::}$ 1.00 28.46-48.85 μg/m$^{3::}$ 1.55 [045-5.77] 48.86-69.06 μg/m$^{3::}$ 2.46 [1.08-5.60] 69.07-88.44 μg/m$^{3::}$ 2.77 [1.32-5.84] 88.45-108.9 μg/m$^{3::}$ 2.94 [1.48-5.85] **Notes:** No TSP threshold for asthma admissions noted. Analysis of lag structure indicated that the acute effect of TSP on admissions started 1 day after TSP concentration increase and remained unchanged for next 4 days. **Notes:** To evaluate the association between TSP generated from burning sugar cane and asthma hospital admissions.
Reference: Bartzokas et al. (2004, 093252) **Period of Study:** Jun 1, 1992–May 31, 2000 **Location:** Athens, Greece	**Outcome:** Respiratory and cardiovascular diseases (combined) **Age Groups:** NR **Study Design:** Time series **N:** 1554 patients **Statistical Analyses:** Simple linear regression and linear stepwise regression, Pearson correlation **Covariates:** Temperature, atmospheric pressure, relative humidity, wind speed **Season:** Warm (May-Sep) and cold (Nov-Mar) **Dose-response Investigated?** No **Statistical Package:** NR **Lags Considered:** NR	**Pollutant:** PM4.5 (black smoke) **Averaging Time:** 10-day moving avg **Mean μg/m^3 (SD):** NR **Monitoring Stations:** 1 **Copollutant (correlation):** N	**PM Increment:** NR **Correlation with Number of Admissions:** Entire year Original: r = 0.18 Smoothed: r = 0.31 Warm period Original: r = 0.19 Smoothed: r = 0.30 Cold period Original: r = 0.18 Smoothed: r = 0.34 *All above values are statistically significant

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Erbas et al. (2005, 073849) **Period of Study:** Jul 1, 1989–Dec 31, 1992 **Location:** Melbourne, Australia	**Outcome (ICD):** COPD (490-492, 494, 496) Asthma (493) **Age Groups:** NR **Study Design:** Time series **N:** NR **Statistical Analyses:** GLM, GAM, Parameter Driven Poisson Regression, Transitional Regression, Seasonal-Trend decomposition based on Loess smoothing for seasonal adjustment **Covariates:** Secular trends, seasonality, relative humidity, dry bulb temp, dew point temp **Season:** NR **Dose-response Investigated?** Yes **Statistical Package:** S-Plus, SAS **Lags Considered:** 0-5 days	**Pollutant:** PM 0.1-1 (API) **Averaging Time:** 24-hs **Mean (min-max):** NR **Monitoring Stations:** 9 **Copollutant (correlation):** NR	**PM Increment:** Increase from the 10th-90th percentile (value NR) **RR Estimate [CI]:** COPD GAM: 0.95 [0.91,1.00] GLM, PDM, TRM: NR Asthma NR **Notes:** This study was used to demonstrate that conclusions are highly dependent on the type of model used
Reference: Halonen et al. (2008, 189507) **Period of Study:** 1998-2004 **Location:** Helsinki, Finland	**Outcome:** Respiratory Hospitalizations & Mortality (ICD 10: J00-99) **Age Groups:** 65+ yrs **Study Design:** time series **N:** NR **Statistical Analyses:** Poisson, GAM **Covariates:** temperature, humidity, influenza epidemics, high pollen episodes, holidays **Dose-response Investigated?** No **Statistical Package:** R **Lags Considered:** lags 0-3 & 5d (0-4) mean	**Pollutant:** $PM_{2.5}$ **Averaging Time:** daily **Mean (SD):** NR **Min:** 1.1 **25th percentile:** 5.5 **50th percentile:** 9.5 **75th percentile:** 11.7 **Max:** 69.5 **Monitoring Stations:** NR **Copollutant:** $PM_{<0.03}$, $PM_{0.03-0.1}$, $PM_{<0.1}$, $PM_{<0.10.29}$, $PM_{2.5-10}$, CO, NO_2 **Co-pollutant Correlation** $PM_{<0.03}$: 0.14 $PM_{0.03-0.1}$: 0.48 $PM_{<0.1}$: 0.35 $PM_{<0.10.29}$: 0.88 $PM_{2.5-10}$: 0.25	**PM Increment:** Interquartile **Percent Change (Lower CI, Upper CI):** All Respiratory Mortality Lag 0: 2.67 (-0.39, 5.82) ‡ Lag 1: 1.59 (-1.43, 4.70) Lag 2: 0.03 (-2.99, 3.16) Lag 3: -0.11 (-3.13, 3.01) 5-d mean: 1.39 (-2.83, 5.81) Pneumonia HA Lag 0: 0.93 (-0.85, 2.75) Lag 1: 2.41 (0.64, 4.21) Lag 2: 1.48 (-0.27, 3.26) Lag 3: 1.91 (0.14, 3.70) 5-d mean: 3.10 (0.60, 5.65) Asthma + COPD HA Lag 0: 2.48 (0.60, 4.39) Lag 1: 2.62 (0.78, 4.49) Lag 2: 1.22(-0.62, 3.10) Lag 3: 0.59 (-1.28, 2.49) 5-d mean: 2.49 (-0.08, 5.12) Other HA Lag 0: 0.05 (-2.38, 2.54) Lag 1: 0.2 (-2.17, 2.62) Lag 2: 2.03 (-0.29, 4.41) Lag 3: 1.72 (-0.63, 4.12) 5-d mean: 1.88 (-1.50, 5.36) *$p < 0.05$, ‡$p < 0.10$

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Llorca et al. (2005, 087825) **Period of Study:** Jan 1, 1992–Dec 31, 1995 **Location:** Torrelavega, Spain	**Outcome (ICD-9):** Respiratory (460-519) and cardiac (390-459) admissions (analyzed combined and individually) **Age Groups:** NR **Study Design:** Time series **N:** 18,137 admissions **Statistical Analyses:** Stepwise multiple linear regression, Poisson regression, Spearman correlation **Covariates:** Influenza, day of week, wind speed, northeast and southwest winds, minimum and maximum temperature **Season:** NR **Dose-response Investigated?** No **Statistical Package:** STATA Intercooled, Release 6 **Lags Considered:** NR	**Pollutant:** TSP (total suspended particles) **Averaging Time:** 24 h **Mean $\mu g/m^3$ (SD):** 48.8 (23.7) **Monitoring Stations:** 3 **Copollutant (correlation):** SO_2: r = -0.400 SH2: r = -0.392 NO: r = -0.109 NO_2: r = -0.120 Other variables: Rain: r = -0.339 Max temp: r = 0.071 Min temp: r = -0.003 Avg temp: r = 0.035 Wind speed: r = -0.357	**PM Increment:** NR **Rate Ratio Estimate [CI]:** Cardiorespiratory Admissions Single-pollutant model: 0.92 [0.86,0.98] Five-pollutant model: 1.05 [0.97,1.14] Respiratory Admissions Single-pollutant model: 0.98 [0.89,1.08] Five-pollutant model: 0.91 [0.80,1.02]
Reference: Michaud et al. (2004, 089900) **Period of Study:** Jan 1997-May 2001 **Location:** Hilo, Hawaii	ED visits **Outcome:** Asthma/COPD (490-496) Respiratory Irritation (506-508) **Age Groups:** All **Study Design:** Time-series **N:** 1,561 ER visits **Statistical Analyses:** Multiple linear regression **Covariates:** Hourly temperature, minimum daily temperature, minimum daily temperature, humidity, year, month, day of the week **Season:** all **Dose-response Investigated?** No **Statistical Package:** STATA 6.0 SAS **Lags Considered:** Previous night, 1,2,3	**Pollutant:** PM_1 **Averaging Time:** 24 h avg **Mean (SD):** 1.91 (2.95) $\mu g/m^3$ **Range (Min, Max):** 0.0, 56.6 $\mu g/m^3$ **Monitoring Stations:** 2 **Notes: Copollutant (correlation):** NR	**PM Increment:** 10 $\mu g/m^3$ **RR Estimate [Lower CI, Upper CI]** **lag:** Asthma, COPD (499-496): Adjusted for day, month & year: 1.11 (0.92, 1.34), 00: 00-6: 00AM 1.14 (1.03, 1.26), lag 1 1.06 (0.83, 0.94), lag 2 0.91 (0.06, 1.05), lag 3 Asthma (493, 495): Adjusted for day, month & year: 1.03 (0.90, 1.42), 00: 00-6: 00AM 1.02 (0.94, 1.21), lag 1 1.02 (0.99, 1.23), lag 2 0.97 (0.69, 1.15), lag 3 Bronchitis (490, 491): Adjusted for day, month & year: 1.02 (0.82, 1.41), 00: 00-6: 00AM 1.07 (1.18, 1.49), lag 1 0.97 (0.60, 1.34), lag 2 0.93 (0.43, 1.18), lag 3 **Notes:** Crude and estimates adjusted for month and year only also presented. **Notes:** Volcanic fog = vog

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Migliaretti et al. (2005, 088689) **Period of Study:** 1997-1999 **Location:** Turin, Italy	**Outcome:** **Cases:** Asthma (493) **Controls:** admissions for non-respiratory or cardiac conditions (460-487, 490-493, 494-496, 500-519, 390-405, 410-429) **Age Groups:** 0-14, 15-64, >64 **Study Design:** Case-control **N:** Cases: 1,401 **Controls:** 201,071 **Statistical Analyses:** Logistic regression **Covariates:** Gender, age, daily mean temperature, season, day of week, holidays, education level **Season:** All **Dose-response Investigated?** No **Lag:** 0-2 d avg	**Pollutant:** TSP **Averaging Time:** Means of daily total levels at stations **Mean (SD):** 105.3 $\mu g/m^3$, (44.2) **Percentiles: 25th:** NR **50th(Median):** 96.0 $\mu g/m^3$ **75th** NR **Monitoring Stations:** 10 **Notes: Copollutant (correlation):** All seasons: NO_3-TSP = 0.80 Winter: NO_3-TSP = 0.77 summer: NO_3-TSP = 0.69	**PM Increment:** 10 $\mu g/m^3$ increase **% Increase, lag 0-2 d avg** **1 pollutant model:** <15: 1.90[0.40, 3.40] 15-64: 2.30 [-0.01, 5.20] >64: 2.30 [1.10, 3.60] Total: 2.30[1.10, 3.60] **% Increase, lag 0-2 d avg** **2 pollutant model:** <15: -0.12 [-0.03, 2.50] 15-64: 0.90 [-0.04, 5.61] >64: 1.2 [-0.01, 4.32] Total: 0.91 [-0.02, 3.11]
Reference: Migliaretti et al. (2004, 087425) **Period of Study:** 1997-1999 **Location:** Turin, Italy	**Outcome:** **Cases:** Asthma (493) **Controls:** non-respiratory or cardiac admissions (460-487, 490-493, 494-496, 500-519, 390-405, 410-429) **Age Groups:** 0-15 **Study Design:** Case-control **N: Cases:** 1,060 **Controls:** 25,523 **Statistical Analyses:** Logistic regression $\mu g/m^3$ increase **Covariates:** Gender, age, daily mean temperature, season, day of week, holidays, solar radiation **Season:** All **Lags Considered:** 1-3 d avg	**Pollutant:** Total suspended particulate **Averaging Time:** Mean of admission day and 3 preceding days **Mean (SD):** 114.5 $\mu g/m^3$, (42.8) **Percentiles:** 25th: NR 50th(Median): 109.9 $\mu g/m^3$ 75th: NR **Monitoring Stations:** 10 **Notes: Copollutant (correlation):** TSP-NO: 0.76	**PM Increment:** 10 $\mu g/m^3$ % Increase, lag 1-3 d avg <4 yrs: 1.8% [0.00, 3.05] 4-15 yrs: 3.0% [0.01, 5.08] all: 1.8% [0.03, 3.02] adjusted for all covariates **Notes:** Multipollutant models also used

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Neuberger et al. (2004, 093249) **Period of Study:** 1999-2000 (1 yr period) **Location:** Vienna and Lower Austria	**Outcome (ICD-9):** Bronchitis, emphysema, asthma, bronchiectasis, extrinsic allergic alveolitis, and chronic airway obstruction (490-496) **Age Groups:** 3.0-5.9 yrs 7-10 yrs 65+ **Study Design:** Time series **N:** 366 days (admissions NR) **Statistical Analyses:** GAM **Covariates:** SO_2, NO, NO_2, O_3, temperature, humidity, and day of the week **Season:** NR **Dose-response Investigated?** Yes **Statistical Package:** S-Plus 2000 **Lags Considered:** 0-14 days	**Pollutant:** PM1 **Averaging Time:** 24 h **Mean** $\mu g/m^3$ **(SD):** NR **Monitoring Stations:** NR **Copollutant (correlation):** NR	**PM Increment:** NR **Effect parameters (Vienna children):** Respiratory Health Male sex = 0.098 Allergy = 0.238 Asthma in family = 0.190 Traffic = 0.112 Log Asthma Score Allergy = 0.210 Asthma in family = 0.112 Rain = 0.257 *only significant coefficients are presented Association with tidal lung function: β = -1.059 (p-value = 0.060) **Notes:** No significant associations between PM and respiratory mortality were found for either sex. Data is also provided for children in the rural area where age, allergy, asthma in family, passive smoking, and PM fraction had significant coefficients.
Reference: Peel et al. (2005, 056305) **Period of Study:** Jan 1993-Aug 2000 **Location:** Atlanta, Georgia	**Hospital Admission/ED:** ED visits **Outcome:** Asthma 493, 786.09 COPD 491, 492, 496 URI 460-466, 477 Pneumonia 480-486 **Age Groups:** All ages. Secondary analyses conducted by age group: Infants 0-1 yrs Pediatric asthma 2-18 yrs Adults > 18 yrs **Study Design:** Case-control All respiratory disease vs. finger wounds **N:** 31 hospitals ED visits NR **Statistical Analyses:** Poisson generalized linear models General linear models **Covariates:** Avg temperature and dew point, pollen counts **Season:** All **Dose-response Investigated?** yes **Statistical Package:** SAS 8.3 S-Plus 2000 **Lags Considered:** 0-7 days and 14 day distributed lag	**Pollutant:** UF (10-100nm) **Averaging Time:** 24 h avg **Mean (SD):** 3800 (40700) **Percentiles:** 10th: 11500 90th: 74600 **PM Component:** Oxygenated hydrocarbons (OH), sulfate, acidity, elemental carbon (EC), organic carbon (OC), water-soluble transition metals **Monitoring Stations:** "Several" **Copollutant (correlation):** PM_{10}: r = -0.13 O_3: r = -0.13 NO_2: r = 0.26 CO: r = 0.10 SO_2: r = 0.24 $PM_{2.5}$: r = -0.16 $PM_{10-2.5}$: r = 0.13	**Increment:** 30,000 #/cm^3 All Respiratory Disease 0.984 [0.968-1.000] URI 0.986 [0.966, 1.006] Asthma 0.999 [0.977, 1.021] Pneumonia 0.997 [0.953, 1.002] COPD 0.982 [0.942, 1.022]

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Simpson et al. (2005, 087438) **Period of Study:** 1996-1999 **Location:** Brisbane, Sydney, Melbourne, and Perth, Australia	**Outcome:** All Respiratory (460-519) Asthma (493) COPD (490-492) Pneumonia, acute bronchitis (466, 480-486) **Age Groups:** All ages, split into f15-64 and > 64 yrs **Study Design:** Time-series **N:** NR ~64,000 admissions **Statistical Analyses:** GAM w/ LOESS smoothers GLM w/ natural and penalized spline smoothers **Covariates:** Temperature, relative humidity, rain, day of the week, public and school holidays, influenza epidemics, and controlled burn events **Season:** All **Dose-response Investigated?** Yes **Statistical Package:** S-Plus R **Lags Considered:** 1-3, 0-1 avg	**Pollutant:** BSP (indicator of particles < 2 μm in diameter) (10^{-4} m^{-1}) **Averaging Time:** 24 h avg **Mean (SD): Means only** Brisbane 0.3 10^{-4} m^{-1} Sydney 0.3 10^{-4} m^{-1} Melbourne 0.3 10^{-4} m^{-1} Perth 0.3 10^{-4} m^{-1} **Range (Min, Max):** Brisbane 0.0, 2.5 10^{-4} m^{-1} Sydney 0.0, 1.6 10^{-4} m^{-1} Melbourne 0.0, 2.2 10^{-4} m^{-1} Perth 0.1, 1.8 10^{-4} m^{-1} **PM Component: Monitoring Stations:** "network of sites across each city" **Notes: Copollutant (correlation):** NR	**PM Increment:** "per unit increase" **RR Estimate [Lower CI, Upper CI]** **lag:** **Single pollutant model** Respiratory > 64 yrs 1.0401 [1.0045, 1.0770] lag1 1.0520 [1.0164, 1.0889] lag2; 1.0451 [1.0093, 1.0821] lag3 1.0552 [1.0082, 1.1045] lag 0-1 avg Asthma 15-64 yrs 1.0641 [1.0006, 1.1315] lag2 1.0893 [1.0240, 1.1587] lag3 Asthma + COPD > 64 yrs 1.0713 [1.0179, 1.1276] lag3 1.0552 [1.0082, 1.1045] lag 0-1 avg Pneumonia & Acute Bronchitis > 64 yrs 1.0587 [1.0013, 1.1193] lag1 1.0636 [1.0056, 1.1249] lag 2 1.0769 [1.0046, 1.1544] lag 0-1 avg **Multipollutant model** Respiratory admissions > 64 yrs No other pollutants: 1.0552 [1.0082, 1.1045] lag 0-1 avg Max 1 h NO_2 1.0028 [0.9513, 1.0572] lag 0-1 avg Max 1 h O_3 1.0534 [1.0058-1.1033] lag 0-1 avg
Reference: Sinclair and Tolsma (2004, 088696) **Period of Study:** 25 Months **Location:** Atlanta, Georgia	Outpatient Visits **Outcome:** Asthma (493) URI (460, 461, 462, 463, 464, 465, 466, 477) LRI (466.1, 480, 481, 482, 483, 484, 485, 486). **Age Groups:** < = 18 y, 18+ y (asthma) All ages (URI//LRI) **Study Design:** Times series **N:** 25 months 260,000 to 275,000 health plan members (August 1998–August 2000) **Statistical Analyses:** Poisson GLM **Covariates:** Season, Day of week, Federal Holidays, Study Months **Season:** NR **Dose-response Investigated?:** No **Statistical Package:** SAS **Lags Considered:** Three 3 d moving averages (0-2, 2-5, 6-8)	**Pollutant:** $PM_{10-2.5-10}$ ($\mu g/m^3$) **Averaging Time:** 24 h avg **Mean (SD):** PM coarse mass ((2.5-10 μm))–9.67 $\mu g/m^3$ (4.74) **Monitoring Stations:** 1 **Copollutant (correlation):** NR	**PM Increment:** 4.74 (1 SD) **RR Estimate [Lower CI, Upper CI]** **lag:** Child Asthma: Coarse PM = 1.053 (S) 3-5 days lag URI: Course PM = 1.021 (S) 3-5 days lag LRI: Coarse PM = 1.07 (S) 3-5 days lag **Notes:** Numerical findings for significant results only presented in manuscript. Results for all lags presented graphically for each outcome (asthma, URI, and LRI).

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Sinclair and Tolsma (2004, 088696) **Period of Study:** 25 Months **Location:** Atlanta, Georgia	**Outpatient Visits** **Outcome:** Asthma (493) URI (460, 461, 462, 463, 464, 465, 466, 477) LRI (466.1, 480, 481, 482, 483, 484, 485, 486). **Age Groups:** < = 18 y, 18+ y (asthma) All ages (URI//LRI) **Study Design:** Times series **N:** 25 months 260,000 to 275,000 health plan members (August 1998–August 2000) **Statistical Analyses:** Poisson GLM **Covariates:** Season, Day of week, Federal Holidays, Study Months **Season:** NR **Dose-response Investigated?:** No **Statistical Package:** SAS **Lags Considered:** Three 3 d moving averages (0-2, 2-5, 6-8)	**Pollutant:** UF (PM_{10-100} nm) **Averaging Time:** 24 h avg **Mean (SD):** PM_{10-100} nm area ($\mu m^2/cm^3$)–249.33 (244.09) **Monitoring Stations:** 1 **Copollutant (correlation):** NR	**PM Increment:** NR **RR Estimate [Lower CI, Upper CI]** **lag:** Adult Asthma: Ultrafine PM area = 1.223 (S) 3-5 days lag URI: Ultrafine PM: = 1.041 (S) 0-2 days lag LRI: Ultrafine PM area = 1.099 (S) 6-8 days lag **Notes:** Numerical findings for significant results only presented in manuscript. Results for all lags presented graphically for each outcome (asthma, URI, and LRI).
Reference: Slaughter et al. (2005, 073854) **Period of Study:** January 1995-June 2001 **Location:** Spokane, WA	Hospital Admissions and ED visits **Outcome:** All respiratory (460-519) Asthma (493) COPD (491,492, 494,496) Pneumonia (480-487) Acute URI not including colds and sinusitis (464, 466, 490) **Age Groups:** All, 15+ years for COPD **Study Design:** Time series **N:** 2373 visit records **Statistical Analyses:** Poisson regression, GLM with natural splines. For comparison also used GAM with smoothing splines and default convergence criteria. **Covariates:** Season, temperature, relative humidity, day of week **Season:** All **Dose-response Investigated?:** No **Statistical Package:** SAS, SPLUS **Lags Considered:** 1 -3 d	**Pollutant:** PM_1 **Averaging Time:** 24 h avg **Range (90% of concentrations):** 3.3-17.6 $\mu g/m^3$ **Monitoring Stations:** One **Copollutant (correlation):** PM_1 $PM_{2.5}$ r = 0.95 PM_{10} r = 0.50 $PM_{10-2.5}$ r = 0.19 CO r = 0.63	**PM Increment:** 10 $\mu g/m^3$ **RR Estimate [Lower CI, Upper CI]** **lag:** ED visits: PM_1 All Respiratory Lag 1: 1.01 [0.98, 1.04] Lag 2: 1.02 [0.99, 1.06] Lag 3: 1.02 [0.99,1.06] Acute Asthma Lag 1: 1.03 [0.97, 1.09] Lag 2: 0.99 [0.93, 1.05] Lag 3: 1.02 [0.96, 1.08] COPD (adult) Lag 1: 0.96 [0.87, 1.05] Lag 2: 1.02 [0.93, 1.12] Lag 3: 0.99 [0.90, 1.09]

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Slaughter et al. (2005, 073854) **Period of Study:** January 1995 through June 2001 **Location:** Spokane, WA	Hospital Admissions and ED visits **Outcome:** All respiratory (460-519) Asthma (493) COPD (491,492, 494,496) Pneumonia (480-487) Acute URI not including colds and sinusitis (464, 466, 490) **Age Groups:** All, 15+ years for COPD **Study Design:** Time series **N:** 2373 visit records **Statistical Analyses:** Poisson regression, GLM with natural splines. For comparison also used GAM with smoothing splines and default convergence criteria. **Covariates:** Season, temperature, relative humidity, day of week **Season:** All **Dose-response Investigated?:** No **Statistical Package:** SAS, SPLUS **Lags Considered:** 1 -3 d	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h avg **Range (90% of Concentrations):** 4.2-20.2 $\mu g/m^3$ **Monitoring Stations:** One **Notes: Copollutant (correlation):** $PM_{2.5}$ PM_1 r = 0.95 PM_{10} r = 0.62 $PM_{10-2.5}$ r = 0.31 CO r = 0.62 Temperature r = 0.21	**PM Increment:** 10 $\mu g/m^3$ **RR Estimate [Lower CI, Upper CI]** **lag:** **ER visits:** $PM_{2.5}$ All Respiratory Lag 1: 1.01 [0.98, 1.04] Lag 2: 1.02 [0.99, 1.04] Lag 3: 1.02 [0.99, 1.05] Acute Asthma Lag 1: 1.03 [0.98, 1.09] Lag 2: 1.00 [0.95, 1.05] Lag 3: 1.01 [0.96, 1.06] COPD (adult) Lag 1: 0.96 [0.89, 1.04] Lag 2: 1.01 [0.93, 1.09] Lag 3: 1.00 [0.93, 1.08] **Hospital Admissions:** $PM_{2.5}$ All Respiratory Lag 1: 0.98 [0.94, 1.01] Lag 2: 0.99 [0.96, 1.03] Lag 3: 1.01 [0.98, 1.05] Asthma Lag 1: 1.01 [0.91, 1.11] Lag 2: 1.03 [0.94, 1.13] Lag 3: 1.02 [0.93, 1.13] COPD (adult) Lag 1: 0.99 [0.91, 1.08] Lag 2: 1.06 [0.98, 1.16] Lag 3: 1.03 [0.94, 1.12]

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Zanobetti and Schwartz (2006, 090195) **Period of Study:** 1995-1999 **Location:** Boston, MA	**Outcome:** Pneumonia (480-487) **Age Groups:** >65 y **Study Design:** Case-crossover, time stratified **N:** 24,857 for Pneumonia **Statistical Analyses:** Condition logistic regression **Covariates:** Season, long term trend, day of-the-wk, mean temperature, relative humidity, barometric pressure, extinction coefficient **Season:** All year **Dose-response Investigated?** No **Statistical Package:** SAS **Lags Considered:** 0-1 **Notes:** Also looked at MI cohort	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Percentiles** (pneumonia cohort)**:** 25th: 7.23 $\mu g/m^3$ 50th(Median): 11.10 75th: 16.14 **PM Component:** Black Carbon (BC), PM non-traffic **Monitoring Stations:** 4-5 monitors **Copollutant (correlation):** $PM_{2.5}$: CO r = 0.52 NO_2 r = 0.55 O_3 r = 0.20 BC r = 0.66 PM non-traffic r = 0.74	**PM Increment:** $PM_{2.5}$ lag 0: 17.17 $\mu g/m^3$ $PM_{2.5}$ lag 0-1 avg: 16.32 $\mu g/m^3$ % change in Pneumonia: 6.48[1.13, 11.43] lag 0 5.56[-0.45, 11.27] mean lag 1
Reference: Zanobetti and Schwartz (2006, 090195) **Period of Study:** 1995-1999 **Location:** Boston, MA	**Outcome:** Pneumonia (480-487) **Age Groups:** >65 y **Study Design:** Case-crossover, time stratified **N:** 24,857 for Pneumonia **Statistical Analyses:** Condition logistic regression **Covariates:** Season, long term trend, day of-the-wk, mean temperature, relative humidity, barometric pressure, extinction coefficient **Season:** All year **Dose-response Investigated?** No **Statistical Package:** SAS **Lags Considered:** 0-1 **Notes:** Also looked at MI cohort	**Pollutant:** BC **Averaging Time:** 24 h **Percentiles** (pneumonia cohort)**:** 5th: 0.42 25th: 0.74 $\mu g/m^3$ 50th(Median): 1.15 75th: 1.72 95th: 2.83 **PM Component:** PM non-traffic **Monitoring Stations:** 4-5 monitors **Copollutant (correlation):** BC: $PM_{2.5}$ r = 0.66 CO r = 0.82 NO_2 r = 0.70 O_3 r = -0.25 PM non-traffic r = -0.01	**PM Increment:** BC lag 0: 2.05 $\mu g/m^3$ BC lag 0-1 avg: 1.69 $\mu g/m^3$ % change in Pneumonia: BC-10.76[4.54, 15.89] lag 0 BC-11.71[4.79, 17.36] mean lag 1

[1]All units expressed in $\mu g/m^3$ unless otherwise specified.

E.3. Short-Term Exposure and Mortality

Table E-16. Short-term exposure – mortality - PM_{10}.

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Aga et al. (2003, 187122) **Period of Study:** ~5 yrs for most cities, during the 1990s **Location:** 28 European cities (APHEA2)	**Outcome:** Non-Accidental Mortality (<800) **Study Design:** Time-series **Statistical Analyses:** Poisson GAM, LOESS **Age Groups:** All ages >65	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** NR **Range (Min, Max):** (15, 66) **Copollutant:** BS **Note:** PM_{10} only measured in 21 cities.	**Increment:** 10 $\mu g/m^3$ **% Increase (Lower CI, Upper CI)** **lag:** All ages Fixed effects: 0.71% (0.60,0.83) 0-1 Random effects: 0.67% (0.47,0.87) 0-1 >65 Fixed effects: 0.79% (0.66,0.92) 0-1 Random effects: 0.74% (0.52,0.95) 0-1 Models with effect modifiers (>65) 24-h NO_2: 25th Percentile: 0.30% (0.07,0.53) 75th Percentile: 0.97% (0.82,1.11) 24-h temperature: 25th Percentile: 0.44% (0.25,0.64) 75th Percentile: 0.91% (0.77,1.05) 24-h relative humidity: 25th Percentile: 0.98% (0.82,1.14) 75th Percentile: 0.52% (0.33,0.71) Age standardized annual mortality rate: 25th Percentile: 0.93% (0.77,1.09) 75th Percentile: 0.61% (0.43,0.79) Proportion individuals >65 25th Percentile: 0.67% (0.50,0.83) 75th Percentile: 0.85% (0.71,0.99) Northwest/Central East: 25th Percentile: 0.81% (0.63,0.98) 75th Percentile: 0.26% (-0.05,0.57) Northwest/South: 25th Percentile: 0.81% (0.63,0.98) 75th Percentile: 1.04% (0.81,1.27)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Analitis et al. (2006, 088177) **Period of Study:** NR **Location:** 29 European cities (APHEA2)	**Outcome:** Mortality: Cardiovascular diseases (390-459) Respiratory diseases (460-519) **Study Design:** Time-series **Statistical Analyses:** 2-stage hierarchical modeling **Age Groups:** All ages	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Median (SD)** unit: Range: 9–64 $\mu g/m^3$ **Range (Min, Max):** NR **Copollutant:** BS **Note:** PM_{10} only measured in 21 cities.	**Increment:** 10 $\mu g/m^3$ **% Increase (Lower CI, Upper CI)** **lag:** Cardiovascular: Fixed effects: 0.64% (0.47, 0.80) 0-1 Random effects: 0.76% (0.47, 1.05) 0-1 0.90% (0.57, 1.23) 0-5 Respiratory: Fixed effects: 0.58% (0.21, 0.95) 0-1 Random effects: 0.71% (0.22, 1.20) 0-1 1.24% (0.49, 1.99) 0-5
Reference: Ballester et al. (2002, 030371) **Period of Study:** 1990–1996 **Location:** 13 Spanish cities	**Outcome:** Mortality: Non-accidental (< 800) Cardiovascular diseases (390-459) Respiratory diseases (460-519) **Study Design:** Ecological time series **Statistical Analyses:** Poisson GAM, LOESS **Age Groups:** All ages	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD): Huelva:** 42.5 (15) Madrid: 37.8 (17.7) Sevilla: 45.1 (14) **Range (Min, Max):** NR **Copollutant:** BS TSP SO_2 **Note:** PM_{10} only measured in 3 cities.	**Increment:** 10 $\mu g/m^3$ **Relative Risk (Lower CI, Upper CI)** **lag:** Non-accidental: Random effects: 1.006 (0.998, 1.015) 0-1 Fixed Effects: 1.005 (1.001, 1.010) 0-1 $PM_{10}+SO_2$: 1.013 (1.006, 1.020) 0-1 Cardiovascular: 1.012 (1.005, 1.018) 0-1 $PM_{10}+SO_2$: Random effects: 1.024 (1.001, 1.048) 0-1 Fixed effects: 1.021 (1.007, 1.035) 0-1 Respiratory: 1.013 (1.001, 1.026) 0-1 $PM_{10}+SO_2$: 1.003 (0.983, 1.023) 0-1
Reference: Bateson and Schwartz (2004, 086244) **Period of Study:** 1988–1991 **Location:** Cook County, Illinois	**Outcome:** Mortality: Heart Disease (390-429) Respiratory (460-519) **Study Design:** Bi-directional case-crossover **Statistical Analyses:** Conditional logistic regression **Age Groups:** ≥ 65	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SE) unit:** 37.6 (15.5) $\mu g/m^3$ **Range (Min, Max):** (3.7, 128) **Copollutant:** NR	**Increment:** 10 $\mu g/m^3$ **% Increase (Lower CI, Upper CI)** **lag:** All-cause: 1.14% (0.44, 1.85) 0-1 Modification of Effect by Prior Diagnosis Myocardial Infarction: 1.98% (-0.25,

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
	Study population: 65,180 elderly residents with history of hospitalization for heart or lung disease		4.26) 0-1 Diabetes: 1.49% (-0.06, 3.07) 0-1 Congestive heart failure: 1.28% (-0.06, 2.64) 0-1 COPD: 0.58% (-0.82, 2.00) 0-1 Conduction Disorders: 0.64% (-0.61, 1.90) 0-1 All other heart or lung diseases: 0.74% (-0.29, 1.79) 0-1 All-cause Men 65: 2.0% (0.3, 3.8) 0-1 75: 1.5% (-0.2, 3.1) 0-1 85: 0.9% (-0.7, 2.5) 0-1 95: 0.3% (-1.3, 1.9) 0-1 All: 1.3% (0.4, 2.3) 0-1 Women 65: 0.1% (-1.6, 1.9) 0-1 75: 0.7% (-1.1, 2.4) 0-1 85: 1.2% (-0.5, 3.0) 0-1 95: 1.8% (0.03, 3.6) 0-1 All: 1.0% (0.1, 1.9) 0-1 Total 65: 1.1% (-0.12, 2.3) 0-1 75: 1.1% (-0.1, 2.3) 0-1 85: 1.2% (-0.0, 2.4) 0-1 95: 1.2% (0.0, 2.4)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			0-1 All: 1.1% (0.4, 1.9) 0-1
Reference: Bell et al. (2009, 191007) **Period of Study:** 1987-2000 **Location:** 84 US Counties	**Outcome:** Mortality **Study Design:** Time-series **Covariates:** socio-economic conditions, long term temperature **Statistical Analysis:** Bayesian hierarchical model **Age Groups:** All	**Pollutant:** PM_{10} **Averaging Time:** 24h **Mean (SD) Unit:** NR **Range (Min, Max):** NR **Copollutant (correlation):** NR	**Increment:** 20% of the population acquiring air conditioning **Percent Change (95% CI) in community-specific PM health effect estimates for mortality** Any AC, including window units Yearly health effect: -30.4 (-80.4-19.6) Summer health effect: 29.9 (-84-144) Winter health effect: -573 (-9100-7955) Central AC Yearly health effect: -39 (-81.4-3.3) Summer health effect: 20. (-60.3-64.3) Winter health effect: -1777 (-5755-2201)
Reference: Bell et al. (2007, 093256) **Period of Study:** 1999-2005 **Location:** US	**Outcome:** Mortality **Age Groups:** 65+ **Study Design:** time series **N:** NR **Statistical Analyses:** Bayesian Hierarchical Regression **Covariates:** time trend, day of week, seasonality, dew point, temperature **Statistical Package:** NR **Lags Considered:** 0-2	**Pollutant:** PM_{10} **Averaging Time:** daily **Mean:** Ni: 0.002 **Min:** Ni: 0.003 **Max:** Ni: 0.021 **Interquartile Range:** Ni: 0.001 **Interquartile Range of Percents:** Ni: 0.01 **Monitoring Stations:** NR **Copollutant:** Al, NH_4+, As, Ca, Cl, Cu, EC, OMC, Fe, Pb, Mg. Ni, NO_3-, K, Si, Na+, $SO_4=$, Ti, V, Zn **Co-pollutant Correlation** Ni, V: 0.48 Ni, EC: 0.30 **Note:** Pollutant concentrations available for all fractions of $PM_{2.5}$	**PM Increment:** Interquartile Range in the fraction of $PM_{2.5}$ **Percent Increase in PM_{10} Health Effect (Lower CI, Upper CI)** Ni: 14.8 (-8.1, 37.7), lag 0 Ni: 14.7 (4.0, 25.3), lag 1 Ni: 14.7 (1.8, 27.5), lag 2 HS education: -31.9 (-82.4, 18.6) median income: -12.3 (-62.3, 37.7) Percent black: 48.7 (-15.8, 113) Percent living in urban area: -20.1 (-102, 61.7) Population: 5.1 (-14.4, 24.5) **Notes:** Interquartile ranges in percent HS education, median income, percent black, percent living in urban area, and population are 5.2 %, $9,223, 17.3%, 11.0%, and 549,283 respectively.

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Bellini et al. (2007, 097787)	**Outcome:** Mortality	**Pollutant:** PM_{10}	**Increment:** 10 $\mu g/m^3$
Period of Study: 1996–2002	All-cause (non-accidental) (< 800)	**Averaging Time:** 24-h avg	**% Increase (Lower CI, Upper CI)**
Location: 15 Italian cities	Cardiovascular (390-459)	**Mean (SD):** NR	**lag:**
	Respiratory (460-519)	**Range (Min, Max):** NR	All-cause:
	Study Design: Meta-analysis	**Copollutant:** SO_2	0.31% (-0.19, 0.74)
	Statistical Analyses: Poisson GLM	NO_2	0-1
	Age Groups: All ages	CO	Winter: 0.08%
		O_3	0-1
			summer: 1.95%
			0-1
			$PM_{10} + O_3$: 0.30%
			0-1
			$PM_{10} + NO_2$: 0.08%
			0-1
			Respiratory:
			0.54% (-0.91, 1.74)
			0-1
			Winter: 0.27%
			0-1
			summer: 3.61%
			0-1
			$PM_{10} + O_3$: 0.55%
			0-1
			$PM_{10} + NO_2$: 0.19%
			0-1
			Cardiovascular:
			0.54% (0.02, 1.02)
			0-1
			Winter: 0.20%
			0-1
			summer: 2.79%
			0-1
			$PM_{10} + O_3$: 0.57%
			0-1
			$PM_{10} + NO_2$: 0.39%
			0-1

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Burnett et al. (2004, 086247) **Period of Study:** 1981–1999 **Location:** 12 Canadian cities	**Outcome:** Mortality: Non-accidental (< 800) **Study Design:** Time-series **Statistical Analyses:** 1. Poisson, natural splines 2. Random effects regression model **Age Groups:** All ages	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** $PM_{2.5}$: 12.8 $PM_{10\text{-}2.5}$: 11.4 **Range (Min, Max):** NR **Copollutant (correlation):** NO_2 O_3 SO_2 CO **Note:** PM_{10} measurement calculated as the sum of $PM_{2.5}$ and $PM_{10\text{-}2.5}$ measurements.	**Increment:** 10 $\mu g/m^3$ **% Increase (Lower CI, Upper CI)** **lag:** 1981–1999 PM_{10}: 0.57% (0.05, 0.89) 1 $PM_{10}+NO_2$: 0.07% (-0.44, 0.58) 1

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Cakmak et al. (2007, 091170)	**Outcome:** Mortality:	**Pollutant:** PM_{10}	**Increment:** 10 $\mu g/m^3$
Period of Study: 1/1997–12/2003	Non-accidental (< 800)	**Averaging Time:** 24-h avg	**% Increase (Lower CI, Upper CI)**
Location: Chile–7 cities	Cardiovascular diseases (390-459)	**Mean (SD):** 84.9	**lag:**
	Respiratory diseases (460-519)	**Range (Min, Max):** NR	Non-accidental:
	Study Design: Time-series	**Copollutant (correlation):** O_3: r = -0.16 to 0.13	0.97% (-1.09, 2.76)
	Statistical Analyses: Poisson	SO_2: r = 0.37 to 0.77	0
	Random effects regression model	CO: r = 0.49 to 0.82	1.31% (-1.56, 3.68)
	Age Groups: All age	**Note:** Correlations are between pollutants for seven monitoring stations.	0-5
	◘ 64		$PM_{10}+O_3+SO_2+CO$: 0.80% (-0.87, 2.28)
	65–74		0
	75–84		◘ 64:
	≥ 85		0.52% (-0.55, 1.51)
			0
			0.49% (-0.51, 1.43)
			0-5
			65-75:
			1.07% (-1.23, 3.03)
			0
			1.31% (-1.57, 3.69)
			0-5
			75-84:
			1.41% (-1.71, 3.94)
			0
			1.93% (-2.57, 5.30)
			0-5
			≥ 85:
			1.56% (-1.94, 4.34)
			0
			2.14% (-2.97, 5.85)
			0-5
			April-September:
			1.03% (-1.17, 2.93)
			0
			1.37% (-1.64, 3.82)
			0-5
			October-March:
			0.07% (-0.07, 0.21)
			0
			0.15% (-0.15, 0.44)
			0-5
			Cardiovascular:
			1.14% (-1.31, 3.21)
			0
			1.49% (-1.82, 4.14)
			0-5
			Respiratory:
			2.03% (-2.75, 5.56)
			0
			3.11% (-5.25, 8.25)
			0-5

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Chen et al. (2008, 190106)	**Outcome** (ICD9: 2001	**Pollutant:** PM_{10}	**Increment:** 10 $\mu g/m^3$
Period of Study: 2001–2004	ICD10: 2002-2004):	**Averaging Time:** 24-h avg	**% Increase (Lower CI, Upper CI)**
Location: Shanghai, China	Mortality:	**Mean (SD):** 102.0	**lag:**
	Non-accidental causes (ICD9 < 800	**Range (Min, Max):** (14.0-566.8)	Non-accidental
	ICD10 A00-R99)	**Copollutant (correlation):**	Single Pollutant: 0.26% (0.14, 0.37)
	Cardiovascular (ICD9 390-459	SO_2	$PM_{10}+SO_2$: 0.08% (-0.07, 0.22)
	ICD10 I00-I99)	r = 0.64	$PM_{10}+NO_2$: 0.01% (-0.14, 0.17)
	Respiratory (ICD9 460-519	NO_2	$PM_{10}+SO_2+NO_2$: 0.00% (-0.16, 0.16)
	ICD10 J00-J98)	r = 0.71	Cardiovascular mortality
	Study Design: Time-series		Single Pollutant: 0.27% (0.10, 0.44)
	Statistical Analyses: Poisson GAM		$PM_{10}+SO_2$: 0.12% (-0.10, 0.34)
	Age Groups: All ages		$PM_{10}+NO_2$: 0.01% (-0.22, 0.25)
			$PM_{10}+SO_2+NO_2$: 0.01% (-0.23, 0.25)
			Respiratory mortality
			Single Pollutant: 0.27% (-0.01, 0.56)
			$PM_{10}+SO_2$: -0.04% (-0.41, 0.33)
			$PM_{10}+NO_2$: -0.05% (-0.45, 0.34)
			$PM_{10}+SO_2+NO_2$: -0.10% (-0.50, 0.30)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Daniels et al. (2004, 087343)	**Outcome:** Mortality:	**Pollutant:** PM_{10}	**Increment:** 10 $\mu g/m^3$
Period of Study: 1987–1994	Total (Non-accidental) mortality	**Averaging Time:** 24-h avg	**% Increase (Lower CI, Upper CI)**
Location: 20 Largest U.S. cities	Cardiovascular-Respiratory (390-448)	**Mean (SD):**	**lag:**
	(480-486, 487, 490-496, 507)	Los Angeles: 46.0	Total (non-accidental):
	Other-cause mortality	New York: 28.8	0.17% (0.03, 0.30)
	Study Design: Time-series	Chicago: 35.6	0
	Statistical Analyses: City-Specific Estimates: Poisson GLM, natural cubic splines	Dallas-Ft. Worth: 23.8	0.20% (0.07, 0.33)
		Houston: 30.0	1
	Combined Estimates: 2-stage Bayesian hierarchical model	San Diego: 33.6	0.28% (0.16, 0.41)
		Santa Ana-Anaheim: 37.4	0-1 avg
	Age Groups: All ages	Phoenix: 39.7	Cardiovascular-Respiratory:
		Detroit: 40.9	0.17% (-0.01, 0.35)
		Miami: 25.7	0
		Philadelphia: 35.4	0.27% (0.09, 0.44)
		Minneapolis: 26.9	1
		Seattle: 25.3	0.30% (0.18, 0.51)
		San Jose: 30.4	0-1 avg
		Cleveland: 45.1	Other-cause:
		San Bernardino: 37.0	0.17% (-0.03, 0.37)
		Pittsburgh: 31.6	0
		Oakland: 26.3	0.12% (-0.07, 0.31)
		Atlanta: 34.4	1
		San Antonio: 23.8	0.20% (0.01, 0.38)
			0-1 avg
			Threshold Models: Total Mortality
			Threshold = 15 $\mu g/m^3$
			0.30% (0.17, 0.42)
			0-1 avg
			Threshold = 0 $\mu g/m^3$
			0.28% (0.16, 0.41)
			0-1 avg
			Threshold = 20 $\mu g/m^3$
			0.30% (0.16, 0.43)
			0-1 avg
Reference: De Leon et al. (2003, 055688)	**Outcome:** Mortality:	**Pollutant:** PM_{10}	**Increment:** 18.16 $\mu g/m^3$
Period of Study: 1/1985–12/1994	Circulatory (390-459)	**Averaging Time:** 24-h avg	**Relative Risk (Lower CI, Upper CI)**
Location: New York, New York	Cancer (140-239)	**Mean (SD):**	**lag:**
	Study Design: Time-series	33.27 $\mu g/m^3$	All Ages
	Statistical Analyses: Poisson GAM	IQR (25th, 75th):	Cancer: 1.014 (1.000, 1.029)
	Age Groups: All ages	(22.67, 40.83)	0-1
	< 75	**Copollutant (correlation):** O_3	-w/out respiratory: 1.011 (0.996, 1.026)
	> 75	CO	0-1
		SO_2	-w/ respiratory: 1.051 (0.998, 1.107)
		NO_2	0-1

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			Circulatory: 1.025 (1.014, 1.035)
			0-1
			-w/out respiratory: 1.022 (1.012, 1.033)
			0-1
			-w/ respiratory: 1.054 (1.022, 1.086)
			0-1
			< 75
			Cancer: 1.003 (0.985, 1.021)
			0-1
			-w/out respiratory: 1.002 (0.983, 1.022)
			0-1
			-w/ respiratory: 1.009 (0.943, 1.078)
			0-1
			Circulatory: 1.027 (1.012, 1.043)
			0-1
			-w/out respiratory: 1.027 (1.011, 1.043)
			0-1
			-w/ respiratory: 1.033 (0.980, 1.089)
			0-1
			> 75
			Cancer: 1.033 (1.009, 1.058)
			0-1
			-w/out respiratory: 1.025 (1.000, 1.050)
			0-1
			-w/ respiratory: 1.129 (1.041, 1.225)
			0-1
			-w/out pneumonia: 1.026 (1.002, 1.050)
			0-1
			-w/ pneumonia: 1.183 (1.058, 1.323)
			0-1
			-w/out COPD: 1.032 (1.008, 1.057)
			0-1
			-w/ COPD: 1.008 (0.849, 1.197)
			0-1
			Circulatory: 1.025 (1.012, 1.038)
			0-1
			-w/out respiratory: 1.022 (1.008, 1.035)
			0-1
			-w/ respiratory: 1.066 (1.027, 1.106)
			0-1
			-w/out pneumonia: 1.023 (1.010, 1.036)
			0-1
			-w/ pneumonia: 1.078 (1.018, 1.141)
			0-1
			-w/out COPD: 1.025 (1.012, 1.038)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			0-1 -w/ COPD: 1.058 (0.991, 1.130) 0-1
Reference: Dominici et al. (2003, 042804) **Period of Study:** 1987–1994 **Location:** 88 U.S. cities	**Outcome:** Mortality: All-cause (non-accidental) (< 800) Cardiac (390-448) Respiratory (490-496) Influenza (487) Pneumonia (480-486, 507) Other causes **Study Design:** Time-series **Statistical Analyses:** 2-stage Bayesian hierarchical model **Age Groups:** < 65 65-74 ≥ 75	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** NR **Range (Min, Max):** NR **Copollutant (correlation):** NR	**Increment:** 10 $\mu g/m^3$ **% Increase (Lower CI, Upper CI)** **lag:** Cardio-respiratory 0.31% (0.15, 0.50) 1 All-cause 0.22% (0.10, 0.38) 1 Other causes 0.13% (-0.05, 0.29) 1
Reference: Dominici et al. (2004) **Period of Study:** 1987–1994 **Location:** 90 U.S. cities (NMMAPS)	**Outcome:** Mortality: Total (non-accidental) **Study Design:** Time-series **Statistical Analyses:** Poisson. GAM, GLM **Age Groups:** All ages	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** NR **Range (Min, Max):** NR	**Increment:** 10 $\mu g/m^3$ **% Increase (Lower CI, Upper CI)** **lag:** ¤ = 3 0.2% (0.05, 0.35)
Reference: Dominici et al. (2004, 096951) **Period of Study:** 1986-1993 **Location:** 10 U.S. cities	**Outcome:** Mortality: Total (non-accidental) **Study Design:** Time-series **Statistical Analyses:** 2-stage Bayesian hierarchical model **Age Groups:** All ages	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** Birmingham 34.8 Canton 28.4 Colorado Springs 27.5 Minneapolis/St. Paul 28.1 Seattle 32.2 Spokane 42.9 Chicago 36.3 Detroit 36.7 New Haven 28.6 Pittsburgh 36.0 New York: 28.8	**Increment:** 10 $\mu g/m^3$ **% Increase (Lower CI, Upper CI)** **lag:** Combined analysis: 0.26% (-0.37, 0.65) 0-1 Separate analysis: 0.28% (-0.12, 0.63) 0-1 Notes: A separate analysis assumes the mortality data does not provide any information on the log relative rates of mortality.
Reference: Dominici et al. (2007, 097361) **Period of Study:** PM_{10}: 1987–2000 $PM_{2.5}$: 1999–2000 **Location:** 100 U.S. counties (NMMAPS)	**Outcome:** Mortality: All-cause (non-accidental) Cardiorespiratory Other-cause **Study Design:** Time-series **Statistical Analyses:** 2-stage Bayesian hierarchical model **Age Groups:** All ages	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** NR **Range (Min, Max):** NR **Copollutant (correlation):** NR	**Increment:** 10 $\mu g/m^3$ **% Increase (Lower CI, Upper CI)** **lag:** PM_{10} All-cause: East: 1987-1994: 0.29% (0.12, 0.46) 1 1995-2000: 0.13% (-0.19, 0.44) 1

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			1987-2000: 0.25% (0.11, 0.39)
			1
			West:
			1987-1994: 0.12% (-0.07, 0.30)
			1
			1995-2000: 0.18% (-0.07, 0.44)
			1
			1987-2000: 0.12% (-0.02, 0.26)
			1
			National:
			1987-1994: 0.21% (0.10, 0.32)
			1
			1995-2000: 0.18% (0.00, 0.35)
			1
			1987-2000: 0.19% (0.10, 0.28)
			1
			Cardiorespiratory:
			East:
			1987-1994: 0.39% (0.16, 0.63)
			1
			1995-2000: 0.30% (-0.13, 0.73)
			1
			1987-2000: 0.34% (0.15, 0.54)
			1
			West:
			1987-1994: 0.17% (-0.07, 0.40)
			1
			1995-2000: 0.13% (-0.23, 0.50)
			1
			1987-2000: 0.14% (-0.05, 0.33)
			1
			National:
			1987-1994: 0.28% (0.14, 0.43)
			1
			1995-2000: 0.21% (-0.03, 0.44)
			1
			1987-2000: 0.24% (0.13, 0.36)
			1
			Other-cause:
			East:
			1987-1994: 0.21% (-0.03, 0.44)
			1
			1995-2000: 0.00% (-0.49, 0.50)
			1
			1987-2000: 0.15% (-0.09, 0.39)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			1 West: 1987-1994: 0.09% (-0.21, 0.38) 1 1995-2000: 0.23% (-0.15, 0.62) 1 1987-2000: 0.17% (-0.07, 0.41) 1 National: 1987-1994: 0.15% (-0.02, 0.32) 1 1995-2000: 0.17% (-0.07, 0.41) 1 1987-2000: 0.15% (0.00, 0.29) 1
Reference: Dominici et al. (2007, 099135) **Period of Study:** 2000–2005 **Location:** 72 U.S. counties representing 69 communities	**Outcome:** Total mortality **Study Design:** Time-series **Statistical Analyses:** 2-stage Bayesian hierarchical model **Age Groups:** All ages	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** NR **Range (Min, Max):** NR **Copollutant (correlation):** NR	The study does not provide results quantitatively. **Note:** The study investigated whether county-specific short-term effects of PM_{10} on mortality are modified by long-term county-specific nickel or vanadium $PM_{2.5}$ concentrations.
Reference: Fischer et al. (2003, 043739) **Period of Study:** 1986–1994 **Location:** The Netherlands	**Outcome:** Mortality: Non-accidental (< 800) Pneumonia (480-486) COPD (490-496) Cardiovascular (390-448) **Study Design:** Time-series **Statistical Analyses:** Poisson GAM, LOESS **Age Groups:** < 45 45-64 65-74 ≥ 75	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Median (SD) unit:** 34 **Range (Min, Max):** (10, 278) **Copollutant:** BS O_3 NO_2 SO_2 CO	**Increment:** 80 $\mu g/m^3$ **Relative Risk (Lower CI, Upper CI)** **lag:** Cardiovascular < 45: 0.906 (0.728, 1.128) 0-6 45-64: 1.023 (0.945, 1.106) 0-6 65-74: 1.002 (0.945, 1.062) 0-6 ≥ 75: 1.016 (0.981, 1.052) 0-6 COPD < 45: 1.153 (0.587, 2.268) 0-6 45-64: 1.139 (0.841, 1.541) 0-6 65-74: 1.166 (0.991, 1.372) 0-6 ≥ 75: 1.066 (0.965, 1.178) 0-6 Pneumonia < 45: 1.427 (0.806, 2.525) 0-6 45-64: 1.712 (1.042, 2.815)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			0-6 65-74: 1.240 (0.879, 1.748) 0-6 ≥ 75: 1.123 (1.011, 1.247) 0-6
Reference: Fischer et al. (2004, 055605) **Period of Study:** 6/2003–8/2003 **Location:** The Netherlands	**Outcome:** Total mortality **Study Design:** NR **Statistical Analyses:** NR **Age Groups:** All ages	**Pollutant:** PM_{10} **Averaging Time:** Weekly avg **Mean (SD):** 2000: 31 2002: 33 2003: 35 IQR (25th, 75th): NR **Copollutant:** O_3	The study does not present quantitative results. Notes: The study estimates the number of deaths attributable to PM_{10} during the summers of 2000, 2002, and 2003.
Reference: Forastiere et al. (2005, 086323) **Period of Study:** 1998-2000 **Location:** Rome, Italy	**Outcome:** Mortality: Ischemic heart disease (410-414) **Study Design:** Time-stratified case-crossover **Statistical Analyses:** Conditional logistic regression **Age Groups:** > 35	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** 52.1 (22.2) IQR (25th, 75th): (36.0, 65.7) **Copollutant (correlation):** PNC: r = 0.38 CO: r = 0.34 NO_2: r = 0.45 SO_2: r = 0.23 O_3: r = 0.13	**Increment:** 29.7 $\mu g/m^3$ **% Increase (Lower CI, Upper CI)** **lag:** 4.8% (0.1, 9.8) 0 4.9% (0.0, 10.1) 1 3.8% (-1.0, 8.9) 2 2.8% (-2.0, 7.7) 3 6.1% (0.6, 11.9) 0-1
Reference: Forastiere et al. (2007, 090720) **Period of Study:** 1998–2001 **Location:** Rome, Italy	**Outcome:** Mortality: Natural (< 800) Malignant neoplasms (140-208) Diabetes mellitus (250) Hypertensive disease (401-405) Previous acute myocardial infarction (410, 412) Other ischemic heart diseases (411, 413-414) Conduction disorders (426) Dysrhythmia (427) Heart failure (428) Cerebrovascular disease (430-438) Peripherical artery disease (440-448) COPD (490-496) **Study Design:** Time-stratified case-crossover **Statistical Analyses:** Conditional logistic regression **Age Groups:** > 35	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean Range (SD) unit:** 51.0 (21.0) $\mu g/m^3$ IQR (25th, 75th): (36.1, 63.0) **Copollutant (correlation):** NR	**Increment:** 10 $\mu g/m^3$ **% Increase (Lower CI, Upper CI)** **lag:** Non-accidental: 1.1% (0.7, 1.6) 0-1 Low income: 1.9% 0-1 Low SES: 1.4% 0-1 High income: 0.0% 0-1 High SES: 0.1% 0-1 Low PM Area: 0.9% (-0.4, 2.1) 0-1 High PM Area: 1.47% (0.4, 2.5) 0-1

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Forastiere et al. (2008, 186937)	**Outcome:** Mortality:	**Pollutant:** PM_{10}	**Increment:** 10 $\mu g/m^3$
Period of Study: 1997–2004	Non-accidental (< 800)	**Averaging Time:** 24-h avg	**% Increase (Lower CI, Upper CI)**
Location: 9 Italian cities	**Study Design:** Time-stratified case-crossover	**Mean Range (SD) unit:**	**lag:**
	Statistical Analyses: Conditional logistic regression	35.1 to 71.5	Total: 0.60% (0.31, 0.89)
	Age Groups: > 35	Range (5th, 95th):	0-1
		Lowest 5th: 14.3	Age
		Highest 95th: 147.0	35-64: -0.20% (-0.77, 0.37)
		Copollutant (correlation): NR	0-1
			65-74: 0.51% (0.05, 0.98)
			0-1
			75-84: 0.59%(0.20, 0.97)
			0-1
			≥ 85: 0.97% (0.53, 1.42)
			0-1
			≥ 65: 0.75% (0.42, 1.09)
			Sex
			Men: 0.72% (0.37, 1.07)
			0-1
			Women: 0.83% (0.33, 1.33)
			0-1
			Median income (by census block)
			Low (< 20th percentile): 0.80% (-0.02, 1.62)
			0-1
			Mid-low (20th-50th percentile): 0.68% (0.25, 1.12)
			0-1
			Mid-high (51st-80th percentile): 0.85% (0.40, 1.30)
			0-1
			High (> 80th percentile): 0.30% (-0.25, 0.86)
			0-1
			Location of death
			Out-of-hospital: 0.71% (0.32, 1.11)
			0-1
			Discharged 2-28 d before death: 1.34% (0.49, 2.20)
			0-1
			In-hospital: 0.65% (0.33, 0.97)
			0-1
			Nursing home: -0.04% (-1.02, 0.95)
			0-1

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Goldberg et al. (2003, 035202) **Period of Study:** 1984–1993 **Location:** Montreal, Quebec, Canada	**Outcome:** Mortality: Congestive Heart Failure (428) **Study Design:** Time-series **Statistical Analyses:** Poisson, natural splines **Age Groups:** ≥ 65	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** PM_{10}: 32.2 (17.6) IQR (25th, 75th): PM_{10}: (19.7, 41.1) **Copollutant (correlation):** $PM_{2.5}$, TSP, Sulfate, CoH, SO_2, NO_2, CO, O_3	This study does not present results quantitatively for PM_{10}
Reference: Goldberg et al. (2003, 035202) **Period of Study:** 1984–1993 **Location:** Montreal, Quebec, Canada	**Outcome:** Mortality: Diabetes (250) **Study Design:** Time-series **Statistical Analyses:** Poisson, natural spline **Age Groups:** ≥ 65	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** PM_{10}: 32.2 (17.6) $\mu g/m^3$ IQR (25th, 75th): PM_{10}: (19.7, 41.1) **Copollutant (correlation):** $PM_{2.5}$, Sulfate, CoH, SO_2, NO_2, CO, O_3	This study does not present results quantitatively for PM_{10}

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Kan and Chen (2003, 087372) **Period of Study:** 1/2000–12/2001 **Location:** Shanghai, China	**Outcome:** Mortality: Non-accidental (< 800) Cardiovascular (390-459) COPD (490-496) **Study Design:** Time-series **Statistical Analyses:** Poisson GAM, LOESS **Age Groups:** All ages < 65 65-75 > 75	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** 91.14 (51.85) **Range (Min, Max):** (17.0, 385.0) **Copollutant (correlation):** SO_2: r = 0.71 NO_2: r = 0.73	**Increment:** 10 $\mu g/m^3$ **Relative Risk (Lower CI, Upper CI)** **lag:** Non-accidental All ages: 1.003 (1.001, 1.005) 0 < 65: 1.001 (0.997, 1.005) 0 65-75: 1.005 (1.001, 1.008) 0 > 75: 1.003 (1.001, 1.006) 0 Cardiovascular All ages: 1.003 (1.000, 1.006) 0 < 65: 1.002 (0.994, 1.010) 0 65-75: 1.003 (0.998, 1.008) 0 > 75: 1.003 (1.000, 1.006) 0 COPD All ages: 1.005 (0.999, 1.011) 0 < 65: 1.004 (0.981, 1.027) 0 65-75: 0.996 (0.986, 1.007) 0 > 75: 1.006 (1.000, 1.012) 0 Multipollutant models SO_2: 1.001 (0.998, 1.003) 0 NO_2: 1.001 (0.998, 1.003) 0 SO_2+NO_2: 1.000 (0.997, 1.003) 0

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Kan and Chen (2003, 087372) **Period of Study:** 1/2000–12/2001 **Location:** Shanghai, China	**Outcome:** Mortality: Non-accidental (< 800) Cardiovascular (390-459) COPD (490-496) **Study Design:** Case-crossover **Statistical Analyses:** Conditional logistic regression **Age Groups:** All ages	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** 91.14 (51.85) IQR (25th, 75th): (54, 114) **Copollutant (correlation):** SO_2: r = 0.71 NO_2: r = 0.73	**Increment:** 10 $\mu g/m^3$ **Odds Ratio (Lower CI, Upper CI)** **lag:** Non-accidental: Bidirectional referent days: 7 d: 1.000 (0.9988, 1.002) 0-1 ma 7 and 14 d: 1.002 (1.000, 1.004) 0-1 ma 7, 14, and 21 d: 1.003 (1.001, 1.005) 0-1 ma Unidirectional referent days: 7 d: 1.015 (1.012, 1.018) 0-1 ma 7 and 14 d: 1.017 (1.015, 1.019) 0-1 ma 7, 14, and 21 d: 1.019 (1.012, 1.021) 0-1 ma Bidirectional referent days (7, 14, and 21 d): Cardiovascular: 1.004 (1.001, 1.007) 0-1 ma COPD: 1.006 (0.999, 1.013) 0-1 ma Non-accidental: $PM_{10}+SO_2$: 0.997 (0.994, 1.025) 0-1 ma $PM_{10}+NO_2$: 0.997 (0.994, 1.025) 0-1 ma $PM_{10}+SO_2+NO_2$: 0.995 (0.992, 1.025) 0-1 ma

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Kan et al. (2005, 087561)	**Outcome:** Mortality:	**Pollutant:** PM_{10}	**Increment:** 10 $\mu g/m^3$
Period of Study: 4/25/2003–5/31/2003	Severe acute respiratory syndrome (SARS)	**Averaging Time:** 24-h avg	Relative Risk (Lower CI, Upper CI)
Location: Beijing, China	**Study Design:** Time-series	**Mean (SD):** 149.1 (8.1)	lag:
	Statistical Analyses: Poisson, GAM, smoothing spline	**Range (Min, Max):** (34, 246)	0.99 (0.96 to 1.03)
	Age Groups: All ages	**Copollutant:**	0
		SO_2	1.00 (0.97 to 1.04)
		NO_2	1
			1.02 (0.98 to 1.06)
			2
			1.04 (0.99 to 1.09)
			3
			1.06 (1.00 to 1.11)
			4
			1.06 (1.00 to 1.12)
			5
			1.05 (0.98 to 1.12)
			6
Reference: Kan et al. (2007, 091267)	**Outcome (ICD10):** Mortality:	**Pollutant:** PM_{10}	**Increment:** 10 $\mu g/m^3$
Period of Study: 3/2004–12/2005	Total (non-accidental) (A00-R99)	**Averaging Time:** 24-h avg	**% Increase (Lower CI, Upper CI)**
Location: Shanghai, China	Cardiovascular (I00-I99)	**Mean (SD):** 107.9 (2.39) $\mu g/m^3$	**lag:**
	Respiratory (J00-J98)	**Range (Min, Max):** (22.0, 403.0)	PM_{10}
	Study Design: Time-series	**Copollutant (correlation):**	Total: 0.16% (0.02, 0.30)
	Statistical Analyses: Poisson GAM, penalized splines	PM_{10}	0-1
	Age Groups: All ages	$PM_{2.5}$: r = 0.84	Cardiovascular: 0.31% (0.10, 0.53)
		$PM_{10-2.5}$: r = 0.88	0-1
		O_3: r = 0.21	Respiratory: 0.33% (-0.08, 0.75)
			0-1
Reference: Kan et al. (2008, 156621)	**Outcome:** Mortality: Total (non-accidental) (A00-R99)	**Pollutant:** PM_{10}	**Increment:** 10 $\mu g/m^3$
Period of Study: 1/2001–12/2004	Cardiovascular (I00-I99)	**Averaging Time:** 24-h avg	**% Increase (Lower CI, Upper CI)**
Location: Shanghai, China	Respiratory (J00-J98)	**Mean (SD):**	**lag:**
	Study Design: Time-series	Warm season: 87.4 (1.8)	Non-accidental
	Statistical Analyses: Poisson GLM, natural splines	Cool season: 116.7 (2.8)	Warm season: 0.21 (0.09, 0.3)
	Age Groups: All ages;	Entire period: 102.0 (1.7)	0-1
	0-4	**Range (Min, Max):** NR	Cool season: 0.26 (0.22, 0.30)
	5-44	**Copollutant (correlation):** SO_2	0-1
	45-64	NO_2	Entire period: 0.25 (0.14, 0.37)
	≥ 65	O_3	0-1
			Female: 0.33 (0.18, 0.48)
			0-1
			Male: 0.17 (0.03, 0.32)
			0-1
			5-44: 0.04 (-0.52, 0.59)
			0-1
			45-64: 0.17 (-0.11, 0.45)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			0-1
			≥ 65: 0.26 (0.15, 0.38)
			0-1
			Cardiovascular
			Warm season: 0.22 (-0.14, 0.58)
			0-1
			Cool season: 0.25 (0.05, 0.45)
			0-1
			Entire period: 0.27 (0.10, 0.44)
			0-1
			Respiratory
			Warm season: -0.28 (-0.93, 0.38)
			0-1
			Cool season: 0.58 (0.25, 0.92)
			0-1
			Entire period: 0.27 (-0.01, 0.56)
			0-1
			Stratified by Educational Attainment
			Nonaccidental:
			Low: 0.33 (0.19, 0.47)
			0-1
			High: 0.18 (0.01, 0.36)
			0-1
			Cardiovascular:
			Low: 0.30 (0.10, 0.51)
			0-1
			High: 0.23 (-0.03, 0.50)
			0-1
			Respiratory:
			Low: 0.36 (0.00, 0.72)
			0-1
			High: 0.02 (-0.43, 0.47)
			0-1

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Keatinge and Donaldson (2006, 087536)	**Outcome:** Mortality: Total (non-accidental)	**Pollutant:** PM_{10}	**Increment:** 10 $\mu g/m^3$
Period of Study: 1991–2002	**Study Design:** Time-series	**Averaging Time:** 24-h avg	**Mortality per 106 (Lower CI, Upper CI)**
Location: London, England	**Statistical Analyses:** Poisson GAM	**Mean (SD):** NR	**lag:**
	Age Groups: ≥ 65	**Range (Min, Max):** NR	PM_{10} + Temp: 2.1 (0.9, 3.3)
		Copollutant: O_3	0-2 avg
		SO_2	PM_{10} + Temp + Acclim: 1.6 (0.4, 2.8)
			0-2 avg
			PM_{10} + Temp + Acclim + Acclim x T: 1.5 (0.3, 2.6)
			0-2 avg
			PM_{10} + Temp + Acclim + Acclim x T + Sun: 1.4 (0.2, 2.5)
			0-2 avg
			PM_{10} + Temp + Acclim + Acclim x T + Sun + Wind: 0.8 (-0.4, 1.9)
			0-2 avg
			PM_{10} + Temp + Acclim + Acclim x T + Sun + Wind + Abs. Humid.: 0.8 (-0.3, 1.9)
			0-2 avg
			PM_{10} + Temp + Acclim + Acclim x T + Sun + Wind + Abs. Humid. + Rain: 0.9 (-0.3, 2.0)
			0-2 avg
			PM_{10} + Temp + Abs. Humid.: 1.9 (0.7, 3.1)
			0-2 avg

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Kettunen et al. (2007, 091242) **Period of Study:** 1998–2004 **Location:** Helsinki, Finland	**Outcome (ICD10):** Mortality: Stroke (I60-I61, I63-I64) **Study Design:** Time-series **Statistical Analyses:** Poisson GAM, penalized thin-plate splines **Age Groups:** ≥ 65	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg Median (SD) unit: Cold Season: 16.3 Warm Season: 16.5 **Range (Min, Max):** Cold Season: (3.1, 136.7) Warm Season: (3.3, 67.4) **Copollutant:** $PM_{2.5}$ $PM_{10-2.5}$ UFP O_3 CO NO_2	**Increment:** Cold Season: 13.8 $\mu g/m^3$ Warm Season: 9.8 $\mu g/m^3$ **% Increase (Lower CI, Upper CI)** **lag:** Cold Season -0.56% (-3.32, 2.29) 0 -0.93% (-3.55, 1.75) 1 -1.68% (-4.30, 1.00) 2 -1.53% (-4.14, 1.14) 3 Warm Season 10.89% (0.95, 21.81) 0 8.56% (-0.88, 18.90) 1 2.06% (-6.76, 11.71) 2 -2.89% (-11.32, 6.34) 3
Reference: Kim et al. (2003, 155899) **Period of Study:** 1/1995–12/1999 **Location:** Seoul, Korea	**Outcome (ICD10):** Mortality: Non-accidental (all except S01-S99, T01-T98) Cardiovascular (I00-I52) Respiratory (J00-J98) Cerebrovascular (I60-I69) **Study Design:** Time-series **Statistical Analyses:** Poisson GAM **Age Groups:** All ages	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** 69.19 (10.36) IQR (25th, 75th): (44.82, 87.95) **Copollutant (correlation):** NR	**Increment:** 10 $\mu g/m^3$ **% Increase (Lower CI, Upper CI)** **lag:** All cause: 2.8% (1.8, 3.7) 0 2.8% (1.9, 3.7) 1 1.4% (0.5, 2.3) 2 3.7% (2.1, 5.4) distributed lag (6-day) Respiratory: 8.3% (4.3, 12.5) 0 6.4% (2.7, 10.2) 1 6.5% (2.7, 10.4) 2 13.9% (6.8, 21.5) distributed lag (6-day) Pneumonia:

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			11.6% (4.2, 19.6)
			0
			9.0% (2.1, 16.3)
			1
			7.7% (0.8, 15.2)
			2
			17.1% (4.1, 31.7)
			distributed lag (6-day)
			COPD:
			4.2% (-1.2, 10.0)
			0
			3.5% (-1.5, 8.9)
			1
			1.4% (-3.7, 6.8)
			2
			12.2% (2.5, 22.9)
			distributed lag (6-day)
			Cardiovascular:
			2.0% (-0.9, 5.0)
			0
			3.3% (0.6, 6.2)
			1
			2.9% (0.1, 5.8)
			2
			4.4% (-0.6, 9.6)
			distributed lag (6-day)
			Myocardial infarction: 2.6% (-2.3, 7.8)
			0
			5.8% (1.0, 10.7)
			1
			5.5% (0.7, 10.6)
			2
			4.9% (-3.4, 13.9)
			distributed lag (6-day)
			Cerebrovascular:
			3.2% (0.8, 5.5)
			0
			3.1% (0.9, 5.3)
			1
			2.4% (0.1, 4.6)
			2
			6.3% (2.3, 10.5)
			distributed lag (6-day)
			Ischemic stroke:
			-0.6% (-5.6, 4.7)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			0
			0.6% (-4.2, 5.7)
			1
			-0.1% (-4.9, 5.1)
			2
			10.3% (1.0, 20.4)
			distributed lag (6-day)
Reference: Kim et al. (2004, 087417)	**Outcome:** Mortality: Non-accidental	**Pollutant:** PM_{10}	**Increment:** 42.11 $\mu g/m^3$
Period of Study: 1/1997–12/2001	**Study Design:** Time-series	**Averaging Time:** 24-h avg	**Relative Risk (Lower CI, Upper CI)**
Location: Seoul, Korea	**Statistical Analyses:** Poisson GAM, LOESS	**Mean (SD):** 68.23 (36.36) $\mu g/m^3$	**lag:**
	Age Groups: All ages	IQR (25th, 75th): (42.56, 84.67)	1.021 (1.009, 1.035)
		Copollutant (correlation): NR	
Reference: Le Tertre et al. (2005, 087560)	**Outcome:** Mortality:	**Pollutant:** PM_{10}	**Increment:** 1.0 $\mu g/m^3$
	Non-accidental ($<$800)	**Averaging Time:** 24-h avg	**β coefficient (SE)**
Period of Study: NR	**Study Design:** Time-series	**Mean (SD):** NR	**lag:**
Location: 21 European cities (APHEA-2)	**Statistical Analyses:** Empirical Bayes	**Range (Min, Max):** NR	Athens: 0.001311 (0.0003)
	Age Groups: All ages	**Copollutant:** NO_2	Barcelona: 0.000575 (0.0002)
			Basel: 0.000462 (0.0005)
			Birmingham: 0.000305 (0.0003)
			Budapest: -0.000248 (0.0005)
			Cracow: 0.000155 (0.0004)
			Erfurt: -0.000465 (0.0004)
			Geneva: -0.000059 (0.0005)
			Helsinki: 0.000389 (0.0004)
			London: 0.000591 (0.0002)
			Lyon: 0.001554 (0.0005)
			Madrid: 0.000372 (0.0003)
			Milan: 0.000901 (0.0002)
			Paris: 0.000411 (0.0003)
			Prague: 0.000097 (0.0002)
			Rome: (0.001333 (0.0003)
			Stockholm: 0.000479 (0.0009)
			Tel Aviv: 0.000522 (0.0003)
			Teplice: 0.000876 (0.0004)
			Torino: 0.000938 (0.0002)
			Zurich: 0.000365 (0.0004)
			Toulouse: NR (NR)
			Overall: 0.00055 (0.000098)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Lee et al. (2007, 093042)	**Outcome (ICD10):** Mortality:	**Pollutant:** PM_{10}	**Increment:** 41.49 $\mu g/m^3$
Period of Study: 1/2000–12/2004	Non-accidental (A00-R99)	**Averaging Time:** 24-h avg	**% Increase (Lower CI, Upper CI)**
Location: Seoul, Korea	**Study Design:** Time-series	**Mean (SD):**	**lag:**
	Statistical Analyses: Poisson GAM	w/ Asian dust days: 70.00 (47.80)	Model with Asian Dust Days
	Age Groups: All ages	w/o Asian dust days: 65.77 (33.60)	0.7% (0.2, 1.3)
		Asian dust days only: 188.49 (142.85)	1-3
		Copollutant:	Model without Asian dust days
		CO	1.0% (0.2, 1.8)
		NO_2	1-3
		SO_2	
		O_3	
Reference: Lee and Shaddick (2007, 156685)	**Outcome (ICD10):** Mortality:	**Pollutant:** PM_{10}	**Increment:** 10 $\mu g/m^3$
Period of Study: 1/1/1993 – 12/31/1997	Non-accidental	**Averaging Time:** 24-h avg	**Relative Risk (Lower CI, Upper CI)**
Location: Cleveland, Ohio	**Study Design:** Time-series	**Mean (SD):** NR	**lag:**
Detroit, Michigan	**Statistical Analyses:** 1. Bayesian, penalized spline	**Range (Min, Max):** NR	Constant model
Minneapolis, Minnesota	2. Likelihood, penalized spline		Cleveland: 1.0049
Pittsburgh, Pennsylvania	**Age Groups:** All ages		1
			Detroit: 1.0046
			1
			Minneapolis: 1.0052
			1
			Pittsburgh: 1.0045
			1
Reference: Martins et al. (2004, 087457)**Period of Study:** 1/1997–12/1999	**Outcome (ICD10):** Mortality:	**Pollutant:** PM_{10}	The study does not present quantitative results.
Location: São Paulo, Brazil	Respiratory (J00-J99)	**Averaging Time:** 24-h avg	
	Study Design: Time-series	**Mean (SD):**	
	Statistical Analyses: Poisson GLM, natural cubic splines	Cerqueira Cesar: 42.5(22.9)	
	Age Groups: ≥ 60	Santa Amaro: 49.6(32.1)	
		Central: 52.1(23.5)	
		Penha: 40.4(23.8)	
		Santana: 72.6(24.5)	
		Sao Miguel Paulista: 68.6(31.0)	
		Range (Min, Max): NR	

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Nawrot et al. (2007, 098619) **Period of Study:** 1/1997–12/2003 **Location:** Flanders, Belgium	**Outcome:** Mortality: Non-accidental (< 800) Cardiovascular (390-459) Respiratory (460-519) **Study Design:** Time-series **Statistical Analyses:** Main analysis: Segmented regression models Sensitivity analysis: Poisson GAM, LOESS **Age Groups:** All ages	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Median (SD) unit:** Winter: 43.3(0.88) Spring: 39.5(0.88) summer: 37.7(0.91) Fall: 37.2(0.88) **Range (Min, Max):** NR **Copollutant (correlation):** NR	**Increment:** Main analysis: NR Sensitivity analysis: 10 $\mu g/m^3$ **% Increase (Lower CI, Upper CI)** **lag:** Highest season-specific PM_{10} quartile versus the lowest season-specific PM_{10} quartile summer: 7.8% (6.1, 9.6) Spring: 6.3% (4.7, 7.8) Autumn: 2.2% (0.58, 3.8) Winter: 1.4% (0.06, 2.9) Warm months (June, July, August): 7.9% (6.2, 9.6) Cold months (December, January, February): 1.5% (0.22, 3.3) Intermediate months (March, April, May, September, October, November): 4.2% (2.9, 5.6) Warmer Periods (April–September) Non-accidental: 1.5% (1.1, 2.0) 0 Respiratory: 2.0% (0.6, 3.7) 0 Cardiovascular: 1.8% (1.1, 2.4) 0
Reference: O'Neill et al. (2004, 087429) **Period of Study:** 1996–1998 1994–7/1995 **Location:** Mexico City, Mexico	**Outcome:** Mortality: Non-accidental **Study Design:** Time-series **Statistical Analyses:** Poisson, natural cubic spline **Age Groups:** All ages	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Range:** Hi-Vol: 46.3–164.0 TEOM: 48.2–107.5 Predicted: 30.2–162.4 Impactor: 58.4 **Range (Min, Max):** Xalostoc Hi-Vol: (40.0, 335.0) TEOM: (16.5, 291.2) Predicted: (60.6, 320.0) Tlalnepantla Hi-Vol: (25.0, 264.0) TEOM: (10.4, 275.9) Predicted: (17.7, 175.0) Merced Hi-Vol: (17.0, 266.0) TEOM: (9.4, 318.7) Predicted: (12.3, 160.8) Cerro de la Estrella	**Increment:** 10 $\mu g/m^3$ **% Increase (Lower CI, Upper CI)** **lag:** TEOM 0.04% (-0.12, 0.20) 0 -0.02% (-0.18, 0.13) 1 -0.01% (-0.27, 0.25) 2 -0.03% (-0.19, 0.13) 3 -0.03% (-0.19, 0.13) 4 -0.05% (-0.21, 0.11) 5 0.05% (-0.25, 0.35) 0-5 Predicted -0.05% (-0.29, 0.19) 0

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
		Hi-Vol: (15.0, 292.0)	0.09% (-0.16, 0.34)
		TEOM: (13.7, 268.3)	1
		Predicted: (11.2, 154.4)	-0.12% (-0.43, 0.20)
		Pedregal (1996-1998)	2
		Hi-Vol: (5.0, 226.0)	-0.02% (-0.26, 0.21)
		TEOM: (7.8, 264.4)	3
		Predicted: (-0.5, 86.3)	-0.14% (-0.37, 0.09)
		Pedregal (1994-1995)	4
		Hi-Vol: (24.0, 114.0)	-0.05% (-0.28, 0.18)
		TEOM: (8.7, 152.5)	5
		Impactor: (15.0, 154.0)	0.00% (-0.39, 0.38)
		Predicted: (3.9, 75.9)	0-5
			Sierra-Anderson High Volume Air Sampler
			0.02% (-0.29, 0.32)
			0
			0.13% (-0.27, 0.54)
			1
			0.21% (-0.10, 0.52)
			2
			0.53% (0.07, 0.99)
			3
			0.11% (-0.20, 0.41)
			4
			0.38% (0.07, 0.70)
			5
			GAM: 2 LOESS terms, default convergence
			1.68% (0.45, 2.93)
			0
			-0.36% (-1.56, 0.86)
			1
			-0.21% (-1.40, 1.00)
			2
			-0.18% (-1.40, 1.05)
			3
			1.31% (0.08, 2.55)
			4
			1.49% (0.25, 2.73)
			5
			1.77% (-0.26, 3.83)
			0-5
			Parametric: cubic splines
			5 df
			1.45% (0.09, 2.83)
			0

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			-0.71% (-2.06, 0.67)
			1
			-0.59% (-1.95, 0.79)
			2
			-0.70% (-2.09, 0.71)
			3
			0.92% (-0.46, 2.32)
			4
			1.17% (-0.19, 2.55)
			5
			1.17% (-1.54, 3.95)
			0-5
			10 df
			1.60% (0.20, 3.02)
			0
			-0.80% (-2.18, 0.60)
			1
			-0.73% (-2.11, 0.68)
			2
			-1.05% (-2.49, 0.40)
			3
			0.64% (-0.79, 2.10)
			4
			1.05% (-0.36, 2.48)
			5
			0.51% (-2.60, 3.71)
			0-5
			2 df
			1.79% (0.48, 3.11)
			0
			-0.09% (-1.38, 1.22)
			1
			0.10% (-1.18, 1.40)
			2
			0.20% (-1.10, 1.52)
			3
			1.60% (0.30, 2.91)
			4
			1.72% (0.43, 3.04)
			5
			1.90% (-0.36, 4.21)
			0-5
Reference: O'Neill et al. (2005, 098094) **Period of Study:** 1996–1998 1996-1999	**Outcome:** Mortality: Non-accidental Cardiovascular (390-460) Respiratory (460-520)	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** Mexico City: 75.8 (31.4)	The study focuses on the temperature–mortality relationship and only includes PM_{10} as a covariate in models.

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Location: Mexico City and Monterrey, Mexico	Other-causes **Study Design:** Time-series **Statistical Analyses:** Poisson, natural cubic splines **Age Groups:** All ages, 0-15, ≥ 65	Monterrey: 50.0 (23.5) **Range (Min, Max):** Mexico City: (18.0, 233.9) Monterrey: (6.2, 230.8) **Copollutant:** O_3	
Reference: O'Neill et al. (2008, 192314) **Period of Study:** 1/1/1998 - 12/30/2002 **Location:** Mexico City, Mexico Santiago, Chile São Paulo, Brazil	**Outcome:** **Study Design:** Time-series **Covariates:** Temperature, Day of Week, Temporal trends, Sex **Statistical Analysis:** Poisson regression **Statistical Package:** S-Plus **Age Groups:** Adults over 21	**Pollutant:** PM_{10} **Averaging Time:** 24h **Mean (SD) $\mu g/m^3$:** Mexico City: 53.8 (24.9) São Paulo: 48.9 (21.9) Santiago: 78.7 (33.0) **Range (Min, Max):** Mexico City: 1.08-192.2 São Paulo: 12.0-171.3 Santiago: 8.0-218.6 **Copollutant:** NR	**Increment:** 10 $\mu g/m^3$ **Percent increase (95% CI) in all-cause adult mortality (>22yrs) by educational level and sex** Mexico City All Adults, Concurrent Day None: 0.76 (0.17-1.36) Primary: 0.27 (-0.19-0.72) Secondary: 0.19 (-0.19-0.57) ≥ 12 yrs: 0.83 (0.03-1.63) All Adults, Lag 1 None: 0.62 (0.02-1.22) Primary: 0.62 (0.17-1.08) Secondary: 0.29 (-0.09-0.90) ≥ 12 yrs: 0.58 (-0.21-1.38) All Adults, Distributed Lags 0-5 None: 0.91 (-0.07-1.89) Primary: 0.48 (-0.27-1.24) Secondary: 0.27 (-0.36-0.90) ≥ 12 yrs: 0.75 (-0.49-2.02) All Adults, *df* (yrs) None: 5.4 Primary: 6.0 Secondary: 6.0 ≥ 12 yrs: 3.0 Women, Concurrent Day None: 0.65 (-0.08-1.38) Primary: 0.48 (-0.13-1.09) Secondary: 0.35 (-0.16-0.86) ≥ 12 yrs: 1.64 (0.69-2.59) Women, Lag 1 None: 0.62 (-0.12-1.36) Primary: 1.03 (0.42-1.64) Secondary: 0.59 (0.08-1.11) ≥ 12 yrs: 1.79 (0.84-2.75) Women, Distributed Lags 0-5 None: 0.46 (-0.74-1.68) Primary: 1.39 (0.42-2.36) Secondary: 0.51 (-0.30-1.33) ≥ 12 yrs: 1.71 (0.61-2.83) Women, *df* (yrs)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			None: 5.4
			Primary: 4.4
			Secondary: 4.8
			≥ 12 yrs: 1.0
			Men, Concurrent Day
			None: 0.75 (-0.21-1.72)
			Primary: 0.52 (-0.11-1.15)
			Secondary: 0.56 (0.08-1.05)
			≥ 12 yrs: 1.20 (0.25-2.17)
			Men, Lag 1
			None: 0.45 (-0.51-1.42)
			Primary: 0.70 (0.06-1.34)
			Secondary: 0.47 (-0.02-0.95)
			≥ 12 yrs: 0.74 (-0.22-1.70)
			Men, Distributed Lags 0-5
			None: 1.24 (-0.25-2.75)
			Primary: 0.65 (-0.39-1.69)
			Secondary: 0.88 (0.11-1.66)
			≥ 12 yrs: 1.07 (-0.41-2.57)
			Men, *df* (yrs)
			None: 3.8
			Primary: 5.6
			Secondary: 4.6
			≥ 12 yrs: 3.8
			São Paulo
			All Adults, Concurrent Day
			None: 0.77 (-0.28-1.82)
			Primary: 1.27 (0.78-1.76)
			Secondary: 0.93 (-0.07-1.94)
			≥ 12 yrs: 2.93 (2.00-2.88)
			All Adults, Lag 1
			None: 0.70 (-0.34-1.76)
			Primary: 1.32 (0.83-1.82)
			Secondary: 1.91 (0.58-2.60)
			≥ 12 yrs: 2.20 (1.27-3.15)
			All Adults, Distributed Lags 0-5
			None: 0.76 (-0.91-2.46)
			Primary: 1.34 (0.55-2.14)
			Secondary: 1.91 (0.35-2.60)
			≥ 12 yrs: 2.20 (1.27-3.15)
			All Adults, *df* (yrs)
			None: 4.0
			Primary: 4.0
			Secondary: 2.8
			≥ 12 yrs: 1.6

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			Women, Concurrent Day
			None: 1.93 (0.87-3.00)
			Primary: 1.72 (1.04-2.41)
			Secondary: 0.85 (-0.21-1.92)
			≥ 12 yrs: 1.84 (0.56-3.13)
			Women, Lag 1
			None: 1.41 (0.34-2.48)
			Primary: 1.64 (0.96-2.33)
			Secondary: 1.43 (0.36-2.50)
			≥ 12 yrs: 2.27 (0.99-3.56)
			Women, Distributed Lags 0-5
			None: 2.00 (0.40-3.63)
			Primary: 2.05 (0.96-3.14)
			Secondary: 1.61 (0.07-3.17)
			≥ 12 yrs: 3.35 (1.49-5.25)
			Women, *df* (yrs)
			None: 2.4
			Primary: 3.6
			Secondary: 1.4
			≥ 12 yrs: 0.8
			Men, Concurrent Day
			None: -0.43 (-2.15-1.32)
			Primary: 1.36 (0.71-2.02)
			Secondary: 1.74 (0.77-2.72)
			≥ 12 yrs: 2.81 (1.71-3.92)
			Men, Lag 1
			None: -0.44 (-2.17-1.33)
			Primary: 1.44 (0.79-2.10)
			Secondary: 1.52 (0.55-2.49)
			≥ 12 yrs: 1.48 (0.38-2.59)
			Men, Distributed Lags 0-5
			None: -0.30 (-3.09-2.56)
			Primary: 1.67 (0.65-2.70)
			Secondary: 1.06 (-0.34-2.49)
			≥ 12 yrs: 3.18 (1.60-4.79)
			Men, *df* (yrs)
			None: 4.4
			Primary: 3.2
			Secondary: 0.8
			≥ 12 yrs: 1.2
			Santiago
			All Adults, Concurrent Day
			None: 1.44 (0.53-2.36)
			Primary: 0.06 (-0.21-0.34)
			Secondary: 0.42 (0.06-0.78)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			≥ 12 yrs: 1.32 (0.60-2.05)
			All Adults, Lag 1
			None: 2.08 (1.16-30.1)
			Primary: 0.53 (0.25-0.81)
			Secondary: 0.55 (0.19-0.91)
			≥ 12 yrs: 1.31 (0.59-2.04)
			All Adults, Distributed Lags 0-5
			None: 3.18 (1.60-4.78)
			Primary: 0.58 (0.10-1.06)
			Secondary: 1.10 (0.48-1.73)
			≥ 12 yrs: 2.00 (0.93-3.07)
			All Adults, *df* (yrs)
			None: 3.6
			Primary: 5.6
			Secondary: 4.0
			≥ 12 yrs: 1.6
			Women, Concurrent Day
			None: 0.91 (-0.06-1.89)
			Primary: 0.31 (-0.06-0.68)
			Secondary: 0.84 (0.33-1.36)
			≥ 12 yrs: 0.60 (-0.32-1.52)
			Women, Lag 1
			None: 1.58 (0.58-2.58)
			Primary: 0.79 (0.42-1.17)
			Secondary: 0.76 (0.25-1.28)
			≥ 12 yrs: 0.53 (-0.39-1.45)
			Women, Distributed Lags 0-5
			None: 1.15 (-0.48-2.80)
			Primary: 1.05 (0.41-1.69)
			Secondary: 1.29 (0.40-2.19)
			≥ 12 yrs: 1.06 (-0.27-2.41)
			Women, *df* (yrs)
			None: 2.6
			Primary: 4.8
			Secondary: 4.4
			≥ 12 yrs: 1.0
			Men, Concurrent Day
			None: 0.05 (-1.02-1.12)
			Primary: -0.11 (-0.5-0.28)
			Secondary: 0.18 (-0.31-0.68)
			≥ 12 yrs: 1.52 (0.70-2.35)
			Men, Lag 1
			None: 0.61 (-0.44-1.68)
			Primary: 0.23 (-0.16-0.62)
			Secondary: 0.49 (0.00-0.98)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			≥ 12 yrs: 1.03 (0.21-1.86)
			Men, Distributed Lags 0-5
			None: 2.08 (0.28-3.91)
			Primary: 0.16 (-0.50-0.82)
			Secondary: 1.27 (0.43-2.12)
			≥ 12 yrs: 1.98 (0.76-3.20)
			Men, *df* (yrs)
			None: 2.8
			Primary: 4.8
			Secondary: 4.4
			≥ 12 yrs: 1.6
			Percent increase (95% CI) in all-cause adult mortality (≥65yrs) by educational level and sex
			Mexico City
			All Adults, Concurrent Day
			None: 0.41 (-0.25-1.08)
			Primary: 0.40 (-0.15-0.95)
			Secondary: 0.50 (-0.01-1.01)
			≥ 12 yrs: 1.51 (0.39-2.63)
			All Adults, Lag 1
			None: 0.20 (-0.47-0.87)
			Primary: 0.80 (0.24-1.36)
			Secondary: 0.60 (0.09-1.12)
			≥ 12 yrs: 1.09 (-0.02-2.22)
			All Adults, Distributed Lags 0-5
			None: 0.27 (-0.83-1.38)
			Primary: 0.99 (0.07-1.91)
			Secondary: 0.30 (-0.56-1.16)
			≥ 12 yrs: 1.83 (0.09-3.59)
			All Adults, *df* (yrs)
			None: 5.6
			Primary: 5.4
			Secondary: 6.0
			≥ 12 yrs: 3.2
			Women, Concurrent Day
			None: 0.49 (-0.30-1.29)
			Primary: 0.39 (-0.33-1.11)
			Secondary: 0.52 (-0.16-1.20)
			≥ 12 yrs: 1.29 (0.12-2.48)
			Women, Lag 1
			None: 0.73 (-0.07-1.54)
			Primary: 1.24 (0.52-1.97)
			Secondary: 0.55 (-0.13-1.23)
			≥ 12 yrs: 1.50 (0.32-2.70)
			Women, Distributed Lags 0-5

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			None: 0.75 (-0.56-2.08)
			Primary: 1.43 (0.29-2.59)
			Secondary: 0.06 (-1.01-1.15)
			≥ 12 yrs: 1.48 (0.10-2.87)
			Women, *df* (yrs)
			None: 5.4
			Primary: 4.2
			Secondary: 4.8
			≥ 12 yrs: 0.6
			Men, Concurrent Day
			None: 0.90 (-0.23-2.04)
			Primary: 0.37 (-0.40-1.16)
			Secondary: 0.78 (0.07-1.49)
			≥ 12 yrs: 1.66 (0.30-3.04)
			Men, Lag 1
			None: -0.15 (-1.27-0.98)
			Primary: 0.26 (-0.53-1.05)
			Secondary: 0.93 (0.22-1.65)
			≥ 12 yrs: 0.95 (-0.41-2.32)
			Men, Distributed Lags 0-5
			None: 0.80 (-0.95-2.58)
			Primary: 0.29 (-0.99-1.58)
			Secondary: 1.06 (-0.08-2.21)
			≥ 12 yrs: 1.76 (-0.35-3.91)
			Men, *df* (yrs)
			None: 3.8
			Primary: 5.6
			Secondary: 4.6
			≥ 12 yrs: 3.8
			São Paulo
			All Adults, Concurrent Day
			None: 0.60 (-0.48-1.70)
			Primary: 0.59 (1.00-2.19)
			Secondary: 1.21 (-0.01-2.44)
			≥ 12 yrs: 2.80 (1.67-3.94)
			All Adults, Lag 1
			None: 0.62 (-0.47-1.72)
			Primary: 1.48 (0.89-2.07)
			Secondary: 2.31 (1.08-3.55)
			≥ 12 yrs: 2.52 (1.40-3.66)
			All Adults, Distributed Lags 0-5
			None: 0.91 (-0.84-2.69)
			Primary: 1.73 (0.79-2.67)
			Secondary: 3.25 (1.39-5.16)
			≥ 12 yrs: 3.63 (2.01-5.29)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			All Adults, *df* (yrs)
			None: 4.0
			Primary: 3.8
			Secondary: 2.6
			≥ 12 yrs: 1.6
			Women, Concurrent Day
			None: 1.82 (0.71-2.94)
			Primary: 1.84 (1.05-2.64)
			Secondary: 0.62 (-0.55-1.81)
			≥ 12 yrs: 1.00 (-0.27-2.29)
			Women, Lag 1
			None: 1.36 (0.25-2.49)
			Primary: 1.76 (0.97-2.56)
			Secondary: 1.57 (0.39-2.76)
			≥ 12 yrs: 1.39 (0.12-2.68)
			Women, Distributed Lags 0-5
			None: 1.80 (0.12-3.51)
			Primary: 1.97 (0.73-3.22)
			Secondary: 1.89 (0.19-3.61)
			≥ 12 yrs: 2.53 (0.70-4.40)
			Women, *df* (yrs)
			None: 2.4
			Primary: 3.4
			Secondary: 1.2
			≥ 12 yrs: 0.8
			Men, Concurrent Day
			None: -0.67 (-2.50-1.19)
			Primary: 1.82 (1.00-2.65)
			Secondary: 2.46 (1.31-3.63)
			≥ 12 yrs: 1.73 (0.47-3.00)
			Men, Lag 1
			None: -0.59 (-2.42-1.26)
			Primary: 1.59 (0.78-2.41)
			Secondary: 2.64 (1.49-3.80)
			≥ 12 yrs: 0.89 (-0.35-2.15)
			Men, Distributed Lags 0-5
			None: 1.50 (-1.52-4.60)
			Primary: 2.46 (1.20-3.74)
			Secondary: 2.24 (0.56-3.95)
			≥ 12 yrs: 1.45 (-0.34-3.29)
			Men, *df* (yrs)
			None: 4.6
			Primary: 3.0
			Secondary: 0.8
			≥ 12 yrs: 1.0

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			Santiago
			All Adults, Concurrent Day
			None: 1.49 (0.54-2.45)
			Primary: 0.28 (-0.03-0.59)
			Secondary: 0.58 (0.13-1.04)
			≥ 12 yrs: 2.32 (1.50-3.15)
			All Adults, Lag 1
			None: 2.20 (1.24-3.17)
			Primary: 0.74 (0.43-1.05)
			Secondary: 0.64 (0.20-1.11)
			≥ 12 yrs: 2.20 (1.36-3.04)
			All Adults, Distributed Lags 0-5
			None: 3.21 (1.54-4.90)
			Primary: 0.92 (0.38-1.46)
			Secondary: 1.46 (0.67-2.25)
			≥ 12 yrs: 4.02 (2.78-5.27)
			All Adults, *df* (yrs)
			None: 3.8
			Primary: 5.2
			Secondary: 4.0
			≥ 12 yrs: 1.8
			Women, Concurrent Day
			None: 1.39 (0.41-2.39)
			Primary: 0.4 (0.01-0.8)
			Secondary: 0.91 (0.29-1.53)
			≥ 12 yrs: 0.87 (-0.02-1.78)
			Women, Lag 1
			None: 1.83 (0.83-2.85)
			Primary: 0.98 (0.58-1.38)
			Secondary: 0.73 (0.11-1.35)
			≥ 12 yrs: 0.76 (-0.15-1.68)
			Women, Distributed Lags 0-5
			None: 2.47 (0.85-4.11)
			Primary: 1.2 (0.52-1.88)
			Secondary: 1.71 (0.65-2.78)
			≥ 12 yrs: 0.87 (-0.02-1.78)
			Women, *df* (yrs)
			None: 2.4
			Primary: 4.8
			Secondary: 4.4
			≥ 12 yrs: 0.6
			Men, Concurrent Day
			None: 0.54 (-0.51-1.61)
			Primary: 0.34 (-0.12-0.80)
			Secondary: 0.25 (-0.40-0.91)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			≥ 12 yrs: 1.97 (1.09-2.86)
			Men, Lag 1
			None: 0.84 (-0.21-1.91)
			Primary: 0.43 (-0.03-0.89)
			Secondary: 0.61 (-0.04-1.26)
			≥ 12 yrs: 1.57 (0.67-2.46)
			Men, Distributed Lags 0-5
			None: 2.41 (0.64-4.22)
			Primary: 0.80 (0.02-1.59)
			Secondary: 1.58 (0.45-2.71)
			≥ 12 yrs: 2.99 (1.66-4.33)
			Men, *df* (yrs)
			None: 2.0
			Primary: 4.4
			Secondary: 4.4
			≥ 12 yrs: 1.8
Reference: Peng et al. (2005, 087463)	**Outcome:** Mortality:	**Pollutant:** PM_{10}	**Increment:** 10 $\mu g/m^3$
Period of Study: 1987–2000	Non-accidental	**Averaging Time:** 24-h avg	**% Increase (Lower CI, Upper CI)**
Location: 100 U.S. cities (NMMAPS)	**Study Design:** Time-series	**Median (SD) unit:** 27.1	**lag:**
	Statistical Analyses: Bayesian semiparametric hierarchical models	**Range (Min, Max):** (13.2, 48.7)	Winter:
		Copollutant (correlation): NR	-0.4% (-0.30, 0.21)
	Age Groups: All ages		0
			0.15% (-0.08, 0.39)
			1
			0.10% (-0.13, 0.33)
			2
			Spring:
			0.32% (0.08, 0.56)
			0
			0.14% (-0.14, 0.42)
			1
			0.05% (-0.21, 0.32)
			2
			Summer:
			0.13% (-0.11, 0.37)
			0
			0.36% (0.11, 0.61)
			1
			-0.03% (-0.27, 0.21)
			2
			Fall:
			0.05% (-0.16, 0.25)
			0
			0.14% (-0.06, 0.34)
			1

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			0.13% (-0.08, 0.35)
			2
			All Seasons:
			0.09% (-0.01, 0.19)
			0
			0.19% (0.10, 0.28)
			1
			0.08% (-0.03, 0.19)
			2
			PM_{10} only (45 cities):
			Winter: 0.15% (-0.16, 0.45)
			1
			Spring: 0.13% (-0.21, 0.48)
			1
			Summer: 0.30% (-0.10, 0.69)
			1
			Fall: 0.07% (-0.23, 0.37)
			1
			PM_{10} + O_3 (45 cities):
			Winter: 0.18% (-0.16, 0.52)
			1
			Spring: 0.10% (-0.30, 0.49)
			1
			Summer: 0.33% (-0.14, 0.81)
			1
			Fall: 0.08% (-0.25, 0.41)
			1
			PM_{10} + O_3 (45 cities):
			Winter: 0.13% (-0.24, 0.49)
			1
			Spring: 0.1% 9(-0.18, 0.56)
			1
			Summer: 0.28% (-0.13, 0.70)
			1
			Fall: -0.01% (-0.34, 0.31)
			1
			PM_{10} + NO_2 (45 cities):
			Winter: 0.21% (-0.18, 0.60)
			1
			Spring: 0.19% (-0.17, 0.54)
			1
			Summer: 0.34% (0.01, 0.68)
			1
			Fall: 0.13% (-0.12, 0.39)
			1

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Penttinen et al. (2004, 087432)	**Outcome:** Mortality:	**Pollutant:** PM_{10}	**Increment:** 10 $\mu g/m^3$
Period of Study: 1988–1996	Total (non-accidental) (< 800)	**Averaging Time:** 24-h avg	**% Increase (Lower CI, Upper CI)**
Location: Helsinki, Finland	Cardiovascular (390-459)	**Median (SD) unit:** 21 $\mu g/m^3$	**lag:**
	Respiratory (460-519)	**Range (Min, Max):** (0.2, 213)	Total (non-accidental)
	Study Design: Time-series	**Copollutant (correlation):**	-0.23% (-1.47, 1.01)
	Statistical Analyses: Poisson GAM, LOESS	O_3: r = -0.09	0
		NO_2: r = 0.50	0.88% (-0.32, 2.08)
	Age Groups: 15-64	CO: r = 0.45	1
	65-74	SO_2: r = 0.61	0.11 (-0.51, 0.73)
	≥ 75	TSP: r = 0.72	0-3 avg
			Cardiovascular
			-1.22% (-3.00, 0.56)
			0
			0.63% (-1.09, 2.35)
			1
			0.08% (-0.96, 0.81)
			0-3 avg
			Respiratory
			3.94% (0.01, 7.87)
			0
			3.96% (0.11, 7.81)
			1
			2.13% (0.03, 4.22)
			0-3 avg

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Qian et al. (2007, 093054)	**Outcome:** Mortality:	**Pollutant:** PM_{10}	**Increment:** 10 $\mu g/m^3$
Period of Study: 2001–2004	Total (non-accidental) (< 800)	**Averaging Time:** 24-h avg	**% Increase (Lower CI, Upper CI)**
Location: Wuhan, China	Cardiovascular (390-459)	**Mean (SD):** 141.8 [3]	**lag:**
	Stroke (430-438)	**Range (Min, Max):** (24.8, 477.8)	Non-accidental
	Cardiac Diseases (390-398)	**Copollutant (correlation):**	0.36% (0.19, 0.53)
	Respiratory (460-519)	NO_2	0
	Cardiopulmonary	SO_2	0.28% (0.12, 0.45)
	Study Design: Time-series	O_3	1
	Statistical Analyses: Poisson GAM, natural splines		0.43% (0.24, 0.62)
			0-1
	Age Groups: All ages		0.08% (-0.15, 0.31)
	< 45		0-4
	≥ 45		< 45
	< 65		0.28% (-0.26, 0.82)
	≥ 65		0
			0.45% (-0.06, 0.96)
			1
			0.53% (-0.08, 1.13)
			0-1
			0.41% (-0.31, 1.13)
			0-4
			≥ 45
			0.36% (0.19, 0.54)
			0
			0.27% (0.10, 0.44)
			1
			0.42% (0.22, 0.62)
			0-1
			0.05% (-0.18, 0.29)
			0-4
			< 65
			0.20% (-0.08, 0.49)
			0
			0.25% (-0.03, 0.52)
			1
			0.33% (0.01, 0.66)
			0-1
			0.01% (-0.38, 0.39)
			0-4
			≥ 65
			0.41% (0.21, 0.61)
			0
			0.30% (0.10, 0.49)
			1
			0.46% (0.24, 0.69)
			0-1
			0.10% (-0.16, 0.37)
			0-4
			Cardiovascular
			0.51% (0.28, 0.75)
			0

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Qian et al. (2008, 156894)	**Outcome:** Mortality:	**Pollutant:** PM_{10}	**Increment:** 10 $\mu g/m^3$
Period of Study: 7/2001–6/2004	Total (non-accidental) (< 800)	**Averaging Time:** 24-h avg	**% Increase (Lower CI, Upper CI)**
Location: Wuhan, China	Cardiovascular (390-459)	**Mean (SD):**	**lag:**
	Stroke (430-438)	Normal temperature: 145.7 (64.6)	Non-accidental:
	Cardiac diseases (390-398, 410-429)	Low temperature: 117.3 (49.5)	Normal:
	Respiratory (460-519)	High temperature: 96.3 (27.9)	All ages: 0.36 (0.17, 0.56)
	Cardiopulmonary (390-459, 460-519)	**Range (Min, Max):** NR	0-1
	Study Design: Time-series	**Copollutant (correlation):**	< 65: 0.23 (-0.10, 0.56)
	Statistical Analyses: Poisson GLM, natural splines and penalized splines	Normal temperature:	0-1
	Age Groups: All ages	NO_2: r = 0/72	≥ 65: 0.51 (0.18, 0.64)
	< 65	SO_2: r = 0.59	0-1
	≥ 65	O_3: r = 0.06	$PM_{10}+NO_2$: 0.07 (-0.17, 0.30)
		Low temperature:	0-1
		NO_2: r = 0.83	$PM_{10}+SO_2$: 0.27 (0.06, 0.47)
		SO_2: r = 0.74	0-1
		O_3: r = 0.19	$PM_{10}+O_3$: 0.38 (0.18, 0.58)
		High temperature:	0-1
		NO_2: r = 0.68	Low:
		SO_2: r = 0.15	All ages: 0.62 (-0.09, 1.34)
		O_3: r = 0.65	0-1
			< 65: 1.78 (0.52, 3.05)
			0-1
			≥ 65: 0.22 (-0.61, 1.05)
			0-1
			$PM_{10}+NO_2$: 0.24 (-0.49, 0.97)
			0-1
			$PM_{10}+SO_2$: 0.45 (-0.27, 1.17)
			0-1
			$PM_{10}+O_3$: 0.72 (0.00, 1.44)
			0-1
			High:
			All ages: 2.20 (0.74, 3.68)
			0-1
			< 65: 2.34 (-0.09, 4.83)
			0-1
			≥ 65: 2.14 (0.42, 3.89)
			0-1
			$PM_{10}+NO_2$: 1.87 (0.42, 3.35)
			0-1
			$PM_{10}+SO_2$: 2.12 (0.67, 3.60)
			0-1
			$PM_{10}+O_3$: 2.15 (0.55, 3.77)
			0-1
			Cardiovascular:
			Normal:
			All ages: 0.39 (0.11, 0.66)
			0-1
			< 65: 0.17 (-0.40, 0.73)
			0-1
			≥ 65: 0.44 (0.14, 0.74)
			0-1

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Ren et al. (2006, 092824)	**Outcome:** Mortality:	**Pollutant:** PM_{10}	The study presents quantitative results associated with an incremental increase in temperature, not PM_{10}.
Period of Study: 1/1996–12/2001	Non-accidental	**Averaging Time:** 24-h avg	
Location: Brisbane, Australia	Cardiovascular (390-448)	**Mean (SD):** 15.84	
	Study Design: Time-series	**Range (Min, Max):** (2.5, 60)	
	Statistical Analyses: Poisson GAM, cubic spline	**Copollutant:** O_3	
	Age Groups: All ages		

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Roberts (2004, 087924)	**Outcome:** Mortality:	**Pollutant:** PM_{10}	**Increment:** 10 $\mu g/m^3$
Period of Study: 1987–1994	Non-accidental (< 800)	**Averaging Time:** 24-h avg	**% Increase (SE)**
Location: Cook County, Illinois	**Study Design:** Time-series	**Median (SD) unit:**	**lag:**
Allegheny County, Pennsylvania	**Statistical Analyses:** Poisson GAM, smooth splines	Cook County	GLM
	Poisson GLM, natural cubic splines	Lower Temp.: 29.24	Cook
	Age Groups: ≥ 65	Middle Temp.: 30.03	□ = 0.5
		Upper Temp.: 52.76	No Interaction: 0.288% (0.157)
		Allegheny County	0
		Lower Temp.: 16.50	Low Temp.: -0.272% (0.380)
		Middle Temp.: 24.97	0
		Upper Temp.: 55.42	Middle Temp.: 0.344% (0.165)
		Range (10th, 90th):	0
		Cook County	Upper Temp.: 0.281% (0.239)
		Lower Tem.: (16.42, 46.42)	0
		Middle Temp.: (14.79, 56.33)	No Interaction: 0.359% (0.149)
		Upper Temp.: (30.81, 82.81)	1
		Allegheny County	Low Temp.: -0.168% (0.372)
		Lower Temp.: (5.14, 34.54)	1
		Middle Temp.: (8.91, 57.91)	Middle Temp.: 0.361% (0.156)
		Upper Temp.: (30.91, 88.99)	1
			Upper Temp.: 0.616% (0.250)
			1
			No Interaction: 0.465% (0.176)
			0-1 ma
			Low Temp.: 0.043% (0.397)
			0-1 ma
			Middle Temp.: 0.506% (0.184)
			0-1 ma
			Upper Temp.: 0.464% (0.256)
			0-1 ma
			No Interaction: 0.633% (0.214)
			0-3 ma
			Low Temp.: 0.365% (0.419)
			0-3 ma
			Middle Temp.: 0.638% (0.222)
			0-3 ma
			Upper Temp.: 0.718% (0.295)
			0-3 ma
			□ = 1
			No Interaction: 0.117% (0.157)
			0
			Low Temp.: -0.351% (0.406)
			0
			Middle Temp.: 0.161% (0.165)
			0
			Upper Temp.: 0.096% (0.264)
			0
			No Interaction: 0.141% (0.150)
			1
			Low Temp.: -0.366% (0.397)
			1

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Roberts (2004, 087924) **Period of Study:** 1987–1994 **Location:** Cook County, Illinois Allegheny County, Pennsylvania	**Outcome:** Mortality: Non-accidental **Study Design:** Time-series **Statistical Analyses:** Poisson GLM **Age Groups:** ≥ 65	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** NR **Range (Min, Max):** Max = 89	The study does not present quantitative results.
Reference: Roberts ((2005, 087992) **Period of Study:** Cook County: 1987–2000. Allegheny County: 1987- 1998 **Location:** Cook County, Illinois Allegheny County, Pennsylvania	**Outcome:** Mortality: Non-accidental **Study Design:** Time-series **Statistical Analyses:** Poisson **Age Groups:** ≥ 65	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** NR **Range (Min, Max):** NR **Copollutant (correlation):** NR	**Increment:** NR **β (SE)** **lag:** Standard Model Cook County 0.000127 (0.000264) 0 -0.000042 (0.000249) 1 -0.000441 (0.000246) 2 Allegheny County 0.000693 (0.000437) 0 0.000356 (0.000423) 1 0.000524 (0.000415) 2 Moving Total Model Cook County 0.000150 (0.000187) k = 2 -0.000047 (0.000153) k = 3 0.000009 (0.000133) k = 4 Allegheny County 0.000633 (0.000310) k = 2 0.000542 (0.000255) k = 3 0.000598 (0.000351) k = 4

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Roberts (2006, 089762) **Period of Study:** 1987–2000 **Location:** Cook County, Illinois Suffolk County, Massachusetts (NMMAPS)	**Outcome:** Mortality: Non-accidental **Study Design:** Time-series **Statistical Analyses:** Poisson GLM **Age Groups:** ≥ 65	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** Cook County: 33.7 (19.4) Suffolk County: 25.9 (11.8) Range (10th, 90th): Cook County: (13.4, 58.1) Suffolk County: (14.0, 41.7) **Copollutant (correlation):** Cook County CO: r = 0.30 NO_2: r = 0.53 SO_2: r = 0.45 O_3: r = 0.44 Suffolk County CO: r = 0.33 NO_2: r = 0.43 SO_2: r = 0.23 O_3: r = 0.36	**Increment:** Cook County: 19.4 $\mu g/m^3$ Suffolk County: 14.0 $\mu g/m^3$ **% Increase (SD)** **lag:** Cook County Standard Model: 0.49% (0.25) 0 Proposed Model: 0.29% (0.16) 0 Standard Model: 0.67% (0.25) 0-2 avg Proposed Model: 0.49% (0.25) 0-2 avg Suffolk County Standard Model: 0.88% (1.27) 0 Proposed Model: 0.85% (0.84) 0 Standard Model: 1.60% (0.71) 0-2 avg Proposed Model: 1.35% (0.73) 0-2 avg
Reference: Roberts and Martin (2006, 097799) **Period of Study:** 1987–2000 **Location:** Cook County, Illinois (NMMAPS)	**Outcome:** Mortality: Non-accidental **Study Design:** Time-series **Statistical Analyses:** Dose-response 1. Piecewise linear relationship (no-threshold) with change point at 25 $\mu g/m^3$ and 50 $\mu g/m^3$ 2. Piecewise linear relationship (threshold), exposure below 25 $\mu g/m^3$ no effect, and exposures above 50 $\mu g/m^3$ having a different effect then exposures between 25 $\mu g/m^3$ and 50 $\mu g/m^3$ **Age Groups:** ≥ 65	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** NR **IQR (25th, 75th):** (23.9, 45.4) Suffolk County: (14.0, 41.7) **Copollutant (correlation):** NR	The study does not present quantitative results.
Reference: Roberts and Martin (2006, 088670) **Period of Study:** 1987–2000 **Location:** 109 U.S. cities (NMMAPS)	**Outcome:** Mortality: Non-accidental Cardiorespiratory **Study Design:** Time-series **Statistical Analyses:** Poisson 2-stage Bayesian hierarchical model **Age Groups:** All ages	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** NR **IQR (25th, 75th):** NR **Copollutant (correlation):** NR	**Increment:** NR **β x 1000 (SE x 1000)** **lag:** Non-accidental Model 1 Base df: 0.079 (0.050) 0 Double df: 0.044 (0.046) 0 Half df: 0.107 (0.052) 0

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			Base df: 0.180 (0.044)
			1
			Double df: 0.149 (0.047)
			1
			Half df: 0.254 (0.048)
			1
			Base df: 0.059 (0.056)
			2
			Double df: 0.024 (0.056)
			2
			Half df: 0.143 (0.054)
			2
			Model 2
			Base df: 0.115 (0.037)
			0-2 ma
			Double df: 0.107 (0.034)
			0-2 ma
			Half df: 0.145 (0.039)
			0-2 ma
			Cardio-respiratory
			Model 1
			Base df: 0.103 (0.068)
			0
			Double df: 0.056 (0.067)
			0
			Half df: 0.134 (0.066)
			0
			Base df: 0.232 (0.060)
			1
			Double df: 0.179 (0.067)
			1
			Half df: 0.309 (0.059)
			1
			Base df: 0.210 (0.078)
			2
			Double df: 0.144 (0.075)
			2
			Half df: 0.305 (0.079)
			2
			Model 2
			Base df: 0.168 (0.047)
			0-2 ma
			Double df: 0.140 (0.044)
			0-2 ma
			Half df: 0.196 (0.051)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			0-2 ma Notes: Model 1 uses current day's mortality count, while Model 2 uses a 3-day moving total mortality count.
Reference: Roberts and Martin (2007, 156917) **Period of Study:** 1987–2000 **Location:** 8 U.S. cities and > 100 U.S. cities (NMMAPS)	**Outcome:** Mortality: Total (non-accidental) Cardiorespiratory **Study Design:** Time-series **Statistical Analyses:** Poisson **Age Groups:** All ages	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** NR **Range (Min, Max):** NR	**Increment:** 10 $\mu g/m^3$ **β x 1000 (SE x 1000)** **lag:** 8 U.S. cities Distributed Lag Model: 0.229 0-2 Weighted Model: 0.315 0-2 Standard Model: 0.276 0 -0.062 1 0.476 2 90 U.S. cities Total (non-accidental) Standard Model: 0.078 (0.039) 0 0.182 (0.037) 1 0.108 (0.036) 2 Moving Total Model: 0.131 (0.023) 0-2 Weighted Model: 0.274 (0.075) 0-2 Cardio-respiratory Standard Model: 0.096 (0.055) 0 0.232 (0.053) 1 0.226 (0.051) 2 Moving Total Model: 0.174 (0.032) 0-2 Weighted Model: 0.389 (0.105) 0-2 Notes: The 8 U.S. cities consist of Chicago, Cleveland, Denver, El Paso, Houston, Nashville, Pittsburgh, and Salt

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			Lake City.
Reference: Roberts and Martin (2007, 156916) **Period of Study:** 1987–2000 **Location:** 10 U.S. cities (NMMAPS)	**Outcome:** Mortality: Non-accidental **Study Design:** Time-series **Statistical Analyses:** Poisson **Age Groups:** ≥ 65	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** Anchorage: 27.32 Chicago: 36.95 Cleveland: 39.83 Detroit: 40.78 El Paso: 40.14 Minneapolis/St. Paul: 28.01 Pittsburgh: 35.09 Salt Lake City: 37.40 Seattle: 28.72 Spokane: 34.52 **Range (Min, Max):** NR	**Increment:** NR **β Coefficient (SE)** **lag:** Pooled Estimates Combined Model (Unconstrained Distributed Lag Model + Piecewise Linear Dose-Response Function) Change-point: 60 $\mu g/m^3$ Slope below: 0.00130 (0.00016) 0-5 Slope above: -0.00163 (0.00026) 0-5 Change-point: 30 $\mu g/m^3$ Slope below: 0.00014 (0.00039) 0-5 Slope above: -0.00003 (0.00015) 0-5 Piecewise Linear Dose-Response Model Change-point: 60 $\mu g/m^3$ Slope below: 0.00044 (0.00011) 3-day ma Slope above: -0.00077 (0.00020) 3- day ma Change-point: 30 $\mu g/m^3$ Slope below: 0.00022 (0.00026) 3-day ma Slope above: -0.00004 (0.00011) 3-day ma Polynomial Distributed Lag Model (degree 2) 0.00046 (0.00011) 0-5
Reference: Samoli et al. (2005, 087436) **Period of Study:** 1990–1997 **Location:** 22 European cities (APHEA-2)	**Outcome:** Mortality: All-cause (non-accidental) (< 800) Cardiovascular (390-459) Respiratory (460-519) **Study Design:** Time-series **Statistical Analyses:** Hierarchical modeling: 1. Poisson GAM, penalized splines 2. Multivariate modeling **Age Groups:** All ages	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg Median (SD) unit: Range: (Stockholm: 14 $\mu g/m^3$ to Torino: 65 $\mu g/m^3$) Percentile (90th): Range: (Stockholm: 27 $\mu g/m^3$ to Torino: 129 $\mu g/m^3$) **Copollutant (correlation):** BS	The study does not present quantitative results.

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Schwartz (2004, 078998) **Period of Study:** 1986–1993 **Location:** 14 U.S. cities	**Outcome:** Mortality: Non-accidental (< 800) **Study Design:** Case-crossover Time-series **Statistical Analyses:** Conditional logistic regression Poisson **Age Groups:** All ages Notes: Case days matched to referent days that had the same temperature.	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** NR **Range (Min, Max):** NR **Copollutant (correlation):** NR	**Increment:** 10 $\mu g/m^3$ **% Increase (Lower CI, Upper CI)** **lag:** Overall: Two stage: 0.36% (0.22, 0.50) 1 Single stage: 0.33% (0.19, 0.46) 1 More winter temperature lags: Two Stage: 0.39% (0.23, 0.56) 1 One stage: 0.32% (0.19, 0.46) 1 Time stratified with temperature matching: Two Stage: 0.39% (0.19, 0.58) 1 One Stage: 0.53% (0.34, 0.72) 1 Poisson regression: 0.40% (0.18, 0.62) 1
Reference: Schwartz (2004, 053506) **Period of Study:** 1986–1993 **Location:** 14 U.S. cities	**Outcome:** Mortality: Non-accidental (< 800) **Study Design:** Case-crossover **Statistical Analyses:** Time-stratified conditional logistic regression **Age Groups:** All ages Notes: Case days matched to referent days based on concentration of gaseous air pollutants. Matched on the following conditions: 1. 24-h avg SO_2 within 1 ppb 2. Daily-maximum O_3 within 2 ppb 3. 24-h avg NO_2 within 1 ppb 4. 24-h avg CO within 0.03 ppm	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Median (SD) unit:** Range: 23 to 36 $\mu g/m^3$ IQR (25th, 75th): Range 25th: 17 to 24 $\mu g/m^3$ Range 75th: 31 to 57 $\mu g/m^3$ **Copollutant (correlation):** CO SO_2 NO_2 O_3	**Increment:** 10 $\mu g/m^3$ **β x 1000 (SE x 1000)** **lag:** Matched on CO: 0.527 (0.251) 0-1 avg Matched on O_3: 0.451 (0.170) 0-1 avg Matched on NO_2: 0.784 (0.185) 0-1 avg Matched on SO_2: 0.811 (0.175) 0-1 avg
Reference: Sharovsky et al. (2004, 156976) **Period of Study:** 7/1996–6/1998 **Location:** São Paulo, Brazil	**Outcome:** Mortality: Myocardial infarction **Study Design:** Time-series **Statistical Analyses:** Poisson GAM **Age Groups:** ≥ 35	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** 58.2 (25.8) **Range (Min, Max):** (23, 186) **Copollutant (correlation):** CO: r = 0.73 SO_2: r = 0.72	**Increment:** 10 $\mu g/m^3$ **β (SE)** **lag:** PM_{10}: 0.001 (0.001) $PM_{10}+CO+SO_2$: 0.0004 (0.0008)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Simpson et al. (2005, 087438) **Period of Study:** 1/1996–12/1999 **Location:** 4 Australian cities	**Outcome:** Mortality: Non-accidental (< 800) Cardiovascular (390-459) Respiratory (460-519) **Study Design:** Time-series meta-analysis **Statistical Analyses:** Poisson GAM, natural splines Poisson GLM, natural splines **Age Groups:** All ages	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** Brisbane: 16.60 Sydney: 16.30 Melbourne: 18.20 **Range (Min, Max):** Brisbane: (2.6, 57.6) Sydney: (3.7, 75.5) Melbourne: (3.3, 51.9) **Copollutant:** $PM_{2.5}$ CO NO_2	**Increment:** 10 $\mu g/m^3$ **% Increase (Lower CI, Upper CI)** **lag:** 0.2% (-0.8, 1.2)
Reference: Slaughter et al. (2005, 073854) **Period of Study:** 1/1995–12/1999 **Location:** Spokane, Washington	**Outcome:** Mortality: Non-accidental (< 800) **Study Design:** Time-series **Statistical Analyses:** Poisson GLM, natural splines **Age Groups:** All ages	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** NR **Range (9th, 95th):** (7.9, 41.9) $\mu g/m^3$ **Copollutant (correlation):** PM_{10} $PM_{10-2.5}$: r = 0.94 CO: r = 0.32	**Increment:** : 25 $\mu g/m^3$ **Relative Risk (Lower CI, Upper CI)** **lag:** 1.00 (0.97, 1.03) 1 0.98 (0.95, 1.01) 2 1.00 (0.97, 1.03) 3
Reference: Staniswalis et al. (2005, 087473) **Period of Study:** 1992–1995 **Location:** El Paso, Texas	**Outcome:** Mortality: Non-accidental (< 800) **Study Design:** Time-series **Statistical Analyses:** Poisson Principal component analysis (PCA) **Age Groups:** All	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** NR **Range (Min, Max):** (0.2, 133.4) **Notes:** The chemical composition and size distribution of PM was not available, therefore, the study used wind speed as a surrogate variable for the PM_{10} composition.	**Increment:** 10 $\mu g/m^3$ **% Increase (Lower CI, Upper CI)** **lag:** Poisson regression: 1.7% 3 PCA: 24-hly measurements: 2.06% 3 Daily avg: 1.7% 3

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Stafoggia et al. (2008, 157005) **Period of Study:** 1997–2004 **Location:** 9 Italian cities	**Outcome:** Mortality: Total (non-accidental) (< 800) Cardiovascular (390-459) Respiratory (460-519) Other natural causes **Study Design:** Time-stratified case-crossover **Statistical Analyses:** Conditional logistic regression **Age Groups:** ≥ 35	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD) unit:** Bologna: 50.4 (31.7) Florence: 37.5 (16.6) Mestre: 48.1 (26.8) Milan: 57.9 (38.0) Palermo: 36.2 (21.7) Pisa: 35.1 (14.9) Rome: 47.3 (19.9) Taranto: 59.8 (18.9) Turin: 71.5 (38.1) **Range (Min, Max):** NR **Copollutant (correlation):** NR	**Increment:** 10 $\mu g/m^3$ **% Increase (Lower CI, Upper CI)** **lag:** Cardiovascular All year: 0.63% (0.31, 1.38) 0-1 Winter: 0.15% (-0.29, 0.59) 0-1 Spring: 0.72% (-0.07, 1.52) 0-1 Summer: 2.90% (1.14, 4.69) 0-1 Fall: 1.37% (0.43, 2.32) 0-1 Apparent Temperature < 50th Percentile: 0.31% (-0.06, 0.67) 0-1 50th-75th Percentile: 2.05% (0.47, 3.66) 0-1 > 75th Percentile: 2.68% (1.20, 4.17) 0-1 Respiratory All year: 0.98% (0.27, 1.70) 0-1 Winter: 0.41% (-0.67, 1.51) 0-1 Spring: 2.99% (1.18, 4.83) 0-1 Summer: 3.89% (0.19, 7.73) 0-1 Fall: 0.45% (-1.11, 2.03) 0-1 Apparent Temperature < 50th Percentile: 0.54% (-0.47, 1.57) 0-1 50th-75th Percentile: 3.15% (0.64, 5.73) 0-1 > 75th Percentile: 4.12% (0.44, 7.93) 0-1 Other natural causes All year: 0.37% (0.09, 0.66) 0-1 Winter: 0.14% (-0.36, 0.63) 0-1 Spring: 0.29% (-0.47, 1.05) 0-1 Summer: 2.15% (0.90, 3.42) 0-1 Fall: 0.70% (-0.41, 1.83) 0-1 Apparent Temperature

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Stölzel et al. (2007, 091374)	**Outcome:**	**Pollutant:** PM_{10}	**Increment:** 23 $\mu g/m^3$
Period of Study: 9/1995–8/2001	Mortality:	**Averaging Time:** 24-h avg	**Relative Risk (Lower CI, Upper CI)**
Location: Erfurt, Germany	Total (non-accidental) (< 800)	**Mean (SD) unit:** : 31.9 (23.2)	**lag:**
	Cardio-respiratory (390-459, 460-519, 785, 786)	IQR (25th, 75th):	Total (non-accidental)
	Study Design: Time-series	(16.5, 39.5)	1.004 (0.980
	Statistical Analyses:	**Copollutant (correlation):**	1.029)
	Poisson GAM	$MC_{0.1\text{-}0.5}$: r = 0.85	0
	Age Groups: All ages	$MC_{0.01\text{-}2.5}$: r = 0.84	1.004 (0.981
		NO: r = 0.54	1.027)
		NO_2: r = 0.62	1
		CO: r = 0.50	0.998 (0.976
			1.021)
			2
			0.984 (0.962
			1.006)
			3
			0.993 (0.972
			1.015)
			4
			0.990 (0.969
			1.012)
			5
			Cardio-respiratory
			1.007 (0.981
			1.034)
			0
			1.006 (0.981
			1.032)
			1
			0.996 (0.971
			1.021)
			2
			0.977 (0.953
			1.002)
			3
			0.994 (0.970
			1.018)
			4
			0.993 (0.969
			1.017)
			5

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Sullivan et al. (2003, 043156)	**Outcome:**	**Pollutant:** PM_{10}	**Increment: :** 16.51 $\mu g/m^3$
Period of Study:	Out-of-hospital cardiac arrest	**Averaging Time:** 24-h avg	**Odds Ratio (Lower CI, Upper CI)**
1985–1994	**Study Design:** Case-crossover	**Median (SD) unit:**	**lag:**
Location: Western Washington	**Statistical Analyses:**	Lag 0: 28.05	Overall
	Conditional logistic regression	Lag 1: 27.97	1.05 (0.87, 1.27)
	Age Groups: 19-79	Lag 2: 28.40	0
	Study Population: Out-of-hospital cardiac arrests: 1,206	**Range (Min, Max):**	0.91 (0.75, 1.11)
		(7.38, 89.83)	1
		Copollutant (correlation):	1.03 (0.82, 1.28)
		SO_2	2
		CO	
		Notes: Study used nephelometry to measure particles and equated the measurements to $PM_{2.5}$ concentrations.	
Reference: Sunyer et al. (2002, 034835)	**Outcome:** Mortality:	**Pollutant:** PM_{10}	**Increment:** 32.7 $\mu g/m^3$
Period of Study: 1985–1995	Respiratory mortality	**Averaging Time:** 24-h avg	**Odds Ratio (Lower CI, Upper CI)**
Location: Barcelona, Spain	**Study Design:** Case-crossover	**Median (SD) unit:** 61.2	**lag:**
	Statistical Analyses: Condition logistic regression	**Range (Min, Max):** (17.3, 240.7)	Asthmatic individuals with 1 ED visit
	Age Groups: >14	**Copollutant:**	0.884 (0.672, 1.162)
	Study population: Asthmatic individuals: 5,610	BS	0-2 avg
		NO_2	Asthmatic individuals with >1 ED visit
		O_3	1.084 (0.661, 1.778)
		SO_2	0-2 avg
		CO	Asthma/COPD individuals with >1 ED visit
			1.011 (0.746, 1.368)
			0-2 avg

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Touloumi et al. (2005, 087477) **Period of Study:** 1990–1997 **Location:** 7 European cities (London, Budapest, Stockholm, Zurich, Paris, Lyon, Madrid) (APHEA2)	**Outcome:** Mortality: Total (non-accidental) (< 800) Cardiovascular (390-459) **Study Design:** Time-series **Statistical Analyses:** Poisson GAM, LOESS **Age Groups:** All ages	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Median (SD) unit:** London: 25.1 Budapest: 40.2 Stockholm: 13.7 Zurich: 27.5 Paris: 22.2 Lyon: 38.5 μ Madrid: 33.4 IQR (25th, 75th): London: (20.3, 33.9) Budapest: (34.3, 45.8) Stockholm: (10.3, 19.1) Zurich: (19.2, 38.5) Paris: (16.0, 33.0) Lyon: (29.7, 50.4) Madrid: (27.6, 41.0) **Copollutant (correlation):** NR	**Increment:** 10 $\mu g/m^3$ **β (x 1000) (SE (x 1000)):** Total (non-accidental) No control: 0.4834 (0.1095) Reported Influenza Data Count ID: 0.4967 (0.1089) I1 ID: 0.4740 (0.1090) MI ID: 0.5019 (0.1096) RI-ID: 0.4735 (0.1091) SF ID: 0.6714 (0.1080) Estimated Influenza Data APHEA-2: 0.5550 (0.1076) I1 EID: 0.5640 (0.1073) MI EID: 0.5872 (0.1100) RI EID: 0.5872 (0.1074) SF EID: 0.6641 (0.1073) Cardiovascular No control: 0.8432 (0.1665) Reported Influenza Data Count ID: 0.8896 (0.1662) I1 ID: 0.8545 (0.1661) MI ID: 0.8693 (0.1674) RI-ID: 0.8649 (0.1665) SF ID: 1.0107 (0.1659) Estimated Influenza Data APHEA-2: 0.9389 (0.1654) I1 EID: 0.9485 (0.1648) MI EID: 1.0440 (0.1686) RI EID: 0.9718 (0.1653) SF EID: 1.0585 (0.1652) **Notes:** I1 = one indicator for all epidemics M1 = multiple indicators, one per epidemic R1 = indicators for intervals indicating the range of influenza counts SF = separate smooth function during epidemic periods.

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Tsai et al. (2003, 050480)	**Outcome:** Mortality:	**Pollutant:** PM_{10}	**Increment:** 67.00 $\mu g/m^3$
Period of Study: 1994–2000	Total (non-accidental) (< 800)	**Averaging Time:** 24-h avg	**Odds Ratio (Lower CI, Upper CI)**
Location: Kaohsiung, Taiwan	Respiratory (460-519)	**Mean (SD):** 81.45	**lag:**
	Circulatory (390-459)	**Range (Min, Max):** (20.50, 232.00)	Total (non-accidental)
	Study Design: Bidirectional case-crossover	**Copollutant:**	1.000 (0.947, 1.056)
		SO_2	0-2 avg
	Statistical Analyses: Conditional logistic regression	NO_2	Respiratory
		CO	1.023 (0.829, 1.264)
	Age Groups: All ages	O_3	0-2 avg
			Circulatory
			0.971 (0.864, 1.092)
			0-2 avg

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Vajanapoom et al. (2002, 042542) **Period of Study:** 1992–1997 **Location:** Bangkok, Thailand	**Outcome:** Mortality: Total (non-accidental) (< 800) Respiratory (460-519) Cardiovascular (390-459) Other-causes **Study Design:** Time-series **Statistical Analyses:** Poisson GAM, LOESS **Age Groups:** All ages 55-64 65-74 ≥ 75	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** 68.0 (23.9) IQR (25th, 75th): (50.1, 80.7) **Copollutant (correlation):** NR	**Increment:** 30 $\mu g/m^3$ **% Increase (Lower CI, Upper CI)** **lag:** Total (non-accidental) All ages: 2.3% (1.3, 3.3) 0-4 ma 55-64: 1.5% (-0.8, 3.9) 0-4 ma 65-74: 4.2% (2.0, 6.3) 0-4 ma ≥ 75: 3.9% (2.1, 5.6) 0-4 ma Cardiovascular All ages: 0.8% (-0.9, 2.4) 0 55-64: -2.5% (-6.3, 1.3) 0 65-74: 2.9% (-0.7, 6.5) 0 ≥ 75: 1.6% (-1.8, 5.0) 0 Respiratory All ages: 5.1% (0.6, 9.6) 0-2 ma 55-64: 1.4% (-11.3, 14.2) 0-2 ma 65-74: 2.8% (-9.5, 15.2) 0-2 ma ≥ 75: 10.2% (-0.1, 20.5) 0-2 ma Other-causes All ages: 2.4% (1.3, 3.5) 0-4 ma 55-64: 1.7% (-1.1, 4.5) 0-4 ma 65-74: 5.6% (3.1, 8.1) 0-4 ma ≥ 75: 3.7% (1.8, 5.6) 0-4 ma

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Vedal et al. (2003, 039044) **Period of Study:** 1/1994–12/1996 **Location:** Vancouver, British Columbia, Canada	**Outcome:** Mortality: Total (non-accidental) (< 800) Respiratory (460-519) Cardiovascular (390-459) **Study Design:** Time-series **Statistical Analyses:** **Poisson** GAM, LOESS **Age Groups:** All ages	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** 14.4 (5.9) **Range (Min, Max):** (4.1, 37.2) **Copollutant (correlation):** O_3: r = 0.48 SO_2: r = 0.76 NO_2: r = 0.84 CO: r = 0.71	The study does not present quantitative results
Reference: Venners et al. (2003, 089931) **Period of Study:** 1/1995–12/1995 **Location:** Chongqing, China	**Outcome:** Mortality: Total (non-accidental) (< 800) **Study Design:** Time-series **Statistical Analyses:** Poisson GAM, cubic spline **Age Groups:** All ages	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** 146.8 **Range (Min, Max):** (44.7, 666.2) **Copollutant:** SO_2 **Notes:** PM_{10} was measured for only 7 months of the study period.	**Increment:** 100 $\mu g/m^3$ **Relative Risk (Lower CI, Upper CI)** **lag:** 1.00 (0.93, 1.07) 0 0.98 (0.91, 1.04) 1 1.00 (0.93, 1.07) 2 0.96 (0.90, 1.03) 3 0.97 (0.90, 1.03) 4 0.99 (0.93, 1.06) 5

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Vichit-Vadakan et al. (2008, 157095) **Period of Study:** 1/1999–12/2003 **Location:** Bangkok, Thailand	**Outcome (ICD10):** Mortality: Non-accidental (A00-R99) Cardiovascular (I00-I99) Ischemic heart diseases (I20-I25) Stroke (I60-I69) Conduction disorder (I44-I49) Respiratory (J00-J98) Lower Respiratory Infection (J10-J22) COPD (J40-J47) Asthma (J45-J46) Senility (R54) **Study Design:** Time-series **Statistical Analyses:** Poisson, natural cubic spline **Age Groups:** All ages 0-4 5-44 18-50 45-64 ≥ 50 ≥ 65 ≥ 75	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** 52.1 (20.1) **Range (Min, Max):** (21.3, 169.2) **Copollutant (correlation):** NR	**Increment:** 10 $\mu g/m^3$ **% Excess Risk (Lower CI, Upper CI)** **lag:** Cause-specific mortality: Nonaccidental: 1.3% (0.8, 1.7) 0-1 Cardiovascular: 1.9% (0.8, 3.0) 0-1 Ischemic heart disease: 1.5% (–0.4, 3.5) 0-1 Stroke: 2.3% (0.6, 4.0) 0-1 Conduction disorders: –0.%3 (–5.9, 5.6) 0-1 Cardiovascular: ≥ 65 1.8 (0.2, 3.3) 0-1 Respiratory All ages: 1.0 (–0.4, 2.4) 0-1 □ 1: 14.6 (2.9, 27.6) 0-1 ≥ 65: 1.3 (–0.8, 3.3) 0-1 LRI: < 5: 7.7 (–3.6, 20.3) 0-1 COPD: 1.3 (–1.8, 4.4) 0-1 Asthma: 7.4 (1.1, 14.1) 0-1 Senility: 1.8 (0.7, 2.8) 0-1 Age-specific for non-accidental 0–4: 0.2 (–2.0, 2.4) 0-1 5–44: 0.9 (0.2, 1.7) 0-1 18–50: 1.2 (0.5, 1.9) 0-1 45–64: 1.1 (0.4, 1.9) 0-1 ≥ 50: 1.4 (0.9, 1.9) 0-1 ≥ 65: 1.5 (0.9, 2.1) 0-1 ≥ 75: 2.2 (1.3, 3.0) 0-1 Sex-specific for non-accidental Male: 1.2 (0.7, 1.7) 0-1

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Villeneuve et al. (2003, 055051)	**Outcome:** Mortality:	**Pollutant:** PM_{10}	**Increment:** 15.4 $\mu g/m^3$
Period of Study: 1986–1999	Non-accidental (< 800)	**Averaging Time:** 24-h avg	**% Increase (Lower CI, Upper CI)**
Location: Vancouver, Canada	Cardiovascular (401-440)	**Mean (SD):**	**lag:**
	Respiratory (460-519)	Daily 14.0	Non-accidental
	Cancer (140-239)	Every 6th Day 19.6	3.7% (-0.5, 8.0)
	Study Design: Time-series	**Range (Min, Max):**	0-2 avg
	Statistical Analyses: Poisson, natural splines	Daily (3.8, 52.2)	2.6% (-0.9, 6.1)
	Age Groups: ≥ 65	Every 6th Day (3.5, 63.0)	0
		Copollutant:	2.7% (-0.7, 6.2)
		SO_2	1
		CO	1.9% (-1.4, 5.3)
		NO_2	2
		O_3	Cardiovascular
		$PM_{2.5}$	3.4% (-2.7, 9.8)
		PM_{10}-2.5	0-2 avg
			5.1% (0.0, 10.4)
			0
			1.3% (-3.8, 6.7)
			1
			0.6% (-4.3, 5.7)
			2
			Respiratory
			PM_{10}
			0.1% (-9.5, 10.8)
			0-2 avg
			1.0% (-7.5, 10.4)
			0
			0.4% (-7.7, 9.3)
			1
			-1.3% (-8.9, 7.1)
			2
			Cancer
			1.2% (-6.9, 10.1)
			0-2 avg
			-2.5% (-8.8, 4.3)
			0
			2.3% (-4.6, 9.6)
			1
			3.3% (-3.7, 10.8)
			2

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Welty et al. (2008, 157134) **Period of Study:** 1987–2000 **Location:** Chicago, Illinois	**Outcome:** Mortality: Total (non-accidental) **Study Design:** Time-series **Statistical Analyses:** Poisson–Gibbs Sampler Bayesian Distributed Lag Model **Age Groups:** All ages	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** NR **Range (Min, Max):** NR **Copollutant (correlation):** NR	**Increment:** 10 $\mu g/m^3$ % Excess Risk (Lower CI, Upper CI) lag: Poisson–Gibbs Sampler 0.17% (0.01, 0.34) 3 -0.24% (-0.73, 0.23) 0-14 Unconstrained: -0.19% (-0.86, 0.48) 0-14 Bayesian Distributed Lag Model -0.21% (-0.86, 0.41) 0-14
Reference: Welty and Zeger (2005, 087484) **Period of Study:** 1987–2000 **Location:** 100 U.S. cities (NMMAPS)	**Outcome:** Mortality: Total (non-accidental) (< 800) **Study Design:** Time-series **Statistical Analyses:** Bayesian hierarchical model **Age Groups:** All ages	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** NR **Range (Min, Max):** NR **Copollutant (correlation):** NR	**Increment:** 10 $\mu g/m^3$ **% Increase (SE)** **lag:** Distributed Lag Model: Seasonally-Temporally Varying Temperature variables: 0, 1-2, 1-7, 1-14 S(t, 1 × years): 0.229 (0.053) 1 S(t, 2 × years): 0.220 (0.053) 1 S(t, 4 × years): 0.187 (0.050) 1 S(t, 8 × years): 0.178 (0.049) 1 Temperature variables: 0, 1-2, 1-7, 1-14, 0×1-2, 0×1-7, 1-2 × 1-7 S(t, 1 × years): 0.195 (0.048) 1 S(t, 2 × years): 0.200 (0.051) 1 S(t, 4 × years): 0.176 (0.050) 1 S(t, 8 × years): 0.149 (0.050) 1 Distributed Lag Model: Nonlinear Temperature variables: 0, 1-2, 1-7, 1-14 S(t, 4 × years): 0.239 (0.053) 1 Temperature variables: 0, 1-2, 1-7, 1-14, 0×1-2, 0×1-7, 1-2 × 1-7 S(t, 4 × years): 0.172 (0.045) 1 Temperature variables: S(0,2), S(1-2,2), S(1-7,2), S(1-14,2)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			S(t, 4 × years): 0.186 (0.046) 1 Temperature variables: S(0,2), S(1-2,2), S(1-7,2), S(1-14,2), S(0×1-2,2), S(0×1-7,2), S(1-2 × 1-7,2) S(t, 4 × years): 0.189 (0.047) 1 Temperature variables: S(0,4), S(1-2,4), S(1-7,4), S(1-14,4) S(t, 4 × years): 0.175 (0.046) 1 Temperature variables: S(0,4), S(1-2,4), S(1-7,4), S(1-14,4), S(0×1-2,4), S(0×1-7,4), S(1-2 × 1-7,4) S(t, 4 × years): 0.190 (0.048) 1 Temperature variables: 0, 1-2, 1-7 S(t, 4 × years): 0.252 (0.053) 1 Temperature variables: 0, 1-2, 1-7, 0×1-2, 0×1-7, 1-2 × 1-7 S(t, 4 × years): 0.186 (0.044) 1 Temperature variables: S(0,2), S(1-2,2), S(1-7,2) S(t, 4 × years): 0.198 (0.046) 1 Temperature variables: S(0,2), S(1-2,2), S(1-7,2), S(0×1-2,2), S(0×1-7,2), S(1-2 × 1-7,2) S(t, 4 × years): 0.201 (0.047) 1 Temperature variables: S(0,4), S(1-2,4), S(1-7,4) S(t, 4 × years): 0.189 (0.045) 1 Temperature variables: S(0,4), S(1-2,4), S(1-7,4), S(0×1-2,2), S(0×1-7,4), S(1-2 × 1-7,2) S(t, 4 × years): 0.205 (0.047) 1 Temperature variables: S(0,4), S(1-2,4) S(t, 4 × years): 0.250 (0.045) 1 Temperature variables: S(0,4), S(1-2,4), S(0×1-2,4) S(t, 4 × years): 0.253 (0.044) 1 Temperature variables: S(0,4) S(t, 4 × years): 0.220 (0.045)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			1 Notes: 0 indicates current-day temperature 1-r indicates avg of lag 1 through lag r temperature S (, ◘) indicates a natural spline smooth with ◘ degrees of freedom. S (t, ◘ x years) indicates the natural spline smooth of time with degrees of freedom equal to ◘ x (number of years of data).
Reference: Wong et al. (2007, 098391) **Period of Study:** 1/1998–12/1998 **Location:** Hong Kong, China	**Outcome:** Mortality: Total (non-accidental) (< 800) Cardiorespiratory (390-519) **Study Design:** Main analysis: Time-series **Sensitivity analysis:** Case-crossover, case-only **Statistical Analyses:** Main analysis: Poisson GAM **Sensitivity analysis:** Conditional logistic regression **Age Groups:** ≥ 30 ≥ 65	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** 48.1 (24.3) **Range (Min, Max):** (15.5, 140.5) **Copollutant:** NO_2 SO_2 O_3	**Increment:** 10 $\mu g/m^3$ **% Excess Risk (Lower CI, Upper CI)** **lag:** Main Analysis Non-accidental Smokers: ≥ 301: .80% (0.35, 3.26) 0 1.77% (0.46, 3.11) 2 ≥ 65: 3.20% (1.36, 5.07) 0 2.42% (0.73, 4.13) 2 Never-smokers ≥ 30: -0.37% (-2.23, 1.52) 0 -0.03% (-1.72, 1.66) 2 ≥ 65P -0.70% (-2.81, 1.46) 0 -0.13% (-2.04, 1.80) 2 Cardiorespiratory Smokers ≥ 30: 1.43% (-0.86, 3.78) 0 2.32% (0.24, 4.44) 2 ≥ 65: 2.98% (0.47, 5.55) 0 2.61% (0.31, 4.95) 2 Never-smokers ≥ 30: 0.02% (-2.75, 2.87) 0 -0.79% (-3.33, 1.82)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			2
			≥ 65: 0.25% (-2.62, 3.19)
			0
			-0.66% (-3.29, 2.04)
			2
			Sensitivity Analysis
			Poisson Regression
			Non-accidental
			≥ 30: 1.81% (0.21, 3.44)
			0
			1.93% (0.32, 3.56)
			2
			1.99% (0.14, 3.87)
			0-3
			≥ 65: 2.31% (0.37, 4.29)
			0
			2.16% (0.20, 4.15)
			2
			2.57% (0.30, 4.89)
			0-3
			Cardiorespiratory
			≥ 30: 1.04% (-1.45, 3.59)
			0
			2.18% (-0.35, 4.77)
			2
			1.66% (-1.24, 4.64)
			0-3
			≥ 65: 1.69% (-0.93, 4.37)
			0
			2.44% (-0.23, 5.18)
			2
			2.30% (-0.80, 5.50)
			0-3
			Case-only: Logistic Regression
			Non-accidental
			≥ 30: 1.79% (0.21, 3.37)
			0
			1.94% (0.33, 3.56)
			2
			≥ 65: 2.30% (0.42, 4.17)
			0
			2.16% (0.26, 4.07)
			2
			Cardiorespiratory
			≥ 30: 1.01% (-1.37, 3.40)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			0
			2.16% (-0.28, 4.61)
			2
			≥ 65: 1.65% (-0.96, 4.27)
			0
			2.42% (-0.27, 5.12)
			2
			Case-crossover
			Non-accidental
			≥ 30: 2.54% (0.35, 4.78)
			0
			1.35% (-0.81, 3.56)
			2
			≥ 65: 3.96% (1.37, 6.63)
			0
			2.20% (-0.35, 4.81)
			2
			Cardiorespiratory
			≥ 30: 0.48% (-2.74, 3.80)
			0
			3.24% (-0.03, 6.61)
			2
			≥ 65: 2.17% (-1.40, 5.86)
			0
			3.43% (-0.13, 7.13)
			2
Reference: Wong et al. (2007, 093278)	**Outcome:** Mortality:	**Pollutant:** PM_{10}	**Increment:** 10 $\mu g/m^3$
Period of Study: 1/1998–12/1998	Total (non-accidental) (< 800)	**Averaging Time:** 24-h avg	**% Excess Risk (Lower CI, Upper CI)**
Location: Hong Kong, China	Cardiorespiratory (390-519)	**Mean (SD):**	**lag:**
	Study Design: Main analysis: Time-series	48.1 (24.3)	Non-accidental
	Sensitivity analysis: Case-only	**Range (Min, Max):**	Exercise
	Statistical Analyses: Main analysis: Poisson GAM, natural cubic spline	(15.5, 140.5)	≥ 30: 0.13% (-1.16, 1.44)
	Sensitivity analysis: Logistic regression	**Copollutant:**	1
	Age Groups: ≥ 30	NO_2	≥ 65: 0.24% (-1.16, 1.67)
	≥ 65	SO_2	1
		O_3	Never-exercise
			≥ 30: 1.04% (0.07, 2.02)
			1
			≥ 65: 1.26% (0.27, 2.27)
			1
			Cardio-respiratory
			Exercise
			≥ 30: 0.46% (-1.43, 2.39)
			1
			≥ 65: 0.30% (-1.65, 2.29)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			1
			Never-exercise
			≥ 30: 0.97% (-0.36, 2.32)
			1
			≥ 65: 0.98% (-0.45, 2.43)
			1
			Difference in % Excess Risk (Exercise vs. Never-Exercise)
			Non-accidental
			Poisson Regression
			≥ 30: -2.86% (-4.03 to -1.67)
			1
			≥ 65: -3.06% (-4.37 to -1.74)
			1
			Case-only
			≥ 30: -2.91% (-4.04 to -1.77)
			1
			≥ 65: -3.12% (-4.38 to -1.84)
			1
			Cardiorespiratory
			Poisson regression
			≥ 30: -2.55% (-4.32 to -0.75)
			1
			≥ 65: -2.64% (-4.48 to -0.76)
			1
			Case-only
			≥ 30: -2.63% (-4.32 to -0.92)
			1
			≥ 65: -2.73% (-4.50 to -0.92)
			1
			Adjusted Case-only
			Non-accidental
			Sex
			≥ 30: -2.88% (-1.73 to -4.01)
			1
			≥ 65: -3.09% (-1.82 to -4.35)
			1
			Education
			≥ 30: -2.94% (-1.80 to -4.07)
			1
			≥ 65: -3.18% (-1.90 to -4.44)
			1
			Job
			≥ 30: -2.88% (-1.74 to -4.02)
			1

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			≥ 65: -3.11% (-1.83 to -4.37)
			1
			Smoking
			≥ 30: -2.82% (-1.66 to -3.96)
			1
			≥ 65: -2.97% (-1.68 to -4.25)
			1
			Illness time
			≥ 30: -2.94% (-1.80 to -4.07)
			1
			≥ 65: -3.16% (-1.88 to -4.42)
			1
			Cardiorespiratory
			Sex
			≥ 30: -2.61% (-0.89 to -4.29)
			1
			≥ 65: -2.71% (-0.90 to -4.48)
			1
			Education
			≥ 30: -2.58% (-0.85 to -4.27)
			1
			≥ 65: -2.77% (-0.95 to -4.54)
			1
			Job
			≥ 30: -2.68% (-0.96 to -4.37)
			1
			≥ 65: -2.68% (-0.88 to -4.46)
			1
			Smoking
			≥ 30: -2.46% (-0.73 to -4.17)
			1
			≥ 65: -2.50% (-0.68 to -4.29)
			1
			Illness Time
			≥ 30: -2.63% (-0.91 to -4.32)
			1
			≥ 65: -2.73% (-0.92 to -4.51)
			1
			Case-only by Exercise Group (Never as Reference)
			Non-accidental
			≥ 30
			Low: -3.34% (-5.77 to -0.85)
			1
			Moderate: -6.32% (-8.55 to -4.03)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			1 High: -1.74% (-3.06 to -0.40) 1 ≥ 65 Low: -3.79% (-6.67 to -0.82) 1 Moderate: -7.78% (-10.39 to -5.10) 1 High: -1.77% (-3.21 to -0.31) 1 Cardiorespiratory ≥ 30 Low: -3.95% (-7.77, 0.04) 1 Moderate: -8.50% (-11.84 to -5.02) 1 High: -0.62% (-2.58, 1.38) 1 ≥ 65 Low: -3.97% (-8.17, 0.43) 1 Moderate: -9.42% (-13.00 to -5.69) 1 High: -0.68% (-2.71, 1.38) 1
Reference: Wong et al. (2002, 025436) **Period of Study:** 1995-1998 **Location:** Hong Kong, China	**Outcome:** Mortality: Respiratory (461-519) COPD (490-496) Pneumonia & Influenza (480-487) Cardiovascular (390-459) IHD (410-414) Cerebrovascular (430-438) **Study Design:** Time-series **Statistical Analyses:** Poisson **Age Groups:** ≥ 30 ≥ 65	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** 51.53 (24.79) **Range (Min, Max):** (14.05, 163.79) **Copollutant (correlation):** NO_2: r = 0.780 SO_2: r = 0.344 O_3: r = 0.538	**Increment:** 10 $\mu g/m^3$ **Relative Risk (Lower CI, Upper CI)** **lag:** Respiratory 1.008 (1.001 to 1.014) 1 COPD 1.017 (1.002, 1.033) 0-3 Pneumonia & Influenza 1.007 (0.999, 1.015) 2 Cardiovascular 1.003 (0.998, 1.016) 2 IHD 1.013 (1.001, 1.025) 0-3 Cerebrovascular 1.007 (0.998, 1.016)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			2
			Respiratory
			$PM_{10}+SO_2+O_3+NO_2$: 1.005 (0.992, 1.010)
			1
			COPD
			$PM_{10}+SO_2+O_3+NO_2$: 0.991 (0.968, 1.015)
			0-3
			$PM_{10}+O_3+NO_2$: 0.993 (0.970, 1.016)
			0-3
			Pneumonia & Influenza
			$PM_{10}+SO_2+O_3+NO_2$: 1.002 (0.991, 1.013)
			2
			IHD
			0.994 (0.978, 1.009)
			0-3
Reference: Wong et al. (2008, 157152)	**Outcome (ICD10):** Mortality:	**Pollutant:** PM_{10}	**Increment:** 10 $\mu g/m^3$
Period of Study: Bangkok: 1999–2003	Natural causes (A00-R99)	**Averaging Time:** 24-h avg	**% Excess Risk (Lower CI, Upper CI)**
Hong Kong: 1996–2002	Cardiovascular (I00-I99)	**Mean (SD):**	**lag:**
Shanghai & Wuhan: 2001–2004	Respiratory (J00-J98)	Bangkok: 52.0	Random Effects (4 cities)
Location: Bangkok, Thailand	**Study Design:** Time-series	Hong Kong: 51.6	Natural causes: 0.55% (0.26, 0.85)
Hong Kong, Shanghai, and Wuhan, China	**Statistical Analyses:** Poisson GLM, natural splines	Shanghai: 102.0	0-1
	Age Groups: All ages	Wuhan: 141.8	Cardiovascular: 0.58% (0.22, 0.93)
	≥ 65	**Range (Min, Max):**	0-1
	≥ 75	Bangkok: (21.3, 169.2)	Respiratory: 0.62% (0.22, 1.02)
		Hong Kong: (13.7, 189.0)	0-1
		Shanghai: (14.0, 566.8)	Random Effects (3 Chinese cities)
		Wuhan: (24.8, 477.8)	Natural causes: 0.37% (0.21, 0.54)
		Copollutant:	0-1
		NO_2	Cardiovascular: 0.44% (0.19, 0.68)
		SO_2	0-1
		O_3	Respiratory: 0.60% (0.16, 1.04)
			0-1
			Sensitivity Analysis
			Random Effects (4 cities)
			Omit $PM_{10} > 95$th: 0.53% (0.27, 0.78)
			0-1
			Omit $PM_{10} > 75$th: 0.53% (0.29, 0.78)
			0-1
			Omit $PM_{10} > 180$ $\mu g/m^3$: 0.65% (0.24, 1.06)
			0-1
			Omit stations with high traffic source: 0.55% (0.26, 0.85)
			0-1

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			Warm season-dichotomous variables: 0.86% (0.11, 1.60) 0-1 Add temperature at lag 1-2 days: 0.51% (0.23, 0.79) 0-1 Add temperature at lag 3-7 days: 0.35% (0.14, 0.57) 0-1 Daily PM_{10} defined by centering: 0.54% (0.26, 0.82) 0-1 Natural spline with (8, 4, 4f: 0.54% (0.26, 0.81) 0-1 Penalized spline: 0.52% (0.26, 0.77) 0-1 Random Effects (3 Chinese cities) Omit $PM_{10} > 95$th: 0.47% (0.21, 0.73) 0-1 Omit $PM_{10} > 75$th: 0.55% (0.24, 0.85) 0-1 Omit $PM_{10} > 180$ $\mu g/m^3$: 0.46% (0.15, 0.76) 0-1 Omit stations with high traffic source: 0.38% (0.20, 0.57) 0-1 Warm season-dichotomous variables: 0.43% (0.10, 0.76) 0-1 Add temperature at lag 1-2 days: 0.36% (0.18, 0.53) 0-1 Add temperature at lag 3-7 days: 0.25% (0.10, 0.40) 0-1 Daily PM_{10} defined by centering: 0.37% (0.21, 0.53) 0-1 Natural spline with (8, 4, 4f: 0.36% (0.23, 0.49) 0-1 Penalized spline: 0.34% (0.23, 0.45) 0-1
Reference: Wong et al. (2008, 157151) **Period of Study:** 1/1996–12/2002 **Location:** Hong Kong	**Outcome (ICD10):** Mortality: Non-accidental (A00-T99 Z00-Z99) Cardiovascular (I00-I99) Respiratory (J00-J98)	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** 51.6 (25.3) **Range (Min, Max):**	**Increment:** 10 $\mu g/m^3$ **% Excess Risk (Lower CI, Upper CI)** **lag:** Non-accidental: Low SDI

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
	Study Design: Time-series	(13.5, 188.5)	0.37 (–0.10, 0.84)
	Statistical Analyses: Poisson GLM, natural splines	**Copollutant:**	0
		NO_2	0.40 (–0.04, 0.84)
	Age Groups: All ages	SO_2	1
		O_3	0.14 (–0.28, 0.57)
			2
			–0.12 (–0.55, 0.30)
			3
			–0.14 (–0.56, 0.28)
			4
			Middle SDI
			0.70 (0.34, 1.07)
			0
			0.48 (0.14, 0.82)
			1
			0.35 (0.02, 0.68)
			2
			0.18 (–0.14, 0.51)
			3
			0.17 (–0.16, 0.50)
			4
			High SDI
			0.22 (–0.29, 0.73)
			0
			0.46 (–0.01, 0.94)
			1
			0.29 (–0.17, 0.75)
			2
			–0.05 (–0.51, 0.40)
			3
			–0.06 (–0.51, 0.40)
			4
			All areas
			0.45 (0.19, 0.72)
			0
			0.40 (0.15, 0.64)
			1
			0.22 (–0.02, 0.45)
			2
			0.00 (–0.24, 0.23)
			3
			0.03 (–0.20, 0.26)
			4
			Cardiovascular:
			Low SDI

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			0.14 (–0.77, 1.06)
			0
			0.64 (–0.21, 1.49)
			1
			0.24 (–0.58, 1.07)
			2
			–0.27 (–1.09, 0.55)
			3
			0.01 (–0.80, 0.83)
			4
			Middle SDI
			0.66 (0.00, 1.34)
			0
			0.49 (–0.13, 1.12)
			1
			0.80 (0.20, 1.40)
			2
			0.65 (0.06, 1.25)
			3
			0.52 (–0.07, 1.12)
			4
			High SDI
			0.83 (–0.08, 1.75)
			0
			0.89 (0.04, 1.75)
			1
			0.12 (–0.70, 0.95)
			2
			–0.09 (–0.91, 0.73)
			3
			0.04 (–0.77, 0.86)
			4
			All areas
			0.52 (0.05, 1.00)
			0
			0.58 (0.14, 1.03)
			1
			0.43 (0.00, 0.86)
			2
			0.14 (–0.28, 0.57)
			3
			0.23 (–0.20, 0.65)
			4
			Respiratory:
			Low SDI

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			0 0.69 (–0.44, 1.82)
			0
			1 0.55 (–0.50, 1.61)
			1
			2 0.36 (–0.66, 1.39)
			2
			3 –0.24 (–1.25, 0.78)
			3
			4 –0.17 (–1.17, 0.85)
			4
			Middle SDI
			0.31 (–0.50, 1.13)
			0
			0.77 (0.01, 1.53)
			1
			0.85 (0.12, 1.59)
			2
			0.66 (–0.07, 1.39)
			3
			0.69 (–0.03, 1.42)
			4
			High SDI
			0.27 (–0.85, 1.40)
			0
			0.72 (–0.32, 1.78)
			1
			1.46 (0.45, 2.47)
			2
			0.70 (–0.30, 1.71)
			3
			0.48 (–0.52, 1.48)
			4
			All areas
			0.39 (–0.20, 0.99)
			0
			0.70 (0.15, 1.26)
			1
			0.89 (0.36, 1.42)
			2
			0.45 (–0.08, 0.98)
			3
			0.43 (–0.10, 0.96)
			4
			High SDI vs. Middle SDI
			Non-accidental: 0.23 (-0.25, 0.72)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			0-1
			Cardiovascular: 0.49 (-0.40, 1.40)
			0-1
			Respiratory: 0.49 (-0.58, 1.58)
			0-1
			High SDI vs. Low SDI
			Non-accidental: 0.12 (-0.42, 0.67)
			0-1
			Cardiovascular: 0.82 (-0.20, 1.86)
			0-1
			Respiratory: -0.15 (-1.39, 1.10)
			0-1
			Trend Test
			Non-accidental: 0.04 (-0.15, 0.22)
			0-1
			Cardiovascular: 0.27 (-0.07, 0.61)
			0-1
			Respiratory: -0.04 (-0.46, 0.37)
			0-1 SDI = Social Deprivation Index. The higher the SDI the lower the SES of the individual.
Reference: Yang et al. (2004, 055603)	**Outcome:** Mortality:	**Pollutant:** PM_{10}	**Increment:** 31.43 $\mu g/m^3$
Period of Study: 1994–1998	Non-accidental (< 800)	**Averaging Time:** 24-h avg	**Odds Ratio (Lower CI, Upper CI)**
Location: Taipei, Taiwan	Circulatory (390-459)	**Mean (SD):** 51.99	**lag:**
	Respiratory (460-519)	**Range (Min, Max):** (13.71, 211.30)	Non-accidental
	Study Design: Bi-directional case-crossover	**Copollutant:**	0.995 (0.971, 1.020)
		SO_2	0
	Statistical Analyses: Conditional logistic regression	NO_2	Respiratory
	Age Groups: All ages	CO	0.986 (0.906, 1.074)
		O_3	0
			Circulatory
			0.988 (0.942, 1.035)
Reference: Zanobetti et al. (2003, 042812)	**Outcome:** Mortality:	**Pollutant:** PM_{10}	**Increment:** 10 $\mu g/m^3$
Period of Study: 1990–1997	Non-accidental (< 800)	**Averaging Time:** 24-h avg	**% Increase (Lower CI, Upper CI)**
Location: 10 European cities (APHEA2)	Circulatory (390-459)	**Mean (SD):**	**lag:**
	Respiratory (460-519)	Athens: 42.7 (12.9)	Cardiovascular
	Study Design: Time-series	Budapest: 41 (9.1)	0.69% (0.31, 1.08)
	Statistical Analyses: Poisson GAM	Lodz: 53.5 (15.5)	0-1 avg
	Age Groups: 15-64	London: 28.8 (13.7)	40-day distributed lag
	65-74	Madrid: 37.8 (17.7)	1.99% (1.44, 2.54)
	≥ 75	Paris: 22.5 (11.5)	4th degree
		Prague: 76.2 (45.7)	1.97% (1.38, 2.55)
		Rome: 58.7 (17.4)	Unrestricted
		Stockholm: 15.5 (7.9)	Respiratory
		Tel Aviv: 50.3 (57.5)	0.74% (-0.17, 1.66)
		Range (Min, Max): NR	0-1 avg

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
		Copollutant (correlation): NR	40-day distributed lag
			4.21% (1.70, 6.79)
			4th degree
			4.20% (1.08, 7.42)
			Unrestricted
			Unrestricted distributed lags
			Cardiovascular
			1.34% (0.89, 1.79)
			20
			1.72% (1.20, 2.25)
			30
			1.97% (1.38, 2.55)
			40
			Respiratory
			1.71% (-0.65, 4.12)
			20
			2.62% (0.19, 5.11)
			30
			4.20% (1.08, 7.42)
			40
			40-day lags
			Non-accidental
			15-64
			-0.25% (-0.87, 0.36)
			4th degree
			-0.01 (-0.76, 0.75)
			Unrestricted
			65-74
			0.78% (0.23, 1.33)
			4th degree
			0.74% (0.02, 1.45)
			Unrestricted
			≥ 75
			1.84% (0.92, 2.78)
			4th degree
			1.94% (1.07, 2.81)
			Unrestricted
			Cardiovascular
			65-74
			2.06% (1.05, 3.09)
			4th degree
			1.62 (0.54, 2.70)
			Unrestricted
			≥ 75
			2.35% (1.42, 3.29)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			4th degree
			2.52% (1.57, 3.48)
			Unrestricted
			Respiratory
			≥ 75
			4.57% (1.25, 7.99)
			4th degree
			4.52% (0.89, 8.28)
			Unrestricted
Reference: Zeka et al. (2005, 088068)	**Outcome (ICD10):** Mortality:	**Pollutant:** PM_{10}	**Increment:** 10 $\mu g/m^3$
Period of Study: 1/1989–12/2000	All-cause (non-accidental) (V01-Y98)	**Averaging Time:** 24-h avg	**% Increase (Lower CI, Upper CI)**
Location: 20 U.S. cities	Heart Disease (I01-I51)	**Mean (SD):**	**lag:**
	IHD (I20-I25)	Birmingham: 31.9 (18.0) $\mu g/m^3$	Single-lag model
	Myocardial infarction (I21, I22)	Boulder: 22.1 (11.3)	All-Cause (non-accidental)
	Dysrhythmias (I46-I49)	Caton: 26.6 (11.5)	0.20% (0.08, 0.32)
	Heart failure (I50)	Chicago: 33.7 (16.4)	0
	Stroke (I60-I69)	Cincinnati: 31.4 (13.9)	0.35% (0.21, 0.49)
	Respiratory (J00-J99)	Cleveland: 37.5 (18.7)	1
	Pneumonia (J12-J18)	Colorado Springs: 24.0 (13.2)	0.24% (0.14, 0.34)
	COPD (J40-J44, J47)	Columbus: 28.5 (12.5)	2
	Study Design: Time-stratified case-crossover	Denver: 28.5 (12.8)	Respiratory
		Detroit: 32.1 (17.7)	0.34% (-0.07, 0.75)
	Statistical Analyses: Conditional logistic regression	Honolulu: 15.9 (6.8)	0
		Minneapolis: 24.7 (12.3)	0.52% (0.15, 0.89)
	Age Groups: All ages	Nashville: 30.1 (12.1)	1
		New Haven: 25.4 (14.4)	0.51% (0.16, 0.86)
		Pittsburgh: 30.2 (18.5)	2
		Provo: 33.7 (22.2)	COPD
		Seattle: 26.4 (14.7)	-0.06% (-0.63, 0.51)
		Salt lake City: 35.0 (20.8) μ	0
		Terra Haute: 29.2 (14.6) μ	0.43% (-0.14, 1.00)
		Youngstown: 30.8 (13.9)	1
		Range (Min, Max): NR	0.39% (-0.16, 0.94)
		Copollutant (correlation): NR	2
			Pneumonia
			0.50% (0.09, 1.09)
			0
			0.59% (-0.12, 1.30)
			1
			0.82% (0.25, 1.39)
			2
			Heart disease
			0.12% (-0.06, 0.30)
			0
			0.30% (0.12, 0.48)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			1
			0.37% (0.17, 0.57)
			2
			IHD
			0.19% (-0.03, 0.41)
			0
			0.41% (0.19, 0.63)
			1
			0.43% (0.10, 0.76)
			2
			Myocardial Infarction
			0.36% (-0.05, 0.77)
			0
			0.17% (-0.18, 0.52)
			1
			0.13% (-0.22, 0.48)
			2
			Heart Failure
			0.17% (-0.63, 0.97)
			0
			-0.01% (-0.81, 0.79)
			1
			0.78% (-0.004, 1.56)
			2
			Dysrhythmias
			-0.23% (-1.41, 0.95)
			0
			0.37% (-0.47, 1.21)
			1
			0.33% (-0.55, 1.21)
			2
			Stroke
			0.09% (-0.49, 0.60)
			0
			0.41% (-0.02, 0.84)
			1
			0.14% (-0.27, 0.55)
			2
			Unconstrained distributed lag model
			All-cause (non-accidental)
			0.45% (0.25, 0.65)
			0-3
			Respiratory
			0.87% (0.38, 1.36)
			0-3

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			COPD
			0.43% (-0.35, 1.21)
			0-3
			Pneumonia
			1.24% (0.46, 2.02)
			0-3
			Heart Disease
			0.50% (0.25, 0.75)
			0-3
			IHD
			0.65% (0.32, 0.98)
			Myocardial Infarction
			0.36% (-0.25, 0.97)
			0-3
			Heart Failure
			0.60% (-0.50, 1.70)
			0-3
			Dysrhythmias
			0.20% (-1.03, 1.43)
			0-3
			Stroke
			0.46% (-0.13, 1.05)
			0-3
Reference: Zeka et al. (2006, 088749)	**Outcome (ICD10):** Mortality:	**Pollutant:** PM_{10}	**Increment:** 10 $\mu g/m^3$
Period of Study: 1/1989–12/2000	All-cause (non-accidental) (V01-Y98)	**Averaging Time:** 24-h avg	**% Increase (Lower CI, Upper CI)**
Location: 20 U.S. cities	Heart Disease (I01-I51)	**Mean (SD):**	**lag: All-cause (non-accidental)**
	Myocardial infarction (I21, I22)	Birmingham: 31.9 (18.0) $\mu g/m^3$	Male: 0.46% (0.28, 0.64)
	Stroke (I60-I69)	Boulder: 22.1 (11.3)	1-2 avg
	Respiratory (J00-J99)	Caton: 26.6 (11.5)	Female: 0.37% (0.17, 0.57)
	Study Design: Time-stratified case-crossover	Chicago: 33.7 (16.4)	1-2 avg
		Cincinnati: 31.4 (13.9)	White
	Statistical Analyses: Conditional logistic regression	Cleveland: 37.5 (18.7)	0.40% (0.22, 0.58)
		Colorado Springs: 24.0 (13.2)	1-2 avg
	Age Groups:	Columbus: 28.5 (12.5)	Black: 0.37% (-0.02, 0.76)
	All ages	Denver: 28.5 (12.8)	1-2 avg
	< 65	Detroit: 32.1 (17.7)	**Age:** < 65: 0.25% (0.01, 0.49)
	65-75	Honolulu: 15.9 (6.8)	1-2 avg
	> 75	Minneapolis: 24.7 (12.3)	75: 0.23% (-0.06, 0.52)
		Nashville: 30.1 (12.1)	1-2 avg
		New Haven: 25.4 (14.4)	> 75: 0.64% (0.44, 0.84)
		Pittsburgh: 30.2 (18.5)	1-2 avg
		Provo: 33.7 (22.2)	**Educational Attainment:** Low (< 8 yrs): 0.62% (0.29, 0.95)
		Seattle: 26.4 (14.7)	1-2 avg
		Salt lake City: 35.0 (20.8)	Medium (8–12 yrs): 0.36% (0.12, 0.60)
		Terra Haute: 29.2 (14.6)	

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
		Youngstown: 30.8 (13.9)	1-2 avg
		Range (Min, Max): NR	High (> 12 yrs): 0.27% (-0.004, 0.54)
		Copollutant (correlation): NR	1-2 avg
			Location of Death: In hospital: 0.22% (0.04, 0.40)
			1-2 avg
			Out of hospital: 0.71% (0.51, 0.91)
			1-2 avg
			Season: Winter: 0.28% (0.04, 0.52)
			1-2 avg
			Summer: 0.19% (-0.22, 0.60)
			1-2 avg
			Transition (spring/fall): 0.49% (0.25, 0.73)
			1-2 avg
			Respiratory
			Male: 0.71% (0.004, 1.42)
			0-3
			Female: 1.04% (0.33, 1.75)
			0-3
			White: 0.88% (0.33, 1.43)
			0-3
			Black: 0.71% (-0.56, 1.98)
			0-3
			Age: < 65: 0.94% (-0.31, 2.19)
			0-3
			65-75: 0.87% (-0.25, 1.99)
			0-3
			> 75: 0.88% (0.17, 1.59)
			0-3
			Educational Attainment: Low (< 8 yrs): 0.82% (-0.32, 1.96)
			0-3
			Medium (8–12 yrs): 0.88% (0.12, 1.64)
			0-3
			High (> 12 yrs): 0.88% (-0.04, 1.80)
			0-3
			Location of Death: In hospital: 0.78% (0.17, 1.39)
			0-3
			Out of hospital: 1.09% (0.25, 1.93)
			0-3
			Season: Winter: -0.007% (-0.87, 0.86)
			0-3
			Summer: 0.69% (-0.68, 2.06)
			0-3
			Transition (spring/fall): 1.57% (0.86,

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			2.28)
			0-3
			Heart Disease
			Male: 0.54% (0.23, 0.85)
			2
			Female: 0.46% (0.15, 0.77)
			2
			White
			0.50% (0.25, 0.75)
			2
			Black: 0.64% (0.13, 1.15)
			2
			Age: < 65: 0.04% (-0.45, 0.53)
			2
			65-75: 0.60% (0.13, 1.07)
			2
			> 75: 0.65% (0.30, 1.00)
			2
			Educational Attainment: Low (< 8 yrs): 0.72% (0.23, 1.21)
			2
			Medium (8–12 yrs): 0.38% (0.07, 0.69)
			2
			High (> 12 yrs): 0.54% (0.13, 0.95)
			2
			Location of Death: In hospital: 0.15% (-0.14, 0.44)
			2
			Out of hospital: 0.93% (0.60, 1.26)
			2
			Season: Winter: 0.41% (-0.002, 0.82)
			2
			Summer: 0.52 (0.03, 1.01)
			2
			Transition (spring/fall): 0.56% (0.13, 0.99)
			2
			Myocardial Infarction
			Male: 0.21% (-0.40, 0.82)
			0
			Female: 0.59% (0.08, 1.10)
			0
			White
			0.24% (-0.27, 0.75)
			0
			Black: 0.99% (0.05, 1.93)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			0
			< 65: 0.12% (-0.76, 1.00)
			0
			65-75: 0.92% (0.21, 1.63)
			0
			> 75: 0.16% (-0.58, 0.90)
			0
			Educational Attainment: Low (< 8 yrs): 0.33% (-0.83, 1.49)
			0
			Medium (8–12 yrs): 0.79% (0.28, 1.30)
			0
			High (> 12 yrs): -0.13% (-0.82, 0.56)
			0
			Location of Death: In hospital: 0.34% (-0.11, 0.79)
			0
			Out of hospital: 0.48% (-0.23, 1.19)
			0
			Season: Winter: 0.32% (-0.37, 1.01)
			0
			Summer: 0.30% (-0.82, 1.42)
			0
			Transition (spring/fall): 0.38% -0.31, 1.07)
			0
			Stroke
			Male: 0.11% (-0.58, 0.80)
			1
			Female: 0.59% (-0.04, 1.22)
			1
			White
			0.48% (0.01, 0.95)
			1
			Black: 0.13% (-0.87, 1.13)
			1
			Age: < 65: 0.09% (-1.09, 1.27)
			1
			65-75: -0.46% (-1.42, 0.50)
			1
			> 75: 0.80% (0.27, 1.33)
			1
			Educational Attainment: Low (< 8 yrs): 0.07% (-1.44, 1.58)
			1
			Medium (8–12 yrs): 0.29% (-0.32, 0.90)
			1

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			High (>12 yrs): 0.52% (-0.28, 1.32)
			1
			Location of Death: In hospital: 0.06% (-0.49, 0.61)
			1
			Out of hospital: 0.87% (0.05, 1.69)
			1
			Season: Winter: -0.09% (-0.93, 0.75)
			1
			Summer: 0.67% (-0.31, 1.65)
			1
			Transition (spring/fall): 0.51% (-0.20, 1.22)
			1
			Contributing causes of disease: All-cause
			Secondary pneumonia present: 0.67% (0.16, 1.18)
			1-2 avg
			Secondary pneumonia absent: 0.34% (0.16, 0.52)
			1-2 avg
			Secondary heart failure present: 0.42% (0.01, 0.83)
			1-2 avg
			Secondary heart failure absent: 0.37% (0.19, 0.55)
			1-2 avg
			Secondary stroke present: 0.85% (0.30, 1.40)
			1-2 avg
			Secondary stroke absent: 0.32% (0.14, 0.50)
			1-2 avg
			Diabetes present: 0.57% (0.02, 1.12)
			1-2 avg
			Diabetes absent: 0.34% (0.14, 0.54)
			1-2 avg
			Respiratory
			Secondary pneumonia present: 1.28% (-0.33, 2.89)
			0-3
			Secondary pneumonia absent: 0.78% (0.15, 1.41)
			0-3
			Secondary heart failure present: 1.48% (0.07, 2.89)
			0-3
			Secondary heart failure absent: 0.79% (0.26, 1.32)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			0-3
			Secondary stroke present: 1.95% (-0.11, 4.01)
			0-3
			Secondary stroke absent: 0.80% (0.29, 1.31)
			0-3
			Diabetes present: 1.96% (-0.22, 4.14)
			0-3
			Diabetes absent: 0.82% (0.31, 1.33)
			0-3
			Heart Disease
			Secondary pneumonia present: 0.66% (-0.63, 1.95)
			2
			Secondary pneumonia absent: 0.49% (0.27, 0.71)
			2
			Secondary stroke present: 0.73% (-0.05, 1.51)
			2
			Secondary stroke absent: 0.48% (0.24, 0.72)
			2
			Diabetes present: 0.34% (-0.42, 1.10)
			2
			Diabetes absent: 0.52% (0.28, 0.76)
			2
			Myocardial Infarction
			Secondary pneumonia present: 1.54% (-1.05, 4.13)
			0
			Secondary pneumonia absent: 0.42% (0.05, 0.79)
			0
			Secondary stroke present: 0.50% (-1.38, 2.38)
			0
			Secondary stroke absent: 0.36% (-0.05, 0.77)
			0
			Diabetes present: 0.70% (-0.38, 1.78)
			0
			Diabetes absent: 0.41% (0.04, 0.78)
			0
			Stroke
			Secondary pneumonia present: 1.74% (0.35, 3.13)
			1
			Secondary pneumonia absent: 0.29%

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			(-0.16, 0.74) 1 Secondary heart failure present: 1.01% (-0.77, 1.79) 1 Secondary heart failure absent: 0.38% (-0.05, 0.81) 1 Diabetes present: 1.02% (-0.53, 2.57) 1 Diabetes absent: 0.37% (-0.08, 0.82) 1

[1]All units expressed in $\mu g/m^3$ unless otherwise specified.

Table E-17. Short-term exposure – mortality - $PM_{10-2.5}$.

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Burnett et al. (2004, 086247) **Period of Study:** 1981–1999 **Location:** 12 Canadian cities	**Outcome:** Mortality: Non-accidental (< 800) **Study Design:** Time-series **Statistical Analyses:** 1. Poisson, natural splines 2. Random effects regression model **Age Groups:** All ages	**Pollutant:** P10-2.5 **Averaging Time:** 24-h avg **Mean (SD):** 11.4 **Range (Min, Max):** NR **Copollutant:** NO_2 O_3 SO_2 CO PM_{10} $PM_{2.5}$ **Note:** PM_{10} measurement calculated as the sum of $PM_{2.5}$ and $PM_{10-2.5}$ measurements.	**Increment:** 10 $\mu g/m^3$ **% Increase (Lower CI, Upper CI)** **lag:** 1981–1999 $PM_{10-2.5}$: 0.31% (-0.66, 1.33) 1 $PM_{10-2.5}+NO_2$: 0.65% (-0.23, 1.59) 1
Reference: Kan et al. (2007, 091267) **Period of Study:** 3/2004–12/2005 **Location:** Shanghai, China	**Outcome (ICD10):** Mortality: Total (non-accidental) (A00-R99) Cardiovascular (I00-I99) Respiratory (J00-J98) **Study Design:** Time-series **Statistical Analyses:** Poisson GAM, penalized splines **Age Groups:** All ages	**Pollutant:** $PM_{10-2.5}$ **Averaging Time:** 24-h avg **Mean (SD):** 56.4 (1.34) **Range (Min, Max):** (8.3, 235.0) **Copollutant (correlation):** PM_{10}: r = 0.88 $PM_{2.5}$: r = 0.48 O_3: r = 0.07	**Increment:** 10 $\mu g/m^3$ **% Increase (Lower CI, Upper CI)** **lag:** Total: 0.12% (-0.13, 0.36) 0-1 Cardiovascular: 0.34% (-0.05, 0.73) 0-1 Respiratory: 0.40% (-0.34, 1.13) 0-1

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Kettunen et al. (2007, 091242) **Period of Study:** 1998–2004 **Location:** Helsinki, Finland	**Outcome (ICD10):** Mortality: Stroke (I60-I61, I63-I64) **Study Design:** Time-series **Statistical Analyses:** Poisson GAM, penalized thin-plate splines **Age Groups:** ≥ 65	**Pollutant:** $PM_{10-2.5}$ **Averaging Time:** 24-h avg **Median (SD) unit:** Cold Season: 6.7 Warm Season: 8.4 **Range (Min, Max):** Cold Season: (0.0, 101.4) Warm Season: (0.0, 42.0) **Copollutant:** O_3, CO, NO_2 PM_{10} $PM_{2.5}$ UFP	**Increment:** Cold Season: 8.3 $\mu g/m^3$ Warm Season: 5.7 $\mu g/m^3$ **% Increase (Lower CI, Upper CI)** **lag:** Cold Season: -1.04% (-6.63, 4.89) 0 -2.49% (-7.57, 2.88) 1. -4.93% (-9.99, 0.41) 2 -4.33% (-9.32, 0.93) 3 Warm Season: 7.05% (-1.88, 16.80) 0 4.38% (-4.26, 13.81) 1: -1.19% (-9.45, 7.84) 2 1.42% (-6.79, 10.34) 3
Reference: Klemm et al. (2004, 056585) **Period of Study:** 8/1998–7/2000 **Location:** Fulton and DeKalb counties, Georgia (ARIES)	**Outcome:** Mortality: Non-accidental (< 800) Cardiovascular (390-459) Respiratory (460-519) Cancer (140-239) **Study Design:** Time-series **Statistical Analyses:** Poisson GLM, natural cubic splines **Age Groups:** < 65, ≥ 65	**Pollutant:** $PM_{10-2.5}$ **Averaging Time:** 24-h avg **Mean (SD):** 9.69 (3.94) **Range (Min, Max):** (1.71, 25.17) **Copollutant:** $PM_{2.5}$ O_3 NO_2 CO SO_2 Acid EC OC SO_4 Oxygenated Hydrocarbons Nonmethane hydrocarbons NO_3	**Increment:** NR **β (SE)** **lag:** Quarterly Knots: 0.00433 (0.00333) 0-1 Monthly Knots: 0.00617 (0.00360) 0-1 Biweekly Knots: 0.00516 (0.00381) 0-1

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Perez et al. (2008, 156020) **Period of Study:** 3/27/2003-12/31/2005 **Location:** Barcelona, Spain	**Outcome:** respiratory mortality **Study Design:** cohort **Covariates:** temperature, humidity **Statistical Analysis:** autoregressive Poisson regression models **Statistical Package:** NR **Age Groups:** All deaths	**Pollutant:** $PM_{10-2.5}$ **Averaging Time:** 24 h **Mean (SD) Unit:** 14.0 (9.5) $\mu g/m^3$ **Range (Min, Max):** 0.1, 93.1 **Copollutant:** $PM_{2.5-1}$, PM_1	**Increment:** 10 $\mu g/m^3$ **Odds Ratio (95%CI)** **Lag** Single Pollutant Model Avg L0-1: 1.000 (0.944-1.060), p = 0.991 L1: 1.002 (0.955-1.052), p = 0.931 L2: 1.070 (1.023-1.118), p = 0.003 Multi-pollutant Model Avg L0-1: 1.002 (0.937-1.071), p = 0.958 L1: 0.998 (0.943-1.056), p = 0.0.936 L2: 1.033 (0.980-1.089), p = 0.226
Reference: Perez et al. (2008, 156020) **Period of Study:** 3/27/2003-12/31/2005 **Location:** Barcelona, Spain	**Outcome:** cardiovascular mortality **Study Design:** cohort **Covariates:** temperature, humidity **Statistical Analysis:** autoregressive Poisson regression models **Statistical Package:** NR **Age Groups:** All deaths	**Pollutant:** $PM_{10-2.5}$ **Averaging Time:** 24 h **Mean (SD) Unit:** 14.0 (9.5) $\mu g/m^3$ **Range (Min, Max):** 0.1, 93.1 **Copollutant:** $PM_{2.5-1}$, PM_1	**Increment:** 10 $\mu g/m^3$ **Odds Ratio (95%CI)** **Lag** Avg L0-1: 1.054 (1.019-1.089), p = 0.002 L1: 1.059 (1.031-1.072), p = 0.000 L2: 1.044 (1.017-1.072), p = 0.001 Multi-pollutant Model Avg L0-1: 1.053 (1.013-1.094), p = 0.009 L1: 1.059 (1.026-1.094), p = 0.001 L2: 1.044 (1.012-1.078), p = 0.007
Reference: Perez et al. (2008, 156020) **Period of Study:** 3/27/2003-12/31/2005 **Location:** Barcelona, Spain	**Outcome:** cerebrovascular mortality **Study Design:** cohort **Covariates:** temperature, humidity **Statistical Analysis:** autoregressive Poisson regression models **Statistical Package:** NR **Age Groups:** All deaths	**Pollutant:** $PM_{10-2.5}$ **Averaging Time:** 24 h **Mean (SD) Unit:** 14.0 (9.5) $\mu g/m^3$ **Range (Min, Max):** 0.1, 93.1 **Copollutant:** $PM_{2.5-1}$, PM_1	**Increment:** 10 $\mu g/m^3$ **Odds Ratio (95%CI)** **Lag** Avg L0-1: 1.087 (1.018-1.161), p = 0.013 L1: 1.086 (1.030-1.145), p = 0.002 L2: 1.051 (0.997-1.108), p = 0.064 Multi-pollutant Model Avg L0-1: 1.103 (1.022-1.191), p = 0.011 L1: 1.098 (1.030-1.171), p = 0.004 L2: 1.076 (1.010-1.146), p = 0.023
Reference: Slaughter et al. (2005, 073854) **Period of Study:** 1/1995–12/1999 **Location:** Spokane, Washington	**Outcome:** Mortality: Non-accidental (< 800) **Study Design:** Time-series **Statistical Analyses:** Poisson GLM, natural splines **Age Groups:** All ages	**Pollutant:** PM_{10}-2.5 **Averaging Time:** 24-h avg **Mean (SD) unit:** NR **Range (9th, 95th):** NR **Copollutant (correlation):** PM1: r = 0.19 $PM_{2.5}$: r = 0.31 PM_{10}: r = 0.94 CO: r = 0.32	This study does not present quantitative results for $PM_{10-2.5}$.

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Stieb et al. (2002, 025205)	**Outcome:** Mortality: All-cause (non-accidental)	**Pollutant:** $PM_{10-2.5}$	**Increment:** 13.0 $\mu g/m^3$
Period of Study: Publication dates of studies: 1985–12/2000 Mortality series: 1958–1999	**Study Design:** Meta-analysis	**Averaging Time:** NR	**% Increase (Lower CI, Upper CI)**
Location: 40 cities (11 Canadian cities, 19 U.S. cities, Santiago, Amsterdam, Erfurt, 7 Korean cities)	**Statistical Analyses:** Random effects model	**Mean (SD):** NR	**lag:**
	Age Groups: All ages	**Range (Min, Max):** NR	Single-pollutant models: 10 studies
		Copollutant: Varied between studies: $PM_{2.5}$, O_3, SO_2, NO_2, CO	$PM_{10-2.5}$: 1.2% (0.5, 1.9)
			Multipollutant models: 6 studies
			$PM_{10-2.5}$: 0.9% (-0.3, 2.0)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Villeneuve et al. (2003, 055051)	**Outcome:** Mortality:	**Pollutant:** $PM_{10-2.5}$	**Increment:** 11.0 $\mu g/m^3$
Period of Study: 1986–1999	Non-accidental (< 800)	**Averaging Time:** 24-h avg	**% Increase (Lower CI, Upper CI)**
Location: Vancouver, Canada	Cardiovascular (401-440)	**Mean (SD):**	**lag:**
	Respiratory (460-519)	Daily: 6.1	Non-accidental
	Cancer (140-239)	Every 6th Day	1.4% (-2.5, 5.4)
	Study Design: Time-series	8.3	0-2 avg
	Statistical Analyses: Poisson, natural splines	**Range (Min, Max):**	1.0% (-1.9, 4.0)
	Age Groups: ≥ 65	Daily: (0.0, 72.0)	0
		Every 6th Day: (0.7, 35.0)	-1.1% (-4.0, 1.8)
		Copollutant:	1
		$PM_{2.5}$	2.0% (-1.0, 5.1)
		PM_{10}	2
		SO_2	Cardiovascular
		CO	5.9% (-0.2, 12.4)
		NO_2	0-2 avg
		O_3	5.9% (1.1, 10.8)
			0
			1.4% (-3.3, 6.4)
			1
			2.2% (-2.0, 6.7)
			2
			Respiratory
			-1.0% (-9.8, 8.8)
			0-2 avg
			-1.5% (-9.4, 7.1)
			0
			-1.5% (-8.4, 6.0)
			1
			0.1% (-6.4, 6.9)
			2
			Cancer
			4.4% (-3.6, 13.1)
			0-2 avg
			3.1% (-2.9, 9.4)
			0
			-1.0% (-6.9, 5.3)
			1
			4.0% (-2.1, 10.4)
			2

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Wilson et al. (2007, 157149)	**Outcome:** Mortality:	**Pollutant:** $PM_{10-2.5}$	**Increment:** 10 $\mu g/m^3$
	Cardiovascular	**Averaging Time:** 24-h avg	**% Excess Risk (Lower CI, Upper CI)**
Period of Study: 1995–1997	**Study Design:** Time-series	**Mean (SD):** NR	**lag:**
Location: Phoenix, Arizona	**Statistical Analyses:** Poisson GAM, nonparametric smoothing spline	**Range (Min, Max):** NR	Central Phoenix: 2.4% (-1.2, 6.1)
		Copollutant (correlation): NR	0-5 ma
	Age Groups:		Middle Phoenix:
	> 25		3.8% (0.3, 7.5)
			0-5 ma
			3.4% (1.0, 5.8)
			1
			3.0% (0.7, 5.4)
			2
			Outer Phoenix: 1.6% (-1.9, 5.2)
			0-5 ma

[1]All units expressed in $\mu g/m^3$ unless otherwise specified.

Table E-18. Short-term exposure – mortality - $PM_{2.5}$ (including PM components/sources).

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Basu et al. (2008, 098716) **Period of Study:** 5/1999–9/2003 **Location:** 9 California counties	**Outcome (ICD10):** Mortality: Non-accidental (V01-Y98) **Study Design:** (1) Main analysis: Case-crossover (2) **Sensitivity analysis:** Time-series **Statistical Analyses:** (1) Main analysis: conditional logistic regression (2) **Sensitivity analysis:** Poisson GAM **Age Groups:** All ages	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-h avg **Mean (SE) unit:** Contra Costa: 8.6 Fresno: 7.6 Kern: 11.3 Los Angeles: 19.8 Orange: 17.0 Riverside: 28.4 Sacramento: 8.8 San Diego: 13.4 Santa Clara: 10.8 IQR (25th, 75th): Contra Costa: (5.8, 10.1) Fresno: (3.8, 9.8) Kern: (8.0, 13.5) Los Angeles: (14.7, 23.3) Orange: (11.8, 21.0) Riverside: (17.9, 36.1) Sacramento: (5.8, 10.1) San Diego: (10.3, 15.8) Santa Clara: (7.2, 13.8) **Copollutant (correlation):** PM_{10} r = 0.45 O_3 (1hr) r = 0.28 O_3 (8hr) r = 0.22 CO r = 0.45 NO_2 r = 0.43	The study does not provide results quantitatively.
Reference: Dominici et al. (2007, 097361) **Period of Study:** PM_{10}: 1987–2000. $PM_{2.5}$: 1999–2000 **Location:** 100 U.S. counties (NMMAPS)	**Outcome:** Mortality: All-cause (non-accidental) Cardiorespiratory Other-cause **Study Design:** Time-series **Statistical Analyses:** 2-stage Bayesian hierarchical model **Age Groups:** All ages	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-h avg **Mean (SD):** NR **Range (Min, Max):** NR **Copollutant (correlation):** NR	**Increment:** 10 $\mu g/m^3$ **% Increase (Lower CI, Upper CI)** **lag:** 1999-2000: All-cause: 0.29% (0.01, 0.57) 1 Cardiorespiratory: 0.38% (-0.07, 0.82) 1

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Dominici et al. (2007, 099135) **Period of Study:** 2000–2005 **Location:** 72 U.S. counties representing 69 communities	**Outcome:** Total mortality **Study Design:** Time-series **Statistical Analyses:** 2-stage Bayesian hierarchical model **Age Groups:** All ages	**Pollutant:** $PM_{2.5}$, Nickel, speciated fine PM, and Vanadium **Averaging Time:** Annual avg **Mean (SD):** NR **Range (Min, Max):** NR **Copollutant (correlation):** NR	The study does not provide results quantitatively. **Note:** The study investigated whether county-specific short-term effects of PM_{10} on mortality are modified by long-term county-specific nickel or vanadium $PM_{2.5}$ concentrations.
Reference: Franklin et al. (2007, 091257) **Period of Study:** 1997–2002 **Location:** 27 U.S. communities	**Outcome:** Mortality: All-cause (non-accidental (< 800) Cardiovascular (390-429) Respiratory (460-519) Stroke (430-438) **Study Design:** Time-stratified case-crossover **Statistical Analyses:** Conditional logistic regression **Age Groups:** All ages	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-h avg **Mean (SD):** 15.7 $\mu g/m^3$ **Range (Min, Max):** NR **Copollutant (correlation):** NR	**Increment:** 10 $\mu g/m^3$ **% Increase (Lower CI, Upper CI)** **lag:** All-cause (non-accidental): 0.67% (-0.12, 1.46) 0 1.21% (0.29, 2.14) 10.82% (0.02, 1.63) 0-1 Respiratory: 1.31% (-0.10, 2.73) 0 1.78% (0.20, 3.36) 1 1.67% (0.19, 3.16) 0-1 Cardiovascular: 0.34% (-0.61, 1.28) 0 0.94% (-0.14, 2.02) 1. 0.54% (-0.47, 1.54) 0-1 Stroke: 0.62% (-0.69, 1.94) 0 1.03% (0.02, 2.04) 1. 0.67% (-0.23, 1.57) 0-1 Age≥ 75: All cause: 1.66% (0.62, 2.70) 1 Respiratory: 1.85% (0.27, 3.44) 1 Cardiovascular: 1.29% (0.15, 2.42) 1 Stroke: 1.52% (0.37, 2.67) 1 Age < 75: All cause: 0.62% (-0.30, 1.55) 1 Respiratory: 1.53% (-0.67, 3.74) 1 Cardiovascular: 0.26% (-1.04, 1.56) 1

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			Stroke: -0.78% (-2.32, 0.76)
			1
			Male: All cause: 1.06% (0.07, 2.06)
			1
			Respiratory: 1.90% (0.14, 3.65)
			1
			Cardiovascular: 0.52% (-0.63, 1.66)
			1
			Stroke: 0.79% (-0.42, 2.02)
			1
			Female: All cause: 1.34% (0.40, 2.27)
			1
			Respiratory: 1.57% (-0.22, 3.35)
			1
			Cardiovascular: 1.30% (0.14, 2.46)
			1
			Stroke: 0.79% (-0.51, 2.09)
			1
			East: All cause: 1.95% (0.50, 3.40)
			1
			Respiratory: 2.66% (0.33, 5.00)
			1
			Cardiovascular: 1.52% (0.06, 2.98)
			1
			Stroke: 1.16% (-0.40, 2.73)
			1
			West: All cause: 0.05% (-1.80, 1.89)
			1
			Respiratory: 0.67% (-2.00, 3.34)
			1 |
			Cardiovascular: 0.11% (-2.03, 2.24)
			1 |
			Stroke: 0.94% (-0.38, 2.26)
			1
			$PM_{2.5} > 15\ \mu g/m^3$: All cause: 1.10% (-0.43, 2.64)
			1
			Respiratory: 1.42% (-0.84, 3.68)
			1
			Cardiovascular: 0.88% (-0.87, 2.62)
			1
			Stroke: 0.91% (-0.28, 2.10)
			1
			$PM_{2.5}$ 15 $\mu g/m^3$: All cause: 1.41% (-0.49, 3.30)
			1

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			Respiratory: 2.46% (-0.49, 5.42)
			1
			Cardiovascular: 1.09% (-1.15, 3.32)
			1
			Stroke: 1.36% (-0.56, 3.27)
			1
			Effect of A/C at percentile of air conditioning prevalence: 25th percentile (45% prevalence of A/C): All cause: 1.50% (0.13, 2.88)
			1
			Respiratory: 2.27% (0.27, 4.27)
			1
			Cardiovascular: 1.04% (-0.54, 2.63)
			1
			Stroke: 1.04% (-0.44, 2.53)
			1
			75th percentile (80% prevalence of A/C): All cause: 0.85% (-0.64, 2.35)
			1
			Respiratory: 1.04% (-1.29, 3.37)
			1
			Cardiovascular: 0.81% (-0.93, 2.61)
			1
			Stroke: 1.03% (-0.76, 2.83)
			1
			Effect of A/C at percentile of air conditioning prevalence in cities with summer peaking $PM_{2.5}$ concentrations: 25th percentile (45% prevalence of A/C): All cause: 1.01% (-0.30, 2.32)
			1
			Respiratory: 0.76% (-1.38, 2.90)
			1
			Cardiovascular: 0.43% (-0.86, 1.72)
			1
			Stroke: -0.18% (-2.08, 1.73)
			1
			75th percentile (77% prevalence of A/C): All cause: -0.55% (-1.95, 0.85)
			1
			Respiratory: -2.08% (-4.47, 0.31)
			1
			Cardiovascular: -1.02% (-2.44, 0.41)
			1
			Stroke: 0.69% (-1.19, 2.57)
			1

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Franklin et al. (2008, 097426) **Period of Study:** 2000–2005 **Location:** 25 U.S. communities	**Outcome (ICD10):** Mortality: Non-accidental (V01-Y98) Respiratory (J00-J99) Cardiovascular (I01-I52) Stroke (I60-J69) **Study Design:** Time-series **Statistical Analyses:** 1st stage: Poisson, cubic spline 2nd stage: Random effects meta-analysis **Age Groups:** All ages	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-h avg **Range Mean (SD):** Winter: 9.6 to 34.4 Spring: 6.7 to 27.6 summer: 7.6 to 26.0 Fall: 9.5 to 32.1 **Range (Min, Max):** NR **Copollutant:** Al, As, Br, Cr, EC, Fe, K, Mn, Na^+, Ni, NO_{3^-}, NH_4, OC, Pb, Si, SO_4^{2-}, V, Zn	**Increment:** 10 $\mu g/m^3$ **% Increase (Lower CI, Upper CI)** **lag:** Non-accidental: 0.74% (0.41, 1.07) 0-1 Cardiovascular: 0.47% (0.02, 0.92) 0-1 Respiratory: 1.01% (-0.03, 2.05) 1-2 Stroke: 0.68% (-0.21, 1.57) 0-1 Winter: 0.15% (-0.42, 0.72) 0-1 Spring: 1.88% (1.29, 2.48) 0-1 Summer: 0.99% (0.35, 1.68) 0-1 Fall: 0.19% (-0.25, 0.64) 0-1 West: 0.51% (0.10, 0.92) 0-1 East & Central: 0.92% (0.44, 1.39) 0-1 % Increase per 10 $\mu g/m^3$ increase in $PM_{2.5}$ for an IQR increase in species to $PM_{2.5}$ mass proportion Univariate analysis Al: 0.58% As: 0.55% Br: 0.38 Cr: 0.33% EC: 0.06% Fe: 0.12% K: 0.41% Mn: 0.14% Na^+: 0.20% Ni: 0.37% NO_{3^-}: -0.49% NH_4: 0.04% OC: -0.02% Pb: 0.17% Si: 0.41% SO_4^{2-}: 0.51% V: 0.30% Zn: 0.23% Multivariate (1) Al: 0.79% Ni: 0.34% SO_4^{2-}: 0.75% Multivariate (2) Al: 0.61% Ni: 0.35%

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Holloman et al. (2004, 087375) **Period of Study:** 1999–2001 **Location:** 7 North Carolina counties	Outcome (ICD10): Mortality: Cardiovascular (I00-I99) **Study Design:** Time-series **Statistical Analyses:** 3-stage Bayesian hierarchical model **Age Groups:** >16	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-h avg **Mean (SD):** NR **Range (Min, Max):** NR **Copollutant (correlation):** NR	**Increment:** 10 $\mu g/m^3$ **% Increase (Lower CI, Upper CI)** **lag:** 2.5% (-3.9 to 9.6) 0 4.0% (-3.3 to 12.2) 1 11.4% (2.8 to 19.8) 2 -1.1% (-7.5 to 5.2) 3
Reference: Hopke et al. (2006, 088390) **Period of Study:** Washington, DC: 8/1988–12/1997. Phoenix, Arizona: 3/1995–6/1998 **Location:** Washington, DC and surrounding counties Phoenix, Arizona	**Outcome:** Mortality: Total (non-accidental) Cardiovascular Cardiovascular-Respiratory **Study Design:** Source-apportionment **Statistical Analyses:** Receptor modeling **Age Groups:** All ages	**Pollutant:** Source-apportioned $PM_{2.5}$: Washington, DC: Soil Traffic Secondary Sulfate Nitrate Residual Oil Wood Smoke Sea Salt Incinerator Primary Coal Phoenix, Arizona: Crustal Traffic Vegetation and Wood Burning Secondary Sulfate Metals Sea Salt Primary Coal **Averaging Time:** 24-h avg **Mean (SD):** NR **Range (Min, Max):** NR **Copollutant (correlation):** NR	The study does not present quantitative results.

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Ito et al. (2006, 088391)	**Outcome:** Mortality:	**Pollutant:** Source-apportioned $PM_{2.5}$:	**Increment:** $PM_{2.5}$ = 28.7 $\mu g/m^3$
Period of Study: 8/1988–12/1997	Total (non-accidental)	Soil	$PM_{2.5}$ Sources 5-95th = Not reported
Location: Washington, DC and surrounding counties	Cardiovascular	Traffic	**% Increase (Lower CI, Upper CI)**
	Cardiovascular-Respiratory	Secondary Sulfate	**lag:**
	Study Design: Time-series	Nitrate	Secondary sulfate (variance-weighted mean percent excess mortality)
	Source-apportionment	Residual Oil	6.7% (1.7, 11.7)
	Statistical Analyses: Poisson GLM, natural splines	Wood Smoke	3
	Age Groups: All ages	Sea Salt	Primary coal-related $PM_{2.5}$ (mean percent excess mortality)
		Incinerator	5.0% (1.0, 9.1)
		Primary Coal	3
		Averaging Time: 24-h avg	Residual oil (mean percent excess mortality)
		Mean (SD):	2.7% (-1.1, 6.5)
		17.8 (8.7)	2
		Range (Min, Max): NR	Traffic-related $PM_{2.5}$ (mean percent excess mortality)
		Copollutant (correlation): NR	2.6% (-1.6, 6.9)
			NR
			Soil-related $PM_{2.5}$ (mean percent excess mortality)
			2.1% (-0.8, 4.9)
			NR
			$PM_{2.5}$ Sensitivity analysis:
			2 df/year: 7.9% (3.3, 12.6)
			3
			4 df/year: 8.3% (3.7, 13.1)
			3
			8 df/year: 8.3% (3.7, 13.2)
			3
			16 df/year: 8.1% (3.1, 13.2)
			3
Reference: Kan et al. (2007, 091267)	**Outcome (ICD10):** Mortality:	**Pollutant:** $PM_{2.5}$	**Increment:** 10 $\mu g/m^3$
Period of Study: 3/2004–12/2005	Total (non-accidental) (A00-R99)	**Averaging Time:** 24-h avg	**% Increase (Lower CI, Upper CI)**
Location: Shanghai, China	Cardiovascular (I00-I99)	**Mean (SD):** 52.3 (1.57)	**lag:**
	Respiratory (J00-J98)	**Range (Min, Max):** (2.0, 330.3)	Total: 0.36% (0.11, 0.61)
	Study Design: Time-series	**Copollutant (correlation):**	0-1
	Statistical Analyses: Poisson GAM, penalized splines	PM_{10}: r = 0.84	Cardiovascular: 0.41% (0.01, 0.82)
	Age Groups: All ages	$PM_{10-2.5}$: r = 0.48	0-1
		O_3: r = 0.31	Respiratory: 0.95% (0.16, 1.73)
			0-1

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Kettunen et al. (2007, 091242)	**Outcome (ICD10):** Mortality:	**Pollutant:** $PM_{2.5}$	**Increment:**
Period of Study: 1998–2004	Stroke (I60-I61, I63-I64)	**Averaging Time:** 24-h avg	Cold Season: 6.7 $\mu g/m^3$
Location: Helsinki, Finland	**Study Design:** Time-series	**Median (SD) unit:**	Warm Season: 5.7 $\mu g/m^3$
	Statistical Analyses: Poisson GAM, penalized thin-plate splines	Cold Season: 8.2	**% Increase (Lower CI, Upper CI)**
	Age Groups: ≥ 65	Warm Season: 7.8	**lag:**
		Range (Min, Max):	Cold Season
		Cold Season: (1.1, 69.5)	-0.19% (-3.77, 3.51)
		Warm Season: (1.1, 41.5)	0
		Copollutant: O_3	-0.17% (-3.73, 3.52)
		CO	1
		NO_2	0.59% (-2.95, 4.26)
		PM_{10}	2
		PM_{10}-2.5	0.46% (-3.10, 4.15)
		UFP	3
			Warm Season
			6.86% (0.37, 13.78)
			0
			7.40% (1.33, 13.84)
			1
			4.01% (-1.79, 10.14)
			2
			-1.72% (-7.38, 4.29)
			3
Reference: Klemm et al. (2004, 056585)	**Outcome:** Mortality:	**Pollutant:** $PM_{2.5}$	**Increment:** NR
Period of Study: 8/1998–7/2000	Non-accidental (< 800)	**Averaging Time:** 24-h avg	**β (SE)**
Location: Fulton and DeKalb counties, Georgia (ARIES)	Cardiovascular (390-459)	**Mean (SD):**	**lag:**
	Respiratory (460-519)	19.62 (8.32)	Quarterly Knots:
	Cancer (140-239)	**Range (Min, Max):**	$PM_{2.5}$: 0.00398 (0.00161)
	Study Design: Time-series	(5.29, 48.01)	0-1
	Statistical Analyses: Poisson GLM, natural cubic splines	**Copollutant:**	Monthly Knots:
		$PM_{10-2.5}$	$PM_{2.5}$: 0.00544 (0.00184)
	Age Groups: < 65	O_3	0-1
	≥ 65	NO_2	Biweekly Knots:
		CO	$PM_{2.5}$: 0.00369 (0.00201)
		SO_2	0-1
		Acid	
		EC	
		OC	
		SO_4	
		Oxygenated Hydrocarbons	
		Nonmethane hydrocarbons	
		NO_3	

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Lippmann et al. (2006, 091165) **Period of Study:** 2000–2003 **Location:** 60 U.S. cities (NMMAPS)	**Outcome:** Mortality: Non-accidental (< 800) **Study Design:** Time-series **Statistical Analyses:** Poisson GLM **Age Groups:** All ages	**Pollutant:** Speciated Fine PM: Al, Ar, Cr, Cu, EC, Fe, Mn, Ni, Nitrate, OC, Pb, Se, Si, Sulfate, V, Zn **Averaging Time:** Annual avg **Mean (SD):** R **Range (Min, Max):** NR	The study does not present quantitative results.
Reference: Mar et al. (2005, 087566) **Period of Study:** 1995–1997 **Location:** Phoenix, Arizona	**Outcome:** Mortality: Non-accidental (< 800) Cardiovascular (390-448) **Study Design:** Time-series **Statistical Analyses:** Poisson GLM **Age Groups:** ≥ 65	**Pollutant:** Source-apportioned $PM_{2.5}$: Soil Traffic Secondary Sulfate Nitrate Residual Oil Wood Smoke Sea Salt Incinerator Primary Coal **Averaging Time:** 24-h avg **Mean (SD):** NR **Range (Min, Max):** NR	**Increment:** $PM_{2.5}$ Sources 5-95th = NR **% Increase (median percent excess risk)** **lag:** Secondary sulfate: 16.0% 0 Traffic: 13.2% 1 Copper (Cu) smelter: 12.0% 0 Sea salt: 10.2% 5 Biomass/wood combustion: 8.6% 3
Reference: Ostro et al. (2006, 087991) **Period of Study:** 1/1999–12/2002 **Location:** 9 California counties (CALFINE)	**Outcome (ICD10):** Mortality: Total mortality (respiratory, cardiovascular, ischemic heart disease, diabetes) Respiratory (J00-J98) Cardiovascular (I00-I99) Ischemic heart disease (I20-I25) Diabetes (E10-E14) **Study Design:** Time-series **Statistical Analyses:** Poisson, natural splines and penalized splines **Age Groups:** All ages > 65	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-h avg **Mean (SD):** Contra Costa: 14 Fresno: 23 Kern: 22 Los Angeles: 21 Orange: 21 Riverside: 29 Sacramento: 14 Santa Clara: 15 San Diego: 16 **Range (Min, Max):** Contra Costa: (1, 77) Fresno: (1, 160) Kern: (1, 155) Los Angeles: (4, 85) Orange: (4, 114) Riverside: (2, 120) Sacramento: (1, 108) Santa Clara: (2, 74) San Diego: (0, 66) **Copollutant (correlation):** NO_2 r = 0.56	**Increment:** 10 $\mu g/m^3$ **% Increase (Lower CI, Upper CI)** **lag:** Penalized splines All ages: All-cause: 0.2% (-0.2, 0.7) 2 0.6% (0.2, 1.0) 0-1 Cardiovascular: 0.3% (-0.1, 0.7) 2 0.6% (0.0, 1.1) 0-1 Respiratory: 1.3% (0.1, 2.6) 2 2.2% (0.6, 3.9) 0-1 > 65: All-cause: 0.2% (-0.2, 0.7) 2 0.7% (0.2, 1.1)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
		CO	0-1
		r = 0.60	Ischemic heart disease: 0.3% (-0.5, 1.0)
		O_3 (1h)	0-1
		r = -0.14	Males: 0.5% (-0.2, 1.2)
		O_3 (8h)	0-1
		r = -0.22	Females: 0.8% (0.3, 1.3)
			0-1
			Whites: 0.8% (0.2, 1.3)
			0-1
			Blacks: 0.1% (-0.9, 1.2)
			0-1
			Hispanics: 0.8% (-0.1, 1.6)
			0-1
			In hospital: 0.6% (-0.1, 1.3)
			0-1
			Out of hospital: 0.6% (0.1, 1.1)
			0-1
			High school graduates: 0.4% (0.0, 0.8)
			0-1
			Non-high school graduates: 0.9% (-0.1, 1.9)
			0-1
			Natural splines
			All cause
			4 df: 0.5% (-0.1, 1.1)
			0-1
			8 df: 0.4% (-0.1, 0.9)
			0-1
			12 df: 0.3% (-0.1, 0.7)
			0-1
			Cardiovascular
			4 df: 0.4% (-0.2, 0.9)
			0-1
			8 df: 0.1% (-0.5, 0.6)
			0-1
			12 df: 0.0% (-0.6, 0.6)
			0-1
			Respiratory
			4 df: 2.1% (0.2, 4.1)
			0-1
			8 df: 1.6% (-0.5, 3.6)
			0-1
			12 df: 1.3% (-0.3, 2.9)
			0-1
			> 65

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			All cause
			4 df: 0.7% (0.0, 1.3)
			0-1
			8 df: 0.4% (-0.1, 0.9)
			0-1
			12 df: 0.3% (-0.1, 0.8)
			0-1
Reference: Ostro et al. (2007, 091354)	**Outcome (ICD10):** Mortality:	**Pollutant:** $PM_{2.5}$	**Increment:** 14.6 $\mu g/m^3$
Period of Study: $PM_{2.5}$ speciation analysis: 1/2000-12/2003. $PM_{2.5}$ analysis: 1/1999-12/2003	Total (non-accidental) mortality	**Averaging Time:** 24-h avg	**% Increase (Lower CI, Upper CI)**
	Respiratory (J00-J98)	**Mean (SD):**	**lag:**
Location: 6 California counties (2000–2003). 9 California counties (1999–2003) (CALFINE)	Cardiovascular (I00-I99)	2000–2003: 19.28	Cardiovascular
	Study Design: Time-series	1999–2003: 18.6	1.6% (0.0, 3.1)
	Statistical Analyses: Poisson, natural splines	**Range (Min, Max): NR**	3
	Age Groups: >65	**Copollutant (correlation):**	**Notes:** The study does not present all estimates quantitatively.
		EC: r = 0.53	
		OC: r = 0.62	
		NO_3: r = 0.65	
		SO_4: r = 0.32	
		Al: r = 0.02	
		Br: r = 0.54	
		Ca: r = 0.23	
		Cl: r = 0.15	
		Cu: r = 0.23	
		Fe: r = 0.38	
		K: r = 0.52	
		Mn: r = 0.21	
		Ni: r = 0.11	
		Pb: r = 0.27	
		S: r = 0.35	
		Si: r = 0.16	
		Ti: r = 0.24	
		V: r = 0.20	
		Zn: r = 0.51	

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Ostro et al. (2008, 097971) **Period of Study:** 1/2000–12/2003 **Location:** 6 California counties	**Outcome (ICD10):** Mortality: Cardiovascular (I00-I99) **Study Design:** Time-series **Statistical Analyses:** Poisson, natural cubic splines and natural splines **Age Groups:**	**Pollutant:** $PM_{2.5}$, EC, OC, NO_3, SO_4, Ca, Cl, Cu, Fe, K, S, Si, Ti, Zn **Averaging Time:** 24-h avg **Mean (SD):** $PM_{2.5}$: 19.28 EC: 0.966 OC: 7.129 NO_3: 5.415 SO_4: 1.908 Ca: 0.080 Cl: 0.094 Cu: 0.007 Fe: 0.124 K: 0.117 S: 0.648 Si: 0.168 Ti: 0.009 Zn: 0.012 **Range (95th):** $PM_{2.5}$: 46.91 EC: 2.57 OC: 15.91 NO_3: 17.46 SO_4: 5.18 Ca: 0.20 Cl: 0.41 Cu: 0.02 Fe: 0.34 K: 0.26 S: 1.70 Si: 0.43 Ti: 0.02 Zn: 0.04	The study does not present quantitative results.
Reference: Perez et al. (2008, 156020) **Period of Study:** 3/27/2003-12/31/2005 **Location:** Barcelona, Spain	**Outcome:** respiratory mortality **Study Design:** cohort **Covariates:** temperature, humidity **Statistical Analysis:** autoregressive Poisson regression models **Statistical Package:** NR **Age Groups:** All deaths	**Pollutant:** $PM_{2.5-1}$ **Averaging Time:** 24 h **Mean (SD) Unit:** 5.5 (3.8) $\mu g/m^3$ **Range (Min, Max):** 0.6, 45.5 **Copollutant:** $PM_{10-2.5}$, PM_1	**Increment:** 10 $\mu g/m^3$ **Odds Ratio (95%CI)** **Lag** Avg L0-1: 0.998 (0.849-1.174), p = 0.981 L1: 1.014 (0.886-1.161), p = 0.838 L2: 1.295 (1.141-1.470), p = 0.000 Multi-pollutant Model Avg L0-1: 0.987 (0.806-1.208), p = 0.898 L1: 1.022 (0.859-1.214), p = 0. L2: 1.206 (1.028-1.416), p = 0.022

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Perez et al. (2008, 156020) **Period of Study:** 3/27/2003-12/31/2005 **Location:** Barcelona, Spain	**Outcome:** cardiovascular mortality **Study Design:** cohort **Covariates:** temperature, humidity **Statistical Analysis:** autoregressive Poisson regression models **Statistical Package:** NR **Age Groups:** All deaths	**Pollutant:** $PM_{2.5-1}$ **Averaging Time:** 24 h **Mean (SD) Unit:** 5.5 (3.8) $\mu g/m^3$ **Range (Min, Max):** 0.6, 45.5 **Copollutant:** $PM_{10-2.5}$, PM_1	**Increment:** 10 $\mu g/m^3$ **Odds Ratio (95%CI)** **Lag** Avg L0-1: 1.100 (1.002-1.207), p = 0.046 L1: 1.112 (1.031-1.200), p = 0.006 L2: 1.078 (0.999-1.163), p = 0.052 Multi-pollutant Model Avg L0-1: 0.994 (0.885-1.116), p = 0.920 L1: 0.984 (0.892-1.086), p = 0.754 L2: 0.981 (0.891-1.079), p = 0.688
Reference: Perez et al. (2008, 156020) **Period of Study:** 3/27/2003-12/31/2005 **Location:** Barcelona, Spain	**Outcome:** cerebrovascular mortality **Study Design:** cohort **Covariates:** temperature, humidity **Statistical Analysis:** autoregressive Poisson regression models **Statistical Package:** NR **Age Groups:** All deaths	**Pollutant:** $PM_{2.5-1}$ **Averaging Time:** 24 h **Mean (SD) Unit:** 5.5 (3.8) $\mu g/m^3$ **Range (Min, Max):** 0.6, 45.5 **Copollutant:** $PM_{10-2.5}$, PM_1	**Increment:** 10 $\mu g/m^3$ **Odds Ratio (95%CI)** **Lag** Avg L0-1: 1.083 (0.897-1.307), p = 0.406 L1: 1.121 (0.964-1.303), p = 0.140 L2: 0.984 (0.841-1.152), p = 0.839 Multi-pollutant Model Avg L0-1: 0.899 (0.712-1.135), p = 0.371 L1: 0.905 (0.743-1.102), p = 0.321 L2: 0.868 (0.711-1.060), p = 0.165
Reference: Rainham et al. (2005, 088676) **Period of Study:** 1981–1999 **Location:** Toronto, Canada	**Outcome:** Mortality: Total (non-accidental) (< 800) Cardiorespiratory (390-459 480-519) Other-causes **Study Design:** Time-series **Statistical Analyses:** Poisson GLM, natural splines **Age Groups:** All ages	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-h avg **Mean (SD):** All years: 17.0 (8.7) Winters (Dec–Feb): 17.2 (6.8) Summers (June–Aug): 18.8 (10.2) **Range (Min, Max):** NR **Copollutant:** CO NO_2 SO_2 O_3	**Increment:** NR **% Increase (Lower CI, Upper CI)** **lag:** Winter and Winter Synoptic Events Winter Total: 0.998% (0.997, 1.000) 2 Cardiorespiratory: 0.998 (0.996, 1.000) 2 Other: 0.998% (0.996, 1.000) 2 Dry Moderate Total: 1.001% (0.996, 1.007) 1 Cardiorespiratory: 1.005 (0.998, 1.011) 1 Other: 0.997% (0.989, 1.006) 0 Dry Polar Total: 0.998% (0.995, 1.001) 2 Cardiorespiratory: 0.995 (0.991, 0.999) 2

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			Other: 1.002% (0.998, 1.005)
			1
			Moist Moderate
			Total: 0.998% (0.993, 1.002)
			2
			Cardiorespiratory: 1.003 (0.995, 1.010)
			1
			Other: 0.997% (0.991, 1.004)
			1
			Moist Polar
			Total: 1.001% (0.998, 1.005)
			1
			Cardiorespiratory: 1.002 (0.997, 1.007)
			2
			Other: 1.003% (0.999, 1.007)
			0
			Moist Tropical
			Total: 1.007% (0.965, 1.203)
			0
			Cardiorespiratory: 1.123 (1.031, 1.224)
			2
			Other: 1.248% (1.123, 1.387)
			0
			Transition
			Total: 1.003% (0.996, 1.009)
			1
			Cardiorespiratory: 0.996 (0.987, 1.004)
			0
			Other: 0.997% (0.990, 1.004)
			0
			summer and summer Synoptic Events
			summer
			Total: 1.000% (1.000, 1.001)
			0
			Cardiorespiratory: 1.001 (1.000, 1.002)
			0
			Other: 1.001% (1.000, 1.002)
			0
			Dry Moderate
			Total: 1.001% (0.999, 1.002)
			2
			Cardiorespiratory: 1.002 (0.999, 1.004)
			2
			Other: 0.999% (0.997, 1.002)
			0

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			Dry Polar
			Total: 1.002% (0.999, 1.005)
			2
			Cardiorespiratory: 0.996 (0.991, 1.000)
			0
			Other: 1.003% (0.999, 1.007)
			2
			Dry Tropical
			Total: 1.016% (1.006, 1.027)
			0
			Cardiorespiratory: 1.017 (1.005, 1.030)
			2
			Other: 1.017% (1.003, 1.031)
			0
			Moist Moderate
			Total: 1.002% (1.000, 1.004)
			2
			Cardiorespiratory: 1.003 (0.999, 1.006)
			2
			Other: 1.004% (1.001, 1.006)
			0
			Moist Polar
			Total: 1.005% (0.998, 1.011)
			1
			Cardiorespiratory: 1.008 (0.997, 1.018)
			0
			Other: 1.003% (0.995, 1.011)
			1
			Moist Tropical
			Total: 0.999% (0.997, 1.001)
			2
			Cardiorespiratory: 0.996 (0.993, 1.000)
			2
			Other: 0.998% (0.995, 1.001)
			1
			Transition
			Total: 1.005% (0.996, 1.014)
			1
			Cardiorespiratory: 1.007 (0.994, 1.020)
			1
			Other: 1.002% (0.996, 1.008)
			2

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Rosenthal et al. (2008, 156925)	**Outcome:** Non-Dead on Arrival (DOA) Out-of-Hospital Cardiac Arrests (OHCA)	**Pollutant:** $PM_{2.5}$	**Increment:** 10 $\mu g/m^3$
Period of Study: 7/2002–7/ 2006	Witnessed non-DOA OHCA	**Averaging Time:** 24-h avg	**Hazard Ratio (Lower CI, Upper CI)**
Location: Indianapolis, Indiana	**Study Design:** Case-crossover	Hourly	**lag:**
	Statistical Analyses: Time-stratified conditional logistic regression	**Mean (SD):**	Out-of-Hospital non-DOA Cardiac Arrests
	Age Groups: All ages	NR	All
	Study Population: Non-DOA OHCA: 1,374	IQR (25th, 75th):	1.02 (0.94, 1.11)
	Witnessed non-DOA OHCA: 511	All non-DOA	0
		All heart rhythms: (9.4, 19.5)	1.00 (0.92, 1.08)
		OHCA: (9.6, 19.5)	1
		Referents: (9.3, 19.5)	0.98 (0.90, 1.06)
		Asystole: (9.2, 19.4)	2
		OHCA: (9.2, 19.7)	1.00 (0.92, 1.08)
		Asystole: (9.2, 19.2)	3
		Witnessed non-DOA hourly	1.02 (0.92, 1.12)
		All heart rhythms: (8.8, 20.7)	0-1 avg
		OHCA: (8.8, 21.9)	1.01 (0.91, 1.12)
		Referents: (8.8, 20.4)	0-2 avg
		Asystole: (8.5, 19.8)	1.02 (0.91, 1.14)
		OHCA: (9.4, 21.3)	0-3 avg
		Referents: (8.3, 19.1)	Asystole
		Copollutant (correlation): NR	1.03 (0.91, 1.17)
			0
			1.00 (0.89, 1.13)
			1
			1.01 (0.90, 1.13)
			2
			0.98 (0.87, 1.10)
			3
			1.03 (0.90, 1.18)
			0-1 avg
			1.05 (0.90, 1.22)
			0-2 avg
			1.04 (0.88, 1.22)
			0-3 avg
			Vfib
			1.08 (0.92, 1.28)
			0
			1.02 (0.87, 1.21)
			1
			0.96 (0.80, 1.14)
			2
			1.10 (0.93, 1.31)
			3
			1.06 (0.88, 1.28)
			0-1 avg
			1.01 (0.82, 1.25)
			0-2 avg
			1.05 (0.83, 1.32)
			0-3 avg
			PEA
			0.92 (0.77, 1.08)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Schwartz et al. (2002, 025312) **Period of Study:** 1979–Late 1980's **Location:** 6 U.S. cities	**Outcome:** Mortality: Total (non-accidental) (< 800) **Study Design:** Time-series **Statistical Analyses:** Hierarchical modeling: 1. Poisson GAM, LOESS 2. Multivariate modeling **Age Groups:** All ages	**Pollutant:** $PM_{2.5}$, $PM_{2.5}$ sources (Traffic, Coal, Residual Oil) **Averaging Time:** 24-h avg **Mean (SD):** $PM_{2.5}$ Range: (Madison: 11.3 to Steubenville: 30.5) Traffic Range: (Steubenville: 1.5 to Boston: 4.8) Coal Range: (Madison: 4.9 to Steubenville: 19.2) Residual Oil Range: (Boston: 0.5 to Steubenville: 0.9) **Range (Min, Max):** NR	The study does not present quantitative results.
Reference: Simpson et al. (2005, 087438) **Period of Study:** 1/1996–12/1999 **Location:** 4 Australian cities	**Outcome:** Mortality: Non-accidental (< 800) Cardiovascular (390-459) Respiratory (460-519) **Study Design:** Time-series meta-analysis **Statistical Analyses:** Poisson GAM, natural splines Poisson GLM, natural splines **Age Groups:** All ages	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-h avg **Mean (SD):** Brisbane: $PM_{2.5}$: 7.50 Sydney: $PM_{2.5}$: 9.00 Melbourne: $PM_{2.5}$: 9.30 Perth: $PM_{2.5}$: 9.0 $\mu g/m^3$ **Range (Min, Max):** Brisbane: $PM_{2.5}$: (1.9, 19.7) Sydney: $PM_{2.5}$: (2.4, 35.3) Melbourne: $PM_{2.5}$: (2.7, 35.1) Perth: $PM_{2.5}$: (2.8, 37.3) **Copollutant:** CO, NO_2	**Increment:** 10 $\mu g/m^3$ % Increase (Lower CI, Upper CI) lag: $PM_{2.5}$ 0.9% (-0.7, 2.5)
Reference: Slaughter et al. (2005, 073854) **Period of Study:** 1/1995–12/1999 **Location:** Spokane, Washington	**Outcome:** Mortality: Non-accidental (< 800) **Study Design:** Time-series **Statistical Analyses:** Poisson GLM, natural splines **Age Groups:** All ages	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-h avg **Mean (SD):** NR **Range (9th, 95th):** $PM_{2.5}$: (4.2, 20.2) **Copollutant (correlation):** $PM_{2.5}$: r = 0.95 PM_{10}: r = 0.62 $PM_{10\text{-}2.5}$: r = 0.31 CO: r = 0.62	**Increment:** $PM_{2.5}$: 10 $\mu g/m^3$ PM_{10}: 25 $\mu g/m^3$ Relative Risk (Lower CI, Upper CI) lag: $PM_{2.5}$ (0.97, 1.04) 1 0.99 (0.96, 1.03) 2 1.00 (0.97, 1.03) 3

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Stieb et al. (2002, 025205) **Period of Study:** Publication dates of studies: 1985–12/2000 Mortality series: 1958–1999 **Location:** 40 cities (11 Canadian cities, 19 U.S. cities, Santiago, Amsterdam, Erfurt, 7 Korean cities)	**Outcome:** Mortality: All-cause (non-accidental) **Study Design:** Meta-analysis **Statistical Analyses:** Random effects model **Age Groups:** All ages	**Pollutant:** $PM_{2.5}$ **Averaging Time:** NR **Mean (SD):** NR **Range (Min, Max):** NR **Copollutant:** Varied between studies: O_3 SO_2 NO_2 CO	**Increment:** $PM_{2.5}$: 18.3 $\mu g/m^3$ **% Increase (Lower CI, Upper CI)** **lag:** Single-pollutant models 18 studies $PM_{2.5}$: 2.0% (1.2, 2.7) Multipollutant models 8 studies $PM_{2.5}$: 1.3% (0.6, 1.9)
Reference: Sullivan et al. (2003, 043156) **Period of Study:** 1985–1994 **Location:** Western Washington	**Outcome:** Out-of-hospital cardiac arrest **Study Design:** Case-crossover **Statistical Analyses:** Conditional logistic regression **Age Groups:** 19-79 Study Population: Out-of-hospital cardiac arrests: 1,206	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-h avg Median (SD) unit: PM_{10} Lag 0: 28.05 Lag 1: 27.97 Lag 2: 28.40 **Range (Min, Max):** PM_{10}: (7.38, 89.83) **Copollutant (correlation):** SO_2, CO Notes: Study used nephelometry to measure particles and equated the measurements to $PM_{2.5}$ concentrations.	**Increment:** PM_{10}: 16.51 $\mu g/m^3$ $PM_{2.5}$: 13.8 $\mu g/m^3$ Odds Ratio (Lower CI, Upper CI) lag: Overall PM_{10} 1.05 (0.87, 1.27) 0 0.91 (0.75, 1.11) 1 1.03 (0.82, 1.28) 2 $PM_{2.5}$ 0.94 (0.88, 1.01) 0.94 (0.88, 1.02) 1 1.00 (0.93, 1.08) 2 $PM_{2.5}$: Stratified by subject characteristics □ 55 0.95 (0.76, 1.18) 0 0.89 (0.71, 1.12) 1 0.95 (0.75, 1.20) 2 ＞55 0.94 (0.88, 1.02) 0 0.95, (0.88, 1.03) 1 1.01 (0.93, 1.10) 2

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			Male
			0.95 (0.87, 1.03)
			0
			0.96 (0.88, 1.04)
			1
			1.01 (0.93, 1.10)
			2
			Female
			0.93 (0.82, 1.06)
			0
			0.92 (0.80, 1.07)
			1
			0.98 (0.83, 1.15)
			2
			White
			0.93 (0.86, 1.01)
			0
			0.95 (0.88, 1.03)
			1
			1.03 (0.95, 1.12)
			2
			Non-White
			1.09 (0.88, 1.36)
			0
			0.96 (0.75, 1.22)
			1
			0.88 (0.68, 1.14)
			2
			Current Smoker
			1.05 (0.92, 1.19)
			0
			0.98 (0.86, 1.12)
			1
			1.06 (0.92, 1.22)
			2
			Nonsmoker
			0.93 (0.85, 1.01)
			0
			0.93 (0.85, 1.02)
			1
			0.97 (0.89, 1.07)
			2
			Drinker
			1.13 (0.92, 1.39)
			0

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			1.15 (0.94, 1.41)
			1
			1.16 (0.92, 1.45)
			2
			Nondrinker
			0.94 (0.86, 1.03)
			0
			0.93 (0.85, 1.02)
			1
			1.00 (0.92, 1.10)
			2
			Activity Level–Unrestricted
			0.96 (0.89, 1.03)
			0
			0.96 (0.89, 1.04)
			1
			1.01 (0.93, 1.10)
			2
			Activity Level–Limited
			0.82 (0.56, 1.20)
			0
			0.70 (0.45, 1.09)
			1
			0.97 (0.65, 1.43)
			2
			$PM_{2.5}$: Stratified by disease state
			Heart disease
			0.95 (0.87, 1.04)
			0
			0.97 (0.89, 1.07)
			1
			1.06 (0.96, 1.16)
			2
			Ischemic Heart Disease
			0.91 (0.80, 1.04)
			0
			0.97 (0.84, 1.11)
			1
			1.09 (0.95, 1.26)
			2
			Active Angina
			0.98 (0.81, 1.20)
			0
			1.07 (0.88, 1.31)
			1

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			1.08 (0.89, 1.32)
			2
			Congestive Heart Failure
			0.91 (0.80, 1.03)
			0
			0.99 (0.87, 1.13)
			1
			1.11 (0.97, 1.26)
			2
			Supraventricular tachycardia
			1.41 (0.97, 2.04)
			0
			1.55 (1.07, 2.25)
			1
			1.23 (0.84, 1.82)
			2
			Bradycardia
			0.97 (0.64, 1.46)
			0
			1.29 (0.85, 1.96)
			1
			1.30 (0.84, 2.01)
			2
			Asthma
			(0.80, 1.27)
			0
			0.92 (0.71, 1.19)
			1
			0.93 (0.71, 1.22)
			2
			COPD
			1.00 (0.86, 1.17)
			1.04 (0.88, 1.23)
			1
			1.08 (0.92, 1.28)
			2
			$PM_{2.5}$: Persons with prior recognized heart disease stratified by smoking status
			All heart disease
			Current smoker
			1.08 (0.92, 1.26)
			0
			1.06 (0.89, 1.26)
			1
			1.29 (1.06, 1.55)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			2
			Nonsmoker
			0.91 (0.82, 1.02)
			0
			0.94 (0.84, 1.05)
			1
			0.99 (0.88, 1.11)
			2
			Ischemic Heart Disease
			Current smoker
			1.06 (0.84, 1.34)
			0
			0.99 (0.75, 1.30)
			1
			1.39 (1.04, 1.86)
			2
			Nonsmoker
			0.86 (0.73, 1.02)
			0
			0.93 (0.78, 1.11)
			1
			0.99 (0.83, 1.18)
			2
			Active Angina
			Current smoker
			1.28 (0.88, 1.86)
			0
			1.26 (0.79, 2.01)
			1
			1.57 (0.99, 2.48)
			2
			Nonsmoker
			0.87 (0.68, 1.12)
			0
			0.93 (0.72, 1.21)
			1
			0.91 (0.70, 1.17)
			2
			Congestive Heart Failure
			Current smoker
			1.00 (0.79, 1.28)
			0
			1.03 (0.78, 1.35)
			1
			1.46 (1.10, 1.96)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			2
			Nonsmoker
			0.88 (0.76, 1.03)
			0
			0.96 (0.82, 1.12)
			1
			0.99 (0.84, 1.17)
			2
			Supraventricular tachycardia
			Current smoker
			12.80 (1.05, 156.57)
			0
			2.56 (0.82, 7.99)
			1
			1.15 (0.46, 2.86)
			2
			Nonsmoker
			1.19 (0.74, 1.90)
			0
			1.35 (0.87, 2.10)
			1
			1.15 (0.73, 1.82)
			2
			Bradycardia
			Nonsmoker
			0.84 (0.14, 4.95)
			0
			0.42 (0.03, 5.34)
			1
			0.51 (0.05, 5.79)
			2
			Nonsmoker
			0.99 (0.63, 1.55)
			0
			1.42 (0.90, 2.24)
			1
			1.39 (0.88, 2.20)
			2
Reference: Thurston et al. (2005, 097949) **Period of Study:** Washington, DC: 8/1988–12/1997. Phoenix, Arizona: 1995–1997 **Location:** Washington, DC and surrounding counties Phoenix, Arizona	**Outcome:** Mortality: Total (non-accidental) (< 800) Cardiovascular (390-448) **Study Design:** Time-series Source-apportionment **Statistical Analyses:** Poisson GLM, natural splines	**Pollutant:** $PM_{2.5}$, and source apportioned $PM_{2.5}$: Crustal Traffic Secondary SO_4 Secondary NO_3 Wood	**Increment:** 10 $\mu g/m^3$ **% Increase:** Total (non-accidental): Secondary sulfate: Phoenix: 5.2% Washington, DC: 3.8% Motor vehicles:

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
	Age Groups: Washington, DC: All ages	Oil	Phoenix: 0.9%
	Phoenix, Arizona: ≥ 65	Salt	Washington, DC: 4.2%
		Incinerator	
		Averaging Time: 24-h avg	
		Median (SD) unit: NR	
		Range (Min, Max): NR	
		Copollutant: $PM_{2.5}$ species (Na, Mg, Al, Si, P, S, Cl, K, Ca, Sc, Ti, V, Cr, Mn, Fe, Co, Ni, Cu, Zn, Ga, Ge, As, Se, Br, Rb, Sr, Y, Zr, Mo, Rh, Pd, Ag, Cd, Sn, Sb, Te, I, Cs, Ba, La, W, Au, Hg, Pb, OC, EC)	
Reference: Villeneuve et al. (2003, 055051)	**Outcome:** Mortality:	**Pollutant:** $PM_{2.5}$	**Increment:**
Period of Study: 1986–1999	Non-accidental (< 800)	**Averaging Time:** 24-h avg	$PM_{2.5}$ (Daily): 9.0 $\mu g/m^3$
Location: Vancouver, Canada	Cardiovascular (401-440)	**Mean (SD):**	$PM_{2.5}$ (6th Day): 15.7 $\mu g/m^3$
	Respiratory (460-519)	Daily	**% Increase (Lower CI, Upper CI)**
	Cancer (140-239)	$PM_{2.5}$: 7.9	**lag:**
	Study Design: Time-series	Every 6th Day	Non-accidental
	Statistical Analyses: Poisson, natural splines	$PM_{2.5}$: 11.6	$PM_{2.5}$ (Daily)
	Age Groups: ≥ 65	**Range (Min, Max):**	-0.1% (-5.1, 5.2)
		Daily	0-2 avg
		$PM_{2.5}$: (2.0, 32.0)	-0.1% (-4.1, 4.1)
		Every 6th Day	0
		$PM_{2.5}$: (1.8, 43.0)	-0.3% (-4.2, 3.7)
		Copollutant:	1
		SO_2	0.5% (-3.3, 4.4)
		CO	2
		NO_2	$PM_{2.5}$ (6th Day)
		O_3	-2.8% (-7.5, 2.1)
			0
			2.0% (-2.6, 7.0)
			1
			4.5% (-0.3, 9.5)
			2
			Cardiovascular
			$PM_{2.5}$ (Daily)
			1.5% (-6.1, 9.7)
			0-2 avg
			4.3% (-1.7, 10.7)
			0
			-1.0% (-7.0, 5.4)
			1
			-0.5% (-6.5, 5.9)
			2
			$PM_{2.5}$ (6th Day)
			-1.5% (-8.9, 6.5)
			0
			-2.0% (-9.3, 5.8)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			1
			3.0% (-4.2, 10.8)
			2
			Respiratory
			$PM_{2.5}$ (Daily)
			-0.7% (-13.1, 13.4)
			0-2 avg
			6.7% (-3.7, 18.3)
			0
			-3.0% (-12.8, 7.9)
			1
			-5.8% (-15.2, 4.7)
			2
			$PM_{2.5}$ (6th Day)
			10.0% (-4.7, 26.8)
			0
			8.3% (-5.4, 24.0)
			1
			0.3% (-12.4, 14.9)
			2
			Cancer
			$PM_{2.5}$ (Daily)
			-0.3% (-9.4, 9.8)
			0-2 avg
			-4.5% (-11.2, 2.8)
			0
			2.7% (-5.0, 11.0)
			1
			2.5% (-5.1, 10.7)
			2
			$PM_{2.5}$ (6th Day)
			-5.1% (-13.8, 4.5)
			0
			-0.3% (-9.7, 11.0)
			1
			0.2% (-9.1, 10.4)
			2

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Wilson et al. (2007, 157149)	**Outcome:** Cardiovascular	**Pollutant:** $PM_{2.5}$	**Increment:** 10 $\mu g/m^3$
Period of Study: 1995–1997	**Study Design:** Time-series	**Averaging Time:** 24-h avg	**% Excess Risk (Lower CI, Upper CI)**
Location: Phoenix, Arizona	**Statistical Analyses:** Poisson GAM, nonparametric smoothing spline	**Mean (SD):** NR	**lag:** Central Phoenix: 11.5% (2.8, 20.9)
		Range (Min, Max): NR	0-5 ma
	Age Groups: > 25	**Copollutant (correlation):** NR	6.6% (1.1, 12.5)
			1
			2.0% (-3.2, 7.5)
			2
			Middle Phoenix: 2.9% (-4.9, 11.4)
			0-5 ma
			6.4% (1.1, 11.9)
			2
			Outer Phoenix: 1.6% (-6.2, 10.0)
			0-5 ma

[1]All units expressed in $\mu g/m^3$ unless otherwise specified.

Table E-19. Short-term exposure – mortality - other PM size fractions.

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Perez et al. (2008, 156020)	**Outcome:** respiratory mortality	**Pollutant:** PM_1	**Increment:** 10 $\mu g/m^3$
Period of Study: 3/27/2003-12/31/2005	**Study Design:** cohort	**Averaging Time:** 24 h	**Odds Ratio (95%CI)**
Location: Barcelona, Spain	**Covariates:** temperature, humidity	**Mean (SD) Unit:** 20.0 (10.3) $\mu g/m^3$	**Lag**
	Statistical Analysis: autoregressive Poisson regression models	**Range (Min, Max):** 1.9, 80.1	Avg L0-1: 1.005 (0.960-1.053), p = 0.824
		Copollutant: $PM_{10-2.5}$, $PM_{2.5-1}$	L1: 1.012 (0.969-1.056), p = 0.599
	Statistical Package: NR		L2: 1.042 (0.998-1.087), p = 0.063
	Age Groups: All deaths		Multi-pollutant Model
			Avg L0-1: 1.007 (0.957-1.059), p = 0.799
			L1: 1.008 (0.961-1.058), p = 0.739
			L2: 1.010 (0.963-1.059), p = 0.678
Reference: Perez et al. (2008, 156020)	**Outcome:** cardiovascular mortality	**Pollutant:** PM_1	**Increment:** 10 $\mu g/m^3$
Period of Study: 3/27/2003-12/31/2005	**Study Design:** cohort	**Averaging Time:** 24 h	**Odds Ratio (95%CI)**
Location: Barcelona, Spain	**Covariates:** temperature, humidity	**Mean (SD) Unit:** 20.0 (10.3) $\mu g/m^3$	**Lag**
	Statistical Analysis: autoregressive Poisson regression models	**Range (Min, Max):** 1.9, 80.1	Avg L0-1: 1.028 (1.000-1.057), p = 0.054
		Copollutant: $PM_{10-2.5}$, $PM_{2.5-1}$	L1: 1.029 (1.003-1.056), p = 0.030
	Statistical Package: NR		L2: 1.023 (0.996-1.050), p = 0.091
	Age Groups: All deaths		Multi-pollutant Model
			Avg L0-1: 1.025 (0.995-1.057), p = 0.688
			L1: 1.028 (1.000-1.058), p = 0.053
			L2: 1.024 (0.995-1.053), p = 0.110

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Perez et al. (2008, 156020) **Period of Study:** 3/27/2003-12/31/2005 **Location:** Barcelona, Spain	**Outcome:** cerebrovascular mortality **Study Design:** cohort **Covariates:** temperature, humidity **Statistical Analysis:** autoregressive Poisson regression models **Statistical Package:** NR **Age Groups:** All deaths	**Pollutant:** PM_1 **Averaging Time:** 24 h **Mean (SD) Unit:** 20.0 (10.3) $\mu g/m^3$ **Range (Min, Max):** 1.9, 80.1 **Copollutant:** $PM_{10-2.5}$, $PM_{2.5-1}$	**Increment:** 10 $\mu g/m^3$ **Odds Ratio (95%CI)** **Lag** Avg L0-1: 1.037 (0.981-1.097), p = 0.202 L1: 1.056 (1.003-1.113), p = 0.039 L2: 1.020 (0.968-1.075), p = 0.460 Multi-pollutant Model Avg L0-1: 1.042 (0.981-1.107), p = 0.179 L1: 1.063 (1.004-1.124), p = 0.035 L2: 1.034 (0.976-1.095), p = 0.255
Reference: Slaughter et al. (2005, 073854) **Period of Study:** 1/1995–12/1999 **Location:** Spokane, Washington	**Outcome:** Mortality: Non-accidental (< 800) **Study Design:** Time-series **Statistical Analyses:** Poisson GLM, natural splines **Age Groups:** All ages	**Pollutant:** PM_1 **Averaging Time:** 24-h avg **Mean (SD):** NR Range (9th, 95th) PM_1: (3.3, 17.6) **Copollutant (correlation):** PM_1 $PM_{2.5}$: r = 0.95 PM_{10}: r = 0.50 PM_{10}2.5: r = 0.19 CO: r = 0.63	This study does not present quantitative results for PM_1.

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Stölzel et al. (2007, 091374)	**Outcome:** Mortality:	**Pollutant:** $MC_{0.1\text{-}0.5}$, $MC_{0.01\text{-}2.5}$	**Increment:**
Period of Study: 9/1995–8/2001	Total (non-accidental) (< 800)	**Averaging Time:** 24-h avg	$MC_{0.1\text{-}0.5}$: 13.1 $\mu g/m^3$
Location: Erfurt, Germany	Cardio-respiratory (390-459, 460-519, 785, 786)	**Mean (SD):**	$MC_{0.01\text{-}2.5}$: 16.8 $\mu g/m^3$
	Study Design: Time-series	$MC_{0.1\text{-}0.5}$: 17.6 (14.8)	**Relative Risk (Lower CI, Upper CI)**
	Statistical Analyses: Poisson GAM	$MC_{0.01\text{-}2.5}$: 22.3 (19.2)	**lag:**
	Age Groups: All ages	IQR (25th, 75th):	Total (non-accidental)
		$MC_{0.1\text{-}0.5}$: (8.4, 21.5)	$MC_{0.1\text{-}0.5}$
		$MC_{0.01\text{-}2.5}$: (10.5, 27.3)	1.010 (0.986
		Copollutant (correlation):	1.034)
		$MC_{0.1\text{-}0.5}$	0
		NO: r = 0.52	1.006 (0.983
		NO_2: r = 0.60	1.029)
		CO: r = 0.58	1
		$MC_{0.01\text{-}2.5}$	1.007 (0.985
		NO: r = 0.51	1.029)
		NO_2: r = 0.58	2
		CO: r = 0.57	0.994 (0.973
			1.016)
			3
			1.002 (0.981
			1.023)
			4
			0.997 (0.976
			1.018)
			5
			$MC_{0.01\text{-}2.5}$
			1.007 (0.985
			1.030)
			0
			1.005 (0.984
			1.026)
			1
			1.003 (0.983
			1.023)
			2
			0.989 (0.970
			1.009)
			3
			1.002 (0.982
			1.022)
			4
			0.998 (0.979
			1.018)
			5
			Cardio-respiratory
			$MC_{0.1\text{-}0.5}$
			1.004 (0.977
			1.031)
			0
			1.004 (0.979
			1.029)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Yamazaki et al. (2007, 090748) **Period of Study:** 1995-1998 **Location:** Hong Kong, China	**Outcome:** Mortality: Intracerebral hemorrhage (431) Ischaemic stroke (434) **Study Design:** Time-stratified case-crossover **Statistical Analyses:** Conditional logistic regression **Age Groups:** ≥ 65	**Pollutant:** PM_7 **Averaging Time:** 1-h avg **Mean (SD):** Warmer Months (April-September): 40.3 Colder Months (October-March): 39.4 **Range (Min, Max):** NR **Copollutant (correlation):** Warmer Months NO_2: r = 0.46 to 0.63 Ox: r = -0.14 to 0.20 Colder Months NO_2: 0.42 to 0.79 Ox: r = -0.36 to -0.14	**Increment:** 30 $\mu g/m^3$ Odds Ratio (Lower CI, Upper CI) lag: 24-h avg concentrations Intracerebral hemorrhage Warmer months: 1.041 (0.984, 1.102) 0 Colder months: 1.005 (0.951, 1.061) 0 Ischaemic stroke Warmer months: 1.027 (0.993, 1.062) 0 Colder months: 1.005 (0.973, 1.039) 0 Exposure measured jointly as 24-h and 1-h mean concentrations Warmer months Intracerebral hemorrhage 1-h with 200 $\mu g/m^3$ threshold: 2.397 (1.476, 3.892) 2 h 24-h: 1.019 (0.960, 1.082) 0 Ischaemic stroke 1-h with 200 $\mu g/m^3$ threshold: 1.051 (0.750, 1.472) 2 h 24-h: 1.018 (0.983, 1.055) 0 Warmer months Intracerebral hemorrhage 1-h with 200 $\mu g/m^3$ threshold: 0.970 (0.712, 1.322) 2 h 24-h: 1.015 (0.958, 1.075) 0 Ischaemic stroke 1-h with 200 $\mu g/m^3$ threshold: 1.040 (0.855, 1.265) 2 h 24-h: 1.003 (0.968, 1.039) 0

[1]All units expressed in $\mu g/m^3$ unless otherwise specified.

E.4. Long-Term Exposure and Cardiovascular Outcomes

Table E-20. Long-term exposure - cardiovascular morbidity outcomes - PM_{10}

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Baccarelli et al. (2008, 157984) **Period of Study:** 1995-2005 **Location:** Italy (Lombardy region)	**Outcome (ICD9 and ICD10):** Deep Vein Thrombosis (DVT) prothrombin time (PT) activated partial thromboplastin time (aPTT) **Age Groups:** 18-84yrs **Study Design:** Case-control (DVT outcome) Cross-sectional (PT and aPTT outcomes) **N:** 871 cases 1210 controls (randomly selected from friends and nonblood relatives of cases frequency matched by age to cases) **Statistical Analyses:** Unconditional logistic regression (DVT outcome) linear regression (PT and aPTT outcomes) **Covariates:** sex, area of residence, education, factor V Leiden or G20210A prothrombin mutation, current use of oral contraceptives or hormone therapy (variables controlled using penalized regression splines with 4 df) age, BMI, day of year (for seasonality), index date, ambient temperature **Season:** covariate **Dose-response Investigated?** Yes **Statistical Package:** STATA v9.0 and R v2.2.0	**Pollutant:** PM_{10} **Averaging Time:** 1 year (immediately preceding the diagnosis date for cases or the date of examination for controls) assessed other averaging periods presented in supplements (90 days, 180 days, 270 days, 2 yrs) **Mean (SD):** NR **Percentiles:** NR **Range (Min, Max):** Range for tertiles of exposure: 1: 12.0–44.2 2: 44.3–48.1 3: 48.2–51.5 **Monitoring Stations:** Monitors from 53 sites exposure assigned by dividing area into 9 regions **Copollutant (correlation):** NR	**PM Increment:** 10 $\mu g/m^3$ **Effect Estimate [Lower CI, Upper CI]: Estimated changes of PT associated with PM_{10}:** Among DVT cases: -0.12 (-0.23, 0.00), p = 0.04 Among Controls: -0.06 (-0.11, 0.00) , p = 0.04 **Estimated changes of aPTT associated with PM_{10}:** Among Controls: -0.09 (-0.19, 0.01) , p = 0.07 Among DVT cases: 0.01 (-0.03, 0.04) , p = 0.78 **Risk of DVT associated with PM_{10} (avg of 1 yr preceding diagnosis/exam date):** | **All subjects:** 1.70 (1.30, 2.23) , p < 0.001 **Sex:** Male: 2.07 (1.50, 2.84) , p < 0.001 Female: 1.40 (1.02, 1.92) , p = 0.04 P for interaction: p = 0.02 **Age:** 18-35yrs: 1.57 (1.11, 2.24) , p = 0.01 36-50yrs: 1.97 (1.41, 2.77) , p < 0.001 51-84yrs: 1.54 (0.90, 2.63) , p = 0.12 P for interaction: p = 0.99 **Premenopausal women with current use of oral contraceptives:** No: 1.53 (0.86, 2.72) , p = 0.14 Yes: 0.87 (0.46, 1.67), p = 0.68 P for interaction: p = 0.11 **Postmenopausal women with current use of hormone therapy:** No: 1.60 (0.72, 3.54), p = 0.24 Yes: 0.85 (0.29, 2.45), p = 0.76 P for interaction: p = 0.27 **Current use of oral contraceptive or hormone replacement therapy:** No: 1.64 (1.05, 2.57), p = 0.03 Yes: 0.97 (0.58, 1.61), p = 0.89 P for interaction: p = 0.048 **Body Mass Index:** 13.3-22.0: 1.47 (0.97, 2.23), p = 0.07; 22.1-24.9: 1.72 (1.17, 2.54), p = 0.006 25.0-53.3: 1.83 (1.03, 3.24), p = 0.04 P for interaction: p = 0.37 **Education:** Elementary/middle school:

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			1.93 (1.35, 2.76), p < 0.001 High school: 1.72 (1.24, 2.39), p = 0.001 College: 1.35 (0.74, 2.45), p = 0.33 P for interaction: p = 0.21 **Deficiencies of natural anticoagulant proteins:** None: 1.66 (1.26, 2.18), p < 0.001 Any: 2.56 (0.91, 7.18), p = 0.07 P for interaction: p = 0.41 **Factor V Leiden or G20210A prothrombin mutation:** None: 1.69 (1.27, 2.23), p < 0.001 Any: 1.79 (1.05, 3.05), p = 0.03 P for interaction: p = 0.83 **Hyperhomocysteinemia:** No: 1.66 (1.26, 2.19), p < 0.001 Yes: 2.19 (1.33, 3.61), p = 0.002 P for interaction: p = 0.25 **Any cause of thrombophilia:** No: 1.59 (1.19, 2.13), p = 0.002 Yes: 1.96 (1.34, 2.87), p < 0.001 P for interaction: p = 0.27 **Year of diagnosis:** 1995-97: 1.61 (1.06, 2.46), p = 0.03 1998-00: 1.34 (0.90, 1.99), p = 0.15 2001-05: 2.14 (1.04, 4.39), p = 0.04 P for interaction: p = 0.12 **Risk of DVT associated with PM_{10} over varying averaging times:** 90 days: 0.91 (0.80, 1.03), p = 0.12 180 days: 0.96 (0.82, 1.13), p = 0.63 270 days: 1.26 (1.01, 1.57), p = 0.04 365 days: 1.70 (1.30, 2.23), p = 0.0001 2 years: 1.47 (1.01, 2.14), p = 0.04 **Risk of DVT associated with PM_{10} (year preceding diagnosis/exam date)** **sensitivity analysis to evaluate the effect of different methods for adjusting for long-term trends:** **Handling of long-term time trends:** Ignored: 1.13 (0.89, 1.42), p = 0.31 Dummy variable for each year: 1.78 (1.31, 2.44), p = 0.0003 Linear term: 1.32 (1.02, 1.69), p = 0.03 Penalized spline, 2 df: 1.54 (1.19, 2.00), p = 0.001 Penalized spline, 3 df: 1.64 (1.26, 2.14), p = 0.0002 Penalized spline, 4 df: 1.70 (1.30, 2.23), p = 0.0001 Penalized spline, 5 df: 1.70 (1.29, 2.22), p = 0.0002

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			Penalized spline, 6 df: 1.66 (1.26, 2.19), p = 0.0003 Penalized spline, 7 df: 1.60 (1.21, 2.13), p = 0.001 Penalized spline, 8 df: 1.55 (1.15, 2.10), p = 0.004
Reference: Baccarelli et al. (2009, 188183) **Period of Study:** 1/1995-9/2005 **Location:** Lombardia Region, Italy	**Outcome:** Deep Vein Thrombosis **Study Design:** Case-control **Covariates:** age, sex, area of residence, BMI, education, medication use **Statistical Analysis:** logistic regression **Statistical Package:** Stata	**Pollutant:** PM_{10} Risk of DVT measured with regards to distance of residence from major road. Specific levels of PM_{10} not given.	**Increment:** NA **Relative Risk (95%CI) of DVT** All subjects, age-adjusted: 1.33 (1.03-1.71), p = 0.03 All subjects, adjusted for covariates: 1.47 (1.10-1.96), p = 0.008 All subjects, adjusted for covariates and background PM_{10} exposure: 1.47 (1.11-1.96), p = 0.008
Reference: Calderon-Garciduenas et al. (2008, 156317) **Period of Study:** Children recruited between Jul 2003 and Dec 2004 **Location:** Mexico (northeast or southwest Mexico city or Polotitlan)	**Outcome (ICD9 and ICD10):** Plasma Endothelin-1 (ET-1) and pulmonary arterial pressure (PAP) **Age Groups:** 6-13 years 7.9 ± 1.3 years **Study Design:** Cross-sectional **N:** 81 children **Statistical Analyses:** Analysis of variance by parametric one-way analysis of variance and the Newman-Keuls multiple comparison post test, Pearson's correlation **Covariates:** doesn't appear to have performed multivariable analyses however, collected information on age, place and length of residency, daily outdoor time, household cooking methods, parents' occupational history, family history of atopic illnesses and respiratory disease, and personal history of otolaryngologic and respiratory symptoms **Season:** No **Dose-response Investigated?** No **Statistical Package:** STATA v8.3, or GraphPad Software, Inc.	**Pollutant:** PM_{10} ($\mu g/m^3$) Exposures assessed quantitatively in Mexico City only no monitors in Polotitlan **Averaging Time:** 1, 2, and 7 days before the exam pollutant concentrations between 0700 and 1900 h were used for the estimates **Mean (SD):** Presented only in figures **Percentiles:** NR **Range (Min, Max):** Presented only in figures **Monitoring Stations:** 4 (2 in northeast and 2 in southwest Mexico City residence and school within 5 miles of one of these monitors) **Copollutant (correlation):** O_3	**PM Increment:** NA **Effect Estimate [Lower CI, Upper CI]:** No health effects models with measured PM concentrations were presented used city of residence to assign exposure no multivariable analyses presented Authors presented (statistically significantly) elevated ET-1 levels among children residing in both areas of Mexico City as compared to Polotitlan (control city): Mean ± SE (pg/mL) Control: 1.23 ± 0.06 Southwest Mexico City: 2.40 ± 0.14 Northeast Mexico City: 2.09 ± 0.10 Mexico City (overall): 2.24 ± 0.12 Authors presented (statistically significantly) elevated PAP levels among children residing in both areas of Mexico City as compared to Polotitlan (control city): Mean ± SE (mmHg) Control: 14.6 ± 0.4 Southwest Mexico City: 16.7 ± 0.6 Northeast Mexico City: 18.6 ± 0.9 Mexico City (overall): 17.3 ± 0.5 Correlation between ET-1 and time spent outdoors: r = 0.31, p = 0.0012 Correlation between PAP and time spent outdoors: r = 0.42, p = 0.0008

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Diez Roux et al. (2008, 156401) **Period of Study:** Baseline data collected June 2000–Aug 2002 Exposure assessed retrospectively between Aug 1982 and baseline date **Location:** USA (6 field centers: Baltimore, MD Chicago, IL Forsyth Co, NC Los Angeles, CA New York, NY St. Paul, MN	**Outcome (ICD9 and ICD10):** Three measures of subclinical atherosclerosis (common carotid intimal-medial thickness (CIMT), coronary artery calcification, and ankle-brachial index (ABI)) **Age Groups:** 44-84 yrs (MESA cohort) **Study Design:** Cross-sectional retrospective cohort **N:** 5172 for coronary calcium analysis 5037 for CIMT analysis 5110 for ABI analysis **Statistical Analyses:** Generalized Additive Models (Binomial regression: presence of calcification Linear regression: CIMT, ABI, amount of calcium among persons with non-zero calcification) **Covariates:** age, sex, race/ethnicity, socioeconomic factors, cardiovascular risk factors (BMI, hypertension, high density lipoprotein and low density lipoprotein cholesterol, smoking, diabetes, diet, physical activity models presented with and without adjustment for cardiovascular RFs) **Season:** NA **Dose-response Investigated?** No **Statistical Package:** NR	**Pollutant:** PM_{10} ($\mu g/m^3$) **Averaging Time:** 20-yr imputed mean **Mean (SD):** 34.1 (7.5) **Percentiles:** NR **Range (Min, Max):** NR **Monitoring Stations:** A spatio-temporal model was used to predict monthly $PM_{2.5}$ exposures based on the geographic location of each participant's residence. **Copollutant (correlation with 20-year imputed mean):** PM_{10} 20-yr observed mean r = 0.93 $PM_{2.5}$ 20-yr imputed mean r = 0.73 PM_{10} 2001 imputed mean r = 0.75 PM_{10} 2001 observed mean r = 0.80 $PM_{2.5}$ 2001 mean r = 0.86	**PM Increment:** 21.0 $\mu g/m^3$ (approx. 10th-90th percentile) **Effect Estimate [Lower CI, Upper CI]:** **CIMT:** Relative difference (95% CI): 1.01 (1.00, 1.02) Adj. for additional CVD RFs: 1.02 (1.00, 1.03) **ABI:** Mean difference (95% CI): 0.002 (-0.005, 0.009) Adj. for additional CVD RFs: 0.001 (-0.006, 0.009) **Coronary calcium:** Relative prevalence (95% CI): 1.02 (0.96, 1.07) Adj. for additional CVD RFs: 1.02 (0.96, 1.08) **Coronary calcium (in those with calcium):** Relative difference (95% CI): 0.98 (0.84, 1.13) Adj. for additional CVD RFs: 1.01 (0.86, 1.18) Found no clear heterogeneity by age, sex, lipid status, smoking status, diabetes status, BMI, education or study site.
Reference: Maheswaran et al. (2005, 088683) **Period of Study:** 1994-1998 **Location:** Sheffield, United Kingdom	**Outcome (ICD9 and ICD10):** Stroke mortality (ICD9: 430-438) and Emergency hospital admissions (ICD10: I60-I69) **Age Groups:** ≥ 45 years **Study Design:** Small area ecological cross-sectional **N:** 1030 census enumeration districts (CEDs) 108 CEDs excluded from PM analyses due to artifacts in the modeled emissions data. The analysis was based on 2979 deaths, 5122 admissions and a population of 199,682 **Statistical Analyses:** Poisson regression **Covariates:** age, sex, socioeconomic deprivation, and smoking prevalence (some models also included age-by-deprivation interaction) **Season:** NA **Dose-response Investigated?** Yes, examined quintiles of exposure **Statistical Package:** SAS	**Pollutant:** PM_{10} ($\mu g/m^3$) **Averaging Time:** 5-yr avg **Mean (SD):** Presented mean values and ranges for each quintile of exposure: 1: 16.0 (< 16.8) 2: 17.5 (≥ 16.8, < 18.2) 3: 18.8 (≥ 18.2, < 19.3) 4: 19.8 (≥ 19.3, < 20.6) 5: 23.3 (≥ 20.6) **Monitoring Stations:** Used air pollution model incorporating point, line and grid sources of pollution and meteorological data. **Copollutant (correlation):** CO (r = 0.82) NO_X (r = 0.87)	**PM Increment:** NA **Effect Estimate [Lower CI, Upper CI]:** **Rate Ratios (95%CI) for stroke mortality adjusted for overdispersion by quintile of PM_{10} level** Adjusted for sex and age: 1: 1 (ref) 2: 0.95 (0.84, 1.08) 3: 1.12 (0.99, 1.27) 4: 1.16 (1.03, 1.32) 5: 1.39 (1.23, 1.58) Adjusted for sex, age, deprivation, and smoking: 1: 1 (ref) 2: 0.94 (0.83, 1.07) 3: 1.08 (0.94, 1.24) 4: 1.12 (0.97, 1.29) 5: 1.33 (1.14, 1.56) **Rate Ratios (95%CI) for emergency hospital admissions because of stroke by quintile of PM_{10} level** Adjusted for sex and age:

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			1: 1 (ref) 2: 1.06 (0.95, 1.17) 3: 1.10 (0.99, 1.23) 4: 1.25 (1.12, 1.38) 5: 1.40 (1.26, 1.55) Adjusted for sex, age, deprivation, and smoking: 1: 1 (ref) 2: 1.01 (0.91, 1.13) 3: 0.98 (0.87, 1.10) 4: 1.08 (0.96, 1.22) 5: 1.13 (0.99, 1.29) **Rate Ratios (95%CI) for stroke mortality in relation to spatially smoothed (using a 1-km radius) modeled outdoor air pollution quintiles** Adjusted for sex, age, socioeconomic deprivation, age by deprivation interaction, and smoking prevalence: 1: 1 (ref) 2: 0.86 (0.75, 0.98) 3: 1.05 (0.92, 1.21) 4: 1.03 (0.89, 1.19) 5: 1.24 (1.05, 1.47) **Rate Ratios (95%CI) for emergency hospital admissions because of stroke in relation to spatially smoothed modeled outdoor air pollution quintiles** Adjusted for sex, age, socioeconomic deprivation, age by deprivation interaction, and smoking prevalence: 1: 1 (ref) 2: 1.05 (0.94, 1.17) 3: 1.07 (0.95, 1.20) 4: 1.06 (0.94, 1.20) 5: 1.15 (1.01, 1.31)
Reference: Maheswaran et al. (2005, 090769) **Period of Study:** 1994-1998 **Location:** Sheffield, United Kingdom	**Outcome (ICD9 and ICD10):** Coronary Heart Disease (CHD) mortality (ICD9: 410-414) and Emergency hospital admissions (ICD10: I20-I25) **Age Groups:** ≥ 45 years **Study Design:** Small area ecological cross-sectional **N:** 1030 census enumeration districts (CEDs) 108 CEDs excluded from PM analyses due to artifacts in the modeled emissions data. Results based on 6857 deaths, 11407 hospital admissions and 199,682 people aged ≥ 45 years **Statistical Analyses:** Poisson regression **Covariates:** age, sex, socioeconomic deprivation, and smoking prevalence	**Pollutant:** PM_{10} ($\mu g/m^3$) **Averaging Time:** 5-yr avg **Mean (SD):** Presented mean values and ranges for each quintile of exposure: 1: 16.0 (< 16.8) 2: 17.5 (≥ 16.8, < 18.2) 3: 18.8 (≥ 18.2, < 19.3) 4: 19.8 (≥ 19.3, < 20.6) 5: 23.3 (≥ 20.6) **Monitoring Stations:** Study used an air pollution model incorporating points, lines, and grids as sources of pollution, and meteorological data.	**PM Increment:** NA **Effect Estimate [Lower CI, Upper CI]:** **Rate Ratios (95%CI) for CHD mortality in relation to modeled outdoor air pollution quintiles, adjusted for overdispersion** Adjusted for sex and age: 1: 1 (ref) 2: 1.06 (0.98, 1.16) 3: 1.10 (1.01, 1.21) 4: 1.23 (1.13, 1.35) 5: 1.30 (1.19, 1.43) Adjusted for sex, age, deprivation, and smoking:

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
	(some models also included age-by-deprivation interaction) **Season:** NA **Dose-response Investigated?** Yes, examined quintiles of exposure **Statistical Package:** SAS	**Copollutant (correlation):** CO (r = 0.82) NO_X (r = 0.87)	1: 1 (ref) 2: 1.03 (0.94, 1.12) 3: 1.00 (0.90, 1.11) 4: 1.08 (0.98, 1.20) 5: 1.08 (0.96, 1.20) Adjusted for sex, age, deprivation, and smoking (spatially smoothed using a 1km radius): 1: 1 (ref) 2: 0.97 (0.89, 1.07) 3: 1.00 (0.90, 1.10) 4: 1.03 (0.93, 1.15) 5: 1.07 (0.96, 1.21) **Rate Ratios (95%CI) for emergency hospital admissions from CHD in relation to modeled outdoor air pollution quintiles** Adjusted for sex and age: 1: 1 (ref) 2: 1.08 (0.98, 1.19) 3: 1.11 (1.01, 1.22) 4: 1.17 (1.07, 1.29) 5: 1.36 (1.23, 1.50) Adjusted for sex, age, deprivation, and smoking: 1: 1 (ref) 2: 1.03 (0.93, 1.13) 3: 0.96 (0.86, 1.07) 4: 0.97 (0.87, 1.08) 5: 1.01 (0.90, 1.14) Adjusted for sex, age, deprivation, and smoking (spatially smoothed using a 1km radius): 1: 1 (ref) 2: 1.01 (0.92, 1.11) 3: 1.04 (0.93, 1.15) 4: 0.97 (0.87, 1.08) 5: 1.07 (0.95, 1.20)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: O'Neill et al. (2007, 156006) **Period of Study:** 2000-2004 **Location:** USA (6 field centers: Baltimore, MD Chicago, IL Forsyth Co, NC Los Angeles, CA New York, NY St. Paul, MN	**Outcome (ICD9 and ICD10):** Creatinine adjusted urinary albumin excretion Assessed 2 ways: continuous log urinary albumin/creatine ration (UACR) and clinically defined micro- or macro-albuminuria (UACR ≥ 25 mg/g) versus normal levels **Age Groups:** 44-84 yrs **Study Design:** Cross-sectional analyses and prospective cohort analyses **N:** 3901 participants free of clinical CVD at baseline **Statistical Analyses:** At baseline: multiple linear regression (continuous outcome) binomial regression (dichotomous outcome) 3-year change: repeated measures model with random subject effects (estimate 3-yr change in log UACR by levels of exposure) **Covariates:** age, gender, race, BMI, cigarette status, ETS, percent dietary protein for repeated measures models: time time x PM_{10} **Season:** NA **Dose-response Investigated?** Yes, examined quartiles of exposure **Statistical Package:** SAS	**Pollutant:** PM_{10} ($\mu g/m^3$) **Averaging Time:** avg of previous month, avg of previous 2 months (recent exposures) 20-yr directly monitored PM_{10} avg, 20-yr imputed PM_{10} avg (longer-term exposures) **Mean (SD):** Previous 20 years: 34.7 (7.0) Previous month: 27.5 (7.9) **Percentiles:** NR **Range (Min, Max):** NR **Monitoring Stations:** NR (used closest monitor to residence to assign exposure 20 year imputed PM_{10} was derived using a space-time model) **Copollutant (correlation):** $PM_{2.5}$	**PM Increment:** 10 $\mu g/m^3$ **Effect Estimate [Lower CI, Upper CI]:** **Adjusted mean differences in log UACR (mg/g) per increase in PM_{10} among participants seen at baseline** **Previous 30 days** Full sample: -0.42 (-0.085, 0.002) Within 10 km: -0.023 (-0.079, 0.034) **Previous 60 days** Full sample: -0.056 (-0.106 to -0.005) Within 10 km: -0.040 (-0.106, 0.025) **20 yr PM_{10} (nearest monitors)** Full sample: -0.019 (-0.072, 0.033) Within 10 km: 0.009 (-0.067, 0.085) **Imputed 20 yr exposure** Full sample: -0.002 (-0.038, 0.035) Within 10 km: 0.016 (-0.033, 0.066) **Adjusted relative prevalence of microalbuminuria vs high-normal and normal levels (below 25 mg/g) per increase in PM_{10} among participants without macroalbuminuria during the baseline visit** Previous 30 days: 0.88 (0.76, 1.02) Previous 60 days: 0.83 (0.70, 0.99) 20 yr PM_{10} (nearest monitors): 0.92 (0.77, 1.08) Imputed 20 yr exposure: 0.98 (0.87, 1.10) **Adjusted mean 3-yr change (SE) in log UACR (mg/g) by quartiles of 1982-2002 exposure to PM_{10} from ambient monitors among participants seen in 2000-20004** **Full sample** Quartile: 18.5 to < 29.3: 0.147 (0.024) 29.3 to < 33.1: 0.159 (0.024) 33.1 to < 36.3: 0.163 (0.024) 36.3 to 55.7: 0.174 (0.023) p-trend: 0.42 **Within 10 km** Quartile: 18.5 to < 29.3: 0.159 (0.030) 29.3 to < 33.1: 0.155 (0.031) 33.1 to < 36.3: 0.167 (0.028) 36.3 to 55.7: 0.152 (0.036) p-trend: 0.99 Interactions with either 20 year or shorter-term PM exposure were not significant ($p < 0.01$) by gender, age, city, race/ethnicity or study site.

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Puett et al, (2008, 156891) **Period of Study:** 1992-2002 **Location:** Northeastern metropolitan US	**Outcome:** Nonfatal myocardial infarction **Study Design:** Cohort **Covariates:** age in months, state of residence, year and season **Statistical Analysis:** Cox proportional hazard **Statistical Package:** SAS **Age Groups:** 30-55	**Pollutant:** PM_{10} **Averaging Time:** 3, 12, 24, 36 and 48 month moving averages **Mean (SD) Unit:** NR **Range (Min, Max):** NR **Copollutant (correlation):** NR	**Increment:** 10 µg/m^3 **Hazard Ratio, 95% CI, 12 month moving average** 0.94 (0.77-1.15)
Reference: Rosenlund et al. (2006, 114678) **Period of Study:** 1992-1994 **Location:** Stockholm County, Sweden	**Outcome (ICD9 and ICD10):** Myocardial infarction (MI) **Age Groups:** 45-70 yrs **Study Design:** Case-control **N:** 1397 cases 1870 controls **Statistical Analyses:** Logistic regression (main analysis) also performed multinomial logistic regression to assess cases as nonfatal, fatal in the hospital within 28 days, and out-of-hospital death within 28 days with all controls as reference **Covariates:** age, sex, and hospital catchment area (frequency matched variables) smoking, physical inactivity, diabetes, SES also assessed but did not include hypertension, BMI, job strain, diet, passive smoking, alcohol consumption, coffee intake, and occupational exposure to motor exhaust and other combustion products **Season:** NA **Dose-response Investigated?** No **Statistical Package:** STATA v8.2	**Pollutant:** PM_{10} (modeled traffic-related pollution also modeled $PM_{2.5}$, but since the PM correlation was high (r = 0.998) only PM_{10} results were presented) (µg/m^3) **Averaging Time:** 30 yrs (PM only assessed during 2000, thus assumed constant levels during 1960-2000) **Median (5th–95th percentile):** **Cases:** 2.6 (0.5-6.0) **Controls:** 2.4 (0.6-5.9) **Range (Min, Max):** NR **Monitoring Stations:** NR **Copollutant (correlation):** NO_2 (r = 0.93) CO (r = 0.66) SO_2	**PM Increment:** 5 μg/m^3 (5th to 95th percentile distribution among controls) **Effect Estimate [Lower CI, Upper CI]:** Association of 30-yr avg exposure to air pollution from traffic with MI Logistic regression All cases: 1.00 (0.79, 1.27) Multinomial logistic regression Nonfatal cases: 0.92 (0.71, 1.19) Fatal cases: 1.39 (0.94, 2.07) In-hospital death: 1.21 (0.75, 1.94) Out-of-hospital death: 1.84 (1.00, 3.40) After adjustment for heating-related SO_2, the estimate for fatal MI was 1.40 (0.86-2.26) for PM_{10}.
Reference: Zanobetti & Schwartz (2007, 091247) **Period of Study:** 1985-1999 **Location:** 21 US cities (Birmingham, Alabama Boulder, Colorado Canton, Ohio Chicago, Illinois Cincinnati, Ohio Cleveland, Ohio Colorado Springs, Colorado Columbus, Ohio Denver, Colorado Detroit, Michigan Honolulu, Hawaii Houston, Texas Minneapolis-St. Paul, Minnesota Nashville, Tennessee	**Outcome (ICD9 and ICD10):** Death, subsequent myocardial infarction (MI ICD9 codes 410.0-410.9), and a first admission for congestive heart failure (CHF ICD9 code 428) **Age Groups:** ≥ 65 yrs **Study Design:** Cohort **N:** 196,000 persons discharged alive following an acute MI **Statistical Analyses:** Cox's Proportional Hazards Regression Meta-regression for city-specific results **Covariates:** age, sex, race, type of MI, number of days of coronary care and intensive care, previous diagnoses for atrial fibrillation, and secondary or previous diagnoses for COPD, diabetes, and hypertension, and for season of initial event (time period, and, sex, race, and type of MI were treated as stratification variables)	**Pollutant:** PM_{10} **Averaging Time:** Yearly averages of pollution for that year and lags up to the 3 previous years (distributed lag) **Mean (SD):** 28.8 (all cities SD not reported) **Percentiles:** 10, 50, and 90 percentiles listed individually for each city (Table 2) **Range (Min, Max):** NR **Monitoring Stations:** NR (obtained data from the US EPA Aerometric Information Retrieval System) **Copollutant (correlation):** None	**PM Increment:** 10 μg/m^3 **Effect Estimate [Lower CI, Upper CI]:** Hazard ratio (95%CI) for an increase in PM for the year of failure and for the distributed lag from the year of failure up to 3 previous years **Death** PM_{10} annual: 1.11 (1.05, 1.19), p = 0.001 Distributed lag model Lag 0: 1.04 (0.96, 1.14), p = 0.336 Lag 1: 1.07 (0.99, 1.14), p = 0.070 Lag 2: 1.14 (1.10, 1.18), p = 0.000 Lag 3: 1.06 (0.99, 1.12), p = 0.077 Sum lags 0-3: 1.34 (1.14, 1.52), p = 0.000 **CHF** PM_{10} annual: 1.11 (1.03, 1.21), p = 0.009

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
New Haven, Connecticut Pittsburgh, Pennsylvania Provo-Orem, Utah Salt Lake City, Utah Seattle, Washington Steubenville, Ohio and Youngstown, Ohio)	**Season:** Assessed as a confounder **Dose-response Investigated?** No **Statistical Package:** NR		Distributed lag model Lag 0: 1.09 (1.01, 1.18), p = 0.030 Lag 1: 1.09 (1.01, 1.19), p = 0.038 Lag 2: 1.13 (1.02, 1.25), p = 0.014 Lag 3: 1.04 (0.97, 1.12), p = 0.260 Sum lags 0-3: 1.41 (1.19, 1.66), p = 0.000 **2nd MI** PM_{10} annual: 1.17 (1.05, 1.31), p = 0.003 Distributed lag model Lag 0: 1.09 (0.92, 1.30), p = 0.325 Lag 1: 1.12 (0.97, 1.30), p = 0.108 Lag 2: 1.15 (1.08, 1.23), p = 0.000 Lag 3: 1.01 (0.94, 1.09), p = 0.783 Sum lags 0-3: 1.43 (1.12, 1.82), p = 0.005 Hazard Ratio (95%CI) for an increase in PM (sum of the previous 3 yrs distributed lag) for the sensitivity analyses **Death** Subjects with follow-up starting after 2nd MI: 1.33 (1.15, 1.55), p = 0.000 Subjects admitted between 1985-1996: 1.45 (1.26, 1.68), p = 0.000 2nd cohort definition (year defined at time of MI): 1.29 (1.15, 1.44), p = 0.000 **CHF** Subjects with follow-up starting after 2nd MI: 1.42 (1.22, 1.65), p = 0.000 Subjects admitted between 1985-1996: 1.51 (1.26, 1.81), p = 0.000 **2nd MI** Subjects admitted between 1985-1996: 1.62 (1.23, 2.13), p = 0.001 **Note:** Age and sex effect modification results presented in Figure 1 used meta-regression to examine predictors of heterogeneity across city and found that most predictors were not significant modifiers of PM (Table 7)

[1]All units expressed in $\mu g/m^3$ unless otherwise specified.

Table E-21. Long-term effects–cardiovascular– $PM_{2.5}$ (including PM components/sources)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Allen et al. (2009, 189644)	**Outcome (ICD9 and ICD10):** Abdominal	**Pollutant:** $PM_{2.5}$ ($\mu g/m^3$)	**PM Increment:** 10 $\mu g/m^3$

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Period of Study: Oct 2000–Sep 2002 (exposure averaging period) outcome assessed in 2002 **Location:** 5 US communities (Chicago, Illinois Forsyth County, North Carolina Los Angeles, California Northern Manhattan and the Bronx, New York and St. Paul, Minnesota) part of MESA (Multi-ethnic Study of Atherosclerosis)	aortic calcium (AAC), a marker of systemic atherosclerosis (quantitative measure of interest was the Agatston score) **Age Groups:** 46-88 years **Study Design:** Cross-sectional **N:** 1,147 participants (sensitivity analysis among 1,269 participants) **Statistical Analyses:** 2-part modeling approach: 1) modeled relative risk of having any AAC using a log link and a Gaussian error model sensitivity analysis used modified Poisson regression with robust error variance 2) multiple linear regression of the log-transformed AAC Agatston score (among those with AAC > 0) sensitivity analysis modeled all participants by adding 1 prior to log-transforming **Covariates:** age, gender, race/ethnicity, BMI, smoking status, pack-year of smoking, diabetes, education, annual income, blood lipid concentration, blood pressure, and medications assessed impact of gender, age, diabetes, obesity, use of lipid-lowering medications, education, income, race/ethnicity, and employment status on heterogeneity of effects (or in sensitivity analyses) **Season:** NA **Dose-response Investigated?** NR **Statistical Package:** SAS v9.1	**Averaging Time:** 2 year averaging period (Oct 2000–Sep 2002) **Mean (SD):** 15.8 (3.6) $\mu g/m^3$ **Percentiles:** NR **Range (Min, Max):** 10.6–24.7 $\mu g/m^3$ **Monitoring Stations:** All monitors with 1) the objective of "population exposure," "regional transport," or "general/background;) and 2) at least 50% data reporting in each of 8 3-month periods over the averaging time used monitors located within 50 km of a study participant's residence **Copollutant (correlation):** (assessed traffic by roadway proximity)	**Effect Estimate [Lower CI, Upper CI]:** Results for fully adjusted models under different participant inclusion, employment status, and roadway proximity criteria. **Presence/Absence of Calcium** **RR (95% CI)** Inclusion criteria: < 10yrs at address: 1.04 (0.89, 1.22) ≥ 10yrs at address: 1.06 (0.96, 1.16) ≥ 10yrs at address & < 10km from monitor: 1.08 (0.98, 1.18) ≥ 20yrs at address: 1.10 (0.99, 1.22) ≥ 20yrs at address & < 10km from monitor: 1.11 (1.00, 1.24) < 10yrs at address & employed: 1.02 (0.87, 1.20) ≥ 20yrs at address & employed: 1.07 (0.89, 1.27) < 10yrs at address & not employed: 1.10 (1.00, 1.22) ≥ 20yrs at address & not employed: 1.16 (1.02, 1.31) < 10yrs at address & near major road: 0.85 (0.69, 1.05) ≥ 20yrs at address & not near major road: 1.10 (0.99, 1.23) **Log-transformed Agatston Score (Agatston > 0)** **% Change (95% CI)** Inclusion criteria: < 10yrs at address: -6.6 (-64.0, 50.9) ≥ 10yrs at address: 8.0 (-29.7, 45.7) ≥ 10yrs at address & < 10km from monitor: 19.7 (-19.6, 58.9) ≥ 20yrs at address: 14.4 (-32.8, 61.7) ≥ 20yrs at address & < 10km from monitor: 24.6 (-24.6, 73.8) < 10yrs at address & employed: 29.1 (-25.7, 83.8) ≥ 20yrs at address & employed: 43.8 (-32.4, 119.9) < 10yrs at address & not employed: -15.1 (-66.3, 36.1) ≥ 20yrs at address & not employed: -14.1 (-72.6, 44.4) < 10yrs at address & near major road: 34.0 (-44.2, 112.1) ≥ 20yrs at address & not near major road: 3.9 (-39.9, 47.8) **Log-transformed Agatston Score (all)** **% Change (95% CI)** Inclusion criteria: < 10yrs at address: -8.5 (-81.3, 64.2) ≥ 10yrs at address: 40.7 (-11.5, 92.8)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			≥ 10yrs at address & < 10km from monitor: 60.7 (5.9, 115.4) ≥ 20yrs at address: 64.1 (-1.73, 129.9) ≥ 20yrs at address & < 10km from monitor: 79.2 (10.1, 148.3) < 10yrs at address & employed: 33.5 (-35.9, 102.9) ≥ 20yrs at address & employed: 55.8 (-37.2, 148.7) < 10yrs at address & not employed: 54.8 (-23.8, 133.4) ≥ 20yrs at address & not employed: 89.3 (-3.7, 182.3) < 10yrs at address & near major road: -30.6 (-141.3, 80.1) ≥ 20yrs at address & not near major road: 51.3 (-8.3, 110.8) **Exploratory/sensitivity analyses (also presented in figures): Detectable AAC** **RR (95%CI):** Among women: 1.14 (1.00, 1.30) Among persons > 65yrs: 1.10 (1.01, 1.19) Among users of lipid-lowering medications: 1.14 (1.00, 1.30) Among Hispanics: 1.22 (1.03, 1.45) Imputing missing covariates among residentially stable participants: 1.08 (0.98, 1.19) **Agatston score** **% change (95%CI):** Among Hispanics: 64 (-4, 133) Among persons earning > $50,000: 72 (5, 139) **Agatston score including those with Agatston = 0** **% change (95%CI):** Fully adjusted model: 41 (-12, 93) Among persons > 65yrs: 75 (8, 143) Among diabetics: 149 (29, 270) Among users of lipid-lowering medications: 121 (25, 217) Among Hispanics: 141 (45, 236) Imputing missing **Covariates:** 49 (1.3, 100.1)
Reference: Auchincloss et al. (2008, 156234) **Period of Study:** Jul 2000–Aug 2002 **Location:** 6 US communities (Baltimore City and Baltimore County, Maryland Chicago, Illinois Forsyth County, North Carolina Los Angeles, California Northern Manhattan and the Bronx, New York	**Outcome (ICD9 and ICD10):** Blood pressure: systolic (SBP), diastolic (DBP), mean arterial (MAP), pulse pressure (PP) Avg of 2nd and 3rd BP measurement used for analyses **Age Groups:** 45-84 years **Study Design:** Cross-sectional (Multi-Ethnic Study of Atherosclerosis baseline examination) **N:** 5,112 persons (free of clinically apparent cardiovascular disease)	**Pollutant:** $PM_{2.5}$ ($\mu g/m^3$) **Averaging Time:** 5 exposure metrics constructed: prior day, avg of prior 2 days, prior 7 days, prior 30 days, and prior 60 days **Mean (SD):** Prior day: 17.0 (10.5) Prior 2 days: 16.8 (9.3) Prior 7 days: 17.0 (6.9) Prior 30 days: 16.8 (5.0) Prior 60 days: 16.7 (4.4)	**PM Increment:** 10 $\mu g/m^3$ (approx. equivalent to difference between 90th and 10th percentile for prior 30 day mean) **Effect Estimate [Lower CI, Upper CI]:** Adjusted mean difference (95% CI) in PP and SBP (mmHg) per 10 $\mu g/m^3$ increase in $PM_{2.5}$ (averaged for the prior 30 days) **Pulse Pressure** Adjustment variables: Person-level **Covariates:** 1.04 (0.25, 1.84) Person-level cov., weather: 1.12 (0.28,

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
and St. Paul, Minnesota) part of MESA (Multi-ethnic Study of Atherosclerosis)	**Statistical Analyses:** Linear regression secondary analyses used log binomial models to fit a binary hypertension outcome **Covariates:** age, sex, race/ethnicity, per capita family income, education, BMI, diabetes status, cigarette smoking status, exposure to ETS, high alcohol use, physical activity, BP medication use, meteorology variables, and copollutants examined site as a potential confounder and effect modifier heterogeneity of effects also examined by traffic-related exposures, age, sex, type 2 diabetes, hypertensive status, cigarette use **Season:** Adjusted for temperature and barometric pressure to adjust for seasonality (because seasons vary by the study sites) Also performed sensitivity analyses adjusting for season to examine the potential for residual confounding not accounted for by weather variables **Dose-response Investigated?** Assessed nonlinear relationships–no evidence of strong threshold/nonlinear effects for $PM_{2.5}$ **Statistical Package:** NR	**Percentiles:** NR **Range (Min, Max):** NR **Monitoring Stations:** Used monitor nearest the participant's residence to calculate exposure metrics **Copollutant (correlation):** SO_2 NO_2 CO Traffic-related exposures (straight-line distance to a highway total road length around a residence)	1.97) Person-level cov., weather, gaseous copollutants: 2.66 (1.61, 3.71) Person-level cov., study site: 0.93 (-0.04, 1.90) Person-level cov., study site, weather: 1.11 (0.01, 2.22) Person-level cov., study site, weather, gaseous copollutants: 1.34 (0.10, 2.59) **Systolic Blood Pressure** Adjustment variables: Person-level **Covariates:** 0.66 (-0.41, 1.74) Person-level cov., weather: 0.99 (-0.15, 2.13) Person-level cov., weather, gaseous copollutants: 2.8 (1.38, 4.22) Person-level cov., study site: 0.86 (-0.45, 2.17) Person-level cov., study site, weather: 1.32 (-0.18, 2.82) Person-level cov., study site, weather, gaseous copollutants: 1.52 (-0.16, 3.21) **Additional results:** Associations became stronger with longer averaging periods up to 30 days. For example: Adjusted (personal covariates and weather) mean differences in PP: Prior day: -0.38 (-0.76, 0.00) Prior 2 days: -0.22 (-0.65, 0.21) Prior 7 days: 0.52 (-0.08, 1.11) Prior 30 days: 1.12 (0.28, 1.97) Prior 60 days: 1.08 (0.11, 2.05) (Pattern held for additional adjustments and for SBP results therefore, only results for 30-day mean differences were presented) **Additional results (not presented):** None of DBP results were statistically significant results for MAP were similar to SBP, though weaker and generally not significant Effect modification: associations between $PM_{2.5}$ and BP were stronger for persons taking medications, with hypertension, during warmer weather, in the presence of high NO_2, residing ≤ 300m from a highway, and surrounded by a high density of roads (Fig 1) associations were not modified for age, sex, diabetes, cigarette smoking, study site, high levels of CO or SO_2, season , nor residence ≤ 400m fro a highway **Note:** supplementary material available on-line
Reference: Calderón-Garcidueñas et al. (2009, 192107) **Period of Study:** 9/11/2004-1/6/2005 **Location:** Mexico City and Polotitlan,	**Outcome:** Flow cytometry **Study Design:** Panel **Covariates:** NR **Statistical Analysis:** Pearson's	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 1, 2 and 7 day averages **Mean (SD) Unit:** 35.89 ± 0.93 $\mu g/m^3$	**Increment:** NR **Flow cytometry results and their statistical significance in control versus exposed children**

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Mexico	Correlation **Statistical Package:** Stata **Age Groups:** 9.7 ± 1.2 years	**Range (Min, Max):** NR **Copollutant:** PM_{10}, O_3	CD3 Exposed: 62.9 ± 1.8 Control: 67.1 ± 1.7 P = 0.1 CD4 Exposed: 39.3 ± 1.3 Control: 38.2 ± 1.4 P = 0.57 CD8 Exposed: 24.0 ± 0.95 Control: 27.3 ± 1.0 P = 0.02 CD4/CD8 Exposed: 1.7 ± 0.14 Control: 1.4 ± 0.07 P = 0.09 CD3-/CD19+ Exposed: 11.8 ± 1.0 Control: 14.8 ± 1.0 P = 0.04 CD56+ Exposed: 11.5 ± 1.2 Control: 12.4 ± 1.5 P = 0.63 CD56+/CD3-NK Exposed: 14.0 ± 9.5 Control: 7.0 ± 2.7 P = 0.003 HLA-DR+ Exposed: 27.5 ± 4.2 Control: 17.0 ± 2.4 P = 0.04 mCD14+ Exposed: 66.5 ± 2.3 Control: 80.6 ± 1.8 P = < 0.001 CD14/CD69 Exposed: 0.20 ± 0.07 Control: 1.0 ± 0.26 P = < 0.001 CD4/CD69 Exposed: 0.08 ± 0.03 Control: 3.1 ± 0.65 P = < 0.001

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Diez Roux et al. (2008, 156401) **Period of Study:** Baseline data collected June 2000–Aug 2002 Exposure assessed retrospectively between Aug 1982 and baseline date **Location:** USA (6 field centers: Baltimore, MD Chicago, IL Forsyth Co, NC Los Angeles, CA New York, NY St. Paul, MN	**Outcome (ICD9 and ICD10):** Three measures of subclinical atherosclerosis (common carotid intimal-medial thickness (CIMT), coronary artery calcification, and ankle-brachial index (ABI)) **Age Groups:** 44-84 yrs **Study Design:** Cross-sectional **N:** 5172 for coronary calcium analysis 5037 for CIMT analysis 5110 for ABI analysis **Statistical Analyses:** Generalized Additive Models (Binomial regression: presence of calcification Linear regression: CIMT, ABI, amount of calcium) **Covariates:** age, sex, race/ethnicity, socioeconomic factors, cardiovascular risk factors (BMI, hypertension, high density lipoprotein and low density lipoprotein cholesterol, smoking, diabetes, diet, physical activity models presented with and without adjustment for cardiovascular RFs) **Season:** NA **Dose-response Investigated?** No **Statistical Package:** NR	**Pollutant:** $PM_{2.5}$ ($\mu g/m^3$) **Averaging Time:** 20-yr imputed mean **Mean (SD):** 21.7 (5.0) **Percentiles:** NR **Range (Min, Max):** NR **Monitoring Stations:** NR Long-term exposure to PM estimated based on residential history reported retrospectively all addresses geocoded ambient AP obtained from US EPA **Copollutant (correlation):** PM_{10} 20-yr observed mean r = 0.64 PM_{10} 20-yr imputed mean r = 0.73 PM_{10} 2001 mean r = 0.43 $PM_{2.5}$ 2001 mean r = 0.64 Due to high correlation among PM exposures, only results of mean 20-yr exposures are reported.	**PM Increment:** 12.5 $\mu g/m^3$ (approx. 10th-90th percentile) **Effect Estimate [Lower CI, Upper CI]:** **CIMT:** Relative difference (95% CI): 1.01 (1.00, 1.01) Adj. for additional CVD RFs: 1.01 (1.00, 1.02) **ABI:** Mean difference (95% CI): 0.000 (-0.006, 0.006) Adj. for additional CVD RFs: -0.001 (-0.006, 0.006) **Coronary calcium:** Relative prevalence (95% CI): 1.01 (0.96, 1.05) Adj. for additional CVD RFs: 1.01 (0.96, 1.06) **Coronary calcium (in those with calcium):** Relative difference (95% CI): 0.99 (0.88, 1.12) Adj. for additional CVD RFs: 1.01 (0.89, 1.14)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Hoffman et al. (2007, 091163) **Period of Study:** 2000-2003 **Location:** Ruhr area of Germany (3 large cities: Essen, Mulheim, and Bochum)	**Outcome (ICD9 and ICD10):** Coronary artery calcification (CAC) **Age Groups:** 45-74 years **Study Design:** Cross-sectional **N:** 4494 participants **Statistical Analyses:** Linear regression (outcome = natural logarithm of CAC score + 1) logistic regression (outcome = CAC score above/below the age- and gender-specific 75th percentile) **Covariates:** city and area of residence, age, sex, education, smoking, ETS, physical inactivity, waist-to-hip ratio, diabetes, blood pressure, and lipids (and household income in a subset) **Season:** NA **Dose-response Investigated?** Yes, PM was also categorized into quartiles for analyses **Statistical Package:** NR	**Pollutant:** $PM_{2.5}$ ($\mu g/m^3$) **Averaging Time:** One year (2002, midpoint of the study) **Mean (SD):** Total: 22.8 (1.5) High traffic exposure (≤ 100m): 22.9 (1.4) Low traffic exposure (> 100m): 22.8 (1.5) **Percentiles:** Q1: 21.54 Q2: 22.59 Q3: 23.75 10^{th}-90^{th} percentile: 3.91 **Range (Min, Max):** NR **Monitoring Stations:** Daily mean $PM_{2.5}$ values for 2002 were estimated with the EURAD model using data from official emission inventories, meteorological information, and regional topographical data. **Copollutant (correlation):** None (Traffic was assessed using distance to roadways) Correlation between modeled daily averages of $PM_{2.5}$ and measured $PM_{2.5}$: 0.86-0.88, depending on season.	**PM Increment:** 3.91 $\mu g/m^3$ (10th-90th percentile) **Effect Estimate [Lower CI, Upper CI]:** **Percent change (95%CI) in CAC associated with an increase in** $PM_{2.5}$ Unadjusted: 12.7 (-7.0, 36.4) Model 1 (adjusted for distance to major road): 12.3 (-7.3, 35.9) Model 2 (model 1 + city and area of residence): 29.7 (0, 68.3) Model 3 (model 2 + age, sex, education): 24.2 (0, 55.1) Model 4 (model 3 + smoking, ETS, physical inactivity, waist-to-hip ratio): 17.9 (-5.3, 46.7) Model 5 (model 4 + diabetes, blood pressure, LDL, HDL, triglycerides): 17.2 (-5.6, 45.5) **Adjusted ORs (95%CI) for the association between the top quarter of PM exposure vs. the low quarter of PM exposure and a CAC score above the age- and sex-specific 75th percentiles** All: 1.22 (0.96, 1.54) No CHD: 1.22 (0.95, 1.57) Men: 1.09 (0.78, 1.53) Women: 1.34 (0.97, 1.87) Age < 60 yrs: 1.18 (0.83, 1.68) Age > 60 yrs: 1.27 (0.93, 1.75) Nonsmokers: 1.17 (0.89, 1.53) Current smokers: 1.30 (0.83, 2.05) Educational level Low: 1.16 (0.86, 1.57) Medium: 1.30 (0.83, 2.05) High: 1.62 (0.81, 3.25) **Additional notes:** No clear dose-response relationship demonstrated when exposure assessed in quartiles (Figure 2) Participants who had not been working full-time during the last 5 years showed stronger effects, with possible dose-response between $PM_{2.5}$ and CAC (results presented in Figure 3)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Hoffman et al. (2006, 091162) **Period of Study:** Dec 2000–Jul 2003 **Location:** Ruhr area of Germany (2 large cities: Essen, Mulheim)	**Outcome (ICD9 and ICD10):** Clinically manifest CHD (defined as self-reported history of a 'hard' coronary event, i.e. myocardial infarction or application of a coronary stent or angioplasty or bypass surgery) **Age Groups:** 45-75 years **Study Design:** Cross-sectional (German Heinz Nixdorf RBCALL study) **N:** 3399 participants **Statistical Analyses:** Multivariable logistic regression **Covariates:** sex, diabetes, hypertension, smoking status, ETS, educational level, physical activity, BMI, triglycerides, age, cigarettes smoked per day, WHR, LDL, HDL, HbAlc, indicator variable for cities, indicator variable for living in northern part of cities. **Statistical Package:** SAS v8.2	**Pollutant:** $PM_{2.5}$ ($\mu g/m^3$) **Averaging Time:** Yearly mean estimated with model for year 2002 (on a spatial scale of 5 km) **Mean (SD):** Total: 23.3 (1.4) High traffic: 23.4 (1.4) Low traffic: 23.3 (1.4) **Percentiles:** NR **Range (Min, Max):** NR **Monitoring Stations:** NR **Copollutant (correlation):** None (Traffic was assessed using distance to roadways)	**Effect Estimate [Lower CI, Upper CI]:** Model 1: $PM_{2.5}$ + high traffic exposure 0.92 (0.36, 2.39) Model 2: model 1 + age, sex 0.83 (0.31, 2.27) Model 3: model 2 + education, diabetes, HbAlc, BMI, WHR, smoking status, ETS, physical activity, city, area of residence 0.56 (0.16, 2.01) Model 4: model 3 + hypertension, lipids 0.55 (0.14, 2.11) Modeled vs. Measured: r = 0.86-0.88, depending on season
Reference: Hoffmann et al, (2009, 190376) **Period of Study:** 2000-2003 **Location:** Ruhr area, Germany	**Outcome:** Peripheral Arterial Disease **Study Design:** **Covariates:** height, weight, medication use, diabetes, physical activity level, smoking, socioeconomic status, education, population density **Statistical Analysis:** NR **Statistical Package:** NR **Age Groups:** 45-75 years	**Pollutant:** $PM_{2.5}$ **Averaging Time:** daily **Mean (SD) Unit:** 22.96 (0.85) **Range (Min, Max):** NR **Copollutant (correlation):** NR	**Increment:** 3.91 $\mu g/m^3$ **Odds Ratio (95%CI) for prevalence of peripheral arterial disease** 0.87 (0.57-1.34)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Kunzli et al. (2005, 087387) **Period of Study:** 1998-2003 **Location:** Los Angeles Basin	**Outcome (ICD9 and ICD10):** Carotid intima-media thickness (CIMT) **Age Groups:** Less than 40 yrs excluded mean age = 59.2 ± 9.8 **Study Design:** Cross-sectional **N:** 798 participants **Statistical Analyses:** Linear regression **Covariates:** age, sex, education, income, smoking, ETS, blood pressure, LDL cholesterol, treatment with antihypertensives or lipid-lowering medications **Season:** NA **Dose-response Investigated?** Yes, assessed $PM_{2.5}$ in quartiles **Statistical Package:** NR	**Pollutant:** $PM_{2.5}$ ($\mu g/m^3$) **Averaging Time:** GIS/geostatics model to estimate 'long-term mean ambient concentrations of $PM_{2.5}$' derived from data collected in 2000, including data from 23 state and local monitoring stations. **Mean (SD):** 20.3 ± 2.6 **Percentiles:** NR **Range (Min, Max):** 5.2, 26.9 **Monitoring Stations:** 23 monitors **Copollutant (correlation):** None	**PM Increment:** 10 $\mu g/m^3$ **Effect Estimate [Lower CI, Upper CI]:** Percent change (95%CI) in CIMT associated with an increase in $PM_{2.5}$ concentration based on a linear model with log intima-media thickness as dependent variable **Total population:** Unadjusted: 5.9 (1.0, 10.9) , p = 0.018 Adjusted for age, sex, education, income: 4.4 (0.0, 9.0) , p = 0.056 Adjusted for above + smoking, ETS, multivitamins, alcohol: 4.2 (-0.2, 8.9) , p = 0.064 **Among Females ≥ 60 years:** Unadjusted: 19.2 (8.8, 30.5) , p = 0.001 Adjusted for age, sex, education, income: 15.7 (5.7, 26.6) , p = 0.002 Adjusted for above + smoking, ETS, multivitamins, alcohol: 13.8 (4.0, 24.5) , p = 0.002 **Among those taking lipid-lowering therapy:** Unadjusted: 15.8 (2.1, 31.2) , p = 0.024 Adjusted for age, sex, education, income: 13.3 (0, 28.5) , p = 0.031 Adjusted for above + smoking, ETS, multivitamins, alcohol: 13.3 (-0.3, 28.8) , p = 0.060 **For the observed contrast between lowest and highest exposure:** Approximately 20 $\mu g/m^3$ → 12.1% (2.0-231%) increase in CIMT. Among nonsmokers: 6.6% (1.0-12.3%). The estimate was small and not significant in current and former smokers. Women: In the range of 6-9% per 10$\mu g/m^3$ Unadjusted means of CIMT across quartiles of exposure were 734, 753, 758, and 774 μm adjusted means trend across exposure groups, p = 0.041 stratified results presented in figures
Reference: Miller et al. (2007, 090130) **Period of Study:** 1994-2003 **Location:** 36 US metropolitan areas (Women's Health Initiative)	**Outcome (ICD9 and ICD10):** First cardiovascular event (myocardial infarction, coronary revascularization, stroke, and death from either coronary heart disease [categorized as "definite" or "possible"] or cerebrovascular disease) **Age Groups:** 50-79 years (median age at enrollment: 63) **Study Design:** Cohort (median follow-up of 6 yrs)	**Pollutant:** $PM_{2.5}$ ($\mu g/m^3$) **Averaging Time:** Annual avg concentration in 2000 (used to represent long-term exposure) **Mean (SD):** Individual exposure: 13.5 (3.7) Citywide avg exposure: 13.5 (3.3) Median: 13.4	**PM Increment: 10** $\mu g/m^3$ **Effect Estimate [Lower CI, Upper CI]:** Estimated Hazards Ratio (95%CI) for the time to the first cardiovascular event or death associated with an increase in $PM_{2.5}$ Any cardiovascular event (first event) Overall: 1.24 (1.09, 1.41)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
	N: 65,893 postmenopausal women without previous cardiovascular disease **Statistical Analyses:** Cox-proportional hazards regression **Covariates:** age, race/ethnicity, smoking status, the number of cigarettes smoked per day, the number of years of smoking, systolic blood pressure, education level, household income, BMI, and presence or absence of diabetes, hypertension, or hypercholesterolemia (also evaluated ETS, occupation, physical activity, diet, alcohol consumption, waist circumference, waist-to-hip ratio, medical history, medications, and presence or absence of a family history of cardiovascular disease as possible confounders in extended models) **Season:** NA **Dose-response Investigated?** **Statistical Package:** SAS v8.0, STATA v8.0	**Percentiles:** Quintile ranges: 1: 3.4, 10.9 2: 11.0, 12.4 3: 12.5, 14.2 4: 14.3, 16.4 5: 16.5, 28.3 IQR: 11.6-18.3 10^{th}-90^{th} Personal: 9.1-18.3 City-wide: 9.3-17.8 **Range (Min, Max):** Personal exposure: 3.4, 28.3 Citywide exposure: 4.0, 19.3 **Monitoring Stations:** 573 monitors the nearest monitor to the location of each residence was used to assign exposure (monitor within 30 mi of residence median of 20 monitors per city (range: 4-78)) **Copollutant (correlation):** PM_{10} SO_2 NO_2 CO O_3	Between cities: 1.15 (0.99, 1.32) Within cities: 1.64 (1.24, 2.18) Coronary heart disease (first event): Overall: 1.21 (1.04, 1.42) Between cities: 1.13 (0.95, 1.35) Within cities: 1.56 (1.11, 2.19) Cerebrovascular disease (first event): Overall: 1.35 (1.08, 1.68); Between cities: 1.20 (0.94, 1.54) Within cities: 2.08 (1.28, 3.40) MI (first event): Overall: 1.06 (0.85, 1.34) Between cities: 0.97 (0.75, 1.25) Within cities: 1.52 (0.91, 2.51) Coronary revascularization (first event): Overall: 1.20 (1.00, 1.43) Between cities: 1.14 (0.93, 1.39) Within cities: 1.45 (0.98, 2.16) Stroke (first event): Overall: 1.28 (1.02, 1.61) Between cities: 1.12 (0.87, 1.45) Within cities: 2.08 (1.25, 3.48) Any death from cardiovascular cause: Overall: 1.76 (1.25, 2.47) Between cities: 1.63 (1.10, 2.40) Within cities: 2.28 (1.10, 4.75) Coronary heart disease death (definite diagnosis): Overall: 2.21 (1.17, 4.16) Between cities: 2.22 (1.06, 4.62) Within cities: 2.17 (0.60, 7.89) Coronary heart disease death (possible diagnosis): Overall: 1.26 (0.62, 2.56) Between cities: 1.20 (0.54, 2.63) Within cities: 1.57 (0.29, 8.51) Cerebrovascular disease death: Overall: 1.83 (1.11, 3.00) Between cities: 1.58 (0.90, 2.78) Within cities: 2.93 (1.03, 8.38) Estimated Hazard Ratios for cardiovascular events associated with an increase in $PM_{2.5}$ according to selected characteristics (presented adjusted H and adjusted H including adjustment for city) Any cardiovascular event: H: 1.24 (1.09, 1.41) H (city): 1.69 (1.26, 2.27) Household income $<$ \$20,000: H: 1.30 (1.10, 1.53) H (city): 1.75 (1.28, 2.40) Household income \$20,000-49,999: H: 1.23 (1.08, 1.41) H (city): 1.69 (1.25, 2.27) Household income $\geq$ \$50,000: H: 1.20

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			(1.02, 1.40)
			6
			H (city): 1.66 (1.22, 2.26)
			P for trend: HR: p = 0.34
			HR (city): p = 0.54
			Education: Not high-school graduate: H: 1.40 (1.11, 1.75)
			H (city): 1.88 (1.32, 2.67)
			Education: High school grad/trade school/GED: H: 1.33 (1.14, 1.55)
			H (city): 1.79 (1.32, 2.44)
			Education: Some college or associate degree: H: 1.26 (1.09, 1.44)
			H (city): 1.74 (1.29, 2.34)
			Education: Bachelor's degree or higher: H: 1.11 (0.94, 1.31)
			H (city): 1.54 (1.13, 2.10)
			P for trend: H: p = 0.07
			H (city): p = 0.15
			Age < 60 yr: H: 1.21 (0.84, 1.73)
			H (city): 1.66 (1.05, 2.61)
			Age 60-69 yr: H: 1.14 (0.93, 1.39)
			H (city): 1.53 (1.09, 2.14)
			Age ≥ 70 yr: H: 1.34 (1.11, 1.63)
			H (city): 1.85 (1.34, 2.56)
			P for trend: H: p = 0.20
			H (city): p = 0.20
			Current smoker: H: 1.68 (1.06, 2.66)
			H (city): 2.28 (1.33, 3.92)
			Former smoker: H: 1.24 (1.01, 1.52)
			H (city): 1.71 (1.23, 2.39)
			Never smoked: H: 1.18 (0.99, 1.40)
			H (city): 1.60 (1.16, 2.21)
			Living with smoker currently: H: 1.28 (0.84, 1.97)
			H (city): 1.65 (0.99, 2.76)
			Living with smoker formerly: H: 1.18 (1.00, 1.38)
			H (city): 1.59 (1.16, 2.16)
			Living with smoker never: H: 1.39 (1.07, 1.80)
			H (city): 1.90 (1.31, 2.78)
			BMI < 22.5: H: 0.99 (0.80, 1.21)
			H (city): 1.35 (0.96, 1.88)
			BMI 22.5-24.7: H: 1.16 (0.96, 1.40)
			H (city): 1.58 (1.14, 2.19)
			BMI 24.8-27.2: H: 1.24 (1.05, 1.45)
			H (city): 1.69 (1.24, 2.30)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			BMI 27.3-30.9: H: 1.38 (1.18, 1.61)
			H (city): 1.88 (1.38, 2.56)
			BMI > 30.9: H: 1.35 (1.12, 1.64)
			H (city): 1.84 (1.33, 2.55)
			P for trend: H: p = 0.003
			H (city): p = 0.007
			Waist-to-hip ratio < 0.74: H: 1.07 (0.90, 1.29)
			H (city): 1.45 (1.05, 2.00)
			Waist-to-hip ratio 0.74-0.77: H: 1.12 (0.95, 1.31)
			H (city): 1.51 (1.11, 2.06)
			Waist-to-hip ratio 0.78-0.80: H: 1.24 (1.07, 1.44)
			H (city): 1.68 (1.23, 2.27)
			Waist-to-hip ratio 0.81-0.86: H: 1.30 (1.13, 1.50)
			H (city): 1.76 (1.30, 2.38)
			Waist-to-hip ratio > 0.86: H: 1.29 (1.11, 1.50)
			H (city): 1.75 (1.29, 2.37)
			Waist circumference < 73 cm: H: 1.05 (0.86, 1.27)
			H (city): 1.43 (1.02, 1.99)
			Waist circumference 73-78 cm: H: 1.20 (1.02, 1.41)
			H (city): 1.63 (1.19, 2.23)
			Waist circumference 79-85 cm: H: 1.22 (1.05, 1.41)
			H (city): 1.66 (1.22, 2.24)
			Waist circumference 86-95 cm: H: 1.33 (1.15, 1.53)
			H (city): 1.80 (1.33, 2.43)
			Waist circumference > 95 cm: H: 1.27 (1.07, 1.51)
			H (city): 1.73 (1.26, 2.36)
			P for trend: H: p = 0.06
			H (city): p = 0.07
			Hormone-replacement therapy-Current Use: H: 1.33 (1.09, 1.61)
			H (city): 1.85 (1.32, 2.58)
			Hormone-replacement therapy-No Current Use: H: 1.16 (0.98, 1.39)
			H (city): 1.57 (1.14, 2.17)
			Diabetes–yes: H: 0.96 (0.67, 1.37)
			H (city): 1.24 (0.78, 1.96)
			Diabetes–no: H: 1.28 (1.12, 1.47)
			H (city): 1.75 (1.30, 2.36)
			Hypertension–yes: H: 1.22 (1.02, 1.45)
			H (city): 1.65 (1.09, 2.27)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			Hypertension–no: H: 1.26 (1.05, 1.51)
			H (city): 1.74 (1.25, 2.40)
			Hypercholesterolemia–yes: H: 1.25 (0.94, 1.67)
			H (city): 1.71 (1.15, 2.54)
			Hypercholesterolemia–no: H: 1.23 (1.07, 1.42)
			H (city): 1.69 (1.25, 2.28)
			Family history of CVD– yes: H: 1.30 (1.12, 1.51)
			H (city): 1.80 (1.32, 2.44)
			Family history of CVD– no: H: 1.07 (0.83, 1.37)
			H (city): 1.46 (1.00, 2.12)
			Time lived in current state: ≥ 20 yr: H: 1.21 (1.06, 1.39)
			H (city): 1.66 (1.23, 2.23)
			Time lived in current state: 10-19 yr: H: 1.39 (1.12, 1.72)' H (city): 1.97 (1.40, 2.79)
			Time lived in current state: ≤ 9 yr: H: 1.54 (1.06, 2.26)
			H (city): 2.24 (1.39, 3.59)
			Health insurance coverage–yes: H: 1.22 (1.07, 1.39)
			H (city): 1.71 (1.27, 2.30)
			Health insurance coverage–no: H: 1.82 (0.81, 4.10)
			H (city): 2.65 (1.12, 6.28)
			Time spent outdoors: < 30 min: H: 1.09 (0.86, 1.39)
			H (city): 1.56 (1.05, 2.31)
			Time spent outdoors: ≥ 30 min
			H: 1.26 (1.05, 1.50)
			H (city): 1.82 (1.29, 2.57)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: O'Neill et al. (2007, 156006) **Period of Study:** 2000-2004 **Location:** USA (6 field centers: Baltimore, MD Chicago, IL Forsyth Co, NC Los Angeles, CA New York, NY St. Paul, MN	**Outcome (ICD9 and ICD10):** Creatinine adjusted urinary albumin excretion Assessed 2 ways: continuous log urinary albumin/creatine ration (UACR) and clinically defined micro- or macro-albuminuria (UACR ≥ 25 mg/g) versus normal levels **Age Groups:** 44-84 yrs **Study Design:** Prospective cohort analyses (MESA cohort) **N:** 3901 participants, free of clinical CVD at baseline **Statistical Analyses:** Multiple linear regression (continuous outcome) binomial regression (dichotomous outcome) **Covariates:** age, gender, race, BMI, cigarette status, ETS, percent dietary protein **Season:** NA **Dose-response Investigated?** Yes, examined quartiles of exposure **Statistical Package:** SAS	**Pollutant:** $PM_{2.5}$ ($\mu g/m^3$) **Averaging Time:** avg of previous month, avg of previous 2 months (recent exposures) 20-yr imputed $PM_{2.5}$ avg (longer-term exposures) **Mean (SD):** Previous month: 16.5 (4.8) **Percentiles:** NR **Range (Min, Max):** NR **Monitoring Stations:** NR (used closest monitor to residence to assign value for recent exposures 20 year $PM_{2.5}$ exposures were imputed using a space-time model.) **Copollutant (correlation):** PM_{10}	**PM Increment:** 10 $\mu g/m^3$ **Effect Estimate [Lower CI, Upper CI]:** **Adjusted mean differences in log UACR (mg/g) per increase in $PM_{2.5}$ among participants seen at baseline** **Previous 30 days** Full sample: -0.017 (-0.087, 0.052) Within 10 km: 0.026 (-0.067, 0.119) **Previous 60 days** Full sample: -0.040 (-0.121, 0.042) Within 10 km: -0.013 (-0.122, 0.097) **Imputed 20 yr exposure** Full sample: 0.002 (-0.048, 0.052) Within 10 km: -0.012 (-0.076, 0.053) **Adjusted relative prevalence of microalbuminuria vs high-normal and normal levels (below 25 mg/g) per increase in $PM_{2.5}$ among participants without macroalbuminuria during the baseline visit** Previous 30 days: 0.94 (0.77, 1.16) Previous 60 days: 0.90 (0.71, 1.14) Imputed 20 yr exposure: 0.98 (0.84, 1.14)
Reference: Solomon et al. (2003, 156994) **Period of Study:** Exposures measures 1966-1969 Health endpoints assessed via questionnaire, year not reported but apparently 30 years after exposure assessment (given the 30 yr residency requirement) **Location:** United Kingdom	**Outcome (ICD9 and ICD10):** Ischemic heart disease (a self-reported history of medically diagnosed angina or heart attack) **Age Groups:** 45 yrs and older **Study Design:** Cross-sectional **N:** 1,166 women **Statistical Analyses:** Log linear modeling **Covariates:** smoking, passive smoking in childhood, tenancy, social class, worked in industry with respiratory hazard, childhood hospital admission for chest problem, diabetes, BMI **Season:** NA **Dose-response Investigated?** No **Statistical Package:** STATA	**Pollutant:** Black smoke ($\mu g/m^3$) **Averaging Time:** Exposure measures performed 1966-1969 women had to live within 5 miles of their current address for the past 30 years to be included **Mean (SD):** 11 wards with pollution measures were categorized into high (mean > 120 $\mu g/m^3$) and low (mean < 50 $\mu g/m^3$) exposure categories when classified according to their black smoke levels during 1966-69 SD not reported **Percentiles:** NR **Range (Min, Max):** NR **Monitoring Stations:** NR **Copollutant (correlation):** SO_2 (health results not presented)	**PM Increment:** Categorical **Effect Estimate [Lower CI, Upper CI]:** Association of particulate pollution in place of residence and ischemic heart disease Low (ref): 1.0 High: 1.0 (0.7, 1.4)

[1]All units expressed in $\mu g/m^3$ unless otherwise specified.

E.5. Long-Term Exposure and Respiratory Outcomes

Table E-22. Long-term exposure - respiratory morbidity outcomes - PM_{10}.

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Ackermann-Liebrich et al. (1997, 077537) **Period of Study:** 1991-1993 **Location:** Switzerland (Aarau, Basel, Davos, Geneva, Lugano, Montana, Payerne, Wald)	**Outcome:** Pulmonary function **Age Groups:** 18-60 yrs **Study Design:** Cross-sectional **N:** 9651 people **Statistical Analyses:** Regression analysis **Covariates:** Age, sex, height, weight, education level, nationality, workplace exposure **Season:** NR **Dose-response Investigated?** No **Statistical Package:** NR	**Pollutant:** PM_{10} **Averaging Time:** Continuously measured, 12 mo. avg. used **Mean (SD):** 21.2 (7.4) **Range:** (10.1-33.4) **Copollutant (correlation):** SO_2: r = 0.93 NO_2: r = 0.91 O_3: r = -0.55 Summer Daytime O_3: r = 0.31 Excess O_3: r = 0.67 Altitude: r = -0.77	**PM Increment:** 10 $\mu g/m^3$ Regression Coefficient β (Lower CI, Upper CI) for air pollutants as predictors of pulmonary function FVC: -0.0345 (-0.0407 to -0.0283) $p < 0.001$ FEV_1: -0.0160 (-0.0225 to -0.0095) $p < 0.001$ Percent Change (Lower CI, Upper CI) associated with increase in avg annual air pollution concentration Healthy Never-smokers FVC: -3.39 $p < 0.001$ FEV_1: -1.59 $p < 0.001$ All Never-smokers FVC: -3.14 $p < 0.001$ FEV_1: -1.06 $p < 0.001$ Former Smokers FVC: -3.03 $p < 0.001$ FEV_1: -0.42 Current Smokers FVC: -3.21 $p < 0.001$ FEV_1: -1.35 $p < 0.001$ All FVC: -3.14 $p < 0.001$ FEV_1: -1.03 $p < 0.001$ Long-term Residents FVC: -3.16 $p < 0.001$ FEV_1: -0.96 $p < 0.001$

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Avol et al. (2001, 020552) **Period of Study:** 1993-1998 **Location:** Southern California	**Outcome:** FVC, FEV_1, MMEF, PEFR **Age Groups:** 10 yrs **Study Design:** cohort **N:** 110 **Statistical Analyses:** Linear regression **Covariates:** Sex, race, cohort entry year, annual avg change in height, weight, BMI **Dose-response Investigated?** No	**Pollutant:** PM_{10} **Averaging Time:** 24 h PM_{10} averaged over 1994 **Mean (SD):** 15.0-66.2	**PM Increment:** 10 $\mu g/m^3$ Mean Change (Lower CI, Upper CI) FVC: -1.8 (-9.1, 5.5) FEV_1: -6.6 (-13.5, 0.3) MMEF: -16.6 (-32.1 to -1.1) PEFR: -34.9 (-59.8 to -10.0)
Reference: Bayer-Oglesby et al. (2005, 086245) **Period of Study:** 1992-2001 **Location:** Switzerland (Lugano, Zurich, Bern, Geneva, Anieres, Biel, Langnau, Payerne, & Montana)	**Outcome:** Respiratory symptoms (chronic cough, bronchitis, cold, dry cough, conjunctivitis, wheeze, sneezing, asthma, & hay fever) **Age Groups:** 6-15 yrs **Study Design:** cross-sectional **N:** 9,591 children **Statistical Analyses:** Logistic regression models **Covariates:** age, sex, nationality, parental education, number of siblings, farming status, low birth weight, breast feeding, smoking, family history of asthma, bronchitis and/or atopy, mother who smokes, indoor humidity, mode of cooking & heating, carpeting, pets, removal of carpets/pets for health reasons, completed questionnaire & month, days max temperature $< 0°C$, mother's belief of association between environmental exposures & respiratory health **Dose-response Investigated?** Yes **Statistical Package:** STATA	**Pollutant:** PM_{10} **Averaging Time:** 12 month avg **Mean (SD):** NR **Range (Min, Max):** NR **Monitoring Stations:** 9 **Copollutant (correlation):** NR	**PM Increment:** 10 $\mu g/m^3$ "Figure 2 shows that declining levels of PM_{10} were associated with declining prevalence of chronic cough, bronchitis, common cold, nocturnal dry cough, and conjunctivitis symptoms. For wheezing, sneezing, asthma, and hay fever, no significant association could be seen with declining PM_{10} levels." "Figure 3 illustrates that, on an aggregate level, across regions the mean change in PM_{10} levels (r pearson = 0.81, p = 0.008). The strongest decline of adjusted prevalence of nocturnal dry cough was observed in Geneva, Lugano, and Anieres, where the strongest reduction of PM_{10} had also been achieved."
Reference: Burr et al. (2004, 087809) **Period of Study:** 3 weeks in July and Jan 1997 and 2 weeks in Nov 1996 and April 1997 **Location:** North Wales, England	**Outcome:** Self-report of symptoms only for wheeze, cough, phlegm, rhinitis, and itchy eyes. **Age Groups:** all **Study Design:** Repeated measures **N:** 386 persons in congested streets and 425 in the uncongested streets in 1996/1997. Of these, 165 and 283 completed the second phase of the study.	**Pollutant:** PM_{10} **Averaging Time:** Mean hourly concentrations **Mean (SD):** SD NR Congested streets – 1996-97 35.2 1998-99 27.2 Uncongested Streets 1996-97 11.6 1998-99 8.2 **Monitoring Stations:** 1 in congested street and 1 in uncongested	Percent change PM_{10} in congested streets: 22.7 Percent change PM_{10} in uncongested streets: 28.9 Uncongested street sampling site was 20 m from the congested street sampler. The opening of the by-pass produced a reduction in pollution in the congested streets. The health effects of these changed is likely to be greater for nasal and ocular symptoms than for lower respiratory symptoms. Uncertainty about the causality arises from low response rates and conflicting trends in respiratory and nasal symptoms.

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Calderón-Garcidueñas et al. (2006, 091253) **Period of Study:** 1999, 2000 **Location:** Southwest Mexico City & Tlaxcala, Mexico	**Outcome:** Hyperinflation, interstitial markings-measured by chest radiograph, and lung function–FVC, FEV_1, PEF, FEF25-75, measured using spirometry tests **Age Groups:** 5-13 yrs **Study Design:** Cohort **N:** 249 (total), 230 (Southwest Mexico City), 19 (Tlaxcala) **Statistical Analyses:** Bayes test, Spearman rank correlation, multiple regression **Covariates:** Age, sex **Dose-response Investigated?** No **Statistical Package:** SAS 8.2	**Pollutant:** PM_{10} **Averaging Time:** 1 yr **Mean (SD):** Mexico City 1999–48 2000–45 Tlaxacala: 1994-2000: < NAAQS std **Monitoring Stations:** Southwest Mexico City–2 Tlxacala–periodic air monitoring data **Copollutant:** O_3	**PM Increment:** NR % Change: % of children with FEV_1 < 80% expected value: Mexico City (n = 77): 7.8% Tlaxacala (n = 19): 0% % children with hyperinflation: Mexico City: 65.6% No hyperinflation: 79 Mild: 72 Moderate: 56 Severe: 23 Tlaxacala: 5.3% No hyperinflation: 18 Mild: 1 Moderate: 0 Severe: 0 % children with interstitial markings: Mexico City: 52.6% Number with: No interstitial markings: 19 Mild: 0 Moderate: 0 Severe: 0 Tlaxacala: 0% No interstitial markings: 109 Mild: 112 Moderate: 9 Severe: 0
Reference: Calderon-Garcidueñas, et al. (2003, 156316) **Period of Study:** Jan 1999-Jun 2000 **Location:** Mexico City, Tuxpam, and Tlaxcala, Mexico	**Outcome:** Respiratory system changes **Age Groups:** 5-17 yrs **Study Design:** Case-control of subjects examined for this study **N:** 174 cases, 27 controls, children **Statistical Analyses:** Chi-square test with Yates correction, Spearman's rank correlation test. **Dose-response Investigated?** No **Statistical Package:** SAS 8.2	**Pollutant:** PM_{10} **Averaging Time:** 12 h (daytime 08: 00-20: 00) and nighttime (20: 00-08: 00) **Mean (SD):** Mexico City Day/Night Jan-Jun 1999 76.0/50.0 Jul-Dec 1999 42.8/22.5 Jan-Jun 2000 75.2/47.5	Daily ambient exposure of children to a complex mixture of air pollutants produces significant chest X-ray abnormalities, a decrease in predicted values of FEF25-75, FEF75, and the FEV_1/FVC ratio in association with interstitial marking on chest X-rays, a mild restrictive pattern by spirometry, peripheral blood abnormalities, and an imbalance of serum cytokines.

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Cavanagh et al. (2007, 098618) **Period of Study:** Mar-Aug 2004 **Location:** Christchurch, New Zealand	**Outcome:** A clinical study of excretion of 1-hydroypyrene (1-OHP) as a marker of PAH exposure **Age Groups:** non-smoking males aged 12-18 yr **Study Design:** Comparison of 2 high pollution events and 2 low pollution events **N:** 89 male students in a boarding school **Statistical Analyses:** Wilcoxon signed rank test for paired observations, Mann-Whitney U test **Season:** Winter **Dose-response Investigated?** No	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (SD):** Autumn Low Outdoor 19 Indoor NA Winter I Outdoor 43 Indoor 38 Winter II Outdoor 72 Indoor 84 Winter Low Outdoor 12 Indoor 16 **Monitoring Stations**: One inside the boarding house, and one outside	Urinary 1-OHP were raised after high-pollutions events. Peaks were slightly higher than for US non-smokers of similar ages and slightly lower than for German non-smokers of similar ages. Urinary 1-OHP was slightly higher in asthmatics compared to non-asthmatics. There were no indoor sources of PAHs (wood-burning stoves, tobacco smoke). Diet is another source of PAHs, but all students ate in the boarding house. These results suggest 1-OHP could be used as a biomarker of ambient air pollution.
Reference: Downs et al. (2007, 092853) **Period of Study:** 1991, 2002 **Location:** Switzerland	**Outcome:** FEV_1, FEV_1 as % of FVC, FEF25-75 **Age Groups:** 18-60 years **Study Design:** Prospective Cohort **N:** 4742 people **Statistical Analyses:** Linear random effects models **Covariates:** Age, sex, height, parental smoking, season, education, nationality, occupational exposure, smoking (status, pack-years), atopy, BMI **Dose-response Investigated?** Yes–linear fit best **Statistical Package:** SAS 9.1, STATA 8.2, R 2.4	**Pollutant:** PM_{10} **Averaging Time:** Annual **Mean:** Mean interval exposure: 238 $\mu g/m^3$/years **Percentiles:** 25th: 197 75th: 287	**PM Increment:** 10 $\mu g/m^3$ reduction in annual mean Percent / absolute reduction in annual decline in lung function over 11-year period (95% CI): Annual decline in FEV_1 reduced by 9% / 3.1 mL (0.03-6.2) Annual decline in FEF_{25-75} reduced by 16% / 11.3 mL/second (4.3-18.2) Annual decline in FEV_1 as a percentage of FVC of 0.06 (0.01-0.12) A reduction in interval exposure of 109 µg per m^3 cubic meter–years (equivalent to a reduction of 10 $\mu g/m^3$ in the annual avg during the mean follow-up time of 10.9 years) was associated with: A reduction of 6.9 mL (95% CI, 2.1 to 11.7) in the annual decline in FEV_1 A 22% reduction in the annual decline in FEF_{25-75} (i.e., by 14.0 mL per second 95% CI, 3.1 to 24.8)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Gauderman et al. (2000, 012531) **Period of Study:** 1993-1997 **Location:** Southern California	**Outcome:** FVC, FEV_1, MMEF, FEF_{75} **Age Groups:** fourth, seventh, or tenth graders **Study Design:** cohort **N:** 3035 subjects **Statistical Analyses:** Linear regression **Covariates:** Height, weight, BMI, asthma, smoking, exercise, room temperature, barometric pressure **Dose-response Investigated?** Yes **Statistical Package:** SAS	**Pollutant:** PM_{10} **Averaging Time:** 24 h avg PM_{10} **Mean (SD):** PM_{10} 51.5 **Copollutant (correlation):** $PM_{2.5}$ r = 0.96 O_3 r = -0.32 $PM_{10-2.5}$ r = 0.92 NO_2 r = 0.65 Inorg. Acid r = 0.68	**PM_{10} Increment:** 51.5 $\mu g/m^3$ % Change (Lower CI, Upper CI) PM_{10}-4th grade FVC -0.58 (-1.14 to -0.02) FEV_1 -0.85 (-1.59 to -0.10) MMEF -1.32 (-2.43 to -0.20) FEF75 -1.63 (-3.14 to -0.11) PM_{10}-7th grade FVC -0.45 (-1.03, 0.13) FEV_1 -0.44 (-1.10, 0.23) MMEF -0.48 (-2.51, 1.59) FEF_{75} -0.50 (-2.26, 1.29) PM_{10}-10th grade FVC 0.07 (-0.99, 1.13) FEV_1 -0.46 (-1.84, 0.94) MMEF -0.71 (-4.87, 3.63) FEF_{75} -1.54 (-5.61, 2.71)
Reference: Gauderman et al. (2002, 026013) **Period of Study:** 1996–2000 **Location:** Southern California	**Outcome:** Lung function development: FEV_1, maximal midexpiratory flow (MMEF) **Age Groups:** Fourth grade children (avg age = 9.9 yrs) **Study Design:** Cohort study **N:** 1678 children, 12 communities **Statistical Analyses:** Mixed model linear regression **Covariates:** Height, BMI, doctor-diagnosed asthma and cigarette smoking in previous year, respiratory illness and exercise on day of test, interaction of each of these variables with sex, barometric pressure, temperature at test time, indicator variables for field technician and spirometer **Dose-response Investigated?** Yes **Statistical Package:** SAS (10)	**Pollutant:** PM_{10} **Averaging Time:** Annual 24 h averages **Mean (SD):** The avg levels were presented in an online data supplement (Figure E1) **Monitoring Stations:** 12 **Copollutant (correlation):** O_3 (10 AM to 6 PM) r = 0.13 O_3 r = -0.37 NO_2 r = 0.64 Acid vapor r = 0.79 $PM_{2.5}$ r = 0.95 $PM_{10-2.5}$ r = 0.95 EC r = 0.86 OC r = 0.97	**PM Increment:** 51.5 $\mu g/m^3$ Association Estimate: None of the pulmonary function tests had a statistically significant correlation with PM_{10} FEV_1 r = -0.12 p = 0.63 MMEF r = -0.22 p = 0.30

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Gauderman et al. (2004, 056569) **Period of Study:** Air pollution data ascertainment: 1994-2000. Spirometry testing: spring 2001- spring 2003 **Location:** 12 Communities in Southern California	**Outcome:** Lung function FVC, FEV_1, MMEF (Maximal midexpiratory flow rate) **Age Groups:** Children, Avg age 10 years **Study Design:** Prospective Cohort Study **N:** 12 Communities 2,034 Children 24,972 child-months **Statistical Analyses:** Linear regression of changes in sex-and-community specific lung growth function and PM **Covariates:** Random effect for communities **Season:** ALL (except for $PM_{2.5}$) **Dose-response Investigated?** No **Statistical Package:** SAS	**Pollutant:** PM_{10} **Averaging Time:** 24-h measurements over each year used to create annual avg Mean: Means are presented in figures only. **Range (Min, Max):** ~15, ~65 **Monitoring Stations:** 12 **Copollutant (correlation):** O_3: r = 0.18 NO_2: r = 0.67 $PM_{2.5}$: r = 0.95 EC: r = 0.85 OC: r = 0.97	**PM Increment:** Most to least polluted community Range: PM_{10}: 51.4 $\mu g/m^3$ EC: 1.2 $\mu g/m^3$ OC: 10.5 $\mu g/m^3$ Difference in Lung Growth [Lower CI, Upper CI]; FVC -60.2 (-190.6 to 70.3) FEV_1 -82.1 (-176.9 to 12.8) MMEF -154.2 (-378.3 to 69.8) EC: FVC -77.7 (-166.7 to 11.3) FEV_1 -87.9 (-146.4 to -29.4) MMEF -165.5 (-323.4 to -7.6) OC: FVC -58.6 (-196.1 to 78.8) FEV_1 -86.2 (-185.6 to 13.3) MMEF -151.2 (-389.4 to 87.1) Correlation with % below 80% predicted Lung function (p-value) PM_{10}: 0.66 (0.02) EC: 0.74 (0.006)
Reference: Gauderman et al. (2007, 090121) **Period of Study:** 1993-2004 **Location:** 12 Southern California Communities	**Outcome:** pulmonary function tests FVC, FEV_1, $MMEF/FEF_{25-75}$ **Age Groups:** Children (mean age 10 at recruitment, followed for 8 years) **Study Design:** Cohort Study (Children's Health Study) **N:** 3677 children (1718 in cohort 1 recruited 1993 and 1959 in cohort 2 recruited 1996) 22686 pulmonary function tests. **Statistical Analyses:** Hierarchical mixed effects model with linear splines **Covariates:** Adjustments for height, height squared, BMI, BMI squared, present asthma status, exercise or respiratory illness on day of test, smoking in previous year, field technician, traffic indicator (distance from freeway, distance from major roads), random effects for participant and community. **Dose-response Investigated?** no **Statistical Package:** SAS	**Pollutant:** PM_{10} **Monitoring Stations:** 1 in each community	**PM Increment:** 51.4 $\mu g/m^3$ Pollutant effect reported as difference in 8 year lung function growth from least to most polluted community. Negative difference indicates growth deficits associated with exposure. For PM_{10} FEV growth deficit is -111

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Goss et al. (2004, 055624) **Period of Study:** 1999-2000 **Location:** USA	**Outcome:** Cystic Fibrosis pulmonary exacerbations, FEV_1 **Age Groups:** > 6 **Study Design:** cohort **N:** 11484 patients **Statistical Analyses:** Logistic regression, t-tests, Mann-Whitney tests, Chi-squared tests, polytomous regression, multiple linear regression **Covariates:** Age, sex, lung function, weight, insurance status, pancreatic insufficiency, airway colonization, genotype, median household income by census tract, zipcode. **Dose-response Investigated?** No **Statistical Package:** STATA, SAS	**Pollutant:** PM_{10} **Averaging Time:** annual mean of 24 h averages **Mean (SD):** 24.8(7.8) mg/m^3 Percentiles: 25th: 20.3 50th(Median): 24.0 75th: 28.9 **Monitoring Stations:** 626	**PM Increment:** 10 μg/m^3 **Odds Ratio Estimate [Lower CI, Upper CI]:** Odds of having 2 or more pulmonary exacerbations as compared to 1 or less in 2000 1.08 (1.02 -1.15) Odds of having 2 or more pulmonary exacerbations as compared to no exacerbations in 2000 1.09 (1.02 -1.17) Decrease in FEV_1 38ml(18-58)
Reference: Hanigan et al, (2008, 156518) **Period of Study:** Fire Season (April-November) from 1996-2005 **Location:** Darwin, Australia	**Outcome:** Respiratory admissions **Study Design:** time-series **Covariates:** Race, age **Statistical Analysis:** Over-dispersed Poisson generalized linear models **Statistical Package:** R **Age Groups:** All	**Pollutant:** PM_{10} **Averaging Time:** Daily levels estimated from visibility data **Mean Unit:** *Only reported for 2005* 15.31 μg/m^3 **Range (Min, Max):** 6.93, 31.12 **Copollutant (correlation):** NR	**Increment:** 10 μg/m^3 **Percent Increase (95% CI)** *Full results reported visually in Figure 3* Total Respiratory Admissions 4.81 % (-1.04-11.01) Indigenous Respiratory Admissions, No Lag 9.40% (1.04-18.46) Non-Indigenous Respiratory Admissions, No Lag 3.14% (-2.99-9.66) Indigenous Respiratory Admissions, Lag 3 15.02% (3.73-27.54) Non-Indigenous Respiratory Admissions, Lag 3 0.67% (-7.55-9.61) Indigenous Asthma Admissions, Lag 1 16.27% (3.55-40.17) Non-Indigenous Asthma Admissions, Lag 1 8.54% (-5.60-24.80)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Ho et al. (2007, 093265) **Period of Study:** Oct 1995-Mar 1996 **Location:** Taiwan, Republic of China	**Outcome:** Asthma **Age Groups:** 10-17 yrs **Study Design:** Screened junior high students for asthma, collected meteorological data to determine the relationship. **N:** 69,367 **Statistical Analyses:** Logistic regression model, the maximum likelihood estimation with Fisher's scoring algorithm, stepwise regression model, Wald statistic, Akaike criteria. GEE, GENMOD **Covariates:** Wind, barometric pressure, temperature, rain, humidity **Season:** Fall-spring **Dose-response Investigated?** No **Statistical Package:** SAS	**Pollutant:** PM_{10} **Averaging Time:** Monthly **Monitoring Stations:** 72	**Odds Ratio from stepwise regression model:** Females (n = 32, 648) 0.993 [0.990-0.997] Males: NS Higher PM_{10} concentration resulted in less asthma prevalence. However, a higher number of rain days seemed to reduce asthma prevalence rain days might interact with PM_{10}.
Reference: Hong et al. (2004, 156565) **Period of Study:** 2001 **Location:** Kerinci, SP7, and Pelalawan, Indonesia	**Outcome:** Respiratory symptoms **Age Groups:** < 12 yrs **Study Design:** Disproportionate random sampling was used to select 100 households from each village. An interviewer interviewed all children through the caregiver/parent to obtain symptoms in the past 2 weeks (cough, cold, phlegm) and the last 12 months. **N:** 382 children **Statistical Analyses:** Chi-square test, analysis of variance, prevalence rates, adjusted odds ratios, multivariate adjusted odds ratios from multiple logistic regression models, allowing for clustering. **Covariates:** Age, gender, no. of children in household, household income, floor area of house, fuel for cooking, no. of smokers in household, personal and family medical history. **Dose-response Investigated?** No **Statistical Package:** SPSS STATA v.7	**Pollutant:** PM_{10} **Averaging Time:** 24 h measurements were taken daily from 2 weeks before the field survey to 1 month after the survey **Mean (SD):** Kerinci 102.9 (49.6) $\mu g/m^3$ SP7 73.7 (41.7) Pelalawan 26.1 (14.5) $P < 0.01$ **Range (Min, Max):** Kerinci 25, 184 SP7 13, 138 Pelalawan 10, 66 **Monitoring Stations:** 3	**PM Increment:** Low (Pelalawan), Medium (SP7), & High (Kerinci) PM Exposure Odds Ratios (95% CI) for Symptoms by village: Cough/cold past 2 wks Pelalawan 1.00 SP7 2.03 (1.04, 3.96) Kerinci 3.17 (1.43, 7.07) Respiratory symptoms last 12 months Pelalawan 1.00 SP7 1.15 (0.58, 2.26) Kerinci 1.42 (0.62, 3.25) Ever had rhinitis w/o flu Pelalawan 1.00 SP7 2.17 (0.57, 8.29) Kerinci 0.56 (0.11, 2.83) Ever had wheezing Pelalawan 1.00 SP7 0.85 (0.35, 2.08) Kerinci 1.18 (0.46, 3.01)
Reference: Horak et al. (2002, 034792) **Period of Study:** 1994-1997 **Location:** Lower Austria	**Outcome:** Lung function growth measured by changes in: 1. FVC (forced vital capacity) 2. FEV_1 3. MEF_{25-75} (midexpiratory flow between 25-75% of the forced vital capacity) **Age Groups:** 2-3 grade schoolchildren (mean age = 8) **Study Design:** Prospective cohort with repeated measures **N:** 975 children **Statistical Analyses:** Linear regression GEE, nonstationary M-dependent correlation structure	**Pollutant:** PM_{10} **Mean (SD):** Winter: 21.0 (4.8) summer: 17.4 (2.8) **Range (Min, Max):** Winter: 9.4-30.5 summer: 11.7-28.9 **Monitoring Stations:** NR, stations were located in the immediate vicinity of each of the 8 elementary schools **Copollutant (correlation):** Winter O_3 (r = -0.581)	**PM Increment:** 1 $\mu g/m^3$ Mean per unit increase in PM (p-value) **Outcome:** difference per day of FVC (mL/day) Summer: 0.001 (0.938) Winter: 0.008 (0.042) Controlling for temperature: Summer: -0.007 (0.417) Winter: -0.003 (0.599) Controlling for O_3: Summer: 0.001 (0.911)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
	Covariates: Gender, atopy, ETS exposure, baseline lung function, first height, height difference, school site **Season:** Winter, summer **Dose-response Investigated?** No	SO_2 (r = 0.520) NO_2 (r = 0.595) summer O_3 (r = -0.429) SO_2 (r = 0.335) NO_2 (r = 0.412)	Winter: 0.010 (0.019) Controlling for NO_2: Summer: -0.018 (0.056) Winter: 0.015 (0.000) Controlling for SO_2: Summer: 0.005 (0.575) Winter: 0.004 (0.492) In non-asthmatic children: Summer: -0.003 (0.710) Winter: 0.009 (0.030) In group not exposed to ETS: Summer: 0.014 (0.154) Winter: 0.012 (0.0018) In group exposed to ETS: Summer: 0.022 (0.088) Winter: 0.003 (0.656) **Outcome:** difference per day of FEV_1 (mL/day) Summer: -0.023 (0.003) Winter: 0.001 (0.885) Controlling for temperature: Summer: -0.034 (0.000) Winter: -0.011 (0.016) Controlling for O_3: Summer: -0.022 (0.008) Winter: 0.004 (0.338) Controlling for NO_2: Summer: -0.038 (0.000) Winter: 0.011 (0.005) Controlling for SO_2: Summer: -0.022 (0.010) Winter: -0.005 (0.358) **Outcome:** difference per day MEF25-75 (mL/day) Summer: -0.090 (0.000) Winter: -0.008 (0.395) Controlling for temperature: Summer: -0.112 (0.000) Winter: -0.013 (0.295) Controlling for O_3: Summer: -0.087 (0.000) Winter: -0.008 (0.434) Controlling for NO_2: Summer: -0.102 (0.000) Winter: 0.005 (0.610) Controlling for SO_2:

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			Summer: -0.095 (0.000)
			Winter: -0.011 (0.474)
Reference: Hwang et al. (2006, 088971)	**Outcome:** Peak expiratory flow rate (PEFR), Forced Expiratory Volume in 1 second (FEV_1), Forced Vital Capacity (FVC), Self reported "frequent coughing," Self reported "shortness of breath," Self reported " irritation of respiratory tract"	**Pollutant:** PM_{10}	**PM Increment:** 10 $\mu g/m^3$
Period of Study: 2001		**Mean (SD):** 55.58 (16.57)	RR Estimate [Lower CI, Upper CI]
Location: Taiwan		Percentiles: 25th: 42.96	Single pollutant model: 1.00 [0.99, 1.02]
		50th(Median): 53.81	Controlling for NO_X: 0.99 [0.97, 1.00]
	Age Groups: 24-55 years (mean = 40)	75th: 70.37	Controlling for CO: 1.00 [0.99, 1.01]
	Study Design: Cohort	**Range (Min, Max):** 29.36, 99.58	Controlling for O_3: 1.00 [0.99, 1.02]
	N: 120 men (60 traffic policemen and 60 controls)	**Monitoring Stations**: 22	
	Statistical Analyses: ANOVA, odds ratios calculated from 2X2 table	**Copollutant (correlation):** NO_X (r = 0.34)	
	Dose-response Investigated? No	SO_2 (r = 0.58)	
		CO (r = 0.27)	
		O_3 (r = 0.28)	
Reference: Hwang et al, (2008, 134420)	**Outcome:** Oral Cleft	**Pollutant:** PM_{10}	**Increment:** 10 $\mu g/m^3$
Period of Study: 2001-2003	**Study Design:** Case-control	**Averaging Time:** hourly	**Odds Ratio (Min CI, Max CI);**
Location: Taiwan	**Covariates:** Maternal age, plurality, gestational age, population density and season of conception	**Mean (SD) Unit:**	Single Pollutant Model
		Average: 54.83 ± 13.07 $\mu g/m^3$	Month 1: 1.01 (0.96-1.06)
	Statistical Analysis: logistic regression	Spring: 64.44 ± 16.21 $\mu g/m^3$	Month 2: 1.00 (0.95-1.05)
	Age Groups: Infants	Summer: 39.11 ± 8.31 $\mu g/m^3$	Month 3: 0.99 (0.95-1.05)
		Fall: 47.76 ± 11.77 $\mu g/m^3$	Two Pollutant Model (O_3 + PM_{10})
		Winter: 68.00 ± 21.88 $\mu g/m^3$	Month 1: 0.99 (0.94-1.04)
		Range (Min, Max):	Month 2: 0.99 (0.94-1.04)
		Average: 20.75-78.05 $\mu g/m^3$	Month 3: 0.98 (0.93-1.04)
		Spring: 23.33-94.33 $\mu g/m^3$	Two Pollutant Model (CO + PM_{10})
		Summer: 17.33-60.00 $\mu g/m^3$	Month 1: 1.01 (0.96-1.06)
		Fall: 21.00-72.00 $\mu g/m^3$	Month 2: 1.00 (0.95-1.05)
		Winter: 21.33-116.00 $\mu g/m^3$	Month 3: 0.99 (0.95-1.05)
		Copollutant (correlation):	Two Pollutant Model (NO_x + PM_{10})
		CO: -0.19	Month 1: 1.02 (0.97-1.08)
		NO_X: 0.56	Month 2: 1.01 (0.95-1.07)
		O_3: 0.39	Month 3: 1.01 (0.95-1.07)
		SO_2: 0.50	Three Pollutant Model (O_3 + CO + PM_{10})
			Month 1: 0.99 (0.94-1.04)
			Month 2: 0.99 (0.94-1.04)
			Month 3: 0.99 (0.93-1.04)
			Three Pollutant Model (O_3 + NO_x + PM_{10})
			Month 1: 1.00 (0.94-1.06)
			Month 2: 0.98 (0.92-1.05)
			Month 3: 1.00 (0.93-1.06)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Ingle et al. (2005, 089014) **Period of Study:** May 2003-April 2004 **Location:** Jalgaon City, India	**Outcome:** Peak expiratory flow rate (PEFR), Forced Expiratory Volume in 1 second (FEV_1), Forced Vital Capacity (FVC), Self reported "frequent coughing," Self reported "shortness of breath," Self reported " irritation of respiratory tract" **Age Groups:** 24-55 years (mean = 40) **Study Design:** Cohort **N:** 120 men (60 traffic policemen and 60 controls) **Statistical Analyses:** ANOVA, odds ratios calculated from 2X2 table **Dose-response Investigated?** No	**Pollutant:** PM_{10} **Mean (SD):** Location-specific means: Prabhat: 224 (27) Ajanta: 269 (41) Icchdevi: 229 (24) **Monitoring Stations:** 3	OR Estimate [p-value] Self reported frequent coughing 2.96 [$p < 0.05$] Self reported shortness of breath 1.22 [$p < 0.05$] Self reported irritation in respiratory tract 7.5 [$p < 0.05$] Observed/expected lung function p-value for difference between groups: FVC (L) Traffic policemen: 0.82 Controls: 0.99 Traffic policemen: Obs = 3.03 ± 1.7 Exp = 3.70 ± 2.8 Controls: Obs = 3.18 ± 0.91 Exp = 3.19 ± 1.71 FEV_1 (L) Traffic policemen: 0.73 Controls: 1.18 Traffic policemen: Obs = 2.27 ± 1.05 Exp = 3.08 ± 2.7 Controls: Obs = 3.61 ± 0.90 Exp = 3.06 ± 0.91 PEFR (L/s) Traffic policemen: 0.66 Controls: 0.92 Traffic policemen: Obs = 6.05 ± 2.15 Exp = 9.21 ± 0.47 Controls: Obs = 5.54 ± 1.85 Exp = 6.11 ± 2.31
Reference: Islam et al. (2007, 090697) **Period of Study:** 2006 **Location:** 12 California communities	**Outcome:** Respiratory symptoms, Asthma **Study Design:** Longitudinal study cohort **Statistical Analyses:** Cox proportional hazards regression **Age Groups:** 7-9 10-11 >11	Pollutants: PM_{10} **Averaging Time:** 24-h avg Copollutants (correlation): O_3 NO_2 EC OC	The study doesn't present quantitative results on PM_{10}.

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Janssen et al. (2003, 133555) **Period of Study:** 4/1997–7/1998 **Location:** Netherlands–24 schools	**Outcome:** Symptoms of asthma and allergic disease (asthma, conjunctivitis, hay fever, itchy rash, eczema, phlegm, bronchitis), skin prick test (SPT) reaction to allergens, lung function (forced vital capacity [FVC], forced expiratory volume in one second [FEV_1], and positive test for fall in $FEV_1 \geq 15\%$ after inhalation of maximal 23 mL hypertonic saline [BHR = bronchial hyper-responsiveness]) **Age Groups:** 7-12 years old **Study Design:** Cohort **N:** 24 schools (see notes) **Statistical Analyses:** Multilevel model **Covariates:** Age, sex, non-Dutch nationality, cooking on gas, current parental smoking, current pet possession, parental education level, number of persons in the household, presence of an unvented water heater in kitchen, questionnaire not filled out by the mother, presence of mold stains in kitchen or living room or bedroom, parental respiratory symptoms, distance of home to motorway, cough or cold at time of lung function measurement, bronchitis or severe cold or flu in 3 weeks preceding measurement, season **Dose-response Investigated?** No **Statistical Package:** MLwiN	**Pollutant:** $PM_{2.5}$ **Averaging Time:** Annual **Mean (SD):** 20.5 $\mu g/m^3$ (2.2) **Percentiles:** 25th: 18.6 50th (Median): 20.4 75th: 22.1 **Range (Min, Max):** 17.3, 24.4	**PM Increment:** 'Difference between the maximum and the minimum of the exposure indicator' (3.5 $\mu g/m^3$) **RR Estimate [Lower CI, Upper CI]** **lag:** Current wheeze 1.51 (0.90, 2.53) Asthma ever 1.03 (0.59, 1.82) Current conjunctivitis 2.08 (1.17, 3.71) Hay fever ever 2.28 (1.13, 4.57) Current itchy rash 1.63 (0.91, 2.89) Eczema ever 1.31 (0.94, 1.83) Current phlegm 1.53 (0.74, 3.19) Current bronchitis 1.71 (0.84, 3.50) Elevated total IgE 1.45 (0.74, 2.84) Any allergen (spt reactivity) 1.33 (0.83, 2.11) Indoor allergens (spt reactivity) 1.17 (0.70, 1.94) Outdoor allergens (spt reactivity) 1.90 (1.06, 3.40) FVC $<$ 85% predicted 0.54 (0.29, 1.00) FEV_1 $<$ 85% predicted 0.88 (0.37, 2.09) BHR 0.93 (0.51, 1.68) **Notes:** Figure 1 of the article illustrates the association between exposures, including $PM_{2.5}$, and various respiratory symptoms among children with and without a positive SPT and positive BHR. In general, the association between $PM_{2.5}$ and respiratory symptoms were higher for children with a positive SPT or BHR, except for the outcome of current phlegm. This effect appeared to be the strongest for children with a positive BHR, particularly for current wheeze and current bronchitis. The authors also reported separate analyses for children with SPT reactivity for indoor and outdoor allergens, but did not report any clear differences between the two groups. The authors did report, in the text, that the OR of $PM_{2.5}$ exposure for children sensitized for outdoor allergens was 7.64 for current itchy rash ($p < 0.05$).

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Kan, et al. (2007, 091383) **Period of Study:** 1987-1992 **Location:** Four Communities in the U.S.: Forsyth County, North Carolina Jackson, Mississippi northwest suburbs of Minneapolis, Minnesota and Washington County, Maryland.	**Outcome:** FEV_1 and FVC **Age Groups:** Middle-aged (mean age was 54.2 years) **Study Design:** Hierarchical regression **N:** 15,792 **Statistical Analyses:** SAS PROC MIXED **Covariates:** Distance to major roads, traffic exposure, age, ethnicity, sex, smoking, environmental tobacco smoke exposure, occupation, education, medical history, BMI. **Dose-response Investigated?** No **Statistical Package:** SPSS Version 11 for traffic density, SAS Version 9.1.2 for statistical analysis	**Pollutant:** PM_{10} **Averaging Time:** 24-h PM_{10} averaged over study period **PM Component:** Vehicle emissions **Monitoring Stations:** 0 **Copollutant:** NO_2 O_3	**RR Estimate (Lower CI, Upper CI):** (Note: for ARIC participants living < 150 meters from major roads) Women FEV_1(mL) Age-adjusted model -29.5 (-52.2 to -6.9) Multivariate model -15.7 (-34.4 to -2.9) FVC (mL) Age-adjusted model -33.2 (-60.4 to -5.9) Multivariate model -24.2 (-46.2,-2.3) FEV_1/FVC (%) Age-adjusted model -0.1(-0.5,0.2) Multivariate model 0.1 (-0.3,0.4) Men FEV_1(mL) Age-adjusted model -38.4 (-76.7,0.6) Multivariate model -6.4 (-38.1,25.3) FVC (mL) Age-adjusted model -17.0(-62.0,28.0) Multivariate model 10.9(-24.7,46.5) FEV_1/FVC (%) Age-adjusted model -0.05 (-0.9,0.0) Multivariate model -0.3 (-0.7,0.2)
Reference: Kim et al. (2005, 087418) **Period of Study:** Mar and Dec 2000 **Location:** Incheon & Ganghwa, Korea	**Outcome:** lung function (FEV_1, FVC) **Age Groups:** middle school students **Study Design:** Panel **N:** 368 children **Statistical Analyses:** Generalized liner model **Covariates:** gender, grade **Season:** Spring and fall **Dose-response Investigated?** No **Statistical Package:** SAS	**Pollutant:** PM_{10} **Averaging Time:** monthly **Mean (SD):** Incheon March 64 December 54 Ganghwa March 64 December 53 **Range (Min, Max):** NR	**PM Increment:** NR **OR Estimate [Lower CI, Upper CI]:** ''The present study showed that the values of FEV_1 and FVC were greater in December than in March for both male and female students at all academic years...Because only the level of PM_{10} was significantly higher for March than for December in both areas, the authors suggest that decrements of pulmonary function in March for both areas are associated with the increased level of PM_{10}''

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Kim et al. (2004, 087383)	**Outcome:** Asthma, bronchitis	**Pollutant:** PM_{10}	**PM Increment:** 1.4 (IQR)
Period of Study: Mar-June (spring) 2001	**Age Groups:** Children (in grades 3-5)	**Averaging Time:** 9 weeks	**OR Estimate [Lower CI, Upper CI]:**
Sep-Nov (fall) 2001	**Study Design:** Cross-sectional	**Mean (SD):** Study Avg 30	Bronchitis
Location: Alameda County, CA	**N:** 1109 children, 871 (long term resident children), 462 (long term related females), 403 (long term related males)	**Monitoring Stations:** 10	All subjects: 1.03 [0.99, 1.07]
		Copollutant (correlation): r2 is approximately 0.9 for all copollutants–Black Carbon (BC), $PM_{2.5}$, NO_X, NO_2, NO (NO_X–NO_2)	LTR subjects: 1.02 [0.98, 1.07]
	Statistical Analyses: 2-stage multiple logistic regression model		LTR females: 1.04 [1.01, 1.09]
	Covariates: respiratory illness before age of 2, household mold/moisture, pests, maternal history of asthma (for asthma) Season: Spring and fall		LTR males: 1.01 [0.95, 1.06]
			Asthma
	Dose-response Investigated? Yes		All subjects: 1.02 [0.96, 1.09]
	Statistical Package: SAS 8.2		LTR subjects: 1.04 [0.97, 1.12]
			LTR females: 1.09 [0.92, 1.29]
			LTR males: 1.02 [0.94, 1.10]
			Asthma excluding outlier school having a larger proportion of Hispanics
			All subjects: 1.06 [0.97, 1.16]
			LTR subjects: 1.08 [0.98, 1.19]
			LTR females: 1.09 [0.96, 1.24]
			LTR males: 1.08 [0.97, 1.19]
Reference: Kumar et al. (2004, 089873)	**Outcome:** Chronic respiratory symptoms & Spirometric ventilatory defect	**Pollutant:** PM_{10}	**PM_{10} Increment:**
Period of Study: 1999-2001	**Age Groups:** > 15 yrs	**Mean (SD):** Study town 112.8 (17.9)	Low vs. High
Location: Mandi Gobindgarh and Morinda, Punjab State, northern India	**Study Design:** Cross-sectional	Reference town 75.8 (2.9)	OR (Lower CI, Upper CI)
	N: 3603 individuals		p-value
	Statistical Analyses: Logistic regression		Chronic respiratory symptoms
			Low 1.00 (ref)
	Covariates: Age, gender, migration, SES, smoking, type of cooking fuel use		High 1.5 (1.2, 1.8)
			< 0.001
	Dose-response Investigated? No		Spirometric ventilatory defect
			Low 1.00 (ref)
			High 2.4 (2.0-2.9)
			< 0.001

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Leonardi et al. (2000, 010272) **Period of Study:** 1996 **Location:** 17 cities of Central Europe (Bulgaria, Czech Republic, Hungary , Poland, Romania, Slovakia)	**Outcome:** Immune biomarkers **Age Groups:** 9-11 **Study Design:** Cross-sectional **N:** 366 school children **Statistical Analyses:** Linear regression **Covariates:** Age, gender, parental smoking, laboratory of analysis, recent respiratory illness **Dose-response Investigated?** No **Statistical Package:** STATA	**Pollutant:** PM_{10} **Averaging Time:** annual PM_{10} **Mean (SD):** PM_{10}: 65 (14) **Range (Min, Max):** PM_{10}: (41, 96) 5th, median, & 95th percentile PM_{10}: 41, 63, 90	**% Change (Lower CI, Upper CI)** **p-value** **PM_{10}** Neutrophils -5 (-33, 36) $>.20$ Total lymphocytes 20 (-6, 54); .150 B lymphocytes 42 (-3, 107); .067 Total T lymphocytes 30 (-2, 73); .072 CD4+ 28 (-10, 82); .177 CD8+ 29 (-5, 75); .097 CD4/CD8 7 (-20, 43) $>.20$ NK 33 (-10, 97); .157 Total IgG 11 (-10, 38) $>.20$ Total IgM 5 (-21, 39) $>.20$ Total IgA11 (-16, 46) $>.20$ Total IgE -8 (-62, 123) $>.20$
Reference: Lichtenfels et al, (2007, 097041) **Period of Study:** 2001-2003 **Location:** São Paulo, Brazil	**Outcome:** Secondary sex ratio **Study Design:** Retrospective Cohort **Covariates:** NR **Statistical Analysis:** Correlation Coefficient **Age Groups:** Infants	**Pollutant:** PM_{10} **Averaging Time:** Annual **Mean (SD) Unit:** 2001: 49.8 (10.5) $\mu g/m^3$ 2002: 48.5 (11.4) $\mu g/m^3$ 2003: 49.4 (14.4) $\mu g/m^3$ **Range (Min, Max):** 31.71-60.96 $\mu g/m^3$ **Copollutant (correlation):** NR	**Increment:** NR **Correlation Coefficient:** $R^2 = 0.7642$, P = 0.13

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Lubinski, et al. (2005, 087563) **Period of Study:** 1993-1997 **Location:** Poland	**Outcome:** Pulmonary function TLC: total lung capacity ITGV: interthoracic gas volume ITGV%TLC: ITGV percent total lung capacity Raw: airway resistance FVC: forced vital capacity FEV_1: forced expiratory volume, 1 second FEV_1%FVC: FEV_1 percent forced vital capacity PEF: peak expiratory flow FEF_{50}: forced expiratory flow **Age Groups:** 18-23 males, healthy **Study Design:** ecological cross-sectional study **N:** 1278 subjects **Statistical Analyses:** Multiple linear regression, ANOVA **Covariates:** report unclear on whether or not there was covariate control, but may include NO_2 and SO_2 **Dose-response Investigated?** No	**Pollutant:** PM_{10} **Averaging Time:** 12 mo **Mean (SD):** A: Highest Pollution Region Katowice 67-125 Krakow 41-49 B: Moderate Pollution Region Bielsko-Biala 29-48 Opole 18-45 Lodz 23-38 Warsaw 35-45 Wroclaw 28-76 Zagan 5-35 C: Lowest Pollution Region Gizycko 5-18 Hel 12-18 Ostroda 23-33 Swinoujscie 7-16 Ustka 12-26 **Copollutant:** NO_2, SO_2	**PM Increment:** 1 $\mu g/m^3$ Slope, multiple regression TLC PM_{10}: -0.05 +SO_2: 0.03 +NO_2: -0.06 ITGV PM_{10}: 0.01 +SO_2: -0.07 +NO_2: -0.07 ITGV%TLC PM_{10}: -0.06 +SO_2: 0.08 +NO_2: 0.00 Raw PM_{10}: 0.075 +SO_2: -0.08 +NO_2: 0.127 FVC PM_{10}: 0.045 +SO_2: 0.045 +NO_2: -0.14 FEV_1 PM_{10}: 0.031 +SO_2: -0.08 +NO_2: -0.12 FEV_1%FVC PM_{10}: 0.00 +SO_2: -0.14 +NO_2: -0.048 PEF PM_{10}: -0.18 +SO_2: 0.056 +NO_2: -0.09 FEF50 PM_{10}: 0.031 +SO_2: -0.11 +NO_2: -0.04
Reference: McConnell et al. (1999, 007028) **Period of Study:** 1993 **Location:** Southern California	**Outcome:** Bronchitis, chronic cough, phlegm **Age Groups:** Children: 4th, 7th, & 10th graders **Study Design:** Cross-sectional **N:** 3676 people **Statistical Analyses:** Logistic regression **Covariates:** Age, sex, race, grade, health insurance **Dose-response Investigated?** Yes	**Pollutant:** PM_{10} **Averaging Time:** yearly avg 24 h PM_{10} **Mean (SD):** 34.8 **Range (Min, Max):** 13.0, 70.7 **Copollutant (correlation):** NO_2 r = 0.74 O_3 r = 0.32 Acid r = 0.54 $PM_{2.5}$ r = 0.90 NO_2 r = 0.83 O_3 r = 0.50 Acid r = 0.71	**PM_{10} Increment:** 19 $\mu g/m^3$ Children w/ asthma Bronchitis: 1.4 (1.1,1.8) Phlegm: 2.1 (1.4, 3.3) Cough: 1.1 (0.8, 1.7) Children w/ wheeze, no asthma Bronchitis: 0.9 (0.7, 1.3) Phlegm: 0.9 (0.6, 1.4) Cough: 1.2 (0.9, 1.8) Children w/ no wheeze, no asthma Bronchitis: 0.7 (0.4, 1.0) Phlegm: 0.8 (0.6, 1.3) Cough: 0.9 (0.7, 1.2)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: McConnell et al. (2003, 049490) **Period of Study:** 1993-99 **Location:** 12 Southern CA communities	**Outcome:** bronchitis symptoms **Age Groups:** 9-19 **Study Design:** communities selected on basis of historic levels of criteria pollutants and low residential mobility. **N:** 475 children **Statistical Analyses:** 3 stage regression combined to give a logistic mixed effects model **Covariates:** sex, ethnicity, allergies history, asthma history, SES, insurance status, current wheeze, current exposure to ETS, personal smoking status, participation in team sports, in utero tobacco exposure through maternal smoking, family history of asthma, amount of time routinely spent outside by child during 2-6 pm. **Dose-response Investigated?** No **Statistical Package:** SAS Glimmix macro	**Pollutant:** PM_{10} **Averaging Time:** 4 year averages **Mean (SD):** .30.8(13.4) $\mu g/m^3$ **Range (Min, Max):** 15.7-63.5 PM Component: particulate organic carbon and elemental carbon **Copollutant (correlation):** $PM_{2.5}$: r = 0.79 $PM_{10-2.5}$: r = 0.79 Inorganic acid: r = 0.72 Organic Acid: r = 0.59 Elemental carbon: r = 0.71 Organic Carbon: r = 0.70 NO_2: r = 0.20 O_3: r = 0.64	**PM Increment:** Between community range 47.8 $\mu g/m^3$ Between community unit 1 $\mu g/m^3$ Within community 1 $\mu g/m^3$ OR Estimate [Lower CI, Upper CI] Between community per range 1.72(0.93-3.20) | Between Community per unit 1.01(1.00-1.02) | Within community per unit 1.04(0.99-1.10)
Reference: McConnell et al. (2002, 023150) **Period of Study:** 1993-1998 **Location:** 12 communities in Southern California (grouped into either high and low pollution communities)	**Outcome:** Asthma (new diagnosis) **Age Groups:** 9-12 yrs, 12-13 yrs, 15-16 yrs **Study Design:** Cohort **N:** 3535 **Statistical Analyses:** Multivariate proportion hazard model **Covariates:** Sex, age, ethnic origin, BMI, child history of allergies and asthma history, SES, maternal smoking, time spent outside, history of wheezing, ownership of insurance (yes/no), number and type of sports played **Dose-response Investigated?** Yes **Statistical Package:** SAS 8.1	**Pollutant:** PM_{10} **Averaging Time:** 4 yrs **Mean (SD):** Low pollution communities: 21.6 (3.8) **High pollution communities:** 43.3 (12.0) **Percentiles:** Low pollution communities: 50th(Median): 20.8 High pollution communities: 50th(Median): 43.3 **Range (Min, Max):** Low pollution communities: 16.62, 27.3 High pollution communities: 33.5, 66.9 **Monitoring Stations:** 12 **Copollutant (correlation):** $PM_{2.5}$: r = 0.96 NO_2: r = 0.65 O_3	**RR Estimate [Lower CI, Upper CI]** **lag:** Low PM communities: 1.0 [ref] 0 sport 1.5 [1.0, 2.2] 1 sport 1.2 [0.7, 1.9] 2 sports 1.7 [0.9, 3.2] ≥ 3 sports High PM communities: 1.0 [ref] 0 sport 1.1 [0.7, 1.7] 1 sport 0.9 [0.5, 1.7] 2 sports 2.0 [1.1, 3.6] ≥3 sports **High vs Low PM_{10} communities: 0.8 (0.6, 1.0)** **Incidence–N (incidence) number of sports:** Low PM communities: 49 (0.023) 0 54 (0.032) 1 22 (0.024) 2 13 (0.033) ≥3 High PM communities: 55 (0.021) 0 36 (0.021) 1 14 (0.018) 2 16 (0.033) ≥3

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: McConnell, et al. (2006, 180226) **Period of Study:** 1996-1999 **Location:** 12 Southern California communities	**Outcome:** Prevalence of bronchitic symptoms (yearly). **Age Groups:** 10-15-years-old **Study Design:** longitudinal cohort **N:** 475 asthmatic children **Statistical Analyses:** Multilevel logistic mixed effects models. **Covariates:** age, second-hand smoke personal smoking history sex, race. **Dose-response Investigated?** No **Statistical Package:** SAS with GLIMMIX macro	**Pollutant:** PM_{10} **Averaging Time:** 365 days **Percentiles:** Community by year (n = 48 = 12 communities ◘ 4 years) 25th: NR 50th(Median): 3.4 75th: NR **Range (Min, Max):** Community by year (n = 48 = 12 communities ◘ 4 years): (0.89, 8.7) **Monitoring Stations**: 12 **Copollutant:** O_3, NO_2, EC, OC Acid vapor (acetic and formic acid)	**PM Increment:** 6.1 $\mu g/m^3$ OR Estimate [Lower CI, Upper CI] PM_{10} Dog (n = 292): 1.60 [1.12: 2.30] No dog (n = 183): 0.89 [0.57: 1.39] PM_{10}*Dog interaction p-value: 0.02 Cat (n = 202): 1.47 [0.96: 2.24] No Cat (n = 273): 1.20 [0.83: 1.73] PM_{10}*Cat interaction p-value: 0.41 Neither pet (n = 112): 0.91 [0.53: 1.56] Cat only (n = 71): 0.84 [0.42: 1.66] Dog only (n = 161): 1.41 [0.91: 2.19] Both pets (n = 131): 1.89 [1.15: 3.10] Results suggest that dog ownership, a source of residential exposure to endotoxin, may worsen the severity of respiratory symptoms from exposure to air pollutants in asthmatic children.
Reference: Meng et al. (2007, 093275) **Period of Study:** November 2000 and September 2001 (collection of health data) **Location:** Los Angeles and San Diego counties	**Outcome:** Poorly controlled asthma vs. controlled asthma **Age Groups:** 18-64, 65+ **Study Design:** Long-term exposure study comparison of cases and controls **N:** 1,609 adults (represented individuals age 18+ who reported ever having been diagnosed as having asthma by a physician and had their address successfully geocoded) **Statistical Analyses:** Logistic regression to evaluate associations between TD (traffic density) and annual avg air pollution concentrations and poorly controlled asthma. Used sample weights that adjusted for unequal probabilities of selection into the CHIS sample. **Covariates:** Age, sex, race/ethnicity, family federal poverty level, county, insurance status, delay in care for asthma, taking medications, smoking behavior, self-reported health status, employment, physical activity **Dose-response Investigated?** yes	**Pollutant:** PM_{10} **Averaging Time:** 24 over 1 year **Copollutant (correlation):** O_3: r = -0.72 NO_2: r = 0.83 $PM_{2.5}$: r = 0.84 CO: r = 0.42 TD: r = 0.14	**PM Increment:** Continuous data: per 10 $\mu g/m^3$ OR Estimate [Lower CI, Upper CI] lag: All Adults: 1.08 [0.82, 1.43] Non-Elederly Adults: 1.14 [0.84, 1.55] Elderly: 0.84 [0.41, 1.73] Women: 1.38 [0.99, 1.94]

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Millstein et al. (2004, 088629) **Period of Study:** Mar-Aug, 1995, and Sep, 1995 to Feb, 1996 Data were taken from the Children's Health Study **Location:** Alpine, Atascadero, Lake Arrowhead, Lake Elsinore, Lancaster, Lompoc, Long Beach, Mira Loma, Riverside, San Dimas, Santa Maria, and Upland, CA	**Outcome:** Wheezing & asthma medication use (ICD9 NR) **Age Groups:** 4th grade students, mostly 9 yrs at the time of the study **Study Design:** Cohort Study, stratified into 2 seasonal groups/ **N:** 2081 enrolled, 2034 provided parent-completed questionnaire. **Statistical Analyses:** Multilevel, mixed-effects logistic model. **Covariates:** Contagious respiratory disease, ambient airborne pollen and other allergens, temperature, sex, age race, allergies, pet cats, carpet in home, environmental tobacco smoke, heating fuel, heating system, water damage in home, education level of questionnaire signer, physician diagnosed asthma. **Season:** Mar-Aug, 1995, and Sep, 1995 to Feb, 1996 **Statistical Package:** GLIMMIX SAS 8.00 macro for generalized linear mixed models. **Lags Considered:** 14	**Pollutant:** PM_{10} **Averaging Time:** Monthly means for PM_{10}. **PM Component:** Nitric acid, formic acid, acetic acid **Monitoring Stations**: 1 central location in each community **Copollutant (correlation):** O_3: r = 0.76 NO_2: r = 0.39 $PM_{2.5}$: r = 0.91	**PM Increment:** IQR 13.39 $\mu g/m^3$ Odds Ratio [lower CI, Upper CI] Annual PM_{10}: 0.93 [0.67, 1.27] March-August PM_{10}: 0.91 [0.46, 1.80] Sep-Feb PM_{10}: 0.65 [0.40, 1.06]
Reference: Neuberger et al. (2004, 093249) **Period of Study:** 6/1999-6/2000 **Location:** Austria (Vienna and a rural area near Linz)	**Outcome:** Questionnaire derived asthma score, and a 1-5 point respiratory health rating by parent **Age Groups:** 7-10 years **Study Design:** Cross-sectional survey **N:** about 2000 children **Statistical Analyses:** mixed models linear regression-used factor analysis to develop the "asthma score" **Covariates:** Pre-existing respiratory conditions, temperature, rainy days, # smokers in household, heavy traffic on residential street, gas stove or heating, molds, sex, age of child, allergies of child, asthma in other family members **Dose-response Investigated?** No **Statistical Package:** NR **Lags Considered:** 4 week avg (preceding interview)	**Pollutant:** PM_{10} **Averaging Time:** 24-h **Copollutant (correlation):** $PM_{2.5}$ (r = 0.94) in Vienna	**PM Increment:** 10 $\mu g/m^3$ **Change in mean associated unit increase in PM (p-value)** **lag** Respiratory Health score Vienna: 0.005 ($p > 0.05$) lag 4 week avg Rural area: 0.008 ($p > 0.05$) lag 4 week avg Asthma score Vienna: 0.006 ($p > 0.05$) lag 4 week avg Rural area: -0.001 ($p > 0.05$) lag 4 week avg

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Oftedal et al. (2008, 093202) **Period of Study:** 2001-2002 **Location:** Oslo, Norway	**Outcome:** Lung function (PEF, $FEF_{25\%}$, $FEF_{50\%}$, FEV_1, FVC) **Age Groups:** 9-10 yrs **Study Design:** Cross-sectional **N:** 1847 children **Statistical Analyses:** Linear regression **Covariates:** Height, age, BMI, birth weight, temperature, maternal smoking, sex **Dose-response Investigated?** Yes **Statistical Package:** SPSS, STATA, S-Plus **Lags Considered:** 1-3	**Pollutant:** PM_{10} **IQR:** PM_{10} in 1st yr of life: 10.3 PM_{10} lifetime: 5.8	**PM Increment:** Per IQR β (Lower CI, Upper CI) PM_{10} in 1st yr of life PEF -72.5 (-122.3 to -22.7) $FEF_{25\%}$ -77.4 (-133.4 to -21.4) $FEF_{50\%}$ -53.9 (-102.6. to -5.2) FEV_1 -6.7 (-24.1, 10.7) FVC 0.5 (-18.5, 19.6) PM_{10} lifetime exposure PEF -66.4 (-109.5 to -23.3) $FEF_{25\%}$ -61.5 (-110.0 to -13.1) $FEF_{50\%}$ -45.6 (-87.7 to -3.5) FEV_1 -7.3 (-22.4, 7.7) FVC -2.1 (-18.6, 14.4)
Reference: Parker et al. (2009, 192359) **Period of Study:** 1999-2005 **Location:** US	**Outcome:** Respiratory allergy/hayfever **Study Design:** Cohort **Covariates:** survey year, age, family structure, usual source of care, health insurance, family income relative to federal poverty level, race/ethnicity **Statistical Analysis:** logistic regression **Statistical Package:** SUDAAN **Age Groups:** 73,198 children aged 3-17 years	**Pollutant:** PM_{10} **Averaging Time:** NR **Median:** 24.1 $\mu g/m^3$ **IQR:** 20.8-28.7 **Copollutant (correlation):** Summer O_3: 0.26 SO_2: -0.19 NO_2: 0.48 $PM_{2.5}$: 0.51 $PM_{10-2.5}$: 0.86	**Increment:** 10$\mu g/m^3$ **Odds Ratio (95% CI)** Single Pollutant Model, variable N Adjusted: 1.04 (0.99-1.09)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Penard-Morand et al. (2005, 087951) **Period of Study:** 03/1999 -- 10/2000 Mean concentrations of NO_2, SO_2, PM_{10}, and O_3 were taken from 01/01/1998 to 12/31/2000 **Location:** 6 French cities: Bordeaux, Clermont-Ferrand, Creteil, Marseille, Strasbourg, Reims.	**Outcome:** Flexural dermatitis Asthma (493) Rhinoconjunctivitis Atopic dermatitis Wheeze Allergic rhinitis Atopy EIB (exercise-induced bronchial reactivity) **Age Groups:** 9-11 years **Study Design:** Cross-sectional **N:** 9615 Children (6672 complete examination and questionnaire info) **Statistical Analyses:** Logistic regression Marginal Model (GENMOD) **Covariates:** Age, Sex, Family history of allergy, Passive smoking Parental education **Season:** All Excluding end of spring and during summer for clinical examinations **Dose-response Investigated?** No **Statistical Package:** SAS	**Pollutant:** PM_{10} **Averaging Time:** 3 years **Mean (SD):** Low concentrations: 26.9 High Concentrations: 23.8 **Range (Min, Max):** Low concentrations: 10-20 High concentrations: 21.5-29.5 **Copollutant (correlation):** NO_2: r =.46 SO_2: r =.76 O_3: r = -.02 **Monitoring Stations**: 16	**PM Increment:** 10 $\mu g/m^3$ (IQR) OR Estimate [Lower CI, Upper CI]: EIB (during exam): 1.43 (1.02-2.01) Flexural dermatitis (during exam): 0.79 (0.59-1.07) Wheeze (past year): 1.05 (0.72-1.54) Asthma (past year): 1.23 (0.77-1.95) Rhinoconjunctivitis (past year): 1.17 (0.86-1.59) Atopic dermatitis (past year): 1.28 (0.96-1.71) Asthma (lifetime): 1.32 (0.96-1.81) Allergic rhinitis (lifetime): 1.32 (1.04-1.68) Atopic dermatitis (lifetime): 1.09 (0.88-1.36) Atopy (lifetime): 0.98(0.80-1.22) Pollen: 1.14 (0.85-1.53) Indoor: 0.91 (0.72-1.15) Moulds: 1.00 (0.53-1.88) Highest correlated pollutant adjustments: EIB (during exam): 1.16 (0.72-1.85) Flexural dermatitis (during exam): 0.93 0(.60-1.43) Wheeze (past year): 1.31 (0.71-2.36) Asthma (past year): 1.25 (0.66-2.37) Rhinoconjunctivitis (past year): 1.22 (0.98-1.68) Atopic dermatitis (past year): 1.63 (1.07-2.49) Asthma (lifetime): 1.11 (0.70-1.74) Allergic rhinitis (lifetime): 1.19 (0.94-1.59) Atopic dermatitis (lifetime): 1.47 (1.07-2.00) Atopy (lifetime): 0.93(0.69-1.26) Pollen: 1.30 (0.98-1.57) Indoor: .83 (0.63-1.12) Moulds: 1.62 (0.64-4.09)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Peters et al., (1999, 087237) **Period of Study:** 1986-1990, 1994 **Location:** Southern California	**Outcome:** Asthma, cough, bronchitis, wheeze **Age Groups:** 4th, 7th, & 10th graders **Study Design:** cohort **N:** 3676 children **Statistical Analyses:** Stepwise logistic regression **Covariates:** Community, grade, race, sex, height, BMI, asthma in parents, hay fever, health insurance, plants in home, mildew in home, passive smoke exposure, pest infestation, carpet, vitamin supplements, active smoking, pets, gas stove, air conditioner **Dose-response Investigated?** Yes	**Pollutant:** PM_{10} **Averaging Time:** 24 h PM_{10} averaged over 1994 Mean based on data collected during 1986-1990, 1994: Alpine 37.4, 21.3 Atascadero 28.0, 20.7 Lake Elsinore 59.5, 34.7 Lake Gregory 38.3, 24.2 Lancaster 47.0, 33.6 Lompoc 30.0, 13.0 Long Beach 49.5, 38.8 Mira Loma 84.9, 70.7 Riverside 84.9, 45.2 San Dimas 67.0, 36.7 Santa Maria 28.0, 29.2 Upland 75.6, 49.0	**PM Increment:** 25 $\mu g/m^3$ OR (Lower CI, Upper CI) for respiratory illness Based on 1986-1990 pollutant levels Ever asthma 0.93 (0.76, 1.13) Current asthma 1.09 (0.86, 1.37) Bronchitis 0.94 (0.74, 1.19) Cough 1.06 (0.93, 1.21) Wheeze 1.05 (0.89, 1.25) Based on 1994 pollutant levels Ever asthma 0.87 (0.67, 1.14) Current asthma 1.11 (0.81, 1.54) Bronchitis 0.90 (0.65, 1.26) Cough 1.14 (0.96, 1.35) Wheeze 1.01 (0.79, 1.29)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Pierse, et al. (2006, 088757) **Period of Study:** 2 years (once in 1998 and once in 2001—surveys) **Location:** Leicestershire, UK	**Outcome:** Cough without a cold Night time cough Current wheeze **Age Groups:** 1-5 years **Study Design:** Cross-sectional (cohorts) **N:** 4400 children **Statistical Analyses:** Binomial generalized linear models (compared with likelihood ratio tests) Spatial variograms (due to the spatial concerns) **Covariates:** Age, Gender Mother/father has asthma Coal heating the home, Smoking by household member in the home, Either parent continued education past 16 years of age, Pre-term birth, Breast feeding, Gas cooking, Presence of pets, Number of cigarettes smoked by mother, Overcrowding, Single parenthood, Diet **Dose-response Investigated?** Yes (Fig. 2 shows evidence of dose-response effect based on surveys, states in discussion). **Statistical Package:** SAS 8.2 S-Plus 6.1	**Pollutant:** PM_{10} **Averaging Time:** annual PM_{10} **Mean (SD):** 1998: 1.47 2001: 1.33 Percentiles: 25th: 1998 (.73) and 2001 (.8) 75th: 1998 (1.93) and 2001 (1.84)	**PM Increment:** 10 $\mu g/m^3$ (IQR) **Unadjusted OR estimates [Lower CI, Upper CI]:** Cough without cold (1998): 1.22 (1.10 to 1.36) Cough without cold (2001): 1.46 (1.27 to 1.68) Night-time cough (1998): 1.11 (1.01 to 1.23) Night-time cough (2001): 1.25 (1.09 to 1.43) Current wheeze (1998): 0.99 (0.89 to 1.10) Current wheeze (2001): 1.09 (0.93 to 1.30) **Adjusted OR Estimate [Lower CI, Upper CI]:** Cough without cold (1998): 1.21 (1.07 to 1.38) Cough without cold (2001): 1.56 (1.32 to 1.84) Night-time cough (1998): 1.06 (0.94 to 1.19) Night-time cough (2001): 1.25 (1.06 to 1.47) Current wheeze (1998): 0.99 (0.88 to 1.12) Current wheeze (2001): 1.28 (1.04 to 1.58) **When the child was originally asymptomatic in 1998:** Unadjusted OR estimates [Lower CI, Upper CI]: Cough without cold (2001): 1.68 (1.39 to 2.03) Night-time cough (2001): 1.21 (1.00 to 1.46) Current wheeze (2001): 1.22 (0.92 to 1.62) Adjusted OR Estimate [Lower CI, Upper CI]: Cough without cold (2001): 1.62 (1.31 to 2.00) Night-time cough (2001): 1.19 (0.96 to 1.47) Current wheeze (2001): 1.42 (1.02 to 1.97)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Qian et al. (2005, 093283) **Period of Study:** 1990-1992 **Location:** Forsythe, NC Minneapolis, MN Jackson, MS.	**Outcome:** FVC, FEV_1, FEV_1/FVC **Age Groups:** middle aged (avg 56.8 years) **Study Design:** cross-sectional **N:** 10,240 people **Statistical Analyses:** regression equations, multiple linear regression analyses **Covariates:** Smoking status, recent use of respiratory medication, current respiratory symptoms, chronic lung diseases, field center **Dose-response Investigated?** No **Statistical Package:** SAS software, version 9.1	**Pollutant:** PM_{10} **Averaging Time:** Annual **Mean (SD):** 27.9 (2.8) Percentiles: 25th: 25.8 50th(Median): 27.5 75th: 30.2 Range (Maximum-Minimum): 12.2 **Monitoring Stations**: 3 (Minneapolis, MN) 5 (Jackson, MS) and 9 (Forsythe, NC) **Copollutant:** O_3	**PM Increment:** 2.8 $\mu g/m^3$ (1 SD) Effect Estimate: In Never Smokers FVC ß = -0.0108, SE = 0.0026, p =.0001 FEV_1 ß = -0.0082, SE = 0.0029, p =.0047 FEV_1/FVC ß = -0.0024, SE = 0.0023, p =.2787 Smoking status Current n = 2377, FVC = -1.96, FEV_1 = -2.23, FEV_1/FVC = -0.94 Former n = 3858, FVC = -1.25, FEV_1 = -1.10, FEV_1/FVC = -0.30 Never n = 4005, FVC = -1.12, FEV_1 = -0.63, FEV_1/FVC = 0.06 Recent Use of Respiratory Medication Yes n = 424, FVC = -2.65, FEV_1 = -3.89, FEV_1/FVC = -3.00 No n = 9816, FVC = -1.41, FEV_1 = -1.20, FEV_1/FVC = -0.24 Current Respiratory Symptoms Yes n = 4340, FVC = -1.68, FEV_1 = -1.70, FEV_1/FVC = -0.63 No n = 5900, FVC = -1.05, FEV_1 = -0.63, FEV_1/FVC = 0.05 Chronic Lung Diseases Yes n = 1374, FVC = -1.95, FEV_1 = -2.31, FEV_1/FVC = -1.18 No n = 8866, FVC = -1.35, FEV_1 = -1.10, FEV_1/FVC = -0.19 Field Center Forsythe, NC n = 3504, FVC = -0.03, FEV_1 = 0.05, FEV_1/FVC = -0.33 Minneapolis, MN n = 3793, FVC = 0.50, FEV_1 = 0.54, FEV_1/FVC = -0.30 Jackson, MS n = 2943, FVC = -0.01, FEV_1 = 0.17, FEV_1/FVC = -0.32

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Rios et al. (2004, 087800) **Period of Study:** 1998-2000 **Location:** the metropolitan area of Rio de Janiero, Brazil, Duque de Caxias (DC) and Seropedica (SR)	**Outcome:** wheezing, asthma, cough at night **Age Groups:** 13-14 yrs **Study Design:** cohort **N:** 4064 students **Statistical Analyses:** chi-squared **Covariates:** sex, type of school, time of residence, domestic smoking, residents per home **Dose-response Investigated?** Yes **Statistical Package:** EpiInfo	**Pollutant:** PM_{10} **Averaging Time:** weekly measurements used to create annual PM estimate **Mean (SD):** DC 1998: 147 1999: 115 2000: 110 Total: 124 SR 1998: 37 1999: 31 2000: 37 Total: 35 **Monitoring Stations**: NR	**PM Increment:** High vs. Low Global Cut-Off Score %, p-val: DC Male: 15.0 Female: 22.3, p < .05† Private School: 16.6 Public School: 19.4, p < .05* < 5yr residence: 20.9 > 5yr residence: 16.8 No domestic smoking exposure: 17.6 Domestic smoking exposure: 20.4, p < .05† < 5 residents per home: 18.4 5+ residents per home: 19.5 SR Male: 12.3 Female: 19.7, p < .05† Private School: 28.3, p < .05*† Public School: 14.7 < 5yr residence: 10.8 > 5yr residence: 16.5 No domestic smoking exposure: 14.8 Domestic smoking exposure: 18.3 < 5 residents per home: 15.6 5+ residents per home: 17.4 Notes: The Global Cut-off Score encompasses replies to the asthma component of ISAAC's written questionnaire that establishes a cut-off from which is defined the presence of asthma for the Brazilian population. *comparing the cities in the same controlled variable †comparing the controlled variable in the same city
Reference: Rojas-Martinez et al. (2007, 091064) **Period of Study:** 1996-1999 **Location:** Mexico City, Mexico	**Outcome:** Lung function: FEV_1, FVC, $FEF_{25-75\%}$ **Age Groups:** Children 8 years old at time of cohort recruitment **Study Design:** school-based "dynamic" cohort study **N:** 3170 children 14,545 observations **Statistical Analyses:** Three-level generalized linear mixed models with unstructured variance-covariance matrix **Covariates:** age, body mass index, height, height by age, weekday spent outdoors, environmental tobacco smoke, previous-day mean air pollutant concentration, time since first test **Dose-response Investigated?** No	**Pollutant:** PM_{10} **Averaging Time:** 6-mo **Mean (SD):** 6-mo averaging SD: NR Mean: 75.6 **Percentiles:** 6-mo averaging 25th: 55.8 50th(Median): 67.5 75th: 92.2 **Monitoring Stations**: 5 sites for PM_{10}, 10 for other pollutants **Copollutant:** O_3 NO_2	**PM Increment:** IQR 6-LC: 36.4 Slope [Lower CI, Upper CI] Girls One-pollutant model FVC: -39 [-47: -31] FEV: -29 [-36: -21] $FEF_{25-75\%}$: -17 [-36: 1] FEV_1/FVC: 0.12 [0.07: 0.17] Two-pollutant model: PM_{10}, 6-LC & O_3 FVC: -30 [-39: -22] FEV: -24 [-31: -16] $FEF_{25-75\%}$: -9 [-26: 9] FEV_1/FVC: 0.10 [0.06: 0.15]

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
	Statistical Package: SA		PM_{10}, 6-LC & NO_2
			FVC: -21 [-30: -13]
			FEV: -17 [-25: -8]
			$FEF_{25-75\%}$: -23 [-43: -4]
			FEV_1/FVC: 0.07 [0.02: 0.13]
			Multipollutant model: PM_{10}, 6-LC, O_3, & NO_2
			FVC: -14 [-23: -5]
			FEV: -11 [-20: -3]
			$FEF_{25-75\%}$: -7 [-27: 12]
			FEV_1/FVC: 0.08 [0.03: 0.13]
			Boys
			One-pollutant model
			FVC: -33 [-41: -25]
			FEV: -27 [-34: -19]
			$FEF_{25-75\%}$: -18 [-34: -2]
			FEV_1/FVC: 0.04 [-0.01: 0.09]
			Two-pollutant model: PM_{10}, 6-LC & O_3
			FVC: -28 [-36: -19]
			FEV: -22 [-30: -15]
			$FEF_{25-75\%}$: -10 [-27: 7]
			FEV_1/FVC: 0.04 [-0.01: 0.09]
			FEV_1/FVC: 0.24 [0.13: 0.34]
			PM_{10}, 6-LC & NO_2
			FVC: -16 [-26: -7]
			FEV: -19 [-27: -10]
			$FEF_{25-75\%}$: -26 [-44: -9]
			FEV_1/FVC: 0.005 [-0.06: 0.05]
			Multipollutant model PM_{10}, 6-LC, O_3, & NO_2
			FVC: -12 [-22: -3]
			FEV: -15 [-23: -6]
			$FEF_{25-75\%}$: -12 [-30: 6]
			FEV_1/FVC: -0.002 [-0.06: 0.05]

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Schikowski et al. (2005, 088637) **Period of Study:** 1985-1994 **Location:** Rhine-Ruhr Basin of Germany [Dortmund (1985, 1990), Duisburg (1990), Gelsenkirchen (1986, 1990), and Herne (1986)]	**Outcome:** Respiratory symptoms & pulmonary function **Age Groups:** age 54-55 **Study Design:** Cross-sectional **N:** 4757 women **Statistical Analyses:** Linear & Logistic regressions, including random effects model **Covariates:** age, smoking, SES, occupational exposure, form of heating, BMI, height **Season:** NR **Dose-response Investigated?** No **Statistical Package:** SAS **Lags Considered:** NR	**Pollutant:** PM_{10} **Averaging Time:** NR Min, P25, Median, Mean, P75, Max Annual Mean 35, 40, 43, 44, 47, 53 Five year Mean 39, 43, 47, 48, 53, 56 **Monitoring Stations**: 7 **Copollutant (correlation):** NR	**PM Increment:** 7 $\mu g/m^3$ OR (Lower CI, Upper CI) for asthma symptoms Annual means Chronic bronchitis 1.00 (0.85, 1.18) Chronic cough 1.03 (0.87, 1.23) Frequent cough 1.01 (0.93, 1.10) COPD 1.37 (0.98, 1.92) $p < 0.1$ FEV_1 0.953 (0.916, 0.989) $p < 0.1$ FVC 0.966 (0.940, 0.992) $p < 0.1$ FEV_1/FVC 0.989 (0.978, 1.000) $p < 0.1$ Five year means Chronic bronchitis 1.13 (0.95, 1.34) Chronic cough 1.11 (0.93, 1.31) Frequent cough 1.05 (0.94, 1.17) COPD 1.33 (1.03, 1.72) $p < 0.1$ FEV_1 0.949 (0.923, 0.975) $p < 0.05$ FVC 0.963 (0.945, 0.982) $p < 0.05$ FEV_1/FVC 0.989 (0.980, 0.997) $p < 0.1$
Reference: Schindler et al, (2009, 191950) **Period of Study:** 1991-2002 **Location:** Switzerland	**Outcome:** Respiratory Symptoms **Study Design:** Prospective Cohort **Statistical Analysis:** Logistic Regression Model **Age Groups:** Adults, 18-60 years of age at start of study **Covariates:** sex, age, level of education, Swiss citizenship, BMI, parental smoking, parental history of asthma/atopy, early respiratory infection, smoking status, pack years, daily number of cigarettes, years since smoking cessation, passive smoking in general/at work, occupational exposure to airbourne irritants	**Pollutant:** PM_{10} **Averaging Time:** Annual **Mean (SD) Unit:** **Range (Min, Max):** **Copollutant (correlation):** NR	**Increment:** 10$\mu g/m^3$ **Odds Ratio (95%CI) of reporting symptoms at second interview** Entire Sample, New Reports Regular Cough: 0.77 (0.62-0.97) Regular Phlegm: 0.74 (0.56-0.99) Chronic Cough or Phlegm: 0.78 (0.62-0.98) Wheezing: 1.01 (0.74-1.39) Wheezing with Dyspnea: 0.70 (0.49-1.01) Wheezing without Cold: 1.06 (0.76-1.50) Entire Sample, Persistent Reports Regular Cough: 0.55 (0.39-0.78) Regular Phlegm: 0.82 (0.52-1.33) Chronic Cough or Phlegm: 0.67 (0.40-1.15) Wheezing: 0.50 (0.32-0.80) Wheezing with Dyspnea: 0.59 (0.30-1.23) Wheezing without Cold: 0.61- (0.35-1.12) Persistent Non-Smokers, New Reports

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			Regular Cough: 0.86 (0.63-1.19)
			Regular Phlegm: 0.70 (0.49-0.99)
			Chronic Cough or Phlegm: 0.71 (0.52-0.99)
			Wheezing: 0.93 (0.60-1.46)
			Wheezing with Dyspnea: 0.77 (0.50-1.20)
			Wheezing without Cold: 1.11 (0.66-1.92)
			Persistent Non-Smokers, Persistent Reports
			Regular Cough: 0.28 (0.14-0.60)
			Regular Phlegm: 0.87 (0.43-1.84)
			Chronic Cough or Phlegm: 0.35 (0.16-0.81)
			Wheezing: 0.53 (0.28-1.08)
			Wheezing with Dyspnea: 0.76 (0.30-2012)
			Wheezing without Cold: 0.61 (0.26-1.52)
			Gender-specific odds ratio (95%CI) of reporting symptoms at second interview
			New Reports
			Regular Cough, p = 0.73
			Men: 0.75 (0.53-1.06)
			Women: 0.81 (0.58-1.15)
			Regular Phlegm, p = 0.41
			Men: 0.85 (0.60-1.20)
			Women: 0.68 (0.46-1.00)
			Chronic Cough or Phlegm: 0.36
			Men: 0.87 (0.63-1.21)
			Women: 0.71 (0.51-0.97)
			Wheezing, p = 0.20
			Men: 0.83 (0.57-1.20)
			Women: 1.20 (0.78-1.87)
			Wheezing with Dyspnea, p = 0.11
			Men: 0.56 (0.36-0.87)
			Women: 1.00.57-1.842
			Wheezing without Cold, p = 0.43
			Men: 0.95 (0.63-1.42)
			Women: 1.25 (0.72-2.17)
			Persistent Reports
			Regular Cough, p = 0.02
			Men: 0.75 (0.48-1.18)
			Women: 0.31 (0.17-0.56)
			Regular Phlegm, p = 0.33
			Men: 0.65 (0.37-1.12)
			Women: 1.04 (0.47-2.34)
			Chronic Cough or Phlegm: 0.47

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			Men: 0.68 (0.39-1.20)
			Women: 0.47 (0.20-1.11)
			Wheezing, p = 0.29
			Men: 0.34 (0.17-0.72)
			Women: 0.57 (0.32-1.01)
			Wheezing with Dyspnea, p = 0.63
			Men: 0.56 (0.16-1.95)
			Women: 0.37 (0.13-1.05)
			Wheezing without Cold, p = 0.57
			Men: 0.34 (0.12-0.91)
			Women: 0.49 (0.21-1.15)
			Odds Ratio (95%CI) of reporting symptoms at second interview with additional adjustment for annual outdoor PM exposure at baseline
			Entire Sample
			Regular Cough, p = 0.0003
			New Reports: 0.77 (0.61-0.97)
			Persistent Reports: 0.55 (0.39-0.78)
			Regular Phlegm, p = 0.13
			New Reports: 0.77 (0.59-1.02)
			Persistent Reports: 0.79 (0.46-1.33)
			Chronic Cough or Phlegm, p = 0.02
			New Reports: 0.78 (0.62-0.98)
			Persistent Reports: 0.64 (0.40-1.02)
			Wheezing, p = 0.002
			New Reports: 0.91 (0.63-1.33)
			Persistent Reports: 0.47 (0.31-0.72)
			Wheezing with Dyspnea, p = 0.03
			New Reports: 0.65 (0.43-0.98)
			Persistent Reports: 0.55 (0.28-1.10)
			Severe Wheezing, p = 0.28
			New Reports: 0.96 (0.66-1.40)
			Persistent Reports: 0.62 (0.34-1.12)
			Non-Smokers
			Regular Cough, p $<$ 0.001
			New Reports: 0.87 (0.63-1.19)
			Persistent Reports: 0.29 (0.16-0.52)
			Regular Phlegm, p = 0.07
			New Reports: 0.70 (0.50-0.99)
			Persistent Reports: 0.67 (0.34-1.33)
			Chronic Cough or Phlegm, p = 0.008
			New Reports: 0.72 (0.52-0.99)
			Persistent Reports: 0.38 (0.17-0.84)
			Wheezing, p = 0.07
			New Reports: 0.87 (0.52-1.48)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			Persistent Reports: 0.48 (0.25-0.91) Wheezing with Dyspnea, p = 0.36 New Reports: 0.76 (0.48-1.19) Persistent Reports: 0.70 (0.27-1.82) Severe Wheezing, p = 0.57 New Reports: 1.11 (0.64-1.93) Persistent Reports: 0.64 (0.26-1.54)
Reference: Sharma et al. (2004, 156974) **Period of Study:** 11/2002–4/2003 **Location:** 3 sections in Kanpur City, India: 1) Indian Institute of Technology Kanpur (IITK) 2) Vikas Nagar (VN) 3) Juhilal Colony (JC)	**Outcome:** Lung function **Age Groups:** 20–55 years **Study Design:** Cohort **N:** 91 people **Statistical Analyses:** Linear regression **Covariates:** NR **Season:** Fall, Winter, spring **Dose-response Investigated?** No **Statistical Package:** Microsoft Excel **Lags Considered:** 1d lag & 5d mov avg	**Pollutant:** PM_{10} **Averaging Time:** 24-h **Mean (SD):** IITK 184 (40) VN 295 (58) JC 293 (90) **PM Component:** Lead, Nickel, Cadmium, Chromium, Iron, Zinc Benzene soluble fraction (includes polycyclic aromatic hydrocarbons [PAHs]) **Copollutant (correlation):** □PEF = mean daily deviations in PEF PM_{10}-□PEF: (-0.52) PM_{10}-$PM_{2.5}$: (0.67) PM_{10}-PM_{10} (1-day lag): (0.45) PM_{10}-$PM_{2.5}$ (1-day lag): (0.46)	**PM Increment:** 1 $\mu g/m^3$ □PEF (difference or change in peak expiratory flow) -0.0318 L/min
Reference: Tager et al. (2005, 087538) **Period of Study:** 4/2000- 6/2000, 2/2001–6/2001, 2/2002–6/2002 **Location:** Los Angeles, California San Francisco, California	**Outcome:** Lung Function (FEV_1, FVC, PEFR, FEF75, FEF_{25-75}, FEF_{25-75}/FVC ratio) **Age Groups:** 16-21 + y/o College Freshman **Study Design:** Retrospective cohort **N:** 255 students 108 Men (M) 147 Women (W) **Statistical Analyses:** Multivariate Linear Regression **Covariates:** Sex, height, weight, area of residence, age, race, ETS exposure, respiratory disease history **Dose-response Investigated?** No	**Pollutant:** PM_{10} **Averaging Time:** Cumulative lifetime exposure Median: Prior to 1987: M: 73 W: 71 1987 and later: M: 36 W: 34 Lifetime: M: 48 W: 45 **Range (Min, Max):** Prior to 1987: M: 34, 117 W: 31, 124 1987 and later: M: 18, 68 W: 20, 61 Lifetime: M: 21, 80 W: 18, 71 **Monitoring Stations**: Between 1 and 3 **Copollutant (correlation):** O_3 prior to 1987: r = 0.68 O_3 1987 and later: r = 0.81 O_3–Lifetime: r = 0.57	**PM Increment:** 1 $\mu g/m^3$ Parameter Estimates (SD) (Lifetime PM_{10}, Interaction PM_{10} FEF_{25-75}/FVC) LnFEF75: M: -0.009 (0.0009), 0.009 (0.007) W: -0.010 (0.0007), 0.008 (0.0005)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Tamura et a. (2003, 087445) **Period of Study:** 1998-1999 **Location:** Bangkok, Thailand	**Outcome:** non-specific respiratory disease (Chronic bronchitis, acute bronchitis, bronchial asthma, dyspnea and wheezing) **Age Groups:** adults **Study Design:** Cross-sectional **N:** 1603 policemen **Statistical Analyses:** Multiple logistic regression **Covariates:** age, smoking status **Dose-response Investigated?** Yes **Statistical Package:** SPSS	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (SD):** Heavily Polluted 80-190 Moderately Polluted 60-69 Control 59 **Monitoring Stations**: 13	**PM Increment:** Heavily Polluted vs. Moderately Polluted vs. Control Number and Prevalence (%) of respiratory disease among heavily polluted, moderately polluted, and control areas. **Heavily Polluted** Chronic bronchitis 16 (3.0) Acute bronchitis 19 (3.5) Bronchial asthma 5 (0.9) Dyspnea & wheezing 49 (9.2) Any 1 of above 69 (13.0) Persistent cough 11 (2.1) Persistent phlegm 27 (1.3) Cough & phlegm 6 (1.1) **Moderately Polluted** Chronic bronchitis 8 (2.4) Acute bronchitis 12 (9.0) Bronchial asthma 2 (0.6) Dyspnea & wheezing 23 (6.8) Any 1 of above 37 (10.9) Persistent cough 1 (0.3) Persistent phlegm 11 (3.3) Cough & phlegm 1 (0.3) Control Chronic bronchitis 6 (1.9) Acute bronchitis 11 (3.3) Bronchial asthma 0 (0.0) Dyspnea & wheezing 23 (7.2) Any 1 of above 31 (9.4) Persistent cough 1 (0.3) Persistent phlegm 8 (2.4) Cough & phlegm 1 (0.3)
Reference: Wheeler and Ben-Schlomo (2005, 089860) **Period of Study:** 1995-1997 **Location:** England	**Outcome:** FEV_1 **Age Groups:** 16-79 yrs **Study Design:** Data from Health Survey for England were coupled geographically with air pollution measurements on a 1 km grid. **N:** 26,426 households with 39,251 adults **Statistical Analyses:** Logistic regression, least squares regression **Covariates:** Age, sex, height, body mass index, smoking status, household passive smoke exposure, inhaler use in the previous 24-hs, doctor diagnosis of asthma. **Dose-response Investigated?** No	**Pollutant:** PM_{10} **Averaging Time:** 1996 annual mean **Mean (SD):** 23.95 (3.58) **Range (Min, Max):** 17.87-43.37	β (95%CI) for Height-age standardized FEV_1 by ambient air quality index p-value Male Good (ref) Poor -0.023 (-0.030 to -0.016) <0.001 Female Good (ref) Poor -0.019 (-0.026 to -0.013) <0.001

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Zhang et al., (2002, 034814) **Period of Study:** 1993-1996 **Location:** 4 Chinese cities (urban and suburban location in each city): Guangzhou, Wuhan, Lanzhou, Chongqing	**Outcome:** Interview-self reports of symptoms: Wheeze (ever wheezy when having a cold) Asthma (diagnosis by doctor) Bronchitis (diagnosis by doctor), Hospitalization due to respiratory disease (ever) Persistent cough (coughed for at least 1 month per year with or apart from colds) Persistent phlegm (brought up phlegm or mucus from the chest for at least 1 month per year with or apart from colds) **Age Groups:** Elementary school students age range: 5.4–16.2 **Study Design:** Cross-sectional **N:** 7,557 returned questionnaires 7,392 included in first stage of analysis **Statistical Analyses:** 2-stage regression approach: Calculated odds ratios and 95% CIs of respiratory outcomes and covariates Second stage consisted of variance-weighted linear regressions that examined associations between district-specific adjusted prevalence rates and district-specific ambient levels of each pollutant. **Covariates:** Age, gender, breast-fed, house type, number of rooms, sleeping in own or shared room, sleeping in own or shared bed, home coal use, ventilation device used, homes smokiness during cooking, eye irritation during cooking, parental smoking, mother's education level, mother's occupation, father's occupation, questionnaire respondent, year of questionnaire administration, season of questionnaire administration, parental asthma prevalence	**Pollutant:** PM_{10} **Averaging Time:** 2 years **Mean (SD):** 151 (56) IQR: 87 **Range (Min, Max):** Gives range (max.–min.): 80 **Monitoring Stations:** 2 types: municipal monitoring stations over a period of 4 years (1993-1996) schoolyards of participating children over a period of 2 years (1995–1996)	**PM Increment:** Interquartile range corresponded to 1 unit of change. RR Estimate [Lower CI, Upper CI] lag: Association between persistent phlegm and PM_{10}: 3.21 (1.55, 6.67) $p < 0.05$ - Between and within city modeled ORs, scaled to interquartile range of concentrations for each pollutant. No associations between any type of respiratory outcome and PM_{10} When scaled to an increment of 50 $\mu g/m^3$ of PM_{10}, ORs were: Wheeze: 1.07 Asthma: 1.18 Bronchitis: 1.53 Hospitalization: 1.17 Persistent cough: 1.20 Persistent phlegm: 1.95

[1]All units expressed in $\mu g/m^3$ unless otherwise specified.

Table E-23. Long-term exposure - respiratory morbidity outcomes - $PM_{10-2.5}$.

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Chattopadhyay et al. (2007, 147471) **Period of Study:** NR **Location:** Three different points in Kolkata, India: North, South, and Central	**Outcome:** pulmonary function tests (respiratory impairments) **Age Groups:** All ages **Study Design:** Cross-sectional **N:** 505 people studied for PFT total population of Kolkata not given **Statistical Analyses:** Frequencies **Covariates:** Meteorologic data (i.e. temperature, wind direction, wind speed, and humidity) **Dose-response Investigated?** No	**Pollutant:** $PM_{<3.3-0.4}$ **Averaging Time:** 8 h **Mean (SD):** North Kolkata: 266.1 Central Kolkata: 435.3 South Kolkata: 449.1 Unit (i.e. $\mu g/m^3$): $\mu g/m^3$ **Monitoring Stations:** 1 **Copollutant (correlation):** PM_{10} $PM_{<10-3.3}$	**PM Increment:** NR Respiratory impairments (SD): North Kolkata Male (n = 137) Restrictive: 4 (2.92) Obstructive: 5 (3.64) Combined Res. And Obs.: 6 (4.37) Total: 15 (10.95) Female (n = 152) Restrictive: 3 (1.97) Obstructive: 5 (3.28) Combined Res. And Obs.: 0

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			Total: 8 (5.26)
			Total (n = 289)
			Restrictive: 7 (2.42)
			Obstructive: 10 (3.46)
			Combined Res. And Obs.: 6 (2.07)
			Total: 23 (7.96)
			Central Kolkata
			Male (n = 44)
			Restrictive: 6 (13.63)
			Obstructive: 1 (2.27)
			Combined Res. And Obs.: 1 (2.27)
			Total: 8 (18.18)
			Female (n = 50)
			Restrictive: 3 (6.00)
			Obstructive: 2 (4.00)
			Combined Res. And Obs.: 0
			Total: 5 (10.00)
			Total (n = 94)
			Restrictive: 9 (9.57)
			Obstructive: 3 (3.19)
			Combined Res. And Obs.: 1 (1.06)
			Total: 13 (13.82)
			South Kolkata
			Male (n = 52)
			Restrictive: 1 (1.92)
			Obstructive: 2 (3.84)
			Combined Res. And Obs.: 3 (5.76)
			Total: 6 (11.53)
			Female (n = 70)
			Restrictive: 2 (2.85)
			Obstructive: 1 (1.42)
			Combined Res. And Obs.: 0
			Total: 3 (4.28)
			Total (n = 122)
			Restrictive: 3 (2.45)
			Obstructive: 3 (2.45)
			Combined Res. And Obs.: 3 (2.45)
			Total: 9 (7.37)
Reference: Chattopadhyay et al. (2007, 147471) **Period of Study:** NR **Location:** Three different points in Kolkata, India: North, South, and Central	**Outcome:** pulmonary function tests (respiratory impairments) **Age Groups:** All ages **Study Design:** Cross-sectional **N:** 505 people studied for PFT total population of Kolkata not given	**Pollutant:** $PM_{<10-3.3}$ **Averaging Time:** 8 h **Mean (SD):** North Kolkata: 269.8 Central Kolkata: 679.2 South Kolkata: 460.1	**PM Increment:** NR Respiratory impairments (SD): North Kolkata Male (n = 137) Restrictive: 4 (2.92) Obstructive: 5 (3.64)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
	Statistical Analyses: Frequencies	Unit (i.e. $\mu g/m^3$): $\mu g/m^3$	Combined Res. And Obs.: 6 (4.37)
	Covariates: Meteorologic data (i.e. temperature, wind direction, wind speed, and humidity)	**Monitoring Stations**: 1	Total: 15 (10.95)
		Copollutant (correlation):	Female (n = 152)
	Dose-response Investigated? No	PM_{10}	Restrictive: 3 (1.97)
		$PM_{<3.3-0.}$	Obstructive: 5 (3.28)
			Combined Res. And Obs.: 0
			Total: 8 (5.26)
			Total (n = 289)
			Restrictive: 7 (2.42)
			Obstructive: 10 (3.46)
			Combined Res. And Obs.: 6 (2.07)
			Total: 23 (7.96)
			Central Kolkata
			Male (n = 44)
			Restrictive: 6 (13.63)
			Obstructive: 1 (2.27)
			Combined Res. And Obs.: 1 (2.27)
			Total: 8 (18.18)
			Female (n = 50)
			Restrictive: 3 (6.00)
			Obstructive: 2 (4.00)
			Combined Res. And Obs.: 0
			Total: 5 (10.00)
			Total (n = 94)
			Restrictive: 9 (9.57)
			Obstructive: 3 (3.19)
			Combined Res. And Obs.: 1 (1.06)
			Total: 13 (13.82)
			South Kolkata
			Male (n = 52)
			Restrictive: 1 (1.92)
			Obstructive: 2 (3.84)
			Combined Res. And Obs.: 3 (5.76)
			Total: 6 (11.53)
			Female (n = 70)
			Restrictive: 2 (2.85)
			Obstructive: 1 (1.42)
			Combined Res. And Obs.: 0
			Total: 3 (4.28)
			Total (n = 122)
			Restrictive: 3 (2.45)
			Obstructive: 3 (2.45)
			Combined Res. And Obs.: 3 (2.45)
			Total: 9 (7.37)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Dales et al., (2008, 156378) **Period of Study: Location:** Windsor, ON	**Outcome:** Pulmonary function and inflammation **Age Groups:** grades 4-6 **Study Design:** cross-sectional prevalence design **Statistical Analyses:** multivariate linear regression **Covariates:** Ethnic background, smokers at home, pets at home, acute respiratory illness, medication use	**Pollutant:** $PM_{10-2.5}$ **Averaging Time:** Annual Mean: 7.25 5th: 6.02 95th: 8.23 Copollutant: SO_2 NO_2	**Increment:** Tertiles of exposure FEV_1: < 7.04: 2.18 ± 0.01 7.04-7.53: 2.19 ± 0.02 > 7.53: 2.14 ± 0.01 FVC: < 7.04: 2.52 ± 0.02 7.04-7.53: 2.53 ± 0.02 > 7.53: 2.48 ± 0.02 eNO: < 7.04: 15.48 ± 0.63 7.04-7.53: 16.73 ± 0.76 > 7.53: 16.59 ± 0.79
Reference: Gauderman et al. (2000, 012531) **Period of Study:** 1993-1997 **Location:** Southern California	**Outcome:** FVC, FEV_1, MMEF, FEF_{75} **Age Groups:** fourth, seventh, or tenth graders **Study Design:** cohort **N:** 3035 subjects **Statistical Analyses:** Linear regression **Covariates:** Height, weight, BMI, asthma, smoking, exercise, room temperature, barometric pressure **Dose-response Investigated?** Yes **Statistical Package:** SAS	**Pollutant:** $PM_{10-2.5}$ **Averaging Time:** 24 h avg PM_{10} & annual avg of 2-week avg $PM_{2.5}$ **Mean (SD):** $PM_{10-2.5}$ 25.6 **Copollutant (correlation):** O_3 r = -0.29 NO_2 r = 0.44 Inorg. Acid r = 0.43	**Increment:** 25.6 $\mu g/m^3$ % Change (Lower CI, Upper CI) $PM_{10-2.5}$-4th grade FVC -0.57 (-1.20 to -0.06) FEV_1 -0.90 (-1.71 to -0.09) MMEF -1.37 (-2.57 to -0.15) FEF_{75} -1.62 (-3.24, 0.04) $PM_{10-2.5}$-7th grade FVC -0.35 (-1.02, 0.31) FEV_1 -0.49 (-1.21, 0.24) MMEF -0.64 (-2.83, 1.60) FEF_{75} -0.74 (-2.65, 1.20) $PM_{10-2.5}$-10th grade FVC -0.17 (-1.32, 0.99) FEV_1 -0.68 (-2.15, 0.81) MMEF -1.41 (-5.85, 3.25) FEF_{75} -2.32 (-6.60, 2.17)
Reference: Gauderman et al. (2002, 026013) **Period of Study:** 1996–2000 **Location:** Southern California	**Outcome:** Lung function development: FEV_1, maximal mid-expiratory flow (MMEF) **Age Groups:** Fourth grade children (avg age = 9.9 yrs) **Study Design:** Cohort study **N:** 1678 children, 12 communities **Statistical Analyses:** Mixed model linear regression **Covariates:** Height, BMI, doctor-diagnosed asthma and cigarette smoking in previous year, respiratory illness and exercise on day of test, interaction of each of these variables with sex, barometric pressure, temperature at test time, indicator variables for field technician and spirometer **Dose-response Investigated?** Yes **Statistical Package:** SAS (10)	**Pollutant:** $PM_{10-2.5}$ **Averaging Time:** Annual 24 h averages **Mean (SD):** The avg levels were presented in an online data supplement (Figure E1) **Monitoring Stations**: 12 **Copollutant (correlation):** O_3 (10 AM to 6 PM) r = 0.10 O_3 r = -0.31 NO_2 r = 0.46 Acid vapor r = 0.63 PM_{10} r = 0.95 $PM_{10-2.5}$ r = 0.81 EC r = 0.71 OC r = 0.96	**PM Increment:** 29.1 $\mu g/m^3$ Association Estimate: $PM_{10-2.5}$ was not correlated with any of the pulmonary function tests that were analyzed

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Leonardi et al. (2000, 010272) **Period of Study:** 1996 **Location:** 17 cities of Central Europe (Bulgaria, Czech Republic, Hungary , Poland, Romania, Slovakia)	**Outcome:** Immune biomarkers **Age Groups:** 9-11 **Study Design:** Cross-sectional **N:** 366 school children **Statistical Analyses:** Linear regression **Covariates:** Age, gender, parental smoking, laboratory of analysis, recent respiratory illness **Dose-response Investigated?** No **Statistical Package:** STATA	**Pollutant:** $PM_{10-2.5}$ **Averaging Time:** subtracting $PM_{2.5}$ from PM_{10} provides avg $PM_{10-2.5}$ **Mean (SD):** $PM_{10-2.5}$: 20 (5) **Range (Min, Max):** $PM_{10-2.5}$: (12, 38) 5th, median, & 95th percentile $PM_{10-2.5}$: 12, 19, 29	**% Change (Lower CI, Upper CI)** **p-value** $PM_{10-2.5}$ Neutrophils 1 (-27, 38) >.20 Total lymphocytes 8 (-15, 38) >.20 B lymphocytes 22 (-16, 76) >.20 Total T lymphocytes 2 (-25, 37) >.20 CD4+ -1 (-30, 41) >.20 CD8+ 3 (-25, 41) >.20 CD4/CD8 0 (-23, 30) >.20 NK 1 (-33, 51) >.20 Total IgG -3 (-21, 18) >.20 Total IgM 19 (-9, 55) >.20 Total IgA 16 (-12, 52) >.20 Total IgE -29 (-70, 70) >.20
Reference: McConnell et al. (2003, 049490) **Period of Study:** 1993-99 **Location:** 12 Southern CA communities	**Outcome:** bronchitic symptoms **Age Groups:** 9-19 **Study Design:** communities selected on basis of historic levels of criteria pollutants and low residential mobility. **N:** 475 children **Statistical Analyses:** 3 stage regression combined to give a logistic mixed effects model **Covariates:** sex, ethnicity, allergies history, asthma history, SES, insurance status, current wheeze, current exposure to ETS, personal smoking status, participation in team sports, in utero tobacco exposure through maternal smoking, family history of asthma, amount of time routinely spent outside by child during 2-6 pm. **Dose-response Investigated?** No **Statistical Package:** SAS Glimmix macro	**Pollutant:** $PM_{10-2.5}$ **Averaging Time:** 4 year avg **Mean (SD):** 17.0(6.4) **Range (Min, Max):** 10.2-35.0 **Copollutant (correlation):** $PM_{2.5}$: r = 0.24 PM_{10}: r = 0.79 Inorganic acid: r = 0.38 Organic Acid: r = 0.35 EC: r = 0.30 OC: r = 0.27 NO_2: r = -0.22 O_3: r = 0.29	**PM Increment:** Between community range 24.8 $\mu g/m^3$ Between community unit 1 $\mu g/m^3$ Within community 1 $\mu g/m^3$ OR Estimate [Lower CI, Upper CI] Between community per range 1.38(0.65-2.92) Between Community per unit 1.01(0.98-1.04) Within community per unit 1.02(0.95-1.10)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Millstein et al. (2004, 088629) **Period of Study:** Mar-Aug, 1995, and Sep, 1995 to Feb, 1996 Data were taken from the Children's Health Study **Location:** Alpine, Atascadero, Lake Arrowhead, Lake Elsinore, Lancaster, Lompoc, Long Beach, Mira Loma, Riverside, San Dimas, Santa Maria, and Upland, CA	**Outcome:** Wheezing & asthma medication use **Age Groups:** 4th grade students, mostly 9 yrs at the time of the study **Study Design:** Cohort Study, stratified into 2 seasonal groups/ **N:** 2081 enrolled, 2034 provided parent-completed questionnaire. **Statistical Analyses:** Multilevel, mixed-effects logistic model. **Covariates:** Contagious respiratory disease, ambient airborne pollen and other allergens, temperature, sex, age race, allergies, pet cats, carpet in home, environmental tobacco smoke, heating fuel, heating system, water damage in home, education level of questionnaire signer, physician diagnosed asthma. **Season:** Mar-Aug, 1995, and Sep, 1995 to Feb, 1996 **Statistical Package:** SAS 8.00 **Lags Considered:** 14	**Pollutant:** $PM_{10-2.5}$ **Averaging Time:** monthly **PM Component:** Nitric acid, formic acid, acetic acid **Monitoring Stations:** 1 central location in each community **Copollutant (correlation):** NO_2: r = 0.29 O_3: r = 0.77 $PM_{2.5}$: r = -0.08	**PM Increment:** IQR 11.44 $\mu g/m^3$ Odds Ratio [lower CI, Upper CI] Annual $PM_{10-2.5}$: 0.96 [0.74, 1.25] March-August $PM_{10-2.5}$: 0.93 [0.54, 1.59] Sep-Feb $PM_{10-2.5}$: 0.68 [0.46, 1.01]
Reference: (Parker et al., 2009, 192359) **Period of Study:** 1999-2005 **Location:** US	**Outcome:** Respiratory allergy/hayfever **Study Design:** Cohort **Covariates:** survey year, age, family structure, usual source of care, health insurance, family income relative to federal poverty level, race/ethnicity **Statistical Analysis:** logistic regression **Statistical Package:** SUDAAN **Age Groups:** 73,198 children aged 3-17 years	**Pollutant:** $PM_{10-2.5}$ **Averaging Time:** NR **Median:** 11.2 $\mu g/m^3$ **IQR:** 8.2-15.2 **Copollutant (correlation):** Summer O_3: 0.16 SO_2: -0.33 NO_2: 0.29 $PM_{2.5}$: 0.02 PM_{10}: 0.86	**Increment:** 10$\mu g/m^3$ **Odds Ratio (95% CI)** Single Pollutant Model, variable N Adjusted: 1.01 (0.95-1.07) Single Pollutant Model, constant N Adjusted: 1.13 (1.04-1.46) Multi-pollutant Model: 1.16 (1.06-1.24)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Zhang et al. (2002, 034814) **Period of Study:** 1993-1996 **Location:** 4 Chinese cities (urban and suburban location in each city): Guangzhou, Wuhan, Lanzhou, Chongqing	**Outcome:** Interview-self reports of symptoms: Wheeze (ever wheezy when having a cold) Asthma (diagnosis by doctor) Bronchitis (diagnosis by doctor), Hospitalization due to respiratory disease (ever) Persistent cough (coughed for at least 1 month per year with or apart from colds) Persistent phlegm (brought up phlegm or mucus from the chest for at least 1 month per year with or apart from colds) **Age Groups:** Elementary school students age range: 5.4–16.2 **Study Design:** Cross-sectional **N:** 7,557 returned questionnaires 7,392 included in first stage of analysis **Statistical Analyses:** 2-stage regression approach: Calculated odds ratios and 95% CIs of respiratory outcomes and covariates Second stage consisted of variance-weighted linear regressions that examined associations between district-specific adjusted prevalence rates and district-specific ambient levels of each pollutant. **Covariates:** Age, gender, breast-fed, house type, number of rooms, sleeping in own or shared room, sleeping in own or shared bed, home coal use, ventilation device used, homes smokiness during cooking, eye irritation during cooking, parental smoking, mother's education level, mother's occupation, father's occupation, questionnaire respondent, year of questionnaire administration, season of questionnaire administration, parental asthma prevalence	**Pollutant:** $PM_{10-2.5}$ **Averaging Time:** 2 years **Mean (SD):** 59 (28) **Percentiles:** 25th: NR 50th(Median): NR 75th: NR IQR: 42 **Range (Min, Max):** Gives range (max.–min.): 80 **Monitoring Stations**: 2 types: municipal monitoring stations over a period of 4 years (1993-1996) schoolyards of participating children over a period of 2 years (1995–1996)	**PM Increment:** Interquartile range corresponded to 1 unit of change. RR Estimate [Lower CI, Upper CI] lag: Association between bronchitis and $PM_{10-2.5}$: 2.20 (1.14, 4.26) $p < 0.05$ Association between persistent cough and $PM_{10-2.5}$: 1.46 (1.12, 1.90) $p < 0.05$ Between and within city associations: Bronchitis: 3.18 (between city) Persistent phlegm (between city): 2.78 When scaled to an increment of 50 $\mu g/m^3$ of $PM_{10-2.5}$ associations (ORs) between respiratory outcome and $PM_{10-2.5}$ were: Wheeze: 1.14 Asthma: 1.34 Bronchitis: 2.56 Hospitalization: 1.58 Persistent cough: 1.57 Persistent phlegm: 3.45

[1]All units expressed in $\mu g/m^3$ unless otherwise specified.

Table E-24. Long-term exposure - respiratory morbidity outcomes - $PM_{2.5}$ (including PM components/sources).

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Annesi-Maesano et al.(2007, 091348)	**Outcome:** EIB, Flexural atopic dermatitis, asthma, rhiniconjuctivitis, allergic rhinitis	**Pollutant:** $PM_{2.5}$	**PM Increment:** High vs. Low
Period of Study: Mar 1999–Oct 2000	**Age Groups:** Children mean 10.4 ± 0.7 yrs	**Averaging Time:** 5-day mean (Mon.-Fri.) over a 13-week to 24-week span	Allergic and respiratory morbidity OR Estimate (Lower CI, Upper CI)
Location: France (Bordeaux, Clermont-Ferrand, Creteil, Marseille, Strasbourg,, & Reims)	**Study Design:** Semi-individual design	Residential Proximity Level	Proximity Level
	N: 5338	**Mean (SD):** Low conc: 8.7	EIB (C) 1.35 (1.10, 1.67)
	Statistical Analyses: Logistic regression	High conc: 20.7	Fl. Atopic dermatitis (C) 2.51 (2.06, 3.06)
	Covariates: Age, sex, family history of allergy, passive smoking	**Range (Min, Max):**	Asthma (P) 1.11 (0.88, 1.39)
	Season: NR	Low conc: (1.6, 12.2)	Atopic asthma (P) 1.43 (1.07, 1.91)
	Dose-response Investigated?	High conc: (12.5, 54.0)	Non-atopic asthma (P) 0.73 (0.49, 1.07)
	No	City Level	Rhiniconjunctivitis (P) 0.94 (0.77, 1.15)
	Statistical Package: SAS	**Mean (SD):** Low conc: 9.6	Atopic dermatitis (P) 1.05 (0.88, 1.27)
		High conc: 23.0	Asthma (L) 1.00 (0.82, 1.22)
		Range (Min, Max):	Allergic Rhinitis (L) 1.09 (0.93, 1.27)
		Low conc: (4.7, 12.7)	Atopic dermatitis (L) 0.94 (0.82, 1.09)
		High conc: (13.0, 54.5)	City Level
			EIB (C) 1.43 (1.15, 1.78)
			Fl. Atopic dermatitis (C) 2.06 (1.69, 2.51)
			Asthma (P) 1.31 (1.04, 1.66)
			Atopic asthma (P) 1.58 (1.17, 2.14)
			Non-atopic asthma (P) 1.00 (0.68, 1.49)
			Rhiniconjunctivitis (P) 0.98 (0.80, 1.20)
			Atopic dermatitis (P) 1.08 (0.90, 1.30)
			Asthma (L) 1.09 (0.89, 1.33)
			Allergic Rhinitis (L) 1.13 (0.97, 1.33)
			Atopic dermatitis (L) 0.95 (0.82, 1.09)
			Notes: C = Current
			P = Past year
			L = Lifetime
			Allergic sensitization OR Estimate (Lower CI, Upper CI)
			Proximity Level
			All allergens 1.19 (1.04, 1.36)
			Indoor allergens 1.29 (1.11, 1.50)
			Outdoor allergens 1.02 (0.85, 1.23)
			Moulds 1.13 (0.78, 1.65)
			City Level
			All allergens 1.32 (1.15, 1.51)
			Indoor allergens 1.51 (1.29, 1.76)
			Outdoor allergens 1.06 (0.88, 1.28)
			Moulds 1.00 (0.69, 1.46)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Bakke et al. (2004, 156246) **Period of Study:** January 1, 1989-June 31, 2002 **Location:** One of Norway's major construction companies	**Outcome:** Spirometric measurements **Age Groups:** All ages, mean = 39 yrs **Study Design:** cohort **N:** 651 male construction workers **Statistical Analyses:** Multiple linear regression models **Covariates:** Age, years for non-smokers and ever smokers **Dose-response Investigated?** No **Statistical Package:** SYSTAT 10.0 and SPSS 11.0	**Pollutant:** Respirable dust **Averaging Time:** 5-8 h **Mean (SD):** Drill and blast workers: 6.3 (2.8) Tunnel concrete workers: 6.1 (3.1) Shotcreting operators: 19 (11) TBM workers: 16 (6.6) Outdoor concrete workers: 1.4 (0.73) Foremen: 0.28 (0.48) Engineers: 0.09 (0.28) **Unit (i.e. $\mu g/m^3$):** mg¤y/m^3 **Monitoring Stations**: 16 tunnel sites visited with sampling equipment **Copollutant (correlation):** Total dust: r = 0.99 ¤ quartz: r = 0.48 NO_2: r = 0.75 CO: r = 0.61 Oil mist: r = 0.83 Oil vapor: r = 0.68 VOC: r = 0.89	**PM Increment:** NR-exposure respirable dust Effect Estimate (Lower CI, Upper CI): Lung function changes predicted by multiple linear regression models using one exposure variable adjusted for age and observation time by non-smokers and ever smokers Non-smokers: ß = -16.0 (-24- -6.8) SE = 4.5 Ever smokers: ß = -9.3 (-17- -1.6) SE = 4.0
Reference: Bakke et al. (2004, 156246) **Period of Study:** January 1, 1989-June 31, 2002 **Location:** One of Norway's major construction companies	**Outcome:** Spirometric measurements **Age Groups:** All ages, mean = 39 yrs **Study Design:** cohort **N:** 651 male construction workers **Statistical Analyses:** Multiple linear regression models **Covariates:** Age, years for non-smokers and ever smokers **Dose-response Investigated?** No **Statistical Package:** SYSTAT 10.0 and SPSS 11.0	**Pollutant:** Total dust **Averaging Time:** 5-8 h **Mean (SD):** Drill and blast workers: 18 (7.8) Tunnel concrete workers: 21 (11) Shotcreting operators: 73 (41) TBM workers: 48 (20) Outdoor concrete workers: 6.5 (3.4) Foremen: 0.78 (1.3) Engineers: 0.27 (0.78) **Unit (i.e. $\mu g/m^3$):** mg¤y/m^3 **Monitoring Stations**: 16 tunnel sites visited with sampling equipment **Copollutant (correlation):** Respirable dust: r = 0.99 ¤ quartz: r = 0.42 NO_2: r = 0.67 CO: r = 0.49 Oil mist: r = 0.81 Oil vapor: r = 0.64 VOC: r = 0.91	**PM Increment:** NR-exposure expirable dust Lung function changes predicted by multiple linear regression models using one exposure variable adjusted for age and observation time by non-smokers and ever smokers Non-smokers: ß = -4.0 (-6.5-1.4) SE = 1.3 Ever smokers: ß = -2.0 (-4.2-0.23) SE = 1.1

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Bennett et al. (2007, 156268) **Period of Study:** 1992-2005 **Location:** Melbourne, Australia	**Outcome:** Respiratory symptoms (from questionnaire) **Age Groups:** All ages, mean = 37.2 yrs **Study Design:** cohort **N:** 1446 **Statistical Analyses:** Logistic regression models **Covariates:** age, gender, use of ß2-agonists, use of inhaled corticosteroids, smoking, year of data collection, and avg daily exposure to $PM_{2.5}$ in the 12 months corresponding to the time frame of symptoms **Dose-response Investigated?** No **Statistical Package:** STATA, version 9	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean (SD):** 6.8 **Range (Min, Max):** (1.8-73.3) **Monitoring Stations:** up to 3	**PM Increment:** NR **Effect Estimate [Lower CI, Upper CI]:** Respiratory symptoms in last 12 months and exposure to ambient $PM_{2.5}$ over the same period Within-person (longitudinal) effects Wheeze: OR = 1.08 (0.79-1.48), p = 0.62 SOB on waking: OR = 1.34 (0.84-2.16), p = 0.22 Cough (AM): OR = 0.74 (0.47-1.15), p = 0.18 Phlegm (AM): OR = 1.55 (0.95-2.53), p = 0.08 Cough w/ phlegm (AM): OR = 1.28 (0.70-2.33), p = 0.42 Asthma attack: OR = 0.91 (0.55-1.49), p = 0.69 Between-person (cross-sectional) effects Wheeze: OR = 1.32 (0.82-2.10), p = 0.25 SOB on waking: OR = 1.29 (0.46-3.60), p = 0.63 Cough (AM): OR = 0.21 (0.07-0.62), p = 0.01 Phlegm (AM): OR = 0.49 (0.16-1.44), p = 0.19 Cough w/ phlegm (AM): OR = 0.28 (0.08-0.97), p = 0.05 Asthma attack: OR = 0.52 (0.17-1.59), p = 0.26

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: (Brauer et al., 2007, 090691) **Period of Study:** 1999-2000 **Location:** The Netherlands	**Outcome:** Allergen sensitivity (any, indoor, outdoor, food, total) IgE > 100 IU/mL Asthma (probable, MD-diagnosed, ever MD-diagnosed) Bronchitis (MD-diagnosed, ever MD-diagnosed) Dry cough at night Itchy rash Itchy rash/eczema Ear/Nose/Throat (ENT) infection Eczema, MD-diagnosed Eczema, ever MD-diagnosed Flu/serious cold, MD-diagnosed Wheeze (ever, early, early frequent, persistent) **Age Groups:** very young children (< 4-years-old) enrolled prenatally **Study Design:** prospective birth cohort study **N:** ~4000 subjects **Statistical Analyses:** multiple logistic regression **Dose-response Investigated?** No	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 12 months **Mean (SD):** SD: NR 16.9 **Percentiles:** 25th: 14.8 50th(Median): 17.3 75th: 18.1 **Range (Min, Max):** (13.5, 25.2) **Monitoring Stations**: 40 **Copollutant (correlation):** Soot: r = 0.97 NO_2: r = 0.93	**PM Increment:** IQR 3.3 $\mu g/m^3$ Notes: Traffic-related pollution ($PM_{2.5}$, soot, NO_2) was associated with respiratory infections, asthma, and allergic sensitization in children during the first four years of life. **Symptom At 4-Years-Old** Wheeze 4-years-old: 1.23 [1.00: 1.51] Early-life: 1.20 [0.99: 1.46] Asthma, MD-diagnosed 4-years-old: 1.15 [0.82: 1.62] Early-life: 1.32 [0.96: 1.83] Dry cough at night 4-years-old: 1.11 [0.94: 1.31] Early-life: 1.14 [0.98: 1.33] Bronchitis, MD-diagnosed 4-years-old: 0.88 [0.66: 1.18] Early-life: 0.86 [0.66: 1.11] ENT infection 4-years-old: 1.13 [0.98: 1.31] Early-life: 1.17 [1.02: 1.34] Flu/serious cold, MD-diagnosed 4-years-old: 1.21 [1.02: 1.42] Early-life: 1.25 [1.07: 1.46] Itchy rash 4-years-old: 0.96 [0.82: 1.11] Early-life: 0.98 [0.85: 1.14] Eczema, MD-diagnosed 4-years-old: 1.00 [0.88: 1.21] Early-life: 0.98 [0.82: 1.17] **Allergen Sensitivity At 4-Yr-Old** Allergen, any: 1.55 [1.13: 2.11] Allergen, indoor: 1.03 [0.69: 1.55] Allergen, outdoor: 0.93 [0.54: 1.58] Allergen, food: 1.75 [1.23: 2.47] Allergen, total IgE > 100 IU/mL: 0.84 [0.59: 1.18] **Cumulative Allergy/Asthma Symptoms At 4-Years-Old** Wheeze, ever: 1.22 [1.06: 1.41] Asthma, ever MD-diagnosed: 1.32 [1.04: 1.69] Asthma, probable: 1.08 [0.90: 1.30] Wheeze, early: 1.16 [1.00: 1.34] Wheeze, persistent: 1.19 [0.96: 1.48] Wheeze, early frequent: 1.19 [0.96: 1.47] Bronchitis, ever MD-diagnosed: 0.96 [0.81: 1.13] Itchy rash/eczema: 0.99 [0.88: 1.13] Eczema, ever MD-diagnosed: 0.98 [0.85: 1.13]

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: (Brauer et al., 2007, 090691) **Period of Study:** 1999-2000 **Location:** The Netherlands	**Outcome:** Allergen sensitivity (any, indoor, outdoor, food, total) IgE > 100 IU/mL Asthma (probable, MD-diagnosed, ever MD-diagnosed) Bronchitis (MD-diagnosed, ever MD-diagnosed) Dry cough at night Itchy rash Itchy rash/eczema Ear/Nose/Throat (ENT) infection Eczema, MD-diagnosed Eczema, ever MD-diagnosed Flu/serious cold, MD-diagnosed Wheeze (ever, early, early frequent, persistent) **Age Groups:** very young children (< 4-years-old) enrolled prenatally **Study Design:** prospective birth cohort study **N:** ~4000 subjects **Statistical Analyses:** multiple logistic regression **Dose-response Investigated?** No	**Pollutant:** Soot (as $PM_{2.5}$ absorbance) **Averaging Time:** 12 months **Mean (SD):** 1.71 **Percentiles:** 25th: 1.33 50th(Median): 1.78 75th: 1.91 **Range (Min, Max):** (0.77, 3.68) **Unit (i.e. $\mu g/m^3$):** 1E-5/m **Monitoring Stations**: 40 **Copollutant (correlation):** NO_2: r = 0.96 $PM_{2.5}$: r = 0.97	**PM Increment:** IQR 0.58 E-5/m Notes: Traffic-related pollution ($PM_{2.5}$, soot, NO_2) was associated with respiratory infections, asthma, and allergic sensitization in children during the first four years of life. **Symptom At 4-Years-Old** Wheeze 4-years-old: 1.18 [0.98: 1.41] Early-life: 1.18 [1.00: 1.40] Asthma, MD-diagnosed 4-years-old: 1.15 [0.85: 1.55] Early-life: 1.30 [0.98: 1.71] Dry cough at night 4-years-old: 1.13 [0.97: 1.30] Early-life: 1.14 [1.00: 1.31] Bronchitis, MD-diagnosed 4-years-old: 0.90 [0.69: 1.16] Early-life: 0.88 [0.69: 1.11] ENT infection 4-years-old: 1.15 [1.01: 1.31] Early-life: 1.16 [1.03: 1.31] Flu/serious cold, MD-diagnosed 4-years-old: 1.18 [1.02: 1.36] Early-life: 1.19 [1.04: 1.37] Itchy rash 4-years-old: 0.94 [0.82: 1.08] Early-life: 0.97 [0.85: 1.10] Eczema, MD-diagnosed 4-years-old: 0.99 [0.84: 1.17] Early-life: 0.97 [0.83: 1.14] **Allergen Sensitivity At 4-Yrs-Old** Allergen, any: 1.45 [1.11: 1.91] Allergen, indoor: 1.02 [0.71: 1.46] Allergen, outdoor: 0.95 [0.59: 1.52] Allergen, food: 1.64 [1.21: 2.23] Allergen, total IgE > 100 IU/mL: 0.80 [0.59: 1.09] **Cumulative Allergy/Asthma Symptoms At 4-Years-Old** Wheeze, ever: 1.18 [1.04: 1.34] Asthma, ever MD-diagnosed: 1.26 [1.02: 1.56] Asthma, probable: 1.06 [0.90: 1.24] Wheeze, early: 1.11 [0.97: 1.26] Wheeze, persistent: 1.18 [0.98: 1.42] Wheeze, early frequent: 1.14 [0.95: 1.37] Bronchitis, ever MD-diagnosed: 0.95 [0.82: 1.10] Itchy rash/eczema: 0.99 [0.89: 1.11] Eczema, ever MD-diagnosed: 0.99 [0.87: 1.12]

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Brauer et al. (2002, 035192) **Period of Study:** NR **Location:** The Netherlands	**Outcome:** Questionnaire derived wheezing, dry nighttime cough, ear, nose and throat infections, skin rash Physician diagnosed asthma, bronchitis, influenza, eczema **Age Groups:** age 2 **Study Design:** Prospective cohort **N:** 4146 children **Statistical Analyses:** Logistic regression **Covariates:** Maternal age, maternal smoking, mattress cover (allergen-free), maternal education, paternal education, gender, gas stove, gas water heater, any other siblings, ethnicity, breastfeeding, mold at home, pets, allergies in mother, allergies in father **Dose-response Investigated?** No	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 4 2-week periods dispersed throughout 1 year, adjusted for temporal trend **Mean (SD):** 16.9 **Percentiles:** 10th: 14.0 25th: 15.0 50th(Median): 17.3 75th: 18.2 90th: 19.1 **Range (Min, Max):** 13.5, 25.2 **Monitoring Stations:** 40 **Copollutant (correlation):** Soot: r = 0.99 NO_2: r = 0.97	**PM Increment:** 3.2 $\mu g/m^3$ OR Estimate [Lower CI, Upper CI]; Unadjusted Wheeze 1.14 (0.99–1.30) Asthma 1.08 (0.84–1.37) Dry cough at night 1.10 (0.95–1.27) Bronchitis 1.00 (0.85–1.18) E, N, T infections 1.14 (0.99–1.33) Flu 1.15 (1.03–1.28) Itchy rash 1.07 (0.95–1.20) Eczema 1.02 (0.90–1.16) Adjusted Wheeze 1.14 (0.98–1.34) Asthma 1.12 (0.84–1.50) Dry cough at night 1.04 (0.88–1.23) Bronchitis 1.04 (0.85–1.26) E, N, T infections 1.20 (1.01–1.42) Flu 1.12 (1.00–1.27) Itchy rash 1.01 (0.88–1.16) Eczema 0.95 (0.83–1.10)
Reference: Brauer et al. (2002, 035192) **Period of Study:** NR **Location:** The Netherlands	**Outcome:** Questionnaire derived wheezing, dry nighttime cough, ear, nose and throat infections, skin rash Physician diagnosed asthma, bronchitis, influenza, eczema **Age Groups:** age 2 **Study Design:** Prospective cohort **N:** 4146 children **Statistical Analyses:** Logistic regression **Covariates:** Maternal age, maternal smoking, mattress cover (allergen-free), maternal education, paternal education, gender, gas stove, gas water heater, any other siblings, ethnicity, breastfeeding, mold at home, pets, allergies in mother, allergies in father **Dose-response Investigated?** No	**Pollutant:** $PM_{2.5}$ "soot" **Averaging Time:** 4 2-week periods dispersed throughout 1 year, adjusted for temporal trend **Mean (SD):** 16.9 10-5/m Percentiles: 10th: 1.16 25th: 1.38 50th(Median): 1.78 75th: 1.92 90th: 2.19 **Range (Min, Max):** 0.77, 3.68 **Unit (i.e. $\mu g/m^3$):** 10-5/m **Monitoring Stations**: 40 **Copollutant (correlation):** $PM_{2.5}$ (r = 0.99) NO_2 (r = 0.96)	**PM Increment:** 0.54 x 10-5/m (equivalent to 0.8 $\mu g/m^3$ elemental carbon) OR Estimate [Lower CI, Upper CI] Unadjusted Wheeze 1.11 [0.99–1.24] Asthma 1.07 [0.87–1.31] Dry cough at night 1.08 [0.95–1.21] Bronchitis 0.98 [0.85–1.12] E, N, T infections 1.12 [0.99–1.27] Flu 1.13 [1.03–1.23] Itchy rash 1.07 [0.97–1.19] Eczema 1.01 [0.91–1.13] Adjusted Wheeze 1.11 [0.97–1.26] Asthma 1.12 [0.88–1.43] Dry cough at night 1.02 [0.88–1.17] Bronchitis 0.99 [0.84–1.17] E, N, T infections 1.15 [1.00–1.33] Flu 1.09 [0.98–1.21] Itchy rash 1.02 [0.91–1.15] Eczema 0.96 [0.85–1.08]

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Brauer et al. (2006, 090757) **Period of Study:** 1997-2001 **Location:** Germany The Netherlands	**Outcome:** Otitis Media (parental report of doctor's diagnosis prior to age 2 years) **Age Groups:** 0-2 years **Study Design:** Prospective Cohort Study **N:** 4,379 children total The Netherlands: 3,714 Germany: 665 **Statistical Analyses:** Logistic regression **Covariates:** Sex, parental atopy, maternal education, siblings, maternal smoking during pregnancy, ETS exposure at home, use of gas for cooking, indoor moulds and dampness, number of siblings, breast-feeding, and presence of pets in the home **Season:** All **Dose-response Investigated?** No	**Pollutant:** $PM_{2.5}$ **PM Component:** Elemental Carbon (EC) **Averaging Time:** 8 weeks (4 2-week periods dispersed throughout 1 year, adjusted for temporal trends) Mean: The Netherlands: $PM_{2.5}$: 16.9 EC: 1.72 Germany: $PM_{2.5}$: 13.4 EC: 1.76 **Range (Min, Max):** The Netherlands: $PM_{2.5}$: 13.5, 25.2 EC: 0.77, 3.68 Germany: $PM_{2.5}$: 12.0, 21.9 EC: 1.40, 4.39 **Monitoring Stations**: 80 (40 for each cohort)	**PM Increment:** $PM_{2.5}$: 3 $\mu g/m^3$ (~ IQR) EC: ~0.5 $\mu g/m^3$ (~ IQR) OR Estimate [Lower CI, Upper CI] The Netherlands: $PM_{2.5}$: At age 1: 1.13 (0.98–1.32) At age 2: 1.13 (1.00–1.27) EC: At age 1: 1.11 (0.98–1.26) At age 2: 1.10 (1.00–1.22) Germany: $PM_{2.5}$: At age 1: 1.19 (0.73–1.92) At age 2: 1.24 (0.84–1.83) EC: At age 1: 1.12 (0.83–1.51) At age 2: 1.10 (0.86–1.41)
Reference: (Burr et al., 2004, 189788) **Period of Study:** 3 weeks in July and Jan 1997 and 2 weeks in Nov 1996 and April 1997 **Location:** North Wales, England	**Outcome:** Self-report of symptoms only for wheeze, cough, phlegm, rhinitis, and itchy eyes. **Age Groups:** all **Study Design:** Repeated measures **N:** 386 persons in congested streets and 425 in the uncongested streets in 1996/1997. Of these, 165 and 283 completed the second phase of the study.	**Pollutant:** $PM_{2.5}$ **Averaging Time:** Mean hourly concentrations **Mean (SD):** Congested Streets 1996-97 21.2 1998-99 16.2 Uncongested Streets 1996-97 6.7 1998-99 4.9 **Monitoring Stations**: 1 in congested street and 1 in uncongested	% change PM_{10} in congested streets: 23.6 % change PM_{10} in uncongested streets: 26.6 Uncongested street sampling site was 20 m from the congested street sampler. The opening of the by-pass produced a reduction in pollution in the congested streets. The health effects of these changed are likely to be greater for nasal and ocular symptoms than for lower respiratory symptoms. Uncertainty about the causality arises from low response rates and conflicting trends in respiratory and nasal symptoms.

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Calderón-Garcidueñas et al. (2006, 091253) **Period of Study:** 1999, 2000 **Location:** Southwest Mexico City & Tlaxcala, Mexico	**Outcome:** Hyperinflation, interstitial markings-measured by chest radiograph, and lung function–FVC, FEV_1, PEF, FEF25-75, measured using spirometry tests **Age Groups:** 5-13 yrs **Study Design:** Cohort1999– **N:** 249 (total), 230 (Southwest Mexico City), 19 (Tlaxcala) **Statistical Analyses:** Bayes test, Spearman rank correlation, multiple regression **Covariates:** Age, sex **Dose-response Investigated?** No **Statistical Package:** SAS 8.2	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 1 yr **Mean (SD):** 21 2000–19 Tlaxacala: 1994-2000: < NAAQS std Mexico City **Monitoring Stations:** Southwest Mexico City–2 Tlxacala–periodic air monitoring data Co**pollutant:** O_3	**PM Increment:** NR % Change: % of children with FEV_1 < 80% expected value: Mexico City (n = 77): 7.8% Tlaxacala (n = 19): 0% % children with hyperinflation: Mexico City: 65.6% Number with: No hyperinflation: 79 Mild: 72 Moderate: 56 Severe: 23 Tlaxacala: 5.3% Number with: No hyperinflation: 18 Mild: 1 Moderate: 0 Severe: 0 % children with interstitial markings: Mexico City: 52.6% Number with: No interstitial markings: 19 Mild: 0 Moderate: 0 Severe: 0 Tlaxacala: 0% Number with: No interstitial markings: 109 Mild: 112 Moderate: 9 Severe: 0

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Cesaroni et al. (2008, 156326) **Period of Study:** Data on PM emissions collected in 2002 cross-sectional survey carried out in 1995 **Location:** Rome, Italy	**Outcome:** Self-reported chronic bronchitis or emphysema, asthma, and rhinitis **Age Groups:** 25-59 yrs **Study Design:** Cross-sectional **N:** 9,488 subjects who had been residents in same place for at least 3 yrs and who had participated in an extension of the ISAAC initiative in Italy in 1994 & 1995 **Statistical Analyses:** GEE with a logit link **Covariates:** sex, age, smoking habits, education level, and variable to account for correlation of data for members of the same family Effect Modifiers: stratified analysis by smoking status (only presented for the traffic score variable) also stratified by education level (data not shown) **Dose-response Investigated:** Wald test to calculate p for trend	**Pollutant:** PM emissions (estimated) Emissions estimated using a model/method based on factors such as vehicle park, driving conditions, emission factors, fuel consumption, fuel properties, road gradients, and climatic conditions **Mean:** 0.12 kg/km^2 SD: 0.081	Odds Ratios for quartiles of PM emissions: Chronic bronchitis or emphysema (n = 397): 1st: 1.00 2nd: 0.96 (0.71, 1.30) 3rd: 0.90 (0.66, 1.23) 4th: 1.05 (0.77, 1.42) p-trend = 0.871 Asthma (n = 472): 1st: 1.00 2nd: 1.10 (0.84, 1.44) 3rd: 0.94 (0.71, 1.24) 4th: 1.06 (0.80, 1.39) p-trend = 0.980 Rhinitis (n = 1227): 1st: 1.00 2nd: 1.41 (1.17, 1.69) 3rd: 1.11 (0.92, 1.34) 4th: 1.37 (1.14, 1.64) p-trend = 0.018
Reference: Dales et al., (2008, 156378) **Period of Study:** **Location:** Windsor, ON	**Outcome:** Pulmonary function and inflammation **Age Groups:** grades 4-6 **Study Design:** cross-sectional prevalence design **Statistical Analyses:** multivariate linear regression **Covariates:** Ethnic background, smokers at home, pets at home, acute respiratory illness, medication use	**Pollutant:** $PM_{2.5}$ **Averaging Time:** Annual **Mean:** 15.4 5th: 14.2 95th: 17.2 **Copollutant:** SO_2 NO_2	**Increment:** Tertiles of exposure FEV_1: < 15.19: 2.16 ± 0.01 15.19-15.96: 2.17 ± 0.02 > 15.96: 2.18 ± 0.01 FVC: < 15.19: 2.51 ± 0.02 15.19-15.96: 2.50 ± 0.02 > 15.96: 2.52 ± 0.02 eNO: < 15.19: 16.08 ± 0.70 15.19-15.96: 15.80 ± 0.76 > 15.96: 16.79 ± 0.72

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Gauderman et al. (2000, 012531) **Period of Study:** 1993-1997 **Location:** Southern California	**Outcome:** FVC, FEV_1, MMEF, FEF_{75} **Age Groups:** fourth, seventh, or tenth graders **Study Design:** cohort **N:** 3035 subjects **Statistical Analyses:** Linear regression **Covariates:** Height, weight, BMI, asthma, smoking, exercise, room temperature, barometric pressure **Dose-response Investigated?** Yes **Statistical Package:** SAS	**Pollutant:** $PM_{2.5}$ **Averaging Time:** annual avg of 2-week avg $PM_{2.5}$ **Mean (SD):** $PM_{2.5}$ 25.9 **Copollutant (correlation):** O_3: r = -0.32 $PM_{10-2.5}$: r = 0.76 NO_2: r = 0.74 Inorg. Acid: r = 0.79	**Increment:** 25.9 $\mu g/m^3$ % Change (Lower CI, Upper CI) $PM_{2.5}$-4th grade FVC -0.47 (-0.94, 0.01) FEV_1 -0.64 (-1.28, 0.01) MMEF -1.03 (-1.95 to -0.09) FEF_{75} -1.31 (-2.57 to -0.03) $PM_{2.5}$-7th grade FVC -0.42 (-0.89, 0.05) FEV_1 -0.32 (-0.88, 0.24) MMEF -0.29 (-1.99, 1.44) FEF_{75} -0.26 (-1.75, 1.25) $PM_{2.5}$-10th grade FVC 0.19 (-0.68, 1.07) FEV_1 -0.25 (-1.41, 0.93) MMEF -0.17 (-3.66, 3.46) FEF_{75} -0.79 (-4.27, 2.82)
Reference: Gauderman et al. (2002, 026013) **Period of Study:** 1996–2000 **Location:** Southern California	**Outcome:** Lung function development: FEV_1, maximal midexpiratory flow (MMEF) **Age Groups:** Fourth grade children (avg age = 9.9 yrs) **Study Design:** Cohort study **N:** 1678 children, 12 communities **Statistical Analyses:** Mixed model linear regression **Covariates:** Height, BMI, doctor-diagnosed asthma and cigarette smoking in previous year, respiratory illness and exercise on day of test, interaction of each of these variables with sex, barometric pressure, temperature at test time, indicator variables for field technician and spirometer **Dose-response Investigated?** Yes **Statistical Package:** SAS (10)	**Pollutant:** $PM_{2.5}$ **Averaging Time:** Annual 24 h averages **Mean (SD):** The avg levels were presented in an online data supplement (Figure E1) **PM Component:** Elemental carbon and organic carbon. **Monitoring Stations**: 12 **Copollutant (correlation):** O_3: (10 AM to 6 PM) r = 0.14 O_3: r = -0.39 NO_2: r = 0.77 Acid vapor: r = 0.87 PM_{10}: r = 0.95 $PM_{10-2.5}$: r = 0.81 EC: r = 0.93 OC: r = 0.89	**PM Increment:** 22.2 $\mu g/m^3$ Association Estimate: Non-statistically significant negative correlation between $PM_{2.5}$ and $FEV_1$and FVC growth rates were observed. MMEF growth rates had a negative correlation with $PM_{2.5}$ (r = -0.43 p = 0.05). $PM_{2.5}$ was not significantly correlated to FEV_1 (r = -0.31 p = 0.25)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: (Gauderman et al., 2004, 056569) **Period of Study:** Air pollution data ascertainment: 1994-2000. Spirometry testing: spring 2001- spring 2003 **Location:** 12 Communities in Southern California	**Outcome:** Lung function FVC, FEV_1, MMEF (Maximal midexpiratory flow rate) **Age Groups:** Children, Avg age 10 years **Study Design:** Prospective Cohort Study **N:** 12 Communities 2,034 children 24,972 child-months **Statistical Analyses:** Linear regression of changes in sex-and-community specific lung growth function and PM Correlation between % with low attained FEV_1 and PM. **Covariates:** Random effect for communities **Dose-response Investigated?** No **Statistical Package:** SAS	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 2-week measurements used to create annual averages Mean: Means are presented in figures only. **Range (Min, Max):** ~6, ~27 **Monitoring Stations:** 12 **Copollutant (correlation):** PM_{10}: r = 0.95 O_3: r = 0.18 NO_2: r = 0.79 EC: r = 0.91 OC: r = 0.91	**PM Increment:** Most to least polluted community Range: 22.8 $\mu g/m^3$ Difference in Lung Growth [Lower CI, Upper CI]; FVC -60.1 (-166.1 to 45.9) FEV_1 -79.7 (-153.0 to ¡6.4) MMEF -168.9 (-345.5 to 7.8) Correlation with % below 80% predicted Lung function (p-value) $PM_{2.5}$: 0.79 (0.002)
Reference: Gauderman et al. (2007, 090121) **Period of Study:** 1993-2004 **Location:** 12 Southern California Communities	**Outcome:** pulmonary function tests FVC, FEV_1, $MMEF/FEF_{25-75}$ **Age Groups:** Children (mean age 10 at recruitment, followed for 8 years) **Study Design:** Cohort Study (Children's Health Study) **N:** 3677 children (1718 in cohort 1 recruited 1993 and 1959 in cohort 2 recruited 1996) 22686 pulmonary function tests. **Statistical Analyses:** Hierarchical mixed effects model with linear splines **Covariates:** Adjustments for height, height squared, BMI, BMI squared, present asthma status, exercise or respiratory illness on day of test, smoking in previous year, field technician, traffic indicator (distance from freeway, distance from major roads), random effects for participant and community. **Dose-response Investigated?** No **Statistical Package:** SAS	**Pollutant:** $PM_{2.5}$ **Monitoring Stations:** 1 in each community	**PM Increment:** 22.8 $\mu g/m^3$ Pollutant effect reported as difference in 8 year lung function growth from least to most polluted community. Negative difference indicate growth deficits associated with exposure. For $PM_{2.5}$ FEV growth deficit is -100
Reference: Gehring et al. (2002, 036250) **Period of Study:** 1995-2002 **Location:** Munich, Germany	**Outcome:** wheezing, cough without infection, dry cough at night, obstructive, spastic or asthmoid bronchitis, respiratory infections, sneezing, runny/stuffed nose **Age Groups:** 0-2 years **Study Design:** Prospective cohort **N:** 1756 infants **Statistical Analyses:** Logistic regression **Covariates:** sex, parental atopy (yes/no), maternal education, siblings (y/n), environmental tobacco smoke at home (y/n), use of gas for cooking	**Pollutant:** $PM_{2.5}$ **Mean (SD):** $PM_{2.5}$ mass: 13.4 $PM_{2.5}$ absorb. 1.77 * 10-5/m Percentiles: $PM_{2.5}$ mass: 10th: 12.2 25th: 12.5 50th(Median): 13.1 75th: 14.0 90th: 14.9 $PM_{2.5}$ absorbance:	**PM Increment:** $PM_{2.5}$ mass: 1.5 $\mu g/m^3$ $PM_{2.5}$ absorb. 0.4 * 10-5/m (IQR) RR Estimate [Lower CI, Upper CI] **Wheeze ($PM_{2.5}$ mass)** Age of 1 yr: All: 0.91 (0.76–1.09) Males: 0.91 (0.72–1.16) Females: 0.94 (0.70–1.27) Age of 2 years: All: 0.96 (0.83–1.12) Males: 0.93 (0.76–1.14) Females: 1.04 (0.83–1.30)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
	(y/n), home dampness (y/n), indoor moulds (y/n), keeping of dogs (y/n) and cats (y/n) study (GINI or LISA)	10th: 1.47 * 10-5	**Cough W/O Infection** ($PM_{2.5}$ mass)
		25th: 1.54 * 10-5	Age of 1 yr: All: 1.34 (1.11–1.61)
	Dose-response Investigated? No	50th(Median): 1.70 * 10-5	Males: 1.43 (1.14–1.80)
		75th: 1.88 * 10-5	Females: 1.19 (0.84–1.70)
		90th: 2.13 * 10-5	**Dry Cough At Night** ($PM_{2.5}$ mass)
		Range (Min, Max):	Age of 1 yr: All: 1.31 (1.07–1.60)
		$PM_{2.5}$ mass: 11.9, 21.9	Males: 1.39 (1.08–1.78)
		$PM_{2.5}$ absorbance:	Females: 1.17 (0.81–1.68)
		1.38 to 4.39 * 10-5	Age of 2 years: All: 1.20 (1.02–1.42)
		$PM_{2.5}$ mass:	Males: 1.25 (1.01–1.55)
		$PM_{2.5}$ absorbance: 1/m	Females: 1.13 (0.86–1.48)
		PM Component: $PM_{2.5}$ mass	**Bronchitis** ($PM_{2.5}$ mass)
		$PM_{2.5}$ absorbance (as a marker of diesel soot)	Age of 1 yr: All: 0.98 (0.80–1.20)
			Males: 0.97 (0.76–1.25)
		Monitoring Stations: 40	Females: 0.98 (0.68–1.41)
		Copollutant (correlation):	Age of 2 years: All: 0.92 (0.78–1.09)
		NO_2: r = 0.99	Males: 0.92 (0.74–1.14)
		$PM_{2.5}$ absorbance and NO_2: r = 0.95	Females: 0.91 (0.68–1.21)
		$PM_{2.5}$ mass and $PM_{2.5}$ absorbance: r = 0.96	**Resp Infections** ($PM_{2.5}$ mass)
			Age of 1 yr: All: 1.04 (0.91–1.19)
			Males: 1.04 (0.87–1.25)
			Females: 1.06 (0.87–1.31)
			Age of 2 years: All: 0.98 (0.80–1.20)
			Males: 0.99 (0.74–1.31): Females: 0.98 (0.73–1.31)
			Sneezing/Runny Nose ($PM_{2.5}$ mass)
			Age of 1 yr: All: 1.01 (0.85–1.20)
			Males: 0.97 (0.77–1.24)
			Females: 1.08 (0.84–1.41)
			Age of 2 years: All: 0.96 (0.82–1.12)
			Males: 0.91 (0.73–1.12)
			Females: 1.04 (0.83–1.31)
			Wheeze ($PM_{2.5}$ absorbance)
			Age of 1 yr: All: 0.93 (0.78–1.12)
			Males: 0.91 (0.71–1.15)
			Females: 1.01 (0.74–1.37)
			Age of 2 years: All: 0.98 (0.84–1.14)
			Males: 0.92 (0.75–1.13)
			Females: 1.07 (0.85–1.36)
			Cough W/O Infection ($PM_{2.5}$ absorbance)
			Age of 1 yr: All: 1.32 (1.10–1.59)
			Males: 1.38 (1.11–1.71)
			Females: 1.25 (0.87–1.78)
			Dry Cough At Night ($PM_{2.5}$ absorbance)
			Age of 1 yr: All: 1.27 (1.04–1.55)
			Males: 1.31 (1.04–1.67)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			Females: 1.16 (0.79–1.71)
			Age of 2 years: All: 1.16 (0.98–1.37)
			Males: 1.17 (0.95–1.44)
			Females: 1.12 (0.84–1.48)
			Bronchitis ($PM_{2.5}$ absorbance)
			Age of 1 yr: All: 0.99 (0.81–1.22)
			Males: 1.00 (0.78–1.27)
			Females: 0.94 (0.63–1.39)
			Age of 2 years: All: 0.94 (0.79–1.12)
			Males: 0.91 (0.72–1.13)
			Females: 0.95 (0.71–1.28)
			Resp Infections ($PM_{2.5}$ absorbance)
			Age of 1 yr: All: 1.03 (0.90–1.18)
			Males: 1.03 (0.86–1.23)
			Females: 1.05 (0.85–1.30)
			Age of 2 years: All: 0.99 (0.80–1.22)
			Males: 0.96 (0.73–1.26)
			Females: 1.04 (0.75–1.43)
			Sneezing/Runny Nose ($PM_{2.5}$ absorbance)
			Age of 1 yr: All: 0.95 (0.79–1.14)
			Males: 0.90 (0.70–1.16)
			Females: 1.06 (0.80–1.39)
			Age of 2 years: All: 0.92 (0.78–1.09)
			Males: 0.83 (0.66–1.05)
			Females: 1.06 (0.83–1.34))
Reference: Goss et al. (2004, 055624) **Period of Study:** 1999-2000 **Location:** USA	**Outcome:** Cystic Fibrosis pulmonary exacerbations, FEV_1 **Age Groups:** Children and adults over the age of 6 **Study Design:** cohort **N:** 11484 patients **Statistical Analyses:** Logistic regression, t-tests, Mann-Whitney tests, Chi-squared tests, polytomous regression, multiple linear regression **Covariates:** Age, sex, lung function, weight, insurance status, pancreatic insufficiency, airway colonization, genotype, median household income by census tract, zipcode. **Dose-response Investigated?** No **Statistical Package:** STATA, SAS	**Pollutant:** $PM_{2.5}$ **Averaging Time:** annual mean of 24 h averages **Mean (SD):** 13.7(4.2) Percentiles: 25th: 11.8 50th(Median): 13.9 75th: 15.9 **Monitoring Stations:** 713	**PM Increment:** 10 $\mu g/m^3$ Odds Ratio Estimate [Lower CI, Upper CI]: Odds of having 2 or more pulmonary exacerbations as compared to 1 or less in 2000 1.21 (1.07 -1.33) Odds of having 1 pulmonary exacerbation as compared to no exacerbations in 2000 0.70 (0.59-0.98) Decrease in FEV_1 155ml(115-194) Decrease in FEV_1 in 2000 after adjusting for FEV_1 in 1999 24ml(7-40)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Hertz-Picciotto et al. (2005, 088678) **Period of Study:** May 1994 to March 1999 **Location:** Teplice and Prachatice, Czech Republic	**Outcome:** Developmental immunotoxicity as assessed by neonatal immunophenotypes **Age Groups:** Not specified: every woman who delivered in the two aforementioned districts were asked to participate **Study Design:** Cohort study **N:** 1397 mother-infant pairs **Statistical Analyses:** Multiple linear regression with lymphocyte percentage as responding variable and pollutant exposure to 14day averaging period before the date of cord blood collection **Covariates:** Season, length of labor, parity, number of previous stillbirths, medication during delivery, working status of mother, maternal education, exposure to active and secondhand smoke, family history of allergy, self-reports of workplace exposure to dust during pregnancy, self-reported maternal chronic or severe respiratory diseases during pregnancy. Ambient temperature and season were controlled for. **Dose-response Investigated?** Yes **Statistical Package:** SUDAAN (version 8)	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h 14 day averages **Mean (SD):** Overall 24 h: 24.8 14 day avg: Teplice: 30.1 Prachatice 19.8 **PM Component:** PAHs **Monitoring Stations:** 2 stations: Teplice and Prachatice	**PM Increment:** 25 $\mu g/m^3$ Adjusted for 3-day temperature and season, $PM_{2.5}$ exposure during the 14 days before birth was associated with reduced T-lymphocyte fractions CD4+, CD3+ and an increase in B-lymphocyte fraction (CD19+). The associations were not quantitatively reported anywhere else in the paper other than in Figure 2 and Table 3
Reference: (Hertz-Picciotto et al., 2007, 135917) **Period of Study:** 1994-98 + follow-ups at up to 4.5 years of age for child **Location:** Czech Republic districts of Teplice and Prachatice	**Outcome:** Lower respiratory illnesses, majority being acute laryngitis, tracheitis, bronchitis. ICD10 codes J04 and J20 **Age Groups:** Birth-4.5 years of age. **Study Design:** longitudinal follow up of a stratified random sample of mother-infant pairs from previous Pregnancy Outcome Study. Low birth weight and preterm births sampled at higher fractions. **N:** 1133 children **Statistical Analyses:** Generalized linear longitudinal models, GEE to adjust for within subject correlations, robust variance estimates were obtained. Model fit judged using Akaike Information criterion. **Covariates:** age of child, breast feeding, environmental tobacco smoke, season, day of week, year of birth, gender, birth weight, pregnancy data including age at delivery, length of gestation, maternal hypertension and diabetes, infant APGAR score, maternal work history, demographics, lifestyle, reproductive and medical histories, temperature, fuel type, other children in household **Dose-response Investigated?** No **Statistical Package:** SUDAAN version 8	**Pollutant:** $PM_{2.5}$ **Averaging Time:** Used 3, 7, 14, 30 and 45 day averages **Mean (SD):** daily mean 22.3 (sd 16 for 3 day avg, 11 for 45 day avg)	**PM Increment:** 25 $\mu g/m^3$ **RR Estimate [Lower CI, Upper CI]** **lag:** Bronchitis, birth-23 months of age Categorical model High 30 day avg $PM_{2.5}$ (greater than 50 $\mu g/m^3$) 2.26(1.81-2.82) Medium 30 day avg $PM_{2.5}$ (between 25 and 50 $\mu g/m^3$) 1.48(1.32-1.65) Continuous model 1.30(1.08-1.58) Bronchitis, 2-4.5 years of age Categorical model High 30 day avg $PM_{2.5}$ (greater than 50 $\mu g/m^3$) 3.66(2.07-6.48) Medium 30 day avg $PM_{2.5}$ (between 25 and 50 $\mu g/m^3$) 1.60(1.41-1.82) Continuous model 1.23(0.94-1.62) Notes: Results of other averaging periods shown in plots.

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: (Hogervorst et al., 2006, 156559) **Period of Study:** NR **Location:** Maastricht, the Netherlands (six schools selected)	**Outcome:** Decreased lung function **Age Groups:** 8-13 years old **Study Design:** Multivariate linear regression (enter method) analysis **N:** 342 children **Statistical Analyses:** ANOVA, Chi square **Covariates:** Independent variables: Age, height, gender, smoking at home by parents, pets, use of ventilation hoods during cooking, presence of unvented geysers, tapestry in the home, indoor/outdoor time, education level of parents. Dependent variables: lung function indices **Dose-response Investigated?** No	**Pollutant:** $PM_{2.5}$ **Averaging Time:** Daily **Mean (SD):** 19.0 (3.2) **Monitoring Stations:** 6 **Copollutant:** PM_{10} TSP	**PM Increment:** 10 $\mu g/m^3$ **RR Estimate [Lower CI, Upper CI]** **lag:** FEV 3.62 [0.50,7.63] FVC 1.80 [-2.10, 5.80] FEF 5.93 [-2.34, 14.89]
Reference: Islam et al. (2007, 090697) **Period of Study:** 1993-2001 **Location:** 12 communities in Southern California, U.S.	**Outcome:** New onset asthma **Age Groups:** 9-10 years **Study Design:** cohort **N:** 2057 **Statistical Analyses:** Cox proportional hazard model **Covariates:** Community, sex, race/ethnicity **Season:** all **Dose-response Investigated?** No **Statistical Package:** SAS V 9.1 **Lags Considered:** 0-2 years	**Pollutant:** $PM_{2.5}$ **Range (Min, Max):** "Low" $PM_{2.5}$ Communities (5.7-8.5) "High" $PM_{2.5}$ Communities (13.7-29.5) **Monitoring Stations:** 12 **Copollutant:** NO_2, acid vapor, PM_{10} and elemental and organic carbon correlated as a "non-ozone package" of pollutants with a similar pattern relative to each other across the 12 communities.	**PM Increment:** NR **IR Estimate [Lower CI, Upper CI]** Low PM FVC □ 90: 19.4 (7.5, 50.5) FVC 90-110: 16.8 (7.0, 40.1) FVC >110: 7.9 (2.9, 21.9) FEV_1 □ 90: 23.7 (9.4, 59.4) FEV_1 90-110: 15.6 (6.5, 37.4) FEV_1 >110: 6.5 (2.3, 18.7) FEF_{25-75} □ 90: 21.1 (8.8, 50.5) FEF_{25-75} 90-110: 11.9 (4.7, 30.0) FEF_{25-75} >110: 6.4 (2.3, 18.2) Overall: 14.2 (7.0, 28.7) High PM FVC □ 90: 14.2 (5.1, 39.6) FVC 90-110: 25.6 (11.1, 59.2) FVC >110: 16.7 (6.5, 42.9) FEV_1 □ 90: 20.8 (8.0, 54.0) FEV_1 90-110: 23.1 (10.0, 53.7) FEV_1 >110: 18.8 (7.5, 47.3) FEF_{25-75} □ 90: 23.8 (10.2, 55.6) FEF_{25-75} 90-110: 23.9 (9.9, 57.7) FEF_{25-75} >110: 15.9 (6.3, 40.5) Overall: 18.4 (9.4, 35.9)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Karr et al. (2007, 090719) **Period of Study:** 1995 to 2000 **Location:** South Coast Air Basin of southern California	**Outcome:** Bronchioloitis **Study Design:** Case-control. Cases included subjects with a record of a single hospitalization with a discharge diagnosis of acute bronchiolitis.10 controls per case were matched on birth date and gestational age. **N:** 18,595 cases 169,472 controls **Statistical Analyses:** Conditional logistic regression to estimate relative risk of hospitalization for bronchiolitis. **Covariates:** Confounders included in the model were: gender, parity, chronic lung disease, cardiac and pulmonary anomalies, SES covariates Age, gestational age, and season of birth were controlled for by matching **Dose-response Investigated?** Yes **Statistical Package:** STATA (Version 8)	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h (lifetime monthly avg from birth & 30 days preceding cases hospitalization) **Mean (SD):** 25 **Percentiles:** 25th: 19 50th(Median): 23 75th: 29 **Range (Min, Max):** 6 to 111 **Monitoring Stations:** 17	**PM Increment:** 10 $\mu g/m^3$ **RR Estimate [Lower CI, Upper CI]** Sub-chronic and chronic exposure: OR = 1.09 (1.04-1.14) Adjusted for adjusted: Sub-chronic OR = 1.10 (1.04, 1.16) Chronic OR = 1.09 (1.03–1.15) Adjusted for CO and NO_2: Sub-chronic OR = 1.14 (1.07, 1.21) Chronic OR = 1.12 (1.06, 1.20) Adjusted for O_3, CO, and NO_2: Chronic OR = 1.15 (1.08, 1.22) Sub-chronic OR = 1.13 (1.06, 1.21)
Reference: (Kim et al., 2004, 087383) **Period of Study:** Mar-June (spring) 2001 Sep-Nov (fall) 2001 **Location:** Alameda County, CA	**Outcome:** Asthma, bronchitis **Age Groups:** Children (grades 3-5) **Study Design:** Cross-sectional **N:** 1109 children, 871 (long term resident children), 462 (long term related females), 403 (long term related males) **Statistical Analyses:** 2-stage multiple logistic regression model **Covariates:** respiratory illness before age of 2, household mold/moisture, pests, maternal history of asthma (for asthma) **Season:** spring and fall **Dose-response Investigated?** Yes **Statistical Package:** SAS 8.2	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 10 weeks **Mean (SD):** Study Avg 12 **Monitoring Stations:** 10 **Copollutant (correlation):** r^2 is approximately 0.9 for all copollutants–Black Carbon (BC), PM_{10}, NO_X, NO_2, NO (NO_X–NO_2)	**PM Increment:** 0.7 (IQR) **OR Estimate [Lower CI, Upper CI]:** Bronchitis All subjects: 1.02 [1.00, 1.08] LTR subjects: 1.03 [1.01, 1.08] LTR females: 1.04 [1.02, 1.05] LTR males: 1.02 [0.99, 1.05] Asthma All subjects: 1.00 [0.96, 1.12] LTR subjects: 1.01 [0.97, 1.06] LTR females: 1.06 [0.99, 1.15] LTR males: 0.99 [0.95, 1.04] Asthma excluding outlier school having a larger proportion of Hispanics All subjects: 1.04 [0.96, 1.12] LTR subjects: 1.03 [0.94, 1.13] LTR females: 1.03 [0.91, 1.17] LTR males: 1.03 [0.94, 1.18]

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Leonardi et al. (2000, 010272) **Period of Study:** 1996 **Location:** 17 cities of Central Europe (Bulgaria, Czech Republic, Hungary , Poland, Romania, Slovakia)	**Outcome:** Immune biomarkers **Age Groups:** 9-11 **Study Design:** Cross-sectional **N:** 366 school children **Statistical Analyses:** Linear regression **Covariates:** Age, gender, parental smoking, laboratory of analysis, recent respiratory illness **Dose-response Investigated?** No **Statistical Package:** STATA	**Pollutant:** $PM_{2.5}$ **Averaging Time:** annual $PM_{2.5}$ **Mean (SD):** $PM_{2.5}$: 46 (10) **Range (Min, Max):** $PM_{2.5}$: (29, 67) 5th, median, & 95th percentile $PM_{2.5}$: 29, 44, 67	**% Change (Lower CI, Upper CI)** **p-value** $PM_{2.5}$ Neutrophils -10 (-45, 46) > .20 Total lymphocytes 49 (11, 101); .008 B lymphocytes 63 (4, 155); .034 Total T lymphocytes 72 (32 123) < .001 CD4+ 80 (34 143) < .001 CD8+ 61 (17, 119); .003 CD4/CD8 16 (-17, 62) > .20 NK 63 (3, 158); .035 Total IgG 24 (2, 52); .034 Total IgM -9 (-32, 22) > .20 Total IgA -1 (-25, 32) > .20 Total IgE -4 (-61, 137) > .20
Reference: McConnell (1999, 007028) **Period of Study:** 1993 **Location:** Southern California	**Outcome:** Bronchitis, chronic cough, phlegm **Age Groups:** Children: 4th, 7th, & 10th graders **Study Design:** Cross-sectional **N:** 3676 people **Statistical Analyses:** Logistic regression **Covariates:** Age, sex, race, grade, health insurance **Dose-response Investigated?** Yes	**Pollutant:** $PM_{2.5}$ **Averaging Time:** Yearly 2 wk avg **Mean (SD):** 15.3 **Range (Min, Max):** 6.7, 31.5 **Copollutant (correlation):** NO_2 r = 0.83 O_3 r = 0.50 Acid r = 0.71	**Child Respiratory symptoms OR Estimate (Lower CI, Upper CI)** **$PM_{2.5}$ Increment:** 15 $\mu g/m^3$ Children w/ asthma Bronchitis: 1.4 (0.9, 2.3) Phlegm: 2.6 (1.2, 5.4) Cough: 1.3 (0.7, 2.4) Children w/ wheeze, no asthma Bronchitis: 0.9 (0.6, 1.4) Phlegm: 1.0 (0.6, 1.8) Cough: 1.1 (0.6, 1.9) Children w/ no wheeze, no asthma Bronchitis: 0.5 (0.3, 1.0) Phlegm: 0.8 (0.4, 1.5) Cough: 0.9 (0.6, 1.3)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: McConnell et al. (2003, 049490) **Period of Study:** 1993-99 **Location:** 12 Southern CA communities	**Outcome:** bronchitic symptoms **Age Groups:** 9-19 **Study Design:** communities selected on basis of historic levels of criteria pollutants and low residential mobility. **N:** 475 children **Statistical Analyses:** 3 stage regression combined to give a logistic mixed effects model **Covariates:** sex, ethnicity, allergies history, asthma history, SES, insurance status, current wheeze, current exposure to ETS, personal smoking status, participation in team sports, in utero tobacco exposure through maternal smoking, family history of asthma, amount of time routinely spent outside by child during 2-6 pm. **Dose-response Investigated?** No **Statistical Package:** SAS Glimmix macro	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 4 year averages **Mean (SD):** 13.8(7.7) **Range (Min, Max):** 5.5-28.5 **Copollutant (correlation):** PM_{10}: r = 0.79 $PM_{10-2.5}$: r = 0.24 Inorganic acid: r = 0.76 Organic Acid: r = 0.58 EC: r = 0.83 OC: r = 0.84 NO_2: r = 0.54 O_3: r = 0.72	**PM Increment:** Between community range 23 $\mu g/m^3$ Between community unit 1 $\mu g/m^3$ Within community 1 $\mu g/m^3$ OR Estimate [Lower CI, Upper CI] Between community per range 1.81(1.14-2.88) Between Community per unit 1.03(1.01-1.05) Within community per unit 1.09(1.01-1.17)
Reference: McConnell et al. (2003, 049490) **Period of Study:** 1993-99 **Location:** 12 Southern CA communities	**Outcome:** bronchitic symptoms **Age Groups:** 9-19 **Study Design:** communities selected on basis of historic levels of criteria pollutants and low residential mobility. **N:** 475 children **Statistical Analyses:** 3 stage regression combined to give a logistic mixed effects model **Covariates:** sex, ethnicity, allergies history, asthma history, SES, insurance status, current wheeze, current exposure to ETS, personal smoking status, participation in team sports, in utero tobacco exposure through maternal smoking, family history of asthma, amount of time routinely spent outside by child during 2-6 pm. **Dose-response Investigated?** No **Statistical Package:** SAS Glimmix macro	**Pollutant:** Elemental Carbon **Averaging Time:** 4 year avg **Mean (SD):** 0.71(0.41) **Range (Min, Max):** 0.1-1.2 **Copollutant (correlation):** $PM_{2.5}$: r = 0.83 PM_{10}: r = 0.71 $PM_{10-2.5}$: r = 0.30 Inorganic acid: r = 0.82 Organic Acid: r = 0.66 Organic Carbon: r = 0.88 NO_2: r = 0.54 O_3: r = 0.68	**PM Increment:** Between community range 1.1 $\mu g/m^3$ Between community unit 1 $\mu g/m^3$ Within community 1 $\mu g/m^3$ OR Estimate [Lower CI, Upper CI] Between community per range 1.64(1.06-2.54) Between Community per unit 1.55(1.05-2.30) Within community per unit 2.63(0.83-8.33)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: McConnell et al. (2003, 049490) **Period of Study:** 1993-99 **Location:** 12 Southern CA communities	**Outcome:** bronchitic symptoms **Age Groups:** 9-19 **Study Design:** communities selected on basis of historic levels of criteria pollutants and low residential mobility. **N:** 475 children **Statistical Analyses:** 3 stage regression combined to give a logistic mixed effects model **Covariates:** sex, ethnicity, allergies history, asthma history, SES, insurance status, current wheeze, current exposure to ETS, personal smoking status, participation in team sports, in utero tobacco exposure through maternal smoking, family history of asthma, amount of time routinely spent outside by child during 2-6 pm. **Dose-response Investigated?** No **Statistical Package:** SAS Glimmix macro	**Pollutant:** Organic Carbon **Averaging Time:** 4 year avg **Mean (SD):** 4.5(2.7) **Range (Min, Max):** 1.4-11.6 **Copollutant (correlation):** $PM_{2.5}$: r = 0.84 PM_{10}: r = .70 $PM_{10-2.5}$: r = 0.27 Inorganic acid: r = 0.83 Organic Acid: r = 0.69 EC: r = 0.88 NO_2: r = 0.67 O_3: r = 0.81	**PM Increment:** Between community range 10.2 $\mu g/m^3$ Between community unit 1 $\mu g/m^3$ Within community 1 $\mu g/m^3$ OR Estimate [Lower CI, Upper CI] Between community per range 1.74(0.89-3.4) Between Community per unit 1.06(0.99-1.13) Within community per unit 1.41(1.12-1.78)
Reference: McConnell, et al. (2006, 180226) **Period of Study:** 1996-1999 **Location:** 12 Southern California communities	**Outcome:** Prevalence of bronchitic symptoms (yearly). **Age Groups:** 10-15-years-old **Study Design:** longitudinal cohort **N:** 475 asthmatic children **Statistical Analyses:** Multilevel logistic mixed effects models. **Covariates:** age, second-hand smoke personal smoking history sex, race. **Dose-response Investigated?** No **Statistical Package:** SAS	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 365 days **Percentiles:** Community by year (n = 48 = 12 communities ◘ 4 years) 25th: NR 50th(Median): 3.4 75th: NR **Range (Min, Max):** Community by year (n = 48 = 12 communities ◘ 4 years): (0.89, 8.7) **Monitoring Stations**: 12 **Copollutant:** O_3 NO_2 EC OC Acid vapor (acetic and formic acid)	**PM Increment:** 3.4 $\mu g/m^3$ OR Estimate [Lower CI, Upper CI] $PM_{2.5}$ Dog (n = 292): 1.56 [1.15: 2.12] No dog (n = 183): 1.03 [0.71: 1.49] $PM_{2.5}$*Dog interaction p-value: 0.06 Cat (n = 202): 1.30 [0.90: 1.88] No Cat (n = 273): 1.36 [0.99: 1.83] $PM_{2.5}$*Cat interaction p-value: 0.87 Neither pet (n = 112): 1.11 [0.71: 1.74] Cat only (n = 71): 0.85 [0.46: 1.57] Dog only (n = 161): 1.53 [1.04: 2.25] Both pets (n = 131): 1.58 [1.02: 2.46] Results suggest that dog ownership, a source of residential exposure to endotoxin, may worsen the severity of respiratory symptoms from exposure to air pollutants in asthmatic children. Although $PM_{2.5}$ was associated at a statistically significant level with ownership of both cats and dogs, it appears that dog ownership (with or without a cat) specifically worsens the association between $PM_{2.5}$ and respiratory symptoms in asthmatic children.

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: (Meng et al., 2007, 093275) **Period of Study:** November 2000 and September 2001 **Location:** Los Angeles and San Diego counties	**Outcome:** Poorly controlled asthma vs. controlled asthma ICD9NR **Age Groups:** 18-64, 65+ **Study Design:** Long-term exposure study comparison of cases and controls **N:** 1,609 adults (represented individuals age 18+ who reported ever having been diagnosed as having asthma by a physician and had their address successfully geocoded) **Statistical Analyses:** Logistic regression to evaluate associations between TD (traffic density) and annual avg air pollution concentrations and poorly controlled asthma. Used sample weights that adjusted for unequal probabilities of selection into the CHIS sample. **Covariates:** Age, sex, race/ethnicity, family federal poverty level, county, insurance status, delay in care for asthma, taking medications, smoking behavior, self-reported health status, employment, physical activity **Dose-response Investigated?** yes	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-hs **Copollutant (correlation):** O_3: r = -0.76 NO_2: r = 0.87 PM_{10}: r = 0.84 CO: r = 0.52 TD: r = 0.13	Results for $PM_{2.5}$ were nonsignificant and not reported quantitatively.
Reference: Millstein, J et al. (2004, 088629) **Period of Study:** Mar-Aug, 1995, and Sep, 1995 to Feb, 1996 Data were taken from the Children's Health Study **Location:** Alpine, Atascadero, Lake Arrowhead, Lake Elsinore, Lancaster, Lompoc, Long Beach, Mira Loma, Riverside, San Dimas, Santa Maria, and Upland, CA	**Outcome:** Wheezing & asthma medication use (ICD 9 NR) **Age Groups:** 4th grade students, mostly 9 yrs at the time of the study **Study Design:** Cohort Study, stratified into 2 seasonal groups/ **N:** 2081 enrolled, 2034 provided parent-completed questionnaire. **Statistical Analyses:** Multilevel, mixed-effects logistic model. **Covariates:** Contagious respiratory disease, ambient airborne pollen and other allergens, temperature, sex, age race, allergies, pet cats, carpet in home, environmental tobacco smoke, heating fuel, heating system, water damage in home, education level of questionnaire signer, physician diagnosed asthma. **Season:** Mar-Aug, 1995, and Sep, 1995 to Feb, 1996 **Statistical Package:** GLIMMIX SAS 8.00 macro for generalized linear mixed models. **Lags Considered:** 14	**Pollutant:** $PM_{2.5}$ **Averaging Time:** Integrated values for successive 2-wk periods **PM Component:** Nitric acid, formic acid, acetic acid **Monitoring Stations**: 1 central location in each community **Copollutant (correlation):** O_3: r = 0.09 NO_2: r = 0.28 PM_{10}: r = 0.33 $PM_{10-2.5}$: r = -0.08	**PM Increment:** IQR: 5.24 $\mu g/m^3$ **Odds Ratio [lower CI, Upper CI]** Annual $PM_{2.5}$: 1.04 [0.83, 1.29] March-August $PM_{2.5}$: 0.91 [0.64, 1.30] Sep-Feb $PM_{2.5}$: 1.18 [0.89, 1.58]

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Morgenstern et al. (2007, 090747) **Period of Study:** Mar 1999-Jul 2000 **Location:** Munich, Germany	**Outcome:** Asthma, wheezing, spastic/obstructive bronchitis. Dry cough at night, respiratory infections, sneezing, runny/stuffed nose without a cold. **Age Groups:** at 1 yr & at 2 yrs **Study Design:** Cohort **N:** 3577 children for the prediction models. Respiratory data available for 3129 children at 1 yr. **Statistical Analyses:** Pearson's correlation coefficient, prediction error expressed as root mean squared error (RMSE), multiple logistic regression with confounding factors, odds ratios **Covariates:** Sex, Parental atopy (genetic predisposition to allergies), environmental tobacco smoke at home, maternal education > or < 12 yrs, sibling, gas stove, home dampness, indoor mold, pets. Since it was not feasible to measure personal exposure to NO_2, $PM_{2.5}$, and $PM_{2.5}$ absorbance, exposure modeling was used. **Statistical Package:** SAS V.8.02	**Pollutant:** $PM_{2.5}$ **Averaging Time:** annual **Mean (SD):** 12.8 Percentiles: 25th: 12.5 50th(Median): 12.9 75th: 13.3 **Range (Min, Max):** 6.8, 15.3 **Monitoring Stations**: 40: traffic, n = 17 and background, n = 23. **Copollutant (correlation):** $PM_{2.5}$ absorbance r = 0.49 NO_2 r = 0.45	**PM Increment:** 1.04 $\mu g/m^3$ **Odds Ratio [Lower CI, Upper CI]** Adjusted OR for $PM_{2.5}$ and: sneezing, runny/stuffed nose during the first year of life was 1.16 [1.01, 1.34] At age 1 yr For wheezing 1.01 [0.87, 1.18] For cough without infection 1.05 [0.88, 1.25] For dry cough at night1.08 [0.86, 1.27] For asthmatic, spastic, or obstructive bronchitis 1.04 [0.90, 1.29] For respiratory infection1.05 [0.88, 1.22] For sneezing, runny or stuffed nose 1.16 [1.01, 1.34] At age 2 yrs For wheezing 1.10 [0.96, 1.25] For cough without infection NA, insufficient sample For dry cough at night 1.03 [0.86, 1.19] For asthmatic, spastic, or obstructive bronchitis 1.05 [0.92, 1.20] For respiratory infection 1.09 [0.94, 1.07] For sneezing, runny or stuffed nose 1.19 [1.04, 1.36]
Reference: Morgenstern et al. (2007, 090747) **Period of Study:** Ma4 1999-Jul 2000 **Location:** Munich, Germany	**Outcome:** Asthma, wheezing, spastic/obstructive bronchitis. Dry cough at night, respiratory infections, sneezing, runny/stuffed nose without a cold. **Age Groups:** at 1 yr & at 2 yrs **Study Design:** Cohort **N:** 3577 children for the prediction models. Respiratory data were available for 3129 children at 1 yr. **Statistical Analyses:** Pearson's correlation coefficient, prediction error expressed as root mean squared error (RMSE), multiple logistic regression with confounding factors, odds ratios **Covariates:** Sex, Parental atopy (genetic predisposition to allergies), environmental tobacco smoke at home, maternal education > or < 12 yrs, sibling, gas stove, home dampness, indoor mold, pets. Since it was not feasible to measure personal exposure to NO_2, $PM_{2.5}$, and $PM_{2.5}$ absorbance, exposure modeling was used. **Statistical Package:** SAS V.8.02	**Pollutant:** $PM_{2.5}$ Absorbance ($PM_{2.5}$ ab) **Averaging Time:** annual **Mean (SD):** 1.7 10 –5 m -1, **Percentiles:** 25th: 1.6 10 –5 m -1 50th(Median): 1.7 10 –5 m -1 75th: 1.8 10 –5 m -1 **Range (Min, Max):** 1.3, 3.2 10 –5 m -1 **Unit (i.e. $\mu g/m^3$):** 10 –5 m -1 **Monitoring Stations**: 40: traffic, n = 17 and background, n = 23.	**PM Increment:** 0.22 x 10 -5 **Odds Ratio [Lower CI, Upper CI]** **no lag** At age 1 yr For wheezing 0.97 [0.77, 1.23] For cough without infection 1.16 [0.87, 1.54] For dry cough at night1.09 [0.78, 1.51] For asthmatic, spastic, or obstructive bronchitis 1.14 [0.88, 1.48] For respiratory infections1.03 [0.86, 1.24] For sneezing, runny or stuffed nose 1.30 [1.03, 1.65] At age 2 yrs For wheezing 1.09 [0.90, 1.33] For cough without infection NR insufficient data For dry cough at night1.18 [0.93, 1.50] For asthmatic, spastic, or obstructive bronchitis 0.85 [0.30, 2.34] For respiratory infections1.05 [0.79, 1.39] For sneezing, runny or stuffed nose 1.27 [1.04, 1.56]

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Oftedal et al. (2008, 093202) **Period of Study:** 2001-2002 **Location:** Oslo, Norway	**Outcome:** Lung function (PEF, FEF25%, FEF50%, FEV_1, FVC) **Age Groups:** 9-10 yrs **Study Design:** Cross-sectional **N:** 1847 children **Statistical Analyses:** Linear regression **Covariates:** Height, age, BMI, birth weight, temperature, maternal smoking, se **Dose-response Investigated?** Yes **Statistical Package:** SPSS, STATA, S-Plus **Lags Considered:** 1-3	**Pollutant:** $PM_{2.5}$ IQR: $PM_{2.5}$ in 1st yr of life: 6.2 $PM_{2.5}$ lifetime: 3.6	**PM Increment:** Per IQR β (Lower CI, Upper CI) $PM_{2.5}$ in 1st yr of life PEF -76.1 (-122.2 to -30.0) FEF25% -75.6 (-127.4 to -23.8) FEF 50% -62.4 (-107.4 to -17.4) FEV_1 -12.7 (-28.8, 3.4) FVC -2.9 (-20.5, 14.7) $PM_{2.5}$ lifetime exposure PEF -57.7 (-94.4 to -21.1) FEF25% -51.8 (-93.1 to -10.6) FEF 50% -48.4 (-84.2 to -12.6) FEV_1 -10.4 (-23.2, 2.4) FVC -3.9 (-17.9, 10.1)
Reference: (Parker et al., 2009, 192359) **Period of Study:** 1999-2005 **Location:** US	**Outcome:** Respiratory allergy/hayfever **Study Design:** Cohort **Covariates:** survey year, age, family structure, usual source of care, health insurance, family income relative to federal poverty level, race/ethnicity **Statistical Analysis:** logistic regression **Statistical Package:** SUDAAN **Age Groups:** 73,198 children aged 3-17 years	**Pollutant:** $PM_{2.5}$ **Averaging Time:** NR **Median:** 13.1 **IQR:** 10.9-15.2 **Copollutant (correlation):** Summer O_3: 0.10 SO_2: 0.21 NO_2: 0.53 $PM_{10-2.5}$: 0.02 PM_{10}: 0.51	**Increment:** 10$\mu g/m^3$ **Odds Ratio (95% CI)** Single Pollutant Model, variable N Adjusted: 1.16 (1.04-1.30) Single Pollutant Model, constant N Adjusted: 1.23 (1.04-1.46) Multi-pollutant Model: 1.29 (1.07-1.56)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Sekine et al. (2004, 090762) **Period of Study:** 1987-1994 **Location:** Nine districts in the Tokyo, Japan metropolitan area: Chuo ward, Ohta ward, Shibuya ward, Itabashi ward, Hachioji City, Tachikawa City, Ome City, Machida City, Tanashi City	**Outcome:** pulmonary function tests **Age Groups:** 30-59 yrs **Study Design:** Cross-sectional and longitudinal **N:** 500 females **Statistical Analyses:** Multiple logistic regression analysis **Covariates:** group (classification by air pollution level), pulmonary function at initial test, age and height at the time of the initial test, number of years investigated, years of residence in the area, type of heater, housing structure, and job status. **Dose-response Investigated?** No **Statistical Package:** SAS	**Pollutant:** Suspended PM (SPM) **Averaging Time:** measured each month for three consecutive days (72 h) **Mean (SD):** 28.1-63.3 **Range (Min, Max):** 3.4-140.6 **Copollutant (correlation):** NO_2	Results of multiple logistic regression analysis for respiratory symptoms Persistent cough Group 3: OR = 1.00 Group 2: OR = 1.02 (0.70-1.48) Group 1: OR = 1.07 (0.67-1.70) Persistent phlegm Group 3: OR = 1.00 Group 2: OR = 1.51 (1.11-2.04) Group 1: OR = 1.78 (1.26-2.53) Asthma Group 3: OR = 1.00 Group 2: OR = 1.99 (0.82-4.83) Group 1: OR = 2.66 (0.98-7.19) Wheeze Group 3: OR = 1.00 Group 2: OR = 1.39 (0.95-2.01) Group 1: OR = 1.34 (0.85-2.11) Breathlessness Group 3: OR = 1.00 Group 2: OR = 0.84 (0.47-1.50) Group 1: OR = 2.70 (1.48-4.91)
Reference: Sharma et al. (2004, 156974) **Period of Study:** 11/2002–4/2003 **Location:** 3 sections in Kanpur City, India 1) Indian Institute of Technology Kanpur (IITK) 2) Vikas Nagar (VN) 3) Juhilal Colony (JC)	**Outcome:** Lung function **Age Groups:** 20–55 years **Study Design:** Cohort **N:** 91 people **Statistical Analyses:** Linear regression **Covariates:** NR **Season:** Fall, Winter, spring **Dose-response Investigated?** No **Statistical Package:** Microsoft Excel **Lags Considered:** 1d lag & 5d mov avg	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-h **Mean (SD):** IITK 158 (22) VN 85 (30) JC 59 (9) **PM Component:** Lead, Nickel, Cadmium, Chromium, Iron, Zinc Benzene soluble fraction (includes polycyclic aromatic hydrocarbons [PAHs]) **Copollutant (correlation):** □PEF = mean daily deviations in PEF $PM_{2.5}$-□PEF: -0.30 $PM_{2.5}$-PM_{10}: 0.67 $PM_{2.5}$-PM_{10} (1-day lag): 0.49 $PM_{2.5}$-$PM_{2.5}$ (1-day lag): 0.88	**PM Increment:** 1 $\mu g/m^3$ □PEF (difference or change in peak expiratory flow) -0.0297 L/min

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: (Singh et al., 2003, 052686) **Period of Study:** NR **Location:** Jaipur, India	**Outcome:** Lung function (peak expiratory flow variability) **Age Groups:** Medical school-aged students **Study Design:** Cross sectional **N:** 313 nonsmoker students **Statistical Analyses:** Amplitude % mean was used as the measure of PEF variability. Mean value of amplitude % mean of peak flow variability were compared for in the two groups by application of Student's t-test. The two groups were: living on campus and commuters. **Dose-response Investigated?** Yes	**Pollutant:** Respirable suspended PM (RSPM) **Averaging Time:** 8 h **Mean (SD):** Roadside: 1,666 Campus: 177 **Monitoring Stations:** 2	It appears that no associations between particulates and the outcome of interest were calculated and reported in this study
Reference: (Solomon et al., 2003, 087441) **Period of Study:** 1966 to 1997 **Location:** United Kingdom: Northern England, North-West Midlands, and Wales.	**Outcome:** Cardio-respiratory morbidity **Age Groups:** 45 yrs and older **Study Design:** Cross-sectional **N:** 1,166 women **Statistical Analyses:** Prevalence ratios were reported for ischemic heart disease, asthma, productive cough, wheeze, and use of an inhaler for asthma or other breathing problems. **Covariates:** Smoked, passive smoking in childhood, tenancy, SES, worked in industry with respiratory hazards, childhood admission to hospital for chest problem, diabetes, BMI were all controlled for as potential confounders. **Dose-response Investigated?** yes **Statistical Package:** STATA	**Pollutant:** Black Smoke **Averaging Time:** Annual	RR Estimate [Lower CI, Upper CI] The findings provide no indication that prolonged residence in places that have had relatively high levels of particulate air pollution causes an important increase in cardio-respiratory morbidity. Prevalence ratios are based on high vs. low pollution with low as referent. Particulate pollution in place of residence: Rr = 1.0 (0.7–1.4) for ischemic heart disease; Rr = 0.7 (0.5-1.0) for asthma Rr = 1.0 (0.7 -1.5) for productive cough
Reference: Suglia et al. (2008, 157027) **Period of Study:** March 1986–October 1992 **Location:** Boston, MA	**Outcome:** lung function **Age Groups:** 18-42 **Study Design:** Prospective cohort **N:** 272 women of childbearing age **Statistical Analyses:** Linear regression **Covariates:** Height, age, weight, race/ethnicity, year, education **Dose-response Investigated?** yes–tertiles of exposure **Statistical Package:** SAS v. 9.0	**Pollutant:** Black Carbon (BC) **Averaging Time:** Annual **Mean (SD):** 0.62 (0.15)	**PM Increment:** 0.22 μg/m^3 (IQR) **Effect Estimate [Lower CI, Upper CI]** FEV_1: -1.08 (-2.5, 0.3) FVC: -0.62 (-1.9, 0.6) $FEF_{25-75\%}$: -2.97 (-5.8 to -0.2) Current Smokers: FEV_1: 0.62 (-2.1, 3.4) FVC: 0.64 (-2.0, 3.3) $FEF_{25-75\%}$: -2.63 (-3.7, 8.9) Former Smokers: FEV_1: -4.40 (-7.8 to -1.0) FVC: -3.11 (-6.1 to -0.2) $FEF_{25-75\%}$: -8.78 (-14.7 to -2.9) Nonsmokers: FEV_1: -0.98 (-2.9, 0.9) FVC: -0.32 (-2.0, 1.4) $FEF_{25-75\%}$: -4.39 (-8.1 to -0.6) Exposure-response relationship presented graphically in Figure 1: the highest BC exposure group had decreases in FEV_1, FVC, and $FEF_{25-75\%}$ compared with the lowest tertile group, although these differences were not statistically significant.

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: (Sunyer et al., 2006, 089771) **Period of Study:** initial selection: 1991-1993, follow-up June 2000-December 2001 **Location:** 21 centers in 10 European countries	**Outcome:** Chronic bronchitis **Age Groups:** Mean age (range) Males- 42.62 (38.12-45.62) Females- 42.57 (39.92-45.69) **Study Design:** Hierarchical models **N:** 6924 **Statistical Analyses:** General additive models (GAM) **Covariates:** Smoking, age at end of education, occupational group, occupational exposures, respiratory infections during childhood, rhinitis, asthma, traffic intensity at household level. **Statistical Package:** STATA-8	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 18 months **Mean (SD):** 3.7-44.9 **Copollutants:** NO_2, SO_2	**PM Increment:** NR **Odds ratio [Lower CI, Upper CI]** Chronic phlegm prevalence at follow up Males: 0.97 [0.70,1.35]
Reference: Zhang et al. (2002, 034814) **Period of Study:** 1993-1996 **Location:** 4 Chinese cities (urban and suburban location in each city): Guangzhou, Wuhan, Lanzhou, Chongqing	**Outcome:** Interview-self reports of symptoms: Wheeze (ever wheezy when having a cold) asthma (diagnosis by doctor) bronchitis (diagnosis by doctor) hospitalization due to respiratory disease (ever) persistent cough (coughed for at least 1 month per year with or apart from colds) persistent phlegm (brought up phlegm or mucus from the chest for at least 1 month per year with or apart from colds). **Age Groups:** Elementary school students age range: 5.4–16.2 **Study Design:** Cross-sectional **N:** 7,557 returned questionnaires 7,392 included in first stage of analysis **Statistical Analyses:** 2-stage regression approach: Calculated odds ratios and 95% CIs of respiratory outcomes and covariates Second stage consisted of variance-weighted linear regressions that examined associations between district-specific adjusted prevalence rates and district-specific ambient levels of each pollutant. **Covariates:** Age, gender, breast-fed, house type, number of rooms, sleeping in own or shared room, sleeping in own or shared bed, home coal use, ventilation device used, homes smokiness during cooking, eye irritation during cooking, parental smoking, mother's education level, mother's occupation, father's occupation, questionnaire respondent, year of questionnaire administration, season of questionnaire administration, parental asthma prevalence.	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 2 years **Mean (SD):** 92 (31) **Percentiles:** 25th: NR 50th(Median): NR 75th: NR IQR: 39 **Range (Min, Max):** Gives range (max.–min.): $PM_{2.5}$-98 **Monitoring Stations**: 2 types: municipal monitoring stations over a period of 4 years (1993-1996) schoolyards of participating children over a period of 2 years (1995–1996)	**PM Increment:** Interquartile range corresponded to 1 unit of change. **RR Estimate [Lower CI, Upper CI]** **lag:** No association between $PM_{2.5}$ and any type of respiratory morbidity. No between or within city association between $PM_{2.5}$ and any type of respiratory morbidity. When scaled to an increment of 50 $\mu g/m^3$ increase in $PM_{2.5}$, association (ORs) between respiratory outcome and $PM_{2.5}$ was: Wheeze: 1.06 Asthma: 1.29 Bronchitis: 1.68 Hospitalization: 1.08 Persistent cough: 1.24 Persistent phlegm: 3.09

[1]All units expressed in $\mu g/m^3$ unless otherwise specified.

Table E-25. Long-term exposure - respiratory morbidity outcomes - other PM size fractions.

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: El-Zein et al. (2007, 093043)	ED Admissions	**Pollutant:** PM from diesel	**PM Increment:** NA
Period of Study: 2000-2004	**Outcome:** Acute respiratory symptoms: asthma, URTI, pneumonia, bronchitis	**Range (Min, Max):** NR	β (p-value):
Location: Beirut, Lebanon	**Age Groups:** < 17	**PM Component:** NR	**2 years pre-ban vs. 2 years post-ban**
	Study Design: Ecological (natural experiment comparing admissions before and after ban on diesel fuel)	**Monitoring Stations:** 1	Oct to Feb
	N: 5 hospitals, 7573 admissions Oct-Feb, 4303 admissions Oct-Dec	**Notes:** Did not look at specific exposure data	All Resp: 0.128 (0.32)
	Statistical Analyses: t-test, Poisson regression	looked at outcome with respect to a timeline that plotted admissions before and after a ban on diesel fuel.	Asthma: -0.176 (0.16)
	Covariates: Month of Year, temperature, humidity, orthogonalized rainfall	**Copollutant:** NR	Bronchitis: 0.505 (0.02)
	Season: Oct-Dec (excluding flu season) and Oct-Feb		Pneumonia: 0.287 (0.17)
	Dose-response Investigated? No		URTI: -0.265 (0.41)
	Statistical Package: NR		Oct to Dec
	Lags Considered: 1-2 years before the ban compared to 1-2 years after the ban		All Resp: -0.022 (0.87)
			Asthma: -0.21 (0.07)
			Bronchitis: 0.2 (0.35)
			Pneumonia: -0.065 (0.78)
			URTI: -0.628 (0.05)
			2 years pre-ban vs. 1 year post-ban
			Oct–Feb
			All Resp: -0.093 (0.45)
			Asthma: -0.208 (0.05)
			Bronchitis: 0.286 (0.32)
			Pneumonia: -0.07 (0.76)
			URTI: -0.715 (0.11)
			Oct to Dec
			All Resp: -0.147 (0.02)
			Asthma: -0.147 (0.00)
			Bronchitis: -0.011 (0.96)
			Pneumonia: -0.214 (0.15)
			URTI: -0.885 (0.06)
			1 years pre-ban vs. 1 year post-ban
			Oct–Feb
			All Resp: -0.165 (0.04)
			Asthma: -0.212 (0.09)
			Bronchitis: 0.059 (0.85)
			Pneumonia: -0.034 (0.84)
			URTI: -1.023 (0.00)
			Oct to Dec
			All Resp: -0.17 (0.00)
			Asthma: -0.131 (0.00)
			Bronchitis: -0.145 (0.001)
			Pneumonia: -0.168 (0.12)
			URTI: -1.036 (0.00)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Kasamatsu et al. (2006, 156627) **Period of Study:** 2001-2002 **Location:** Shenyang, China	**Outcome:** FVC, FEV_1, PEF, FEF_{75} **Age Groups:** School Children aged 8-10 **Study Design:** Children in three schools in three types of areas (commercial city area, residential city area, residential suburban area) invited to participate **N:** 322 children participated, 244 have complete data. **Statistical Analyses:** Genralized estimating equations **Covariates:** age, height, **Dose-response Investigated?** no **Statistical Package:** SAS **Lags**: Considered: previous quarter.	**Pollutant:** PM_7 **Averaging Time:** avg of 4 separate 2-7 consecutive day measurements within each designated measurement month of the quarter **Mean (SD):** School A 7/2001 86.4(14.2) 10/2001 114.1(35.1) 1/2002 118.2(28.2) 4/2002 182.7(102.1) School B 7/2001 90.1(8.3) 10/2001 161.5(45.7) 1/2002 118.8(28.2) 4/2002 152.0(31.3) School C 7/2001 78.1(16.9) 10/2001 131.2(29.6) 1/2002 142.2(37.6) 4/2002 173.6(121.5) **PM Component:** mainly pollutants associated with coal heating **Monitoring Stations**: 1 at each location	**PM Increment:** 63.0 $\mu g/m^3$ **Mean change of pulmonary function value [Lower CI, Upper CI] at lag 0** Boys FVC -0.095(-0.170,-0.019) FEV_1 -0.088(-0.158,-0.019) PEF -0.170(-0.365,0.032) FEF_{75} -0.063(-0.183,0.050) Girls FVC -0.082(-0.145,-0.019) FEV_1 -0.069(-0.126,-0.006) PEF 0.095(-0.095,0.290) FEF_{75} -0.032(-0.151,0.082) Mean change of pulmonary function value [Lower CI, Upper CI] at lag 1(previous quarter) Boys FVC -0.145(-0.189,-0.095) FEV_1 -0.095(-0.139,-0.057) PEF -0.082(-0.208,0.050) FEF_{75} 0.013(-0.063,0.088) Girls FVC -0.126(-0.170,-0.088) FEV_1 -0.101(-0.139,-0.063) PEF -0.101(-0.227,0.025) FEF_{75} -0.057(-0.132,0.019)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Kasamatsu et al.(2006, 156627) **Period of Study:** 2001-2002 **Location:** Shenyang, China	**Outcome:** FVC, FEV_1, PEF, FEF_{75} **Age Groups:** School Children aged 8-10 **Study Design:** Children in three schools in three types of areas (commercial city area, residential city area, residential suburban area) invited to participate **N:** 322 children participated, 244 have complete data. **Statistical Analyses:** Genralized estimating equations **Covariates:** age, height, **Dose-response Investigated?** no **Statistical Package:** SAS **Lags:** Considered: previous quarter.	**Pollutant:** $PM_{2.1}$ **Averaging Time:** avg of 4 separate 2-7 consecutive day measurements within each designated measurement month of the quarter **Mean (SD):** School A 7/2001 47.6(6.4) 10/2001 54.2(20.5) 1/2002 68.9(15.8) 4/2002 115.8(76.7) School B 7/2001 45.6(6.5) 10/2001 74.4(27.1) 1/2002 63.3(17.9) 4/2002 96.3(27.6) School C 7/2001 42.5(9.5) 10/2001 59.7(13.1) 1/2002 76.4(22.1) 4/2002 123.0(100.9) **PM Component:** mainly pollutants associated with coal heating **Monitoring Stations:** 1 at each location	**PM Increment:** 42.1 $\mu g/m^3$ Mean change of pulmonary function value [Lower CI, Upper CI] at lag 0 Boys FVC -0.126(-0.181,-0.076) FEV_1 -0.122(-0.173,-0.076) PEF -0.164(-0.303,-0.025) FEF_{75} -0.046(-0.131,0.038) Girls FVC -0.110(-0.156,-0.067) FEV_1 -0.101(-0147,-0.059) PEF 0.008(-0.131,0.147) FEF_{75} -0.055(-0.139,0.030) Mean change of pulmonary function value [Lower CI, Upper CI] at lag 1(previous quarter) Boys FVC -0.099(-0.145,-0.053) FEV_1 -0.059(-0.106,-0.020) PEF -0.040(-0.158,0.086) FEF_{75} 0.026(-0.046,0.092) Girls FVC -0.086(-0.125,-0.046) FEV_1 -0.066(-0.106,-0.026) PEF -0.079(-0.198,0.040) FEF_{75} -0.033(-0.106,0.040)

[1]All units expressed in $\mu g/m^3$ unless otherwise specified.

E.6. Long-Term Exposure and Cancer

Table E-26. Long-term exposure - cancer outcomes - PM_{10}.

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: (Abbey et al., 1999, 047559) **Period of Study:** 1977-1992 **Location:** California	**Outcome (ICD9):** Lung Cancer Mortality (162) **Age Groups:** 27-95 at baseline **Study Design:** Cohort (AHSMOG) **N:** 6,338 nonsmoking CA Seventh-Day Adventists **Statistical Analyses:** time-dependent, gender-specific, Cox proportional hazards regression models **Covariates:** age, smoking, education, occupation, BMI	**Pollutant:** PM_{10} **Averaging Time:** monthly estimates from 1966-1992 **Mean (SD):** 51.24 (16.63) **Percentiles:** IQR: 24.08 **Range (Min, Max):** 0, 83.9 **Correlations:** SO_4: r = 0.68) SO_2: r = 0.31 O_3: r = 0.77 NO_2: r = 0.56 **Lag:** 3 years	**PM Increment:** 24.08 (IQR) RR, males: 3.36 [1.57, 7.19] RR, females: 1.33 [0.60, 2.96] PM_{10} **above 100μg/m^3 (days per year)** IQR: 43 days/year Males: 2.38 (1.42, 3.97) Females: 1.08 (0.55, 2.13)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Beeson et al. (1998, 048890) **Period of Study:** 1977-1992 **Location:** California	**Outcome (ICD9:** Lung Cancer Mortality (ICDO-1: 162, ICDO-2: C34.0-C34.9) **Age Groups:** 27-95 at baseline **Study Design:** Cohort (AHSMOG) **N:** 6,338 nonsmoking CA Seventh-Day Adventists (non-Hispanic white) **Statistical Analyses:** time-dependent, gender-specific, Cox proportional hazards regression models **Covariates:** Smoking, Education, Age, Alcohol **Statistical Package:** SAS **Lags Considered:** 3 yr	**Pollutant:** PM_{10} **Averaging Time:** averaged monthly estimates from 1966-1992 **Mean (SD):** 51 (16.52) **Percentiles:** IQR: 24 **Range (Min, Max):** 0, 84	**PM Increment:** 24 (IQR) RR, males: 5.21 [1.94, 13.99] RR, females: Positive, but not statistically significant
Reference: Binkova et al. (2007, 156273) **Period of Study:** February 6-20, 2001 **Location:** Prague, Czech Republic	**Outcome:** Total DNA adducts (bulky aromatic PAH-DNA adducts and ... **Age Groups:** 22-50 yrs **Study Design:** Case Control **N:** 53 occupationally exposed policemen and 52 control policemen **Statistical Analyses:** Multivariate logistic regression, Mann-Whitney u-test **Covariates:** Smoking. Vitamin C, polymorphisms of XPD repair gene in exon 23 and 6 and GSTM 1 and XRCC1 genes **Season:** Winter	**Pollutant:** PM_{10} **Range (Min, Max):** 32-55 **Monitoring Stations:** 2 (and personal monitors)	No relationship between short term exposure to C-PAHs evaluated by personal monitors and DNA adduct level. Genetic damage was observed in city policemen working in winter outdoors in the Prague downtown area they had slightly elevated aromatic DNA adduct levels, which was statistically significant for a distinct DNA adduct spot that could originate from ambient exposure to B[□]P. **Total PAH-DNA adducts**: p = 0.065 Exposed: 0.92 ± 0.28 adducts/10^8 nucleotids Control: 0.82 ± 0.23 adducts/10^8 nucleotids **B[□]P-like adducts:** Exposed: 0.122 ± 0.36 adducts/10^8 nucleotids Control: 0.099 ± 0.035 adducts/10^8 nucleotids Multiple regression "like" B[□]P-DNA adduct for air pollution exposure group: B = 0.016, p = 0.01
Reference: (Liu et al., 2009, 190292) **Period of Study:** 1995-2005 **Location:** Taiwan	**Outcome:** Bladder Cancer Mortality (ICD-9 188) **Age Groups:** 50-69 **Study Design:** case-crossover **Statistical Analysis:** Multiple Logistic Regression **Statistical Package:** NR **Covariates:** none **Dose-response Investigated?** No	**Pollutant:** PM_{10} **Averaging Time:** annual mean of 24h avg **Tertiles (median):** T1: □52.80 T2: 53.04-71.72 T3: 72.24-90.29 **Copollutant:** O_3, CO, NO_2, SO_2 **Copollutant (correlation):** NR **Monitoring Sattions:** 64	**Increment:** **Odds Ratio (Min CI, Max CI)** **Lag** T1 vs. T1: 1.00 (ref) T2 vs. T1: 1.08 (0.83 · 1.41) T3 vs. T1: 1.39 (1.06 · 1.83) P for trend = .020

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: (Pope CA et al., 2002, 024689) **Period of Study:** 1982-1998 **Location:** 50 US states, District of Columbia, and Puerto Rico	**Outcome (ICD9):** Lung cancer mortality (162) **Age Groups:** Ages > 30 years **Study Design:** Longitudinal cohort (Cancer Prevention Study II) **N:** 1.2 million people **Statistical Analyses:** Cox proportional hazard, generalized additive **Covariates:** Age, sex, race, education, smoking status, marital status, occupational exposure, diet, body-mass index, alcohol consumption	**Pollutant:** PM_{10} **Mean (SD):** 1982-1998: 28.8(5.9)	Effect estimates: Effect estimates were recorded in Figure 5 and not presented quantitatively anywhere else
Reference: Sram et al, (2007, 188457) **Period of Study:** January and March of 2004 **Location:** Prague, Czech Republic	**Outcome:** Chromosomal aberrations **Study Design:** Panel **Covariates:** urinary cotinine, plasma levels of vitamins A, E and C, folate, total cholesterol, HDL and LDL cholesterols, and triglycerides **Statistical Analysis:** bivariate correlations, ANOVA, Mann-Whitney, Kruskal-Wallis and Spearman rank correlation **Statistical Package:** STATISTICA **Age Groups:** 61 city policemen, aged 34 ± 8 years, spending 8+ hours outdoors	**Pollutant:** PM_{10} **Averaging Time:** NR **Mean (SD) Unit:** January: 55.6 $\mu g/m^3$ March: 36.4 $\mu g/m^3$ **Copollutant:** $PM_{2.5}$	Results not given by PM increment.
Reference: Sram et al, (2007, 188457) **Period of Study:** January and March of 2004 **Location:** Prague, Czech Republic	**Outcome:** Chromosomal aberrations **Study Design:** Panel **Covariates:** urinary cotinine, plasma levels of vitamins A, E and C, folate, total cholesterol, HDL and LDL cholesterols, and triglycerides **Statistical Analysis:** bivariate correlations, ANOVA, Mann-Whitney, Kruskal-Wallis and Spearman rank correlation **Statistical Package:** STATISTICA **Age Groups:** 61 city policemen, aged 34 ± 8 years, spending 8+ hours outdoors	**Pollutant:** $PM_{2.5}$ **Averaging Time:** NR **Mean (SD) Unit:** January: 44.4 $\mu g/m^3$ March: 24.8 $\mu g/m^3$ **Copollutant:** PM_{10}	Results not given by PM increment.
Reference: (Tarantini et al., 2009, 192010 **Period of Study:** NR **Location:** Brescia, Italy	**Outcome:** DNA methylation content estimated by Alu, LINE-1 and *iNOS* analysis **Study Design:** Panel **Covariates:** age, BMI, smoking, number of cigarettes/day **Statistical Analysis:** mixed effects models **Statistical Package:** NR **Age Groups:** 63 male workers between 27 and 55 years, mean age 44.	**Pollutant:** PM_{10} **Averaging Time:** NR **Mean (SD) Unit:** NR **Individual Exposure Range:** 73.4-1220 $\mu g/m^3$ **Copollutant (correlation):** NR	**Difference in DNA Methylation before and after work exposure, mean (SE)** Alu (%5mC): 0.00 (0.08), p = 0.99 LINE-1 (%5mC): 0.02 (0.11), p = 0.89 iNOS (%5mC): -0.61 (0.26), p = 0.02

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: (Vineis et al., 2006, 192089)	**Outcome:** lung cancer	**Pollutant:** PM_{10}	**Increment:** 10 $\mu g/m^3$
Period of Study: 1990-1999	**Study Design:** nested case-control	**Averaging Time:** NR	**Odds Ratios (Min CI, Max CI) for increase in lung cancer per increment increase in PM_{10}**
Location: 10 European countries	**Covariates:** age, sex, country, smoking status, time since recruitment, education, BMI, physical activity, intake of fruit, vegetables, meat, alcohol and energy	**Mean by Country ($\mu g/m^3$):**	0.91 (0.70-1.18)
		France	
		Ile-de-France	
		1990-1994: 22.3	
		1995-1999: 19.9	
		Northeast France	
		1990-1994: 30.2	
		1995-1999: 29.5	
		Italy	
		Turin	
		1990-1994: 73.4	
		1995-1999: 61.1	
		Florence	
		1990-1994: 40.4	
		1995-1999: 33.3	
		United Kingdom	
		Oxford	
		1990-1994: 29.0	
		1995-1999: 25.5	
		Cambridge	
		1990-1994: NR	
		1995-1999: 25.4	
		The Netherlands	
		Utrecht	
		1990-1994: 42.8	
		1995-1999: 40.0	
		Bilthoven	
		1990-1994: 39.0	
		1995-1999: 37.2	
		Germany	
		Heidelberg	
		1990-1994: NR	
		1995-1999: 27.0	
		Potsdam	
		1990-1994: 32.0	
		1995-1999: 28.9	
		Range (Min, Max): NR	
		Copollutant: NO_2, O_3, SO_2	

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: (Wei et al., 2009, 192361)	**Outcome:** Urinary 8-OHdG increase	**Pollutant:** $PM_{2.5}$	**Increment:** 166.29 $\mu g/m^3$
Period of Study: 11/17/2006 - 1/13/2007	**Study Design:** Panel	**Averaging Time:** 24h	**8-OHdG Concentrations, pre and post-work shift, subjects averaged**
Location: Peking, China	**Covariates:** NR	**Median:** 154.87 $\mu g/m^3$	Pre-work: 1.83
	Statistical Analysis: analysis of variance model with autoregressive terms	**IQR:** 166.29	Post-work: 6.92
	Statistical Package: SAS	**Copollutant (correlation):** NA	**Concentration Changes (95%CI) of 8-OHdG per IQR Increase**
	Age Groups: Two nonsmoking security guards, ages 18 and 20		Pre-work: 0.256 (0.040, 0.472), p = 0.021
			Post-work: 2.370 (0.907, 3.833), p = 0.002

[1]All units expressed in $\mu g/m^3$ unless otherwise specified.

Table E-27. Long-term exposure - cancer outcomes - $PM_{2.5}$ (including PM components/sources).

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Baccarelli et al, (2009, 188183)	**Outcome:** DNA methylation of LINE-1 and Alu	**Pollutant:** $PM_{2.5}$	**Increment:** SD for each lag
Period of Study: 1/1999-6/2007	**Study Design:** Panel	**Averaging Time:** NR	**Correlation Coefficient (95% CI)**
Location: Boston, Massachusetts	**Covariates:** age, BMI, smoking status, pack-years, statin use, fasting blood glucose, diabetes mellitus, percent lymphocytes and neutrophils in differential blood count, day of the week, season, temperature	**Mean (SD) Unit:**	**Lag for LINE-1 Methylation**
		4h: 12.2 (7.7) $\mu g/m^3$	4h: -0.07 (-0.13, -0.01), p = 0.03
		1d: 10.9 (6.3) $\mu g/m^3$	1d: -0.09 (-0.16, -0.02), p = 0.008
		2d: 10.6 (5.2) $\mu g/m^3$	2d: -0.10 (-0.17, -0.03), p = 0.003
		3d: 10.4 (4.8) $\mu g/m^3$	3d: -0.10 (-0.17, --0.04), p = 0.003
		4d: 10.3 (4.3) $\mu g/m^3$	4d: -0.10 (-0.16, -0.03), p = 0.004
		5d: 10.2 (3.9) $\mu g/m^3$	5d: -0.10 (-0.16, -0.03), p = 0.004
		6d: 10.3 (3.5) $\mu g/m^3$	6d: -0.11 (-0.17, -0.04), p = 0.001
		7d: 10.3 (3.3) $\mu g/m^3$	7d: -0.13 (-0.19, -0.06), p < 0.001
	Statistical Analysis: mixed effects models	**Copollutants:** Black carbon, Sulfate	**Correlation Coefficient (95% CI)**
	Statistical Package: SAS		**Lag for Alu Methylation**
	Age Groups: 719 elderly individuals, mean age 73.3, range 55-100 years		4h: 0.03 (-0.03, 0.09), p = 0.28
			1d: -0.01 (-0.07, 0.05), p = 0.74
			2d: -0.01 (-0.07, 0.05), p = 0.82
			3d: -0.01 (-0.07, 0.05), p = 0.78
			4d: -0.01 (-0.07, 0.05), p = 0.75
			5d: -0.01 (-0.07, 0.05), p = 0.84
			6d: -0.01 (-0.07, 0.05), p = 0.74
			7d: -0.01 (-0.07, 0.05), p = 0.71
			Correlation Coefficient (95% CI)
			LINE-1 Methylation and moving averages of pollutant levels
			4h: -0.04 (-0.11, 0.03), p = 0.24
			7d: -0.11 (-0.18, -0.05), p = 0.001

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Binkova et al. (2007, 156273) **Period of Study:** February 6-20, 2001 **Location:** Prague, Czech Republic	**Outcome:** Bulky aromatic PAH-DNA adducts **Age Groups:** 22-50 yrs **Study Design:** Case Control **N:** 53 exposed policemen and 52 control policemen **Statistical Analyses:** Multivariate logistic regression, Mann-Whitney, Rank-Sum U-test **Covariates:** Smoking. Vitamin C, polymorphisms of XPD repair gene in exon 23 and 6 and GSTM 1 gene **Season:** Winter	**Pollutant:** $PM_{2.5}$ **Range (Min, Max):** 27-38 **c-PAHs: range** = 18-22 ng/m^3 **B[a]P: range** = 2.5-3.1 ng/m^3 **Monitoring Stations:** 2	Genetic damage was observed in city policemen working in winter outdoors in the Prague downtown area they had slightly elevated aromatic DNA adduct levels, which was more pronounced for a distinct DNA adduct spot that could originate from ambient exposure to B[□]P. **Total DNA-adduct level** Exposed: 0.92 ± 0.28 adducts/108 nucleotides Control: 0.82 ± 0.23 adducts/108 nucleotides p = 0.065 **"Like" B[□]P-derived DNA adducts** Exposed: 0.122 ± 0.036 Control: 0.101 ± 0.035 p < 0.01 **Multiple Regression (exposed vs. control)** B = 0.016, p = 0.011
Reference: Brunekreef et al, (2009, 191947) **Period of Study:** 1987-1996 **Location:** The Netherlands	**Outcome:** Air pollution related lung cancer deaths (ICD-9 162) **Study Design:** Case-cohort **Covariates** Individual: sex, age, Quetelet index, smoking status, passive smoking status, educational level, occupation, occupational exposure, marital status, alcohol use, intake of vegetables, fruits, energy, saturatured and monounsaturated fatty acids, trans fatty acids, total fiver, folic acid and fish Area-level: Percent of population with income below the 40th percentile and above the 80th percentile **Statistical Analysis:** Cox proportional hazards **Statistical Package:** Stata, SPSS, R **Age Groups:** 120,000 adults aged 55-69 years at enrollment	**Pollutant:** $PM_{2.5}$, estimated from PM_{10} levelsf **Averaging Time:** 24hr **50th Percentile:** 28 μg/m^3 **Range (Min, Max):** 23-37 **Copollutant (correlation):** NO_2: 0.75 Black Smoke: 0.84 NO: 0.69 SO_2: 0.43	**Increment:** 10μg/m^3 **Relative Risk (95% CI) for associations between $PM_{2.5}$ and lung cancer incidence** Case Cohort Unadjusted: 0.93 (0.71-1.22) Adjusted: 0.67 (0.41-1.10) Unadjusted Complete: 0.87 (0.60-1.25) Full Cohort Unadjusted: 0.96 (0.79-1.18) Adjusted: 0.81 (0.63-1.04) Unadjusted Complete: 0.92 (0.74-1.15)
Reference: Liu et al. (2008, 156708) **Period of Study:** 1995-2005 **Location:** Taiwan	**Outcome:** Brain cancer deaths ICD9: 191 **Age Groups:** 29 yrs of age or younger **Study Design:** matched case-control by sex, years of birth and death **N:** 340 matched pairs **Statistical Analyses:** Conditional logistic regression **Covariates:** age, gender, urbanization level of residence, nonpetrochemical air pollution exposure level	No direct measures of pollutants used an index to assign petrochemical air pollution exposure (each municipality was assigned an exposure by dividing the number of workers per municipality employed in the petrochemical industry by the municipalities total population). Study participants divided into tertiles based on this index.	People who lived in the group of municipalities with the highest levels of air pollutants arising from petrochemical sources were at a statistically significant increased risk for brain cancer development compared to the group living in municipalities with the lowest petrochemical air pollution exposure index. **Effect Measure:** OR (95%CI) Tertile 1: 1. ?0 Tertile 2: 1.54 (0.98-2.42) Tertile 3: 1.65 (1.00-2.73) P for trend < 0.01

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Nafstad et al. (2004, 087949) **Period of Study:** 1972/73-1998 **Location:** Oslo, Norway	**Outcome:** Lung cancer ICD7 162.1-162.9 **Age Groups:** 40-49 yr old men **Study Design:** Cohort **N:** 16,209 males **Statistical Analyses:** Cox regression models (proportional hazards) **Covariates:** age at inclusion, smoking habits, education **Season:** all year	PM values had small variations in exposure level, and strong correlations with another pollutant of interest (SO_2) and were not considered in analyses. **Copollutants:** SO_2 NO_X	No effect estimates for PM
Reference: (Pope CA and Burnett, 2007, 090928) **Period of Study:** 1982-1998 **Location:** 50 US states, District of Columbia, and Puerto Rico	**Outcome:** Lung cancer mortality (162) **Age Groups:** > 30 years **Study Design:** Longitudinal cohort (Cancer Prevention II Study) **N:** 415,000 CPS II patients with information involving PM_{10} **Statistical Analyses:** Cox proportional hazard, incorporating a spatial random-effects component **Covariates:** Age, sex, race, education, ETS, smoking status, marital status, occupational exposure, diet, body-mass index, alcohol consumption	**Pollutant:** $PM_{2.5}$ **Mean (SD):** 1979–1983: 21.1(4.6) 1999-2000: 14.0(3.0) **Average:** 17.7(3.7) **Averaging time:** 1982-1998	**PM Increment:** 10 $\mu g/m^3$ **RR Estimate [Lower CI, Upper CI]** **lag:** **Lung Cancer:** 1979-1983: 1.08[1.01, 1.16] 1999-2000: 1.13[1.04, 1.22] Avg: 1.14[1.04, 1.23] RR results were also presented in Figures 2-5. Authors found that $PM_{2.5}$ had the strongest association with increased risk of all-cause, cardiopulmonary, and lung cancer mortality.
Reference: Sram et al, (2007, 188457) **Period of Study:** 2/6/2001-2/20/2001 **Location:** Prague, Czech Republic	**Outcome:** Chromosomal aberrations **Study Design:** Panel **Covariates:** urinary cotinine, plasma levels of vitamins A, E and C **Statistical Analysis:** bivariate correlations, ANOVA, Mann-Whitney, Kruskal-Wallis and Spearman rank correlation **Statistical Package:** STATISTICA, SAS **Age Groups:** 53 city policemen, aged 22-50 years, spending 8+ hours outdoors	**Pollutant:** PM_{10} **Averaging Time:** NR **Range:** 32-55$\mu g/m^3$ **Copollutant:** $PM_{2.5}$	Results not given by PM increment.
Reference: Sram et al, (2007, 188457) **Period of Study:** 2/6/2001-2/20/2001 **Location:** Prague, Czech Republic	**Outcome:** Chromosomal aberrations **Study Design:** Panel **Covariates:** urinary cotinine, plasma levels of vitamins A, E and C **Statistical Analysis:** bivariate correlations, ANOVA, Mann-Whitney, Kruskal-Wallis and Spearman rank correlation **Statistical Package:** STATISTICA, SAS **Age Groups:** 53 city policemen, aged 22-50 years, spending 8+ hours outdoors	**Pollutant:** $PM_{2.5}$ **Averaging Time:** NR **Range:** 27-38$\mu g/m^3$ **Copollutant:** PM_{10}	Results not given by PM increment.

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Tovalin et al. (Tovalin et al., 2006, 091322) **Period of Study:** April-May 2002 **Location:** Mexico City and Puebla	**Outcome:** DNA damage (comet tail length) **Age Groups:** 18-60 **Study Design:** Panel Study **N:** 55 male workers **Statistical Analyses:** Mann-Whitney test, Chi-square, Spearman's correlation, logistic regression **Statistical Package:** SPSS and STATA	**Pollutant:** $PM_{2.5}$ Personal monitoring values observed in this study reported in Tovalin et al. 2003 **Median Personal Exposure to $PM_{2.5}$:** Mexico City Outdoor Worker: 133 $\mu g/m^3$ Indoor Worker: 86.6 $\mu g/m^3$ Puebla Outdoor Worker: 122 $\mu g/m^3$ Indoor Worker: 78.3 $\mu g/m^3$	OR for being a highly damaged worker: 1.02 (1.01-1.04), p = 0.03 Correlation between comet tail length and PM 2.5: 0.57, p = 0.000 OR for being a highly damaged worker: 1.03, p ◘ 0.07 **Comet Tail Length** Outdoor Worker: 46.80 μm Indoor Worker: 30.11 μm $p < 0.01$ **Percent Highly DNA Damaged Cells** Outdoor Worker: 68% Indoor Worker: 20%

[1]All units expressed in $\mu g/m^3$ unless otherwise specified.

Table E-28. Long-term exposure - cancer outcomes - other PM size fractions.

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: (Pope CA et al., 2002, 024689) **Period of Study:** 1982-1998 **Location:** 50 US states, District of Columbia, and Puerto Rico	**Outcome:** Lung cancer mortality (162) **Age Groups:** Ages > 30 years who were members of a household with at least one individual ≥45yrs. **Study Design:** Longitudinal cohort (Cancer Prevention Study II) **N:** 359,000 CPS II participants with information regarding PM_{15} and $PM_{15} – PM_{2.5}$ **Statistical Analyses:** Cox proportional hazard, incorporating a spatial random-effects component **Covariates:** Age, sex, race, education, ETS, smoking status, marital status, occupational exposure, diet, body-mass index, alcohol consumption Smoking covariates adjusted for: Indicator: current smoker, former smoker, pipe or cigar smoker, started smoking before or after age 18 Continuous, current and former smokers: years smoked, years smoked squared, cigarettes per day, cigarettes per day squared, number of hours per day exposed to passive cigarette smoke.	**Pollutant:** PM_{15} **Mean (SD):** 1979-1983: 40.3(7.7) **Pollutant:** $PM_{15-2.5}$ **Mean (SD):** 1979-1983: 19.2(6.1) **Averaging Time:** 1979-1983	Relative risks effect estimates were recorded in Figure 5 and not presented quantitatively anywhere else.

[1]All units expressed in $\mu g/m^3$ unless otherwise specified.

E.7. Long-Term Exposure and Reproductive Effects

Table E-29. Long-term exposure - reproductive outcomes - PM_{10}.

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Bell at al. (2007, 091059) **Period of Study:** 1999-2002 **Location:** Connecticut–Fairfield, Hartford, New Haven, New London, Windham, Massachusetts–Barnstable, Berkshire, Bristol, Essex, Hampden, Middlesex, Norfolk, Plymouth, Suffolk, Worcester	**Outcome:** Low birth weight **Age Groups:** Neonates **Study Design:** Cross-sectional **N:** 358,504 births **Statistical Analyses:** Multiple logistic and linear regressions **Covariates:** Child's sex, mother's education, tobacco use, mother's marital status, mother's race, time prenatal care began, mother's age, birth order, gestation length **Dose-response Investigated?** No **Statistical Package:** NR	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (SD):** 22.3 (5.3) **Monitoring Stations:** NR **Copollutant:** NO_2, CO, SO_2 **Gestation exposure correlation:** $PM_{2.5}$: r = 0.77 NO_2: r = 0.55	**PM Increment:** 7.4 $\mu g/m^3$ (IQR) **Difference in birth weight [Lower CI, Upper CI]** **per IQR for the gestational period:** -8.2 [-11.1 to -5.3] **Difference in birth weight by race of mother [Lower CI, Upper CI]:** Black: -7.9 [-16.0, 0.2] White: -9.0 [-12.2 to -5.9] **Range among trimester models for change in birth weight per IQR increase (min, max)** **trimester:** -6.6 to -4.7 3rd **OR Estimate for birth weight < 2500 g [Lower CI, Upper CI]** **per IQR for the gestational period:** 1.027 [0.991, 1.064] **Notes:** Analyses using first births alone yielded similar results. Two pollutant models for uncorrelated pollutants were analyzed but not presented quantitatively.
Reference: Brauer et al. (2008, 156292) **Period of Study:** 1999-2002 **Location:** Vancouver, BC	**Outcome:** Preterm birth, SGA, LBW **Age Groups: Study Design:** Cross-sectional **N:** 70,249 births **Statistical Analyses:** Logistic regression **Covariates:** Sex, parity, month and year of birth, maternal age and smoking, neighborhood level income and education **Statistical Package:** SAS	**Pollutant:** PM_{10} **Averaging Time:** 24-h **Mean (SD):** 12.7 **Range (Min, Max):** 5.6, 35.4 **Monitoring Stations:** 19 **Copollutant:** NO NO_2 CO SO_2 O_3	**PM Increment:** 1 $\mu g/m^3$ **Effect Estimate [Lower CI, Upper CI]** **pollutant assessed for entire pregnancy period:** SGA: 1.02 (0.99, 1.05) LBW: 1.01 (0.95, 1.08) Preterm (< 30 weeks): 1.13 (0.95, 1.35)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Chen et al. (2002, 024945)	**Outcome:** birth weight	**Pollutant:** PM_{10}	**PM Increment:** 10 $\mu g/m^3$
Period of Study: 1991-1999	**Age Groups:** single births with gestational age between 37-44 weeks and maternal all ages	**Averaging Time:** 24 h	**Effect Estimate [Lower CI, Upper CI]:**
Location: Washoe County, Nevada	**Study Design:** cross-sectional	**Mean (SD):** 31.53 (22.32)	Using continuous pollutant variables
	N: 33,859 single births	**Percentiles: 25th:** 16.80	Model one–PM_{10}
	Statistical Analyses: multiple linear and logistic regression	**50th(Median):** 26.30	1 trimester
	Covariates: infant sex, maternal residential city, education, medical risk factors, active tobacco use, drug use, alcohol use, prenatal care, mother's age, race and ethnicity of mothers and weight gain of mothers	**75th:** 39.35	Crude model: ß = -0.186 (0.225)
		Range (Min, Max): (0.97-157.32)	Adjusted model: ß = -0.082 (0.221)
		Monitoring Stations: 4	2 trimester
		Copollutant: CO	Crude model: ß = 0.045 (0.223)
		O_3	Adjusted model: ß = -0.020 (0.221)
	Dose-response Investigated? No		3 trimester
	Statistical Package: SPSS 10.0		Crude model: ß = -0.509 (0.231)
			Adjusted model: ß = -0.395 (0.227)
			Whole
			Crude model: ß = -0.823 (0.459)
			Adjusted model: ß = -0.726 (0.483)
			Model two
			CO and PM_{10}
			3 trimester
			Crude model: ß = -1.044 (0.457)
			Adjusted model: ß = -1.078 (0.445)
			O_3 and PM_{10}
			3 trimester
			Crude model: ß = -1.035 (0.385)
			Adjusted model: ß = -0.966 (0.378)
			Model three
			PM_{10}, O_3, and CO
			3 trimester
			Crude model: ß = -1.070 (0.458)
			Adjusted model: ß = -1.102 (0.446)
			Whole
			Crude model: ß = -1.413 (0.733)
			Adjusted model: ß = -1.332 (0.738)
			Using categorical pollutant variables–3 trimester
			Model 1–PM_{10}
			Adjusted model: ß = -10.243 (5.235)
			Model 2
			PM_{10} and CO
			Adjusted model: ß = -11.883 (6.108)
			PM_{10} and O_3 Adjusted model: ß = -9.144 (5.860)
			Model 3
			PM_{10}, CO, and O_3 Adjusted model: ß = -10.937 (6.222)
			Using logistic regression (ref value = < 19.72 $\mu g/m^3$
			Exposure to PM_{10} at 3 trimester at > 44.74 $\mu g/m^3$: OR = 1.105 (0.714-1.709)
			Between 19.72-44.74 $\mu g/m^3$: OR = 1.050 (0.811-1.360)
			Notes: Crude model: model with air-pollutant variables controlled with gestational age only. Adjusted model:

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Dales et al. (2004, 087342) **Period of Study:** Jan 1, 1984–Dec 31, 1999 **Location:** Canada (12 cities)	**Outcome:** SIDS (a sudden, unexplained death of a child < 1 year of age for which a clinical investigation and autopsy fail to reveal a cause of death) **Age Groups:** Infants < 1 yr **Study Design:** Time-series **N:** Total population of 12 cities: 10,310,309 1556 cases of SIDS over study period **Statistical Analyses:** Random-effects regression model for count data (a linear association between air pollution and the incidence of SIDS was assumed on the logarithmic scale) **Covariates:** weather factors (daily mean temp, daily mean relative humidity, maximum change in barometric pressure, all measured on the day of death), length of time-period adjustment, seasonal indicator variables, and size-fractionated PM **Season:** Used piece-wise constant functions in time that varied by 3, 6, or 12 months **Dose-response Investigated?** No **Statistical Package:** NR	**Pollutant:** PM_{10} **Averaging Time:** 24-hs (PM measures every 6 days gaseous pollutants every day) **Mean (IQR):** PM_{10}: 23.43 (15.56) **Range (Min, Max):** IQR presented above **Monitoring Stations:** When data were available from more than one monitoring site, they were averaged **Copollutant:** $PM_{2.5}$ PM_{10} CO NO_2 O_3 SO_2	**Notes:** The abstract reports no association between increased daily rates of SIDS and fine particles measured every sixth day. However, no effect estimates presented for PM (only gaseous pollutants adjusted for PM).
Reference: Dugandzic et al. (2006, 088681) **Period of Study:** 1/1/1988–12/31/2000 **Location:** Nova Scotia, Canada	**Outcome:** Low birth weight (LBW) (< 2500 grams) **Age Groups:** Babies born ≥ 37 weeks (full term) **Study Design:** cross-sectional **N:** 74,284 births **Statistical Analyses:** Logistic regression **Covariates:** Maternal age, parity, prior fetal death, prior neonatal death, prior low birth weight infant, smoking during pregnancy, neighborhood family income, infant gender, gestational age, weight change, year of birth **Season:** All **Dose-response Investigated?** Yes **Statistical Package:** SAS	**Pollutant:** PM_{10} **Averaging Time:** 24-h **Mean (SD):** **Percentiles: 25th:** 14 **50th(Median):** 16 **75th:** 19 **Range (Min, Max):** Max: 53 **Monitoring Stations:** 18 **Copollutant:** SO_2, O_3 **Notes:** Only three stations monitored more than one pollutant. Daily data were available for gaseous pollutants while particulate levels were measured every sixth day.	**PM Increment:** 1) IQR (5 $\mu g/m^3$) 2) Quartiles (first quartile is the reference) **Exposure period: first trimester** Unadjusted model 2nd quartile: 1.24 (0.95, 1.62) 3rd quartile: 1.25 (0.96, 1.62) 4th quartile: 1.28 (1.00, 1.65) Per IQR: 1.09 (1.00, 1.18) Adjusted model 2nd quartile: 1.24 (0.94, 1.64) 3rd quartile: 1.24 (0.95, 1.64) 4th quartile: 1.33 (1.02, 1.74) Per IQR: 1.09 (1.00, 1.19) Adjusted for Birth Year model 2nd quartile: 1.14 (0.86, 1.52) 3rd quartile: 1.08 (0.82, 1.44) 4th quartile: 1.11 (0.84, 1.48) Per IQR: 1.03 (0.94, 1.14) **Exposure period: second trimester** Unadjusted model 2nd quartile: 0.98 (0.76, 1.28) 3rd quartile: 1.09 (0.84, 1.40) 4th quartile: 1.00 (0.77, 1.28) Per IQR: 1.00 (0.91, 1.09) Adjusted model

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			2nd quartile: 1.02 (0.77, 1.34) 3rd quartile: 1.16 (0.89, 1.51) 4th quartile: 1.09 (0.83, 1.42) Per IQR: 1.02 (0.93, 1.12) Adjusted for Birth Year model 2nd quartile: 0.99 (0.75, 1.31) 3rd quartile: 1.10 (0.84, 1.45) 4th quartile: 1.01 (0.76, 1.34) Per IQR: 1.00 (0.90, 1.10) **Exposure period: third trimester** Unadjusted model 2nd quartile: 0.93 (0.72, 1.20) 3rd quartile: 1.07 (0.83, 1.37) 4th quartile: 0.92 (0.71, 1.18) Per IQR: 0.95 (0.87, 1.05) Adjusted model 2nd quartile: 0.96 (0.73, 1.26) 3rd quartile: 1.14 (0.88, 1.48) 4th quartile: 1.03 (0.79, 1.35) Per IQR: 0.99 (0.89, 1.09) Adjusted for Birth Year model 2nd quartile: 0.92 (0.70, 1.21) 3rd quartile: 1.04 (0.80, 1.36) 4th quartile: 0.92 (0.69, 1.22) Per IQR: 0.94 (0.85, 1.05)
Reference: Gilboa, et al. (2005, 087892) **Period of Study:** January 1, 1996-December 31, 2000 **Location:** Seven Counties in Texas, USA: (Bexar, Dallas, El Paso, Harris, Hidalgo, Tarrant, Travis)	**Outcome:** Birth defects **Age Groups:** newborn babies **Study Design:** Case-control **N:** 5,338 newborn babies 4574 controls **Statistical Analyses:** Logistic regression **Covariates:** alcohol consumption during pregnancy, attendant of delivery (i.e., the person who delivered the baby (physician/nursemaid-wife vs. other)), gravidity, marital status, maternal age, maternal education, maternal illness, maternal race/ethnicity, parity, place of delivery, plurality, prenatal care, season of conception, and tobacco use during pregnancy control frequency matched to cases by vital status, year and maternal county of residence **Season:** covariate in model **Dose-response Investigated?** Yes **Statistical Package:** SAS v 8.2	**Pollutant:** PM_{10} **Averaging Time:** NR **Percentiles: 25th:** < 19.5 **50th(Median):** 19.5- < 23.8 **75th:** 23.8- < 29.0 **100th:** ≥ 29.0 **Monitoring Stations:** The Environmental Protection Agency provided raw data for hourly (for gases) or daily (for PM) air pollution concentrations for the seven study counties **Copollutant:** CO, NO_2, O_3, SO_2	**PM Increment:** calculated as quartiles of avg concentration during weeks 3-8 of pregnancy **Isolated Cardiac Defects** **Aortic artery and valve defects:** 25th: 0.40 (0.15, 1.03) 50th: 0.45 (0.18, 1.13) 75th: 0.68 (0.28, 1.65) **Atrial septal defects:** 25th: 1.41 (0.86, 2.31) 50th: 2.13 (1.34, 3.37) 75th: 2.27 (1.43, 3.60) **Pulmonary artery and valve defects:** 25th: 1.14 (0.62, 2.10) 50th: 0.79 (0.41, 1.55) 75th: 0.68 (0.33, 1.40) **Ventricular septal defects:** 25th: 0.83 (0.61, 1.11) 50th: 1.12 (0.85, 1.48) 75th: 0.98 (0.73, 1.32) **Multiple Cardiac Defects**

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			Conotruncal defects:
			25th: 1.13 (0.79, 1.62)
			50th: 1.20 (0.84, 1.72)
			75th: 1.26 (0.86, 1.84)
			Endocardial cushion and mitral valve defects:
			25th: 0.82 (0.54, 1.25)
			50th: 0.66 (0.42, 1.05)
			75th: 0.63 (0.38, 1.03)
			Isolated Oral Clefts
			Cleft lip with or without palate:
			25th: 1.29 (0.90, 1.85)
			50th: 1.45 (1.01, 2.07)
			75th: 1.37 (0.94,2.00)
			Cleft palate:
			25th: 0.99 (0.55, 1.78)
			50th: 1.14 (0.64, 2.03)
			75th: 1.11 (0.60, 2.06)
			Individual Birth Defects
			Aortic valve stenosis:
			25th: 0.91 (0.53, 1.57)
			50th: 0.86 (0.50, 1.50)
			75th: 1.12 (0.63, 1.99)
			Atrial septal defects:
			25th: 1.10 (0.89, 1.35)
			50th: 1.28 (1.04, 1.57)
			75th: 1.26 (1.03, 1.55)
			Coarctation of the aorta:
			25th: 0.78 (0.53, 1.15)
			50th: 0.68 (0.45, 1.02)
			75th: 0.75 (0.48, 1.15)
			Endocardial cushion defects:
			25th: 0.87 (0.49, 1.55)
			50th: 1.12 (0.64, 1.96)
			75th: 0.89 (0.47, 1.65)
			Ostium secundum:
			25th: 1.15 (0.85, 1.55)
			50th: 1.13 (0.83, 1.53)
			75th: 1.06 (0.77, 1.48)
			Pulmonary artery atresia without ventricular septal defects:
			25th: 1.93 (1.08, 3.45)
			50th: 2.01 (1.11, 3.64)
			75th: 0.86 (0.41, 1.83)
			Pulmonary valve stenosis:
			25th: 1.16 (0.88, 1.55)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			50th: 1.25 (0.94, 1.66) 75th: 1.27 (0.94, 1.71) **Tetralogy of Fallot:** 25th: 1.21 (0.72, 2.01) 50th: 1.40 (0.84, 2.33) 75th: 1.45 0.85, 2.48) **Ventricular septal defects:** 25th: 1.06 (0.90, 1.24) 50th: 1.10 (0.94, 1.29) 75th: 1.08 (0.92, 1.27)
Reference: Gouveia et al. (2004, 055613) **Period of Study:** 1997 **Location:** São Paulo, Brazil	**Outcome:** birth weight **Age Groups:** singleton full term live births within 1000 g to 5500 g **Study Design:** Cross sectional study **N:** 179,460 live births **Statistical Analyses:** GAM and Logistic regression models **Covariates:** maternal age, length of gestation, season, infant gender, maternal education, number of antenatal care visits, parity, and the type of delivery **Season:** All seasons **Dose-response Investigated?** Yes **Statistical Package:** S-Plus 2000	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (SD):** 60.3 (25.2) **Range (Min, Max):** (25.5-153.0) **Monitoring Stations:** maximum of 12 sites **Copollutant (correlation):** CO: r = 0.9 SO_2 NO_2 O_3	**PM Increment:** 10 $\mu g/m^3$ **Mean [Lower CI, Upper CI]:** Changes in birth weight (in g) First trimester = -13.7 (-27.0, -0.4) Second trimester = -4.4 (-18.9, 10.1) Third trimester = 14.6 (0.0, 29.2) **RR Estimate [Lower CI, Upper CI]:** (RR estimates are adjusted odds ratios for low birth weight according to quartiles of air pollution in each trimester of pregnancy.) 1st quartile First trimester = 1 (REF) Second trimester = 1 (REF) Third trimester = 1 (REF) 2nd quartile First trimester = 1.105 (0.994, 1.229) Second trimester = 1.003 (0.904, 1.113) Third trimester = 1.004 (0.914, 1.104) 3rd quartile First trimester = 1.049 (0.903, 1.219) Second trimester = 1.074 (0.920, 1.254) Third trimester = 1.003 (0.861, 1.169) 4th quartile First trimester = 1.144 (0.878, 1.491) Second trimester = 1.252 (1.028, 1.525) Third trimester = 0.970 (0.780, 1.205) Multiple linear regression coefficients (SE) obtained from single, dual, and three pollutant models Single pollutant model = -1.37 (0.68) Two pollutant (PM_{10} and CO) = -0.51 (0.87) Two pollutant (PM_{10} and SO_2) = -0.94 (0.75) Three pollutant = -0.47 (0.88)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Ha et al. (2003, 042552) **Period of Study:** Jan 1995-Dec 1999 **Location:** Seoul, South Korea	**Outcome:** Post-neonate total and respiratory mortality **Age Groups:** 1 month-1 yr 2 yr-65 yr, > 65 yr **Study Design:** Time-series **N:** 1045 post-neonate deaths, 67,597 2-65 yr old deaths, 100,316 > 65 yr old deaths **Statistical Analyses:** Generalized additive model **Covariates:** Seasonality, temperature, relative humidity, day of the week **Dose-response Investigated?** No **Statistical Package:** S Plus **Lags Considered:** 0, 1, 2, 3, 4, 5, 6, 7, moving averages from 1-5 days	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (SD):** 69.2 (31.6) **Percentiles: 25th:** 44.8 50th(Median): 64.2 75th: 87.7 **Range (Min, Max):** 10.5 $\mu g/m^3$, 245.4 $\mu g/m^3$ **Monitoring Stations:** 27 **Copollutant (correlation):** NO_2: r = 0.73 SO_2: r = 0.62 O_3: r = -0.02 CO: r = 0.63	**PM Increment:** 42.9 $\mu g/m^3$ **RR Estimate [Lower CI, Upper CI]** **lag:** Total Mortality: 1 month-1 yr (post-neonates): 1.142 [1.096, 1.190] lag 0 2 yr-65 yr: 1.008 [1.006, 1.010] lag 0 > 65 yr (elderly): 1.023 [1.023, 1.024] lag 0 Respiratory Mortality: 1 month-1 yr (post-neonates): 2.018 [1.784, 2.283] lag 0 2 yr-65 yr: 1.066 [1.044, 1.090] lag 0 > 65 yr (elderly): 1.063 [1.055, 1.072] lag 0
Reference: Hansen, et al. (2006, 089818) **Period of Study:** July 1, 2000- June 30, 2003 **Location:** Brisbane, Australia	**Outcome:** Pre-term birth (< 37 weeks) **Age Groups:** newborn babies **Study Design:** Cross-sectional **N:** 1583 live pre-terms births 28,200 singleton live births **Statistical Analyses:** Multiple logistic regression models **Covariates:** Neonate gender, mother's age, parity, indigenous status, number of antenatal visits, marital status, number of previous abortions/miscarriages, type of delivery, and index of SES **Season:** all **Dose-response Investigated?** Yes **Statistical Package:** SAS version 8.2	**Pollutant:** PM_{10} **Averaging Time:** recorded hourly, averaged daily **Mean (SD):** 19.6 (9.4) **Range (Min, Max):** 4.9, 171.7 **Monitoring Stations:** 5 **Copollutant (correlation):** Fine PM or bsp, 0.1 to < 2.5 μg in diameter (0.58 to 0.76) O_3 (0.54 to 0.83) NO_2 (0.54 to 0.75) PM_{10} (0.80 to 0.93) **Note:** Correlations presented are for the individual pollutant across monitoring stations (not correlations between PM_{10} and the pollutant.)	**PM Increment:** Trimester One 4.5 $\mu g/m^3$ Last 90 days prior to birth 5.7 $\mu g/m^3$ **Odds Ratio [Lower CI, Upper CI]:** Trimester one 1.15 [1.06, 1.25] Last 90 days prior to birth 1.04 [0.92, 1.16]
Reference: Hansen et al. (2007, 090703) **Period of Study:** Jul 2000–Jun 2003 **Location:** Brisbane, Australia	**Outcome:** Birth weight and Small for Gestational Age (SGA < 10th percentile for age and gender) head circumference (HC) and crown-heel length (CHL) among subsample **Study Design:** Cross-sectional **N:** 26,617 births (birth weight analysis) and 21,432 (HC and CHL analyses) **Statistical Analyses:** Logistic (SGA) and linear (birth weight, HC, CHL) regressions **Covariates:** gender, gestational age (with a quadratic term), maternal age, parity, number of previous abortions/miscarriages, marital status, indigenous status, number of antenatal visits, type of delivery, an index of SES, and season of birth **Season:** assessed as a covariate	**Pollutant:** PM_{10} **Averaging Time:** Trimester and monthly averages were used in analyses (calculated as the mean of daily values hourly data was use to calculate daily means city-wide avg used) **Mean (SD):** 19.6 (9.4) **Percentiles:** 25th: 14.6 50th: 18.1 75th: 22.7 **Range (Min, Max):** (4.9, 171.7) **Monitoring Stations:** 5 **Copollutant (correlation):** By trimesters: PM_{10} T1:	**PM Increment:** IQR (8.1 $\mu g/m^3$) **Effect Estimate [Lower CI, Upper CI]:** **Change (β) in mean birth weight (g) associated with trimester-specific exposures** Trimester 1: Continuous exposure: -3.2 (-11.9, 5.5) Quartiles of exposure: 1: Ref 2: -4.7 (-19.7, 10.2) 3: 4.2 (-12.9, 21.3) 4: -0.2 (-19.2, 18.8) p-trend: 0.864 Trimester 2: Continuous exposure: 0.4 (-9.4, 10.2)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
	Dose-response Investigated? Yes, assessed exposures as quartiles **Statistical Package:** SAS v8.2	PM_{10} T2: r = 0.12	Quartiles of exposure:
		PM_{10} T3: r = -0.55	1: Ref
		O_3 T1: r = 0.77	2: 12.7 (-2.3, 27.6)
		O_3 T2: r = 0.28	3: 7.6 (-10.6, 25.7)
		O_3 T3: r = -0.61	4: 1.0 (-18.7, 20.7)
		NO_2 T1: r = 0.32	p-trend: 0.922
		NO_2 T2: r = -0.65	Trimester 3:
		NO_2 T3: r = -0.17	Continuous exposure: 3.6 (-6.9, 14.0)
		visibility reducing particles (bsp) T1: r = 0.82	Quartiles of exposure:
		visibility reducing particles (bsp) T2: r = -.15	1: Ref
		visibility reducing particles (bsp) T3: r = -0.50	2: 2.9 (-12.8, 18.7)
		PM_{10} T1: r = 0.12	3: 18.5 (0.0, 36.9)
		PM_{10} T2:	4: 4.3 (-15.8, 24.4)
		PM_{10} T3: r = 0.04	p-trend: 0.524
		O_3 T1: r = -0.11	**ORs for SGA associated with trimester-specific exposures**
		O_3 T2: r = 0.80	Trimester 1:
		O_3 T3: r = 0.18	Continuous exposure: 1.04 (0.96, 1.12)
		NO_2 T1: r = 0.77	Quartiles of exposure:
		NO_2 T2: r = 0.25	1: Ref
		NO_2 T3: r = -0.72	2: 1.23 (1.07, 1.42)
		visibility reducing particles (bsp) T1: r = 0.23	3: 1.12 (0.95, 1.31)
		visibility reducing particles (bsp) T2: r = 0.80	4: 1.12 (0.94, 1.34)
		visibility reducing particles (bsp) T3: r = -0.24	p-trend: 0.361
		PM_{10} T1: r = -0.55	Trimester 2:
		PM_{10} T2: r = 0.04	Continuous exposure: 0.95 (0.88, 1.04)
		PM_{10} T3:	Quartiles of exposure:
		O_3 T1: r = -0.56	1: Ref
		O_3 T2: r = -0.18	2: 0.96 (0.83, 1.11)
		O_3 T3: r = 0.81	3: 1.06 (0.89, 1.25)
		NO_2 T1: r = -0.20	4: 0.98 (0.81, 1.18)
		NO_2 T2: r = 0.75	p-trend: 0.962
		NO_2 T3: r = 0.22	Trimester 3:
		visibility reducing particles (bsp) T1: r = -0.62	Continuous exposure: 0.93 (0.85, 1.03)
		visibility reducing particles (bsp) T2: r = 0.19	Quartiles of exposure:
		visibility reducing particles (bsp) T3: r = 0.79	1: Ref
			2: 0.90 (0.78, 1.04)
			3: 0.81 (0.68, 0.96)
			4: 0.86 (0.71, 1.04)
			p-trend: 0.098
			Change (β) in mean head circumference (HC
			cm) associated with trimester-specific exposures
			Trimester 1:
			Continuous exposure: -0.01 (-0.04, 0.02)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			Quartiles of exposure:
			1: Ref
			2: -0.02 (-0.07, 0.04)
			3: -0.02 (-0.08, 0.04)
			4: -0.02 (-0.08, 0.05)
			p-trend: 0.605
			Trimester 2:
			Continuous exposure: -0.01 (-0.04, 0.02)
			Quartiles of exposure:
			1: Ref
			2: 0.03 (-0.02, 0.08)
			3: 0.00 (-0.06, 0.06)
			4: -0.01 (-0.08, 0.05)
			p-trend: 0.538
			Trimester 3:
			Continuous exposure: 0.02 (-0.02, 0.05)
			Quartiles of exposure:
			1: Ref
			2: 0.02 (-0.04, 0.07)
			3: 0.07 (0.01, 0.13)
			4: 0.04 (-0.03, 0.11)
			p-trend: 0.171
			Change (β) in mean crown-heel length (CHL
			cm) associated with trimester-specific exposures
			Trimester 1:
			Continuous exposure: 0.00 (-0.05, 0.05)
			Quartiles of exposure:
			1: Ref
			2: 0.02 (-0.07, 0.11)
			3: 0.01 (-0.10, 0.11)
			4: 0.04 (-0.07, 0.16)
			p-trend: 0.511
			Trimester 2:
			Continuous exposure: 0.07 (0.01, 0.13)
			Quartiles of exposure:
			1: Ref
			2: 0.10 (0.01, 0.18)
			3: 0.11 (0.00, 0.21)
			4: 0.13 (0.01, 0.24)
			p-trend: 0.049
			Trimester 3:
			Continuous exposure: -0.01 (-0.07, 0.05)
			Quartiles of exposure:
			1: Ref

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			2: -0.02 (-0.11, 0.07) 3: 0.10 (-0.01, 0.21) 4: -0.01 (-0.13, 0.10) p-trend: 0.883
Reference: (Hansen et al., 2009, 192362) **Period of Study:** 1/1997-12/2004 **Location:** Brisbane, Australia	**Outcome:** birth defects- artery and valve, atrial and ventricular septal, conotruncal, endocardial cushion and mitral valve, cleft lip and palate **Study Design:** Case-control **Covariates:** mother's age, marital status, indigenous status, previous pregnancies, last menstrual period, area-level socioeconomic status, distance to a pollution monitor **Statistical Analysis:** Conditional logistic regression **Statistical Package:** R **Age Groups:** neonates	**Pollutant:** PM_{10} **Averaging Time:** daily **Mean (SD) Unit:** 18.0 $\mu g/m^3$ **Range (Min, Max):** (4.4, 151.7) **Copollutant (correlation):** NR	**Increment:** 4$\mu g/m^3$ **Odds Ratios (95% CI) for risk of defect** Aortic Artery and Valve Defects All Births, Matched: 1.10 (0.76-1.56) Births ◘ 12km to Monitor: 1.83 (1.16-2.98) Births ◘ 6km to Monitor: 1.43 (0.73-2.90) All Births, Unmatched: 1.09 (0.84-1.39) Atrial Septal Defects All Births, Matched: 1.06 (0.86-1.30) Births ◘ 12km to Monitor: 1.07 (0.84-1.37) Births ◘ 6km to Monitor: 0.88 (0.60-1.27) All Births, Unmatched: 1.14 (0.98-1.33) Pulmonary Artery and Valve Defects All Births, Matched: 0.90 (0.61-1.29) Births ◘ 12km to Monitor: 0.69 (0.43-1.08) Births ◘ 6km to Monitor: 1.46 (0.76-2.73) All Births, Unmatched: 0.99 (0.78-1.24) Ventricular Septal Defects All Births, Matched: 0.87 (0.73-1.04) Births ◘ 12km to Monitor: 0.85 (0.69-1.03) Births ◘ 6km to Monitor: 0.90 (0.68-1.18) All Births, Unmatched: 1.15 (1.02-1.30) Conotruncal Defects All Births, Matched: 0.80 (0.54-1.19) Births ◘ 12km to Monitor: 0.94 (0.55-1.49) Births ◘ 6km to Monitor: 0.66 (0.27-1.45) All Births, Unmatched: 0.97 (0.74-1.24) Endocardial Cushion and Mitral Valve Defects All Births, Matched: 1.29 (0.82-2.04) Births ◘ 12km to Monitor: 1.28 (0.75-2.19) Births ◘ 6km to Monitor: 0.90 (0.44-1.86) All Births, Unmatched: 0.94 (0.68-1.26) Cleft Lip

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			All Births, Matched: 1.05 (0.72-1.51)
			Births ◘ 12km to Monitor: 1.16 (0.72-1.82)
			Births ◘ 6km to Monitor: 1.03 (0.56-1.82)
			All Births, Unmatched: 1.01 (0.79-1.27)
			Cleft Palate
			All Births, Matched: 0.69 (0.50-0.93)
			Births ◘ 12km to Monitor: 0.53 (0.29-0.87)
			Births ◘ 6km to Monitor: 0.71 (0.49-1.00)
			All Births, Unmatched: 0.89 (0.72-1.10)
			Cleft Lip with or without Cleft Palate
			All Births, Matched: 1.05 (0.84-1.30)
			Births ◘ 12km to Monitor: 1.03 (0.79-1.34)
			Births ◘ 6km to Monitor: 0.83 (0.58-1.19)
			All Births, Unmatched: 1.04 (0.89-1.21)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Jalaludin et al. (2007, 156601) **Period of Study:** 1998-2000 **Location:** Sydney, Australia	**Outcome:** Gestational age (categorized: preterm birth: < 37 weeks term birth: ≥ 37 weeks but < 42 weeks) **Age Groups:** infants **Study Design:** Cross-sectional **N:** 123,840 singleton births of > 20 weeks gestation **Statistical Analyses:** Logistic regression **Covariates:** sex of child, maternal age, maternal smoking during pregnancy, gestational age at first antenatal visit, whether mother identifies as being Aboriginal or Torres Strait Islander, whether first pregnancy, season of conception, SES, (temperature and relative humidity were not significant in single variable models and therefore, were not included) **Season:** examined as covariate and effect modifier **Dose-response Investigated?** No **Statistical Package:** SAS v8	**Pollutant:** PM_{10} **Averaging Time:** 24 h averages used to calculate the mean concentration over the first trimester, the 3 months preceding birth, the first month after the estimated date of conception, and the month prior to delivery **Mean (SD):** (24 hr averages) All year: 16.3 (6.38) summer: 18.2 (7.20) Autumn: 17.0 (6.23) Winter: 14.5 (5.57) Spring: 15.7 (5.82) **Monitoring Stations:** 14 stations within the Sydney metropolitan area (levels averaged to provide one estimate for the entire study area) **Copollutant (correlation):** PM_{10} $PM_{2.5}$ (r = 0.83) CO (r = 0.28) NO_2 (r = 0.48) O_3 (r = 0.50) SO_2 (r = 0.42) **Notes:** Correlations between monitoring stations measuring PM_{10} ranged from 0.67 to 0.91	**PM Increment:** 1 $\mu g/m^3$ **Effect Estimate [Lower CI, Upper CI]:** ORs (air pollutant concentration during the 1st trimester and preterm birth by season) Autumn: 1.462 (1.267, 1.688) Winter: 1.343 (1.190, 1.516) Spring: 1.119 (0.973, 1.288) Summer: 0.913 (0.889, 0.937) ORs (air pollutant concentrations during different exposure periods and preterm birth for all of Sydney and among only those residing within 5 km of a monitoring station) 1 month preceding birth Sydney: 0.991 (0.979, 1.003) 5km: 1.008 (0.993, 1.022) 3 months preceding birth Sydney: 0.989 (0.975, 1.004) 5km: 1.012 (0.995, 1.030) 1st month of gestation Sydney: 0.983 (0.973, 0.993) 5km: 0.957 (0.914, 1.002) 1st trimester Sydney: 0.987 (0.973, 1.001) 5km: 1.009 (0.978, 1.041) **Notes:** Authors note that effect of PM_{10} on preterm birth for infants conceived during the autumn did not remain in 2 pollutant models (ORs between 0.77 and 1.04)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Kaiser et al. (2004, 076674) **Period of Study:** 1995-1997 **Location:** 25 US counties (23 metropolitan areas): Jackson, AL Fresno, CA Los Angeles, CA Sacramento, CA San Diego, CA San Francisco, CA Denver, CO Hartford, CT Cook, IL Baltimore, MD Wayne, MI St. Louis, MO Bronx, NY Kings, NY New York, NY Philadelphia, PA El Paso, TX Harris, TX Dallas, TX Oklahoma, OK Tulsa, OK Providence, RI Salt Lake City, UT King, WA Milwaukee, WI	**Outcome:** Postneonatal death: All cause, SIDS (798.0) Respiratory disease (460-519) **Age Groups:** infants between 1-12 months **Study Design:** Attributable risk assessment **N:** 700,000 infants (# deaths NR) **Statistical Analyses:** Risk assessment methods described in: Kunzli et al. Public-health impact of outdoor and traffic-related air pollution: a European assessment. Lancet 2000, 356: 795-801. **Covariates:** Maternal education, maternal ethnicity, parental marital status, maternal smoking during pregnancy, infant's month and year of birth, avg temperature in the first 2 months of life **Season:** All adjusted for month/year of birth **Dose-response Investigated?** NR **Statistical Package:** NR **Lags Considered:** Annual, county-level mean	**Pollutant:** PM_{10} **Averaging Time:** "annual mean levels" in each county **Mean (SD):** 28.4 **Range (Min, Max):** County range: 18.0, 44.8 **Monitoring Stations:** NR **Notes:** 14 out of 25 counties had PM_{10} levels $> 25\ \mu g/m^3$	**PM Increment:** Analysis 1: 16.4 $\mu g/m^3$ (difference between reference level of 12 $\mu g/m^3$ and observed mean level of 28.4 $\mu g/m^3$) Analysis 2: 13 $\mu g/m^3$ (difference between reference level of 12 $\mu g/m^3$ and 25 $\mu g/m^3$) **AR Estimate [Lower CI, Upper CI]:** Analysis 1: All cause 6% [3, 11] SIDS 16% [9, 23] Respiratory 24% [7, 44] Attributable # deaths per 100,000 infants: All cause 14.7 [7.3, 25.6] SIDS 11.7 [6.8, 16.6] Respiratory 2.3 [0.7, 4.1] Analysis 2: All cause 5% [2, 8] SIDS 12% [7, 18] Respiratory 19% [6, 34] Attributable # deaths per 100,000 infants: All cause 10.9 [5.5, 19.1] SIDS 9.0 [5.3, 12.8] Respiratory 1.8 [0.5, 3.2] **Notes:** -Authors did not extrapolate attributable cases below 12 $\mu g/m^3$ (i.e., reference level was set at 12 $\mu g/m^3$) -Attributable risks are based on the RRs reported by Woodruff et al, 1997 for a 10 $\mu g/m^3$ increase: All cause 1.04 [1.02-1.07] SIDS 1.12 [1.07, 1.17] Respiratory 1.20 [1.06, 1.36]
Reference: (Kim et al., 2007, 156642) **Period of Study:** May 1, 2001–May 31, 2004 **Location:** Seoul, Korea	**Outcome (ICD9 and ICD10):** LBW (low birth weight, less than 2500 g at later than gestational week 37), premature delivery (birth before the completion of the 37th week), stillbirth (intrauterine fetal death), IUGR (birth weight lower than the 10th percentile for the given gestational age), and congenital anomaly (a defect in the infant's body structure) **Age Groups:** Infants **Study Design:** Cross-sectional (women visiting the clinic for prenatal care were recruited with follow-up until discharge after delivery) **N:** 1514 observations (births) **Statistical Analyses:** multiple logistic and linear regression (in addition, for birth weight, used generalized additive model to account for long-term trends and nonlinear relationships between the response variable and the predictors, and	**Pollutant:** PM_{10} **Averaging Time:** Used hourly exposure levels to calculate avg exposure levels at each trimester, each month of pregnancy, and 6 weeks before delivery from the nearest monitoring station (based on home address of mother) also created categories within each pregnancy period (< 25th percentile [referent], 25th to 50th percentile, and > 50th percentile) **Mean (SD):** Range of PM means across pregnancy periods: 88.7-89.7 **Monitoring Stations:** 27 stations	**PM increment:** 10$\mu g/m^3$ **Preterm:** 1st Trimester Odds Ratios: Crude: 0.95 (0.90, 1.01) Adj 1: 0.93 (0.87, 1.00) Adj 2: 0.93 (0.85, 1.01) 2nd Trimester Odds Ratios: Crude: 0.99 (0.94, 1.06) Adj 1: 0.98 (0.92, 1.04) Adj 2: 1.00 (0.93, 1.07) 3rd Trimester Odds Ratios: Crude: 1.02 (0.98, 1.06) Adj 1: 1.05 (1.00, 1.10) Adj 2: 1.05 (0.99, 1.11)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
	to produce smoothed plots of the relationship between PM and birth weight) **Covariates:** Adjustment 1: infant sex, infant order, maternal age and education, paternal education, season of birth Adjustment 2: adjustment 1 factors plus alcohol, maternal BMI, maternal weight prior to delivery (collected information on smoking, ETS, parity, past history of illnesses, history of illnesses during pregnancy but did not use in analyses due to small numbers or non-significance) **Season:** adjusted for season of delivery **Dose-response Investigated?** Yes **Statistical Package:** SAS 8.01, S-Plus 2000		**LBW**: 1st Trimester Odds Ratios: Crude: 1.02 (0.93, 1.12) Adj 1: 1.03 (0.93, 1.14) Adj 2: 1.07 (0.96, 1.19) 2nd Trimester Odds Ratios: Crude: 1.03 (0.94, 1.14) Adj 1: 1.04 (0.93, 1.17) Adj 2: 1.07 (0.94, 1.22) 3rd Trimester Odds Ratios: Crude: 1.04 (0.97, 1.11) Adj 1: 1.05 (0.97, 1.14) Adj 2: 1.05 (0.96, 1.16) **IUGR**: 1st Trimester Odds Ratios: Crude: 1.07 (0.97, 1.19) Adj 1: 1.07 (0.95, 1.21) Adj 2: 1.14 (0.99, 1.31) 2nd Trimester Odds Ratios: Crude: 0.97 (0.85, 1.12) Adj 1: 0.97 (0.82, 1.13) Adj 2: 0.93 (0.77, 1.13) 3rd Trimester Odds Ratios: Crude: 0.82 (0.68, 0.99) Adj 1: 0.88 (0.72, 1.08) Adj 2: 0.85 (0.67, 1.08) **Birth defect**: 1st Trimester Odds Ratios: Crude: 1.08 (0.98, 1.20) Adj 1: 1.12 (1.00, 1.25) Adj 2: 1.08 (0.95, 1.22) 2nd Trimester Odds Ratios: Crude: 1.09 (0.99, 1.21) Adj 1: 1.11 (0.98, 1.26) Adj 2: 1.16 (1.00, 1.34) 3rd Trimester Odds Ratios: Crude: 1.00 (0.90, 1.11) Adj 1: 0.97 (0.86, 1.08) Adj 2: 0.97 (0.87, 1.10) **Stillbirth**: 1st Trimester Odds Ratios: Crude: 0.83 (0.76, 0.90) Adj 1: 0.93 (0.85, 1.02) Adj 2: 0.95 (0.85, 1.02) 2nd Trimester Odds Ratios:

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			Crude: 0.99 (0.93, 1.05)
			Adj 1: 1.03 (0.95, 1.11)
			Adj 2: 1.07 (0.98, 1.17)
			3rd Trimester Odds Ratios:
			Crude: 1.14 (1.10, 1.18)
			Adj 1: 1.09 (1.04, 1.15)
			Adj 2: 1.08 (1.02, 1.14)
			LBW (categorical PM exposure):
			1st Trimester ORs:
			< 25th: 1.0
			25th-50th: 0.5 (0.1, 3.2)
			> 50th: 1.0 (0.3, 3.8)
			3rd Trimester ORs:
			< 25th: 1.0
			25th-50th: 1.3 (0.2, 10.4)
			> 50th: 3.0 (0.5, 18.5)
			6 wk before birth ORs:
			< 25th: 1.0
			25th-50th: 3.2 (0.3, 33.7)
			> 50th: 5.2 (0.6, 47.6)
			Changes in Birth Weight (95%CI) per 10 $\mu g/m^3$ increase in PM concentration: 1st trimester: 7.8 (1.2, 14.5)
			2nd trimester: -0.3 (-7.3, 6.8)
			3rd trimester: -2.1 (-7.5, 3.4)
			1st month: 4.4 (-1.0, 9.8)
			2nd month: 6.4 (0.6, 12.2)
			3rd month: 4.3 (-1.5, 10.2)
			4th month: 3.0 (-3.7, 9.6)
			5th month: -3.9 (-10.5, 2.7)
			6th month: 0.1
			(-5.7, 5.8)
			7th month: 0.1 (-5.1, 5.3)
			8th month: 0.0 (-4.5, 4.5)
			9th month: 1.8 (-2.3, 5.9)
			Last 6 wk: -4.8 (-9.9, 0.4)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Lee et al. (2003, 043202) **Period of Study:** Jan 1, 1996-Dec 31 1998 **Location:** Seoul, South Korea	**Outcome:** Low birth weight (LBW), < 2500 g **Age Groups:** child-bearing age women and their newborn children – delivered at 37-44 gestational weeks **Study Design:** Cross-section **N:** 388,905 full-term single births **Statistical Analyses:** Generalized additive model, LOESS, Akaike's criterion, **Covariates:** Infant sex, birth order, maternal age, parental education level, time trend and gestational age. **Season:** All **Dose-response Investigated?** Yes **Statistical Package:** NR	**Pollutant:** PM_{10} **Averaging Time:** Arithmetic avg of hourly measurements at 20 stations **Mean (SD):** 71.1 (30.1) **Percentiles: 25th:** 47.4 **50th(Median):** 67.6 **75th:** 89.3 **Range (Min, Max):** 18.4, 236.9 **Monitoring Stations:** 20 **Copollutant (correlation):** 1st trimester: PM_{10}–CO: 0.47 PM_{10}–SO_2: 0.78 PM_{10}–NO_2: 0.66 2nd trimester: PM_{10}–CO: 0.68 PM_{10}–SO_2: 0.82 PM_{10}–NO_2: 0.81 3rd trimester: PM_{10}–CO: 0.69 PM_{10}–SO_2: 0.85 PM_{10}–NO_2: 0.80	**PM Increment:** IQR, 41.9 **RR Estimate [Lower CI, Upper CI]:** 1st trimester: 1.03 [1.00, 1.07] 2nd trimester: 1.04 [1.00, 1.08] 3rd trimester: 1.00 [0.95, 1.04] All trimesters: 1.06 [1.01, 1.10] Low exposure in last 5 months using IQR during last 5 months: 0.94 [0.85, 1.05] Low exposure in first 5 months using IQR during first 5 months: 1.04 [1.01, 1.08] **Notes:** Birth weight was decreased by 19.6 g for an IQR increase in the 2nd trimester. The OR for LBW increased for female children, fourth or higher order child, mother < 20 yrs of age, and low parental education level.

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Leem et al. (2006, 089828) **Period of Study:** 2001-2002 **Location:** Incheon, Korea	**Outcome (ICD9 and ICD10): Age Groups:** Pre-term delivery **Study Design:** Cross-sectional **N:** Cases: 2,082 Controls: 50,031 **Statistical Analyses:** Log-binomial regression (corrected for overdispersion used the log link function) **Covariates:** Maternal age, parity, sex, season of birth, and education level of each parent **Season:** Controlled as a covariate **Dose-response Investigated?** Yes, assessed quartiles of exposure **Statistical Package:** NR	**Pollutant:** PM_{10} **Averaging Time:** Trimesters (daily hourly data used to calculate) **Range (Min, Max):** Reported ranges within quartiles by trimester: 1st Trimester: 4: 64.57-106.39 3: 53.84-64.56 2: 45.95-53.83 1: 26.99-45.94 3rd Trimester: 4: 65.63-95.91 3: 56.07-65.62 2: 47.07-56.06 1: 33.12-47.06 **Monitoring Stations:** 27 monitoring stations pollutant levels for each area were predicted from the levels recorded at the monitors using ordinary block kriging **Copollutant (correlation):** SO_2 (r = 0.13) NO_2 (r = 0.37) CO (r = 0.27)	**Effect Estimate [Lower CI, Upper CI]:** **Crude and Adjusted RR for preterm delivery and exposure during the 1st trimester** Crude Quartiles of exposure: 4: 1.07 (0.95, 1.21) 3: 1.02 (0.90, 1.15) 2: 1.06 (0.94, 1.20) 1: 1.00 Adjusted Quartiles of exposure: 4: 1.27 (1.04, 1.56) 3: 1.13 (0.94, 1.37) 2: 1.14 (0.97, 1.34) 1: 1.00 p-trend: 0.39 **Crude and Adjusted RR for preterm delivery and exposure during the 3rd trimester** Crude Quartiles of exposure: 4: 1.06 (0.94, 1.20) 3: 1.06 (0.94, 1.19) 2: 1.05 (0.93, 1.18) 1: 1.00 Adjusted Quartiles of exposure: 4: 1.09 (0.91, 1.30) 3: 1.04 (0.90, 1.21) 2: 1.05 (0.91, 1.20) 1: 1.00 p-trend: 0.33

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Lin et al. (2004, 095787) **Period of Study:** 1/98-12/00 **Location:** São Paulo, Brazil	**Outcome:** Neonatal death **Age Groups:** Neonates (infants 0-28 days after birth) **Study Design:** Time series **N:** 1096 days, 6697 deaths **Statistical Analyses:** Poisson regression (GAM) **Covariates:** Non-parametric LOESS smoothers to control for: time (long term trend), temperature, humidity, and day of week Also controlled for holidays with linear term **Season:** All **Dose-response Investigated?** No **Statistical Package:** NR **Lags Considered:** Lag 0, "moving averages from 2 to 7 days" **Notes:** No explicit control for season apart from temperature	**Pollutant:** PM_{10} **Averaging Time:** Daily values **Mean (SD):** 48.62 (21.18) **Range (Min, Max):** 13.9, 157.3 **Monitoring Stations:** NR (indicated more than 1) **Copollutant (correlation):** CO r = 0.71 NO_2 r = 0.76 SO_2 r = 0.80 O_3 r = 0.36	**PM Increment:** 1 $\mu g/m^3$ **Log relative rate (standard error)** **lag** Single pollutant model 0.0017 (0.0008) lag 0 This translates to a 4.0% [95% CI: 0.3, 7.9] increase in neonatal mortality for a 23.3 $\mu g/m^3$ increase in PM_{10} Two-pollutant model 0.0000 (0.0011) lag 0 **Notes:** -In two pollutant model with PM_{10} and SO_2 (which are highly correlated), effect of PM disappeared and effect of SO_2 remained constant - results from pollutant moving averages from 2 to 7 days not reported, authors indicate effects only found for lag 0 (same day levels) - confidence intervals reported in abstract are incompatible with βs/standard errors and plotted results in text: abstract indicates a 4% increase in mortality with 95% CI: 2-6 for a 23.3 $\mu g/m^3$ increase in PM_{10}

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: (Lin et al., 2004, 089827) **Period of Study:** 1995-1997 **Location:** Taipei and Kaoshiung, Taiwan	**Outcome:** Low birth weight (< 2500 grams) **Age Groups:** newborns **Study Design:** Cross-sectional **N:** 92,288 infants **Statistical Analyses:** Logistic regression **Covariates:** Gender, birth order, gestational weeks, season of birth, maternal age, maternal education, copollutants **Season:** All **Dose-response Investigated?** Yes **Statistical Package:** NR **Lags Considered:** The 9-month pregnancy period for each infant, and each trimester	**Pollutant:** PM_{10} **Averaging Time:** NR, "daily measurements" **Mean (SD):** Reported by monitoring station: Taipei: 1. 48.78 2. 46.29 3. 48.79 4. 50.80 5. 52.54 Kaohsiung 1. 69.99 2. 63.39 3. 64.89 4. 75.79 5. 77.27 **Monitoring Stations:** 10 (5 in each city) **Notes:** All pregnant women/infants included in study lived within 3 km of an air quality monitoring station Pollution assigned based on nearest air quality station to the maternal residence **Co-pollutant:** CO, SO_2, O_3, NO_2	**PM Increment:** Tertiles Entire pregnancy T1: < 46.4 ppb T2: 46.4-63.1 ppb T3: > 63.1 ppb First trimester T1: < 45.8 ppb T2: 45.8-67.6 ppb T3: > 67.6 ppb Second trimester T1: < 44.6 ppb T2: 44.6-64.2 ppb T3: > 64.2 ppb Third trimester T1: < 43.7 ppb T2: 43.7-63.7 ppb T3: > 63.7 ppb **RR Estimate [Lower CI, Upper CI]** Entire pregnancy T1: 1.00 T2: 0.96 [0.83, 1.11] T3: 0.87 [0.71, 1.05] First trimester T1: 1.00 T2: 0.96 [0.84, 1.09] T3: 0.97 [0.80, 1.17] Second trimester T1: 1.00 T2: 1.03 [0.90, 1.17] T3: 1.00 [0.83, 1.21] Third trimester T1: 1.00 T2: 1.02 [0.90, 1.16] T3: 0.97 [0.81, 1.17] **Notes:** RR for births in Kaoshiung vs. Taipei: 1.13 [1.03, 1.24]
Reference: Lipfert et al. (2000, 004103) **Period of Study:** 1990 **Location:** U.S.	**Outcome:** Infant mortality including respiratory mortality (traditional definition, ICD9 460-519), expanded definition (adds ICD9 769 and 770) **Age Groups:** Infants **Study Design:** Cross-sectional **N:** 2,413,762 infants in 180 counties (Ns differ for various models) **Statistical Analyses:** Logistic regression **Covariates:** mother's smoking,	**Pollutant:** PM_{10} **Averaging Time:** Yearly avg used **Mean (SD):** 33.1 (9.17) (based on 180 counties) **Range (Min, Max):** (16.9, 59) **Monitoring Stations:** NR **Copollutant (correlation):** PM_{10} SO_4^{2-} (r = 0.10) $NSPM_{10}$–non-sulfate portion of PM_{10}	**PM Increment:** NR (present regression coefficients) **Effect Estimate [Lower CI, Upper CI]:** Presented regression coefficients (standard errors) (3 PM exposures regressed jointly) bold = p < 0.05 Cause of death: All Birth weight: All PM_{10}: 0.0114 (0.0015)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
	education, marital status, and race	(r = 0.91)	SO_4^{2-}: -0.0002 (0.0061)
	month of birth	CO (r = 0.27)	$NSPM_{10}$: 0.0115 (0.0014)
	and county avg heating degree days	SO_2 (r = 0.04)	Cause of death: All
	Dose-response Investigated? NR	**Notes:** TSP-based sulfate was adjusted for compatibility with the PM_{10}-based data	Birth weight: LBW
	Statistical Package: NR		PM_{10}: 0.0088 (0.0019)
			SO_4^{2-}: 0.0265 (0.0080)
			$NSPM_{10}$: 0.0086 (0.0020)
			Cause of death: All
			Birth weight: normal
			PM_{10}: 0.0092 (0.0024)
			SO_4^{2-}: -0.0488 (0.0098)
			$NSPM_{10}$: 0.0096 (0.0024)
			Cause of death: All neonatal
			Birth weight: All
			PM_{10}: 0.0126 (0.0018)
			SO_4^{2-}: 0.0267 (0.0076)
			$NSPM_{10}$: 0.0126 (0.0018)
			Cause of death: All neonatal
			Birth weight: LBW
			PM_{10}: 0.0086 (0.0022)
			SO_4^{2-}: 0.0388 (0.0088)
			$NSPM_{10}$: 0.0093 (0.0022)
			Cause of death: All neonatal
			Birth wt: normal
			PM_{10}: 0.0123 (0.0041)
			SO_4^{2-}: -0.0334 (0.0169)
			$NSPM_{10}$: 0.0125 (0.0040)
			Cause of death: All post neonatal
			Birth wt: All
			PM_{10}: 0.0091 (0.0024)
			SO_4^{2-}: -0.0474 (0.0100)
			$NSPM_{10}$: 0.0096 (0.0024)
			Cause of death: All post neonatal
			Birth wt: LBW
			PM_{10}: 0.0096 (0.0043)
			SO_4^{2-}: -0.0247 (0.0173)
			$NSPM_{10}$: 0.0101 (0.0042)
			Cause of death: All post neonatal
			Birth wt: normal
			PM_{10}: 0.0074 (0.0030)
			SO_4^{2-}: -0.0569 (0.0121)
			$NSPM_{10}$: 0.0080 (0.0029)
			Cause of death: SIDS
			Birth weight: All
			PM_{10}: 0.0138 (0.0038)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			SO_4^{2-}: -0.1078 (0.0151)
			$NSPM_{10}$: 0.0149 (0.0037)
			Cause of death: SIDS
			Birth weight: LBW
			PM_{10}: 0.0115 (0.0088)
			SO_4^{2-}: -0.1378 (0.0337)
			$NSPM_{10}$: 0.0146 (0.0085)
			Cause of death: SIDS
			Birth weight: normal
			PM_{10}: 0.0137 (0.0042)
			SO_4^{2-}: -0.0995 (0.0168)
			$NSPM_{10}$: 0.0147 (0.0041)
			Cause of death: All respiratory (ICD9: 460-519, 769, 770)
			Birth weight: All
			PM_{10}: 0.0168 (0.0034)
			SO_4^{2-}: 0.0706 (0.0146)
			$NSPM_{10}$: 0.0166 (0.0034)
			Cause of death: All respiratory (ICD9: 460-519, 769, 770)
			Birth weight: LBW
			PM_{10}: 0.0144 (0.0038)
			SO_4^{2-}: 0.0821 (0.0158)
			$NSPM_{10}$: 0.0139 (0.0038)
			Cause of death: All respiratory (ICD9: 460-519, 769, 770)
			Birth weight: normal
			PM_{10}: 0.0177 (0.0091)
			SO_4^{2-}: 0.0001 (0.0392)
			$NSPM_{10}$: 0.0118 (0.0090)
			Cause of death: Respiratory disease (ICD9: 460-519)
			Birth weight: All
			PM_{10}: 0.0133 (0.0089)
			SO_4^{2-}: 0.0093 (0.0384)
			$NSPM_{10}$: 0.0134 (0.0089)
			Cause of death: Respiratory disease (ICD9: 460-519)
			Birth weight: LBW
			PM_{10}: 0.0092 (0.0137)
			SO_4^{2-}: 0.0434 (0.0580)
			$NSPM_{10}$: 0.0089 (0.0138)
			Cause of death: Respiratory disease (ICD9: 460-519)
			Birth weight: normal
			PM_{10}: 0.0126 (0.0120)
			SO_4^{2-}: -0.0177 (0.0509)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			$NSPM_{10}$: 0.0128 (0.0119)
			Associations with SIDS by smoking status
			Smoking status: Yes
			Birth weight: Normal
			PM_{10}: 0.0202 (0.0073)
			SO_4^{2-}: -0.0722 (0.0284)
			$NSPM_{10}$: 0.0206 (0.0071)
			Smoking status: No
			Birth weight: Normal
			PM_{10}: 0.0104 (0.0051)
			SO_4^{2-}: -0.114 (0.021)
			$NSPM_{10}$: 0.0117 (0.005)
			Smoking status: Yes
			Birth weight: LBW
			PM_{10}: 0.0322 (0.0130)
			SO_4^{2-}: -0.0958 (0.0483)
			$NSPM_{10}$: 0.0345 (0.0125)
			Smoking status: No
			Birth weight: LBW
			PM_{10}: -0.0044 (0.012)
			SO_4^{2-}: -0.0172 (0.047)
			$NSPM_{10}$: -0.0007 (0.012)
			Mean risks (95%CI) between post neonatal SIDS among normal birth weight babies
			pollutants regressed one at a time
			PM_{10}: 1.20 (1.02, 1.42)
			SO_4^{2-}: 0.43 (0.37, 0.51)
			$NSPM_{10}$: 1.33 (1.18, 1.50)
Reference: Maisonet et al. (2001, 016624) **Period of Study:** 1994-1996 **Location:** Northeastern U.S. (6 cities: Boston, Hartford, Philadelphia, Pittsburgh, Springfield, Washington DC)	**Outcome:** Low birth weight (LBW): infants with a birth weight < 2,500 g and having a gestational age between 37 and 44 weeks **Age Groups:** Term live births (singleton) **Study Design:** Cross-sectional **N:** 89,557 infants **Statistical Analyses:** Logistic regression (LBW) and linear regression (for reductions in birth weight) **Covariates:** gestational age, gender, birth order, maternal age, race/ethnicity, years of education, marital status, adequacy of prenatal care, previous induced or spontaneous abortions, weight gain during pregnancy, maternal prenatal smoking, and alcohol consumption season **Season:** Yes, as covariate **Dose-response Investigated?** Yes, categorical exposure variables assessed	**Pollutant:** PM_{10} **Averaging Time:** Trimester averages calculated using 24-h measurements taken every 6 days **Range (Min, Max):** Ranges for categories of exposure: 1st Trimester < 25th: < 24.821 25 to < 50th: 24.821, 30.996 50 to < 75th: 30.997, 36.142 75 to < 95th: 36.143, 46.547 ≥ 95th: ≥ 46.548 2nd Trimester < 25th: < 24.702 25 to < 50th: 24.702, 30.294 50 to < 75th: 30.295, 35.410 75 to < 95th: 35.411, 43.928	**PM Increment:** 10 $\mu g/m^3$ for analyses assessing exposures continuously **Effect Estimate [Lower CI, Upper CI]:** ORs for term LBW by trimester 1st Trimester Crude < 25th: 1.00 25 to < 50th: 1.02 (0.90, 1.14) 50 to < 75th: 0.90 (0.65, 1.24) 75 to < 95th: 0.87 (0.58, 1.30) ≥ 95th: 0.89 (0.60, 1.33) Continuous: 0.93 (0.77, 1.13) 1st Trimester Adjusted < 25th: 1.00 25 to < 50th: 1.02 (0.94, 1.11) 50 to < 75th: 0.90 (0.78, 1.03) 75 to < 95th: 0.85 (0.73, 1.00) ≥ 95th: 0.83 (0.70, 0.97)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
	Statistical Package: STATA	≥ 95th: ≥ 43.929	Continuous: 0.93 (0.85, 1.00)
		3rd Trimester	2nd Trimester Crude
		< 25th: < 24.702	< 25th: 1.00
		25 to < 50th: 24.702, 30.162	25 to < 50th: 1.01 (0.93, 1.10)
		50 to < 75th: 30.163, 35.642	50 to < 75th: 0.90 (0.66, 1.21)
		75 to < 95th: 35.643, 43.588	75 to < 95th: 0.92 (0.62, 1.34)
		≥ 95th: ≥ 43.589	≥ 95th: 0.90 (0.61, 1.33)
		Monitoring Stations: 3-4 per city	Continuous: 0.95 (0.78, 1.16)
		Copollutants: CO, SO_2	2nd Trimester Adjusted
			< 25th: 1.00
			25 to < 50th: 1.06 (0.97, 1.15)
			50 to < 75th: 0.95 (0.85, 1.07)
			75 to < 95th: 0.91 (0.79, 1.05)
			≥ 95th: 0.77 (0.63, 0.95)
			Continuous: 0.93 (0.85, 1.02)
			3rd Trimester Crude
			< 25th: 1.00
			25 to < 50th: 0.94 (0.85, 1.05)
			50 to < 75th: 0.86 (0.58, 1.25)
			75 to < 95th: 0.86 (0.57, 1.29)
			≥ 95th: 0.92 (0.61, 1.38)
			Continuous: 0.95 (0.75, 1.20)
			3rd Trimester Adjusted
			< 25th: 1.00
			25 to < 50th: 0.98 (0.87, 1.10)
			50 to < 75th: 0.92 (0.76, 1.11)
			75 to < 95th: 0.88 (0.75, 1.04)
			≥ 95th: 0.91 (0.77, 1.07)
			Continuous: 0.96 (0.88, 1.06)
			Adjusted ORs by race/ethnicity
			Whites:
			1st Trimester
			< 25th: 1.00
			25 to < 50th: 1.13 (0.96, 1.33)
			50 to < 75th: 1.00 (0.92, 1.08)
			75 to < 95th: 1.00 (0.91, 1.09)
			≥ 95th: 0.92 (0.81, 1.04)
			Continuous: 0.94 (0.90, 0.98)
			2nd Trimester
			< 25th: 1.00
			25 to < 50th: 0.88 (0.77, 1.02)
			50 to < 75th: 0.95 (0.89, 1.02)
			75 to < 95th: 0.95 (0.84, 1.07)
			≥ 95th: 0.89 (0.64, 1.26)
			Continuous: 0.96 (0.89, 1.04)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			3rd Trimester
			< 25th: 1.00
			25 to < 50th: 0.84 (0.64, 1.11)
			50 to < 75th: 0.91 (0.83, 1.01)
			75 to < 95th: 0.80 (0.71, 0.90)
			≥ 95th: 1.03 (0.86, 1.24)
			Continuous: 0.95 (0.90, 1.00)
			African Americans:
			1st Trimester
			< 25th: 1.00
			25 to < 50th: 1.01 (0.98, 1.05)
			50 to < 75th: 0.88 (0.79, 0.98)
			75 to < 95th: 0.83 (0.70, 0.97)
			≥ 95th: 0.81 (0.67, 0.99)
			Continuous: 0.93 (0.85, 1.01)
			2nd Trimester
			< 25th: 1.00
			25 to < 50th: 1.10 (0.93, 1.30)
			50 to < 75th: 0.95 (0.80, 1.12)
			75 to < 95th: 0.88 (0.69, 1.11)
			≥ 95th: 0.75 (0.54, 1.03)
			Continuous: 0.92 (0.80, 1.05)
			3rd Trimester
			< 25th: 1.00
			25 to < 50th: 1.08 (0.92, 1.27)
			50 to < 75th: 0.89 (0.70, 1.12)
			75 to < 95th: 0.94 (0.75, 1.18)
			≥ 95th: 0.83 (0.71, 0.97)
			Continuous: 0.99 (0.87, 1.11)
			Hispanics:
			1st Trimester
			< 25th: 1.00
			25 to < 50th: 0.83 (0.64, 1.06)
			50 to < 75th: 0.86 (0.70, 1.05)
			75 to < 95th: 0.79 (0.68, 0.93)
			≥ 95th: 1.36 (1.06, 1.75)
			Continuous: 0.96 (0.84, 1.09)
			2nd Trimester
			< 25th: 1.00
			25 to < 50th: 1.16 (0.84, 1.61)
			50 to < 75th: 0.86 (0.63, 1.19)
			75 to < 95th: 0.98 (0.71, 1.34)
			≥ 95th: 0.68 (0.38, 1.21)
			Continuous: 0.92 (0.81, 1.05)
			3rd Trimester

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			< 25th: 1.00 25 to < 50th: 0.77 (0.55, 1.07) 50 to < 75th: 1.12 (0.76, 1.66) 75 to < 95th: 0.93 (0.65, 1.31) ≥ 95th: 0.90 (0.55, 1.47) Continuous: 0.96 (0.80, 1.15)
Reference: Mannes et al.(2005, 087895) **Period of Study:** January 1, 1998-December 31, 2000 **Location:** Metropolitan Sydney, Australia	**Outcome:** Risk of SGA and birth weight **Age Groups:** all singleton births > 20 weeks and ≥ 400 grams birth weight and maternal all ages **Study Design:** Cross-sectional **N:** 138,056 singleton births **Statistical Analyses:** Logistic and linear regression models **Covariates:** sex of child, maternal age, gestational age, maternal smoking, gestational age at first antenatal visit, maternal indigenous status, whether first pregnancy, season of birth, socioeconomic status **Season:** All seasons included as covariate **Dose-response Investigated?** No **Statistical Package:** SAS v8.02	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (SD):** 16.8 (7.1) **25th:** 12.3 **50th(Median):** 15.7 **75th:** 19.9 **Range (Min, Max):** (3.8-104.0) **Monitoring Stations:** up to 14 **Copollutants (correlations):** CO: r = 0.26 NO_2: r = 0.47 O_3: r = 0.52 $PM_{2.5}$: r = 0.81	**PM Increment:** 1 $\mu g/m^3$ Risk of SGA All births One month before birth: OR = 1.01 (1.00-1.03) Third trimester: OR = 1.00 (0.99-1.013) Second trimester: OR = 1.01 (1.00-1.04) First trimester: OR = 1.00 (0.98-1.02) 5 km births One month before birth: OR = 1.00 (0.99-1.02) Third trimester: OR = 1.01 (0.99-1.02) Second trimester: OR = 1.02 (1.01-1.03) First trimester: OR = 1.01 (0.99-1.02) Change in birth weight All births One month before birth: ß = -1.21 (-2.31- -0.11) Third trimester: ß = -0.95 (-2.30–0.40) Second trimester: ß = -2.05 (-3.36- -0.74) First trimester: ß = -0.14 (-1.37- 1.09) 5 km births One month before birth: ß = -2.98 (-4.25- -1.71) Third trimester: ß = -3.84 (-5.35- -2.33) Second trimester: ß = -4.28 (-5.79- -2.77) First trimester: ß = -2.57 (-4.04- -1.10) Key second trimester findings Single pollutant model: ß = -4.28 (-5.79- -2.77) 2 pollutant (PM_{10} and CO): ß = -3.72 (-6.29- -1.15) 2 pollutant (PM_{10} and NO_2): ß = -2.65 (-4.32- -0.98) 2 pollutant (PM_{10} and O_3): ß = -5.47 (-7.06- -3.88) 4 pollutant (PM_{10}, NO_2, CO and O_3): ß = -3.27 (-7.05-0.51) Controlling for exposures in other pregnancy periods: ß = -3.03 (-4.85- -1.21)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Pereira et al. (1998, 007264) **Period of Study:** Jan 1991–Dec 1992 **Location:** Sao Paulo, Brazil **Notes:** Paper does not focus on PM as a pollutant of interest.	**Outcome:** Intrauterine mortality (fetuses over 28 weeks of pregnancy) **Study Design:** Time-series **N:** 730 days with PM measures **Statistical Analyses:** Poisson regression **Covariates:** Season, day of the week and weather (temperature and relative humidity) **Season:** Assessed by including 24 indicator variables for month and year **Dose-response Investigated?** No **Statistical Package:** NR **Lags Considered:** Paper focuses on other pollutants (lags for PM not reported)	**Pollutant:** PM_{10} **Averaging Time:** 24 hr mean **Mean (SD):** 65.04 (27.28) **Range (Min, Max):** (14.80, 192.80) **Monitoring Stations:** 13 (averaged to provide city-wide pollutant level) **Copollutants (correlation):** NO_2 (r = 0.45) SO_2 (r = 0.74) CO (r = 0.41) O_3 (r = 0.25)	**PM Increment:** NR (reported only regression coefficients for PM) **Effect Estimate [Lower CI, Upper CI]:** Regression coefficients (standard errors) for pollutants when considered separately and simultaneously in the completed model: Separately: 0.0008 (0.0006) Simultaneously: -0.0005 (0.0010)
Reference: Ritz et al. (2000, 012068) **Period of Study:** 1989-1993 **Location:** Southern California	**Outcome:** Preterm birth (treated dichotomously as birth at < 37 weeks gestation also analyzed continuously) **Age Groups:** infants (born vaginally between 26-44 weeks of gestation) **Study Design:** Cross-sectional **N:** 97,158 births **Statistical Analyses:** Logistic and linear regression **Covariates:** maternal age, race, education, parity, interval since the previous live birth, access to prenatal care, infant sex, previous low weight or preterm births, smoking (reported as "pregnancy complications") to examine effect modification, authors conducted stratified analysis by region, birth and conception seasons, maternal age, race, education, and infant gender **Season:** Some models included season of birth or conception also assessed as effect modifier in stratified analyses **Dose-response Investigated?** Examined adequacy of linear or log-linear relation using indicator terms for pollutant-avg quartiles results presented in Fig 2 (dose-response demonstrated for last 6 weeks exposure period) **Statistical Package:** NR	**Pollutant:** PM_{10} **Averaging Time:** 24-h averages at 6 day intervals averaged pollutant measures for 1, 2, 4, 6, 8, 12, and 26 weeks before birth and the whole pregnancy period **Mean (SD):** 6 weeks before birth: 47.5 (15.0) 1st month of pregnancy: 49.3 (16.9) **Range (Min, Max):** 6 weeks before birth: 12.3-152.3 1st month of pregnancy: 9.5-178.8 **Monitoring Stations:** 17 stations (PM measured at only 8 stations) **Copollutants (correlations):** 6 weeks before birth: CO (r = 0.43) NO_2 (r = 0.74) O_3 (r = 0.20) 1st month of pregnancy: CO (r = 0.37) NO_2 (r = 0.71) O_3 (r = 0.23) **Notes:** Averaged pollutant measures taken at the air monitoring station closest to the residence	**PM Increment:** 50 $\mu g/m^3$ **Effect Estimate [Lower CI, Upper CI]:** **All 8 stations** 6 weeks before birth Crude: 1.20 (1.09, 1.33) 2 exposure periods: 1.18 (1.07, 1.31) Other risk factors: 1.15 (1.04, 1.26) Other RFs plus season: 1.15 (1.03, 1.29) Multipollutant model: 1.19 (1.01, 1.40) 1st month of pregnancy Crude: 1.16 (1.06, 1.26) 2 exposure periods: 1.13 (1.04, 1.24) Other risk factors: 1.09 (1.00, 1.19) Other RFs plus season: 1.09 (0.99, 1.20) Multipollutant model: 1.12 (0.97, 1.29) **Coastal stations only** 6 weeks before birth Crude: 1.22 (1.00, 1.49) 2 exposure periods: 1.28 (1.04, 1.56) Other risk factors: 1.13 (0.93, 1.38) Other RFs plus season: 1.18 (0.92, 1.51) Multipollutant model: 1.42 (097, 2.01) 1st month of pregnancy Crude: 1.28 (1.06, 1.54) 2 exposure periods: 1.32 (1.09, 1.59) Other risk factors: 1.17 (0.97, 1.40) Other RFs plus season: 0.99 (0.79, 1.24) Multipollutant model: 1.09 (0.83, 1.41) **Inland stations only** 6 weeks before birth Crude: 1.27 (1.12, 1.44) 2 exposure periods: 1.27 (1.11, 1.44)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			Other risk factors: 1.19 (1.05, 1.35)
			Other RFs plus season: 1.27 (1.10, 1.48)
			Multipollutant model: 1.18 (0.97, 1.43)
			1st month of pregnancy
			Crude: 1.16 (1.04, 1.29)
			2 exposure periods: 1.16 (1.04, 1.29)
			Other risk factors: 1.09 (0.98, 1.21)
			Other RFs plus season: 1.09 (0.97, 1.24)
			Multipollutant model: 1.11 (0.93, 1.33)
			Crude estimates for last 6 weeks exposure by season
			Fall: 1.08 (0.88, 1.31)
			Summer: 1.06 (0.87, 1.29)
			Winter: 1.33 (1.07, 1.65)
			Spring: 1.81 (1.41, 2.31)
			Reduction in mean gestation length for each increase in PM_{10} during last 6 weeks before birth (linear regression analysis)
			Crude: 0.66 (± 0.24) days
			Adj: 0.90 (± 0.27) days
			Notes: Effect estimates remain stable when excluding SGA or LBW children or when restricting preterm births to SGA or LBW children only (results not presented)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Ritz, et al. (2002, 023227) **Period of Study:** 1987-1993 **Location:** Southern California (July 1990–July 1993 for Los Angeles, 1989 for Riverside, 1988–1989 for San Bernardino, and 1987–1989 for Orange counties	**Outcome:** 1) aortic defects 2) defects of the atrium and atrium septum 3) endocardial and mitral valve defects 4) pulmonary artery and valve defects 5) conotruncal defects including tetralogy of Fallot, transposition of great vessels, truncus arteriosus communis, double outlet right ventricle, and aorticopulmonary window and 6) ventricular septal defects not included in the conotruncal category. **Age Groups:** all live born infants and fetal deaths diagnosed between 20 weeks of gestation and 1 year after birth **Study Design:** case-control **N:** 10,649 infants and fetuses **Statistical Analyses:** hierarchical (two-level) regression model, polytomous logistic regression, linear model **Covariates:** gender, no prenatal care, multiple births, no siblings, maternal race, maternal age, maternal education, born before 1990, season of conception, **Season:** all **Dose-response Investigated?** Yes, for ozone and CO, study found a clear dose-response pattern for aortic septum and valve and ventricular septal defects and possibly for conotruncal and pulmonary artery and valve defects **Statistical Package:** SAS	**Pollutant:** PM_{10} **Averaging Time:** 24 h (every 6 days) **PM Component:** vehicle emissions **Monitoring Stations:** 11 (for PM_{10}) **Copollutants (correlations):** CO: r = 0.32 NO_2(NR) O_3 (NR)	**Notes:** The authors did not observe consistently increased risks and dose-response patterns for PM_{10} after controlling for the effects of CO and ozone on these cardiac defects. (Quantitative results not shown).
Reference: Ritz et al. (2006, 089819) **Period of Study:** 1989-2000 **Location:** 389 South Coast Air Basin (SoCAB) zip codes	**Outcome:** total infant deaths during the first year of life as well as all respiratory causes of death (ICD-9 codes 460-519, 769, 770.4, 770.7, 770.8, and 770.9 and ICD-10 codes J00-J98, P22.0, P22.9, P27.1, P27.9, P28.0, P28.4, P28.5, and P28.9) and sudden infant death syndrome (SIDS) (ICD-9 code 798.0 and ICD-10 code R95). **Age Groups:** infants 0-1 yr **Study Design:** Case-control **N:** 2,975,059 births and 19,664 infant deaths Cases, n = 13,146 Controls, n = 151,015 **Statistical Analyses:** Conditional logistic regression analysis **Covariates:** risk factors available on birth and/or death certificates (maternal age, race/ethnicity, and education, level of prenatal care, infant gender, parity, birth country, and death season) **Season:** Death season (spring, summer, autumn, winter) **Dose-response Investigated?** Yes **Statistical Package:** NR	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (SD):** Two weeks before death: 46.2 One month before death: 46.3 Two months before death: 46.3 Six months before death: 46.3 **Range (Min, Max):** Two weeks before death: (21.0-83.5) One month before death: (25.0-77.2) Two months before death: (27.6-74.2) Six months before death: (31.3-69.5) **Monitoring Stations:** maximum of 31 **Copollutants (correlation):** Two weeks before death CO: r = 0.33 NO_2: r = 0.48 O_3: r = 0.12 One month before death CO: r = 0.33 NO_2: r = 0.48 O_3: r = 0.12	**PM Increment:** 10 $\mu g/m^3$ **Effect Estimate [Lower CI, Upper CI]:** **All-cause death** **2 mo before death** Single-pollutant model: $<25^{th}$ = 1.04 (1.01-1.06) 25^{th}-75^{th} = 0.96 (0.89-1.04) $>75^{th}$ = 1.14 (1.03-1.27) Multiple-pollutant model: $<25^{th}$ = 1.02 (0.99-1.05) 25^{th}-75^{th} = 0.92 (0.84-1.00) $>75^{th}$ = 1.07 (0.95-1.20) **SIDS** **2 mo before death:** Single-pollutant model: $<25^{th}$ = 1.03 (0.99-1.08) 25^{th}-75^{th} = 0.94 (0.81-1.08) $>75^{th}$ = 1.13 (0.93-1.36) Multiple-pollutant model: $<25^{th}$ = 1.01 (0.95-1.07) 25^{th}-75^{th} = 0.90 (0.76-1.06)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
		Two months before death	$>75^{th}$ = 0.99 (0.80-1.24)
		CO: r = 0.32	**Respiratory death**
		NO_2: r = 0.48	**2 wk before death**
		O_3: r = 0.12	**Postneonatal deaths (28 d to 1 y)**
		Six months before death	Single-pollutant model:
		CO: r = 0.29	$<25^{th}$ = 1.05 (1.01-1.10)
		NO_2: r = 0.44	25^{th}-75^{th} = 1.13 (1.01-1.10)
		O_3: r = 0.16	$>75^{th}$ = 1.46 (1.13-1.88)
			Multiple-pollutant model:
			$<25^{th}$ = 1.04 (0.98-1.09)
			25^{th}-75^{th} = 1.09 (0.86-1.38)
			$>75^{th}$ = 1.40 (1.03-1.89)
			Postneonatal deaths (28 d to 3 mo)
			Single-pollutant model:
			$<25^{th}$ = 1.01 (0.95-1.08)
			25^{th}-75^{th} = 1.16 (0.82-1.63)
			$>75^{th}$ = 1.44 (0.96-2.17)
			Multiple-pollutant model:
			$<25^{th}$ = 1.00 (0.92-1.09)
			25^{th}-75^{th} = 0.97 (0.67-1.42)
			$>75^{th}$ = 1.23 (0.76-2.00)
			Post neonatal deaths (4-12 mo)
			Single-pollutant model:
			$<25^{th}$ = 1.12 (1.02-1.23)
			25^{th}-75^{th} = 1.08 (0.81-1.44)
			$>75^{th}$ = 1.41 (1.02-1.96)
			Multiple-pollutant model:
			$<25^{th}$ = 1.07 (1.00-1.15)
			25^{th}-75^{th} = 1.02 (0.75-1.40)
			$>75^{th}$ = 1.36 (0.92-2.01)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Rogers et al. (2006, 091232)	**Outcome:** VLBW	**Pollutant:** PM_{10}	**PM Increment:** Quartile
Period of Study: 1986-1988	Term, AGA, Preterm AGA, Preterm, SGA	**Averaging Time:** annual	**Notes:** Statistically significant increases in the odds of VLBW and preterm AGA births are associated with living in a county with a PM_{10} point source. Preterm AGA births are also associated with living in an area with very high (4th quartile) estimated PM_{10} exposure.
Location: Georgia, USA	**Age Groups:** Newborns and their mothers (< 19 to ≥ 35-years-old)	Preterm SGA:	
	Study Design: case-control	**50th(Median):** 3.38	
	N: 325 infants (69 preterm SGA	Preterm AGA:	
	59 preterm AGA	**50th(Median):** 7.84	
	197 term AGA) and their mothers	Term AGA:	
	Statistical Analyses: logistic regression	**50th(Median):** 3.23	
	Covariates: maternal age, maternal race, maternal education, active and passive smoking, birth season, prepregnancy weight, pregnancy weight gain, maternal toxemia, anemia, asthma	**Monitoring Stations:** NR	
		Percent Mothers Residing In County With Industrial Point Source	
	Dose-response Investigated? Yes, used	Preterm SGA: 60.9%	
	Statistical Package: SUDAAN	Preterm AGA: 79.7%	
	Cochran-Armitage test for trend to determine whether the observed proportions of cases and controls differed in a linear manner across exposure categories.	Term AGA: 60.4%	
		Percent Mothers Residing In Pm_{10} Quartile (based on environmental transport model)	
		Preterm SGA	
		1st quartile (< 1.48): 31.9%	
		2nd quartile (1.48-3.74): 18.8%	
		3rd quartile (3.75-15.07): 26.1%	
		4th quartile (> 15.07): 23.2%	
		Preterm AGA	
		1st quartile (< 1.48): 16.9%	
		2nd quartile (1.48-3.74): 22.1%	
		3rd quartile (3.75-15.07): 28.8%	
		4th quartile (> 15.07): 32.2%	
		Term AGA	
		1st quartile (< 1.48): 24.7%	
		2nd quartile (1.48-3.74): 28.4%	
		3rd quartile (3.75-15.07): 27.9%	
		4th quartile (> 15.07): 19.3%	
			Delivery of VLBW vs. Term AGA infant
			County with point source
			2.54 [1.46, 4.22]
			PM_{10} quartile
			1st quartile: reference
			2nd quartile:
			0.81 [0.42, 1.55]
			3rd quartile:
			0.85 [0.45, 1.16]
			4th quartile:
			1.94 [0.98, 3.83]
			Delivery of Preterm AGA vs. Term AGA infant
			County with point source
			4.31 [1.88: 9.87]
			PM_{10} quartile
			1st quartile: reference
			2nd quartile:
			1.56 [0.56: 4.35]
			3rd quartile:
			1.19 [0.44: 3.23]
			4th quartile:
			3.68 [1.44: 9.44]
			Delivery of Preterm AGA vs. Preterm SGA infant
			County with point source
			2.07 [0.83: 5.16]
			PM_{10} quartile
			1st quartile: reference
			2nd quartile:
			1.96 [0.59: 6.43]
			3rd quartile:
			2.10 [0.66: 6.73]
			4th quartile:
			2.58 [0.78: 8.51]

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Romieu et al. (2004, 093074)	**Outcome:** Respiratory-related infant mortality ICD9 (460–519)	**Pollutant:** PM_{10}	**PM Increment:** 20 $\mu g/m^3$
Period of Study: 1997 to 2001	ICD10 (J00-J99)	**Averaging Time:** 24 h	**RR Estimate [Lower CI, Upper CI]**
Location: Ciudad Juarez, Mexico	**Age Groups:** 1 month to 1 year	**Mean (SD):** 1997: 33.04 (20.67) $\mu g/m^3$	**lag:**
	Study Design: Case crossover	1998: 35.25 (17.32) $\mu g/m^3$	**Total mortality:**
	N: 216 respiratory-related deaths	1999: 45.92 (28.69) $\mu g/m^3$	OR = 1.02 (0.94–1.11) lag 1
	N = 412 other causes and N = 628 total deaths	2000: 43.38 (23.77) $\mu g/m^3$	OR = 1.03 (0.95-1.12) lag 2
	Statistical Analyses: The acute effects of air pollution was modeled on both total and respiratory-related mortality as a function of the pollution levels on the same day and preceding days and over two- and three-day averages before the date of death. Case-crossover with semi-symmetric bidirectional referent selection was the approach used. Data were stratified by day of the week and calendar month. Data were analyzed with conditional logistic regression. Second and third polynomial distributed lag models were used to study lag structure. BIC was used to determine lag length.	2001: 39.46 (29.43) $\mu g/m^3$	OR = 1.03 (0.94-1.13 ac2
		Monitoring Stations: 5 stations in Ciudad Juarez	OR = 1.04 (0.95-1.15) ac3
		2 stations in El Paso (close to US-Mexico border)	**Respiratory mortality**
		Copollutant (correlation): O_3: r = 0.01	OR = 0.95 (0.83-1.09) lag 1
		Notes: Ciudad Juarez monitors measured PM_{10} every 6 days while El Paso monitors measured on a daily basis.	OR = 1.04 (0.91-1.19) lag 2
			OR = 0.98 (0.81-1.19) ac2
			OR = 0.97 (0.74-1.26) ac3
			Higher SES
			OR = 0.82 (0.59, 1.14) lag 1
			OR = 1.08 (0.84, 1.40) lag 2
			OR = 0.89 (0.58, 1.35) ac2
			OR = 0.97 (0.52, 1.82) ac3
			Medium SES
			OR = 0.99 (0.79, 1.27) lag 1
			OR = 1.11 (0.86, 1.43) lag 2
			OR = 1.03 (0.73, 1.45) ac2
			OR = 1.17 (0.72, 1.90) ac3
			Lower SES
			OR = 1.61 (0.97-2.66) lag 1
			OR = 1.07 (0.65, 1.75) lag 2
			OR = 2.56 (1.06-6.17) ac2
			OR = 1.76 (0.59, 5.23) ac3
			Notes:
			ac2 and ac3 represent cumulative PM_{10} ambient levels over two or three days before death.

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Sagiv et al. (2005, 087468) **Period of Study:** 1/1/1997–12/31/2001 **Location:** Allegheny county, Beaver county, Lackawanna county, Philadelphia county, Pennsylvania, U.S.A.	**Outcome:** Preterm birth (< 36 weeks) **Age Groups:** Babies born between 20 and 44 weeks **Study Design:** Time series **N:** 3704 observation days, 187,997 births **Statistical Analyses:** Poisson regression multivariable mixed-effects model with a random intercept for each county to incorporate count-level information. **Covariates:** Temperature, dew point temperature, mean 6-week level of copollutants (CO, NO_2, and SO_2), long-term preterm birth trends **Season:** All **Dose-response Investigated?** Yes **Statistical Package:** NR **Lags Considered:** 1, 2, 3, 4, 5, 6, 7	**Pollutant:** PM_{10} **Averaging Time:** Daily used to calculate 6-week period **Mean (SD):** 6-week period 27.1 (8.3) Daily 25.3 (14.6) **Percentiles:** 6-week period **50th (Median):** 26.0 Daily **50th (Median):** 21.6 **Range (Min, Max):** 6-week period: 8.7, 68.9 Daily: 2.0, 156.3 **Monitoring Stations:** NR **Copollutant (correlation):** Daily PM_{10}–daily SO_2: r = 0.46 Also considered CO, NO_2 and O_3 as copollutants.	**PM Increment:** 1) 50 $\mu g/m^3$ 2) Quartiles (first quartile is the reference) **Exposure period: 6 weeks before birth** Per 50 $\mu g/m^3$: 1.07 (0.98, 1.18) 2nd quartile: 1.00 (0.95, 1.05) 3rd quartile: 1.04 (0.99, 1.09) 4th quartile: 1.03 (0.98, 1.08) **Exposure period: 1-day acute time windows** Per 50 $\mu g/m^3$: 2-day lag: 1.10 (1.00, 1.21) 5-day lag: 1.07 (0.98, 1.18) **Notes:** Within the article, authors provide a Figure 1 displaying a graph of the relative risk (RR) and 95% confidence intervals (CI) for 1- to 7-day lags. While the authors report the 2- and 5-day lag RRs and 95% CIs in the text, the others are not specifically reported. However, the figure shows the approximate RRs per 50 $\mu g/m^3$ as indicated below: 1-day lag: 1.05 3-day lag: 1.05 4-day lag: 1.00 6-day lag: 0.97 7-day lag: 1.03

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: (Salam et al., 2005, 087885)	**Outcome:** Birth weight	**Pollutant:** PM_{10}	**PM Increment:** IQR (interquartile range)
Period of Study: 1975–1987	Low birth weight (LBW	**Averaging Time:** Monthly	**Outcome: birth weight (g)**
Location: Southern California	$<$2500 g)	**Mean (SD):** Entire pregnancy: 45.8 (12.9)	Single-pollutant model
	Intrauterine growth retardation (IUGR)	First trimester: 46.6 (15.9)	Entire pregnancy
	Age Groups: Children born full-term (between 37 and 44 weeks)	Second trimester: 45.4 (14.8)	18 $\mu g/m^3$: -19.9 (-43.6, 3.8)
	Study Design: Cohort study	Third trimester: 45.4 (15.5)	First trimester
	N: 3901 children	**Monitoring Stations:** 1 or 3 (See notes)	20 $\mu g/m^3$: -3.0 (-22.7, 16.7)
	Statistical Analyses: Linear mixed-effects	**Copollutant (correlation):** Entire pregnancy	Second trimester
	Logistic regression	PM_{10}-$O_{3[10-6]}$: r = 0.54	19 $\mu g/m^3$: -15.7 (-36.1, 4.7)
	Covariates: Maternal age, months since last live birth, parity, maternal smoking during pregnancy, SES, marital status at childbirth, gestational diabetes, child's sex, child's race/ethnicity, child's grade in school (4th, 7th, and 10th), Julian day of birth	PM_{10}-$O_{3[24\ hr]}$: r = 0.20	Third trimester
		PM_{10}-NO_2: r = 0.55	20 $\mu g/m^3$: -21.7 (-42.2 to -1.1)
		PM_{10}-CO: r = 0.41	Multipollutant model
		First trimester	(included O_3
		PM_{10}-$O_{3[10-6]}$: r = 0.54	(24 hr) in model
	Season: All	PM_{10}-$O_{3[24\ hr]}$: r = 0.34	third trimester exposure)
	Dose-response Investigated? Yes	PM_{10}-NO_2: r = 0.48	20 $\mu g/m^3$: -10.8 (-31.8, 10.2)
	Statistical Package: SAS	PM_{10}-CO: r = 0.29	**Outcome: IUGR (ORs)**
		Second trimester	Single-pollutant model
		PM_{10}-$O_{3[10-6]}$: r = 0.50	Entire pregnancy
		PM_{10}-$O_{3[24\ hr]}$: r = 0.27	18 $\mu g/m^3$: 1.1 (0.9, 1.3)
		PM_{10}-NO_2: r = 0.53	First trimester
		PM_{10}-CO: r = 0.35	20 $\mu g/m^3$: 1.0 (0.9, 1.2)
		Third trimester	Second trimester
		PM_{10}-$O_{3[10-6]}$: r = 0.52	19 $\mu g/m^3$: 1.0 (0.9, 1.2)
		PM_{10}-$O_{3[24\ hr]}$: r = 0.31	Third trimester
		PM_{10}-NO_2: r = 0.52	20 $\mu g/m^3$: 1.1 (0.9, 1.3)
		PM_{10}-CO: r = 0.37	**Outcome: LBW**
		Notes: Exposure estimates were calculated by spatially interpolated monthly averages which were based off of three monitoring stations located within 50 km of the ZIP code region of maternal birth residences.	Single-pollutant model
			Entire pregnancy
			18 $\mu g/m^3$: 1.3 (0.8, 2.2)
			First trimester
			20 $\mu g/m^3$: 1.0 (0.7, 1.5)
			Second trimester
			19 $\mu g/m^3$: 1.2 (0.8, 1.7)
			Third trimester
			20 $\mu g/m^3$: 1.3 (0.9, 1.9)
			Notes: Numbers reported for birth weight outcome are the effects on birth weight outcome (the change in birth weight in grams) across the IQR (which vary depending on air pollutant and duration of exposure measurement).

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: (Sokol et al., 2006, 098539) **Period of Study:** 1/1996-12/1998 **Location:** Los Angeles, California	**Outcome:** Semen Quality **Study Design:** Panel **Statistical Analysis:** Univariate and Multivariate Regression **Statistical Package:** SAS **Age Groups:** Males ranging 19-35 in age	**Pollutant:** PM_{10} **Averaging Time:** 0-9d, 10-14d and 70-90d **Mean (SD) Unit:** 35.74 ± 13.83 $\mu g/m^3$ **Copollutant (correlation):** O_3, NO_2, CO	PM_{10} specific results are given in Figures 3-5. PM_{10} was not significantly correlated with sperm quality.
Reference: (Suh et al., 2007, 157028) **Period of Study:** 2001-2004 **Location:** Seoul, Korea	**Outcome:** Birth weight **Age Groups:** prenatal follow-up for newborns **Study Design:** based prospective cohort study **N:** 199 pregnant mothers **Statistical Analyses:** ANCOVA, generalized linear models **Covariates:** infant's sex, maternal age, maternal and paternal education, parity, presence of illness during pregnancy, delivery month, gestational age (squared) **Dose-response Investigated?** Yes **Statistical Package:** SAS	**Pollutant:** PM_{10} **Averaging Time:** 24-h **Mean (SD):** 1st trimester: 76.41 (28.80) 2nd trimester: 77.84 (31.63) 3rd trimester: 95.61 (26.15) **Percentiles:** 1st trimester **25th:** 55.28 **50th(Median):** 71.09 **75th:** 92.38 2nd trimester **25th:** 48.65 **50th(Median):** 72.36 **75th:** 108.00 3rd trimester **25th:** 77.10 **50th(Median):** 96.35 **75th:** 116.68 **Range (Min, Max):** 1st trimester (21.00, 151.65) 2nd trimester (31.45, 139.13) 3rd trimester (23.45, 172.75) **Monitoring Stations:** 27 **Copollutant:** CO SO_2 NO_2	**PM Increment:** Trimester ≥ 90th%ile compared to < 90th%ile Least-square (ANCOVA) mean (SE) **All Genotypes** 1st trimester < 90th%ile, N(%): 158 (90.3%): 3253 (37) ≥ 90th%ile, N(%): 17 (9.7%): 2841 (145) p-Value for mean birth weight for ≥ 90th%ile PM_{10} vs. for < 90th%ile PM_{10} Adjusted: 0.009 Adjusted, with CO: 0.041 Adjusted, with NO_2: 0.092 Adjusted, with SO_2: 0.012 2nd trimester < 90th%ile, N(%): 153 (89.5%): 3253 (39) ≥ 90th%ile, N(%): 18 (10.5%): 3026 (157) p-Value for mean birth weight for ≥ 90th%ile PM_{10} vs. for < 90th%ile PM_{10} Adjusted: 0.177 Adjusted, with CO: 0.203 Adjusted, with NO_2: 0.151 Adjusted, with SO_2: 0.151 3rd trimester < 90th%ile, N(%): 162 (90.5%): 3226 (38) ≥ 90th%ile, N(%): 17 (9.5%): 3122 (140) p-Value for mean birth weight for ≥ 90th%ile PM_{10} vs. for < 90th%ile PM_{10} Adjusted: 0.487 Adjusted, with CO: 0.748 Adjusted, with NO_2: 0.420 Adjusted, with SO_2: 0.466 **Genotype** MspI TT 1st trimester < 90th%ile, N(%): 60 (34.3%): 3350 (64) ≥ 90th%ile, N(%): 5 (2.9%): 3001 (229) p-Value for mean birth weight for ≥ 90th%ile PM_{10} vs. for < 90th%ile PM_{10} Adjusted: 0.147 Adjusted, with CO: 0.186

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			Adjusted, with NO_2: 0.430
			Adjusted, with SO_2: 0.155
			2nd trimester
			< 90th%ile, N(%): 59 (34.5%): 3335 (66)
			≥ 90th%ile, N(%): 6 (3.5%): 3281 (249)
			p-Value for mean birth weight for ≥ 90th%ile PM_{10} vs. for < 90th%ile PM_{10}
			Adjusted: 0.833
			Adjusted, with CO: 0.833
			Adjusted, with NO_2: 0.778
			Adjusted, with SO_2: 0.806
			3rd trimester
			< 90th%ile, N(%): 61 (34.1%): 3327 (65)
			≥ 90th%ile, N(%): 6 (3.4%): 3227 (300)
			p-Value for mean birth weight for ≥ 90th%ile PM_{10} vs. for < 90th%ile PM_{10}
			Adjusted: 0.749
			Adjusted, with CO: 0.980
			Adjusted, with NO_2: 0.635
			Adjusted, with SO_2: 0.687
			Genotype MspI TC/CC
			1st trimester
			< 90th%ile, N(%): 98 (56.0%): 3193 (48)
			≥ 90th%ile, N(%): 12 (6.9%): 2799 (169)
			p-Value for mean birth weight for ≥ 90th%ile PM_{10} vs. for < 90th%ile PM_{10}
			Adjusted: 0.033
			Adjusted, with CO: 0.073
			Adjusted, with NO_2: 0.150
			Adjusted, with SO_2: 0.036
			2nd trimester
			< 90th%ile, N(%): 94 (55.0%): 3200 (52)
			≥ 90th%ile, N(%): 12 (7.0%): 2933 (176)
			p-Value for mean birth weight for ≥ 90th%ile PM_{10} vs. for < 90th%ile PM_{10}
			Adjusted: 0.161
			Adjusted, with CO: 0.172
			Adjusted, with NO_2: 0.152
			Adjusted, with SO_2: 0.158
			3rd trimester
			< 90th%ile, N(%): 101 (56.4%): 3165 (49)
			≥ 90th%ile, N(%): 11 (6.2%): 3087 (147)
			p-Value for mean birth weight for ≥ 90th%ile PM_{10} vs. for < 90th%ile PM_{10}

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			Adjusted: 0.626
			Adjusted, with CO: 0.978
			Adjusted, with NO_2: 0.551
			Adjusted, with SO_2: 0.614
			Genotype NcoI IleIle
			1st trimester
			< 90th%ile, N(%): 87 (49.7%): 3244 (52)
			≥ 90th%ile, N(%): 7 (4.0%): 2983 (232)
			p-Value for mean birth weight for ≥ 90th%ile PM_{10} vs. for < 90th%ile PM_{10}
			Adjusted: 0.289
			Adjusted, with CO: 0.344
			Adjusted, with NO_2: 0.641
			Adjusted, with SO_2: 0.293
			2nd trimester
			< 90th%ile, N(%): 82 (48.0%): 3243 (55)
			≥ 90th%ile, N(%): 11 (6.4%): 3185 (207)
			p-Value for mean birth weight for ≥ 90th%ile PM_{10} vs. for < 90th%ile PM_{10}
			Adjusted: 0.790
			Adjusted, with CO: 0.783
			Adjusted, with NO_2: 0.707
			Adjusted, with SO_2: 0.733
			3rd trimester
			< 90th%ile, N(%): 90 (50.3%): 3239 (53)
			≥ 90th%ile, N(%): 9 (5.0%): 2944 (198)
			p-Value for mean birth weight for ≥ 90th%ile PM_{10} vs. for < 90th%ile PM_{10}
			Adjusted: 0.161
			Adjusted, with CO: 0.279
			Adjusted, with NO_2: 0.134
			Adjusted, with SO_2: 0.150
			Genotype NcoI IleVal/ValVal
			1st trimester
			< 90th%ile, N(%): 71 (40.6%): 3262 (56)
			≥ 90th%ile, N(%): 10 (5.7%): 2773 (171)
			p-Value for mean birth weight for ≥ 90th%ile PM_{10} vs. for < 90th%ile PM_{10}
			Adjusted: 0.009
			Adjusted, with CO: 0.031
			Adjusted, with NO_2: 0.058
			Adjusted, with SO_2: 0.010
			2nd trimester
			< 90th%ile, N(%): 71 (41.5%): 3264 (61)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			≥ 90th%ile, N(%): 7 (4.1%): 2862 (208)
			p-Value for mean birth weight for ≥ 90th%ile PM_{10} vs. for < 90th%ile PM_{10}
			Adjusted: 0.076
			Adjusted, with CO: 0.093
			Adjusted, with NO_2: 0.063
			Adjusted, with SO_2: 0.061
			3rd trimester
			< 90th%ile, N(%): 72 (40.2%): 3207 (58)
			≥ 90th%ile, N(%): 8 (4.5%): 3262 (180)
			p-Value for mean birth weight for ≥ 90th%ile PM_{10} vs. for < 90th%ile PM_{10}
			Adjusted: 0.777
			Adjusted, with CO: 0.607
			Adjusted, with NO_2: 0.843
			Adjusted, with SO_2: 0.791
Reference: Tsai et al. (2006, 098312) **Period of Study:** 1994-2000 **Location:** Kaohsiung, Taiwan	**Outcome:** post neonatal mortality **Age Groups:** infants more than 27 days and less than 1 year **Study Design:** Case-crossover study **N:** 207 deaths **Statistical Analyses:** Conditional logistic regression **Covariates:** temperature, humidity **Dose-response Investigated?** No **Statistical Package:** SAS, version 8.2	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (SD):** 81.45 $\mu g/m^3$ **Percentiles: 25th:** 44.50 **50th(Median):** 79.20 **75th:** 111.50 **Range (Min, Max):** (20.50-232.00) **Monitoring Stations:** 6 **Copollutant:** SO_2 NO_2 CO O_3	**PM Increment:** 67.00 $\mu g/m^3$ **Effect Estimate [Lower CI, Upper CI]:** OR = 1.040 (0.340-3.177) **Note:** Air pollution levels at the dates of infant death were compared with air pollution levels 1 week before and 1 week after death a cumulative lag up to 2 previous days was used to assign exposure.
Reference: (Wilhelm and Ritz, 2005, 188761) **Period of Study:** 1994-2000 **Location:** Los Angeles County, California, U.S.	**Outcome:** Term low birth weight (LBW) (< 2500 g at ≥ 37 completed weeks gestation), Vaginal birth < 37 completed weeks gestation **Age Groups:** LBW: ≥ 37 completed weeks Preterm births: < 37 completed weeks **Study Design:** Cross-sectional **N:** For LBW: 136,134 For preterm birth: 106,483 **Statistical Analyses:** Logistic regression **Covariates:** Maternal age, maternal race, maternal education, parity, interval since previous live birth, level of prenatal care, infant sex, previous LBW or preterm infant, birth season, other pollutants (CO, NO_2, O_3, PM_{10}), gestational age (in birth weight analysis) **Dose-response Investigated?** Yes **Statistical Package:** NR	**Pollutant:** PM_{10} **Averaging Time:** 24 hr (every 6 days) Entire pregnancy Trimesters of pregnancy Months of pregnancy 6 weeks before birth **Mean (SD):** First trimester: 42.2 Third trimester: 41.5 6 weeks before birth: 39.1 **Range (Min, Max):** First trimester: 26.3, 77.4 Third trimester: 25.7, 74.6 6 weeks before birth: 13.0, 103.7 **Monitoring Stations:** Zip-code-level analysis: 8	**PM Increment:** 1) 10 $\mu g/m^3$ 2) 3 levels: a) < 25%ile (reference) b) 25%-75%ile c) ≥ 75%ile **Incidence of LBW (third trimester exposure)** < 32.8 $\mu g/m^3$: 2.0 (1.8, 2.2) 32.8 to < 43.4 $\mu g/m^3$: 2.0 (1.9, 2.1) ≥ 43.4 $\mu g/m^3$: 2.2 (2.0, 2.4) **Incidence of preterm birth (first trimester exposure)** < 32.9 $\mu g/m^3$: 8.7 (8.3, 9.2) 32.9 to < 43.9 $\mu g/m^3$: 8.8 (8.5, 9.1) ≥ 43.9 $\mu g/m^3$: 8.6 (8.1, 9.0) **Incidence of preterm birth (6 weeks before birth exposure)** < 31.8 $\mu g/m^3$: 8.8 (8.4, 9.3)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
		Address-level analysis: 6	31.8 to < 44.1 $\mu g/m^3$: 8.6 (8.3, 8.9)
		Copollutant (correlation):	≥ 44.1 $\mu g/m^3$: 8.8 (8.4, 9.2)
		First trimester: PM_{10}-CO: r = 0.12	**Outcome: LBW**
		PM_{10}-NO_2: r = 0.29	**Exposure Period: Third trimester**
		PM_{10}-O_3: r = -0.01	**Address-level analysis:**
		PM_{10}-$PM_{2.5}$: r = 0.43	Single-pollutant model:
		Third trimester: PM_{10}-CO: r = 0.32	Distance ◘ 1 mile
		PM_{10}-NO_2: r = 0.45	Per 10 $\mu g/m^3$: 1.22 (1.05, 1.41)
		PM_{10}-O_3: r = -0.08	33.4 to < 44.7 $\mu g/m^3$: 1.08 (0.76, 1.52)
		PM_{10}-$PM_{2.5}$: r = 0.52	≥ 44.7 $\mu g/m^3$: 1.48 (1.00, 2.19)
		6 weeks before birth: PM_{10}-CO: r = 0.36	Multipollutant model:
		PM_{10}-NO_2: r = 0.49	Distance ◘ 1 mile
		PM_{10}-O_3: r = -0.16	Per 10 $\mu g/m^3$: 1.36 (1.12, 1.65)
		PM_{10}-$PM_{2.5}$: r = 0.60	33.4 to < 44.7 $\mu g/m^3$: 1.16 (0.77, 1.74)
			≥ 44.7 $\mu g/m^3$: 1.58 (0.95, 2.62)
			Single-pollutant model:
			1 < distance ◘ 2 mile
			Per 10 $\mu g/m^3$: 0.98 (0.90, 1.06)
			33.4 to < 44.7 $\mu g/m^3$: 0.95 (0.80, 1.13)
			≥ 44.7 $\mu g/m^3$: 0.96 (0.78, 1.18)
			Multipollutant model:
			1 < distance ◘ 2 mile
			Per 10 $\mu g/m^3$: 1.02 (0.92, 1.14)
			33.4 to < 44.7 $\mu g/m^3$: 0.93 (0.77, 1.12)
			≥ 44.7 $\mu g/m^3$: 1.02 (0.79, 1.32)
			Single-pollutant model:
			2 < distance ◘ 4 mile
			Per 10 $\mu g/m^3$: 1.03 (0.99, 1.08)
			33.9 to < 45.0 $\mu g/m^3$: 1.04 (0.96, 1.14)
			≥ 45.0 $\mu g/m^3$: 1.08 (0.97, 1.20)
			Multipollutant model:
			2 < distance ◘ 4 mile
			Per 10 $\mu g/m^3$: 1.04 (0.98, 1.09)
			33.9 to < 45.0 $\mu g/m^3$: 1.02 (0.92, 1.12)
			≥ 45.0 $\mu g/m^3$: 1.06 (0.93, 1.21)
			Zip-code-level analysis
			Single-pollutant model:
			Per 10 $\mu g/m^3$: 1.03 (0.97, 1.09)
			33.2 to < 43.6 $\mu g/m^3$: 0.98 (0.86, 1.11)
			≥ 43.6 $\mu g/m^3$: 1.03 (0.88, 1.21)
			Multipollutant model:
			Per 10 $\mu g/m^3$: 1.07 (0.99, 1.15)
			33.2 to < 43.6 $\mu g/m^3$: 0.97 (0.85, 1.12)
			≥ 43.6 $\mu g/m^3$: 1.09 (0.90, 1.31)
			Outcome: LBW

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			Exposure Period: Entire pregnancy period
			Address-level analysis:
			Multipollutant model:
			Per 10 $\mu g/m^3$: 1.24 (0.91, 1.70)
			Outcome: Preterm Birth
			Exposure Period: First trimester of pregnancy
			Address-level analysis:
			Single-pollutant model:
			Distance ◘ 1 mile
			Per 10 $\mu g/m^3$: 1.00 (0.93, 1.09)
			33.3 to < 45.1 $\mu g/m^3$: 1.07 (0.90, 1.26)
			≥ 45.1 $\mu g/m^3$: 1.12 (0.91, 1.38)
			Multipollutant model:
			Distance ◘ 1 mile
			Per 10 $\mu g/m^3$: 1.00 (0.90, 1.12)
			33.3 to < 45.1 $\mu g/m^3$: 1.12 (0.92, 1.36)
			≥ 45.1 $\mu g/m^3$: 1.17 (0.90, 1.50)
			Single-pollutant model:
			1 < distance ◘ 2 mile
			Per 10 $\mu g/m^3$: 1.01 (0.97, 1.05)
			33.7 to < 45.3 $\mu g/m^3$: 1.03 (0.95, 1.12)
			≥ 45.3 $\mu g/m^3$: 1.07 (0.97, 1.19)
			Multipollutant model:
			1 < distance ◘ 2 mile
			Per 10 $\mu g/m^3$: 1.04 (0.99, 1.10)
			33.7 to < 45.3 $\mu g/m^3$: 1.07 (0.98, 1.17)
			≥ 45.3 $\mu g/m^3$: 1.13 (1.00, 1.27)
			Single-pollutant model:
			2 < distance ◘ 4 mile
			Per 10 $\mu g/m^3$: 1.01 (0.99, 1.03)
			34.1 to < 45.5 $\mu g/m^3$: 1.03 (0.99, 1.08)
			≥ 45.5 $\mu g/m^3$: 1.02 (0.96, 1.07)
			Multipollutant model:
			2 < distance ◘ 4 mile
			Per 10 $\mu g/m^3$: 0.99 (0.97, 1.02)
			34.1 to < 45.5 $\mu g/m^3$: 0.99 (0.95, 1.04)
			≥ 45.5 $\mu g/m^3$: 0.94 (0.89, 1.01)
			Zip-code-level analysis
			Single-pollutant model:
			Per 10 $\mu g/m^3$: 0.99 (0.96, 1.01)
			33.3 to < 44.2 $\mu g/m^3$: 1.01 (0.95, 1.08)
			≥ 44.2 $\mu g/m^3$: 0.98 (0.90, 1.05)
			Multipollutant model:
			Per 10 $\mu g/m^3$: 0.99 (0.96, 1.03)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			33.3 to < 44.2 $\mu g/m^3$: 1.03 (0.97, 1.11)
			≥ 44.2 $\mu g/m^3$: 1.01 (0.92, 1.11)
			Outcome: Preterm birth
			Exposure Period: 6 weeks before birth
			Address-level analysis:
			Single-pollutant model:
			Distance ◘ 1 mile
			Per 10 $\mu g/m^3$: 1.02 (0.95, 1.10)
			32.5 to < 44.8 $\mu g/m^3$: 1.09 (0.92, 1.29)
			≥ 44.8 $\mu g/m^3$: 1.12 (0.92, 1.37)
			Multipollutant model:
			Distance ◘ 1 mile
			Per 10 $\mu g/m^3$: 1.06 (0.97, 1.16)
			32.5 to < 44.8 $\mu g/m^3$: 1.09 (0.90, 1.31)
			≥ 44.8 $\mu g/m^3$: 1.17 (0.91, 1.49)
			Single-pollutant model:
			1 < distance ◘ 2 mile
			Per 10 $\mu g/m^3$: 1.00 (0.96, 1.03)
			32.3 to < 45.3 $\mu g/m^3$: 0.99 (0.91, 1.07)
			≥ 45.3 $\mu g/m^3$: 0.99 (0.89, 1.10)
			Multipollutant model:
			1 < distance ◘ 2 mile
			Per 10 $\mu g/m^3$: 1.01 (0.97, 1.06)
			32.3 to < 45.3 $\mu g/m^3$: 1.00 (0.92, 1.10)
			≥ 45.3 $\mu g/m^3$: 1.02 (0.91, 1.16)
			Single-pollutant model:
			2 < distance ◘ 4 mile
			Per 10 $\mu g/m^3$: 0.99 (0.98, 1.01)
			33.1 to < 45.3 $\mu g/m^3$: 1.00 (0.96, 1.05)
			≥ 45.3 $\mu g/m^3$: 0.98 (0.93, 1.03)
			Multipollutant model:
			2 < distance ◘ 4 mile
			Per 10 $\mu g/m^3$: 1.00 (0.98, 1.02)
			33.1 to < 45.3 $\mu g/m^3$: 1.01 (0.96, 1.05)
			≥ 45.3 $\mu g/m^3$: 0.98 (0.92, 1.04)
			Zip-code-level analysis
			Single-pollutant model:
			Per 10 $\mu g/m^3$: 1.02 (0.99, 1.04)
			32.1 to < 44.3 $\mu g/m^3$: 1.01 (0.95, 1.07)
			≥ 44.3 $\mu g/m^3$: 1.04 (0.96, 1.12)
			Multipollutant model:
			Per 10 $\mu g/m^3$: 1.02 (0.99, 1.06)
			32.1 to < 44.3 $\mu g/m^3$: 1.02 (0.95, 1.09)
			≥ 44.3 $\mu g/m^3$: 1.04 (0.95, 1.14)
			Notes: multipollutant model adds CO,NO_2, and O_3 in addition to the main

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			pollutant of interest, PM_{10}.
Reference: Woodruff et al. (1997, 084271) **Period of Study:** 1989-1991 **Location:** 86 Metropolitan Statistical Areas in the US (counties with populations less than 100,000 were excluded)	**Outcome:** Postneonatal mortality (death of an infant between 1 month and 1 yr of age) 1) all post neonatal deaths 2) normal birth weight (NBW, ≥ 2500 g) SIDS deaths 3) NBW respiratory deaths 4) low birth weight (LBW) respiratory death Respiratory deaths: ICD9 codes 460-519 SIDS: ICD9 code 798.0 **Age Groups:** infants (1 month–1yr of age) **Study Design:** Cross-sectional **N:** 3,788,079 infants **Statistical Analyses:** Logistic regression **Covariates:** maternal education, maternal race, parental marital status, maternal smoking during pregnancy avg temperature during the first 2 months of life infant's month and year of birth assessed race as an effect modifier (p-val for interaction terms > 0.2) **Dose-response Investigated?** Yes **Statistical Package:** NR	**Pollutant:** PM_{10} **Averaging Time:** Mean of 1st 2 months of life analyzed as tertiles of exposure and as continuous exposure **Mean (SD):** 31.4 (7.8) **Range (Min, Max):** Overall: 11.9-68.8 Low category: < 28.0 Medium category: 28.1-40.0 High category: > 40.0 **Monitoring Stations:** NR	**PM Increment:** 10 $\mu g/m^3$ (for continuous exposure analysis) **Adjusted ORs for cause-specific post neonatal mortality by pollution category (tertiles)** All causes Low: Ref Medium: 1.05 (1.01, 1.09) High: 1.10 (1.04, 1.16) SIDS, NBW: Low: Ref Medium: 1.09 (1.01, 1.17) High: 1.26 (1.14, 1.39) Respiratory death, NBW: Low: Ref Medium: 1.08 (0.87, 1.33) High: 1.40 (1.05, 1.85) Respiratory death, LBW: Low: Ref Medium: 0.93 (0.73, 1.18) High: 1.18 (0.86, 1.61) All other causes: Low: Ref Medium: 1.03 (0.97, 1.08) High: 0.97 (0.90, 1.04) **Adjusted ORs for a continuous 10 $\mu g/m^3$ change in exposure** All causes: 1.04 (1.02, 1.07) SIDS, NBW: 1.12 (1.07, 1.17) Respiratory death, NBW: 1.20 (1.06, 1.36) Respiratory death, LBW: 1.05 (0.91, 1.22) All other causes: 1.00 (0.99, 1.00)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Woodruff et al (2008, 098386) **Period of Study:** 1999-2002 **Location:** US counties with > 250,000 residents (96 counties)	**Outcome:** Postneonatal deaths Respiratory mortality (ICD10: J000-99, plus bronchopulmonary dysplasia [BPD] P27.1) SIDS (ICD10: R95) Ill-defined causes (R99); All other deaths evaluated as a control category **Age Groups:** Infants aged > 28 days and < 1 yr **Study Design:** Cross-sectional **N:** 3,583,495 births (6,639 post neonatal deaths) **Statistical Analyses:** Logistic GEE (exchangeable correlation structure) **Covariates:** maternal race/ethnicity, marital status, age, education, primiparity, county-level poverty and per capita income levels, year and month of birth dummy variables to account for time trend and seasonal effects, and region of the country sensitivity analyses performed among only those mothers with smoking information (adjustment for smoking had no effect on the estimates) **Season:** Adjusted for year and month of birth dummy variables to account for time trend and seasonal effects **Dose-response Investigated?** Evaluated the appropriateness of a linear form from analysis based on quartiles of exposure and concluded that linear form was appropriate (data not shown) **Statistical Package:** SAS	**Pollutant:** PM_{10} **Averaging Time:** Measured continuously for 24 h once every 6 days exposure assigned by calculating avg concentration of pollutant during first 2 months of life **Median and IQR (25th-75th percentile):** Survivors: 28.9 (23.3-34.4) All causes of death: 29.1 (23.9-34.5) Respiratory: 29.8 (24.3-36.5) SIDS: 28.6 (23.5-33.8) SIDS + ill-defined: 28.8 (23.9-33.9) Other causes: 29.2 (23.9-34.5) **Percentiles:** see above **PM Component:** Not assessed, but controlled for region of the country to account for PM composition variation **Monitoring Stations:** NR **Copollutant (correlation):** PM_{10} $PM_{2.5}$ (r = 0.34) CO (r = 0.18) SO_2 (r = 0.00) O_3 (r = 0.20) **Notes:** Monthly averages calculated if there were at least 3 available measures for PM Assigned exposures using the avg concentration of the county of residence	**PM Increment:** IQR (11 $\mu g/m^3$) **Effect Estimate [Lower CI, Upper CI]:** Adjusted ORs for single pollutant models All causes: 1.04 (0.99, 1.10) Respiratory: 1.18 (1.06, 1.31) SIDS: 1.02 (0.89, 1.16) Ill-defined + SIDS: 1.06 (0.97, 1.16) Other causes: 1.02 (0.96, 1.07) Adjusted ORs for multipollutant models (including CO, O_3, SO_2) Respiratory: 1.16 (1.04, 1.30) SIDS: 1.02 (0.90, 1.16) OR for deaths coded as BPD per increase in IQR: 1.19 (0.85, 1.65) OR for respiratory post neonatal death stratified by birth weight NBW only: 1.19 (1.05, 1.36) LBW only: 1.12 (0.95, 1.31) OR for respiratory deaths removing region of US as a confounding variable: 1.30 (1.04, 1.61) OR for respiratory deaths assessing exposure as quartiles Highest vs Lowest quartile: 1.31 (1.00, 1.71) OR for respiratory deaths among only those deaths that occurred during the first 90 days (most closely matched exposure metric of the avg over the first 2 months of life): 1.25 (1.06, 1.47)
Reference: (Suh et al., 2007, 157028) **Period of Study:** 2001-2004 **Location:** Seoul, Korea	**Outcome:** Birth weight **Age Groups:** prenatal follow-up for newborns **Study Design:** based prospective cohort study **N:** 199 pregnant mothers **Statistical Analyses:** ANCOVA, generalized linear models **Covariates:** infant's sex, maternal age, maternal and paternal education, parity, presence of illness during pregnancy, delivery month, gestational age (squared) **Dose-response Investigated?** Yes **Statistical Package:** SAS	**Pollutant:** PM_{10} **Averaging Time:** 24-h **Mean (SD):** 1st trimester: 76.41 (28.80) 2nd trimester: 77.84 (31.63) 3rd trimester: 95.61 (26.15) **Percentiles:** 1st trimester **25th:** 55.28 **50th(Median):** 71.09 **75th:** 92.38 2nd trimester **25th:** 48.65 **50th(Median):** 72.36 **75th:** 108.00 3rd trimester **25th:** 77.10 **50th(Median):** 96.35 **75th:** 116.68 **Range (Min, Max):** 1st trimester (21.00, 151.65)	**PM Increment:** Trimester ≥ 90th%ile compared to < 90th%ile Least-square (ANCOVA) mean (SE) **All Genotypes** 1st trimester < 90th%ile, N(%): 158 (90.3%): 3253 (37) ≥ 90th%ile, N(%): 17 (9.7%): 2841 (145) p-Value for mean birth weight for ≥ 90th%ile PM_{10} vs. for < 90th%ile PM_{10} Adjusted: 0.009 Adjusted, with CO: 0.041 Adjusted, with NO_2: 0.092 Adjusted, with SO_2: 0.012 2nd trimester < 90th%ile, N(%): 153 (89.5%): 3253 (39) ≥ 90th%ile, N(%): 18 (10.5%): 3026 (157) p-Value for mean birth weight for ≥ 90th%ile PM_{10} vs. for < 90th%ile PM_{10}

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
		2nd trimester (31.45, 139.13)	Adjusted: 0.177
		3rd trimester (23.45, 172.75)	Adjusted, with CO: 0.203
		Monitoring Stations: 27	Adjusted, with NO_2: 0.151
		Copollutant:	Adjusted, with SO_2: 0.151
		CO	3rd trimester
		SO_2	< 90th%ile, N(%): 162 (90.5%): 3226 (38)
		NO_2	≥ 90th%ile, N(%): 17 (9.5%): 3122 (140)
			p-Value for mean birth weight for ≥ 90th%ile PM_{10} vs. for < 90th%ile PM_{10}
			Adjusted: 0.487
			Adjusted, with CO: 0.748
			Adjusted, with NO_2: 0.420
			Adjusted, with SO_2: 0.466
			Genotype MspI TT
			1st trimester
			< 90th%ile, N(%): 60 (34.3%): 3350 (64)
			≥ 90th%ile, N(%): 5 (2.9%): 3001 (229)
			p-Value for mean birth weight for ≥ 90th%ile PM_{10} vs. for < 90th%ile PM_{10}
			Adjusted: 0.147
			Adjusted, with CO: 0.186
			Adjusted, with NO_2: 0.430
			Adjusted, with SO_2: 0.155
			2nd trimester
			< 90th%ile, N(%): 59 (34.5%): 3335 (66)
			≥ 90th%ile, N(%): 6 (3.5%): 3281 (249)
			p-Value for mean birth weight for ≥ 90th%ile PM_{10} vs. for < 90th%ile PM_{10}
			Adjusted: 0.833
			Adjusted, with CO: 0.833
			Adjusted, with NO_2: 0.778
			Adjusted, with SO_2: 0.806
			3rd trimester
			< 90th%ile, N(%): 61 (34.1%): 3327 (65)
			≥ 90th%ile, N(%): 6 (3.4%): 3227 (300)
			p-Value for mean birth weight for ≥ 90th%ile PM_{10} vs. for < 90th%ile PM_{10}
			Adjusted: 0.749
			Adjusted, with CO: 0.980
			Adjusted, with NO_2: 0.635
			Adjusted, with SO_2: 0.687
			Genotype MspI TC/CC
			1st trimester
			< 90th%ile, N(%): 98 (56.0%): 3193 (48)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			≥ 90th%ile, N(%): 12 (6.9%): 2799 (169)
			p-Value for mean birth weight for ≥ 90th%ile PM_{10} vs. for < 90th%ile PM_{10}
			Adjusted: 0.033
			Adjusted, with CO: 0.073
			Adjusted, with NO_2: 0.150
			Adjusted, with SO_2: 0.036
			2nd trimester
			< 90th%ile, N(%): 94 (55.0%): 3200 (52)
			≥ 90th%ile, N(%): 12 (7.0%): 2933 (176)
			p-Value for mean birth weight for ≥ 90th%ile PM_{10} vs. for < 90th%ile PM_{10}
			Adjusted: 0.161
			Adjusted, with CO: 0.172
			Adjusted, with NO_2: 0.152
			Adjusted, with SO_2: 0.158
			3rd trimester
			< 90th%ile, N(%): 101 (56.4%): 3165 (49)
			≥ 90th%ile, N(%): 11 (6.2%): 3087 (147)
			p-Value for mean birth weight for ≥ 90th%ile PM_{10} vs. for < 90th%ile PM_{10}
			Adjusted: 0.626
			Adjusted, with CO: 0.978
			Adjusted, with NO_2: 0.551
			Adjusted, with SO_2: 0.614
			Genotype NcoI IleIle
			1st trimester
			< 90th%ile, N(%): 87 (49.7%): 3244 (52)
			≥ 90th%ile, N(%): 7 (4.0%): 2983 (232)
			p-Value for mean birth weight for ≥ 90th%ile PM_{10} vs. for < 90th%ile PM_{10}
			Adjusted: 0.289
			Adjusted, with CO: 0.344
			Adjusted, with NO_2: 0.641
			Adjusted, with SO_2: 0.293
			2nd trimester
			< 90th%ile, N(%): 82 (48.0%): 3243 (55)
			≥ 90th%ile, N(%): 11 (6.4%): 3185 (207)
			p-Value for mean birth weight for ≥ 90th%ile PM_{10} vs. for < 90th%ile PM_{10}
			Adjusted: 0.790
			Adjusted, with CO: 0.783
			Adjusted, with NO_2: 0.707
			Adjusted, with SO_2: 0.733
			3rd trimester

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			< 90th%ile, N(%): 90 (50.3%): 3239 (53)
			≥ 90th%ile, N(%): 9 (5.0%): 2944 (198)
			p-Value for mean birth weight for ≥ 90th%ile PM_{10} vs. for < 90th%ile PM_{10}
			Adjusted: 0.161
			Adjusted, with CO: 0.279
			Adjusted, with NO_2: 0.134
			Adjusted, with SO_2: 0.150
			Genotype NcoI IleVal/ValVal
			1st trimester
			< 90th%ile, N(%): 71 (40.6%): 3262 (56)
			≥ 90th%ile, N(%): 10 (5.7%): 2773 (171)
			p-Value for mean birth weight for ≥ 90th%ile PM_{10} vs. for < 90th%ile PM_{10}
			Adjusted: 0.009
			Adjusted, with CO: 0.031
			Adjusted, with NO_2: 0.058
			Adjusted, with SO_2: 0.010
			2nd trimester
			< 90th%ile, N(%): 71 (41.5%): 3264 (61)
			≥ 90th%ile, N(%): 7 (4.1%): 2862 (208)
			p-Value for mean birth weight for ≥ 90th%ile PM_{10} vs. for < 90th%ile PM_{10}
			Adjusted: 0.076
			Adjusted, with CO: 0.093
			Adjusted, with NO_2: 0.063
			Adjusted, with SO_2: 0.061
			3rd trimester
			< 90th%ile, N(%): 72 (40.2%): 3207 (58)
			≥ 90th%ile, N(%): 8 (4.5%): 3262 (180)
			p-Value for mean birth weight for ≥ 90th%ile PM_{10} vs. for < 90th%ile PM_{10}
			Adjusted: 0.777
			Adjusted, with CO: 0.607
			Adjusted, with NO_2: 0.843
			Adjusted, with SO_2: 0.791

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Tsai et al. (2006, 098312) **Period of Study:** 1994-2000 **Location:** Kaohsiung, Taiwan	**Outcome:** post neonatal mortality **Age Groups:** infants more than 27 days and less than 1 year **Study Design:** Case-crossover study **N:** 207 deaths **Statistical Analyses:** Conditional logistic regression **Covariates:** temperature, humidity **Dose-response Investigated?** No **Statistical Package:** SAS, version 8.2	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (SD):** 81.45 $\mu g/m^3$ **Percentiles: 25th:** 44.50 **50th(Median):** 79.20 **75th:** 111.50 **Range (Min, Max):** (20.50-232.00) **Monitoring Stations:** 6 **Copollutant:** SO_2 NO_2 CO O_3	**PM Increment:** 67.00 $\mu g/m^3$ **Effect Estimate [Lower CI, Upper CI]:** OR = 1.040 (0.340-3.177) **Note:** Air pollution levels at the dates of infant death were compared with air pollution levels 1 week before and 1 week after death a cumulative lag up to 2 previous days was used to assign exposure.
Reference: (Wilhelm and Ritz, 2005, 188761) **Period of Study:** 1994-2000 **Location:** Los Angeles County, California, U.S.	**Outcome:** Term low birth weight (LBW) (< 2500 g at ≥ 37 completed weeks gestation), Vaginal birth < 37 completed weeks gestation **Age Groups:** LBW: ≥ 37 completed weeks Preterm births: < 37 completed weeks **Study Design:** Cross-sectional **N:** For LBW: 136,134 For preterm birth: 106,483 **Statistical Analyses:** Logistic regression **Covariates:** Maternal age, maternal race, maternal education, parity, interval since previous live birth, level of prenatal care, infant sex, previous LBW or preterm infant, birth season, other pollutants (CO, NO_2, O_3, PM_{10}), gestational age (in birth weight analysis) **Dose-response Investigated?** Yes **Statistical Package:** NR	**Pollutant:** PM_{10} **Averaging Time:** 24 hr (every 6 days) Entire pregnancy Trimesters of pregnancy Months of pregnancy 6 weeks before birth **Mean (SD):** First trimester: 42.2 Third trimester: 41.5 6 weeks before birth: 39.1 **Range (Min, Max):** First trimester: 26.3, 77.4 Third trimester: 25.7, 74.6 6 weeks before birth: 13.0, 103.7 **Monitoring Stations:** Zip-code-level analysis: 8 Address-level analysis: 6 **Copollutant (correlation):** **First trimester:** PM_{10}-CO: r = 0.12 PM_{10}-NO_2: r = 0.29 PM_{10}-O_3: r = -0.01 PM_{10}-$PM_{2.5}$: r = 0.43 **Third trimester:** PM_{10}-CO: r = 0.32 PM_{10}-NO_2: r = 0.45 PM_{10}-O_3: r = -0.08 PM_{10}-$PM_{2.5}$: r = 0.52 **6 weeks before birth:** PM_{10}-CO: r = 0.36 PM_{10}-NO_2: r = 0.49 PM_{10}-O_3: r = -0.16 PM_{10}-$PM_{2.5}$: r = 0.60	**PM Increment:** 1) 10 $\mu g/m^3$ 2) 3 levels: a) < 25%ile (reference) b) 25%-75%ile c) ≥ 75%ile **Incidence of LBW (third trimester exposure)** < 32.8 $\mu g/m^3$: 2.0 (1.8, 2.2) 32.8 to < 43.4 $\mu g/m^3$: 2.0 (1.9, 2.1) ≥ 43.4 $\mu g/m^3$: 2.2 (2.0, 2.4) **Incidence of preterm birth (first trimester exposure)** < 32.9 $\mu g/m^3$: 8.7 (8.3, 9.2) 32.9 to < 43.9 $\mu g/m^3$: 8.8 (8.5, 9.1) ≥ 43.9 $\mu g/m^3$: 8.6 (8.1, 9.0) **Incidence of preterm birth (6 weeks before birth exposure)** < 31.8 $\mu g/m^3$: 8.8 (8.4, 9.3) 31.8 to < 44.1 $\mu g/m^3$: 8.6 (8.3, 8.9) ≥ 44.1 $\mu g/m^3$: 8.8 (8.4, 9.2) **Outcome: LBW** **Exposure Period: Third trimester** **Address-level analysis:** Single-pollutant model: Distance ◘ 1 mile Per 10 $\mu g/m^3$: 1.22 (1.05, 1.41) 33.4 to < 44.7 $\mu g/m^3$: 1.08 (0.76, 1.52) ≥ 44.7 $\mu g/m^3$: 1.48 (1.00, 2.19) Multipollutant model: Distance ◘ 1 mile Per 10 $\mu g/m^3$: 1.36 (1.12, 1.65) 33.4 to < 44.7 $\mu g/m^3$: 1.16 (0.77, 1.74) ≥ 44.7 $\mu g/m^3$: 1.58 (0.95, 2.62) Single-pollutant model:

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			1 < distance ◘ 2 mile
			Per 10 $\mu g/m^3$: 0.98 (0.90, 1.06)
			33.4 to < 44.7 $\mu g/m^3$: 0.95 (0.80, 1.13)
			≥ 44.7 $\mu g/m^3$: 0.96 (0.78, 1.18)
			Multipollutant model:
			1 < distance ◘ 2 mile
			Per 10 $\mu g/m^3$: 1.02 (0.92, 1.14)
			33.4 to < 44.7 $\mu g/m^3$: 0.93 (0.77, 1.12)
			≥ 44.7 $\mu g/m^3$: 1.02 (0.79, 1.32)
			Single-pollutant model:
			2 < distance ◘ 4 mile
			Per 10 $\mu g/m^3$: 1.03 (0.99, 1.08)
			33.9 to < 45.0 $\mu g/m^3$: 1.04 (0.96, 1.14)
			≥ 45.0 $\mu g/m^3$: 1.08 (0.97, 1.20)
			Multipollutant model:
			2 < distance ◘ 4 mile
			Per 10 $\mu g/m^3$: 1.04 (0.98, 1.09)
			33.9 to < 45.0 $\mu g/m^3$: 1.02 (0.92, 1.12)
			≥ 45.0 $\mu g/m^3$: 1.06 (0.93, 1.21)
			Zip-code-level analysis
			Single-pollutant model:
			Per 10 $\mu g/m^3$: 1.03 (0.97, 1.09)
			33.2 to < 43.6 $\mu g/m^3$: 0.98 (0.86, 1.11)
			≥ 43.6 $\mu g/m^3$: 1.03 (0.88, 1.21)
			Multipollutant model:
			Per 10 $\mu g/m^3$: 1.07 (0.99, 1.15)
			33.2 to < 43.6 $\mu g/m^3$: 0.97 (0.85, 1.12)
			≥ 43.6 $\mu g/m^3$: 1.09 (0.90, 1.31)
			Outcome: LBW
			Exposure Period: Entire pregnancy period
			Address-level analysis:
			Multipollutant model:
			Per 10 $\mu g/m^3$: 1.24 (0.91, 1.70)
			Outcome: Preterm Birth
			Exposure Period: First trimester of pregnancy
			Address-level analysis:
			Single-pollutant model:
			Distance ◘ 1 mile
			Per 10 $\mu g/m^3$: 1.00 (0.93, 1.09)
			33.3 to < 45.1 $\mu g/m^3$: 1.07 (0.90, 1.26)
			≥ 45.1 $\mu g/m^3$: 1.12 (0.91, 1.38)
			Multipollutant model:
			Distance ◘ 1 mile
			Per 10 $\mu g/m^3$: 1.00 (0.90, 1.12)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			33.3 to < 45.1 $\mu g/m^3$: 1.12 (0.92, 1.36)
			≥ 45.1 $\mu g/m^3$: 1.17 (0.90, 1.50)
			Single-pollutant model:
			1 < distance ≤ 2 mile
			Per 10 $\mu g/m^3$: 1.01 (0.97, 1.05)
			33.7 to < 45.3 $\mu g/m^3$: 1.03 (0.95, 1.12)
			≥ 45.3 $\mu g/m^3$: 1.07 (0.97, 1.19)
			Multipollutant model:
			1 < distance ≤ 2 mile
			Per 10 $\mu g/m^3$: 1.04 (0.99, 1.10)
			33.7 to < 45.3 $\mu g/m^3$: 1.07 (0.98, 1.17)
			≥ 45.3 $\mu g/m^3$: 1.13 (1.00, 1.27)
			Single-pollutant model:
			2 < distance ≤ 4 mile
			Per 10 $\mu g/m^3$: 1.01 (0.99, 1.03)
			34.1 to < 45.5 $\mu g/m^3$: 1.03 (0.99, 1.08)
			≥ 45.5 $\mu g/m^3$: 1.02 (0.96, 1.07)
			Multipollutant model:
			2 < distance ≤ 4 mile
			Per 10 $\mu g/m^3$: 0.99 (0.97, 1.02)
			34.1 to < 45.5 $\mu g/m^3$: 0.99 (0.95, 1.04)
			≥ 45.5 $\mu g/m^3$: 0.94 (0.89, 1.01)
			Zip-code-level analysis
			Single-pollutant model:
			Per 10 $\mu g/m^3$: 0.99 (0.96, 1.01)
			33.3 to < 44.2 $\mu g/m^3$: 1.01 (0.95, 1.08)
			≥ 44.2 $\mu g/m^3$: 0.98 (0.90, 1.05)
			Multipollutant model:
			Per 10 $\mu g/m^3$: 0.99 (0.96, 1.03)
			33.3 to < 44.2 $\mu g/m^3$: 1.03 (0.97, 1.11)
			≥ 44.2 $\mu g/m^3$: 1.01 (0.92, 1.11)
			Outcome: Preterm birth
			Exposure Period: 6 weeks before birth
			Address-level analysis:
			Single-pollutant model:
			Distance ≤ 1 mile
			Per 10 $\mu g/m^3$: 1.02 (0.95, 1.10)
			32.5 to < 44.8 $\mu g/m^3$: 1.09 (0.92, 1.29)
			≥ 44.8 $\mu g/m^3$: 1.12 (0.92, 1.37)
			Multipollutant model:
			Distance ≤ 1 mile
			Per 10 $\mu g/m^3$: 1.06 (0.97, 1.16)
			32.5 to < 44.8 $\mu g/m^3$: 1.09 (0.90, 1.31)
			≥ 44.8 $\mu g/m^3$: 1.17 (0.91, 1.49)
			Single-pollutant model:

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			1 < distance ≤ 2 mile
			Per 10 $\mu g/m^3$: 1.00 (0.96, 1.03)
			32.3 to < 45.3 $\mu g/m^3$: 0.99 (0.91, 1.07)
			≥ 45.3 $\mu g/m^3$: 0.99 (0.89, 1.10)
			Multipollutant model:
			1 < distance ≤ 2 mile
			Per 10 $\mu g/m^3$: 1.01 (0.97, 1.06)
			32.3 to < 45.3 $\mu g/m^3$: 1.00 (0.92, 1.10)
			≥ 45.3 $\mu g/m^3$: 1.02 (0.91, 1.16)
			Single-pollutant model:
			2 < distance ≤ 4 mile
			Per 10 $\mu g/m^3$: 0.99 (0.98, 1.01)
			33.1 to < 45.3 $\mu g/m^3$: 1.00 (0.96, 1.05)
			≥ 45.3 $\mu g/m^3$: 0.98 (0.93, 1.03)
			Multipollutant model:
			2 < distance ≤ 4 mile
			Per 10 $\mu g/m^3$: 1.00 (0.98, 1.02)
			33.1 to < 45.3 $\mu g/m^3$: 1.01 (0.96, 1.05)
			≥ 45.3 $\mu g/m^3$: 0.98 (0.92, 1.04)
			Zip-code-level analysis
			Single-pollutant model:
			Per 10 $\mu g/m^3$: 1.02 (0.99, 1.04)
			32.1 to < 44.3 $\mu g/m^3$: 1.01 (0.95, 1.07)
			≥ 44.3 $\mu g/m^3$: 1.04 (0.96, 1.12)
			Multipollutant model:
			Per 10 $\mu g/m^3$: 1.02 (0.99, 1.06)
			32.1 to < 44.3 $\mu g/m^3$: 1.02 (0.95, 1.09)
			≥ 44.3 $\mu g/m^3$: 1.04 (0.95, 1.14)
			Notes: multipollutant model adds CO, NO_2, and O_3 in addition to the main pollutant of interest, PM_{10}.

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Woodruff et al. (1997, 084271) **Period of Study:** 1989-1991 **Location:** 86 Metropolitan Statistical Areas in the US (counties with populations less than 100,000 were excluded)	**Outcome:** Postneonatal mortality (death of an infant between 1 month and 1 yr of age) 1) all post neonatal deaths 2) normal birth weight (NBW, ≥ 2500 g) SIDS deaths 3) NBW respiratory deaths 4) low birth weight (LBW) respiratory death Respiratory deaths: ICD9 codes 460-519 SIDS: ICD9 code 798.0 **Age Groups:** infants (1 month–1yr of age) **Study Design:** Cross-sectional **N:** 3,788,079 infants **Statistical Analyses:** Logistic regression **Covariates:** maternal education, maternal race, parental marital status, maternal smoking during pregnancy avg temperature during the first 2 months of life infant's month and year of birth assessed race as an effect modifier (p-val for interaction terms > 0.2) **Dose-response Investigated?** Yes **Statistical Package:** NR	**Pollutant:** PM_{10} **Averaging Time:** Mean of 1st 2 months of life analyzed as tertiles of exposure and as continuous exposure **Mean (SD):** 31.4 (7.8) **Range (Min, Max):** Overall: 11.9-68.8 Low category: < 28.0 Medium category: 28.1-40.0 High category: > 40.0 **Monitoring Stations:** NR	**PM Increment:** 10 $\mu g/m^3$ (for continuous exposure analysis) **Adjusted ORs for cause-specific post neonatal mortality by pollution category (tertiles)** All causes Low: Ref Medium: 1.05 (1.01, 1.09) High: 1.10 (1.04, 1.16) SIDS, NBW: Low: Ref Medium: 1.09 (1.01, 1.17) High: 1.26 (1.14, 1.39) Respiratory death, NBW: Low: Ref Medium: 1.08 (0.87, 1.33) High: 1.40 (1.05, 1.85) Respiratory death, LBW: Low: Ref Medium: 0.93 (0.73, 1.18) High: 1.18 (0.86, 1.61) All other causes: Low: Ref Medium: 1.03 (0.97, 1.08) High: 0.97 (0.90, 1.04) **Adjusted ORs for a continuous 10 $\mu g/m^3$ change in exposure** All causes: 1.04 (1.02, 1.07) SIDS, NBW: 1.12 (1.07, 1.17) Respiratory death, NBW: 1.20 (1.06, 1.36) Respiratory death, LBW: 1.05 (0.91, 1.22) All other causes: 1.00 (0.99, 1.00)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Woodruff et al (2008, 098386) **Period of Study:** 1999-2002 **Location:** US counties with > 250,000 residents (96 counties)	**Outcome:** Postneonatal deaths Respiratory mortality (ICD10: J000-99, plus bronchopulmonary dysplasia [BPD] P27.1) SIDS (ICD10: R95) Ill-defined causes (R99); All other deaths evaluated as a control category **Age Groups:** Infants aged > 28 days and < 1 yr **Study Design:** Cross-sectional **N:** 3,583,495 births (6,639 post neonatal deaths) **Statistical Analyses:** Logistic GEE (exchangeable correlation structure) **Covariates:** maternal race/ethnicity, marital status, age, education, primiparity, county-level poverty and per capita income levels, year and month of birth dummy variables to account for time trend and seasonal effects, and region of the country sensitivity analyses performed among only those mothers with smoking information (adjustment for smoking had no effect on the estimates) **Season:** Adjusted for year and month of birth dummy variables to account for time trend and seasonal effects **Dose-response Investigated?** Evaluated the appropriateness of a linear form from analysis based on quartiles of exposure and concluded that linear form was appropriate (data not shown) **Statistical Package:** SAS	**Pollutant:** PM_{10} **Averaging Time:** Measured continuously for 24 h once every 6 days exposure assigned by calculating avg concentration of pollutant during first 2 months of life **Median and IQR (25th-75th percentile):** Survivors: 28.9 (23.3-34.4) All causes of death: 29.1 (23.9-34.5) Respiratory: 29.8 (24.3-36.5) SIDS: 28.6 (23.5-33.8) SIDS + ill-defined: 28.8 (23.9-33.9) Other causes: 29.2 (23.9-34.5) **Percentiles:** see above **PM Component:** Not assessed, but controlled for region of the country to account for PM composition variation **Monitoring Stations:** NR **Copollutant (correlation):** PM_{10} $PM_{2.5}$ (r = 0.34) CO (r = 0.18) SO_2 (r = 0.00) O_3 (r = 0.20) **Notes:** Monthly averages calculated if there were at least 3 available measures for PM Assigned exposures using the avg concentration of the county of residence	**PM Increment:** IQR (11 $\mu g/m^3$) **Effect Estimate [Lower CI, Upper CI]:** Adjusted ORs for single pollutant models All causes: 1.04 (0.99, 1.10) Respiratory: 1.18 (1.06, 1.31) SIDS: 1.02 (0.89, 1.16) Ill-defined + SIDS: 1.06 (0.97, 1.16) Other causes: 1.02 (0.96, 1.07) Adjusted ORs for multipollutant models (including CO, O_3, SO_2) Respiratory: 1.16 (1.04, 1.30) SIDS: 1.02 (0.90, 1.16) OR for deaths coded as BPD per increase in IQR: 1.19 (0.85, 1.65) OR for respiratory post neonatal death stratified by birth weight NBW only: 1.19 (1.05, 1.36) LBW only: 1.12 (0.95, 1.31) OR for respiratory deaths removing region of US as a confounding variable: 1.30 (1.04, 1.61) OR for respiratory deaths assessing exposure as quartiles Highest vs Lowest quartile: 1.31 (1.00, 1.71) OR for respiratory deaths among only those deaths that occurred during the first 90 days (most closely matched exposure metric of the avg over the first 2 months of life): 1.25 (1.06, 1.47)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Jedrychowski, et al., (2007, 156607) **Period of Study:** Jan 2001-Feb 2004 **Location:** Krakow, Poland	**Outcome:** Birth weight (grams), birth length (cm) **Age Groups:** pregnant women 18-35 years **Study Design:** Prospective cohort **N:** 493 women **Statistical Analyses:** Linear regression **Covariates:** Environmental tobacco smoke (# cigarettes smoked daily in presence of pregnant woman), season of birth, size of mother, parity, gestational age, gender of child, vitamin A intake **Season:** All **Dose-response Investigated?** Yes **Statistical Package:** NR **Lags Considered:** Two consecutive days in the second trimester	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 48 h period **Percentiles: 50th(Median):** 35.3 **Range (Min, Max):** 10.3, 294.9 **Monitoring Stations:** No stations, personal monitoring **Notes:** PM measured during a two day period in the second trimester by Personal Environmental Monitoring Sampler (PEMS)	**PM Increment:** in 1 $\mu g/m^3$ and tertiles T1: < 27.0 $\mu g/m^3$ T2: 27.0-46.2 $\mu g/m^3$ T3: ≥ 46.2 $\mu g/m^3$ **Mean [Lower CI, Upper CI]:** Birth weight (g) For ln unit PM: β = -172.39 (p = 0.02) Tertiles: T1: ref T2: β = -16.510 [-94.630, 61.610] T3: β = -109.956 [-196.649 to -23.263] In low Vitamin A group (< 1,378 μg) T1: ref T2: β = -68.354 [-165.643, 28.935] T3: β = -185.070 [-293.393 to -76.747] In high Vitamin A group (> 1,378 μg) T1: ref T2: β = 64.262 [-70.464, 198.988] T3: β = 38.593 [-109.853, 187.039] Birth length (cm) For ln unit PM: β = -1.39 (p = 0.00) Tertiles: T1: ref T2: β = -0.288 [-0.790, 0.214] T3: β = -0.810 [-1.367 to -0.253] In low Vitamin A group (< 1,378 μg) T1: ref T2: β = -0.514 [-1.114, 0.086] T3: β = -1.100 [-1.768 to -0.432] In high Vitamin A group (> 1,378 μg) T1: ref T2: β = 0.039 [-0.896, 0.974] T3: β = -0.301 [-1.326, 0.724]

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: (Lipfert et al., 2000, 004103) **Period of Study:** 1990 **Location:** U.S.	**Outcome (ICD9 and ICD10):** Infant mortality including respiratory mortality (traditional definition, ICD9 460-519), expanded definition (adds ICD9 769 and 770) **Age Groups:** Infants **Study Design:** Cross-sectional **N:** 2,413,762 infants in 180 counties (Ns differ for various models) **Statistical Analyses:** Logistic regression **Covariates:** mother's smoking, education, marital status, and race month of birth and county avg heating degree days **Dose-response Investigated?** NR **Statistical Package:** NR	**Pollutant:** SO_4^{2-}/ $NSPM_{10}$ (regressed jointly) **Averaging Time:** Yearly avg used **Mean (SD):** 33.1 (9.17) (based on 180 counties) **Range (Min, Max):** (16.9, 59) **Monitoring Stations:** NR **Copollutant:** PM_{10} $NSPM_{10}$ CO SO_2 **Notes:** TSP-based sulfate was adjusted for compatibility with the PM_{10}-based data	**PM Increment:** NR (present regression coefficients) **Effect Estimate [Lower CI, Upper CI]:** Presented regression coefficients (standard errors) (3 PM exposures regressed jointly) bold = $p < 0.05$ Cause of death: All Birth weight: All SO_4^{2-}: -0.0002 (0.0061) $NSPM_{10}$: 0.0115 (0.0014) Cause of death: All Birth weight: LBW SO_4^{2-}: 0.0265 (0.0080) $NSPM_{10}$: 0.0086 (0.0020) Cause of death: All Birth weight: normal SO_4^{2-}: -0.0488 (0.0098) $NSPM_{10}$: 0.0096 (0.0024) Cause of death: All neonatal Birth weight: All SO_4^{2-}: 0.0267 (0.0076) $NSPM_{10}$: 0.0126 (0.0018) Cause of death: All neonatal Birth weight: LBW SO_4^{2-}: 0.0388 (0.0088) $NSPM_{10}$: 0.0093 (0.0022) Cause of death: All neonatal Birth wt: normal SO_4^{2-}: -0.0334 (0.0169) $NSPM_{10}$: 0.0125 (0.0040) Cause of death: All post neonatal Birth wt: All PM_{10}: 0.0091 (0.0024) SO_4^{2-}: -0.0474 (0.0100) $NSPM_{10}$: 0.0096 (0.0024) Cause of death: All post neonatal Birth wt: LBW SO_4^{2-}: -0.0247 (0.0173) $NSPM_{10}$: 0.0101 (0.0042) Cause of death: All post neonatal Birth wt: normal SO_4^{2-}: -0.0569 (0.0121) $NSPM_{10}$: 0.0080 (0.0029) Cause of death: SIDS Birth weight: All SO_4^{2-}: -0.1078 (0.0151) $NSPM_{10}$: 0.0149 (0.0037) Cause of death: SIDS Birth weight: LBW SO_4^{2-}: -0.1378 (0.0337) $NSPM_{10}$: 0.0146 (0.0085)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: (Liu et al., 2007, 090429) **Period of Study:** 1985-2000 **Location:** 3 Canadian cities: Calgary, Edmonton, and Montreal	**Outcome:** intrauterine growth restriction (IUGR) **Age Groups:** singleton term live births (37-42 wks gestation) **Study Design:** retrospective cohort **N:** 386,202 singleton live births **Statistical Analyses:** Multiple logistic regression **Covariates:** maternal age, parity, infant gender, season, and city of residence at time period of birth **Season:** All seasons **Dose-response Investigated?** No **Statistical Package:** NR	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h (6-day schedule) **Mean (SD):** 12.2 **Percentiles: 25th:** 6.3 **50th(Median):** 9.7 **75th:** 15 **PM Component:** metals and organic matter such as polycyclic aromatic hydrocarbons **Monitoring Stations:** Calgary (4), Edmonton (2), and Montreal (8) **Copollutant (correlation):** SO_2: $r = 0.44$, $p < 0.0001$ NO_2: $r = 0.41$, $p < 0.0001$ CO: $r = 0.31$, $p < 0.0001$ O_3: $r = -0.14$, $p < 0.0001$	**PM Increment:** 10 $\mu g/m^3$ **Effect Estimate** **single-pollutant model [Lower CI, Upper CI]:** 1st trimester OR = 1.07 (1.03-1.10) 2nd trimester OR = 1.06 (1.03-1.10) 3rd trimester OR = 1.06 (1.03-1.10) **Effect Estimate** **multi-pollutant model [Lower CI, Upper CI]:** 1st trimester OR= 1.03 (0.99-1.06) 2nd trimester OR= 1.01 (0.98-1.05) 3rd trimester OR= 1.03 (0.99-1.06) **Note:** ORs and CIs estimated from Figs. 6 and 7

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Loomis et al. (1999, 087288) **Period of Study:** Jan 1, 1993–Jul 31,1995 **Location:** Mexico City (southwestern section)	**Outcome (ICD9 and ICD10):** Infant mortality (daily counts of deaths) All ICD9 codes, excluding accidents, poisoning, and violence (ICD9 ≥800) **Age Groups:** Children < 1 yr of age **Study Design:** Time-series **N:** 942 deaths (days were the unit of observation) **Statistical Analyses:** Poisson regression (generalized additive model) **Covariates:** Final models controlled for mean temp of 3 days before death and nonparametrically smoothed periodic cycles **Season:** Yes (considered) **Dose-response Investigated?** Loess smoother **Statistical Package:** NR **Lags Considered:** 0-5 (also considered lags with avg exposure levels during "windows" of 2 to 4 days)	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-h **Mean (SD):** 27.4 (10.5) **Percentiles:** Lower quartile: 20 Median: 26 Upper quartile: 34 **Range (Min, Max):** 4, 85 **Monitoring Stations:** one **Copollutant:** O_3 NO_2 NO NO_X SO_2 **Notes:** Pearson correlation coefficients ranging from 0.52 to 0.71	**PM Increment:** 10 $\mu g/m^3$ **Effect Estimate [Lower CI, Upper CI]:** %Change in infant mortality Lags 0-5 (single day) presented in Figure 1: Lag0,1,2: No association (results not presented) Lag3: 4.8 (0.97, 8.61) Lag4: 4.2 (0.37, 7.93) %Change in mortality when avg exposure levels during "windows" of 2 to 4 days were considered Two Days: No lag: -1.36 (-5.51, 2.8) Lag1: -0.95 (-5.10, 3.20) Lag2: 2.78 (-1.33, 6.89) Lag3: 4.93 (0.86, 9.01) Three Days: No lag: -0.81 (-5.29, 3.67) Lag1: 1.99 (-2.46, 6.45) Lag2: 4.54 (0.12, 8.96) Lag3: 6.87 (2.48, 11.26) Four Days: No lag: 1.95 (-2.76, 6.66) Lag1: 3.74 (-0.95, 8.42) Lag2: 5.87 (1.21, 10.53) Multipollutant models (3-day mean w/ 3-day lag) 1 pollutant model: 6.87 (2.48, 11.26) 2 pollutant models: w/ O_3: 6.24 (1.35, 11.14) w/ NO_2: 5.91 (-0.76, 12.59) 3 pollutant model (w/ O_3 and NO_2): 6.30 (-0.54, 13.15)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Mannes et al. (2005, 087895) **Period of Study:** January 1, 1998-December 31, 2000 **Location:** metropolitan Sydney, Australia	**Outcome:** risk of small for gestational age (SGA) and birth weight **Age Groups:** all singleton births > 20 weeks and ≥ 400 grams birth weight and maternal all ages **Study Design:** cross-sectional **N:** 138,056 singleton births **Statistical Analyses:** Logistic and linear regression models **Covariates:** sex of child, maternal age, gestational age, maternal smoking, gestational age at first antenatal visit, maternal indigenous status, whether first pregnancy, season of birth, and socioeconomic status (SES) **Season:** All seasons included as covariate. **Dose-response Investigated?** No **Statistical Package:** SAS System for Windows v8.02	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean (SD):** 9.4 (5.1) **Percentiles: 25th:** 6.5 **50th(Median):** 8.4 **75th:** 11.2 **Range (Min, Max):** (2.4- 82.1) **Monitoring Stations:** up to 14 **Copollutant (correlation):** CO: r = 0.53 NO_2: r = 0.66 O_3: r = 0.36 PM_{10}: r = 0.81	**PM Increment:** 1 $\mu g/m^3$ Risk of SGA All births One month before birth: OR = 1.01 (0.99-1.03) Third trimester: OR = 0.99 (0.97-1.02) Second trimester: OR = 1.03 (1.01-1.05) First trimester: OR = 0.99 (0.97-1.01) 5 km births One month before birth: OR = 1.01 (0.97-1.04) Third trimester: OR = 1.00 (0.95-1.05) Second trimester: OR = 1.00 (0.96-1.05) First trimester: OR = 0.99 (0.94-1.04) Change in birth weight All births One month before birth: ß = -2.48 (-4.58- -0.38) Third trimester: ß = -0.98 (-3.74–1.78) Second trimester: ß = -4.10 (-6.79- -1.41) First trimester: ß = 0.36 (-2.29- 3.01) 5 km births One month before birth: ß = -2.70 (-6.80-1.40) Third trimester: ß = -2.83 (-9.00-3.34) Second trimester: ß = 1.54 (-4.59-7.67) First trimester: ß = 1.89 (-1.99-5.77)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Parker et al. (2005, 087462) **Period of Study:** 1999-2000 **Location:** California	**Outcome:** small for gestational age (SGA) and birth weight **Age Groups:** infants delivered at 40 weeks gestation maternal all ages **Study Design:** cross-sectional **N:** 18,247 singleton births **Statistical Analyses:** Linear and logistic regression models **Covariates:** maternal race, maternal Hispanic origin, marital status, parity, maternal education, and maternal age **Season:** season of delivery (covariate) **Dose-response Investigated?** Yes **Statistical Package:** STATA	**Pollutant:** $PM_{2.5}$ **Averaging Time:** NR (measurement taken every 6 days) **Mean (SD):** 15.42 (5.08) **PM Component:** metals, polycyclic aromatic hydrocarbons **Monitoring Stations:** 40 **Copollutant (correlation):** $PM_{2.5}$-CO: r = 0.6 **Notes:** Mean calculated for 9-month exposure. The following means (SDs) are calculated for trimester: First: 15.70 (6.26) Second: 15.40 (6.53) Third: 14.29 (6.35) PM categorized into quartiles: Q1: < 11.9 Q2: 11.9-13.9 Q3: 13.9-18.4 Q4: > 18.4	**PM Increment:** < 11.9 $\mu g/m^3$ Referent **PM Increment:** 11.9-13.9 $\mu g/m^3$ **Effect Estimate [Lower CI, Upper CI]:** First Trimester Birth weight: ß = -5.7 (-27.9-16.5) SGA: OR = 1.02 (0.84-1.23) Second Trimester Birth weight: ß = 11.3 (-12.2-34.9) SGA: OR = 0.89 (0.73-1.09) Third Trimester Birth weight: ß = 8.3 (-13.1-29.8) SGA: OR = 1.00 (0.83-1.19) **PM Increment:** 13.9-18.4 $\mu g/m^3$ **Effect Estimate [Lower CI, Upper CI]:** First Trimester Birth weight: ß = -2.5 (-24.5-19.5) SGA: OR = 1.12 (0.93-1.34) Second Trimester Birth weight: ß = -17.2 (-39.4-4.9) SGA: OR = 1.05 (0.88-1.26) Third Trimester Birth weight: ß = -8.1 (-30.2-13.9) SGA: OR = 0.98 (0.82-1.18) **PM Increment:** > 18.4 $\mu g/m^3$ **Effect Estimate [Lower CI, Upper CI]:** First Trimester Birth weight: ß = -35.8 (-58.4--13.3) SGA: OR = 1.26 (1.04-1.51) Second Trimester Birth weight: ß = -46.6 (-68.6- -24.6) SGA: OR = 1.24 (1.04-1.49) Third Trimester Birth weight: ß = -31.6 (-52.0- -11.1) SGA: OR = 1.21 (1.02-1.43)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: (Parker and Woodruff, 2008, 189095) **Period of Study:** 2001-2003 **Location:** US	**Outcome:** Low birth weight **Study Design:** cohort **N:** 785,965 Singleton births delivered at 40 weeks gestation **Statistical Analyses:** GEE regression models linear and logistic regression **Covariates:** race/ethnicity, parity, maternal age **Season:** season of delivery **Statistical Package:** SUDAAN	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 9-months **Mean (SD):** 14.5 25th: 12.1 75th: 17.6 **Copollutant (correlation):** SO_2, NO_2 CO O_3	**PM Increment:** 10 $\mu g/m^3$ **Change in Birth weight (9 month exposure):** Unadjusted: 19.4 (9.8, 29.0) Adjusted for maternal factors: 18.4 (9.2, 27.7) **Stratified by region:** Industrial Midwest: -15.3 (-43.4, 12.9) Northeast: -9.8 (-11.9, 26.6) Northwest: 27.5 (5.5, 49.4) Southern CA: 5.5 (-9.6, 20.5) Southeast: 7.3 (-11.9, 26.6) Southwest: 72.3 (34.0, 110.5) Upper Midwest: -0.7 (-62.0, 60.6) **Multipollutant models:** $PM_{2.5}$ + $PM_{10-2.5}$: 14.2 (4.3, 24.1) $PM_{2.5}$ + $PM_{10-2.5}$ + SO_2 + CO + NO_2 + O_3: 28.6 (14.2, 43.0)
Reference: Rich et al. (2009, 180122) **Period of Study:** 1999-2003 **Location:** New Jersey, United States	**Outcome:** Small for gestational age **Study Design:** Retrospective Cohort **Covariates:** month and calendar year of birth, apparent temperature, pregnancy complications **Statistical Analysis:** Polytomous logistic regression **Statistical Package:** SAS **Age Groups:** Gestational age 37-42 wks	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24h **Mean (SD) Unit:** *All values are for first trimester, other trimesters are available in paper Reference Births: 13.8 (2.5) SGA Births: 13.9 (2.5) VSGA Births: 13.9 (2.4) **Range (Min, Max):** 2.0, 29.0 **Copollutant (correlation):** *All values are for first trimester, other trimesters are available in paper NO_2: 0.01 SO_2: 0.17 CO: 0.25	*All values are for first trimester, other trimesters are available in paper **Increment:** 4 $\mu g/m^3$ **Percent Change in Risk (95% CI)** SGA: 4.5 (0.5-8.7) VSGA: 2.6 (-4.4-10.0) **Percent Change in Risk (95% CI) for single and two pollutant models** Single, SGA: 4.6 (-0.3-9.8) Single, VSGA: 4.5 (-4.0-13.4) Two ($PM_{2.5}$ & NO_2), SGA: 4.5 (-0.4-9.7) Two ($PM_{2.5}$ & NO_2), VSGA: 3.2 (-5.2-12.4) **Percent Change in Risk (95% CI) by pregnancy complication in third trimester** SGA Any Complication No: 4.7 (0.6-9.0) Yes: 2.2 (-6.1-11.3) Placental Abruption No: 4.0 (0.3-7.9) Yes: 11.7 (-21.7-59.5) Placental Praevia No: 3.9 (0.2-7.8) Yes: 23.2 (-20.9-91.9) Pre-eclampsia No: 4.2 (0.4-8.2) Yes: 2.7 (-13.8-22.3) Gestational Hypertension No: 4.3 (0.4-8.4) Yes: 3.9 (-7.8-17.1) Premature Rupture of the Membrane

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			No: 3.7 (-0.1-7.7)
			Yes: 14.6 (-3.3-35.9)
			Gestational Diabetes
			No: 4.6 (0.8-8.6)
			Yes: -9.3 (-24.7-9.3)
			VSGA
			Any Complication
			No: 1.5 (-6.1-9.7)
			Yes: 12.6 (0.1-26.7)
			Placental Abruption
			No: 4.1 (-2.6-11.2)
			Yes: 7.6 (-29.8-64.9)
			Placental Praevia
			No: 4.1 (-2.5-11.2)
			Yes: 3.2 (-43.0-86.9)
			Pre-eclampsia
			No: 4.4 (-2.6-11.9)
			Yes: 3.9 (-15.7-28.1)
			Gestational Hypertension
			No: 3.2 (-4.0-10.9)
			Yes: 12.9 (-3.3-31.9)
			Premature Rupture of the Membrane
			No: 3.3 (-3.5-10.5)
			Yes: 21.9 (-3.6-54.2)
			Gestational Diabetes
			No: 4.3 (-2.5-11.5)
			Yes: 1.4 (-27.0-40.9)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Ritz et al. (2007, 096146) **Period of Study:** Jan 1, 2003–Dec 31, 2003 **Location:** Los Angeles, California	**Outcome:** Preterm births (infants delivered before 37 weeks) **Age Groups:** Births **Study Design:** Case-control nested within a birth cohort (cases and controls matched on zip code and birth month) Phase 1: cross-sectional including all birth cohort Phase 2: nested case-control of survey respondents **N:** Phase 1: Birth cohort consisted of 58,316 eligible births. Phase II: 2,543 **Statistical Analyses:** Logistic regression **Covariates:** Birth certificate information: maternal age, race/ethnicity, parity, education, season of birth survey information: maternal smoking, alcohol consumption, living with a smoker, and marital status during pregnancy income (imputed) occupation and pregnancy weight gain considered but not included in final models **Season:** Yes **Dose-response Investigated?** Yes, examined categories of exposure **Statistical Package:** NR	**Pollutant:** $PM_{2.5}$ **Averaging Time:** daily or every 3rd day used to calculate the entire pregnancy, the first trimester, and the last 6 weeks before delivery only reported first trimester exposures for PM **Range (Min, Max):** NR Ranges for 3 categories reported: Low (ref): ◘ 18.63 Mid: 18.64-21.36 High: > 21.36 **Monitoring Stations:** Each zip code was linked to the nearest monitoring station (number not reported) **Copollutant (correlation):** CO NO_2 O_3 **Notes:** Daily or every 3rd day measurements used for mean calculations	**PM Increment:** Reported analyses using exposure categories **Effect Estimate [Lower CI, Upper CI]:** Birth cohort (phase I) Crude: Low: 1.0 Mid: 0.96 (0.90, 1.03) High: 1.05 (0.99, 1.12) Adj for birth cert Covariates: Low: 1.0 Mid: 1.01 (0.93, 1.09) High: 1.10 (1.01, 1.20) Survey respondents (phase II) Crude: Low: 1.0′ Mid: 1.11 (0.90, 1.36) High: 1.27 (1.06, 1.53) Adj for birth cert Covariates: Low: 1.0 Mid: 1.14 (0.90, 1.46) High: 1.27 (0.99, 1.64) Adj for all Covariates: Low: 1.0 Mid: 1.15 (0.90, 1.47) High: 1.29 (1.00, 1.67) Two-phase model: * Low: 1.0 Mid: 0.98 (0.84, 1.15) High: 1.07 (0.85, 1.35) *method to reduce potential selection bias and increase statistical efficiency
Reference: (Slama et al., 2007, 093216) **Period of Study:** 1/1998 -1/1999 **Location:** Munich, Germany	**Outcome:** Birth weight offspring at term **Study Design:** Cohort study **N:** 1016 births **Statistical Analyses:** Poisson model **Covariates:** Maternal passive smoking, maternal age, gestational duration, sex of child, parity, maternal education, maternal size, prepregnancy weight, other pollutants ($PM_{2.5}$, $PM_{2.5}$ absorbance, NO_2), season of conception **Dose-response Investigated?** Yes **Statistical Package:** STATA	**Pollutant:** $PM_{2.5}$ (estimated based on larger PM size fractions) **Averaging Time:** Entire pregnancy period and trimesters **Mean (SD):** 14.4 **Percentiles: 25th:** 13.5 **50th(Median):** 14.4 **75th:** 15.4 **Monitoring Stations:** Spatial component: 40 Temporal component: 1 **Copollutant (correlation):** p.a. = pregnancy avg trim. = trimester $PM_{2.5}$ (p.a.)–$PM_{2.5}$ (1st trim.): 0.85 $PM_{2.5}$ (p.a.)–$PM_{2.5}$ (2nd trim.): 0.77 $PM_{2.5}$ (p.a.)–$PM_{2.5}$ (3rd trim.): 0.87 $PM_{2.5}$ (p.a.)–NO_2 (p.a.): 0.45 $PM_{2.5}$ (p.a.)–NO_2 (1st trim.): 0.18 $PM_{2.5}$ (p.a.)–NO_2 (2nd trim.): 0.32 $PM_{2.5}$ (p.a.)–NO_2 (3rd trim.): 0.37 $PM_{2.5}$ (1st trim.)–$PM_{2.5}$ (2nd trim.): 0.40 $PM_{2.5}$ (1st trim.)–$PM_{2.5}$ (3rd trim.): 0.68	**PM Increment:** 1) 1 $\mu g/m^3$ 2) Quartiles: a) 1st (reference) (7.2–13.5 $\mu g/m^3$) b) 2nd (13.5–14.4 $\mu g/m^3$) c) 3rd (14.4–15.4 $\mu g/m^3$) d) 4th (15.41–17.5 $\mu g/m^3$) **Prevalence ratios (PRs) of birth weight < 3000 g during exposure over the whole pregnancy** **Single-pollutant models** Unadjusted models 2nd quartile: 1.07 (0.65, 1.73); 3rd quartile: 1.38 (0.91, 2.09) 4th quartile: 1.45 (0.92, 2.25) Per 1 $\mu g/m^3$: 1.06 (0.95, 1.19) Adjusted models 2nd quartile: 1.08 (0.63, 1.82); 3rd quartile: 1.34 (0.86, 2.13) 4th quartile: 1.73 (1.15, 2.69); Per 1 $\mu g/m^3$: 1.13 (1.00, 1.29) **Multipollutant models** Adjusted models 2nd quartile: 1.01 (0.57, 1.85) 3rd quartile: 1.12 (0.64, 1.87) 4th quartile: 1.36 (0.72, 2.45); Per

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
		$PM_{2.5}$ (1st trim.)–NO_2 (p.a.): 0.48	1 $\mu g/m^3$: 1.07 (0.91, 1.26)
		$PM_{2.5}$ (1st trim.)–NO_2 (1st trim.): 0.15	**Single-pollutant models** (restricted analysis to $PM_{2.5}$ absorbance below the median)
		$PM_{2.5}$ (1st trim.)–NO_2 (2nd trim.): 0.41	Per 1 $\mu g/m^3$: 1.15 (0.89, 1.52)
		$PM_{2.5}$ (1st trim.)–NO_2 (3rd trim.): 0.39	**Prevalence ratios (PRs) of birth weight < 3000 g**
		$PM_{2.5}$ (2nd trim.)–$PM_{2.5}$ (3rd trim.): 0.51	**Multipollutant models (simultaneous adjustment of 3rd trimester $PM_{2.5}$ and whole pregnancy $PM_{2.5}$)**
		$PM_{2.5}$ (2nd trim.)–NO_2 (p.a.): 0.23	$PM_{2.5}$ (whole pregnancy)
		$PM_{2.5}$ (2nd trim.)–NO_2 (1st trim.): -0.03	Per 1 $\mu g/m^3$: 0.96 (0.75, 1.19)
		$PM_{2.5}$ (2nd trim.)–NO_2 (2nd trim.): 0.17	$PM_{2.5}$ (3rd trimester)
		$PM_{2.5}$ (2nd trim.)–NO_2 (3rd trim.): 0.30	Per 1 $\mu g/m^3$: 1.17 (0.98, 1.40)
		$PM_{2.5}$ (3rd trim.)–NO_2 (p.a.): 0.39	**Prevalence ratios (PRs) of birth weight < 3000 g during exposure over the whole pregnancy (adjustment for season of conception)**
		$PM_{2.5}$ (3rd trim.)–NO_2 (1st trim.): 0.33	4th quartile: 1.68 (1.05, 2.75); Per 1 $\mu g/m^3$: 1.12 (0.97, 1.28)
		$PM_{2.5}$ (3rd trim.)–NO_2 (2nd trim.): 0.21	**Prevalence ratios (PRs) of birth weight < 3000 g during exposure over first trimester of pregnancy**
		$PM_{2.5}$ (3rd trim.)–NO_2 (3rd trim.): 0.23	Each trimester separately
		$PM_{2.5}$ (p.a.)- $PM_{2.5}$ absorbance (p.a.): 0.69	2nd quartile: 1.14 (0.74, 1.96); 3rd quartile: 1.28 (0.84, 2.10)
		$PM_{2.5}$ (p.a.)- $PM_{2.5}$ abs (1st trim.): 0.33	4th quartile: 1.65 (1.02, 2.60)
		$PM_{2.5}$ (p.a.)- $PM_{2.5}$ abs (2nd trim.): 0.48	Per 1 $\mu g/m^3$: 1.10 (0.99, 1.20)
		$PM_{2.5}$ (p.a.)- $PM_{2.5}$ abs (3rd trim.): 0.52	All trimesters adjusted simultaneously
		$PM_{2.5}$ (1st trim.)- $PM_{2.5}$ abs (p.a.): 0.68	2nd quartile: 0.97 (0.60, 1.73); 3rd quartile: 0.98 (0.57, 1.75)
		$PM_{2.5}$ (1st trim.)- $PM_{2.5}$ abs (1st trim.): 0.27	4th quartile: 1.22 (0.71, 2.18)
		$PM_{2.5}$ (1st trim.)- $PM_{2.5}$ abs (2nd trim.): 0.53	Per 1 $\mu g/m^3$: 1.03 (0.90, 1.17)
		$PM_{2.5}$ (1st trim.)- $PM_{2.5}$ abs (3rd trim.): 0.51	**Prevalence ratios (PRs) of birth weight < 3000 g during exposure over second trimester of pregnancy**
		$PM_{2.5}$ (2nd trim.)- $PM_{2.5}$ abs(p.a.): 0.41	Each trimester separately
		$PM_{2.5}$ (2nd trim.)- $PM_{2.5}$ abs (1st trim.): 0.08	2nd quartile: 0.83 (0.52, 1.32); 3rd quartile: 1.08 (0.71, 1.60)
		$PM_{2.5}$ (2nd trim.)- $PM_{2.5}$ abs (2nd trim.): 0.29	4th quartile: 0.94 (0.61, 1.47)
		$PM_{2.5}$ (2nd trim.)- $PM_{2.5}$ abs (3rd trim.): 0.41	Per 1 $\mu g/m^3$: 1.01 (0.92, 1.12)
		$PM_{2.5}$ (3rd trim.)- $PM_{2.5}$ abs (p.a.): 0.62	All trimesters adjusted simultaneously
		$PM_{2.5}$ (3rd trim.)- $PM_{2.5}$ abs (1st trim.): 0.48	2nd quartile: 0.75 (0.46, 1.24)
		$PM_{2.5}$ (3rd trim.)- $PM_{2.5}$ abs (2nd trim.): 0.36	3rd quartile: 0.86 (0.56, 1.30);
		$PM_{2.5}$ (3rd trim.)- $PM_{2.5}$ abs (3rd trim.): 0.37	4th quartile: 0.75 (0.48, 1.23)
			Per 1 $\mu g/m^3$: 0.94 (0.84, 1.06)
			Prevalence ratios (PRs) of birth weight < 3000 g during exposure over third trimester of pregnancy
			Each trimester separately
			2nd quartile: 1.30 (0.80, 2.17)
			3rd quartile: 1.44 (0.85, 2.27)
			4th quartile: 1.90 (1.20, 2.82)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			Per 1 μg/m^3: 1.14 (1.02, 1.24) All trimesters adjusted simultaneously 2nd quartile: 1.34 (0.79, 2.30) 3rd quartile: 1.48 (0.86, 2.58) 4th quartile: 1.91 (1.00, 3.20) Per 1 μg/m^3: 1.14 (0.99, 1.29) **Prevalence ratios (PRs) of birth weight < 3000 g during exposure over third trimester of pregnancy (adjustment for season of conception)** All trimesters adjusted simultaneously Per 1 μg/m^3: 1.25 (1.04, 1.50) **Sensitivity analysis**(bootstrapped PR) 2nd quartile: 0.98 (0.63, 1.61); 3rd quartile: 1.22 (0.82, 2.02) 4th quartile: 1.57 (1.02, 2.57) Per 1 μg/m^3: 1.11 (0.98, 1.27) **Estimated increments in prevalence of birth weight of < 3000 g during exposure 9 months after birth** Per 1 μg/m^3: 7% (-7%, 22%)
Reference: (Slama et al., 2007, 093216) **Period of Study:** 1/1998 -1/1999 **Location:** Munich, Germany	**Outcome:** Birth weight offspring at term **Study Design:** Cohort study **N:** 1016 births **Statistical Analyses:** Poisson model **Covariates:** Maternal passive smoking, maternal age, gestational duration, sex of child, parity, maternal education, maternal size, prepregnancy weight, other pollutants ($PM_{2.5}$, $PM_{2.5}$ absorbance, NO_2), season of conception **Dose-response Investigated?** Yes **Statistical Package:** STATA	**Pollutant:** $PM_{2.5}$ absorbance (estimated) **Averaging Time:** Entire pregnancy period and trimesters **Mean (SD):** 1.76 * **Percentiles: 25th:** 1.61* **50th(Median):** 1.72* **75th:** 1.89 * **Unit (i.e. μg/m^3):** 10^{-5}/m **Monitoring Stations:** Spatial component: 40 Temporal component: 1 **Copollutant (correlation):** p.a. = pregnancy avg trim. = trimester abs = absorbance $PM_{2.5}$ abs (p.a.)–$PM_{2.5}$ abs (1st trim.): 0.54 $PM_{2.5}$ abs (p.a.)–$PM_{2.5}$ abs (2nd trim.): 0.84 $PM_{2.5}$ abs (p.a.)–$PM_{2.5}$ abs (3rd trim.): 0.55 $PM_{2.5}$ abs (p.a.)–$PM_{2.5}$ (p.a.): 0.69 $PM_{2.5}$ abs (p.a.)–$PM_{2.5}$ (1st trim.): 0.68 $PM_{2.5}$ abs (p.a.)–$PM_{2.5}$ (2nd trim.): 0.41 $PM_{2.5}$ abs (p.a.)–$PM_{2.5}$ (3rd trim.): 0.62 $PM_{2.5}$ abs (p.a.)–NO_2 (p.a.): 0.67 $PM_{2.5}$ abs (p.a.)–NO_2 (1st trim.): 0.34 $PM_{2.5}$ abs (p.a.)–NO_2 (2nd trim.): 0.63 $PM_{2.5}$ abs (p.a.)–NO_2 (3rd trim.): 0.36 $PM_{2.5}$ abs (1st trim.)–$PM_{2.5}$ abs (2nd trim.): 0.32 $PM_{2.5}$ abs (1st trim.)–$PM_{2.5}$ abs (3rd trim.): -	**PM Increment:** 1) 0.5 * 10^{-5}/m 2) Quartiles: a) 1st (reference) (1.29–1.61) b) 2nd (1.61–1.72) c) 3rd (1.72–1.89) d) 4th (1.89–3.10) **Prevalence ratios (PRs) of birth weight < 3000 g during exposure over the whole pregnancy** **Single-pollutant models** Unadjusted models 2nd quartile: 1.19 (0.74, 1.99) 3rd quartile: 1.56 (0.98, 2.50); 4th quartile: 1.52 (0.96, 2.46) Per 0.5 * 10^{-5}/m: 1.25 (0.90, 1.70) Adjusted models 2nd quartile: 1.21 (0.73, 1.97) 3rd quartile: 1.63 (0.98, 2.57); 4th quartile: 1.78 (1.10, 2.70) Per 0.5 * 10^{-5}/m: 1.45 (1.06, 1.87) **Multipollutant models** Adjusted models 2nd quartile: 1.19 (0.70, 2.01) 3rd quartile: 1.55 (0.80, 2.80); 4th quartile: 1.46 (0.67, 2.90) Per 0.5 * 10^{-5}/m: 1.33 (0.76, 2.38) **Prevalence ratios (PRs) of birth weight < 3000 g during exposure over the whole pregnancy (adjustment for season of conception)** 4th quartile: 1.72 (1.08, 2.73) Per 0.5 * 10^{-5}/m: 1.38 (0.96, 1.86)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
		0.26 $PM_{2.5}$ abs (1st trim.)–$PM_{2.5}$ (p.a.): 0.33 $PM_{2.5}$ abs (1st trim.)–$PM_{2.5}$ (1st trim.): 0.27 $PM_{2.5}$ abs (1st trim.)–$PM_{2.5}$ (2nd trim.): 0.08 $PM_{2.5}$ abs (1st trim.)–$PM_{2.5}$ (3rd trim.): 0.48 $PM_{2.5}$ abs (1st trim.)–NO_2 (p.a.): 0.29 $PM_{2.5}$ abs (1st trim.)–NO_2 (1st trim.): 0.84 $PM_{2.5}$ abs (1st trim.)–NO_2 (2nd trim.): 0.16 $PM_{2.5}$ abs (1st trim.)–NO_2 (3rd trim.): -0.39 $PM_{2.5}$ abs (2nd trim.)–$PM_{2.5}$ abs (3rd trim.): 0.31 $PM_{2.5}$ abs (2nd trim.)–$PM_{2.5}$ (p.a.): 0.48 $PM_{2.5}$ abs (2nd trim.)–$PM_{2.5}$ (1st trim.): 0.53 $PM_{2.5}$ abs (2nd trim.)–$PM_{2.5}$ (2nd trim.): 0.29 $PM_{2.5}$ abs (2nd trim.)–$PM_{2.5}$ (3rd trim.): 0.36 $PM_{2.5}$ abs (2nd trim.)–NO_2 (p.a.): 0.61 $PM_{2.5}$ abs (2nd trim.)–NO_2 (1st trim.): 0.19 $PM_{2.5}$ abs (2nd trim.)–NO_2 (2nd trim.): 0.85 $PM_{2.5}$ abs (2nd trim.)–NO_2 (3rd trim.): 0.17 $PM_{2.5}$ abs (3rd trim.)–$PM_{2.5}$ (p.a.): 0.52 $PM_{2.5}$ abs (3rd trim.)–$PM_{2.5}$ (1st trim.): 0.51 $PM_{2.5}$ abs (3rd trim.)–$PM_{2.5}$ (2nd trim.): 0.41 $PM_{2.5}$ abs (3rd trim.)–$PM_{2.5}$ (3rd trim.): 0.37 $PM_{2.5}$ abs (3rd trim.)–NO_2 (p.a.): 0.40 $PM_{2.5}$ abs (3rd trim.)–NO_2 (1st trim.): -0.34 $PM_{2.5}$ abs (3rd trim.)–NO_2 (2nd trim.): 0.21 $PM_{2.5}$ abs (3rd trim.)–NO_2 (3rd trim.): 0.88	**Prevalence ratios (PRs) of birth weight < 3000 g during exposure over the whole pregnancy** **Single-pollutant models** (restricted analysis to $PM_{2.5}$ below the median) Per 0.5 * 10^{-5}/m: 1.67 (0.66, 3.73) **Prevalence ratios (PRs) of birth weight < 3000 g during exposure over first trimester of pregnancy** Each trimester separately 2nd quartile: 1.15 (0.73, 1.80) 3rd quartile: 1.01 (0.61, 1.53); 4th quartile: 1.04 (0.70, 1.57) Per 0.5 * 10^{-5}/m: 1.03 (0.82, 1.28) All trimesters adjusted simultaneously 2nd quartile: 0.90 (0.52, 1.58) 3rd quartile: 0.82 (0.45, 1.31); 4th quartile: 0.88 (0.53, 1.42) Per 0.5 * 10^{-5}/m: 1.02 (0.77, 1.29) **Prevalence ratios (PRs) of birth weight < 3000 g during exposure over second trimester of pregnancy** Each trimester separately 2nd quartile: 1.33 (0.85, 2.22) 3rd quartile: 1.76 (1.07, 2.91); 4th quartile: 1.83 (1.11, 2.81) Per 0.5 * 10^{-5}/m: 1.27 (1.04, 1.54) All trimesters adjusted simultaneously 2nd quartile: 1.30 (0.77, 2.16) 3rd quartile: 1.63 (0.93, 2.73); 4th quartile: 1.99 (1.12, 3.33) Per 0.5 * 10^{-5}/m: 1.21 (0.93, 1.54) **Prevalence ratios (PRs) of birth weight < 3000 g during exposure over third trimester of pregnancy** Each trimester separately 2nd quartile: 1.30 (0.85, 2.09) 3rd quartile: 0.92 (0.55, 1.50); 4th quartile: 1.50 (1.00, 2.27) Per 0.5 * 10^{-5}/m: 1.20 (0.98, 1.44) All trimesters adjusted simultaneously 2nd quartile: 0.99 (0.64, 1.62) 3rd quartile: 0.71 (0.40, 1.20); 4th quartile: 1.14 (0.68, 1.91) Per 0.5 * 10^{-5}/m: 1.15 (0.92, 1.42) **Prevalence ratios (PRs) of birth weight < 3000 g during exposure over first trimester of pregnancy** **(adjustment for season of conception)**

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			All trimesters adjusted simultaneously 4th quartile: 0.73 (0.38, 1.38) Per 0.5 * 10^{-5}/m: 0.93 (0.41, 1.32) **Prevalence ratios (PRs) of birth weight < 3000 g during exposure over second trimester of pregnancy (adjustment for season of conception)** All trimesters adjusted simultaneously 4th quartile: 2.45 (1.22, 4.77) Per 0.5 * 10^{-5}/m: 1.14 (0.70, 1.64) **Prevalence ratios (PRs) of birth weight < 3000 g during exposure over third trimester of pregnancy** **(adjustment for season of conception)** All trimesters adjusted simultaneously 4th quartile: 1.19 (0.60, 2.48) Per 0.5 * 10^{-5}/m: 1.29 (0.90, 1.75) **Sensitivity analysis** (bootstrapped PR) 2nd quartile: 1.19 (0.76, 1.91) 3rd quartile: 1.52 (0.99, 2.34); 4th quartile: 1.62 (1.06, 2.55) Per 0.5 * 10^{-5}/m: 1.35 (1.01, 1.83) **Estimated increments in prevalence of birth weight < 3000 g during exposure 9 months after birth** Per 0.5 * 10^{-5}/m: 18% (-16%, 57%)
Reference: Wilhelm et al. (2005, 088668) **Period of Study:** 1994-2000 **Location:** Los Angeles County, California, U.S.	**Outcome:** Term low birth weight (LBW) (< 2500 g at ≥ 37 completed weeks gestation) Vaginal birth < 37 completed weeks gestation **Age Groups:** LBW: ≥ 37 completed weeks Preterm births: < 37 completed weeks **Study Design:** cross-sectional study **N:** For LBW: 136,134 For preterm birth: 106,483 **Statistical Analyses:** Logistic regression **Covariates:** Maternal age, maternal race, maternal education, parity, interval since previous live birth, level of prenatal care, infant sex, previous LBW or preterm infant, birth season, other pollutants (not specified in birth weight analyses, also adjusted for gestational age) **Dose-response Investigated?** Yes **Statistical Package:** NR	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24hr (every 3 days) Entire pregnancy Trimesters of pregnancy Months of pregnancy 6 weeks before birth **Mean (SD):** First trimester: 21.9 Third trimester: 21.0 6 weeks before birth: 21.0 **Range (Min, Max):** First trimester: 11.8-38.9 Third trimester: 11.8-.38.9 6 weeks before birth: 9.9-48.5 **Monitoring Stations:** Zip-code-level analysis: 9 Address-level analysis: 8 **Copollutant (correlation):** First trimester $PM_{2.5}$-CO: 0.57 $PM_{2.5}$-NO_2: 0.73 $PM_{2.5}$-O_3: -0.55 $PM_{2.5}$-PM_{10}: 0.43 Third trimester: $PM_{2.5}$-CO: 0.67	**PM Increment:** 1) 10 $\mu g/m^3$ 2) 3 levels: a) < 25%ile (reference) b) 25%-75%ile c) ≥ 75%ile **Incidence of LBW (third trimester exposure)** < 17.1 $\mu g/m^3$: 2.4 (2.0, 2.8) 17.1 to < 24.0 $\mu g/m^3$: 2.2 (2.0, 2.5) ≥ 24.0 $\mu g/m^3$: 2.1 (1.7, 2.4) **Incidence of preterm birth (first trimester exposure)** < 18.0 $\mu g/m^3$: 10.6 (9.6, 11.7) 18.0 to < 25.4 $\mu g/m^3$: 8.8 (8.1, 9.5) ≥ 25.4 $\mu g/m^3$: 9.0 (8.1, 10.0) **Incidence of preterm birth (6 weeks before birth exposure)** < 16.5 $\mu g/m^3$: 8.2 (7.4, 9.1) 16.5 to < 24.7 $\mu g/m^3$: 8.8 (8.2, 9.4) ≥ 24.7 $\mu g/m^3$: 9.6 (8.7, 10.5) **Outcome: Preterm birth** **Exposure Period: First trimester of pregnancy** **Address-level analysis:** Single-pollutant model: Distance ◘ 1 mile

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
		$PM_{2.5}$-NO_2: 0.78	Per 10 $\mu g/m^3$: 0.85 (0.70, 1.02)
		$PM_{2.5}$-O_3: -0.60	18.1 to $<$ 25.2 $\mu g/m^3$: 0.91 (0.72, 1.16)
		$PM_{2.5}$-PM_{10}: 0.52	$\geq$ 25.2 $\mu g/m^3$: 0.83 (0.60, 1.14)
		6 weeks before birth: $PM_{2.5}$-CO: 0.63	Single-pollutant model: 1 $<$ distance $\leq$ 2 mile
		$PM_{2.5}$-NO_2: 0.74	Per 10 $\mu g/m^3$: 0.85 (0.74, 0.99)
		$PM_{2.5}$-O_3: -0.60	18.3 to $<$ 25.2 $\mu g/m^3$: 0.81 (0.69, 0.94)
		$PM_{2.5}$-PM_{10}: 0.60	$\geq$ 25.2 $\mu g/m^3$: 0.79 (0.65, 0.97)
			Multipollutant model1 $<$ distance $\leq$ 2 mile
			Per 10 $\mu g/m^3$: 1.18 (0.84, 1.65)
			Single-pollutant model: 2 $<$ distance $\leq$ 4 mile
			Per 10 $\mu g/m^3$: 0.83 (0.78, 0.88)
			18.5 to $<$ 24.9 $\mu g/m^3$: 0.79 (0.74, 0.85)
			$\geq$ 24.9 $\mu g/m^3$: 0.76 (0.70, 0.84)
			Zip-code-level analysis: Single-pollutant model: Per 10 $\mu g/m^3$: 0.73 (0.67, 0.80)
			18.0 to $<$ 25.4 $\mu g/m^3$: 0.70 (0.61, 0.80)
			$\geq$ 25.4 $\mu g/m^3$: 0.64 (0.53, 0.76)
			Outcome: Preterm birth
			Exposure Period: 6 weeks before birth
			Address-level analysis:
			Single-pollutant model: Distance $\leq$ 1 mile
			Per 10 $\mu g/m^3$: 1.09 (0.91, 1.30)
			16.8 to $<$ 24.1 $\mu g/m^3$: 1.21 (0.97, 1.51)
			$\geq$ 24.1 $\mu g/m^3$: 1.25 (0.93, 1.68)
			Single-pollutant model: 1 $<$ distance $\leq$ 2 mile
			Per 10 $\mu g/m^3$: 1.08 (0.97, 1.21)
			17.2 to $<$ 24.5 $\mu g/m^3$: 0.94 (0.82, 1.08)
			$\geq$ 24.5 $\mu g/m^3$: 1.04 (0.87, 1.24)
			Single-pollutant model: 2 $<$ distance $\leq$ 4 mile
			Per 10 $\mu g/m^3$: 1.05 (0.99, 1.10)
			17.3 to $<$ 24.6 $\mu g/m^3$: 1.06 (1.00, 1.13)
			$\geq$ 24.6 $\mu g/m^3$: 1.08 (0.99, 1.17)
			Zip-code-level analysis
			Single-pollutant model: Per 10 $\mu g/m^3$: 1.10 (1.00, 1.21)
			16.5 to $<$ 24.7 $\mu g/m^3$: 1.06 (0.94, 1.20)
			$\geq$ 24.7 $\mu g/m^3$: 1.19 (1.02, 1.40)
			(See Notes[1])
			Multipollutant model
			Per 10 $\mu g/m^3$: 1.12 (0.90, 1.40)
			$\geq$ 24.6 $\mu g/m^3$: 1.12 (0.82, 1.52)
			Notes: [1] In the table, the 75%ile is noted as 24.7 $\mu g/m^3$. However, the text notes the 75%ile as 24.3 $\mu g/m^3$.

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Woodruff et al. (2006, 088758) **Period of Study:** 1999-2000 **Location:** California	**Outcome (ICD10):** SIDS (R95) Respiratory mortality (J00-J99) Bronchopulmonary dysplasia (P27.1) External accidents (V01-Y98) Ill-defined and unspecified causes of mortality (R99) **Age Groups:** > 28 days old **Study Design:** Matched case-control (matched on date of birth and birth weight) **N:** 3877 infants **Statistical Analyses:** Conditional logistic regression **Covariates:** Maternal race, education, parity, age, marital status **Dose-response Investigated?** Yes **Statistical Package:** STATA	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 hrs (every 6 days) (time period between birth and post neonatal death for the infant who died and the same period for its four matched surviving infants) **Percentiles:** Infants who died of all causes (cases) **25th:** 13.4 **50th(Median):** 19.2 **75th:** 23.6 Matched controls **25th:** 13.5 **50th(Median):** 18.4 **75th:** 22.7 **Monitoring Stations:** 73 (from 39 counties)	**PM Increment:** 10 $\mu g/m^3$ **RR Estimate [Lower CI, Upper CI]** **lag:** All-cause mortality: Unadjusted: 1.15 (1.00, 1.32) Adjusted: 1.07 (0.93, 1.24) Cause-specific mortality: Respiratory (all): Unadjusted: 2.15 (1.15, 4.02) Adjusted: 2.13 (1.12, 4.05) Respiratory (excluding deaths due to BPD): Adjusted: 1.42 (0.66, 3.03) Respiratory (BPD alone): Unadjusted: 6.00 (1.40, 27.76) Respiratory (low birth weight infants only): Unadjusted: 3.09 (1.14, 8.40) Respiratory (normal birth weight infants only): Unadjusted: 1.66 (0.74, 3.70) Respiratory (with matched $PM_{2.5}$ averaged over all monitors in county) Adjusted: 2.28 (0.94, 5.52) Respiratory (averaging all $PM_{2.5}$ measurements in county over the 2-year study period): Adjusted: 2.26 (0.83, 6.21) SIDS: Unadjusted: 0.86 (0.61, 1.22) Adjusted: 0.82 (0.55, 1.23) SIDS (includes ICD10 code R99: ill-defined and unspecified causes of mortality): Adjusted: 1.03 (0.79, 1.35) External causes: Unadjusted: 0.91 (0.56, 1.47) Adjusted: 0.83 (0.50, 1.39) Compare against the lowest quartile, estimates for respiratory-specific mortality were provided: 2nd quartile: 1.28 (0.47, 3.51) 3rd quartile: 1.75 (0.65, 4.72) 4th quartile: 2.35 (0.85, 6.54)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Woodruff et al. (2008, 098386) **Period of Study:** 1999-2002 **Location:** US counties with > 250,000 residents (96 counties)	**Outcome (ICD10):** Postneonatal deaths: Respiratory mortality (J000-99, plus bronchopulmonary dysplasia [BPD] P27.1) SIDS (R95) Ill-defined causes (R99) All other deaths evaluated as a control category **Age Groups:** Infants aged > 28 days and < 1 yr **Study Design:** Cross-sectional **N:** 3,583,495 births (6,639 post neonatal deaths) **Statistical Analyses:** Logistic GEE (exchangeable correlation structure) **Covariates:** maternal race/ethnicity, marital status, age, education, primiparity, county-level poverty and per capita income levels, year and month of birth dummy variables to account for time trend and seasonal effects, and region of the country sensitivity analyses performed among only those mothers with smoking information (adjustment for smoking had no effect on the estimates) **Season:** Adjusted for year and month of birth dummy variables to account for time trend and seasonal effects **Dose-response Investigated?** Evaluated the appropriateness of a linear form from analysis based on quartiles of exposure and concluded that linear form was appropriate (data not shown) **Statistical Package:** SAS	**Pollutant:** $PM_{2.5}$ **Averaging Time:** Measured continuously for 24 h once every 6 days exposure assigned by calculating avg concentration of pollutant during first 2 months of life **Median and IQR (25th-75th percentile):** Survivors: 14.8 (11.7-18.7) All causes of death: 14.9 (12.0-18.6) Respiratory: 14.8 (11.5-18.5) SIDS: 14.5 (12.0-17.5) SIDS + ill-defined: 14.8 (12.1-18.5) Other causes: 14.9 (12.0-18.6) **Percentiles:** See above **PM Component:** Not assessed, but controlled for region of the country to account for PM composition variation **Monitoring Stations:** NR **Copollutant (correlation):** PM_{10} (r = 0.34) $PM_{2.5}$ CO (r = 0.35) SO_2 (r = 0.21) O_3 (r = -0.10) **Notes:** Monthly averages calculated if there were at least 3 available measures for PM Assigned exposures using the avg concentration of the county of residence	**PM Increment:** IQR (7 $\mu g/m^3$) **Effect Estimate [Lower CI, Upper CI]:** Adjusted ORs for single pollutant models All causes: 1.04 (0.98, 1.11) Respiratory: 1.11 (0.96, 1.29) SIDS: 1.01 (0.86, 1.20) Ill-defined + SIDS: 1.06 (0.97, 1.17) Other causes: 1.03 (0.96, 1.12) Adjusted ORs for multipollutant models (including CO, O_3, SO_2) Respiratory: 1.05 (0.89, 1.24) SIDS: 1.04 (0.87, 1.23) OR for respiratory deaths assessing exposure as quartiles Highest vs Lowest quartile: 1.39 (1.04, 1.85)

[1]All units expressed in $\mu g/m^3$ unless otherwise specified.

E.8. Long-Term Exposure and Mortality

Table E-30. Long-term exposure – mortality - PM_{10}.

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: (Breitner et al., 2009, 188439) **Period of Study:** 10/1/1991 to 3/31/2002 **Location:** Efurt, Germany	**Outcome:** Mortality, excluding infants and ICD-9 ≥ 800 **Study Design:** Time-series **Covariates:** seasonal and weekday variations, influenza epidemics, air temperature, relative humidity **Statistical Analysis:** semiparametric Poisson regression, polynomial distributed lag (PDL) **Statistical Package:** R **Age Groups:** All	**Pollutant:** PM_{10} **Averaging Time:** daily **Mean (SD) Unit:** 1 (10/1/1991-8/31/1995): 50.6 ± 32.2 $\mu g/m^3$ 2 (9/1/1995-2/28/1998): 41.1 ± 28.4 $\mu g/m^3$ 3 (3/1/1998-3/31/2002): 24.3 ± 15.4 $\mu g/m^3$ Total: 38.0 ±28.3 $\mu g/m^3$ **Range (Min, Max):** NR **Copollutant:** NO_2, CO, UFP	**Increment:** IQR **Relative Risk (95% CI)** **Lag** New City Limits 6-day IQR: 17.2 PDL: 0.997 (0.972-1.022) Mean of lags 0-5: 0.995 (0.971-1.019) Old City Limits 6-day IQR: 17.2 PDL: 1.004 (0.978-1.031) Mean of lags 0-5: 1.001 (0.976-1.027) New City Limits 15-day IQR: 14.5 PDL: 1.008 (0.982-1.036) Mean of lags 0-14: 1.006 (0.981-1.032) Old City Limits 15-day IQR: 14.5 PDL: 1.019 (0.991-1.048) Mean of lags 0-14: 1.017 (0.990-1.044) Multiday Moving Averages, 6-day Overall IQR: 24.2 Overall RR (95% CI): 0.998 (0.976-1.021) Period 1: 0.996 (0.969-1.024) Period 2: 1.013 (0.972-1.056) Period 3: 0.949 (0.897-1.004) Multiday Moving Averages, 15-day Overall IQR: 22.3 Overall RR (95% CI): 1.020 (0.993-1.093) Period 1: 1.017 (0.984-1.051) Period 2: 1.012 (0.973-1.071) Period 3: 0.978 (0.911-1.051)
Reference: (Slama et al., 2007, 093216) **Period of Study:** 1/1998 -1/1999 **Location:** Munich, Germany	**Outcome:** Birth weight offspring at term **Study Design:** Cohort study **N:** 1016 births **Statistical Analyses:** Poisson model **Covariates:** Maternal passive smoking, maternal age, gestational duration, sex of child, parity, maternal education, maternal size, prepregnancy weight, other pollutants ($PM_{2.5}$, $PM_{2.5}$ absorbance, NO_2),	**Pollutant:** $PM_{2.5}$ (estimated based on larger PM size fractions) **Averaging Time:** Entire pregnancy period and trimesters **Mean (SD):** 14.4 **Percentiles: 25th:** 13.5 **50th(Median):** 14.4 **75th:** 15.4	**PM Increment:** 1) 1 $\mu g/m^3$ 2) Quartiles: a) 1st (reference) (7.2–13.5 $\mu g/m^3$) b) 2nd (13.5–14.4 $\mu g/m^3$) c) 3rd (14.4–15.4 $\mu g/m^3$) d) 4th (15.41–17.5 $\mu g/m^3$) **Prevalence ratios (PRs) of birth weight < 3000 g during exposure over the**

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
	season of conception **Dose-response Investigated?** Yes **Statistical Package:** STATA	**Monitoring Stations:** Spatial component: 40 Temporal component: 1 **Copollutant (correlation):** p.a. = pregnancy avg trim. = trimester $PM_{2.5}$ (p.a.)–$PM_{2.5}$ (1st trim.): 0.85 $PM_{2.5}$ (p.a.)–$PM_{2.5}$ (2nd trim.): 0.77 $PM_{2.5}$ (p.a.)–$PM_{2.5}$ (3rd trim.): 0.87 $PM_{2.5}$ (p.a.)–NO_2 (p.a.): 0.45 $PM_{2.5}$ (p.a.)–NO_2 (1st trim.): 0.18 $PM_{2.5}$ (p.a.)–NO_2 (2nd trim.): 0.32 $PM_{2.5}$ (p.a.)–NO_2 (3rd trim.): 0.37 $PM_{2.5}$ (1st trim.)–$PM_{2.5}$ (2nd trim.): 0.40 $PM_{2.5}$ (1st trim.)–$PM_{2.5}$ (3rd trim.): 0.68 $PM_{2.5}$ (1st trim.)–NO_2 (p.a.): 0.48 $PM_{2.5}$ (1st trim.)–NO_2 (1st trim.): 0.15 $PM_{2.5}$ (1st trim.)–NO_2 (2nd trim.): 0.41 $PM_{2.5}$ (1st trim.)–NO_2 (3rd trim.): 0.39 $PM_{2.5}$ (2nd trim.)–$PM_{2.5}$ (3rd trim.): 0.51 $PM_{2.5}$ (2nd trim.)–NO_2 (p.a.): 0.23 $PM_{2.5}$ (2nd trim.)–NO_2 (1st trim.): -0.03 $PM_{2.5}$ (2nd trim.)–NO_2 (2nd trim.): 0.17 $PM_{2.5}$ (2nd trim.)–NO_2 (3rd trim.): 0.30 $PM_{2.5}$ (3rd trim.)–NO_2 (p.a.): 0.39 $PM_{2.5}$ (3rd trim.)–NO_2 (1st trim.): 0.33 $PM_{2.5}$ (3rd trim.)–NO_2 (2nd trim.): 0.21 $PM_{2.5}$ (3rd trim.)–NO_2 (3rd trim.): 0.23 $PM_{2.5}$ (p.a.)- $PM_{2.5}$ absorbance (p.a.): 0.69 $PM_{2.5}$ (p.a.)- $PM_{2.5}$ abs (1st trim.): 0.33 $PM_{2.5}$ (p.a.)- $PM_{2.5}$ abs (2nd trim.): 0.48 $PM_{2.5}$ (p.a.)- $PM_{2.5}$ abs (3rd trim.): 0.52 $PM_{2.5}$ (1st trim.)- $PM_{2.5}$ abs (p.a.): 0.68 $PM_{2.5}$ (1st trim.)- $PM_{2.5}$ abs (1st trim.): 0.27 $PM_{2.5}$ (1st trim.)- $PM_{2.5}$ abs (2nd trim.): 0.53 $PM_{2.5}$ (1st trim.)- $PM_{2.5}$ abs (3rd trim.): 0.51 $PM_{2.5}$ (2nd trim.)- $PM_{2.5}$ abs(p.a.): 0.41 $PM_{2.5}$ (2nd trim.)- $PM_{2.5}$ abs (1st trim.): 0.08 $PM_{2.5}$ (2nd trim.)- $PM_{2.5}$ abs (2nd trim.): 0.29 $PM_{2.5}$ (2nd trim.)- $PM_{2.5}$ abs (3rd trim.): 0.41 $PM_{2.5}$ (3rd trim.)- $PM_{2.5}$ abs (p.a.): 0.62 $PM_{2.5}$ (3rd trim.)- $PM_{2.5}$ abs (1st trim.): 0.48 $PM_{2.5}$ (3rd trim.)- $PM_{2.5}$ abs (2nd trim.): 0.36 $PM_{2.5}$ (3rd trim.)- $PM_{2.5}$ abs (3rd trim.): 0.37	**whole pregnancy** **Single-pollutant models** Unadjusted models 2nd quartile: 1.07 (0.65, 1.73); 3rd quartile: 1.38 (0.91, 2.09) 4th quartile: 1.45 (0.92, 2.25) Per 1 $\mu g/m^3$: 1.06 (0.95, 1.19) Adjusted models 2nd quartile: 1.08 (0.63, 1.82); 3rd quartile: 1.34 (0.86, 2.13) 4th quartile: 1.73 (1.15, 2.69); Per 1 $\mu g/m^3$: 1.13 (1.00, 1.29) **Multipollutant models** Adjusted models 2nd quartile: 1.01 (0.57, 1.85) 3rd quartile: 1.12 (0.64, 1.87) 4th quartile: 1.36 (0.72, 2.45); Per 1 $\mu g/m^3$: 1.07 (0.91, 1.26) **Single-pollutant models** (restricted analysis to $PM_{2.5}$ absorbance below the median) Per 1 $\mu g/m^3$: 1.15 (0.89, 1.52) **Prevalence ratios (PRs) of birth weight < 3000 g** **Multipollutant models (simultaneous adjustment of 3rd trimester $PM_{2.5}$ and whole pregnancy $PM_{2.5}$)** $PM_{2.5}$ (whole pregnancy) Per 1 $\mu g/m^3$: 0.96 (0.75, 1.19) $PM_{2.5}$ (3rd trimester) Per 1 $\mu g/m^3$: 1.17 (0.98, 1.40) **Prevalence ratios (PRs) of birth weight < 3000 g during exposure over the whole pregnancy (adjustment for season of conception)** 4th quartile: 1.68 (1.05, 2.75); Per 1 $\mu g/m^3$: 1.12 (0.97, 1.28) **Prevalence ratios (PRs) of birth weight < 3000 g during exposure over first trimester of pregnancy** Each trimester separately 2nd quartile: 1.14 (0.74, 1.96); 3rd quartile: 1.28 (0.84, 2.10) 4th quartile: 1.65 (1.02, 2.60) Per 1 $\mu g/m^3$: 1.10 (0.99, 1.20) All trimesters adjusted simultaneously 2nd quartile: 0.97 (0.60, 1.73); 3rd quartile: 0.98 (0.57, 1.75) 4th quartile: 1.22 (0.71, 2.18) Per 1 $\mu g/m^3$: 1.03 (0.90, 1.17) **Prevalence ratios (PRs) of birth weight < 3000 g during exposure over second trimester of pregnancy**

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			Each trimester separately 2nd quartile: 0.83 (0.52, 1.32); 3rd quartile: 1.08 (0.71, 1.60) 4th quartile: 0.94 (0.61, 1.47) Per 1 μg/m^3: 1.01 (0.92, 1.12) All trimesters adjusted simultaneously 2nd quartile: 0.75 (0.46, 1.24) 3rd quartile: 0.86 (0.56, 1.30); 4th quartile: 0.75 (0.48, 1.23) Per 1 μg/m^3: 0.94 (0.84, 1.06) **Prevalence ratios (PRs) of birth weight < 3000 g during exposure over third trimester of pregnancy** Each trimester separately 2nd quartile: 1.30 (0.80, 2.17) 3rd quartile: 1.44 (0.85, 2.27) 4th quartile: 1.90 (1.20, 2.82) Per 1 μg/m^3: 1.14 (1.02, 1.24) All trimesters adjusted simultaneously 2nd quartile: 1.34 (0.79, 2.30) 3rd quartile: 1.48 (0.86, 2.58) 4th quartile: 1.91 (1.00, 3.20) Per 1 μg/m^3: 1.14 (0.99, 1.29) **Prevalence ratios (PRs) of birth weight < 3000 g during exposure over third trimester of pregnancy (adjustment for season of conception)** All trimesters adjusted simultaneously Per 1 μg/m^3: 1.25 (1.04, 1.50) **Sensitivity analysis**(bootstrapped PR) 2nd quartile: 0.98 (0.63, 1.61); 3rd quartile: 1.22 (0.82, 2.02) 4th quartile: 1.57 (1.02, 2.57) Per 1 μg/m^3: 1.11 (0.98, 1.27) **Estimated increments in prevalence of birth weight of < 3000 g during exposure 9 months after birth** Per 1 μg/m^3: 7% (-7%, 22%)
Reference: (Slama et al., 2007, 093216) **Period of Study:** 1/1998 -1/1999 **Location:** Munich, Germany	**Outcome:** Birth weight offspring at term **Study Design:** Cohort study **N:** 1016 births **Statistical Analyses:** Poisson model **Covariates:** Maternal passive smoking, maternal age, gestational duration, sex of child, parity, maternal education, maternal size, prepregnancy weight, other pollutants ($PM_{2.5}$, $PM_{2.5}$ absorbance, NO_2), season of conception **Dose-response Investigated?** Yes **Statistical Package:** STATA	**Pollutant:** $PM_{2.5}$ absorbance (estimated) **Averaging Time:** Entire pregnancy period and trimesters **Mean (SD):** 1.76 * **Percentiles: 25th:** 1.61* **50th(Median):** 1.72* **75th:** 1.89 * **Unit (i.e. μg/m^3):** 10^{-5}/m **Monitoring Stations:** Spatial component: 40 Temporal component: 1 **Copollutant (correlation):**	**PM Increment:** 1) 0.5 * 10^{-5}/m 2) Quartiles: a) 1st (reference) (1.29–1.61) b) 2nd (1.61–1.72) c) 3rd (1.72–1.89) d) 4th (1.89–3.10) **Prevalence ratios (PRs) of birth weight < 3000 g during exposure over the whole pregnancy** **Single-pollutant models** Unadjusted models 2nd quartile: 1.19 (0.74, 1.99) 3rd quartile: 1.56 (0.98, 2.50); 4th quartile: 1.52 (0.96, 2.46)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
		p.a. = pregnancy avg	Per 0.5 * 10^{-5}/m: 1.25 (0.90, 1.70)
		trim. = trimester	Adjusted models
		abs = absorbance	2nd quartile: 1.21 (0.73, 1.97)
		$PM_{2.5}$ abs (p.a.)–$PM_{2.5}$ abs (1st trim.): 0.54	3rd quartile: 1.63 (0.98, 2.57);
		$PM_{2.5}$ abs (p.a.)–$PM_{2.5}$ abs (2nd trim.): 0.84	4th quartile: 1.78 (1.10, 2.70)
		$PM_{2.5}$ abs (p.a.)–$PM_{2.5}$ abs (3rd trim.): 0.55	Per 0.5 * 10^{-5}/m: 1.45 (1.06, 1.87)
		$PM_{2.5}$ abs (p.a.)–$PM_{2.5}$ (p.a.): 0.69	**Multipollutant models** Adjusted models
		$PM_{2.5}$ abs (p.a.)–$PM_{2.5}$ (1st trim.): 0.68	2nd quartile: 1.19 (0.70, 2.01)
		$PM_{2.5}$ abs (p.a.)–$PM_{2.5}$ (2nd trim.): 0.41	3rd quartile: 1.55 (0.80, 2.80);
		$PM_{2.5}$ abs (p.a.)–$PM_{2.5}$ (3rd trim.): 0.62	4th quartile: 1.46 (0.67, 2.90)
		$PM_{2.5}$ abs (p.a.)–NO_2 (p.a.): 0.67	Per 0.5 * 10^{-5}/m: 1.33 (0.76, 2.38)
		$PM_{2.5}$ abs (p.a.)–NO_2 (1st trim.): 0.34	**Prevalence ratios (PRs) of birth weight < 3000 g during exposure over the whole pregnancy (adjustment for season of conception)**
		$PM_{2.5}$ abs (p.a.)–NO_2 (2nd trim.): 0.63	4th quartile: 1.72 (1.08, 2.73)
		$PM_{2.5}$ abs (p.a.)–NO_2 (3rd trim.): 0.36	Per 0.5 * 10^{-5}/m: 1.38 (0.96, 1.86)
		$PM_{2.5}$ abs (1st trim.)–$PM_{2.5}$ abs (2nd trim.): 0.32	**Prevalence ratios (PRs) of birth weight < 3000 g during exposure over the whole pregnancy**
		$PM_{2.5}$ abs (1st trim.)–$PM_{2.5}$ abs (3rd trim.): -0.26	**Single-pollutant models**
		$PM_{2.5}$ abs (1st trim.)–$PM_{2.5}$ (p.a.): 0.33	(restricted analysis to $PM_{2.5}$ below the median)
		$PM_{2.5}$ abs (1st trim.)–$PM_{2.5}$ (1st trim.): 0.27	Per 0.5 * 10^{-5}/m: 1.67 (0.66, 3.73)
		$PM_{2.5}$ abs (1st trim.)–$PM_{2.5}$ (2nd trim.): 0.08	**Prevalence ratios (PRs) of birth weight < 3000 g during exposure over first trimester of pregnancy**
		$PM_{2.5}$ abs (1st trim.)–$PM_{2.5}$ (3rd trim.): 0.48	Each trimester separately
		$PM_{2.5}$ abs (1st trim.)–NO_2 (p.a.): 0.29	2nd quartile: 1.15 (0.73, 1.80)
		$PM_{2.5}$ abs (1st trim.)–NO_2 (1st trim.): 0.84	3rd quartile: 1.01 (0.61, 1.53);
		$PM_{2.5}$ abs (1st trim.)–NO_2 (2nd trim.): 0.16	4th quartile: 1.04 (0.70, 1.57)
		$PM_{2.5}$ abs (1st trim.)–NO_2 (3rd trim.): -0.39	Per 0.5 * 10^{-5}/m: 1.03 (0.82, 1.28)
		$PM_{2.5}$ abs (2nd trim.)–$PM_{2.5}$ abs (3rd trim.): 0.31	All trimesters adjusted simultaneously
		$PM_{2.5}$ abs (2nd trim.)–$PM_{2.5}$ (p.a.): 0.48	2nd quartile: 0.90 (0.52, 1.58)
		$PM_{2.5}$ abs (2nd trim.)–$PM_{2.5}$ (1st trim.): 0.53	3rd quartile: 0.82 (0.45, 1.31);
		$PM_{2.5}$ abs (2nd trim.)–$PM_{2.5}$ (2nd trim.): 0.29	4th quartile: 0.88 (0.53, 1.42)
		$PM_{2.5}$ abs (2nd trim.)–$PM_{2.5}$ (3rd trim.): 0.36	Per 0.5 * 10^{-5}/m: 1.02 (0.77, 1.29)
		$PM_{2.5}$ abs (2nd trim.)–NO_2 (p.a.): 0.61	**Prevalence ratios (PRs) of birth weight < 3000 g during exposure over second trimester of pregnancy**
		$PM_{2.5}$ abs (2nd trim.)–NO_2 (1st trim.): 0.19	Each trimester separately
		$PM_{2.5}$ abs (2nd trim.)–NO_2 (2nd trim.): 0.85	2nd quartile: 1.33 (0.85, 2.22)
		$PM_{2.5}$ abs (2nd trim.)–NO_2 (3rd trim.): 0.17	3rd quartile: 1.76 (1.07, 2.91);
		$PM_{2.5}$ abs (3rd trim.)–$PM_{2.5}$ (p.a.): 0.52	4th quartile: 1.83 (1.11, 2.81)
		$PM_{2.5}$ abs (3rd trim.)–$PM_{2.5}$ (1st trim.): 0.51	Per 0.5 * 10^{-5}/m: 1.27 (1.04, 1.54)
		$PM_{2.5}$ abs (3rd trim.)–$PM_{2.5}$ (2nd trim.): 0.41	All trimesters adjusted simultaneously
		$PM_{2.5}$ abs (3rd trim.)–$PM_{2.5}$ (3rd trim.): 0.37	2nd quartile: 1.30 (0.77, 2.16)
		$PM_{2.5}$ abs (3rd trim.)–NO_2 (p.a.): 0.40	3rd quartile: 1.63 (0.93, 2.73);
		$PM_{2.5}$ abs (3rd trim.)–NO_2 (1st trim.): -0.34	4th quartile: 1.99 (1.12, 3.33)
		$PM_{2.5}$ abs (3rd trim.)–NO_2 (2nd trim.): 0.21	
		$PM_{2.5}$ abs (3rd trim.)–NO_2 (3rd trim.): 0.88	

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			Per 0.5 * 10^{-5}/m: 1.21 (0.93, 1.54)
			Prevalence ratios (PRs) of birth weight < 3000 g during exposure over third trimester of pregnancy
			Each trimester separately
			2nd quartile: 1.30 (0.85, 2.09)
			3rd quartile: 0.92 (0.55, 1.50);
			4th quartile: 1.50 (1.00, 2.27)
			Per 0.5 * 10^{-5}/m: 1.20 (0.98, 1.44)
			All trimesters adjusted simultaneously
			2nd quartile: 0.99 (0.64, 1.62)
			3rd quartile: 0.71 (0.40, 1.20);
			4th quartile: 1.14 (0.68, 1.91)
			Per 0.5 * 10^{-5}/m: 1.15 (0.92, 1.42)
			Prevalence ratios (PRs) of birth weight < 3000 g during exposure over first trimester of pregnancy
			(adjustment for season of conception)
			All trimesters adjusted simultaneously
			4th quartile: 0.73 (0.38, 1.38)
			Per 0.5 * 10^{-5}/m: 0.93 (0.41, 1.32)
			Prevalence ratios (PRs) of birth weight < 3000 g during exposure over second trimester of pregnancy (adjustment for season of conception)
			All trimesters adjusted simultaneously
			4th quartile: 2.45 (1.22, 4.77)
			Per 0.5 * 10^{-5}/m: 1.14 (0.70, 1.64)
			Prevalence ratios (PRs) of birth weight < 3000 g during exposure over third trimester of pregnancy
			(adjustment for season of conception)
			All trimesters adjusted simultaneously
			4th quartile: 1.19 (0.60, 2.48)
			Per 0.5 * 10^{-5}/m: 1.29 (0.90, 1.75)
			Sensitivity analysis (bootstrapped PR)
			2nd quartile: 1.19 (0.76, 1.91)
			3rd quartile: 1.52 (0.99, 2.34);
			4th quartile: 1.62 (1.06, 2.55)
			Per 0.5 * 10^{-5}/m: 1.35 (1.01, 1.83)
			Estimated increments in prevalence of birth weight < 3000 g during exposure 9 months after birth Per 0.5 * 10^{-5}/m: 18% (-16%, 57%)
Reference: Wilhelm et al. (2005, 088668) **Period of Study:** 1994-2000 **Location:** Los Angeles County, California, U.S.	**Outcome:** Term low birth weight (LBW) (< 2500 g at ≥ 37 completed weeks gestation) Vaginal birth < 37 completed weeks gestation **Age Groups:** LBW: ≥ 37 completed weeks Preterm births: < 37 completed weeks	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24hr (every 3 days) Entire pregnancy Trimesters of pregnancy Months of pregnancy 6 weeks before birth	**PM Increment:** 1) 10 $\mu g/m^3$ 2) 3 levels: a) < 25%ile (reference) b) 25%-75%ile c) ≥ 75%ile **Incidence of LBW (third trimester exposure)**

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
	Study Design: cross-sectional study **N:** For LBW: 136,134 For preterm birth: 106,483 **Statistical Analyses:** Logistic regression **Covariates:** Maternal age, maternal race, maternal education, parity, interval since previous live birth, level of prenatal care, infant sex, previous LBW or preterm infant, birth season, other pollutants (not specified in birth weight analyses, also adjusted for gestational age) **Dose-response Investigated?** Yes **Statistical Package:** NR	**Mean (SD):** First trimester: 21.9 Third trimester: 21.0 6 weeks before birth: 21.0 **Range (Min, Max):** First trimester: 11.8-38.9 Third trimester: 11.8-.38.9 6 weeks before birth: 9.9-48.5 **Monitoring Stations:** Zip-code-level analysis: 9 Address-level analysis: 8 **Copollutant (correlation):** First trimester $PM_{2.5}$-CO: 0.57 $PM_{2.5}$-NO_2: 0.73 $PM_{2.5}$-O_3: -0.55 $PM_{2.5}$-PM_{10}: 0.43 Third trimester: $PM_{2.5}$-CO: 0.67 $PM_{2.5}$-NO_2: 0.78 $PM_{2.5}$-O_3: -0.60 $PM_{2.5}$-PM_{10}: 0.52 6 weeks before birth: $PM_{2.5}$-CO: 0.63 $PM_{2.5}$-NO_2: 0.74 $PM_{2.5}$-O_3: -0.60 $PM_{2.5}$-PM_{10}: 0.60	< 17.1 $\mu g/m^3$: 2.4 (2.0, 2.8) 17.1 to < 24.0 $\mu g/m^3$: 2.2 (2.0, 2.5) ≥ 24.0 $\mu g/m^3$: 2.1 (1.7, 2.4) **Incidence of preterm birth (first trimester exposure)** < 18.0 $\mu g/m^3$: 10.6 (9.6, 11.7) 18.0 to < 25.4 $\mu g/m^3$: 8.8 (8.1, 9.5) ≥ 25.4 $\mu g/m^3$: 9.0 (8.1, 10.0) **Incidence of preterm birth (6 weeks before birth exposure)** < 16.5 $\mu g/m^3$: 8.2 (7.4, 9.1) 16.5 to < 24.7 $\mu g/m^3$: 8.8 (8.2, 9.4) ≥ 24.7 $\mu g/m^3$: 9.6 (8.7, 10.5) **Outcome: Preterm birth** **Exposure Period: First trimester of pregnancy** **Address-level analysis:** Single-pollutant model: Distance ◘ 1 mile Per 10 $\mu g/m^3$: 0.85 (0.70, 1.02) 18.1 to < 25.2 $\mu g/m^3$: 0.91 (0.72, 1.16) ≥ 25.2 $\mu g/m^3$: 0.83 (0.60, 1.14) Single-pollutant model: 1 < distance ◘ 2 mile Per 10 $\mu g/m^3$: 0.85 (0.74, 0.99) 18.3 to < 25.2 $\mu g/m^3$: 0.81 (0.69, 0.94) ≥ 25.2 $\mu g/m^3$: 0.79 (0.65, 0.97) Multipollutant model1 < distance ◘ 2 mile Per 10 $\mu g/m^3$: 1.18 (0.84, 1.65) Single-pollutant model: 2 < distance ◘ 4 mile Per 10 $\mu g/m^3$: 0.83 (0.78, 0.88) 18.5 to < 24.9 $\mu g/m^3$: 0.79 (0.74, 0.85) ≥ 24.9 $\mu g/m^3$: 0.76 (0.70, 0.84) **Zip-code-level analysis:** Single-pollutant model: Per 10 $\mu g/m^3$: 0.73 (0.67, 0.80) 18.0 to < 25.4 $\mu g/m^3$: 0.70 (0.61, 0.80) ≥ 25.4 $\mu g/m^3$: 0.64 (0.53, 0.76) **Outcome: Preterm birth** **Exposure Period: 6 weeks before birth** **Address-level analysis:** Single-pollutant model: Distance ◘ 1 mile Per 10 $\mu g/m^3$: 1.09 (0.91, 1.30) 16.8 to < 24.1 $\mu g/m^3$: 1.21 (0.97, 1.51) ≥ 24.1 $\mu g/m^3$: 1.25 (0.93, 1.68) Single-pollutant model: 1 < distance ◘ 2 mile Per 10 $\mu g/m^3$: 1.08 (0.97, 1.21) 17.2 to < 24.5 $\mu g/m^3$: 0.94 (0.82, 1.08)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			≥ 24.5 $\mu g/m^3$: 1.04 (0.87, 1.24)
			Single-pollutant model: 2 < distance ◘ 4 mile
			Per 10 $\mu g/m^3$: 1.05 (0.99, 1.10)
			17.3 to < 24.6 $\mu g/m^3$: 1.06 (1.00, 1.13)
			≥ 24.6 $\mu g/m^3$: 1.08 (0.99, 1.17)
			Zip-code-level analysis
			Single-pollutant model: Per 10 $\mu g/m^3$: 1.10 (1.00, 1.21)
			16.5 to < 24.7 $\mu g/m^3$: 1.06 (0.94, 1.20)
			≥ 24.7 $\mu g/m^3$: 1.19 (1.02, 1.40)
			(See Notes[1])
			Multipollutant model
			Per 10 $\mu g/m^3$: 1.12 (0.90, 1.40)
			≥ 24.6 $\mu g/m^3$: 1.12 (0.82, 1.52)
			Notes: [1] In the table, the 75%ile is noted as 24.7 $\mu g/m^3$. However, the text notes the 75%ile as 24.3 $\mu g/m^3$.

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Woodruff et al. (2006, 088758) **Period of Study:** 1999-2000 **Location:** California	**Outcome (ICD10):** SIDS (R95) Respiratory mortality (J00-J99) Bronchopulmonary dysplasia (P27.1) External accidents (V01-Y98) Ill-defined and unspecified causes of mortality (R99) **Age Groups:** > 28 days old **Study Design:** Matched case-control (matched on date of birth and birth weight) **N:** 3877 infants **Statistical Analyses:** Conditional logistic regression **Covariates:** Maternal race, education, parity, age, marital status **Dose-response Investigated?** Yes **Statistical Package:** STATA	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 hrs (every 6 days) (time period between birth and post neonatal death for the infant who died and the same period for its four matched surviving infants) **Percentiles:** Infants who died of all causes (cases) **25th:** 13.4 **50th(Median):** 19.2 **75th:** 23.6 Matched controls **25th:** 13.5 **50th(Median):** 18.4 **75th:** 22.7 **Monitoring Stations:** 73 (from 39 counties)	**PM Increment:** 10 $\mu g/m^3$ **RR Estimate [Lower CI, Upper CI]** **lag:** All-cause mortality: Unadjusted: 1.15 (1.00, 1.32) Adjusted: 1.07 (0.93, 1.24) Cause-specific mortality: Respiratory (all): Unadjusted: 2.15 (1.15, 4.02) Adjusted: 2.13 (1.12, 4.05) Respiratory (excluding deaths due to BPD): Adjusted: 1.42 (0.66, 3.03) Respiratory (BPD alone): Unadjusted: 6.00 (1.40, 27.76) Respiratory (low birth weight infants only): Unadjusted: 3.09 (1.14, 8.40) Respiratory (normal birth weight infants only): Unadjusted: 1.66 (0.74, 3.70) Respiratory (with matched $PM_{2.5}$ averaged over all monitors in county) Adjusted: 2.28 (0.94, 5.52) Respiratory (averaging all $PM_{2.5}$ measurements in county over the 2-year study period): Adjusted: 2.26 (0.83, 6.21) SIDS: Unadjusted: 0.86 (0.61, 1.22) Adjusted: 0.82 (0.55, 1.23) SIDS (includes ICD10 code R99: ill-defined and unspecified causes of mortality): Adjusted: 1.03 (0.79, 1.35) External causes: Unadjusted: 0.91 (0.56, 1.47) Adjusted: 0.83 (0.50, 1.39) Compare against the lowest quartile, estimates for respiratory-specific mortality were provided: 2nd quartile: 1.28 (0.47, 3.51) 3rd quartile: 1.75 (0.65, 4.72) 4th quartile: 2.35 (0.85, 6.54)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Woodruff et al. (2008, 098386) **Period of Study:** 1999-2002 **Location:** US counties with > 250,000 residents (96 counties)	**Outcome (ICD10):** Postneonatal deaths: Respiratory mortality (J000-99, plus bronchopulmonary dysplasia [BPD] P27.1) SIDS (R95) Ill-defined causes (R99) All other deaths evaluated as a control category **Age Groups:** Infants aged > 28 days and < 1 yr **Study Design:** Cross-sectional **N:** 3,583,495 births (6,639 post neonatal deaths) **Statistical Analyses:** Logistic GEE (exchangeable correlation structure) **Covariates:** maternal race/ethnicity, marital status, age, education, primiparity, county-level poverty and per capita income levels, year and month of birth dummy variables to account for time trend and seasonal effects, and region of the country sensitivity analyses performed among only those mothers with smoking information (adjustment for smoking had no effect on the estimates) **Season:** Adjusted for year and month of birth dummy variables to account for time trend and seasonal effects **Dose-response Investigated?** Evaluated the appropriateness of a linear form from analysis based on quartiles of exposure and concluded that linear form was appropriate (data not shown) **Statistical Package:** SAS	**Pollutant:** $PM_{2.5}$ **Averaging Time:** Measured continuously for 24 h once every 6 days exposure assigned by calculating avg concentration of pollutant during first 2 months of life **Median and IQR (25th-75th percentile):** Survivors: 14.8 (11.7-18.7) All causes of death: 14.9 (12.0-18.6) Respiratory: 14.8 (11.5-18.5) SIDS: 14.5 (12.0-17.5) SIDS + ill-defined: 14.8 (12.1-18.5) Other causes: 14.9 (12.0-18.6) **Percentiles:** See above **PM Component:** Not assessed, but controlled for region of the country to account for PM composition variation **Monitoring Stations:** NR **Copollutant (correlation):** PM_{10} (r = 0.34) $PM_{2.5}$ CO (r = 0.35) SO_2 (r = 0.21) O_3 (r = -0.10) **Notes:** Monthly averages calculated if there were at least 3 available measures for PM Assigned exposures using the avg concentration of the county of residence	**PM Increment:** IQR (7 $\mu g/m^3$) **Effect Estimate [Lower CI, Upper CI]:** Adjusted ORs for single pollutant models All causes: 1.04 (0.98, 1.11) Respiratory: 1.11 (0.96, 1.29) SIDS: 1.01 (0.86, 1.20) Ill-defined + SIDS: 1.06 (0.97, 1.17) Other causes: 1.03 (0.96, 1.12) Adjusted ORs for multipollutant models (including CO, O_3, SO_2) Respiratory: 1.05 (0.89, 1.24) SIDS: 1.04 (0.87, 1.23) OR for respiratory deaths assessing exposure as quartiles Highest vs Lowest quartile: 1.39 (1.04, 1.85)

[1]All units expressed in $\mu g/m^3$ unless otherwise specified.

E.9. Long-Term Exposure and Mortality

Table E-31. Long-term exposure – mortality - PM_{10}.

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: (Breitner et al., 2009, 188439) **Period of Study:** 10/1/1991 to 3/31/2002 **Location:** Efurt, Germany	**Outcome:** Mortality, excluding infants and ICD-9 ≥ 800 **Study Design:** Time-series **Covariates:** seasonal and weekday variations, influenza epidemics, air temperature, relative humidity **Statistical Analysis:** semiparametric Poisson regression, polynomial distributed lag (PDL) **Statistical Package:** R **Age Groups:** All	**Pollutant:** PM_{10} **Averaging Time:** daily **Mean (SD) Unit:** 1 (10/1/1991-8/31/1995): 50.6 ± 32.2 $\mu g/m^3$ 2 (9/1/1995-2/28/1998): 41.1 ± 28.4 $\mu g/m^3$ 3 (3/1/1998-3/31/2002): 24.3 ± 15.4 $\mu g/m^3$ Total: 38.0 ±28.3 $\mu g/m^3$ **Range (Min, Max):** NR **Copollutant:** NO_2, CO, UFP	**Increment:** IQR **Relative Risk (95% CI)** **Lag** New City Limits 6-day IQR: 17.2 PDL: 0.997 (0.972-1.022) Mean of lags 0-5: 0.995 (0.971-1.019) Old City Limits 6-day IQR: 17.2 PDL: 1.004 (0.978-1.031) Mean of lags 0-5: 1.001 (0.976-1.027) New City Limits 15-day IQR: 14.5 PDL: 1.008 (0.982-1.036) Mean of lags 0-14: 1.006 (0.981-1.032) Old City Limits 15-day IQR: 14.5 PDL: 1.019 (0.991-1.048) Mean of lags 0-14: 1.017 (0.990-1.044) Multiday Moving Averages, 6-day Overall IQR: 24.2 Overall RR (95% CI): 0.998 (0.976-1.021) Period 1: 0.996 (0.969-1.024) Period 2: 1.013 (0.972-1.056) Period 3: 0.949 (0.897-1.004) Multiday Moving Averages, 15-day Overall IQR: 22.3 Overall RR (95% CI): 1.020 (0.993-1.093) Period 1: 1.017 (0.984-1.051) Period 2: 1.012 (0.973-1.071) Period 3: 0.978 (0.911-1.051)

Table E-32. Long-term exposure – mortality - $PM_{10-2.5}$.

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: (Chen et al., 2005, 087942)	**Outcome:** Mortality: CHD	**Pollutant:** $PM_{10-2.5}$	**Increment:** 10 $\mu g/m^3$
Period of Study: 1973-1998	**Study Design:** Cohort	**Averaging Time:** 25 years	**Relative Risk (Lower CI, Upper CI)**
Location: San Francisco, San Diego, Los Angeles, CA	**Statistical Analyses:** Cox proportion hazards model	**Mean (SD):** 25.4	**lag:** Males
	Age Groups: > 25	**Range (Min, Max):** NR	$PM_{10-2.5}$: 0.93 (0.68, 1.29)
		Copollutant: NO_2	0-1
		O_3	$PM_{10-2.5} + NO_2$: 0.86 (0.62, 1.20)
		SO_2	0-1
			$PM_{10-2.5} + SO_2$: 0.90 (0.64,1.27)
			0-1
			$PM_{10-2.5} + O_3$: 1.01 (0.67,1.51)
			0-1
			Females
			$PM_{10-2.5}$: 1.20 (0.95, 1.53)
			0-1
			$PM_{10-2.5} + NO_2$: 1.19 (0.92, 1.54)
			0-1
			$PM_{10-2.5} + SO_2$: 1.31 (1.03,1.68)
			0-1
			$PM_{10-2.5} + O_3$: 1.47 (1.10,1.96)
			0-1
Reference: Goss et al. (2004, 055624)	**Outcome:** Mortality	**Pollutant:** $PM_{2.5}$	**Increment:** 10 $\mu g/m^3$
Period of Study: 1999-2000	**Study Design:** Cohort Study (Cystic Fibrosis Cohort)	**Averaging Time:** Annual avg	$PM_{2.5}$: 1.32 (0.91 – 1.93)
Location: United States	**Statistical Analyses:** Logistic Regression	**Mean (SD) unit:** $PM_{2.5}$: 13.7 (4.2)	
	Age Groups: > 6 yrs	**IQR:** $PM_{2.5}$: 11.8-15.9	
		Copollutant: O_3	
		NO_2	
		SO_2	
		CO	
Reference: (Lipfert et al., 2006, 189271)	**Outcome:** Mortality	**Pollutant:** $PM_{10-2.5}$	**Increment:** 12
Period of Study: 1989-1996	**Study Design:** Retrospective Cohort	**Mean (SD):** 16.0 (5.1)	1.07 (1.01, 1.13)
Location: Various parts of the Untied States	**Statistical Analyses:** Cox proportional hazards regression		
	Age Groups: Male US veterans between ages of 39 and 63 (Avg. age: 51)		
Reference: McDonnell et al. (2000, 010319)	**Outcome:** Mortality	**Pollutant:** $PM_{10-2.5}$	**Increment:** IQR
Period of Study: 1973-1977	**Study Design:** Cohort (AHSMOG airport cohort)	**Averaging Time:** monthly averages	All Cause: 1.05 (0.92-1.20)
Location: California	**Statistical Analyses:** Cox regression models	**Mean (SD):** $PM_{10-2.5}$: 27.3 (8.6)	Resp: 1.19 (0.88, 1.62)
	Age Groups: Males, 27 yrs +	**IQR:** 9.7	Lung Cancer: 1.25 (0.63-2.49)
		Copollutant: O_3: 0.70	
		SO_2: 0.31	
		NO_2: 0.23	
		SO_4: 0.47	

[1]All units expressed in $\mu g/m^3$ unless otherwise specified.

Table E-33. Long-term exposure – mortality - $PM_{2.5}$ (including PM components/sources).

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Abrahamowicz et al. (2003, 086292) **Period of Study:** 1982-1989 **Location:** 151 Cities	**Outcome:** Mortality: All-causes **Study Design:** Case-cohort study **Statistical Analyses:** Cox proportion-hazards model flexible regression spline generalization **Age Groups:** > 18	**Pollutant:** $PM_{2.5}$ **Averaging Time:** Annual **Mean (SD):** 18.2 **Range (Min, Max):** (9.0, 33.5) **Copollutant:** Sulfates	**Relative Risk (Min CI, Max CI)** **Estimated from graph (Figure 1):** log HR for a 24.5 $\mu g/m^3$ increase in $PM_{2.5}$ over time Years 0: 0.5 (-1.1, 1.6) 2: 0.6 (0.2, 0.9) 4: 0.6 (0.3, 0.8) 6: 0.8 (0.3, 1.1) 8: -1.0 (-1.5, 1.0)
Reference: Abrahamowicz et al. (2003, 086292) **Period of Study:** 1982-1989 **Location:** 151 Cities	**Outcome:** Mortality: All-causes **Study Design:** Case-cohort study **Statistical Analyses:** Cox proportion-hazards model flexible regression spline generalization **Age Groups:** > 18	**Pollutant:** Sulfates **Averaging Time:** Annual **Mean (SD):** 18.2 **Range (Min, Max):** (9.0, 33.5) **Copollutant:** $PM_{2.5}$	**Relative Risk (Min CI, Max CI)** **Estimated from graph (Figure 1):** Log HR for a 19.9 $\mu g/m^3$ increase in Sulfates over time Years 0: 0.1 (-0.2, 0.7) 2: 0.1 (-0.2, 0.4) 4: 0.0 (-0.4, 0.3) 6: 0.3 (-0.1, 0.5) 8: 0.4 (-0.4, 1.6)
Reference: Ballester et al. (2008, 189977) **Period of Study:** 2001-2002 **Location:** Europe	**Outcome:** Mortality- All-causes **Study Design:** Health Impact Assessment **Statistical Analyses:** Aphesis Network **Age Groups:** > 30	**Pollutant:** $PM_{2.5}$ **Averaging Time:** Annual **Mean (SD):** NR **Range (Min, Max):** NR	**Potential Reduction in the total burden of mortality (min CI, max CI) for four different decreases in annual $PM_{2.5}$ using a conservative estimate** Reduction to 25 $\mu g/m^3$ - 0.4 (0.1, 0.8) Reduction to 20 $\mu g/m^3$ - 0.8 (0.2, 1.6) Reduction to 15 $\mu g/m^3$ - 1.6 (0.4, 3.1) Reduction to 10 $\mu g/m^3$ - 3.0 (0.8, 5.8)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Beelen et al. (2008, 156263) **Period of Study:** 1987-1996 **Location:** Netherlands	**Outcome:** Mortality: Total (non-accidental) (< 800) Cardio-respiratory (390-448, 490-496, 487, 480-486, 507) Pulmonary (460-519) Cardiovascular (400-440) Lung Cancer (162) Other-causes **Study Design:** Case-cohort study and prospective cohort **Statistical Analyses:** Cox proportion-hazards model **Age Groups:** 55-69	**Pollutant:** $PM_{2.5}$ **Averaging Time:** Annual **Mean (SD):** 28.3 (2.1) $\mu g/m^3$ **Range (Min, Max):** (23.0, 36.8) **Copollutant (correlation):** NO_2: (> 0.8) BS: (> 0.8) SO_2: (> 0.6)	**Increment:** 11 $\mu g/m^3$ **Relative Risk (Min CI, Max CI)** **RR for the association between exposures to $PM_{2.5}$ and cause specific mortality** Natural Cause: Full cohort: 1.06 (0.97, 1.16) Case cohort: 0.86 (0.66, 1.13) Cardiovascular: Full cohort: 1.04 (0.90, 1.21) Case cohort: 0.83 (0.60, 1.15) Respiratory: Full cohort: 1.07 (0.75, 1.52) Case cohort: 1.02 (0.56, 1.88) Lung Cancer: Full cohort: 1.06 (0.82, 1.38) Case cohort: 0.87 (0.52, 1.47) Other cause: Full cohort: 1.08 (0.96, 1.23) Case cohort: 0.85 (0.65, 1.12) **RR for the association between exposures to BS and cause specific mortality** Natural Cause: Full cohort: 1.05 (1.00, 1.11) Case cohort: 0.97 (0.83, 1.13) Cardiovascular: Full cohort: 1.04 (0.95, 1.13) Case cohort: 0.98 (0.81, 1.18) Respiratory: Full cohort: 1.22 (0.99, 1.50) Case cohort: 1.29 (0.91, 1.83) Lung Cancer: Full cohort: 1.03 (0.88, 1.20) Case cohort: 1.03 (0.77, 1.38) Other cause: Full cohort: 1.04 (0.97, 1.12) Case cohort: 0.91 (0.78, 1.07)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Breitner et al. (2009, 188439) **Period of Study:** 10/1/1991 to 3/31/2002 **Location:** Efurt, Germany	**Outcome:** Mortality, excluding infants and ICD-9 ≥ 800 **Study Design:** Time-series **Covariates:** seasonal and weekday variations, influenza epidemics, air temperature, relative humidity **Statistical Analysis:** semiparametric Poisson regression, polynomial distributed lag (PDL) **Statistical Package:** R **Age Groups:** All	**Pollutant:** $PM_{2.5}$ **Averaging Time:** daily **Mean (SD) Unit:** 1 (10/1/1991-8/31/1995): 50.6 ± 32.2 $\mu g/m^3$ 2 (9/1/1995-2/28/1998): 41.1 ± 28.4 $\mu g/m^3$ 3 (3/1/1998-3/31/2002): 24.3 ± 15.4 $\mu g/m^3$ Total: 38.0 ±28.3 $\mu g/m^3$ **Range (Min, Max):** NR **Copollutant:** NO_2, CO, UFP	**Increment:** IQR **Relative Risk (95% CI)** **Lag** New City Limits 6-day IQR: 13.3 PDL: 1.009 (0.984-1.035) Mean of lags 0-5: 1.004 (0.981-1.027) Old City Limits 6-day IQR: 13.3 PDL: 1.017 (0.990-1.044) Mean of lags 0-5: 1.010 (0.986-1.035) New City Limits 15-day IQR: 11.5 PDL: 1.019 (0.988-1.050) Mean of lags 0-14: 1.017 (0.992-1.042) Old City Limits 15-day IQR: 11.5 PDL: 1.030 (0.997-1.063) Mean of lags 0-14: 1.025 (0.999-1.052) Multiday Moving Averages, 6-day Overall IQR: 13.3 Overall RR (95% CI): 1.004 (0.981-1.027) Period 1: NR Period 2: 1.017 (0.990-1.044) Period 3: 0.974 (0.937-1.013) Multiday Moving Averages, 15-day Overall IQR: 11.5 Overall RR (95% CI): 1.017 (0.992-1.042) Period 1: NR Period 2: 1.016 (0.988-1.045) Period 3: 1.016 (0.971-1.063)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Brunekreef et al. (2009, 191947)	**Outcome:** All cause mortality (ICD-9 400-440, 460-519, > 800)	**Pollutant:** $PM_{2.5}$, estimated from PM_{10} levelsf	**Increment:** 10μg/m^3
Period of Study: 1987-1996	**Study Design:** Case-cohort	**Averaging Time:** 24hr	**Relative Risk (95 % CI) for $PM_{2.5}$ concentrations and cause specific mortality**
Location: The Netherlands	**Covariates**	**50th Percentile:** 28 μg/m^3	Case Cohort
	Individual: sex, age, Quetelet index, smoking status, passive smoking status, educational level, occupation, occupational exposure, marital status, alcohol use, intake of vegetables, fruits, energy, saturated and monounsaturated fatty acids, trans fatty acids, total fiver, folic acid and fish	**Range (Min, Max):** 23-37	Natural Cause: 0.86 (0.66-1.13)
		Copollutant (correlation):	Cardiovascular: 0.83 (0.60-1.15)
		NO_2: 0.75	Respiratory: 1.02 (0.56-1.88)
		Black Smoke: 0.84	Lung Cancer: 0.87 (0.52-1.47)
		NO: 0.69	Noncardiopulmonary, non-lung cancer: 0.85 (0.65-1.23)
	Area-level: Percent of population with income below the 40th percentile and above the 80th percentile	SO_2: 0.43	Full Cohort
	Statistical Analysis: Cox proportional hazards		Natural Cause: 1.06 (0.97-1.16)
	Statistical Package: Stata, SPSS, R		Cardiovascular: 1.04 (0.90-1.21)
	Age Groups: 120,000 adults aged 55-69 years at enrollment		Respiratory: 1.07 (0.75-1.52)
			Lung Cancer: 1.06 (0.82-1.38)
			Noncardiopulmonary, non-lung cancer: 1.08 (0.72-1.19)
			Relative Risk (95%CI) for $PM_{2.5}$ concentrations and cause specific mortality in full cohort analysis by confounder model
			Natural Cause Mortality
			Unadjusted: 1.11 (1.04-1.20)
			Smoking: 1.04 (0.96-1.13)
			Smoking, area-level income: 1.06 (0.97-1.16)
			Cardiovascular Mortality
			Unadjusted: 1.09 (0.97-1.23)
			Smoking: 1.02 (0.90-1.16)
			Smoking, area-level income: 1.04 (0.90-1.21)
			Respiratory Mortality
			Unadjusted: 1.23 (0.92-1.65)
			Smoking: 1.10 (0.81-1.50)
			Smoking, area-level income: 1.07 (0.75-1.52)
			Lung Cancer Mortality
			Unadjusted: 1.17 (0.95-1.46)
			Smoking: 1.06 (0.85-1.33)
			Smoking, area-level income: 1.06 (0.82-1.38)
			Noncardiopulmonary, Non-Lung Cancer Mortality
			Unadjusted: 1.10 (1.00-1.22)
			Smoking: 1.05 (0.94-1.16)
			Smoking, area-level income: 1.08 (0.96-1.22)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Chen et al. (2005, 087942)	**Outcome:** Mortality: CHD	**Pollutant:** $PM_{2.5}$	**Increment:** 10 $\mu g/m^3$
Period of Study: 1973-1998	**Study Design:** Cohort	**Averaging Time:** 25 years	**Relative Risk (Lower CI, Upper CI)**
Location: San Francisco, San Diego, Los Angeles, CA	**Statistical Analyses:** Cox proportion hazards model	**Mean (SD):** 29.0	**lag:** Males
	Age Groups: > 25	**Range (Min, Max):** NR	$PM_{2.5}$: 0.89 (0.69, 1.17)
		Copollutant: NO_2, O_3, SO_2	0-1
			$PM_{2.5}+NO_2$: 0.82 (0.61, 1.10); 0-1
			$PM_{2.5}+SO_2$: 0.86 (0.65,1.14)
			0-1
			$PM_{2.5}+O_3$: 0.92 (0.65,1.29)
			0-1
			Females
			$PM_{2.5}$: 1.19 (0.96, 1.47)
			0-1
			$PM_{2.5}+NO_2$: 1.18 (0.95, 1.47); 0-1
			$PM_{2.5}+SO_2$: 1.36 (1.05,1.74)
			0-1
			$PM_{2.5}+O_3$: 1.61 (1.17,2.22)
			0-1
Reference: Eftim et al. (2008, 099104)	**Outcome (ICD-9):** All non-accidental causes (< 800)	**Pollutant:** $PM_{2.5}$	**Increment:** 10 $\mu g/m^3$
Period of Study: 2000-2002	**Study Design:** Cross-sectional	**Averaging Time:** Annual avg	**% Increase in Mortality for overall exposure period and individual year (95%CI Min, 95%CI Max):**
Location: USA, Same cities as six cities and ACS cohorts	**Statistical Analyses:** Log-linear regression, Poisson	**Mean (SD):** |	ACS (adjusted for age, sex)
	Age Groups: > 65	ACS: 13.6 (2.8)	Overall: 10.8 (8.6, 13.0)
		SCS: 14.1 (3.1)	2000: 10.9 (7.3, 14.6)
		Range (Min, Max): ACS: (6.0, 25.1); SCS: (9.6, 19.1)	2001: 9.1 (5.3, 12.7)
			2002: 10.1 (6.0, 14.3)
			SCS (adjusted for age, sex)
			Overall: 20.8 (14.8, 27.1)
			2000: 17.8 (9.8, 26.4)
			2001: 16.5 (7.4, 25.0)
			2002: 33.5 (19.2, 49.3)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Enstrom et al. (2005, 087356)	**Outcome:** Mortality: Cardiovascular-respiratory (390-448)	**Pollutant:** $PM_{2.5}$	**Relative Risk (Lower CI, Upper CI)**
Period of Study: 1973-2002	(480-486, 487, 490-496, 507)	**Averaging Time:** Annual	**RR from causes for both sexes by county from 1973-2002**
Location: 25 California Colonies	**Study Design:** Retrospective cohort	**Mean (SD):** 23.4	Alameda: 0.962 (0.926,0.999)
11 California Colonies (EPA IPN study)	**Statistical Analyses:** Cox proportional hazards regression model, SAS PHREG	**Range (Min, Max):** (13.1 $\mu g/m^3$, 36.1)	Butte: 0.999 (0.910,1.096)
	Age Groups: 35 or older		Contra Costa: 0.999 (0.943,1.058)
			Fresno: 0.935 (0.872,1.002)
			Humboldt: 0.992 (0.900,1.092)
			Kern: 0.944 (0.872,1.023)
			Marin: 0.939 (0.867,1.016)
			Napa: 0.949 (0.868,1.038)
			Orange: 0.990 (0.948,1.034)
			Riverside: 0.959 (0.906,1.015)
			Sacramento: 0.998 (0.944,1.055)
			San Bernardino: 0.992 (0.938,1.049)
			San Diego: 0.992 (0.954, 1.033)
			San Francisco: 0.963 (0.914,1.014)
			San Joaquin: 0.925 (0.816,1.049)
			San Mateo: 0.949 (0.899, 1.003)
			Santa Barbara: 0.968 (0.878,1.068)
			Santa Clara: 0.955 (0.910,1.003)
			Santa Cruz: 0.890 (0.793,0.999)
			Solano: 0.901 (0.815,0.995)
			Sonoma: 0.968 (0.884,1.060)
			Stanislaus: 0.984 (0.904,1.072)
			Tulare: 1.047 (0.979,1.119)
			Ventura: 0.967 (0.872,1.072)
			RR from all causes for 11 counties for both sexes (EPA IPN study)
			Santa Barbara: 0.968 (0.878,1.068)
			Contra Costa: 0.999 (0.943,1.058)
			Alameda: 0.962 (0.926,0.999)
			Butte: 0.999 (0.910,1.096)
			San Francisco: 0.963 (0.914,1.014)
			Santa Clara: 0.955 (0.910,1.003)
			Fresno: 0.935 (0.872,1.002)
			San Diego: 0.992 (0.954,1.033)
			Kern: 0.944 (0.872,1.023)
			Riverside: 0.959 (0.906,1.015)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Filleul et al. (2005, 087357) **Period of Study:** 1974-1976 **Location:** 7 cities in France	**Outcome:** Non-accidental causes (< 800), cardiopulmonary disease (401-440 and 460-519), lung cancer (162) **Age Groups:** 25–59 years **Study Design:** Cohort **N:** 14,284 people **Statistical Analyses:** Cox proportional hazard, regression **Covariates:** Sex, smoking habits, educational level, body-mass index (BMI), occupational exposure **Statistical Package:** Proc Phreg SAS	**Pollutant:** Total suspended particles (TSP) **Averaging Time:** NR **Mean (SD):** NR **Range (Min, Max):** (45, 243) **PM Component:** NR **Monitoring Stations:** 1 station **Copollutant (correlation):** BS r = 0.87 SO_2 r = 0.17 NO r = 0.84 NO_2 r = 0.60	**Increment:** 10 $\mu g/m^3$ Adjusted mortality rate ratios: 24 areas: All non-accidental causes: 1.00[0.99, 1.01] Lung cancer: 0.97[0.94, 1.01] Cardiopulmonary disease: 1.01[0.99, 1.03] 18 areas: All non-accidental causes: 1.05[1.02, 1.08] Lung cancer: 1.00[0.92, 1.10] Cardiopulmonary disease: 1.06[1.01, 1.12]
Reference: Fuentes et al. (2006, 097647) **Period of Study:** June 2000 **Location:** Conterminous U.S.	**Outcome:** Mortality: **Study Design:** Time-series **Statistical Analyses:** Generalized Poisson Regression **Age Groups:** 0-14, 15-64, > 65 **Covariates:** temperature, pressure, dew point, wind speed, elevation, age, ethnicity	**Pollutant:** $PM_{2.5}$ **Averaging Time:** monthly **Mean (SD):** 6.60 (0.76) **Copollutant:** PM_{10}, O_3	**Increment:** 10 $\mu g/m^3$ $PM_{2.5}$: 1.066 (1.064, 1.069) PM_{10}: 1.030 (1.028, 1.032)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Janes et al. (2007, 090927)	**Outcome:** Mortality:	**Pollutant:** $PM_{2.5}$	**Increment:** 1 $\mu g/m^3$
Period of Study: 2000 to 2002	**Study Design:** Time-series	**Averaging Time:** Annual Avg	**% Increase (Lower CI, Upper CI)**
Location: 113 US counties	**Statistical Analyses:** Cox proportional hazards model	**Mean (SD):** NR	**lag:** Overall % Increase by age-sex stratum
		Range (Min, Max): NR	Age Category
	Age Groups: 65-74		65-74: Male: 1.48 (0.93,2.03)
	75-84		Female: 0.83 (0.24,1.43)
	85+		75-84: Male: 0.85 (0.34,1.35)
			Female: 0.77 (0.28,1.27)
			85+: Male: 0.70 (0.03,1.38)
			Female: 0.59 (0.05,1.12)
			National Trend % Increase by age-sex stratum
			Age Category
			65-74: Male: 3.55 (2.77,4.34)
			Female: 1.97 (1.12,2.83)
			75-84: Male: 2.48 (1.83,3.14)
			Female: 2.29 (1.66,2.93)
			85+: Male: 1.38 (0.52,2.26)
			Female: 1.65 (1.01,2.29)
			Local Trend % Increase by age-sex stratum
			Age Category
			65-74: Male: 0.04 (-0.58,0.67)
			Female: -0.03 (-0.71,0.66)
			75-84: Male: -0.34 (-0.87,0.19)
			Female: -0.31 (-0.82, 0.21)
			85+: Male: $<$ 0.01 (-0.71,0.73)
			Female: -0.22 (-0.74,0.31)
			*Local trends are county specific deviations from national trends
Reference: Jerrett et al. (2003, 087380)	**Outcome:** Mortality	**Pollutant:** Sulfates	**Increment:** 19.9 (Range)
Period of Study: 1982	**Study Design:** multilevel, individual-ecologic analysis	**Mean (SD):** 10.6	All Cause: SO_4: 1.17 (1.07, 1.27)
Location: 151 cities from ACS		**Range (Min, Max):** 3.6,23.5	SO_4 + CO: 1.16 (1.10, 1.23)
	Statistical Analysis: Cox proportional hazards model		SO_4 + NO_2: 1.16 (1.08, 1.24)
	Covariates: Smoking, education, occupational exposures, BMI, marital status, alcohol consumption, gender		SO_4 + O_3: 1.17 (1.11, 1.24)
			SO_4 + SO_2: 1.05 (0.98, 1.12)
			CPD: SO_4: 1.25 (1.16, 1.35)
			SO_4 + CO: 1.28 (1.18, 1.39)
			SO_4 + NO_2: 1.29 (1.17, 1.42)
			SO_4 + O_3: 1.27 (1.17, 1.38)
			SO_4 + SO_2: 1.13 (1.03, 1.24)
			Lung Cancer: SO_4: 1.31 (1.09, 1.58)
			SO_4 + CO: 1.26 (1.03, 1.53)
			SO_4 + NO_2: 1.31 (1.05, 1.65)
			SO_4 + O_3: 1.30 (1.07, 1.59)
			SO_4 + SO_2: 1.37 (1.08, 1.73)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Jerrett et al. (2005, 087600) **Period of Study:** 1982-2000 **Location:** Los Angeles, California	**Outcome:** Mortality: Non- accidental (< 800) IHD (410-414) Cardiopulmonary (400-440, 460-519) Lung Cancer (162) Other Cancers (140-149,160, 161, 163-239) Other causes **Study Design:** Retrospective Cohort **Statistical Analyses:** Cox regression hazards model kriging, radial basis function multiquadric interpolator **Age Groups:** All ages	**Pollutant:** $PM_{2.5}$ **Averaging Time:** Annual avg **Mean (SD):** NR **Range (Min, Max):** NR **Copollutant:** O_3	**Increment:** 10 $\mu g/m^3$ **Relative Risk (Lower CI, Upper CI)** All Causes - $PM_{2.5}$ Only: 1.24 (1.11,1.37) 44 Ind. Covariates together + $PM_{2.5}$: 1.17 (1.03,1.32) 44 Ind. Covariates together + $PM_{2.5}$ + O_3: 1.20 (1.07,1.34) 44 Ind. Covariates together + intersection within freeways within 500 m + $PM_{2.5}$ + O_3: 1.17 (1.05,1.31) IHD - $PM_{2.5}$ Only: 1.49 (1.20,1.85) 44 Ind. Covariates together + $PM_{2.5}$: 1.39 (1.12,1.73) 44 Ind. Covariates together + $PM_{2.5}$ + O_3: 1.45 (1.15,1.82) 44 Ind. Covariates together + intersection within freeways within 500 m + $PM_{2.5}$ + O_3: 1.38 (1.11,1.72) Cardiopulmonary - $PM_{2.5}$ Only: 1.20 (1.04,1.39) 44 Ind. Covariates together + $PM_{2.5}$ + O_3: 1.19 (1.02,1.38) 44 Ind. Covariates together + intersection within freeways within 500 m + $PM_{2.5}$ + O_3: 1.13 (0.97,1.31) Lung Cancer - $PM_{2.5}$ Only: 1.60 (1.09,2.33) 44 Ind. Covariates together + $PM_{2.5}$: 1.44 (0.98,2.11) 44 Ind. Covariates together + intersection within freeways within 500 m + $PM_{2.5}$ + O_3: 1.46 (0.99,2.16) Other Cancers - $PM_{2.5}$ Only: 1.09 (0.85,1.40) 44 Ind. Covariates together + $PM_{2.5}$ + O_3: 1.08 (0.83,1.39) 44 Ind. Covariates together + intersection within freeways within 500 m + $PM_{2.5}$ + O_3: 1.08 (0.83,1.39) All Other Causes - $PM_{2.5}$ Only: 1.11 (0.74,1.67) 44 Ind. Covariates together + $PM_{2.5}$ + O_3: 0.95 (0.64,1.39) 44 Ind. Covariates together + intersection within freeways within 500 m + $PM_{2.5}$ + O_3: 1.02 (0.71,1.48)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Laden et al. (2006, 087605)	**Outcome:** Total mortality	**Pollutant:** $PM_{2.5}$	**Increment:** 10 $\mu g/m^3$
Period of Study: 1974-1998	Non-accidental (< 800)	**Averaging Time:** Annual avg	**Relative Risk (Lower CI, Upper CI)**
Period 1: 1974-1989	Cardiovascular (400-440)	**Mean (SD):** Period 1	**lag:**
Period 2: 1990-1998	Respiratory (485-496)	Portage: 11.4	**Period 1:** Portage: 1.00
Location: Nine US Cities	Lung Cancer (162)	Topeka: 12.4	Topeka: 1.06 (0.86, 1.31)
Watertown, MA	Other	Watertown: 15.4	Watertown: 1.06 (0.87, 1.28)
Kingston, TN	**Study Design:** Prospective Cohort	Harriman: 20.9	Harriman: 1.19 (0.98, 1.44)
Harriman, TN	**Statistical Analyses:** Cox proportional hazards regression	St Louis: 19.2	St Louis: 1.15 (0.96, 1.38)
St. Louis, MO	**Age Groups:** 25-74	Steubenville: 29.0	Steubenville: 1.31 (1.10, 1.57)
Steubenville, OH		Period 2	**Period 2:** Portage: NR
Portage, WI		Portage: 10.2	Topeka: 1.01 (0.83, 1.22)
Wyocena, WI		Topeka: 13.1	Watertown: 0.82 (0.67, 1.00)
Pardeeville, WI		Watertown: 12.1	Harriman: 1.10 (0.91, 1.33)
Topeka, KS		Harriman: 18.1	St Louis: 0.96 (0.80, 1.15)
		St. Louis: 13.4	Steubenville: 1.06 (0.89, 1.27)
		Steubenville: 22.0	**Complete Period:** Portage: 1.00
			Topeka: 1.03 (0.89, 1.19)
			Watertown: 0.95 (0.83, 1.08)
			Harriman: 1.15 (1.01, 1.32)
			St. Louis: 1.05 (0.93, 1.20)
			Steubenville: 1.18 (1.04, 1.34)
			RR for complete follow up Avg. $PM_{2.5}$
			Total Mortality: 1.16 (1.07, 1.26)
			Cardiovascular: 1.28 (1.13, 1.44)
			Respiratory: 1.08 (0.79, 1.49)
			Lung Cancer: 1.27 (0.96, 1.69)
			Other: 1.02 (0.90, 1.17)
			RR for period one Avg. $PM_{2.5}$
			Total Mortality: 1.18 (1.09, 1.27)
			Cardiovascular: 1.28 (1.14, 1.43)
			Respiratory: 1.21 (0.89, 1.66)
			Lung Cancer: 1.20 (0.91, 1.58)
			Other: 1.05 (0.93, 1.19)
			Decrease in Avg. $PM_{2.5}$ **over the two periods**
			Total Mortality: 0.73 (0.57, 0.95)
			Cardiovascular: 0.69 (0.46, 1.01)
			Respiratory: 0.43 (0.16, 1.13)
			Lung Cancer: 1.06 (0.43, 2.62)
			Other: 0.85 (0.56, 1.27)
Reference: Lipfert et al. (2006, 088756)	**Outcome:** Mortality	**Pollutant:** Sulfate	**Increment:** 8
Period of Study: 1989-1996	**Study Design:** Retrospective Cohort	**Mean (SD) from 1976-81:** 10.7 (3.6)	1.045 (0.944, 1.157)
Location: Various parts of the Untied States	**Statistical Analyses:** Cox proportional hazards regression		
	Age Groups: Male US veterans between ages of 39 and 63 (Avg. age: 51)		

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Lipfert et al. (2006, 088756)	**Outcome:** Mortality	**Pollutant:** $PM_{2.5}$	**Increment:** 8
Period of Study: 1989-1996	**Study Design:** Retrospective Cohort	**Mean (SD):** 14.3 (3.2)	1.118 (1.038, 1.203)
Location: Various parts of the Untied States	**Statistical Analyses:** Cox proportional hazards regression		
	Age Groups: Male US veterans between ages of 39 and 63 (Avg age 51)		

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Lipfert et al. (2006, 088218) **Period of Study:** 1997-2002 **Location:** Various parts of the Untied States	**Outcome:** Mortality: Non- accidental (< 800) **Study Design:** Retrospective cohort **Statistical Analyses:** Cox proportional hazards regression AIC **Age Groups:** Male US veterans between ages of 39 and 63 (Avg. age: 51)	**Pollutant:** $PM_{2.5}$ **Averaging Time:** Annual avg **Mean (SD):** 15.02 (4.80) $\mu g/m^3$ (2000-2003) **Range (Min, Max):** (3.29, 24.96) **Copollutant (correlation):** As: r = 0.443 Cr: r = 0.379 Cu: r = 0.530 Fe: r = 0.379; Pb: r = 0.489 Mn: r = 0.389; Ni: r = 0.140 Se: r = 0.312; V: r = 0.197 Zn: r = 0.420; OC: r = 0.620 EC: r = 0.544; | SO_4: r = 0.827 NO_3: r = 0.649 NO_2: r = 0.641 Peak CO: r = 0.040 Peak O_3: r = 0.222 Peak SO_2: r = 0.714	**Increment:** 10 $\mu g/m^3$ **% Increase per 10 $\mu g/m^3$ increase in $PM_{2.5}$** **Single-Pollutant Model** As: -5.23% Cr: -2.11% Cu: 2.12% Fe: 2.81% Pb: -2.40% Mn: -1.20% Ni: 3.75% Se: -0.30% V: 5.08% Zn: 1.52% OC: -0.02% EC: 9.16% SO_4: 3.04% NO_3: 6.60% NO_2: 6.92% Peak CO: -0.61% Peak O_3: 4.95% Peak SO_2: -4.20% **Multiple Pollutants model- Pollutant with traffic density** NO_3: 3.42% SO_4: -2.73% EC: 6.27% Ni: 2.51% V: 3.27% **Pollutant with NO_3** EC: 5.93% Ni: 2.31% V: 3.11% **Pollutant with Peak O_3** **Traffic density: 2.40%** EC: 10.79% Fe: 5.94% NO_3: 7.57% $PM_{2.5}$: 8.97% V: 4.93% Ni: 3.65% SO_4: 6.75% Cu: 1.55% OC: 0.21%

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Krewski et al. (2009, 191193) **Period of Study:** 1979-2000 **Location:** 48 contiguous states US	**Outcome:** Death **Study Design:** cohort **Covariates:** demographic, socioeconomic and ecologic characteristics **Statistical Analysis:** Cox proportional-hazards model **Statistical Package:** NR **Age Groups:** Adults of at least 30 years	**Pollutant:** $PM_{2.5}$ **Averaging Time:** NR **Mean Unit:** 1979-1983: 21.20 $\mu g/m^3$ 1999-2000: 14.02 $\mu g/m^3$ **Range (Min, Max):** 1979-1983: 10.77-30.01 1999-2000: 5.80-22.20 **Copollutant:** SO_4^{2-}, SO_2, PM_{15}, TSP, O_3, NO_2, CO	**Increment:** 10$\mu g/m^3$ **Hazard Ratio (95% CI)** **MSA & DIFF** Increment Change: 10.78 (1.043-1.115) Change 5-15 $\mu g/m^3$: 1.128 (1.077-1.183) Change 10-20 $\mu g/m^3$: 1.079 (1.048-1.112) **HR (95% CI)** **Los Angeles** Parsimonious ecologic covariates: 1.126 (1.014-1.251) **HR (95% CI)** **15 year time window** Group A: 0.98 (0.92-1.06) Group B: 1.01 (0.99-1.02) **HR (95% CI)** **Third follow-up, 7 Ecologic Variables** 1979-1983: 1.044 (1.028-1.060) 1999-2000: 1.057 (1.036-1.079) **HR (95% CI)** **Nationwide analysis, 1999-2000** Standard Cox: 1.03 (1.01-1.05) Random Effects Cox: 1.06 (1.04-1.08) **Increment:** 1.5 $\mu g/m^3$ **HR (95% CI)** **28 County, 3 year model** All 7 ecologic covariates: 0.977 (0.932-1.025)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Krewski et al. (2009, 191193)	**Outcome:** Death from cardiopulmonary disease	**Pollutant:** $PM_{2.5}$	**Increment:** 10 $\mu g/m^3$
Period of Study: 1979-2000	**Study Design:** cohort	**Averaging Time:** NR	**Hazard Ratio (95% CI)**
Location: 48 contiguous states US	**Covariates:** demographic, socioeconomic and ecologic characteristics	**Mean Unit:**	**MSA & DIFF**
	Statistical Analysis: Cox proportional-hazards model	1979-1983: 21.20 $\mu g/m^3$	Increment Change: 1.078 (1.077-1.182)
	Statistical Package: NR	1999-2000: 14.02 $\mu g/m^3$	Change 5-15 $\mu g/m^3$: 1.208 (1.132-1.290)
	Age Groups: Adults of at least 30 years	**Range (Min, Max):**	Change 10-20 $\mu g/m^3$: 1.127 (1.081-1.174)
		1979-1983: 10.77-30.01	**HR (95% CI)**
		1999-2000: 5.80-22.20	**Los Angeles**
		Copollutant: SO_4^{2-}, SO_2, PM_{15}, TSP, O_3, NO_2, CO	Parsimonious ecologic covariates: 1.086 (0.939-1.285)
			HR (95% CI)
			15 year time window
			Group A: 1.00 (0.90-1.11)
			Group B: 1.05 (1.03-1.07)
			HR (95% CI)
			Third follow-up, 7 Ecologic Variables
			1979-1983: 1.094 (1.070-1.118)
			1999-2000: 1.138 (1.106-1.172)
			HR (95% CI)
			Nationwide analysis, 1999-2000
			Standard Cox: 1.09 (1.06-1.12)
			Random Effects Cox: 1.13 (1.10-1.16)
			Increment: 1.5 $\mu g/m^3$
			HR (95% CI)
			28 County, 3 year model
			All 7 ecologic covariates: 0.940 (0.875-1.011)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Krewski et al. (2009, 191193)	**Outcome:** Death from ischemic heart disease	**Pollutant:** $PM_{2.5}$	**Increment:** 10 $\mu g/m^3$
Period of Study: 1979-2000	**Study Design:** cohort	**Averaging Time:** NR	**Hazard Ratio (95% CI)**
Location: 48 contiguous states US	**Covariates:** demographic, socioeconomic and ecologic characteristics	**Mean Unit:**	**MSA & DIFF**
	Statistical Analysis: Cox proportional-hazards model	1979-1983: 21.20 $\mu g/m^3$	Increment Change: 1.196 (1.177-1.407)
	Statistical Package: NR	1999-2000: 14.02 $\mu g/m^3$	Change 5-15 $\mu g/m^3$: 1.484 (1.311-1.680)
	Age Groups: Adults of at least 30 years	**Range (Min, Max):**	Change 10-20 $\mu g/m^3$: 1.283 (1.186-1.387)
		1979-1983: 10.77-30.01	**HR (95% CI)**
		1999-2000: 5.80-22.20	**Los Angeles**
		Copollutant: SO_4^{2-}, SO_2, PM_{15}, TSP, O_3, NO_2, CO	Parsimonious ecologic covariates: 1.263 (10.22-1.563)
			HR (95% CI)
			Third follow-up, 7 Ecologic Variables
			1979-1983: 1.184 (1.146-1.222)
			1999-2000: 1.242 (1.191-1.295)
			HR (95% CI)
			Nationwide analysis, 1999-2000
			Standard Cox: 1.15 (1.11-1.20)
			Random Effects Cox: 1.24 (1.19-1.29)
			Increment: 1.5 $\mu g/m^3$
			HR (95% CI)
			28 County, 3 year model
			All 7 ecologic covariates: 1.072 (0.980-1.172)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Krewski et al. (2009, 191193) **Period of Study:** 1979-2000 **Location:** 48 contiguous states US	**Outcome:** Death from lung cancer **Study Design:** cohort **Covariates:** demographic, socioeconomic and ecologic characteristics **Statistical Analysis:** Cox proportional-hazards model **Statistical Package:** NR **Age Groups:** Adults of at least 30 years	**Pollutant:** $PM_{2.5}$ **Averaging Time:** NR **Mean Unit:** 1979-1983: 21.20 $\mu g/m^3$ 1999-2000: 14.02 $\mu g/m^3$ **Range (Min, Max):** 1979-1983: 10.77-30.01 1999-2000: 5.80-22.20 **Copollutant:** SO_4^{2-}, SO_2, PM_{15}, TSP, O_3, NO_2, CO	**Increment:** 10$\mu g/m^3$ **Hazard Ratio (95% CI)** **MSA & DIFF** Increment Change: 1.142 (1.057-1.234) Change 5-15 $\mu g/m^3$: 1.236 (1.114-1.372) Change 10-20 $\mu g/m^3$: 1.143 (1.071-1.221) **HR (95% CI)** **Los Angeles** Parsimonious ecologic covariates: 1.311 (0.897-1.915) **HR (95% CI)** **15 year time window** Group A: 1.08 (0.87-1.35) Group B: 1.07 (1.02-1.13) **HR (95% CI)** **Third follow-up, 7 Ecologic Variables** 1979-1983: 1.092 (1.033-1.154) 1999-2000: 1.138 (1.057-1.225) **HR (95% CI)** **Nationwide analysis, 1999-2000** Standard Cox: 1.11 (1.04-1.18) Random Effects Cox: 1.14 (1.06-1.23) **Increment:** 1.5 $\mu g/m^3$ **HR (95% CI)** **28 County, 3 year model** All 7 ecologic covariates: 0.985 (0.832-1.166)
Reference: Krewski et al. (2009, 191193) **Period of Study:** 1979-2000 **Location:** 48 contiguous states US	**Outcome:** Death from diabetes **Study Design:** cohort **Covariates:** demographic, socioeconomic and ecologic characteristics **Statistical Analysis:** Cox proportional-hazards model **Statistical Package:** NR **Age Groups:** Adults of at least 30 years	**Pollutant:** $PM_{2.5}$ **Averaging Time:** NR **Mean Unit:** 1979-1983: 21.20 $\mu g/m^3$ 1999-2000: 14.02 $\mu g/m^3$ **Range (Min, Max):** 1979-1983: 10.77-30.01 1999-2000: 5.80-22.20 **Copollutant:** SO_4^{2-}, SO_2, PM_{15}, TSP, O_3, NO_2, CO	**Increment:** 1.5 $\mu g/m^3$ **HR (95% CI)** **28 County, 3 year model** All 7 ecologic covariates: 1.083 (0.723-1.621)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Krewski et al. (2009, 191193) **Period of Study:** 1979-2000 **Location:** 48 contiguous states US	**Outcome:** Death from endocrine disease **Study Design:** cohort **Covariates:** demographic, socioeconomic and ecologic characteristics **Statistical Analysis:** Cox proportional-hazards model **Statistical Package:** NR **Age Groups:** Adults of at least 30 years	**Pollutant:** $PM_{2.5}$ **Averaging Time:** NR **Mean Unit:** 1979-1983: 21.20 $\mu g/m^3$ 1999-2000: 14.02 $\mu g/m^3$ **Range (Min, Max):** 1979-1983: 10.77-30.01 1999-2000: 5.80-22.20 **Copollutant:** SO_4^{2-}, SO_2, PM_{15}, TSP, O_3, NO_2, CO	**Increment:** 1.5 $\mu g/m^3$ **HR (95% CI)** **28 County, 3 year model** All 7 ecologic covariates: 1.143 (0.835-1.564)
Reference: Krewski et al. (2009, 191193) **Period of Study:** 1979-2000 **Location:** 48 contiguous states US	**Outcome:** Death from digestive cancer **Study Design:** cohort **Covariates:** demographic, socioeconomic and ecologic characteristics **Statistical Analysis:** Cox proportional-hazards model **Statistical Package:** NR **Age Groups:** Adults of at least 30 years	**Pollutant:** $PM_{2.5}$ **Averaging Time:** NR **Mean Unit:** 1979-1983: 21.20 $\mu g/m^3$ 1999-2000: 14.02 $\mu g/m^3$ **Range (Min, Max):** 1979-1983: 10.77-30.01 1999-2000: 5.80-22.20 **Copollutant:** SO_4^{2-}, SO_2, PM_{15}, TSP, O_3, NO_2, CO	**Increment:** 10 $\mu g/m^3$ **HR (95% CI)** **Los Angeles** Parsimonious ecologic covariates: 1.199 (0.817-1.758)
Reference: Krewski et al. (2009, 191193) **Period of Study:** 1979-2000 **Location:** 48 contiguous states US	**Outcome:** Death cancers other than lung and digestive **Study Design:** cohort **Covariates:** demographic, socioeconomic and ecologic characteristics **Statistical Analysis:** Cox proportional-hazards model **Statistical Package:** NR **Age Groups:** Adults of at least 30 years	**Pollutant:** $PM_{2.5}$ **Averaging Time:** NR **Mean Unit:** 1979-1983: 21.20 $\mu g/m^3$ 1999-2000: 14.02 $\mu g/m^3$ **Range (Min, Max):** 1979-1983: 10.77-30.01 1999-2000: 5.80-22.20 **Copollutant:** SO_4^{2-}, SO_2, PM_{15}, TSP, O_3, NO_2, CO	**Increment:** 10 $\mu g/m^3$ **HR (95% CI)** **Los Angeles** Parsimonious ecologic covariates: 1.012 (0.788-1.299)
Reference: Krewski et al. (2009, 191193) **Period of Study:** 1979-2000 **Location:** 48 contiguous states US	**Outcome:** Deaths from causes other than CPD, IHD and lung cancer **Study Design:** cohort **Covariates:** demographic, socioeconomic and ecologic characteristics **Statistical Analysis:** Cox proportional-hazards model **Statistical Package:** NR **Age Groups:** Adults of at least 30 years	**Pollutant:** $PM_{2.5}$ **Averaging Time:** NR **Mean Unit:** 1979-1983: 21.20 $\mu g/m^3$ 1999-2000: 14.02 $\mu g/m^3$ **Range (Min, Max):** 1979-1983: 10.77-30.01 1999-2000: 5.80-22.20 **Copollutant:** SO_4^{2-}, SO_2, PM_{15}, TSP, O_3, NO_2, CO	**Increment:** 10 $\mu g/m^3$ **Hazard Ratio (95% CI)** **MSA & DIFF** Increment Change: 1.010 (0.968-1.055) Change 5-15 $\mu g/m^3$: 1.026 (0.970-1.085) Change 10-20 $\mu g/m^3$: 1.016 (0.981-1.053) **HR (95% CI)** **Third follow-up, 7 Ecologic Variables** 1979-1983: 0.983 (0.960-1.007) 1999-2000: 0.953 (0.923-0.984)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: McDonnell et al. (2000, 010319) **Period of Study:** 1973-1977 **Location:** California	**Outcome:** Mortality **Study Design:** Cohort (AHSMOG airport cohort) **Statistical Analyses:** Cox regression models **Age Groups:** Males, 27 yrs +	**Pollutant:** $PM_{2.5}$ **Averaging Time:** monthly averages **Mean (SD):** 31.9 (10.7) **IQR:** 24.3 **Copollutants (correlation):** O_3: 0.68 SO_2: 0.18 NO_2: -0.08; SO_4: 0.33	**Increment:** IQR All Cause: 1.22 (0.95-1.58) Resp: 1.64 (0.93-2.90) Lung Cancer: 2.23 (0.56-8.94)
Reference: Miller et al. (2007, 090130) **Period of Study:** 1994-1998 **Location:** 36 US Metropolitan Areas	**Outcome:** CVD Mortality **Study Design:** Prospective Cohort (WHI) **Statistical Analyses:** Cox proportional hazards regression **Age Groups:** postmenopausal women ages 50-79	**Pollutant:** $PM_{2.5}$ **Averaging Time:** annual avg (2000) **Mean (SD):** 13.4 **IQR:** 11.6, 18.3 **Range:** 3.4, 28.3	**Increment:** 10 $\mu g/m^3$ CVD Death: 1.76 (1.25, 2.47) CHD Death: 2.21 (1.17, 4.16) CV Death: 1.83 (1.11, 3.00)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Naess et al. (2007, 090736) **Period of Study:** 1992-1998 **Location:** Oslo, Norway	**Outcome:** Mortality: Non-accidental (< 800) Lung cancer (162) COPD (490-496) Cardiovascular (390-459) **Study Design:** Prospective Cohort **Statistical Analyses:** Cox proportional hazards regression model **Age Groups:** 51-70, 71-90	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 4 year avg **Mean (SD):** $PM_{2.5}$: 15 **Range (Min, Max):** $PM_{2.5}$: (7, 22) **Copollutant (correlation):** NO_2: r = 0.95	**Relative Risk (CI min, CI max)** RR for deaths from all causes Men (ages 51-70) $PM_{2.5}$ exposure (in $\mu g/m^3$) 6.56-11.45: 1.00 11.46-14.25: 0.96 (0.89, 1.04) 14.26-18.43: 1.12 (1.03, 1.22) 18.44-22.34: 1.48 (1.36, 1.60) Men (ages 71-90) $PM_{2.5}$ exposure (in $\mu g/m^3$) 6.56-11.45: 1.00 11.46-14.25: 0.99 (0.93, 1.06) 14.26-18.43: 1.10 (1.03, 1.17) 18.44-22.34: 1.19 (1.12, 1.27) Women (ages 51-70) $PM_{2.5}$ exposure (in $\mu g/m^3$) 6.56-11.45: 1.00 11.46-14.25: 0.96 (0.87, 1.07) 14.26-18.43: 1.08 (0.98, 1.20) 18.44-22.34: 1.44 (1.30, 1.59) Women (ages 71-90) $PM_{2.5}$ exposure (in $\mu g/m^3$) 6.56-11.45: 1.00 11.46-14.25: 1.03 (0.97, 1.09) 14.26-18.43: 1.07 (1.01, 1.12) 18.44-22.34: 1.11 (1.05, 1.16) **Increment:** 10 $\mu g/m^3$ RR for death from CVD and lung cancer Men (ages 51-70) CVD- $PM_{2.5}$: 1.11 (1.06, 1.16) COPD- $PM_{2.5}$: 1.32 (1.17, 1.49) Lung Cancer- $PM_{2.5}$: 1.07 (0.98, 1.17) Women (ages 51-70) CVD: $PM_{2.5}$: 1.16 (1.09, 1.24) COPD: $PM_{2.5}$: 1.18 (1.03, 1.34) Lung Cancer: $PM_{2.5}$: 1.23 (1.10, 1.37) Men (ages 71-90) CVD: $PM_{2.5}$: 1.06 (1.03, 1.09) COPD: PM_{10}: 1.13 (1.04, 1.24) $PM_{2.5}$: 1.14 (1.04, 1.24) Lung Cancer: $PM_{2.5}$: 1.08 (0.98, 1.19) Women (ages 71-90) CVD: $PM_{2.5}$: 1.02 (1.00, 1.05) COPD: $PM_{2.5}$: 1.09 (1.00, 1.18) Lung Cancer: $PM_{2.5}$: 1.16 (1.03, 1.31)
Reference: Naess et al. (2007, 090736) **Period of Study:** 1992-1998	**Outcome:** Mortality: Lung cancer (162) COPD (490-496)	**Pollutant:** $PM_{2.5}$ **Averaging Time:** (Month-year) avg	**Relative Risk (CI min, CI max)** **RR on All-cause mortality of $PM_{2.5}$ in**

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Location: Oslo, Norway	Cardiovascular (390-459) Psychiatric causes (290, 292-302, 304, 306-319) Stomach cancer (151) Violence (800-999) **Study Design:** Multilevel cohort **Statistical Analyses:** WinBUGS **Age Groups:** 50-74	**Range Mean (SD):** 14.2 (3.6) **IQ Range (1st, 4th):** (6.6, 22.3) **Copollutant (correlation):** PM_{10}: r = 0.95 | NO_2: r = 0.87	**Men Age 50-74** Primary Education: $PM_{2.5}$: 1.06 (1.00, 1.11) Individual: 1.34 (1.24, 1.43) Neighborhood: 1.22 (1.16, 1.28) Manual Class: $PM_{2.5}$: 1.06 (1.01, 1.12) Individual: 1.28 (1.20, 1.37) Neighborhood: 1.20 (1.14, 1.26) Income below median: $PM_{2.5}$: 1.05 (1.00, 1.12) Individual: 1.44 (1.35, 1.53) Neighborhood: 1.16 (1.11, 1.21) Not owner occupied: $PM_{2.5}$: 1.06 (1.00, 1.13) Individual: 1.24 (1.12, 1.36) Neighborhood: 1.11 (1.05, 1.17) Lives in flat dwelling: $PM_{2.5}$: 1.04 (0.98, 1.11) Individual: 1.19 (1.09, 1.31) Neighborhood: 1.10 (1.04, 1.17) More than one person per room in dwelling: $PM_{2.5}$: 1.10 (1.02, 1.18) Individual: 1.05 (0.98, 1.13) Neighborhood: 1.01 (0.96, 1.05) **RR on All-cause mortality of $PM_{2.5}$ in Women Age 50-74** Primary Education Only: $PM_{2.5}$: 1.05 (1.00, 1.11) Individual: 1.32 (1.23, 1.42) Neighborhood: 1.18 (1.12, 1.24) Manual Class: $PM_{2.5}$: 1.07 (1.01, 1.13) Individual: 1.27 (1.18, 1.36) Neighborhood: 1.18 (1.12, 1.24) Income below median: $PM_{2.5}$: 1.05 (1.01, 1.10) Individual: 1.52 (1.41, 1.63) Neighborhood: 1.13 (1.09, 1.18) Not owner occupied: $PM_{2.5}$: 1.07 (1.01, 1.14) Individual: 1.24 (1.12, 1.38) Neighborhood: 1.08 (1.02, 1.14) Lives in a flat dwelling: $PM_{2.5}$: 1.05 (0.99, 1.11) Individual: 1.21 (1.09, 1.34) Neighborhood: 1.09 (1.02, 1.15) More than one person per room in dwelling: $PM_{2.5}$: 1.11 (1.04, 1.19) Individual: 1.07 (0.99, 1.14) Neighborhood: 1.01 (0.96, 1.05) **RR for Interquartile Increase (MI) in**

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			$PM_{2.5}$ for different causes of death
			CVD: Age and sex adjusted: 1.11 (1.07, 1.15)
			Primary education only: M1 + Individual: 1.07 (1.04, 1.11)
			M1 + Neighborhood: 1.03 (1.00, 1.07)
			Manual Class: M1 + Individual: 1.08 (1.04, 1.11)
			M1 + Neighborhood: 1.06 (1.02, 1.10)
			Income below Median: M1 + Individual: 1.07 (1.03, 1.11)
			M1 + Neighborhood: 1.02 (0.98, 1.05)
			Not owner occupied: M1 + Individual: 1.05 (1.01, 1.09)
			M1 + Neighborhood: 1.03 (0.99, 1.07): Living in a Flat dwelling
			M1 + Individual: 1.04 (1.00, 1.08)
			M1 + Neighborhood: 1.01 (0.97, 1.05)
			Crowded household: M1 + Individual: 1.10 (1.05, 1.14)
			M1 +Neighborhood: 1.10 (1.06, 1.15)
			Pulmonary Cancer: Age and sex adjusted: 1.12 (1.05, 1.19)
			Primary education only: M1 + Individual: 1.09 (1.01, 1.17)
			M1 + Neighborhood: 1.05 (0.98, 1.13)
			Manual Class: M1 + Individual: 1.09 (1.01, 1.17)
			M1 + Neighborhood: 1.10 (1.06, 1.13)
			Income below Median: M1 + Individual: 1.09 (1.01, 1.17)
			M1 + Neighborhood: 1.02 (0.95, 1.10)
			Not owner occupied: M1 + Individual: 1.07 (1.00, 1.15)
			M1 + Neighborhood: 1.04 (0.97, 1.12)
			Living in a Flat dwelling: M1 + Individual: 1.03 (0.96, 1.11)
			M1 + Neighborhood: 1.00 (0.92, 1.08)
			Crowded household: M1 + Individual: 1.10 (1.03, 1.14)
			M1 +Neighborhood: 1.11 (1.04, 1.20)
			COPD: Age and sex adjusted: 1.17 (1.09, 1.25)
			Primary education only: M1 + Individual: 1.13 (1.05, 1.22)
			M1 + Neighborhood: 1.09 (1.01, 1.19)
			Manual Class: M1 + Individual: 1.14 (1.05, 1.23)
			M1 + Neighborhood: 1.12 (1.04, 1.22)
			Income below Median: M1 + Individual: 1.13 (1.04, 1.22)
			M1 + Neighborhood: 1.06 (0.97, 1.15)
			Not owner occupied: M1 + Individual:

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			1.10 (1.02, 1.19) M1+ Neighborhood: 1.07 (0.99, 1.16) Living in a Flat dwelling: M1+ Individual: 1.08 (1.00, 1.18) M1+ Neighborhood: 1.03 (0.95, 1.13) Crowded household: M1+ Individual: 1.16 (1.07, 1.26) M1+Neighborhood: 1.16 (1.07, 1.26) Estimates for psychiatric diseases, genetic cancer and violent death
Reference: Nerriere et al. (2005, 088630) **Period of Study:** Grenoble (2001) Paris (2002) Rouen (2002-2003) Strasbourg (2003) **Location:** Four French Cities- Grenoble, Rouen, Paris, and Strasbourg	**Outcome:** Mortality: Lung Cancer (162) **Study Design:** Time-series **Statistical Analyses:** GIS **Age Groups:** 30-71 year old nonsmoking adults	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 48-h avg **Mean Range:** 17 to 49 $\mu g/m^3$	**Increment:** 10 $\mu g/m^3$ **% Increase (Lower CI, Upper CI)** % increase in lung cancer deaths attributable to $PM_{2.5}$ exposure France: 8 (1, 16) Grenoble: 10 (3, 19) Rouen: 10 (2, 19) Strasbourg: 24 (4, 40)
Reference: Ozkaynak and Thurston (1987, 072960) **Period of Study:** 1980 **Location:** U.S.	**Outcome:** Total Mortality **Study Design:** Cross-sectional **Statistical Analyses:** Multiple regression analysis	**Pollutant:** Sulfate **Averaging Time:** Annual avg **Mean Range:** Sulfate: 11.1 (3.5)	**Range of estimated total mortality effects of air pollutions:** Sulfate: 4-9% "Sulfate concentration was consistently found to be a significant predictor of mortality in the models considered. Fine particle mass coefficients were also often found to be statistically significant in the mortality regressions."

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Pope et al. (2004, 055880) **Period of Study:** 1982-2000 **Location:** Metropolitan areas in all 50 states in the US	**Outcome:** Mortality: Cardiovascular Diseases (390-459) Diabetes (250) Respiratory Disease (460-519) **Study Design:** Prospective Cohort **Statistical Analyses:** Cox proportional hazards regression **Age Groups:** > 30	**Pollutant:** $PM_{2.5}$ **Averaging Time:** Annual avg **Mean (SD):** 17.1 (3.7) **Range (Min, Max):** NR	**Increment:** 10 $\mu g/m^3$ **Relative Risk (Lower CI, Upper CI)** All cardiovascular disease plus diabetes: $PM_{2.5}$: 1.12 (1.08, 1.15) Former Smoker: 1.26 (1.23, 1.28) Current Smoker: 1.94 (1.90, 1.99) Ischemic Heart Disease: $PM_{2.5}$: 1.18 (1.14, 1.23) Former Smoker: 1.33 (1.29, 1.37) Current Smoker: 2.03 (1.96, 2.10) Diabetes: $PM_{2.5}$: 0.99 (0.86, 1.14) Former Smoker: 1.05 (0.94, 1.16) Current Smoker: 1.35 (1.20, 1.53) All other Cardiovascular Diseases: $PM_{2.5}$: 0.84 (0.71, 0.99) Former Smoker: 1.22 (1.09, 1.38) Current Smoker: 1.78 (1.56, 2.04) Diseases of the respiratory system: $PM_{2.5}$: 0.92 (0.86, 0.98) Former Smoker: 2.16 (2.04, 2.28) Current Smoker: 3.88 (3.66, 4.11) COPD: $PM_{2.5}$: 0.84 (0.77, 0.93) Former Smoker: 4.93 (4.48, 5.42) Current Smoker: 9.85 (8.95, 10.84) All other respiratory diseases: $PM_{2.5}$: 0.86 (0.73, 1.02) Former Smoker: 1.54 (1.36, 1.74) Current Smoker: 1.83 (1.57, 2.12)
Reference: Pope et al. (2007, 091256) **Period of Study:** 1960-1975 **Location:** New Mexico, Arizona, Utah, and Nevada	**Outcome (ICD7&8):** Mortality: Cardiovascular (ICD 7: 400-468, 331, 332 ICD 8: 390-458) Respiratory (ICD 7: 470-527 ICD 8: 460-519) Influenza/ pneumonia (ICD 7: 480-483, 490-493, ICD 8: 470-474, 480-486) **Study Design:** Retrospective Cohort **Statistical Analyses:** Poisson regression model GAM SAS **Age Groups:** All smelter workers > 18	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-h avg **Mean (SD):** NR **Range (Min, Max):** NR	The study does not present quantitative results results are presented in figures. The References found that the strike-related estimated percent decrease in mortality was 2.5% (1.1-4.0),

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Pope et al. (2009, 190107) **Period of Study:** 1978-1982, 1997-2001 **Location:** 211 US counties and 51 metropolitan areas	**Outcome:** Increased life expectancy **Study Design:** Cross-sectional **Statistical Analysis:** Cross-sectional regression **Age Groups:** Adults ≥45 years	**Pollutant:** $PM_{2.5}$ **Averaging Time:** daily, quarterly and annual **Mean (SD) Unit:** 1979-1983: 20.61 ± 4.36 $\mu g/m^3$ 1999-2000: 14.10 ± 2.86 $\mu g/m^3$ **Range (Min, Max):** NR **Copollutant (correlation):** NR	**Increment:** 10 $\mu g/m^3$ **Regression Coefficient ± SD** 211 County Units Intercept: 1.75 ± 0.27 Reduction in $PM_{2.5}$: 0.61 ± 0.20 Change in Income: 0.13 ± 0.01 Change in Population: 0.06 ± 0.02 Change in Black Population: -2.70 ± 0.64 Change in Lung Cancer Mortality Rate: -0.06 ± 0.02 Change in COPD Mortality Rate: -0.08 ± 0.02 R: 0.53 51 Metropolitan Areas Intercept: 2.09 ± 0.36 Reduction in $PM_{2.5}$: 0.95 ± 0.23 Change in Income: 0.11 ± 0.02 Change in Population: 0.05 ± 0.02 Change in Black Population: -5.98 ± 1.99 Change in Lung Cancer Mortality Rate: 0.02 ± 0.03 Change in COPD Mortality Rate: -0.19 ± 0.05 R: 0.74
Reference: Rainham et al. (2005, 088676) **Period of Study:** 1981-1999 **Location:** Toronto, Canada	**Outcome:** Total deaths (ICD9 < 800), cardiorespiratory (390-459), non-cardiorespiratory (ICD9-NR) **Study Design:** Time-series **Statistical Analyses:** Generalized linear models were used **Season:** Winter (December–February) Summer (June–August) **Statistical Package:** S-Plus 6.1	**Pollutant:** $PM_{2.5}$ **Averaging Time:** NR **Mean (SD):** All years: 17.0 (8.7) $\mu g/m^3$ Winters: 17.2 (6.8) Summers: 18.8 (10.2) Avg Winter values: Dry Moderate: 17.0 (1.0) Dry Polar: 17.5 (0.5) Dry Tropical: No Comparison Moist Moderate: 17.1 (0.8) Moist Polar: 17.5 (0.6) Moist Tropical: 16.5 (3.6) Transition: 16.7 (1.0) Avg summer values: Dry Moderate: 18.4 (0.9) Dry Polar: 19.0 (1.2) Dry Tropical: 18.5 (2.4) Moist Moderate: 19.2 (1.2) Moist Polar: 17.5 (2.0) Moist Tropical: 19.8 (1.1) Transition: 17.6 (1.5)	**Mortality risk for winter season and within winter synoptic weather categories** **RR Estimate [Lower CI, Upper CI]:** Winter: Total: 0.998[0.997, 1.000] Cardioresp: 0.998[0.996, 1.000] Other: 0.998 [0.996, 1.000] Dry Moderate: Total: 1.001[0.996, 1.007] Cardioresp: 1.005[0.998, 1.011] Other: 1.002 [0.998, 1.005] Dry Polar: Total: 0.998[0.995, 1.001] Cardioresp: 0.995[0.991, 0.999] Other: 1.002 [0.998, 1.005] Dry Tropical: NA Moist Moderate: Total: 0.998[0.993, 1.002] Cardioresp: 1.003[0.995, 1.010] Other: 0.997 [0.991, 1.004] Moist Polar: Total: 1.001[0.998, 1.005] Cardioresp: 1.002[0.997, 1.007] Other: 1.003 [0.999, 1.007] Moist Tropical: Total: 1.007[0.965, 1.203] Cardioresp: 1.123[1.031, 1.224]

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			Other: 1.248 [1.123, 1.387]
			Transition Total: 1.003[0.996, 1.009]
			Cardioresp: 0.996[0.987, 1.004]
			Other: 0.997 [0.990, 1.004]
			Mortality risk for summer season and within summer synoptic weather categories
			RR Estimate [Lower CI, Upper CI]: Summer: Total: 1.000[1.000, 1.001]
			Cardioresp: 1.001[1.000, 1.002]
			Other: 1.001[1.000, 1.002]
			Dry Moderate: Total: 1.001[0.999, 1.002]
			Cardioresp: 1.002[0.999, 1.004]
			Other: 0.999[0.997, 1.002]
			Dry Polar: Total: 1.002[0.999, 1.005]
			Cardioresp: 0.996[0.991, 1.000]
			Other: 1.003[0.999, 1.007]
			Dry Tropical: Total: 1.016[1.006, 1.027]
			Cardioresp: 1.017[1.005, 1.030]
			Other: 1.017 [1.003, 1.031]
			Moist Moderate: Total: 1.002[1.000, 1.004]
			Cardioresp: 1.003[0.999, 1.006]
			Other: 1.004 [1.001, 1.006]
			Moist Polar: Total: 1.005[0.998, 1.011]
			Cardioresp: 1.008[0.997, 1.018]
			Other: 1.003 [0.995, 1.011]
			Moist Tropical: Total: 0.999[0.997, 1.001]
			Cardioresp: 0.996[0.993, 1.000]
			Other: 0.998 [0.995, 1.001]
			Transition: Total: 1.005[0.996, 1.014]
			Cardioresp: 1.007[0.994, 1.020]
			Other: 1.002 [0.996, 1.008]
Reference: Roman et al. (2008, 156921)	**Outcome:** Mortality	**Pollutant:** $PM_{2.5}$	Quantitative results are not presented in the text, but can be found graphically in Figure 3.
Period of Study: 2006	**Study Design:** Expert Judgment Study	**Averaging Time:** annual average	
Location: U.S.	**Statistical Analyses:** Standard best practices for expert elicitation	**Mean (SD):** 4-30	"Most of the experts' central estimates fall at or above the 2002 ACS median (0.6% per $\mu g/m^3$) and below the original Six Cities median (1.2% per $\mu g/m^3$)."

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Schwartz, et al (2008, 156921) **Period of Study:** 1979-1988 **Location:** Six U.S. metropolitan areas: Boston, Massachusetts Knoxville, Tennessee St. Louis, Missouri Steubenville, Ohio Madison, Wisconsin and Topeka, Kansas	**Outcome:** Mortality **Study Design:** Poisson regression with GAM **Statistical Analyses:** Weighted linear regression **Season:** all **Dose-response Investigated?** No **Statistical Package:** S-plus	**Pollutant:** $PM_{2.5}$ **Averaging Time:** daily **Mean (SD):** Boston-16.5 Knoxville-21.1 St. Louis-19.2 Steubenville-30.5 Madison-11.3 Topeka-12.2 SD not reported **Range (Min, Max):** (0,35) **Monitoring Stations:** 6	**PM Increment:** 10 $\mu g/m^3$ The difference between mean $PM_{2.5}$ concentrations of 10 $\mu g/m^3$ and 20 $\mu g/m^3$ is associated with about a 1.5% increase in deaths.
Reference: (Schwartz et al., 2008, 156963) **Period of Study:** 1979-1998 **Location:** Watertown, MA Kingston and Harriman, TN St Louis, MO Steubenville, OH Portage, Wyocena Pardeeville WI Topeka, KS	**Outcome:** Mortality: Non-accidental (<800) **Study Design:** Cross-sectional **Statistical Analyses:** Cox proportional hazards regression penalized splines Bayesian Model Averaging **Age Groups:** >18	**Pollutant:** $PM_{2.5}$ **Averaging Time:** Annual avg **Mean (SD):** 17.5 (6.8) **Range (Min, Max):** (8, 40)	**Increment:** 10 $\mu g/m^3$ **Relative Risk (Lower CI, Upper CI)** **Estimated from Figure 4:** All Cause Mortality - Year before Death 0: 1.10 (1.00, 1.21) 1: 1.03 (0.98, 1.08) 2: 1.01 (1.00, 1.02) 3: 1.00 (0.99, 1.01) 4: 1.00 (0.99, 1.01) 5: 1.00 Lung Cancer Mortality - Year Before Death **Estimated from Figure 5** 0: 1.18 (1.00, 1.48) 1: 1.12 (0.98, 1.33) 2: 1.08 (0.92, 1.22) 3: 1.02 (1.01, 1.03) 4: 1.01 (1.00, 1.02) 5: 1.01 RR per 10 $\mu g/m^3$ increase of $PM_{2.5}$ exposure Level Of Increase **Estimated from Figure 3** 10 $\mu g/m^3$: 1.15 20 $\mu g/m^3$: 1.29 30 $\mu g/m^3$: 1.46 40 $\mu g/m^3$: 1.64

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Tainio et al. (2005, 087444) **Period of Study:** 1997-Present **Location:** Helsinki, Finland	**Outcome (ICD10):** Mortality: Cardiopulmonary (I11-I70 and J15-J47) Lung Cancer (C34) Other causes **Study Design:** Time-series simulation **Statistical Analyses:** Monte Carlo Simulation **Age Groups:** All ages	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-h avg **Mean (SD):** 10.7 **Range (Min, Max):** NR	**Estimated Deaths Per Year (Min CI, Max CI) Associated with Primary $PM_{2.5}$ Emissions from buses in Helsinki in 2020 for different bus strategies** Cardiopulmonary Mortality Current Fleet: 15.9 (0, 46.6) Modern Diesel: 7.9 (0, 23.0) Diesel with particle trap: 3.9 (0, 12) Natural gas bus: 2.3 (0, 6.8) Lung Cancer Mortality Current Fleet: 2.2 (0, 6.1) Modern Diesel: 1.1 (0, 3.0) Diesel with particle trap: 0.6 (0, 1.6) Natural gas bus: 0.3 (0, 0.9) Total Mortality Current Fleet: 18.1 (0, 55.0) Modern Diesel: 9.0 (0, 27.0) Diesel with particle trap: 4.4 (0, 14.1) Natural Gas Bus: 2.6 (0, 8.0)
Reference: Villeneuve et al. (2002, 042576) **Period of Study:** 1974-1991 **Location:** Six US Cities: Steubenville, OH, St. Louis, MO, Portage, WI, Topeka, KS, Watertown, MA, Kingston/ Harriman, TN	**Outcome (ICD10):** Mortality: Non-accidental (< 800) **Study Design:** Prospective Cohort **Statistical Analyses:** Poisson, EPICURE **Age Groups:** All ages < 60 ≥ 60	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-h avg **Mean (SD):** Portage: 10.9 (7.2) Topeka: 12.1 (7.1) Harriman: 20.7 (9.4) Watertown: 14.9 (8.4) St. Louis: 18.7 (10.6) Steubenville: 28.6 (21.0) Overall: 18.6 **Range (Min, Max):** NR	**Increment:** 18.6 $\mu g/m^3$ **Relative Risk (Min CI, Max CI)** RR of all cause mortality for exposure of $PM_{2.5}$ by age group Exposure to $PM_{2.5}$ remained fixed over entire study period < 60: 1.89 (1.32, 2.69) > 60: 1.21 (1.02, 1.43) Total: 1.31 (1.12, 1.52) Exposure to $PM_{2.5}$ was defined according to 13 calendar periods* (no smoothing) < 60: 1.52 (1.15, 2.00) > 60: 1.11 (0.95, 1.29) Total: 1.19 (1.04, 1.36) Exposure to $PM_{2.5}$ was defined according to 13 calendar periods* (smoothed) < 60: 1.43 (1.10, 1.85) > 60: 1.09 (0.93, 1.26) Total: 1.16 (1.02, 1.32) Time dependent estimate of $PM_{2.5}$ received during the previous two years < 60: 1.42 (1.09, 1.82) > 60: 1.08 (0.94, 1.25) Total: 1.16 (1.02, 1.31) Time dependent estimate of $PM_{2.5}$ received 3-5 years before current year < 60: 1.35 (1.08, 1.67) > 60: 1.08 (0.95, 1.22) Total: 1.14 (1.02, 1.27)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			Time dependent estimate of $PM_{2.5}$ received >5 years before current year $<$60: 1.34 (1.11, 1.59) $>$60: 1.09 (0.99, 1.20) Total: 1.14 (1.05, 1.23) * The calendar periods used were: 1970-1978, 1979, 1980, 1981, 1982, 1983, 1984, 1985, 1986, 1987, 1988, 1989, and 1990+. RR of all cause mortality and $PM_{2.5}$ exposure by city Portage: 1.16 (0.96, 1.39) Topeka: 1.06 (0.89, 1.27) Harriman Men: 1.04 (0.79, 1.36) Women: 0.96 (0.69, 1.31) All: 1.13 (0.95, 1.35) Watertown Men: 1.20 (0.95, 1.51) Women: 1.06 (0.78, 1.43) All: 1.32 (1.11, 1.51) St. Louis Men: 0.97 (0.76, 1.24) Women: 1.13 (0.86, 1.49) Steubenville Men: 1.39 (1.11, 1.74) Women: 1.22 (0.93, 1.61)
Reference: Willis et al. (2003, 089922) **Period of Study:** 1982-1989 **Location:** US Metropolitan areas in all 50 states	**Outcome:** Mortality: All causes Lung Cancer (162) Cardiopulmonary (401-440, 460-519) **Study Design:** Prospective Cohort **Statistical Analyses:** Cox proportional hazards model **Age Groups:** All ages	**Pollutant:** Sulfates **Averaging Time:** Annual avg **Mean (SD):** 10.6 $\mu g/m^3$ **Range (Min, Max):** 3.6, 23.5 **Copollutant:** CO, NO_2, O_3, SO_2	All Cause, Metropolitan Scale: 1.25 (1.13, 1.37) All Cause, County Scale: 1.50 (1.30, 1.73) CPD, Metropolitan Scale: 1.29 (1.15, 1.46) CPD, County Scale: 1.75 (1.48, 2.08)
Reference: Zanobetti and Schwartz (2009, 188462) **Period of Study:** 1999-2005 **Location:** 112 US Cities	**Outcome:** Mortality, all causes, excluding ICD codes S00-U99 **Study Design:** Time-series **Covariates:** Region, season **Statistical Analysis:** Poisson regression **Age Groups:** All	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24h **Mean (SD) Unit:** $\mu g/m^3$ Birmingham AL - 16.5 Phoenix AZ - 11.4 LittleRock AR - 14.3 Fresno CA - 19.4 Bakersfield CA - 21.7 Los Angeles CA - 19.9 Anaheim CA - 16.3 Rubidoux CA - 24.9 Sacramento CA - 13.0 El Cajon CA - 13.5	**Increment:** 10$\mu g/m^3$ **Percent Increase (95% CI) in mortality by increment of $PM_{2.5}$, combined by season** All Cause Mortality Overall: 0.98 (0.75-1.22) Winter: 0.56 (0.17-0.94) Spring: 2.57 (1.96-3.19) Summer: 0.25 (-0.13-0.63) Autumn: 0.95 (0.56-1.34) CVD Overall: 0.85 (0.46-1.24) Winter: 0.70 (0.04-1.36) Spring: 2.18 (1.22-3.15)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
		Denver CO - 10.3	Summer: -0.03 (-0.75-0.69)
		Hartford CT - 11.6	Autumn: 0.92 (0.17-1.68)
		New Haven CT - 13.7	MI
		Wilmington DE - 15.1	Overall: 1.18 (0.48-1.89)
		Davie FL - 8.4	Winter: 1.29 (-0.14-2.75)
		Miami FL - 9.4	Spring: 2.12 (0.53-3.74)
		Jacksonville FL - 10.6	Summer: -0.03 (-1.46-1.42)
		Pensacola FL - 12.4	Autumn: 1.24 (0.12-2.36)
		Tampa FL - 11.9	Stroke
		Orlando FL - 10.3	Overall: 1.78 (0.96-2.62)
		Palm beach FL - 7.9	Winter: 1.93 (0.34-3.54)
		Pinellas FL - 10.4	Spring: 2.04 (-0.02-4.13)
		Atlanta GA - 17.6	Summer: 1.64 (0.05-3.26)
		Chicago IL - 15.9	Autumn: 1.69 (0.06-3.35)
		Gary IN - 15.3	Respiratory
		Indianapolis IN - 16.3	Overall: 1.68 (1.04-2.33)
		Cedar Rapids IA - 11.0	Winter: 0.86 (-0.16-1.88)
		Des Moines IA - 10.5	Spring: 4.62 (3.08-6.18)
		Davenport IA - 12.3	Summer: 0.78 (-0.49-2.06)
		Louisville KY - 15.9	Autumn: 1.45 (0.19-2.72)
		Baton Rouge LA - 13.4	**Percent Increase (95% CI) in mortality by increment in $PM_{2.5}$ combined by region**
		Avondale LA - 12.3	
		New Orleans LA - 12.6	All Cause Mortality
		Baltimore MD - 15.6	Humid Subtropical and Maritime: 1.02 (0.65-1.38)
		Springfield MA - 12.3	
		Boston MA - 12.4	Warm Summer Continental: 1.19 (0.73-1.64)
		Worcester MA - 11.3	
		Holland MI - 12.1	Hot Summer Continental: 1.14 (0.55-1.73)
		Grand Rapids MI - 13.6	Dry: 1.18 (-0.70-3.10)
		Detroit MI - 16.2	Dry, Continental: 1.26 (-0.21-2.76)
		Minneapolis MN - 11.1	Mediterranean: 0.50 (0.00-1.01)
		Kansas MO - 12.0	CVD
		St Louis MO - 14.5	Humid Subtropical and Maritime: 0.78 (0.05-1.51)
		Omaha NE - 10.4	
		Elizabeth NJ - 14.7	Warm Summer Continental: 1.43 (0.67-2.19)
		Albuquerque NM - 6.7	Hot Summer Continental: 0.43 (-0.53-1.40)
		New York NY - 14.8	
		Bath NY - 9.6	Dry: 3.11 (-0.02-6.33)
		Durham NC - 14.3	Dry, Continental: 1.67 (-0.75-4.16)
		Winston NC - 14.7	Mediterranean: 0.16 (-0.46-0.79)
		Greensborough NC - 14.2	MI
		Charlotte NC - 15.3	Humid Subtropical and Maritime: 0.97 (-0.29-2.26)
		Raleigh NC - 14.3	Warm Summer Continental: 1.50 (0.05-2.97)
		Middletown OH - 16.4	
		Youngstown OH - 15.6	Hot Summer Continental: 0.64 (-0.96-2.28)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
		Cleveland OH - 16.4	Dry: 4.25 (-2.38-11.33)
		Columbus OH - 16.2	Dry, Continental: 0.60 (-7.42-9.32)
		Cincinnati OH - 17.1	Mediterranean: 1.85 (-0.66-4.41)
		Steubenville OH - 17.0	Stroke
		Toledo OH - 14.9	Humid Subtropical and Maritime: 2.94 (1.59-4.32)
		Dayton OH - 16.2	Warm Summer Continental: 1.85 (0.04-3.69)
		Akron OH - 16.0	Hot Summer Continental: 0.77 (-1.77-3.38)
		Warren OH - 15.3	Dry: 1.82 (-6.98-11.45)
		Oklahoma OK - 9.9	Dry, Continental: 2.49 (-2.32-7.53)
		Tulsa OK - 11.1	Mediterranean: 0.95 (-0.66-2.59)
		Bend OR - 7.8	Respiratory
		Medford OR - 9.9	Humid Subtropical and Maritime: 0.91 (-0.25-2.08)
		Klamath OR - 10.6	Warm Summer Continental: 2.12 (0.89-3.36)
		Eugene OR - 8.0	Hot Summer Continental: 3.36 (1.95-4.79)
		Portland OR - 8.8	Dry: 5.81 (-0.04-12.00)
		Gettysburg PA - 13.4	Dry, Continental: -0.31 (-5.89-5.61)
		Pittsburgh PA - 15.7	Mediterranean: 1.06 (-0.36-2.50)
		State College PA - 13.2	
		Carlisle PA - 15.1	
		Harrisburg PA - 15.5	
		Erie PA - 13.1	
		Scranton PA - 11.8	
		Allentown PA - 14.2	
		Wilkes Barre PA - 12.8	
		Mercer PA - 14.1	
		Easton PA - 14.0	
		Philadelphia PA - 14.5	
		Washington PA - 14.7	
		Providence RI - 11.5	
		Charleston SC - 12.1	
		Taylors SC - 15.3	
		Columbia SC - 14.0	
		Spartanburg SC - 14.2	
		Nashville TN - 14.0	
		Knoxville TN - 16.0	
		Memphis TN - 13.5	
		San Antonio TX - 9.4	
		Dallas TX - 12.9	
		El Paso TX - 9.2	
		Houston TX - 12.9	
		Port Arthur TX - 11.5	
		Ft Worth TX - 12.2	
		Austin TX - 10.4	
		Salt Lake UT - 11.5	
		Provo UT - 9.5	

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
		WDC VA - 15.2	
		Annandale VA - 14.0	
		Dumbarton VA - 13.6	
		Chesapeake VA - 12.7	
		Norfolk VA - 12.7	
		Richmond VA - 14.3	
		Seattle WA - 10.1	
		Tacoma WA - 11.2	
		Spokane WA - 9.1	
		Dodge WI - 11.1	
		Milwaukee WI - 13.2	
		Waukesha WI - 13.2	
		Range (Min, Max): NR	
		Copollutant (correlation): NR	
Reference: Zeger et al. (2007, 157176)	**Outcome:** Mortality	**Pollutant:** $PM_{2.5}$	**Increment:** 10 $\mu g/m^3$
Period of Study: 2000-2002	**Study Design:** Retrospective Cohort (MCAPS)	**Averaging Time:** 3 year avg	65+: 1.076 (1.044, 1.108)
Location: 250 largest US counties	**Statistical Analyses:** log-linear regression models (GAM)		Eastern US: 1.125 (1.091, 1.159)
	Covariates: age, gender, race, county-level SES, education and COPD SMR		Central US: 1.196 (1.115, 1.277)
	Age Groups: 65+		Western US: 1.029 (0.994, 1.064)
	65-74, 75-84, 85+		65-74: 1.156 (1.117, 1.196)
			75-84: 1.081 (1.042, 1.121)
			85+: 0.995 (0.956, 1.035)
Reference: Zeger et al. (2008, 191951)	**Outcome:** Mortality	**Pollutant:** $PM_{2.5}$	**Increment:** 10 $\mu g/m^3$
Period of Study: 2000-2005	**Study Design:** Retrospective Cohort	**Averaging Time:** Annual	**Relative Risk (Min CI, Max CI)**
Location: 4568 zip codes in urban areas	**Statistical Analysis:** Log-linear regression model	**Median (SD) Unit:**	**Lag**
	Age Groups: ≥65	Eastern: 14.0 $\mu g/m^3$	Risk estimate for increase in mortality per increase in $PM_{2.5}$, all ages
		Central: 10.7 $\mu g/m^3$	Eastern Region
		Western: 13.1 $\mu g/m^3$	Age: 1.155 (1.130-1.180)
		All: 13.2 $\mu g/m^3$	Age + SES: 1.105 (1.084-1.125)
		Range (IQR):	Age + SES + COPD: 1.068 (1.049-1.087)
		Eastern: 12.3-15.3	Central Region
		Central: 9.8-12.2	Age: 1.178 (1.133-1.222)
		Western: 10.4-18.5	Age + SES: 1.089 (1.052-1.125)
		All: 11.1-14.9	Age + SES + COPD: 1.132 (1.095-1.169)
		Copollutant (correlation): NR	Western Region
			Age: 1.003 (0.981-1.025)
			Age + SES: 0.997 (0.978-1.016)
			Age + SES + COPD: 0.989 (0.970-1.008)
			Risk estimate for increase in mortality per increase in $PM_{2.5}$, ages 65-74
			Eastern Region
			Age: 31.1 (26.8-35.5)
			Age + SES: 17.3 (14.6-20.0)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			Age + SES + COPD: 11.4 (8.8-14.1)
			Central Region
			Age: 39.0 (29.7-48.2)
			Age + SES: 16.5 (10.9-22.1)
			Age + SES + COPD: 20.4 (15.0-25.8)
			Western Region
			Age: 6.0 (2.3-9.6)
			Age + SES: -2.1 (-5.0-0.8)
			Age + SES + COPD: -1.5 (-4.2-1.1)
			Risk estimate for increase in mortality per increase in $PM_{2.5}$, ages 75-84
			Eastern Region
			Age: 17.6 (14.9-20.4)
			Age + SES: 12.4 (10.1-14.6)
			Age + SES + COPD: 8.9 (6.8-11.0)
			Central Region
			Age: 17.5 (12.7-22.2)
			Age + SES: 8.8 (4.6-13.0)
			Age + SES + COPD: 12.0 (7.6-16.4)
			Western Region
			Age: 0.4 (-2.0-2.7)
			Age + SES: 0.3 (-1.8-2.5)
			Age + SES + COPD: -0.2 (-2.2-1.9)
			Risk estimate for increase in mortality per increase in $PM_{2.5}$, aged ≥85
			Eastern Region
			Age: -1.4 (-3.5-0.8)
			Age + SES: 1.4 (-0.7-3.5)
			Age + SES + COPD: 1.7 (-0.3-3.7)
			Central Region
			Age: -2.1 (-5.9-1.6)
			Age + SES: -0.7 (-4.2-2.8)
			Age + SES + COPD: -0.3 (-4.0-3.3)
			Western Region
			Age: -5.2 (-7.2-3.2)
			Age + SES: 0.9 (-0.8-2.7)
			Age + SES + COPD: -0.5 (-2.5-1.5)

[1]All units expressed in $\mu g/m^3$ unless otherwise specified.

Table E-34. Long-term exposure - central nervous system outcomes - PM.

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Author: Calderón-Garcidueñas et al. (2008, 192369) **Period of Study:** NR **Location:** Mexico City (polluted city) and	**Outcome (ICD9 and ICD10):** COX2 (cyclooxygenase), IL-1β, CD14 in lungs, OB (olfactory bulb), frontal cortex, hippocampus, substantia nigrae, periaqueductal gray and vagus nerves	**PM Size:** No measure of PM used Mexico City as the "polluted city" and Tlaxcala and Veracruz as the "control cities"	**PM Increment:** NA **Effect Estimate [Lower CI, Upper CI]:** RT-PCR sample results from Control and Mexico City (MC) lung, CNS, PNS

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Tlaxcala and Veracruz (control cities), Mexico	**Age Groups Analyzed:** Subjects 2-45 yrs of age mean=25.1 ± 1.5 yrs **Study Design:** Cross-sectional **N:** 47 deceased subjects with complete autopsies and neuropathological examinations (each subject had to be considered clinically healthy and cognitively and neurologically intact prior to death) (primarily cause of death: accidents resulting in immediate death) **Statistical Analyses:** NR likely used T-tests in addition, stated using "parametric procedure that considers the differences among variances of the variables of interest" **Covariates:** Age, gender, place of birth, place of residency, occupation, smoking habits, clinical histories, cause of death, and time between death and autopsy **Season:** NR **Dose-response Investigated? (Yes/No):** No **Statistical package:** Stata	**Averaging Time:** NA **Mean (SD):** NA **Percentiles:** NA **Range (Min, Max):** NA **Unit (i.e. $\mu g/m^3$):** NA **Number of Monitoring Stations:** NA **Co-pollutant (correlation):** NA	(peripheral nervous system) tissues and p-value for the difference between the means concentrations are normalized to the amount of GAPDH cDNA COX2 (cyclooxygenase-2) lung Controls: $15.9 \pm 6.7 \times 10^6$ MC residents: $42.3 \pm 7.4 \times 10^6$ p-value: 0.015 IL-1β lung Controls: $3.08 \pm 1.87 \times 10^6$ MC residents: $4.51 \pm 2.6 \times 10^6$ p-value: 0.60 COX2 OB (olfactory bulb) Controls: $12.9 \pm 3.0 \times 10^5$ MC residents: $38.7 \pm 5.5 \times 10^5$ p-value: 0.0002 IL-1β OB Controls: $3.4 \pm 0.8 \times 10^4$ MC residents: $7.7 \pm 1.0 \times 10^4$ p-value: 0.003 CD14 OB Controls: 0.01 ± 0.001 MC residents: 0.04 ± 0.01 p-value: 0.04 COX2 frontal Controls: $2.6 \pm 0.4 \times 10^5$ MC residents: $5.0 \pm 0.7 \times 10^5$ p-value: 0.008 IL-1β frontal Controls: $0.6 \pm 0.2 \times 10^4$ MC residents: $6.2 \pm 1.3 \times 10^4$ p-value: 0.0002 COX2 hippocampus Controls: $1.9 \pm 0.5 \times 10^5$ MC residents: $1.6 \pm 8.7 \times 10^5$ p-value: 0.1 IL-1β hippocampus Controls: $1.8 \pm 0.2 \times 10^4$ MC residents: $3.0 \pm 0.5 \times 10^4$ p-value: 0.06 COX2 substantia nigrae Controls: 0.16 ± 0.06 MC residents: 0.97 ± 0.2 p-value: 0.03 IL-1β substanita nigrae

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			Controls: 0.01 ± 0.005
			MC residents: 0.09 ± 0.03
			p-value: 0.06
			CD14 substantia nigrae
			Controls: 0.02 ± 0.005
			MC residents: 0.03 ± 0.007
			p-value: 0.7
			COX2 periaqueductal gray
			Controls: 0.10 ± 0.03
			MC residents: 0.45 ± 0.12
			p-value: 0.12
			IL-1β periaqueductal gray
			Controls: 0.009 ± 0.003
			MC residents: 0.07 ± 0.02
			p-value: 0.09
			COX2 left vagus
			Controls: 0.65 ± 0.18
			MC residents: 2.68 ± 0.82
			p-value: 0.03
			COX2 right vagus
			Controls: 0.43 ± 0.09
			MC residents: 3.68 ± 0.8
			p-value: 0.0002
			IL-1β left vagus
			Controls: 0.1 ± 0.03
			MC residents: 1.3 ± 0.73
			p-value: 0.06
			IL-1 β right vagus
			Controls: 0.15 ± 0.09
			MC residents: 0.87 ± 0.53
			p-value: 0.66
			CD14 left vagus
			Controls: 0.07 ± 0.01
			MC residents: 0.79 ± 0.41
			p-value: 0.01
			CD14 right vagus
			Controls: 0.05 ± 0.01
			MC residents: 0.31 ± 0.1
			p-value: 0.02
			Distribution of subjects with expression of Aβ42 as a function of age and residency
			Groups: No (%) with Aβ42 expression
			Controls < 25yr APOE 3/3 (n = 6): 0 (0)
			Controls > 25yr APOE 3/3 (n = 3): 0 (0)
			MC E2 or E3 < 25yr (n = 17): 10 (58.82)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			MC E2 or E3 > 25yr (n=10): 8 (80)
			MC E4 (n=8): 8 (100)
			Controls E4 (n=3): 2 (66)
			Distribution of subjects with expression of α-synuclein as a function of age and residency
			Groups: No (%) with α-synuclein expression
			Controls < 25yr APOE 3/3 (n=6): 0 (0)
			Controls > 25yr APOE 3/3 (n=3): 0 (0)
			MC E2 or E3 < 25yr (n=17): 4 (23.5)
			MC E2 or E3 > 25yr (n=10): 3 (30)
			MC E4 (n=8): 2 (25)
			Controls E4 (n=3): 0 (0)
Reference: Chen and Schwartz (2009, 179945)	**Outcome:** change in central nervous system function	**Pollutant:** PM_{10}	**Increment:** 10 $\mu g/m^3$
Period of Study: 1989-1991	**Study Design:** Panel	**Averaging Time:** 1year	**Regression Coefficient β (95% CI)**
Location: US	**Covariates:** age, sex, race/ethnicity, individual socioeconomic position, lifestyle factors, household and neighborhood characteristics, conventional CVD risk factors	**Mean (SD) Unit:** 37.2 ± 12.8 $\mu g/m^3$	Crude
		Copollutant: O_3	SRTT: 2.14 (-0.08-4.36)
			SDST: 0.08 (0.04-0.13)
			SDLT Trials: 0.22 (0.13-0.31)
			SDLT Total: 0.44 (0.23-0.65)
	Statistical Analysis: Pearson Chi-square tests and t-tests, as appropriate		Model 1: adjusted for age, sex, race/ethnicity
	Statistical Package: STATA		SRTT: 2.03 (-0.15-4.20)
	Age Groups: 20-59 years		SDST: 0.10 (0.05-0.15)
			SDLT Trials: 0.23 (0.14-0.32)
			SDLT Total: 0.48 (0.27-0.68)
			Model 2: Model 1 + socioeconomic factors
			SRTT: -0.11 (-2.38-2.16)
			SDST: 0.01 (-0.04-0.06)
			SDLT Trials: 0.01 (-0.08-0.10)
			SDLT Total: -0.07 (-0.27-0.13)
			Model 3: Model 2 + lifestyle factors
			SRTT: -0.36 (-2.58-1.85)
			SDST: 0.00 (-0.04-0.05)
			SDLT Trials: 0.09 (0.00-0.17)
			SDLT Total: 0.12 (-0.07-0.31)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Author: Suglia et al. (2008, 157027) **Period of Study:** 1986-2001 **Location:** Boston, Massachusetts	**Outcome (ICD9 and ICD10):** Cognition: Kaufman Brief Intelligence Test, K-BIT (vocabulary and matrices subscales and composite IQ score) Wide Range Assessment of Memory and Learning, WRAML (psychometric instrument with subscales on verbal memory, visual memory, learning, and overall general index scale) All cognition scores have a mean of 100 and SD = 15. **Age Groups Analyzed:** Cognitive tests administered when children were 8-11 yrs of age **Study Design:** Cross-sectional **N:** 202 children **Statistical Analyses:** Linear regression **Covariates:** Child's age at cognitive assessment, gender, primary language spoken at home, and maternal education (model 1 "demographic factors") sensitivity analyses performed with further adjustment for in-utero and postnatal secondhand tobacco smoke exposure (via questionnaire during follow-ups and urinary cotinine levels) (model 2) birth weight (model 3) and blood lead level (model 4) **Season:** Separate land-use regression models were fit for the warm (May-Oct) and cold (Nov-Apr) seasons used average of two seasons as measure of average lifetime BC exposure **Dose-response Investigated? (Yes/No):** No **Statistical package:** SAS (v9.0)	**PM Size:** Black carbon (BC) **Averaging Time:** Lifetime exposure Estimated 24hr measures of traffic using a spatiotemporal land-use regression model using data from > 80 locations in Greater Boston (6021 pollution measurements from 2127 unique exposure days) Predictors in the land-use regression analysis were the BC level at a central station (to capture average concentrations on that day), meteorological conditions, weekday/weekend, and measure of traffic activity (GIS-based measures of cumulative traffic density within 100m, population density, distance to nearest major roadway, % urbanization) Used the average of the cold and warm seasons as the measure of average lifetime BC exposure **Mean (SD):** 0.56 (0.13) μg/m^3 **Percentiles:** NR **Range (Min, Max):** NR **Unit (i.e. μg/m^3):** **Number of Monitoring Stations:** > 80 locations **Co-pollutant (correlation):** NA	**PM Increment:** 0.4 μg/m^3 **Effect Estimate [Lower CI, Upper CI]:** Change in subscale score (95%CI) per IQR (0.4 μg/m^3) increase in log BC level K-BIT Vocabulary: Adj for demographic factors: -2.0 (-5.3, 1.3) Adj for above factors + secondhand smoke: -2.0 (-5.3, 1.4) Adj for above factors + birth weight: -2.0 (-5.4, 1.3) Adj for above factors + blood lead level: -2.2 (-5.5, 1.1) Matrices: Adj for demographic factors: -4.2 (-7.7, -0.7) Adj for above factors + secondhand smoke: -4.0 (-7.5, -0.4) Adj for above factors + birth weight: -4.0 (-7.6, -0.5) Adj for above factors + blood lead level: -4.0 (-7.6, -0.5) Composite: Adj for demographic factors: -3.4 (-6.6, -0.3) Adj for above factors + secondhand smoke: -3.3 (-6.4, -0.1) Adj for above factors + birth weight: -3.3 (-6.5, -0.2) Adj for above factors + blood lead level: -3.4 (-6.6, -0.3) WRAML Verbal: Adj for demographic factors: -1.1 (-4.6, 2.3) Adj for above factors + secondhand smoke: -1.2 (-4.7, 2.3) Adj for above factors + birth weight: -1.3 (-4.7, 2.2) Adj for above factors + blood lead level: -1.3 (-4.8, 2.2) Visual: Adj for demographic factors: -5.2 (-8.6, -1.7) Adj for above factors + secondhand smoke: -5.3 (-8.8, -1.8) Adj for above factors + birth weight: -5.3 (-8.8, -1.8) Adj for above factors + blood lead level: -5.4 (-8.9, -1.9) Learning: Adj for demographic factors: -2.7 (-6.5, 1.1) Adj for above factors + secondhand smoke: -2.6 (-6.5, 1.2) Adj for above factors + birth weight: -2.6 (-6.5, 1.3) Adj for above factors + blood lead level: -2.8 (-6.6, 1.1) General:

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)

[1]All units expressed in $\mu g/m^3$ unless otherwise specified.

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Annex F. Source Apportionment Studies

Table F-1. Epidemiologic studies of ambient PM sources, factors, or constituents.

Reference: Andersen et al. (2007, 093201) **Location:** 1 monitor in Copenhagen, Denmark/ 6 years, but apportionment done for 1.5 year only (2002-2003) **Particle Size:** PM_{10}	**Subjects:** NR **Exposure:** NR	**n:** NR	**Number of constituents considered for grouping:** 31	**Grouping method:** PCA + PMF/CMB hybrid (COPREM) **# of groups:** 12, but only 6 used in relating to health effects, and CO, NO_2	**Groups/Factors/ Sources:** Road, Vehicle, Salt, Biomass, Oil, Coal, Rock, Lime, $NaNO_3$, NH_4NO_3, $(NH_4)_2SO_3$, $(NH_4SO_4$	**PM variables used:** Mass contribution of sources
	Results: Single pollutant models: Biomass, secondary compounds, oil, and crustal significantly associated with CVD HA (4 day moving ave). Biomass and secondary components significantly associated with respiratory HA (5 day moving average). No significant effects for asthma HA in children (6 day moving average). **Two pollutant models:** Crustal effect for CVD admissions remained robust. Biomass effect for respiratory admissions was highest. Effect of vehicle source remained robust for asthma admissions in children in presence of other PM_{10} sources.					
Reference: Bell et al. (Bell et al., 2009, 191007) **Location:** $PM_{2.5}$: 2000-2005 (6 years)/106 US counties/EPA composition data PM_{10}: 1987-2000/100 counties/EPA composition data **Particle Size:** PM_{10}, $PM_{2.5}$	**Subjects:** NR **Exposure:** NR	**n:** NR	**Number of constituents considered for grouping:** 16 elements + NO_3, SO_4, EC, OC	**Grouping method:** NR **# of groups:** NR	**Groups/Factors/ Sources:** NR	**PM variables used:** Every component (16 elements + NO_3, SO_4,EC, OC)
	Results: Mortality: Ni significantly increased PM_{10} mortality risks. However, effect of Ni was not significant when NY City was removed, in a sensitivity analysis conducted by selectively removing cities from the overall estimate. **Hospital Admissions:** CVD and respiratory HAs higher in counties with higher EC, Ni, and V $PM_{2.5}$. In CVD association between $PM_{2.5}$, RR and V robust to inclusion of EC or V, and V robust to inclusion of EC.					
Reference: Cakmak et al. (2009, 191995) **Location:** 1 monitor in Santiago, Chile **Particle Size:** $PM_{2.5}$	**Subjects:** NR **Exposure:** 1998-2009 (8.3 years)	**n:** NR	**Number of constituents considered for grouping:** 16 elements + CO, NO_2,SO_2, EC, OC	**Grouping method:** PCA **# of groups:** 4	**Groups/Factors/ Sources:** Vehicle (CO, NO_2, EC, OC), Soil (Al, Ca, Fe, Si), Combustion (Cr, Cu, Fe, Mn, Zn), Factor 4 (Br, Cl, Pb)	**PM variables used:** individual components, then groupings
	Results: Individual components: EC, OC only stat. sign. risk estimates for total, cardiac, and respiratory mortality for 1 day lag after adjustment for other elements. **Groupings.:** Lag 1. Vehicle factor: Increased total mortality, cardiac mortality, and respiratory mortality. Soil factor: increased cardiac mortality and respiratory mortality (but smaller than vehicle factor RRs). Combustion factor: greatest RR for respiratory mortality, but significant for total and cardiac mortality. Factor 4: increased total, cardiac, and respiratory mortality. Point estimates for Factor 1 significantly different from Factors 3 and 4. Elderly had higher risk estimates for combustion and soil sources. No significant effect modification by gender or season.					

Note: Hyperlinks to the reference citations throughout this document will take you to the NCEA HERO database (Health and Environmental Research Online) at http://epa.gov/hero. HERO is a database of scientific literature used by U.S. EPA in the process of developing science assessments such as the Integrated Science Assessments (ISA) and the Integrated Risk Information System (IRIS).

Reference: Franklin et al. (2008, 155779) **Location:** STN/25 communities/2000-2005 (6 years) **Particle Size:** $PM_{2.5}$	**Subjects:** NR **Exposure:** NR	**n:** NR	**Number of constituents considered for grouping:** 15 elements + EC, OC, NO_3	**Grouping method:** NR **# of groups:** NR	**Groups/Factors/ Sources:** NR	**PM variables used:** Every component
	Results: The $PM_{2.5}$-mortality association was significantly modified by Al, As, Sulfate, Ni, and Si. When including a combination of species proportions and using backwards elimination Al, sulfate, and Ni remained significant. Al and Ni explained most of the residual heterogeneity.					
Reference: Gent et al. (2009, 180399) **Location:** 2 monitors in New Haven, CT/ 3.5 years **Particle Size:** $PM_{2.5}$	**Subjects:** Children with physician diagnosed asthma and symptoms or medication use in previous 12 months, and resided within 30km of New Haven county monitor **Exposure:** NR	**n:** 149 children	**Number of constituents considered for grouping:** 17 elements + EC	**Grouping method:** PCA **# of groups:** 6	**Groups/Factors/ Sources:** Vehicle (EC, Zn, Pb, Cu, Se), road dust (Si, Fe, Al, Ca, Ba, Ti), sulfur (S, P), biomass burning, (K) oil (V, Ni), sea salt (Na, Cl) In addition, effects of NO_2, CO, SO_2, and O_3 were included in the health outcomes model	**PM variables used:** Groupings and individual elements
	Results: Overall: Trace elements originating from motor vehicle, road dust, biomass burning, and oil sources associated with symptoms and/or medication use. No associations with S or sea salt. **Specific Results:** $PM_{2.5}$ mass from motor vehicle or road dust associated with increased odds of respiratory symptoms or inhaler use. Reduced odds of wheeze or inhaler use with same day S. Significant reductions odds of wheeze with biomass burning. **Co-pollutant:** Positive effects of motor vehicles and road dust on wheeze were robust to the inclusion of gaseous copollutants. However, NO_2 increases association with wheeze.					
Reference: Ito et al. (2006, 188554) **Location:** Washington, DC **Particle Size:** $PM_{2.5}$	**Subjects:** NR **Exposure:** NR	**n:** NR	**Number of constituents considered for grouping:** NR	**Grouping method:** Comparison of: PMF; (absolute) PCA; UNMIX **# of groups:** 6-10 **Groups/ Factors/ Sources:** Different research groups gave different names to sources	**Sources for which association with health was analyzed:** Soil, traffic, Secondary SO_4, NO_3 (Wash DC only), residual oil (Wash DC only), Wood smoke/ biomass combustion, Sea salt, incinerator (Wash DC only), primary coal (Wash DC only), Cu smelter (Phoenix only)	**PM variables used:** Mass contribution of sources
	Results: Overall, $PM_{2.5}$ effects observed at lag 3. Lag structure of association varied across source types, but consistent across investigators for total (non-accidental mortality): soil factor - mostly positive at various lags (not significant); secondary sulfate - strongest association at lag 3; nitrate - mostly negative except at lag 3; residual oil - strongest association at lag 2 (not significant); wood-burning - increasing association as lag increases (not significant); incinerator - significant negative associations at lag 0; primary coal - significant association at lag 3.					
Reference: Laden et al. (2000, 012102) **Location:** Monitors in 6 Eastern US cities (Harvard Six Cities	**Subjects:** NR **Exposure:** NR	**n:** NR	**Number of constituents considered for grouping:** 15 elements	**Grouping method:** PCA **# of groups:** 8	**Groups/ Factors/ Sources:** Soil/Crustal (PM fine), Mobile vehicle exhaust (PM fine), Coal (PM fine), Fuel oil; Metals, Salt' Manganese, Residual	**PM variables used:** Tracers: Si, V, Cl, Pb, Se

Study) **Particle Size:** NR	**Results:** Lag 0-1 average for all results. Over all 6 cities, mobile source factor (using Pb as tracer) had greatest association with daily mortality (3.4%) with 10 $\mu g/m^3$ increase. The greatest effects for mortality due to mobile sources were observed in Madison (Portage), Knoxville (Kingston-Harriman), and St. Louis, although the Madison results were not statistically significant. The coal source factor was only significant in Boston (Watertown) - 2.8% increase in mortality and the overall percent increase was also significant (1.1%). Deaths from pneumonia attributable to coal combustion sources was 7.9% (CI 3.1-12.7%) and statistically significant. The crustal factor was not associated with mortality in any city, although this factor was not a significant predictor in the regression model for Boston (Watertown) due to its low contribution to $PM_{2.5}$ mass. For specific elements included simultaneously, S, Pb, and Ni were significantly associated with overall mortality (3.0, 1.6, 1.5%, respectively). Boston had the greatest percent increase in mortality for S (7.9%), Knoxville for Pb (15.0%), and Steubenville for Ni (8.2%), although the CIs are all quite large. **Reanalysis results** (Schwartz, 2003): Effects changed slightly. New percent increases in mortality for combined cities are 3.5 and 0.79 for traffic and coal, respectively. The coal factor in Boston decreased to 2.1% increased mortality. A residual oil factor in Boston and Steubenville resulted in at 22.9% increase in daily deaths (but was not significant in the original analysis).					
Reference: Lanki et al. (2006, 088412) **Location:** Monitors in Helsinki, Finland, Amsterdam,The Netherlands and Erfurt, Germany **Particle Size:** UF/$PM_{2.5}$	**Subjects:** NR **Exposure:** NR	**n:** NR	**Number of constituents considered for grouping:** 13 elements	**Grouping method:** Absolute PCA **# of groups:** 5	**Groups/ Factors/ Sources:** Crustal; long range transported; oil combustion; soil; traffic	**PM variables used:** Tracers: Si (crustal); S (long-range transport); Ni (oil combustion); Cl (salt); ABS (local traffic).
	Results: Highest observed effects were for crustal sources and salt at lag 3 (when analyzing sources), but not consistent or significant. In multipollutant models only ABS associated with ST-segment depression, but wide CIs. When examining indicator elements of a source, local traffic found to be the most toxic, but when examined per IQR long-range transport and traffic had similar effects.					
	Results: All had significant associations with mortality. Traffic density and EC had the largest effects.					
Reference: Lippmann et al. (2006, 091165) **Location:** U.S. **Particle Size:** PM_{10} for risk estimates, $PM_{2.5}$ for speciation data	**Subjects:** NR **Exposure:** NR	**n:** NR	**Number of constituents considered for grouping:** NR	**Grouping method:** No grouping was performed **# of groups:** NR	**Groups/ Factors/ Sources:** NR	**PM variables used:** Mass contribution of 16 constituents
	Results: The strongest predictions of the variation in PM_{10} risk estimates across the 90 NMMAPs MSAs was for Ni and V. Elevated, but nonsignificant increases were associated with EC, Zn, SO_4^{2-}, Cu, Pb, and OC. Al and Si had the lowest values.					
Reference: Mar et al. (2000, 001760) **Location:** 1 monitor in Phoenix, AZ **Particle Size:** NR	**Subjects:** Elderly only **Exposure:** NR	**n:** NR	**Number of constituents considered for grouping:** 10 elements, OC, EC, CO, NO_2; SO_2	**Grouping method:** Unspecified type of factor analysis **# of groups:** 3 or 5	**Groups/ Factors/ Sources:** Motor exhaust/road dust, soil, vegetative burning, local SO_2, regional SO_4	**PM variables used:** First used individual constituents: S, Zn, Pb, K, OC, EC, TC (AL+Si+Ca+Fe+Ti), then factor scores
	Results: Cardiovascular mortality associated with $PM_{2.5}$ mass on lag 1 and 4 (6 and 4%, respectively). EC and TC associated with CV mortality for lag 1 (RR = 1.05); OC was weakly associated with CV mortality for lags 1 and 3. For total mortality, regional sulfate was positively associated at lag 0, but negatively associated at lag 3. The local SO_2 and the soil factors were negatively associated with total mortality. For CV mortality, secondary sulfate was positively associated at lag 0, motor vehicle at lag 1, and vegetative burning at lag 3. **Reanalysis results** (Mar, 2003): Similar associations were observed.					
Reference: Mar et al. (2006, 086143) **Location:** Phoenix, AZ **Particle Size:** $PM_{2.5}$	**Subjects:** NR **Exposure:** NR	**n:** NR	**Number of constituents considered for grouping:** NR	**Grouping method:** Comparison of: PMF (absolute); PCA; UNMIX **# of groups:** 6-10 **Groups/ Factors/ Sources:** Different labs gave different names to sources (see Hopke et al, table 2)	**Sources for which association with health was analyzed:** Soil, Traffic, secondary SO_4, NO_3, (Wash DC only), residual oil (Wash DC only), woodsmoke/ biomass combustion, sea salt, incinerator (Wash DC only); primary coal (Wash DC only); Cu smelter (Phoenix only)	**PM variables used:** Mass contribution of sources
	Results: Using daily $PM_{2.5}$ data found the following associations with cardiovascular mortality: Secondary sulfate - greatest effect observed for all sources and at lag 0; traffic - associated at lag 1; copper smelter associated at lag 0; sea salt - had the greatest statistical significance and observed at lag 5; biomass/wood burning - less consistent lag structure but greatest association at lag 3; soil - did not show an association or consistent lag structure. For total (non-accidental) mortality associations were weaker and consistently observed for only: copper smelter - lag 0; sea salt - lag 5.					

Reference: Ostro et al. (2007, 091354) **Location:** Monitors in 6 CA counties, some with 2 monitors, for 4 years **Particle Size:** $PM_{2.5}$	**Subjects** NR **Exposure:** NR	**n:** NR	**Number of constituents considered for grouping:** 15 elements, EC, OC; NO_3; SO_4, $PM_{2.5}$ mass	**Grouping method:** No grouping was performed **# of groups:** NA	**Groups/ Factors/ Sources:** NR	**PM variables used:** Mass contribution of every constituent
	Results: Effects were greater during the winter months. In the all year analysis, at 3-day lag associations observed for EC, OC, NO_3 and Zn. During winter months (Oct -March) effects observed for most species for both all-cause and cardiovascular mortality at lag 3 (EC, OC, SO_4, Ca, Fe, K, Mn, Pb, S, Si, Ti, Zn) and (OC, NO_3, SO_4, Fe, Mn, S, V, Zn), respectively.					
Reference: Ostro et al. (2009, 191971) **Location:** Monitors in 6 CA counties, some with 2 monitors/4 years **Particle Size:** $PM_{2.5}$	**Subjects** NR **Exposure:** NR	**n:** NR	**Number of constituents considered for grouping:** 9 elements,EC, OC, $PM_{2.5}$ mass, SO_4, NO_3	**Grouping method:** No grouping was performed **# of groups:** NA	**Groups/ Factors/ Sources:** NR	**PM variables used:** Mass contribution of every constituent
	Results: The following associations were observed with cardiovascular mortality: $PM_{2.5}$ (lag 3); EC (lag 2); NO_3 (lag 3); SO_4 (lag 3); Fe (lag 2); K (lag 2); S (lag 3); Ti (lag 2); Zn (lag 3).					
Reference: Peng et al. (2009, 191998) **Location:** 119 urban communities STN data/2000-2006 **Particle Size:** $PM_{2.5}$	**Subjects:** Medicare enrollees 65 or older **Exposure:** NR	**n:** NR	**Number of constituents considered for grouping:** SO_4, NO_3, Si, EC, OCM, Na, NH_4	**Grouping method:** NR **# of groups:** NR	**Groups/Factors/ Sources:** Only suggested in discussion	**PM variables used:** Tracers
	Results: CVD HA's: EC associated with same-day CVD HA's in single and multi-pollutant models. In single pollutant models associations also observed for sulfate, nitrate, OCM, and ammonium. However, the sulfate, nitrate, OCM, and ammonium associations were reduced in the multi-pollutant models. **Respiratory HA's:** OCM associated with same-day respiratory HA's in single and multi-pollutant models. Some evidence for sulfate associations at one and two-day lag.					
Reference: Penttinen et al. (2006, 087988) **Location:** Helsinki 1996-1997 (7 months) **Particle Size:** $PM_{2.5}$	**Subjects:** Adult asthma subjects, max 2 km from single monitor **Exposure:** NR	**n:** 78	**Number of constituents considered for grouping:** Unknown	**Grouping method:** PCA **# of groups:** 6	**Groups/Factors/ Sources:** Long range (PM mass, S, K, Zn), local combustion-traffic (Cu, Zn, Mn, Fe), soil (Si, Al, Ca, Fe, Mn), oil (V, Ni), salt (Na, Cl), unidentified	**PM variables used:** every component individually, then groupings
	Results: Long range $PM_{2.5}$ associated with decreased mean PEF in the morning at lag 1. Local combustion $PM_{2.5}$ associated with decreased mean PEF in the evening for lag 1. Local combustion $PM_{2.5}$ associated with decreased mean PEF in the afternoon and evening for 5-day mean lag. Negative significant association between long-range $PM_{2.5}$ and asthma symptom prevalence at lag 3. Sea-salt $PM_{2.5}$ negatively associated with bronchodilator use at lag 3 and 5-day mean lag. Sea-salt $PM_{2.5}$ negatively associated with corticosteroid use for 5-day mean lag. Unidentified $PM_{2.5}$ negatively associated with corticosteroid use at lag 1. Most consistent negative responses for local combustion, although not always significant. No consistent or significant associations between 5-day average concentrations of elements and PEF, cough, asthma symptoms, or medication use.					
Reference: Riediker et al. (2004, 091261) **Location:** Inside 9 state police patrol cars **Particle Size:** $PM_{2.5}$	**Subjects:** Healthy male young police officers **Exposure:** 4 consecutive days	**n:** 9	**Number of constituents considered for grouping:** 10 elements; 3 gaseous pollutants; 2 physical variables	**Grouping method:** PCA **# of groups:** 4 when 13+2 constituents included; 3 when only 9 "PM-associated" constituents included	**Groups/ Factors/ Sources:** Soil; automotive steel wear; gasoline combustion; speed-changing traffic	**PM variables used:** Mass contribution or score of sources
	Results: Using two different factor analysis models found most significant effects (MCL, SDNN, PNN50, supraventricular ectopic beats, % neutrophils, % lymphocytes, MCV, von Willebrand Factor, and protein C) were for "speed-change factor" (i.e., Cu, S, aldehydes). Some associations observed for "crustal" and none for "steel wear" and "gasoline."					

Reference: Sarnat et al. (2008, 097972) **Location:** 1monitor in Atlanta. GA for 2 yrs **Particle Size:** $PM_{2.5}$	**Subjects:** NR **Exposure:** NR	**n:** NR	**Number of constituents considered for grouping:** NR	**Grouping method:** Comparison of: PMF, CMB-LGO, "*a priori* decision" **# of groups:** 9,11 (6 of them common between methods)	**Groups/ Factors/ Sources:** gasoline, diesel, wood smoke/ biomass burning, soil, secondary SO_4/ammonium sulfate, seconddary nitrate/ ammonium nitrate, metal processing, railroad, bus and highway, cement kiln, power plants, other OC, ammonium bisulfate	**PM variables used:** Mass contribution or score of sources, and tracers
	Results: Sulfate secondary associated with 1.2 - 2.0% increase in RD visits, significant negative association RD visits and primary emissions from coal-fired power plants. CVD significantly associated with other OC (1.014), biomass (1.033), diesel and gas for CMB-LGO. For PMF and CVD visits: diesel (1.025), gas, wood smoke, metal processing (1.013). Year-long associations: PMF diesel, EC, CMB-LGO gas, Zn and biomass combustion sources (CMB-LGO biomass burning, PMF wood smoke, and K). Diesel and gas sources association with RD in the warm season (1.2-2.1% per IQR).					
Reference: Schreuder et al. (2006, 097959) **Location:** 1monitor in Spokane, WA for 7 years **Particle Size:** $PM_{2.5}$	**Subjects:** NR **Exposure:** NR	**n:** NR	**Number of constituents considered for grouping:** 11 elements, TC, NO_3	**Grouping method:** Comparison of: PMF, UNMIX, Multilinear Engine **# of groups:** 8	**Groups/ Factors/ Sources:** Vegetative burning; As-rich Vehicle; SO_4; NO_3; Soil; Cu-rich; Marine	**PM variables used:** Tracers: TC (vegetative burning); As (As-rich); Zn (vehicle); Si (soil)
	Results: Si, As, and Zn were not associated with any health outcomes; while an IQR increase in TC (vegetative burning) was associated with a 2% increase in respiratory ED visits.					
Reference: Tsai et al. (2000, 006251) **Location:** 3 NJ sites for 2 summers (ATEOS study) **Particle Size:** NR	**Subjects:** NR **Exposure:** NR	**N:** NR	**Number of constituents considered for grouping:** 8 metals, IPM, FPM, SO_4, CX, DCM, ACE, CO	**Grouping method:** Unspecified type of factor analysis **# of groups:** 5	**Groups/ Factors/ Sources:** Oil burning, motor emissions, resuspended dust, secondary aerosol, industrial sources	**PM variables used:** individual constituents, then factor scores, then tracers
	Results: RR associated with 10 $\mu g/m^3$ increases: Newark - 1.03 for industrial and total daily deaths; 1.02 for sulfate and total daily deaths; 1.04 for sulfate and cardiopulmonary deaths. Camden - 1.11 for oil burning sources and total daily deaths; 1.10 industrial and total daily deaths; 1.12 for oil burning sources and cardiopulmonary daily deaths; 1.02 for sulfate and cardiopulmonary daily deaths					
Reference: Yue et al. (2007, 097968) **Location:** 1monitor in German city, 30.000 samples **Particle Size:** UF/$PM_{2.5}$	**Subjects:** Adult males **Exposure:** CAD	**n:** 56, data collected 12 times over 6 month for every subject, but extended period of missing PM data	**Number of constituents considered for grouping:** Apportionment based on particle size distribution.	**Grouping method:** PMF **# of groups:** 5	**Groups/ Factors/ Sources:** Airborne soil, local traffic, local fuel combustion, remote traffic (diesel), secondary aerosols	**PM variables used:** Mass contribution or score of sources
	Results: Overall, repolarization parameters influenced by traffic-related particles; vWF increased in response to traffic-related particles and combustion-generated aerosols. All source factors contributed to increasing CRP levels.					

Table F-2. Human clinical studies of ambient PM sources, factors, or constituents.

Study: Gong et al. (2003, 042106) **Location:** Los Angeles, CA **Particle Size:** $PM_{2.5}$	**Subjects:** Adult 18-45, healthy vs. asthmatic **Exposure:** CAPs, healthy and asthmatic subjects exposed at different times	**N:** 12 healthy, 12 asthmatic	**Constituents considered for grouping:** 7 elements, EC, NO_3, SO_4	**Grouping method:** PCA **# of groups:** 4 (note: OC data was unavailable)	**Groups/ Factors/ Sources:** Crustal (Al Si CA K Fe),S (2 metrics of SO_4 + elemental S), Total Mass + NO_3, EC	**PM variables used:** Total mass, then tracers: SO_4, EC, Fe

	Results: Fe and EC associated with a decrease in ST-segment voltage 2 days post-exposure. EC associated with an increase in ST-segment voltage immediately following exposure. Sulfate content associated with a decrease in systolic BP 4 h post-exposure.					
Study: Gong et al. (2005, 087921) **Location:** Los Angeles, CA **Particle Size:** $PM_{2.5}$	**Subjects:** Elderly, COPD vs. healthy/ CAPs **Exposure:** NO_2 (full factorial)	**N:** 6 healthy, 18 COPD	**Constituents considered for grouping:** 7 elements + EC	**Grouping method:** PCA **# of groups:** 3 (note: OC was unavailable)	**Groups/ Factors/ Sources:** Crustal (Al Si CA K Fe), S (= SO_4),Na	**PM variables used:** Total mass, then tracers:, SO_4, Si, Fe, EC
	Results: Mass concentration of CAPs not observed to significantly affect lung function. However, sulfate content was associated with a decrease lung function (FEV_1 and FVC), which was enhanced by coexposure to NO_2.					
Reference: Huang et al. (2003, 087377) **Location:** Chapel Hill, NC **Particle Size:** $PM_{2.5}$	**Subjects:** Healthy adults **Exposure:** CAPs	**N:** 35 male; 2 female	**Constituents considered for grouping:** 8 elements and SO_4	**Grouping method:** PCA **# of groups: 2**	**Groups/ Factors/ Sources:** Fe/SO_4/Se/V/Zn/Cu	**PM variables used:** Factor scores, then mass contribution of all 9 constituents
	Results: Associations observed between sulfate,Zn, and Se content and increases in BAL neutrophils. Increases in fibrinogen associated with Cu, Zn, and V content.					
Reference: Urch et al. (2004, 055629) **Location:** Toronto, Canada **Particle Size:** $PM_{2.5}$	**Subjects:** Healthy adults 19-50 yrs/CAPs **Exposure:** O_3	**N:** 23	**Constituents considered for grouping:** unknown	**Grouping method:** No grouping was performed **# of groups:** NA	**Groups/ Factors/ Sources:** NR	**PM variables used:** Every constituent in univariate analysis, then OC and SO_4 in multivariate analysis
	Results: CAPs-induced increase in diastolic BP significantly associated with carbon content of the particles.					
Reference: Urch et al. (2004, 055629) **Location:** Toronto, Canada **Particle Size:** $PM_{2.5}$	**Subjects:** Healthy adults/CAPs **Exposure:** O_3	**N:** 24	**Constituents considered for grouping:** 14 elements, EC, OC	**Grouping method:** No grouping was performed **# of groups:** NA	**Groups/ Factors/ Sources:** NR	**PM variables used:** Every constituent in univariate analysis, then OC and SO_4 in multivariate analysis
	Results: Both organic and EC content of CAPs associated with an increase in brachial artery vasoconstriction.					

Table F-3. Toxicological studies of ambient PM sources, factors, or constituents

Reference: Batalha et al. (2002, 088109) **Location:** Boston, MA **Particle Size:** $PM_{2.5}$	**Subjects:** Rats **Exposure:** CAPs (3-day mean CAPs concentration range: 126.1-481.0 $\mu g/m^3$) CAPs (3-day mean CAPs concentration range: 126.1-481.0 $\mu g/m^3$)	**n:** 7-10 rats × 2 levels CAPs × 2 levels SO_2 × 6 runs in different seasons	**Constituents considered for grouping:** 20 elements; OC; EC	**Grouping method:** Previous study in same city (Clarke et al.), and PCA of this experiment's data **# of groups:**4	**Groups/ Factors/ Sources:** V/Ni, S, Al/Si, Br/Pb	**PM variables used:** 4 tracers (Si, SO_4, V, Pb) and EC, OC in univariate step. 4 tracers (Si, SO_4, V, Pb) in multivariate step
	Results: Univariate analyses for first day not significant for L/W ratio. Univariate analyses for second and third day and second+third day mean were similar. Presented second+third day mean regression data. CAPs mass, Si, Pb, SO_4, EC, OC significant for decreased L/W ratio in normal+CB rats exposed to CAPs. Si, SO_4 significant for decreased L/W ratio in normal rats. Si, OC significant for decreased L/W ratio in CB rats. Multivariate analysis using normal+CB rats for Si, SO_4, V, Pb - only Si remained significant with decreased L/W ratio.					
Reference: Becker et al. (2005, 088590) **Location:** Chapel Hill, NC; repeated	**Subjects:** Normal human bronchial epithelial and human AM **Exposure:** ($2\text{-}3X10^5$ cells/mL; 11 or 50 $\mu g/mL$)	**n:** NR	**Constituents considered for grouping:** 12 elements	**Grouping method:** PCA **# of groups:** 2	**Groups/ Factors/ Sources:** Cr/Al/Si/Ti/Fe/Cu ("crustal"), Zn/As/V/Ni/Pb/ Se	**PM variables used:** NR

sampling for 1 year **Particle Size:** PM_{10}	**Results:** Cr/Al/Si/Ti/Fe/Cu associated with IL-8 release in normal human bronchial epithelial cells and IL-6 release in AM. Zn/As/V/Ni/Pb/Se not associated with any endpoints. Stepwise linear regression with individual constituents Fe and Si associated with IL-6 release in AM. Cr associated with IL-8 release in NHBE cells.					
Reference: Clarke et al. (2000, 013252) **Location:** Boston, MA **Particle Size:** $PM_{2.5}$	**Subjects:** Dogs **Exposure:** CAPs (average for all studies, paired: 203.4, crossover: 360.8 $\mu g/m^3$) repeated exposure with several weeks in between	**n:** 10 dogs, 20 paired exposures, 24 crossover	**Constituents considered for grouping:** 19 elements, black C	**Grouping method:** PCA **# of groups:** 4 for exposure in paired runs,6 for exposure in crossover runs	**Groups/ Factors/ Sources:** V/Ni, S, Al/Si, Br/Pb, S, Na/Cl Cr	**PM variables used:** All elements, then factor scores
	Results: No significant differences between baseline, sham, or CAPs group for BAL cell differential percentages. Total BAL protein increased with CAPs compared to sham. No significant hematological effects with CAPs exposure. Mixed linear regression analyses (statistics not provided): Al and Ti (3-day avg. concentrations) associated with dose-dependent decreases in BAL AM and increases in BAL PMN percentages. Sulfate associated with increased WBC. BC, Al, Mn, Si, Zn, Ti, V, Fe, Ni associated with increased blood PMN. Na associated with increased blood lymphocytes. Al, Mn, Si associated with decreased blood lymphocytes. CAPs mass and BC associated with decreased blood eosinophils. CAPs mass associated with decreased platelet count. Regression using results of factor analysis: None for 3-day avg. concentration for BAL parameters. V/Ni for increased AM percentage and Br/Pb for increased PMN percentage for 3rd-day only concentration. V/Ni and Al/Si for increased blood PMN percentage and decreased blood lymphocyte percentage. Al/Si also for increased WBC counts. Na/Cl for increased blood lymphocyte percentage. S for decreased RBC and hemoglobin.					
Reference: Duvall et al. (2008, 097969) **Location:** 5 US cities **Particle Size:** $PM_{2.5}$	**Subjects** Primary human airway epithelial cells (100,000 cells/mL; dose not provided) **Exposure:** NR	**n:** NR	**Constituents considered for grouping:** NR	**Grouping method:** CMB, but not on coarse and ultrafine **# of groups:** 6 or 7	**Groups/ Factors/ Sources:** Mobile, residual, oil, wood, soil, secondary SO_4, secondary NO_3	**PM variables used:** Mass contribution of constituents, then mass contribution of sources
	Results: Linear regression with individual constituents: Sulfate associated with increased IL-8 mRNA expression. Sr associated with increased COX-2 and decreased HO-1 mRNA expressions. K associated with decreased HO-1 mRNA expression. **Linear regression with sources:** Significance levels not provided.					
Reference: Godleski et al. (2002, 156478) **Location:** Boston, MA **Particle Size:** NR	**Subjects:** Rats **Exposure:** CAPs (3-day mean CAPs concentration range: 126.1-481.0 $\mu g/m^3$)	**n:** 7-10 rats × 2 levels CAPs × 2 levels SO_2 × 6 runs in different seasons	**Constituents considered for grouping:** 20 elements, OC, EC	**Grouping method:** Previous study in same city (Clarke et al.), and PCA of this experiment's data **# of groups:** 4	**Groups/ Factors/ Sources:** V/Ni, S, Al/Si/Ca, Br/Pb	**PM variables used:** 4 tracers (I, SO_4,V, Pb) and EC, OC
	Results: Increased percent of PMNs in BALF in CAPs-exposed rats at 24 h. CAPs affected lung tissue mRNA involved in pro-inflammation, immune, and vascular endothelial responses. **Linear regression:** Increased PMN associated with CAPs mass, Br, Pb, SO_4, EC, and OC.					
Reference: Gurgueira et al. (2002, 036535) **Location:** Boston, MA **Particle Size:** $PM_{2.5}$	**Subjects** Rats (Sprague Dawley) **Exposure:** CAPs (avg. mass concentration 600 $\mu g/m^3$); also carbon black and ROFA	**n:** 13 experiments (1 rat/group at each time point)	**Constituents considered for grouping:** 20 elements	**Grouping method:** No grouping was performed **# of groups:** NA	**Groups/ Factors/ Sources:** NR	**PM variables used:** Mass contribution of every constituent
	Results: Increased oxidative stress in heart and lungs following CAPs exposure (and ROFA exposure). **Univariate regression:** Mn, Zn, Fe, Cu, and Ca most significant responses for lung ($r^2 > 0.40$). Al, Si, Ti, Fe, and total mass most significant response for heart ($r2 > 0.49$).					
Reference: Kodavanti et al. (2005, 087946) **Location:** RTP, NC **Particle Size:** $PM_{2.5}$	**Subjects** Rats (SH and WKY) **Exposure:** CAPs (144-2758 $\mu g/m^3$)	**n:** 6 1-day , 1-strain runs, 7 2-day, 2-strain runs, 4-9 rats per run.	**Constituents considered for grouping:** NR	**Grouping method:** No grouping was performed **# of groups:** NA	**Groups/ Factors/ Sources:** NR	**PM variables used:** Mass contribution of every constituent
	Results: No significant correlations between biologic responses and exposure variables (i.e., CAP mass, OC, inorganic C, sulfate, and other major elemental constituents). Al, Cu, Zn correlated with biologic responses when constituents normalized per unit mass of CAP ($\mu g/mg$). Zn correlated with plasma fibrinogen in SH rats ($p = 0.0023$).					

Reference: Lippmann et al. (2005, 087453) **Location:** Rural location upwind from NYC **Particle Size:** $PM_{2.5}$	**Subjects:** Mice (C57 and ApoE) **Exposure:** CAPs (avg. mass concentration 113 $\mu g/m^3$)	**n:** C57: 3-6 mice/group $ApoE^{-/-}$: 9-10 mice/group	**Constituents considered for grouping:** 19 elements + OC, EC, NO_3	**Grouping method:** (Absolute) PCA **# of groups:** 4	**Groups/ Factors/ Sources:** Regional SO_4 (S/Si/OC); Resuspended soil (CA/Fe/Al/Si);RO power plants (V/Ni/Se); Traffic and unknown	**PM variables used:** Mass contribution of sources
	Results: ApoE null mice: Resuspended soil associated with decreased HR during exposure, but increased HR after exposure. Secondary sulfate associated with decreased HR after exposure. Residual oil associated with increased RMSSD and SDNN in afternoon following exposure. Secondary sulfate associated with decreased RMSSD and SDNN in night following exposure. Resuspended soil associated with increased RMSSD at night following exposure. PM mass associated with decreased HR during exposure and decreased RMSSD at night following exposure. **C57 mice**: Motor vehicle/other source category associated with decrease in RMSSD in afternoon following exposure					
Reference: Lippmann et al. (2006, 091165) **Location:** Rural location upwind from NYC **Particle Size:** $PM_{2.5}$	**Subjects:** Mice ($ApoE^{-/-}$) **Exposure:** CAPs (avg. mass concentration 85.6 $\mu g/m^3$)	**n:** 12 $ApoE^{-/-}$ mice (6/group)	**Number of constituents considered for grouping:** NR	**Grouping method:** No grouping was performed **# of groups:** NR	**Groups/ Factors/ Sources:** NR	**PM variables used:** Mass contribution of every constituent in CAPs portion of study, contribution of 16 constituents in epi portion
	Results: Lag for HR elevations on 14 days with wind from NW was same day. Lag for SDNN reduction on 14 days with wind from NW was 0, 1 and 2. **GAM analysis:** B coefficient significant for Ni and HR (but not V, Cr, or Fe). B coefficient significant for Ni and log SDNN (but not V, Cr, or Fe).					
Reference: Maciejczyk and Chen (2005, 087456) **Location:** Rural; upwind from NYC **Particle Size:** $PM_{2.5}$	**Subjects:** NR **Exposure:** CAPs (90000/well; 300 μg/mL)	**n:** 110 samples	**Constituents considered for grouping:** 19 elements + OC, EC, NO_3	**Grouping method:** (Absolute) PCA **# of groups:** 4	**Groups/ Factors/ Sources:** Regional SO_4 Soil; Unknown Oil combustion	**PM variables used:** Mass contribution of sources
	Results: Correlation: V and Ni positively correlated with NF-¤B. Oil combustion correlated the greatest with NF-¤B (0.316). Significance not provided. Only 2% of mass contribution originates from this source.					
Reference: Nikolov et al. (2008, 156808) **Location:** Boston, MA **Particle Size:** NR	**Subjects:** Dogs **Exposure:**	**n:** 8 dogs, 24 exposure-days in 1997-98; 4 dogs, 21 exposure-days in 2001-02	**Constituents considered for grouping:** 13 elements, BC, EC, OC	**Grouping method:** Compared 3 factor-analytic models within a SEM model **# of groups:** 4	**Groups/ Factors/ Sources:** Oil Combustion V/Ni; Power Plants S ;Road dust Al/Si ;Motor vehicles BC/OC/EC	**PM variables used:** Mass contribution of every constituent
	Results: Univariate response for respiratory outcomes: road dust and oil combustion associated with decreased respiratory frequency; motor vehicles associated with increased respiratory frequency; motor vehicles associated with increased PEF; road dust associated with decreased penh and motor vehicles associated with increased penh. **Multivariate responses for respiratory outcome**: Road dust associated with decreased respiratory rate; Motor vehicles associated with increased airway irritation.					
Reference: Rhoden et al. (2004, 087969) **Location:** Boston, MA **Particle Size:** $PM_{2.5}$	**Subjects** Rats (Sprague Dawley) **Exposure:** CAPs (avg. mass concentration range 150-2520 $\mu g/m^3$) acetylcysteine full factorial	**n:** 4-8 rats (1-2 per group - sham, CAPs, sham/NAC, CAP/NAC) 10 exposures	**Constituents considered for grouping:** 20 elements	**Grouping method:** No grouping was performed **# of groups:** NA	**Groups/ Factors/ Sources:** NR	**PM variables used:** Mass contribution of every constituent
	Results: Increased oxidative stress and inflammation in lungs of CAPs animals that was attenuated with NAC. **Univariate regression**: Al, Si, Fe, K, Pb, and Cu most significantly correlated with lung TBARS. No significant correlations for lung carbonyls or lung PMN.					

Reference: Saldiva et al. (2002, 025988) **Location:** Boston, MA **Particle Size:** $PM_{2.5}$	**Subjects:** Rats (Sprague Dawley **Exposure:** CAPs (3-day avg. mass concentration range 126.1-481 $\mu g/m^3$)	**n:** 7-10 rats/group (air/sham, SO_2/sham, air/CAP, SO_2/CAP) × 6 runs in different seasons	**Constituents considered for grouping:** 15 elements (used Clarke 2000 to select tracers)	**Grouping method:** Previous study in same city (Clarke et al. 2000) **# of groups:** 6	**Groups/ Factors/ Sources:** V/Ni S Al/Si Br/Pb _____ Na/Cl Cr	**PM variables used:** Mass contribution of 8 elelments in univariate step. Tracers (Si, SO_4, V, Pb, Br, Cl) and EC, OC in multivariate step.
	Results: Increased percent and number of PMN in majority of air and SO_2 rats exposed to CAPs, but significance levels not provided. Other responses (protein, LDH, NAG) were variable and depended upon the CAPs exposure. No CAPs effect on histopathology. **Linear regression**: V, Br, Pb, SO_4, EC, OC, Si, CAP mass associated with increased PMN and lymphocytes for normal+CB rats. Only V not associated with PMN in normal rats. Lymphocyte response due to CB rats, but not observed for SO_4 , Si, or mass in this group. Br, Pb, SO_4, EC, OC, Si associated with increased total protein in CB rats. Cl and V associated with decreased LDH in CB rats. No BAL effects in normal rats exposed to CAPs. V, Br, Pb, EC, OC, and Cl associated with increased neutrophil density in lungs of normal rats.					
Reference: Seagrave et al. (2006, 091291) **Location:** 4 SE US sites for 2 seasons **Particle Size:** $PM_{2.5}$	**Subjects** Rats (Fisher 344) **Exposure:** 0.75, 1.5 and 3 mg/rat via intratracheal instillation	**n:** 5 rats/dose	**Constituents considered for grouping:** NR	**Grouping method:** CMB **# of groups:** 13	**Groups/ Factors/ Sources:** secondary NO_3; secondary NH_4; secondary SO_4; coke production; vegetative detritus; natural gas combust; road dust; wood combust; meat cooking gasoline; diesel other OM; other mass	**PM variables used:** Mass contribution of every constituent, then mass contribution of sources
	Results: Potency depended upon season and site of sample collection. In general, effects were greater in the winter. **PLS analysis:** 2 major constituents identified (OC, Pb, hopanes/steranes, nitrate, As for first and major metal oxides for the second), gasoline most important predictor for both constituents, with diesel influencing second constituent and nitrate influencing first constituent. First constituent affected cytotoxic responses, second constituent affected inflammatory responses.					
Reference: Veranth et al. (2006, 087479) **Location:** 8 sites in the Western US **Particle Size:** $PM_{2.5}$	**Subjects** BEAS-2B cells (35000 cells/cm2; 10, 20, 40, 80 μg/cm2) **Exposure:** Loose surface soil sweepings through mechanical tumbler and cascade impactor	**n:** 6; 16 runs over 6 months.	**Constituents considered for grouping:** 13 elements, TC, 5 OC variables, 4 EC variables, 2 ions, EU, one ratio (Ca: Al), OP, CO3	**Grouping method:** PLS **# of groups:** NR	**Groups/ Factors/ Sources:** NR	**PM variables used:** Mass contribution every constituent (?)
	Results: Dose-related increase in IL-6 and decreases in cell viability for all soil types. IL-8 responses more variable and dependent upon soil type. Univariate correlations. Low correlations for all constituents tested with IL-6. Highest correlations for EC1 (R2 = 0.50) and pyrolyzed OC (R2 = 0.46), then Ca/Al (R2 = 0.21). Carbonate carbon, EC3, and Sr correlated with IL-8 (R2 = 0.27, 0.13, and 0.25, respectively). EC and Ni correlated with IL-8 trend over the range of 10-80 μg/cm2 (R2 = 0.39 and 0.27, respectively). Multivariate redundancy analysis OC1, OC3, OC2, EC2, Br, EC1, Ni correlated with IL-8 release, decreased viability, and decreased IL-6 at low and high doses. Ni, EC1, and EC2 correlated with IL-6 release at the high dose, decreased IL-6 at the low dose, decreased IL-8 release, and decreased viability. Br was negatively associated.					
Reference: Wellenius et al. (2003, 055691) **Location:** Boston, MA **Particle Size:** $PM_{2.5}$	**Subjects:** Dogs **Exposure:** CAPs (avg. mass concentration range 161.3-957.3 $\mu g/m^3$) repeated exposure with several weeks in between	**n:** 6 dogs, 20 exposures	**Constituents considered for grouping:** 15 elements (+EC OC?) (used Clarke et al. 2000)	**Grouping method:** Previous study in same city (Clarke et al. 2000) **# of groups:** 6 (but did not use all in analysis of health effects)	**Groups/ Factors/ Sources:** V/Ni S Al/Si Br/Pb _____ Na/Cl Cr	**PM variables used:** **Univariate:** Mass Number Ni, S, Si, BC **Multivariate:** Ni, S, Si , BC
	Results: ST-segment elevation increased with CAPs. **Univariate regression:** Si and Pb associated with peak ST-segment elevation and integrated ST-segment change.CAPs mass or number concentration were not associated with any change. **Multivariate regression:** Si associated with peak ST-segment elevation and integrated ST-segment change.					

Reference: Zhang et al. (2008, 192008) **Location:** Metro area of Denver, CO / 45 samples through 1 year	**Subjects:** Alveolar macrophage cell line (NR8383); 1x106 cells/ml **Exposure:** Soluble components exposure concentration range from 20-200 pg of PM/cell	**n:** 45 PM samples, 3 runs	**Constituents considered for grouping:** 43 + EC, OC	**Grouping method:** PMF **# of groups:** 9	**Groups/Factors/ Sources:** Mobile, Water soluble carbon, Sulfate, Soil, Iron, Cd and Zn point source, Pb, Pyrotechniques, Platinum	**PM variables used:** Mass contribution of sources
Particle Size: 2.5; filtered to 0.22 um	**Results:** Started with regression on 9 sources, then 3 (water-soluble carbon factor, soil dust source, iron source). Soil dust source was not significant. Final regression model excluded 3 days of outliers (Fe source most significant, then water-soluble carbon factor, then soil dust source) for ROS effects, with adjusted R2 of 0.774. Fe source likely associated with industrial source and includes high loadings of water-soluble Fe and Ti (not identified); water-soluble C factor derived from both secondary organic aerosol and biomass smoke (largely consists of polar organic compounds); soil dust source identified by water-soluble resuspended dust elements and contains Mg and Ca.					

Annex F References

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Note: Hyperlinks to the reference citations throughout this document will take you to the NCEA HERO database (Health and Environmental Research Online) at http://epa.gov/hero. HERO is a database of scientific literature used by U.S. EPA in the process of developing science assessments such as the Integrated Science Assessments (ISA) and the Integrated Risk Information System (IRIS).

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