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 Aerosol Speciation Sampler Model 450	URG Corp.
MiniVol	Battery-Powered Portable Low-Volume Sampler	Air Metrics Inc. (Eugene, OR)
PC-BOSS	Particle Concentrator-Brigham Young University Organic Sampling System	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, commercialized by Thermo Andersen Instruments; now withdrawn from market
RAMS	Real-Time Ambient Mass Sampler (modified Tapered Element Oscillation Microbalance with diffusion denuder and Nafion 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. Flash 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
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)

Abbreviation	Instrument	Manufacturer / Research Institute
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 Scientific Co. (Berkeley, CA)
AE-16	Magee AE-16 aethalometer (BC)	Magee Scientific Co.
AE-20	Magee AE-20 dual wavelength aethalometer (BC)	Magee Scientific Co.
AE-21	Magee AE-21 dual-wavelength aethalometer (BC)	Magee Scientific Co.
AE-31	Magee AE-31 seven color aethalometer (BC)	Magee Scientific Co.
DRI-PA	DRI Photoacoustic Analyzer (BC)	Droplet Measurement Technologies, Inc. (Boulder, CO)
MAAP	Multi-Angle Absorption Photometer, Model 5012 (BC)	Thermo Scientific 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 3 stages)	University of California–Davis (Davis, CA)
DRUM-8	Davis Rotating-Drum Uniform Size-Cut Monitor (0.09- > 5.0 µm in 8 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 Orifice Uniform Deposit Impactor	MSP Corp. (Minneapolis, MN)
MOUDI-100	MOUDI Model 100 (0.18-18 µm in 8 stages)	MSP Corp.
MOUDI-110	MOUDI Model 110 (0.056-18 µm in 10 stages)	MSP Corp.
Nano-MOUDI	Nano MOUDI (0.010-0.056 µm in 3 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.

Abbreviation	Instrument	Manufacturer / Research Institute
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 µm)	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 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[d] 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-200-VSCC 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. 15567, 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
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

Manufacturer[a]	Sampler Name	Size Cut[b]	Description	FRM or FEM[c]	Designation #	FRN
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 inlet (Watson et al., 1983, 045084) 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, while the other half is retained by the inlet) and 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% andc 84%, $[d_{16}/d_{84}]^{0.5}$).

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

[d] 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 et al. (2008, 156355)

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

Observable	Analytical Accuracy[a]	Precisionb	Minimum Detectable Limit (MDL)	Interferences	Comparability	Data Completeness
$PM_{2.5}$ mass	± 5% [4]	± 10% [4]	0.04 µg/m^3 [c] to ~1 µg/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% [h 6,7]
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 Teflon4 and ± 1−14% on nylon filters13	± 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% EC: ± 20% TC: ± 10% [4]	OC: 0.1 µg/m^3 [f] to 0.8 µg/m^3 [d] EC: 0.03 µg/m^3 [d] to 0.1 µg/m^3 [f] TC: 0.8 µg/m^3 [d 1,6]	Subject to adsorption (positive artifact) and volatilization (negative artifact) of organic gases to and from quartz-fiber filters	OC: Within 20 to 50% EC: Within 20 to 200% TC: Within 20% [4,17,22]	86 to 100% [6,7]
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: C, H, N, and S	C: 1.5%; H: 3%; N: 3%; S: 5% [30]	± 2% [30]	C: 0.3 µg/m^3 H:0.09 µg/m^3 N: 0.03 µg/m^3 S: 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 (R^2 = 0.87) [31]	N/A

Observable	Analytical Accuracy[a]	Precisionb	Minimum Detectable Limit (MDL)	Interferences	Comparability	Data Completeness
Neutral polyols and polyether	GC/MS: ± 4-8% [32]	GC/MS: ± 23% [33,34] Typically ± 20%, ranged from ± 10 to ± 30% [l 32,35,36,37,38] HPLC/MS: ± 5-26% [39]	GC/MS: Levoglucosan: 10 ng/m^3 [g 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 (R^2 = 0.97) with HPLC/MS; and (R^2 = 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
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 cm^2 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, 157360); [5]Solomon et al. (2001, 157193); [6]Watson et al. (2005, 157124); [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]Fitz 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. (2000, 012225); [36]Fine et al. (2004, 141283); [37]Yue et al. (2004, 157169); [38]Rinehart et al. (2006, 115184); [39]Wan and Yu (2006, 157104); [40]Poore (2000, 012839); [41]Fraser et al. (2003, 040266); [42]Engling et al. (2006, 156422); [43]Yu et al. (2005, 157167); [44]Tran et al. (2000, 013025); [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, 046318); [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, 157209); [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 (2002, 051444); [62]Butler et al. (2003, 156313); [63]Chow et al. (2006, 146622); [64]Russell et al. (2004, 082453); [65]Grover et al. (2006, 138080); [66]Grover et al. (2005, 090044); [67]Schwab et al. (2006, 098449); [68]Hauck et al. (2004, 156525); [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. (2005, 155925); [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, 136787); [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. (2006, 098785); [90]Lim et al. (2003, 037037); [91]Watson and Chow (2002, 037873); [92]Venkatachari et al. (2006, 105918); [93]Bae et al. (2004, 156243); [94]Arhami et al. (2006, 156224); [95]Park et al. (2005, 156843); [96]Bae et al. (2004, 098680); [97]Chow et al. (2006, 156350); [98]Arnott et al. (2005, 156227); [99]Bond et al. (1999, 156281); [100]Virkkula et al. (2005, 157097); [101]Petzold et al. (2002, 156863); [102]Park et al. (2006, 098104); [103]Arnott et al. (1999, 020650); [104]Peters et al. (2001, 016925); [105]Pitchford et al. (1997, 156872); [106]Rees et al. (2004, 097164); [107]Watson et al. (2000, 010354); [108]Lee et al. (2005, 156680); [109]Hering et al. (2004, 155837); [110]Watson et al. (1998, 198805); [111]Chakrabarti et al. (2004, 157426); [112]Mathai et al. (1990, 156741); [113]Kidwell and Ondov (2001, 017092); [114]Stanier et al. (2004, 095955); [115]Khlystov et al. (2005, 156635); [116]Takahama et al. (2004, 157038); [117]Chow et al. (2005, 156348); [118]Zhang et al. (2002, 157181); [119]Subramanian et al. (2004, 081203); [120]Chow et al. (2006, 155207); [121]Birch and Cary (1996, 026004); [122]Birch (1998, 024953); [123]Birch and Cary (1996, 002352); [124]NIOSH (1996, 156810); [125]NIOSH (1999, 156811); [126]Chow et al. (1993, 077459); [127]Chow et al. (2007, 156354); [128]Ellis and Novakov (1982, 156416); [129]Peterson and Richards (2002, 156861); [130]Schauer et al. (2003, 037014); [13]1Middlebrook et al. (2003, 042932); [132]Wenzel et al. (2003, 157139); [133]Jimenez et al. (2003, 156611); [134]Phares et al. (2003, 156866); [135]Qin and Prather (2006, 156895); [136]Zhang et al. (2005, 157185); [137]Bein et al. (2005, 156265); [138]Drewnick et al. (2004, 155754); [139]Drewnick et al. (2004, 155755); [140]Lake et al. (2003, 156669); [141]Lake et al. (2004, 088411)

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]
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 stationary phase column used during TD analysis. Incomplete thermal desorption of analytes because of strong affinity with filter matrix.	Possible contaminants from solvents and complicated 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 R^2 = 0.731 [59]

	Analytical Accuracy		Precision		MDL		Interferences		
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

[1]Chow (1995, 077012); [2]Watson and Chow (2001, 157123); [3] Watson et al. (1983, 045084); [4]Fehsenfeld et al. (2004, 157360); [5]Solomon et al. (2001, 157193); [6]Watson et al. (2005, 157124); [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]Fitz 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. (2000, 012225); [36]Fine et al. (2004, 141283); [37]Yue et al. (2004, 157169); [38]Rinehart et al. (2006, 115184); [39]Wan and Yu (2006, 157104); [40]Poore (2000, 012839); [41]Fraser et al. (2003, 040266); [42]Engling et al. (2006, 156422); [43]Yu et al. (2005, 157167); [44]Tran et al. (2000, 013025); [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, 046318); [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, 157209); [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 (2002, 051444); [62]Butler et al. (2003, 156313); [63]Chow et al. (2006, 146622); [64]Russell et al. (2004, 082453); [65]Grover et al. (2006, 138080); [66]Grover et al. (2005, 090044); [67]Schwab et al. (2006, 098449); [68]Hauck et al. (2004, 156525); [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. (2005, 155925); [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, 136787); [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. (2006, 098785); [90]Lim et al. (2003, 037037); [91]Watson and Chow (2002, 037873); [92]Venkatachari et al. (2006, 105918); [93]Bae et al. (2004, 156243); [94]Arhami et al. (2006, 156224); [95]Park et al. (2005, 156843); [96]Bae et al. (2004, 098680); [97]Chow et al. (2006, 156350); [98]Arnott et al. (2005, 156227); [99]Bond et al. (1999, 156281); [100]Virkkula et al. (2005, 157097); [101]Petzold et al. (2002, 156863); [102]Park et al. (2006, 098104); [103]Arnott et al. (1999, 020650); [104]Peters et al. (2001, 016925); [105]Pitchford et al. (1997, 156872); [106]Rees et al. (2004, 097164); [107]Watson et al. (2000, 010354); [108]Lee et al. (2005, 156680); [109]Hering et al. (2004, 155837); [110]Watson et al. (1998, 198805); [111]Chakrabarti et al. (2004, 157426); [112]Mathai et al. (1990, 156741); [113]Kidwell and Ondov (2001, 017092); [114]Stanier et al. (2004, 095955); [115]Khlystov et al. (2005, 156635); [116]Takahama et al. (2004, 157038); [117]Chow et al. (2005, 156348); [118]Zhang et al. (2002, 157181); [119]Subramanian et al. (2004, 081203); [120]Chow et al. (2006, 155207); [121]Birch and Cary (1996, 026004); [122]Birch (1998, 024953); [123]Birch and Cary (1996, 002352); [124]NIOSH (1996, 156810); [125]NIOSH (1999, 156811); [126]Chow et al. (1993, 077459); [127]Chow et al. (2007, 156354); [128]Ellis and Novakov (1982, 156416); [129]Peterson and Richards (2002, 156861); [130]Schauer et al. (2003, 037014); [131]Middlebrook et al. (2003, 042932); [132]Wenzel et al. (2003, 157139); [133]Jimenez et al. (2003, 156611); [134]Phares et al. (2003, 156866); [135]Qin and Prather (2006, 156895); [136]Zhang et al. (2005, 157185); [137]Bein et al. (2005, 156265); [138]Drewnick et al. (2004, 155754); [139]Drewnick et al. (2004, 155755); [140]Lake et al. (2003, 156669); [141]Lake et al. (2004, 088411)

Source: Chow et al. (2008, 156355)

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

Instrument and Measurement Principle	Averaging Time	Analytical Accuracy[a]	Precisionb	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 avgc,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]-92% [6]
FDMS TEOM. 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]	95-99%[65,68] 57-65%[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 [72] FRM mass [73,74]	N/A

Instrument and Measurement Principle	Averaging Time	Analytical Accuracy[a]	Precisionb	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 (~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 DTEOM 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	± 3 µg for 24-h avg concentrations < 100 µg/m^3 and 2% for 100 to 1,000 µg/m^3 ± 8 µg for 1-h concentrations < 100 µg/m^3 and 8% for 100 to 1000 µg/m^3	± 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]	93-99%[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: ± 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 (R^2 >0.80). Comparability depends on conversion factor used.	>80-98% 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]	Precisionb	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 μg/m^3, R^2 = 0.99).
[j] Specified as "Zero stability" by the manufacturer.
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, 157360); [5]Solomon et al. (2001, 157193); [6]Watson et al. (2005, 157124); [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]Fitz 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. (2000, 012225); [36]Fine et al. (2004, 141283); [37]Yue et al. (2004, 157169); [38]Rinehart et al. (2006, 115184); [39]Wan and Yu (2006, 157104); [40]Poore (2000, 012839); [41]Fraser et al. (2003, 040266); [42]Engling et al. (2006, 156422); [43]Yu et al. (2005, 157167); [44]Tran et al. (2000, 013025); [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, 046318); [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, 157209); [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 (2002, 051444); [62]Butler et al. (2003, 156313); [63]Chow et al. (2006, 146622); [64]Russell et al. (2004, 082453); [65]Grover et al. (2006, 138080); [66]Grover et al. (2005, 090044); [67]Schwab et al. (2006, 098449); [68]Hauck et al. (2004, 156525); [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. (2005, 155925); [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, 136787); [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. (2006, 098785); [90]Lim et al. (2003, 037037); [91]Watson and Chow (2002, 037873); [92]Venkatachari et al. (2006, 105918); [93]Bae et al. (2004, 156243); [94]Arhami et al. (2006, 156224); [95]Park et al. (2005, 156843); [96]Bae et al. (2004, 098680); [97]Chow et al. (2006, 156350); [98]Arnott et al. (2005, 156227); [99]Bond et al. (1999, 156281); [100]Virkkula et al. (2005, 157097); [101]Petzold et al. (2002, 156863); [102]Park et al. (2006, 098104); [103]Arnott et al. (1999, 020650); [104]Peters et al. (2001, 016925); [105]Pitchford et al. (1997, 156872); [106]Rees et al. (2004, 097164); [107]Watson et al. (2000, 010354); [108]Lee et al. (2005, 156680); [109]Hering et al. (2004, 155837); [110]Watson et al. (1998, 198805); [111]Chakrabarti et al. (2004, 157426); [112]Mathai et al. (1990, 156741); [113]Kidwell and Ondov (2001, 017092); [114]Stanier et al. (2004, 095955); [115]Khlystov et al. (2005, 156635); [116]Takahama et al. (2004, 157038); [117]Chow et al. (2005, 156348); [118]Zhang et al. (2002, 157181); [119]Subramanian et al. (2004, 081203); [120]Chow et al. (2006, 155207); [121]Birch and Cary (1996, 026004); [122]Birch (1998, 024953); [123]Birch and Cary (1996, 002352); [124]NIOSH (1996, 156810); [125]NIOSH (1999, 156811); [126]Chow et al. (1993, 077459); [127]Chow et al. (2007, 156354); [128]Ellis and Novakov (1982, 156416); [129]Peterson and Richards (2002, 156861); [130]Schauer et al. (2003, 037014); [131]Middlebrook et al. (2003, 042932); [132]Wenzel et al. (2003, 157139); [133]Jimenez et al. (2003, 156611); [134]Phares et al. (2003, 156866); [135]Qin and Prather (2006, 156895); [136]Zhang et al. (2005, 157185); [137]Bein et al. (2005, 156265); [138]Drewnick et al. (2004, 155754); [139]Drewnick et al. (2004, 155755); [140]Lake et al. (2003, 156669); [141]Lake et al. (2004, 088411)

Source: Chow et al. (2008, 156355)

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

Instrument and Measurement Principle	Averaging Time	Analytical Accuracya	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]	20-43%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
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

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

Source: Chow et al. (2008, 156355)

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

Instrument and Measurement Principle	Averaging Time	Analytical Accuracy[a]	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 µg/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 µg/m^3 for 24-h avg [83,84] 0.24 µg/m^3 to 0.45 µg/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->94%[6,20,83-85]
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 s	N/A	N/A	0.5 µg/m^3 for 30-s 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 µg/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 µg/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]	0.05-0.1 µg/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]	65-70%[20]

Instrument and Measurement Principle	Averaging Time	Analytical Accuracy[a]	Precision	MDL	Interferences	Comparability	Data Completion
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 supersaturation 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 µg/m^3 for 1-h avg[e]	N/A	N/A	N/A
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 µg/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&P8400N 20	94-98%[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.

[1]Chow (1995, 077012); [2]Watson and Chow (2001, 157123); [3] Watson et al. (1983, 045084); [4]Fehsenfeld et al. (2004, 157360); [5]Solomon et al. (2001, 157193); [6]Watson et al. (2005, 157124); [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]Fitz 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. (2000, 012225); [36]Fine et al. (2004, 141283); [37]Yue et al. (2004, 157169); [38]Rinehart et al. (2006, 115184); [39]Wan and Yu (2006, 157104); [40]Poore (2000, 012839); [41]Fraser et al. (2003, 040266); [42]Engling et al. (2006, 156422); [43]Yu et al. (2005, 157167); [44]Tran et al. (2000, 013025); [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, 046318); [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, 157209); [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 (2002, 051444); [62]Butler et al. (2003, 156313); [63]Chow et al. (2006, 146622); [64]Russell et al. (2004, 082453); [65]Grover et al. (2006, 138080); [66]Grover et al. (2005, 090044); [67]Schwab et al. (2006, 098449); [68]Hauck et al. (2004, 156525); [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. (2005, 155925); [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, 136787); [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. (2006, 098785); [90]Lim et al. (2003, 037037); [91]Watson and Chow (2002, 037873); [92]Venkatachari et al. (2006, 105918); [93]Bae et al. (2004, 156243); [94]Arhami et al. (2006, 156224); [95]Park et al. (2005, 156843); [96]Bae et al. (2004, 098680); [97]Chow et al. (2006, 156350); [98]Arnott et al. (2005, 156227); [99]Bond et al. (1999, 156281); [100]Virkkula et al. (2005, 157097); [101]Petzold et al. (2002, 156863); [102]Park et al. (2006, 098104); [103]Arnott et al. (1999, 020650); [104]Peters et al. (2001, 016925); [105]Pitchford et al. (1997, 156872); [106]Rees et al. (2004, 097164); [107]Watson et al. (2000, 010354); [108]Lee et al. (2005, 156680); [109]Hering et al. (2004, 155837); [110]Watson et al. (1998, 198805); [111]Chakrabarti et al. (2004, 157426); [112]Mathai et al. (1990, 156741); [113]Kidwell and Ondov (2001, 017092); [114]Stanier et al. (2004, 095955); [115]Khlystov et al. (2005, 156635); [116]Takahama et al. (2004, 157038); [117]Chow et al. (2005, 156348); [118]Zhang et al. (2002, 157181); [119]Subramanian et al. (2004, 081203); [120]Chow et al. (2006, 155207); [121]Birch and Cary (1996, 026004); [122]Birch (1998, 024953); [123]Birch and Cary (1996, 002352); [124]NIOSH (1996, 156810); [125]NIOSH (1999, 156811); [126]Chow et al. (1993, 077459); [127]Chow et al. (2007, 156354); [128]Ellis and Novakov (1982, 156416); [129]Peterson and Richards (2002, 156861); [130]Schauer et al. (2003, 037014); [13]1Middlebrook et al. (2003, 042932); [132]Wenzel et al. (2003, 157139); [133]Jimenez et al. (2003, 156611); [134]Phares et al. (2003, 156866); [135]Qin and Prather (2006, 156895); [136]Zhang et al. (2005, 157185); [137]Bein et al. (2005, 156265); [138]Drewnick et al. (2004, 155754); [139]Drewnick et al. (2004, 155755); [140]Lake et al. (2003, 156669); [141]Lake et al. (2004, 088411)

Source: Chow et al. (2008, 156355)

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 (ADIS) 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 µg/m^3 [82]	N/A	Within 15% of filter and continuous SO_4^{2-} See Weber et al. [82] for details.	100%[7]
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 µg/m^3 and >30% at conc. < 2 µg/m^3 [b] [84]	0.48 µg/m^3 [85]	SO_4^{2-} to SO_2 conversion and volatilization efficiency appears to depend on ambient composition [84]	10-30% lower than filter SO_4^{2-} [20,21,84]	84- 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]	80-98%[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 µg/m^3 for 24-h avg [89] 0.5 µg/m^3 for 15-min avgd	SO_4^{2-} to SO_2 conversion efficiency depends on ambient composition [89]	~20% lower than filter SO_4^{2-} [89]	88-90%[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%
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 µg/m^3 [82,88]	Consistent water quality is essential for good precision.	Within 30% of filter and other continuous SO_4^{2-} [20,21]	65-70%[20,21]

Instrument and Measurement Principle	Averaging Time	Analytical Accuracy[a]	Precision	MDL	Interferences	Comparability	Data Completeness
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.	1h	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
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.	1h	N/A	N/A	0.1 µg/m^3 for 1-h avg	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]	93-98%[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 = 0.01-0.2, R^2>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

[1]Chow (1995, 077012); [2]Watson and Chow (2001, 157123); [3] Watson et al. (1983, 045084); [4]Fehsenfeld et al. (2004, 157360); [5]Solomon et al. (2001, 157193); [6]Watson et al. (2005, 157124); [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]Fitz 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. (2000, 012225); [36]Fine et al. (2004, 141283); [37]Yue et al. (2004, 157169); [38]Rinehart et al. (2006, 115184); [39]Wan and Yu (2006, 157104); [40]Poore (2000, 012839); [41]Fraser et al. (2003, 040266); [42]Engling et al. (2006, 156422); [43]Yu et al. (2005, 157167); [44]Tran et al. (2000, 013025); [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, 046318); [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, 157209); [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 (2002, 051444); [62]Butler et al. (2003, 156313); [63]Chow et al. (2006, 146622); [64]Russell et al. (2004, 082453); [65]Grover et al. (2006, 138080); [66]Grover et al. (2005, 090044); [67]Schwab et al. (2006, 098449); [68]Hauck et al. (2004, 156525); [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. (2005, 155925); [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, 136787); [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. (2006, 098785); [90]Lim et al. (2003, 037037); [91]Watson and Chow (2002, 037873); [92]Venkatachari et al. (2006, 105918); [93]Bae et al. (2004, 156243); [94]Arhami et al. (2006, 156224); [95]Park et al. (2005, 156843); [96]Bae et al. (2004, 098680); [97]Chow et al. (2006, 156350); [98]Arnott et al. (2005, 156227); [99]Bond et al. (1999, 156281); [100]Virkkula et al. (2005, 157097); [101]Petzold et al. (2002, 156863); [102]Park et al. (2006, 098104); [103]Arnott et al. (1999, 020650); [104]Peters et al. (2001, 016925); [105]Pitchford et al. (1997, 156872); [106]Rees et al. (2004, 097164); [107]Watson et al. (2000, 010354); [108]Lee et al. (2005, 156680); [109]Hering et al. (2004, 155837); [110]Watson et al. (1998, 198805); [111]Chakrabarti et al. (2004, 157426); [112]Mathai et al. (1990, 156741); [113]Kidwell and Ondov (2001, 017092); [114]Stanier et al. (2004, 095955); [115]Khlystov et al. (2005, 156635); [116]Takahama et al. (2004, 157038); [117]Chow et al. (2005, 156348); [118]Zhang et al. (2002, 157181); [119]Subramanian et al. (2004, 081203); [120]Chow et al. (2006, 155207); [121]Birch and Cary (1996, 026004); [122]Birch (1998, 024953); [123]Birch and Cary (1996, 002352); [124]NIOSH (1996, 156810); [125]NIOSH (1999, 156811); [126]Chow et al. (1993, 077459); [127]Chow et al. (2007, 156354); [128]Ellis and Novakov (1982, 156416); [129]Peterson and Richards (2002, 156861); [130]Schauer et al. (2003, 037014); [13]1Middlebrook et al. (2003, 042932); [132]Wenzel et al. (2003, 157139); [133]Jimenez et al. (2003, 156611); [134]Phares et al. (2003, 156866); [135]Qin and Prather (2006, 156895); [136]Zhang et al. (2005, 157185); [137]Bein et al. (2005, 156265); [138]Drewnick et al. (2004, 155754); [139]Drewnick et al. (2004, 155755); [140]Lake et al. (2003, 156669); [141]Lake et al. (2004, 088411)

Source: Chow et al. (2008, 156355)

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

Instrument and Measurement Principle	Averaging Time	Analytical Accuracy[a]	Precision	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 µg/m^3 [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]	[88] 0.05 µg/m^3	Consistent water quality is essential for good precision	~5% lower than all-sampler avg[c] 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 µg/m^3 [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 µg/m^3 for 1-h avg[d]	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] 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

[1]Chow (1995, 077012); [2]Watson and Chow (2001, 157123); [3] Watson et al. (1983, 045084); [4]Fehsenfeld et al. (2004, 157360); [5]Solomon et al. (2001, 157193); [6]Watson et al. (2005, 157124); [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]Fitz 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. (2000, 012225); [36]Fine et al. (2004, 141283); [37]Yue et al. (2004, 157169); [38]Rinehart et al. (2006, 115184); [39]Wan and Yu (2006, 157104); [40]Poore (2000, 012839); [41]Fraser et al. (2003, 040266); [42]Engling et al. (2006, 156422); [43]Yu et al. (2005, 157167); [44]Tran et al. (2000, 013025); [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, 046318); [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, 157209); [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 (2002, 051444); [62]Butler et al. (2003, 156313); [63]Chow et al. (2006, 146622); [64]Russell et al. (2004, 082453); [65]Grover et al. (2006, 138080); [66]Grover et al. (2005, 090044); [67]Schwab et al. (2006, 098449); [68]Hauck et al. (2004, 156525); [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. (2005, 155925); [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, 136787); [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. (2006, 098785); [90]Lim et al. (2003, 037037); [91]Watson and Chow (2002, 037873); [92]Venkatachari et al. (2006, 105918); [93]Bae et al. (2004, 156243); [94]Arhami et al. (2006, 156224); [95]Park et al. (2005, 156843); [96]Bae et al. (2004, 098680); [97]Chow et al. (2006, 156350); [98]Arnott et al. (2005, 156227); [99]Bond et al. (1999, 156281); [100]Virkkula et al. (2005, 157097); [101]Petzold et al. (2002, 156863); [102]Park et al. (2006, 098104); [103]Arnott et al. (1999, 020650); [104]Peters et al. (2001, 016925); [105]Pitchford et al. (1997, 156872); [106]Rees et al. (2004, 097164); [107]Watson et al. (2000, 010354); [108]Lee et al. (2005, 156680); [109]Hering et al. (2004, 155837); [110]Watson et al. (1998, 198805); [111]Chakrabarti et al. (2004, 157426); [112]Mathai et al. (1990, 156741); [113]Kidwell and Ondov (2001, 017092); [114]Stanier et al. (2004, 095955); [115]Khlystov et al. (2005, 156635); [116]Takahama et al. (2004, 157038); [117]Chow et al. (2005, 156348); [118]Zhang et al. (2002, 157181); [119]Subramanian et al. (2004, 081203); [120]Chow et al. (2006, 155207); [121]Birch and Cary (1996, 026004); [122]Birch (1998, 024953); [123]Birch and Cary (1996, 002352); [124]NIOSH (1996, 156810); [125]NIOSH (1999, 156811); [126]Chow et al. (1993, 077459); [127]Chow et al. (2007, 156354); [128]Ellis and Novakov (1982, 156416); [129]Peterson and Richards (2002, 156861); [130]Schauer et al. (2003, 037014); [131]Middlebrook et al. (2003, 042932); [132]Wenzel et al. (2003, 157139); [133]Jimenez et al. (2003, 156611); [134]Phares et al. (2003, 156866); [135]Qin and Prather (2006, 156895); [136]Zhang et al. (2005, 157185); [137]Bein et al. (2005, 156265); [138]Drewnick et al. (2004, 155754); [139]Drewnick et al. (2004, 155755); [140]Lake et al. (2003, 156669); [141]Lake et al. (2004, 088411)

Source: Chow et al. (2008, 156355)

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

Instrument and Measurement Principle	Averaging Time	Analytical Accuracy[a]	Precision	MDL	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 µg/m^3 EC, TC: not applicable, since it measures only OC [90]	N/A	15-22% 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 µg/m^3 EC: 0.5 µg/m^3 TC: 0.5 µg/m^3 [90]	N/A	20 to 60% lower TC than filter TC by TOR or TOT.[91,92]	56-60%[6,91]
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 µg/m^3 EC: 0.5 µg/m^3 TC: 0.4 µg/m^3 [90]	N/A	8% higher OC and 20% lower EC than R&P-5400 [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 µg/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 differences in temperature and analysis protocols. [92,95,96]	80-89%[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 µg/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]	75-90%[6]

Instrument and Measurement Principle	Averaging Time	Analytical Accuracy[a]	Precision	MDL	Interferences	Comparability	Data Completeness
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 µg/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 babs and is converted to BC using σ_{abs} of 6.6 m_2/g.	1 min	N/A	12%[9,101]	BC[h]: 0.05 µg/m^3 (or b_{abs} = 0.33 Mm^{-1} for 10-min avg) 0.02 µg/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 (R^2 = 0.96) and up to 40% higher than Sunset EC. [102]	N/A
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 babs, which is converted to BC using σ_{abs}s = 5 m^2/g for the 1047 nm instrument and σ_{abs} = 10 m2/g for the 532 nm instrument.	5 s	N/A	N/A	BC[i]: 0.04 µg/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 (R^2 >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 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%[6]

Instrument and Measurement Principle	Averaging Time	Analytical Accuracy[a]	Precision	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] 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; R^2 = 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 m2/g at 880 nm, Lim et al. (2003, 037037) 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.

[1]Chow (1995, 077012); [2]Watson and Chow (2001, 157123); [3] Watson et al. (1983, 045084); [4]Fehsenfeld et al. (2004, 157360); [5]Solomon et al. (2001, 157193); [6]Watson et al. (2005, 157124); [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]Fitz 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. (2000, 012225); [36]Fine et al. (2004, 141283); [37]Yue et al. (2004, 157169); [38]Rinehart et al. (2006, 115184); [39]Wan and Yu (2006, 157104); [40]Poore (2000, 012839); [41]Fraser et al. (2003, 040266); [42]Engling et al. (2006, 156422); [43]Yu et al. (2005, 157167); [44]Tran et al. (2000, 013025); [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, 046318); [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, 157209); [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 (2002, 051444); [62]Butler et al. (2003, 156313); [63]Chow et al. (2006, 146622); [64]Russell et al. (2004, 082453); [65]Grover et al. (2006, 138080); [66]Grover et al. (2005, 090044); [67]Schwab et al. (2006, 098449); [68]Hauck et al. (2004, 156525); [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. (2005, 155925); [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, 136787); [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. (2006, 098785); [90]Lim et al. (2003, 037037); [91]Watson and Chow (2002, 037873); [92]Venkatachari et al. (2006, 105918); [93]Bae et al. (2004, 156243); [94]Arhami et al. (2006, 156224); [95]Park et al. (2005, 156843); [96]Bae et al. (2004, 098680); [97]Chow et al. (2006, 156350); [98]Arnott et al. (2005, 156227); [99]Bond et al. (1999, 156281); [100]Virkkula et al. (2005, 157097); [101]Petzold et al. (2002, 156863); [102]Park et al. (2006, 098104); [103]Arnott et al. (1999, 020650); [104]Peters et al. (2001, 016925); [105]Pitchford et al. (1997, 156872); [106]Rees et al. (2004, 097164); [107]Watson et al. (2000, 010354); [108]Lee et al. (2005, 156680); [109]Hering et al. (2004, 155837); [110]Watson et al. (1998, 198805); [111]Chakrabarti et al. (2004, 157426); [112]Mathai et al. (1990, 156741); [113]Kidwell and Ondov (2001, 017092); [114]Stanier et al. (2004, 095955); [115]Khlystov et al. (2005, 156635); [116]Takahama et al. (2004, 157038); [117]Chow et al. (2005, 156348); [118]Zhang et al. (2002, 157181); [119]Subramanian et al. (2004, 081203); [120]Chow et al. (2006, 155207); [121]Birch and Cary (1996, 026004); [122]Birch (1998, 024953); [123]Birch and Cary (1996, 002352); [124]NIOSH (1996, 156810); [125]NIOSH (1999, 156811); [126]Chow et al. (1993, 077459); [127]Chow et al. (2007, 156354); [128]Ellis and Novakov (1982, 156416); [129]Peterson and Richards (2002, 156861); [130]Schauer et al. (2003, 037014); [13]1Middlebrook et al. (2003, 042932); [132]Wenzel et al. (2003, 157139); [133]Jimenez et al. (2003, 156611); [134]Phares et al. (2003, 156866); [135]Qin and Prather (2006, 156895); [136]Zhang et al. (2005, 157185); [137]Bein et al. (2005, 156265); [138]Drewnick et al. (2004, 155754); [139]Drewnick et al. (2004, 155755); [140]Lake et al. (2003, 156669); [141]Lake et al. (2004, 088411)

Source: Chow et al. (2008, 156355)

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)

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

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. (2001, 016925)[104]: RTP [97] dataset

CV was 1.7%, 2.3%, 3.4%, 6.4% for the PQ200, Partisol 2000, RAAS2.5100, 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. (2001, 016925)[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.

Atlanta Supersite, GA: 8/3/99 to 9/1/99
Four km NW of downtown, within 200 m of a bus maintenance yard and several warehouse facilities, representative of a mixed commercial-residential neighborhood.

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
$PM_{10\text{-}2.5}$	1.67	Polycarbonate	None Na_2CO_3/Citric
URG-PCM	16.7	Teflon (P)	Acid
ARA-PCM	16.7	Teflon (N/A)	Na_2CO_3/Citric acid
PC-BOSS (operated by TVA)	105	Teflon (W)	CIF

Solomon et al. (2003, 156994)[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 R^2 >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 the filters, which may have enhanced loss of semi-volatile components, resulting in a lower mass on the filter.

Site / Period / Sampler / Configuration					Summary of Findings
PC-BOSS (operated by BYU)	150	Teflon (W)		CIF	**Butler et al. (2003, 156313)[62]** The sum of individual species accounted for ~78% of the RAAS-100 FRM $PM_{2.5}$ mass concentration. TEOM explained ~82 to 92% of the species sum of RAAS with R^2 = 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. (2005, 128139)[73]** RAMS $PM_{2.5}$ adjusted using particle concentrator efficiency of 0.5. Good correlation between SES-TEOM and Radiance Research M903s (R^2 = 0.80), while medium correlation was found between CAMM and Radiance Research M903 (R^2 = 0.64) or RAMS and Radiance Research M903 (R^2 = 0.63). CAMM = (0.75 ± 0.03) SES-TEOM + (2.51 ± 0.51); R^2 = 0.78; N = 196 RAMS = (0.85 ± 0.06) SES-TEOM + (5.34 ± 1.04); R^2 = 0.52; N = 96 RAMS = (0.91 ± 0.07) CAMM + (5.71 ± 1.20); R^2 = 0.43; N = 196 Semi-volatile material explains the difference between RAMS and SES TEOM. CAMM = (0.75 ± 0.08) R&P-2025 FRM + (2.47 ± 1.02); R^2 = 0.76; N = 31 RAMS = (0.97 ± 0.22) R&P-2025 FRM + (2.39 ± 3.42); R^2 = 0.64; N = 13 SES-TEOM = (1.07 ± 0.05) R&P-2025 FRM + (-1.34 ± 0.71); R^2 = 0.95; N = 26 CAMM vs. FRM yielded lower slopes (0.75) with high intercepts.
$PM_{2.5}$ Sampler	**Flow Rate (L/Min)**	**Filter Type[a]**		**Denuder[b]**	
R&P-2025 FRM	16.7	Teflon (N/A)		None	
$PM_{2.5}$ Continuous Sampler	**Flow Rate (L/Min)**	**Inlet Temperature**	**Dryer**	**Other**	
TEOM	16.7	30 °C	Nafion	$PM_{2.5}$	
SES-TEOM	16.7	30 °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	
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. (2004, 148859)[18]: Rees et al. (2004, 097164)[106]** MOUDI PM_{10} = 0.80 Dichot PM_{10}, R^2 = 0.85 MOUDI $PM_{2.5}$ = 1.03 Dichot $PM_{2.5}$, R^2 = 0.78 MOUDI $PM_{2.5}$ = 1.01 FRM $PM_{2.5}$, R^2 = 0.78 Dichot $PM_{2.5}$ = 0.97 FRM $PM_{2.5}$ + 0.02; R^2 = 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. Ultrafine (< 100 nm) mass (PM_{0-10}) measurements had high uncertainties (~30%) 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, R^2 = 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. **Rees et al. (2004, 097164)[106]** SES-TEOM $PM_{2.5}$ = 1.02 FRM $PM_{2.5}$ + 0.65; R^2 = 0.95 Volatilization did not affect SES-TEOM performance when $PM_{2.5}$ mass >20-30 µg/m^3. When ambient temperature was < -6 °C, and when mass was low, SES-TEOM was lower (up to 50%) than FRM or Dichot.
Sampler	**Flow Rate (L/Min)**	**Filter Type[a]**		**Denuder**	
MOUDI-110	30	Teflon (P,d)		None	
And-241 Dichot	16.7	Teflon (P		None	
R&P-2000 $PM_{2.5}$ FRM	16.7	Teflon (W)		None	
$PM_{2.5}$ Continuous Sampler	**Flow Rate (L/Min)**	**Inlet Temperature**	**Dryer**	**Other**	
SES-TEOM	16.7	30 °C	Nafion	$PM_{2.5}$	
DAASS	N/A	30 °C	Nafion or None	$PM_{2.5}$	

Site / Period / Sampler / Configuration	Summary of Findings

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 Type[a]	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. (2006, 146622)[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 (R^2 >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 (R^2) 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_4NO_3) 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 (R^2 >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 (R^2 >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 µg/m^3.

Grover et al. (2006, 138080) [65]

PC-BOSS $PM_{2.5}$ = (0.88 ± 0.04) FDMS-TEOM + (6.7 ± 4.3); R^2 = 95; n = 29

PC-BOSS $PM_{2.5}$ = (1.11 ± 0.07) D-TEOM + (7.5 ± 6.1); R^2 = 0.90; n = 29

TEOM50C $PM_{2.5}$ = (0.80 ± 0.01) TEO$m^3$0C + (1.1 ± 3.1); R^2 = 0.91; n = 507

TEO$m^3$0C $PM_{2.5}$ = (0.50 ± 0.01) FDMS-TEOM -(1.7 ± 6.9); R^2 = 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_4NO_3. 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}
Sampler	**Flow Rate (L/Min)**	**Filter Type[a]**		**Denuder[b]**
PC-BOSS $PM_{2.5}$	150	Teflon (W)		CIF

Site / Period / Sampler / Configuration					Summary of Findings
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. (2004, 082453)[64]; Lee et al. (2005, 156680)[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); R^2 = 0.81 SES-TEOM = (0.92 ± 0.03) RAMS + (1.52 ± 0.77); R^2 = 0.80 SES-TEOM = (1.01 ± 0.03) CAMM + (-1.91 ± 0.79); R^2 = 0.83 Correlation of Radiance Research M903 and SES-TEOM was good (R^2 = 0.95), while that of Radiance Research M903 with CAMM or RAMS was poor (R^2 ~ 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); R^2 = 0.89 RAMS = (1.10 ± 0.08) R&P-2025 + (0.68 ± 1.28); R^2 = 0.89 SES-TEOM = (1.09 ± 0.07) R&P-2025 + (0.21 ± 1.27); R^2 = 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. (2004, 155878)[69]; Hering et al. (2004, 155837)[109]** Dichot $PM_{2.5}$ = 0.83 MOUDI + 1.23; R^2 = 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; R^2 = 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 than the pressure drop across the filter. Vaporization from the TEOM had a time constant between 10 and 100 min depending on ambient and TEOM filter temperatures, the vapor pressure, and
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)		$NaHCO_3$	
Continuous Sampler	**Flow Rate (L/Min)**	**Inlet Temperature**	**Dryer**	**Other**	
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	

Site / Period / Sampler / Configuration					Summary of Findings
					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.
SMPS-3936	0.3	Ambient	None	Number to mass assuming spherical particles of 1.6 g/cc density	**Chakrabarti et al. (2004, 157426)[111]** Good agreement between MOUDI $PM_{0.15}$ and Nano-BAM $PM_{0.15}$ (MOUDI PM0.15 = 0.97 Nano-BAM $PM_{0.15}$ + 0.60; R^2 = 0.92; n = 24) Nano-BAM captured peak $PM_{0.15}$ 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 northwest 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[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

Grover et al. (2005, 090044)[66] (2003 measurements):

D-TEOM = (0.98 ± 0.02) FDMS-TEOM + (-0.6 ± 5.3); R^2=0.85; n = 426; excludes 38 data points when FDMS-TEOM $PM_{2.5}$ was higher than D-TEOM $PM_{2.5}$ by ~21 µg/m^3.

RAMS = (0.93 ± 0.02) FDMS-TEOM + (2.4 ± 8.2); R^2 = 0.81; n = 337

FDMS-TEOM = (0.96 ± 0.06) PC-BOSSconstructed mass + (-0.3 ± 3.9); R^2 = 0.90; n = 33

R&P-2025 FRM = (0.96 ± 0.06) FDMS-TEOM + (-9.3 ± 3.9); R^2 = 0.90; n = 29

The R&P-2025 FRM $PM_{2.5}$ was, on avg, ~32% lower than FDMSTEOM. Losses of NH_4NO_3 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. (2005, 155925)[76] (2001 measurements)

D-TEOM $PM_{2.5}$ and Radiance Research M903s light scattering (with and without dryers) showed good correlation.

D-TEOM = (3.69 ± 0.09) Radiance Research M903no-dryer + (2.74 ± 0.89); R^2 = 0.84; n = 299

D-TEOM = (3.79 ± 0.10) Radiance Research M903dryed + (4.08 ± 0.84); R^2 = 0.83; n = 312

Radiance Research M903no-dryer = (1.03 ± 0.01) Radiance Research M903dryed + (0.34 ± 0.05); R^2 = 0.98; n = 513; absorbed water did not affect relationship to $PM_{2.5}$.

CAMM and RAMS compared poorly (R^2 = 0 to 0.25) with D-TEOM, Radiance Research M903s and among themselves.

RAMS correlated well with D-TEOM for $PM_{2.5}$ >30 µg/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 (R^2 = 0.62) and OC by Sunset OCEC (R^2 = 0.61) suggesting that D-TEOM measured $PM_{2.5}$ mass with minimum loss of SVOCs. RAMS showed R^2 of 0.20 (NO_3^-) to 0.30 (OC), while CAMM showed no correlation.

Site / Period / Sampler / Configuration					Summary of Findings
LINDON, UT; 01/29/03 to 02/12/03					**Grover et al. (2005, 090044)**[66]
Sampler	**Flow Rate (L/Min)**	**Filter Type**[a]		**Denuder**[b]	RAMS required regular maintenance.
PC-BOSS $PM_{2.5}$	150	Teflon (W)		CIF	RAMS = (0.92 ± 0.03) FDMS-TEOM + (1.3 ± 3.9); R^2 = 0.69; n = 332
CONTINUOUS SAMPLER	**FLOW RATE (L/MIN)**	**INLET TEMPERATURE**	**DRYER**	**OTHER**	PC-BOSS constructed mass = (0.89 ± 0.21) FDMS-TEOM + (1.8 ± 2.8); R^2 = 0.66; n = 11
TEOM	16.7	30 °C	None	$PM_{2.5}$	TEOM @ 30 °C $PM_{2.5}$ was consistently lower than FDMS-TEOM and the difference was consistent with concentrations SVOCs and NH_4NO_3 measured by PC-BOSS.
FDMS-TEOM	16.7	30 °C	Nafion	$PM_{2.5}$	
RAMS	16.7	30 °C	Nafion	$PM_{2.5}$ Denuder used	
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.					**Lee et al. (2005, 128139)**[73]
Sampler	**Flow Rate (L/Min)**	**Filter Type**[a]		**Denuder**[b]	Radiance Research M903dryer = (0.78 ± 0.01) Radiance Research M903no dryer + (0.30 ± 0.03); R^2 = 0.95
Harvard Impactor $PM_{2.5}$	10	Teflon (N/A)		N/A	Radiance Research M903s vs. CAMM, R^2 = 0.78
Continuous Sampler	**Flow Rate (L/Min)**	**Inlet Temperature**	**Dryer**	**Other**	Radiance Research M903s vs. RAMS, R^2 = 0.63
SES-TEOM	16.7	35 °C	Nafion	$PM_{2.5}$	Radiance Research M903s vs. SES-TEOM, R^2 = 0.72
CAMM	0.3	N/A	Nafion	$PM_{2.5}$	CAMM = (0.60 ± 0.03) SES-TEOM + (2.0 ± 0.42); R^2 = 0.71; N = 185
RAMS	16.7	30 °C	Nafion	$PM_{2.5}$ TEA & CIF denuders With particle concentrator	RAMS = (0.71 ± 0.04) SES-TEOM + (2.51 ± 0.59); R^2 = 0.63; N = 185
Radiance Research M903	N/A	N/A	Nafion	bscat	RAMS = (0.93 ± 0.06) CAMM + (2.44 ± 0.68); R^2 = 0.55; N = 185
Radiance Research M903	N/A	N/A	None	bscat	Both RAMS and CAMM under-measured ambient $PM_{2.5.}$ CAMM = (0.70 ± 0.06) HI + (0.16 ± 0.96); R^2 = 0.87; N = 22 SES-TEOM = (1.0 ± 0.10) HI + (-0.68 ± 1.74); R^2 = 0.89; N = 15
BALTIMORE SUPERSITE, MD; 05/17/01 to 06/11/01. Located near a freeway and bus yard.					**Lee et al. (2005, 128139)**[73]
Sampler	**Flow Rate (L/Min)**	**Filter Type**		**Denuder**	Radiance Research M903dryed = (0.65 ± 0.02) Radiance Research M903no dryer + (1.80 ± 0.20); R^2 = 0.75, suggesting influence from particle-bound water.
RAAS-100 $PM_{2.5}$ FRM	16.7	Teflon		None	High correlation (R^2 = 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	$PM_{2.5}$	CAMM = (0.32 ± 0.07) SES-TEOM + (9.45 ± 1.61); R^2 = 0.14; N = 120
RAMS	16.7	30 °C	Nafion	$PM_{2.5}$ TEA & CIF denuders; No particle	RAMS = (0.82 ± 0.10) SES-TEOM + (6.41 ± 2.09); R^2 = 0.38; N = 120
Radiance Research M903	N/A	N/A	Nafion	bscat	RAMS = (0.71 ± 0.12) CAMM + (11.3 ± 2.23); R^2 = 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); R^2 = 0.60; N = 7 RAMS = (1.05 ± 0.12) RAAS-100 FRM + (4.80 ± 2.60); R^2 = 0.90; N = 11 SES-TEOM = (0.86 ± 0.10) RAAS-100 FRM + (2.96 ± 1.99); R^2 = 0.90; N = 10

Site / Period / Sampler / Configuration					Summary of Findings
SEATTLE, WA; 01/28/01 to 02/21/01 Urban area near major highway and interstate, 8 km southeast of downtown.					**Lee et al. (2005, 156680)**[108] Radiance Research M903dryed = 0.94 ± 0.00 Radiance Research M903no dryer; R^2 = 1.0. Correlation of Radiance Research M903 vs. SES-TEOM, R^2 = 0.80, while that of Radiance Research M903 with CAMM was R^2 = 0.84 and with RAMS was R^2 = 0.72. CAMM = (1.07 ± 0.05) RAMS + (1.03 ± 0.55); R^2 = 0.61 SES-TEOM = (0.95 ± 0.03) RAMS + (1.24 ± 0.38); R^2 = 0.72 SES-TEOM = (0.87 ± 0.03) CAMM + (0.55 ± 0.37); R^2 = 0.74 SES-TEOM likely lost semi-volatile organic matter. Continuous $PM_{2.5}$ samplers were similar to filter $PM_{2.5}$ sampler. Number of samples was small (~7). 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.
SAMPLER	**FLOW RATE (L/MIN)**	**FILTER TYPE**[a]		**DENUDER**[b]	
MASS $PM_{2.5}$	16.7	Teflon (N/A)		Na_2CO_3	
Continuous Sampler	**Flow Rate (L/Min)**	**Inlet Temperature**	**Dryer**	**Other**	
SES-TEOM	16.7	30 °C	Nafion	$PM_{2.5}$	
CAMM	0.3	Ambient	Nafion	$PM_{2.5}$	
RAMS	16.7	30 °C	Nafion	$PM_{2.5}$ TEA & CIF denuders	
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. (2006, 098449)**[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. Urban site: BAM = (1.02 ± 0.02) FDMS-TEOM + 1.72; R^2 = 0.93; n = 244 FDMS-TEOM = (1.25 ± 0.02) FRM – (0.63 ± 0.26); R^2 = 0.95; n = 238 BAM = (1.28 ± 0.03) FRM + (1.27 ± 0.38); R^2 = 0.88; n = 320 Rural site: FDMS-TEOM = (1.09 ± 0.02) FRM – (0.004 ± 0.18); R^2 = 0.95; n = 349 $PM_{2.5}$ 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.
Sampler	**Flow Rate (L/Min)**	**Filter Type**[a]		**Denuder**[b]	
R&P-2025 $PM_{2.5}$ FRM	16.7	Teflon (N/A)		None	
R&P-2300 $PM_{2.5}$	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	$PM_{2.5}$	
BAM	16.7	"smart" heater on @ RH >44%		$PM_{2.5}$	

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; $NaHCO_3$: 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 µg/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

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Source: Chow et al. (2008, 156355)

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 (2001, 017092; 2004, 155898)** 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.
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 µg/m^3 to 30 µg/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_4NO_3. 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%.

Source: Chow et al. (2008, 156355)

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 Four 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.(2003, 156994)**[17]
Sampler	**Flow Rate (L/Min)**	**Filter Type**[a]	**Denuder**[b]	$PM_{2.5}$ 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.
R&P-2000 FRM	16.7	Quartz (P)	None	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).
RAAS-400	24	Nylon (P)	MgO	
SASS	6.7	Nylon (P)	MgO	A small positive artifact (few tenths of $\mu g/m^3$) might be present when using Na_2
MASS-400	16.7	Teflon (P)-Nylon (P	Na_2CO_3	
MASS-450	16.7	Quartz (P)	None	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.
R&P-2300	10	Nylon (P)	Na_2CO_3	
VAPS	15	Polycarbonatec (front & back-up)	Na_2CO_3	$PM_{2.5}$ NO_3- R&P-2000 FRM and MOUDI-100 samplers are consistently lower than other samplers.
URG-PCM	16.7	Teflon (P)-Cellulose-fiber (W	Na_2CO_3	**Weber et al. (2003, 157129)**[82]
ARA-PCM	16.7	Teflon (N/A)-Nylon (N/A)	Na_2CO_3/Citric acid	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.
PC-BOSS (TVA)	105	Teflon (W)-Nylon (P)	CIF	
PC-BOSS (BYU)	150	Teflon (W)-Nylon (P)	CIF	Except for ARA-N, good correlations (R^2 = 0.70 to 0.90) were found during the 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.
PC-BOSS (BYU)	150	Quartz (P)-CIF (S)	CIF	
MOUDI-100	30	Teflon (N/A-Quartz (N/A	None	
Continuous Sampler	**Flow Rate (L/Min)**	**Denuder**	**Analysis Method**[b]	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 (~0.1 $\mu g/m^3$, except for ARA-N, which had 0.5 $\mu g/m^3$).
ADI-N	1	Activated Carbon	NO_X Chemiluminescence	
ARA-N	3	Potassium iodide (KI) and dual sodium chlorite ($NaClO_2$)	NO_X Chemiluminescence	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).
PILS-IC	5	Two URG annular glass denuders in series containing citric acid and $CaCO_3$	IC	
ECN	16.7	Rotating annular wet denuder system	IC	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.
TT	5	Wet parallel plate denuder	IC	

SITE / PERIOD / SAMPLER / CONFIGURATION				SUMMARY OF FINDINGS
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. (2004, 148859)[18]; Takahama et al. (2004, 157038)[116]**
Sampler	**Flow Rate (L/Min)**	**Filter Type[a]**	**Denuder[b]**	More than 70% (~0.5 µg/m^3) of NO_3 mass was lost from MOUDI samplers during summer.
MOUDI-110	30	Teflon (W) Teflon (W)	None	MOUDI NO_3 = 0.27 CMU; R^2 = 0.40; Summer MOUDI NO_3 = 0.99 CMU; R^2 = 0.49; winter
CMU	16.7	Nylon (W)	MgO/Citric acid	**Wittig et al. (2004, 103413)[85]**
R&P-2000 FRM	16.7	Teflon (W)	None	Avg conversion efficiency to NO_X (tested using NH_4NO_3 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 µg/m^3; R^2 = 0.84
				Underestimation 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.
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. (2005, 099030)[87]**
Sampler	**Flow Rate (L/Min)**	**Filter Type[a]**	**Denuder[b]**	Maximum NO_3- volatilization was observed during summer (Jun-Aug), while the lowest volatilization was observed during winter (Dec-Feb).
DRI-SFS	113	Quartz (Pellulose	Al_2O_3	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.
RAAS-400	24	Quartz (P)-Nylon (P)	Na_2CO_3	
RAAS-400	24	Quartz (P)-Quartz (P)	None	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.
RAAS-100 FRM	16.7	Quartz (P)	None	
Continuous Sampler	**Flow Rate (L/Min)**	**Denuder**	**Analysis Method[b]**	Front-quartz non-volatilized NO_3- concentrations were similar for DRISFS (0.52 ± 0.26 µg/m^3) and RAAS-100 FRM (0.81 ± 0.33 µg/m^3) for warm months (May-Sep). With preceding denuders, the DRI-SFS PNO_3 concentration (3 ± 1.9 µg/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.
R&P-8400N	5	Activated Carbon	NO_X Chemiluminescence	
Sampler	**Flow Rate (L/Min)**	**Filter Type[a]**	**Denuder[b]**	**Chow et al. (2005, 156348)[117]**
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	
Dionex-IC	5	Parallel plate wet denuder	IC	

SITE / PERIOD / SAMPLER / CONFIGURATION	SUMMARY OF FINDINGS
	High correlation (R^2 >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. (2006, 138080)[65]
	Dionex-IC NO_3 = (0.71 ± 0.04) PC-BOSS NO_3 + (3.2 ± 1.1); R^2 = 0.91; n = 29
	R&P-8400N = (1.10 ± 0.06) PC-BOSS NO_3 - (0.8 ± 1.8); R^2 = 0.93; n = 29
	R&P-8400N = (0.55 ± 0.01) Dionex-IC + (1.4 ± 1.8); R^2 = 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.

SITE / PERIOD / SAMPLER / CONFIGURATION	SUMMARY OF FINDINGS
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. **Sampler** / **Flow Rate (L/Min)** / **Filter Type**[a] / **Denuder**[b] SASS / 6.7 / Nylon (N/A) / MgO **Continuous Sampler** / **Flow Rate (L/Min)** / **Denuder** / **Analysis Method**[b] R&P-8400N / 5 / Activated Carbon / NO_X Chemiluminescence	**Harrison et al. (2004, 136787)**[83] Corrections were made to R&P-8400N data for software calculation error, conversion efficiency, gas analyzer efficiency, vacuum drift and sample flow drift. The relative uncertainty of R&P-8400N measurements averaged 8.7%, ranging from 6.3% to 23%. Data capture >95%. R&P-8400N underestimated SASS filter NO_3^- by ~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 ambientRH 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**[b] 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. (2004, 099003)**[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 NO_3 concentration. At the rural site, AMS had a slope of 0.51 and R^2 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 Type**[a] / **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. (2006, 115897)**[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 NO_3 over all concentration ranges, except at <1 µg/m^3. Urban: R&P-8400N = 0.59 R&P-2300 NO_3 + 0.28; R^2 = 0.88; n = 305 Rural: R&P-8400N = 0.73 R&P-2300 NO_3 + 0.01; R^2 = 0.90; n~161; however concentrations were low with 95% of data < 1 µg/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 Type**[a] / **Denuder**[b]	**Fine et al. (2003, 155775)**[19] MOUDI = 0.68 HEADS; R^2 = 0.88 ADI-N Sized = 0.80 HEADS; R^2 = 0.79

SITE / PERIOD / SAMPLER / CONFIGURATION				SUMMARY OF FINDINGS
MOUDI	30	Teflon (P)	None	ADI-N Sized = 1.12 MOUDI; R^2 = 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. 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. 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 (R^2 = 0.67) for the 1-2 µm size range NO_3 relative to other size ranges (R^2 < = 0.56). This is possibly due to NO_3^- in the form of non-volatilized sodium nitrate ($NaNO_3$) than volatilized NH_4NO_3 in the 1-2 µm size range. Single particle analysis also indicated this possibility of $NaNO_3$ in the 1-2 µm range.
HEADS	10	Teflon (N/A) -GF--GF	Carbonate	
Continuous Sampler	**Flow Rate (L/Min)**	**Denuder**	**Analysis Method**[b]	
ADI-N Sized	0.9	Activated Carbon	NO_X Chemiluminescence	
RUBIDOUX, CA; 07/01/03 to 07/31/03 Located in the eastern section of SoCAB in the northwest 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. (2005, 090044)**[66] R&P-8400N = (0.65 ± 0.07) PC-BOSS + (3.3 ± 2.4); R^2 = 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_4NO_3 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 (R^2 ≥ 0.89). At the rural site, R^2 ranged from 0.61 to 0.83, except for the AMS versus R&P2300 comparison, with an R^2 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	NO_X Chemiluminescence	
PILS-IC	5	Na_2CO_3 and Citric acid	IC	
AMS	0.1	None	Mass Spectrometry	

SITE / PERIOD / SAMPLER / CONFIGURATION	SUMMARY OF FINDINGS

[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_2O_3: Aluminum oxide; GF: Na_2CO_3 impregnated Glass Fiber Filters; IC: Ion chromatography; MgO: Magnesium oxide; Na_2CO_3: Sodium carbonate; $NaHCO_3$: 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.

[1]Chow (1995, 077012); [2]Watson and Chow (2001, 157123); [3] Watson et al. (1983, 045084); [4]Fehsenfeld et al. (2004, 157360); [5]Solomon et al. (2001, 157193); [6]Watson et al. (2005, 157124); [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]Fitz 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. (2000, 012225); [36]Fine et al. (2004, 141283); [37]Yue et al. (2004, 157169); [38]Rinehart et al. (2006, 115184); [39]Wan and Yu (2006, 157104); [40]Poore (2000, 012839); [41]Fraser et al. (2003, 040266); [42]Engling et al. (2006, 156422); [43]Yu et al. (2005, 157167); [44]Tran et al. (2000, 013025); [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, 046318); [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, 157209); [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 (2002, 051444); [62]Butler et al. (2003, 156313); [63]Chow et al. (2006, 146622); [64]Russell et al. (2004, 082453); [65]Grover et al. (2006, 138080); [66]Grover et al. (2005, 090044); [67]Schwab et al. (2006, 098449); [68]Hauck et al. (2004, 156525); [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. (2005, 155925); [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, 136787); [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. (2006, 098785); [90]Lim et al. (2003, 037037); [91]Watson and Chow (2002, 037873); [92]Venkatachari et al. (2006, 105918); [93]Bae et al. (2004, 156243); [94]Arhami et al. (2006, 156224); [95]Park et al. (2005, 156843); [96]Bae et al. (2004, 098680); [97]Chow et al. (2006, 156350); [98]Arnott et al. (2005, 156227); [99]Bond et al. (1999, 156281); [100]Virkkula et al. (2005, 157097); [101]Petzold et al. (2002, 156863); [102]Park et al. (2006, 098104); [103]Arnott et al. (1999, 020650); [104]Peters et al. (2001, 016925); [105]Pitchford et al. (1997, 156872); [106]Rees et al. (2004, 097164); [107]Watson et al. (2000, 010354); [108]Lee et al. (2005, 156680); [109]Hering et al. (2004, 155837); [110]Watson et al. (1998, 198805); [111]Chakrabarti et al. (2004, 157426); [112]Mathai et al. (1990, 156741); [113]Kidwell and Ondov (2001, 017092); [114]Stanier et al. (2004, 095955); [115]Khlystov et al. (2005, 156635); [116]Takahama et al. (2004, 157038); [117]Chow et al. (2005, 156348); [118]Zhang et al. (2002, 157181); [119]Subramanian et al. (2004, 081203); [120]Chow et al. (2006, 155207); [121]Birch and Cary (1996, 026004); [122]Birch (1998, 024953); [123]Birch and Cary (1996, 002352); [124]NIOSH (1996, 156810); [125]NIOSH (1999, 156811); [126]Chow et al. (1993, 077459); [127]Chow et al. (2007, 156354); [128]Ellis and Novakov (1982, 156416); [129]Peterson and Richards (2002, 156861); [130]Schauer et al. (2003, 037014); [131]Middlebrook et al. (2003, 042932); [132]Wenzel et al. (2003, 157139); [133]Jimenez et al. (2003, 156611); [134]Phares et al. (2003, 156866); [135]Qin and Prather (2006, 156895); [136]Zhang et al. (2005, 157185); [137]Bein et al. (2005, 156265); [138]Drewnick et al. (2004, 155754); [139]Drewnick et al. (2004, 155755); [140]Lake et al. (2003, 156669); [141]Lake et al. (2004, 088411)

Source: Chow et al. (2008, 156355)

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 Four 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. (2003, 156994)[17]**
Sampler	**Flow Rate (L/Min)**	**Filter Type[a]**	**Denuder[b]**	$PM_{2.5}$ 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.
R&P-2000 FRM	16.7	Quartz (P)	None	
RAAS-400	24	Teflon (P)	None	
SASS	6.7	Teflon (P)	None	While avg mass was within 10%, daily variability was >50% of filter RR.
MASS-450	16.7	Quartz (P)	None	
R&P-2300	10	Quartz (P)	None	All samplers, except for the PC-BOSS (TVA), correlated well (R^2 >0.90) with daily filter RR.
VAPS	15	Quartz (P)	XAD-4	PC-BOSS (TVA) had instrument leaks.
URG-PCM	16.7	Teflon (P)-Cellulose-fiber (W)		The R&P-2000 FRM, on avg, agreed within 1% of filter RR.
ARA-PCM	16.7	Teflon (N/A)	Na_2CO_3/Citric acid	MOUDI-100 was ~13% low compared to filter RR.
ARA-PCM	16.7	Nylon (N/A)	Na_2CO_3/Citric acid	**Weber et al. (2003, 157129)[82]; Zhang et al. (2002, 157181)[118]**
PC-BOSS (TVA)	105	Teflon (W)	CIF	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 µg/m^3.
PC-BOSS (TVA)	105	Quartz (P)	CIF	
PC-BOSS (BYU)	150	Teflon (W)	CIF	Good correlations (R^2 = 0.76 to 0.94) were found during the second half of the study, except for TT versus ADI.
PC-BOSS (BYU)	150	Quartz (P)	CIF	
MOUDI-100	30	Teflon (N/A) Quartz (N/A)	None	
Continuous Sampler	**Flow Rate (L/Min)**	**Denuder**	**Analysis Method[b]**	Good correlation (R^2 = 0.84) was found between continuous and filter-based SO_4^{2-}
ADI-S	2.7	Activated Carbon	SO_2, UV Fluorescence	Continuous RR = (1.15 ± 0.15), Filter RR + (0.41 ± 1.73)
PILS-IC	5	Two URG annular glass denuders in series containing citric acid & $CaCO_3$	IC	Variability among continuous SO_4^{2-} instruments (RSD = 13%) was similar to that for NO_3^- instruments. Filter sample variability was low (RSD = 8%) indicating more uniformity among samplers.
ECN	16.7	Rotating annular wet denuder system	IC	
TT	5	Wet parallel plate denuder	IC	The ECN and TT instruments were within 15%, PILS-IC was within 20% and ADI-S was within 26% of filter RR.
PITTSBURGH SUPERSITE, PA; 070/1/01 to 08/01/02 6 km east of downtown in a park on the top of a hill				**Cabada et al. (2004, 148859)[18]; Takahama et al. (2004, 157038)[116]**
Sampler	**Flow Rate (L/Min)**	**Filter Type[a]**	**Denuder[b]**	MOUDI SO_4^{2-} 0.80 CMU; R^2 = 0.95; Summer
MOUDI-110	30	Teflon (W)	None	MOUDI SO_4^{2-} 0.97 CMU; R^2 = 0.48; winter
CMU	16.7	Teflon (W)	MgO/Citric acid	**Wittig et al. (2004, 103413)[85]**

SITE/PERIOD/SAMPLER/ CONFIGURATION				SUMMARY OF FINDINGS
R&P-2000 FRM	16.7	Teflon (W)	None	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.
Continuous Sampler	**Flow Rate (L/Min)**	**Denuder**	**Analysis Method[b]**	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.
R&P-8400S	5	Activated Carbon	SO_2 UV Fluorescence	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 µg/m^3; R^2 = 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.				**Fine et al. (2003, 155775)[19]** MOUDI explained 85% of HEADS SO_4^{2-} (R^2 = 0.89; n = 40)
Sampler	**Flow Rate (L/Min)**	**Filter Type[a]**	**Denuder[b]**	
MOUDI	30	Teflon (P)	None	
HEADS	10	Teflon (N/A) GF-GF[c]	Carbonate	
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, 60m above sea level, in a clearing surrounded by deciduous and evergreen trees and no major cities within 20 km of the site.				**Drewnick et al. (2003, 099160)[21]; Hogrefe et al. (2004, 099003)[20]** Data completeness: 89-93% for R&P-8400S, 94-98% for AMS, 81-98% for CASM, and 65-70% for PILS-IC.
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	
TEOM-ACCU	16.7	Zefluor (N/A)	None	
Continuous Sampler	**Flow Rate (L/Min)**	**Denuder**	**Analysis Method[b]**	
R&P-8400S	5	Activated Carbon	SO_2 UV Fluorescence	
PILS-IC	5	Na_2CO_3 and Citric acid	IC	
AMS	0.1	None	Mass Spectrometry	
CASM	5	Na_2CO_3 and Carbon and a Nafion dryer	SO_2 UV Fluorescence	The urban site data showed good correlations (R^2 = 0.87 to 0.94) with slopes ranging from 0.97 to 1.01. At the rural site, the variability was large (R^2 = 0.73 to 0.91) with slopes ranging from 0.76 to 1.32. SO_4 from PILS-IC was overestimated by ~25% when compared to the AMS at the rural site.
				Filter samples were within 5% of each other, except for comparison of ACCU with R&P-2300 at the rural site, with high correlations (R^2 = 0.97 to 1.0). ACCU underestimated SO_4^{2-} by ~15%.
				Continuous versus 6-h SCS filter comparisons showed high R^2 (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)
				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.
				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 (~

SITE/PERIOD/SAMPLER/ CONFIGURATION	SUMMARY OF FINDINGS
	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. (2006, 115897)**[84] Data capture was above 85%. Data loss was primarily due to frequent flash strip failures, every 2 wk and without warning. Data were adjusted for span and zero drifts, measured conversion efficiency, flow drift, and blanks. 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. Urban South Bronx site: R&P-8400S = 0.82 TEOM-ACCU + 1.15; R^2 = 0.84; n = 513 R&P-8400S = 0.74 R&P-2300 + 1.14; R^2 = 0.81; n = 322 Rural Whiteface mountain: R&P-8400S = 0.75 TEOM-ACCU + 0.22; R^2 = 0.95; n = 207 R&P-8400S = 0.78 R&P-2300 + 0.17; R^2 = 0.85; n = 198 Required weekly or biweekly maintenance by trained personnel **Schwab et al. (2006, 098785)**[89] TE-5020 = 0.78 ACCU – 0.2; R^2 = 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.

Integrated Sampler	Flow Rate (L/Min)	Filter Type[a]	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-8400S	5	Activated Carbon	SO_2 pulsed fluorescence
TE-5020 (07/14/04 to 11/01/04)	5	Na_2CO_3	SO_2 pulsed fluorescence

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 Typea Denuderb	**Grover et al. (2006, 138080)**[65] Dionex-IC SO_4^{2-} (1.03 ± 0.03) PC-BOSS SO_4 + (0.2 ± 0.3); R^2 = 0.98; n = 27 R&P-8400S SO_4^{2-} (0.95 ± 0.05) Dionex-IC SO_4 + (0.3 ± 0.6); R^2 = 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

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.
[b]Al_2O_3: Aluminum oxide; IC: Ion chromatography; CIF: Charcoal Impregnated 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]Na_2CO_3 impregnated.
[d]37 mm filter.

[1]Chow (1995, 077012); [2]Watson and Chow (2001, 157123); [3] Watson et al. (1983, 045084); [4]Fehsenfeld et al. (2004, 157360); [5]Solomon et al. (2001, 157193); [6]Watson et al. (2005, 157124); [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]Fitz 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. (2000, 012225); [36]Fine et al. (2004, 141283); [37]Yue et al. (2004, 157169); [38]Rinehart et al. (2006, 115184); [39]Wan and Yu (2006, 157104); [40]Poore (2000, 012839); [41]Fraser et al. (2003, 040266); [42]Engling et al. (2006, 156422); [43]Yu et al. (2005, 157167); [44]Tran et al. (2000, 013025); [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, 046318); [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, 157209); [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 (2002, 051444); [62]Butler et al. (2003, 156313); [63]Chow et al. (2006, 146622); [64]Russell et al. (2004, 082453); [65]Grover et al. (2006, 138080); [66]Grover et al. (2005, 090044); [67]Schwab et al. (2006, 098449); [68]Hauck et al. (2004, 156525); [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. (2005, 155925); [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, 136787); [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. (2006, 098785); [90]Lim et al. (2003, 037037); [91]Watson and Chow (2002, 037873); [92]Venkatachari et al. (2006, 105918); [93]Bae et al. (2004, 156243); [94]Arhami et al. (2006, 156224); [95]Park et al. (2005, 156843); [96]Bae et al. (2004, 098680); [97]Chow et al. (2006, 156350); [98]Arnott et al. (2005, 156227); [99]Bond et al. (1999, 156281); [100]Virkkula et al. (2005, 157097); [101]Petzold et al. (2002, 156863); [102]Park et al. (2006, 098104); [103]Arnott et al. (1999, 020650); [104]Peters et al. (2001, 016925); [105]Pitchford et al. (1997, 156872); [106]Rees et al. (2004, 097164); [107]Watson et al. (2000, 010354); [108]Lee et al. (2005, 156680); [109]Hering et al. (2004, 155837); [110]Watson et al. (1998, 198805); [111]Chakrabarti et al. (2004, 157426); [112]Mathai et al. (1990, 156741); [113]Kidwell and Ondov (2001, 017092); [114]Stanier et al. (2004, 095955); [115]Khlystov et al. (2005, 156635); [116]Takahama et al. (2004, 157038); [117]Chow et al. (2005, 156348); [118]Zhang et al. (2002, 157181); [119]Subramanian et al. (2004, 081203); [120]Chow et al. (2006, 155207); [121]Birch and Cary (1996, 026004); [122]Birch (1998, 024953); [123]Birch and Cary (1996, 002352); [124]NIOSH (1996, 156810); [125]NIOSH (1999, 156811); [126]Chow et al. (1993, 077459); [127]Chow et al. (2007, 156354); [128]Ellis and Novakov (1982, 156416); [129]Peterson and Richards (2002, 156861); [130]Schauer et al. (2003, 037014); [13]1Middlebrook et al. (2003, 042932); [132]Wenzel et al. (2003, 157139); [133]Jimenez et al. (2003, 156611); [134]Phares et al. (2003, 156866); [135]Qin and Prather (2006, 156895); [136]Zhang et al. (2005, 157185); [137]Bein et al. (2005, 156265); [138]Drewnick et al. (2004, 155754); [139]Drewnick et al. (2004, 155755); [140]Lake et al. (2003, 156669); [141]Lake et al. (2004, 088411)

Source: Chow et al. (2008, 156355)

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 Four 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. (2003, 156994)[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. OC positive artifacts ranged from 2 to 4 µg/m^3 **EC:** $PM_{2.5}$ 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. 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. Major difference in EC is due to the carbon analysis protocol and optical monitoring correction (i.e., transmittance, reflectance). **Lim et al. (2003, 037037)[90]**
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	
R&P-2300	10	Quartz (P)-Quartz (P)	None		NIOSH 5040-TOT	
VAPS	15	Quartz (P)	XAD-4		NIOSH 5040-TOT	
URG-PCM	16.7	Quartz (P)-Quartz (P)	XAD-4		Front: NIOSH 5040-TOT; Backup: custom-TOTd	
ARA-PCM	16.7	Quartz (N/A)-Quartz (N/A)	CIF		IMPROVE_TOR	
PC-BOSS (TVA)	150	Quartz (P)-CIF (N/A)	CIF		Front: IMPROVE_TOR; Backup: TPV	
PC-BOSS (BYU)	150	Quartz (P)-CIF (S)	CIF		TPB	
MOUDI-100	30	Al Foil-Quartz (N/A)[f]	None		Custom-TOR to suit Al[e]	
Continuous Sampler	**Flow Rate (L/Min)**	**Denuder**	**OC**	**EC**	**Comments**	

SITE/PERIOD/SAMPLER/ CONFIGURATION						SUMMARY OF FINDINGS
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	TC concentrations measured by the RU-OGI and R&P-5400 correlated reasonably well (R^2 = 0.83), with a slope of 0.96. The ratio of the mean RU-OGI to mean R&P-5400 TC was 1.02.
RU-OGI	16.1	None	700 in He	850 in 2% O_2	TOT; Dynamic blank for adsorption correction	R&P-5400 OC was 8% lower than the RU-OGI (R^2 = 0.73), while the R&P-5400 EC was 20% higher than RU-OGI (R^2 = 0.74).
R&P-5400	16.7	None	275 in air	750 in air	No pyrolysis correction	OC measured by ADI-C was lower than R&P-5400 and RUOGI by 15% and 22%, respectively.
PSAP	1.26	None		b_{abs}@ 565 nm	10m^2/g factor	EC from PSAP and AE-16 correlated well (R^2 = 0.97). PSAP was lower by ~50%, compared with AE-16, R&P-5400 and RU-OGI.
AE-16	4	None		b_{abs}@ 880 nm	12.6 m^2/g factor	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).

SITE/PERIOD/SAMPLER/ CONFIGURATION						SUMMARY OF FINDINGS
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. (2004, 081203)**[119] 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. 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- to 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 μg/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.
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	
	16.7	Non-denuded blank (UDB)	Teflon (P/W)-Quartz (P)-CIG (S)	None	NIOSH 5040-TOT	
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.						**Bae et al. (2004, 156243)**[93]**; Bae et al. (2004, 098680)**[96] Denuder breakthrough was 0.17 ± 0.15 μg/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 summertime OC artifact, on an absolute basis.
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**	

SITE/PERIOD/SAMPLER/ CONFIGURATION						SUMMARY OF FINDINGS
Sunset OCEC	8	CIF	340, 500, 615, 870°C in 100% He	550, 625, 700, 775, 850, 900 °C in 2% O2, 98% He	ACE Asia TOT; CH4 FID detector	Comparison of continuous Sunset TC and OC with 24-h filter samples showed good correlations (R^2) 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 (R^2 = 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 (P) (QBT)	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
RAAS-100 FRM	16.7	Quartz (P)	None	IMPROVE_TOR

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 (Lmin)	Filter Type/Pack[a]	Denuder[b]	Analysis Method[c]

Watson and Chow (2002, 037873)[91]; Chow et al. (2005, 156348)[117]; Chow et al. (2006, 155207)[120]; Watson et al. (2005, 157124)[6]; Park et al. (2006, 098104)[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 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 (R^2 < 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 (babs) from DRI-PA (1047 nm), MAAP (670 nm), and AE

SITE/PERIOD/SAMPLER/ CONFIGURATION						SUMMARY OF FINDINGS
PC-BOSS	150	Quartz (P)-CIG (S)†	CIF		TPV	(880 nm) analyzers with the filter IMPROVE_TOR EC, gave a σ_{abs} 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.
Continuous Sampler	**Flow Rate (L/Min)**	**Denuder**	**OC**	**EC**	**Comments**	
R&P-5400	16.7	None	375°C in air	750°C in air	No pyrolysis	**Grover et al. (2006, 138080)**[65]
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	R&P-5400 TC = (0.50 ± 0.01) Sunset TC + (3.6 ± 1.5); R^2 = 0.73; n = 480 Sunset TC = (0.63 ± 0.05) PC-BOSS TC + (4.1 ± 3.2); R^2 = 0.86; n = 29 R&P-5400 TC = (0.41 ± 0.02) PC-BOSS TC + (6.7 ± 1.6); R^2 = 0.91; n = 29

SITE/PERIOD/SAMPLER/ CONFIGURATION	SUMMARY OF FINDINGS

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.

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**
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

Park et al. (2005, 156843)[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 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 northwest 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 N2)
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 meas-ured	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. (2005, 090044)[66]

Sunset OCEC TC = (0.90 ± 0.06) PC-BOSS + (2.0 ± 2.1); R^2 = 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 λ m^2/g factor, where λ is in nm
AMS	N/A	None	N/A	N/A	~ 1 µm cut-point

Venkatachari et al. (2006, 105918)[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; R^2 = 0.86; n = 506

Sunset Optical EC = 0.62 ± 0.05 AE-20 BC; R^2 = 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); R^2 = 0.67; n = 16

Sunset OC = (0.98 ± 0.11) R&P-5400 OC - (0.47 ± 0.17); R^2 = 0.44; n = 327

SITE/PERIOD/SAMPLER/ CONFIGURATION	SUMMARY OF FINDINGS
	R&P-5400 OC = (0.60 ± 0.47) R&P-2300 OC + (0.58 ± 0.82); R^2 = 0.58; n = 17 Organic matter measurements by AMS showed reasonable correlation (R^2 = 0.76) with filter (R&P-2300) OC, while being poorly correlated with continuous OC by Sunset (R^2 = 0.32) and R&P-5400 (R^2 = 0.36) Sunset EC = (1.21 ± 0.44) R&P-2300 EC – (0.03 ± 0.13); R^2 = 0.94; n = 16 Sunset EC = (1.35 ± 0.12) R&P-5400 EC + (0.06 ± 0.04); R^2 = 0.61; n = 327 R&P-5400 EC = (0.49 ± 0.46) R&P-2300 EC + (0.09 ± 0.26); R^2 = 0.77; n = 15 Sunset TC = (0.86 ± 0.39) R&P-2300 TC – (0.06 ± 0.69); R^2 = 0.77; n = 16 Sunset TC = (1.31 ± 0.10) R&P-5400 TC – (1.15 ± 0.15); R^2 = 0.59; n = 327 R&P-5400 TC = (0.77 ± 0.58) R&P-2300 TC + (0.35 ± 1.37); R^2 = 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% O2 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 N2. For Quartz filters: Heated from 50 °C to 800 °C at a ramp rate of 28 °C/min in 70% N2 and 30% O2; 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

[1]Chow (1995, 077012); [2]Watson and Chow (2001, 157123); [3] Watson et al. (1983, 045084); [4]Fehsenfeld et al. (2004, 157360); [5]Solomon et al. (2001, 157193); [6]Watson et al. (2005, 157124); [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]Fitz 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. (2000, 012225); [36]Fine et al. (2004, 141283); [37]Yue et al. (2004, 157169); [38]Rinehart et al. (2006, 115184); [39]Wan and Yu (2006, 157104); [40]Poore (2000, 012839); [41]Fraser et al. (2003, 040266); [42]Engling et al. (2006, 156422); [43]Yu et al. (2005, 157167); [44]Tran et al. (2000, 013025); [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, 046318); [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, 157209); [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 (2002, 051444); [62]Butler et al. (2003, 156313); [63]Chow et al. (2006, 146622); [64]Russell et al. (2004, 082453); [65]Grover et al. (2006, 138080); [66]Grover et al. (2005, 090044); [67]Schwab et al. (2006, 098449); [68]Hauck et al. (2004, 156525); [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. (2005, 155925); [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, 136787); [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. (2006, 098785); [90]Lim et al. (2003, 037037); [91]Watson and Chow (2002, 037873); [92]Venkatachari et al. (2006, 105918); [93]Bae et al. (2004, 156243); [94]Arhami et al. (2006, 156224); [95]Park et al. (2005, 156843); [96]Bae et al. (2004, 098680); [97]Chow et al. (2006, 156350); [98]Arnott et al. (2005, 156227); [99]Bond et al. (1999, 156281); [100]Virkkula et al. (2005, 157097); [101]Petzold et al. (2002, 156863); [102]Park et al. (2006, 098104); [103]Arnott et al. (1999, 020650); [104]Peters et al. (2001, 016925); [105]Pitchford et al. (1997, 156872); [106]Rees et al. (2004, 097164); [107]Watson et al. (2000, 010354); [108]Lee et al. (2005, 156680); [109]Hering et al. (2004, 155837); [110]Watson et al. (1998, 198805); [111]Chakrabarti et al. (2004, 157426); [112]Mathai et al. (1990, 156741); [113]Kidwell and Ondov (2001, 017092); [114]Stanier et al. (2004, 095955); [115]Khlystov et al. (2005, 156635); [116]Takahama et al. (2004, 157038); [117]Chow et al. (2005, 156348); [118]Zhang et al. (2002, 157181); [119]Subramanian et al. (2004, 081203); [120]Chow et al. (2006, 155207); [121]Birch and Cary (1996, 026004); [122]Birch (1998, 024953); [123]Birch and Cary (1996, 002352); [124]NIOSH (1996, 156810); [125]NIOSH (1999, 156811); [126]Chow et al. (1993, 077459); [127]Chow et al. (2007, 156354); [128]Ellis and Novakov (1982, 156416); [129]Peterson and Richards (2002, 156861); [130]Schauer et al. (2003, 037014); [131]Middlebrook et al. (2003, 042932); [132]Wenzel et al. (2003, 157139); [133]Jimenez et al. (2003, 156611); [134]Phares et al. (2003, 156866); [135]Qin and Prather (2006, 156895); [136]Zhang et al. (2005, 157185); [137]Bein et al. (2005, 156265); [138]Drewnick et al. (2004, 155754); [139]Drewnick et al. (2004, 155755); [140]Lake et al. (2003, 156669); [141]Lake et al. (2004, 088411)

Source: Chow et al. (2008, 156355)

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

ATLANTA SUPERSITE, GA: 08/03/99 to 09/01/99
Four km NW of downtown, within 200 m of a bus maintenance yard and several warehouse facilities, representative of a mixed commercial-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
PALMS	N/A $PM_{2.5}$ cyclone Nafion (17 days) / None (4 days) 0.35-2.5 Light scattering	LDI, ArF 193 nm $2x10^9$ to $5x10^9$ W/cm^2	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 0.2-2.5 Aerosol TOF	LDI, Nd: YAG 266 nm laser ~ $1x10^8$ W/cm^2	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 $1x10^8$ to $2x10^8$ W/cm^2	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. (2003, 042932)[131]; Wenzel et al. (2003, 157139)[132]; Jimenez et al. (2003, 156611)[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 (R^2 = 0.93) than PALMS (R^2 = 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 (R^2 = 0.79), and the data uniformly scattered around a 1:1 line. NO_3 comparison was poor (R^2 = 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.

ATLANTA SUPERSITE, GA: 08/03/99 to 09/01/99
Four km NW of downtown, within 200 m of a bus maintenance yard and several warehouse facilities, representative of a mixed commercial-residential neighborhood.

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. (2003, 156866)[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 β attenuation monitor $PM_{2.5}$ mass concentration

Qin and Prather (2008, 156985)[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]	Hit Rates[b]	Mass Spectrometer[c]
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

ATLANTA SUPERSITE, GA: 08/03/99 to 09/01/99
Four km NW of downtown, within 200 m of a bus maintenance yard and several warehouse facilities, representative of a mixed commercial-residential neighborhood.

Zhang et al. (2005, 157185)[136]; Bein et al. (2005, 156265)[137]

The AMS observed 75% of the SO_4^{2-} measured by R&P-8400S (R^2 = 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 (R^2 = 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]	Hit Rates[b]	Mass Spectrometer[c]
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

Drewnick et al. (2004, 155754)[138]; Drewnick et al. (2004, 155755)[139]; Hogrefe et al. (2004, 099003)[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_4NO_3 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 R^2 of 0.46.

AMS SO_4 showed good agreement with R&P-8400S at both the rural and urban locations (R^2 = 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 (R^2 = 0.91) with near zero intercept (0.22 µg/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 (5-10%).

ATLANTA SUPERSITE, GA: 08/03/99 to 09/01/99
Four km NW of downtown, within 200 m of a bus maintenance yard and several warehouse facilities, representative of a mixed commercial-residential neighborhood.

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]	Hit Rates[b]	Mass Spectrometer[c]
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. (2003, 156669)[140], Lake et al. (2004, 088411)[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.

aEI: Electon Impact; LDI: Laser Desorption / Ionization
bHit 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
cTOF: Time fo Flight

[1]Chow (1995, 077012); [2]Watson and Chow (2001, 157123); [3] Watson et al. (1983, 045084); [4]Fehsenfeld et al. (2004, 157360); [5]Solomon et al. (2001, 157193); [6]Watson et al. (2005, 157124); [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]Fitz 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. (2000, 012225); [36]Fine et al. (2004, 141283); [37]Yue et al. (2004, 157169); [38]Rinehart et al. (2006, 115184); [39]Wan and Yu (2006, 157104); [40]Poore (2000, 012839); [41]Fraser et al. (2003, 040266); [42]Engling et al. (2006, 156422); [43]Yu et al. (2005, 157167); [44]Tran et al. (2000, 013025); [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, 046318); [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, 157209); [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 (2002, 051444); [62]Butler et al. (2003, 156313); [63]Chow et al. (2006, 146622); [64]Russell et al. (2004, 082453); [65]Grover et al. (2006, 138080); [66]Grover et al. (2005, 090044); [67]Schwab et al. (2006, 098449); [68]Hauck et al. (2004, 156525); [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. (2005, 155925); [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, 136787); [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. (2006, 098785); [90]Lim et al. (2003, 037037); [91]Watson and Chow (2002, 037873); [92]Venkatachari et al. (2006, 105918); [93]Bae et al. (2004, 156243); [94]Arhami et al. (2006, 156224); [95]Park et al. (2005, 156843); [96]Bae et al. (2004, 098680); [97]Chow et al. (2006, 156350); [98]Arnott et al. (2005, 156227); [99]Bond et al. (1999, 156281); [100]Virkkula et al. (2005, 157097); [101]Petzold et al. (2002, 156863); [102]Park et al. (2006, 098104); [103]Arnott et al. (1999, 020650); [104]Peters et al. (2001, 016925); [105]Pitchford et al. (1997, 156872); [106]Rees et al. (2004, 097164); [107]Watson et al. (2000, 010354); [108]Lee et al. (2005, 156680); [109]Hering et al. (2004, 155837); [110]Watson et al. (1998, 198805); [111]Chakrabarti et al. (2004, 157426); [112]Mathai et al. (1990, 156741); [113]Kidwell and Ondov (2001, 017092); [114]Stanier et al. (2004, 095955); [115]Khlystov et al. (2005, 156635); [116]Takahama et al. (2004, 157038); [117]Chow et al. (2005, 156348); [118]Zhang et al. (2002, 157181); [119]Subramanian et al. (2004, 081203); [120]Chow et al. (2006, 155207); [121]Birch and Cary (1996, 026004); [122]Birch (1998, 024953); [123]Birch and Cary (1996, 002352); [124]NIOSH (1996, 156810); [125]NIOSH (1999, 156811); [126]Chow et al. (1993, 077459); [127]Chow et al. (2007, 156354); [128]Ellis and Novakov (1982, 156416); [129]Peterson and Richards (2002, 156861); [130]Schauer et al. (2003, 037014); [13]1Middlebrook et al. (2003, 042932); [132]Wenzel et al. (2003, 157139); [133]Jimenez et al. (2003, 156611); [134]Phares et al. (2003, 156866); [135]Qin and Prather (2006, 156895); [136]Zhang et al. (2005, 157185); [137]Bein et al. (2005, 156265); [138]Drewnick et al. (2004, 155754); [139]Drewnick et al. (2004, 155755); [140]Lake et al. (2003, 156669); [141]Lake et al. (2004, 088411)

Source: Chow et al. (2008, 156355)

Table A-17. 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 fiber filters 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 first 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 fireplace 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
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, 156277); Blanchard et al. (2002, 047598)	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

Reference	Sample Type	TD Unit	Analytical Instrument	Total Analysis Time
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 area 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

[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-18. Relevant Spatial Scales for PM_{10}, $PM_{2.5}$, and $PM_{10-2.5}$ Measurement

Spatial Scales	PM_{10}	$PM_{2.5}$	$PM_{10-2.5}$
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 PM 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 m. 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 PM 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 m. 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 PM 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 $PM_{10-2.5}$ exposure. Monitors located in populated areas that are nearly adjacent to large industrial point sources of $PM_{10-2.5}$ 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 adjacent to 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 PM 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 PM 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 PM 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 PM 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 PM 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-19. Major routine operating air monitoring networks[a]

Network	Lead Agency	Number of Sites	Initiated	Measurement Parameters	Location of Information and/or Data
STATE / LOCAL / FEDERAL NETWORKS					
NCore[b] – National Core Monitoring Network	EPA	75	2008	O_3, NO/NO_2/NO_Y, SO_2, CO, $PM_{2.5}$/$_{PM10-2.5}$, $PM_{2.5}$ speciation, NH_3, HNO_3, surface meteorology[c]	http://www.epa.gov/ttN/Amtic/monstratdoc.html
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/

Network	Lead Agency	Number of Sites	Initiated	Measurement Parameters	Location of Information and/or Data
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
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 1960s	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 1960s	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[d], Hg	http://www.epa.gov/ttN/Amtic/airtoxpg.html
State/Local Air Toxics Monitoring	EPA	250+	1987	VOCs, Carbonyls, PM_{10} metals[d], 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[f]	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

Network	Lead Agency	Number of Sites	Initiated	Measurement Parameters	Location of Information and/or Data
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					
UV Index – EPA Sunrise Program[g]	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	NASA co-located networks	22 + other participants	1998	Aerosol spectral optical depths, aerosol size distributions, and precipitable water	http://aeronet.gsfc.nasa.gov/index.html
MPLNET – Micro-pulse Lidar Network		8	2000	Aerosols and cloud layer heights	http://mplnet.gsfc.nasa.gov/
PRIMENet – Park Research & Intensive Monitoring of Ecosystems NETwork[h]	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]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.
[b]NCore is a network proposed to replace NAMS, as a component of SLAMS; NAMS are currently designated as national trends sites.
[c]surface meteorology includes wind direction and speed, temperature, precipitation, relative humidity, solar radiation (PAMS only).
[d]PM_{10} metals may include arsenic, beryllium, cadmium, chromium, lead, manganese, nickel, and others.
[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.1.3. Monitor Distribution with Respect to Population Density

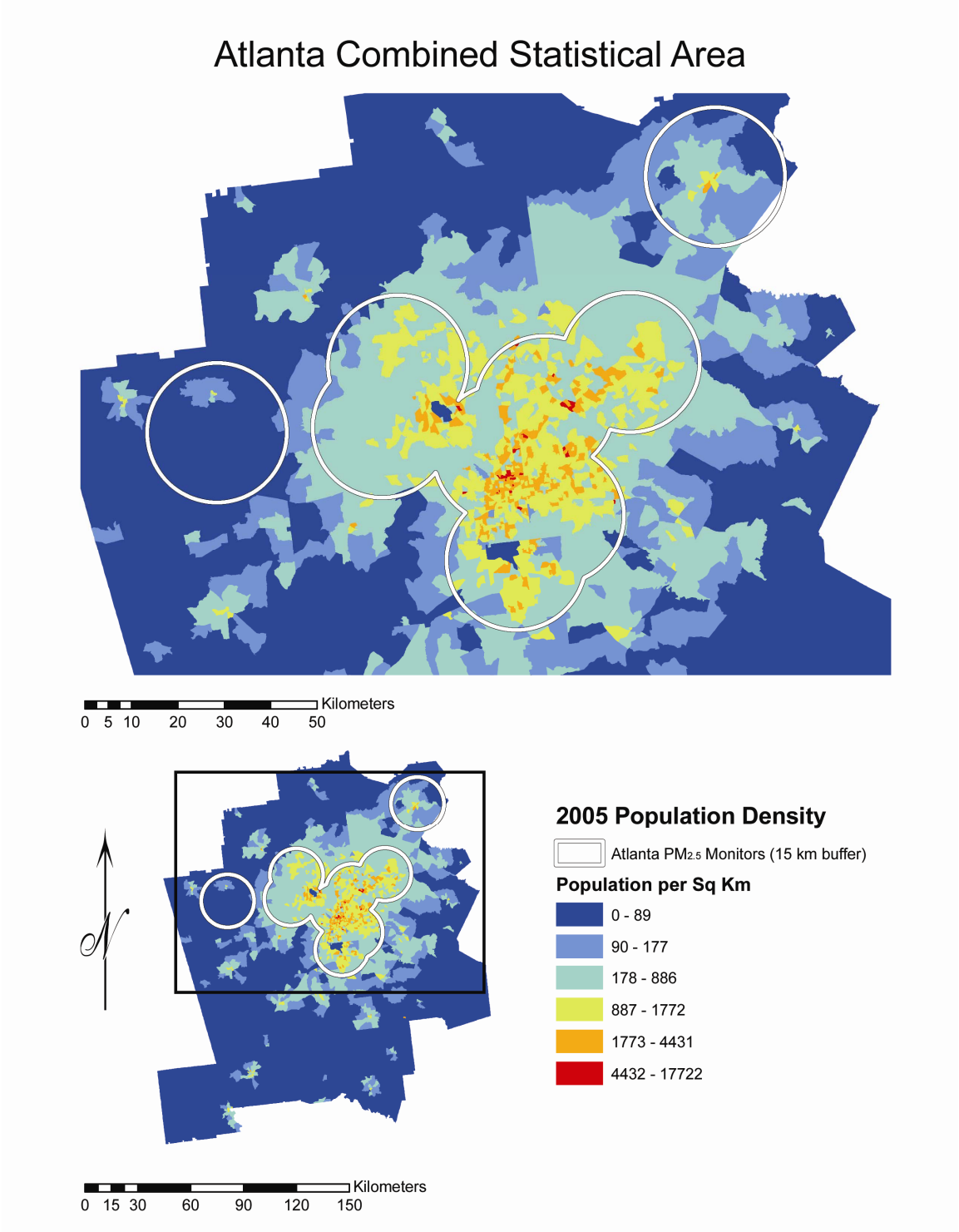


Figure A-1. $PM_{2.5}$ monitor distribution in comparison with population density, Atlanta, GA.

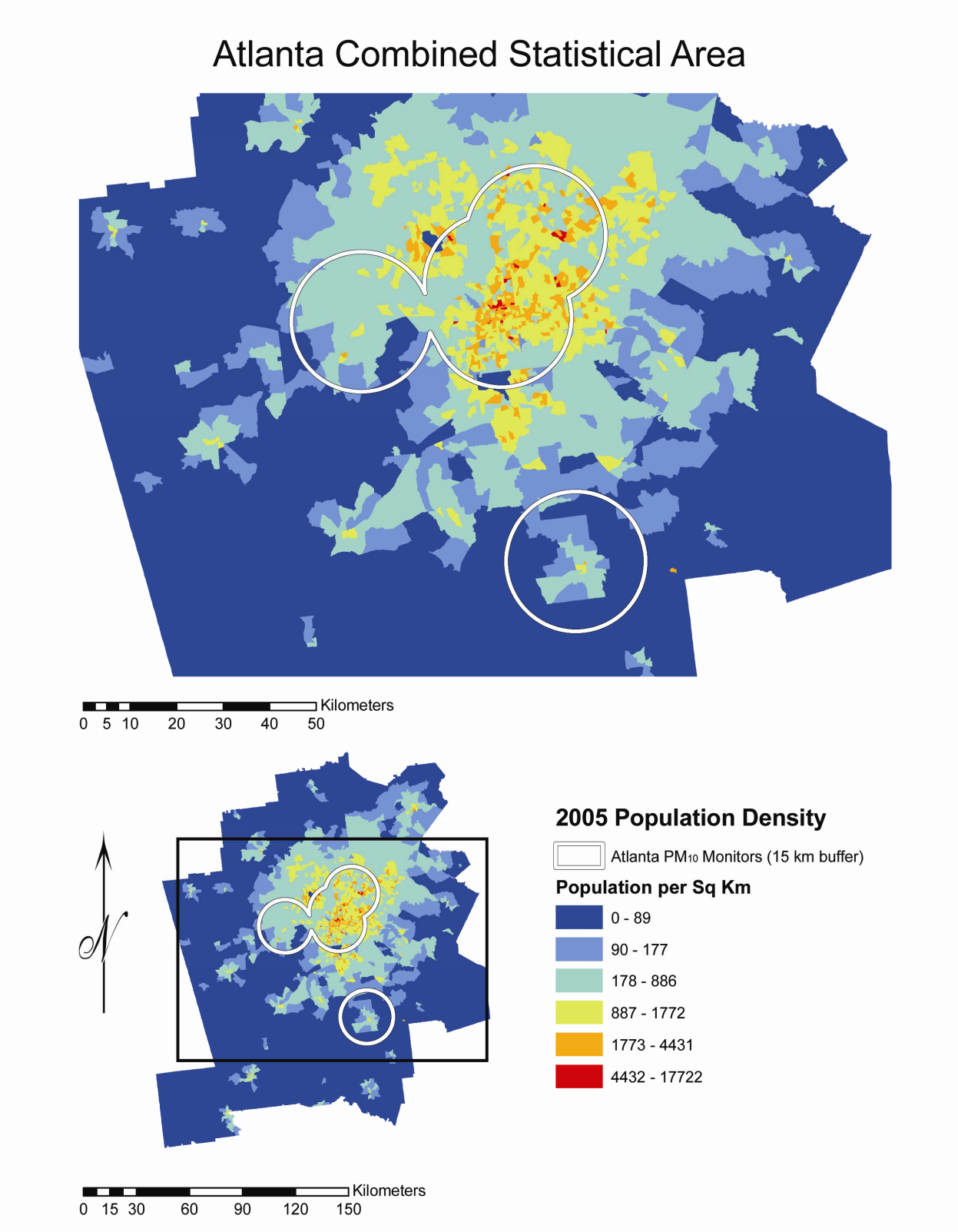


Figure A-2. PM_{10} monitor distribution in comparison with population density, Atlanta, GA.

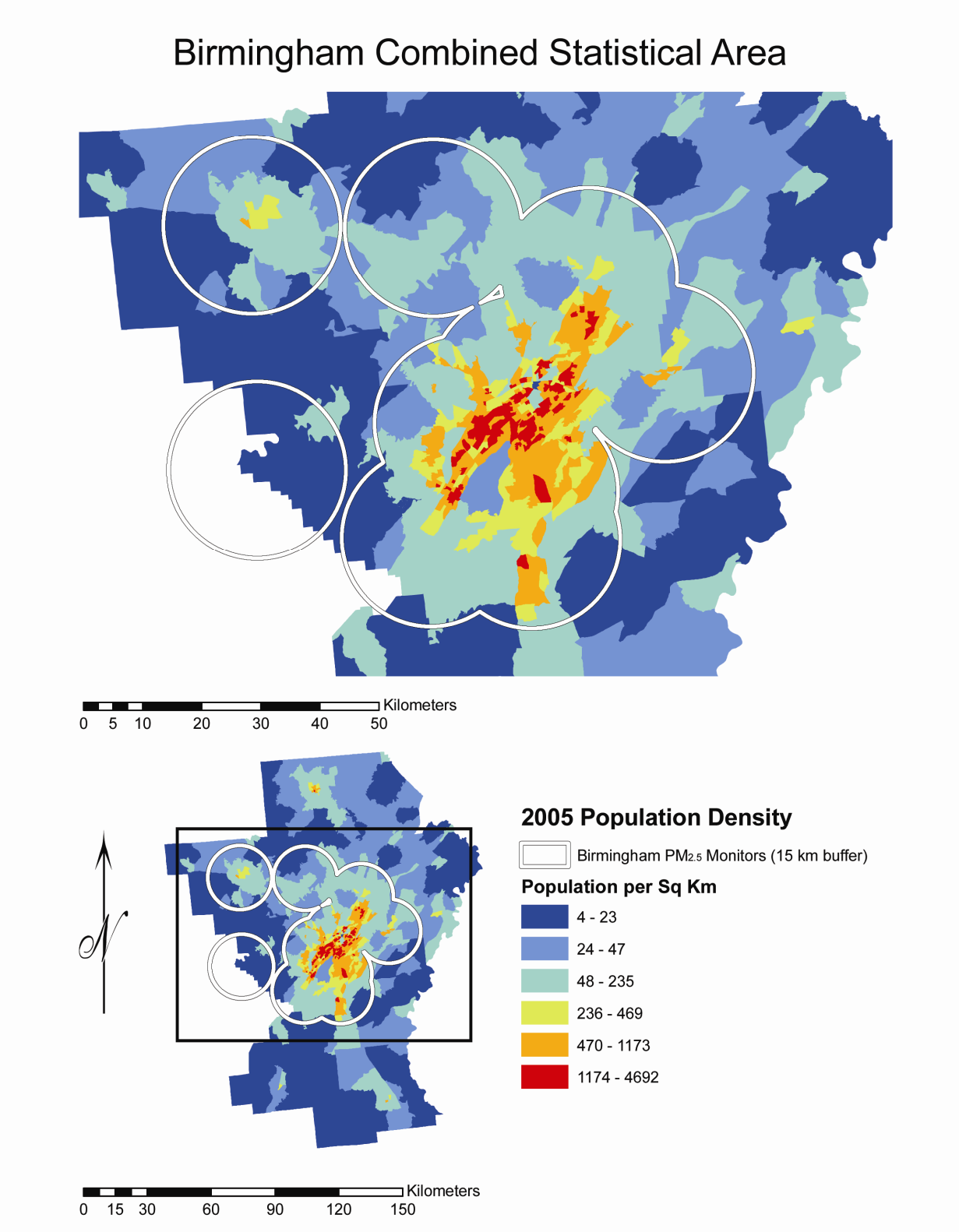


Figure A-3. **$PM_{2.5}$ monitor distribution in comparison with population density, Birmingham, AL.**

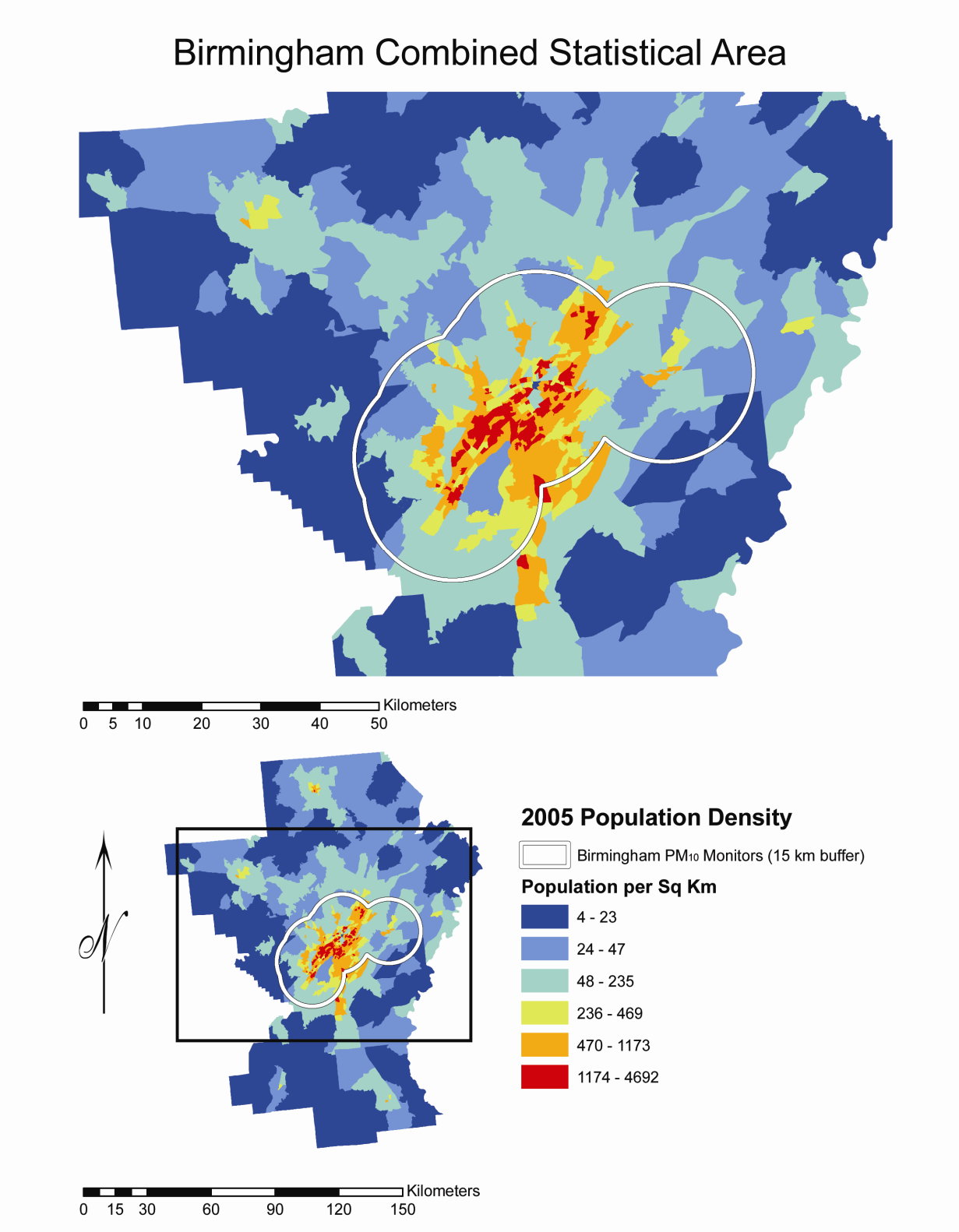


Figure A-4. **PM_{10} monitor distribution in comparison with population density, Birmingham, AL.**

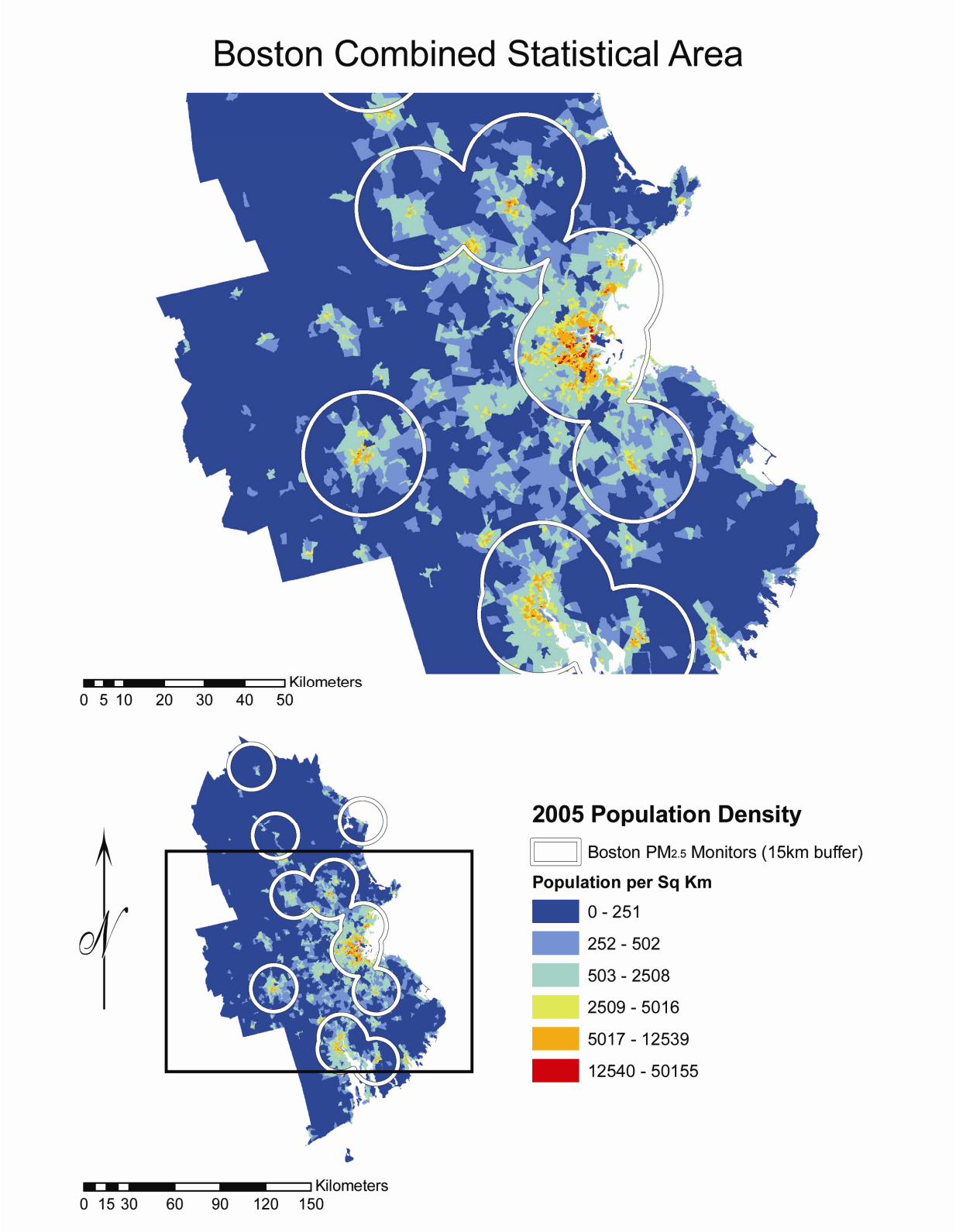


Figure A-5. $PM_{2.5}$ monitor distribution in comparison with population density, Boston, MA.

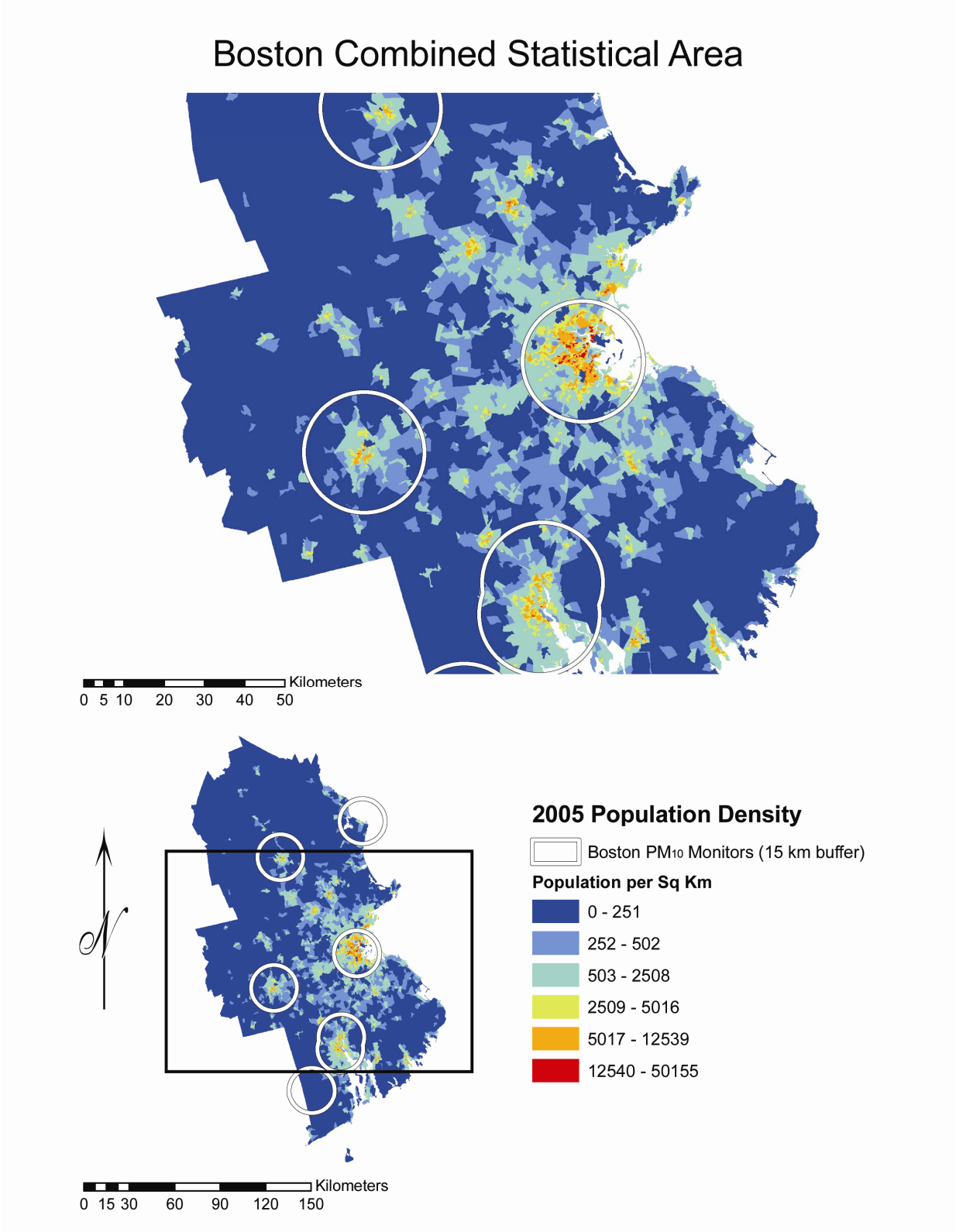


Figure A-6. PM_{10} monitor distribution in comparison with population density, Boston, MA.

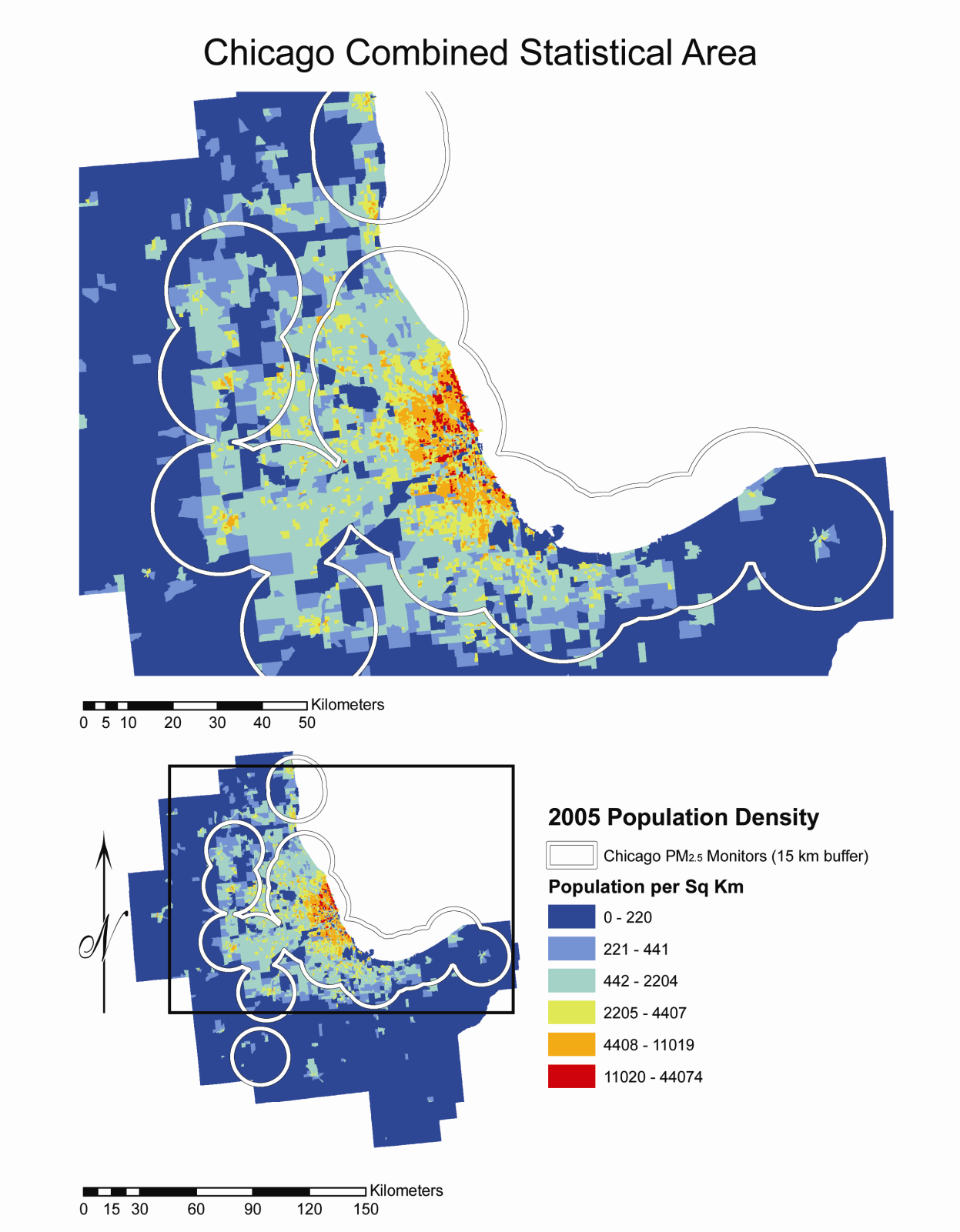


Figure A-7. $PM_{2.5}$ monitor distribution in comparison with population density, Chicago, IL.

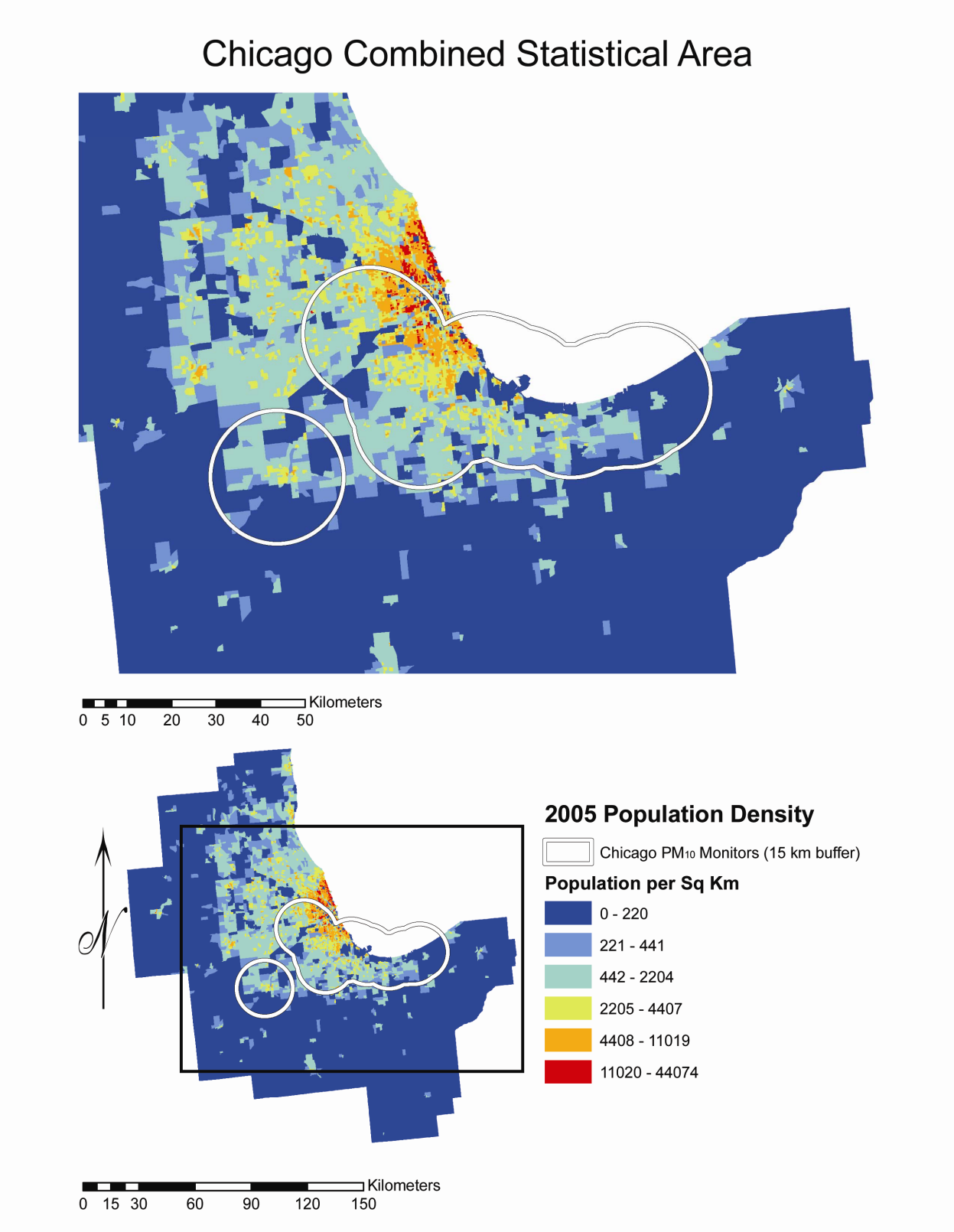


Figure A-8. PM_{10} monitor distribution in comparison with population density, Chicago, IL.

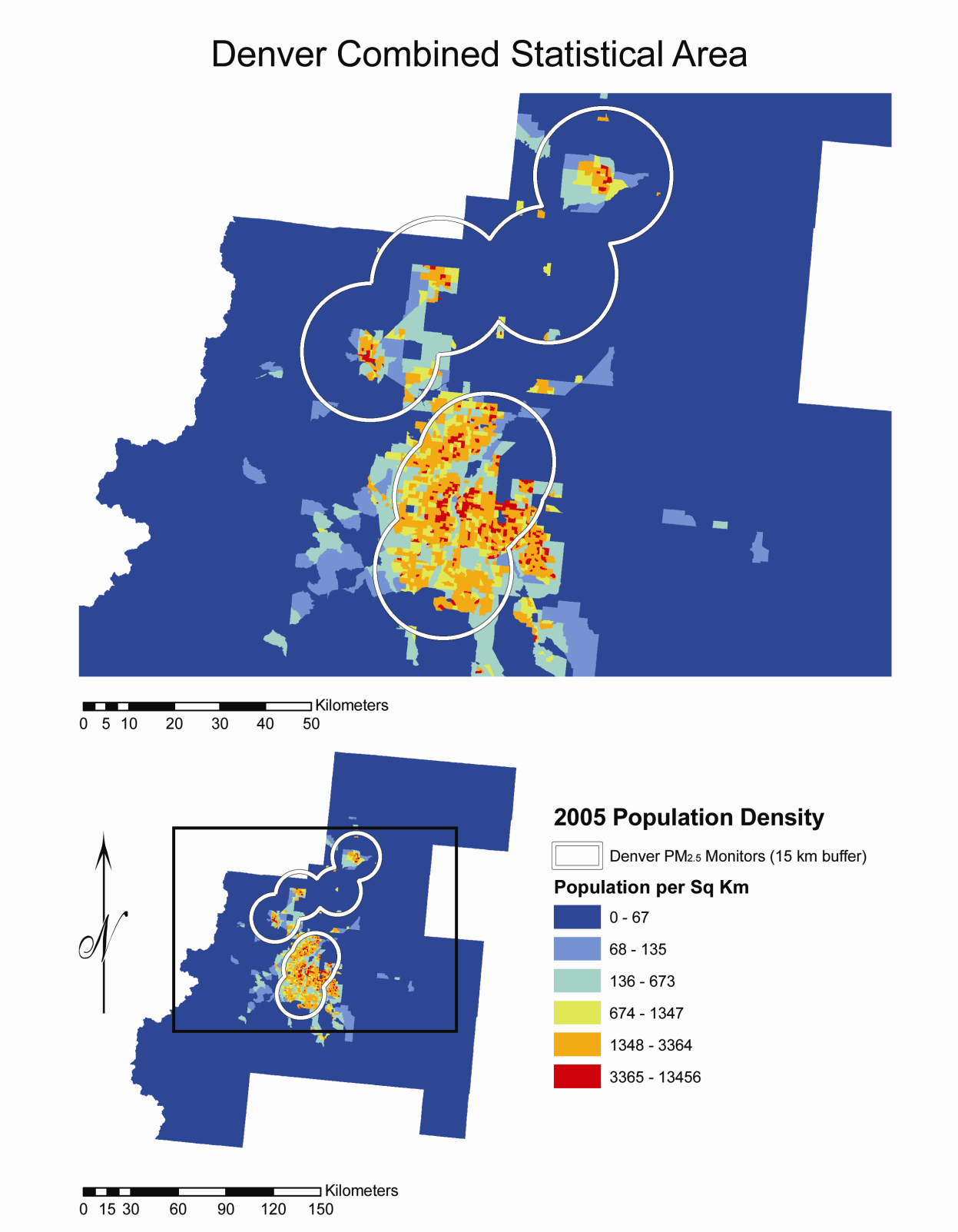


Figure A-9. $PM_{2.5}$ monitor distribution in comparison with population density, Denver, CO.

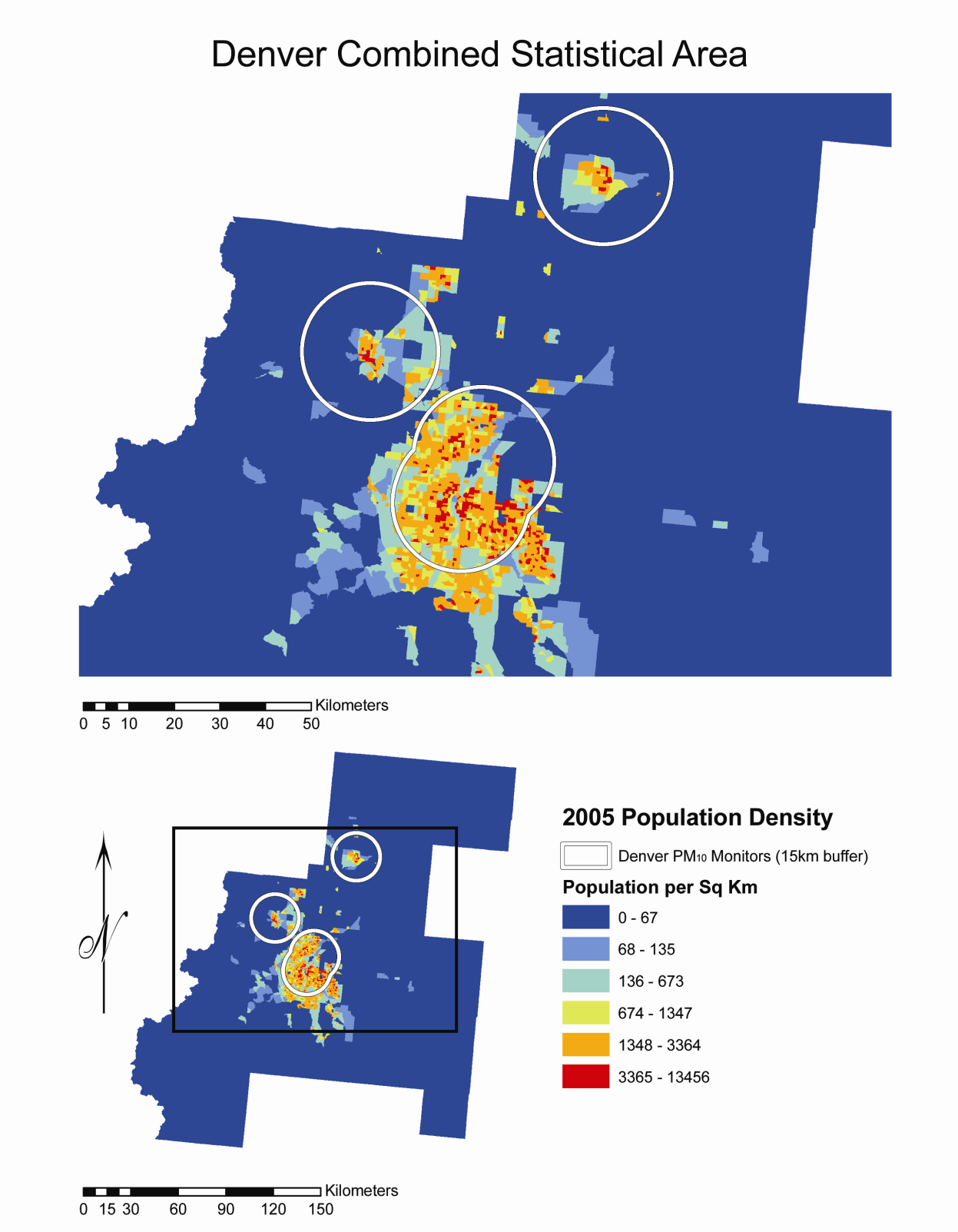


Figure A-10. PM_{10} monitor distribution in comparison with population density, Denver, CO.

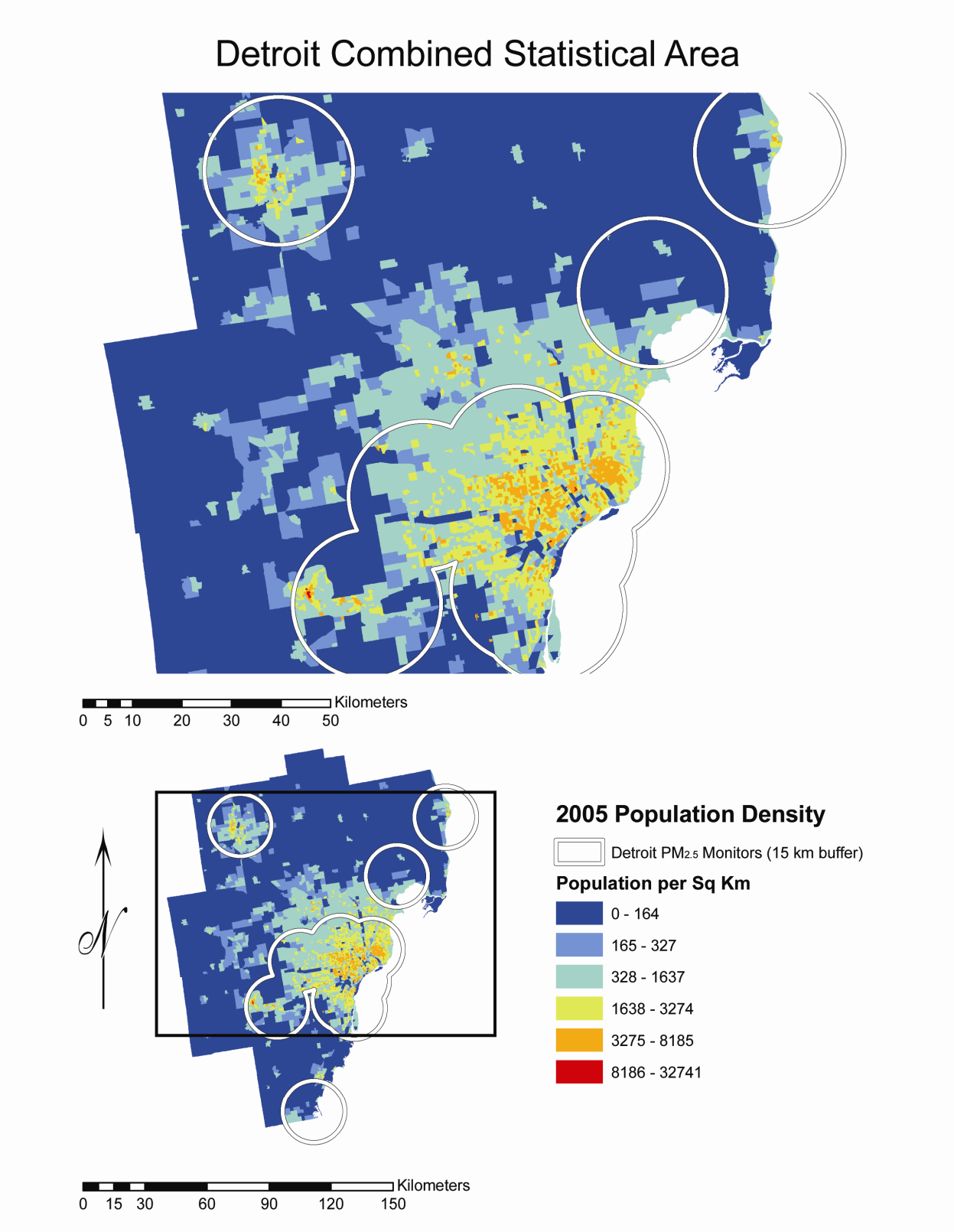


Figure A-11. $PM_{2.5}$ monitor distribution in comparison with population density, Detroit, MI.

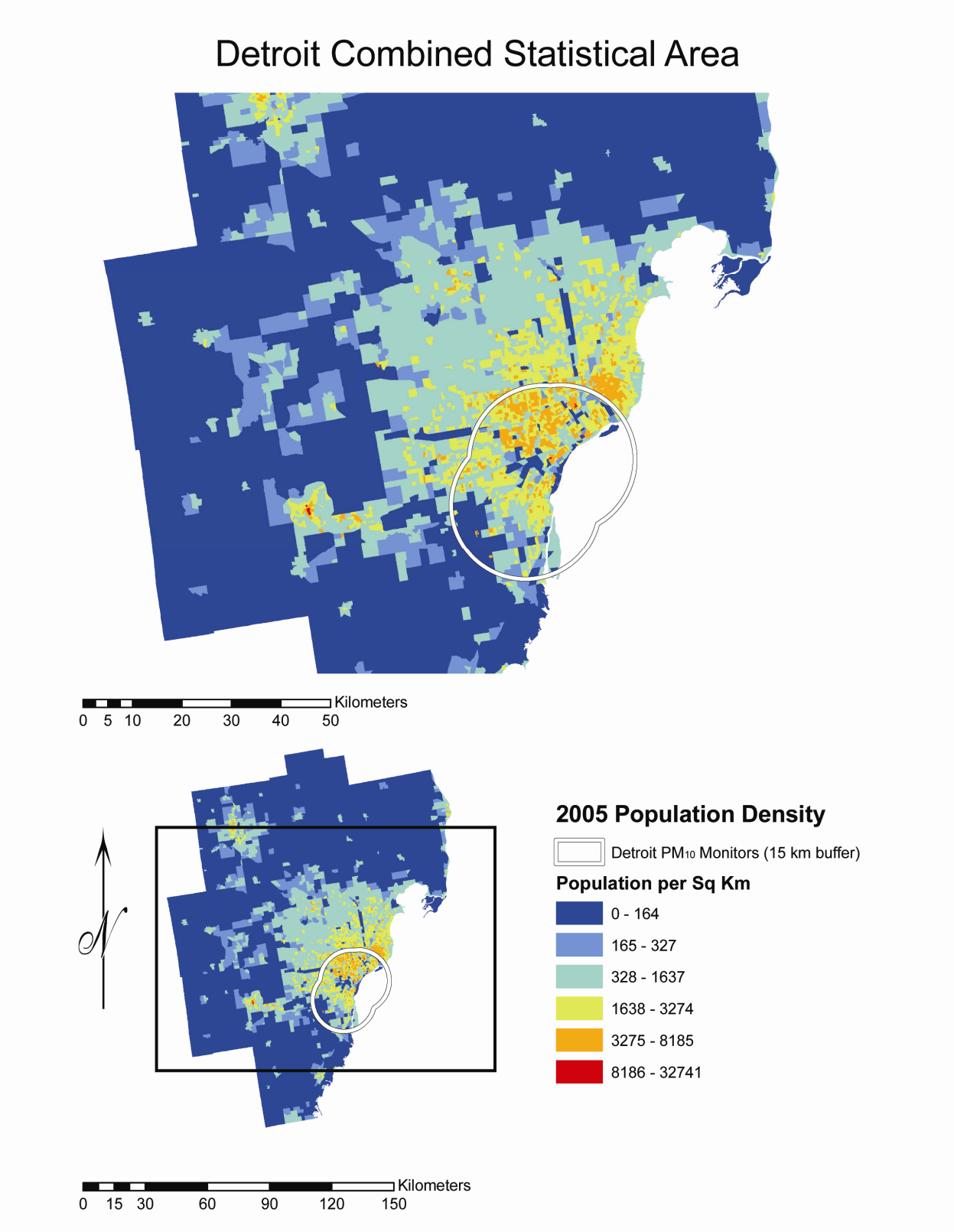


Figure A-12. PM_{10} monitor distribution in comparison with population density, Detroit, MI.

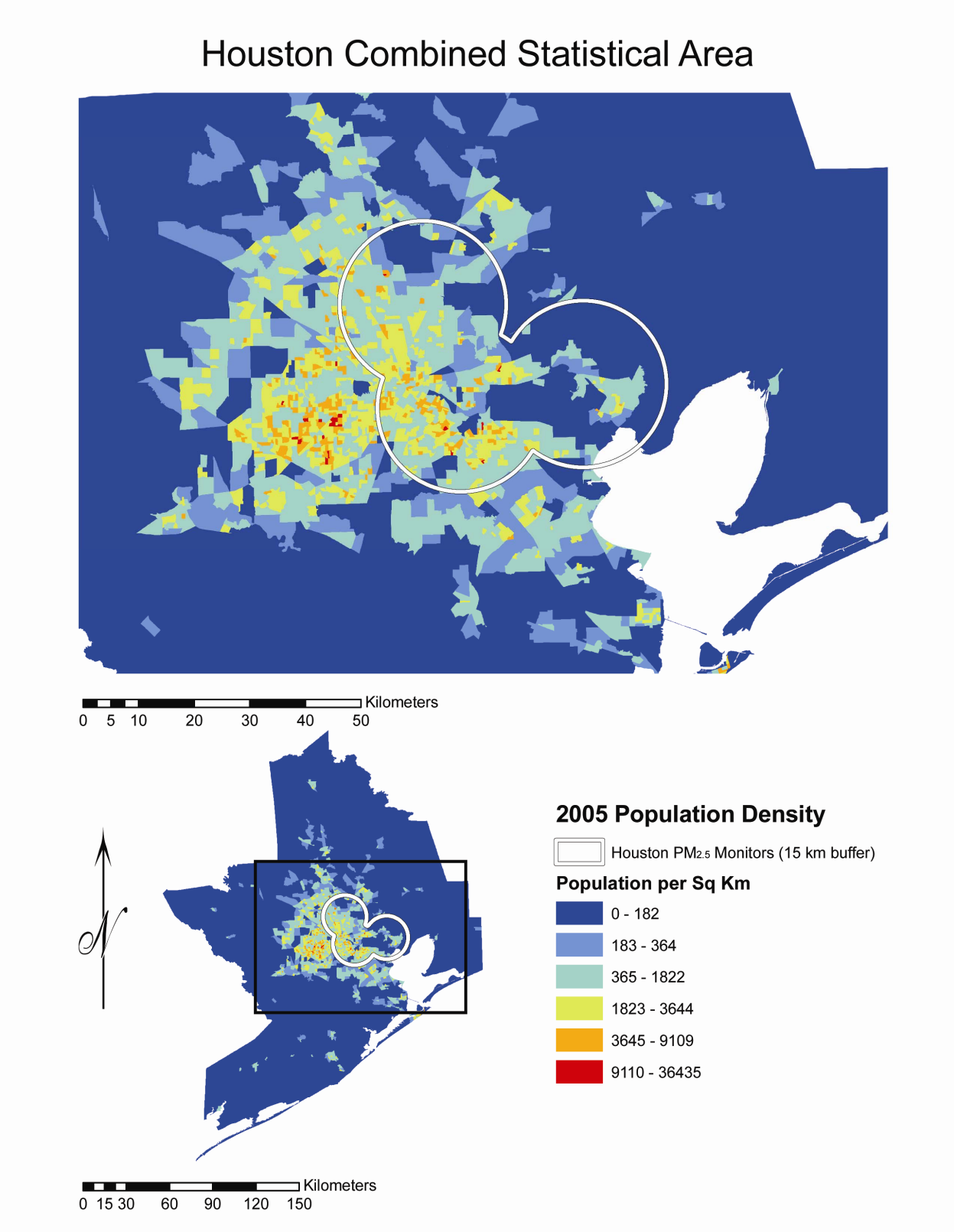


Figure A-13. $PM_{2.5}$ monitor distribution in comparison with population density, Houston, TX.

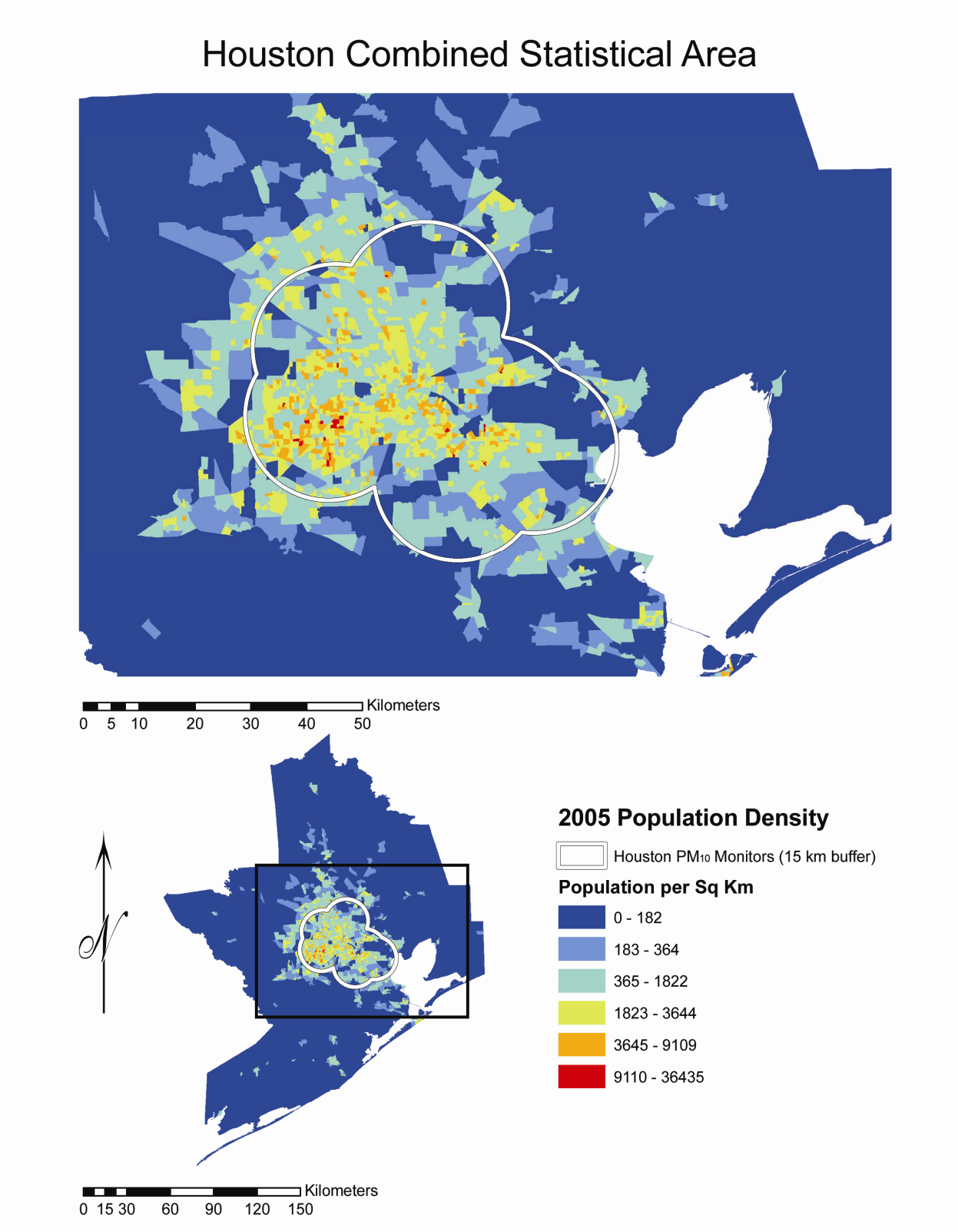


Figure A-14. PM_{10} monitor distribution in comparison with population density, Houston, TX.

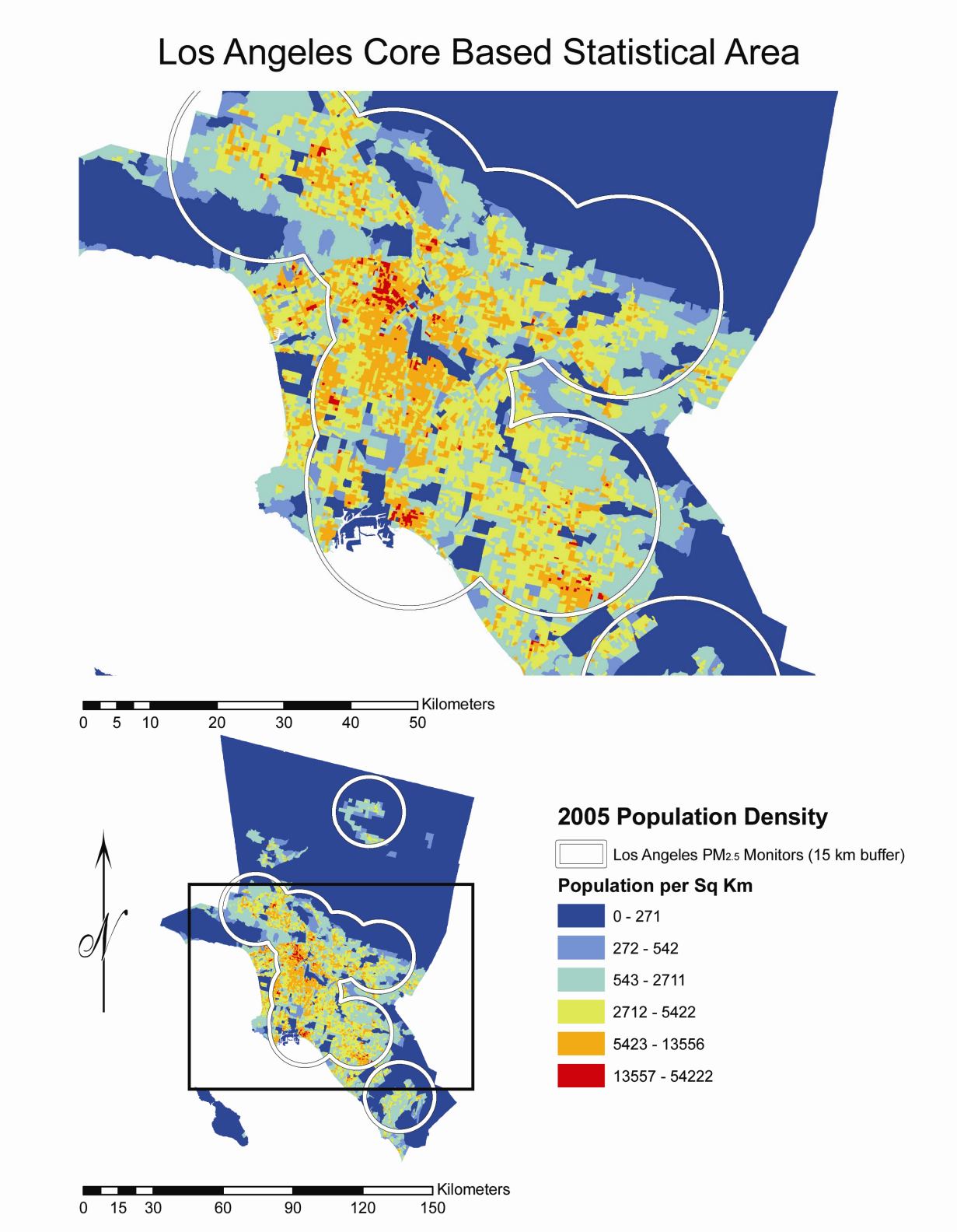


Figure A-15. $PM_{2.5}$ monitor distribution in comparison with population density, Los Angeles, CA.

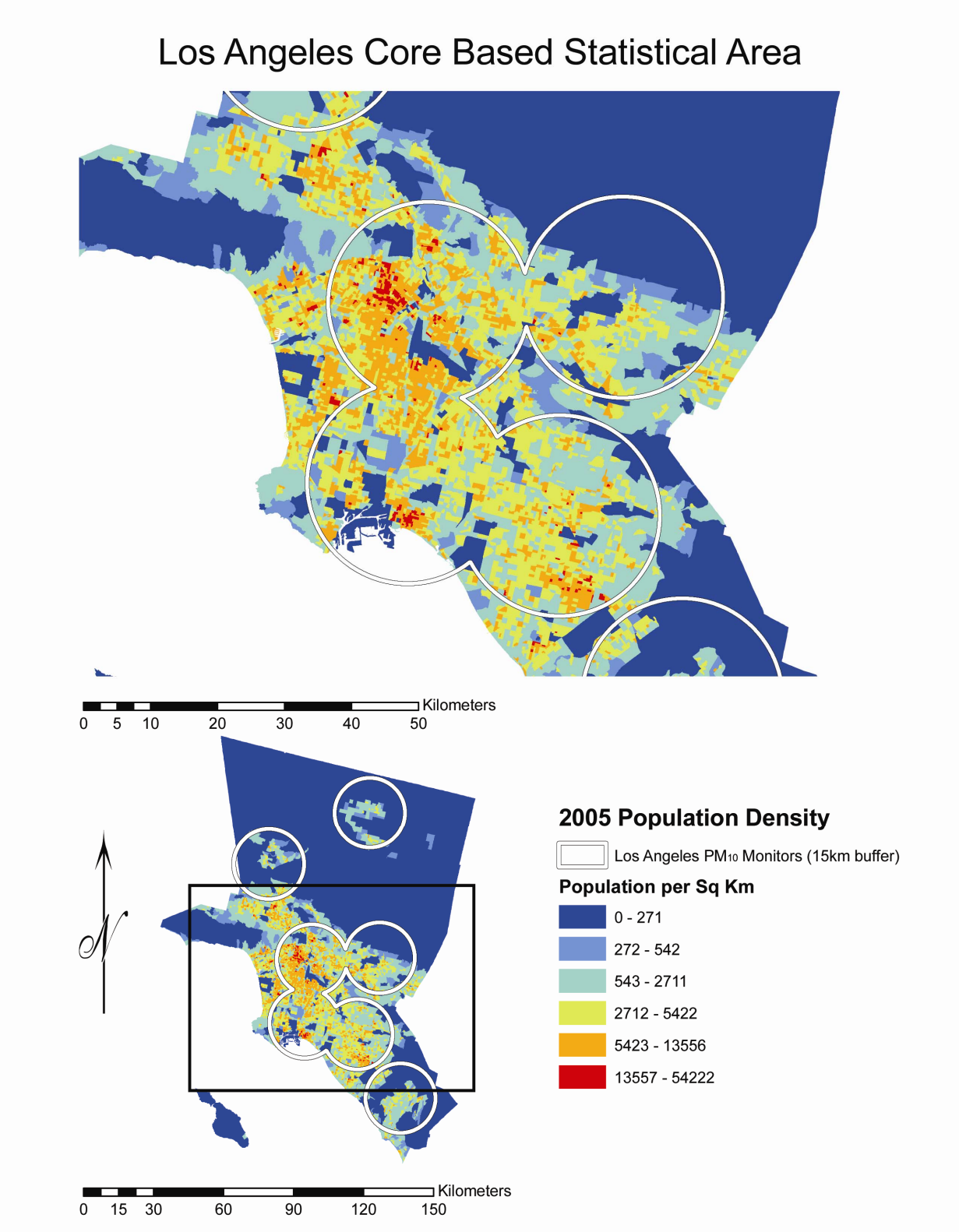


Figure A-16. PM_{10} monitor distribution in comparison with population density, Los Angeles, CA.

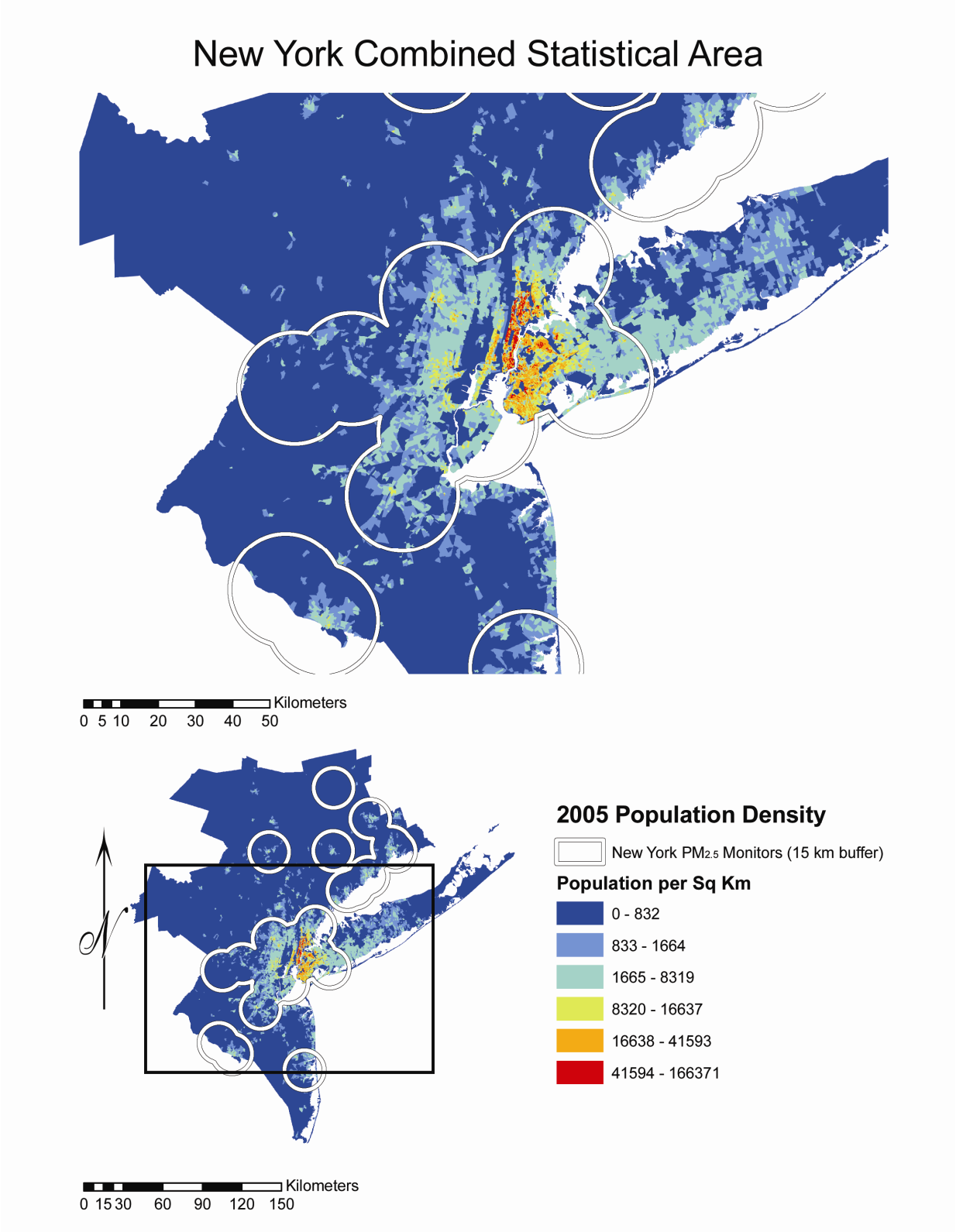


Figure A-17. $PM_{2.5}$ monitor distribution in comparison with population density, New York, NY.

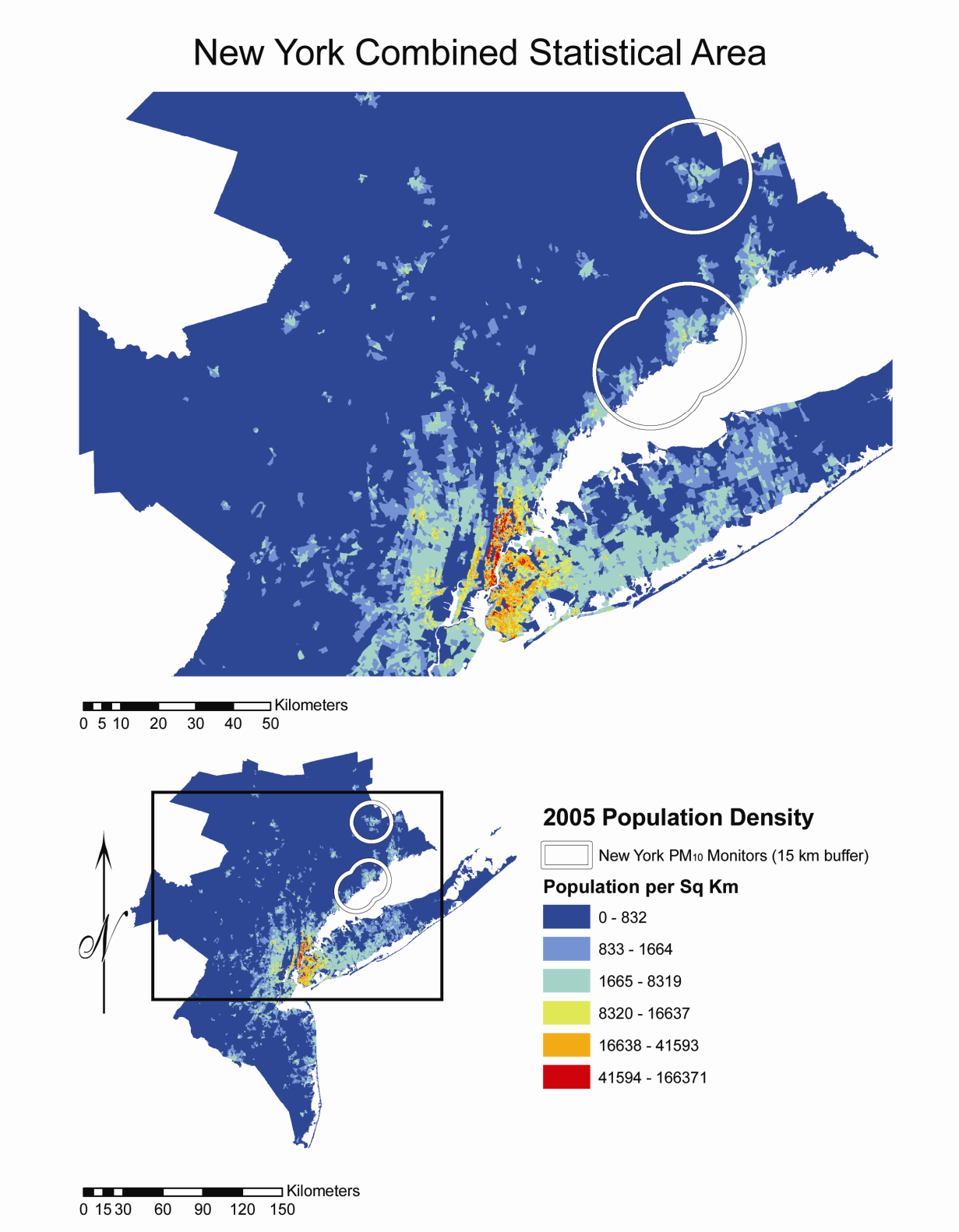


Figure A-18. PM_{10} monitor distribution in comparison with population density, New York, NY.

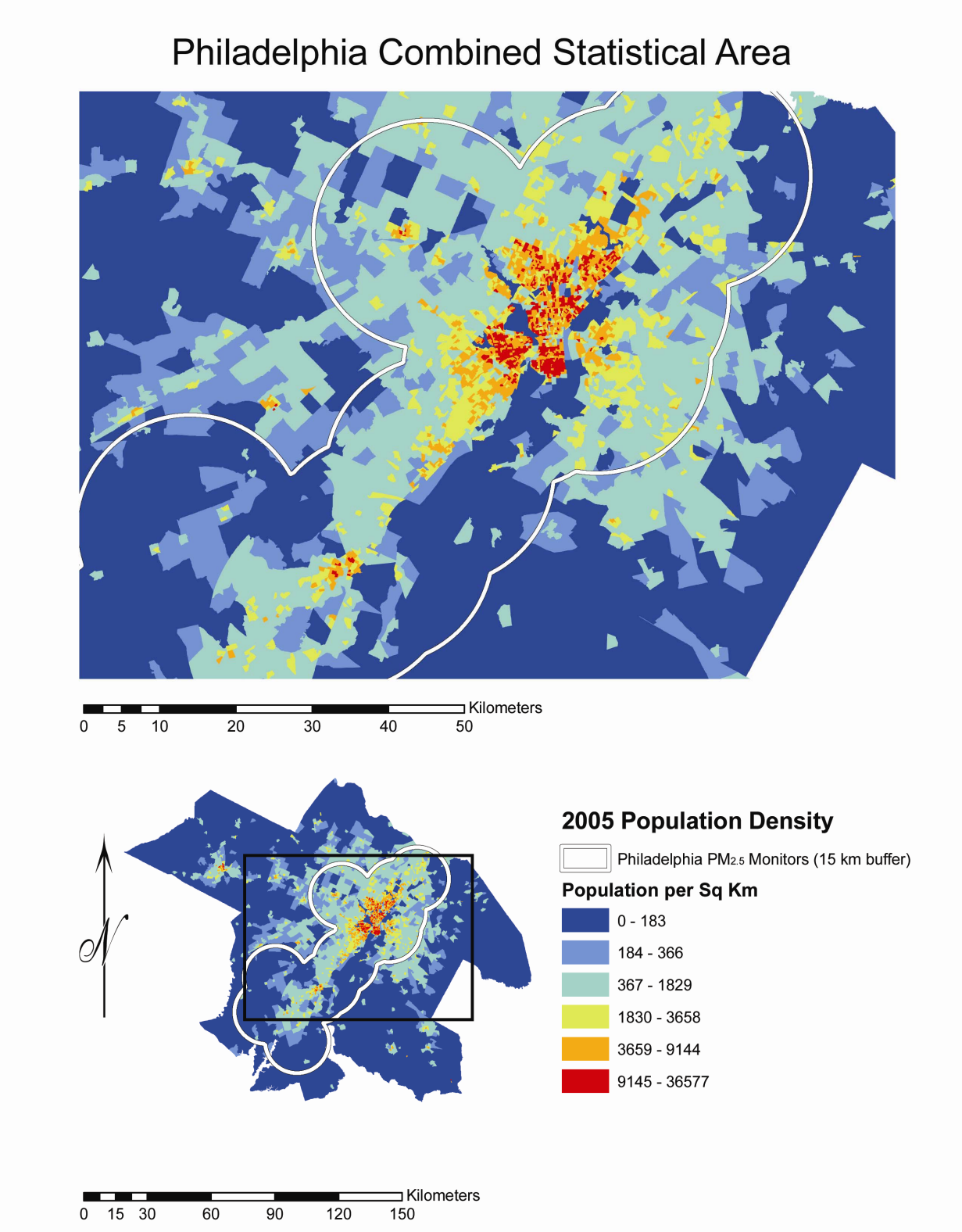


Figure A-19. $PM_{2.5}$ monitor distribution in comparison with population density, Philadelphia, PA.

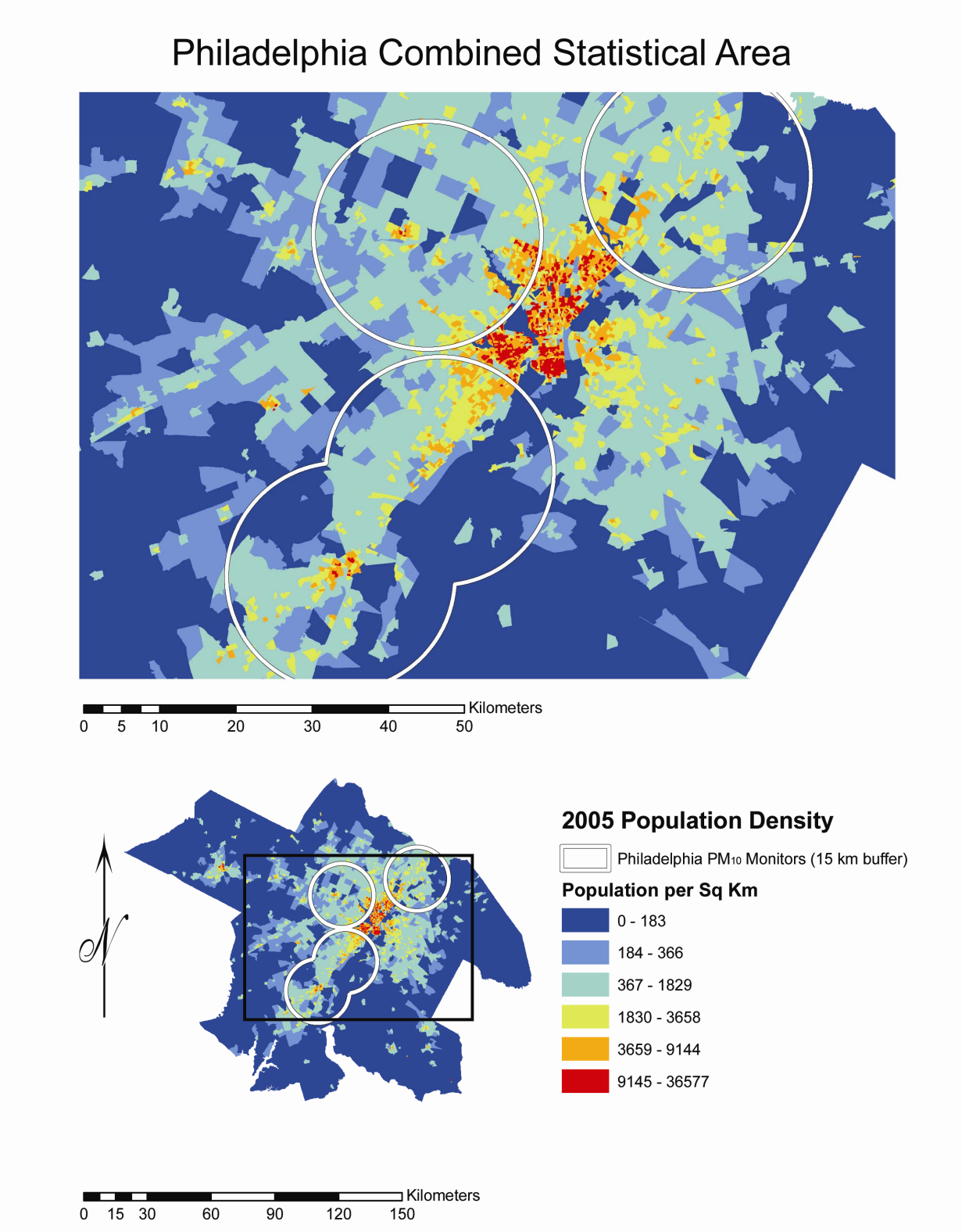


Figure A-20. PM_{10} monitor distribution in comparison with population density, Philadelphia, PA.

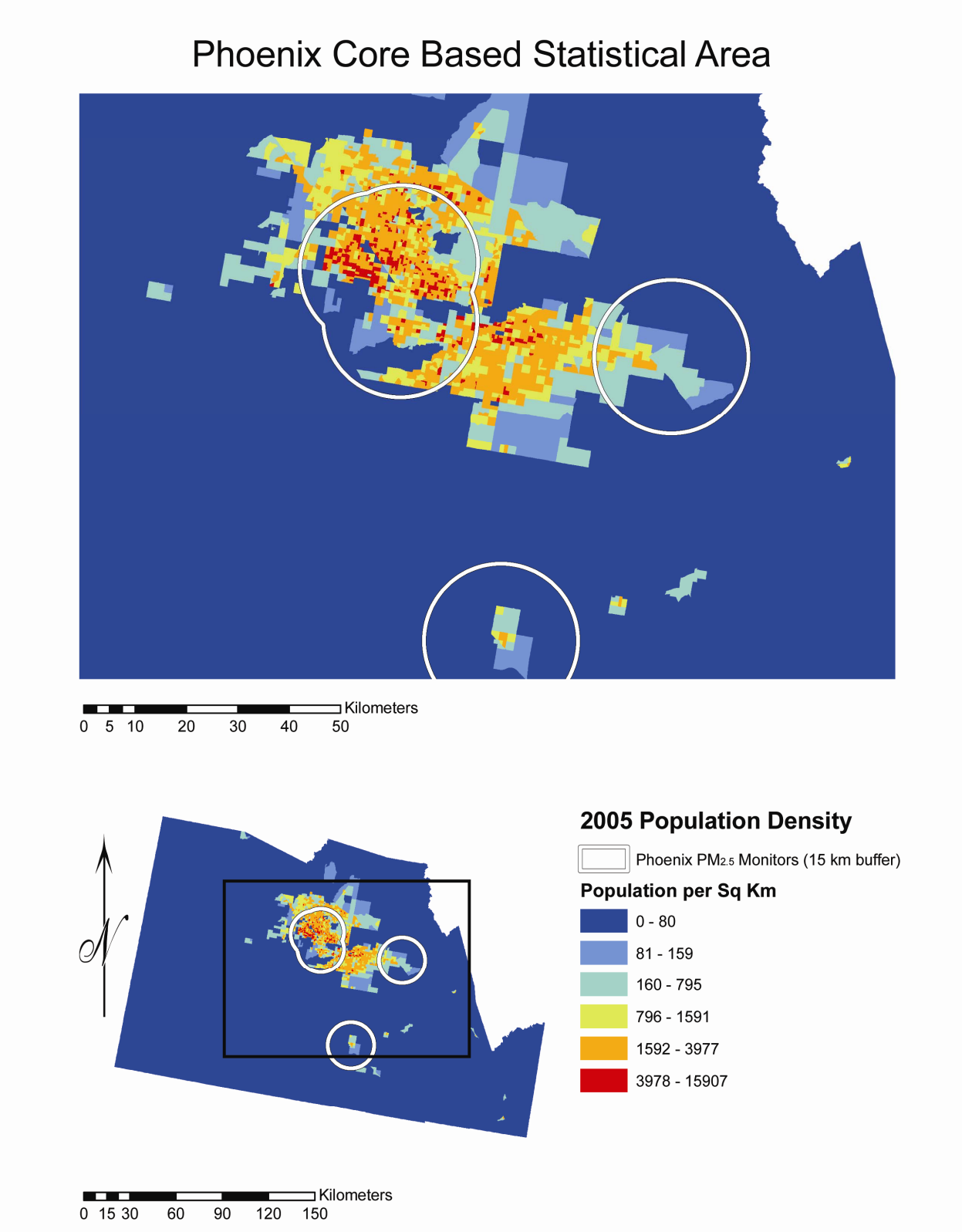


Figure A-21. $PM_{2.5}$ monitor distribution in comparison with population density, Phoenix, AZ.

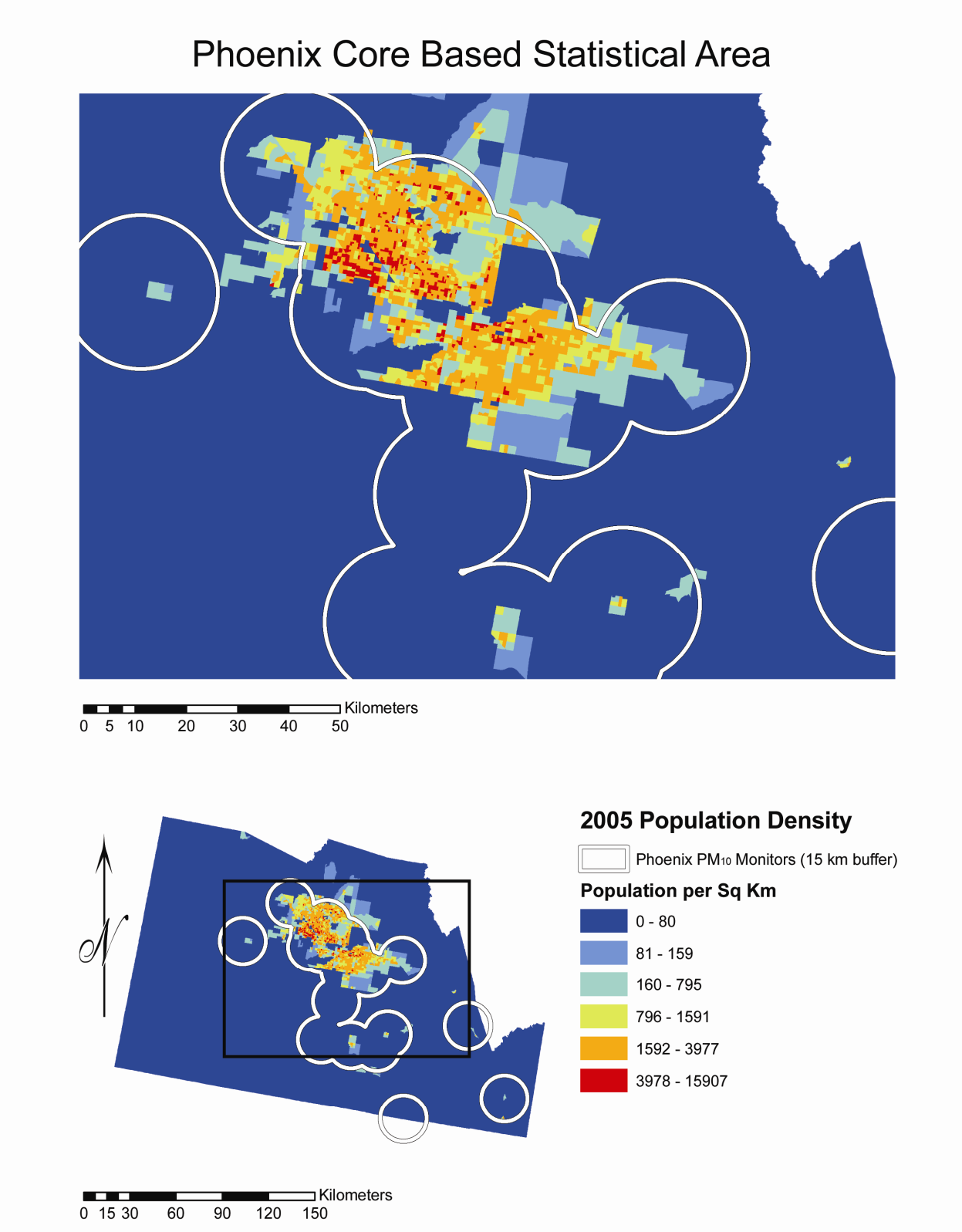


Figure A-22. PM_{10} monitor distribution in comparison with population density, Phoenix, AZ.

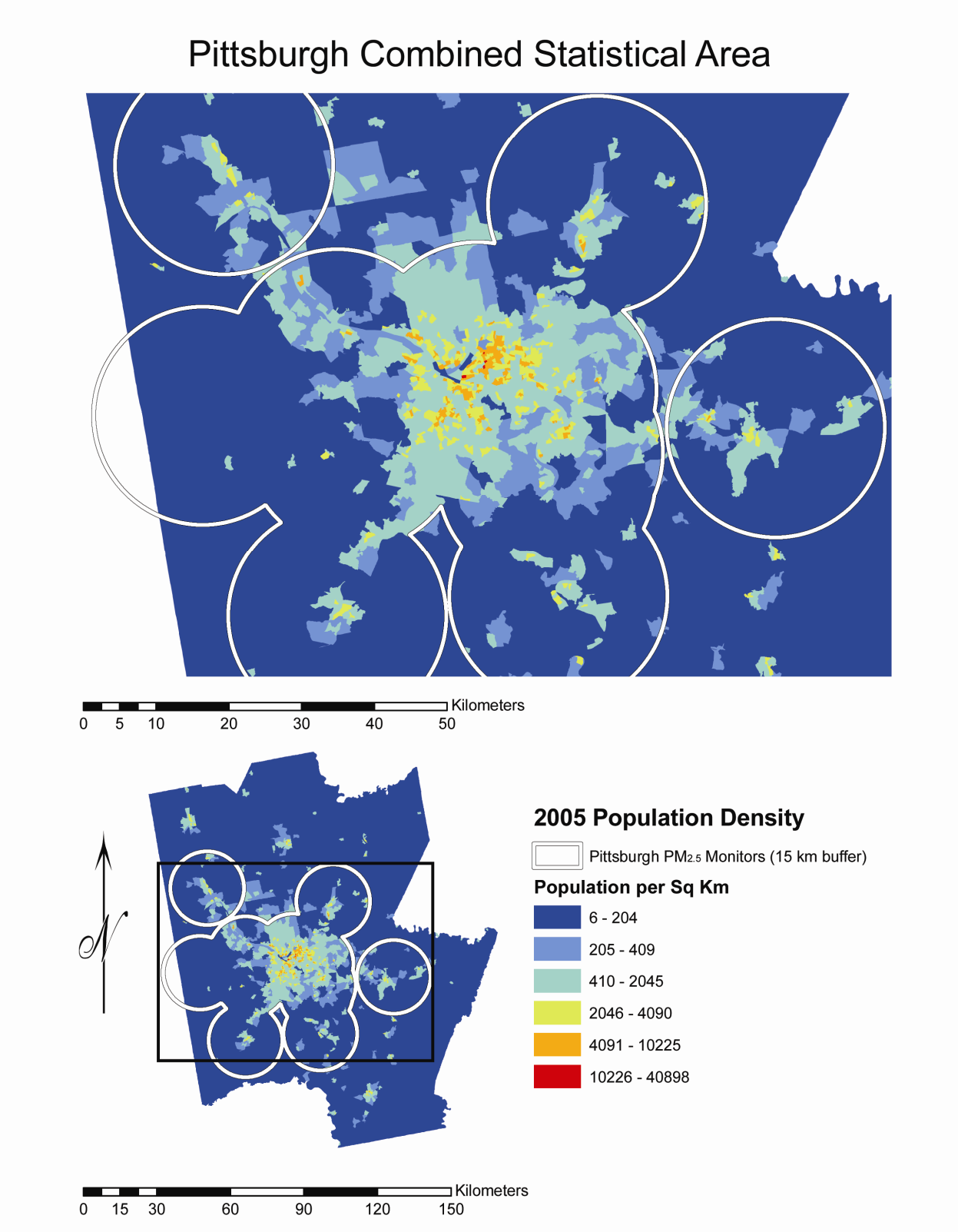


Figure A-23. $PM_{2.5}$ monitor distribution in comparison with population density, Pittsburgh, PA.

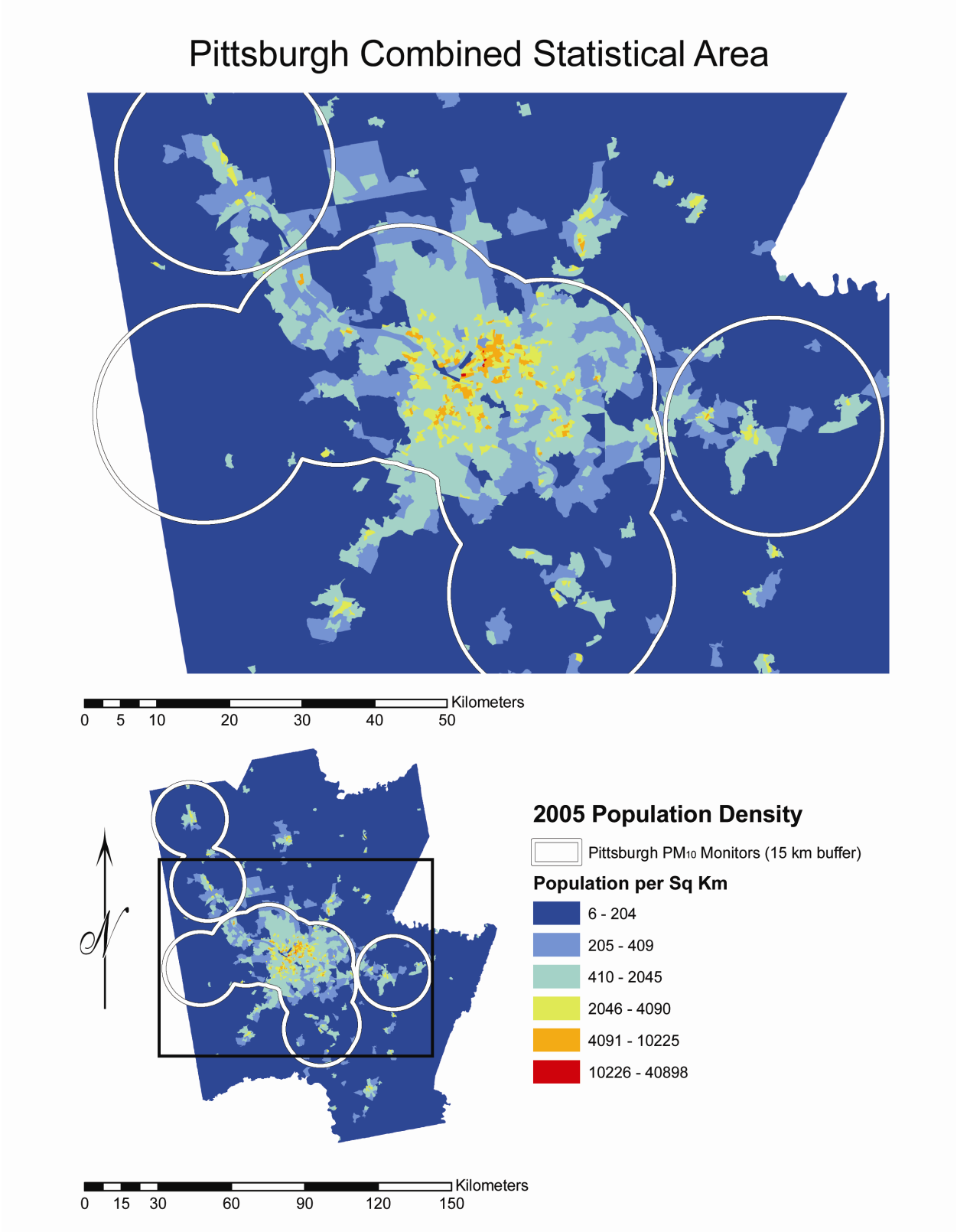


Figure A-24. PM_{10} monitor distribution in comparison with population density, Pittsburgh, PA.

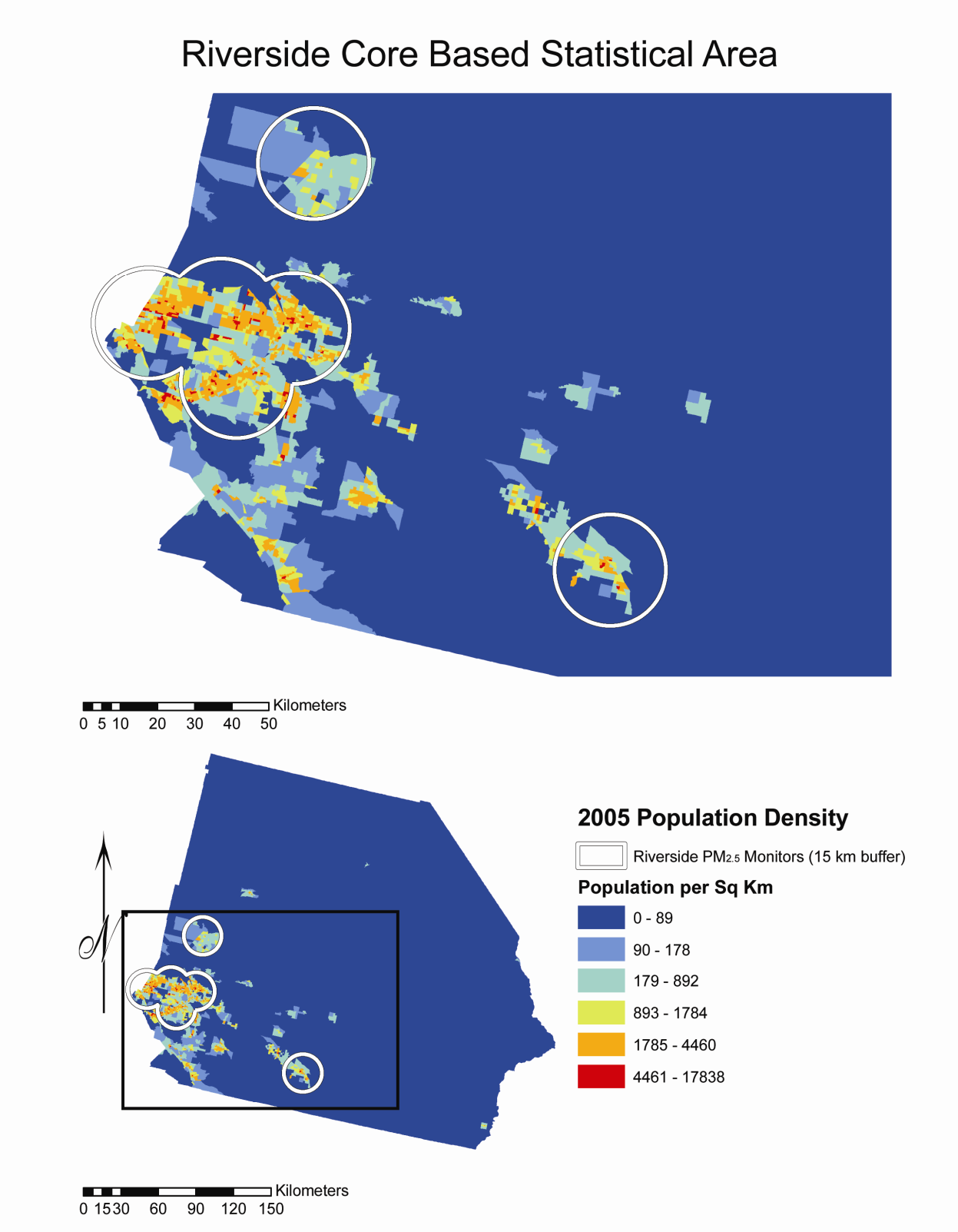


Figure A-25. $PM_{2.5}$ monitor distribution in comparison with population density, Riverside, CA.

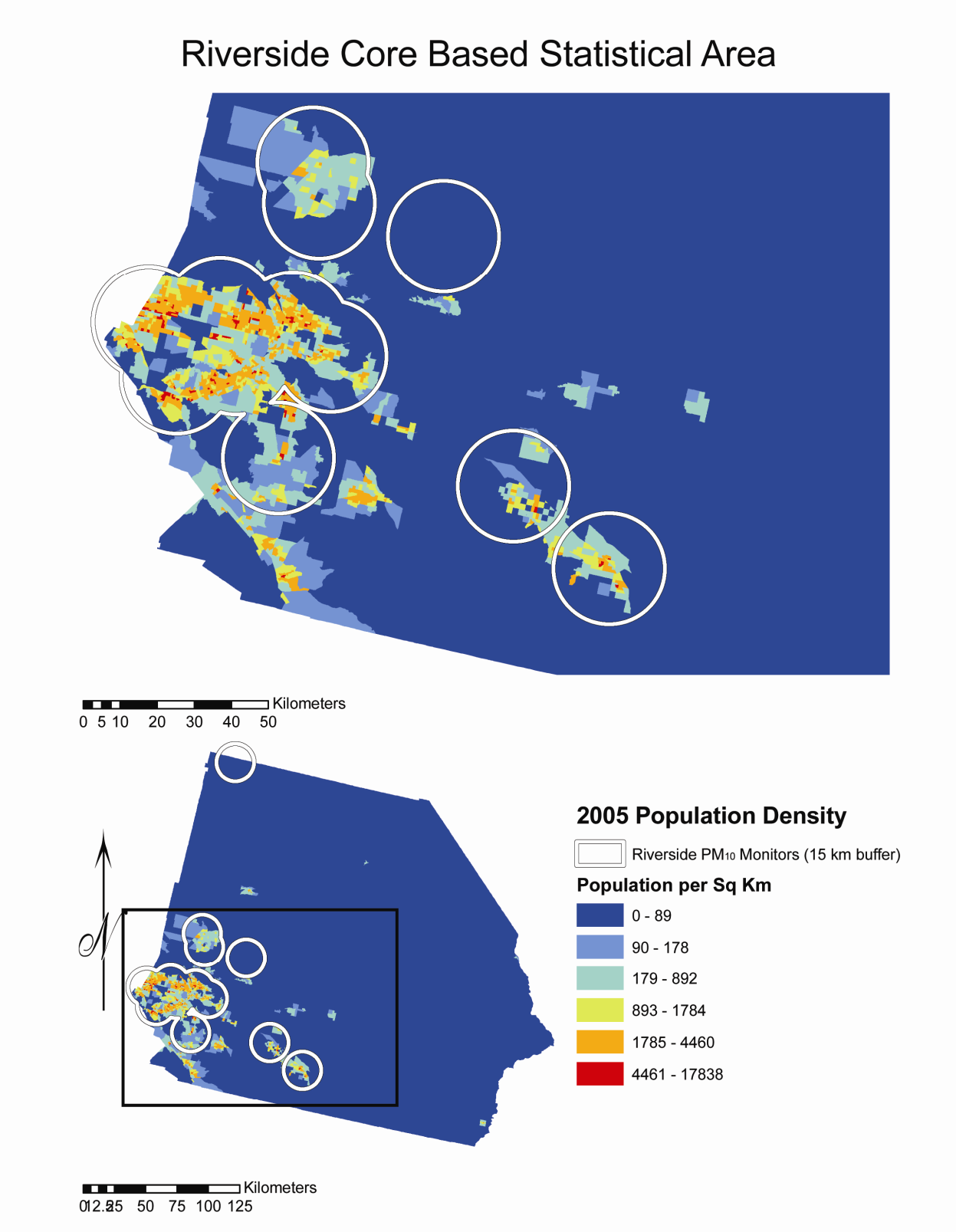


Figure A-26. PM_{10} monitor distribution in comparison with population density, Riverside, CA.

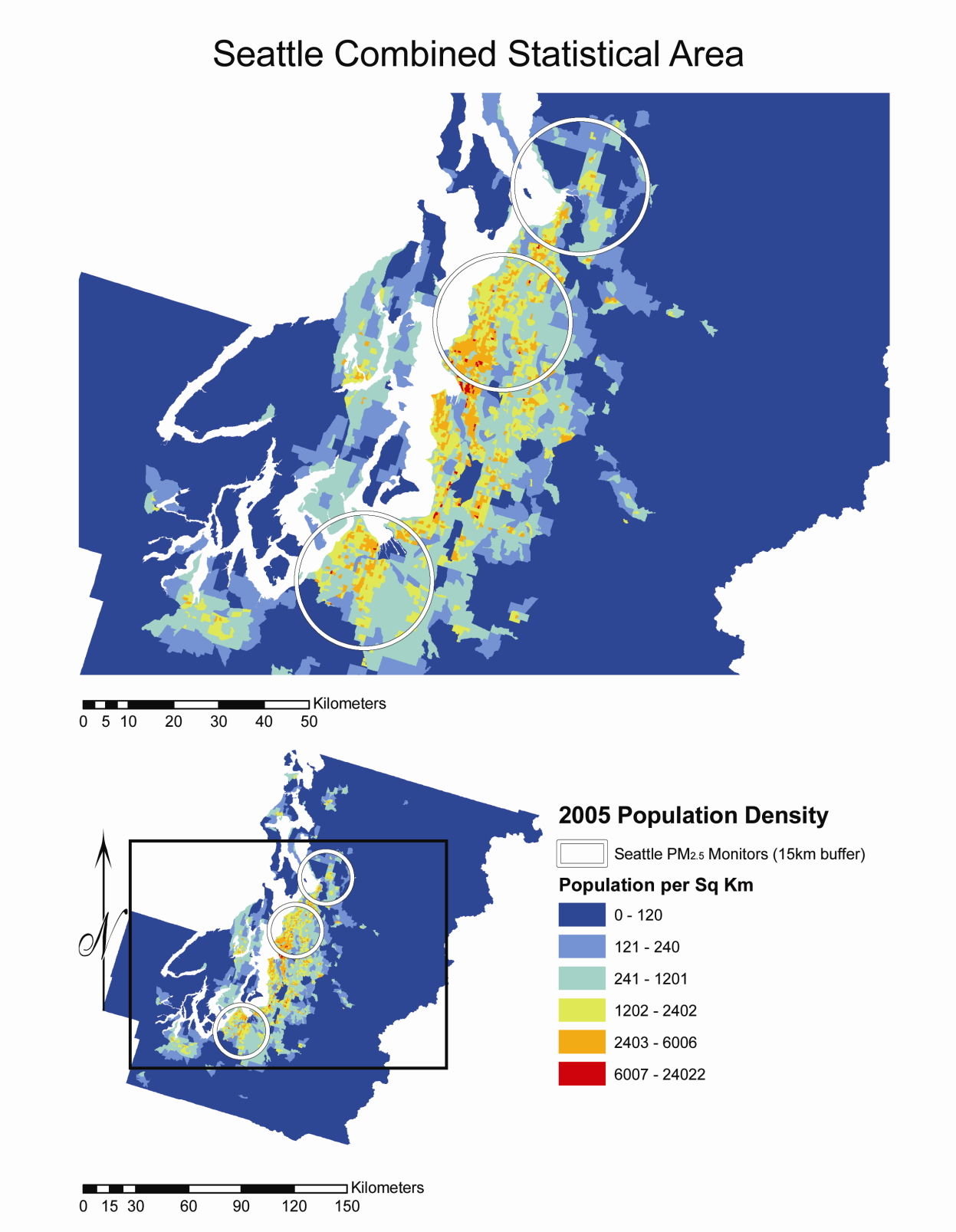


Figure A-27. $PM_{2.5}$ monitor distribution in comparison with population density, Seattle, WA.

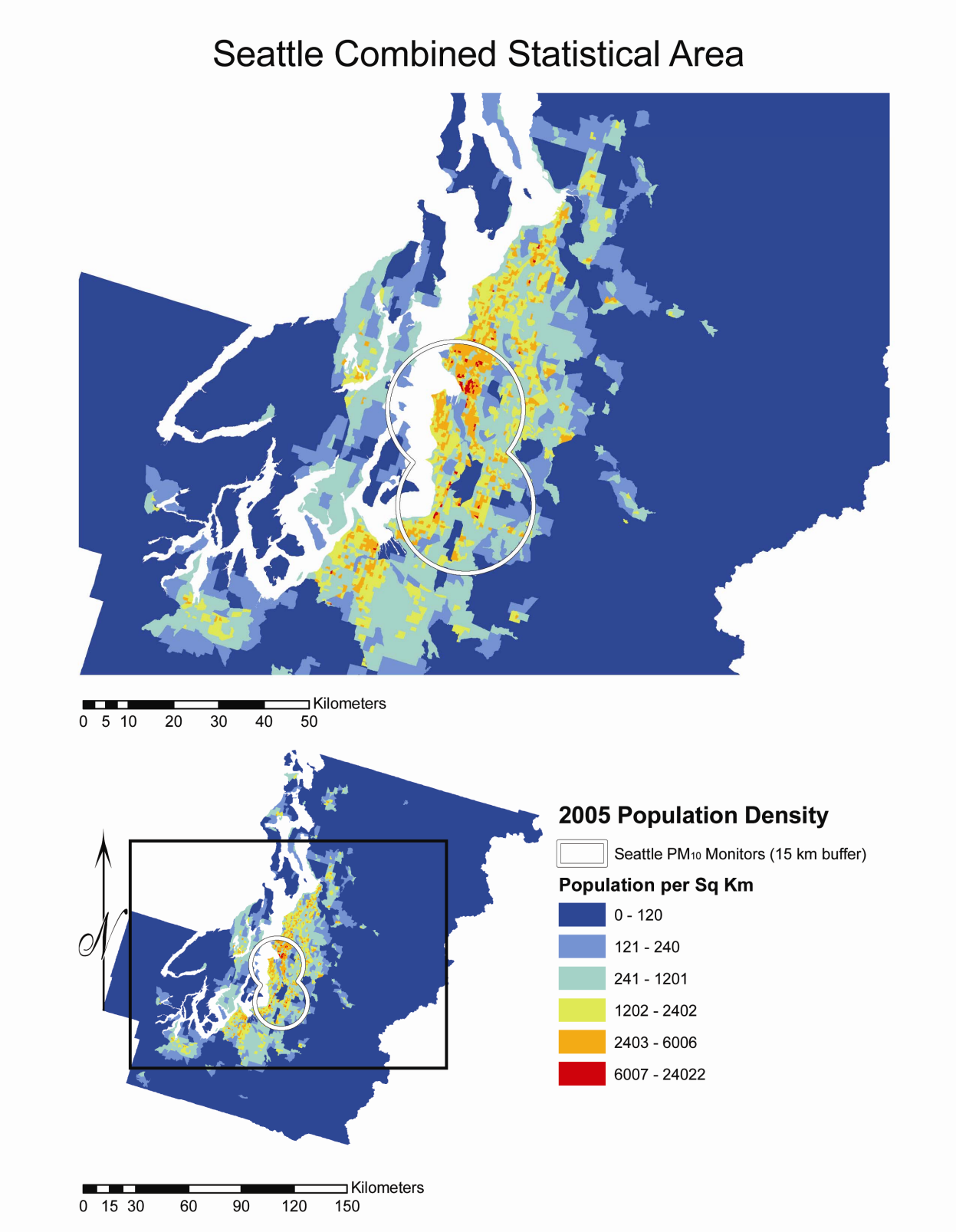


Figure A-28. PM_{10} monitor distribution in comparison with population density, Seattle, WA.

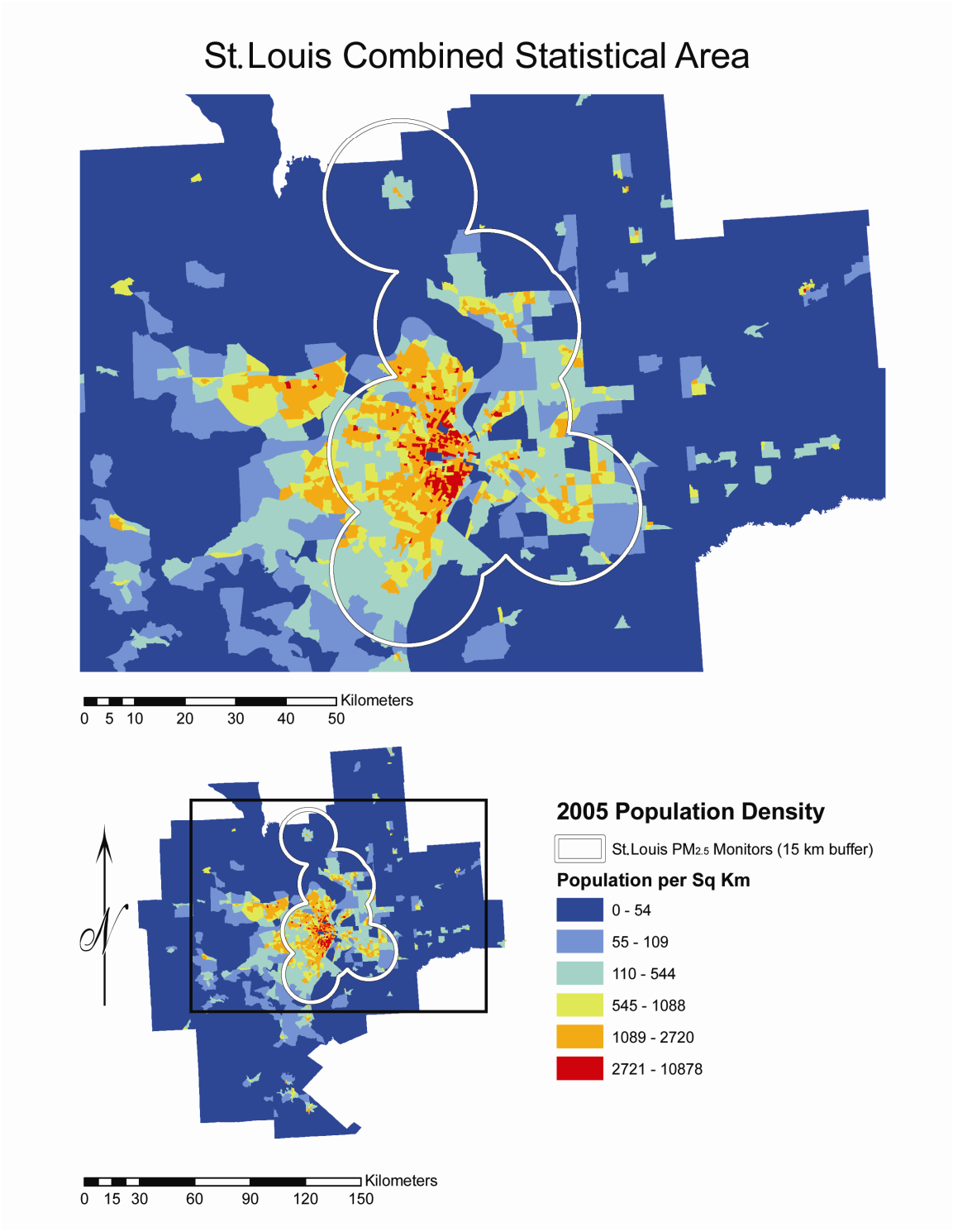


Figure A-29. $PM_{2.5}$ monitor distribution in comparison with population density, St. Louis, MO.

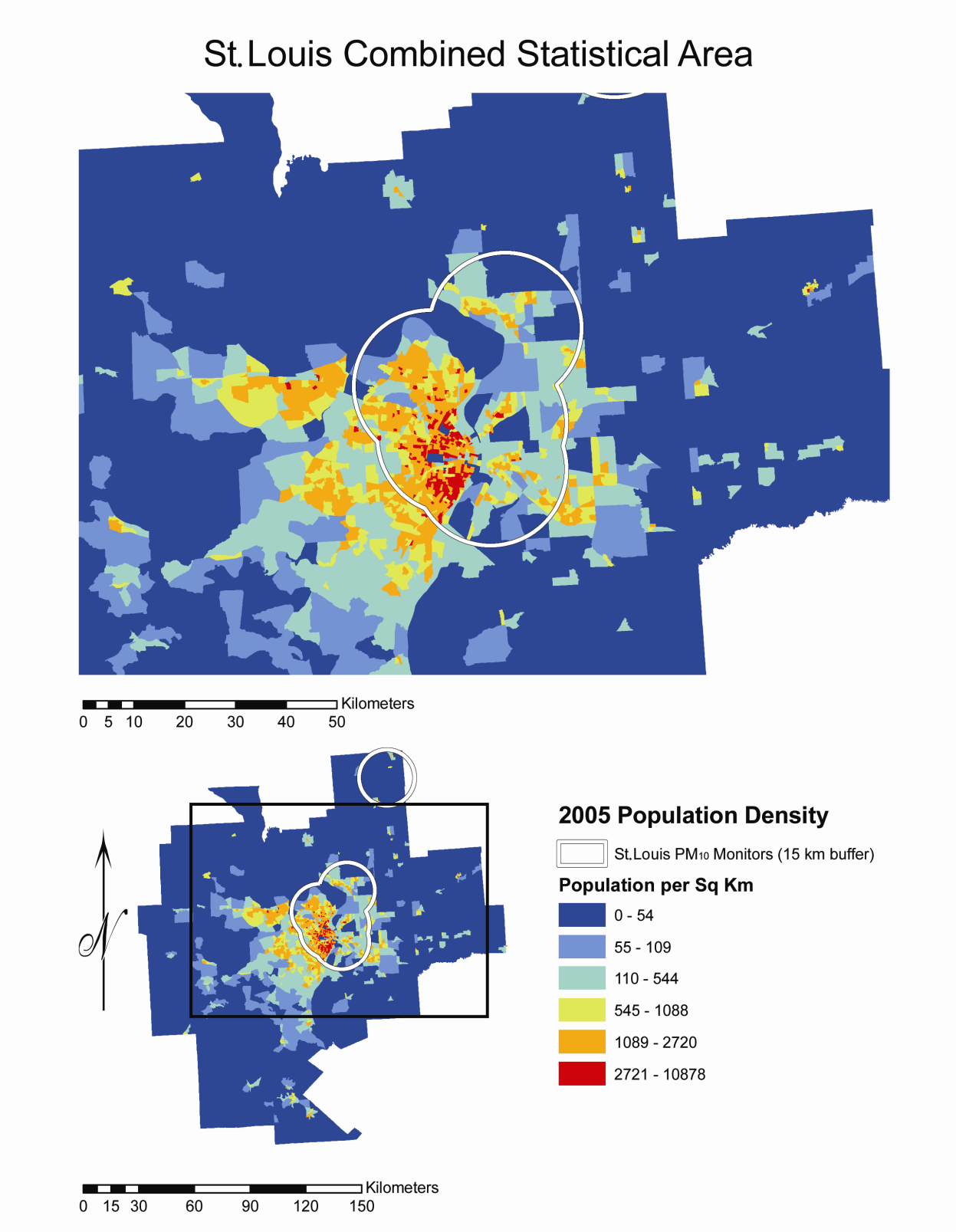


Figure A-30. PM_{10} monitor distribution in comparison with population density, St. Louis, MO.

A.2. Ambient PM Concentration

A.2.1. Speciation Trends Network Site Data

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

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

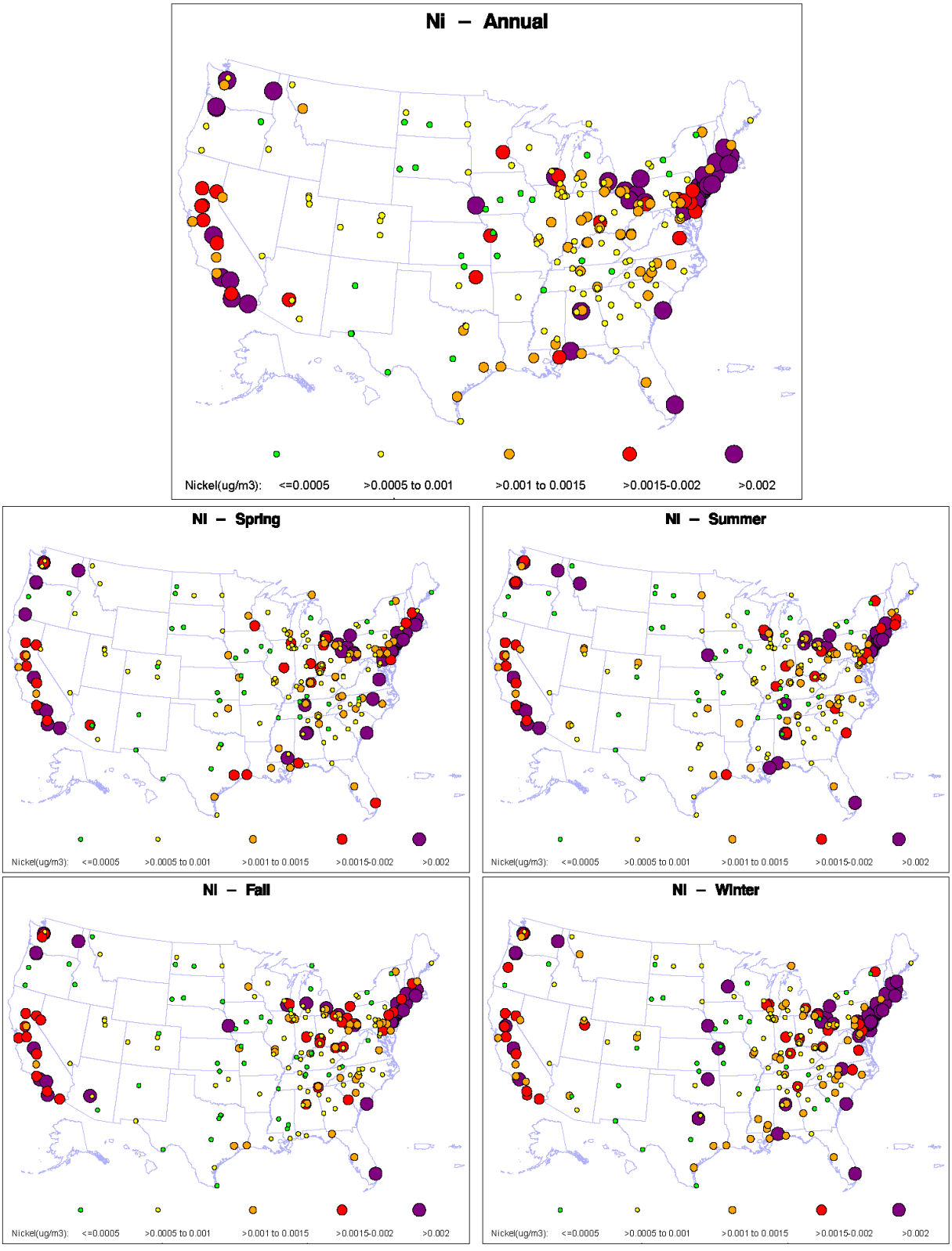


Figure A-33. Three-yr avg of 24-h $PM_{2.5}$ Ni concentrations measured at CSN sites across the U.S., 2005-2007

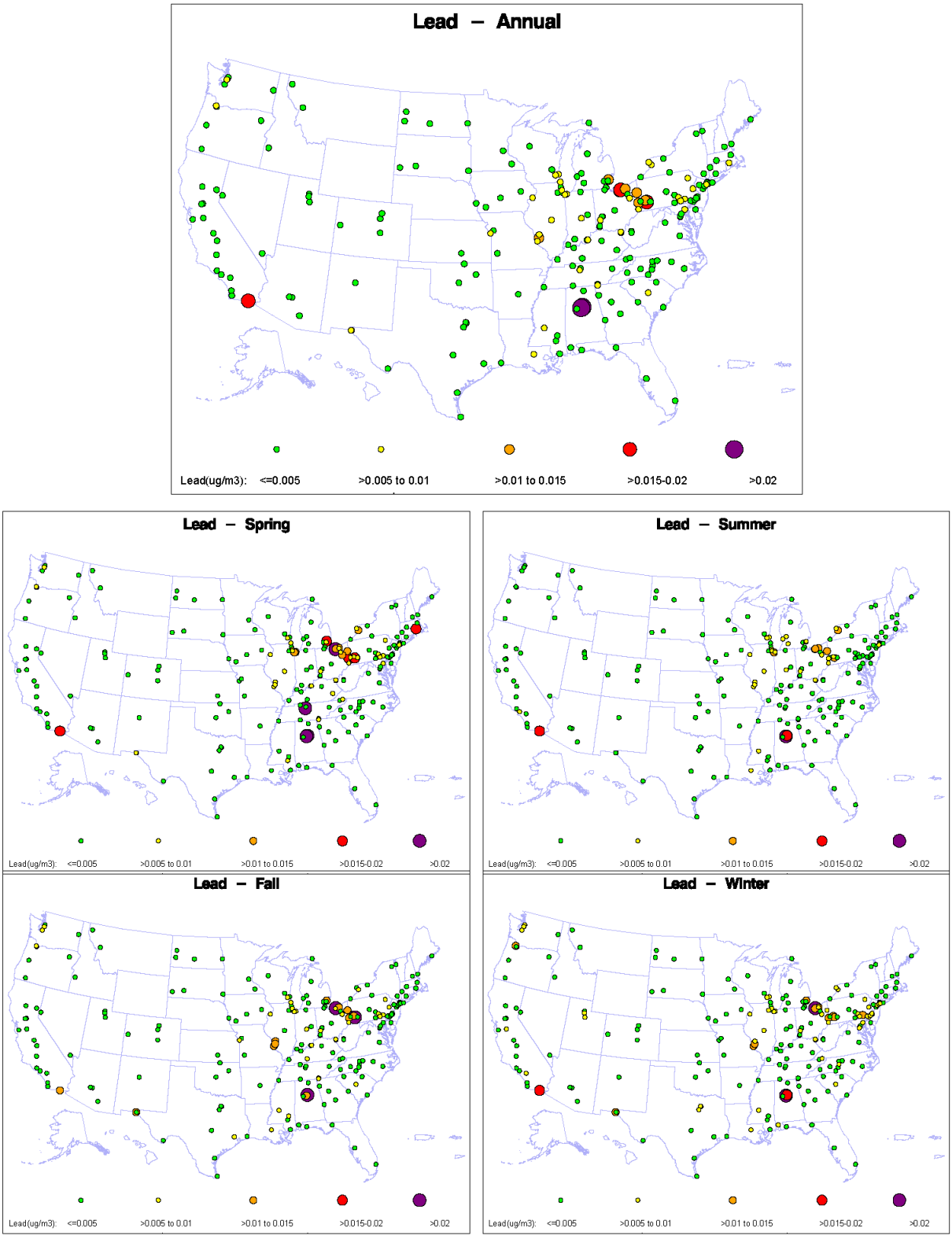


Figure A-34. Three-yr avg of 24-h $PM_{2.5}$ Pb concentrations measured at CSN sites across the U.S., 2005-2007

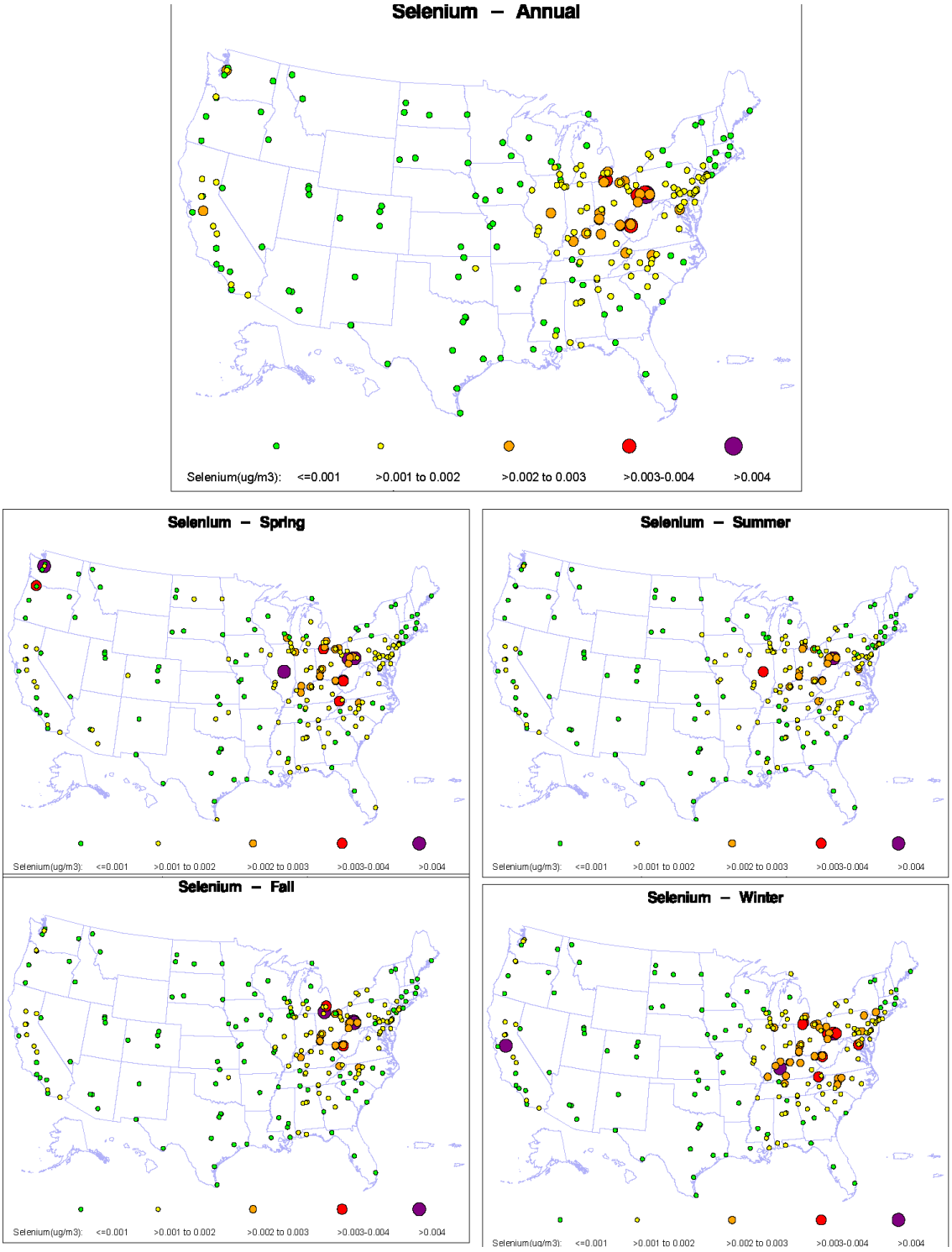


Figure A-35. Three-yr avg of 24-h $PM_{2.5}$ Se concentrations measured at CSN sites across the U.S., 2005-2007

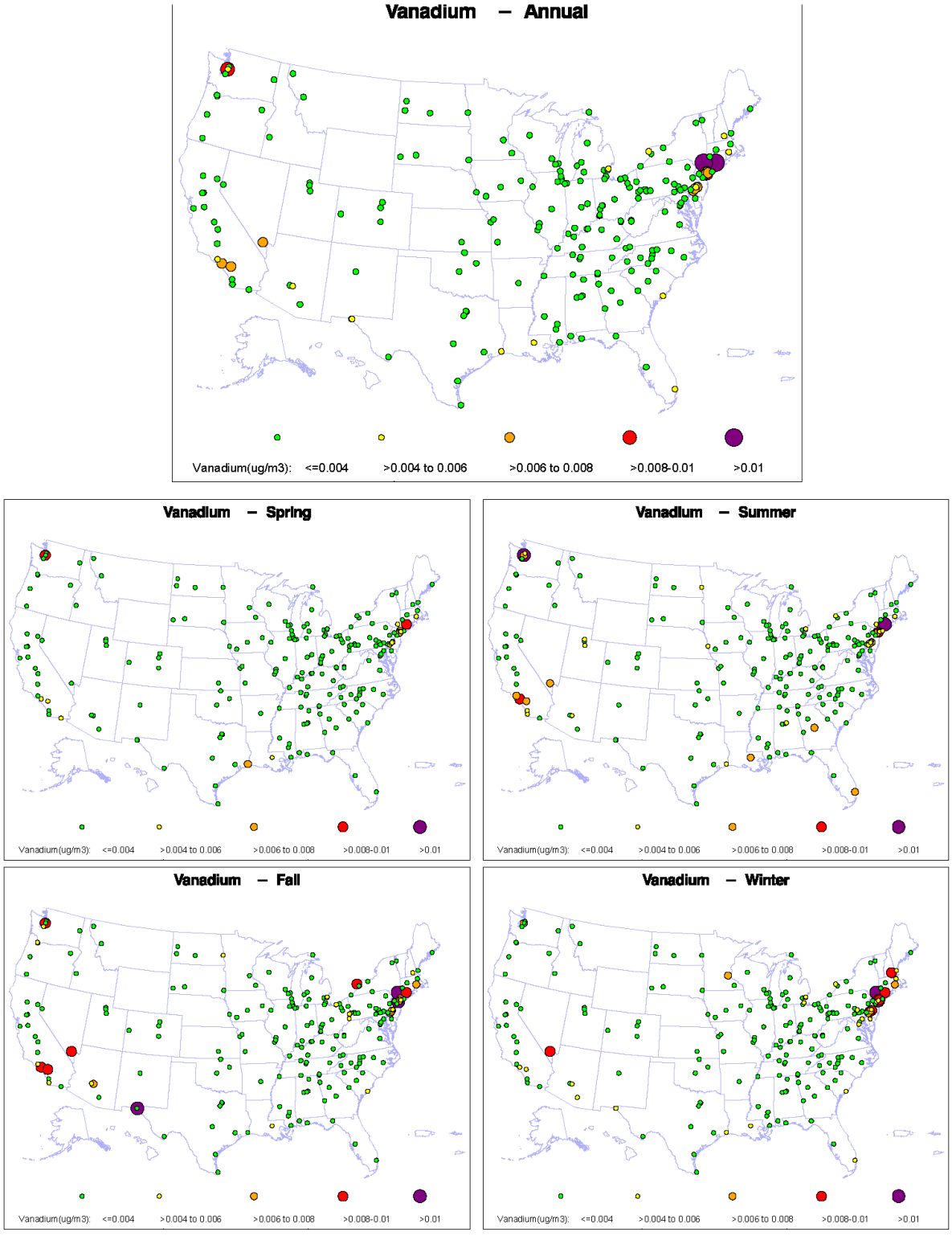


Figure A-36. Three-yr avg of 24-h $PM_{2.5}$ V concentrations measured at CSN sites across the U.S., 2005-2007

A.2.2. Intraurban Variability

The following figures and tables exemplify the intraurban variability among $PM_{2.5}$, $PM_{10-2.5}$ 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.

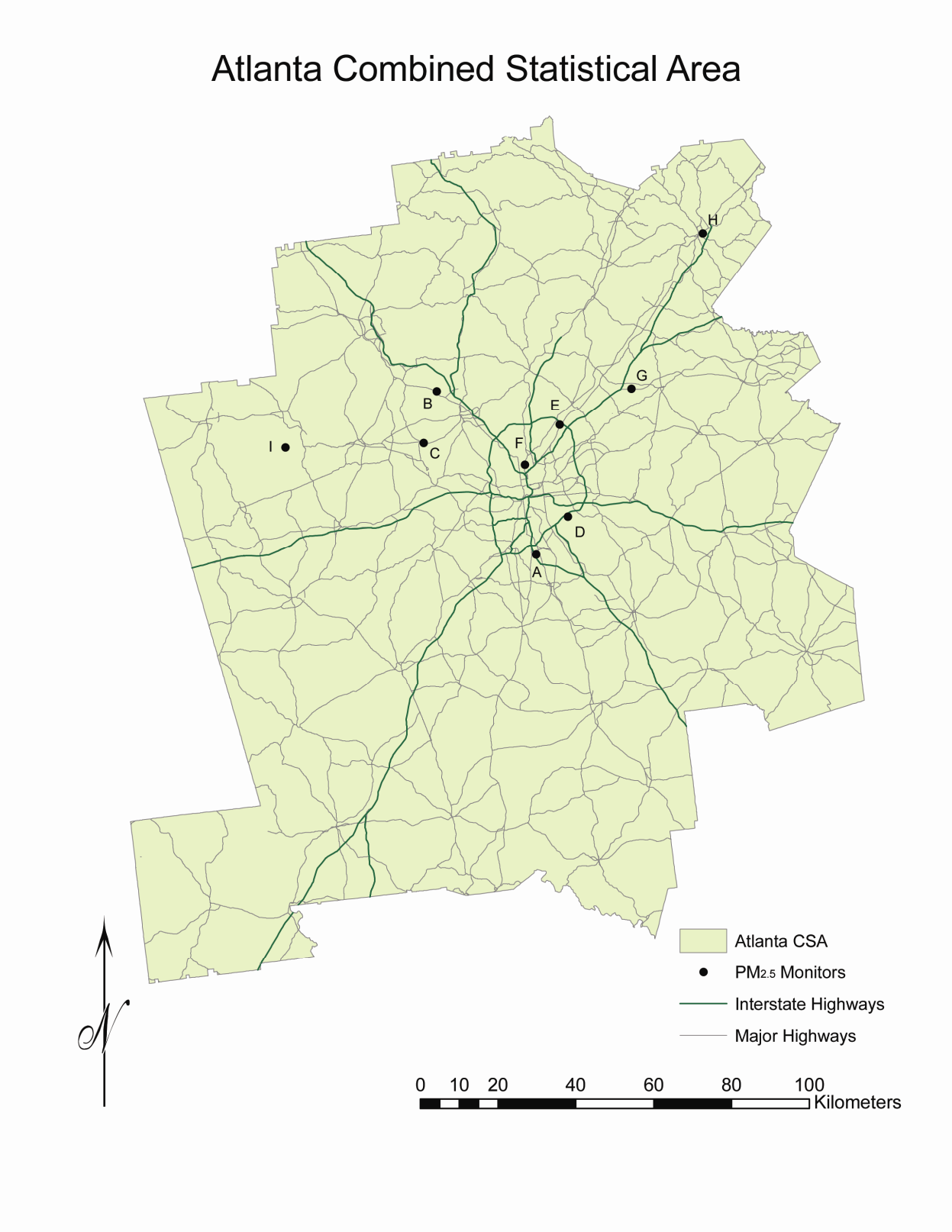


Figure A-37. $PM_{2.5}$ monitor distribution and major highways, Atlanta, GA.

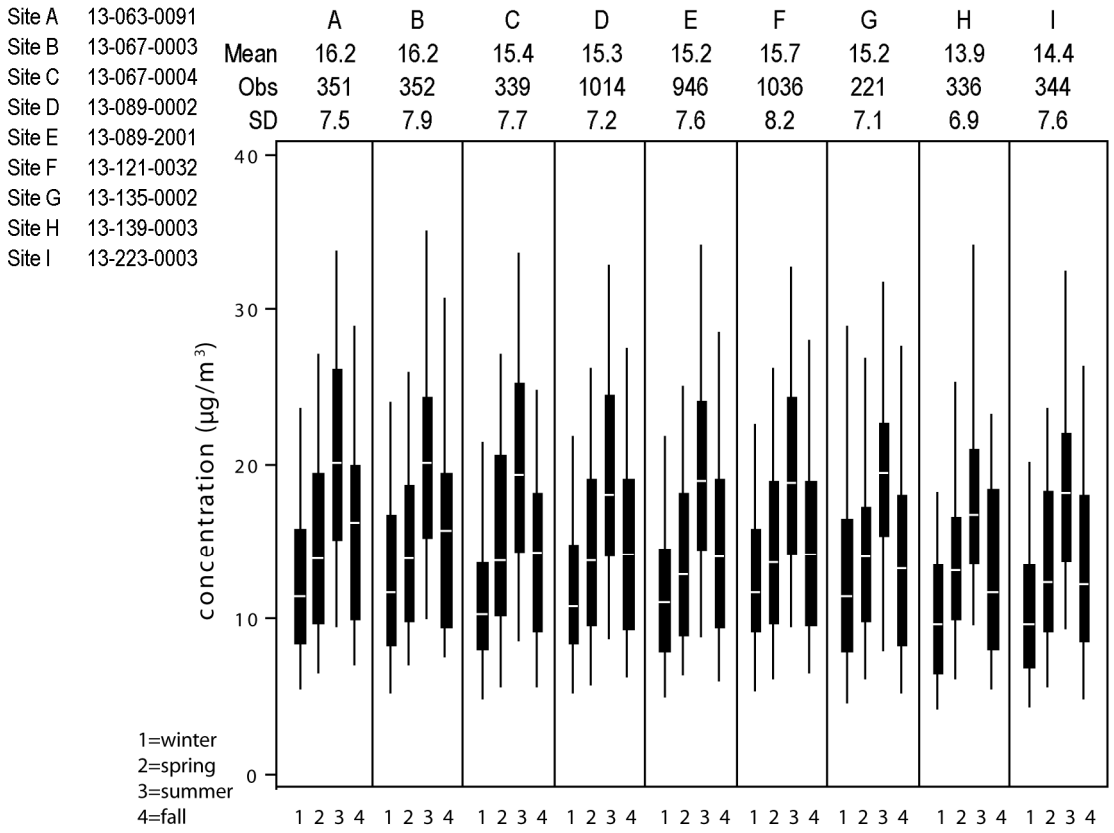


Figure A-38. Box plots illustrating the seasonal distribution of 24-h avg $PM_{2.5}$ concentrations for Atlanta, GA.

Table A-20. 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				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)
		LEGEND			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

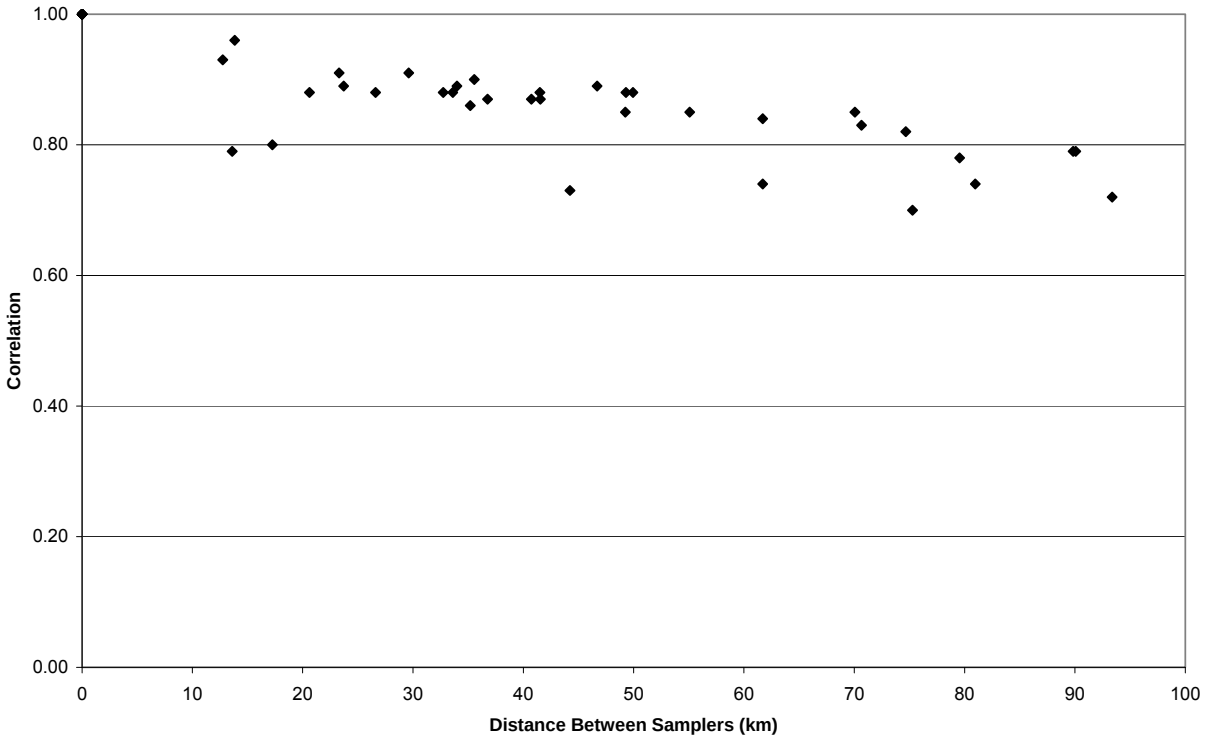


Figure A-39. $PM_{2.5}$ inter-sampler correlations as a function of distance between monitors for Atlanta, GA.

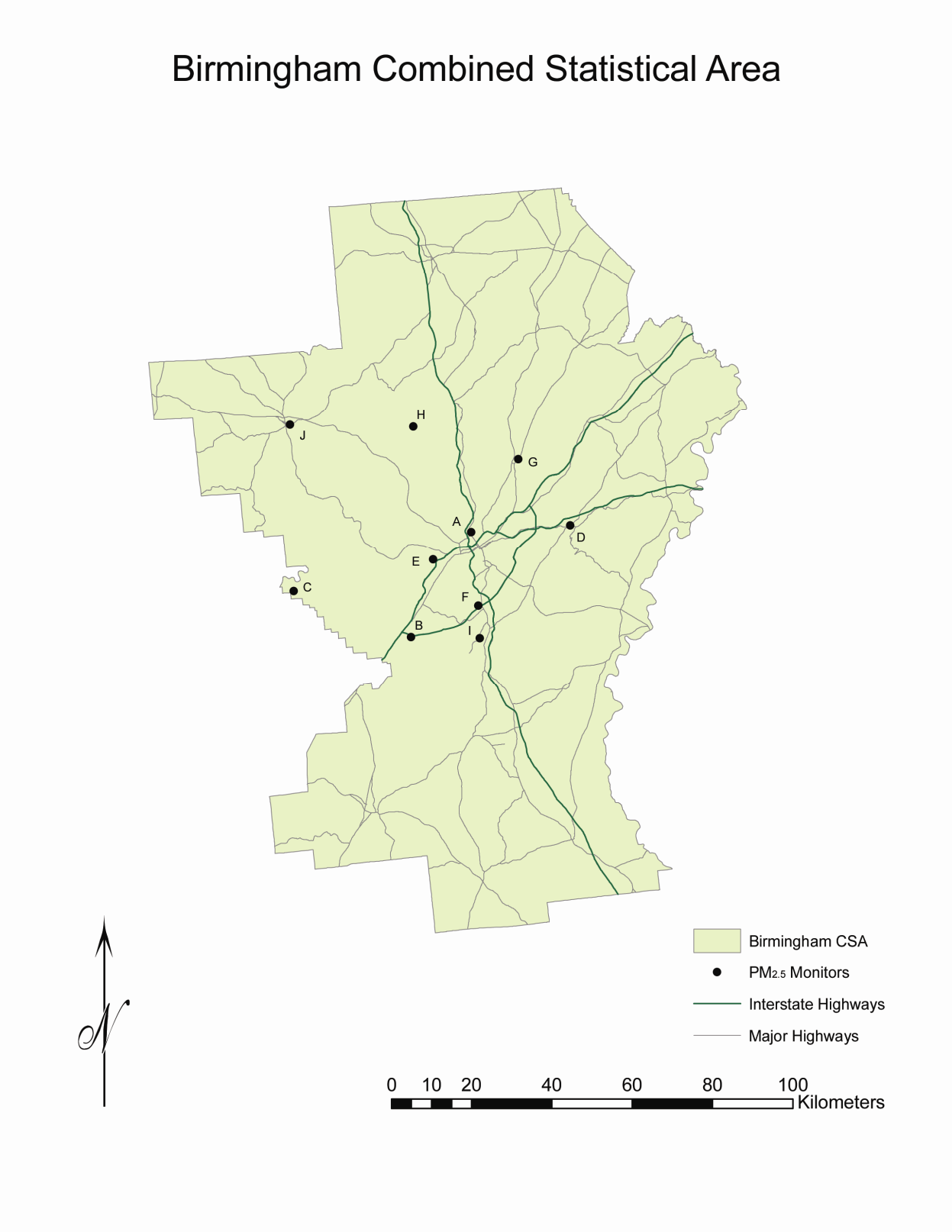


Figure A-40. $PM_{2.5}$ monitor distribution and major highways, Birmingham, AL.

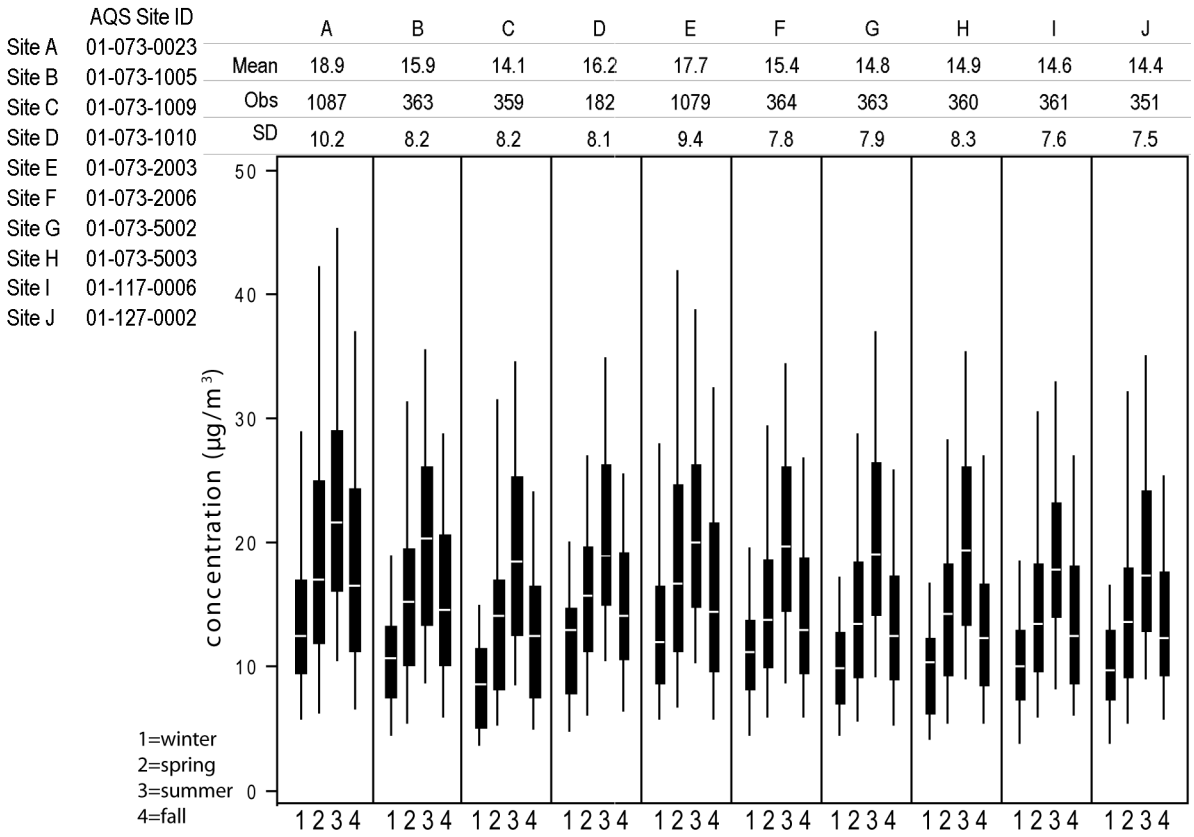


Figure A-41. Box plots illustrating the seasonal distribution of 24-h avg $PM_{2.5}$ concentrations for Birmingham, AL.

Table A-21. 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)
		LEGEND			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

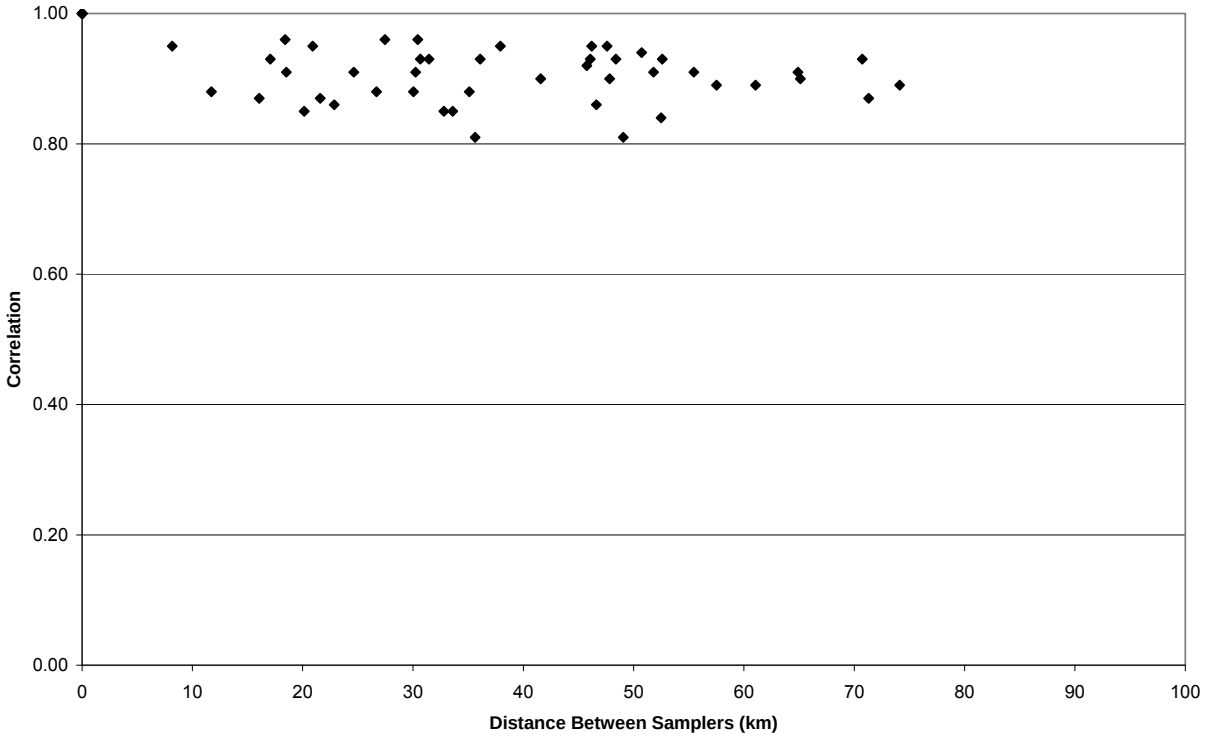


Figure A-42. $PM_{2.5}$ inter-sampler correlations as a function of distance between monitors for Birmingham, AL.

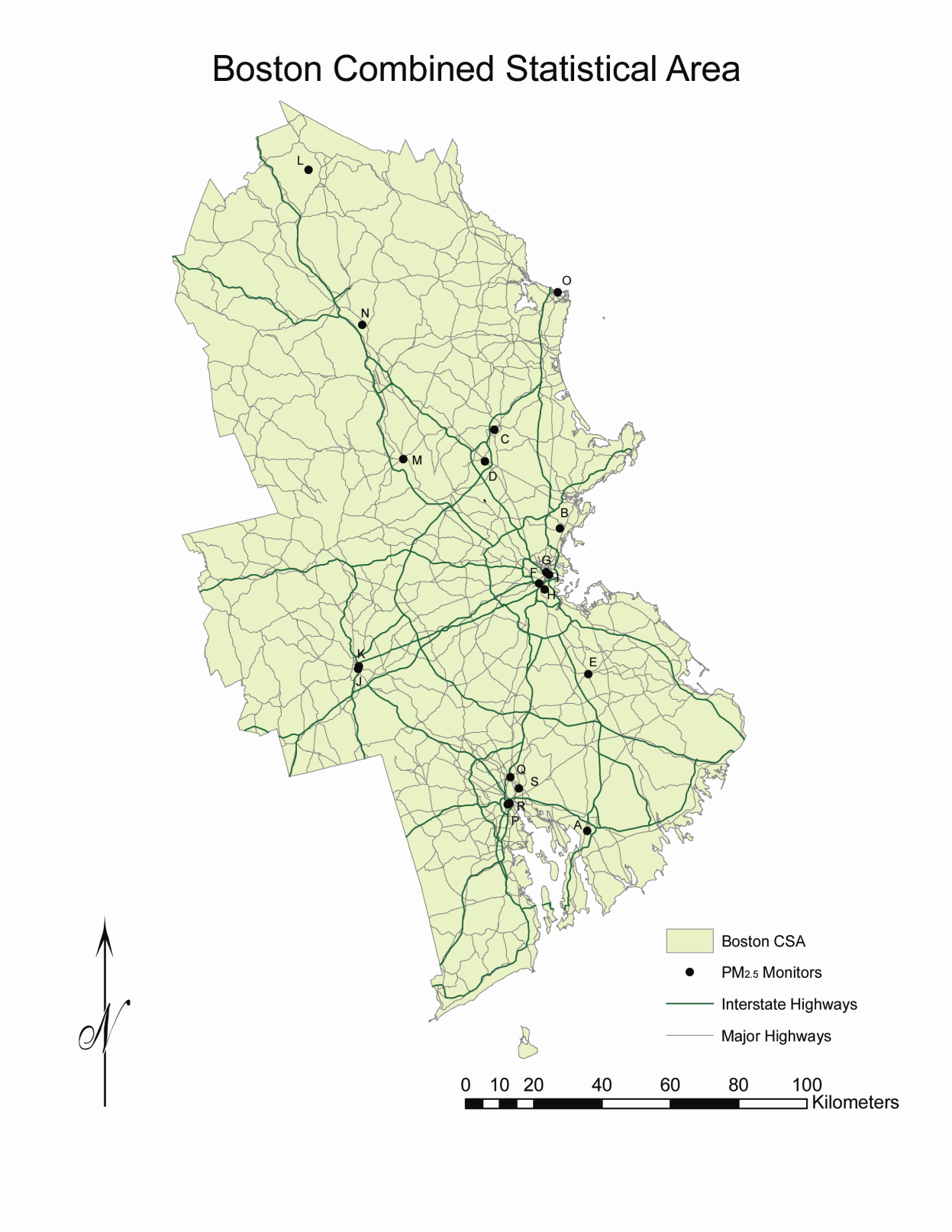


Figure A-43. $PM_{2.5}$ monitor distribution and major highways, Boston, MA.

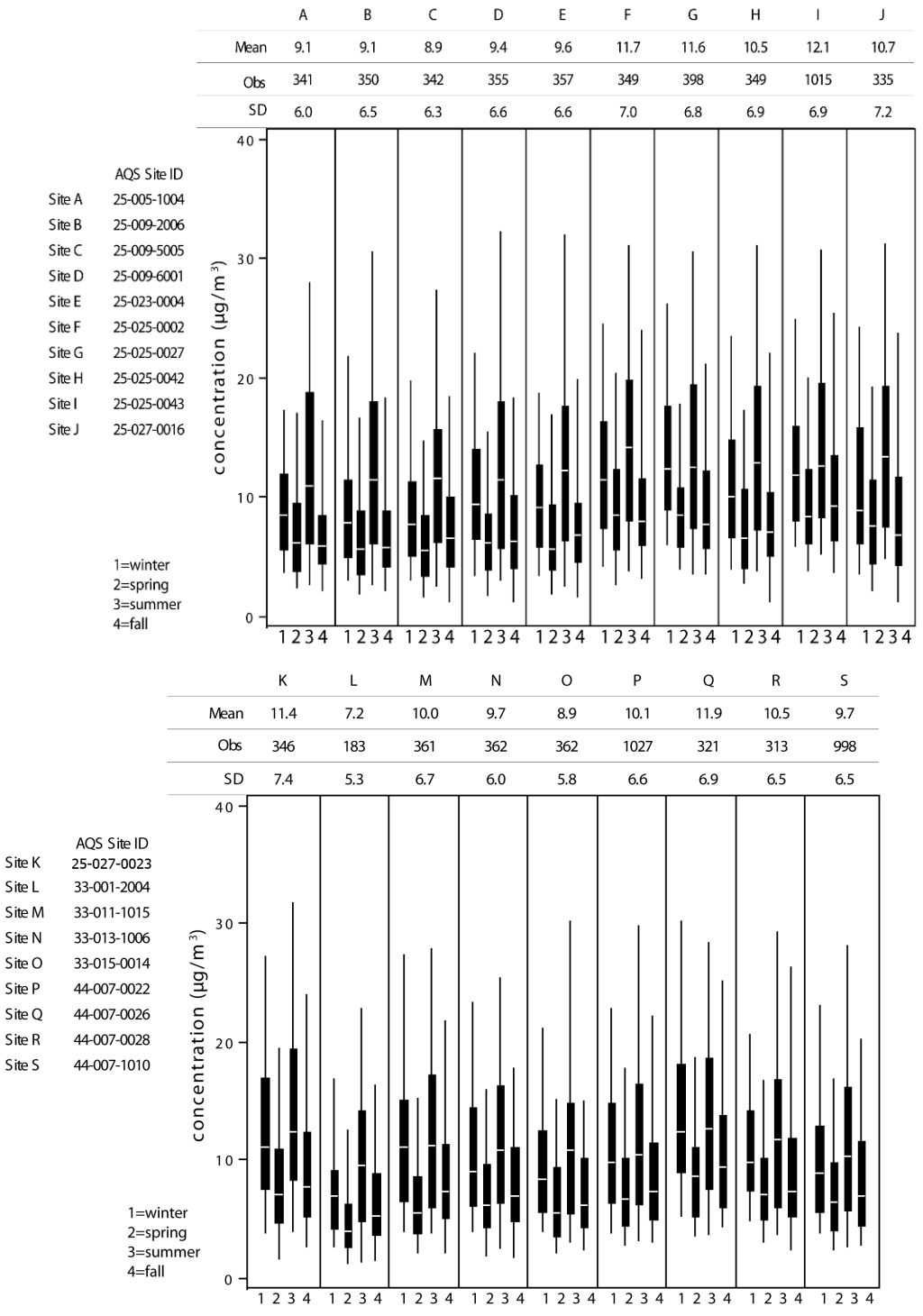


Figure A-44. Box plots illustrating the seasonal distribution of 24-h avg $PM_{2.5}$ concentrations for Boston, MA.

Table A-22. Inter-sampler correlation statistics for each pair of $PM_{2.5}$ monitors reporting to AQS for Boston, MA.

Site	A	B	C	D	E	F	G	H	I	J
A	1.00	0.80	0.77	0.71	0.84	0.79	0.78	0.79	0.79	0.77
	(0.0, 0.00)	(6.6, 0.21)	(6.2, 0.22)	(6.9, 0.23)	(4.8, 0.19)	(8.1, 0.23)	(7.7, 0.24)	(6.8, 0.22)	(7.9, 0.25)	(7.5, 0.24)
	341	326	318	323	329	318	319	325	338	310
B		1.00	0.92	0.87	0.87	0.90	0.90	0.90	0.90	0.85
		(0.0, 0.00)	(4.1, 0.17)	(4.1, 0.18)	(4.7, 0.19)	(6.3, 0.21)	(6.2, 0.23)	(4.9, 0.19)	(7.1, 0.26)	(5.5, 0.21)
		350	328	331	339	326	323	333	343	317
C			1.00	0.90	0.85	0.90	0.89	0.90	0.88	0.86
			(0.0, 0.00)	(3.5, 0.17)	(5.3, 0.21)	(6.3, 0.23)	(6.3, 0.24)	(5.0, 0.20)	(6.8, 0.26)	(6.2, 0.21)
			342	321	331	316	318	326	336	311
D				1.00	0.80	0.88	0.88	0.86	0.86	0.87
				(0.0, 0.00)	(5.6, 0.20)	(5.8, 0.21)	(5.8, 0.22)	(4.6, 0.19)	(7.0, 0.26)	(5.8, 0.19)
				355	336	324	329	332	345	313
E					1.00	0.90	0.90	0.89	0.87	0.87
					(0.0, 0.00)	(5.9, 0.19)	(5.8, 0.21)	(5.0, 0.19)	(6.9, 0.24)	(5.4, 0.20)
					357	330	333	340	350	322
F						1.00	0.94	0.94	0.92	0.92
						(0.0, 0.00)	(3.8, 0.14)	(3.5, 0.15)	(4.5, 0.17)	(5.4, 0.18)
						349	324	324	339	310
G							1.00	0.94	0.94	0.89
							(0.0, 0.00)	(4.0, 0.16)	(4.3, 0.15)	(5.7, 0.20)
							398	325	338	308
H								1.00	0.93	0.89
								(0.0, 0.00)	(4.7, 0.19)	(5.0, 0.17)
								349	342	318
I									1.00	0.86
									(0.0, 0.00)	(6.9, 0.23)
									1015	330
J										1.00
										(0.0, 0.00)
										335

LEGEND
Pearson R
(P90, COD)
n

Site	K	L	M	N	O	P	Q	R	S
A	0.77	0.61	0.71	0.68	0.73	0.87	0.81	0.85	0.86
	(8.1, 0.23)	(8.3, 0.29)	(8.0, 0.23)	(7.9, 0.23)	(7.0, 0.22)	(5.3, 0.18)	(7.2, 0.23)	(5.6, 0.20)	(5.2, 0.18)
	320	173	324	334	331	326	292	285	306
B	0.86	0.80	0.87	0.83	0.88	0.86	0.80	0.85	0.85
	(6.6, 0.21)	(6.2, 0.23)	(5.3, 0.19)	(6.0, 0.21)	(4.7, 0.18)	(5.6, 0.19)	(7.9, 0.26)	(5.7, 0.21)	(6.0, 0.19)
	329	175	331	341	336	335	300	288	314
C	0.86	0.89	0.93	0.90	0.93	0.83	0.79	0.81	0.82
	(6.9, 0.21)	(4.8, 0.23)	(4.4, 0.17)	(4.6, 0.19)	(3.8, 0.18)	(5.9, 0.21)	(7.8, 0.26)	(6.2, 0.23)	(6.0, 0.21)
	321	173	323	335	328	329	290	281	309
D	0.88	0.79	0.91	0.85	0.86	0.80	0.75	0.79	0.80
	(6.4, 0.19)	(5.7, 0.25)	(3.5, 0.16)	(4.7, 0.19)	(4.2, 0.18)	(6.2, 0.20)	(7.8, 0.25)	(6.2, 0.21)	(5.8, 0.20)
	325	174	329	339	334	342	300	287	321
E	0.87	0.72	0.83	0.79	0.84	0.91	0.86	0.88	0.91
	(6.3, 0.20)	(8.3, 0.27)	(5.8, 0.17)	(6.3, 0.20)	(4.8, 0.18)	(4.5, 0.17)	(6.3, 0.22)	(4.9, 0.18)	(3.9, 0.17)
	333	179	338	347	343	343	306	295	324
F	0.91	0.78	0.90	0.85	0.85	0.89	0.86	0.88	0.89
	(4.7, 0.17)	(9.6, 0.33)	(5.3, 0.18)	(6.4, 0.20)	(7.5, 0.22)	(5.2, 0.16)	(6.0, 0.16)	(4.9, 0.16)	(5.5, 0.17)
	323	168	323	334	330	336	295	281	316
G	0.90	0.77	0.90	0.85	0.87	0.88	0.86	0.87	0.88
	(5.0, 0.19)	(9.0, 0.33)	(5.3, 0.19)	(6.3, 0.20)	(7.0, 0.22)	(5.5, 0.17)	(5.3, 0.17)	(5.2, 0.17)	(5.7, 0.19)
	320	172	326	335	329	383	296	282	356
H	0.90	0.75	0.88	0.83	0.84	0.89	0.86	0.87	0.88
	(4.4, 0.17)	(9.4, 0.30)	(4.9, 0.18)	(5.6, 0.21)	(6.8, 0.21)	(4.5, 0.16)	(6.0, 0.19)	(4.5, 0.16)	(5.1, 0.17)
	327	175	332	341	336	335	299	289	314
I	0.87	0.75	0.86	0.82	0.83	0.88	0.84	0.85	0.87
	(6.1, 0.20)	(10.0, 0.36)	(6.7, 0.22)	(7.2, 0.23)	(8.2, 0.25)	(6.1, 0.20)	(6.0, 0.16)	(6.0, 0.18)	(6.3, 0.21)
	341	181	352	356	357	957	314	306	936
J	0.95	0.73	0.87	0.84	0.80	0.90	0.86	0.87	0.88
	(3.0, 0.14)	(9.2, 0.28)	(5.2, 0.18)	(5.9, 0.20)	(7.5, 0.22)	(5.0, 0.17)	(5.9, 0.20)	(5.3, 0.17)	(5.2, 0.18)
	316	167	314	326	323	321	283	272	302
K	1.00	0.71	0.88	0.85	0.81	0.89	0.86	0.87	0.88
	(0.0, 0.00)	(10.3, 0.31)	(6.0, 0.16)	(6.5, 0.19)	(8.2, 0.22)	(5.2, 0.16)	(5.8, 0.18)	(5.5, 0.16)	(5.5, 0.18)
	346	170	326	337	332	331	296	286	313
L		1.00	0.89	0.91	0.90	0.68	0.63	0.72	0.69
		(0.0, 0.00)	(6.7, 0.24)	(5.9, 0.23)	(4.8, 0.21)	(10.0, 0.29)	(12.1, 0.35)	(9.1, 0.30)	(9.8, 0.29)
		183	176	181	177	181	153	149	164
M			1.00	0.94	0.90	0.83	0.81	0.82	0.84
			(0.0, 0.00)	(3.8, 0.13)	(4.6, 0.16)	(5.5, 0.16)	(7.4, 0.20)	(5.8, 0.17)	(5.1, 0.16)
			361	341	336	345	300	288	326
N				1.00	0.90	0.77	0.75	0.78	0.78
				(0.0, 0.00)	(4.4, 0.17)	(6.7, 0.19)	(8.1, 0.22)	(6.4, 0.20)	(6.2, 0.19)
				362	346	347	309	297	327
O					1.00	0.80	0.75	0.79	0.80
					(0.0, 0.00)	(5.8, 0.19)	(8.8, 0.25)	(6.8, 0.21)	(6.0, 0.19)
					362	348	304	292	330
P						1.00	0.95	0.97	0.97
						(0.0, 0.00)	(3.6, 0.14)	(2.0, 0.09)	(2.1, 0.08)

LEGEND
Pearson R
(P90, COD)
n

Site	K	L	M	N	O	P	Q	R	S
						1027	307	299	943
Q							1.00	0.92	0.94
							(0.0, 0.00)	(3.1, 0.13)	(4.0, 0.16)
							321	268	290
R								1.00	0.94
								(0.0, 0.00)	(2.7, 0.12)
								313	280
S									1.00
									(0.0, 0.00)
									998

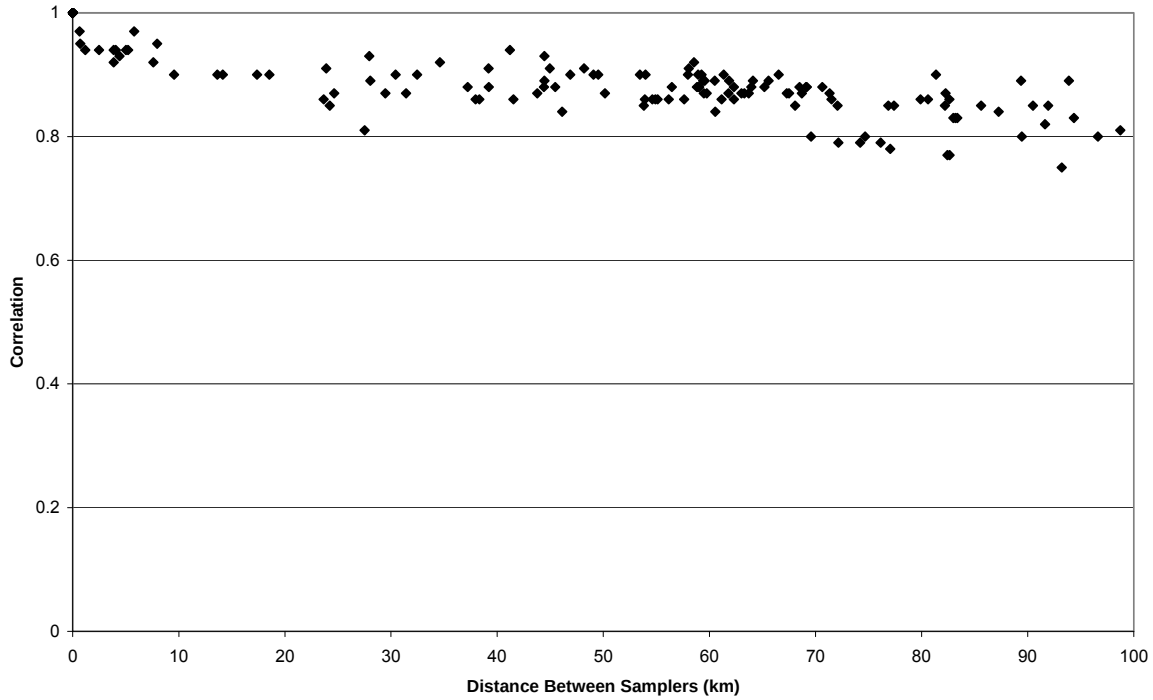


Figure A-45. $PM_{2.5}$ inter-sampler correlations as a function of distance between monitors for Boston, MA.

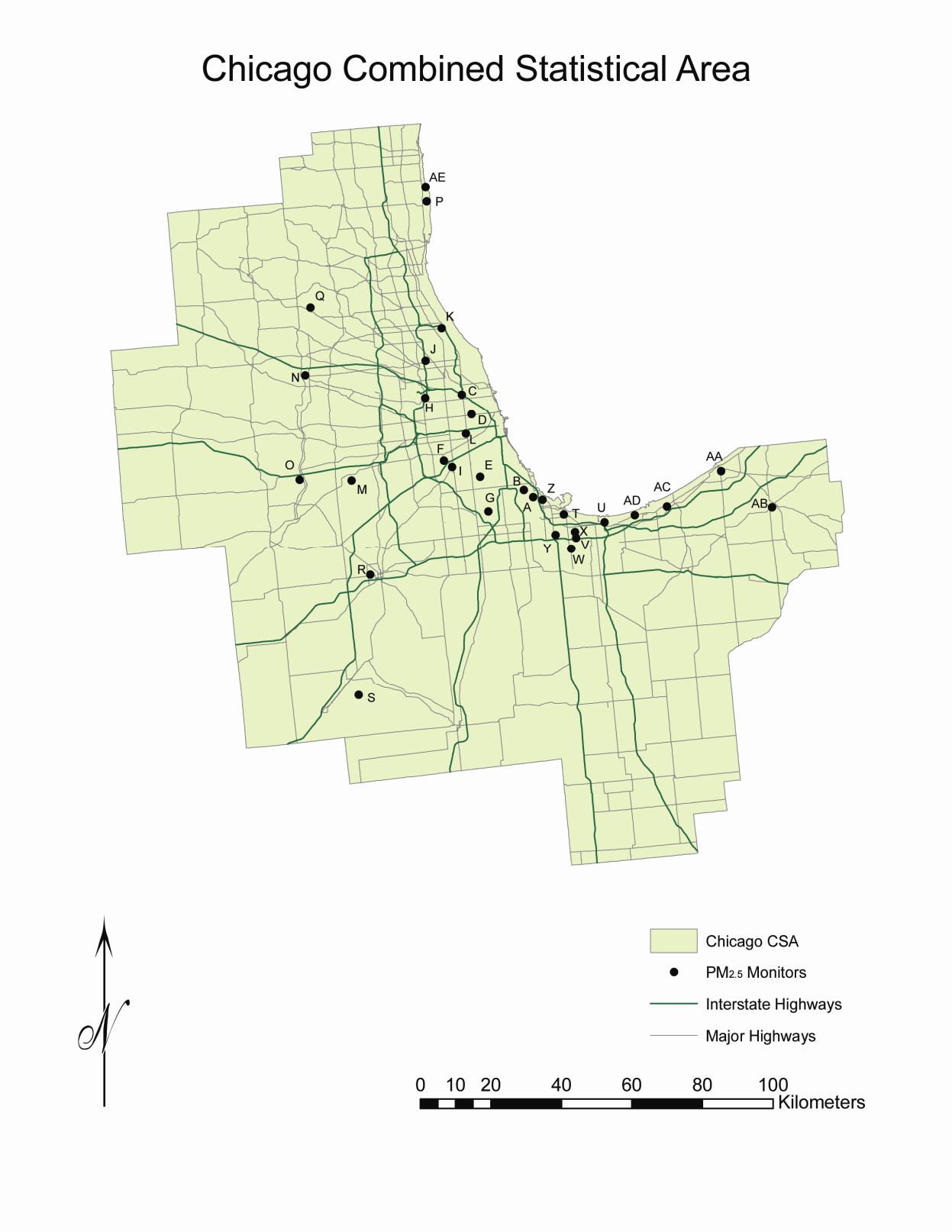
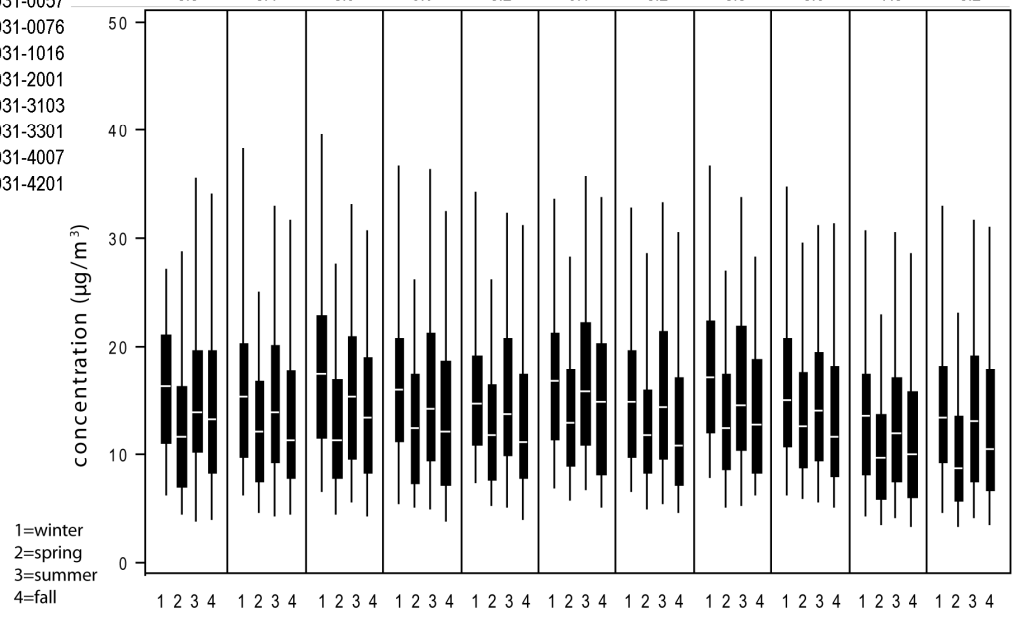
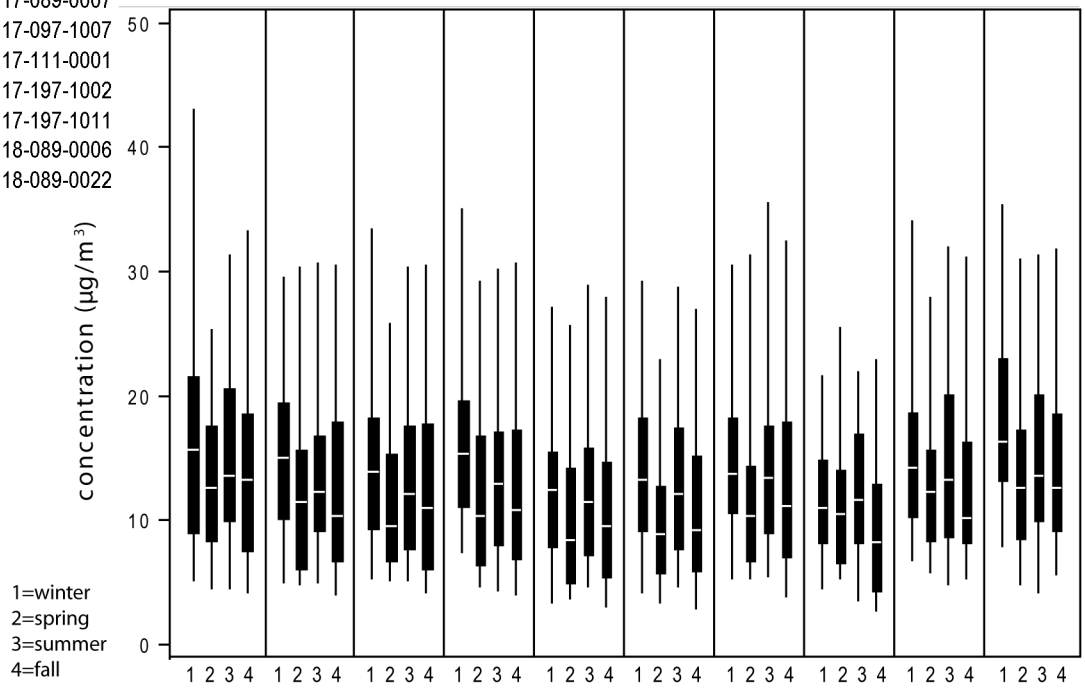


Figure A-46. $PM_{2.5}$ monitor distribution and major highways, Chicago, IL.

	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											



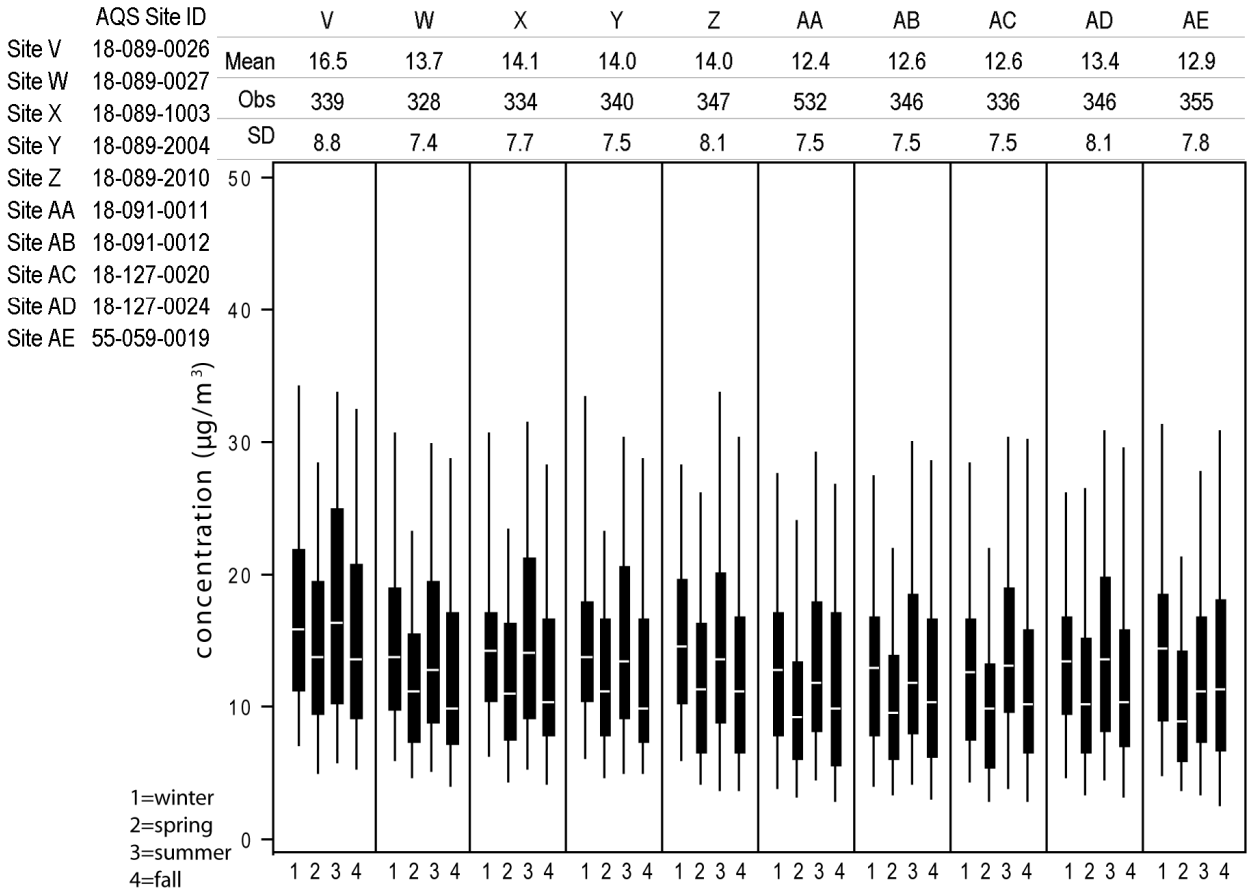


Figure A-47. Box plots illustrating the seasonal distribution of 24-h avg $PM_{2.5}$ concentrations for Chicago, IL.

Table A-23. 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
										(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					**LEGEND**						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

	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)
								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

LEGEND
R
(P90, COD)
N

P	Q	R	S	T	U	V	W	X	Y	Z	AA	AB	AC	AD	AE
															(0.0, 0.00)
															355

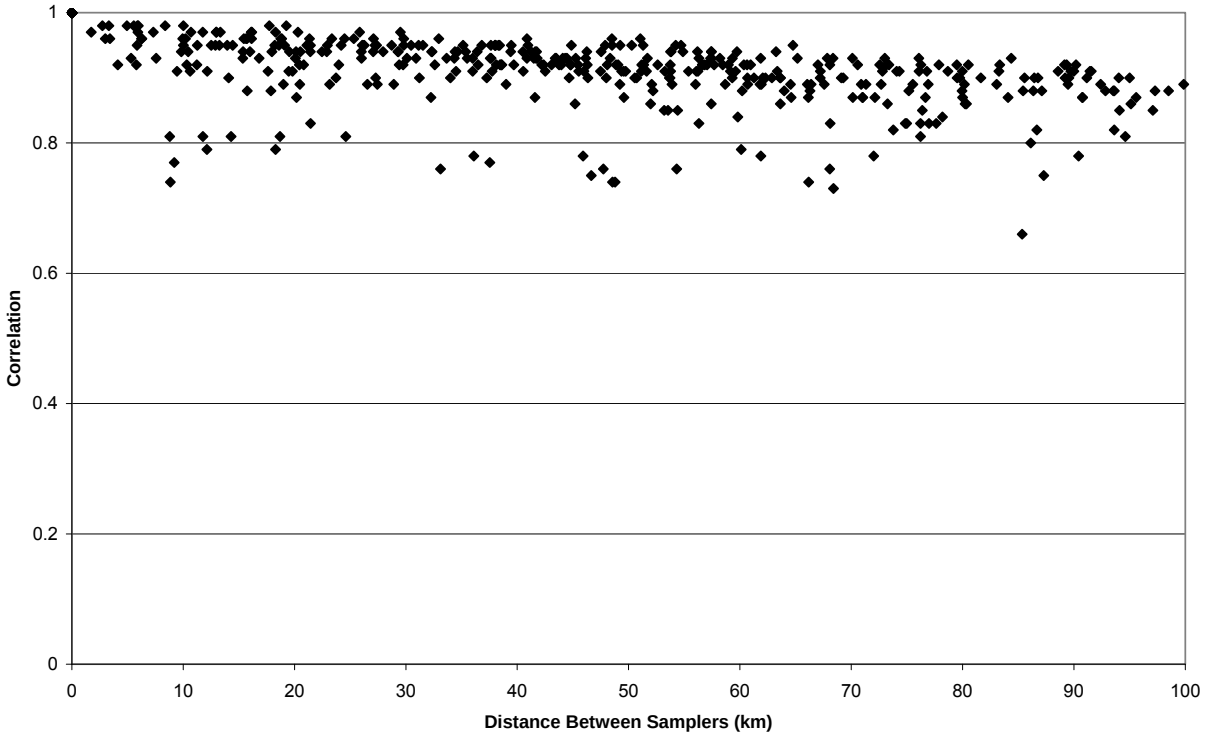


Figure A-48. $PM_{2.5}$ inter-sampler correlations as a function of distance between monitors for Chicago, IL.

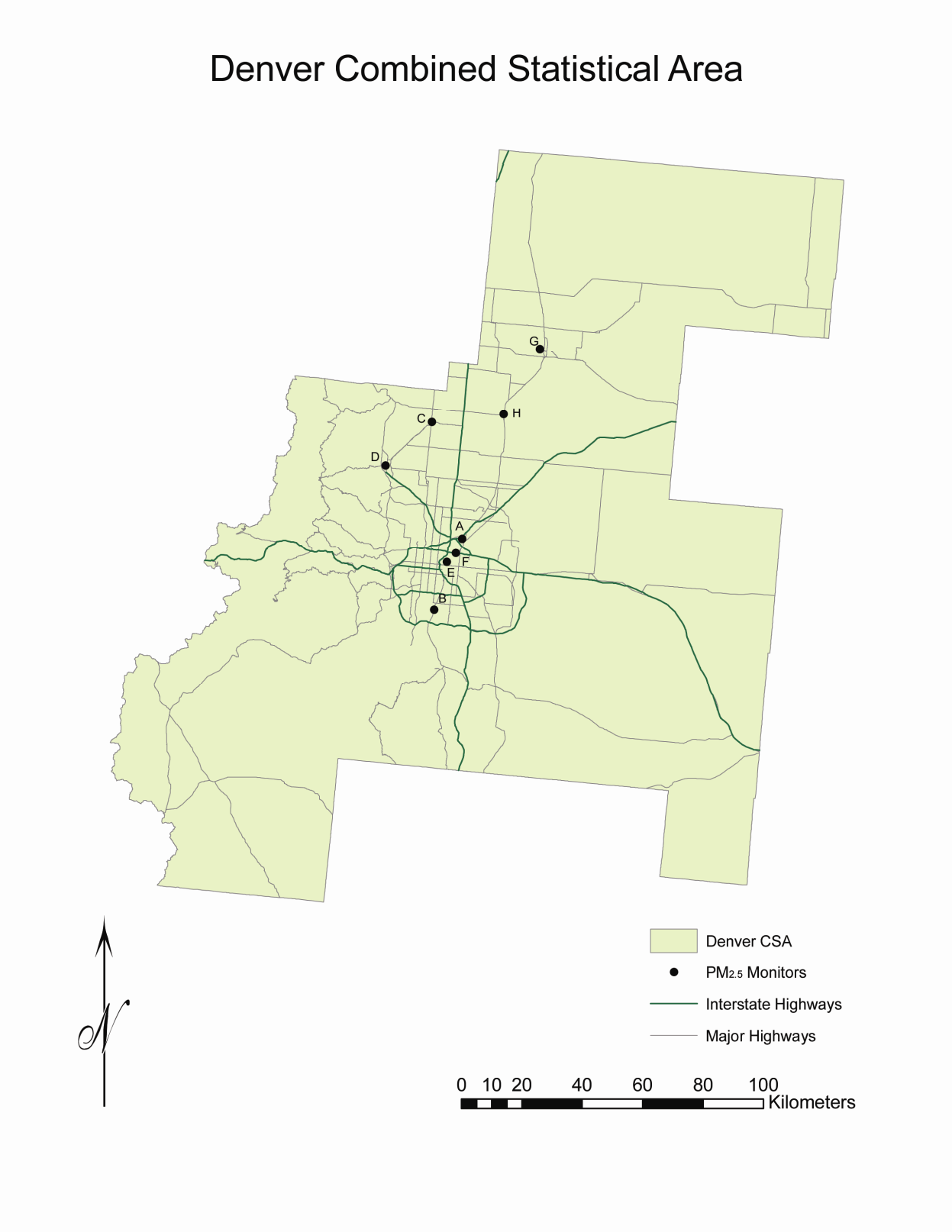


Figure A-49. $PM_{2.5}$ monitor distribution and major highways, Denver, CO.

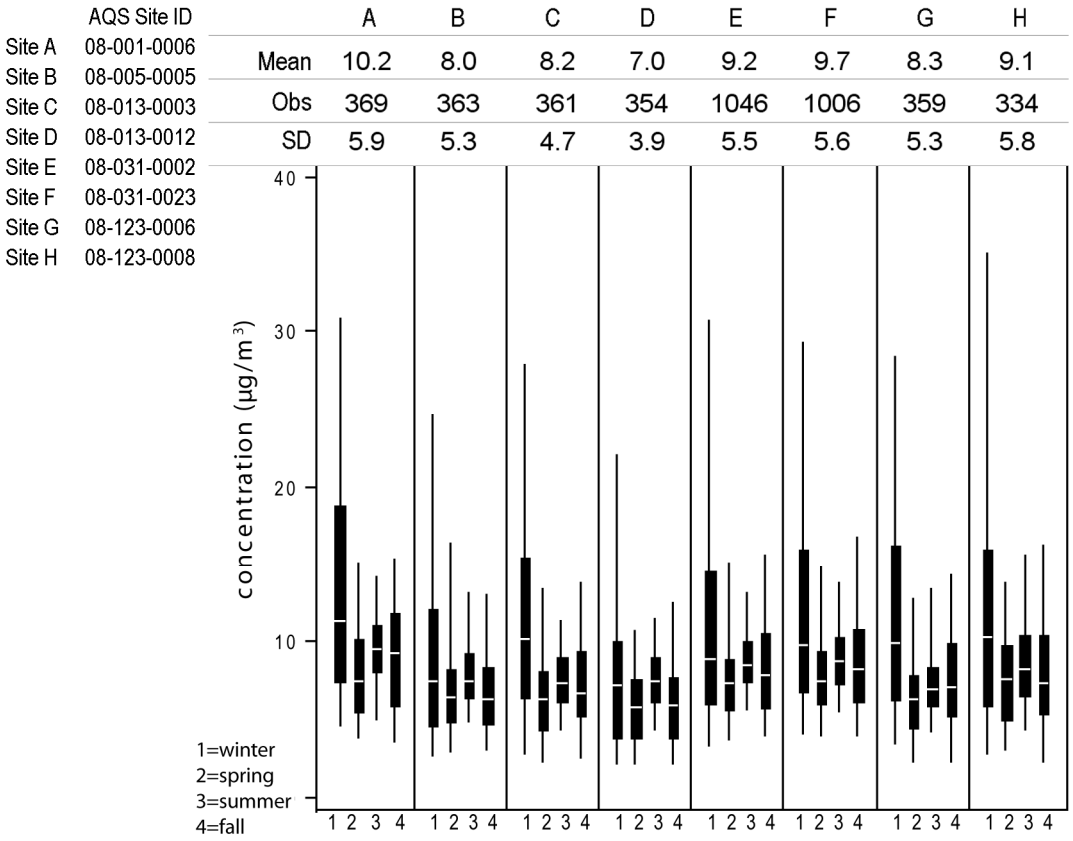


Figure A-50. Box plots illustrating the seasonal distribution of 24-h avg $PM_{2.5}$ concentrations for Denver, CO.

Table A-24. 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
		LEGEND			(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
								(0.0, 0.00)
								334

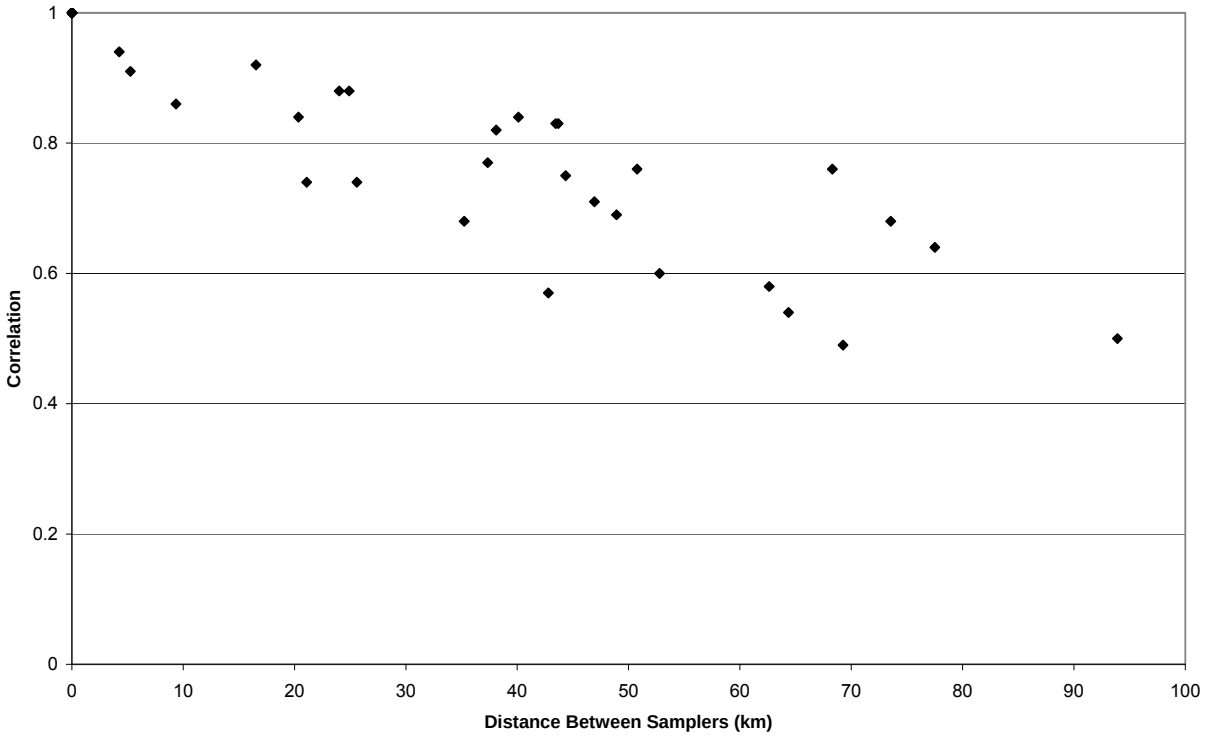


Figure A-51. $PM_{2.5}$ inter-sampler correlations as a function of distance between monitors for Denver, CO.

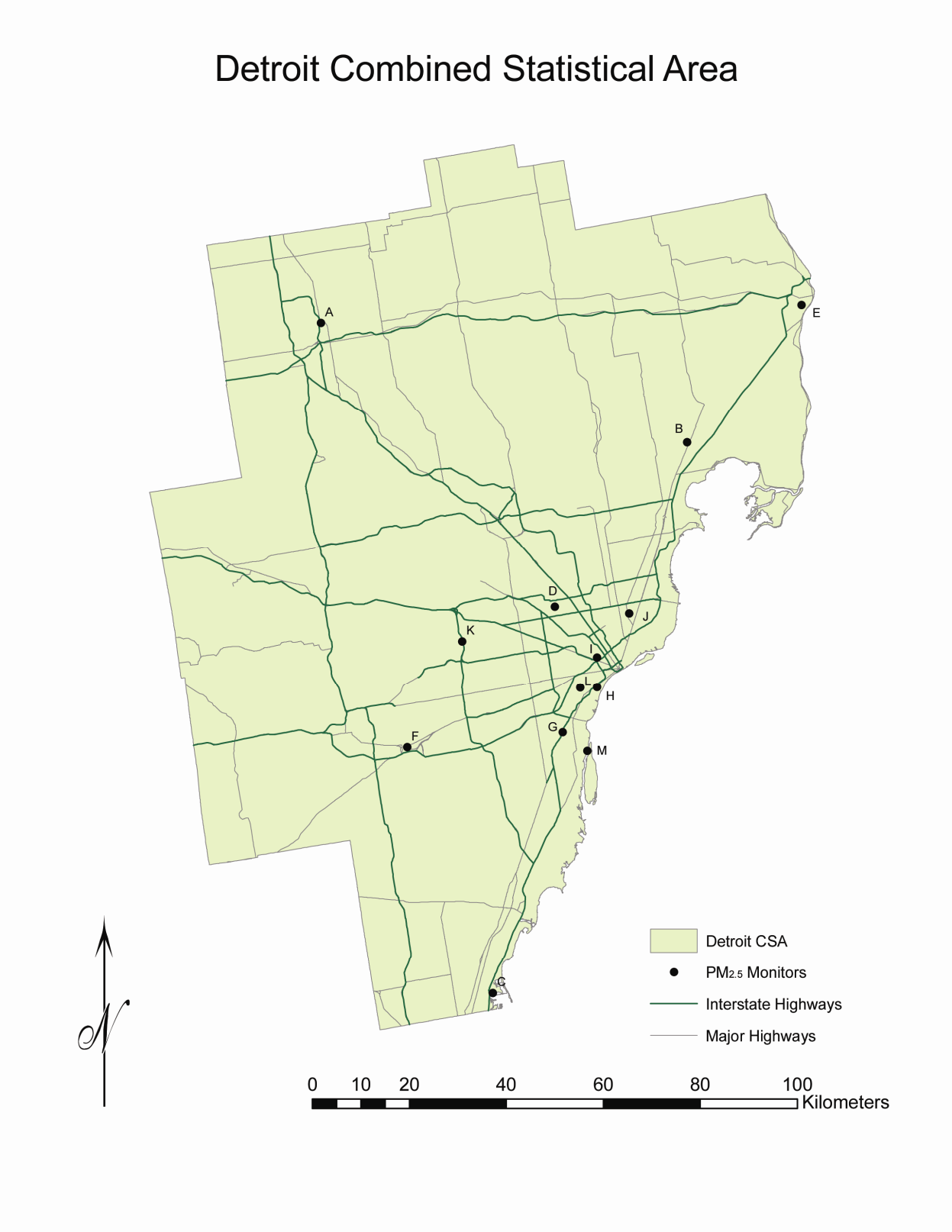


Figure A-52. $PM_{2.5}$ monitor distribution and major highways, Detroit, MI.

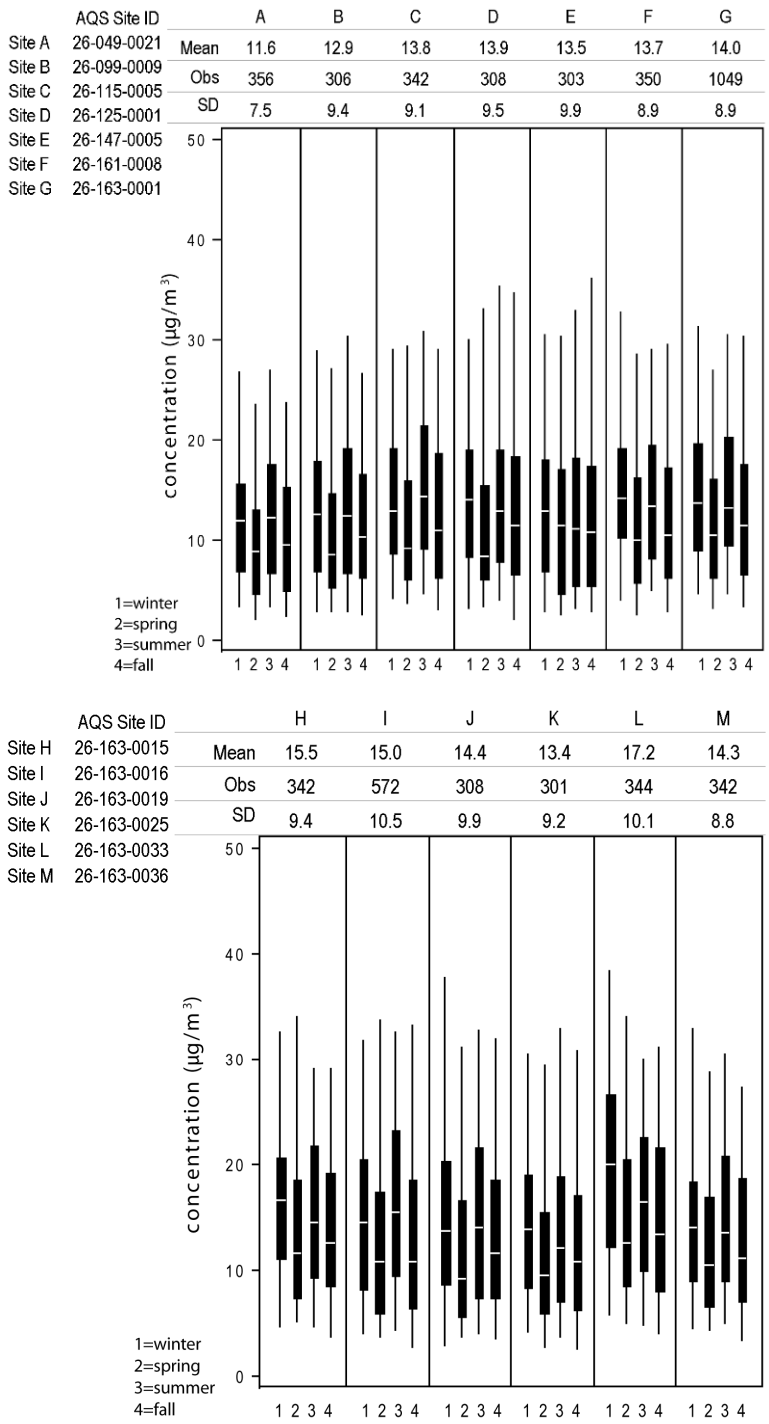


Figure A-53. Box plots illustrating the seasonal distribution of 24-h avg $PM_{2.5}$ concentrations for Detroit, MI.

Table A-25. 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			**LEGEND**						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

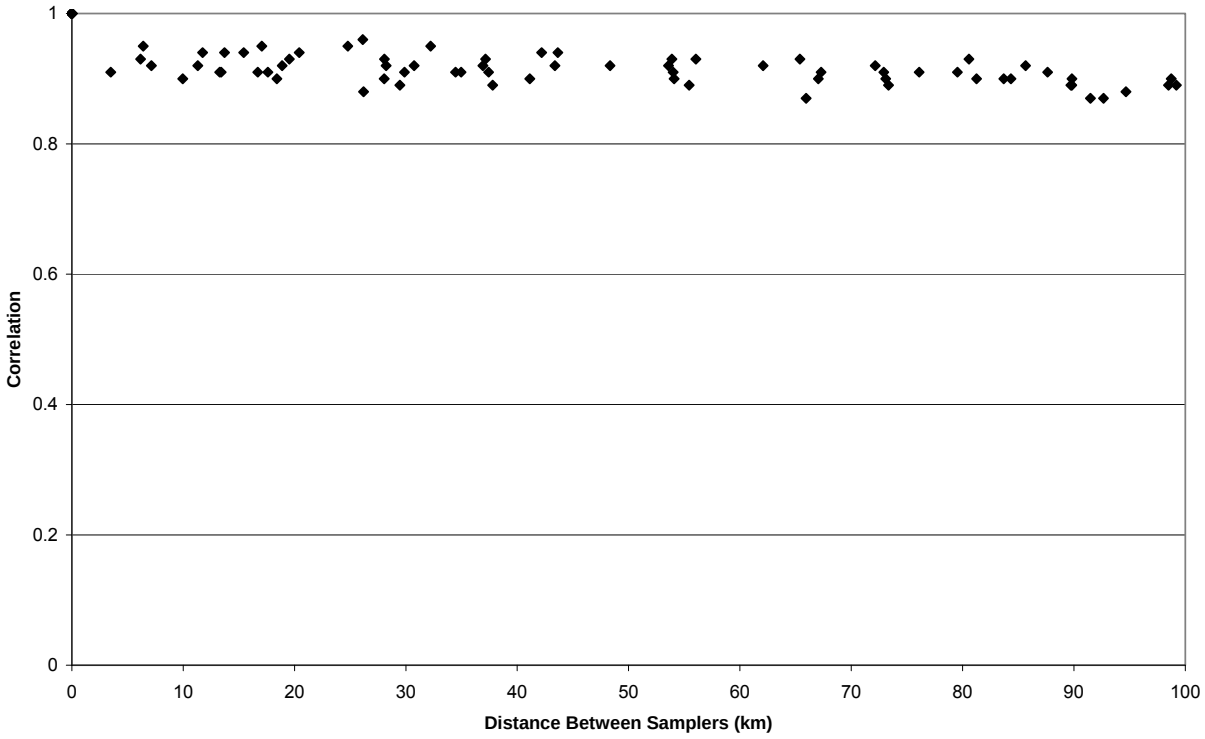


Figure A-54. $PM_{2.5}$ inter-sampler correlations as a function of distance between monitors for Detroit, MI.

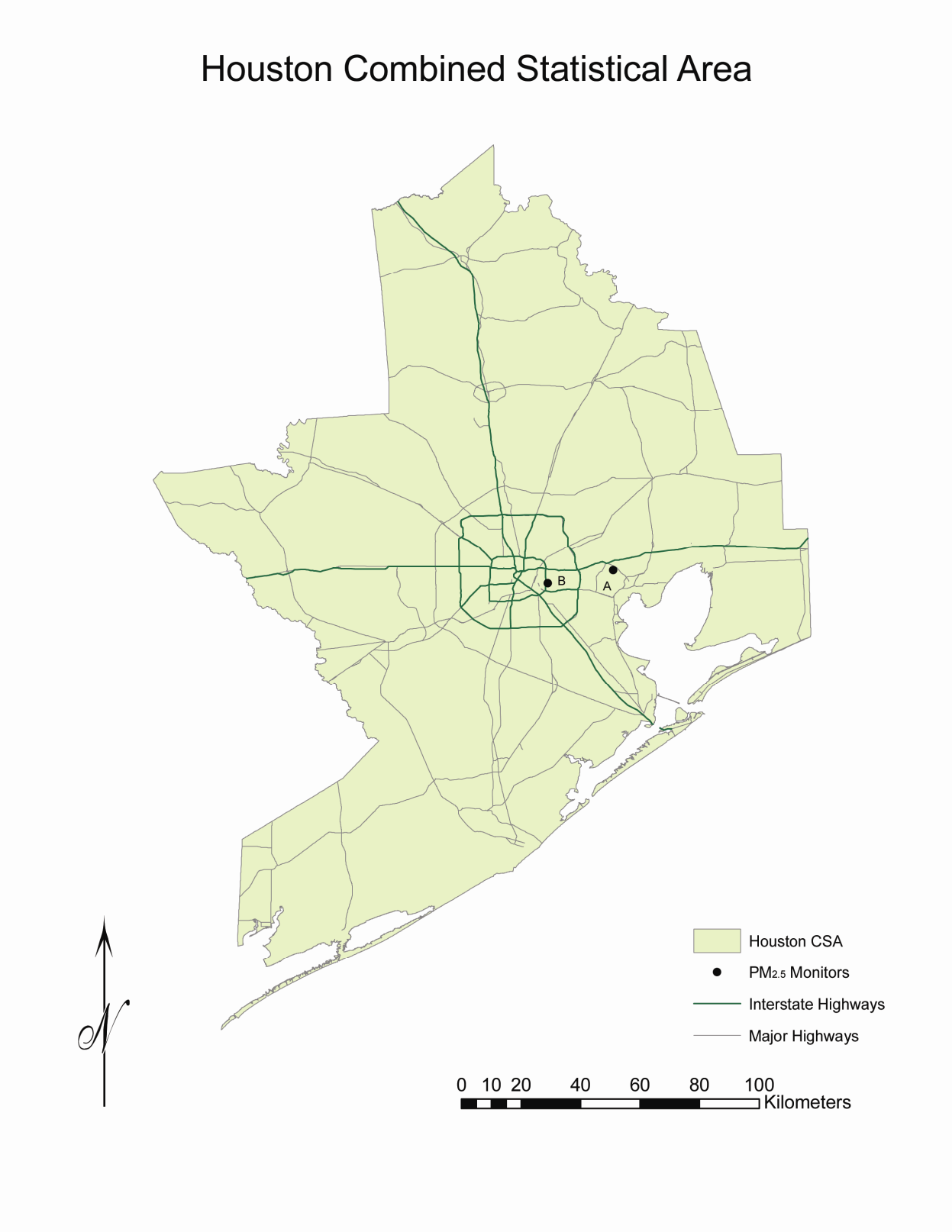


Figure A-55. $PM_{2.5}$ monitor distribution and major highways, Houston, TX.

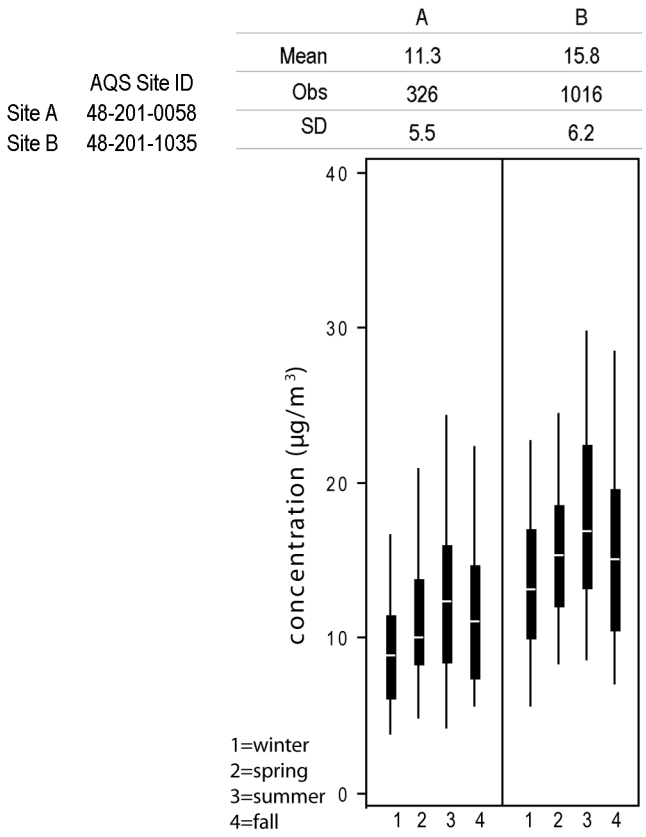


Figure A-56. Box plots illustrating the seasonal distribution of 24-h avg $PM_{2.5}$ concentrations for Houston, TX.

Table A-26. 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
LEGEND		
R		
(P90, COD)		
N		

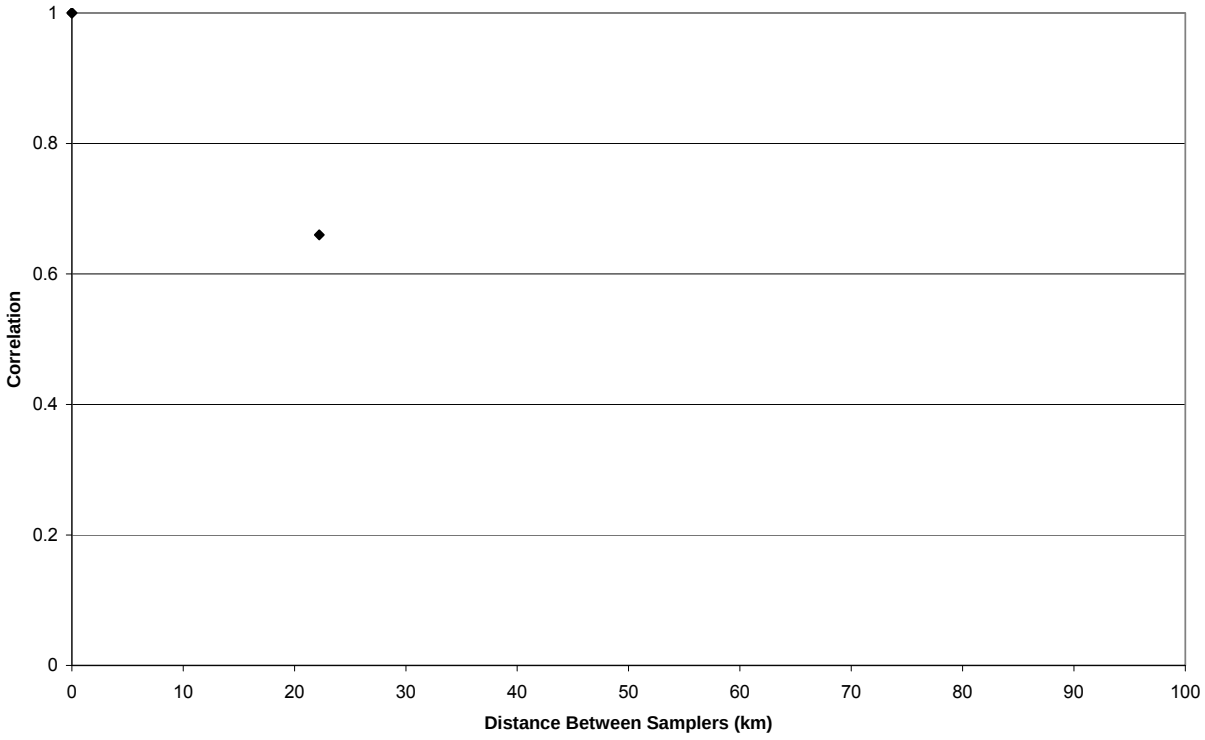


Figure A-57. $PM_{2.5}$ inter-sampler correlations as a function of distance between monitors for Houston, TX.

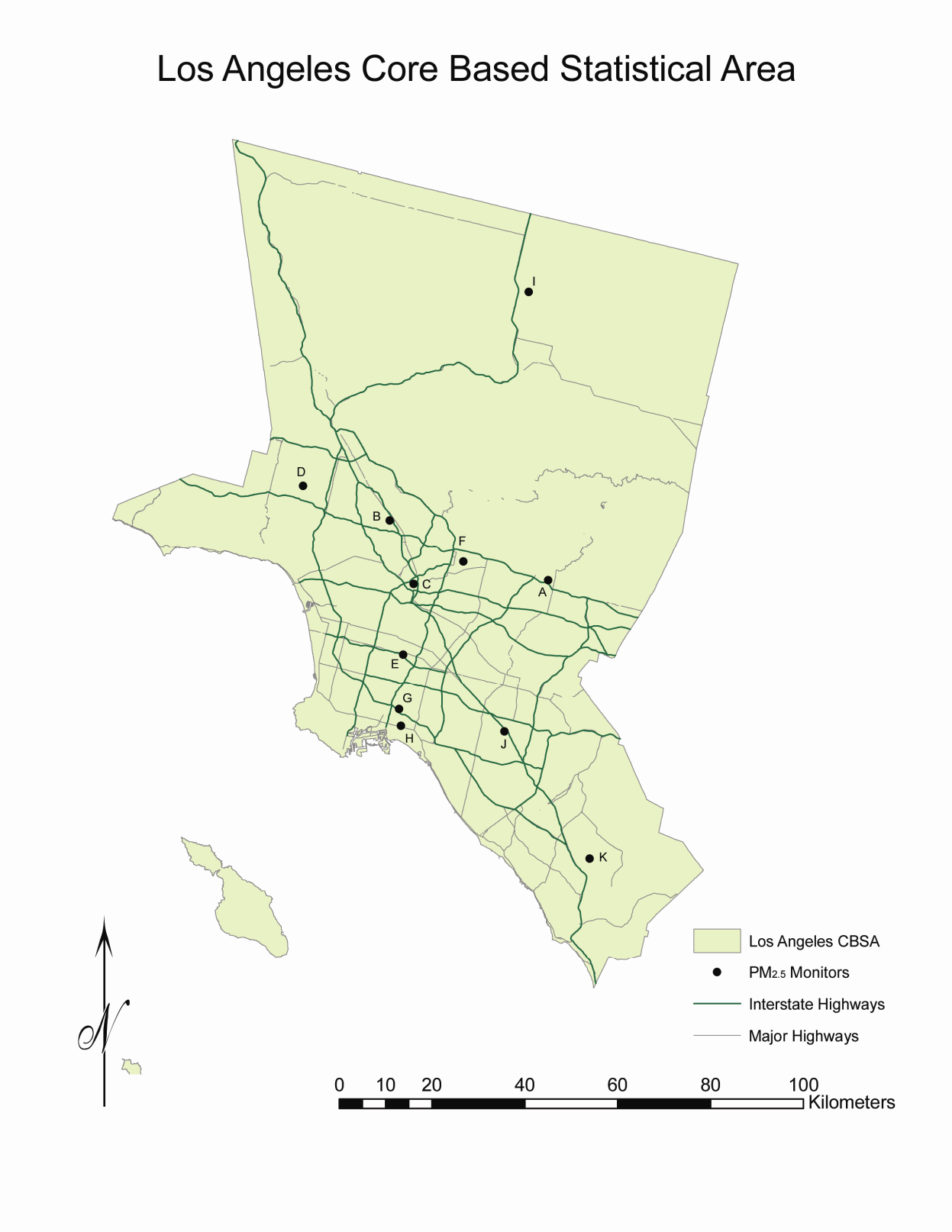


Figure A-58. $PM_{2.5}$ monitor distribution and major highways, Los Angeles, CA.

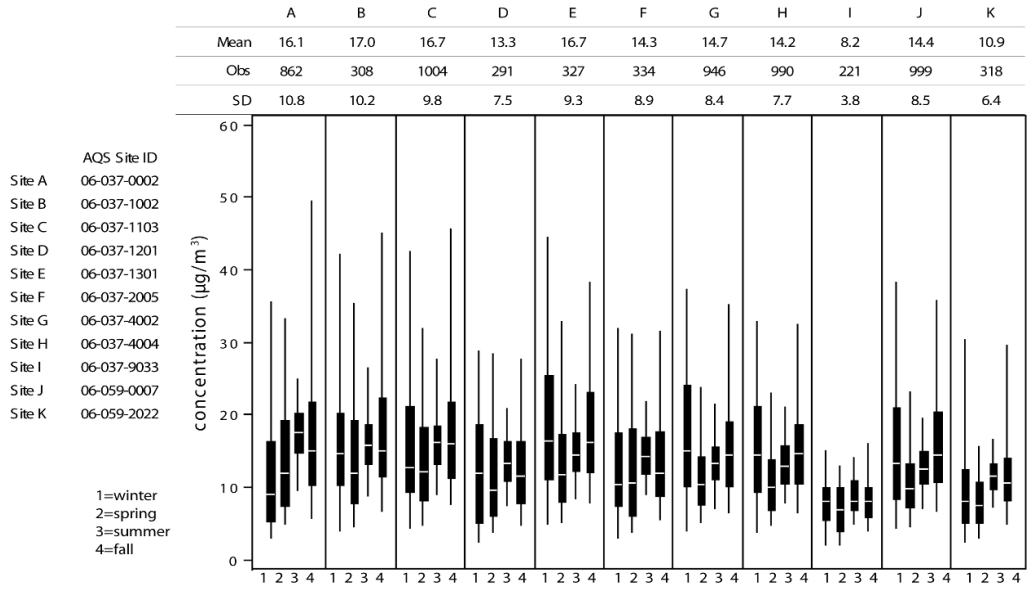


Figure A-59. Box plots illustrating the seasonal distribution of 24-h avg $PM_{2.5}$ concentrations for Los Angeles, CA.

Table A-27. Inter-sampler correlation statistics for each pair of $PM_{2.5}$ monitors reporting to AQS for Los Angeles, CA.

	A	B	C	D	E	F	G	H	I	J	K
A	1.00	0.86	0.87	0.81	0.80	0.88	0.68	0.64	0.30	0.70	0.82
	(0.0, 0.00)	(9.0, 0.18)	(7.7, 0.16)	(9.0, 0.19)	(9.7, 0.21)	(5.8, 0.14)	(11.5, 0.22)	(12.4, 0.23)	(18.0, 0.36)	(10.5, 0.21)	(11.4, 0.23)
	862	252	803	238	262	269	761	793	179	804	259
B		1.00	0.92	0.87	0.83	0.88	0.77	0.73	0.31	0.74	0.71
		(0.0, 0.00)	(5.5, 0.11)	(9.1, 0.19)	(9.0, 0.15)	(7.6, 0.15)	(9.8, 0.17)	(11.6, 0.18)	(24.1, 0.38)	(11.9, 0.19)	(15.0, 0.27)
		308	293	250	278	279	268	282	177	292	277
C			1.00	0.80	0.89	0.92	0.84	0.79	0.29	0.82	0.78
			(0.0, 0.00)	(9.6, 0.20)	(5.8, 0.11)	(6.4, 0.13)	(9.0, 0.15)	(10.0, 0.17)	(18.6, 0.38)	(9.4, 0.16)	(13.2, 0.25)
			1004	274	315	319	880	913	213	920	305
D				1.00	0.69	0.77	0.63	0.60	0.41	0.64	0.60
				(0.0, 0.00)	(10.9, 0.23)	(7.4, 0.18)	(11.3, 0.22)	(11.1, 0.22)	(14.8, 0.31)	(9.6, 0.21)	(11.6, 0.23)
				291	263	263	256	268	164	274	261
E					1.00	0.79	0.95	0.92	0.34	0.88	0.76
					(0.0, 0.00)	(9.1, 0.19)	(5.9, 0.11)	(7.6, 0.13)	(19.7, 0.39)	(8.2, 0.15)	(13.7, 0.27)
					327	301	289	301	192	307	291
F						1.00	0.70	0.70	0.33	0.69	0.72
						(0.0, 0.00)	(10.5, 0.18)	(9.2, 0.19)	(14.8, 0.34)	(9.8, 0.19)	(9.9, 0.21)
						334	290	302	184	311	293
G							1.00	0.96	0.23	0.92	0.78
							(0.0, 0.00)	(4.0, 0.09)	(17.0, 0.35)	(5.4, 0.12)	(11.0, 0.21)
							946	859	194	882	277
H								1.00	0.26	0.91	0.78
								(0.0, 0.00)	(15.3, 0.34)	(5.9, 0.12)	(9.5, 0.21)
								990	208	914	294
I									1.00	0.21	0.31
									(0.0, 0.00)	(18.3, 0.35)	(9.7, 0.28)
									221	205	180
J										1.00	0.84
										(0.0, 0.00)	(9.8, 0.19)
										999	298
K											1.00
											(0.0, 0.00)
											318

LEGEND
Pearson R
(P90, COD)
n

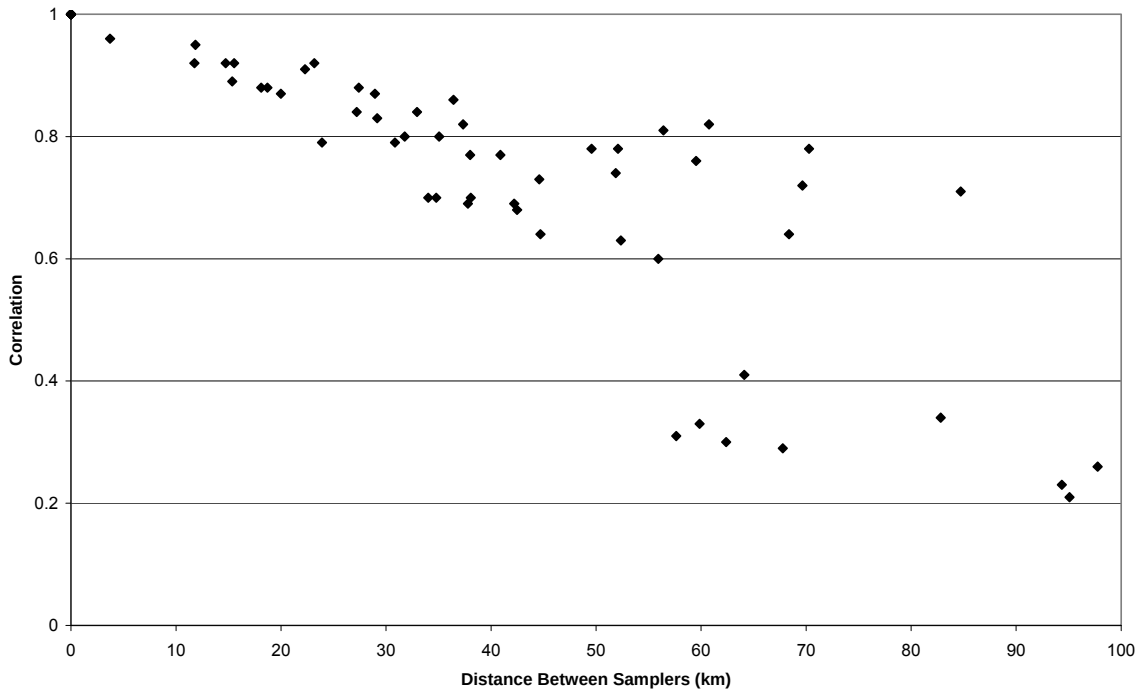


Figure A-60. $PM_{2.5}$ inter-sampler correlations as a function of distance between monitors for Los Angeles, CA.

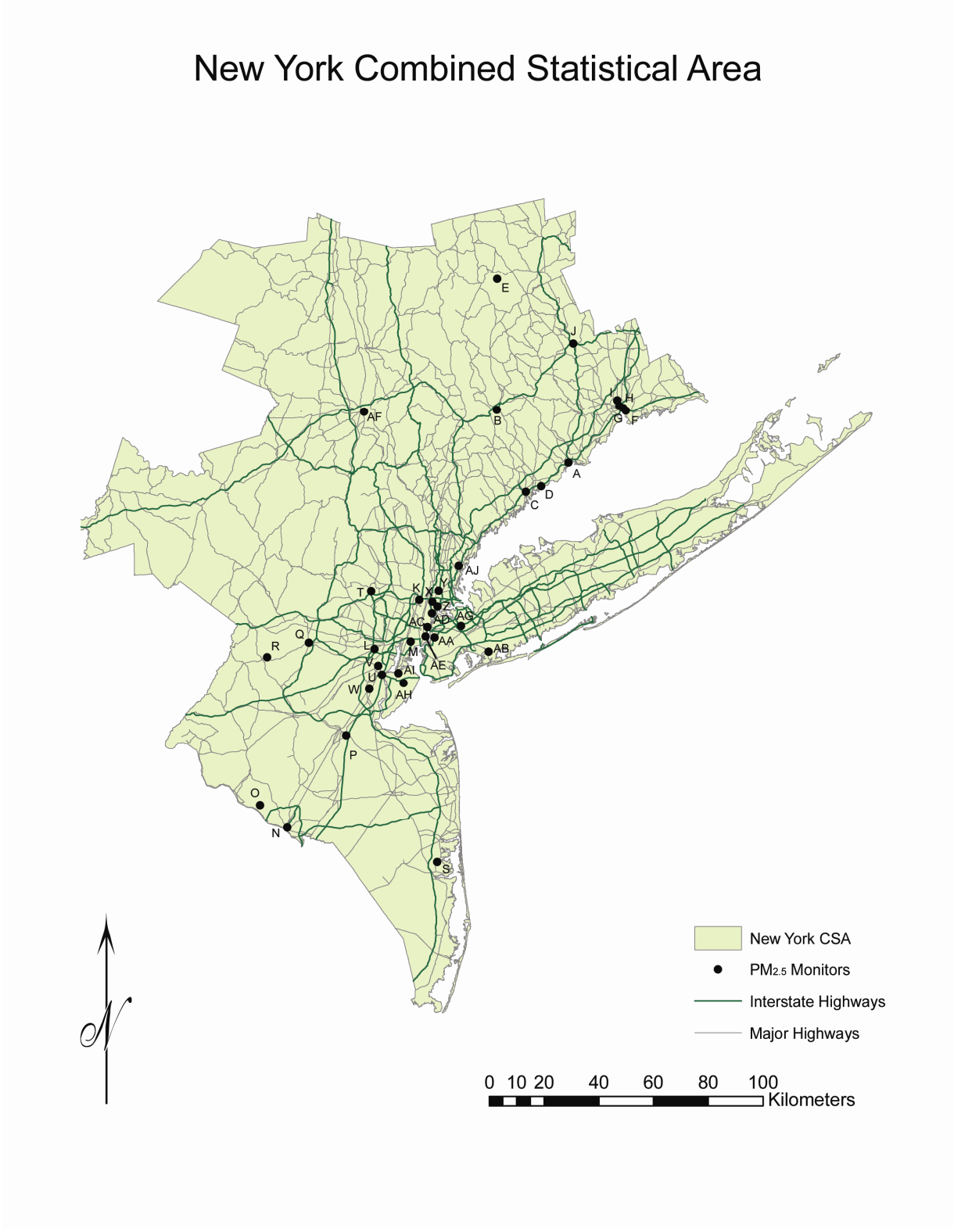
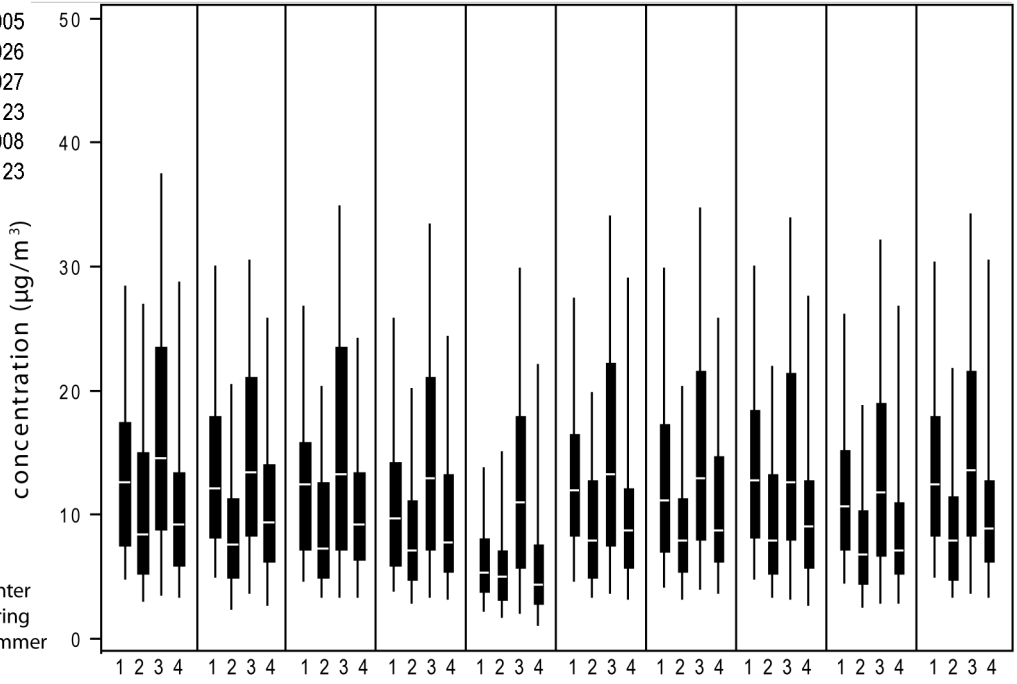


Figure A-61. $PM_{2.5}$ monitor distribution and major highways, New York City, NY.

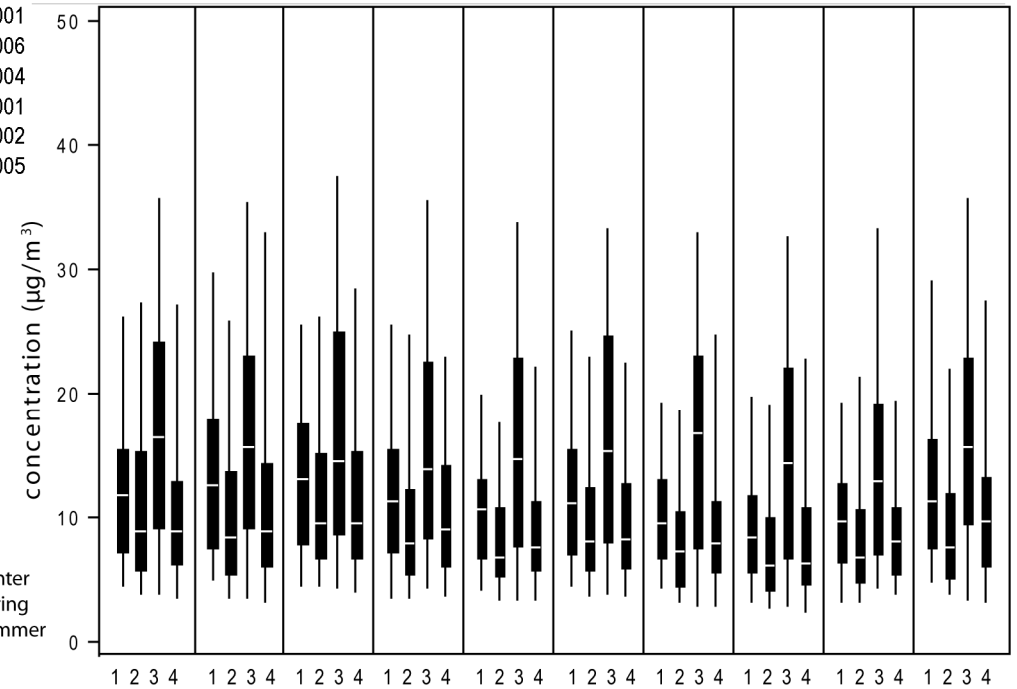
	AQS Site ID
Site A	09-001-0010
Site B	09-001-1123
Site C	09-001-3005
Site D	09-001-9003
Site E	09-005-0005
Site F	09-009-0026
Site G	09-009-0027
Site H	09-009-1123
Site I	09-009-2008
Site J	09-009-2123

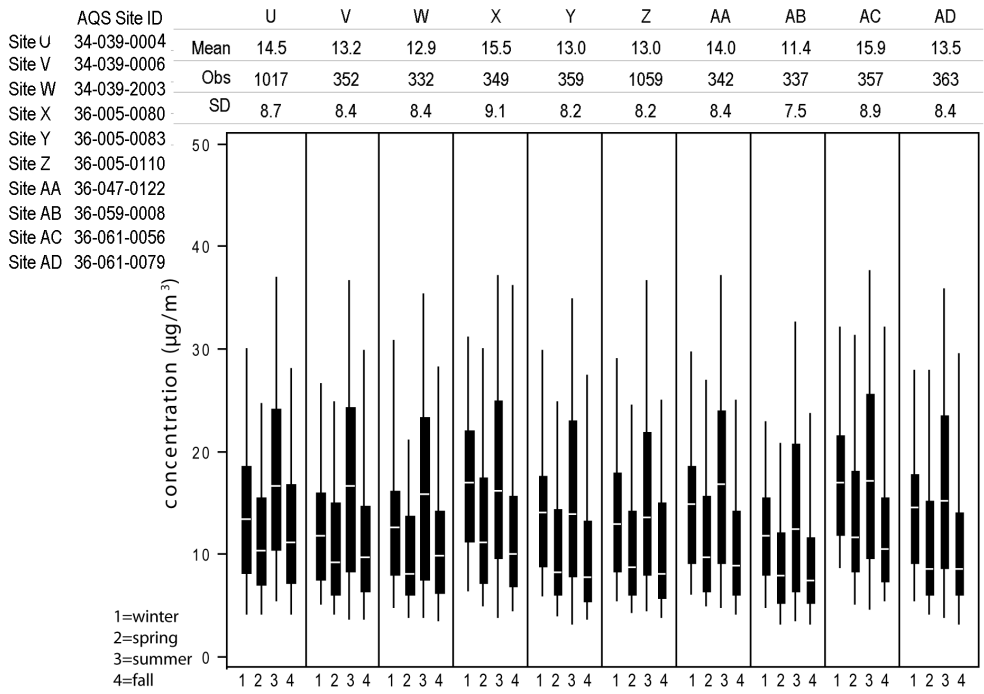
	A	B	C	D	E	F	G	H	I	J
Mean	13.2	12.5	12.3	11.2	8.0	12.1	12.4	12.8	11.1	12.6
Obs	349	339	332	565	341	341	992	338	344	352
SD	8.7	8.3	8.1	7.6	6.8	8.0	8.1	8.6	7.5	8.2



	AQS Site ID
Site K	34-003-0003
Site L	34-013-0015
Site M	34-017-1003
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

	K	L	M	N	O	P	Q	R	S	T
Mean	13.2	13.3	13.7	12.3	10.8	12.1	11.3	10.0	10.6	12.9
Obs	345	334	559	545	313	336	336	357	550	330
SD	8.9	8.8	8.5	7.7	7.0	7.7	7.7	7.4	6.8	8.7





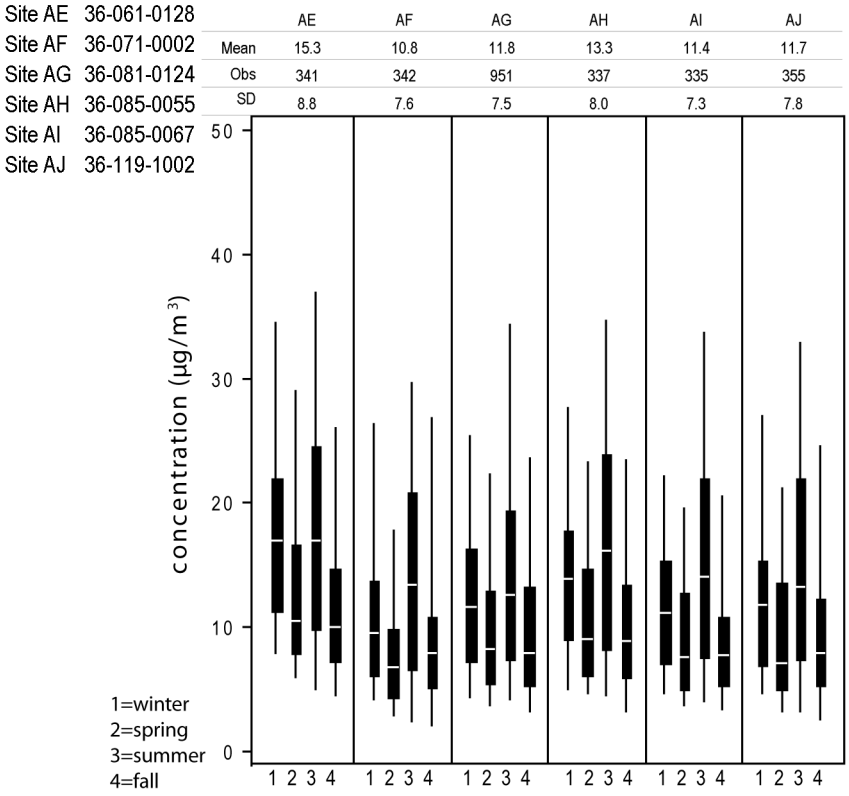


Figure A-62. Box plots illustrating the seasonal distribution of 24-h avg $PM_{2.5}$ concentrations for New York, NY.

Table A-28. Inter-sampler correlation statistics for each pair of $PM_{2.5}$ monitors reporting to AQS for New York, 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
											(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												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

LEGEND
R
(P90, COD)
N

	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
	(10.8, 0.23)	(5.9, 0.16)	(8.6, 0.20)	(6.5, 0.18)	(6.8, 0.18)	(9.0, 0.21)	(5.9, 0.16)	(6.8, 0.17)	(7.3, 0.18)	(7.9, 0.20)	(8.4, 0.22)	(7.0, 0.17)	(8.8, 0.23)	(5.8, 0.16)	(6.6, 0.17)	(6.8, 0.18)	(7.1, 0.18)	(5.5, 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, 0.20)	(6.1, 0.14)	(7.8, 0.18)	(6.4, 0.15)	(6.0, 0.15)	(7.8, 0.18)	(4.4, 0.11)	(5.4, 0.13)	(5.6, 0.15)	(6.1, 0.16)	(7.5, 0.20)	(5.3, 0.12)	(7.4, 0.20)	(6.7, 0.15)	(4.7, 0.11)	(5.7, 0.14)	(6.0, 0.15)	(3.5, 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, 0.19)	(7.3, 0.16)	(8.1, 0.20)	(7.1, 0.17)	(6.9, 0.17)	(9.7, 0.21)	(5.6, 0.14)	(6.2, 0.15)	(7.0, 0.17)	(5.9, 0.16)	(9.6, 0.23)	(6.6, 0.15)	(9.2, 0.23)	(5.4, 0.14)	(4.8, 0.12)	(6.5, 0.16)	(5.3, 0.14)	(3.7, 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, 0.32)	(11.3, 0.34)	(14.9, 0.40)	(11.7, 0.36)	(12.1, 0.36)	(15.2, 0.41)	(11.5, 0.34)	(13.1, 0.36)	(13.9, 0.38)	(10.1, 0.33)	(15.7, 0.43)	(13.1, 0.35)	(15.0, 0.42)	(7.6, 0.26)	(11.3, 0.32)	(12.5, 0.36)	(9.4, 0.31)	(9.8, 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, 0.19)	(6.7, 0.15)	(8.5, 0.19)	(6.8, 0.16)	(6.6, 0.16)	(8.2, 0.19)	(5.0, 0.12)	(6.4, 0.14)	(6.7, 0.15)	(5.6, 0.16)	(8.4, 0.20)	(6.3, 0.14)	(8.0, 0.21)	(6.4, 0.16)	(4.6, 0.12)	(5.7, 0.15)	(5.3, 0.15)	(4.1, 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, 0.21)	(6.3, 0.15)	(7.8, 0.18)	(7.0, 0.16)	(6.3, 0.15)	(8.3, 0.17)	(5.4, 0.13)	(5.7, 0.14)	(7.1, 0.15)	(7.5, 0.17)	(8.1, 0.19)	(6.4, 0.14)	(8.2, 0.20)	(6.7, 0.17)	(5.2, 0.13)	(6.1, 0.15)	(6.1, 0.16)	(5.0, 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, 0.22)	(6.6, 0.15)	(8.4, 0.18)	(7.1, 0.16)	(6.9, 0.16)	(7.5, 0.17)	(5.2, 0.13)	(5.6, 0.14)	(6.4, 0.15)	(7.3, 0.19)	(7.9, 0.19)	(5.7, 0.13)	(7.3, 0.20)	(6.9, 0.17)	(5.6, 0.14)	(6.8, 0.16)	(6.4, 0.17)	(5.0, 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, 0.20)	(7.1, 0.17)	(8.7, 0.21)	(7.4, 0.17)	(6.9, 0.17)	(9.4, 0.22)	(5.7, 0.15)	(6.5, 0.16)	(7.2, 0.18)	(6.2, 0.17)	(9.6, 0.24)	(6.3, 0.16)	(9.2, 0.24)	(5.5, 0.13)	(5.1, 0.14)	(6.7, 0.18)	(5.8, 0.16)	(4.1, 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, 0.22)	(6.6, 0.16)	(9.0, 0.19)	(6.7, 0.16)	(6.8, 0.17)	(8.8, 0.19)	(6.1, 0.14)	(7.1, 0.16)	(7.3, 0.17)	(8.2, 0.19)	(9.0, 0.21)	(6.9, 0.15)	(8.9, 0.22)	(6.4, 0.16)	(6.4, 0.15)	(7.5, 0.16)	(7.7, 0.18)	(5.6, 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, 0.21)	(3.9, 0.11)	(5.7, 0.14)	(3.4, 0.10)	(4.3, 0.12)	(6.0, 0.15)	(3.8, 0.12)	(4.2, 0.12)	(4.3, 0.12)	(8.5, 0.19)	(6.2, 0.17)	(3.8, 0.11)	(6.2, 0.18)	(7.4, 0.17)	(5.9, 0.13)	(5.0, 0.13)	(6.5, 0.15)	(4.8, 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, 0.20)	(3.9, 0.11)	(4.5, 0.12)	(2.9, 0.08)	(4.0, 0.10)	(6.3, 0.15)	(4.5, 0.12)	(4.2, 0.11)	(4.1, 0.11)	(8.1, 0.18)	(6.3, 0.17)	(4.2, 0.10)	(5.9, 0.17)	(6.8, 0.17)	(5.4, 0.12)	(4.0, 0.11)	(6.4, 0.14)	(5.4, 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, 0.22)	(5.4, 0.13)	(3.8, 0.09)	(3.5, 0.09)	(4.7, 0.11)	(4.9, 0.11)	(4.7, 0.12)	(3.5, 0.10)	(3.4, 0.09)	(8.3, 0.20)	(4.5, 0.12)	(3.5, 0.10)	(4.5, 0.13)	(8.5, 0.21)	(5.0, 0.14)	(4.3, 0.10)	(6.8, 0.16)	(5.7, 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, 0.17)	(6.5, 0.16)	(6.5, 0.15)	(5.7, 0.13)	(4.5, 0.13)	(8.2, 0.18)	(5.9, 0.14)	(5.4, 0.15)	(5.3, 0.14)	(5.6, 0.17)	(8.1, 0.18)	(5.3, 0.14)	(7.7, 0.18)	(7.5, 0.20)	(4.9, 0.14)	(4.6, 0.13)	(5.3, 0.14)	(5.4, 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, 0.16)	(7.2, 0.18)	(9.9, 0.22)	(7.3, 0.18)	(6.4, 0.16)	(11.1, 0.24)	(6.7, 0.18)	(8.6, 0.19)	(8.2, 0.20)	(5.2, 0.15)	(10.3, 0.25)	(8.4, 0.18)	(10.6, 0.25)	(7.0, 0.18)	(6.1, 0.16)	(6.1, 0.18)	(4.7, 0.14)	(5.4, 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, 0.15)	(6.2, 0.14)	(7.4, 0.17)	(5.0, 0.12)	(4.0, 0.11)	(8.9, 0.19)	(5.9, 0.14)	(6.8, 0.15)	(6.4, 0.14)	(5.3, 0.14)	(8.9, 0.21)	(6.4, 0.14)	(8.3, 0.20)	(6.7, 0.16)	(5.5, 0.13)	(4.7, 0.12)	(3.5, 0.10)	(5.0, 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, 0.19)	(5.0, 0.14)	(8.2, 0.20)	(6.2, 0.14)	(5.4, 0.15)	(9.7, 0.22)	(6.3, 0.16)	(7.3, 0.17)	(7.3, 0.18)	(6.9, 0.19)	(9.9, 0.24)	(6.9, 0.16)	(9.5, 0.24)	(4.8, 0.14)	(6.0, 0.15)	(6.3, 0.17)	(5.3, 0.14)	(5.5, 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, 0.20)	(7.6, 0.21)	(10.9, 0.26)	(8.2, 0.21)	(7.0, 0.20)	(11.6, 0.28)	(8.6, 0.21)	(10.0, 0.23)	(9.1, 0.24)	(7.0, 0.21)	(11.2, 0.30)	(9.5, 0.22)	(11.0, 0.30)	(6.2, 0.17)	(7.6, 0.20)	(7.8, 0.22)	(5.7, 0.17)	(6.3, 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, 0.00)	(10.4, 0.22)	(10.5, 0.24)	(10.5, 0.21)	(9.2, 0.19)	(12.5, 0.25)	(8.6, 0.19)	(9.3, 0.21)	(9.4, 0.20)	(5.0, 0.16)	(11.6, 0.26)	(9.9, 0.20)	(11.5, 0.25)	(10.3, 0.22)	(7.2, 0.17)	(8.5, 0.18)	(5.5, 0.14)	(8.1, 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, 0.00)	(6.0, 0.15)	(4.5, 0.12)	(4.8, 0.13)	(6.7, 0.16)	(5.2, 0.14)	(4.9, 0.13)	(5.6, 0.14)	(8.4, 0.20)	(6.9, 0.18)	(4.9, 0.12)	(7.2, 0.20)	(6.3, 0.17)	(6.2, 0.14)	(5.8, 0.14)	(7.3, 0.17)	(5.9, 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, 0.00)	(3.9, 0.10)	(5.0, 0.12)	(5.4, 0.12)	(6.9, 0.15)	(5.2, 0.13)	(4.9, 0.12)	(9.9, 0.22)	(5.0, 0.12)	(5.2, 0.12)	(5.9, 0.14)	(9.6, 0.23)	(6.9, 0.17)	(5.8, 0.12)	(8.4, 0.18)	(7.5, 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, 0.00)	(2.8, 0.09)	(6.1, 0.14)	(5.0, 0.13)	(4.4, 0.12)	(4.2, 0.12)	(8.2, 0.19)	(6.6, 0.16)	(4.3, 0.11)	(6.1, 0.16)	(7.0, 0.18)	(5.8, 0.15)	(4.0, 0.10)	(6.4, 0.15)	(5.3, 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
					(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

LEGEND
R
(P90, COD)
N

	S	T	U	V	W	X	Y	Z	AA	AB	AC	AD	AE	AF	AG	AH	AI	AJ
													(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

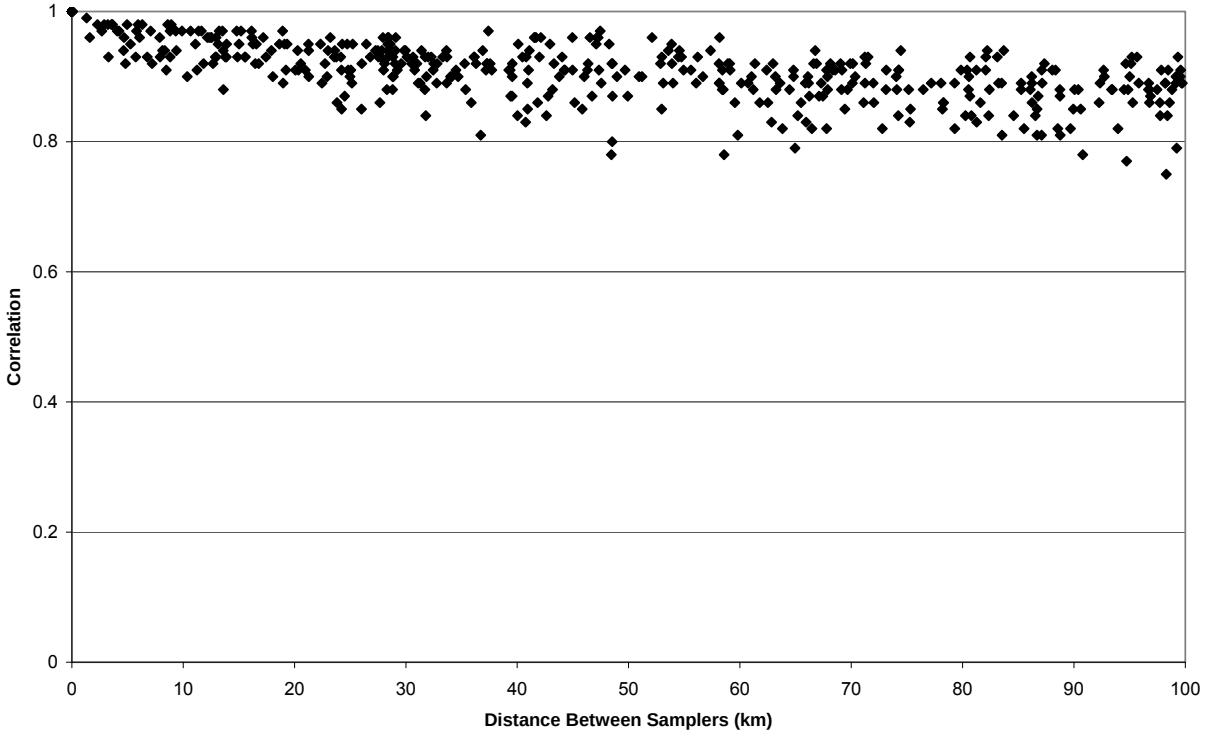


Figure A-63 $PM_{2.5}$ inter-sampler correlations as a function of distance between monitors for New York, NY.

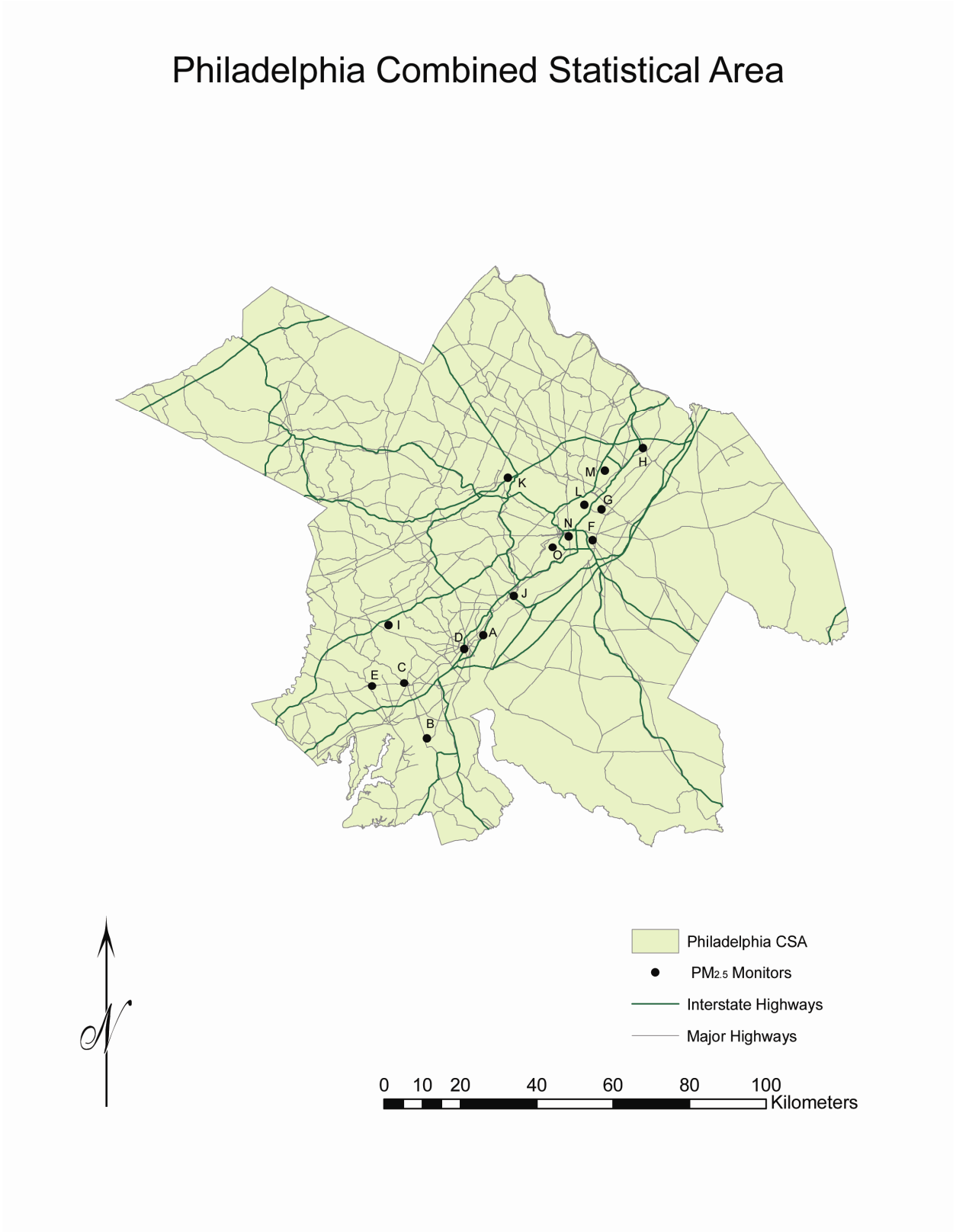


Figure A-64. $PM_{2.5}$ monitor distribution and major highways, Philadelphia, PA.

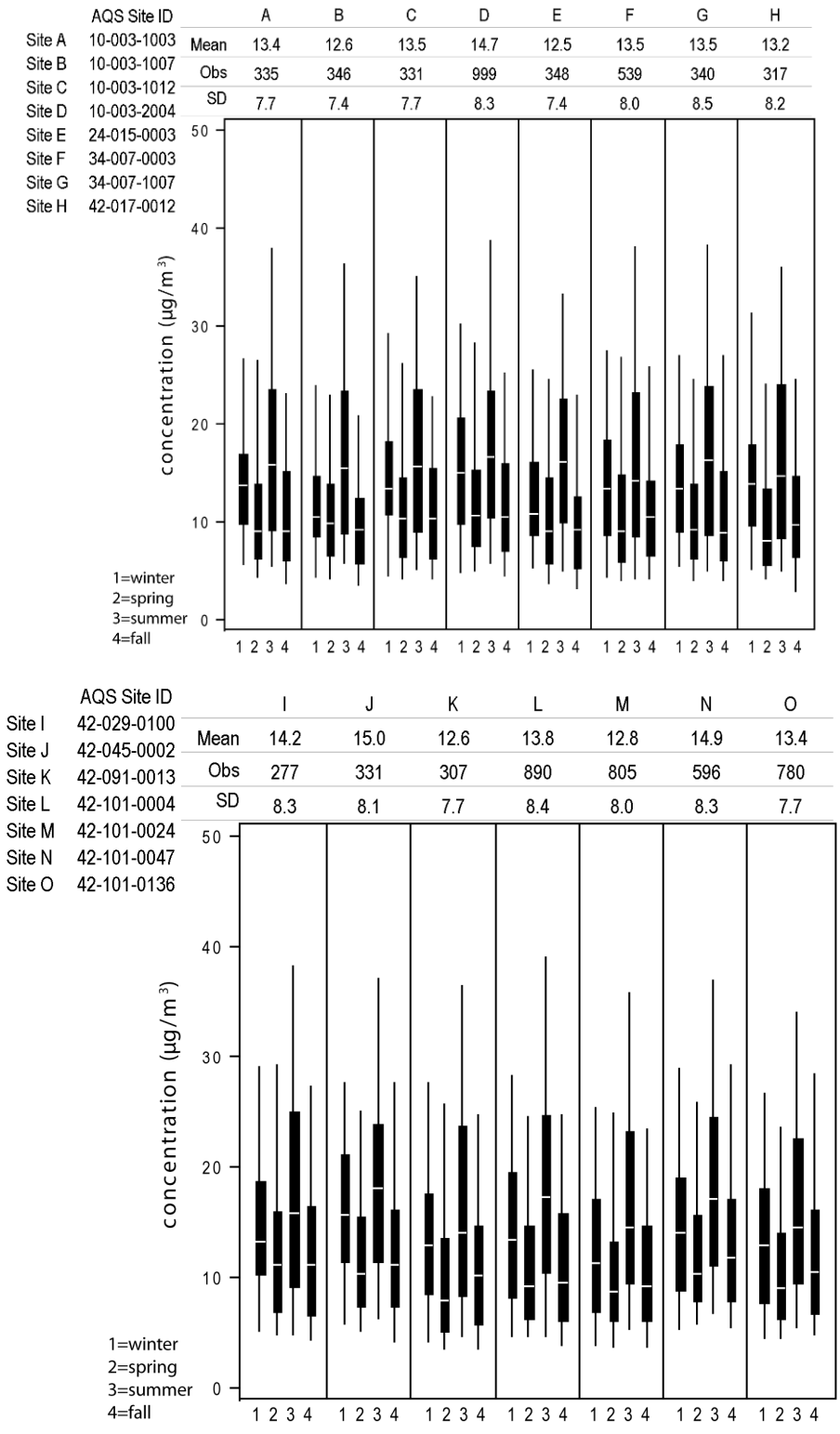


Figure A-65. Box plots illustrating the seasonal distribution of 24-h avg $PM_{2.5}$ concentrations for Philadelphia, PA.

Table A-29. 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)
			LEGEND						277	248	228	235	215	196	195
			R												
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

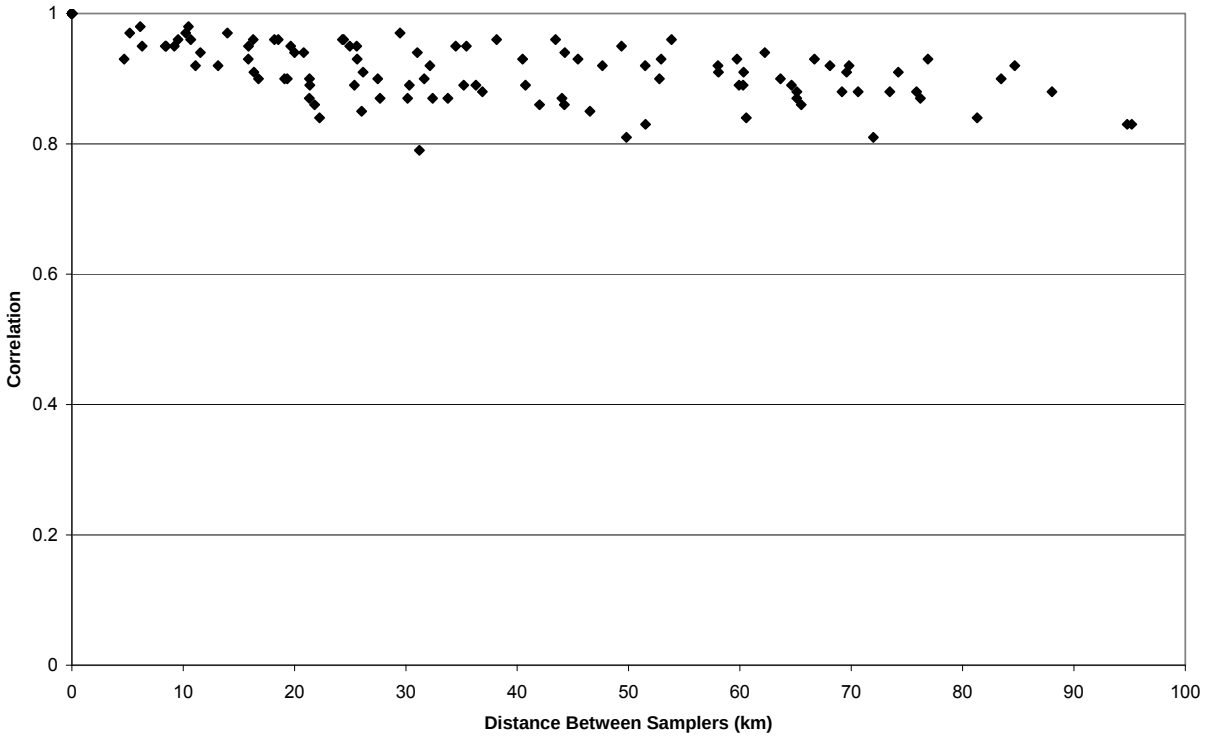


Figure A-66. $PM_{2.5}$ inter-sampler correlations as a function of distance between monitors for Philadelphia, PA.

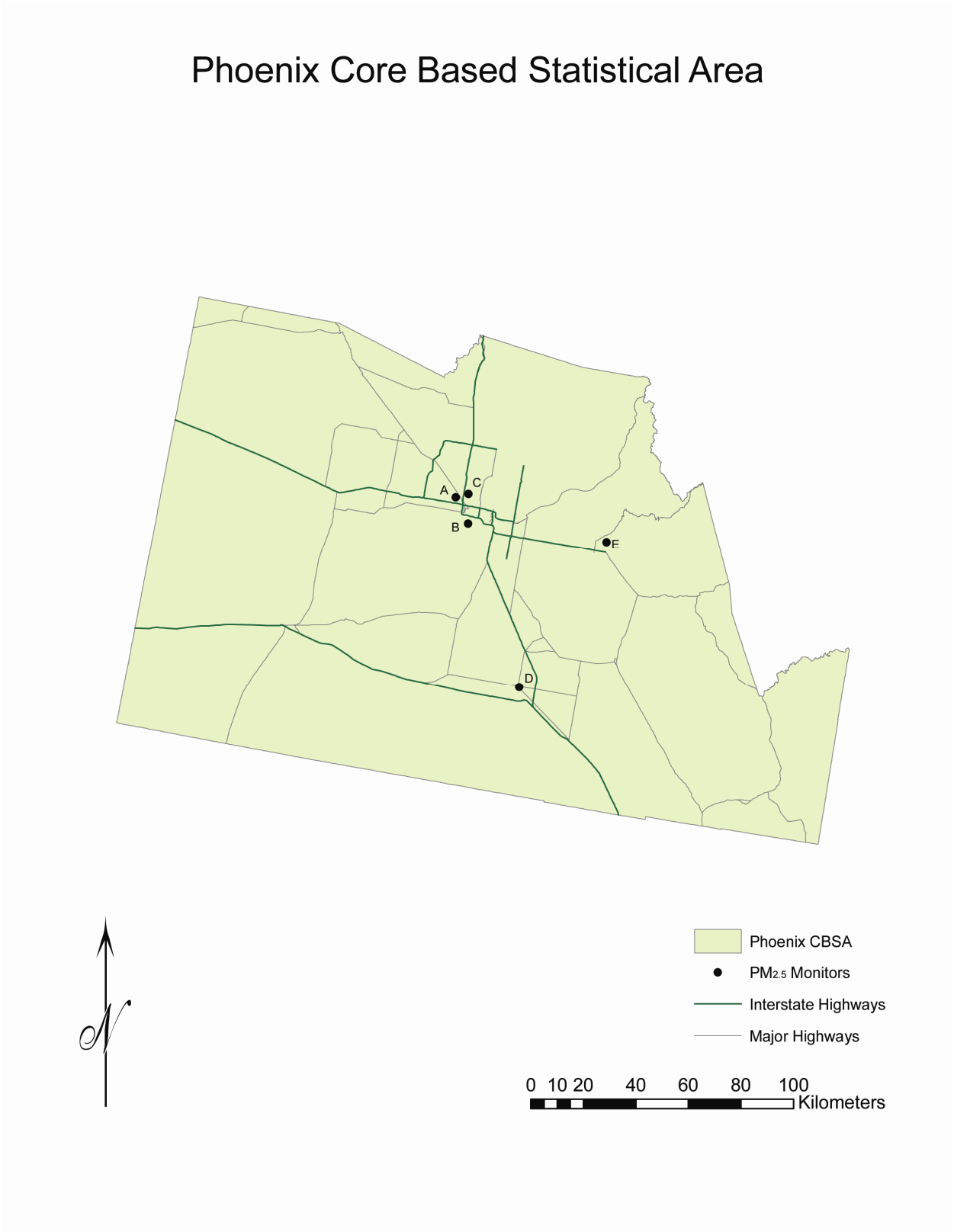


Figure A-67. $PM_{2.5}$ monitor distribution and major highways, Phoenix, AZ.

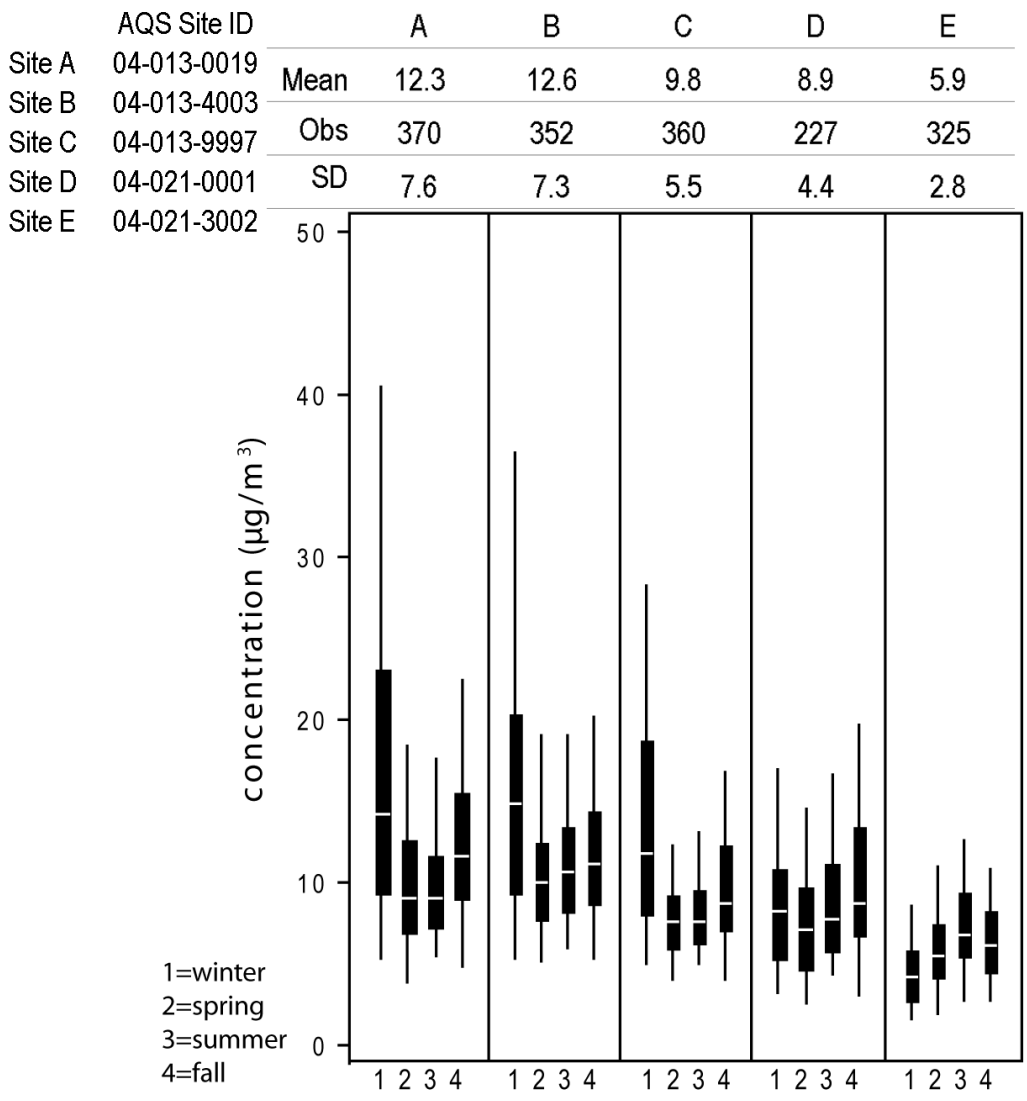


Figure A-68. Box plots illustrating the seasonal distribution of 24-h avg $PM_{2.5}$ concentrations for Phoenix, AZ.

Table A-30. 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)
			360	216	315
D	**LEGEND**			1.00	0.51
	R			(0.0, 0.00)	(7.8, 0.27)
	(P90, COD)			227	200
E	**N**				1.00
					(0.0, 0.00)
					325

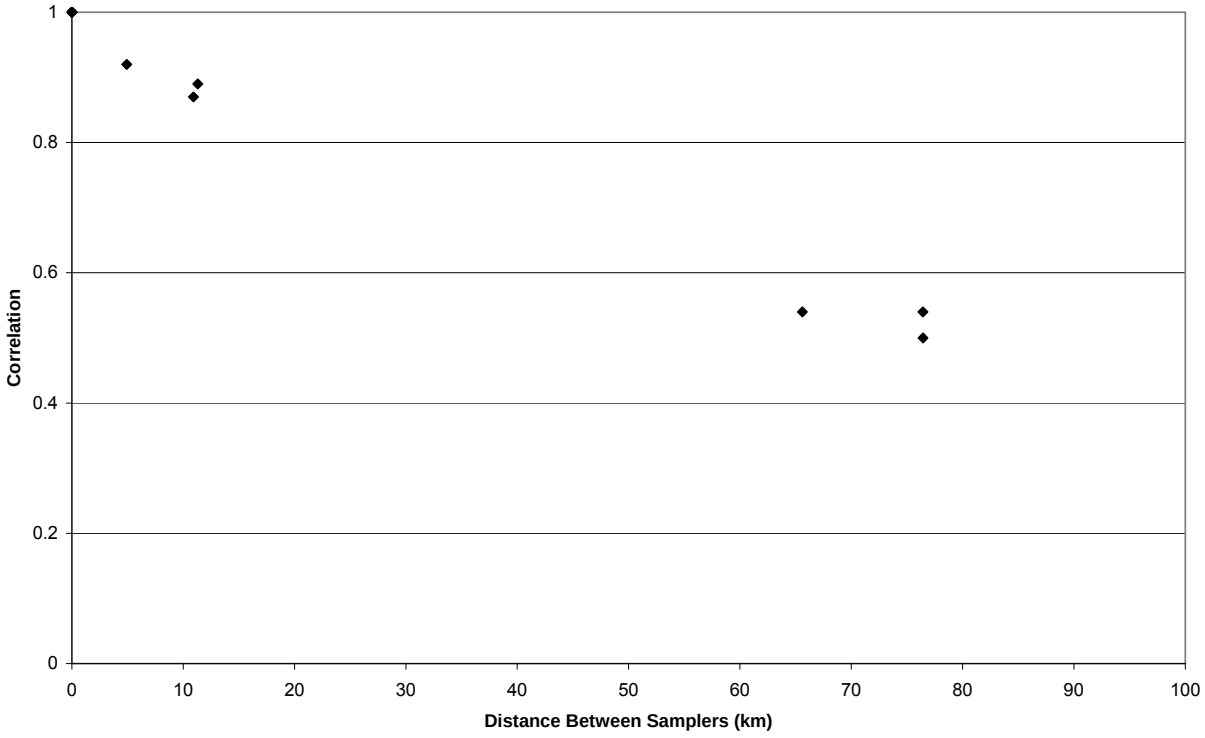


Figure A-69. $PM_{2.5}$ inter-sampler correlations as a function of distance between monitors for Phoenix, AZ.

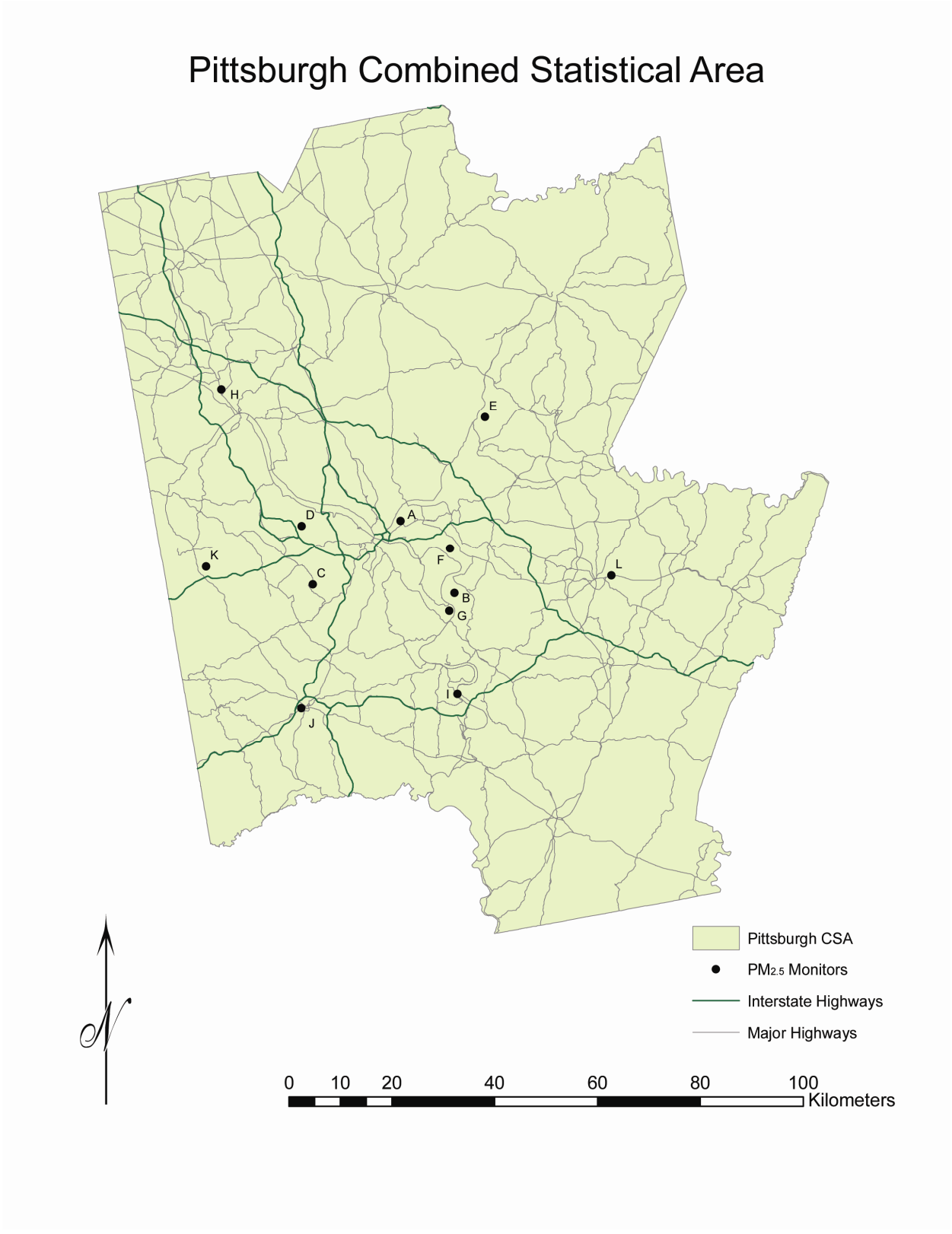


Figure A-70. $PM_{2.5}$ monitor distribution and major highways, Pittsburgh, PA.

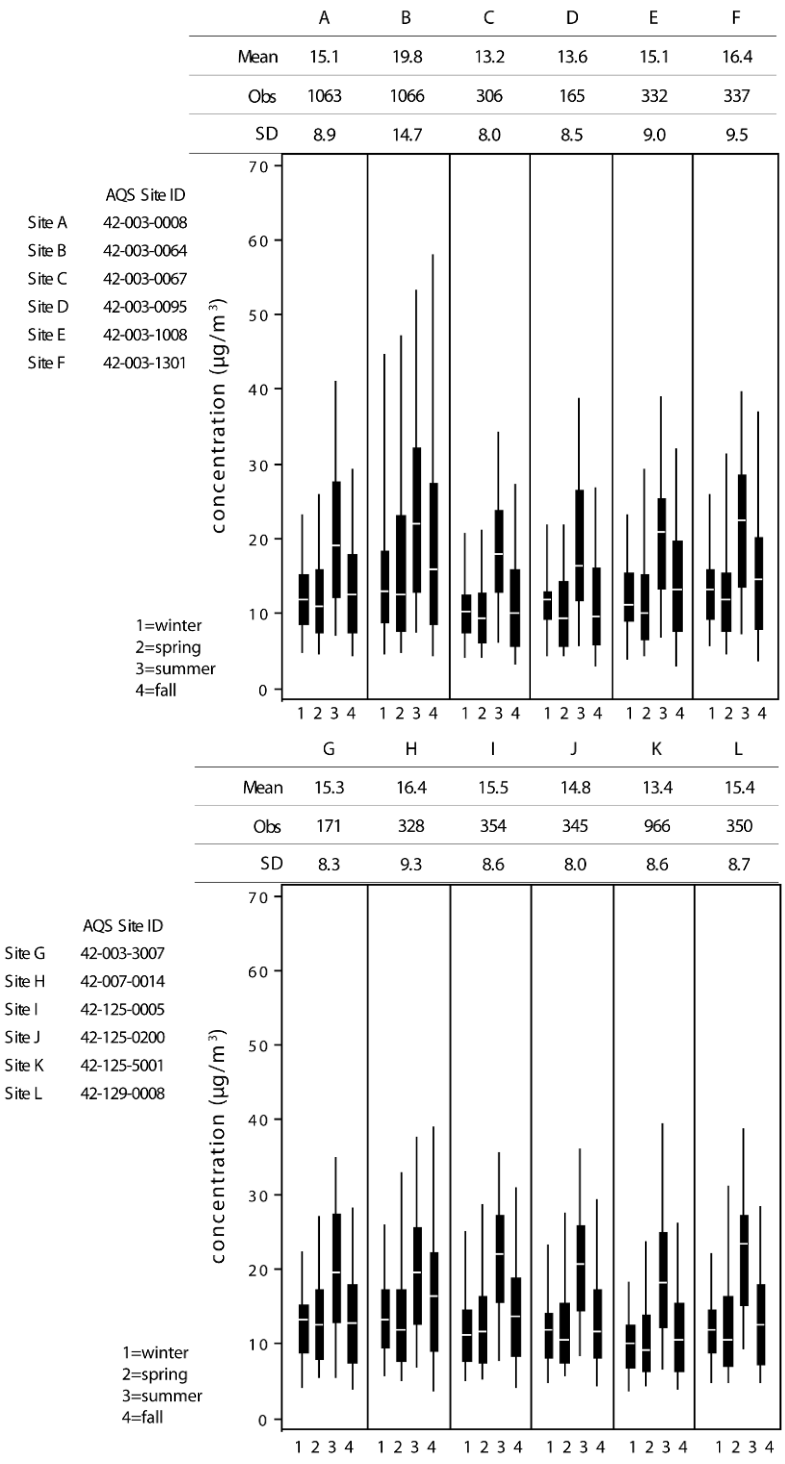


Figure A-71. Box plots illustrating the seasonal distribution of 24-h avg $PM_{2.5}$ concentrations for Pitsburgh, PA.

Table A-31. Inter-sampler correlation statistics for each pair of $PM_{2.5}$ monitors reporting to AQS for Pittsburgh, PA.

	A	B	C	D	E	F	G	H	I	J	K	L
A	1.00	0.79	0.95	0.92	0.93	0.95	0.95	0.85	0.90	0.93	0.91	0.88
	(0.0, 0.00)	(15.9, 0.19)	(5.6, 0.13)	(4.7, 0.11)	(4.7, 0.11)	(4.9, 0.10)	(3.8, 0.10)	(6.4, 0.13)	(6.4, 0.13)	(5.0, 0.12)	(6.0, 0.13)	(5.6, 0.12)
	1063	1035	298	164	323	329	170	319	344	337	934	340
B		1.00	0.71	0.65	0.80	0.85	0.76	0.69	0.71	0.68	0.68	0.67
		(0.0, 0.00)	(16.9, 0.24)	(17.4, 0.25)	(14.4, 0.19)	(12.5, 0.14)	(15.7, 0.20)	(17.0, 0.19)	(15.7, 0.21)	(17.8, 0.23)	(19.3, 0.25)	(15.9, 0.21)
		1066	303	165	329	335	171	324	350	341	938	346
C			1.00	0.93	0.90	0.91	0.94	0.80	0.93	0.96	0.95	0.91
			(0.0, 0.00)	(2.8, 0.09)	(6.6, 0.16)	(8.7, 0.17)	(6.0, 0.14)	(9.4, 0.19)	(6.7, 0.15)	(4.6, 0.12)	(4.5, 0.10)	(6.5, 0.15)
			306	144	282	282	148	268	290	286	270	286
D				1.00	0.84	0.87	0.91	0.79	0.89	0.91	0.97	0.85
				(0.0, 0.00)	(6.4, 0.15)	(8.5, 0.16)	(5.8, 0.13)	(9.2, 0.17)	(5.9, 0.13)	(4.6, 0.11)	(3.1, 0.08)	(6.5, 0.15)
				165	153	161	158	156	158	155	146	157
E					1.00	0.90	0.90	0.84	0.85	0.86	0.88	0.83
					(0.0, 0.00)	(6.4, 0.13)	(6.5, 0.13)	(6.8, 0.14)	(8.3, 0.16)	(7.7, 0.16)	(7.6, 0.15)	(7.3, 0.15)
					332	313	157	295	320	315	290	318
F						1.00	0.91	0.82	0.88	0.88	0.89	0.86
						(0.0, 0.00)	(6.7, 0.13)	(7.4, 0.14)	(7.1, 0.15)	(7.9, 0.15)	(8.8, 0.17)	(7.0, 0.14)
						337	167	302	327	319	296	322
G							1.00	0.78	0.94	0.93	0.90	0.91
							(0.0, 0.00)	(7.3, 0.16)	(4.0, 0.10)	(5.0, 0.11)	(6.6, 0.15)	(5.0, 0.13)
							171	159	163	159	149	161
H								1.00	0.80	0.78	0.82	0.70
								(0.0, 0.00)	(8.4, 0.15)	(8.2, 0.17)	(9.0, 0.18)	(9.2, 0.18)
								328	317	309	288	314
I									1.00	0.93	0.89	0.88
									(0.0, 0.00)	(5.0, 0.11)	(7.2, 0.16)	(6.0, 0.13)
									354	334	310	339
J										1.00	0.93	0.88
										(0.0, 0.00)	(5.5, 0.12)	(5.9, 0.13)
										345	302	331
K											1.00	0.86
											(0.0, 0.00)	(6.9, 0.15)
											966	306
L												1.00
												(0.0, 0.00)
												350

LEGEND
Pearson R
(P90, COD)
n

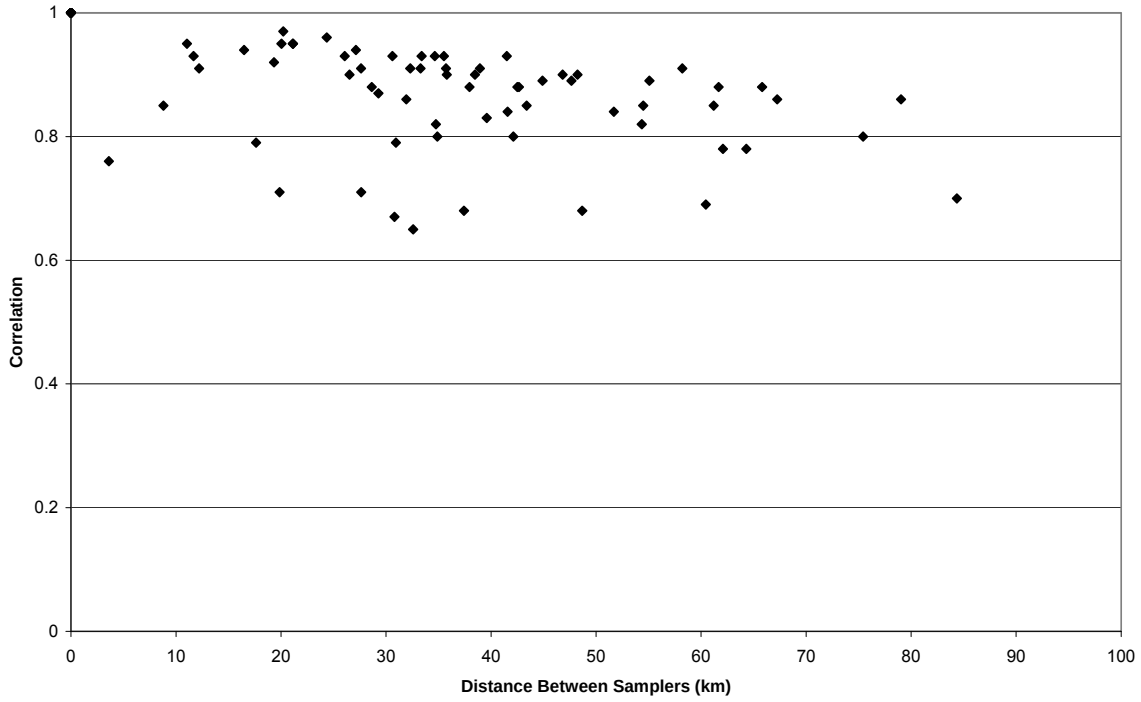


Figure A-72. $PM_{2.5}$ inter-sampler correlations as a function of distance between monitors for Pittsburgh, PA.

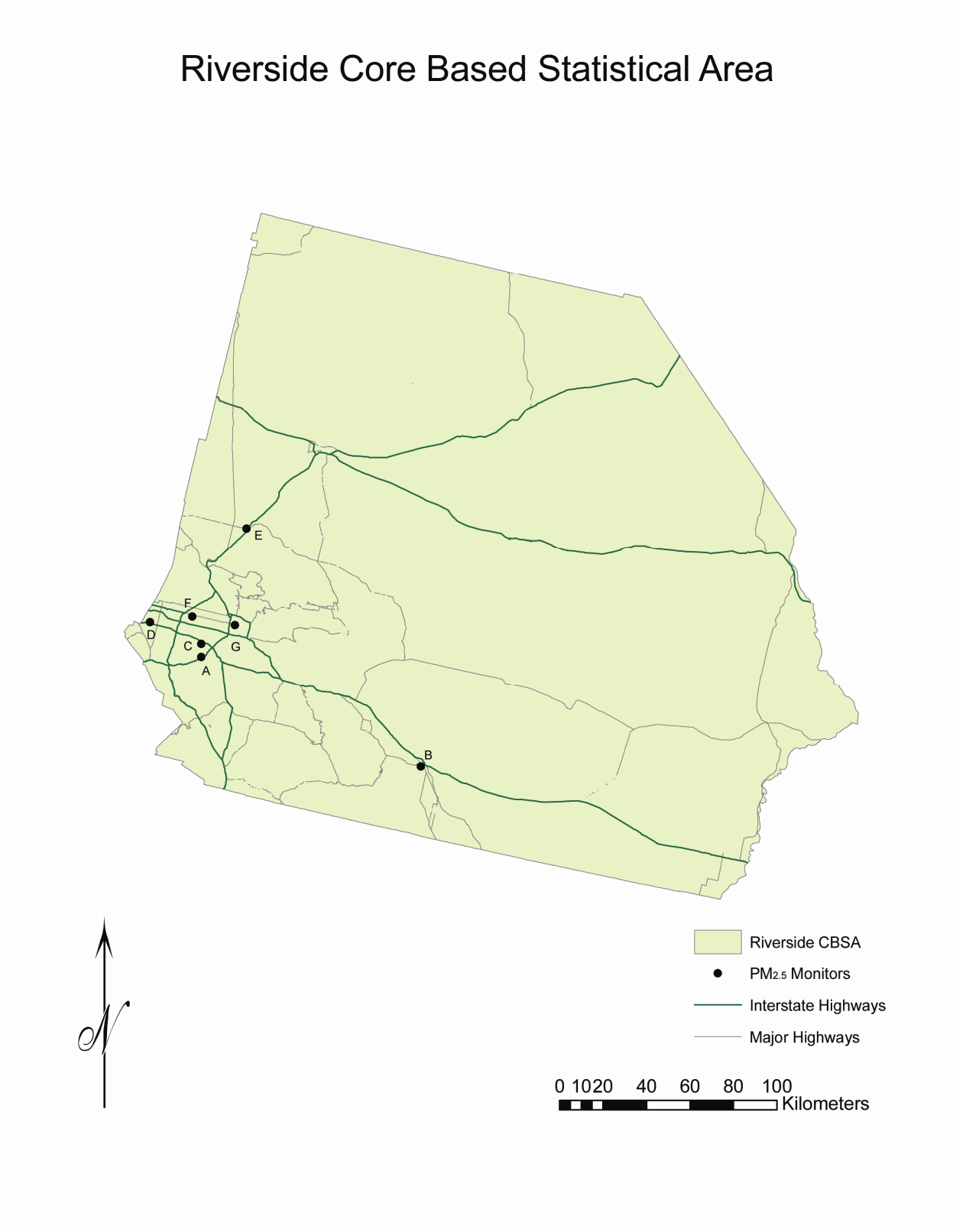


Figure A-73. $PM_{2.5}$ monitor distribution and major highways, Riverside, CA.

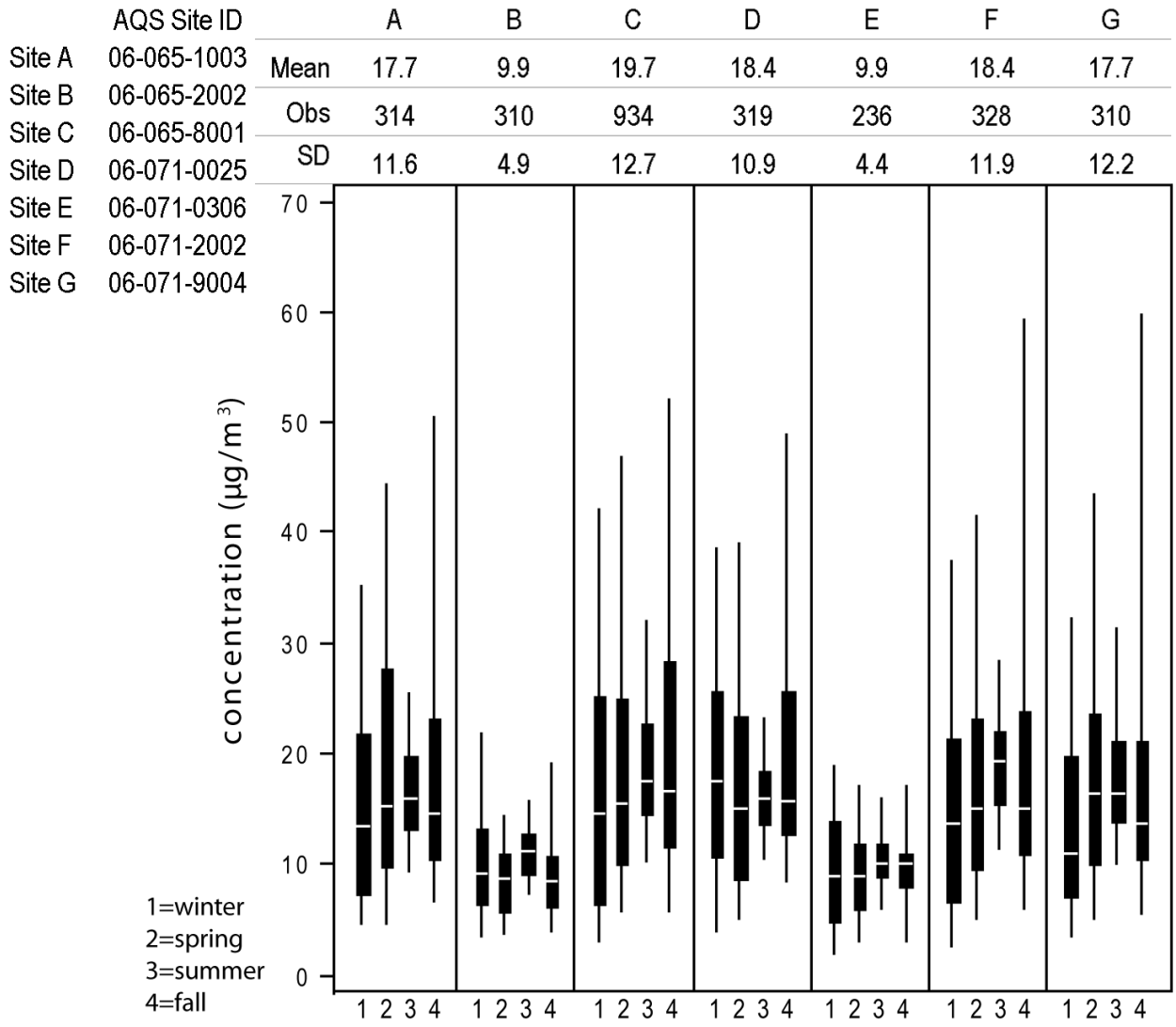


Figure A-74. Box plots illustrating the seasonal distribution of 24-h avg $PM_{2.5}$ concentrations for Riverside, CA.

Table A-32. 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)
		LEGEND		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

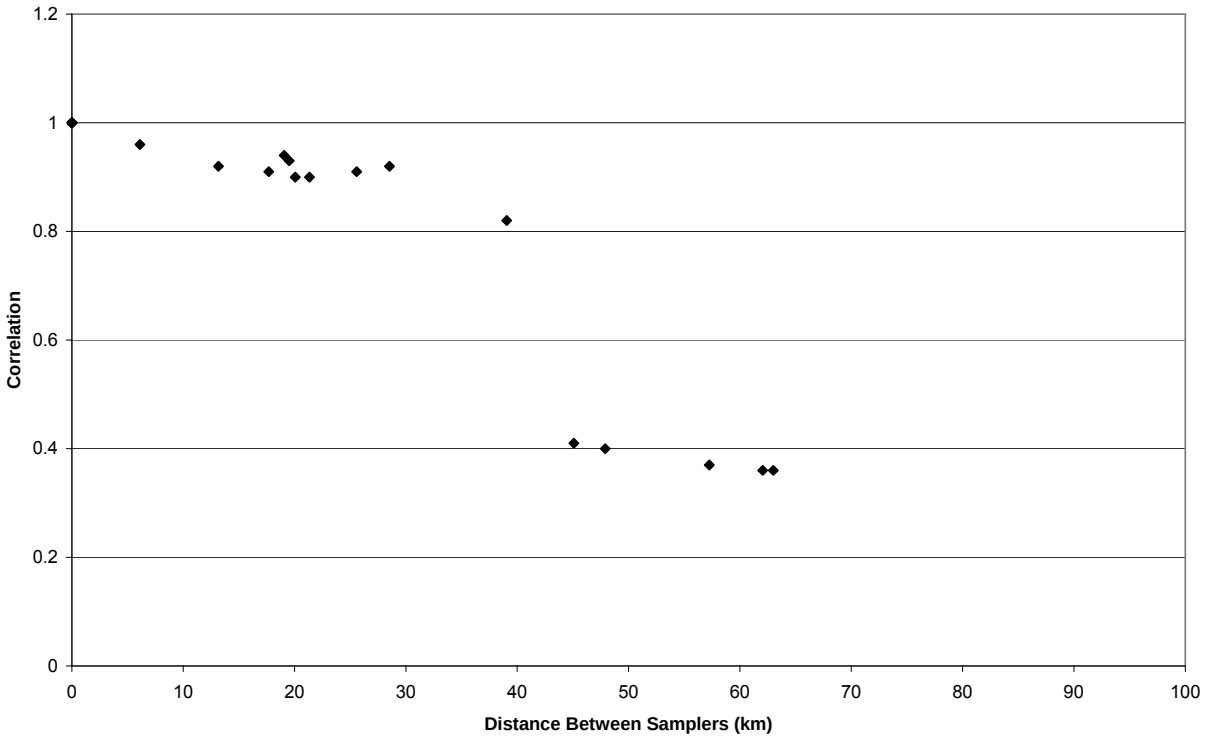


Figure A-75. $PM_{2.5}$ inter-sampler correlations as a function of distance between monitors for Riverside CA.

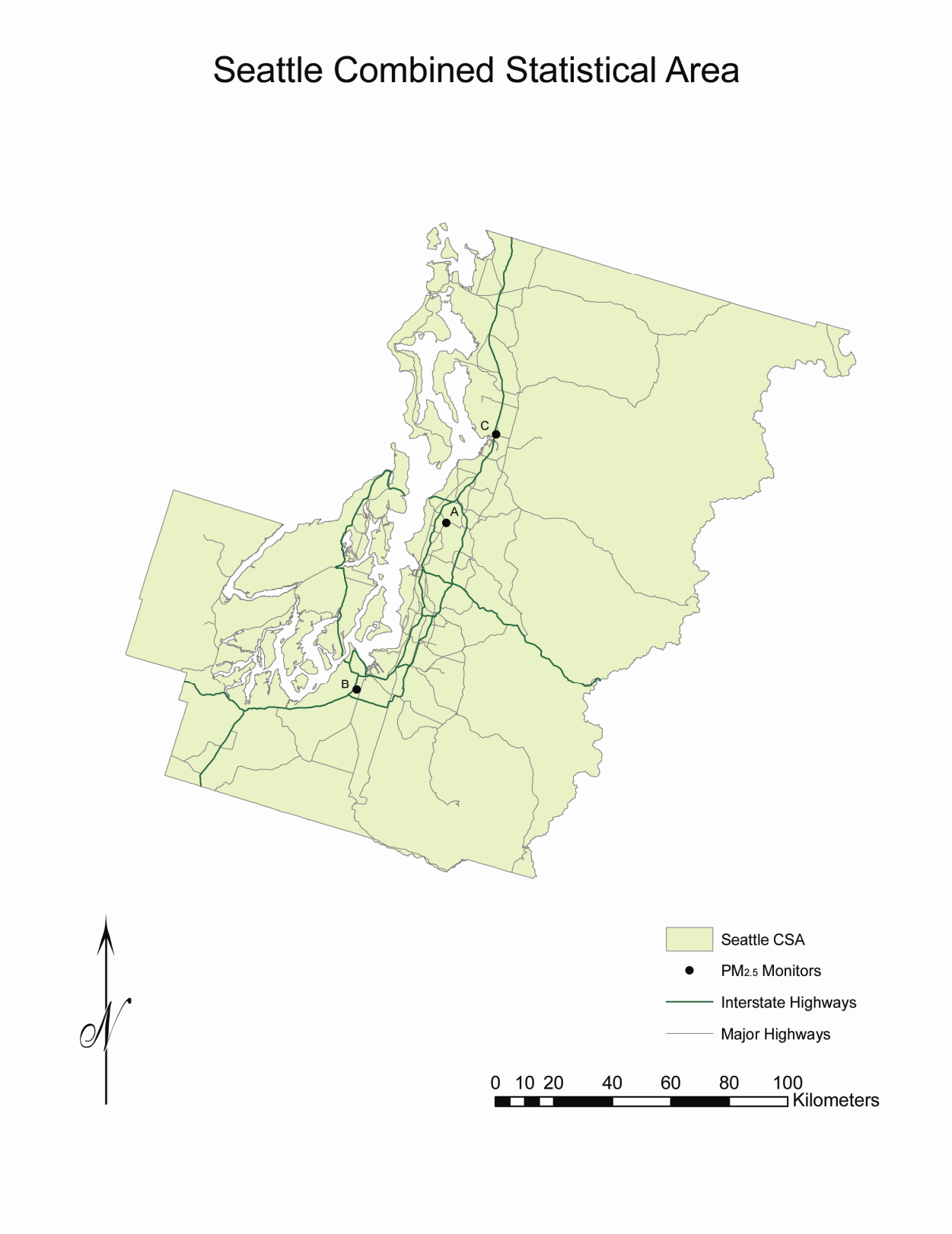


Figure A-76. $PM_{2.5}$ monitor distribution and major highways, Seattle, WA.

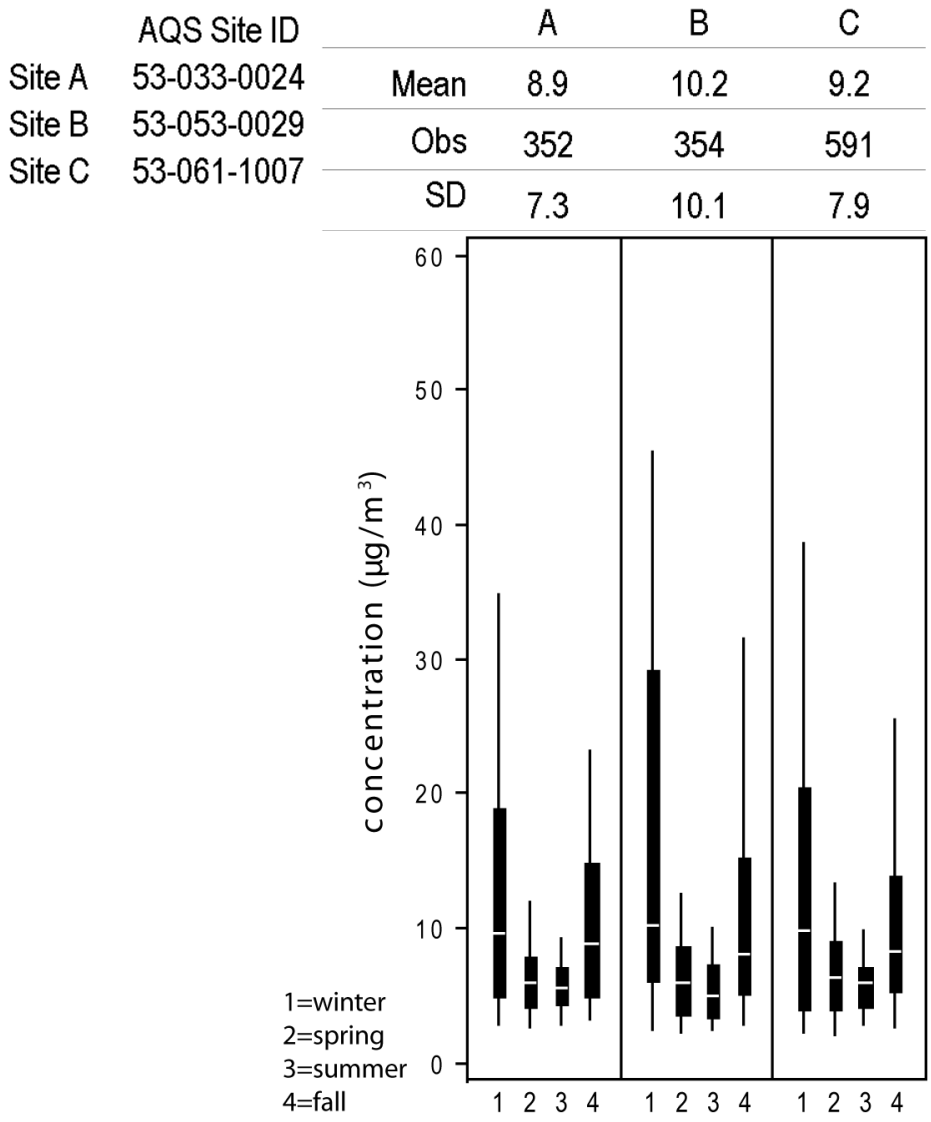


Figure A-77. Box plots illustrating the seasonal distribution of 24-h avg $PM_{2.5}$ concentrations for Seattle, WA.

Table A-33. 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	**LEGEND**		1.00	0.80
	R		(0.0, 0.00)	(7.8, 0.20)
	(P90, COD)		354	335
C	**N**			1.00
				(0.0, 0.00)
				591

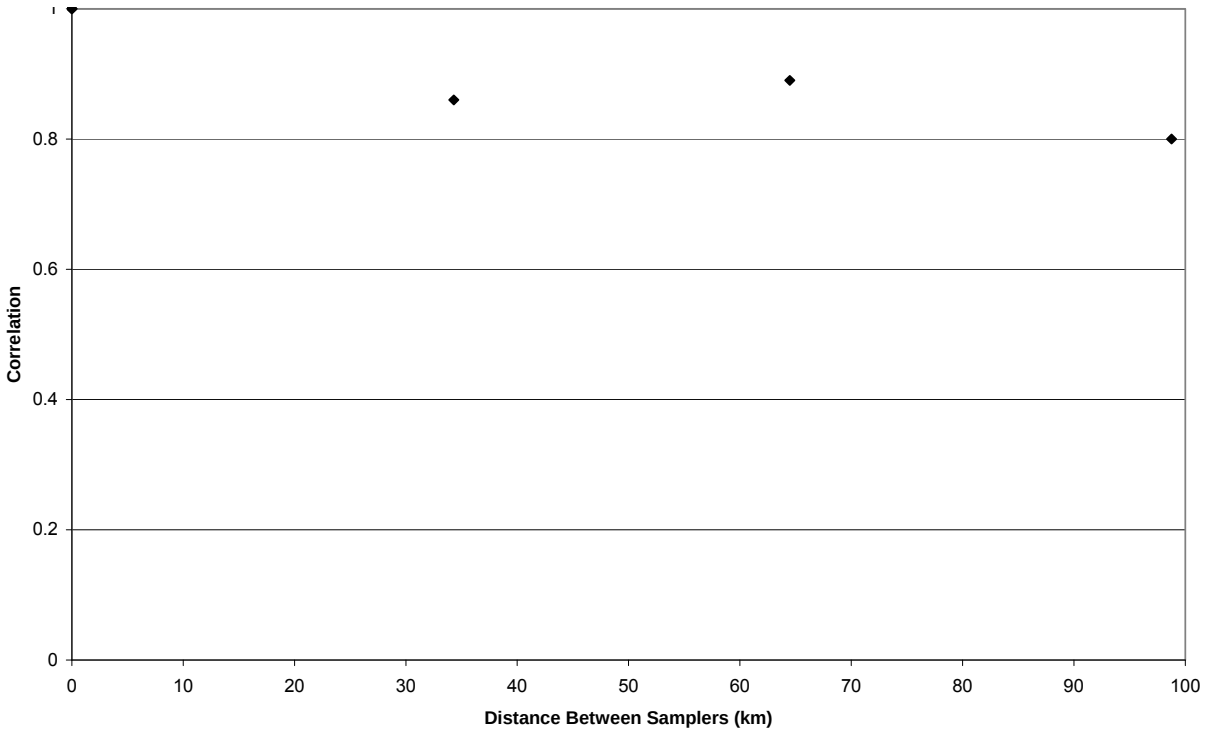


Figure A-78. $PM_{2.5}$ inter-sampler correlations as a function of distance between monitors for Seattle, WA.

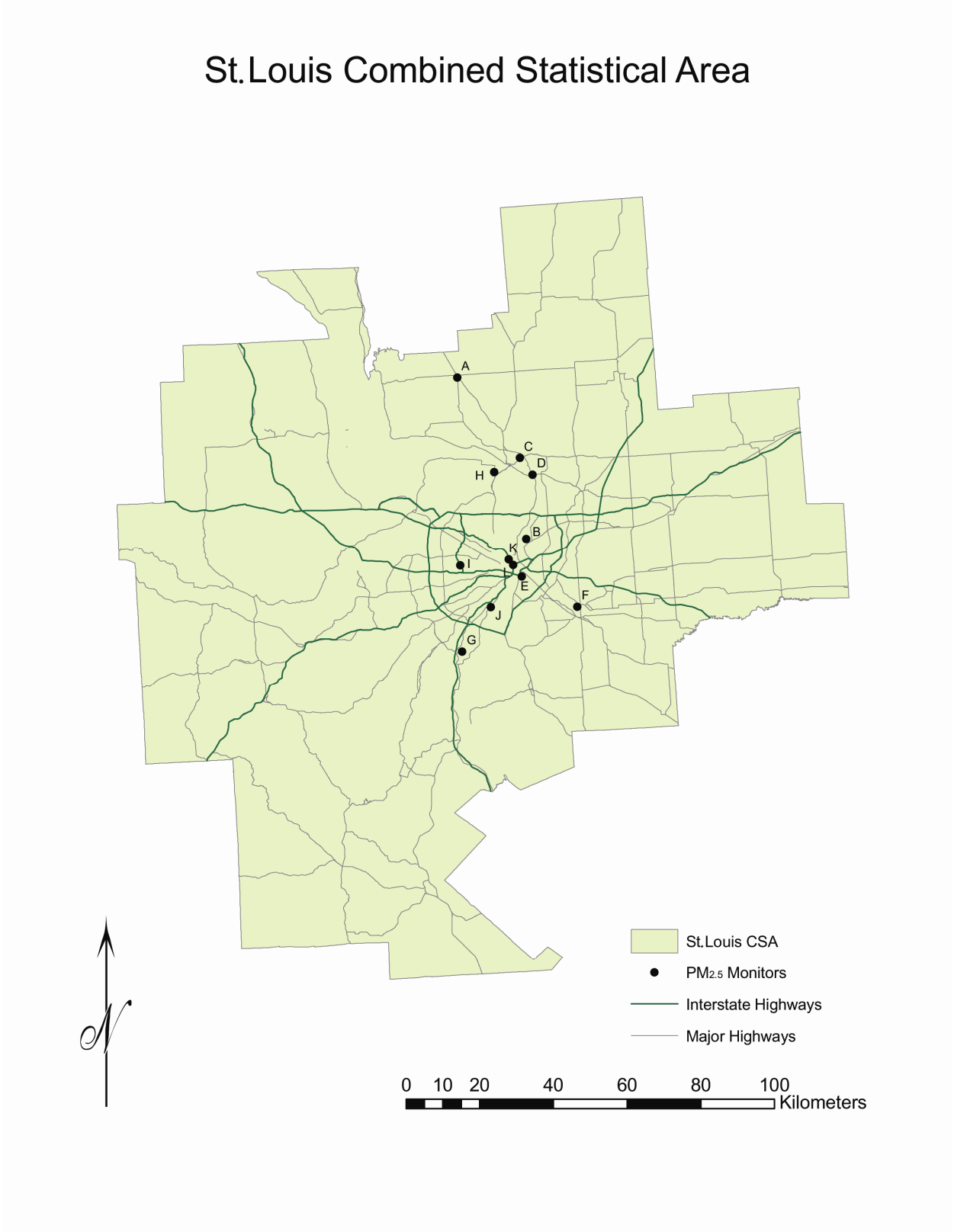


Figure A-79. $PM_{2.5}$ monitor distribution and major highways, St. Louis, MO.

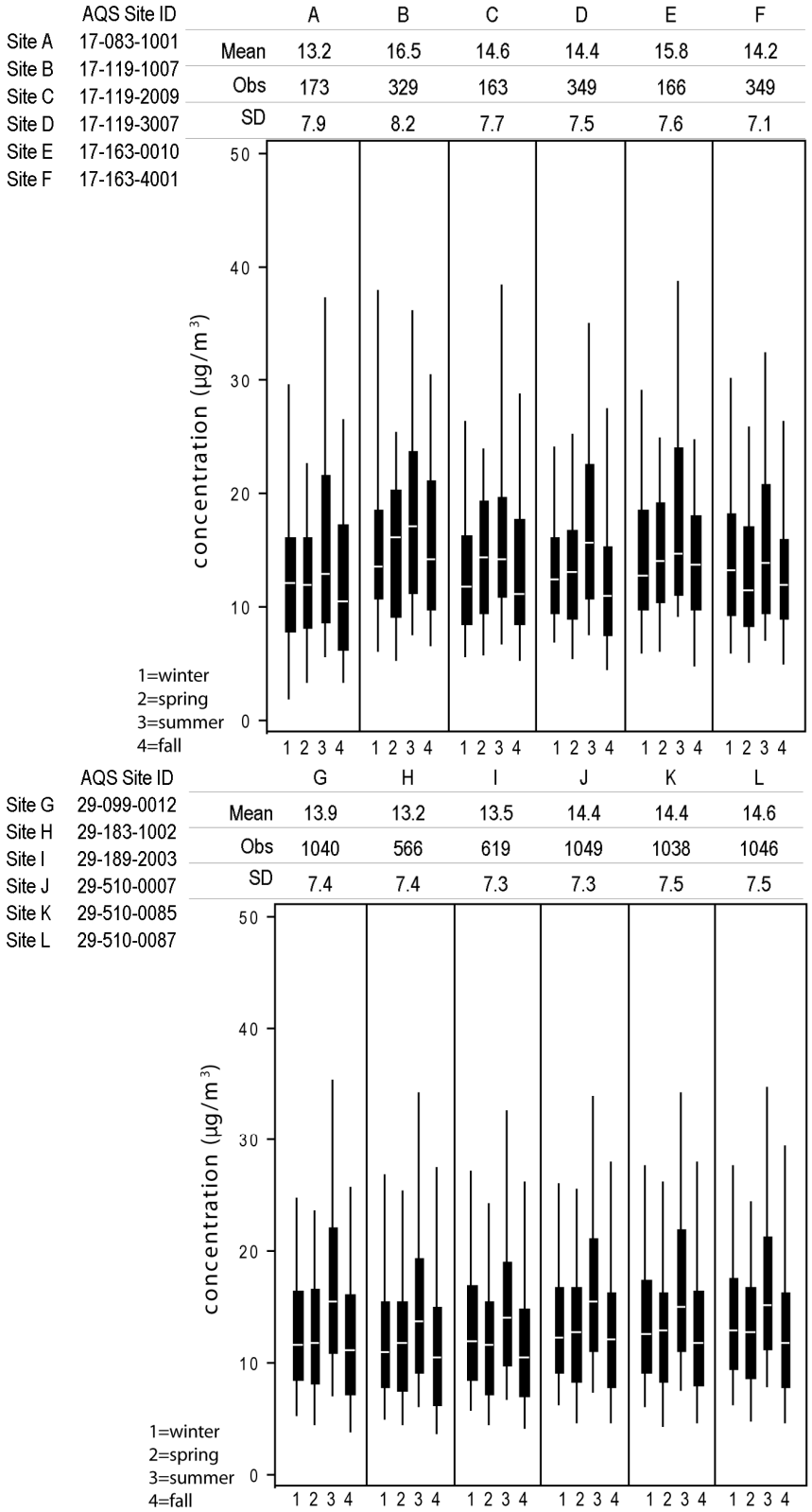


Figure A-80. Box plots illustrating the seasonal distribution of 24-h avg $PM_{2.5}$ concentrations for St. Louis, MO.

Table A-34. 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)
		LEGEND				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

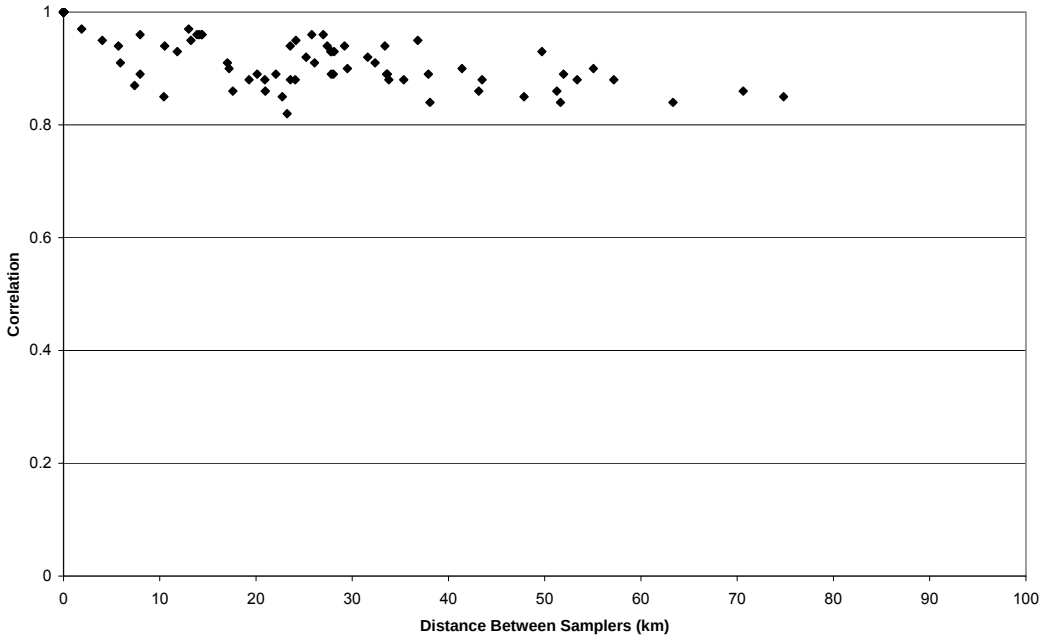


Figure A-81 $PM_{2.5}$ inter-sampler correlations as a function of distance between monitors for St. Louis, MO.

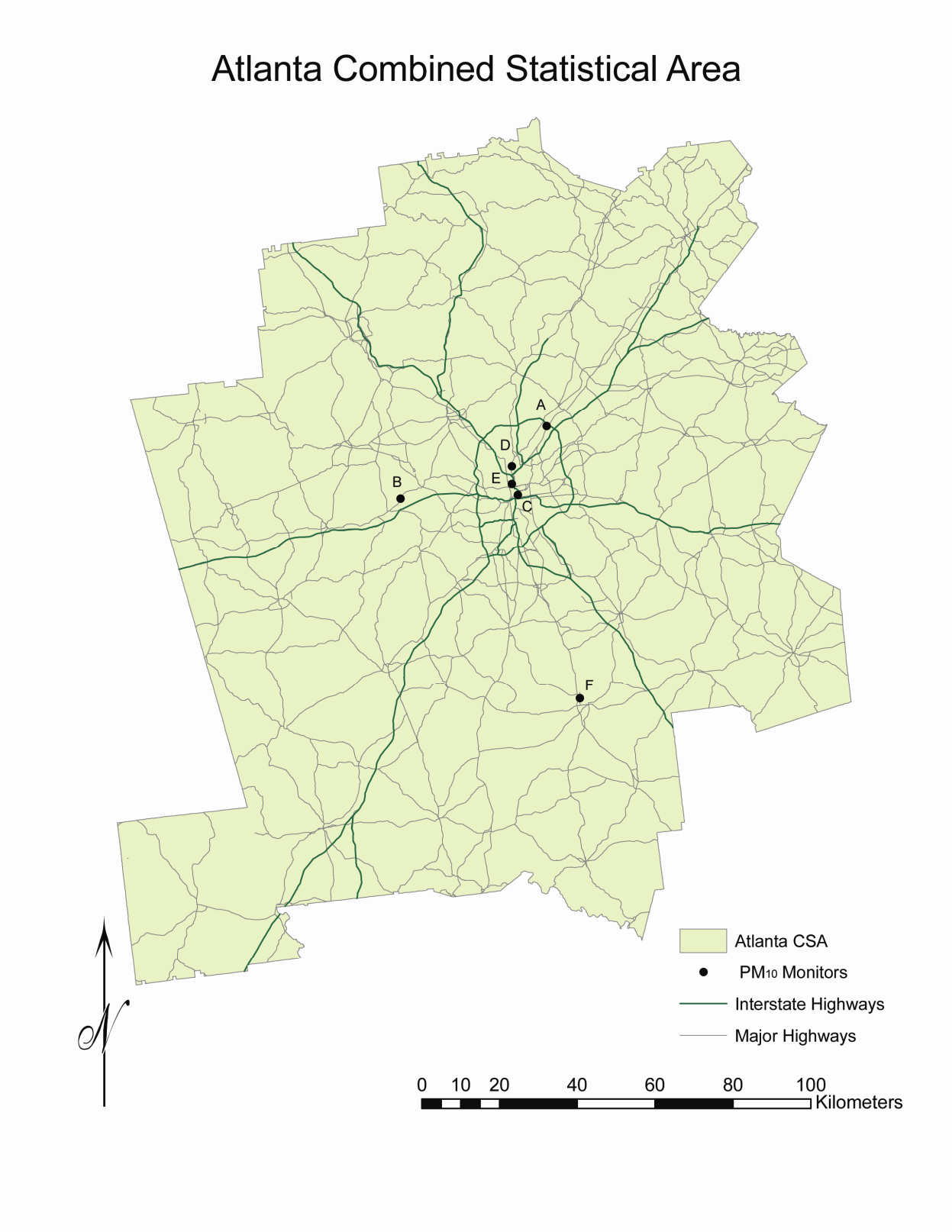


Figure A-82. PM_{10} monitor distribution and major highways, Atlanta, GA.

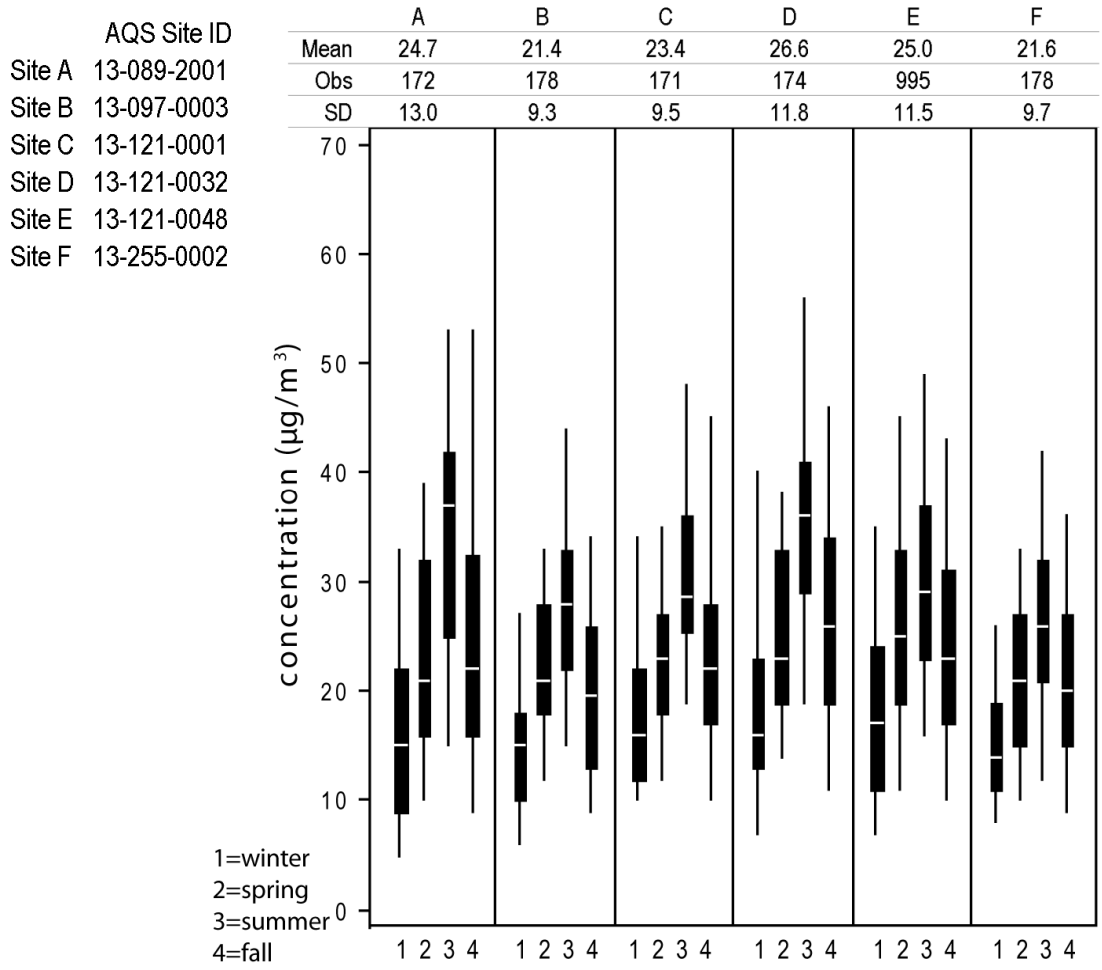


Figure A-83. Box plots illustrating the seasonal distribution of 24-h avg PM_{10} concentrations for Atlanta, GA.

Table A-35. 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	**LEGEND**			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

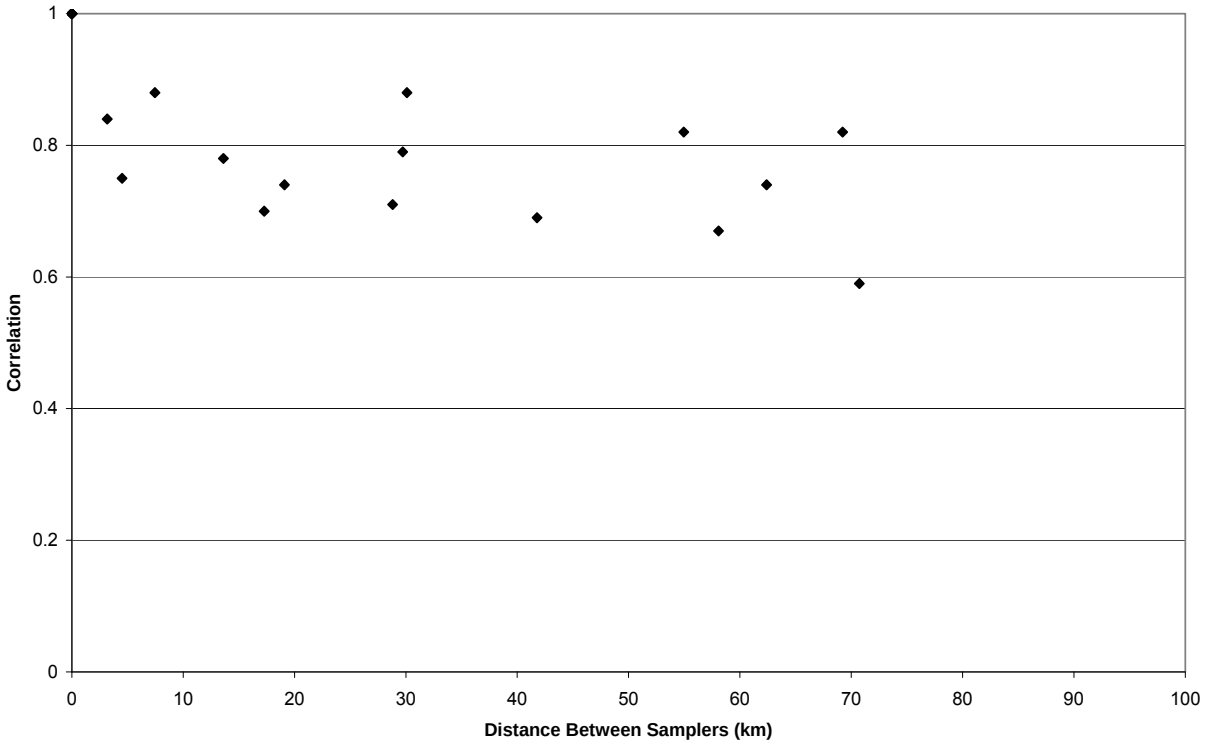


Figure A-84. PM_{10} inter-sampler correlations as a function of distance between monitors for Atlanta, GA.

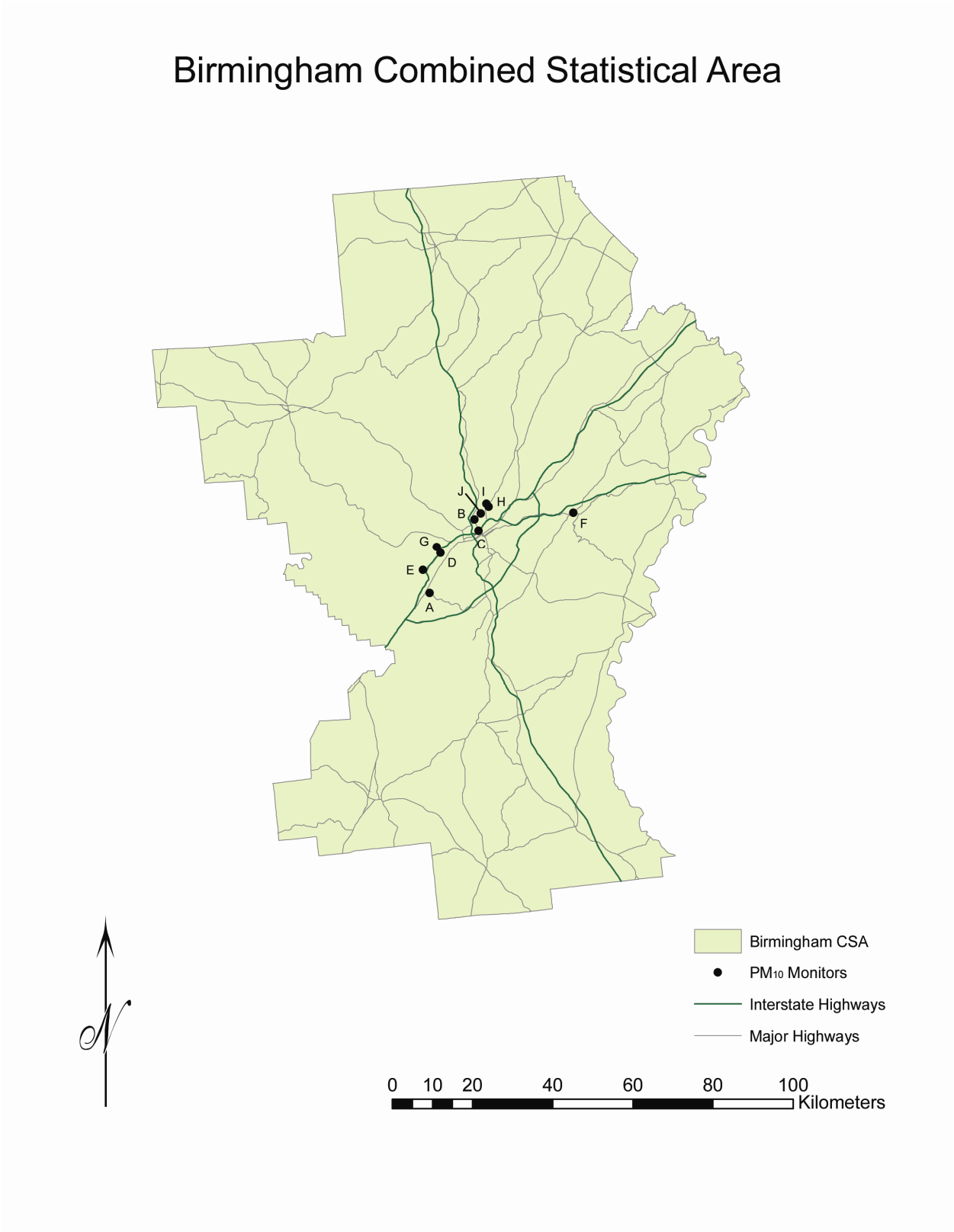


Figure A-85. PM_{10} monitor distribution and major highways, Birmingham, AL.

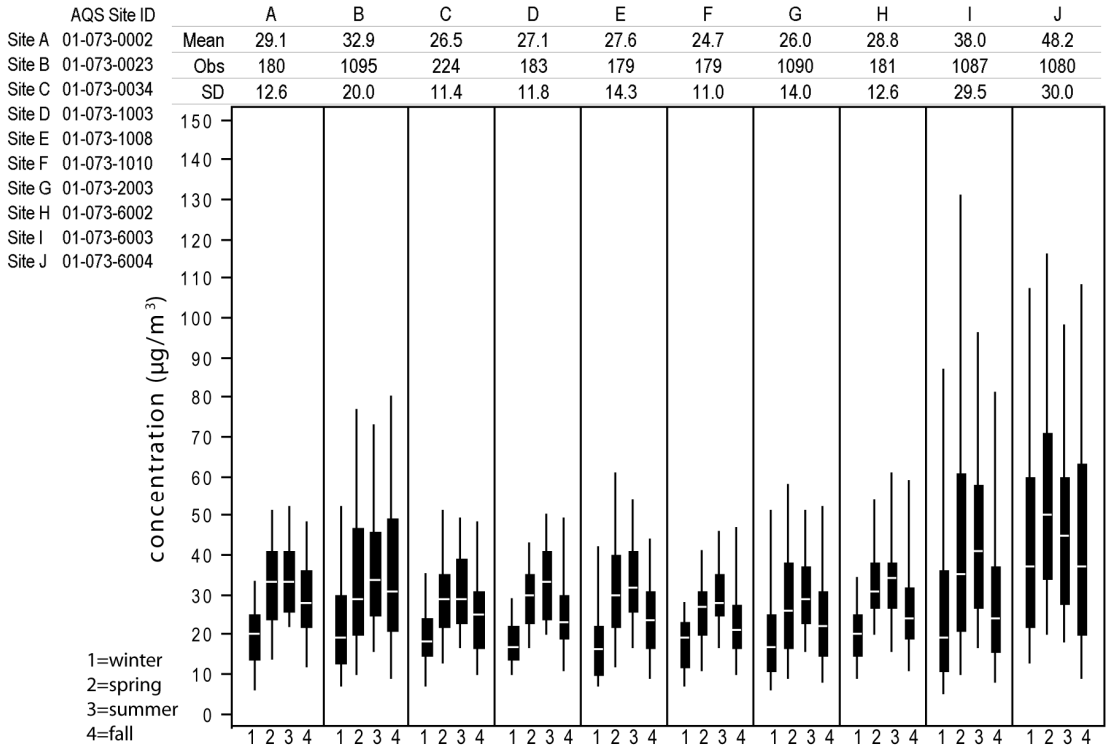


Figure A-86. Box plots illustrating the seasonal distribution of 24-h avg PM_{10} concentrations for Birmingham, AL.

Table A-36. 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
		LEGEND				(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

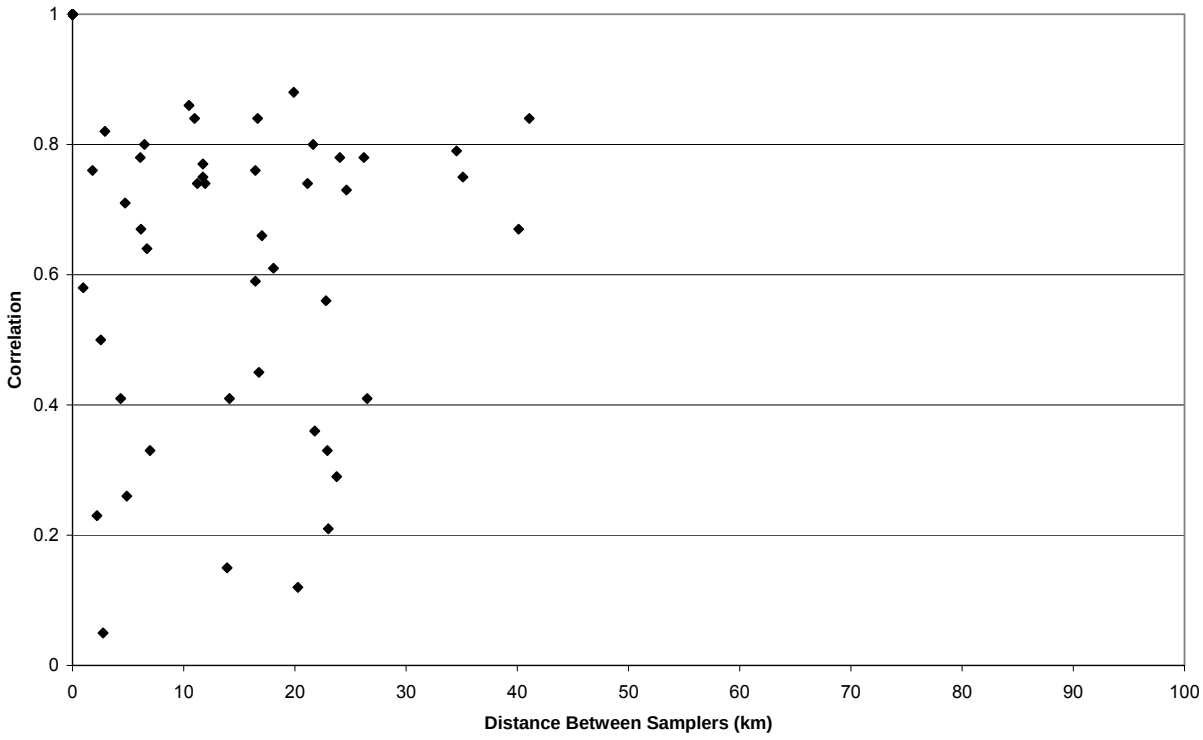


Figure A-87 PM_{10} inter-sampler correlations as a function of distance between monitors for Birmingham, AL.

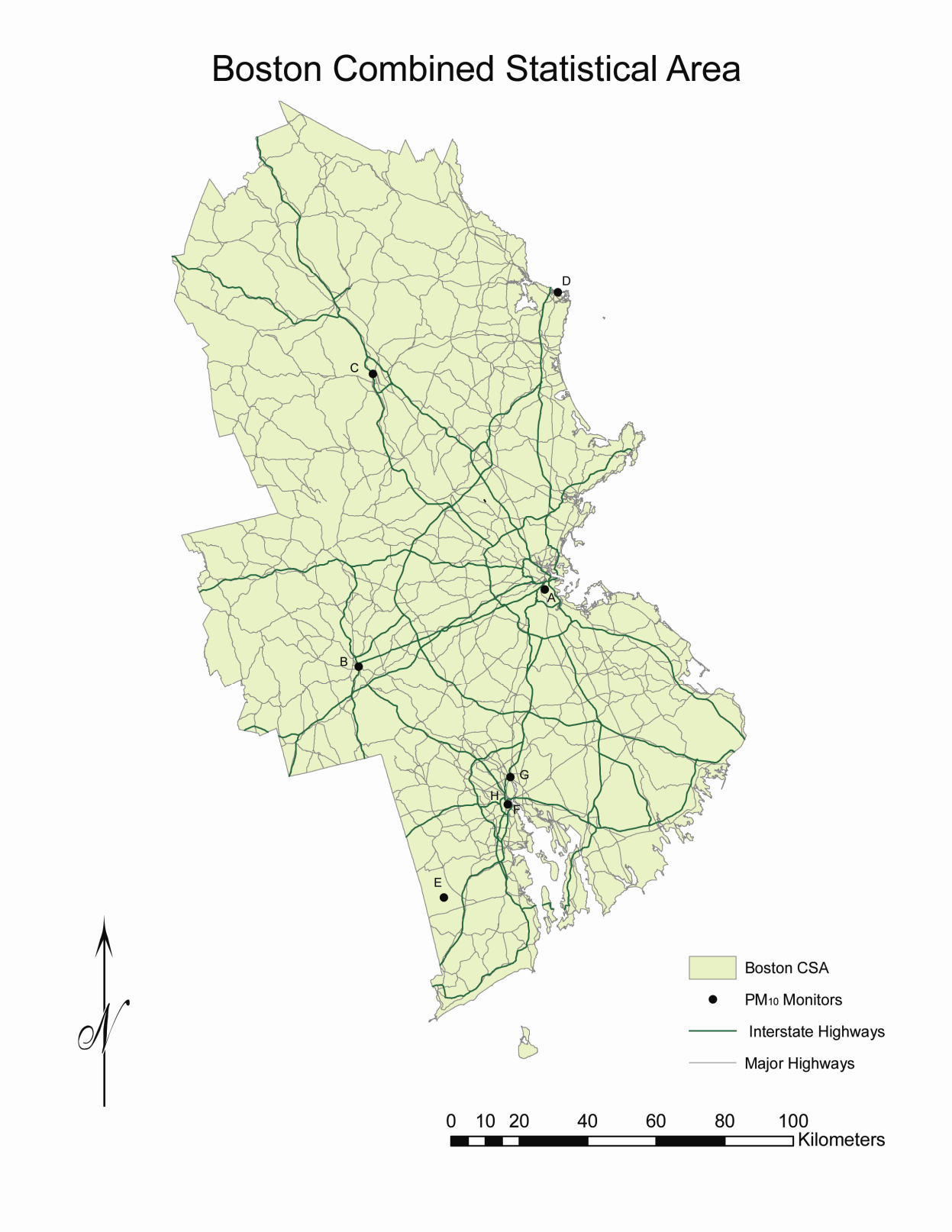


Figure A-88. PM_{10} monitor distribution and major highways, Boston, MA.

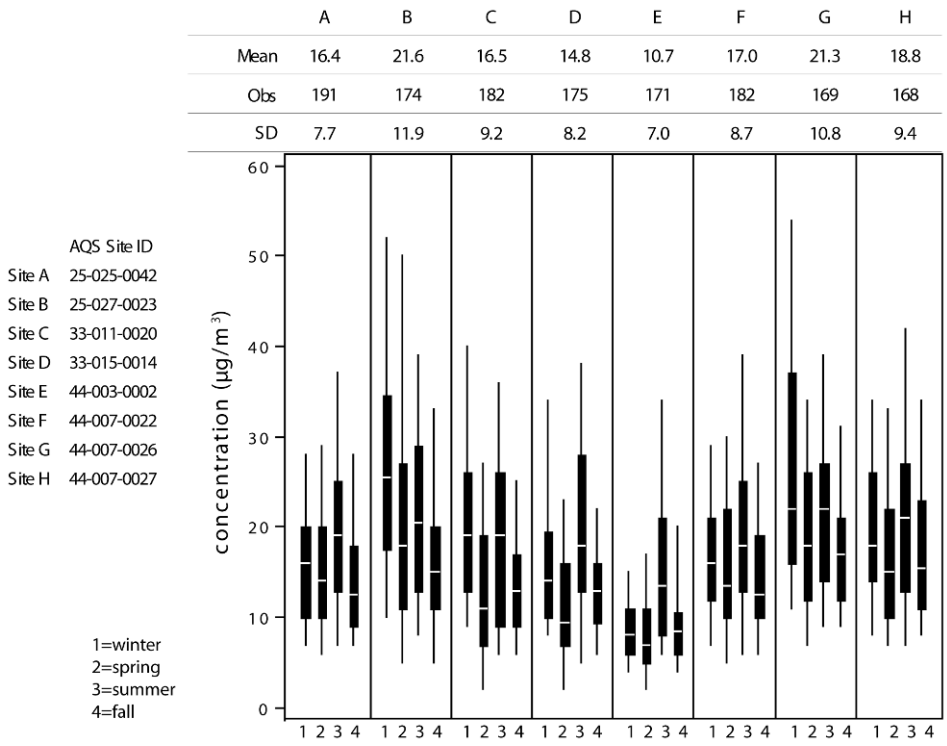


Figure A-89. Box plots illustrating the seasonal distribution of 24-h avg PM_{10} concentrations for Boston, MA.

Table A-37. Inter-sampler correlation statistics for each pair of PM_{10} monitors reporting to AQS for Boston, MA.

Site	A	B	C	D	E	F	G	H
A	1.00	0.69	0.69	0.73	0.71	0.84	0.70	0.79
	(0.0, 0.00)	(15.0, 0.22)	(12.0, 0.20)	(10.0, 0.22)	(13.0, 0.30)	(8.0, 0.14)	(15.0, 0.20)	(10.0, 0.17)
	191	169	179	173	171	182	169	167
B		1.00	0.66	0.56	0.45	0.69	0.77	0.65
		(0.0, 0.00)	(17.0, 0.24)	(19.0, 0.28)	(24.0, 0.39)	(15.0, 0.21)	(12.0, 0.17)	(16.0, 0.20)
		174	167	161	158	169	156	154
C			1.00	0.72	0.47	0.62	0.64	0.59
			(0.0, 0.00)	(10.0, 0.22)	(17.0, 0.33)	(12.0, 0.21)	(16.0, 0.26)	(16.0, 0.24)
			182	170	168	179	166	164
D				1.00	0.63	0.68	0.59	0.69
				(0.0, 0.00)	(11.0, 0.29)	(10.0, 0.23)	(19.0, 0.30)	(13.0, 0.26)
				175	163	173	161	158
E					1.00	0.84	0.58	0.80
					(0.0, 0.00)	(13.0, 0.29)	(22.0, 0.38)	(15.0, 0.33)
					171	171	161	157
F						1.00	0.81	0.95
						(0.0, 0.00)	(11.0, 0.16)	(5.0, 0.11)
						182	169	167
G							1.00	0.79
							(0.0, 0.00)	(10.0, 0.13)
							169	154
H								1.00
								(0.0, 0.00)
								168

LEGEND
Pearson R
(P90, COD)
n

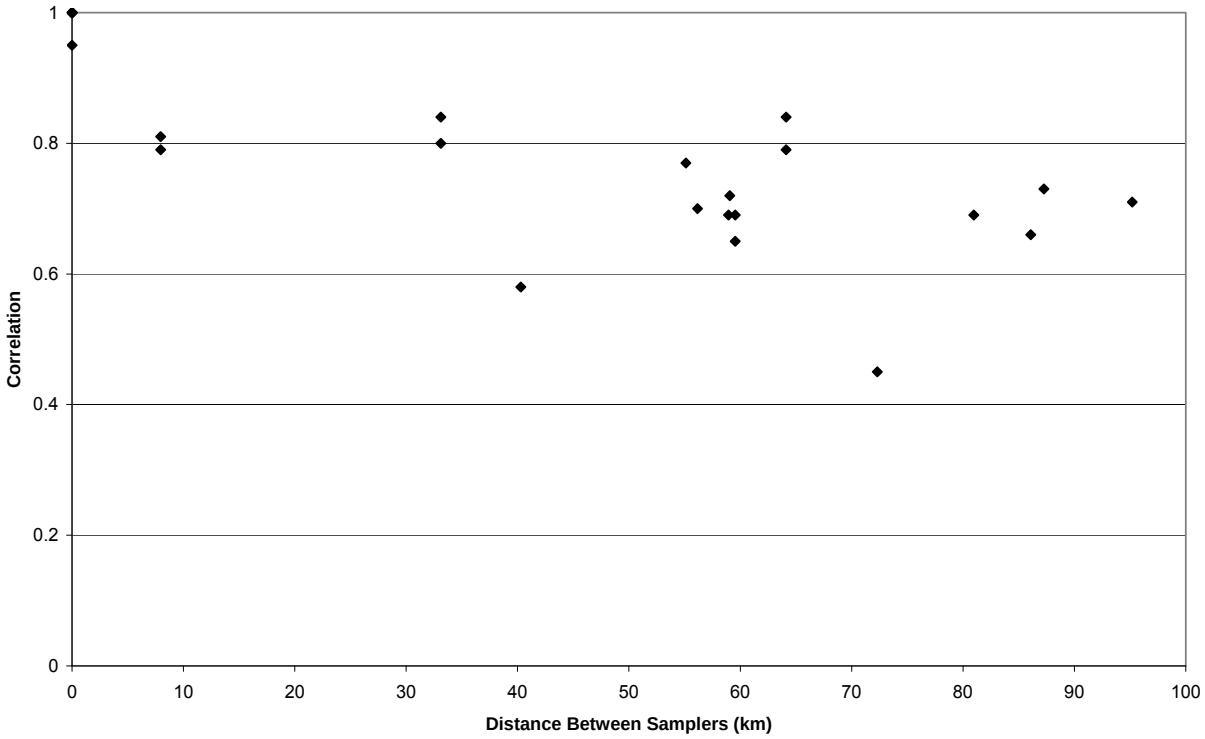


Figure A-90 PM_{10} inter-sampler correlations as a function of distance between monitors for Boston, MA.

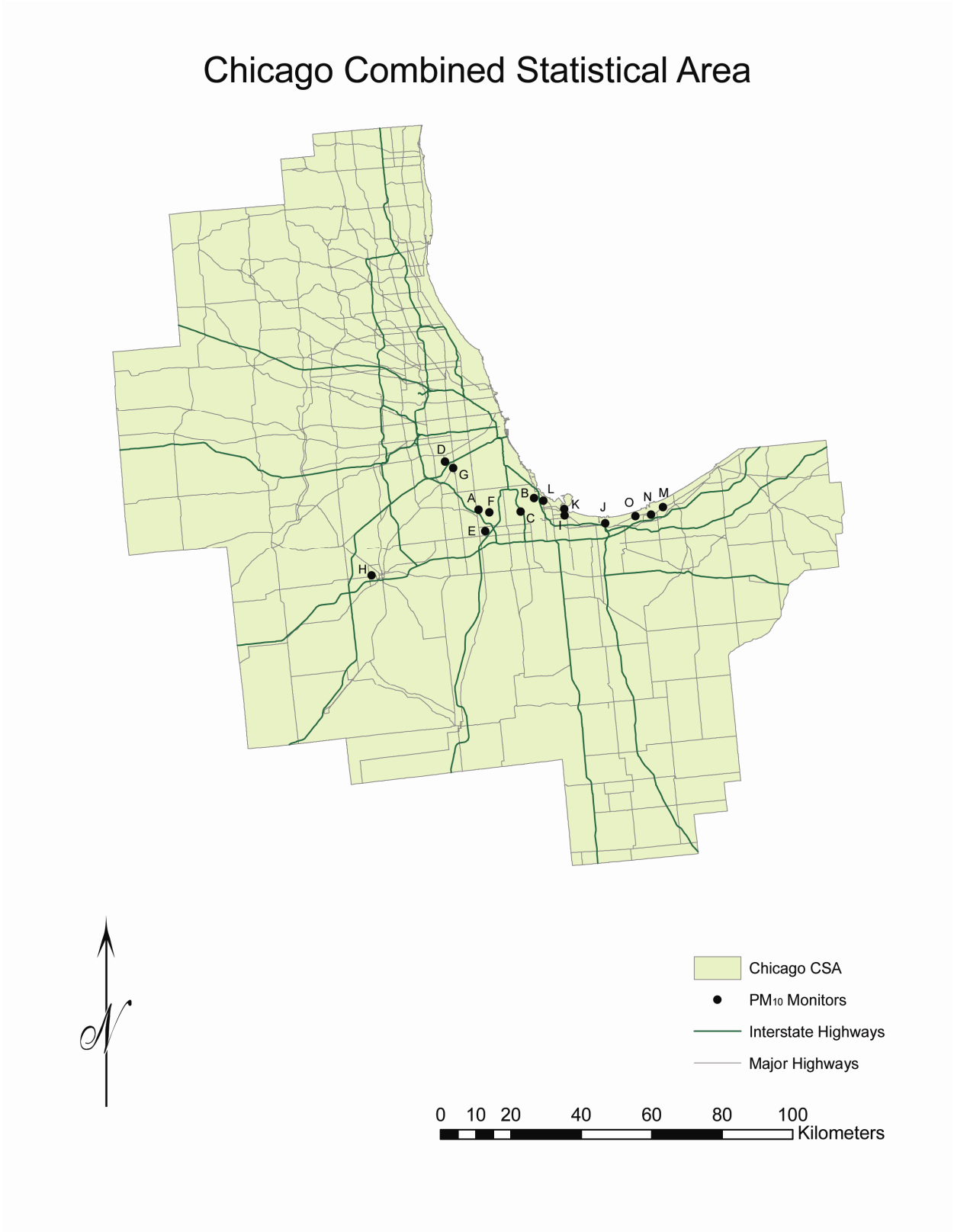


Figure A-91. PM_{10} monitor distribution and major highways, Chicago, IL.

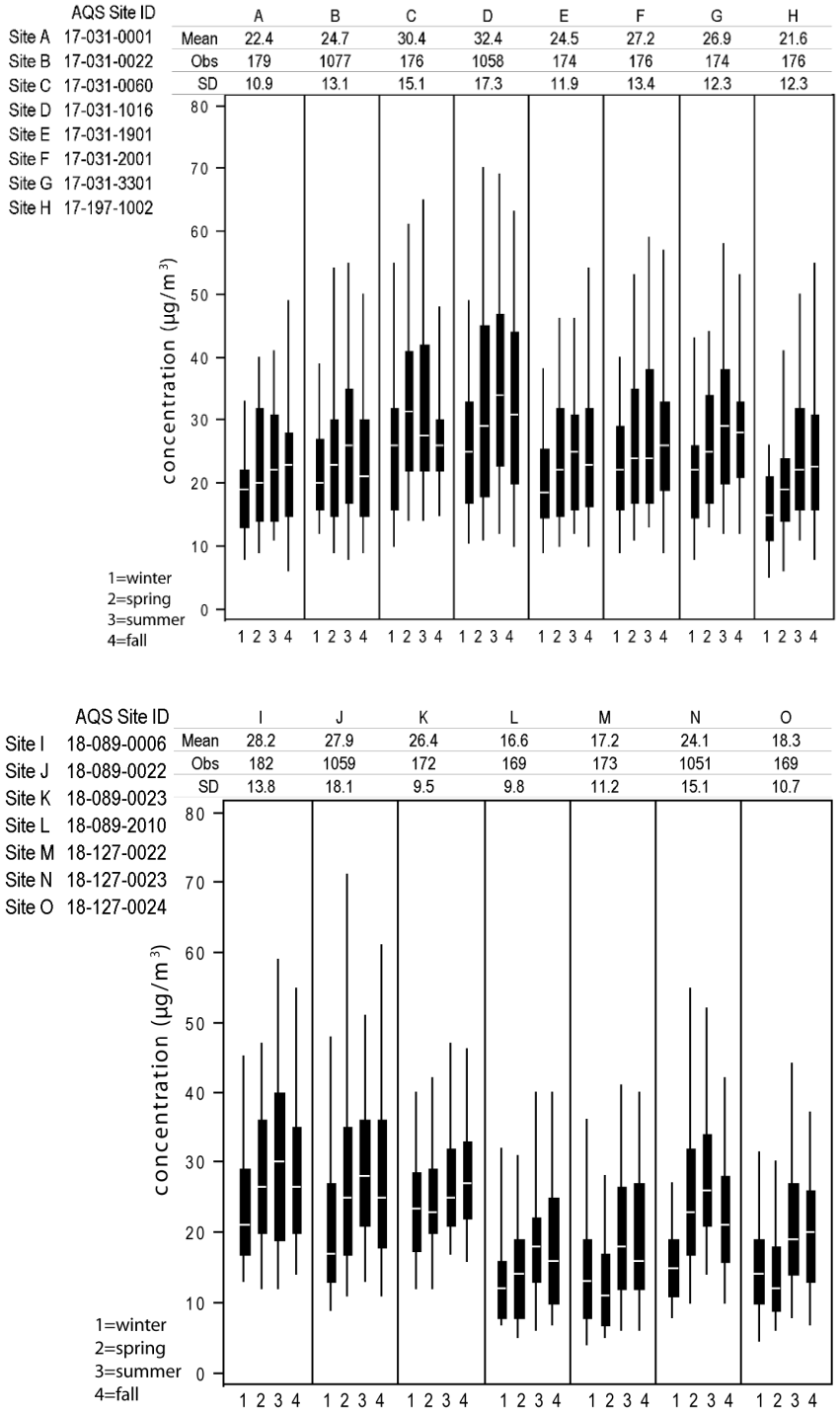


Figure A-92. Box plots illustrating the seasonal distribution of 24-h avg PM_{10} concentrations for Chicago, IL.

Table A-38. 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)
			LEGEND						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

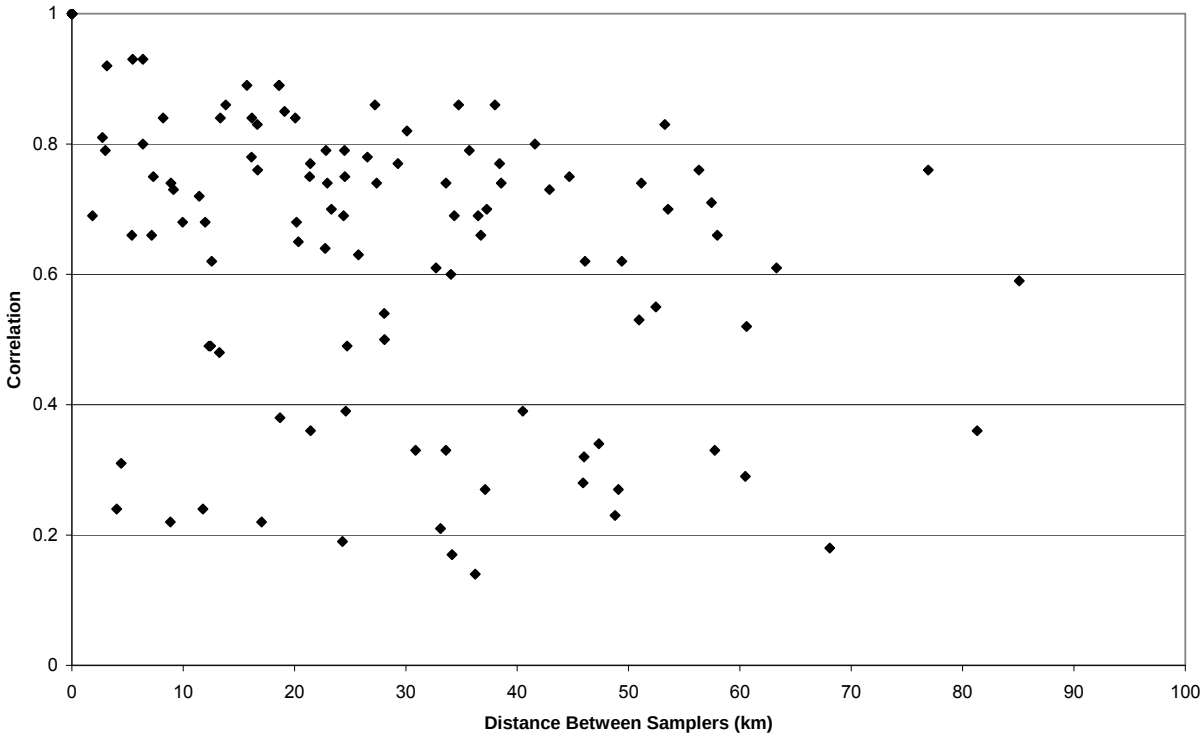


Figure A-93. PM_{10} inter-sampler correlations as a function of distance between monitors for Chicago, IL.

Figure A-94. PM_{10} monitor distribution and major highways, Denver, CO.

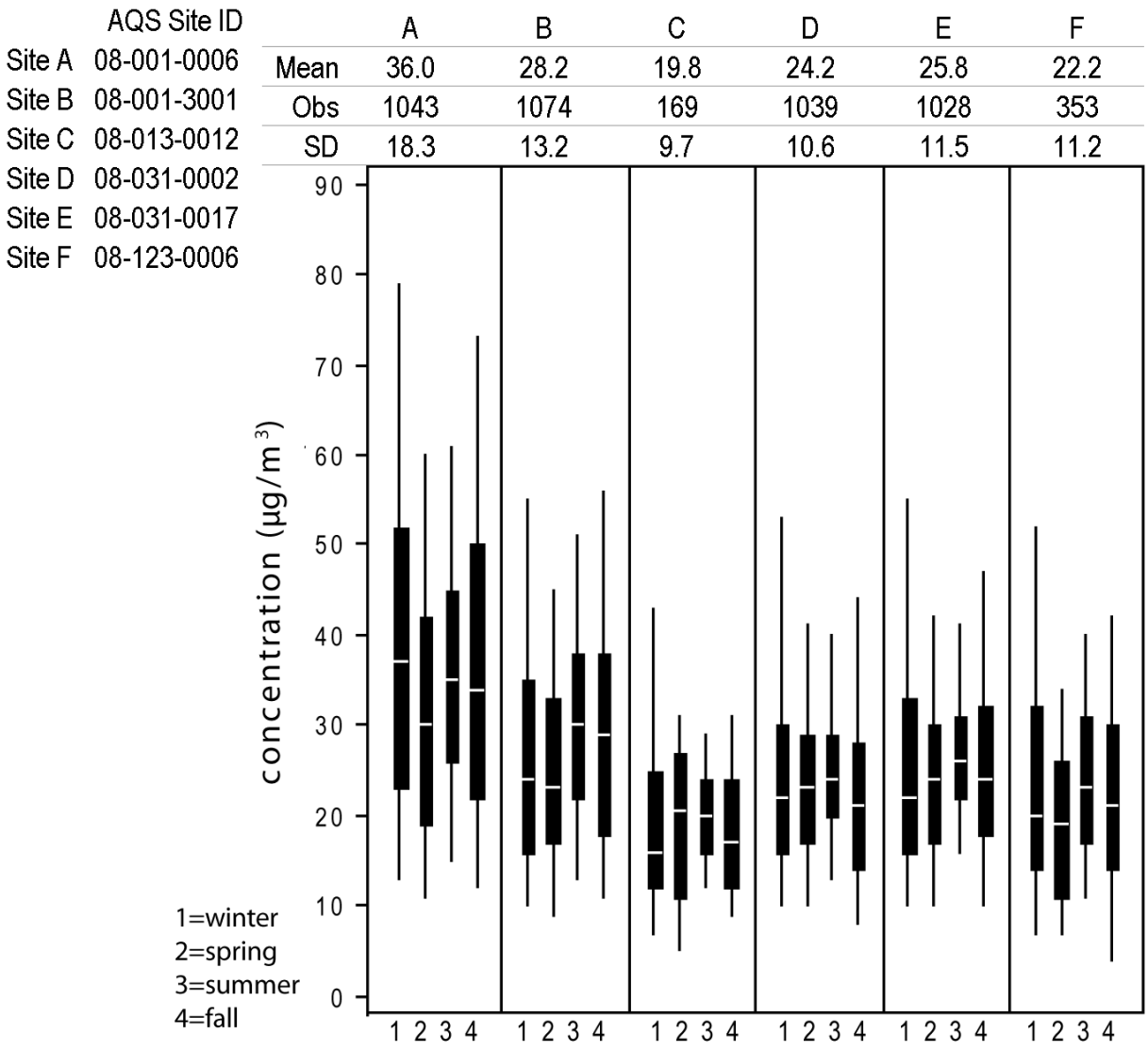


Figure A-95. Box plots illustrating the seasonal distribution of 24-h avg PM_{10} concentrations for Denver, CO.

Table A-39. 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)
	LEGEND		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
						(0.0, 0.00)
						353

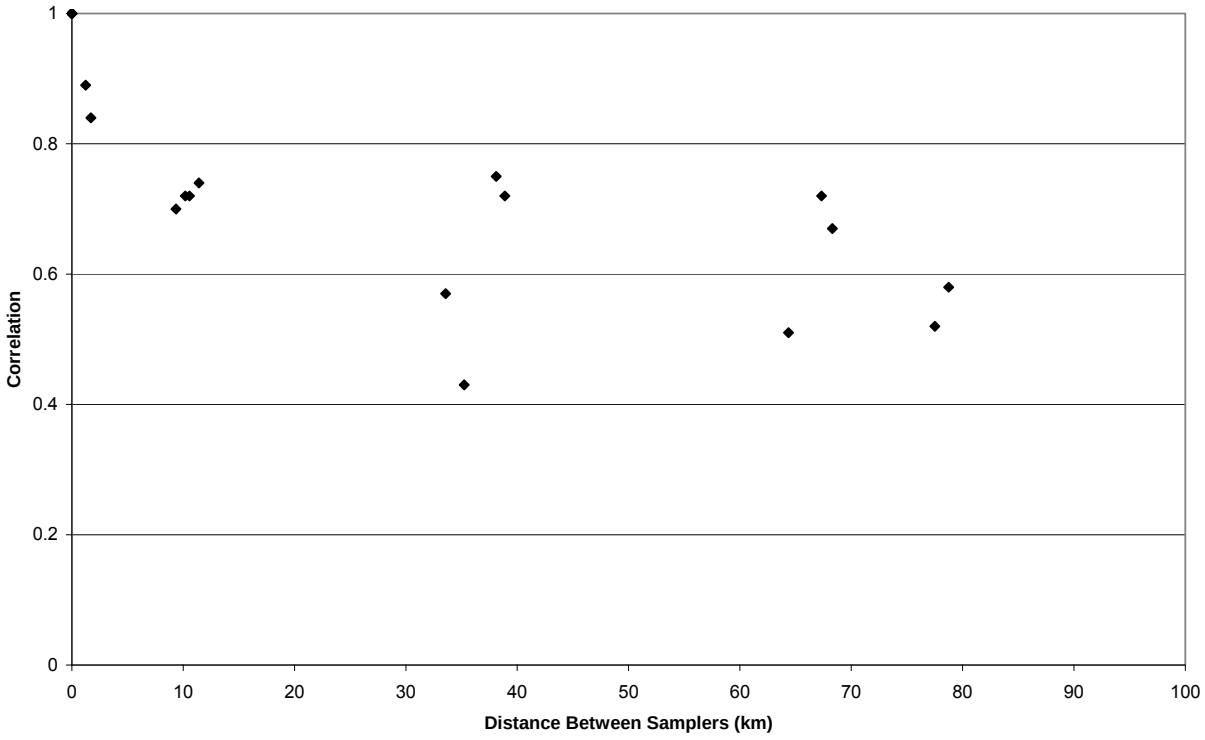


Figure A-96. PM_{10} inter-sampler correlations as a function of distance between monitors for Denver, CO.

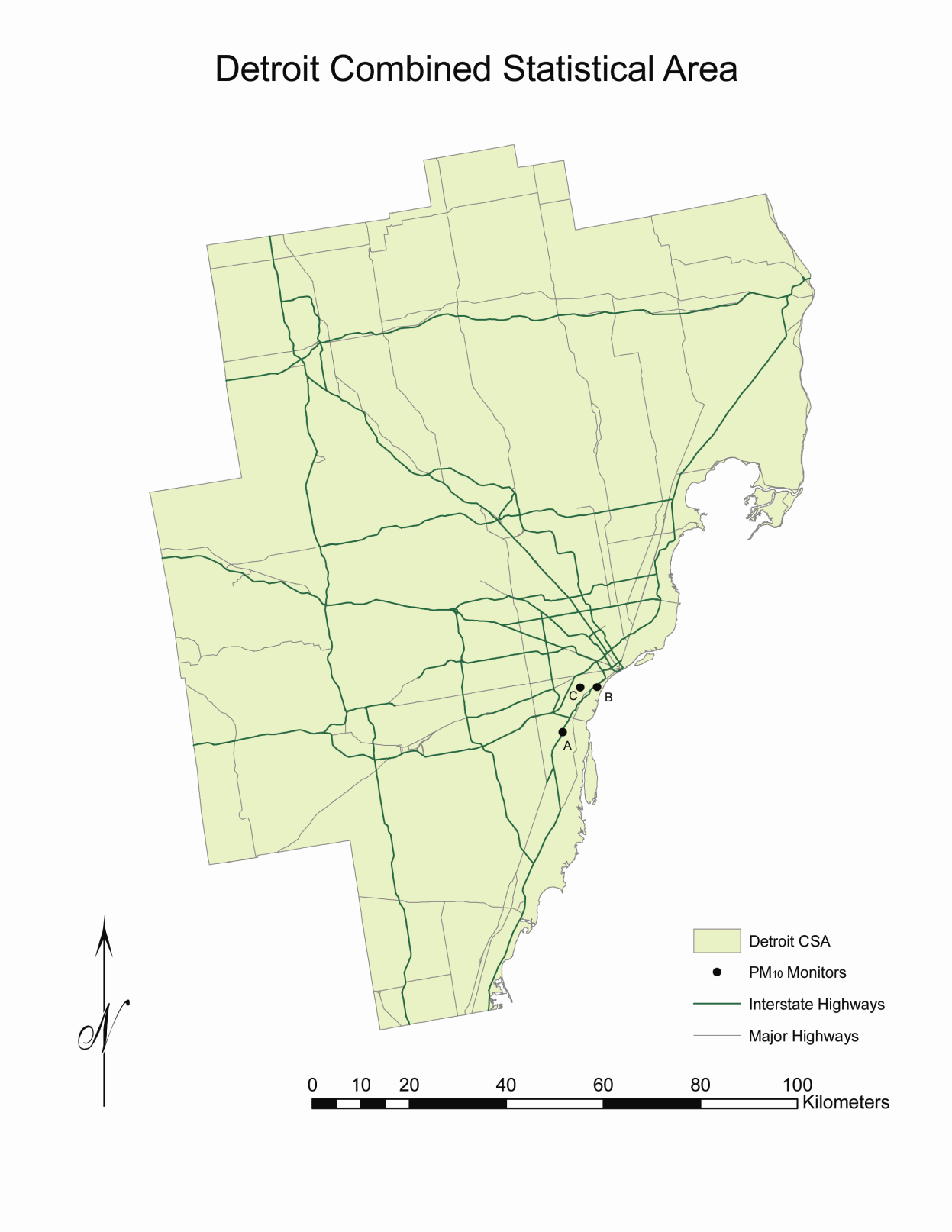


Figure A-97. PM_{10} monitor distribution and major highways, Detroit, MI.

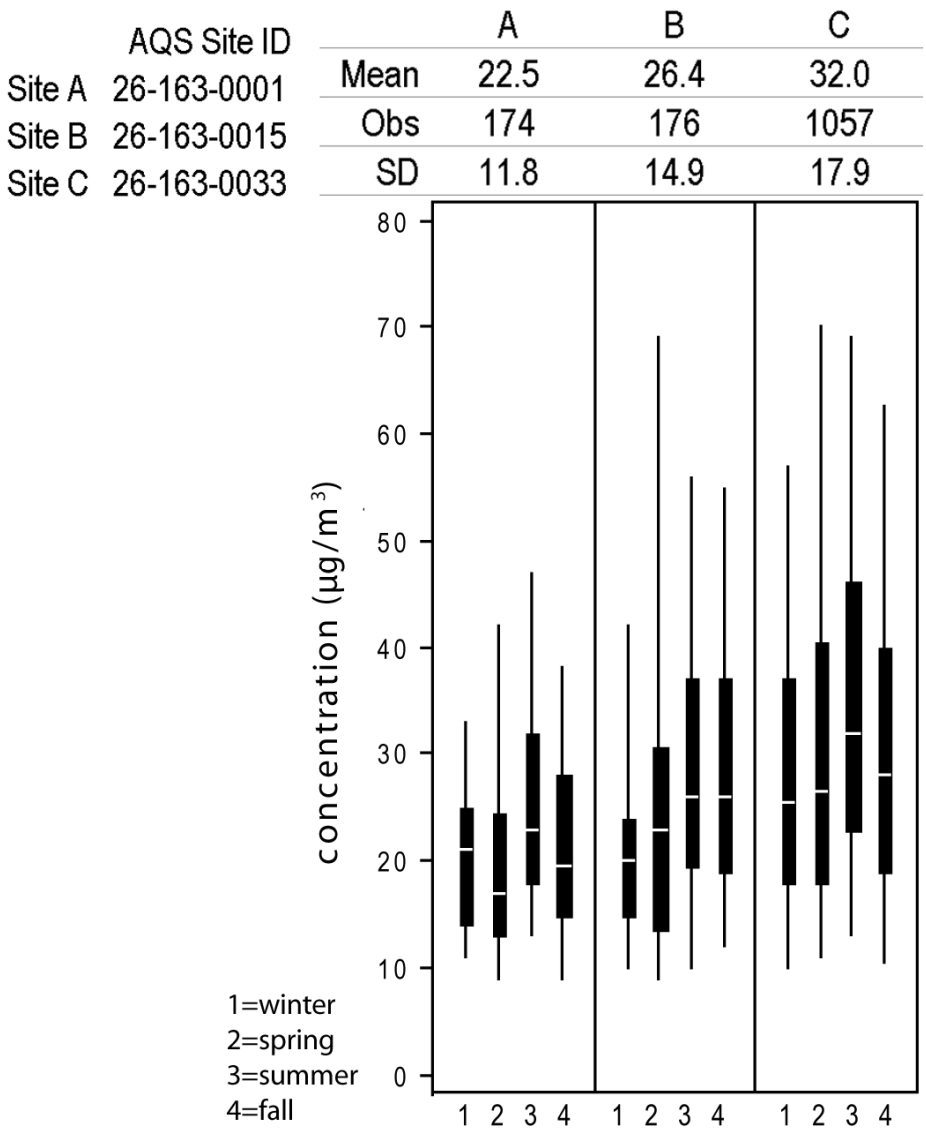


Figure A-98. Box plots illustrating the seasonal distribution of 24-h avg PM_{10} concentrations for Detroit, MI.

Table A-40. 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
	LEGEND	(0.0, 0.00)	(21.0, 0.21)
	R	176	174
C	(P90, COD)		1.00
	N		(0.0, 0.00)
			1057

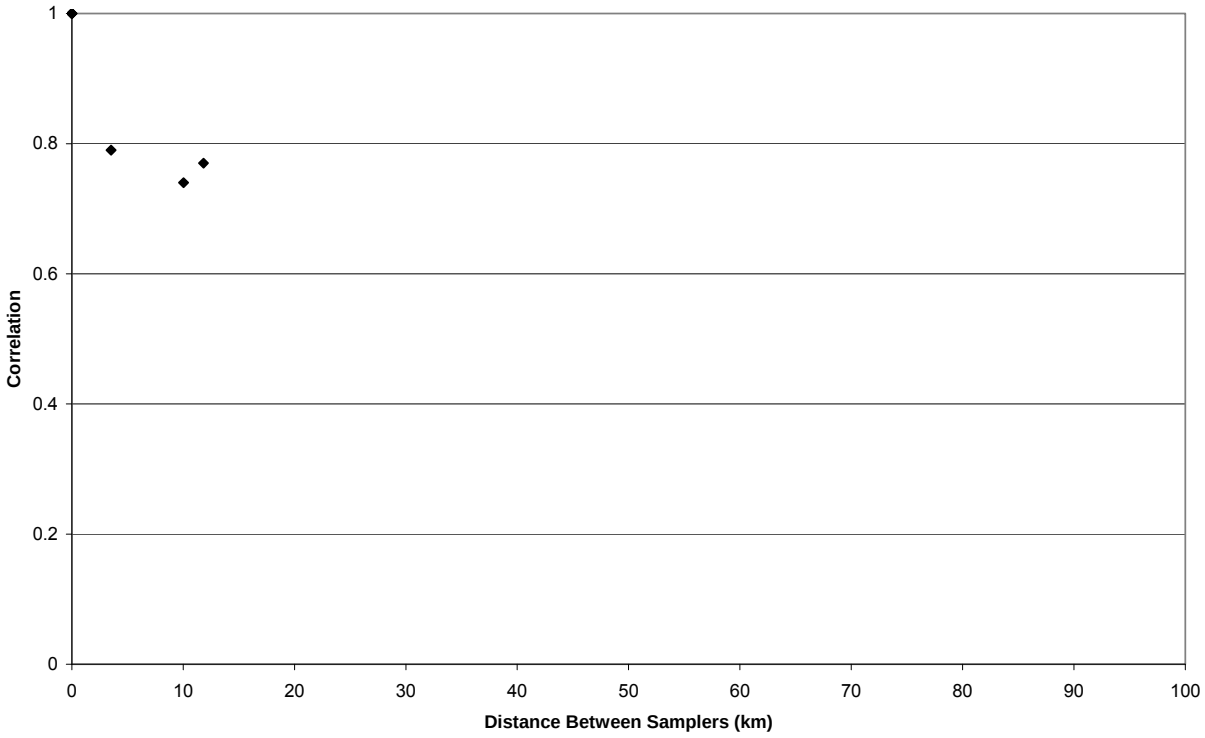


Figure A-99. PM_{10} inter-sampler correlations as a function of distance between monitors for Detroit, MI.

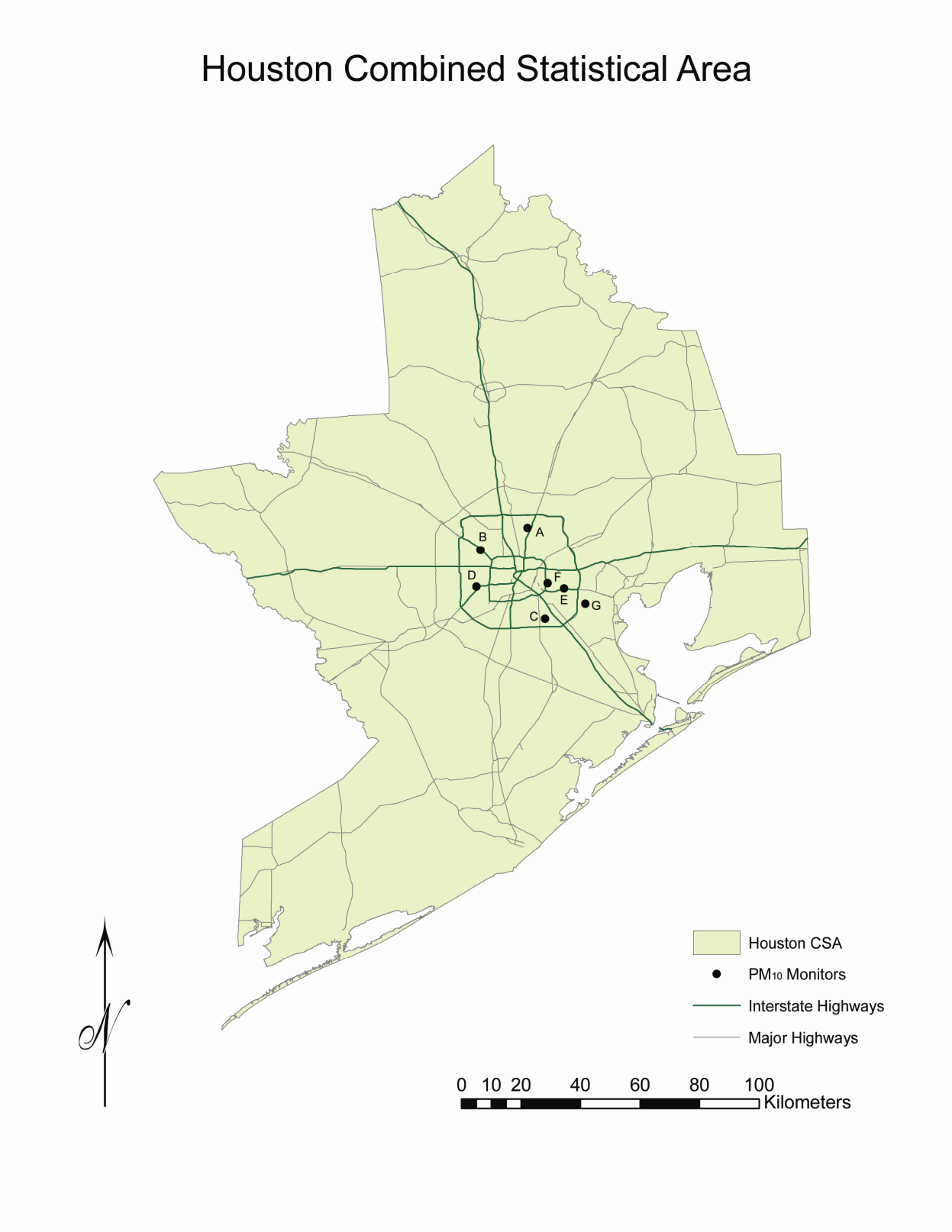


Figure A-100. PM_{10} monitor distribution and major highways, Houston, TX.

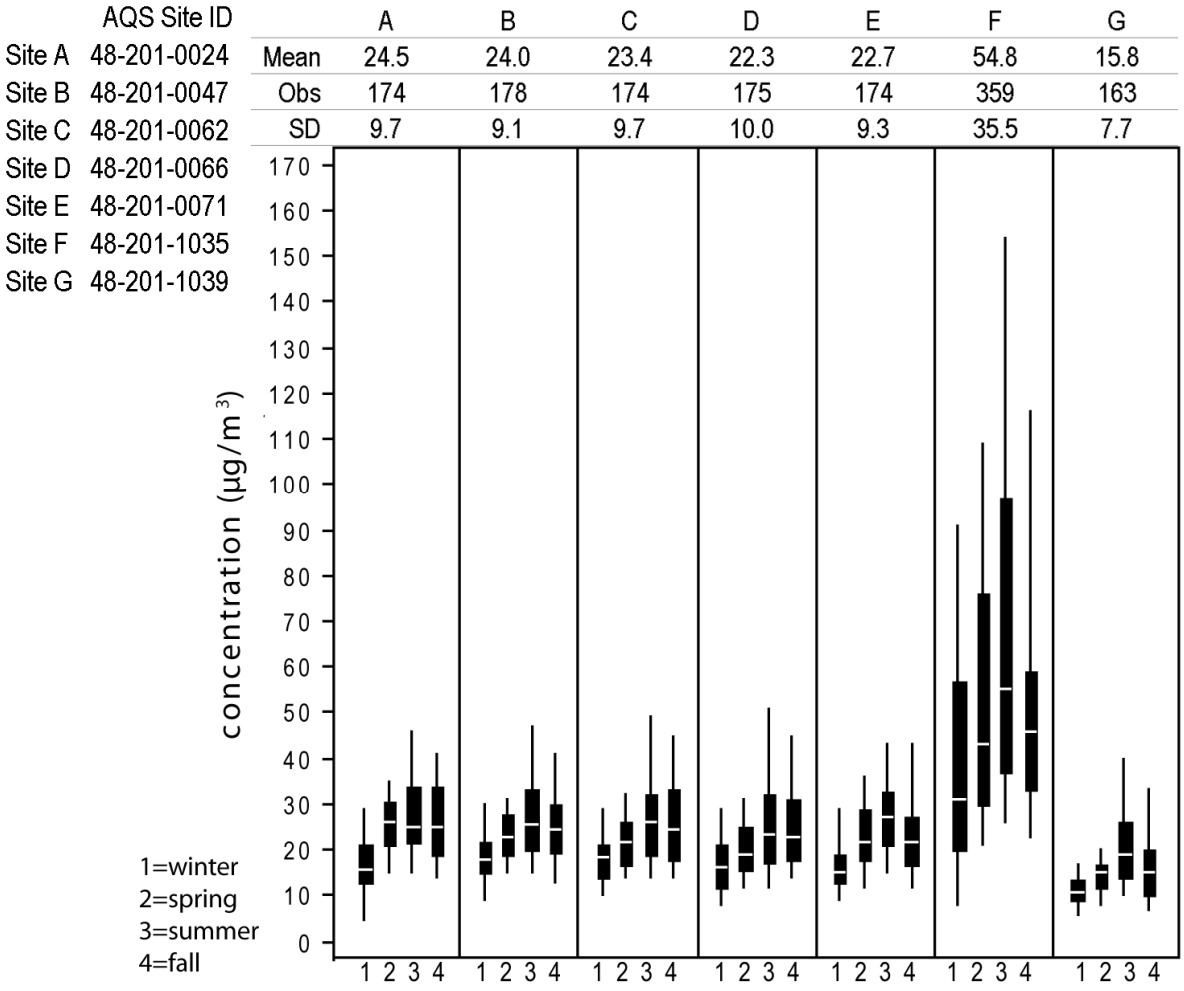


Figure A-101. Box plots illustrating the seasonal distribution of 24-h avg PM_{10} concentrations for Houston, TX.

Table A-41. 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
	LEGEND				(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

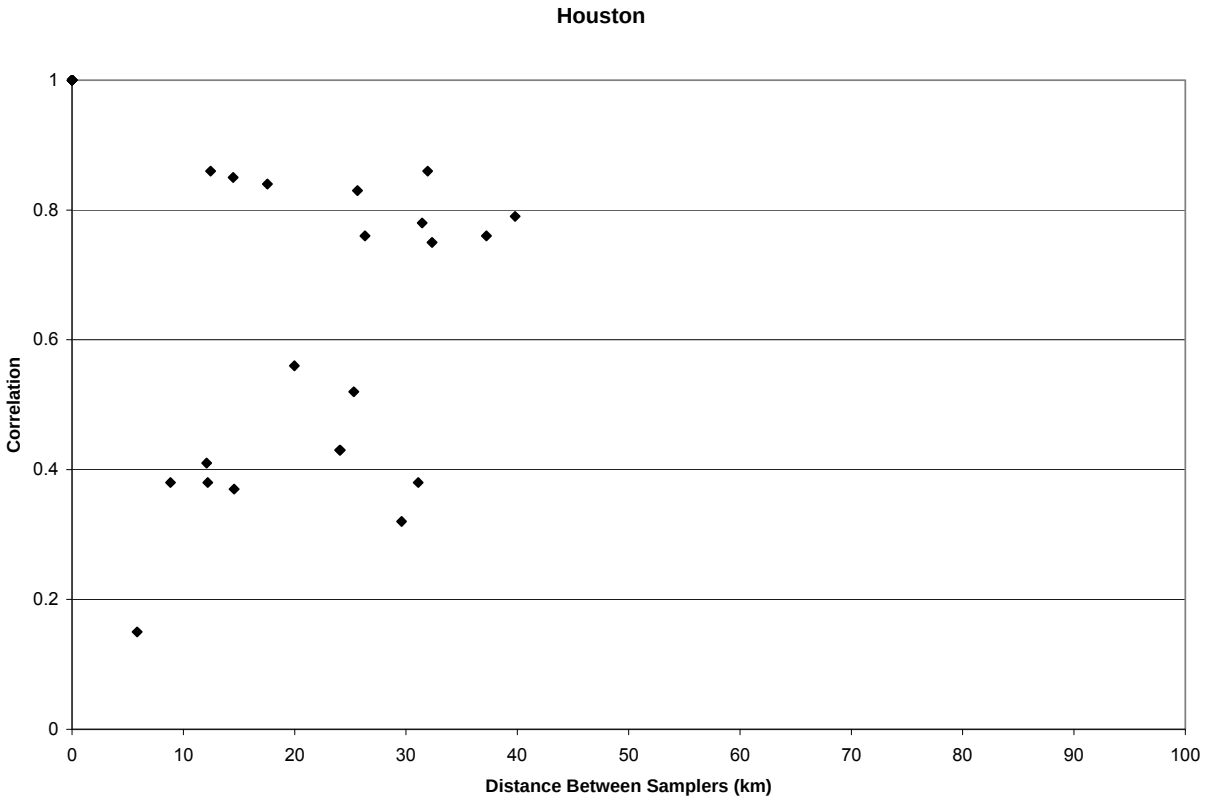


Figure A-102. PM_{10} inter-sampler correlations as a function of distance between monitors for Houston, TX.

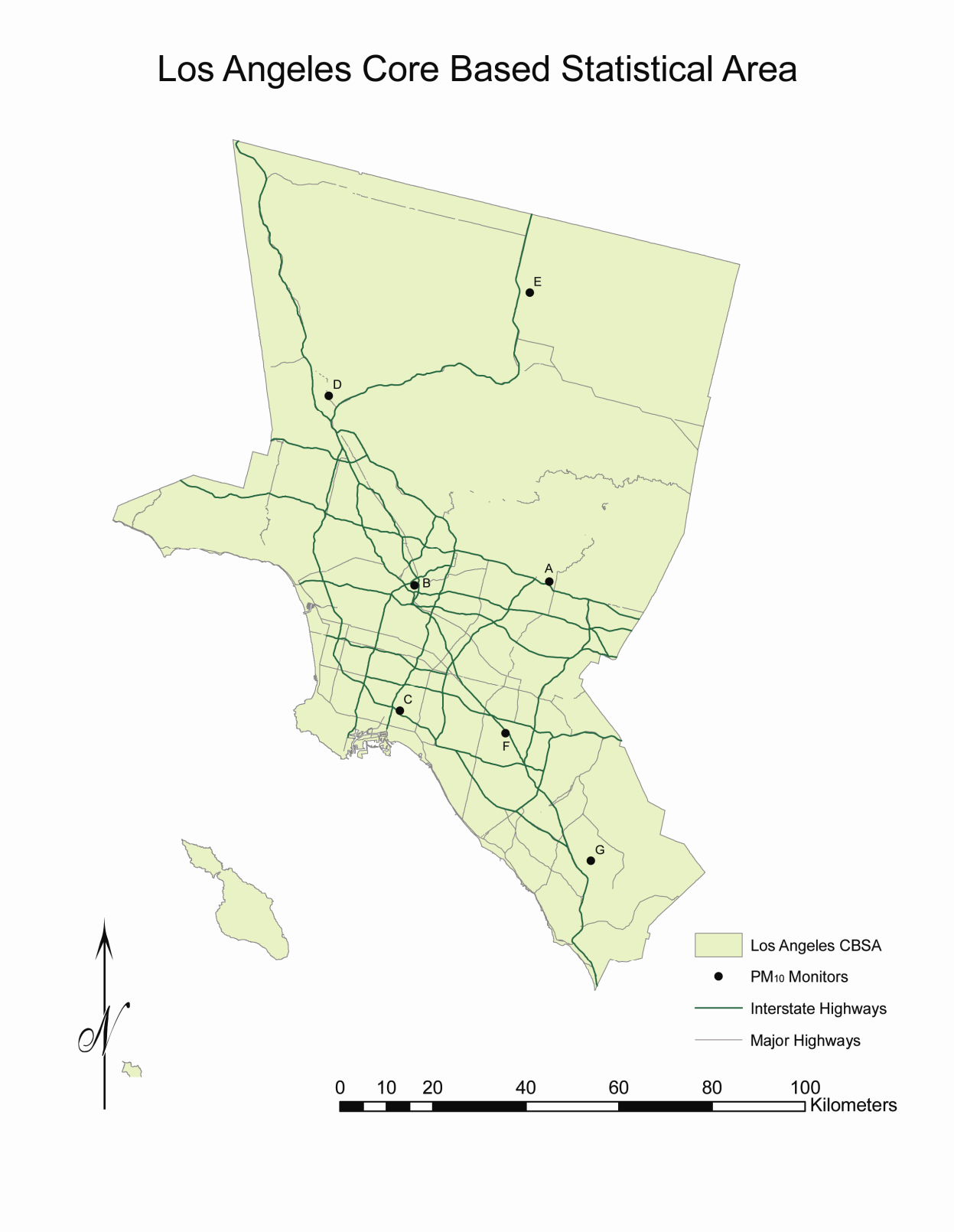


Figure A-103. PM_{10} monitor distribution and major highways, Los Angeles, CA.

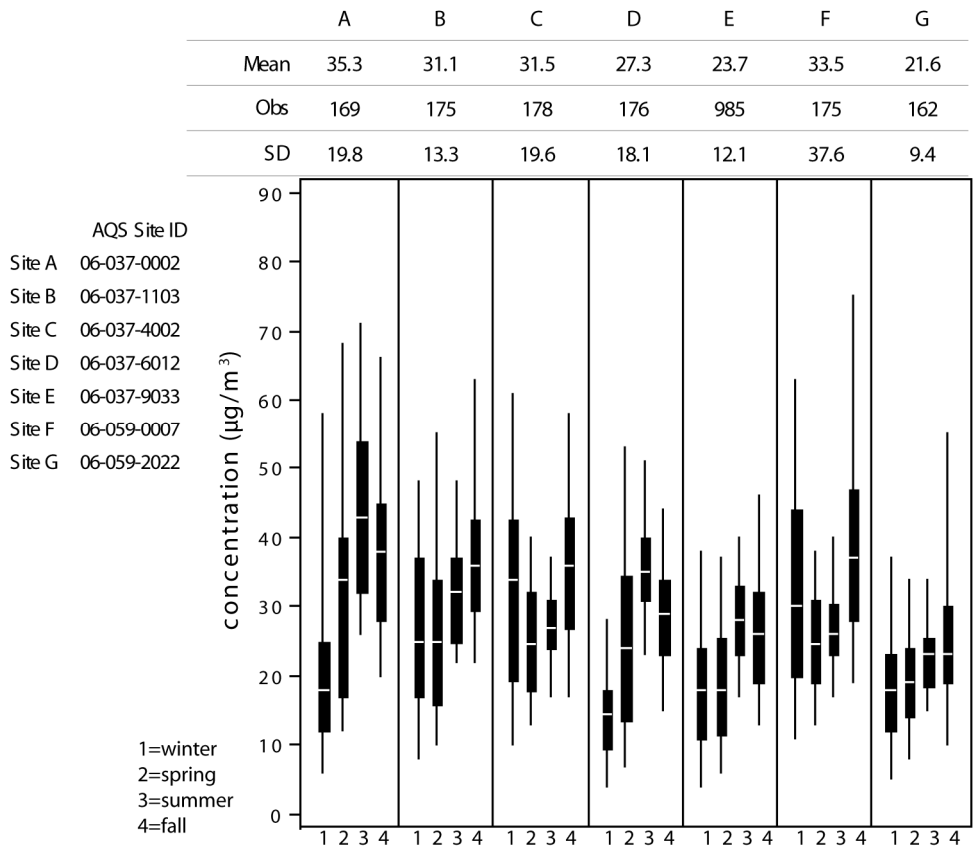


Figure A-104. Box plots illustrating the seasonal distribution of 24-h avg PM_{10} concentrations for Los Angeles, CA.

Table A-42. Inter-sampler correlation statistics for each pair of PM_{10} monitors reporting to AQS for Los Angeles, CA.

Site	A	B	C	D	E	F	G
A	1.00	0.73	0.44	0.73	0.47	0.41	0.65
	(0.0, 0.00)	(17.0, 0.17)	(27.0, 0.24)	(24.0, 0.22)	(28.0, 0.26)	(29.0, 0.24)	(30.0, 0.28)
	169	153	154	157	169	155	143
B		1.00	0.61	0.57	0.52	0.42	0.73
		(0.0, 0.00)	(14.0, 0.14)	(21.0, 0.24)	(23.0, 0.23)	(15.0, 0.16)	(20.0, 0.23)
		175	159	159	173	162	149
C			1.00	0.65	0.43	0.93	0.73
	LEGEND		(0.0, 0.00)	(27.0, 0.28)	(22.0, 0.24)	(11.0, 0.11)	(21.0, 0.22)
	Pearson R		178	158	176	159	148
D	**(P90,tCOD)**			1.00	0.70	0.65	0.57
				(0.0, 0.00)	(16.0, 0.20)	(26.0, 0.28)	(19.5, 0.24)
	n			176	175	161	150
E					1.00	0.29	0.38
					(0.0, 0.00)	(26.0, 0.25)	(20.0, 0.24)
					985	173	159
F						1.00	0.65
						(0.0, 0.00)	(21.5, 0.22)
						175	150
G							1.00
							(0.0, 0.00)
							162

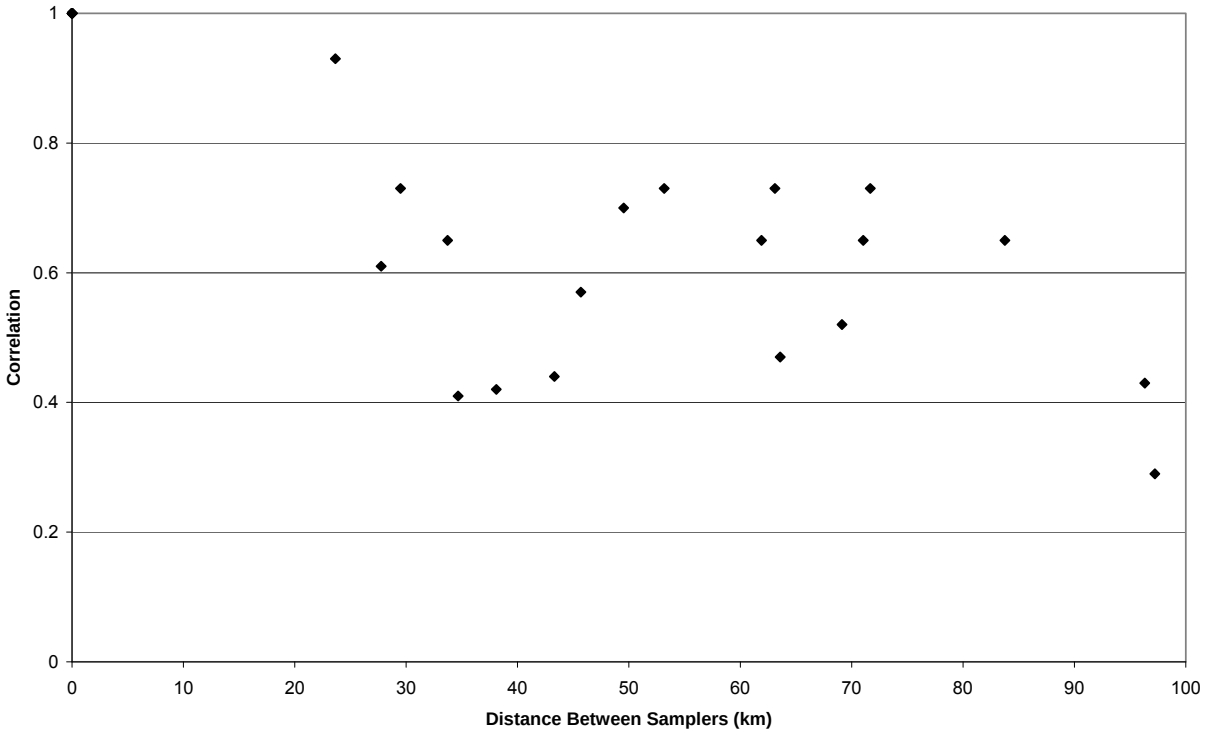


Figure A-105. PM_{10} inter-sampler correlations as a function of distance between monitors for Los Angeles, CA.

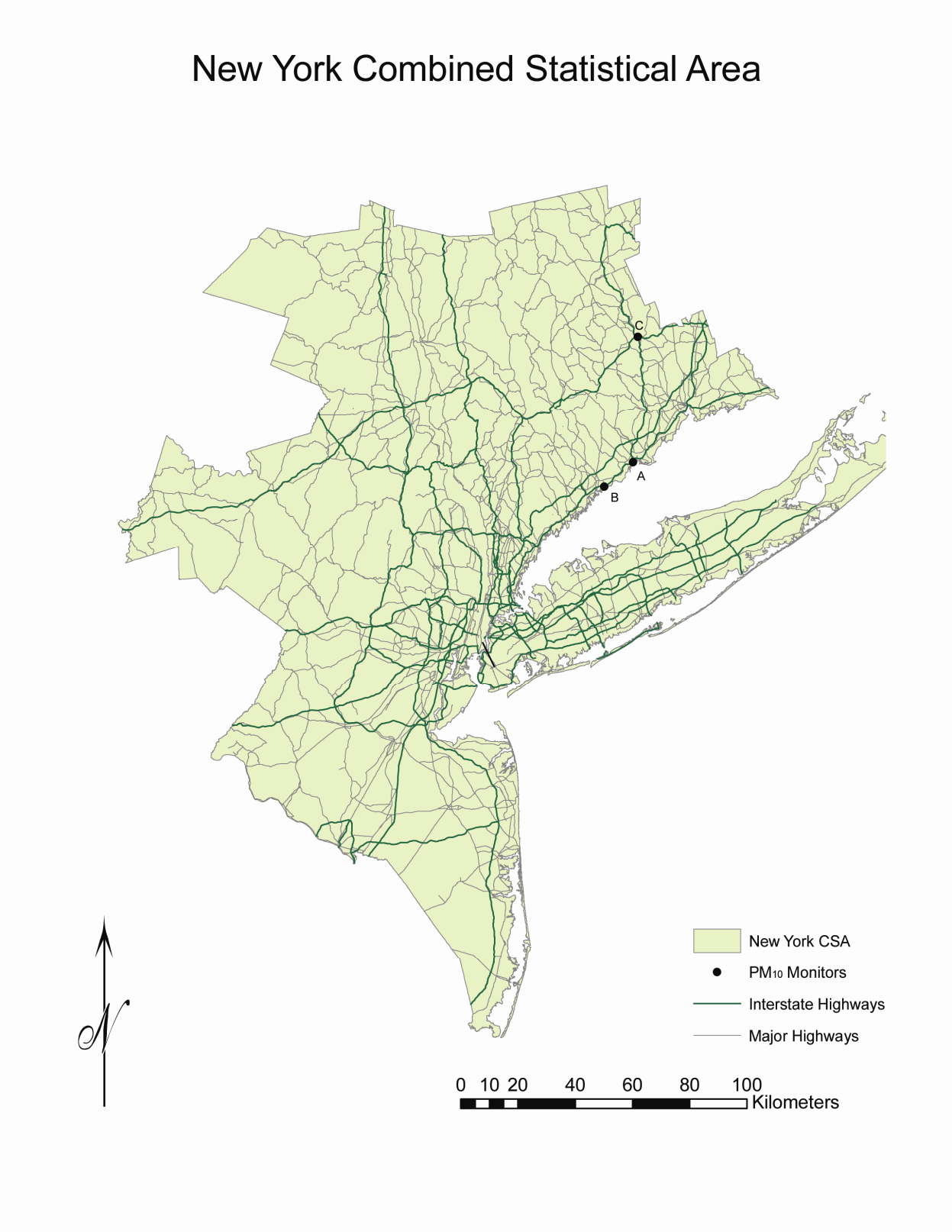


Figure A-106. PM_{10} monitor distribution and major highways, New York, NY.

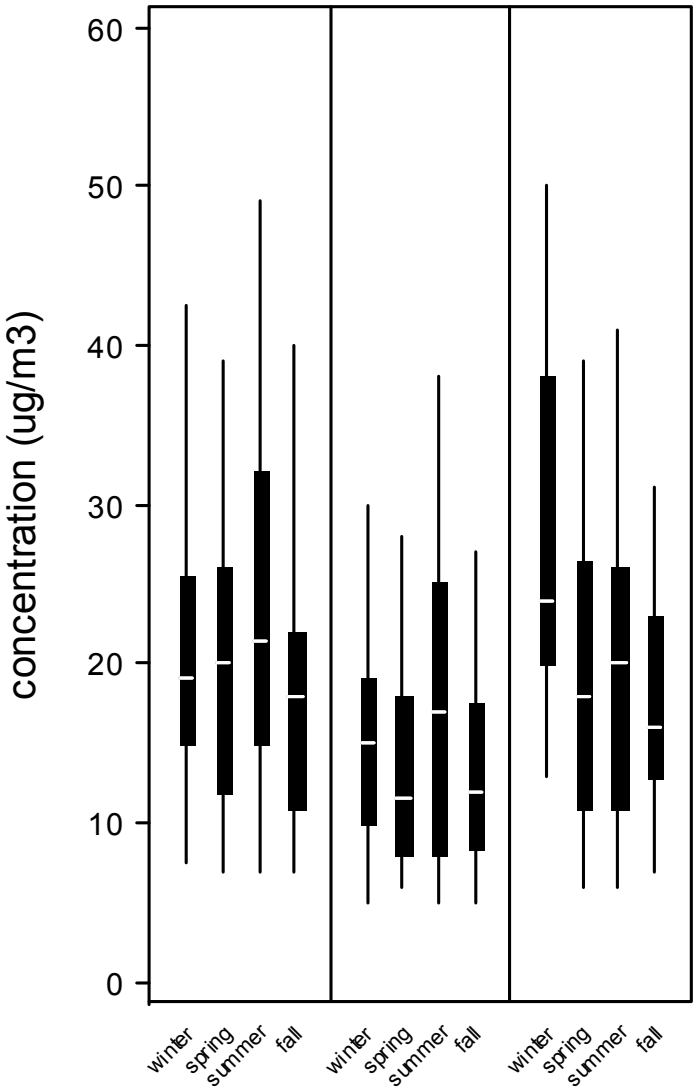


Figure A-107. Box plots illustrating the seasonal distribution of 24-h avg PM_{10} concentrations for New York, NY.

Table A-43. Inter-sampler correlation statistics for each pair of PM_{10} monitors reporting to AQS for New York, 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
	LEGEND	(0.0, 0.00)	(18.0, 0.25)
	R	169	166
C	(P90, COD)		1.00
	N		(0.0, 0.00)
			178

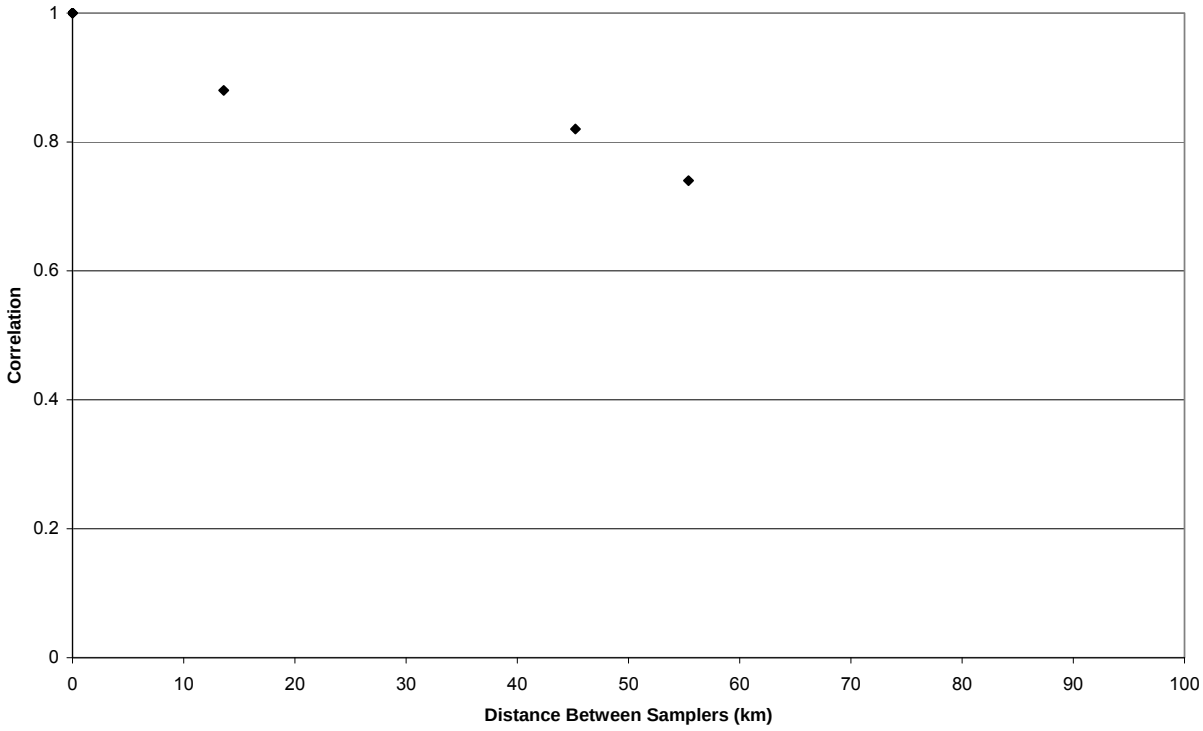


Figure A-108. PM_{10} inter-sampler correlations as a function of distance between monitors for New York, NY.

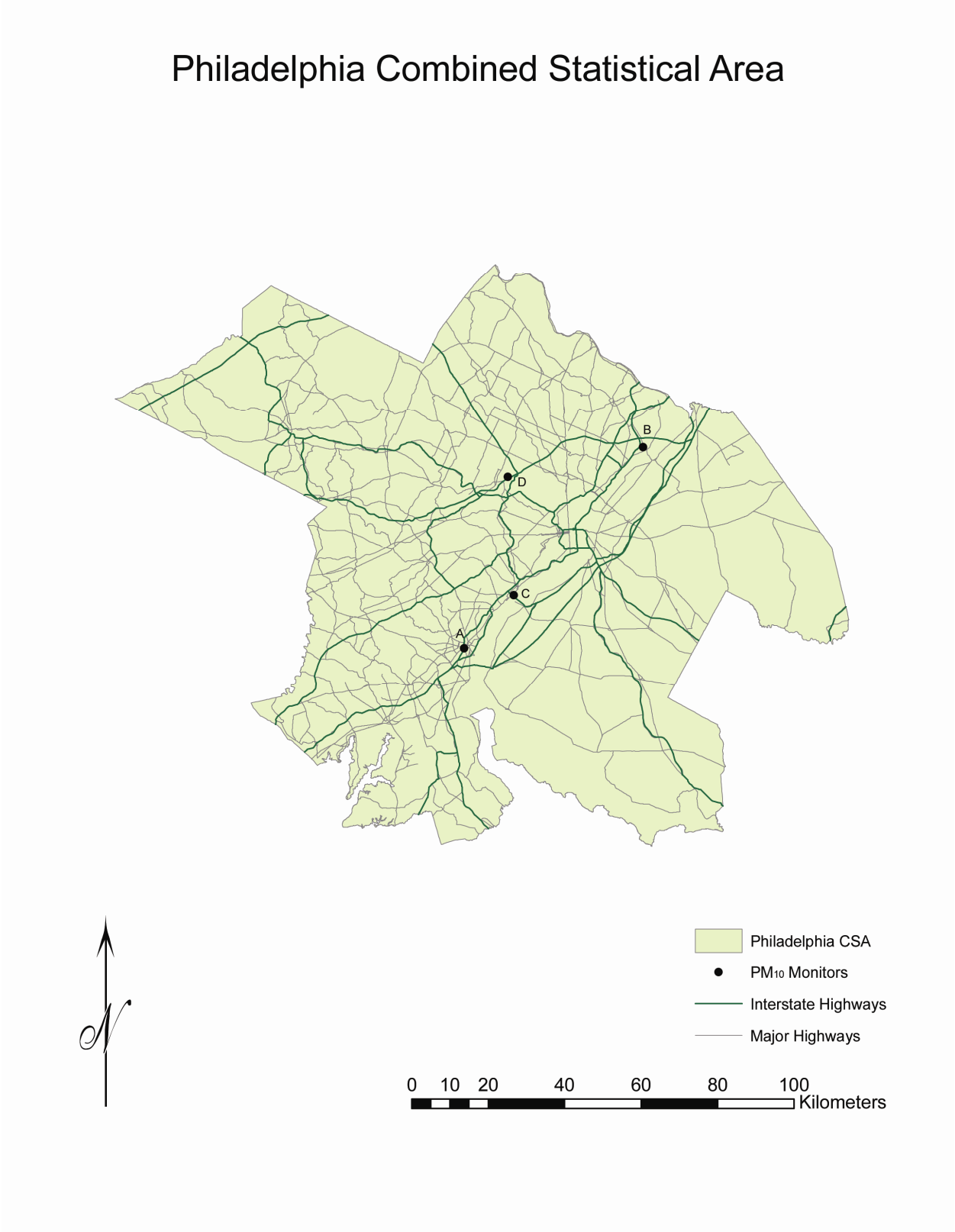


Figure A-109. PM_{10} monitor distribution and major highways, Philadelphia, PA.

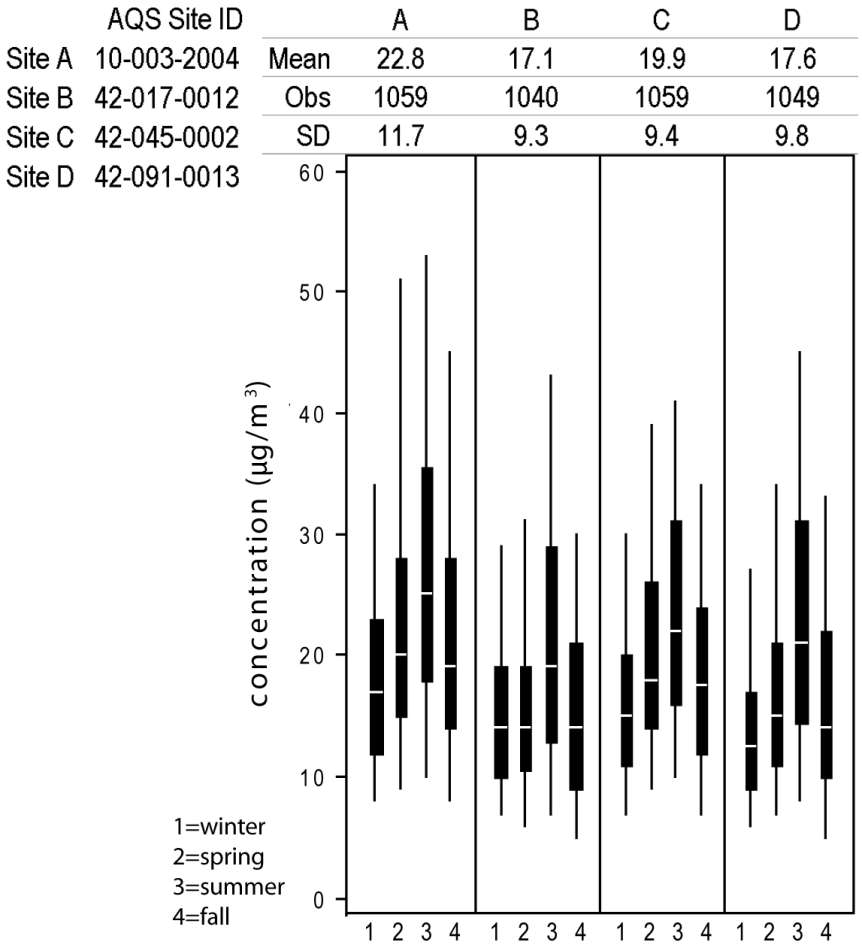


Figure A-110. Box plots illustrating the seasonal distribution of 24-h avg PM_{10} concentrations for Philadelphia, PA.

Table A-44. 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
	LEGEND		(0.0, 0.00)	(11.0, 0.19)
	R		1059	1014
D	(P90, COD)			1.00
	N			(0.0, 0.00)
				1049

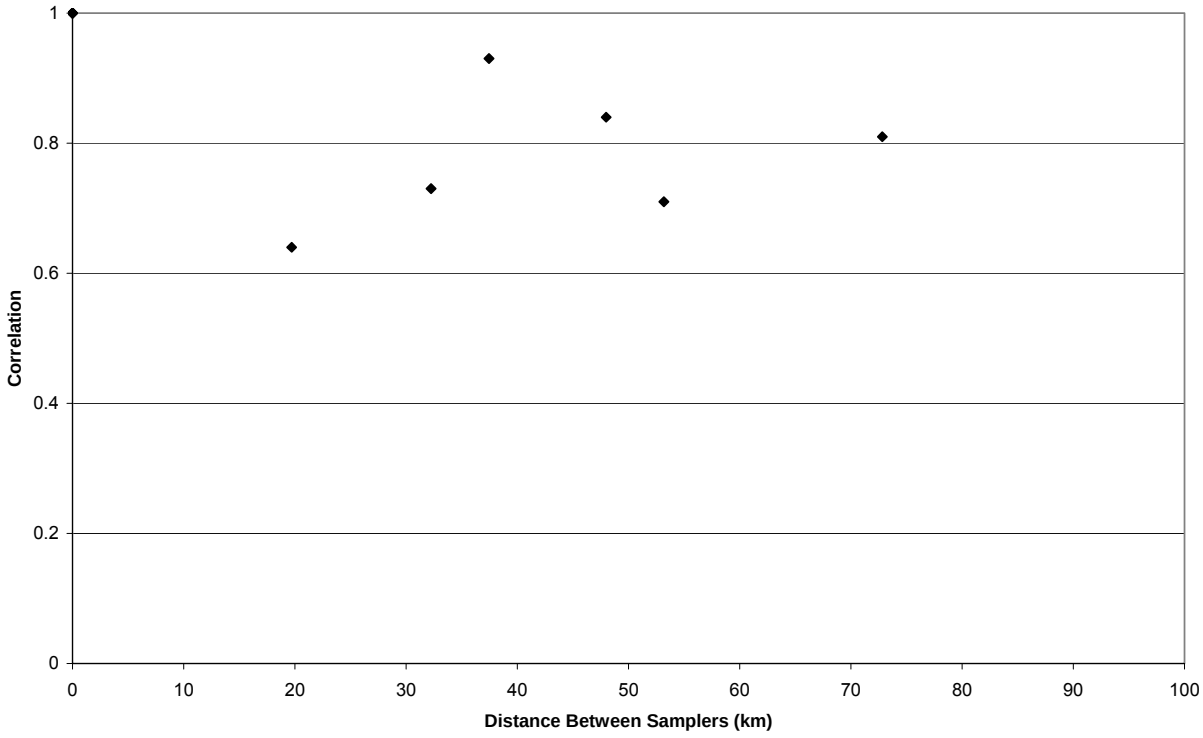


Figure A-111. PM_{10} inter-sampler correlations as a function of distance between monitors for Philadelphia, PA.

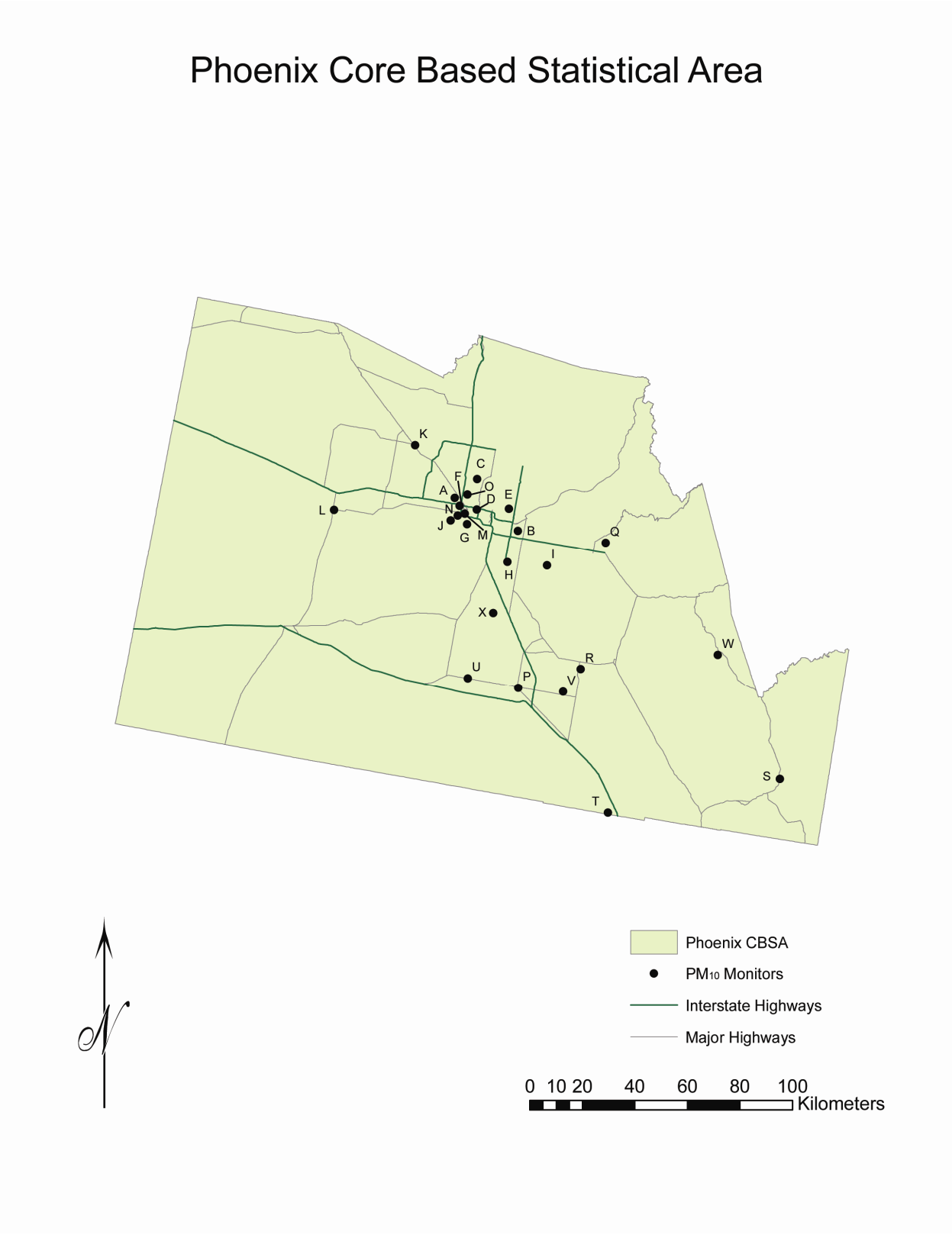
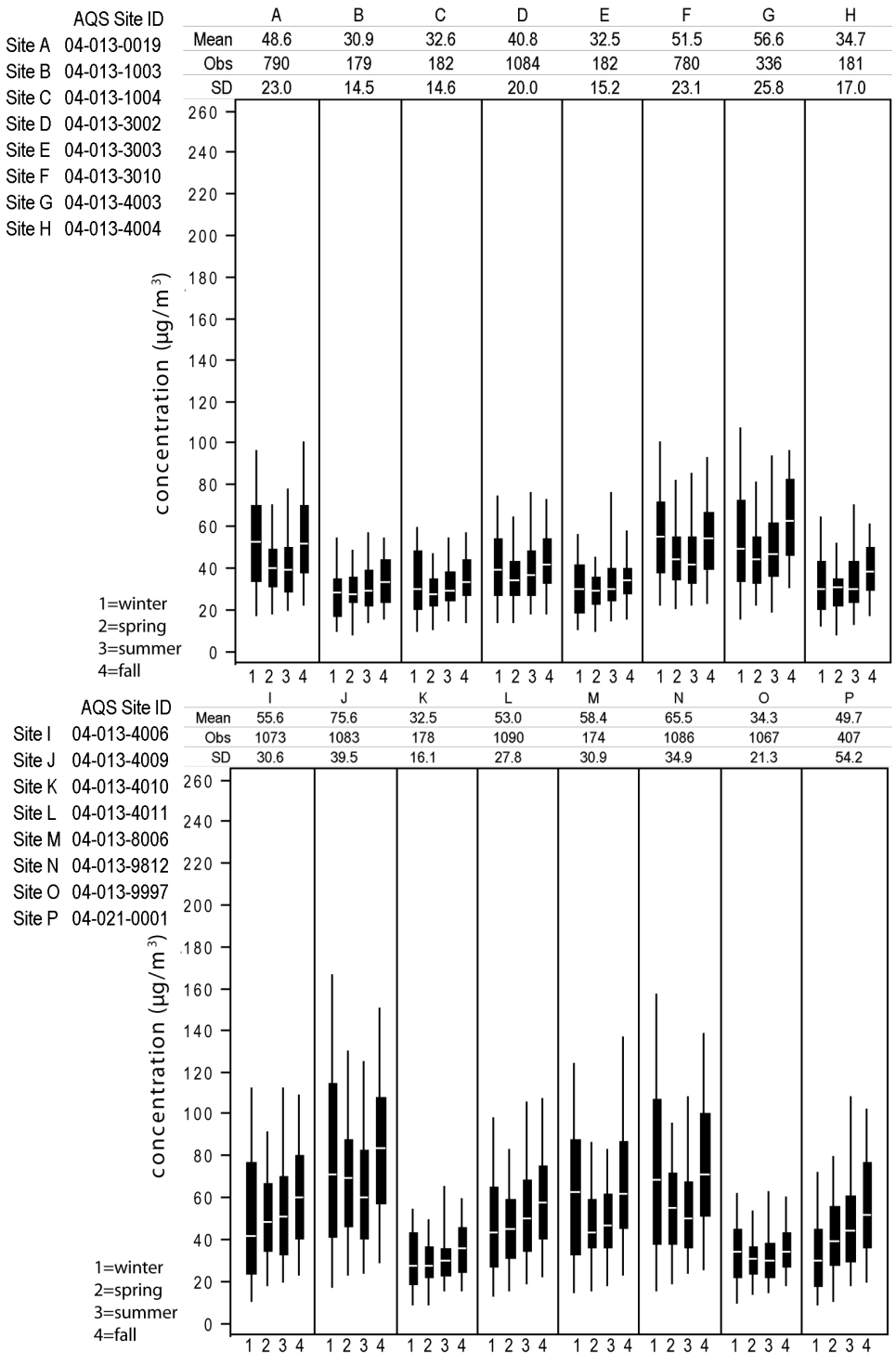


Figure A-112. PM_{10} monitor distribution and major highways, Phoenix, AZ.


AQS Site ID
Site A 04-013-0019
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

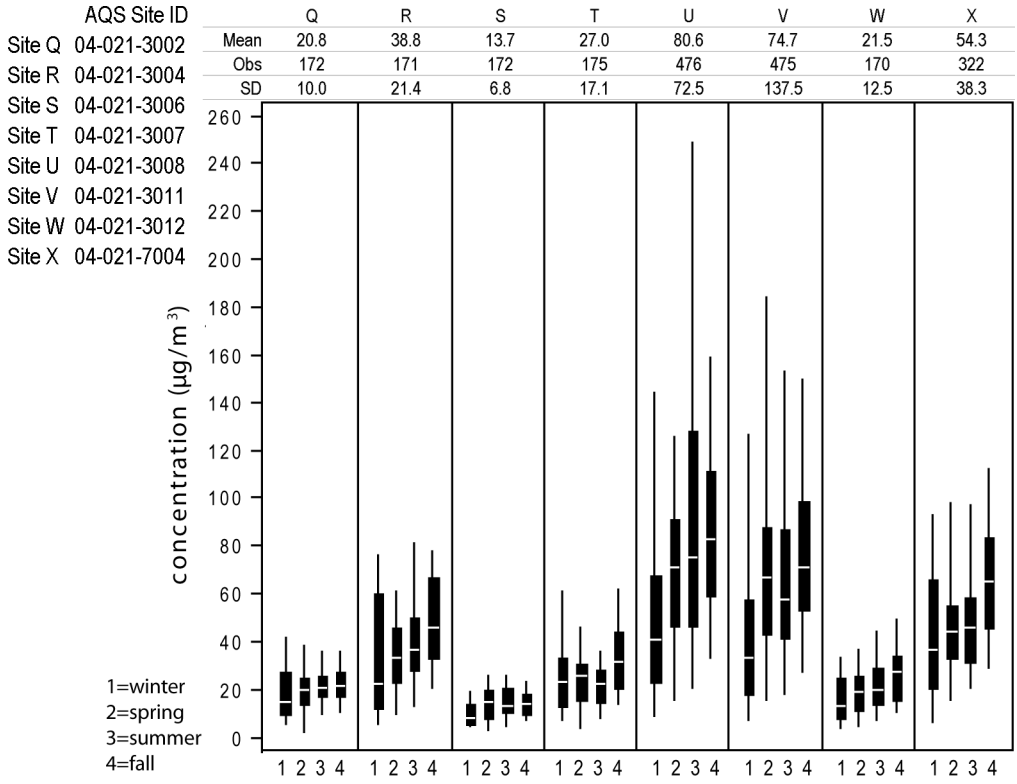


Figure A-113. Box plots illustrating the seasonal distribution of 24-h avg PM_{10} concentrations for Phoenix, AZ.

Table A-45. 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)
			LEGEND					181	177	178	177	180	167
I			**R (P90, COD) N**						1.00	0.79	0.76	0.69	0.68
									(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

	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
			LEGEND				(0.0, 0.00)	(94.0, 0.47)	(71.0, 0.39)	(18.0, 0.24)	(51.5, 0.37)
			R				175	169	174	165	150
U			**(P90, COD)**					1.00	0.54	0.52	0.71
			N					(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
X											1.00
											(0.0, 0.00)
											322

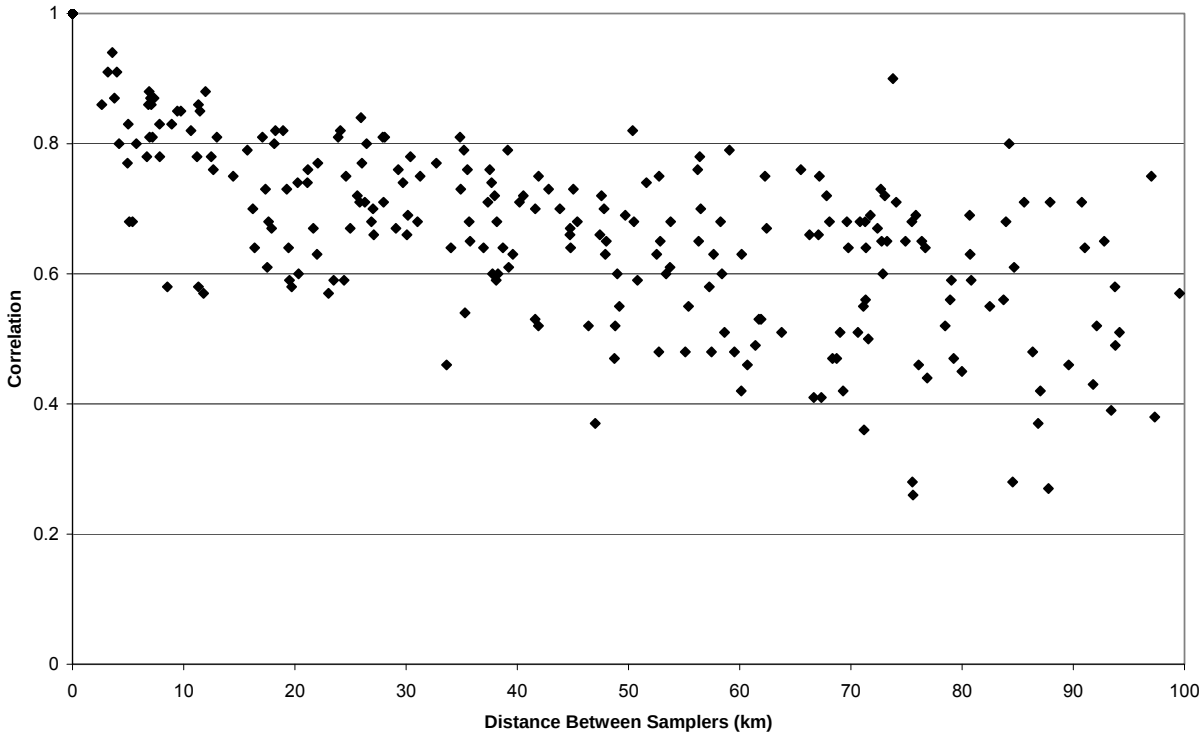


Figure A-114. PM_{10} inter-sampler correlations as a function of distance between monitors for Phoenix, AZ.

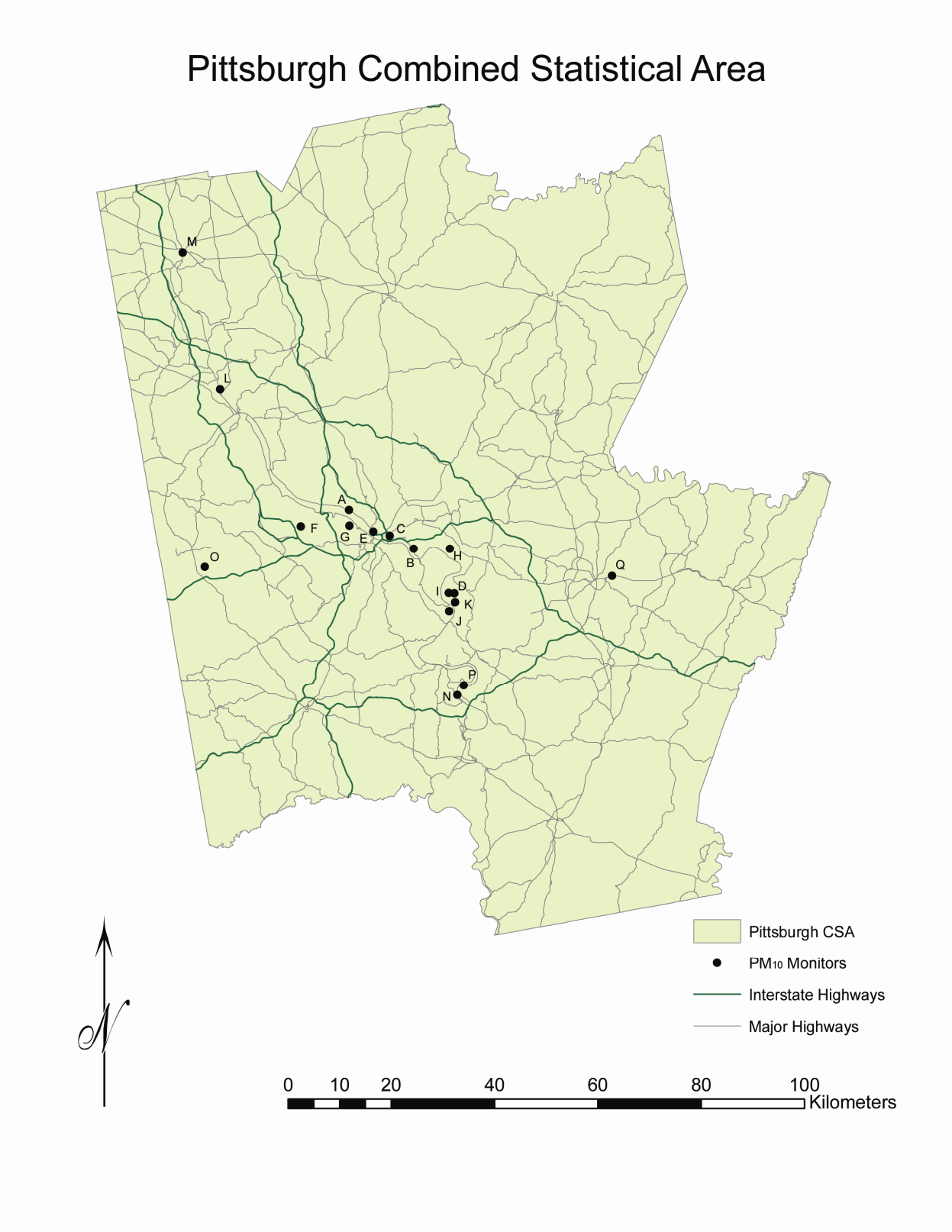


Figure A-115. PM_{10} monitor distribution and major highways, Pittsburgh, PA.

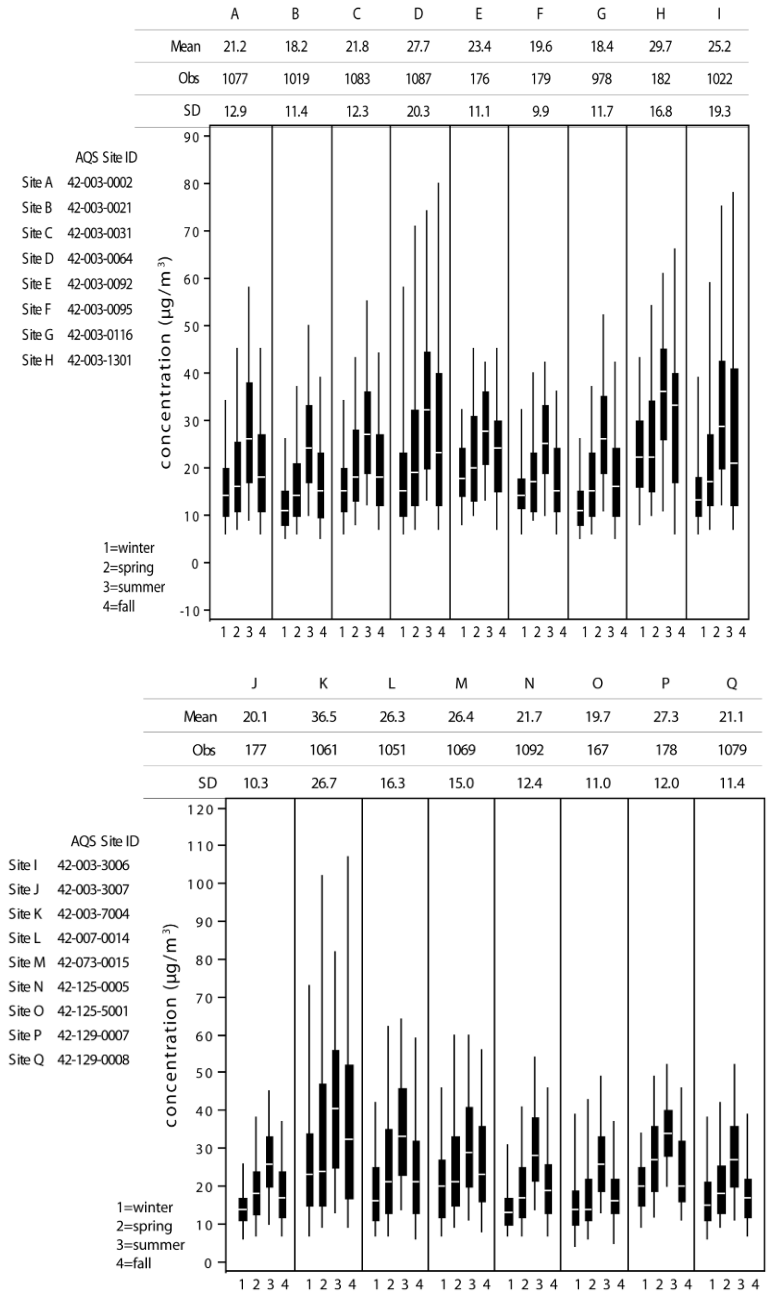


Figure A-116. Box plots illustrating the seasonal distribution of 24-h avg PM_{10} concentrations for Pittsburgh, PA.

Table A-46. Inter-sampler correlation statistics for each pair of PM_{10} monitors reporting to AQS for Pittsburgh, PA.

Site	A	B	C	D	E	F	G	H	I
A	1.00	0.93	0.93	0.80	0.92	0.89	0.93	0.79	0.86
	(0.0, 0.00)	(9.0, 0.15)	(8.0, 0.14)	(23.0, 0.21)	(8.0, 0.12)	(14.0, 0.18)	(8.0, 0.14)	(16.0, 0.17)	(18.0, 0.18)
	1077	1002	1065	1070	175	178	960	181	1005
B		1.00	0.96	0.80	0.91	0.92	0.97	0.81	0.89
		(0.0, 0.00)	(8.0, 0.15)	(29.0, 0.24)	(11.0, 0.20)	(6.0, 0.16)	(5.0, 0.10)	(25.0, 0.29)	(22.0, 0.20)
		1019	1007	1012	163	166	911	169	954
C			1.00	0.81	0.94	0.93	0.94	0.77	0.87
			(0.0, 0.00)	(23.0, 0.20)	(6.0, 0.11)	(7.0, 0.12)	(8.0, 0.13)	(21.0, 0.22)	(19.0, 0.17)
			1083	1075	173	176	966	179	1010
D				1.00	0.72	0.66	0.76	0.83	0.88
				(0.0, 0.00)	(21.0, 0.20)	(26.0, 0.24)	(27.0, 0.24)	(14.0, 0.18)	(16.0, 0.14)
				1087	176	179	970	182	1014
E					1.00	0.90	0.90	0.78	0.77
					(0.0, 0.00)	(10.0, 0.14)	(10.0, 0.17)	(20.0, 0.20)	(20.0, 0.19)
					176	173	154	175	166
F						1.00	0.94	0.70	0.74
						(0.0, 0.00)	(7.0, 0.12)	(25.0, 0.27)	(25.0, 0.22)
						179	157	178	168
G							1.00	0.70	0.87
							(0.0, 0.00)	(22.0, 0.28)	(20.0, 0.19)
							978	160	910
H								1.00	0.76
								(0.0, 0.00)	(17.0, 0.20)
								182	171
I									1.00
									(0.0, 0.00)
									1022

LEGEND
Pearson R
(P90, COD)
n

	J	K	L	M	N	O	P	Q
A	0.84	0.76	0.88	0.85	0.86	0.77	0.78	0.86
	(14.0, 0.20)	(40.0, 0.30)	(15.0, 0.18)	(16.0, 0.19)	(11.0, 0.16)	(16.0, 0.22)	(15.0, 0.19)	(11.0, 0.15)
	176	1044	1033	1052	1074	166	177	1061
B	0.93	0.76	0.88	0.81	0.91	0.76	0.83	0.88
	(7.0, 0.16)	(43.0, 0.36)	(19.0, 0.23)	(20.0, 0.26)	(10.0, 0.16)	(12.0, 0.19)	(18.0, 0.28)	(10.0, 0.18)
	164	986	982	994	1016	157	165	1003
C	0.90	0.75	0.88	0.83	0.89	0.78	0.88	0.90
	(8.0, 0.13)	(39.0, 0.30)	(14.0, 0.17)	(15.0, 0.19)	(9.0, 0.12)	(12.0, 0.18)	(13.0, 0.19)	(9.0, 0.12)
	174	1049	1039	1057	1080	164	175	1067
D	0.73	0.84	0.80	0.78	0.76	0.57	0.64	0.74
	(24.0, 0.22)	(24.0, 0.22)	(20.0, 0.18)	(20.0, 0.20)	(25.0, 0.20)	(28.0, 0.26)	(20.0, 0.25)	(26.0, 0.21)
	177	1055	1043	1061	1084	167	178	1071
E	0.86	0.65	0.83	0.80	0.84	0.77	0.84	0.85
	(10.0, 0.16)	(36.0, 0.29)	(16.0, 0.16)	(14.0, 0.17)	(12.0, 0.14)	(14.0, 0.19)	(13.0, 0.16)	(11.0, 0.15)
	171	169	169	172	176	161	172	174
F	0.90	0.57	0.82	0.75	0.86	0.83	0.84	0.86
	(7.0, 0.12)	(41.0, 0.34)	(20.0, 0.20)	(19.0, 0.22)	(11.0, 0.14)	(9.0, 0.15)	(16.0, 0.22)	(9.0, 0.14)
	174	172	172	175	179	164	175	177
G	0.92	0.73	0.87	0.78	0.89	0.81	0.84	0.86
	(7.0, 0.13)	(45.0, 0.35)	(18.0, 0.21)	(19.0, 0.24)	(9.0, 0.15)	(11.0, 0.17)	(17.0, 0.26)	(10.0, 0.16)
	156	955	938	952	975	146	157	967
H	0.74	0.68	0.77	0.78	0.74	0.60	0.65	0.76
	(23.0, 0.26)	(26.0, 0.22)	(15.0, 0.18)	(17.0, 0.18)	(21.0, 0.22)	(27.0, 0.29)	(19.0, 0.22)	(21.5, 0.24)
	176	175	175	178	182	167	177	180
I	0.79	0.83	0.82	0.78	0.81	0.66	0.69	0.78
	(22.0, 0.20)	(30.0, 0.25)	(16.0, 0.17)	(18.0, 0.20)	(20.0, 0.17)	(26.0, 0.24)	(21.0, 0.25)	(22.0, 0.19)
	166	992	978	998	1019	158	167	1009
J	1.00	0.66	0.79	0.72	0.88	0.78	0.86	0.86
	(0.0, 0.00)	(44.5, 0.33)	(18.0, 0.20)	(18.0, 0.22)	(8.0, 0.13)	(11.0, 0.17)	(16.0, 0.21)	(8.0, 0.15)
	177	170	170	173	177	163	173	175
K		1.00	0.74	0.75	0.70	0.47	0.58	0.68
		(0.0, 0.00)	(31.0, 0.26)	(33.0, 0.24)	(40.0, 0.30)	(44.0, 0.36)	(34.0, 0.30)	(43.0, 0.30)
		1061	1017	1035	1058	160	171	1048
L			1.00	0.87	0.85	0.70	0.74	0.80
			(0.0, 0.00)	(13.0, 0.16)	(16.0, 0.17)	(22.0, 0.24)	(17.0, 0.21)	(18.0, 0.19)
			1051	1025	1048	160	171	1035
M				1.00	0.74	0.64	0.67	0.77
				(0.0, 0.00)	(18.0, 0.21)	(19.0, 0.26)	(17.0, 0.22)	(18.0, 0.19)
				1069	1067	163	174	1053
N					1.00	0.72	0.86	0.86
					(0.0, 0.00)	(13.0, 0.18)	(14.0, 0.20)	(10.0, 0.14)
					1092	167	178	1076
O						1.00	0.75	0.69
						(0.0, 0.00)	(18.0, 0.25)	(14.0, 0.19)
						167	163	165
P							1.00	0.84
							(0.0, 0.00)	(15.0, 0.21)
							178	176
Q								1.00
								(0.0, 0.00)
								1079

LEGEND
Pearson R
(P90, COD)
n

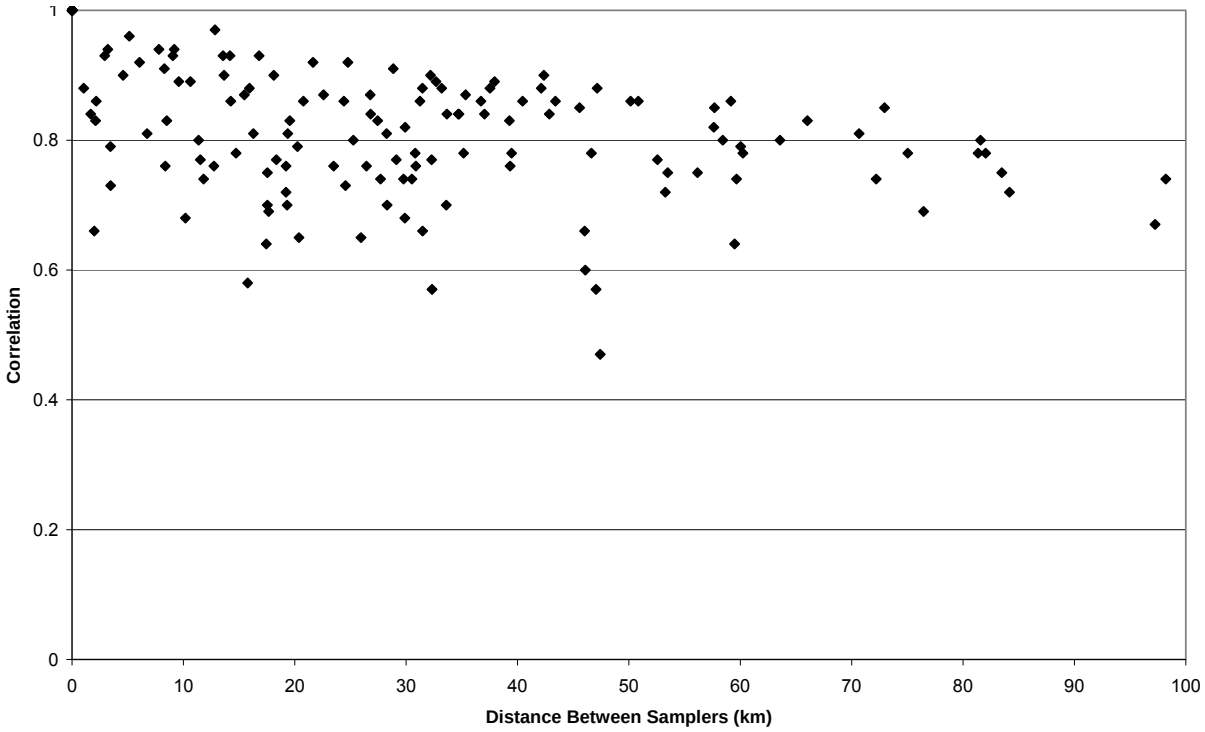


Figure A-117. PM_{10} inter-sampler correlations as a function of distance between monitors for Pittsburgh, PA.

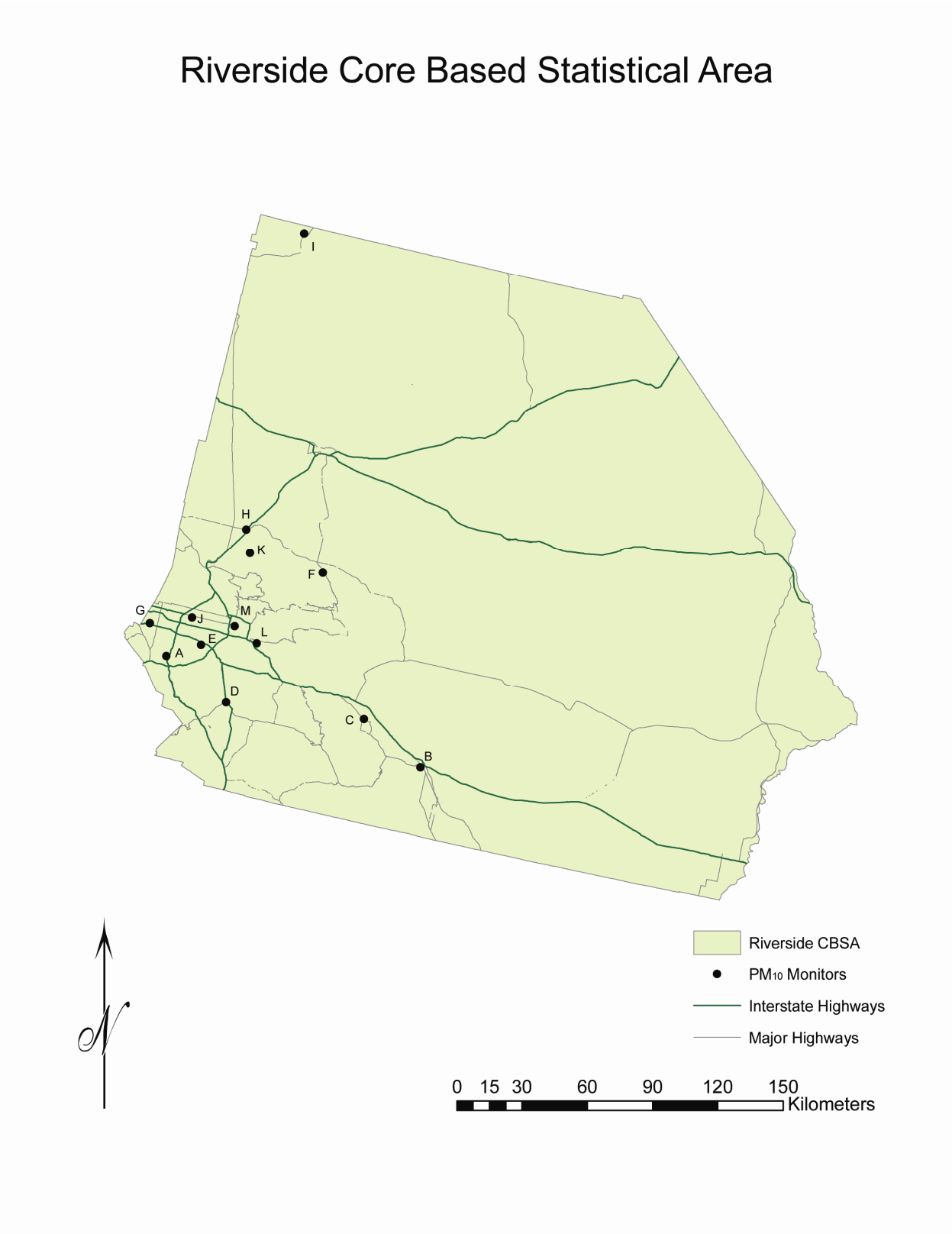


Figure A-118. PM_{10} monitor distribution and major highways, Riverside, CA.

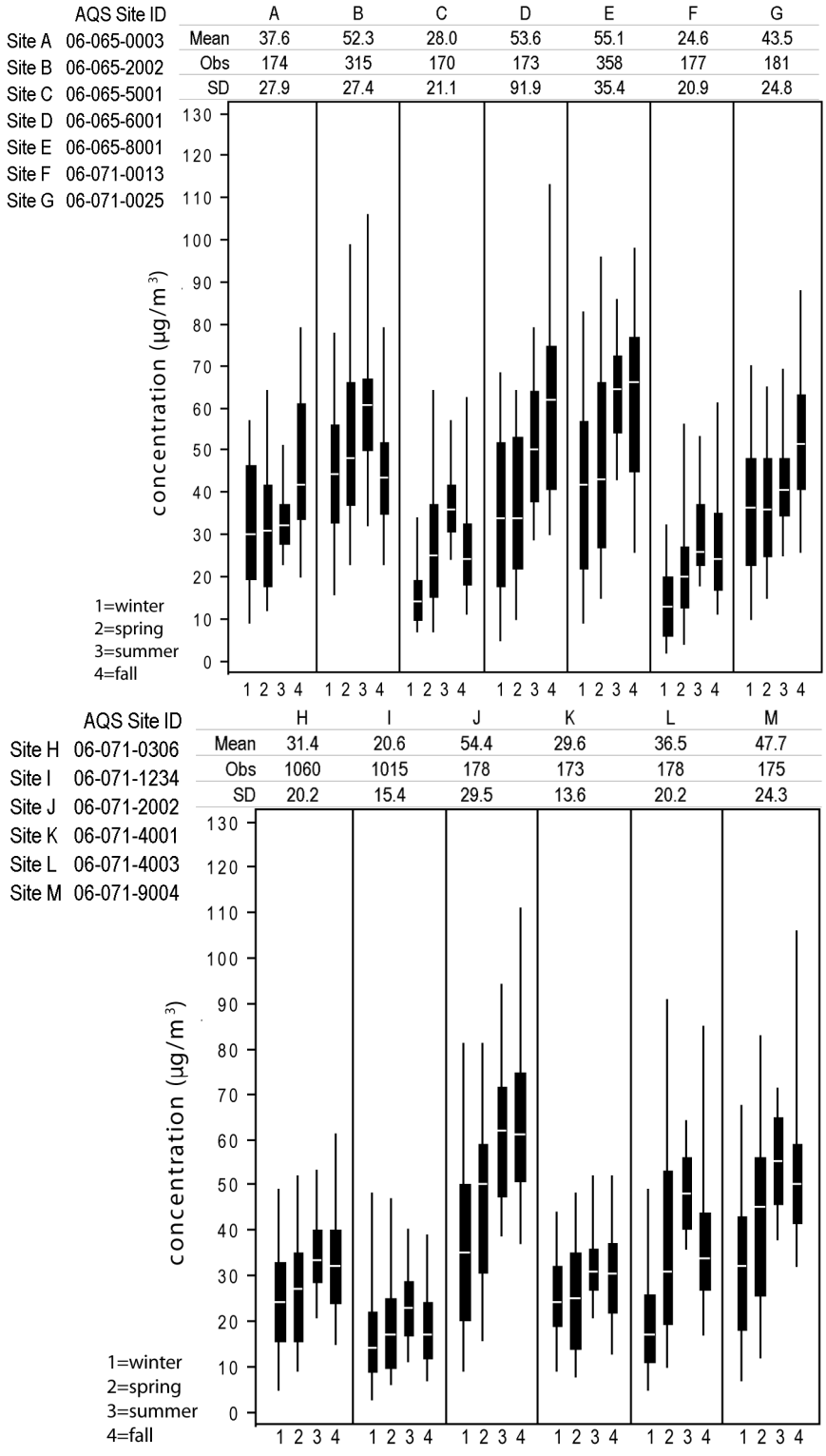


Figure A-119. Box plots illustrating the seasonal distribution of 24-h avg PM_{10} concentrations for Riverside, CA.

Table A-47. 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)
		LEGEND							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

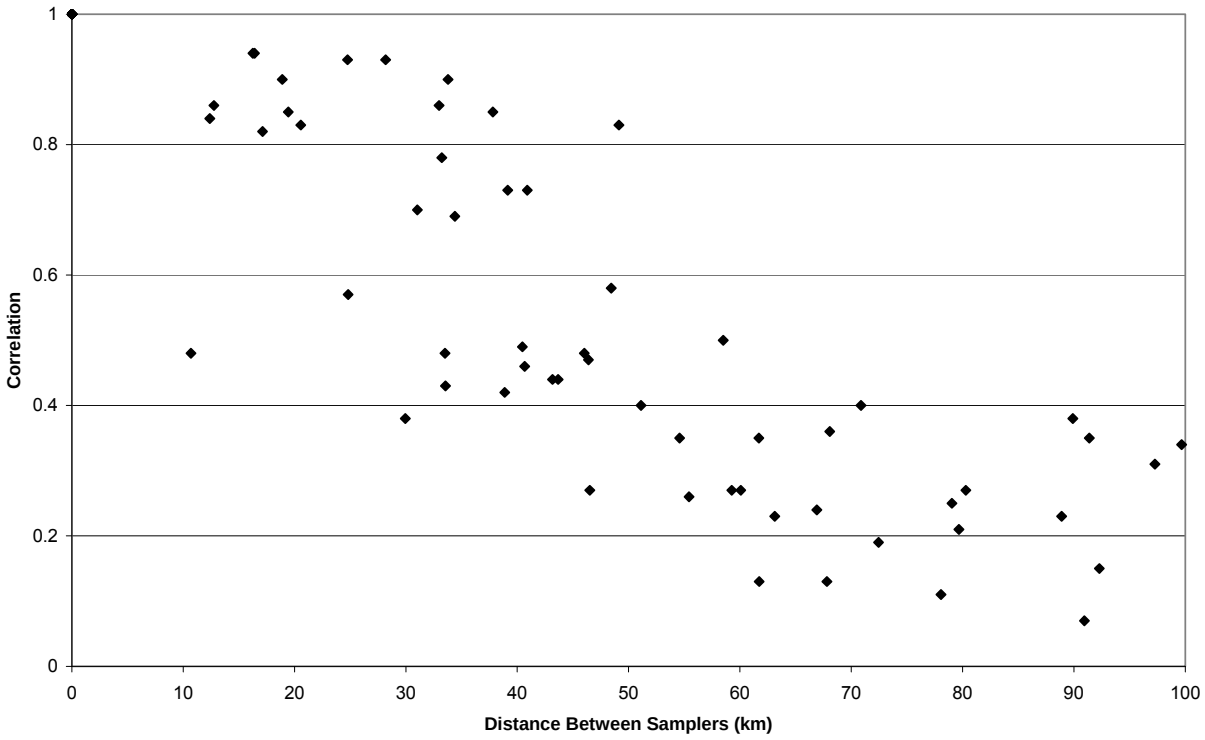


Figure A-120. PM_{10} inter-sampler correlations as a function of distance between monitors for Riverside, CA.

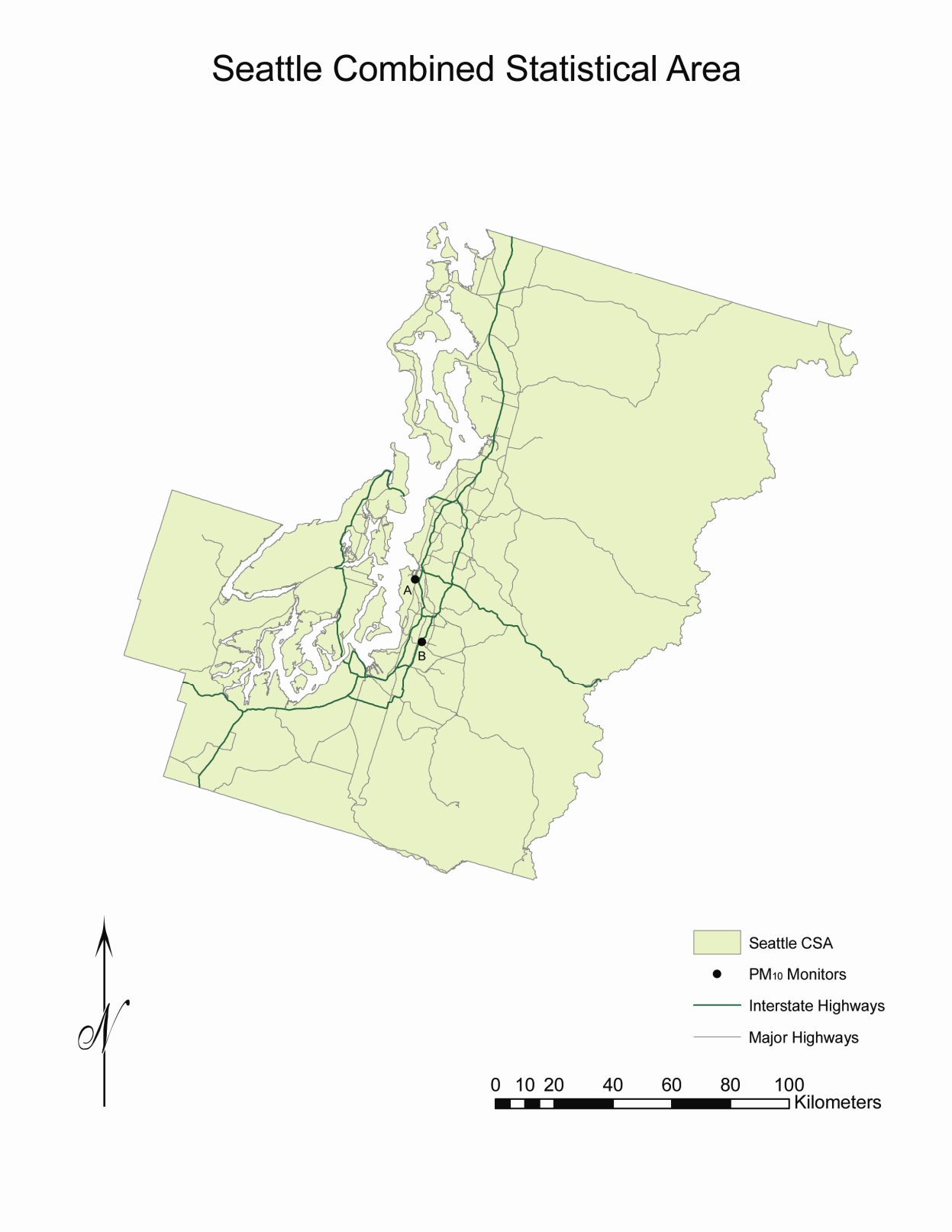


Figure A-121. PM_{10} monitor distribution and major highways, Seattle, WA.

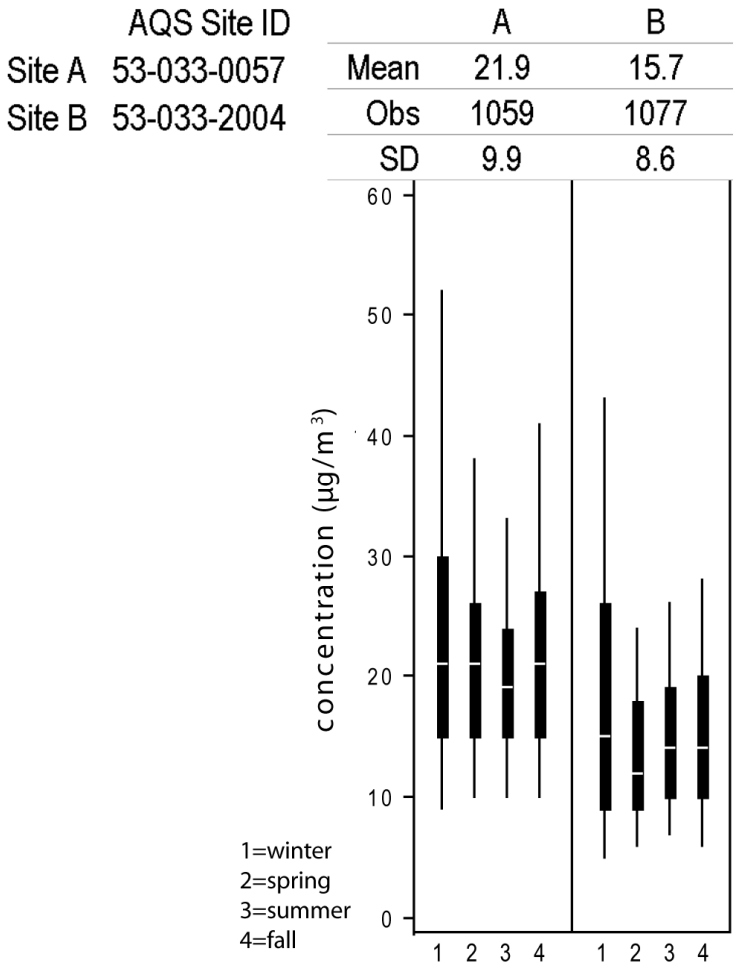


Figure A-122. Box plots illustrating the seasonal distribution of 24-h avg PM_{10} concentrations for Seattle, WA.

Table A-48. 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	**LEGEND**	1.00
	R	(0.0, 0.00)
	(P90, COD)	1077
	N	

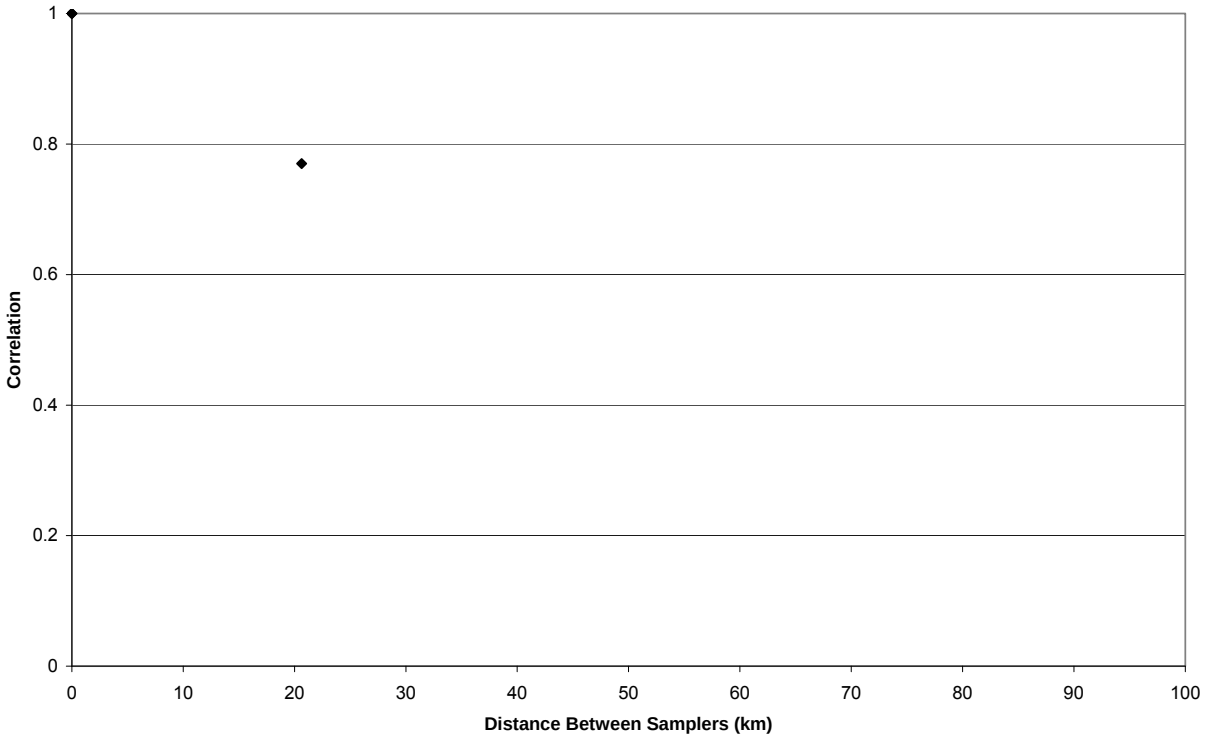


Figure A-123. PM_{10} inter-sampler correlations as a function of distance between monitors for Seattle, WA.

Figure A-124. PM_{10} monitor distribution and major highways, St. Louis, MO.

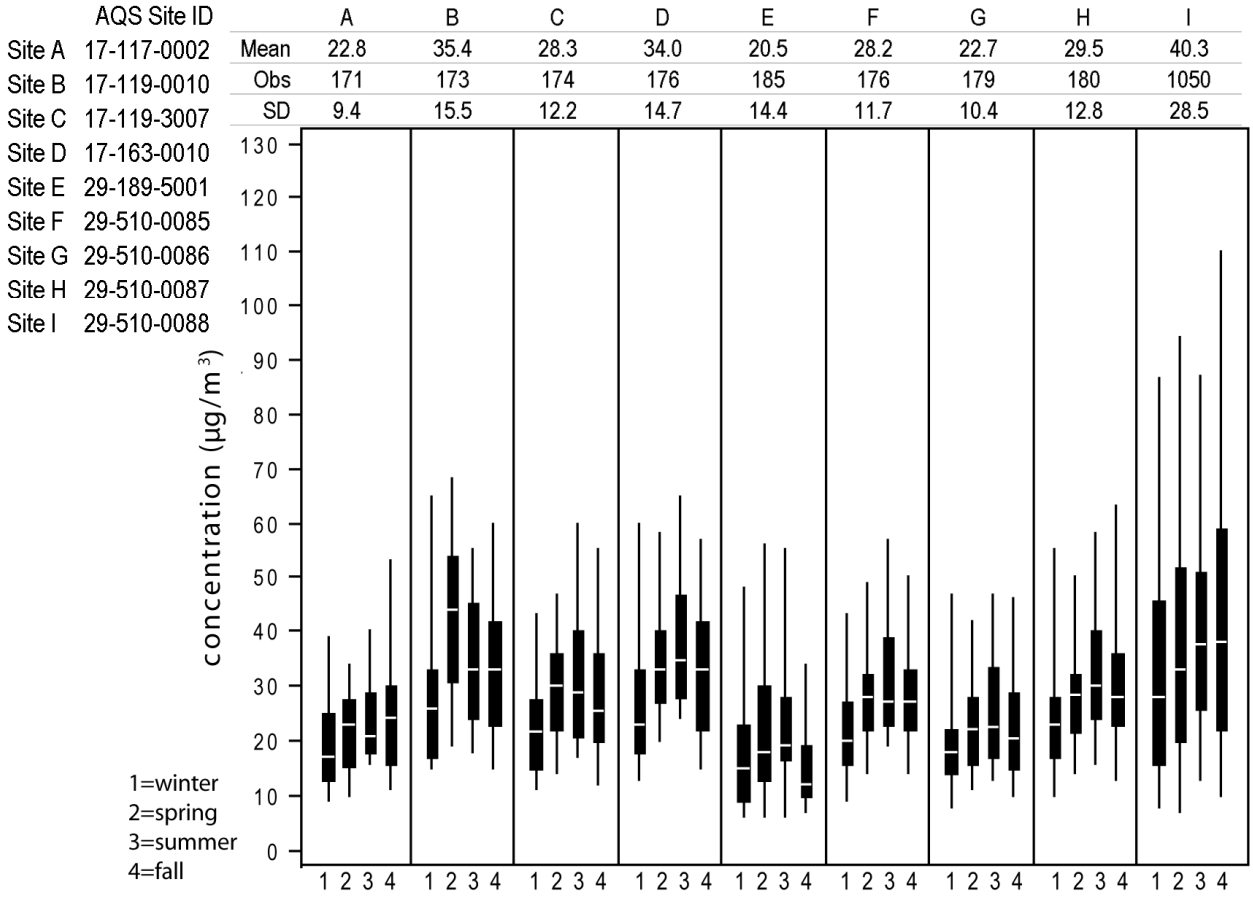


Figure A-125. Box plots illustrating the seasonal distribution of 24-h avg PM_{10} concentrations for St. Louis, MO.

Table A-49. 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)
		LEGEND				176	173	174	169
G		**R**					1.00	0.83	0.65
		(P90, COD)					(0.0, 0.00)	(16.0, 0.19)	(47.0, 0.32)
		N					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

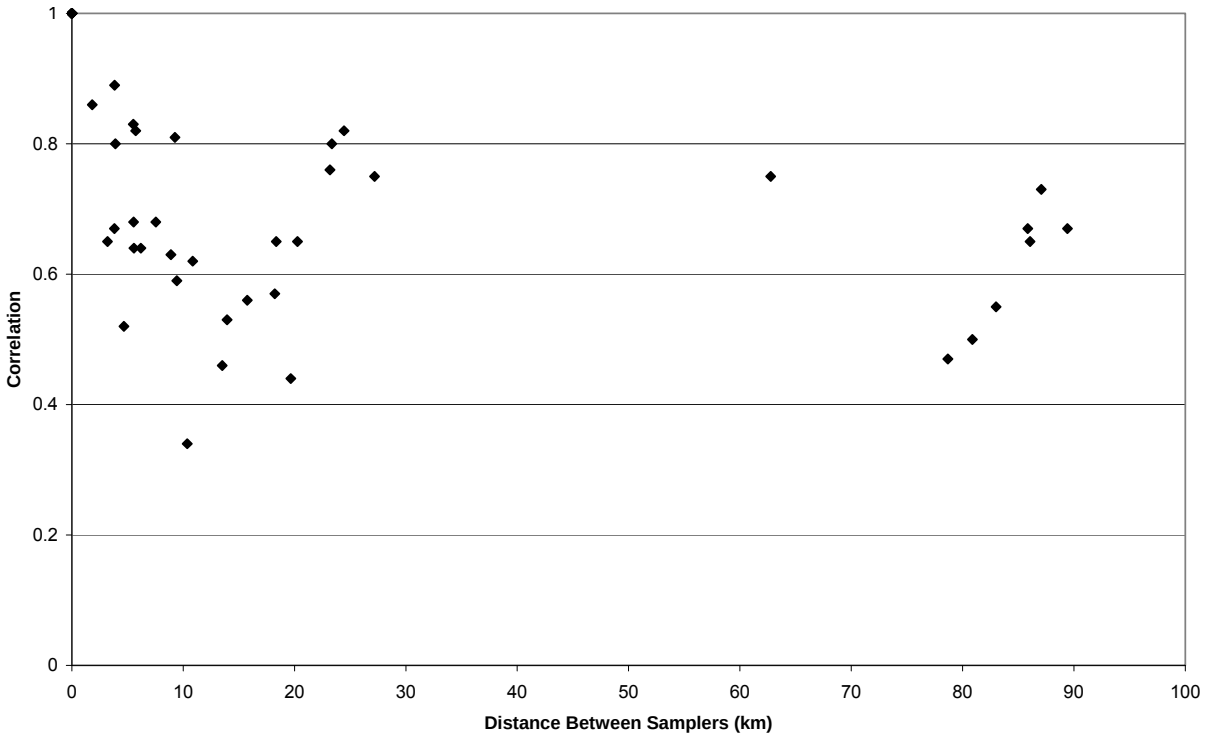


Figure A-126. PM_{10} inter-sampler correlations as a function of distance between monitors for St. Louis, MO.

Table A-50. 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

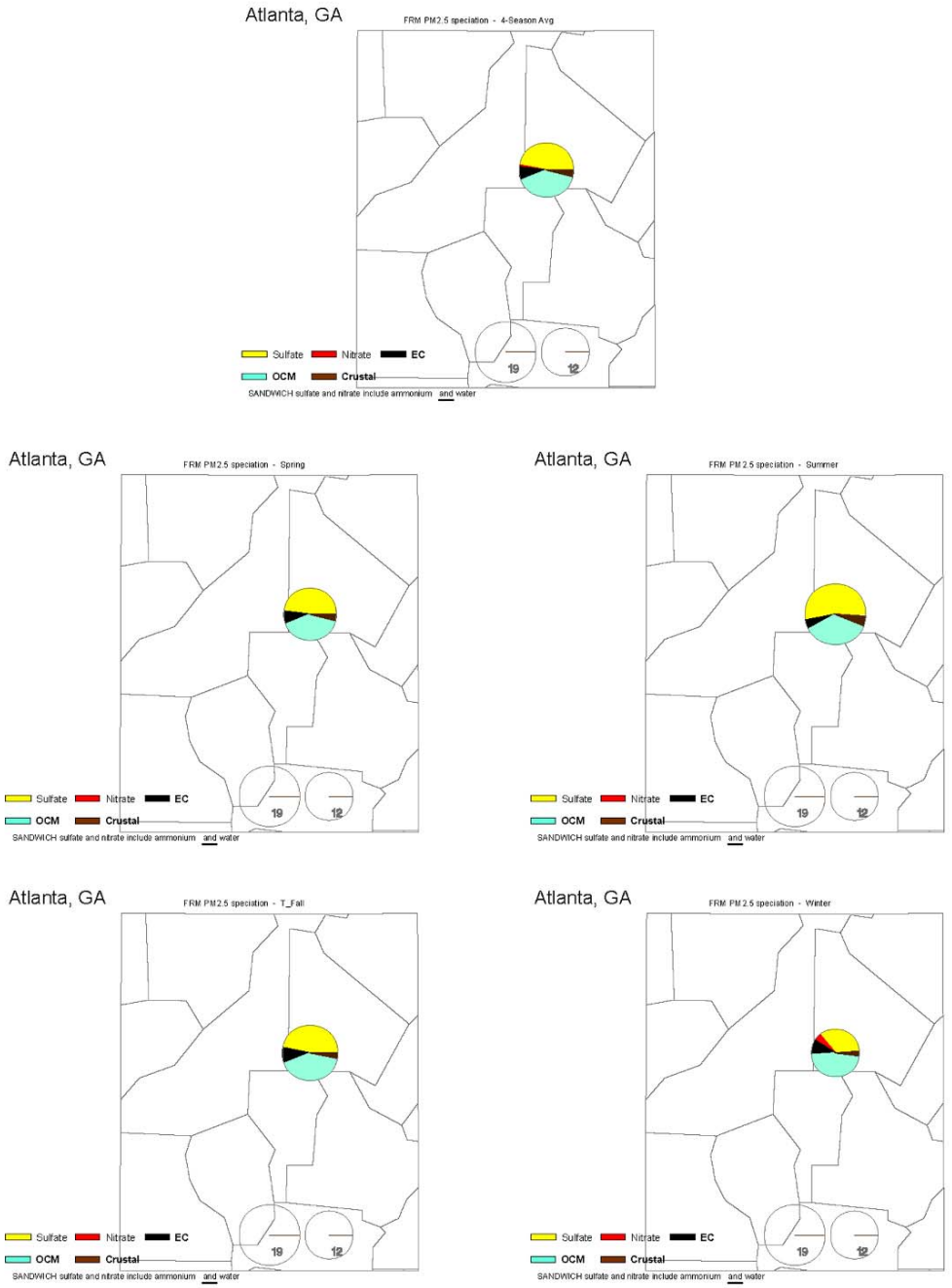


Figure A-127. Seasonally averaged $PM_{2.5}$ speciation data for 2005-2007 for a) annual, b) spring, c) summer, d) fall and e) winter derived using the SANDWICH method in Atlanta, GA.

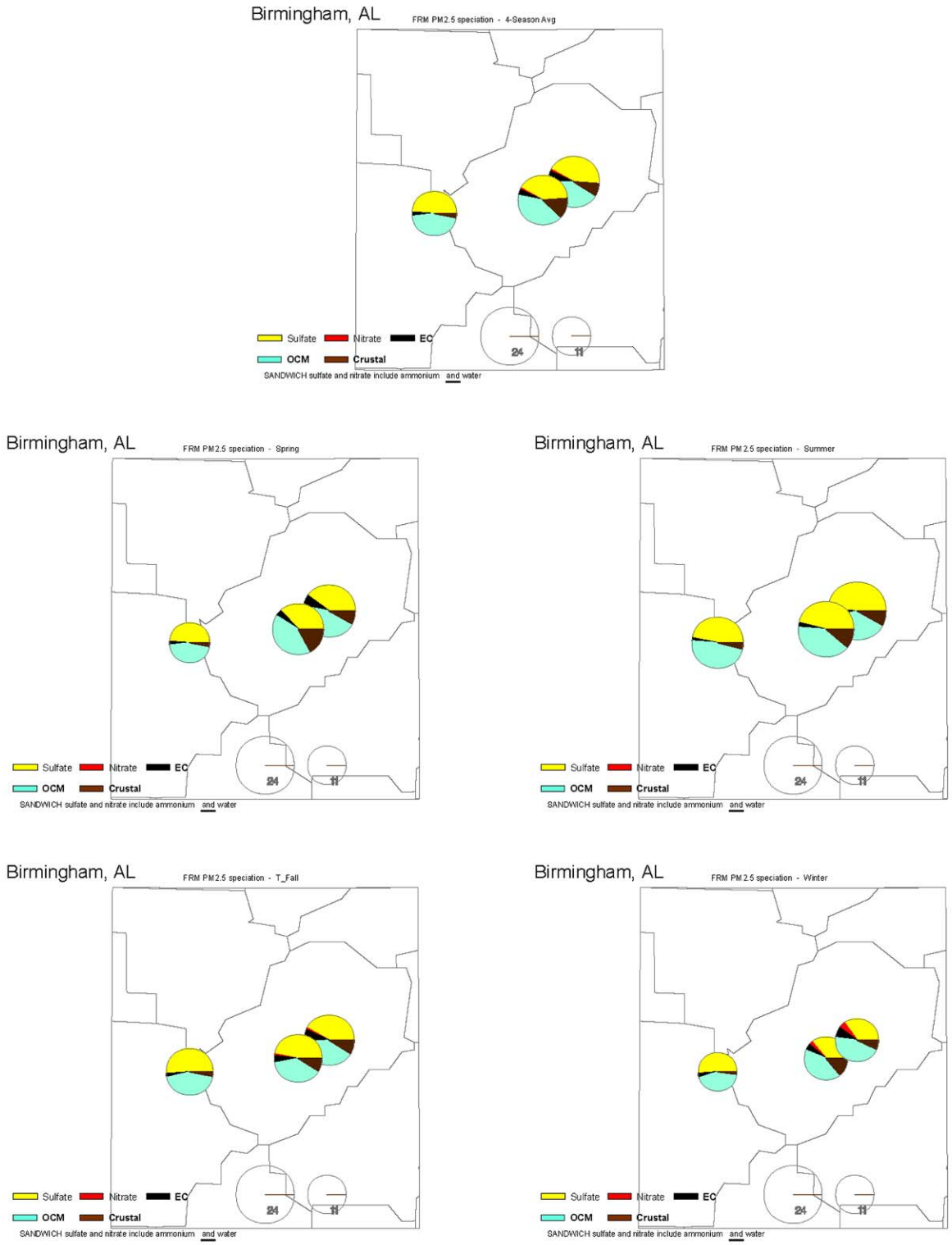


Figure A-128. Seasonally averaged $PM_{2.5}$ speciation data for 2005-2007 for a) annual, b) spring, c) summer, d) fall and e) winter derived using the SANDWICH method in Birmingham, AL.

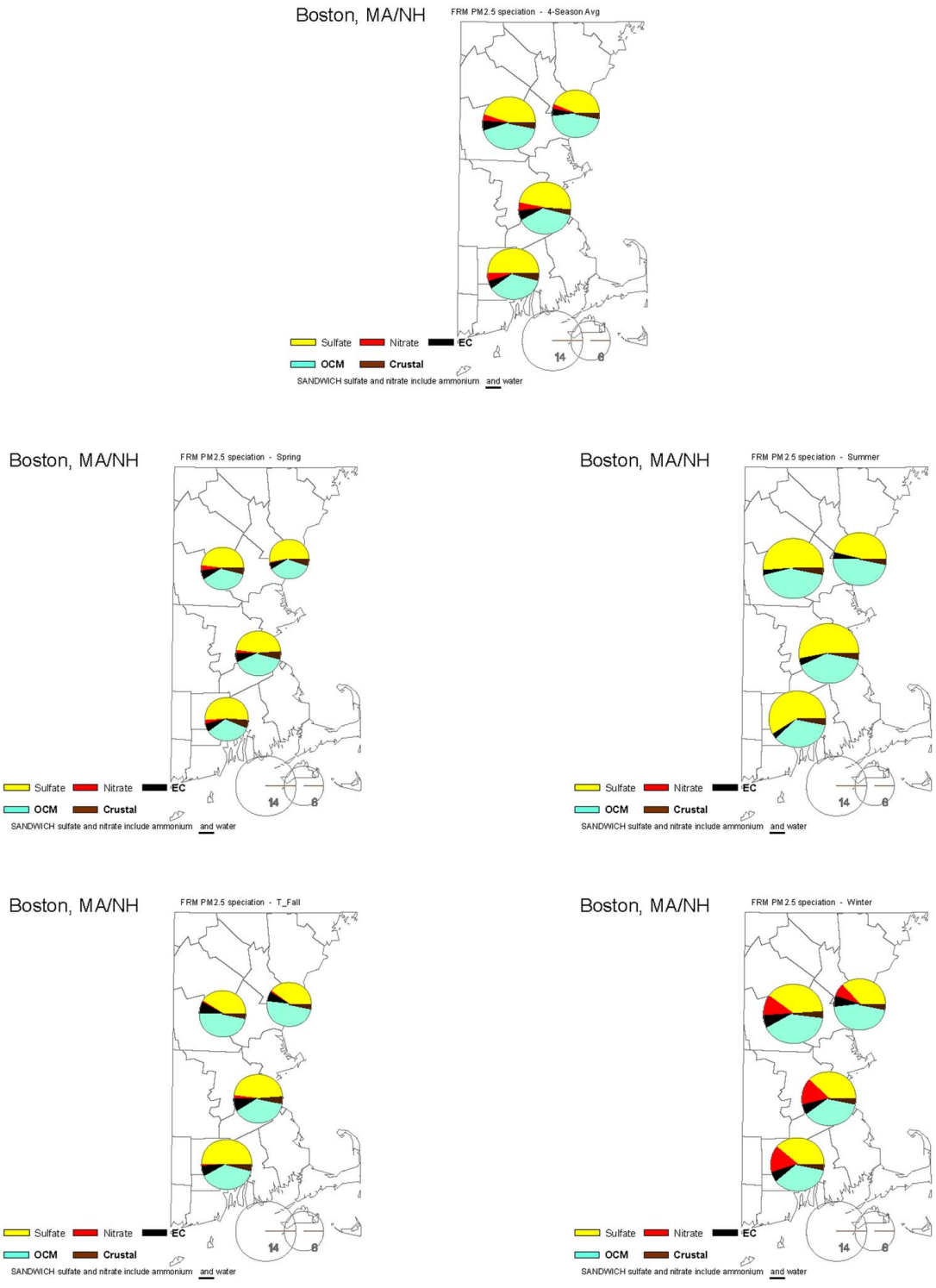


Figure A-129. Seasonally averaged $PM_{2.5}$ speciation data for 2005-2007 for a) annual, b) spring, c) summer, d) fall and e) winter derived using the SANDWICH method in Boston, MA.

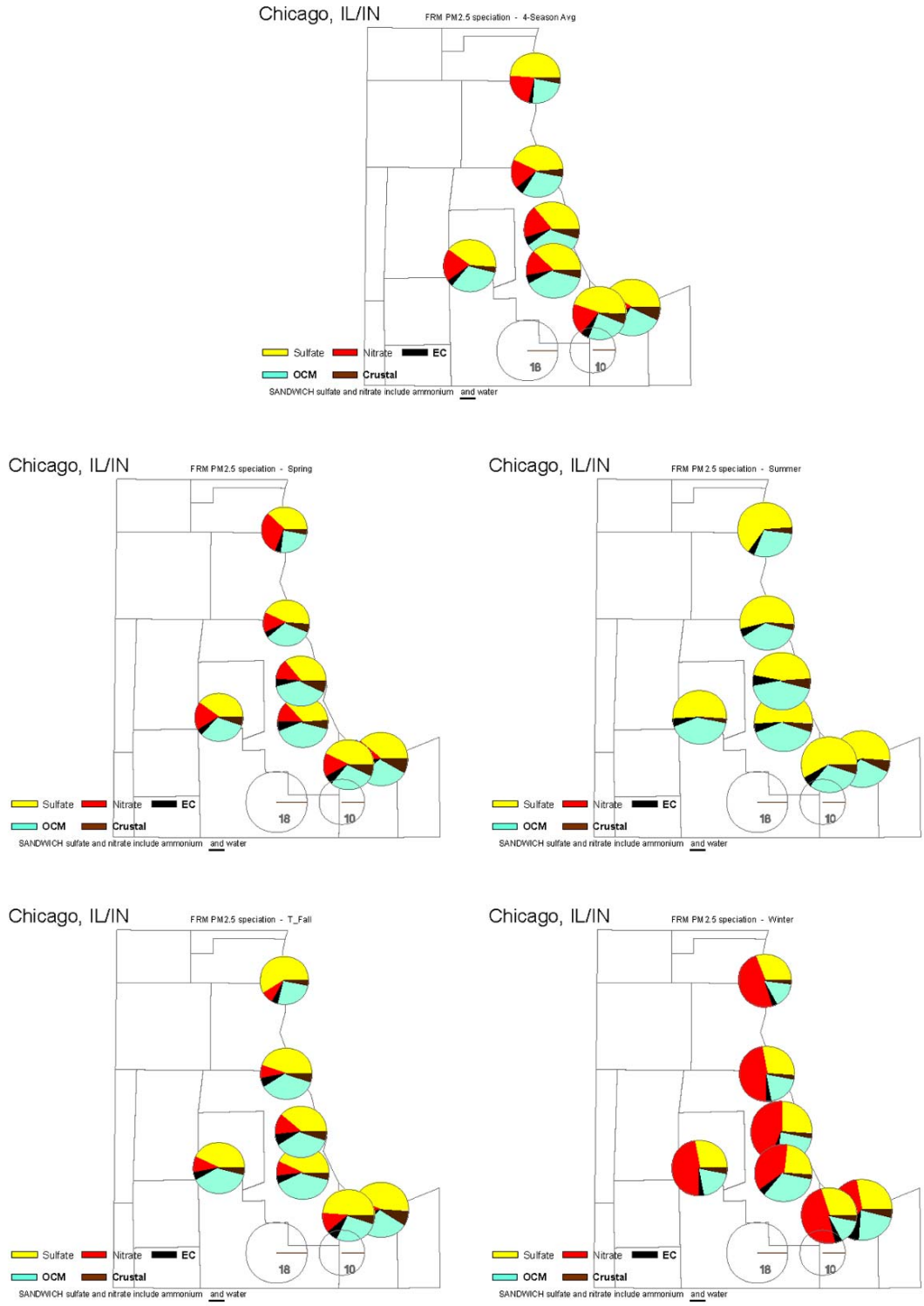


Figure A-130. Seasonally averaged $PM_{2.5}$ speciation data for 2005-2007 for a) annual, b) spring, c) summer, d) fall and e) winter derived using the SANDWICH method in Chicago, IL.

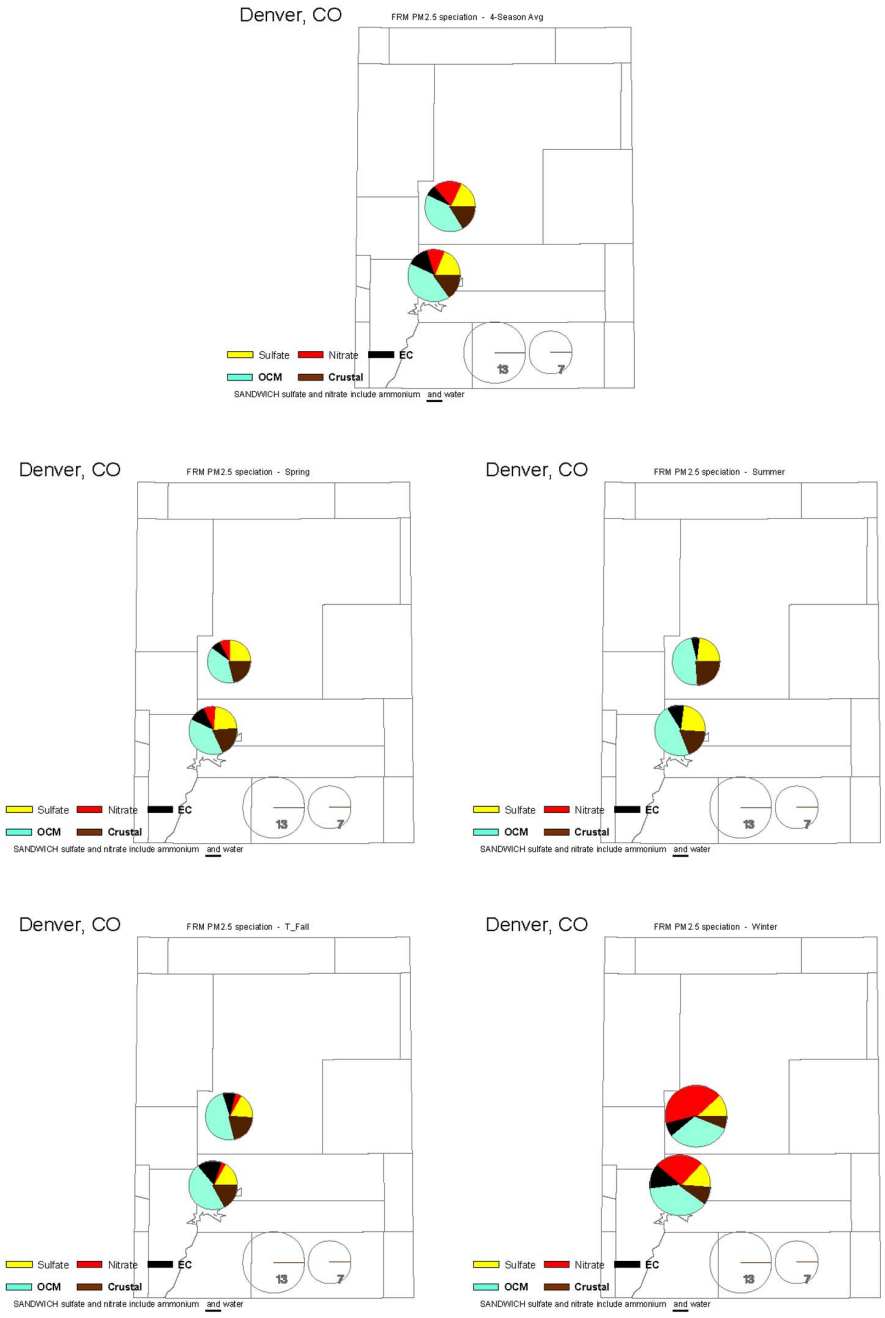


Figure A-131. Seasonally averaged $PM_{2.5}$ speciation data for 2005-2007 for a) annual, b) spring, c) summer, d) fall and e) winter, derived using the SANDWICH method in Denver, CO.

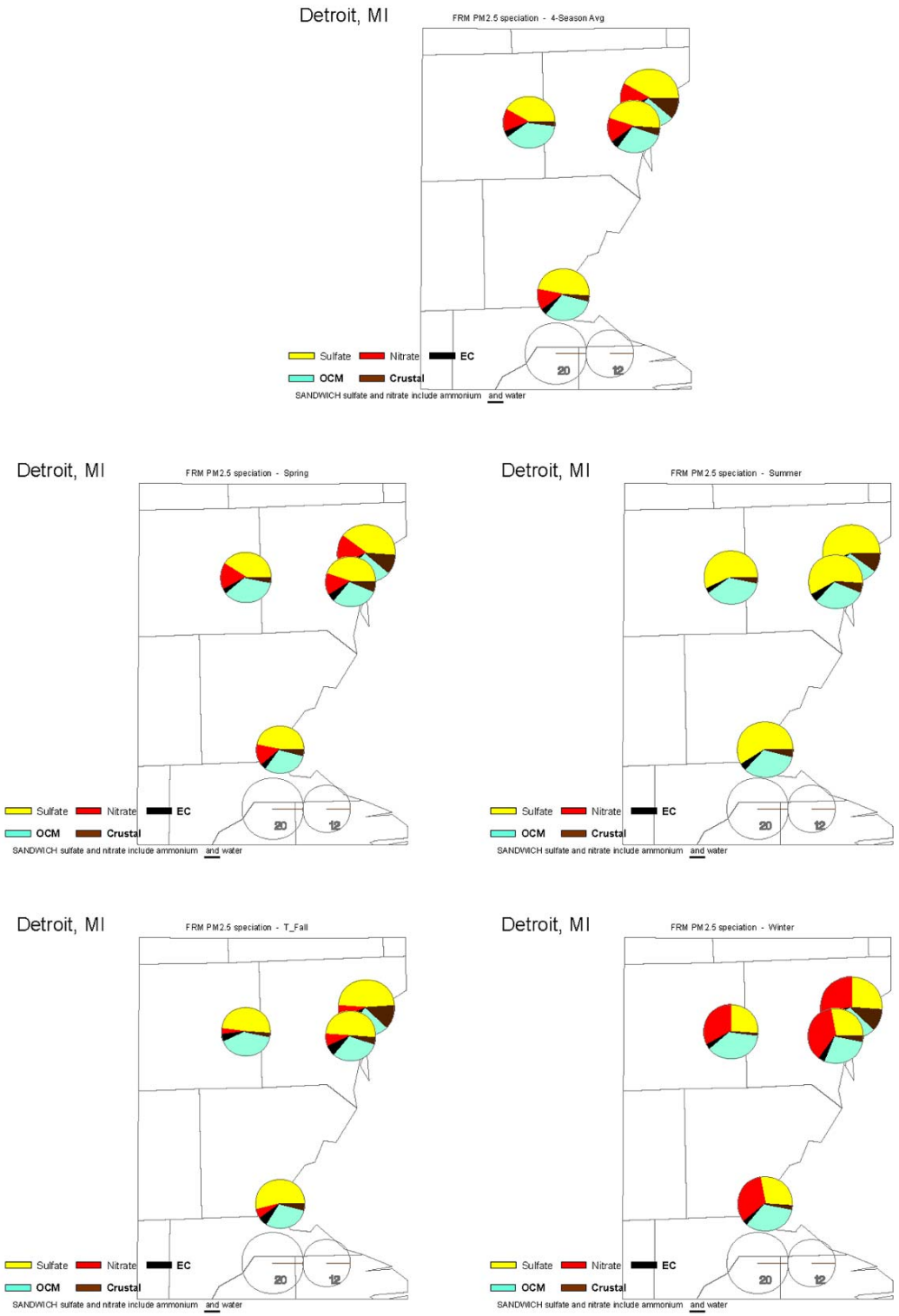


Figure A-132. Seasonally averaged $PM_{2.5}$ speciation data for 2005-2007 for a) annual, b) spring, c) summer, d) fall and e) winter derived using the SANDWICH method in Detroit, MI.

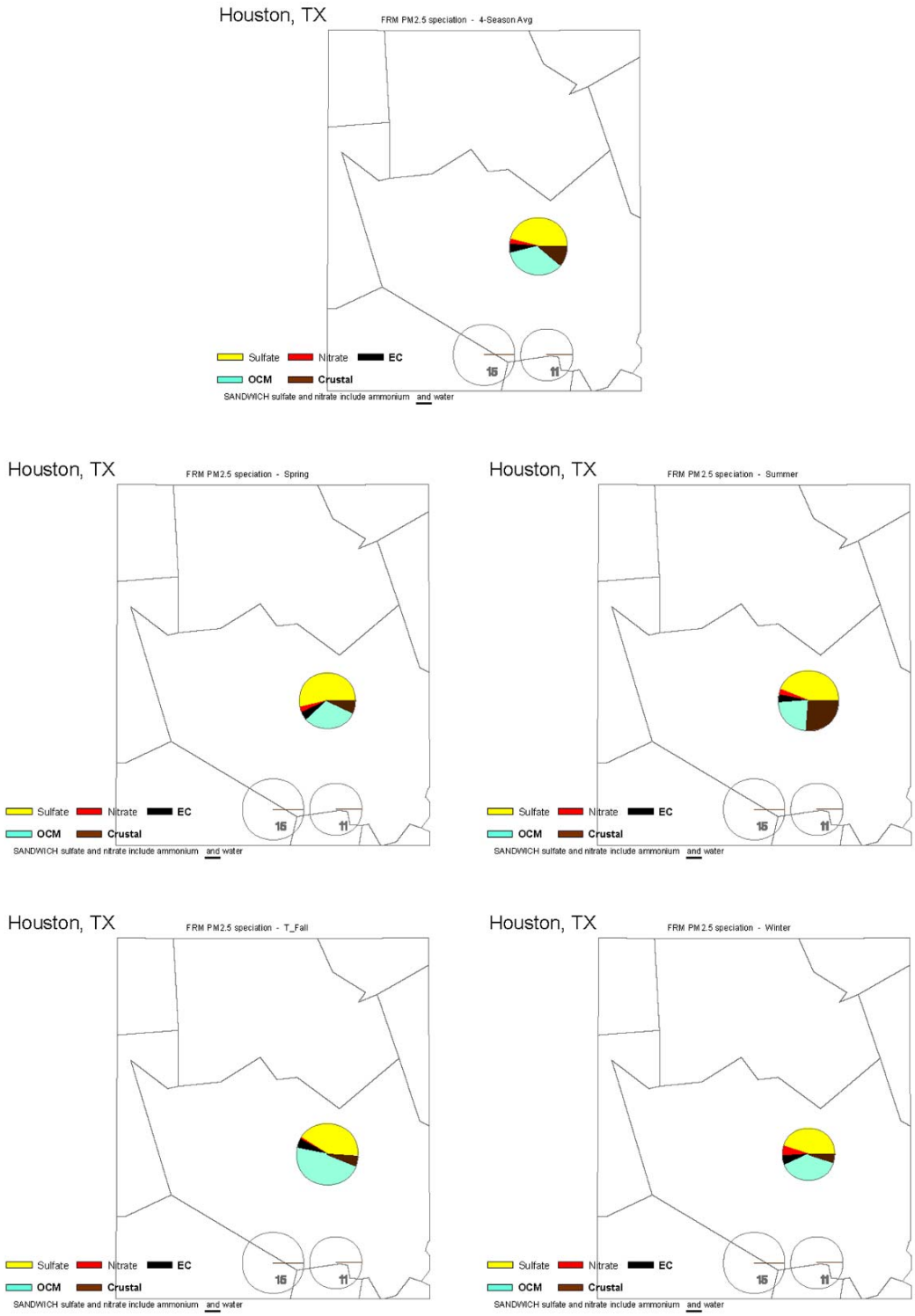


Figure A-133. Seasonally averaged $PM_{2.5}$ speciation data for 2005-2007 for a) annual, b) spring, c) summer, d) fall and e) winter derived using the SANDWICH method in Houston, TX.

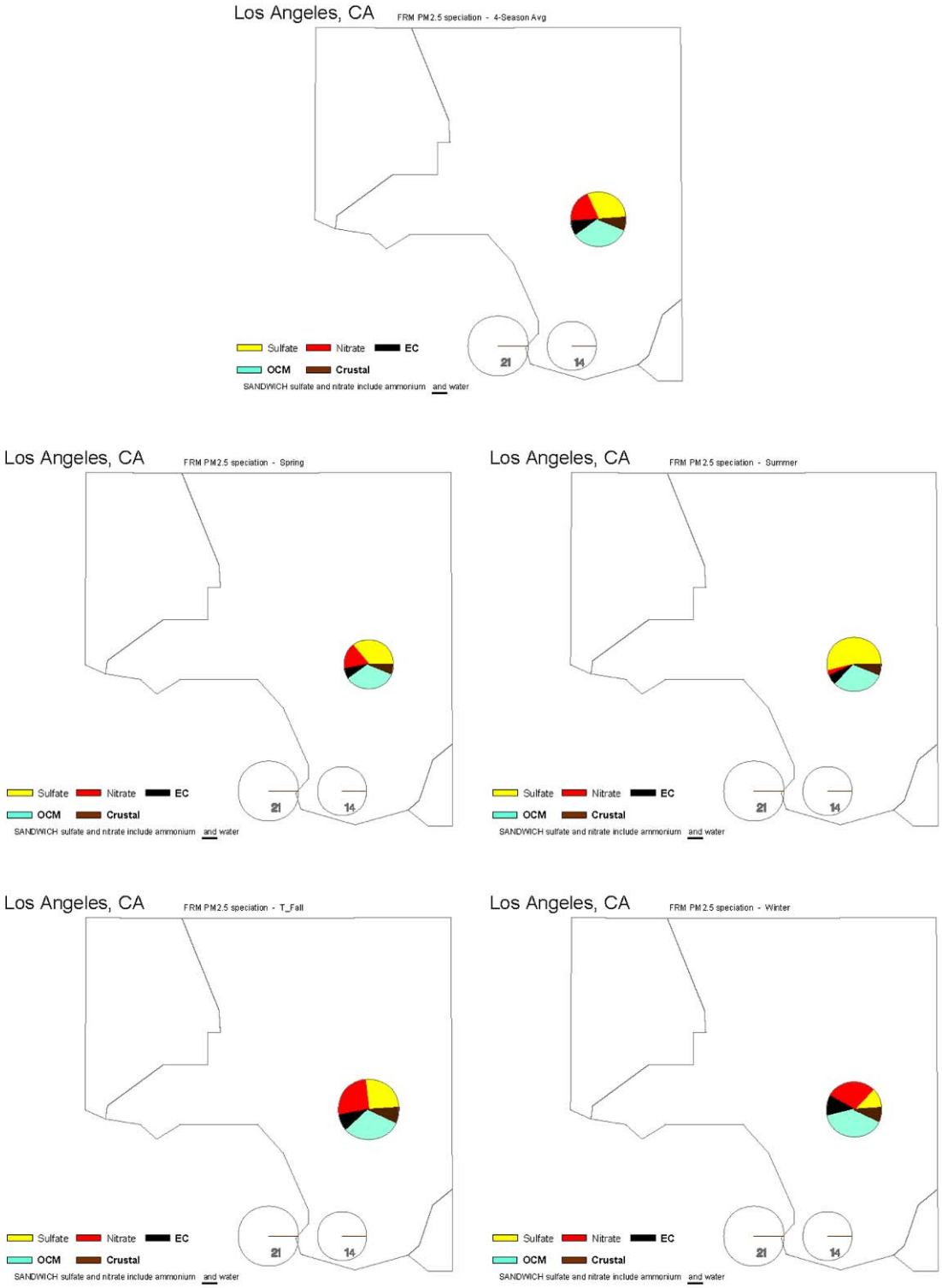


Figure A-134. Seasonally averaged $PM_{2.5}$ speciation data for 2005-2007 for a) annual, b) spring, c) summer, d) fall and e) winter derived using the SANDWICH method in Los Angeles, CA.

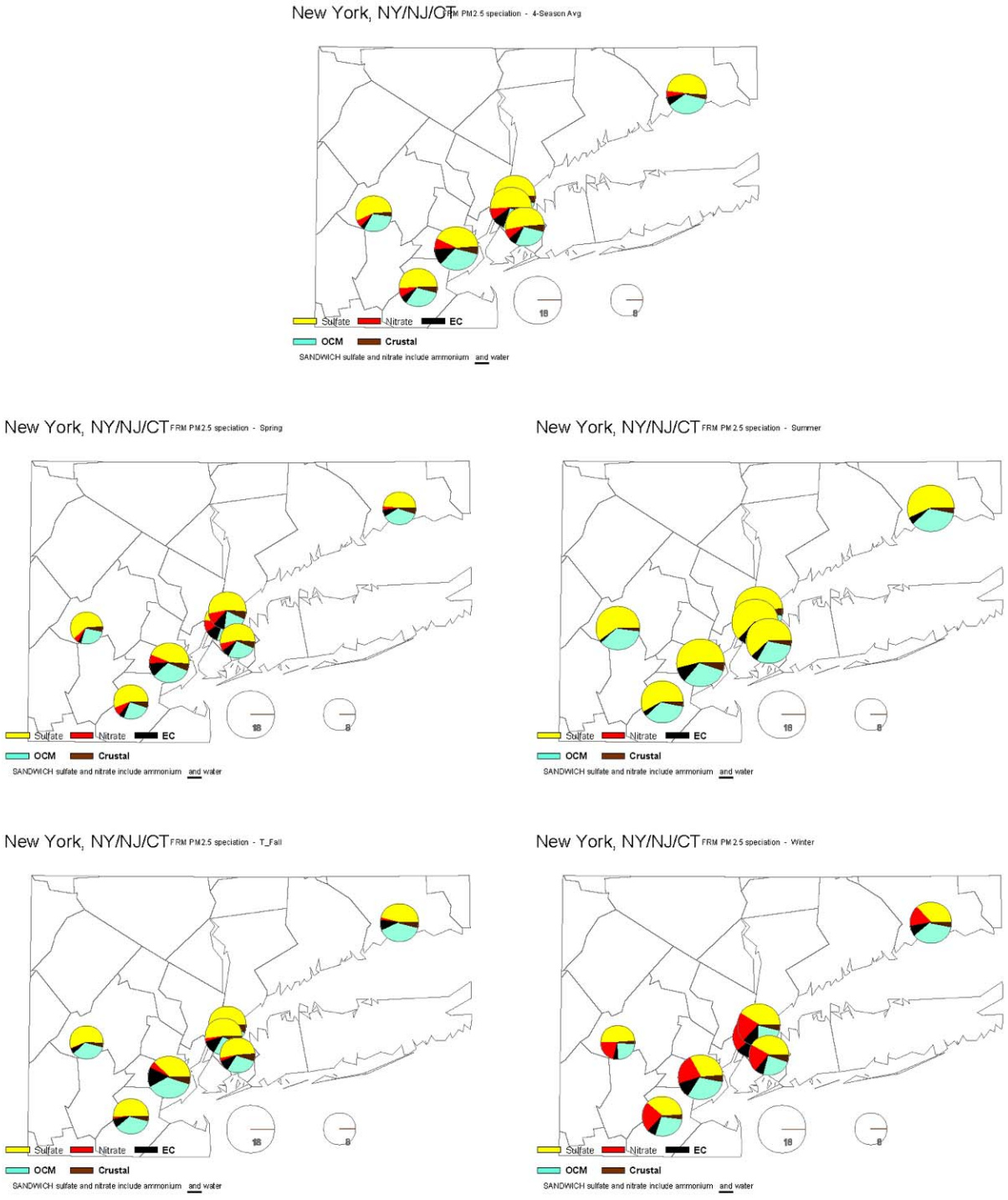


Figure A-135. Seasonally averaged $PM_{2.5}$ speciation data for 2005-2007 for a) annual, b) spring, c) summer, d) fall and e) winter derived using the SANDWICH method in New York, NY.

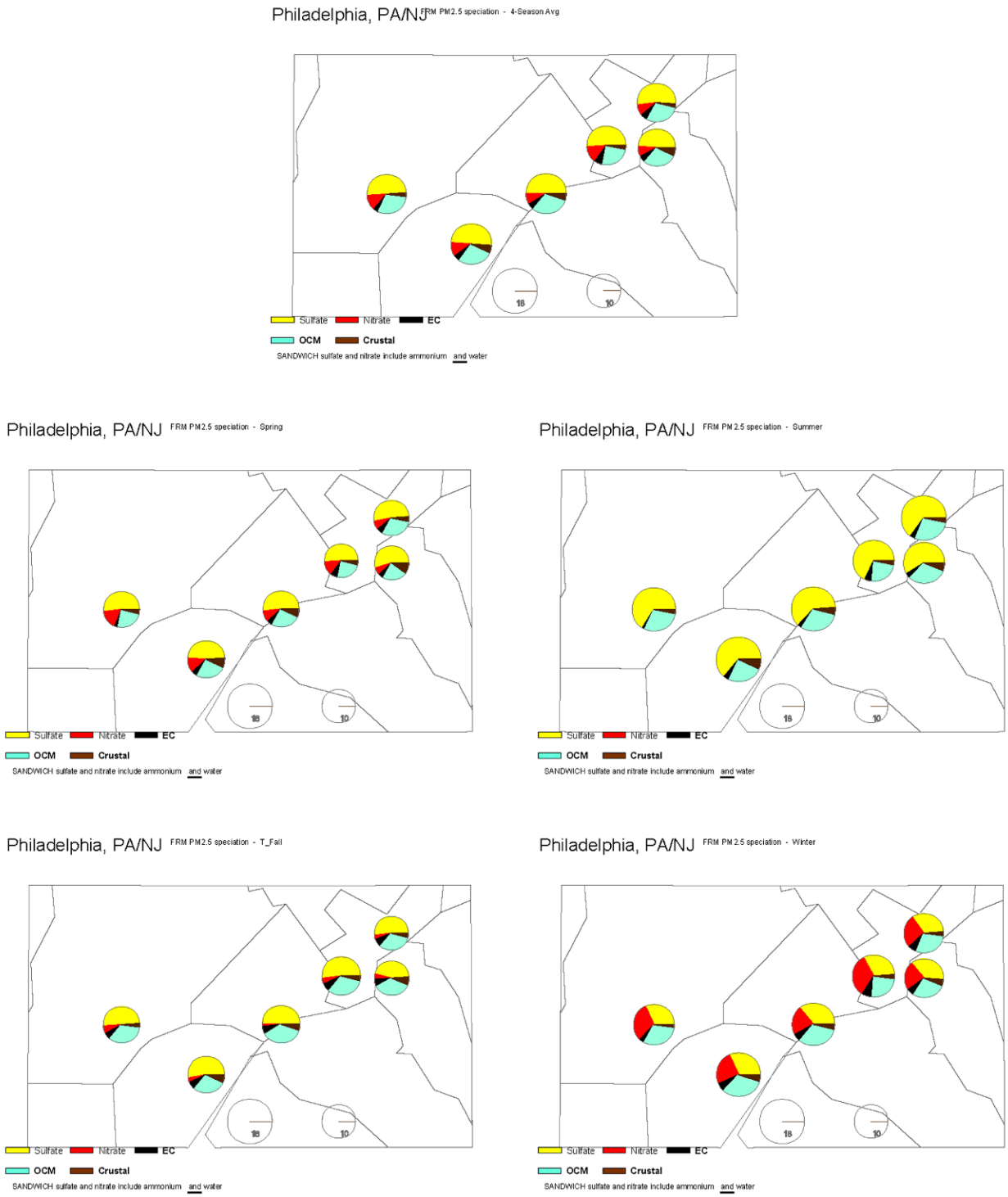


Figure A-136. Seasonally averaged $PM_{2.5}$ speciation data for 2005-2007 for a) annual, b) spring, c) summer, d) fall and e) winter derived using the SANDWICH method in Philadelphia, PA.

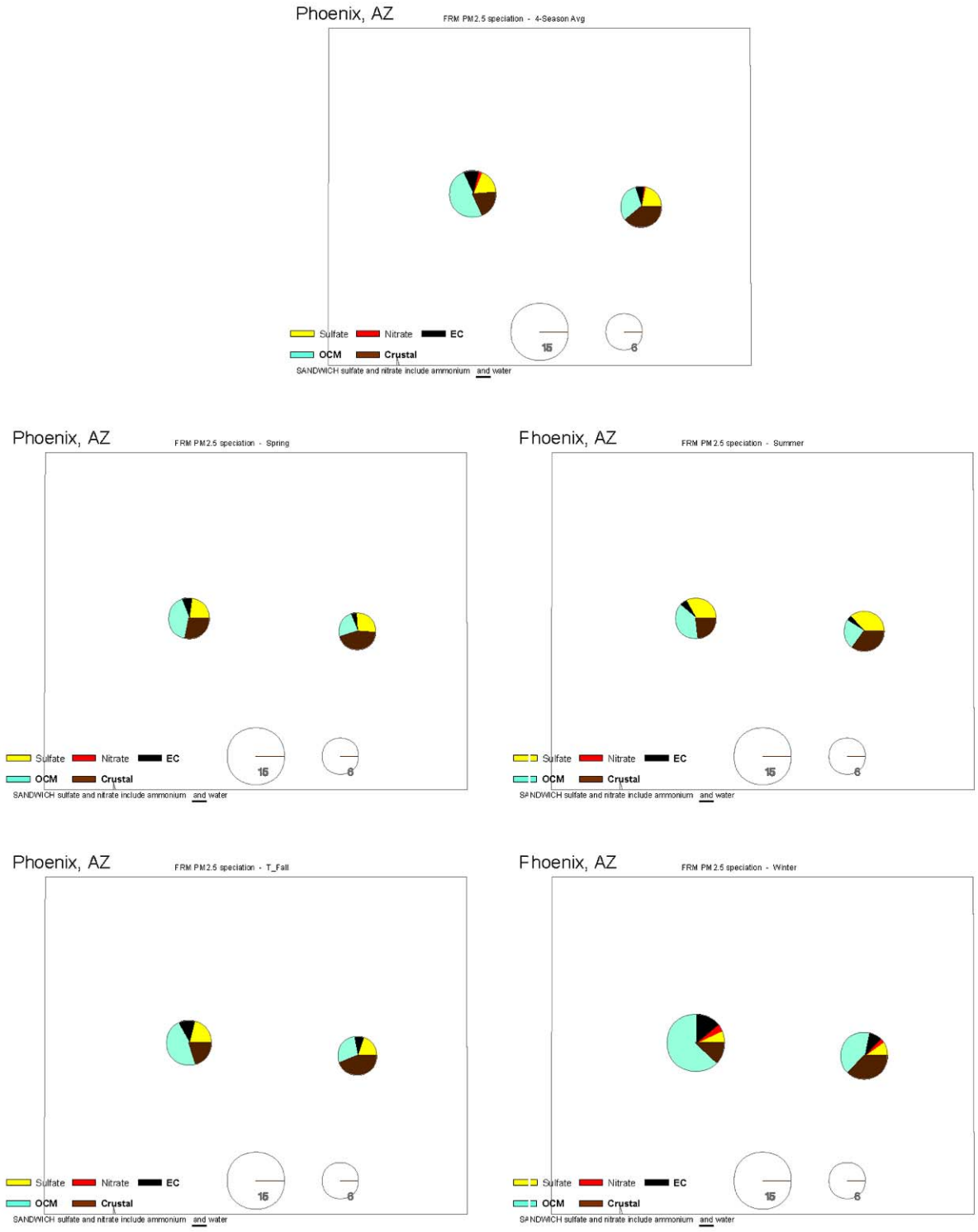


Figure A-137. Seasonally averaged $PM_{2.5}$ speciation data for 2005-2007 for a) annual, b) spring, c) summer, d) fall and e) winter derived using the SANDWICH method in Phoenix, AZ.

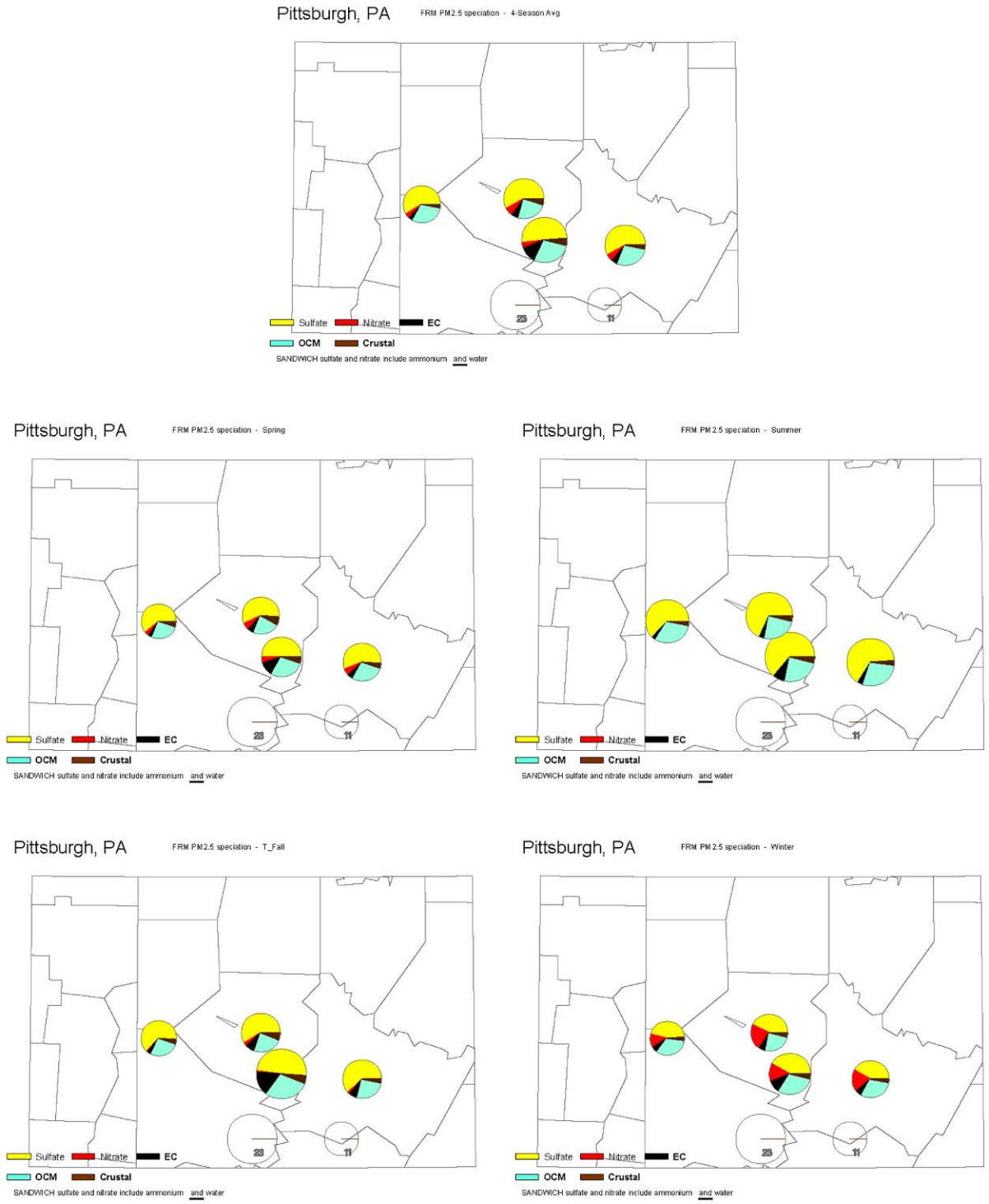


Figure A-138. Seasonally averaged $PM_{2.5}$ speciation data for 2005-2007 for a) annual, b) spring, c) summer, d) fall and e) winter derived using the SANDWICH method in Pittsburgh, PA.

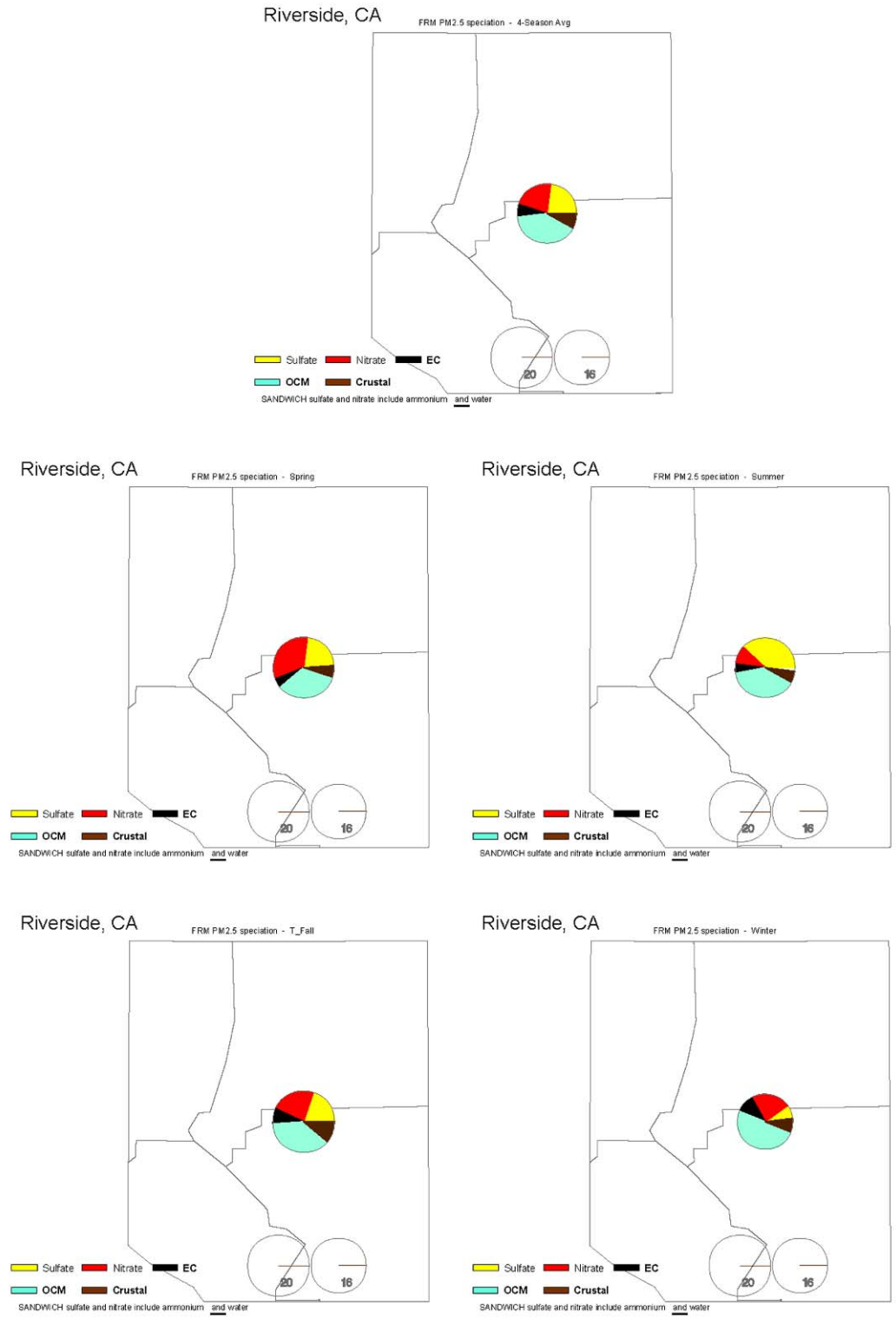


Figure A-139. Seasonally averaged $PM_{2.5}$ speciation data for 2005-2007 for a) annual, b) spring, c) summer, d) fall and e) winter derived using the SANDWICH method in Riverside, CA.

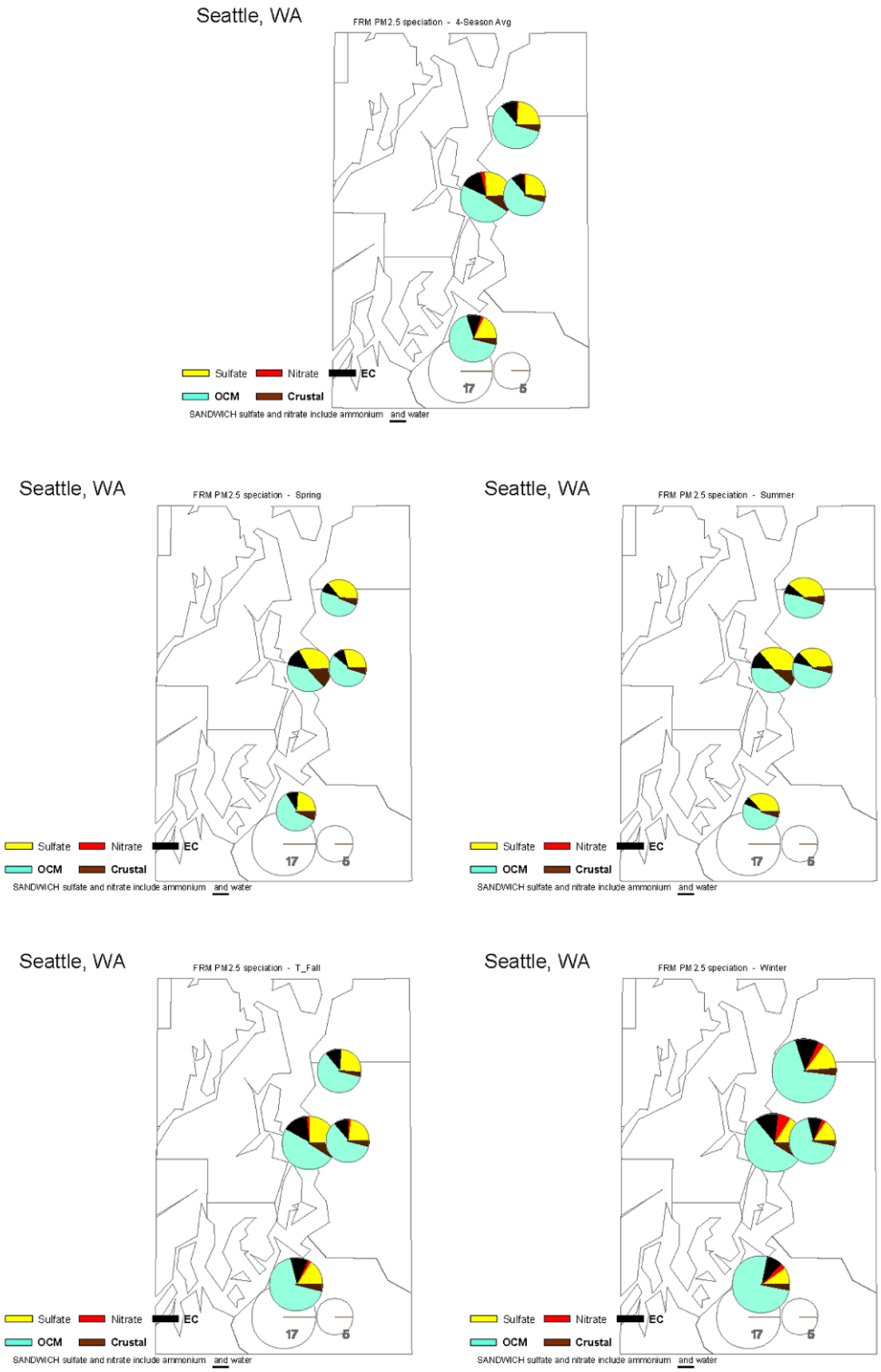


Figure A-140. Seasonally averaged $PM_{2.5}$ speciation data for 2005-2007 for a) annual, b) spring, c) summer, d) fall and e) winter derived using the SANDWICH method in Seattle, WA.

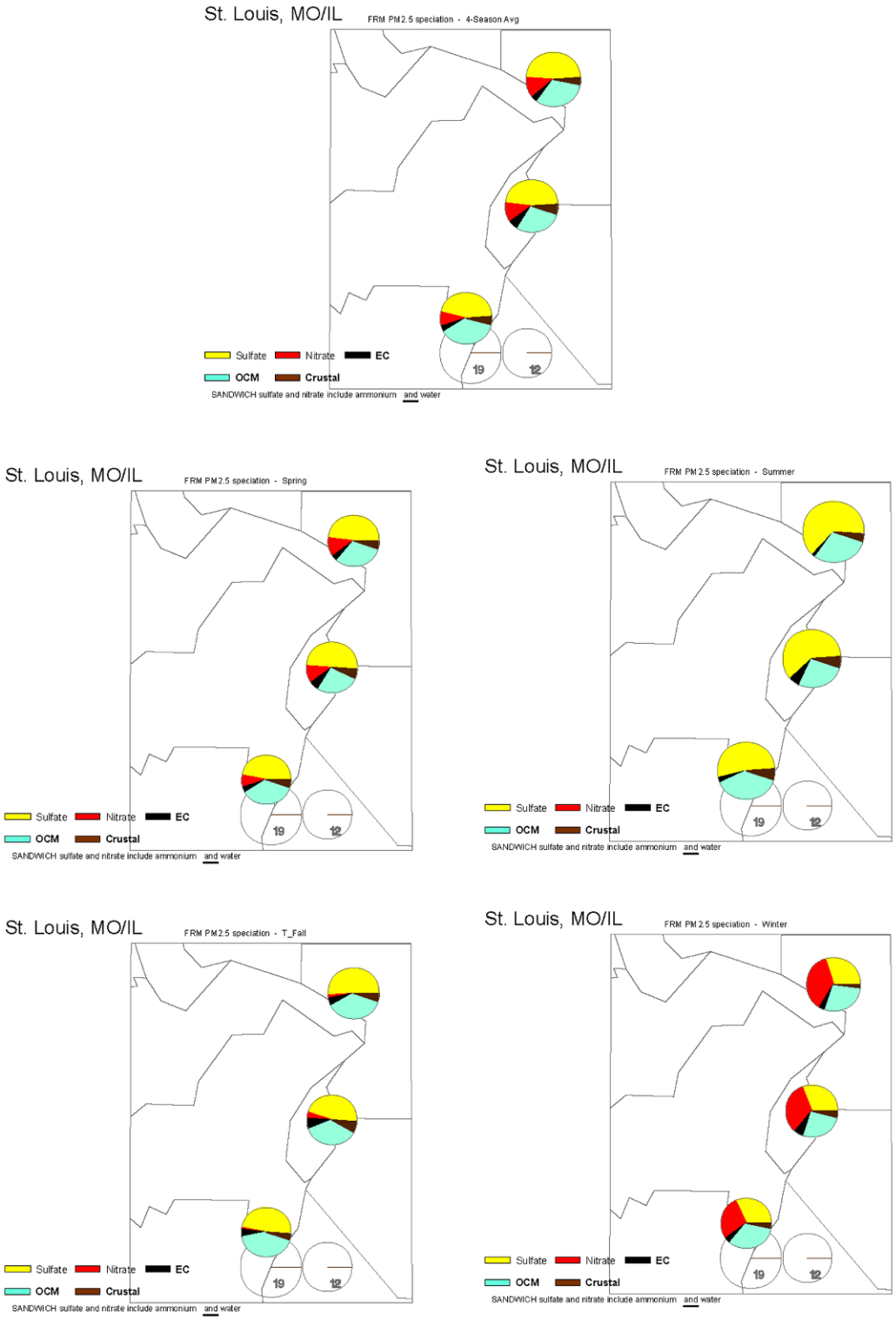


Figure A-141. Seasonally averaged $PM_{2.5}$ speciation data for 2005-2007 for a) annual, b) spring, c) summer, d) fall and e) winter derived using the SANDWICH method in St. Louis, MO.

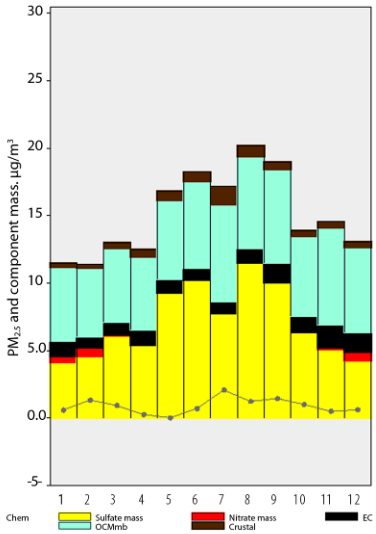


Figure A-142. Seasonal patterns in $PM_{2.5}$ chemical composition from city-wide monthly average values for Atlanta, GA, 2005-2007. The gray line represents the difference in OCM calculated using material balance and blank corrected OC×1.4.

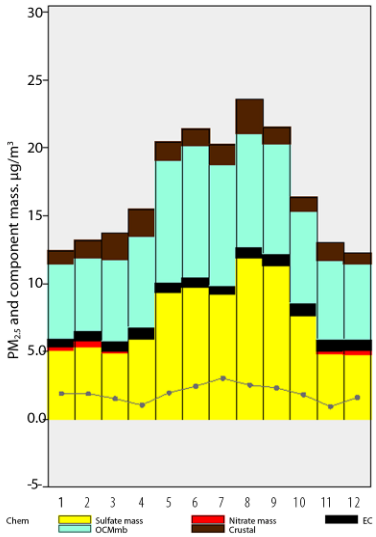


Figure A-143. Seasonal patterns in $PM_{2.5}$ chemical composition from city-wide monthly average values for Birmingham, AL, 2005-2007. The gray line represents the difference in OCM calculated using material balance and blank corrected OC×1.4.

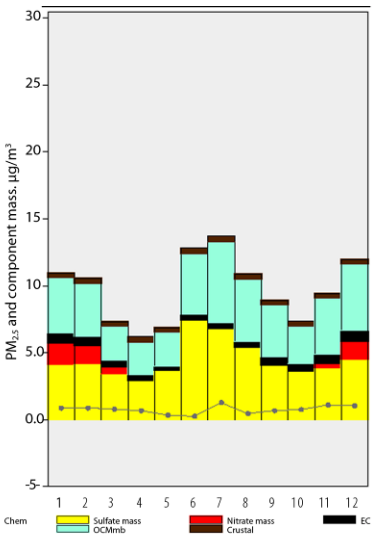


Figure A-144. Seasonal patterns in $PM_{2.5}$ chemical composition from city-wide monthly average values for Boston, MA, 2005-2007. The gray line represents the difference in OCM calculated using material balance and blank corrected OC×1.4.

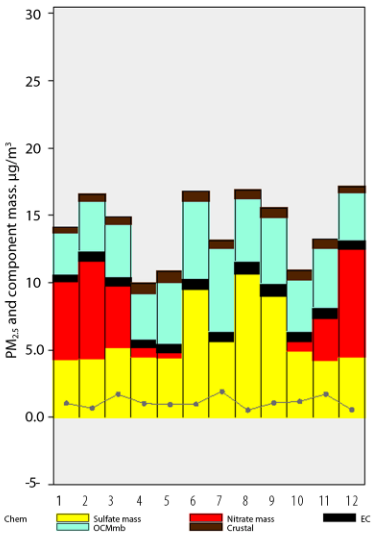


Figure A-145. Seasonal patterns in $PM_{2.5}$ chemical composition from city-wide monthly average values for Chicago, IL, 2005-2007. The gray line represents the difference in OCM calculated using material balance and blank corrected OC×1.4.

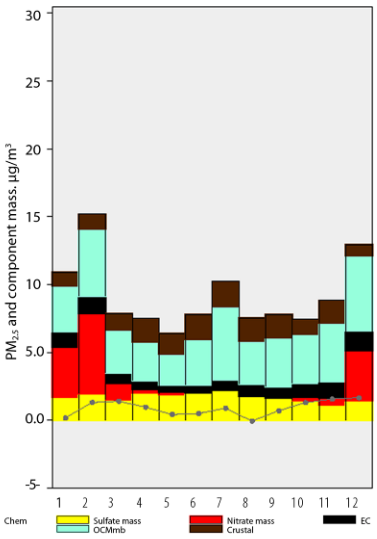


Figure A-146. Seasonal patterns in $PM_{2.5}$ chemical composition from city-wide monthly average values for Denver, CO, 2005-2007. The gray line represents the difference in OCM calculated using material balance and blank corrected OC×1.4.

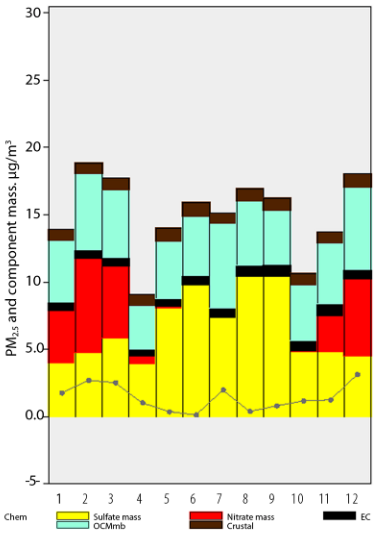


Figure A-147. Seasonal patterns in $PM_{2.5}$ chemical composition from city-wide monthly average values for Detroit, MI, 2005-2007. The gray line represents the difference in OCM calculated using material balance and blank corrected OC x 1.4.

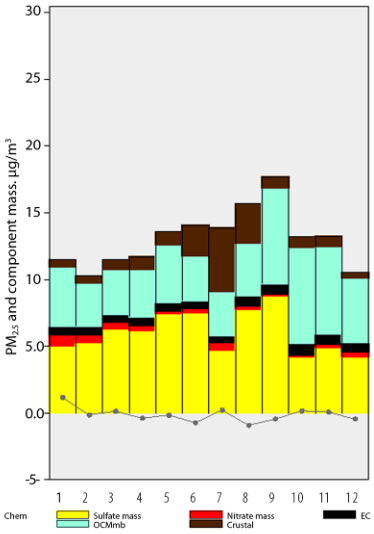


Figure A-148. Seasonal patterns in $PM_{2.5}$ chemical composition from city-wide monthly average values for Houston, TX, 2005-2007. The gray line represents the difference in OCM calculated using material balance and blank corrected OC×1.4.

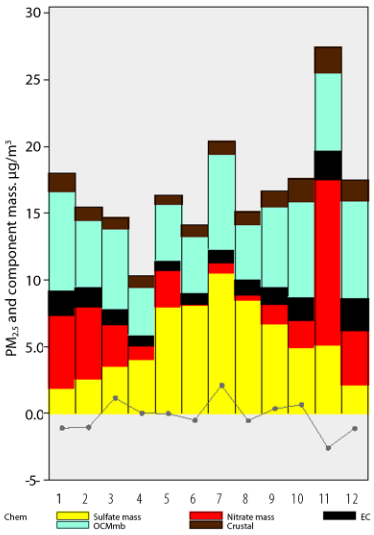


Figure A-149. Seasonal patterns in $PM_{2.5}$ chemical composition from city-wide monthly average values for Los Angeles, CA, 2005-2007. The gray line represents the difference in OCM calculated using material balance and blank corrected OC×1.4.

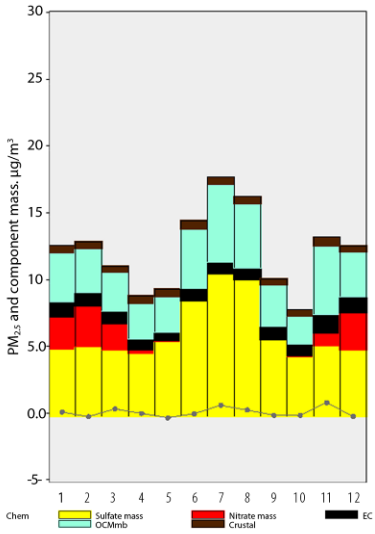


Figure A-150. Seasonal patterns in $PM_{2.5}$ chemical composition from city-wide monthly average values for New York, NY, 2005-2007. The gray line represents the difference in OCM calculated using material balance and blank corrected OC×1.4.

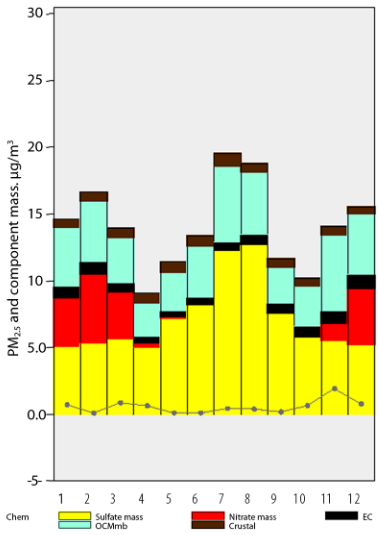


Figure A-151. Seasonal patterns in $PM_{2.5}$ chemical composition from city-wide monthly average values for Philadelphia, PA, 2005-2007. The gray line represents the difference in OCM calculated using material balance and blank corrected OC×1.4.

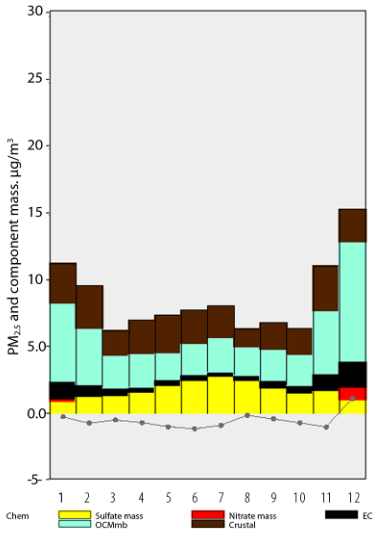


Figure A-152. Seasonal patterns in $PM_{2.5}$ chemical composition from city-wide monthly average values for Phoenix, AZ, 2005-2007. The gray line represents the difference in OCM calculated using material balance and blank corrected OC×1.4.

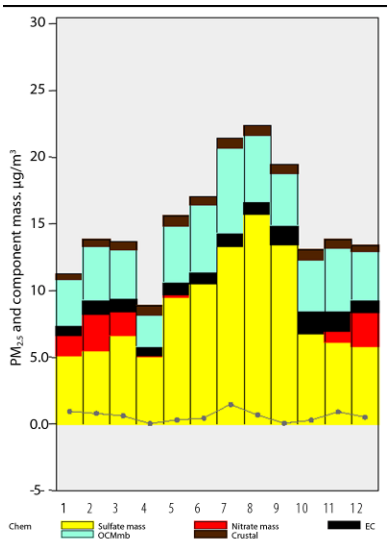


Figure A-153. Seasonal patterns in $PM_{2.5}$ chemical composition from city-wide monthly average values for Pittsburgh, PA, 2005-2007. The gray line represents the difference in OCM calculated using material balance and blank corrected OC×1.4.

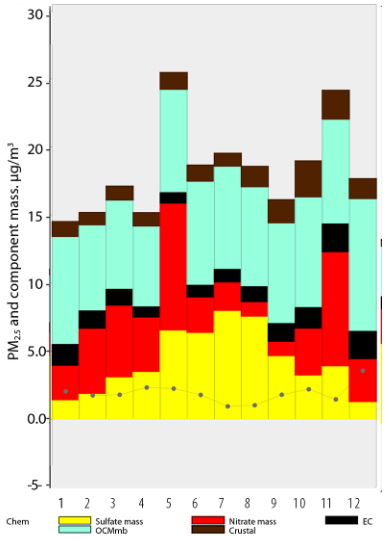


Figure A-154. Seasonal patterns in $PM_{2.5}$ chemical composition from city-wide monthly average values for Riverside, CA, 2005-2007. The gray line represents the difference in OCM calculated using material balance and blank corrected OC×1.4.

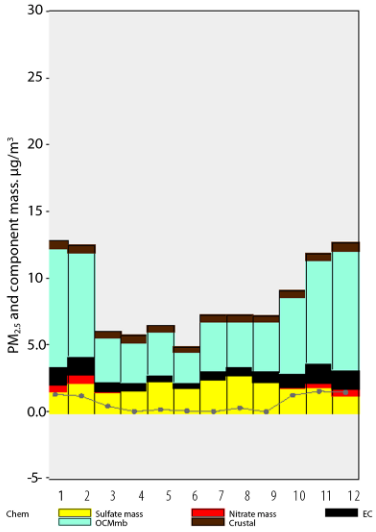


Figure A-155. Seasonal patterns in $PM_{2.5}$ chemical composition from city-wide monthly average values for Seattle, WA, 2005-2007. The gray line represents the difference in OCM calculated using material balance and blank corrected OC×1.4.

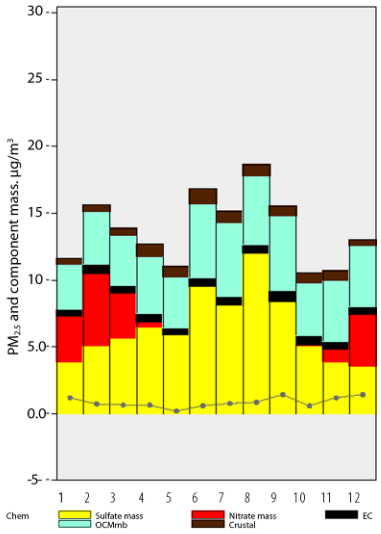


Figure A-156. Seasonal patterns in $PM_{2.5}$ chemical composition from city-wide monthly average values for St. Louis, MO, 2005-2007. The gray line represents the difference in OCM calculated using material balance and blank corrected OC×1.4.

A.2.4. Diel Trends

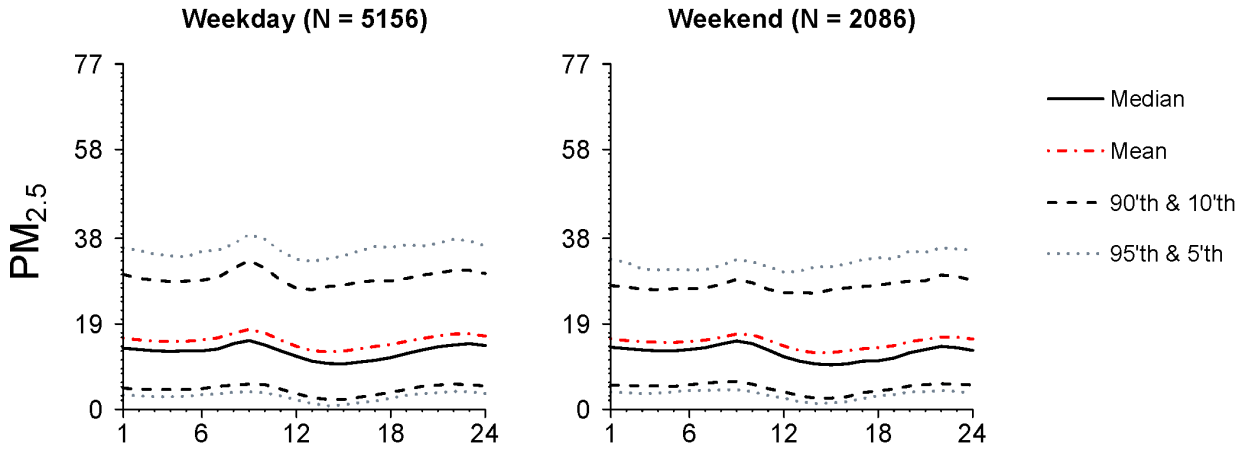


Figure A-157. Diel plots generated from all available hourly FRM-like $PM_{2.5}$ data, stratified by weekday (left) and weekend (right), in Atlanta, GA. Included are the number of monitor days (N) and the median, mean, 5th, 10th, 90th and 95th percentiles for each hour.

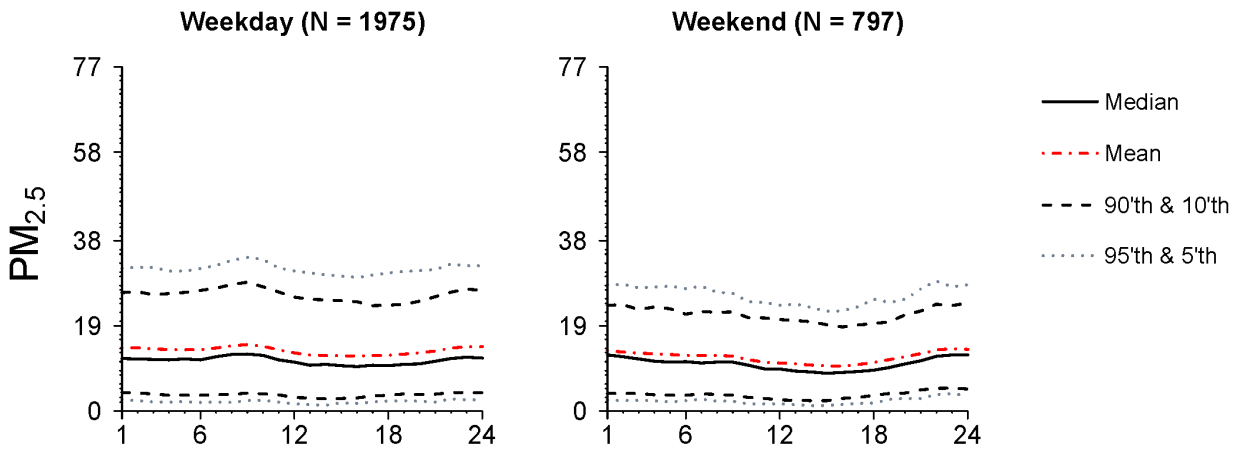


Figure A-158. Diel plots generated from all available hourly FRM-like $PM_{2.5}$ data, stratified by weekday (left) and weekend (right), in Chicago, IL. Included are the number of monitor days (N) and the median, mean, 5th, 10th, 90th and 95th percentiles for each hour.

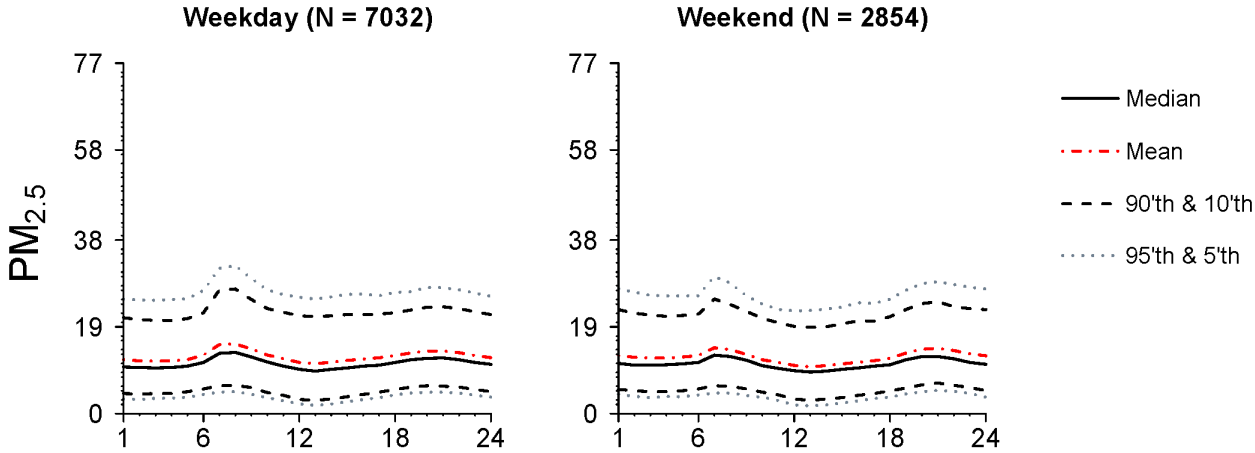


Figure A-159. Diel plots generated from all available hourly FRM-like $PM_{2.5}$ data, stratified by weekday (left) and weekend (right), in Houston, TX. Included are the number of monitor days (N) and the median, mean, 5th, 10th, 90th and 95th percentiles for each hour.

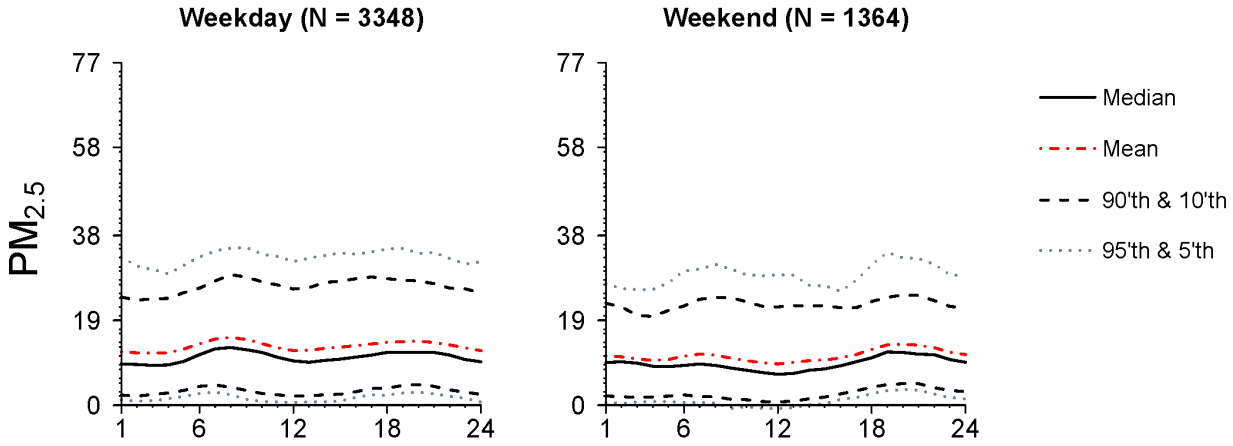


Figure A-160. Diel plots generated from all available hourly FRM-like $PM_{2.5}$ data, stratified by weekday (left) and weekend (right), in New York, NY. Included are the number of monitor days (N) and the median, mean, 5th, 10th, 90th and 95th percentiles for each hour.

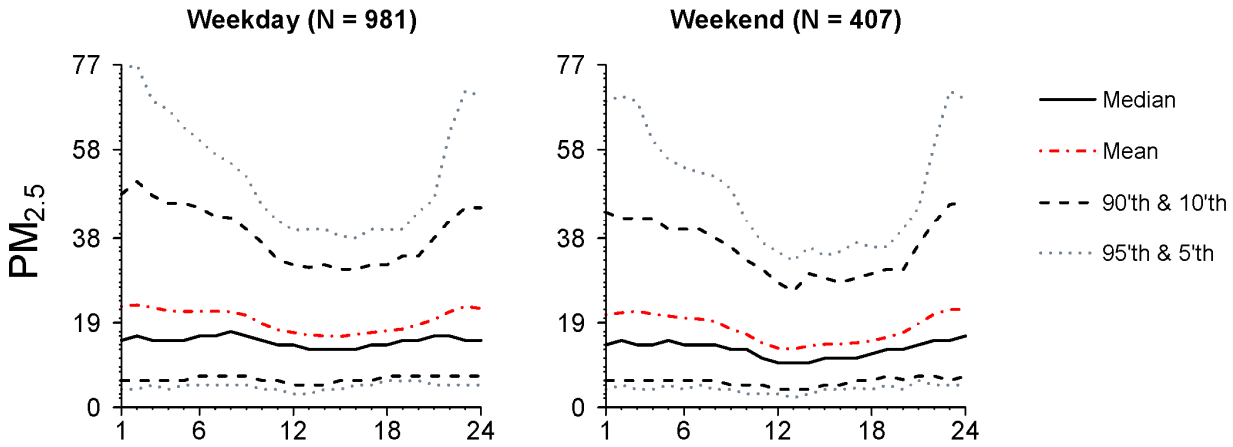


Figure A-161. Diel plots generated from all available hourly FRM-like $PM_{2.5}$ data, stratified by weekday (left) and weekend (right), in Pittsburgh, PA. Included are the number of monitor days (N) and the median, mean, 5th, 10th, 90th and 95th percentiles for each hour.

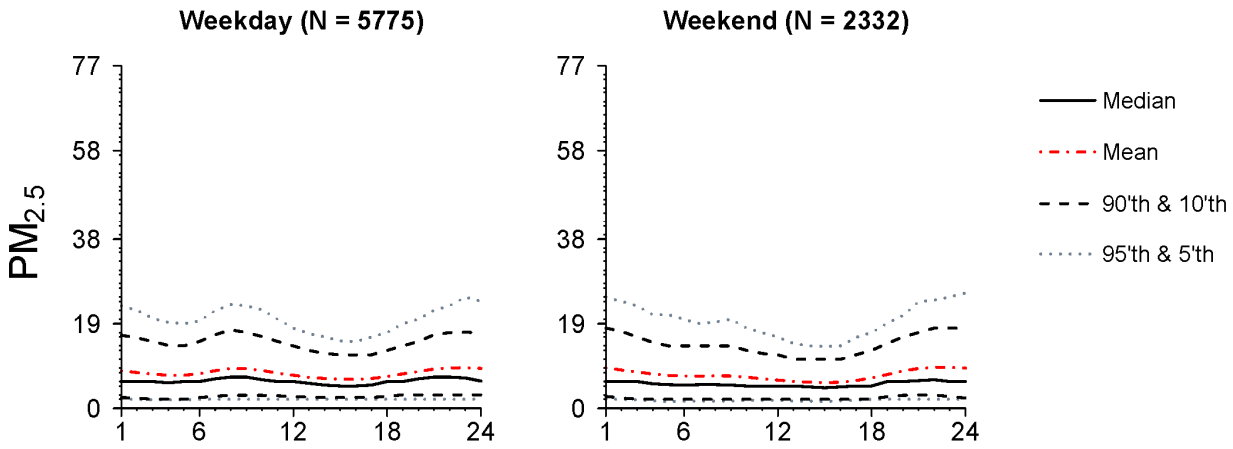


Figure A-162. Diel plots generated from all available hourly FRM-like $PM_{2.5}$ data, stratified by weekday (left) and weekend (right), in Seattle, WA. Included are the number of monitor days (N) and the median, mean, 5th, 10th, 90th and 95th percentiles for each hour.

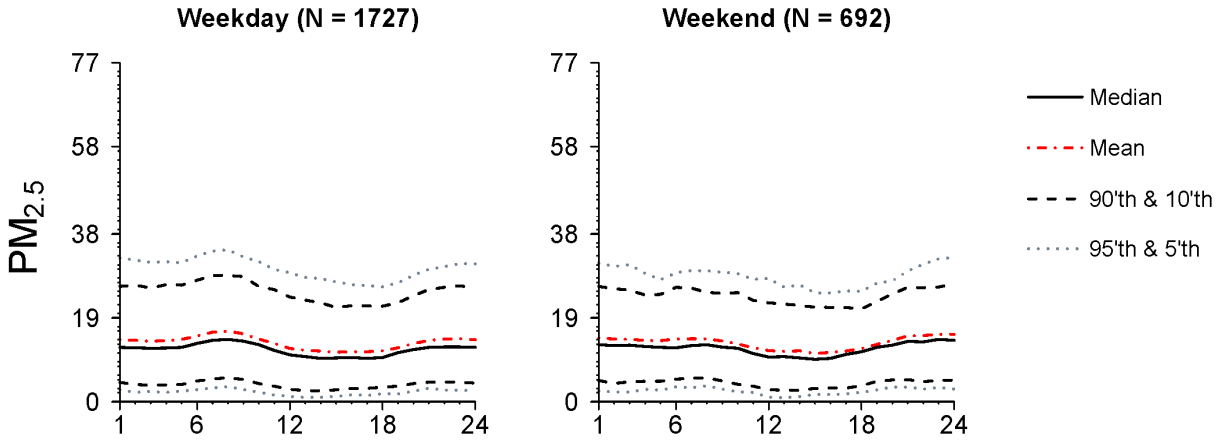


Figure A-163. Diel plots generated from all available hourly FRM-like $PM_{2.5}$ data, stratified by weekday (left) and weekend (right), in St. Louis, MO. Included are the number of monitor days (N) and the median, mean, 5th, 10th, 90th and 95th percentiles for each hour.

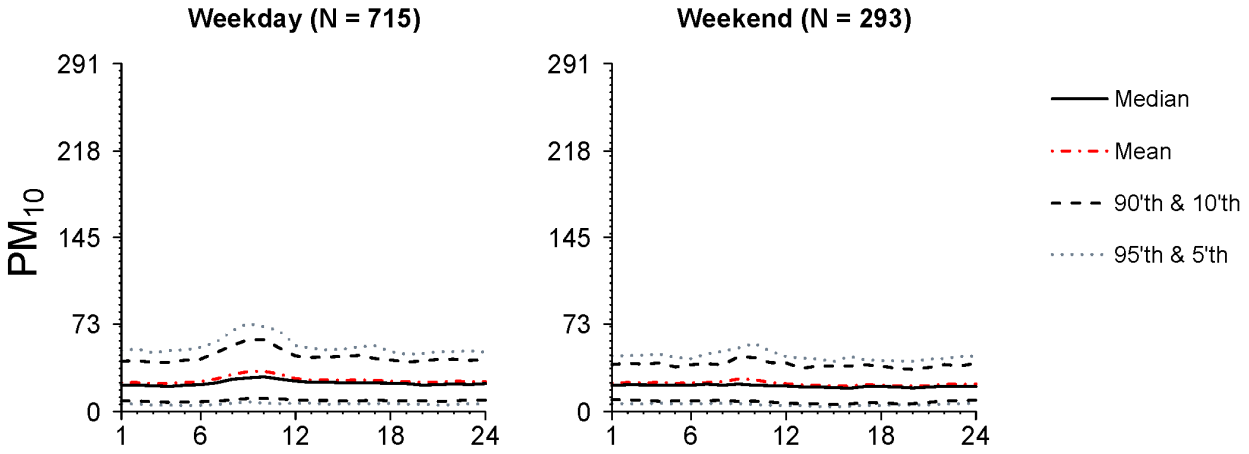


Figure A-164. Diel plot generated from all available hourly FRM/FEM PM_{10} data, stratified by weekday (left) and weekend (right), in Atlanta, GA. Included are the number of monitor days (N) and the median, mean, 5th, 10th, 90th and 95th percentiles for each hour.

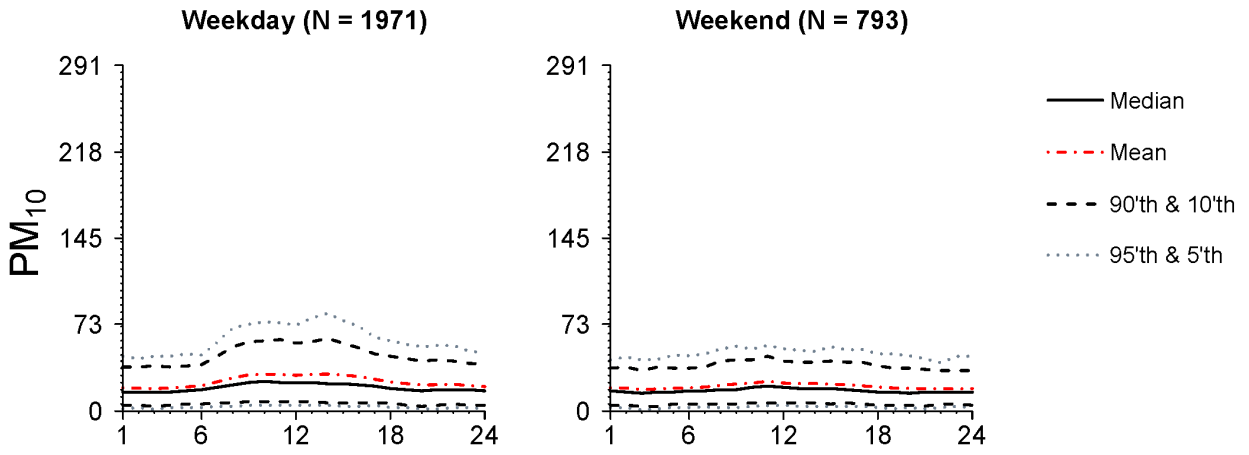


Figure A-165. Diel plot generated from all available hourly FRM/FEM PM_{10} data, stratified by weekday (left) and weekend (right), in Chicago, IL. Included are the number of monitor days (N) and the median, mean, 5th, 10th, 90th and 95th percentiles for each hour.

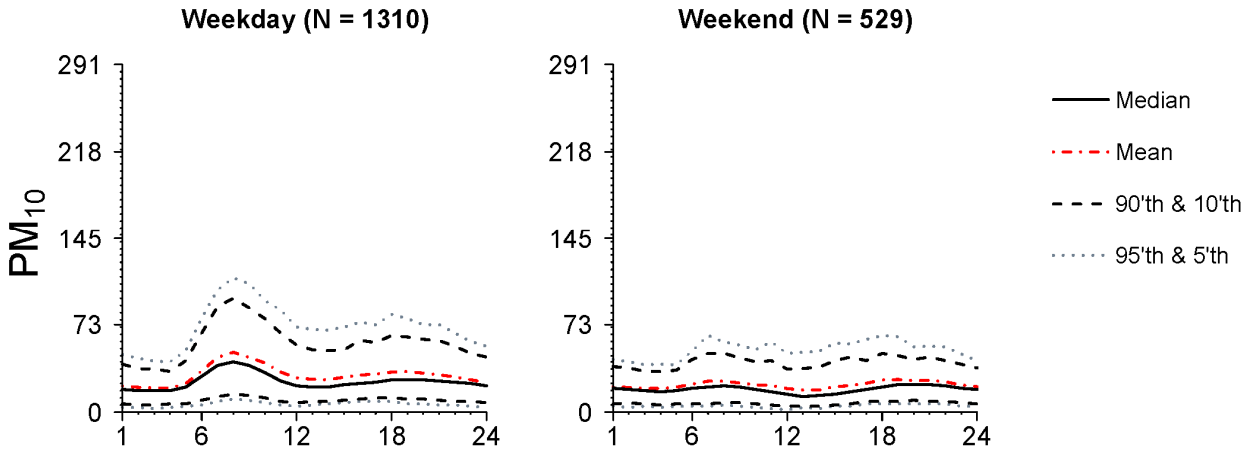


Figure A-166. Diel plot generated from all available hourly FRM/FEM PM_{10} data, stratified by weekday (left) and weekend (right), in Denver, CO. Included are the number of monitor days (N) and the median, mean, 5th, 10th, 90th and 95th percentiles for each hour.

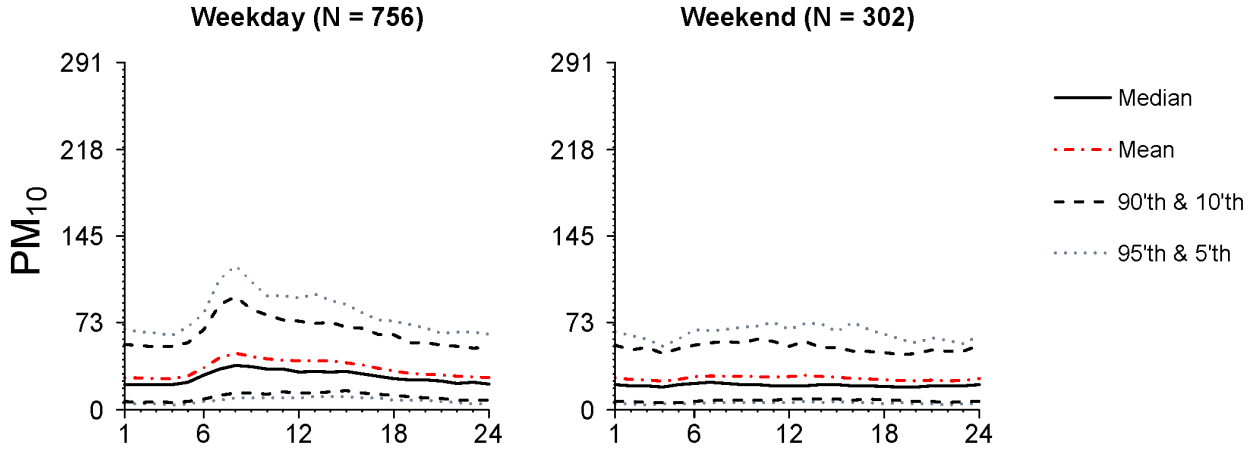


Figure A-167. Diel plot generated from all available hourly FRM/FEM PM_{10} data, stratified by weekday (left) and weekend (right), in Detroit, MI. Included are the number of monitor days (N) and the median, mean, 5th, 10th, 90th and 95th percentiles for each hour.

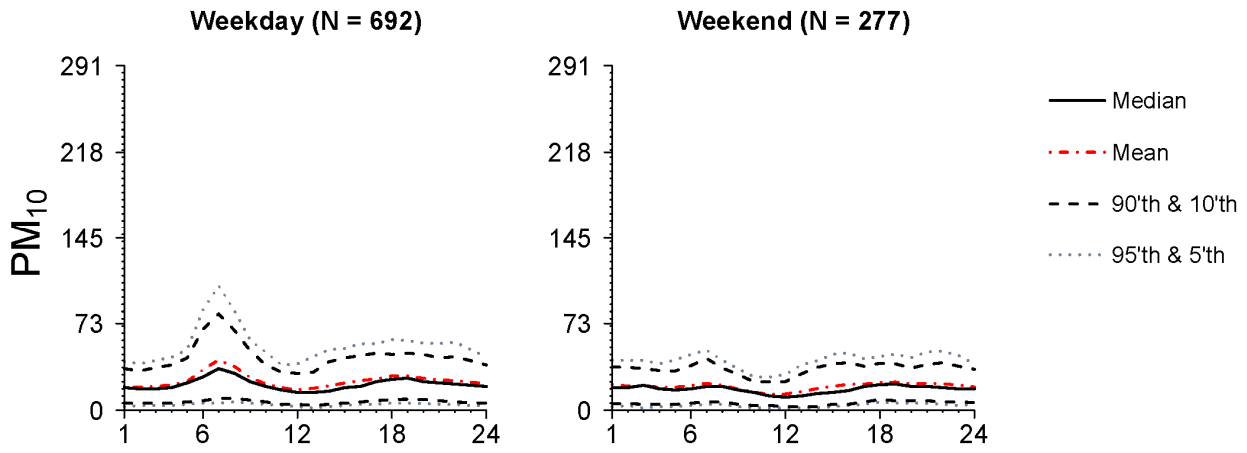


Figure A-168. Diel plot generated from all available hourly FRM/FEM PM_{10} data, stratified by weekday (left) and weekend (right), in Los Angeles, CA. Included are the number of monitor days (N) and the median, mean, 5th, 10th, 90th and 95th percentiles for each hour.

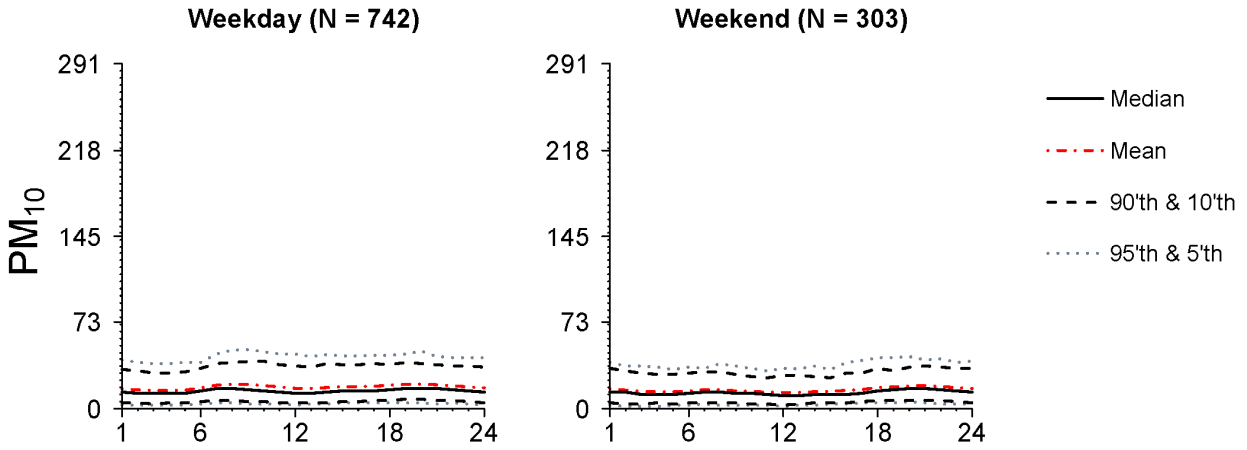


Figure A-169. Diel plot generated from all available hourly FRM/FEM PM_{10} data, stratified by weekday (left) and weekend (right), in Philadelphia, PA. Included are the number of monitor days (N) and the median, mean, 5th, 10th, 90th and 95th percentiles for each hour.

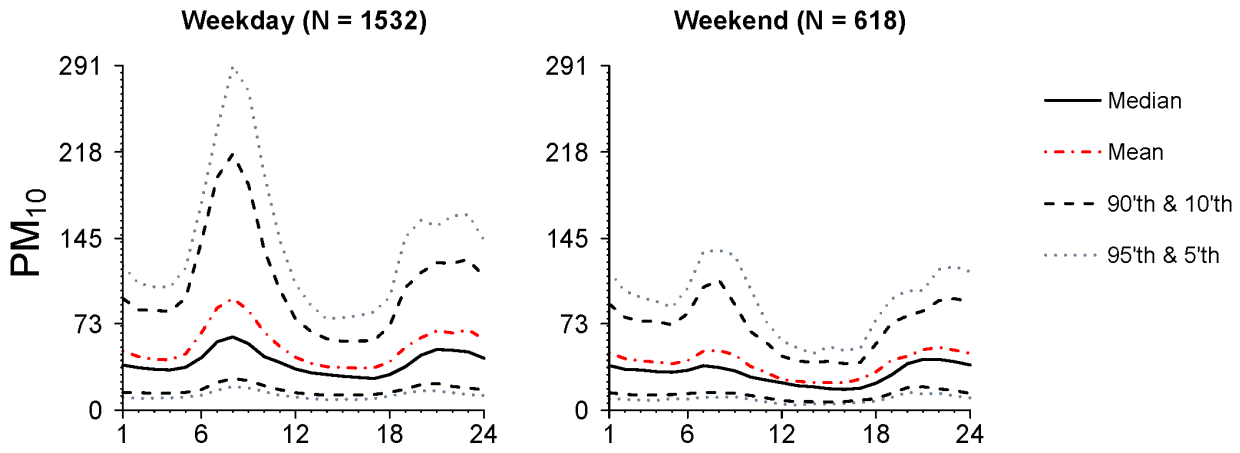


Figure A-170. Diel plot generated from all available hourly FRM/FEM PM_{10} data, stratified by weekday (left) and weekend (right), in Phoenix, AZ. Included are the number of monitor days (N) and the median, mean, 5th, 10th, 90th and 95th percentiles for each hour.

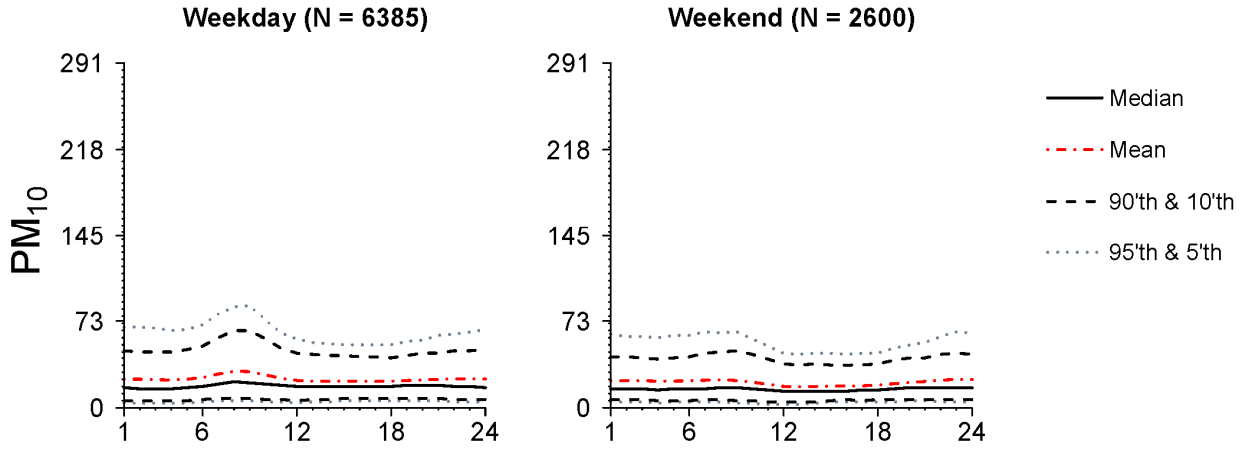


Figure A-171. Diel plot generated from all available hourly FRM/FEM PM_{10} data, stratified by weekday (left) and weekend (right), in Pittsburgh, PA. Included are the number of monitor days (N) and the median, mean, 5th, 10th, 90th and 95th percentiles for each hour.

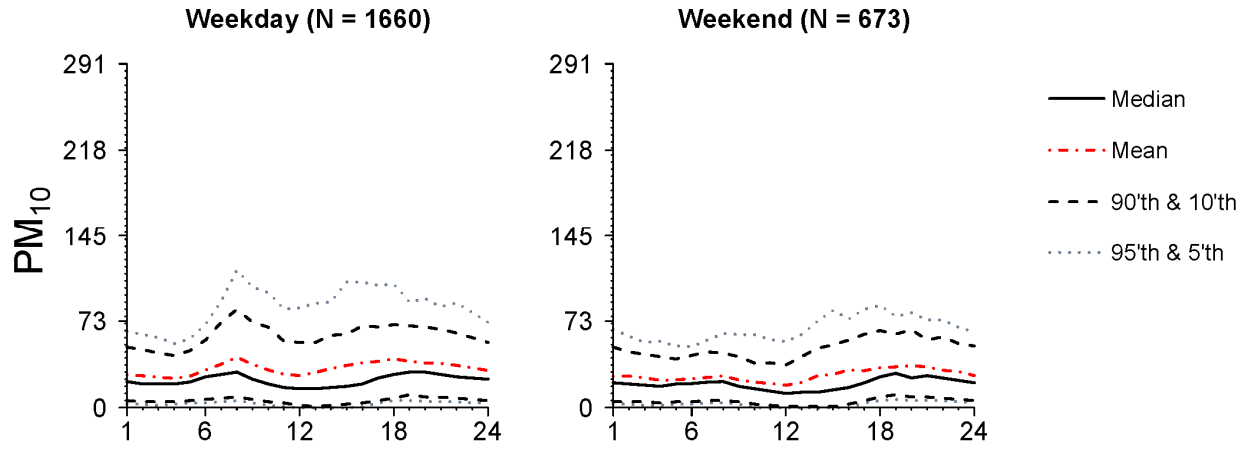


Figure A-172. Diel plot generated from all available hourly FRM/FEM PM_{10} data, stratified by weekday (left) and weekend (right), in Riverside, CA. Included are the number of monitor days (N) and the median, mean, 5th, 10th, 90th and 95th percentiles for each hour.

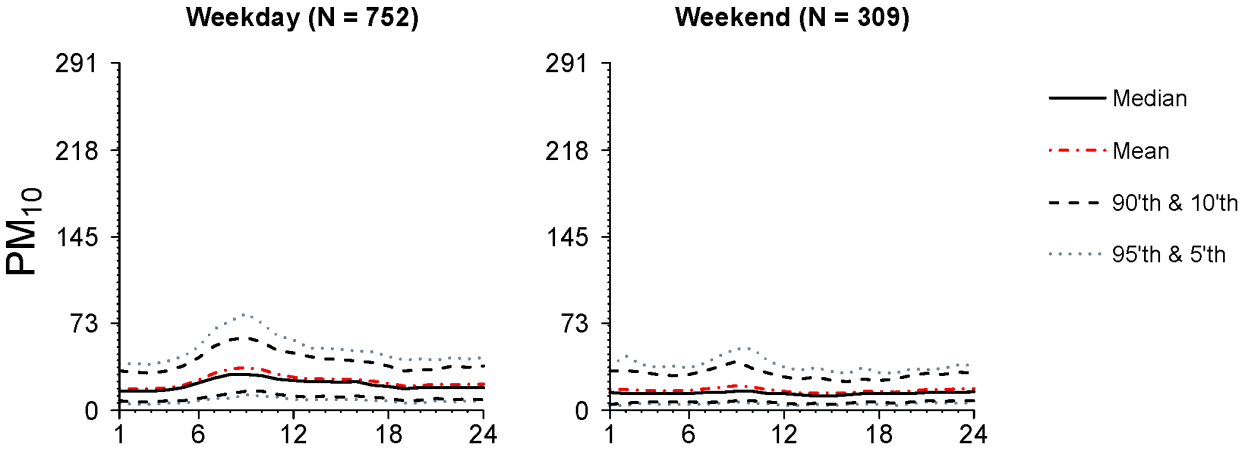


Figure A-173. Diel plot generated from all available hourly FRM/FEM PM_{10} data, stratified by weekday (left) and weekend (right), in Seattle, WA. Included are the number of monitor days (N) and the median, mean, 5th, 10th, 90th and 95th percentiles for each hour.

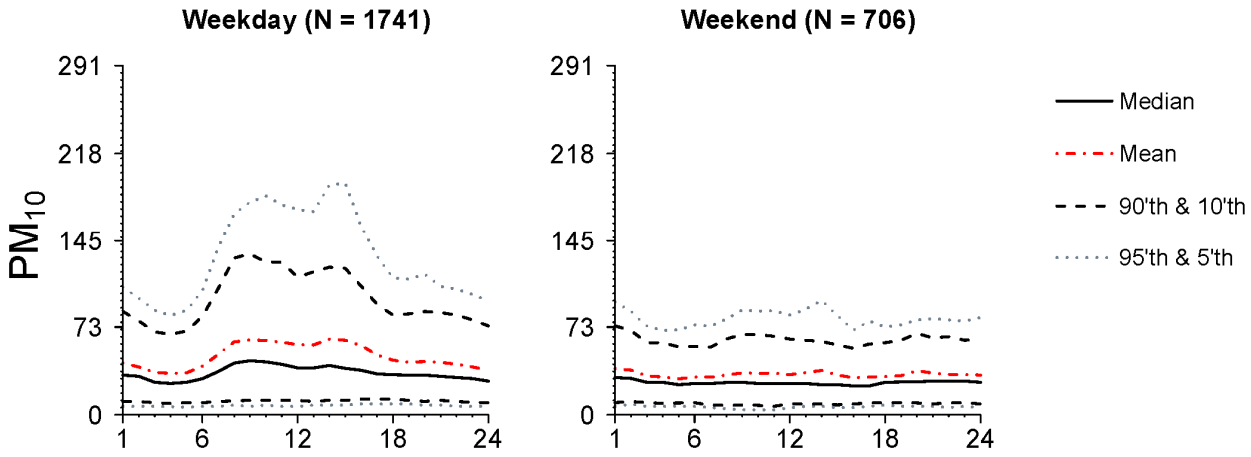


Figure A-174. Diel plot generated from all available hourly FRM/FEM PM_{10} data, stratified by weekday (left) and weekend (right), in St. Louis, MO. Included are the number of monitor days (N) and the median, mean, 5th, 10th, 90th and 95th percentiles for each hour.

A.2.5. Copollutant Measurements

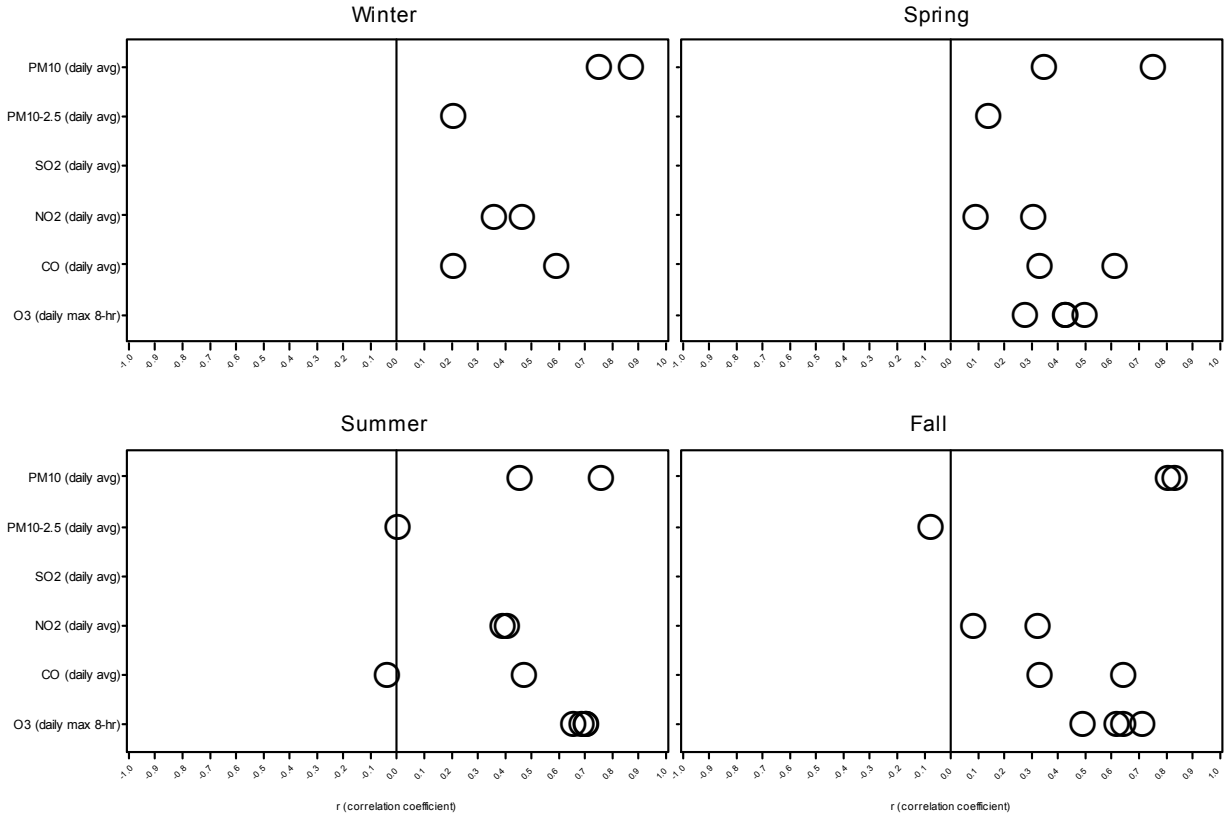


Figure A-175. Correlations between 24-h $PM_{2.5}$ and co-located 24-h avg PM_{10}, $PM_{10-2.5}$, SO_2, NO_2 and CO and daily maximum 8-h avg O_3 for Atlanta, GA, stratified by season (2005-2007). One point is included for each available monitor pair.

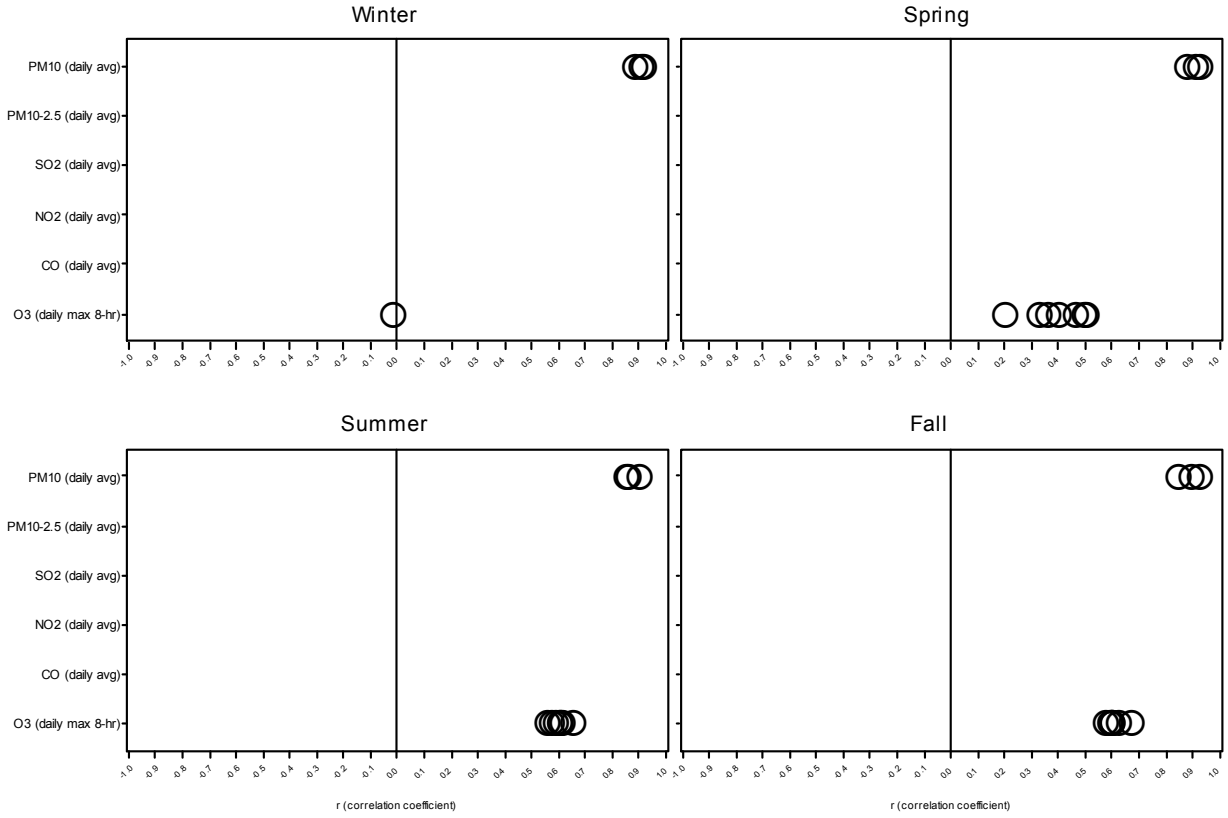


Figure A-176. Correlations between 24-h $PM_{2.5}$ and co-located 24-h avg PM_{10}, $PM_{10-2.5}$, SO_2, NO_2 and CO and daily maximum 8-h avg O_3 for Birmingham, AL, stratified by season (2005-2007). One point is included for each available monitor pair.

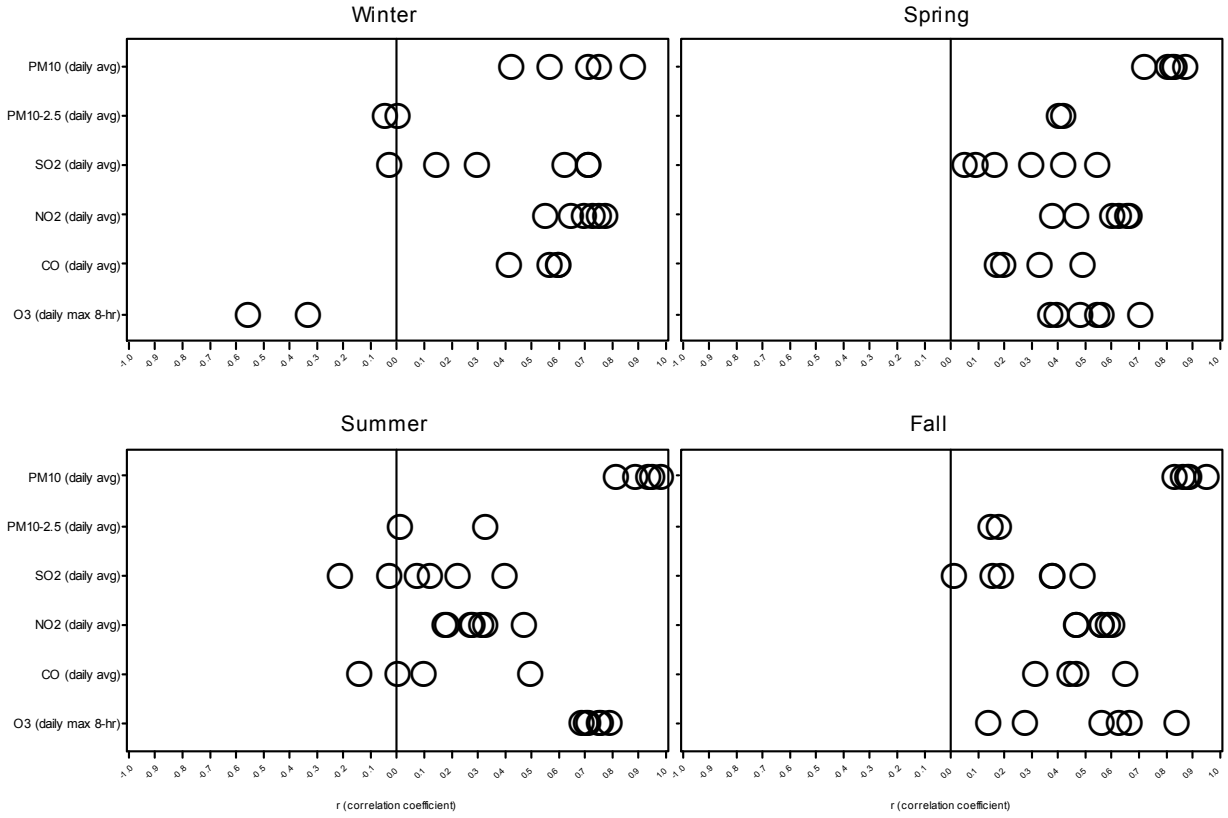


Figure A-177. Correlations between 24-h $PM_{2.5}$ and co-located 24-h avg PM_{10}, $PM_{10-2.5}$, SO_2, NO_2 and CO and daily maximum 8-h avg O_3 for Boston, MA, stratified by season (2005-2007). One point is included for each available monitor pair.

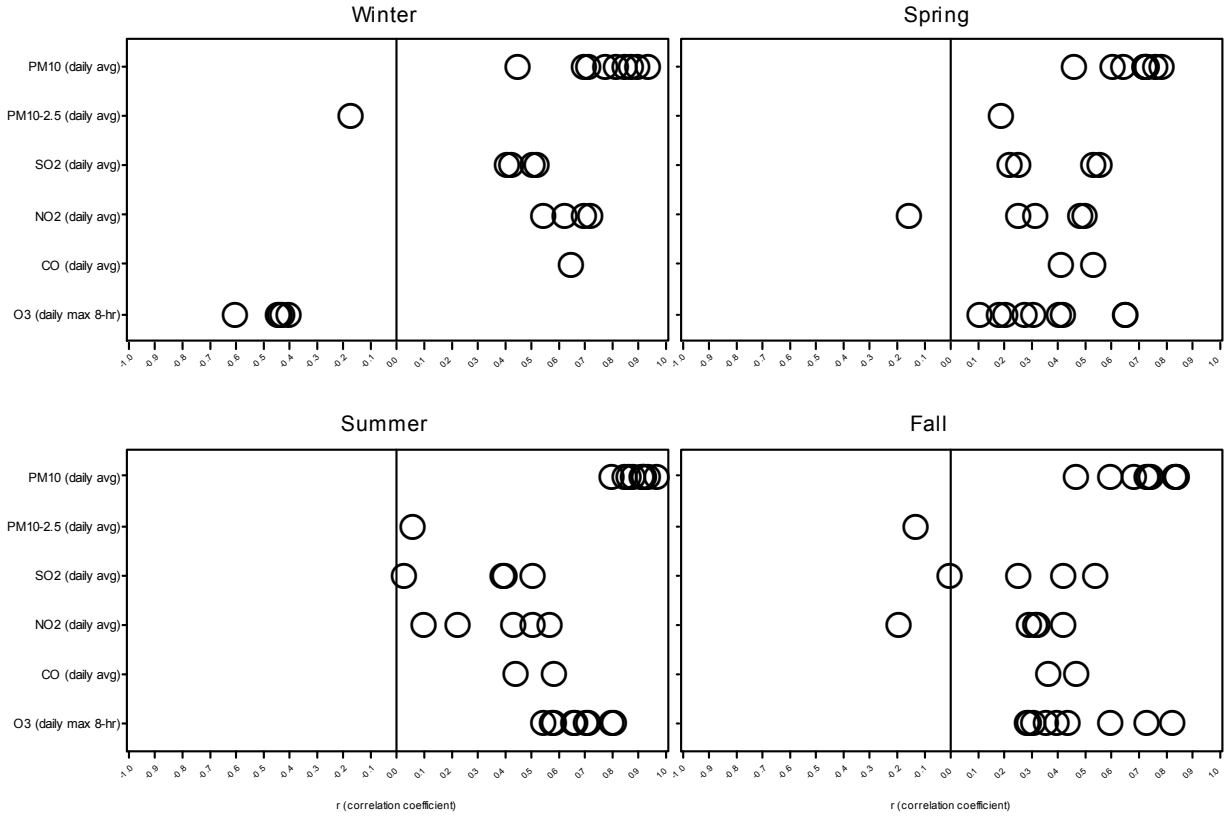


Figure A-178. Correlations between 24-h $PM_{2.5}$ and co-located 24-h avg PM_{10}, $PM_{10-2.5}$, SO_2, NO_2 and CO and daily maximum 8-h avg O_3 for Chicago, IL, stratified by season (2005-2007). One point is included for each available monitor pair.

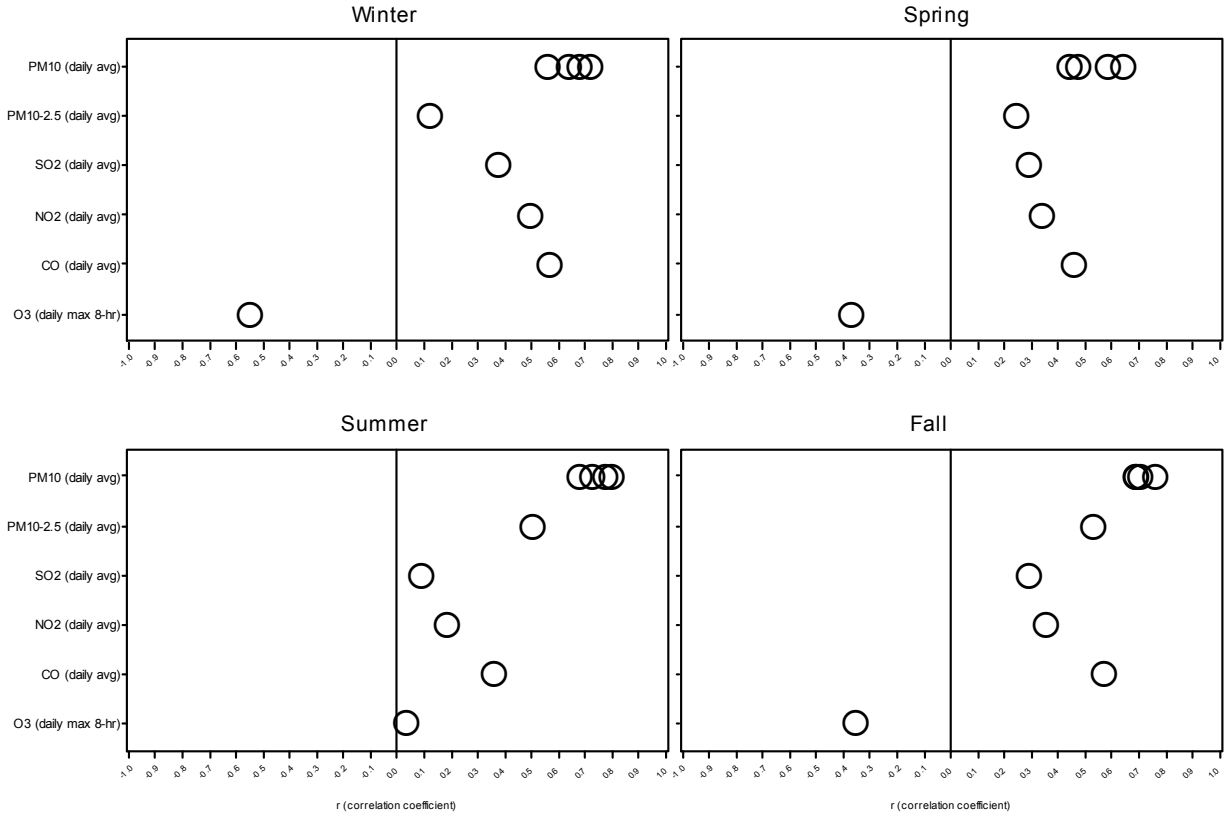


Figure A-179. Correlations between 24-h $PM_{2.5}$ and co-located 24-h avg PM_{10}, $PM_{10-2.5}$, SO_2, NO_2 and CO and daily maximum 8-h avg O_3 for Denver, CO, stratified by season (2005-2007). One point is included for each available monitor pair.

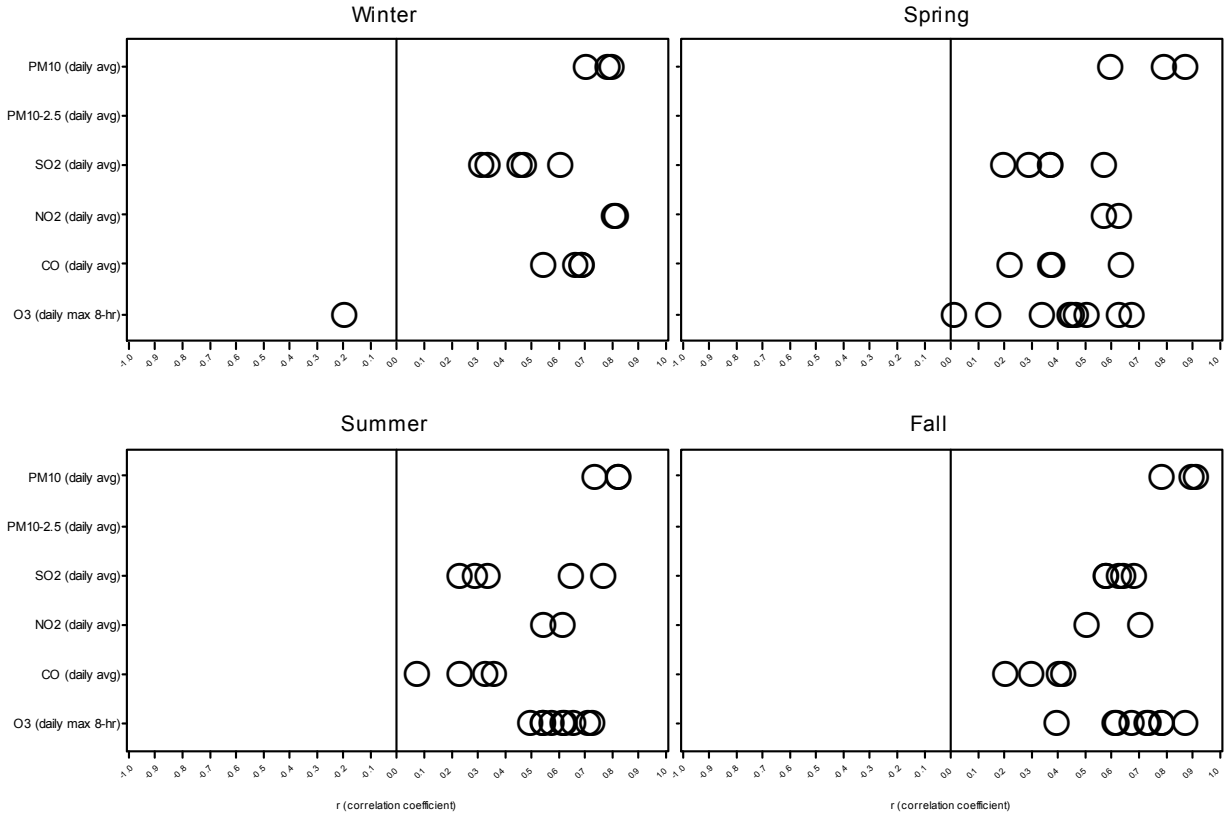


Figure A-180. Correlations between 24-h $PM_{2.5}$ and co-located 24-h avg PM_{10}, $PM_{10-2.5}$, SO_2, NO_2 and CO and daily maximum 8-h avg O_3 for Detroit, MI, stratified by season (2005-2007). One point is included for each available monitor pair.

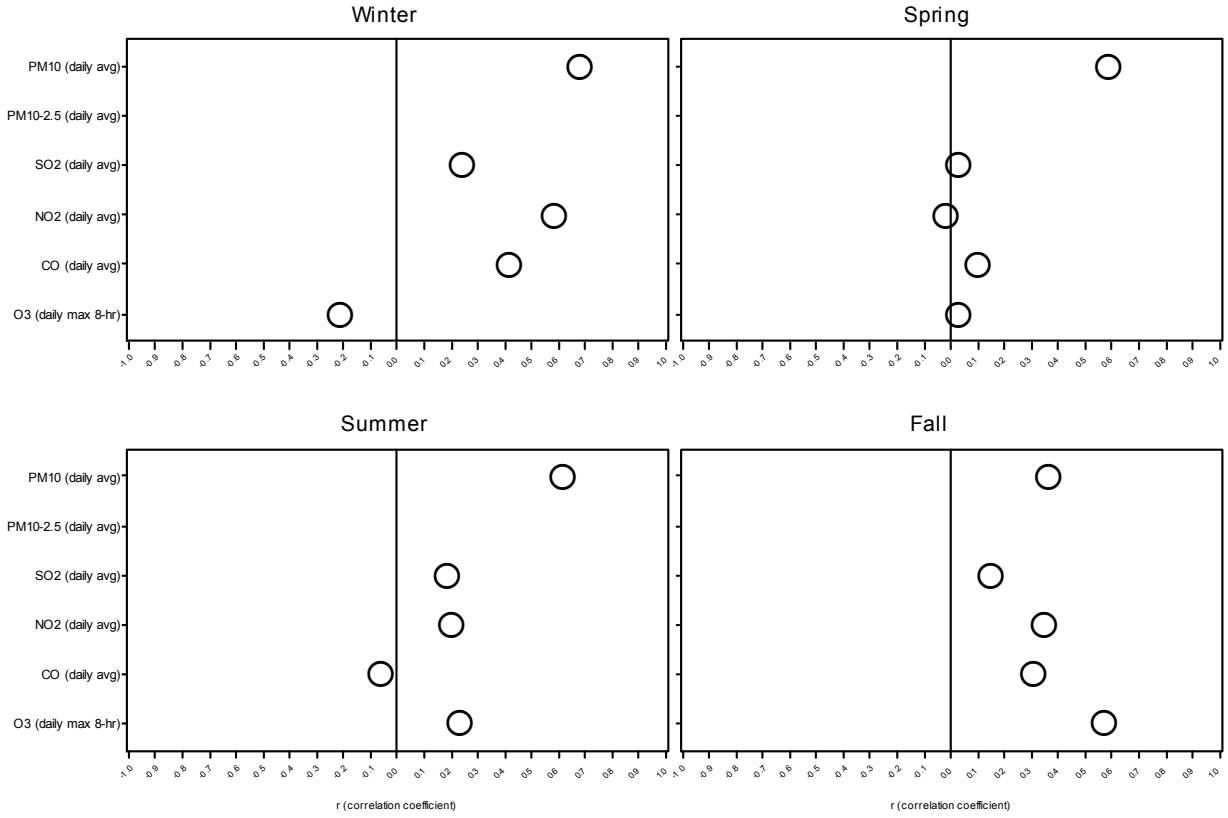


Figure A-181. Correlations between 24-h $PM_{2.5}$ and co-located 24-h avg PM_{10}, $PM_{10-2.5}$, SO_2, NO_2 and CO and daily maximum 8-h avg O_3 for Houston, TX, stratified by season (2005-2007). One point is included for each available monitor pair.

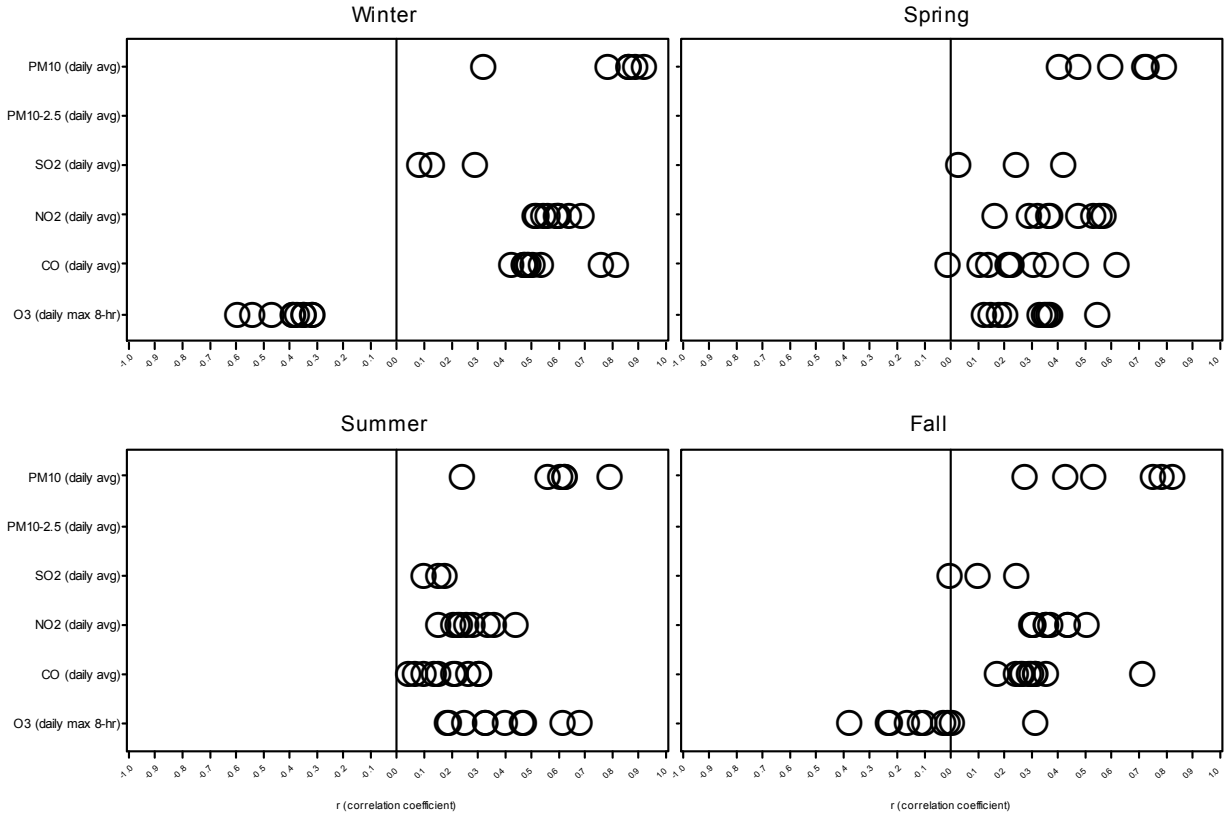


Figure A-182. Correlations between 24-h $PM_{2.5}$ and co-located 24-h avg PM_{10}, $PM_{10-2.5}$, SO_2, NO_2 and CO and daily maximum 8-h avg O_3 for Los Angeles, CA, stratified by season (2005-2007). One point is included for each available monitor pair.

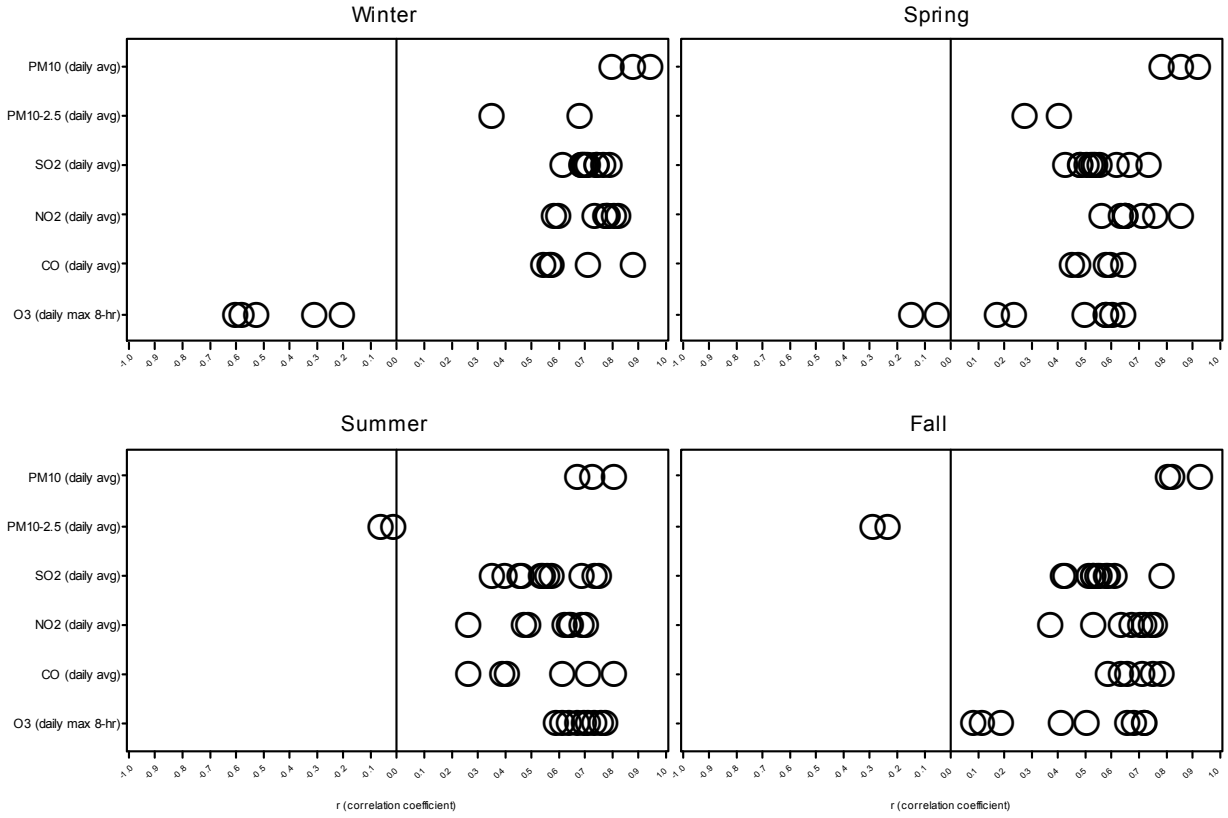


Figure A-183. Correlations between 24-h $PM_{2.5}$ and co-located 24-h avg PM_{10}, $PM_{10-2.5}$, SO_2, NO_2 and CO and daily maximum 8-h avg O_3 for New York, NY, stratified by season (2005-2007). One point is included for each available monitor pair.

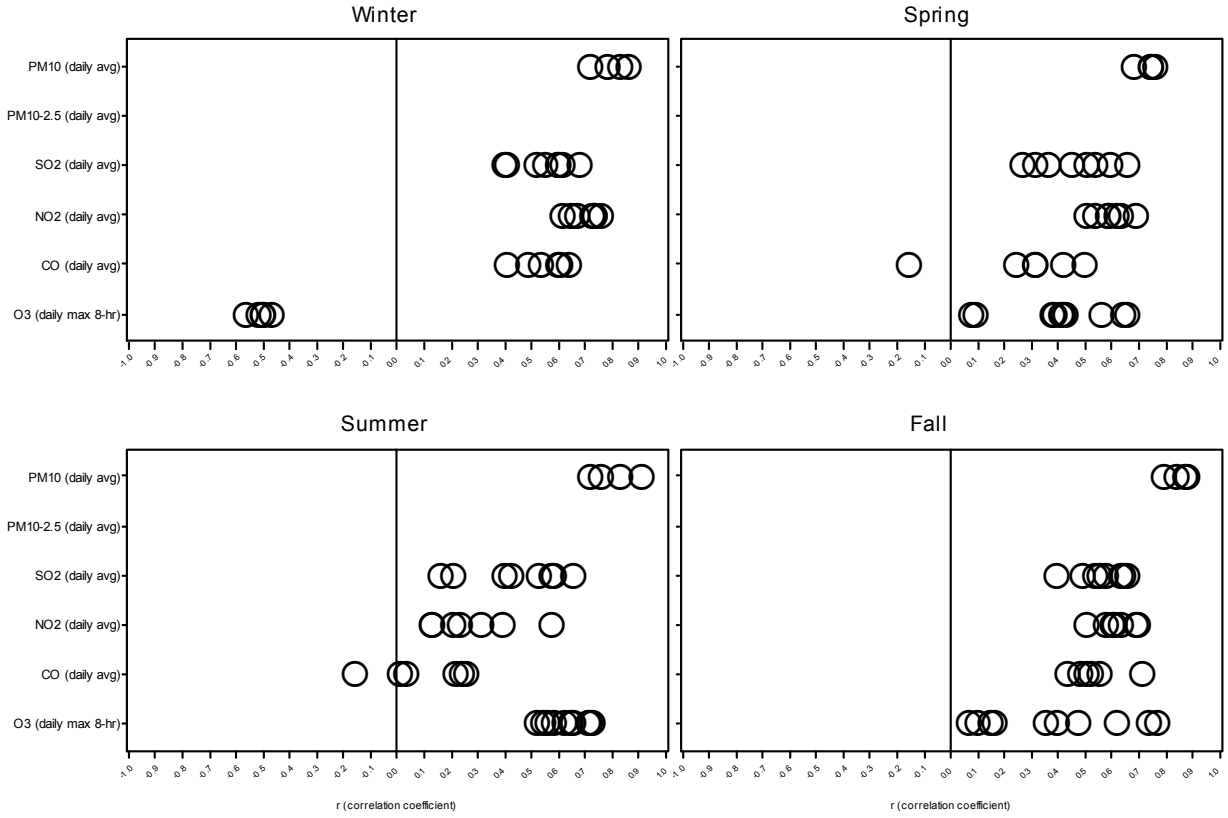


Figure A-184. Correlations between 24-h $PM_{2.5}$ and co-located 24-h avg PM_{10}, $PM_{10-2.5}$, SO_2, NO_2 and CO and daily maximum 8-h avg O_3 for Philadelphia, PA, stratified by season (2005-2007). One point is included for each available monitor pair.

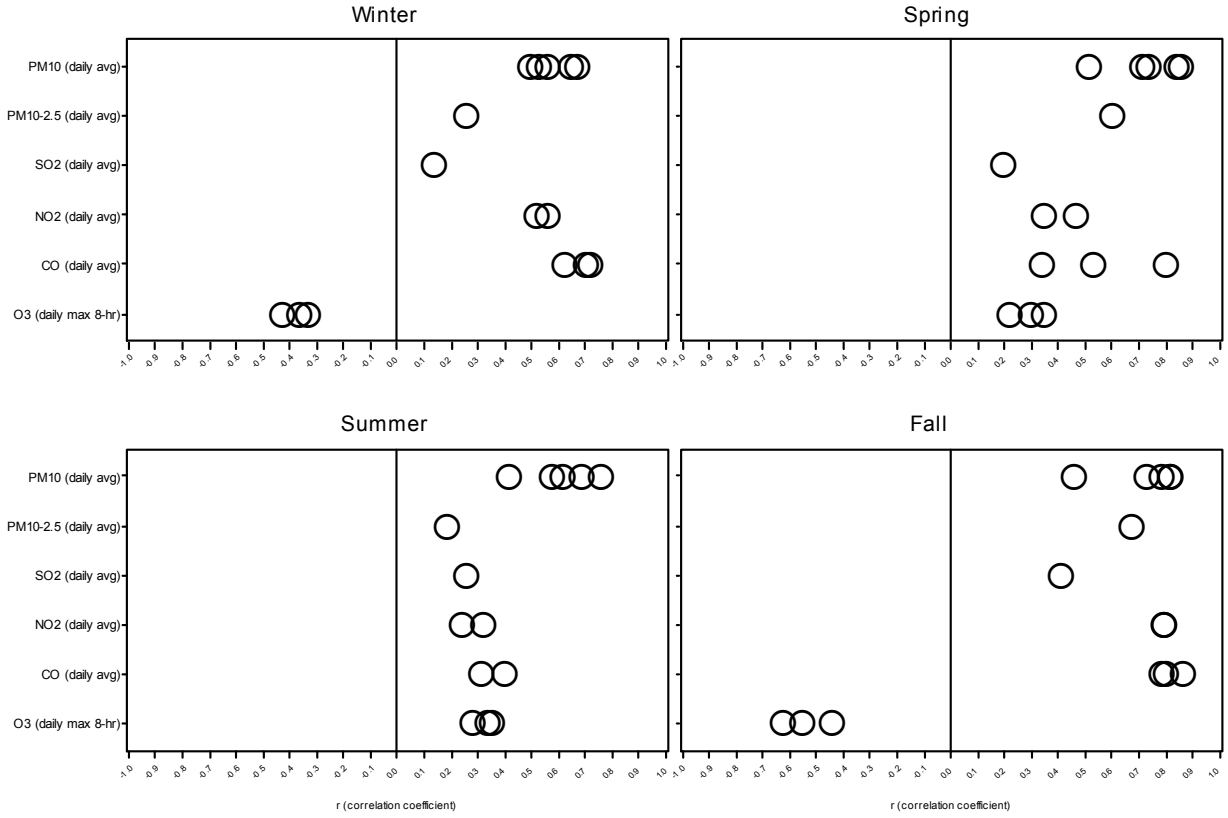


Figure A-185. Correlations between 24-h $PM_{2.5}$ and co-located 24-h avg PM_{10}, $PM_{10-2.5}$, SO_2, NO_2 and CO and daily maximum 8-h avg O_3 for Phoenix, AZ, stratified by season (2005-2007). One point is included for each available monitor pair.

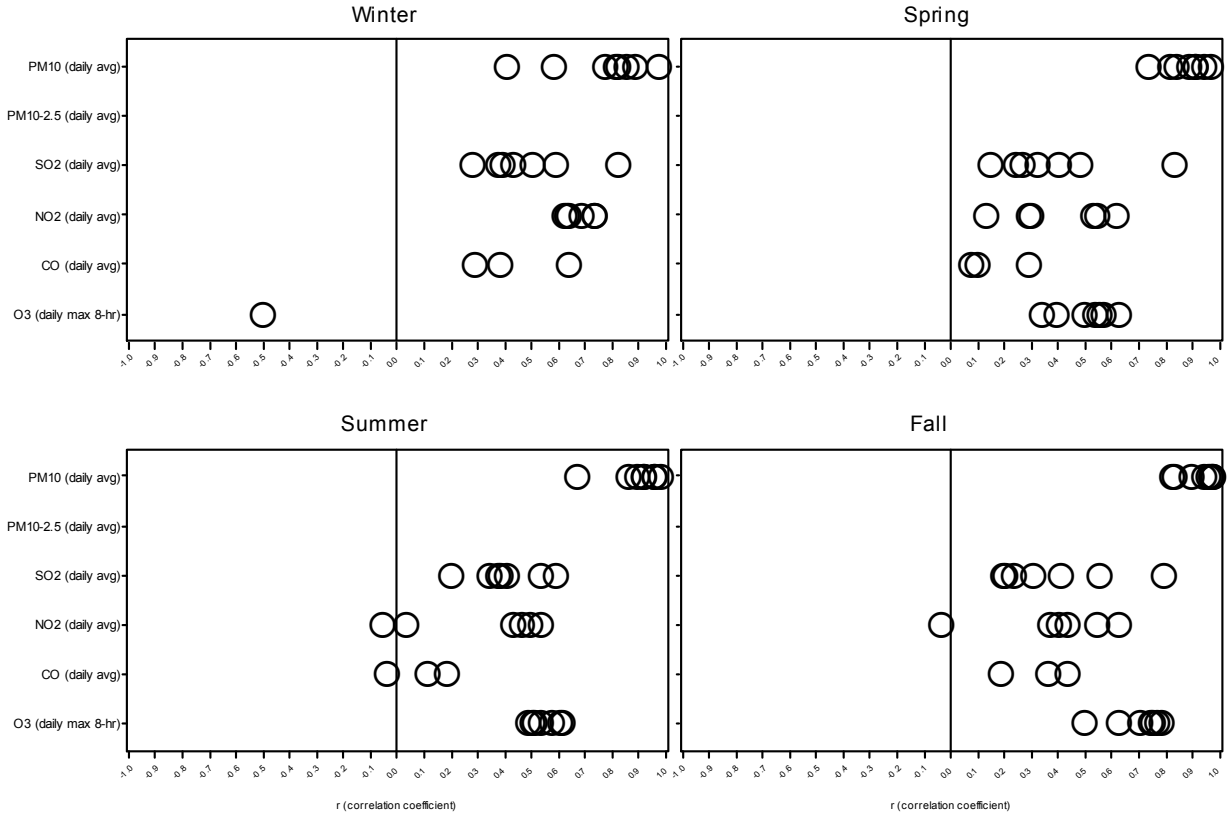


Figure A-186. Correlations between 24-h $PM_{2.5}$ and co-located 24-h avg PM_{10}, $PM_{10-2.5}$, SO_2, NO_2 and CO and daily maximum 8-h avg O_3 for Pittsburgh, PA, stratified by season (2005-2007). One point is included for each available monitor pair.

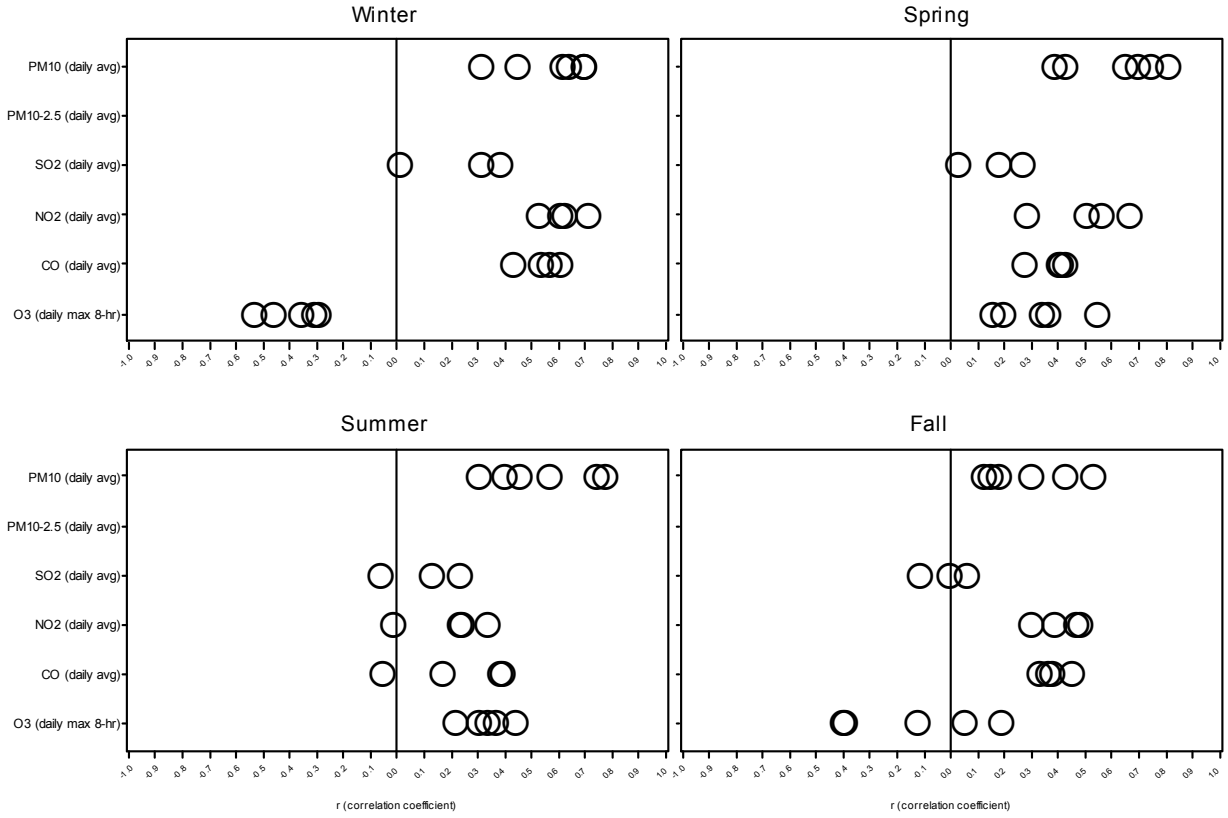


Figure A-187. Correlations between 24-h $PM_{2.5}$ and co-located 24-h avg PM_{10}, $PM_{10-2.5}$, SO_2, NO_2 and CO and daily maximum 8-h avg O_3 for Riverside, CA, stratified by season (2005-2007). One point is included for each available monitor pair.

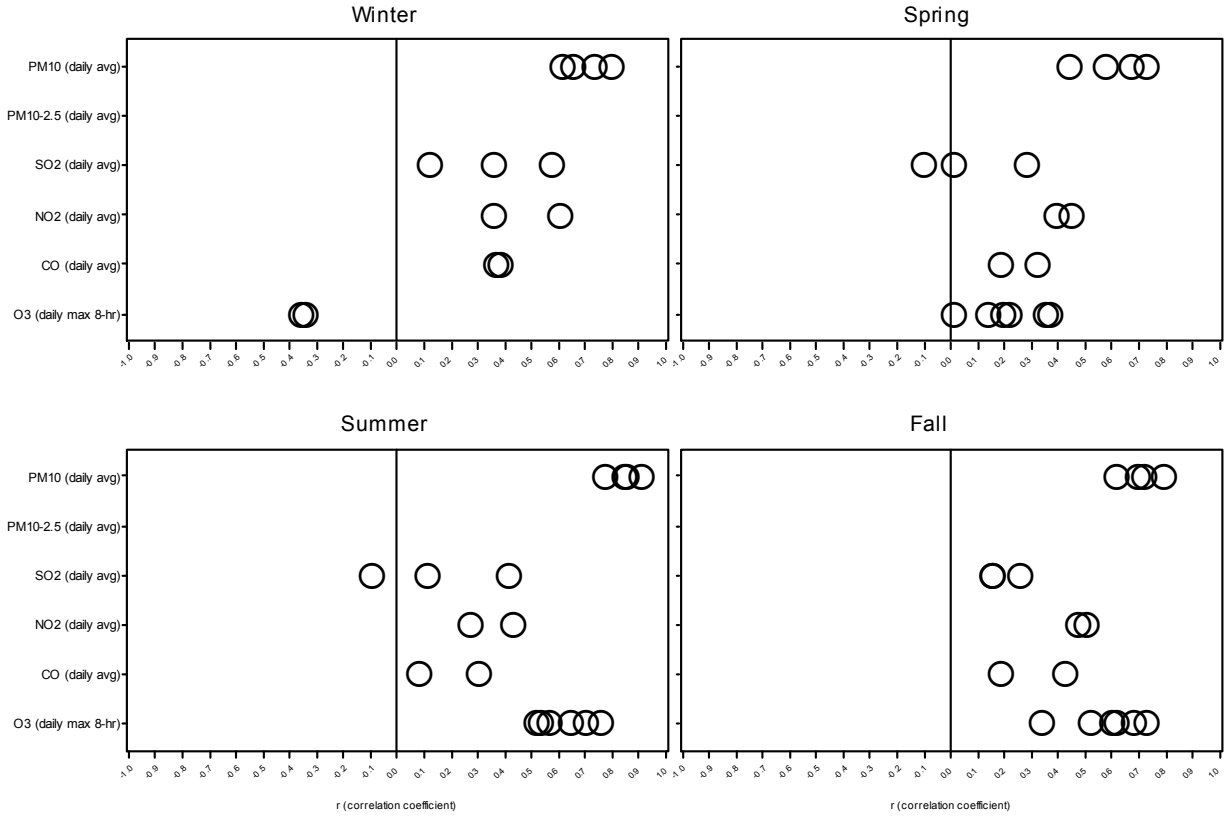


Figure A-188. Correlations between 24-h $PM_{2.5}$ and co-located 24-h avg PM_{10}, $PM_{10-2.5}$, SO_2, NO_2 and CO and daily maximum 8-h avg O_3 for St. Louis, MO, stratified by season (2005-2007). One point is included for each available monitor pair.

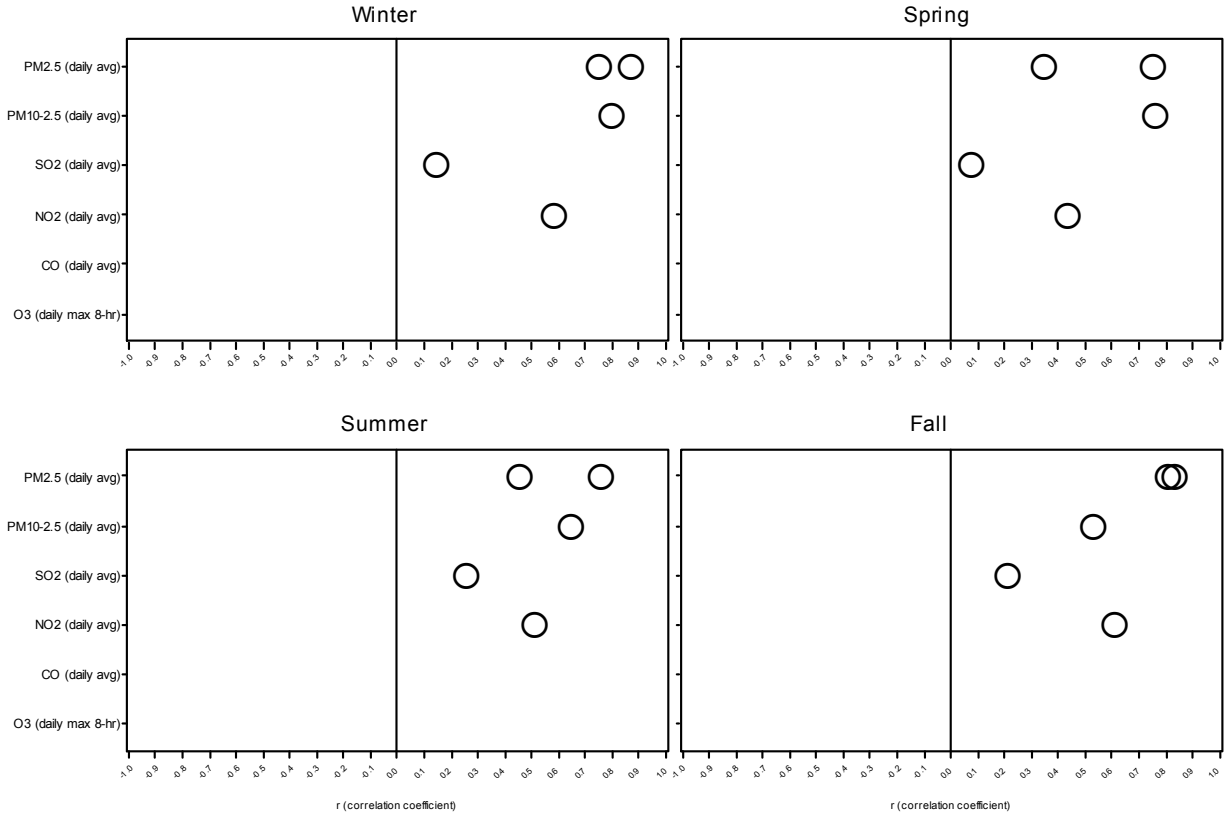


Figure A-189. Correlations between 24-h PM_{10} and co-located 24-h avg $PM_{2.5}$, $PM_{10-2.5}$, SO_2, NO_2 and CO and daily maximum 8-h avg O_3 for Atlanta, GA, stratified by season (2005-2007). One point is included for each available monitor pair.

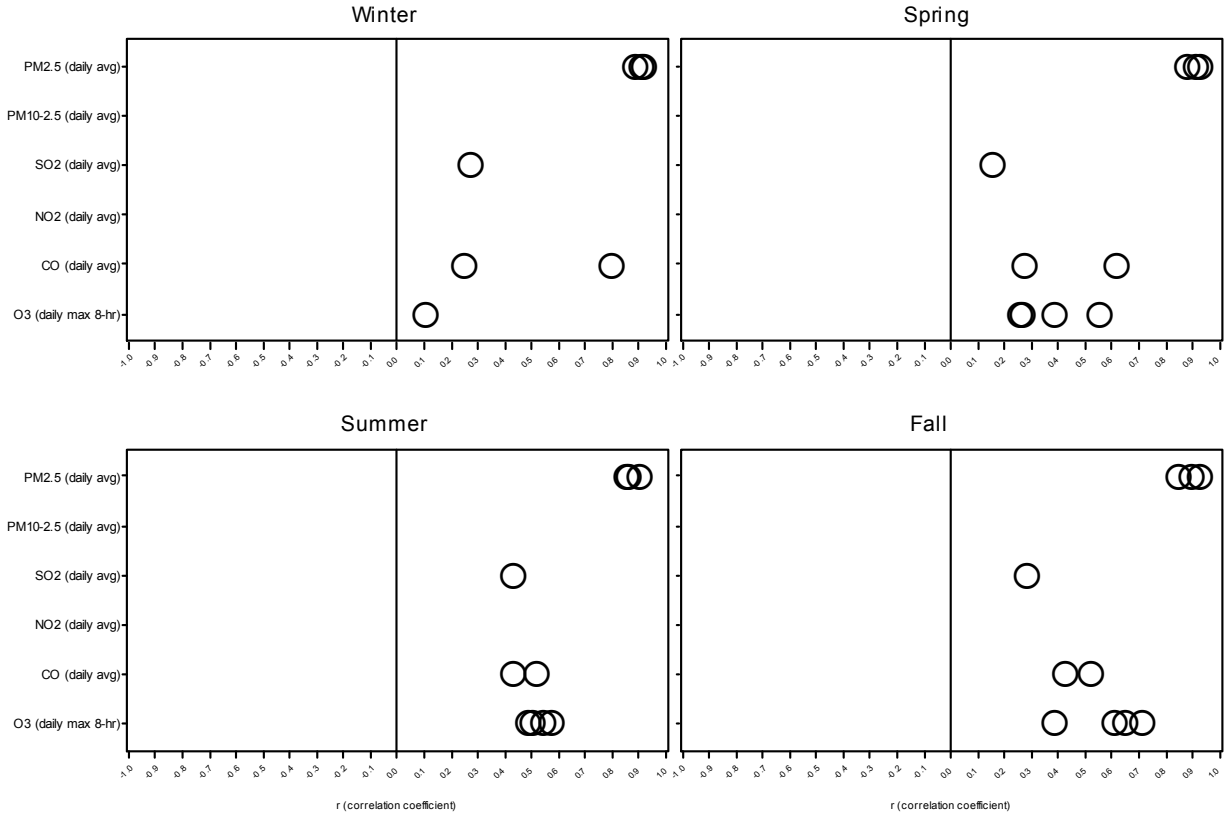


Figure A-190. Correlations between 24-h PM_{10} and co-located 24-h avg $PM_{2.5}$, $PM_{10-2.5}$, SO_2, NO_2 and CO and daily maximum 8-h avg O_3 for Birmingham, AL, stratified by season (2005-2007). One point is included for each available monitor pair.

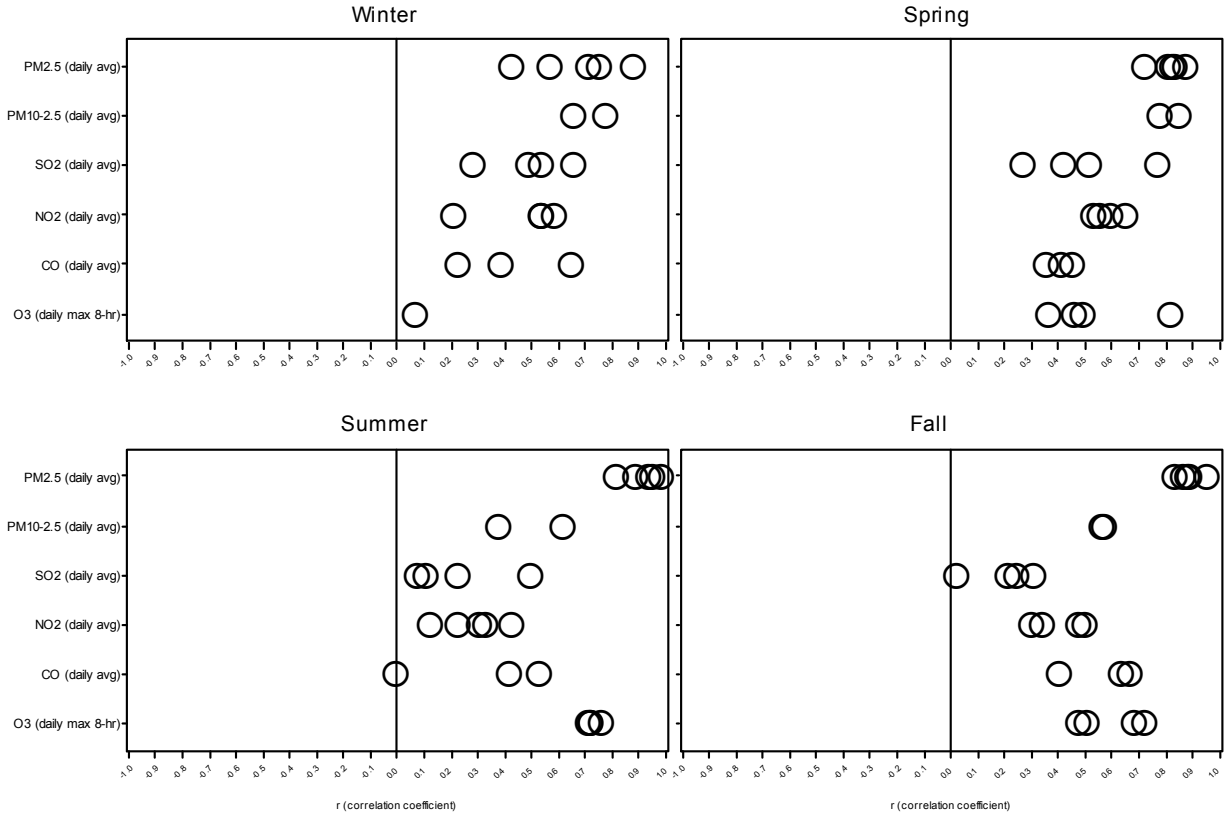


Figure A-191. Correlations between 24-h PM_{10} and co-located 24-h avg $PM_{2.5}$, $PM_{10-2.5}$, SO_2, NO_2 and CO and daily maximum 8-h avg O_3 for Boston, MA, stratified by season (2005-2007). One point is included for each available monitor pair.

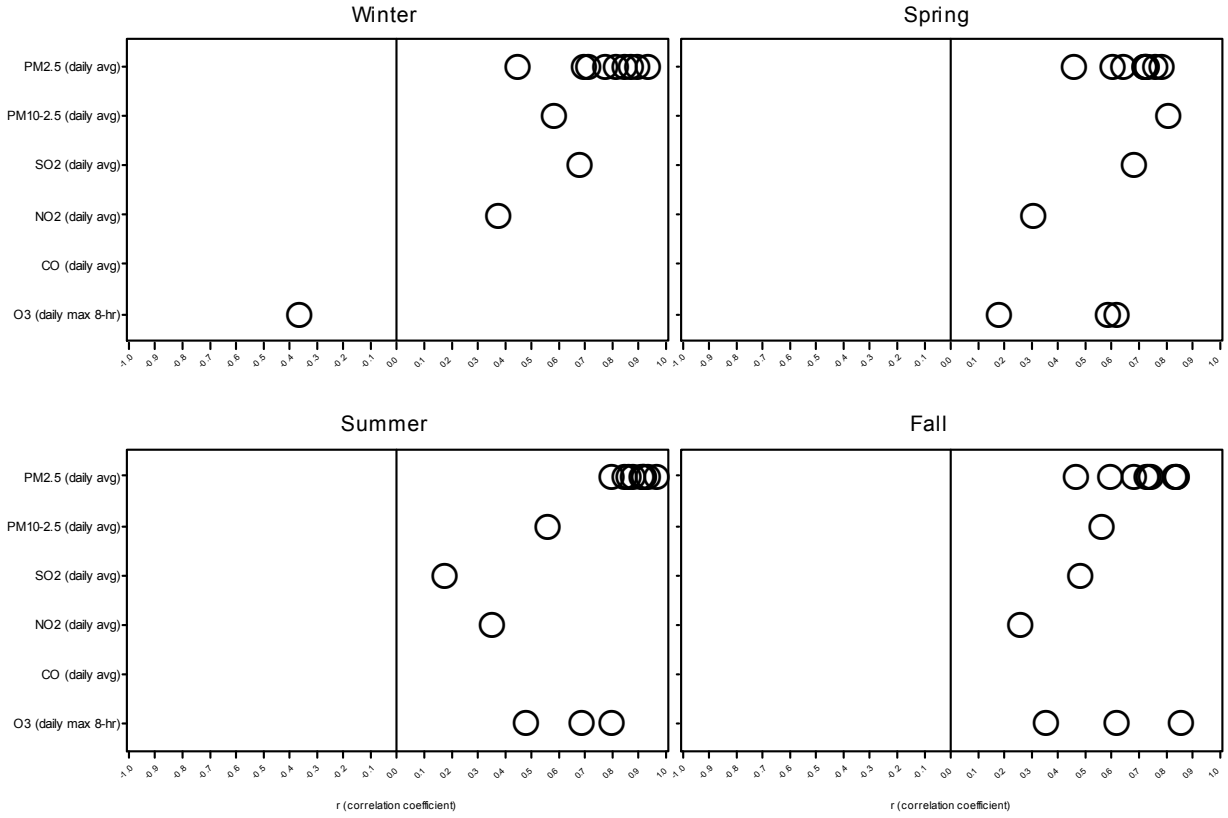


Figure A-192. Correlations between 24-h PM_{10} and co-located 24-h avg $PM_{2.5}$, $PM_{10-2.5}$, SO_2, NO_2 and CO and daily maximum 8-h avg O_3 for Chicago, IL, stratified by season (2005-2007). One point is included for each available monitor pair.

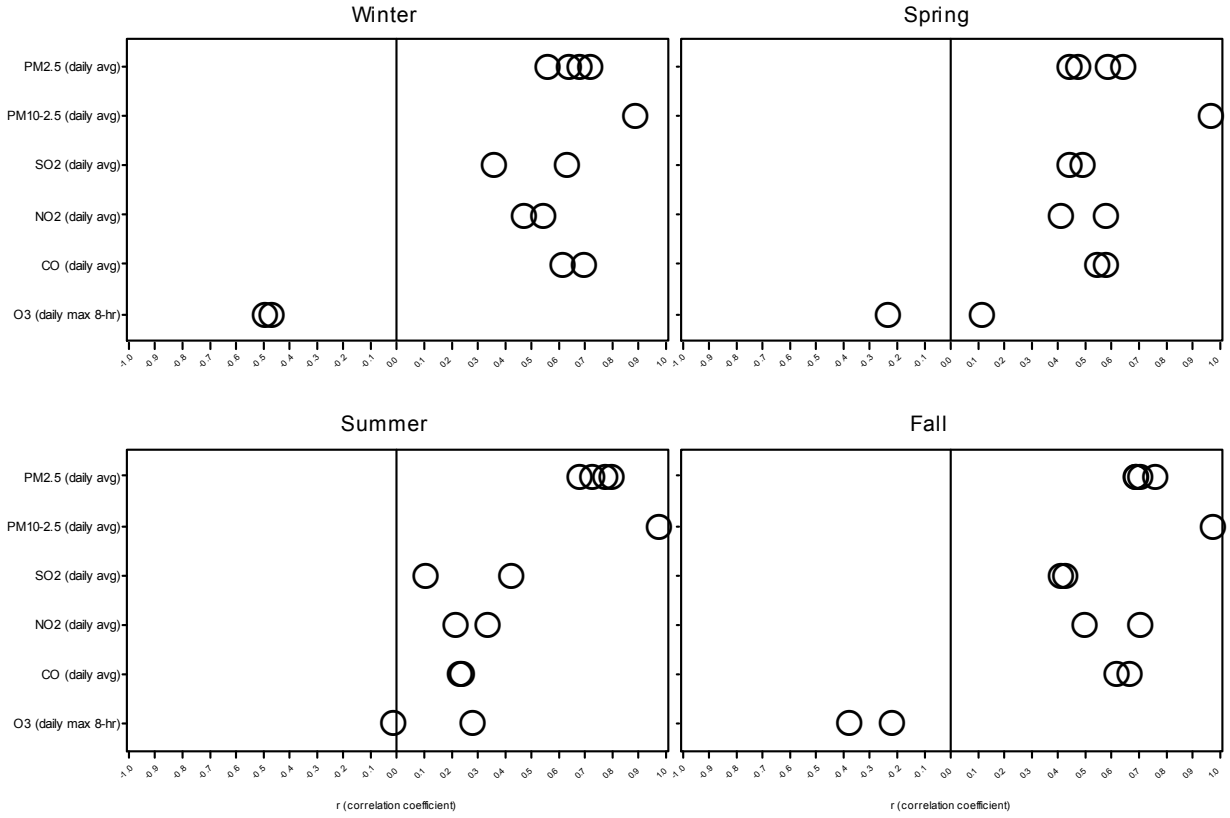


Figure A-193. Correlations between 24-h PM_{10} and co-located 24-h avg $PM_{2.5}$, $PM_{10-2.5}$, SO_2, NO_2 and CO and daily maximum 8-h avg O_3 for Denver, CO, stratified by season (2005-2007). One point is included for each available monitor pair.

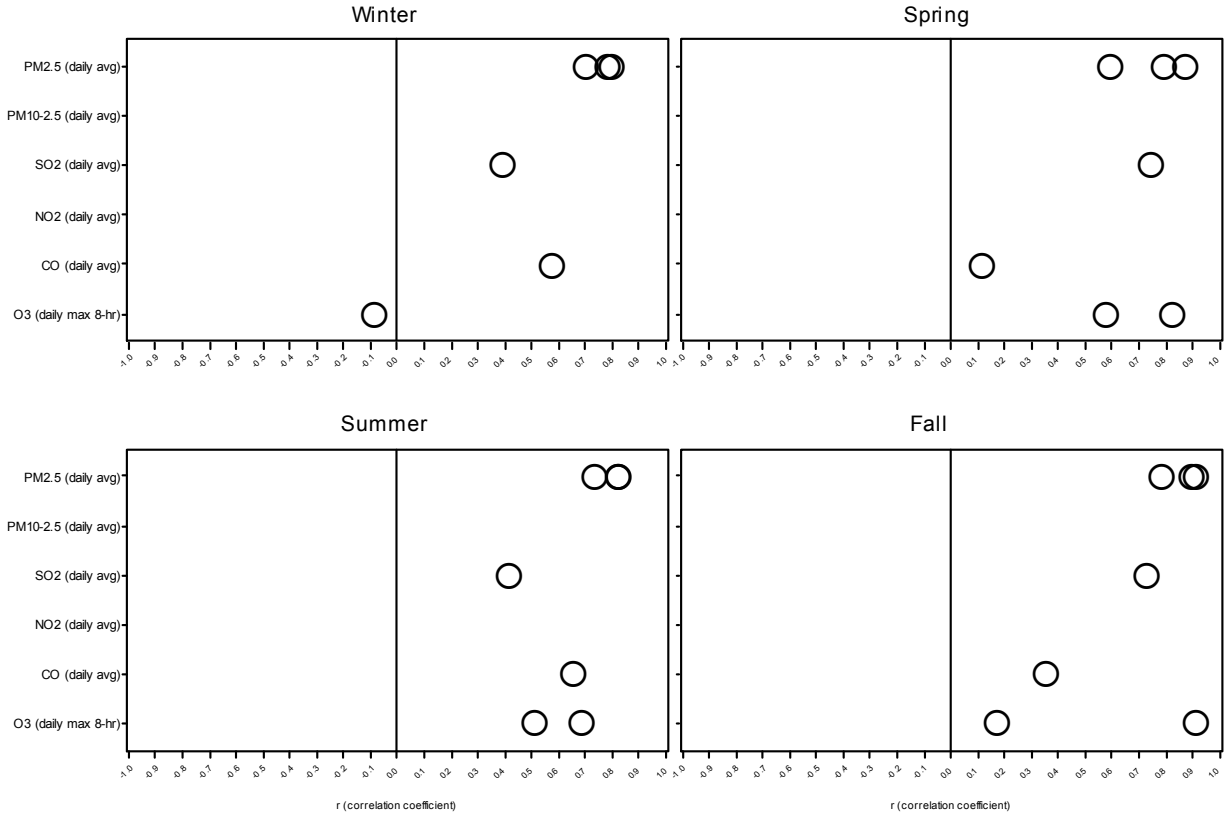


Figure A-194. Correlations between 24-h PM_{10} and co-located 24-h avg $PM_{2.5}$, $PM_{10-2.5}$, SO_2, NO_2 and CO and daily maximum 8-h avg O_3 for Detroit, MI, stratified by season (2005-2007). One point is included for each available monitor pair.

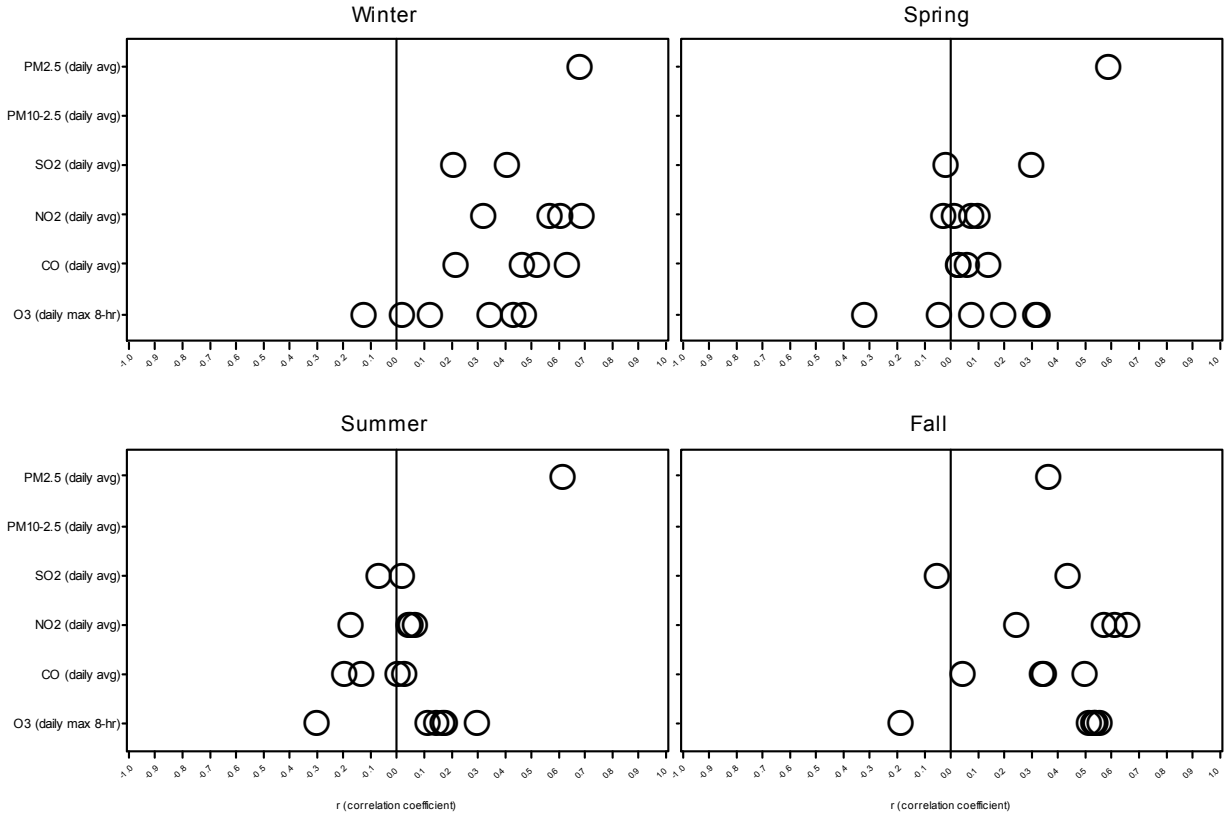


Figure A-195. Correlations between 24-h PM_{10} and co-located 24-h avg $PM_{2.5}$, $PM_{10-2.5}$, SO_2, NO_2 and CO and daily maximum 8-h avg O_3 for Houston, TX, stratified by season (2005-2007). One point is included for each available monitor pair.

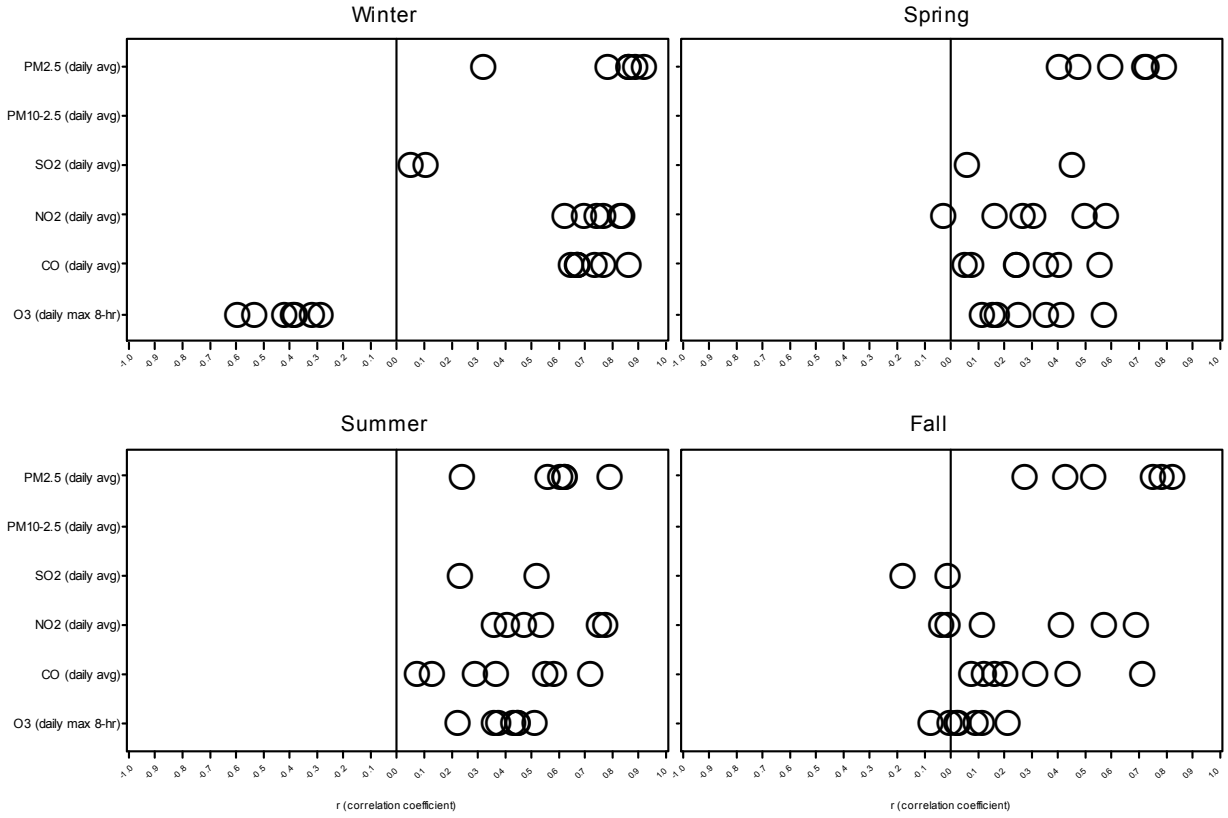


Figure A-196. Correlations between 24-h PM_{10} and co-located 24-h avg $PM_{2.5}$, $PM_{10-2.5}$, SO_2, NO_2 and CO and daily maximum 8-h avg O_3 for Los Angeles, CA, stratified by season (2005-2007). One point is included for each available monitor pair.

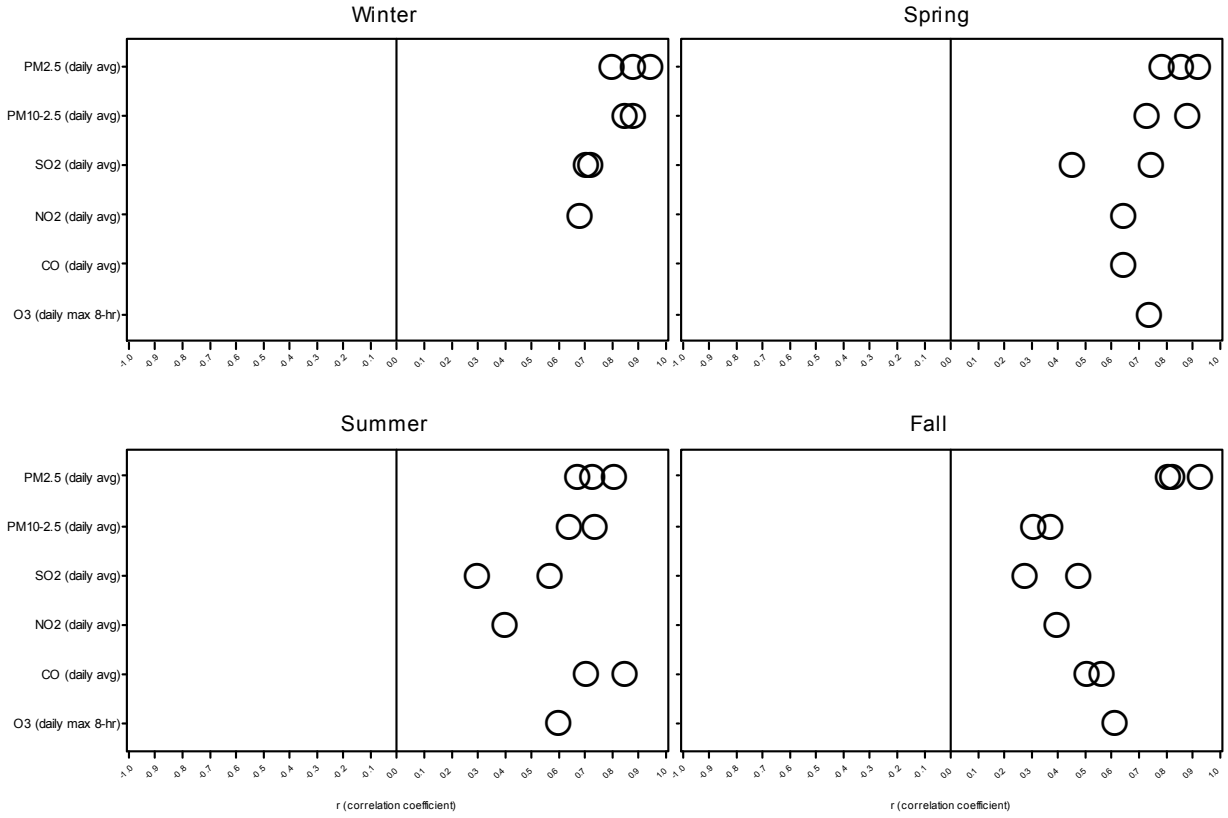


Figure A-197. Correlations between 24-h PM_{10} and co-located 24-h avg $PM_{2.5}$, $PM_{10-2.5}$, SO_2, NO_2 and CO and daily maximum 8-h avg O_3 for New York, NY, stratified by season (2005-2007). One point is included for each available monitor pair.

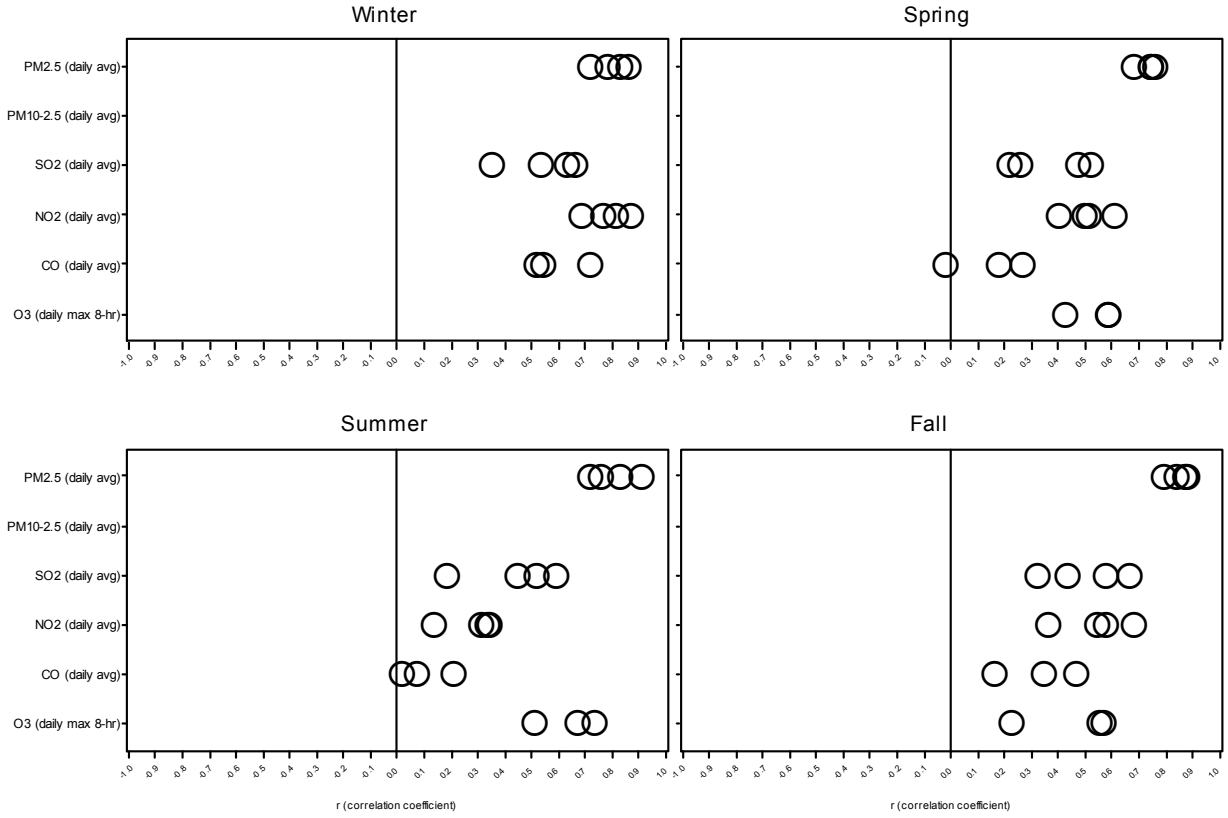


Figure A-198. Correlations between 24-h PM_{10} and co-located 24-h avg $PM_{2.5}$, $PM_{10-2.5}$, SO_2, NO_2 and CO and daily maximum 8-h avg O_3 for Philadelphia, PA, stratified by season (2005-2007). One point is included for each available monitor pair.

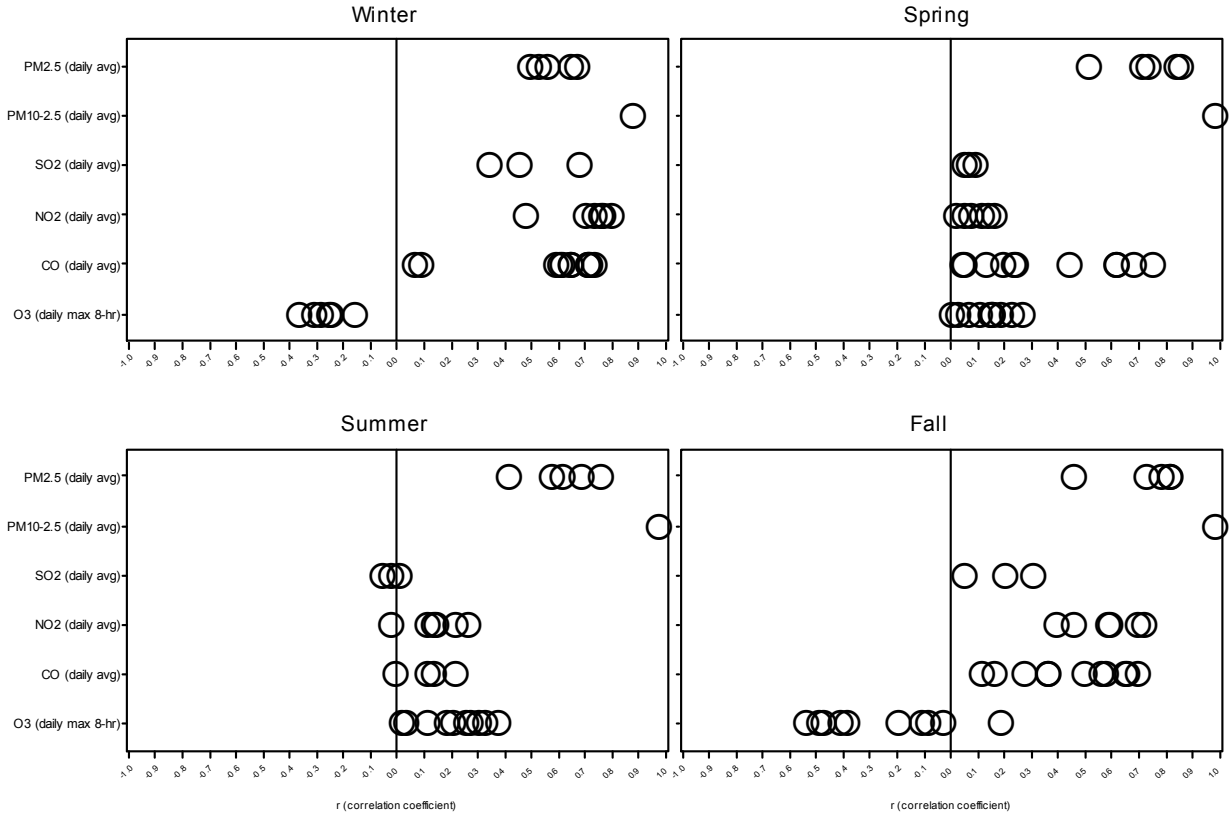


Figure A-199. Correlations between 24-h PM_{10} and co-located 24-h avg $PM_{2.5}$, $PM_{10-2.5}$, SO_2, NO_2 and CO and daily maximum 8-h avg O_3 for Phoenix, AZ, stratified by season (2005-2007). One point is included for each available monitor pair.

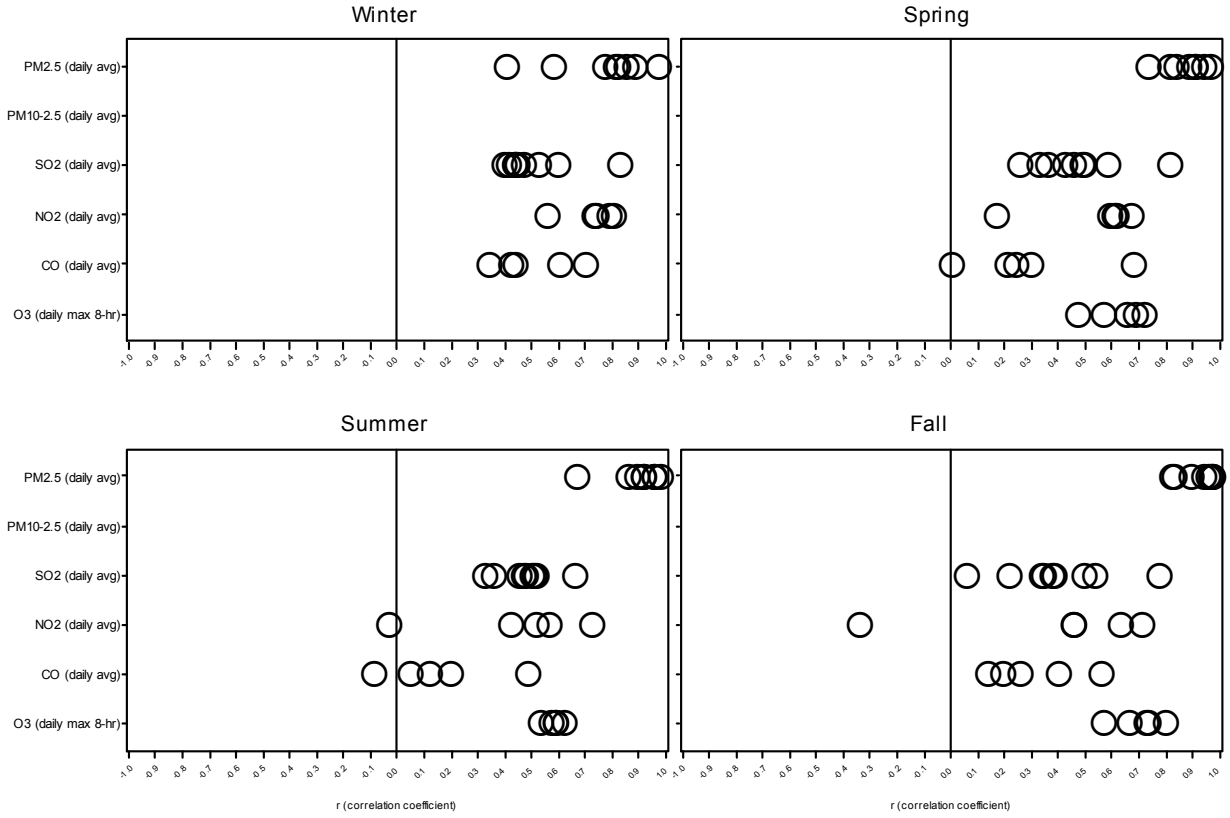


Figure A-200. Correlations between 24-h PM_{10} and co-located 24-h avg $PM_{2.5}$, $PM_{10-2.5}$, SO_2, NO_2 and CO and daily maximum 8-h avg O_3 for Pittsburgh, PA, stratified by season (2005-2007). One point is included for each available monitor pair.

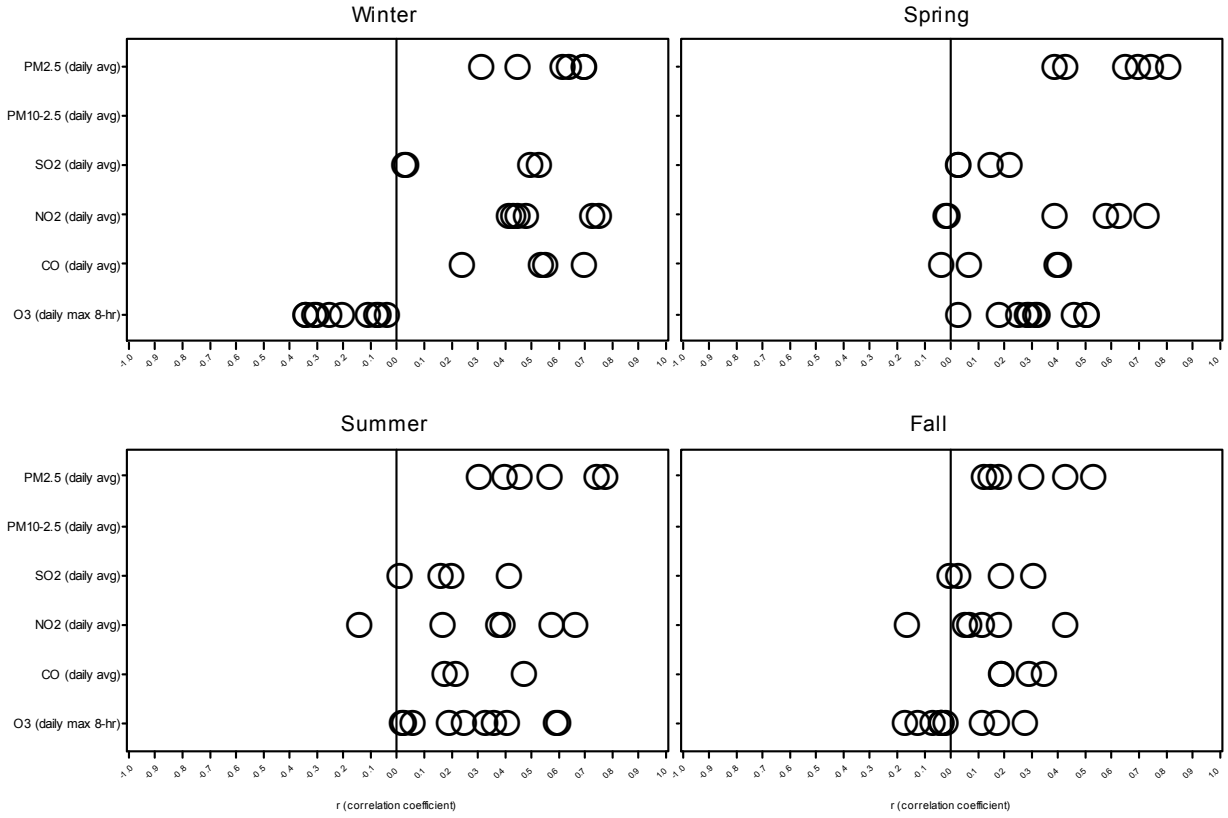


Figure A-201. Correlations between 24-h PM_{10} and co-located 24-h avg $PM_{2.5}$, $PM_{10-2.5}$, SO_2, NO_2 and CO and daily maximum 8-h avg O_3 for Riverside, CA, stratified by season (2005-2007). One point is included for each available monitor pair.

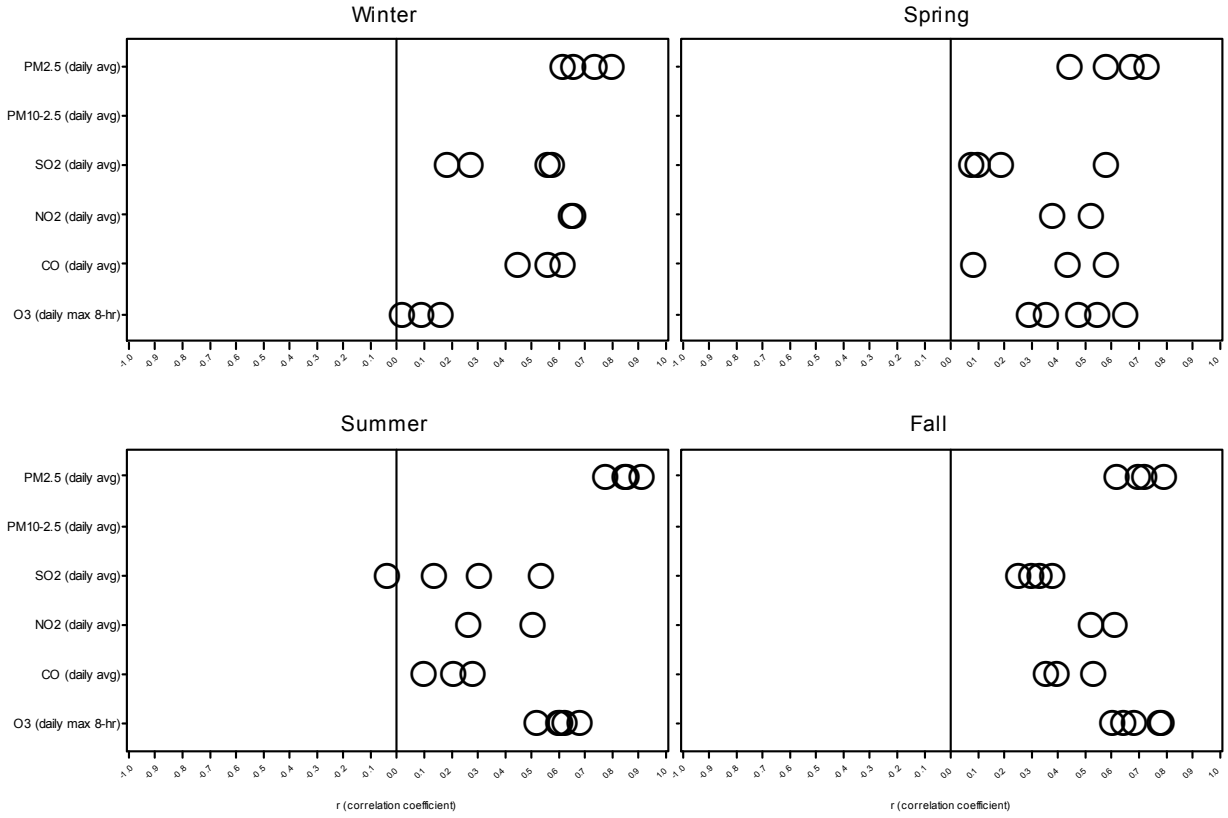


Figure A-202. Correlations between 24-h PM_{10} and co-located 24-h avg $PM_{2.5}$, $PM_{10-2.5}$, SO_2, NO_2 and CO and daily maximum 8-h avg O_3 for St. Louis, MO, stratified by season (2005-2007). One point is included for each available monitor pair.

A.3. Source Apportionment

A.3.1. Type of Receptor Models

Table A-51. 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 reflects the historical solution in which source profiles 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 (Equations A-1 or A-2). These equations are solved for the source contribution estimates when ambient concentrations and source profiles are input. The single-sample effective variance least squares is the most commonly used solution method because it incorporates uncertainties of ambient concentrations and source profiles 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 profiles should avoid collinearity, which occurs when chemical compositions of various source emissions are not sufficiently different.[121] $$C_{iklmn} = \sum_{j=1}^{J} F_{ijm} T_{ijklmn} S_{iklmn}$$ **for i = 1 to I** **Equation A-1** $$C_{it} = \sum_{j} F_{ij} S_{jt} + E_{it}$$ **Equation A-2** **Data Needs** CMB requires source profiles, 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 profiles are preferably obtained in the same geographical region as the ambient samples, although using source profiles from different regions is commonly practiced in the literature. The practitioner needs to decide the source profiles 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 R^2, deviation X^2, residue/ uncertainty ratio, and MPIN matrix that are useful for refining 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 profiles. Quantifies 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 specific chemical markers. Typically does not apportion secondary particle constituents to sources. Must be combined with profile 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-52. Different receptor models used in the Supersites source apportionment studies: factor analysis.

Receptor Model	Description	Strengths and Weaknesses
PMF PMF_x (PMF_2 and PMF_3) 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 PMF_2 and PMF_3. PMF_2 solves the CMB equations (i.e., Equations A-2 and A-3) using an iterative minimization algorithm. Source profiles 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 profile and contribution should not contain negative values. There is rotational ambiguity in all two-way factor analyses (i.e.,F_{jt} and S_{jt} matrices may be rotated and still fit 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 $F_{jt}.F_{key}$ can further bind individual elements in F_{jt} 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: $$Q \text{ or } \chi^2 = \sum_i \sum_t [E_{it}/\sigma_{it}]^2 = \sum_i \sum_t [(C_{it} - \sum_j F_{ij} S_{jt})/\sigma_{it}]^2 \quad \textbf{Equation A-3}$$ Where the weighing factor, σit, represents the magnitude of Eit, PMFx limits solutions of Equation A-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 Fkey 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 X^2 and standard deviation of each matrix element. The practitioner needs to interpret the results linking them to source profiles 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 profiles 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 profiles or existing profiles to verify the representativeness of calculated factor profiles and uncertainties of factor contributions. Relies on many parameters/initial conditions adjustable to model input; sensitive to the preset parameters.
ME2[125] 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 Equation A-1 or A-2, ME2 solves all models in which the data values are fitted by sums of products of unknown (and known) factor elements. The first part of the algorithm interprets instructions from the user and generates a table that specifies 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 profiles 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 flexible. 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-specified 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 Equations A-2 and A-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" defined 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 advance[127], which reports the R^2 and signal-to-noise (S/N) ratio associated with the first 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 signifies 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 (F_{ij}) and contribution (S_{it}) matrices. It also calculates the uncertainty associated with the factor elements and model performance measures including: (1) R^2, (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., R^2, 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 profiles 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, Equation A-2 is modified to: $$C_k = \sum_j ER_{ij}\left(\frac{\chi}{Q}\right)_{j,t} + E_k$$ **Equation A-4** where $ER_{i,j}$ is interpreted as the emission rate of species i from stationary source j and (X/Q,t is the meteorological dispersion factor averaged over the time interval t. Equation A-4 is solved for ERi,j and (X/Q,t simultaneously by a nonlinear fit minimizing the objective function, FUN: $$FUN = \sum_{i=1}^{l}\sum_{t=1}^{m}\sum_{j=1}^{n}\left[ER_{ij}\left(\frac{\chi}{Q}\right)_{j,t}^{PDRM} - C_k\right]^2$$ **Equation A-5** Because the number of solutions for a product of unknowns is infinite, additional constraints are set up for (X/Q,t on the basis of the Gaussian plume model, thus: $$LB\left(\frac{\chi}{Q}\right)_{j,t}^{Met} \le \left(\frac{\chi}{Q}\right)_{j,t}^{PDRM} \le UB\left(\frac{\chi}{Q}\right)_{j,t}^{Met}$$ $$\left(\frac{\chi}{Q}\right)_{j,t}^{Met} = \frac{1}{2\pi\sigma_y\sigma_z u}\exp\left(-\frac{1}{2}\frac{y^2}{\sigma_y^2}\right)\left\{\exp\left[-\frac{1}{2}\left(\frac{z-h}{\sigma_z}\right)^2\right] + \exp\left[-\frac{1}{2}\left(\frac{z+h}{\sigma_z}\right)^2\right]\right\}$$ **Equations A-6 & A-7** Equations A-6 and A-7 limit the solution of Equation A-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 Equation A-7: transport speed (u), lateral and vertical dispersion parameters (σy and σz), 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 configuration 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 fit. 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 significantly 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. Equation A- 2 is modified to: $$X_k = \sum_i T_i P_{ik} + E_k$$ **Equation A-8** $$Y_n = \sum_i U_i C_{in} + D_k$$ **Equation A-9** 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 A-8 and A-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. Rx and Ry, $$R_x = 1 - \text{var}(E)/\text{var}(X)$$ **Equation A-10** $$R_y = 1 - \text{var}(D)/\text{var}(Y)$$ **Equation A-11** 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.

Henry (1997, 020941)
Lewis et al. (2003, 088413)
Ogulei et al. (2006, 119975)
Park et al. (2005, 156844)
Paatero (1997, 087001)
Paatero et al. (2002, 156836)
Paatero (1999, 156835)
Henry (2003, 156540)

Source: Watson et al. (2008, 157128)

Table A-53. 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., Ci,t) can be expressed as: $$C_{i,t} = \sum_{j} \frac{1}{EF_{i,pj}} C_{pj,t} + Z_{i,t} = \sum_{j} \left(\frac{F_{ij}}{F_{pj}}\right) C_{pj,t} + Z_{i,t}$$ **Equation A-12** 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 Zi,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 profiles, edges of a two-way scatter plot 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 specific 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 identification than for quantification.
NNLS [131,132] The MatLab Optimization Toolbox provides a function "lsqnonneg" for performing the NNLS calculation.	**Principle** NNLS also solves the EF equation (Equation A-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.: $$EF_{i,pj} \geq 0$$ **Equation A-13** 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 coefficients. **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 coefficients for an EF/MLR problem and these coefficients can be related to the source contributions.	**Strengths** Implemented by many statistical software packages. Generate only non-negative EFs or regression coefficients. 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 specific. 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	**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 defined as the fraction of SOA that would result from the reactions of a particular VOC: $$[\text{SOA}] = \sum_i FAC_i \times ([\text{VOC}_i]_0 \times \text{Fraction of VOC } i \text{ reacted})$$ **Equation A-14** where $[\text{VOC}_i]_0$ is the emission rate of VOC_i and [SOA] is the formation rate of SOA. Equation A-14 can be viewed as an extension of Equation -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 significant 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 modifications 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 influence 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. Difficult to verify.

Grosjean and Seinfeld (1989, 045643)
Darns et al. (1970, 156379)
Reimann and De Caritat (2000, 013269)
Lawson and Hanson (1974, 156673)
Wang and Hopke (1989, 157105)

Source: Watson et al. (2008, 157128)

Table A-54. 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., O_3 in Equation A-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: $$CPF(\theta) = \frac{m_{\Delta\theta}}{n_{\Delta\theta}}$$ **Equation A-15** where $m\Delta\theta$ is the number of occurrences in the direction sector $\theta \rightarrow \theta + \Delta\theta$ that exceeds the specified 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.	**Strengths** Infer the direction of sources or factors relative to the receptor site. Provide verification for the source identification made by factor analysis method. Easy to implement. **Weaknesses** Criterion for the threshold is subjective. Absolute source contribution (or fractional contribution) may be influenced 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: $$S(\theta) = \frac{\sum_i K\left(\frac{\theta - W_i}{\Delta\theta}\right) \times S_i}{\sum_i K\left(\frac{\theta - W_i}{\Delta\theta}\right)}$$ **Equation A-16** where Wi is the wind direction for the ith sample and Si is the contribution from a specific 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 Δθ. If Δθ is too large, S(θ) will be too smooth and meaningful peaks could be lost. If it is too small, S(θ) will have too many small, meaningless peaks. Δθ needs to be chosen according to the project-specific spatial distribution of sources. NPR also estimates the confidence intervals of S(θ) based on the asymptotic normal distribution of the kernel estimates, thus: $$\Delta S(\theta) = \frac{\sum_i K\left(\frac{\theta - W_i}{\Delta\theta}\right) \times (S_i - S(\theta))^2}{\left(\sum_i K\left(\frac{\theta - W_i}{\Delta\theta}\right)\right)^2}$$ **Equation A-17** **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 confidence level associated with it.	**Strengths** Infer the direction of sources or factors relative to the receptor site. Provide verification for the source identification 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 influenced 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 ($\tau_{i,j}$, 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 $\tau_{i,j}$ and averaged over all samples to produce the directional dependent pollutant concentration/source contribution for the period of interest: $$\overline{S_j} = \sum_i S_i \left(\frac{\tau_{ij}}{\sum_i \tau_{ij}} \right) / N$$ **Equation A-18** 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 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. 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 efficiency, dispersion, and deposition. Difficult 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: $$PSCF_{ij} = \frac{\text{Sum of "high" residence time in cell } (i, j)}{\text{Sum of all residence time in cell } (i, j)}$$ **Equation A-19** 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.	**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 field" for each trajectory ignoring the effects of chemical reactions and deposition. Back trajectories are first 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: $$Q(x, y, t|x', y', z') = \frac{1}{2\pi(at')^2}\exp\left[-\frac{1}{2}\left(\left(\frac{X - x'(t')}{at'}\right)^2 + \left(\frac{Y - y'(t')}{at'}\right)^2\right)\right]$$ **Equation A-20** 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 field, Q, for a given trajectory is then integrated over the upwind period, τ, to produce a two-dimensional "natural" (nonweighted) transport field: $$T_k(x, y|x', y') = \frac{\int_{-\tau}^{0} Q(x, y, t|x', y', z')}{\int_{-\tau}^{0} dt'}$$ **Equation A-21** After the transport field 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 field.[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 field 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 field is unclear. Difficult 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] $$S_{i,k} = S_k \frac{\text{resident time in cell } i}{\text{average residence time in each cell}}$$ **Equation A-22** 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 identified when cells are crossed by a very small number of trajectories. Although some corrections were proposed147 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 field and source strength). Physical meaning of the RTWC field is unclear. Difficult 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, 188974)
[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-55. 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	N/A	5.968	0.5247	5.729	0.4058	7.4822	0.9315	8.4853	2.3478
Antimony	Sb	0.01	N/A	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	N/A	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	N/A	3.4536	1.0411	2.5657	0.1388	2.163	1.0444	0.1236	0.056
Chloride ion	Cl-	0.39	N/A								
Chromium	Cr	0.01	N/A	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	N/A	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	N/A	0	0.0404	0	0.022	0	0.1041	0	0.0183
Iron	Fe	1.27	N/A	2.916	0.3827	4.5713	0.2661	5.5128	2.1152	1.4708	0.2216
Lanthanum	La	0	N/A	0	0.2462	0	0.1341	0	0.6521	0	0.1146
Lead	Pb	0.08	N/A	0.068	0.0336	0.067	0.0074	0.0288	0.0284	0.0097	0.0063
Magnesium	Mg	0.14	N/A								
Manganese	Mn	0.01	N/A	0.0284	0.0139	0.087	0.009	0.1372	0.0509	0.016	0.002
Mercury	Hg	0	N/A	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	N/A	0.0072	0.0019	0.0081	0.0015	0.0091	0.0057	0.04	0.0065
Nitrate	NO_3^-	0.06	N/A	0	0.2116	0	0.094	0	0.6371	0	0.0772
Organic carbon	OC	59.37	N/A	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	N/A	0.9372	0.6322	0	0.0324	0.1603	0.044	0.0689	0.0144
Potassium	K	0.01	N/A	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	N/A	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	N/A								
Strontium	Sr			0.1964	0.0686	0.0395	0.0078	0.0313	0.0112	0.0094	0.0031
Sulfate	SO_4^-			10.1716	8.9405	1.1604	0.2003	0.8688	1.3788	2.3243	3.4523
Sulfur	S	0.37	N/A	2.948	2.729	0.598	0.0509	0.2808	0.3884	0.6304	0.9627

		Motor Vehicle Exhaust - Gasoline		Coal Combustion		Highway Road Dust		Unpaved Road Dust		Refinery	
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	N/A	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	NH4+	0.34	N/A	0.3476	0.1352	0	0.025	0	0.1317	0.3281	0.5565
Sodium ion	Na+										
Carbonate	CO_3^-										
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	N/A	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 %	Uncertainty	Weight %	Uncertainty	Weight %	Uncertainty	Weight %	Uncertainty	Weight %	Uncertainty
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		
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		

		Residential Wood Burning		Oil Combustion		DE		Fly Ash		Incinerator	
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	NH4+	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	CO_3^-			0	0.0214	0.2577	0.4463				
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-56. $PM_{2.5}$ receptor model results ($\mu g/m^3$)

Sampling Site	Measured $PM_{2.5}$ Concentration	Vegetative Burning	Road Dust, Soil	$(NH_4)_2SO_4$	NH_4NO_3	Tailpipe	Brake Wear
Albany, NY 2000-2001	20.9	5.5	1.9	2.4	4.6	2.9	0.0
Birmingham, AL, 2000-2001	16.2	3.3	1.4	3.7	2.4	5.7	0.0
Houston, TX, 2000-2001	12.4	3.1	2.6	1.6	2.2	2.6	0.0
Long Beach, CA, 2000-2001	30.0	4.6	1.3	2.1	16.3	4.1	0.4
Las Vegas, NV, 2000-2001	2.5	1.0	2.0	0.5	0.3	1.5	0.0
El Paso, TX, 2000-2001	5.5	0.7	2.8	0.7	0.3	2.0	0.3
Westbury, NY, 2000-2001	11.5	1.7	0.7	5.2	2.2	5.3	0.0

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

Table A-57. PM_{10} receptor model results (mass percent)

Sampling Site	Wood Smoke	Diesel	Gasoline Vehicles	Natural Gas Combustion	Vegetative Detritus	Tire Wear Debris
Apline, CA, 1994-1995	15.00	33.19	46.46		5.31	
Apline, CA, 1995	9.92	58.78	11.47		19.63	
Apline, CA, 1995	10.97	65.64	10.81		12.66	
Atascadero, CA, 1994-1995	44.22	22.16	26.44			6.91
Atascadero, CA, 1995	21.36	38.99	12.41		17.89	9.43
Atascadero, CA, 1995	73.45	18.11			3.14	5.31
Lake Arrowhead, CA, 1994-1995	6.86	46.55	33.92	2.73	9.85	
Lake Arrowhead, CA, 1995	4.85	65.20	7.40	4.95	17.65	
Lake Arrowhead, CA, 1995	9.91	38.90	46.70	0.79	3.66	
Lake Elsinore, CA, 1994-1995	12.72	44.01	18.61		4.21	20.42
Lake Elsinore, CA, 1995	17.13	74.72		0.26	7.81	
Lake Elsinore, CA, 1995[2]	6.84	38.48	10.85	0.21	15.55	28.01
Lancaster, CA, 1994-1995	22.49	43.14	20.56	0.45	3.73	9.78
Lancaster, CA, 1995	3.69	46.18	12.66	0.20	8.21	29.17
Lancaster, CA, 1995	34.89	37.30	7.33	0.61	7.78	11.93
Lompoc, CA, 1994-1995		18.16	49.65		5.89	26.38
Lompoc, CA, 1995	13.09	51.27	14.73		20.73	
Lompoc, CA, 1995		79.42	10.19		10.87	
Long Beach, CA, 1994-1995	10.12	43.24	16.49	0.13	3.97	26.00
Long Beach, CA, 1995	2.38	70.25	5.47	0.86	6.79	14.11
Long Beach, CA, 1995	14.32	56.80	6.15	0.72	5.34	16.61
Mira Loma, CA, 1994-1995	4.68	48.87	18.10		8.82	19.52
Mira Loma, CA, 1995	5.20	53.72	6.65		18.79	15.71
Mira Loma, CA, 1995	27.97	41.88	8.87		11.50	9.85
Riverside, CA, 1994-1995	14.14	46.67	12.03		6.83	20.31
Riverside, CA, 1995	6.20	52.15	7.93	0.16	14.54	19.06
Riverside, CA, 1995	25.28	47.65			6.91	20.17
San Dimas, CA, 1995	7.62	71.35	4.87	0.15	8.35	
San Dimas, CA, 1995	22.01	61.34	4.48	0.23	3.70	7.85
Santa Maria, CA, 1994-1995	18.66	23.99	22.03		5.58	8.15
Santa Maria, CA, 1995	12.94	52.57	11.87	0.27	9.63	12.78
Santa Maria, CA, 1995	12.24	48.13	10.79	0.47	18.04	15.05
Upland, CA, 1994-1995	20.33	46.39	14.08		4.49	14.70
Upland, CA, 1995	7.33	68.69	3.50	0.17	9.19	11.25
Upland, CA, 1995	28.10	46.52	4.90	0.33	10.30	9.81

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

A.4. Exposure Assessment

A.4.1. Exposure Assessment Study Findings

Table A-58. Exposure Assessment Study Summaries

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. Two portable carts containing continuous air pollution monitors were used to measure group-level micro-environmental exposures to traffic related pollutants, including $PM_{2.5}$, 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.
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	$PM_{2.5}$ 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 $PM_{2.5}$ were positively associated with FeNO. For example, an interquartile increase of 4 µg/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 µg/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 µg/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 for 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 outdoor (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. O samples 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	$PM_{2.5}$
Microenvironment Size	$PM_{2.5}$
Ambient Size	$PM_{2.5}$
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 trace elements (TE). 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 a lower income and centrally located community than in a comparatively higher income 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 I measurements. Participants also carried personal pumps in small bags to obtain P measurements. Start times for indoor and personal monitors were always within a few minutes of each other. Gravimetric 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	$PM_{2.5}$ concentrations were higher than I concentrations, which were higher than O concentrations. In healthy non-smoking adults, moderate median for correlation between P and I; modest median for correlation between I and O; and minimal median correlation between P and O longitudinal 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.

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 to 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)	S
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 Penetration, air exchange rate, deposition rate, and NH^{4+} for each residence.

Allen et al. (2007, 154226)

Period	Heating seasonOctober-February; Non-heating season March-September
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)	S (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}. Individual 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 S, instead of techniques that give average estimates of infiltration across homes.

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 $\mu g/m^3$). 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.

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	I/O suggest that chemicals such as Cl^-, Na^+, Al, Co, Cu, Fe, Mn, Ti, V, Zn, and EC were derived from the migration of outdoor particles (I/O <1 or ~1).

Barn et al. (2008, 156252)

Study Design	Measure indoor F_{inf} 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 F_{inf} 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	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 F_{inf} ($p< 0.001$).

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 $\mu g/m^3$ 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 50 m 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, 007894)

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	ETS, pets, cleaning, traffic load
Personal Method	NR
Personal Size	NR
Microenvironment Size	PM_{10}
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 coarse particles; outdoor air identified as a source of indoor fine particles ($PM_{1.0}$ and $PM_{2.5}$)
Personal Method	Harvard impactors (HI) 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.

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 4 L/min.
Personal Size	$PM_{2.5}$
Microenvironment Size	$PM_{2.5}$
Ambient Size	$PM_{2.5}$
Component(s)	SO_4^{2-}
Primary Findings	In both cities, personal and indoor $PM_{2.5}$ were highly correlated with outdoor concentrations.

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 Fe, Mn, and Cr are reported in this study out of 28 elements sampled.
Primary Findings	Personal samples had significantly higher concentration of Fe, Mn, and Cr 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.

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%), mold (27%), being present during cooking (27%), and aerosol use (17%).

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	$PM_{2.5}$
Component(s)	NR
Primary Findings	Higher correlation was observed between P and I compared with the correlation between either P and ambient (A) or I and A.

Delfino et al. (2004, 056897)

Study Design	Panel study with repeated measures
Period	Sep-Oct 1999 or Apr-Jun 2000
Location	Alpine, California
Population	Children
Age Groups	9-17 yr
Indoor Source	No
Personal Method	Personal dataRAM (pDR) carried at waist level using a fanny pack, shoulder harness, or vest.
Personal Size	0.1-10 μm
Microenvironment Size	PM_{10} and $PM_{2.5}$; 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 $PM_{2.5}$, 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	The strongest positive associations were between FeNO and 2-day average pollutant concentrations. Per IQR increases 1.1 ppb FeNO/24 μg/m^3 personal $PM_{2.5}$.; 0.7 ppb FeNO/0.6 μg/m^3 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.

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 were well correlated with the outdoor concentration (R^2 = 0.89).

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_1, $PM_{2.5}$, PM_{10}, presence of children and activities of children in classrooms, infiltrated vehicular exhaust
Personal Method	Harvard PEM, Teflon filters Dust Trak Condensation particle counter
Personal Size	NR
Microenvironment Size	PM_1, $PM_{2.5}$, PM_{10}
Ambient Size	PM_1, $PM_{2.5}$, PM_{10}
Component(s)	NO_3^-, SO_4^{2-}
Primary Findings	High levels of PM_{10} and $PM_{2.5}$ measured indoors and outdoors. PM_{10} more variable spatially than $PM_{2.5}$. I/O ratio for PM_{10} and $PM_{2.5}$ close to 1 at almost all sites. Ratio of PM_1 smaller 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.

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	24-h integrated filter sample
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)	SO_4^{2-}
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 molecular epidemiology studies of carcinogenic environmental pollutants, particularly PAHs
Period	12 months
Location	Prague, Czech Republic (2 sites); Košice, Slovak republic; Sofia, Bulgaria
Population	Policeman and busdrivers usually working through busy streets in 8-10 h 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)	Extractable organic matter (EOM), B[a]P, c-PAHs
Primary Findings	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.

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.

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	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.

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	NR
Personal Method	Measured using personal exposure monitors (MSP Inc) with nozzle to remove particles > 4 µm
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	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 $PM_{2.5}$. Based on comparison of trace metals in central I site vs. P samples, resident activities result in exposure to higher concentration of soluble trace metals.

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.

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 μm 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, TC
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}, $PM_{2.5}$, $PM_{10-2.5}$, UFP
Component(s)	soot, sulfate
Primary Findings	Correlation between 24-h average central site and indoor concentrations was lower for UFP than for $PM_{2.5}$, soot, or SO_4^{2-}, probably related to greater losses during infiltration due to smaller particle size. Infiltration factors for UFP and $PM_{2.5}$ were low.

Hopke et al. (2003, 095544)

Study Design	Exposure assessment
Period	26 July to 22 August 1998
Location	Retirement facility in Towson, MD
Population	Elderly residents
Age Groups	Mean age of 84
Indoor Source	Ammonium sulfate and ammonium nitrate, secondary sulfate, OC, and motor vehicle exhaust
Personal Method	Inertial impactor PEM in the breathing zone of the subjects
Personal Size	$PM_{2.5}$
Microenvironment Size	$PM_{2.5}$
Ambient Size	$PM_{2.5}$
Component(s)	SO_4^{2-}
Primary Findings	Personal exposures were influenced by a combination of indoor and outdoor factors. Indoor factors included gypsum, personal grooming products, and an unknown indoor source. Outdoor factor included SO_4^{2-}, soil, and an unknown factor. Outdoor factors accounted for 63% of personal exposure, and SO42- was the largest ambient contributor to personal exposure (48%).

Jacquemin et al. (2007, 156600)

Study Design	Assessment of relationship between outdoor and personal concentrations of $PM_{2.5}$ absorbance and sulfur among post-myocardial infarction patients
Period	January 2004-June 2004
Location	Barcelona, Spain
Population	Survivors of a myocardial infarction exposed to ETS
Age Groups	n = 38, including 32 and 15 over age 64.
Indoor Source	ETS
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)	S
Primary Findings	Ambient measurements of light extinction and S can be used as surrogates to personal $PM_{2.5}$ exposure, especially for those exposed to ETS.

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 EC, 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^-).

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)
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 a very strong determinant of the total personal exposure to $PM_{2.5}$, and it explained about 31% of variance between the subjects.

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 $_{PM2.5}$ 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 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.

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. UFP measured using TSI P-TRAK particle counters in which isopropyl alcohol condenses to form droplets that can be easily counted by a photodetector as they pass through a laser beam.
Personal Size	$PM_{2.5}$, UFP (0.02-1.0um)
Microenvironment Size	$PM_{2.5}$, 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
Indoor Source	NR
Personal Method	$PM_{2.5}$ gravimetric filter measurement, UFP (0.02-1 µm) P-TRAK device, reflectance reflectometer measurement of $PM_{2.5}$ filter
Personal Size	$PM_{2.5}$, UFP (0.02-1 µm)
Microenvironment Size	NR
Ambient Size	$PM_{2.5}$, UFP (0.02-1 µm)
Component(s)	Absorbance of $PM_{2.5}$ filter
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	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

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	Integrated 24-h filter sample
Personal Size	$PM_{2.5}$
Microenvironment Size	$PM_{2.5}$
Ambient Size	$PM_{2.5}$
Component(s)	BS
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	Sometimes ETS
Personal Method	Integrated 48-h filter sample
Personal Size	$PM_{2.5}$
Microenvironment Size	$PM_{2.5}$
Ambient Size	$PM_{2.5}$, PM_{10}
Component(s)	NR
Primary Findings	Throughout the study, the highest correlations were those between personal exposures and indoor concentrations, which suggests that indoor sources were important. Correlations were generally lower between ambient concentrations and personal exposures.

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
Indoor Source	NR
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)	EC, SO_4^{2-}
Primary Findings	Ambient $PM_{2.5}$ and SO_4^{2-} are strong predictors of respective personal exposures. Ambient SO_4^{2-} is a strong predictor of personal exposure to $PM_{2.5}$. Because $PM_{2.5}$ has substantial indoor sources and SO_4^{2-} does not, the investigators; concluded that personal exposure to SO_4^{2-} accurately reflects exposure to ambient $PM_{2.5}$ and therefore the ambient component of personal exposure to $PM_{2.5}$ as well.

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	Integrated 48-h filter samples
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	Personal exposures were influenced by both indoor and ambient sources, and indoor levels exceeded ambient levels for $PM_{2.5}$ as well as for VOCs and eight other compounds. Correlation between personal and indoor $PM_{2.5}$ was 0.60 ($p < 0.001$).

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.

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.

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 his/her age was not specified)
Personal Method	Harvard Personal Environmental Monitor for $PM_{2.5}$ ($HPEM_{2.5}$)
Personal Size	$PM_{2.5}$, PM_{10}
Microenvironment Size	$PM_{2.5}$, PM_{10}
Ambient Size	$PM_{2.5}$, PM_{10}
Primary Findings	Average personal $PM_{2.5}$ exposure was similar to ambient $PM_{2.5}$ concentrations but much higher than average indoor concentrations. Personal, indoor, and outdoor $PM_{2.5}$ and PM_{10}, as well as the ratio PM2.5/PM10, were all significantly higher during the winter. Personal $PM_{2.5}$ and PM_{10} exposures were highest for the children in the study.

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.

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	NR
Age Groups	NR
Indoor Source	NR
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	$PM_{2.5}$
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).
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.

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	Integrated filter samples with a dichotomous virtual impactor to separate $PM_{10\text{-}2.5}$ from $PM_{2.5}$
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	Wood burning made statistically significant contributions to personal exposure to K, Ca, and Zn. Cl, Mn, Cu, Rb, Pb, and BS were found to be potential personal exposures from wood smoke, but their association was not always statistically significant. S had no significant association with personal exposure to wood smoke.

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	Integrated filter samples with cyclones for $PM_{2.5}$ and PM_1 cut points
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	Personal exposure to Cl, K, Ca, Ti, Fe, and Cu in PM2.5 were significantly higher than outdoor and central site ambient concentrations, and personal exposure to Cl, Ca, Ti, Fe, and Br were also significantly higher than indoor levels. In most cases, indoor concentrations were not higher than outdoor concentrations.

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	Integrated filter samples for $PM_{2.5}$
Personal Size	$PM_{2.5}$
Microenvironment Size	NR
Ambient Size	$PM_{2.5}$
Component(s)	EC, OC
Primary Findings	Indoor $PM_{2.5}$ was significant influenced by indoor OC sources. Indoor EC sources were predominantly of outdoor origin.

Naumova et al. (2003, 089213)

Study Design	RIOPA Study-PAH partitioning indoor and outdoor pollutants 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	$PM_{2.5}$
Microenvironment Size	$PM_{2.5}$
Ambient Size	$PM_{2.5}$
Component(s)	OC, EC
Primary Findings	Both EC and OC were associated with gas/particle partitioning of PAHs, with EC being a better predictor. High correlation between EC and OC suggests that PAHs adsorb onto PM containing EC during combustion.

Nerriere et al. (2005, 089481)

Study Design	Exposure assessment with 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	NR
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, but the opposite is true for NO_2.

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
Personal Method	$PM_{2.5}$ Harvard Personal Environment Monitors ($HPEM_{2.5}$)
Personal Size	$PM_{2.5}$
Microenvironment Size	NR
Ambient Size	$PM_{2.5}$
Component(s)	SO_4^{2-}, ABS (light absorbing carbon)
Primary Findings	Thermal inversions were associated with personal exposures as well as ambient $PM_{2.5}$ concentrations and likely caused observed spatial variability. However, ambient sampling locations were correlated in time. Similar observations were made for SO_4^{2-} and ABS.

Rojas-Bracho et al. (2004, 054772)

Study Design	Cohort study with repeated measures.
Period	Winter or summer of 1996-1997
Location	Boston, Massachusetts
Population	COPD patients
Age Groups	Adult
Indoor Source	Housecleaning, cooking, transport in motor vehicles, low-effort home activities, moderate-effort home activities, activities in public places, and resting or sleeping.
Personal Method	PEM
Personal Size	$PM_{2.5}$, PM_{10}, and $PM_{10-2.5}$
Microenvironment Size	$PM_{2.5}$, PM_{10}, & $PM10\text{-}2.5$
Ambient Size	NR
Component(s)	NR
Primary Findings	During both seasons, personal exposures were higher than indoor or outdoor means, except during the winter when 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:26 January 1997–4 June 1998 Basel, Switzerland 3 February 1997–23 January 1998 Milan, Italy 10 March 1997–23 May 1998 Oxford, UK November 1998–7 October 1999 Prague, Czech Republic 3 June 1997–4 June 1998 Helsinki, Finland 26 September 1996–10 December 1997
Location	Athens, Greece; Basel, Switzerland; Milan, Italy; Oxford, UK; Prague, Czech Republic; Helsinki, Finland
Population	Adults
Age Groups	25-55 yr
Indoor Source	NR
Personal Method	Integrated 48-h $PM_{2.5}$ filter samples
Personal Size	$PM_{2.5}$
Microenvironment Size	$PM_{2.5}$
Ambient Size	$PM_{2.5}$
Copollutant(s)	NO_2
Primary Findings	Personal $PM_{2.5}$ and NO_2 levels were associated with subjects' level of annoyance. Highest annoyance levels occurred while in traffic.

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 sample
Personal Size	$PM_{2.5}$
Microenvironment Size	NR
Ambient Size	NR
Component(s)	4-6 ring PAHs on indoor particle
Primary Findings	Indoor concentration of 4-6-ring PAH were linked to outdoor industrial sources in residences without any major indoor source, but with industrial facility as the main outdoor source. This 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); NO_3 (volatile component)
Primary Findings	Infiltration rate for $PM_{2.5}$ was intermediate, while BC was highest and NO_3 lowest. Infiltration rate varied with particle size, air exchange rate, outdoor NO_3. $PM_{2.5}$ infiltration was lowest for volatile components. Outdoor volatile $PM_{2.5}$ components may be less representative of indoor exposure to volatile $PM_{2.5}$ of ambient origin. 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	NR
Personal Method	Integrated filter gravimetric measurement
Personal Size	$PM_{2.5}$
Microenvironment Size	NR
Ambient Size	$PM_{2.5}$
Component(s)	SO_4^{2-}; EC
Primary Findings	24-h ambient measurements are more representative of personal particle exposure than gases, and 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 also made.
Population	School children and seniors
Age Groups	NR
Indoor Source	$PM_{2.5}$
Personal Method	NR
Personal Size	$PM_{2.5}$
Microenvironment Size	NR
Ambient Size	$PM_{2.5}$
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.

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	NR
Personal Method	Integrated filter and real-time nephelometer at floor height and at a height of 110 cm
Personal Size	TSP, inhalable PM
Microenvironment Size	NR
Ambient Size	NR
Copollutant(s)	NR
Primary Findings	The study results suggest that young children are exposed to more inhalable PM and TSP because PM becomes resuspended from the floor with motion.

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	NR
Personal Method	PM_{10} measured with integrated filter samples
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 Wolverhampton, UK
Population	NR
Indoor Source	Mn,Al, NO_3, Cl^- (wind-blown dust), Cu and Zn^-
Personal Method	Active sampling using Casella sampler (filter)-
Personal Size	Respirable PM
Microenvironment Size	Respirable PM
Ambient Size	Respirable PM
Component(s)	NO_3^-, metals (Zn, Cu, Mn, Al), SO_4^{2-}, Cl^-
Primary Findings	The indoor particulate concentration was driven by ambient concentration, and meteorological-induced changes in ambient PM were detected indoors.

Strand et al. (2007, 157018)

Study Design	Cohort
Period	Winter of 1999-2000 and winter of 2000-2001
Location	Denver, Colorado
Population	Asthmatic children
Indoor Source	NR
Personal Method	Modeling/extrapolation from fixed-site ambient monitoring (multiple methods)
Personal Size	NR
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 FEV1 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}, $PM_{2.5}$, PM_1, $PM_{10\text{-}2.5}$, $PM_{2.5\text{-}1}$
Microenvironment Size	NR
Ambient Size	PM_{10}, $PM_{2.5}$, $PM_{10\text{-}2.5\text{-}}$
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.

Thornburg et al. (2004, 157052)

Study Design	PM exposure studies
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	Resuspension of PM_{10} from a carpet and cooking
Personal Method	Harvard impactors and PEMs, 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
Age Groups	Adult
Indoor Source	Fungi, bacteria
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.

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	$PM_{10-2.5}$, $PM_{2.5}$ 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.

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%.

Vallejo et al. (2006, 157081)

Study Design	Panel study
Period	4/2002-8/2002
Location	Mexico City, Mexico
Population	Health young, non-smoking adults
Age Groups	Mean age 27 yr
Indoor Source	NR
Personal Method	pDR nephelometric method
Personal Size	$PM_{2.5}$
Microenvironment Size	NR
Ambient Size	NR
Component(s)	NR
Primary Findings	The descriptive analysis showed that overall outdoor median concentration of $PM_{2.5}$ was higher than the indoor concentration. 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.

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	ETS, cooking
Personal Method	Integrated filter gravimetric measurement. Light absorbance.
Personal Size	$PM_{2.5}$
Microenvironment Size	NR
Ambient Size	$PM_{2.5}$
Component(s)	Absorbance
Primary Findings	Children living near busy roads had 35% higher personal exposure to ‘soot’ than children living in urban background locations.

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	Condensation particle counters
Personal Size	UFP (10-100 nm)
Microenvironment Size	UFP (10–100 nm)
Ambient Size	PM_{10}
Primary Findings	UFP exposure predicted oxidative DNA damage.

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)	S
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
Population	Health-compromised adults, non-smokers
Age Groups	Adults
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.

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)	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)	OC and EC
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.

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	Children and adults (6-89 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 $PM_{2.5}$ concentrations.

Wichmann et al. (2005, 086240)

Study Design	Exposure assessment
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	NR
Personal Method	Personal impactor
Personal Size	PM_{10}
Microenvironment Size	PM_{10}
Ambient Size	PM_{10}
Component(s)	Absorbance coefficient measurements
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	Raleigh and Chapel Hill, North Carolina
Population	Elderly persons
Age Groups	> 50 yr
Indoor Source	Occasional ETS
Personal Method	Integrated filter samples
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	When comparing cohorts, there was no statistically significant difference between $PM_{2.5}$ exposure. Little spatial variability was observed regarding $PM_{2.5}$ concentrations; this was observed to a lesser extent for PM_{10} as well.

Wilson and Brauer (2006, 088933)

Study Design	Exposure assessment
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)	SO_4^{2-}
Primary Findings	It was observed that ambient $PM_{2.5}$ exposure, estimated with the SO_4^{2-} method, accounted for 71% of measured ambient concentration and 44% of measured total personal exposure. No correlation between nonambient exposure and ambient concentration was observed.

Wu et al. (2006, 179950)

Study Design	Panel study
Period	9/3/2002-11/1/2002
Location	Pullman, WA
Population	Asthmatic adults
Age Groups	18-52 yr
Indoor Source	No
Personal Method	Co-located Harvard Personal Environmental Monitors (HPEM2.5; Harvard School of Public Health, Boston, MA)\
Personal Size	PM2.5
Microenvironment Size	PM2.5
Ambient Size	PM2.5
Component(s)	Levoglucosan (LG); Elemental Carbon (EC); Organic Carbon (OC)
Primary Findings	The authors observed significant variability between subjects for burning and nonburning episodes. The authors postulated that activity patterns contribute to this variability and that central-site measurements of LG might not be a good surrogate for biomass combustion smoke exposurefor this reason.

Wu et al. (2005, 086397)

Study Design	Panel study
Period	1999-2000
Location	Alpine, CA
Population	Asthmatic children
Age Groups	9-17 yr
Indoor Source	NR
Personal Method	pDR
Personal Size	$PM_{2.5}$
Microenvironment Size	$PM_{2.5}$
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, although 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.

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 produce nearly equal results. 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.

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	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	PEM and HI
Personal Size	NR
Microenvironment Size	NR
Ambient Size	NR
Component(s)	SO_4^{2-} , OC, EC, and trace elements
Primary Findings	Secondary SO_4^{2-} and vehicle emissions were significant contributors of personal PM exposure and residential indoor PM concentrations.

Zhao et al. (2007, 156182)

Study Design	Comprehensive analysis of the sources of PM_{15} exposure on children with moderate to severe asthma in urban-poor settings.
Period	Two 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	PEM
Personal Size	$PM_{2.5}$
Microenvironment Size	$PM_{2.5}$
Ambient Size	$PM_{2.5}$
Component(s)	EC, Cl, Si, NO_3
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	Exposure assessment
Period	Winter Period of 2005 and 2006
Location	Baden-Wuerttemberg, Germany
Population	School children
Age Groups	NR
Personal Method	NR
Personal Size	NR
Microenvironment Size	They only reported concentrations for $PM_{2.5}$. PM ranging in size from 0.02 to >20 μm were collected and analyzed but only $PM_{2.5}$ concentration were reported.
Ambient Size	They only reported concentrations for $PM_{2.5}$. PM ranging in size from 0.02 to >20 μm were collected and analyzed but only $PM_{2.5}$ concentration were reported.
Primary Findings	The impaction of PM was strongly influenced by specific weather conditions. Time resolution of measurements in classrooms showed variation in particle concentration depending on the type of building and indoor activities. E'[Concentrations of very small particles indoors and in ambient air measured by condensation particle counter were influenced by traffic emissions.

Table A-59. Examples of studies showing developments with UFP sampling methods since the 2004 PM AQCD.

Reference	PM Size Ranges	PM Constituents	Instruments	Study Description
Biswas et al. (2005, 150694)			CPC (water)	Water-based CPC performance evaluation.
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 evaluation.
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 compared best 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.
Petäjä et al. (2006, 156021)			CPC (water)	Water-based CPC performance evaluation.
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-60. Summary of in-vehicle studies of exposure assessment.

Reference	Study Design	Mode of Transport	Exposures	Primary Findings
Briggs et al. (2008, 156294)	UFP (P-Trak), PM_{10}, $PM_{2.5}$, and PM_1 (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: PM_1-PM_{10} (µg/m^3), UFP (p cm^{-3}) Avg Car Exposure: PM_{10} 5.87 (3.09) $PM_{2.5}$ 3.01 (1.10) PM_1 1.82 (1.10) UFP 21639 (14379) Avg Walking Exposure: PM_{10} 27.56 (13.16) $PM_{2.5}$ 6.59 (3.12) PM_1 3.37 (3.40) UFP 30334 (17245)	In-car concentrations of $PM_{2.5}$, PM_1, 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 average 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.
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	Car	15-min median (1000p/cm^3): School indoor 13.6 School outdoor 16.6 Residence indoor 11.2 Residence outdoor 24.0 In-vehicle 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.
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 measured UFP (CPCs, SMPS, EAD), BC (aethalometer), NOX (chemiluminescence), PM-bound PAHs (UV-photoionization), and 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/cm^3) 13-43 $PM_{2.5}$ (µg/m^3) 7.9-45 BC (µg/m^3) 0.74-3.3 Freeway range of medians: UFP (1000p/cm^3) 47-190 $PM_{2.5}$ (µg/m^3) 25-110 BC (µg/m^3) 2.4-13	Measurements of freeway UFP, BC, PM-bound PAH, and NO_x 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).
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.

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 ($\mu g/m^3$), components (% of mass) Bus: $PM_{2.5}$ 20-58 $(NH_4O_3$ 5-8 $(NH_4)_2SO_4$ 10-18 OC 17-39 EC 8-20 Crustal 15-18 Non-crustal 2-3 Unknown 6-24 Minibus: $PM_{2.5}$ 25-55 $(NH_4O_3$ 4-13 $(NH_4)_2SO_4$ 7-22 OC 22-37 EC 9-19 Crustal 12-13 Non-crustal 3-3 Unknown 4-26 Metro: $PM_{2.5}$ 24-41 $(NH4O_3$ 5-8 $(NH4)2SO_4$ 10-21 OC 35-42 EC 9-13 Crustal 10-16 Non-crustal 2-4 Unknown 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.
Gulliver and Briggs (2004, 053238)	PM_{10}, $PM_{2.5}$, and PM_1 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	Walking concentrations, Units: $\mu g/m^3$ Walk, Car, Background PM_{10} 38.2,43.2, 26.6 $PM_{2.5}$ 15.1, 15.5 PM_1 7.1, 7.0	In-car PM_{10} concentrations were elevated compared with walking and background. $PM_{2.5}$ and PM_1 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.
Gulliver and Briggs (2007, 155814)	TSP, PM_{10}, $PM_{2.5}$, and PM_1 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 concentrations, Units: $\mu g/m^3$ Walk, Car, Background TSP-PM_{10} 19.1 (19.8) 18.2 (18.0) 4.9 (5.1) $PM_{10-2.5}$ 22.1 (22.8) 15.1 (14.2) 10.0 (9.0) $PM_{2.5}$ -1 10.9 (10.4) 8.3 (8.4) 7.6 (7.1) PM_1 4.8 (3.4) 2.9 (2.6) 4.2 (2.4)	Walking exposures larger than car and background, and car exposures were generally larger than background except for PM_1. Peak exposures during walking were significantly higher than peak in-car exposures.
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/m^3 Winter 2005: Bus Control c-PAH 7.1 (3.7) 9.4 (5.5) B[a]P 1.3 (0.7) 1.8 (1.0) Smmer 2006: Bus Control c-PAH 1.8 (0.5) 2.0 (0.8) B[a]P 0.2 (0.1) 0.3 (0.2) Wnter 2006: Bus Control c-PAH 5.4 (3.5) 4.1 (1.7) B[a]P 1.0 (0.5) 0.8 (0.4)	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.

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 ($\mu g/m^3$) PAH (ng/m^3) Windows closed: BC PAH Ambient: 2.5,27 CNG:2.3, 57 TO: 7.1, 190 Diesel:11, 290 Windows open: BC PAH Ambient: 1.9,26 CNG:1.5, 43 TO: 2.3, 42 Diesel: 3.9, 58	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.

Table A-61. Summary of personal PM exposure studies with no indoor source during 2002-2008.

Reference / Location	Personal	Micro	Ambient
SOUTHWEST			
Delfino et al. (2004, 056897) Alpine, California	Method: pDR, Units = $\mu g/m^3$ Last 2-h $PM_{2.5}$ 34.4 (33.7) Diurnal $PM_{2.5}$ 55.7 (31.6) Nocturnal $PM_{2.5}$ 22.3 (13.6) 1-h max $PM_{2.5}$ 151.0 (120.3) 4-h max $PM_{2.5}$ 87.5 (55.3) 8-h max $PM_{2.5}$ 67.6 (39.0) 24-h $PM_{2.5}$ 37.9 (19.9)	Method: HI, Units = $\mu g/m^3$ 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, Units = $\mu g/m^3$ Diurnal PM_{10}35.1 (11.3) Nocturnal PM_{10} 23.3 (8.4) 1-h max PM_{10} 54.4 (13.8) 4-h max PM_{10} 44.5 (12.4) 8-h max PM_{10} 39.8 (11.2) 24-h PM_{10} 23.6 (9.1) 24-h $PM_{2.5}$ 10.3 (5.6)
Delfino et al. (2006, 090745) Riverside and Whittier, California	Method: PEM, Units = $\mu g/m^3$ Riverside: n13 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: n32 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, Units = $\mu g/m^3$ Riverside: 24-h $PM_{2.5}$ 36.63 (23.46) 24-h PM_{10} 70.82 (29.36) Whittier: 24-h $PM_{2.5}$ 18.0 (12.14) 24-h PM_{10} 35.73 (16.6)
Turpin et al. (2007, 157062) Los Angeles County, CA (and Elizabeth, NJ, Houston, TX)	Method: PEM, Units = $\mu g/m^3$ Avg of 48-h $PM_{2.5}$ Child 40.2 Adult 29.2	Method: HI, Units = $\mu g/m^3$ Avg of 48-h $PM_{2.5}$: 16.2	Method: HI, Units = $\mu g/m^3$ Avg of 48-h $PM_{2.5}$: 19.2
Wu et al. (2005, 157155) Alpine, CA	Method: pDR, Units = $\mu g/m^3$ n11 Avg of 24-h $PM_{2.5}$ 11.4 (7.8)	Method: pDR, Units = $\mu g/m^3$ n14 Avg of 24-h $PM_{2.5}$ 5.6 (2.9) Method: HI n14 Avg of 24-h $PM_{2.5}$ 9.8 (2.5)	Method: pDR, Units = $\mu g/m^3$ n8 Avg of 24-h $PM_{2.5}$ 14.0 (11.4) Method: HI n8 Avg of 24-h $PM_{2.5}$ 14.3 (7.8)

Reference / Location	Personal	Micro	Ambient
NORTHWEST			
Jansen et al. (2005, 082236) Seattle, Washington, USA	NR	Method: HI, Units = μg/m^3 Indoor home: PM_{10} 11.93 $PM_{2.5}$ 7.29 Outdoor home: PM_{10} 13.47 $PM_{2.5}$ 10.47	Method: HI, Units = μg/m^3 PM_{10} 18.0 $PM_{2.5}$ 14.0
Koenig et al. (2003, 156653) Seattle, WA	13.4 ± 3.2 μg/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 (μg/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 (μg/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 (μg/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
Mar et al. (2005, 087566) Seattle, WA USA	Method: HI, Units = μg/m^3 $PM_{2.5}$: Healthy: 9.3 (8.4) CVD: 10.8 (8.4) COPD: 10.5 (7.2)	Method: HI, Units = μg/m^3 $PM_{2.5}$: 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, Units = μg/m^3 $PM_{2.5}$: 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)
Trenga et al. (2006, 155209) Seattle, Washington	Method: PEM, Units = μg/m^3 Median $PM_{2.5}$ Child 11.3 Adult 8.5	Method: HI, Units = μg/m^3 Median $PM_{2.5}$ Child 7.5 Adult 7.6	Method: HI, Units = μg/m^3 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
Wu et al. (2006, 179950) Pullman, WA	During non-burning times: 13.8 (11.1) During burning episodes: 19.0 (11.8)		
SOUTHCENTRAL			
Turpin et al. (2007, 157062) Houston (and Elizabeth, NJ, and Los Angeles County, CA)	Houston, Units = μg/m^3 (48-hr avg) Child: 36.6 Adult: 37.2	Houston: 17.1	Houston: 14.7

Reference / Location	Personal	Micro	Ambient
MIDWEST			
Adgate et al. (2002, 030676) Battle Creek, East St. Paul, and Phillips, Minnesota, constituting the Minneapolis-St. Paul metropolitan area.	$PM_{2.5}$, Units = $\mu g/m^3$ 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)	$PM_{2.5}$, Units = $\mu g/m^3$ 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)	$PM_{2.5}$, Units = $\mu g/m^3$ 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	$PM_{2.5}$, Units = $\mu g/m^3$ Athens (rural): 17.61 (17.81) Koebel (urban): 14.59 (13.05) New Albany (suburb): 13.93 (12.25)	$PM_{2.5}$, Units = $\mu g/m^3$ Indoor Athens (rural): 17.20 (13.56) Koebel (urban): 14.98 (12.30) New Albany (suburb): 16.52 (13.53)	$PM_{2.5}$, Units = $\mu g/m^3$ Athens (rural): 13.66 (8.91) Koebel (urban): 13.89 (9.29) New Albany (suburb): 12.72 (8.86)
Sarnat et al. (2006, 089784) Steubenville, OH	Mean (SD): $PM_{2.5}$, Units = $\mu g/m^3$ Summer n = 169 mean (SD) = 19.9 (9.4) Fall mean (SD) = 20.1 (11.6)		Mean (SD): $PM_{2.5}$, Units = $\mu g/m^3$ Summer n = 65 mean (SD) = 20.1 (9.3) Fall mean (SD) = 19.3 (12.2)
SOUTHEAST			
Wallace and Williams (2005, 057485) Raleigh, North Carolina	Units = $\mu g/m^3$ $PM_{2.5}$ pers = 23.0 (16.4) $PM_{2.5}$ pers/$PM_{2.5}$ out = 1.31 (0.99)	Units = $\mu g/m^3$ $PM_{2.5}$ in = 19.4 (16.5) $PM_{2.5}$ in/$PM_{2.5}$ out = 1.08 (1.05)	Units = $\mu g/m^3$ $PM_{2.5}$ 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. (b) measured 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. (b) measured using PEMs. (c) measured using HI samplers. (d) measured 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 $PM_{10-2.5}$ (d) 210, 8.6, 10.0, 62.3 (a) Relative standard deviation of the presented arithmetic mean. (b) measured using PEMs. (c) measured using HI samplers. (d) measured by difference in PEM PM_{10} monitor and co-located HI $PM_{2.5}$ mass concentrations.

Reference / Location	Personal	Micro	Ambient
NORTHEAST			
Koutrakis et al. (2005, 095800) Baltimore, MD Boston, MA	$PM_{2.5}$, Units = $\mu g/m^3$: (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	NR	$PM_{2.5}$, Units = $\mu g/m^3$: (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)
Sarnat et al. (2005, 087531) Boston, Massachusetts. Comparisons to a previous study in Baltimore are made.	Units = $\mu g/m^3$: 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	Units = $\mu g/m^3$: 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
Turpin et al. (2007, 157062) Elizabeth, NJ, (and Houston, TX, and Los Angeles County, CA+	48-h avg $PM_{2.5}$, Units = $\mu g/m^3$: Elizabeth Child: 54.0 Adult: 44.8	Elizabeth: 20.1	Elizabeth: 20.4

Table A-62. Summary of PM species exposure studies.

Reference	Particle Sizes Measured	Component	Results	Primary Findings
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: Outdoor, Indoor, Personal S 334.4,272.1,351.6 Ca 232.2,85.0,174.1 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 NiNA -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 ambient 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.
Brunekreef et al. (2005, 090486)	Personal, Micro & Ambient: $PM_{2.5}$	NO_3^-	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.
Brunekreef et al. (2005, 090486)	Personal, Micro, and Ambient: $PM_{2.5}$	SO_4^{2-}, NO_3^-	Mean, units = $\mu g/m^3$: SO_4^{2-}: P, I, O Amsterdam 4.6 4.7 5.9 Helsinki 2.7 3.0 5.0 NO_3^-: P, I, O Amsterdam 1.4 1.4 4.0 Helsinki 0.2 0.3 1.3	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.
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 of the elevated personal metal levels.

Reference	Particle Sizes Measured	Component	Results	Primary Findings
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 SO_4^{2-}, 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) B[a]P cPAHs	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) 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)	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 higher.
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-SMWinter Summer EOM (µg/m^3) 14.93 4.96 EOM2 (%)23.9 13.4 B[a]P (µg/m^3)3.5 0.28 c-PAHsb (µg/m^3) 24.69 2.29 Prague-LB Winter Summer EOM (µg/m^3) 10.86 3.72 EOM2 (%)27.9 14.1 B[a]P (µg/m^3)2.9 0.17 c-PAHsb (µg/m^3) 20.36 1.32 Košice Winter Summer EOM (µg/m^3) 15.3 1.67 EOM2 (%)26.4 6.9 B[a]P (µg/m^3)1.37 0.15 c-PAHsb (µg/m^3) 11.87 1.2 Sofia Winter Summer EOM (µg/m^3) 24.6 3.95 EOM2 (%)27.37 13.3 B[a]P (µg/m^3)4.84 0.36 c-PAHsb (µg/m^3) 36.44 2.43	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.
Gadkari et al. (2007, 156459)	Personal: Respirable PM (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 personal exposure to ambient RPM is related to local traffic and soil resuspension. They felt that indoor activities or ventilation determined indoor levels of RPM.

Reference	Particle Sizes Measured	Component	Results	Primary Findings
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 = $\mu g/m^3$: 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)	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.
Hanninen et al. (2004, 056812)	Personal: $PM_{2.5}$ Micro: NR Ambient: $PM_{2.5}$	$PM_{2.5}$ -bound S	Indoor Outdoor Athens 5.3 (2.0) 7.6 (5.1) Basel 2.6 (1.6) 3.3 (1.6) Helsinki 1.6 (1.3) 2.2 (1.5) Prague 3.1 (1.3) 4.0 (1.5)	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.
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.
Jacquemin et al. (2007, 192372)	Personal: $PM_{2.5}$ Micro: NA Ambient: $PM_{2.5}$	S	Mean, units = $\mu g/m^3$: 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."
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 = $\mu g/m^3$: Amsterdam, Helsinki P,O,P,O $PM_{2.5}$ 14.5, 15.7, 9.4, 11.4 Abs 1.4, 1.6, 1.3, 1.9 S 912.3, 1299.9, 605.3, 1435.7 Zn 13.2, 18.3, 11.7, 18.6 Fe 57.0, 71.3, 41.6, 79.2 K 87.4, 70.3, 103.1, 93.9 Ca 72.9, 40.2, 68.5, 36.4 Cu 5.4, 2.5, 4.3, 1.8 Si 29.7, 13.7, 79.5, 93.9 Cl 40.8, 72.7, 9.8, 44.2	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).

Reference	Particle Sizes Measured	Component	Results	Primary Findings
Johannesson et al. (2007, 156614)	Personal, Micro, and Ambient: $PM_{2.5}$, PM_1	BS	$BS_{2.5}$ 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 PM_1/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. 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.
Kim et al. (2005, 156640)	Personal: $PM_{2.5}$ Micro: NR Ambient: $PM_{2.5}$	SO_4^{2-}, EC, Ca^{2+}, Mn^{2+}, K, Na^+	Mean (SD), Units = $\mu g/m^3$: 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.
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 - 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 * 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.

Reference	Particle Sizes Measured	Component	Results	Primary Findings
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^{2-}: (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^{2-} are strong predictors of respective personal exposures. Ambient SO_4^{2-} is a strong predictor of personal exposure to $PM_{2.5}$. Because $PM_{2.5}$ has substantial indoor sources and SO_4^{2-} does not, the investigators concluded that personal exposure to SO_4^{2-} 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)	Personal: PM_5 Micro: NR Ambient: PM_5	Pb Ni Cd Cu Cr Fe Mn	Personal samples, Units = $\mu g/m^3$: Mean ± SD Type Pb 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 $\mu g/m^3$ Cd 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 $\mu g/m^3$ Mn 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 $\mu g/m^3$ K Occupational 3.473 ± 4.691 $\mu g/m^3$ Residential 4.589 ± 4.619 $\mu g/m^3$ 24-h integrated Check	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
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 280 (7.0), 67 (7.2), 22 (2.9), 110 (7.5), 1.4 As 4.7 (1.6), 3.7 (1.8), 2.6 (2.7), 6 (—), 1.4 Br 4.7 (2.2), 3.9 (2.0), 2.4 (2.5), 6.2 (2.5), 1.6 Ca 260 (2.0), 120 (2.1), 30 (1.6), 280 (2.9), 3.3 Cd 23 (1.4), 19 (1.8), 7 (—), 43 (2.2), — Cl 400 (3.0), 270 (3.9), 220 (5.2), 380 (3.9), 1.0 Cu 120 (1.3), 88 (1.7), 2.3 (2.8), 230 (2.1), 37.1 Fe 59 (2.3),30 (3.8), 19 (3.5), 85 (2.9), 1.6 Ga 0.9 (2.1), 0.6 (2.2), 0.2 (2.2), 2.0 (3.4), 2.4 K 250 (2.4), 180 (2.7), 93 (2.0), 130 (4.0), 1.7 Mg 260 (2.1), 130 (3.1), 140 (2.9), 120 (2.8), 0.7 Mn 2.1 (2.6), 1.8 (2.4), 2.2 (1.5), 3.5 (3.0), 0.8 Na 2100 (1.6) ,1800 (1.7), 1100 (3.2), 2700 (1.9), 1.6 Ni 11 (2.2), 8.6 (2.5), 18 (—), 23 (2.9),— P 110 (2.1), 70 (2.2), 27 (1.8), 86 (2.4), 2.5 Pb 26 (1.7), 19 (1.8), 9.4 (2.8), 32 (2.0), 1.9 S 1200 (1.9), 1200 (2.0), 890 (4.8), 1.2 Se 8.4 (1.5), 6.8 (1.7), 2.3 (1.8), 16 (2.2), 2.8 Si 740 (3.4), 360 (2.9), 95 (2.2), 570 (3.8), 2.6 Sn 35 (1.5), 27 (1.8), 0 (—), 68 (2.6), — Ti 6.2 (1.7), 2.8 (2.2), 1.1 (2.0), 6.1 (3.2), 2.3 V 1.8 (1.5), 1.4 (1.9), 4 (—), — Zn 18 (2.4), 15 (2.2), 13 (2.5), 23 (2.4), 0.9	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
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 Al, As, Br, Ca, Cl, Cr, Cu, Fe, K, Mn, Ni, Pb, Si, S, Ti, V	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, 1105, 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 0 ,0, 1, 1 Pb 2, 2, 5, 5 Si 109, 65, 66, 62 S 289, 289, 468, 492 Ti 4, 3, 3, 6 V 0, 1, 2, 3	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.
Maitre et al. (2002, 156726)	Personal: PM_4 Micro: NR Ambient: PM_4	PAH, benxene-toluene-xylenes (BTX), aldehydes, BaP PAHc, formaldehyde, acetaldehyde	Median Personal Ambient Resp µg/m^3 124, 124 (mean) BaP ng/m^3 0.28,0.14 PAHc ng/m^3 1.19,1.56 PAH ng/m^3 13.14,12.26 Benzene µg/m^3 23.5, 17 Toluene µg/m^3 94.5, 52 Xylene µg/m^3 74, 39 BTX µg/m^3 192, 108 Formaldehyde µg/m^3 21, 17.5 Acetaldehyde µg/m^3 17, 10.5 Aldehyde µg/m^3 38, 28	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.
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) Outdoor: EC: 1144.1 (968.1) OC: 3777.7 (2520.1) S: 1232.3 (633.2) Si: 141.1 (171.3)	Use of central-site $PM_{2.5}$ as an exposure surrogate underestimates the bandwidth of the distribution of exposures to PM of ambient origin.

Reference	Particle Sizes Measured	Component	Results	Primary Findings
Molnár et al. (2005, 156772)	Personal: $PM_{2.5}$ Micro and Ambient: $PM_{10-2.5}$ and $PM_{2.5}$	BS S Cl K Ca Mn Fe Cu Zn Br Rb Pb	Median, unit = ng/m^3 Wood burners Ref 1-sided p-value BS 0.97, 0.74, 0.053 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	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.
Molnar et al. (2006, 156773)	Personal: $PM_{2.5}$ and PM_1 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 (ng/m^3) 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 ($\mu g/m^3$) 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 PM_1 Mean, median, range ($\mu g/m^3$) 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	$PM_{2.5}$ 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 than 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
			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 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 PM_{1} 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	
Na and Cocker (2005, 156790)	Personal: $PM_{2.5}$ Micro: NR Ambient: $PM_{2.5}$	EC, OC	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.
Noulett et al. (2006, 155999)	Personal: $PM_{2.5}$ Micro: NR Ambient: $PM_{2.5}$	SO_4^{2-} ABS (light absorbing carbon)	Measurement Mean s.d. Ambient $_{SO4}^{2-}$ 2.72* 3.11 Ambient ABS 1.4** 1.0 Personal SO_4^{2-} 1.33* 1.47 Personal ABS 1.0** 1.7 * Mean SO_4^{2-} values reported in µg/m^3 ** Mean ABS values reported in 10-5/m^{-1}	SO_4^{2-} and light absorbing carbon concentrations had higher personal-ambient correlations and less variability. This indicates that SO_4^{2-} and ABS were of outdoor origin, while $PM_{2.5}$ mass was of varied indoor and outdoor origin.

Reference	Particle Sizes Measured	Component	Results	Primary Findings
Salma et al. (2007, 113852)	Personal: $PM_{10-2.0}$ and $PM_{2.0}$ 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.
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. NOX 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.
Sarnat et al. (2006, 089784)	Personal: $PM_{2.5}$ Micro: NR Ambient: $PM_{2.5}$	SO_4^{2-} EC	Mean (SD), units = $\mu g/m^3$: Personal Ambient SO_4^{2-} 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.
Shilton et al. (2002, 049602)	Personal, Micro, and Ambient: Respirable PM	Respirable PM, metals (Zn, Cu, Mn, Al), SO_4^{2-}, NO_3^-, and Cl^-	IndoorOutdoor 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^{2-} (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;

Reference	Particle Sizes Measured	Component	Results	Primary Findings
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.	EC OC	Work Area EC, OC, EC/TC Office 0.31 (3.72), 11.29 (1.63) Dock 0.53 (3.24), 5.01 (1.76), 3% (3.10) Yard 0.73 (2.89), 7.77 (1.65), 9% (2.49) Shop 1.54 (3.52), 10.37 (2.00), 8% (2.21) Non-smokers on-site: 12% (2.13) Clerk 0.09 (9.98), 15.97 (1.31) Dock worker 0.76 (2.13), 13.89 (1.45),1% (10.19) Mechanic 2.00 (3.82), 16.89 (1.64), 5% (1.96) Hostler 0.88 (3.04), 14.89 (1.86), 10% (2.71) Non-smokers off-site 5% (2.09) Pickup/deliver driver 1.09 (2.46), 12.40 (1.54) Long haul driver 1.12 (1.91), 19.26 (2.30),8% (2.13) Smokers On-Site 7% (1.82) Clerk 1.19 (1.70), 32.25 (1.70), NR Dock worker 0.98 (1.93), 24.02 (1.87) Mechanic 2.41 (2.27), 24.35 (1.78) Hostler 1.74 (2.21), 43.92 (2.03) Smokers off-site Pickup & Delivery drivers 1.33 (3.84), 24.24 (2.14) Long haul drivers 1.37 (2.40),32.81 (3.23)	

Reference	Particle Sizes Measured	Component	Results	Primary Findings
Sørensen et al. (2003, 157000)	Personal: $PM_{2.5}$ Micro: NR Ambient: $PM_{2.5}$	BS	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.
Sorenson et al. (2005, 089428)	Personal: $PM_{2.5}$ and BS Micro: $PM_{2.5}$ and BS Ambient: Street monitoring station and roof of a campus building $PM_{2.5}$ and BS	BS	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: 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)	Indoor sources of PM and BS were shown to be greatly influenced by indoor sources.
Sram et al. (2007, 192084)	Personal: PM_{10}, $PM_{2.5}$ Micro: NR Ambient: PM_{10}, $PM_{2.5}$	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 ng/m^3	Ambient air exposure to c-PAHs increased fluorescent in situ hybridization (FISH) cytogenetic parameters in non-smoking policemen exposed to ambient PM

Reference	Particle Sizes Measured	Component	Results	Primary Findings
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 (µgC/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%.
Wallace and Williams (2005, 057485)	Personal: $PM_{2.5}$ Indoor Micro: $PM_{2.5}$ Outdoor Micro: $PM_{2.5}$	S	Mean (SD), units = ng/m^3: Personal: 1046 (633) Indoor: 1098 (652) Outdoor: 1951 (1137)	Generally, F_{inf} provides a reliable estimate of personal exposure. S can be used in lieu of personal exposure to PM because it is generally derived from outdoors.
Wu et al. (2006, 179950)	Personal: $PM_{2.5}$ Micro: $PM_{2.5}$ Ambient: $PM_{2.5}$	LG EC OC	Mean personal exposure (µg/m^3): 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.

Reference	Particle Sizes Measured	Component	Results	Primary Findings
Zhao et al. (2007, 156182)	Personal, Micro, and Ambient: $PM_{2.5}$	EC, Cl, Si, NO_3	Units = $\mu g/m^3$: 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 NO_3^- and motor vehicle exhaust 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.

Table A-63. Summary of personal PM exposure source apportionment studies.

Reference	Study Design	Results	Primary Findings
Hopke et al. (2003, 095544)	Source apportionment of personal and indoor central and apartment and outdoor $_{PM2.5}$. Baltimore retirement home with 10 elderly subjects. July-Aug 1998.	% control P,I, C, O External Secondary SO_4^{2-} 46.3, 64.0, 79.0, 64.0 Unknown 13.6, 14.5, 17.4, 14.5 Soil 2.8, 3.1, 3.6, 3.1 Internal Gypsum 0.7, 0.4, 0.0, 0.0 Activity 36.2, 17.8, 0.0, 0.0 Personal care 0.4, 0.3, 0.0, 0.0	63% of personal exposure could be attributed to outdoor sources (with 46% from SO_4^{2-}), 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 and residences and central outdoor $PM_{2.5}$ around Seattle with 10 elderly subjects and 10 asthmatic children. The purpose of the article was to compare PMF2 and PMF3 methods. Seattle Sep 2000 and May 2001	PMF2: % control P,I,O Veg burn 28.8, 47.6, 56.7 Mobile 0.0, 3.6, 7.5 Fuel oil 0.0, 0.0, 6.7 S, Mn, Fe 8.1, 0.0, 0.0 Secondary 0.0, 34.5, 20.9 Cl-rich 9.9, 3.6, 3.7 Crustal 25.2, 10.7, 4.5 Crustal 2 27.9, 0.0, 0.0 PMF3: % control P,I, O Veg burn 41.0, 57.4, 71.3 Mobile 7.2, 4.3, 8.2 Secondary 19.3, 13.8, 18.0 Crustal 32.5, 24.,5 2.5	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 and residential indoor and residential outdoor and central outdoor $PM_{2.5}$, Raleigh and Chapel Hill NC with 38 subjects. Summer 2000 and Spring 2001.	% control P, I, R,O Motor vehicle 10.0, 9.4, 17.2, 19.4 Soil 3.5, 3.7, 9.3, 8.5 Secondary SO_4^{2-} 15.9, 22.5, 59.3, 61.9 Secondary NO_3^- 4.4, 4.7, 7.6, 7.8 ETS 7.0, 10.0, 0.0, 0.0 Personal care and activity 8.0, 19.1, 0.0, 0.0 CU-factor mix with indoor soil 0.4, 1.2, 0.0, 0.0 Cooking 52.5, 53.6, 0.0, 0.0	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, 194618)	Source apportioned infiltration for personal and residential indoor and residential outdoor and central outdoor $_{PM2.5}$. 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 Indoor (Outdoor Origin) Mechanically generated 2, 17 Primary Combustion 43, 43 Secondary Formation* 55, 40 *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 and residential indoor and residential outdoor and central outdoor $PM_{2.5}$, $PM_{2.5}$ samples from 219 homes were used for this analysis. 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).	SO_4^{2-}: R, O, I, P O 1.0 I 0.54-0.76 1.0 P 0.54-0.73 0.84-0.90 1.0 C = O: R, O, I, P O 1.0 I 0.12-0.61 1.0 P -0.13-0.69 0.07-0.77 1.0 CH: R, O, I, P O 1.0 I -0.08-0.35 1.0 P -0.07-0.19 0.41-0.85 1.0	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 and indoor school and outdoor school $PM_{2.5}$. Denver with 56 asthmatic children. Oct 2002-March 2003 and Oct 2003-March 2004.	% contr P IO Secondary SO_4^{2-} 4.3 8.9 9.6 Soil 6.6 4.2 12.4 Secondary NO_3^- 9.4 2.8 40.8 Motor vehicle 13.3 26.5 26.5 Cl-based cleaning 2.8 0.4 0.0 Cooking 54.8 30.2 0.0 ETS 9.2 2.1 0.0	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-}Fe components. Denver. Winter 1999-2000 and 2000-2001.	Estimation method, Mean (SD, range): PMF: 7.42 (1.93, 3.43-12.89) Extrapolation Method: Using SO_4^{2-}: 6.38 (1.60, 3.20-10.97) Using SO_4^{2-} and Fe: 6.50 (1.36, 3.54-10.12) Using SO_4^{2-} and Fe, temperature adjusted: 7.02 (1.48, 3.79-11.02) Using SO_4^{2-} (no gamma): 8.23 (2.06, 4.12-14.14)	Similar results were found with each technique.

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Table A-64. 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 copollutants. 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 of 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 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	$PM_{2.5}$ (mean, coefficient of variation (CV)): 1.14 (0.71) EC: 0.89 (0.64) Ca: 1.16 (1.90) Fe: 0.69 (1.40) K: 1.10 (0.95) Si: 1.04 (1.31) Na: 1.05 (1.84) Cl: 3.18 (3.79) Zn: 0.83 ± (1.13) S: 0.76 ± (0.32) V: 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	$PM_{2.5}$: Open Windows: 0.98 Closed Windows: 0.64 EC: 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 of 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 $_{PM2.5}$: 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
Diapouli et al. (2008, 190893)	Objective: To characterize the PM_{10f} $PM_{2.5}$, 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)
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 Finf and a correction factor that results from division of BPM (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}: SO_4^{2-}: 0.3, NO_3^-: 0.1, Cl^-: 0.6, Na^{2+}: 0.9, NH_4^+: 0.1, Mg: 0.6, Ca^{2+}: 1.4, EC: 0.7, OC: 1.1 $PM_{2.5}$: SO_4^{2-}: 0.4, NO_3^-: 0.2, Cl^-: 0.5, Na^{2+}: 0.6, NH_4^+: 0.3, Mg: 0.5, Ca^{2+}: 1.6

Reference	Study Design	F_{inf}	I/O
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 S (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 S: 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
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 SO_4^{2-}: 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

Reference	Study Design	F_{inf}	I/O
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.	NO_3^-- SO_4^{2-}: 0.03 SO_4^{2-}: 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 BS: 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 S or Pb): 0.4-0.9	S (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) Pb (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, 195017)	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 BS: 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 S: 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.	SO_4^{2-}: 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
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-65. Summary of PM – copollutant exposure studies.

<table>
<tr><th>Reference</th><th>PM metric</th><th>Copollutant metric</th><th>Association between PM and copollutant</th><th>Primary findings</th></tr>
<tr><td>Fruin et al. (2008, 097183)</td><td>In-vehicle UFP, BC, PM-bound PAH</td><td>In-vehicle NO_X, CO</td><td>
<table>
<tr><th>R</th><th>UFP</th><th>$PM_{2.5}$</th><th>NO</th><th>BC</th><th>CO</th><th>CO_2</th></tr>
<tr><td>UFP</td><td>1</td><td>0.71</td><td>0.97</td><td>0.95</td><td>0.63</td><td>0.72</td></tr>
<tr><td>$PM_{2.5}$</td><td></td><td>1</td><td>0.69</td><td>0.89</td><td>0.66</td><td>0.68</td></tr>
<tr><td>NO</td><td></td><td></td><td>1</td><td>0.91</td><td>0.78</td><td>0.85</td></tr>
<tr><td>BC</td><td></td><td></td><td></td><td>1</td><td>0.65</td><td>0.74</td></tr>
<tr><td>CO</td><td></td><td></td><td></td><td></td><td>1</td><td>0.94</td></tr>
<tr><td>CO_2</td><td></td><td></td><td></td><td></td><td></td><td>1</td></tr>
</table>
Note that these correlations are computed from data presented by Fruin et al. (2008, 097183) for mean concentrations at different loc ations.
</td><td>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.</td></tr>
<tr><td>Schwartz et al. (2007, 090220)</td><td>Ambient and personal $PM_{2.5}$ data from the Baltimore panel study</td><td>Ambient and personal O_3 and NO_2 data from the Baltimore panel study.</td><td>Median β for regressions:
<table>
<tr><th></th><th>Ambient $PM_{2.5}$</th><th>Ambient O_3</th><th>Ambient NO_2</th></tr>
<tr><td>Personal $PM_{2.5}$</td><td>0.0143</td><td>-0.0016</td><td>0.0115</td></tr>
<tr><td>Personal $PM_{2.5}$ of ambient origin</td><td>0.0183</td><td>-0.0037</td><td>0.0124</td></tr>
<tr><td>Personal SO_4^{2-}</td><td>0.0051</td><td>0.0035</td><td>0.0006</td></tr>
<tr><td>Personal O_3</td><td>0.0014</td><td>0.0010</td><td>0.0009</td></tr>
<tr><td>Personal NO2</td><td>0.0015</td><td>0.0009</td><td>0.0010</td></tr>
</table>
</td><td>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.</td></tr>
<tr><td>Tolbert et al. (2007, 090316)</td><td>Ambient PM_{10}, $PM_{10-2.5}$, $PM_{2.5}$, EC, OC, TC, SO_4^{2-}, water-soluble metals, oxygenated hydrocarbons</td><td>Ambient O_3, NO_2, CO, SO_2</td><td>
<table>
<tr><th></th><th>PM_{10}</th><th>O_3</th><th>NO_2</th><th>CO</th><th>SO_2</th><th>PMc</th><th>$PM_{2.5}$</th></tr>
<tr><td>PM_{10}</td><td>1.0</td><td></td><td></td><td></td><td></td><td></td><td></td></tr>
<tr><td>O_3</td><td>0.6</td><td>1.0</td><td></td><td></td><td></td><td></td><td></td></tr>
<tr><td>NO_2</td><td>0.5</td><td>0.4</td><td>1.0</td><td></td><td></td><td></td><td></td></tr>
<tr><td>CO</td><td>0.5</td><td>0.3</td><td>0.7</td><td>1.0</td><td></td><td></td><td></td></tr>
<tr><td>SO_2</td><td>0.2</td><td>0.2</td><td>0.4</td><td>0.3</td><td>1.0</td><td></td><td></td></tr>
<tr><td>PMc</td><td>0.7</td><td>0.4</td><td>0.5</td><td>0.4</td><td>0.2</td><td>1.0</td><td></td></tr>
<tr><td>$PM_{2.5}$</td><td>0.8</td><td>0.6</td><td>0.6</td><td>0.4</td><td>0.2</td><td>0.5</td><td>1.0</td></tr>
<tr><td>SO_4^{2-}</td><td>0.7</td><td>0.6</td><td>0.1</td><td>0.1</td><td>0.1</td><td>0.3</td><td>0.8</td></tr>
<tr><td>EC</td><td>0.6</td><td>0.4</td><td>0.6</td><td>0.7</td><td>0.2</td><td>0.5</td><td>0.7</td></tr>
<tr><td>OC</td><td>0.7</td><td>0.5</td><td>0.6</td><td>0.6</td><td>0.2</td><td>0.5</td><td>0.7</td></tr>
<tr><td>TC</td><td>0.7</td><td>0.5</td><td>0.7</td><td>0.6</td><td>0.2</td><td>0.5</td><td>0.7</td></tr>
<tr><td>Metals</td><td>0.7</td><td>0.4</td><td>0.3</td><td>0.4</td><td>0.1</td><td>0.5</td><td>0.7</td></tr>
<tr><td>OHC</td><td>0.5</td><td>0.4</td><td>0.2</td><td>0.3</td><td>0.1</td><td>0.4</td><td>0.5</td></tr>
<tr><td>SO_4^{2-}</td><td>EC</td><td>OC</td><td>TC</td><td>Metals</td><td>OHC</td><td></td><td></td></tr>
<tr><td>SO_4^{2-}</td><td>1.0</td><td></td><td></td><td></td><td></td><td></td><td></td></tr>
<tr><td>EC</td><td>0.3</td><td>1.0</td><td></td><td></td><td></td><td></td><td></td></tr>
<tr><td>OC</td><td>0.3</td><td>0.8</td><td>1.0</td><td></td><td></td><td></td><td></td></tr>
<tr><td>TC</td><td>0.3</td><td>0.9</td><td>1.0</td><td>1.0</td><td></td><td></td><td></td></tr>
<tr><td>Metals</td><td>0.7</td><td>0.5</td><td>0.5</td><td>0.5</td><td>1.0</td><td></td><td></td></tr>
<tr><td>OHC</td><td>0.5</td><td>0.4</td><td>0.4</td><td>0.4</td><td>0.5</td><td>1.0</td><td></td></tr>
</table>
</td><td>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.</td></tr>
<tr><td>Brook et al. (2007, 091153)</td><td>Anbient PM_{10}, $PM_{10-2.5}$, $PM_{2.5}$, SO_4^{2-}, and trace metals in 10 Canadian cities.</td><td>Ambient NO_2, NO</td><td>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)</td><td>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.</td></tr>
</table>

Reference	PM metric	Copollutant metric	Association between PM and copollutant	Primary findings
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: $PM_{2.5}$ UFPCO $PM_{2.5}$ 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.
Lai et al. (2004, 056811)	Microenvironmental and personal $PM_{2.5}$ and trace elements for personal exposure (P), residential indoor (RI), residential outdoor (RO), and workplace (WI) measurements.	Microenvironmental and personal VOCs, NO_2, and CO.	| R ($PM_{2.5}$) | P | RI | RO | WI | | --- | --- | --- | --- | --- | | TVOC | 0.21 | 0.21 | 0.41 | -0.32 | | NO_2 | -0.1 | -0.02 | -0.16 | 0.09 | | CO | -0.07 | NR | NR | NR |	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. (2004, 054418; 2007, 138816)	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.

Reference	PM metric	Copollutant metric	Association between PM and copollutant	Primary findings
Sarnat et al. (2001, 019401)	Fixed site and personal $PM_{2.5}$ monitors.	Ambient O_3, NO_2, SO_2, and CO	R: $PM_{2.5}$ 1; O_3 0.67; NO_2 0.37; SO_2 ---; CO 0.15 $PM_{2.5}$: O_3 -0.72; NO_2 0.75; SO_2 -0.17; CO 0.69 O_3: NO_2 -0.71; SO_2 0.41; CO -0.67 NO_2: SO_2 -0.17; CO 0.76 SO_2: CO -0.12 CO: 1	Strong association between ambient NO_2 and personal $PM_{2.5}$ suggests that ambient gas may be a suitable surrogate for personal exposure.

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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 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 yr)	Carbon - 99mTc 108 nm CMD (σg = 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 yr) Smokers (n = 10; 51 ± 10 yr) COPD patients (n = 7; 69 ± 10 yr)	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. 81mKr 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 yr) 6 healthy 5 asthmatic 4 smokers	Carbon - 99mTc 87 nm CMD (σg = 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.
Wiebert et al. (2006, 157146)	Healthy subjects (4M, 5F; 56 ± 9 yr) Asthmatics (2M, 3F; 59 ± 6 yr) Control (1M; 50 yr)	Carbon - 99mTc 34 nm CMD (σg = 1.5) Technegas Generator	Slow deep aerosol inhalations with 10 s 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.

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).

Table B- 2. Ultrafine disposition in animals.

Reference	Study Group	Aerosol	Study Protocol	Observations
Bermudez et al.(2004, 056707)	Fischer 344 rats, females (6 wk) B3C3F1 mice, females (6 wk) Hamsters, females (6 wk)	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 wk 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) post-exposure. 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 (~2× control groups) out to 52 wk without signs of recovery. Epithelial permeability was 3-4× control in high exposed rats through 4 wk post exposure, but approached control by 13 wk . 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 in vitro studies	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 min and of 253-675 nm by 50-60 min 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 min. At 50-60 min, 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 (106 cell/mLuman red blood cells (RBC; 8 × 106 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 wk 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.

Reference	Study Group	Aerosol	Study Protocol	Observations
Dorman et al. (2004, 155752)	Crl: CD rats Males (6 wk 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 wk) 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 × 106 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 MnCl2 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 MnCl2 (8.2 ± 3.6%) as a percent of the amount instilled.
Oberdörster et al. (2004, 055639)	Fisher 344 rats Males (14 wk; 284 ± 9 g)	13C (36 nm CMD, 1.7 σg) 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 13C analysis. Tissue 13C-levels were determined by isotope ratio mass spectroscopy and background corrected for 13C levels in unexposed controls (n = 3).	At 1 day postexposure, the lungs of rats exposed to ultrafine 13C particles contained 1.34 ± 0.22 µg of 13C (1.39 µg/g-lung) following background corrected. By 7 days postexposure, the 13C concentration had decreased to 0.59 µg/g-lung. There was a significant and persistent increase in 13C 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 13C 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)	65ZnCl2 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 wk postexposure. Pike: instilled (0.12 µg Zn in 10 µL) in right or both olfactory chambers, assayed 2 wk 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.
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 1 mo.	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 wk 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 of age.

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 yr) between 11 and 90 yr 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 yr of age (9.3 ± 5.2 min) versus older subjects (15.4 ± 5.0 min). 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 yr) and ten elderly (2 M, 5 F; 63 ± 5 yr) 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 min. 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.
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 yr, mean 30 yr) 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 ~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 yr olds vs. 4.6 ± 3.2 mm/min in individuals >30 yr of age. However, it should be noted that only 2 subjects were greater than 45 yr 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 yr 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 yr) had 1-h clearance of 34 ± 14% which was significantly greater than the 22 ± 8% found in the older subjects (n = 5; >54 yr). Separated by 5.4 wk (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 yr, n = 13; 25-29 yr, n = 8; 30-49 yr, n = 7; 50-64, n = 9; >65 yr, 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 wk 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 wk, and 3 wk, 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.

Reference	Animal	Particles	Study Protocol	Observed Effect(s)
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 yr) 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-mo-old rats compared to 74 days in 23-mo-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 yr	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 µg/m^3; Session 2: median 243 µg/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 µg/m^3), acetaldehyde (75 µg/m^3), benzene (30 µg/m^3), 1,3-butadiene (6.3 µg/m^3); Session 2: NO_2 (0.09 ppm), CO (9.1 ppm), formaldehyde (64 µg/m^3), acetaldehyde (40 µg/m^3), benzene (20 µg/m^3), 1,3-butadiene (3.9 µg/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-prostaglandin2α 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 yr	Ultrafine and fine zinc oxide **Particle Size:** UF: <0.1 µm; Fine: 0.1-1.0 µm **Particle Number/Count:** UF: 4.6×10^7/cm^3; Fine: 1.9×10^5/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 wk. **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 yr	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 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.
Reference: Brauner et al. (2007, 091152) **Subjects:** 29 healthy adults **Gender:** 20 M/9 F **Age:** 20-40 yr	Urban traffic particles **Particle Number/Count:** 6–700nm: 10,067/cm^3 **Concentration:** $PM_{2.5}$: 9.7 µg/m^3; $PM_{10\text{-}2.5}$: 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 O_3 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.

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. (2008, 156293) **Subjects:** 42 healthy older adults (21 couples) **Age:** 60-75 yr	Indoor air particles **Particle Number/Count:** 10-700 nm: 10,016/cm^3 **Concentration:** Coarse: 9.4 µg/m^3; Fine: 12.6 µg/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 µg/m^3,, and fine concentration from 12.6 to 4.7 µg/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-α, 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 yr, F avg 26 yr	Urban traffic particles **Particle Number/Count:** 11,600/cm^3 **Concentration:** $PM_{2.5}$: 10.5 µg/m^3; $PM_{10-2.5}$: 13.8 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, while O_3 concentrations were lower with filtered air. **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- α) 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 yr	DE 2002 Cummins B-series diesel engine (6BT5.9G6, 5.9 L) operating at load **Concentration:** Fine PM: 100, 200 µg/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 wk. 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 were observed at 6 h following the start of exposure (4 h post-exposure). No diesel-induced increase in C-reactive protein observed 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 yr	DE 2002 Cummins B-series diesel engine (6BT5.9G6, 5.9 L) **Concentration:** Fine PM: 100, 200 µg/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 wk. 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 µg/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.
Reference: Danielsen et al. (2008, 156382) **Subjects:** 13 healthy adults **Gender:** 6 M/7 F **Age:** 20-56 yr	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 µg/m^3; Session 2: median 243 µg/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 µg/m^3), acetaldehyde (75 µg/m^3), benzene (30 µg/m^3), 1,3-butadiene (6.3 µg/m^3); Session 2: NO_2 (0.09 ppm), CO (9.1 ppm), formaldehyde (64 µg/m^3), acetaldehyde (40 µg/m^3), benzene (20 µg/m^3), 1,3-butadiene (3.9 µg/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 yr	Fine CAPs (Chapel Hill, NC) **Concentration:** Mean: 40.5 µg/m^3, Range: 21.2-80.3 µg/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).

Study	Pollutant	Exposure	Findings
Reference: Fakhri et al. (2009, 191914) **Subjects:** 50 adults (40 healthy, 10 asthmatic) **Gender:** 24 M/26 F **Age:** 19-48 yr	Fine CAPs (Toronto) **Concentration:** 127 ± 62 µg/m^3 with and without co-exposure to O_3 (114 ± ppb)	Exposures conducted through a facemask which covered the subject's nose and mouth. Subjects were exposed to CAPs, O_3, CAPs + O_3 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 O_3, 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 O_3 and SDNN, rMSSD, HF power and LF power (statistically significant for LF power). Diastolic blood pressure was observed to increase with exposure to CAPs + O_3, 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 yr	Ultrafine EC **Particle Size:** CMD ~25 nm **Particle Number/Count:** 10 µg/m^3: ~2.0 × 10^6/cm^3; 25 µg/m^3: ~7.0 × 10^6/cm^3; 50 µg/m^3: ~10.8 × 10^6/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 effect of ultrafine particle exposure on leukocyte counts or leukocyte expression of adhesion molecules observed in healthy subjects exposed at rest to 10 µg/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.
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 yr, Healthy: avg 73 yr	Fine CAPs (Los Angeles) **Particle Size:** 85% of mass between 0.1 and 2.5 µm **Concentration:** Mean: 194 µg/m^3, Range: 135-229 µg/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 wk. **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 yr, Healthy: avg 32 yr	Coarse CAPs (Los Angeles) **Particle Size:** 80% of mass between 2.5 and 10 µm, 20% of mass <2.5 µm **Concentration:** Mean: 157 µg/m^3, Range: 56-218 µg/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 wk. **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 yr, Healthy: 24 ± 8 yr	Ultrafine CAPs (Los Angeles) **Particle Number/Count:** 145,000/cm^3, Range 39,000-312,000/cm^3 **Concentration:** Mean-100 µg/m^3, Range-13-277 µg/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 wk. **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.

Study	Pollutant	Exposure	Findings
Reference: Graff et al. (2009, 191981) **Subjects:** 14 healthy adults **Gender:** 8 M/6 F **Age:** 20-34 yr	Coarse CAPs (Chapel Hill, NC) **Concentration:** 89 ± 49.5 µg/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 µg/m^3 increase in CAPs concentration (p = 0.01). D-dimer concentration decreased 11.3% per 10 µg/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 µg/m^3 increase in CAPs concentration. No other changes in HRV were observed following 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 yr	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, 091375) **Subjects:** 16 healthy adults **Gender:** 10 M/6 F **Age:** 19-59 yr	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 × 10^5/cm^3, < 100 nm: 0.85 × 10^5/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 wk. 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 yr	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, 180257) **Subjects:** 10 healthy adults **Gender:** 4 M/6 F **Age:** 18-40 yr	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 2 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.

Study	Pollutant	Exposure	Findings
Reference: Lundback et al. (2009, 191967) **Subjects:** 12 healthy adults **Gender:** M **Age:** 21-30 yr	DE Idling Volvo diesel engine (TD45, 4.5 L, 4 cylinders, 680 rpm) using Gasoil E10 **Particle Number/Count:** $1.26 \times 10^6/cm^3$ **Concentration:** 330 $\mu g/m^3$	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 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 $\mu g/m^3$). **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.
Reference: Mills et al. (2005, 095757) **Subjects:** 30 healthy adults **Gender:** M **Age:** 20-38 yr	DE Idling 1991 Volvo diesel engine (TD45, 4.5 L, 4 cylinders, 680 rpm) **Particle Number/Count:** $1.2 \times 10^6/cm^3$ **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 wk. 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-α observed 6 h post-exposure.
Reference: Mills et al. (2007, 091206) **Subjects:** 20 older adults with prior myocardial infarction **Gender:** M **Age:** 60 ± 1 yr	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:** $1.26 \times 10^6/cm^3$ **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 wk. 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 yr, Healthy: 54 ± 2 yr	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 wk. **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, 156853) **Subjects:** 5 healthy adults **Gender:** M **Age:** 20-31 yr	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 wk. 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 yr, Healthy: 20-42 yr	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 µg/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 wk. Other diesel emissions measured, 100 µg/m^3 exposure: NO_2 (16.5 ppb), NO (0.96 ppm), CO (0.51 ppm); 200 µg/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 µg/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 µg/m^3. Plasma levels of endothelin-1 were observed to increase following DE exposure (200 µg/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: M**etabolic syndrome: 31-48 yr, Healthy: 24-39 yr	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 µg/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 wk. Other diesel emissions measured, 100 µg/m^3 exposure: NO_2 (20.6 ppb), NO (0.95 ppm), CO (0.47 ppm); 200 µg/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 µg/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 yr	Carbon and ammonium nitrate particles **Concentration:** With co-exposure to 0.2ppm O_3: 255 µg/m^3, Without co-exposure to 0.2ppm O_3: 313 µg/m^3	Subjects exposed for 4 h with intermittent exercise (30-min periods) to filtered air, particles, and particles + O_3 in a crossover study design. Exposures were separated by at least 3 wk. **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 O_3 resulted in a significant decrease in SDNN as well as changes to both high and low frequency power normalized to the difference between total and very low frequency power.
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 yr, Healthy: 56-75 yr	Ultrafine carbon **Particle Size:** <10-300 nm; mode at 20-30 nm **Concentration:** Ultrafine carbon: 50 µg/m^3; SO_2: 200 ppb	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 SO_2, 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 yr	Gasoline emissions 2.5 hp gasoline engine running 10 s each min during exposure and in the min prior to exposure **Particle Size:** PM1.0 **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 ($p < 0.01$).

Study	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:** Ultrafine CAPs: 18-35 yr, Coarse CAPs: 18-35 yr	CAPs (Chapel Hill, NC) **Particle Size:** Ultrafine 0.049 ± 0.009 µm; Coarse 3.59 ± 0.58 µm **Concentration:** Ultrafine 47.0 ± 20.2 µg/m^3; Coarse 89.0 ± 49.5 µg/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.
Reference: Samet et al. (2009, 191913) **Subjects:** 19 healthy adults **Gender:** 10 M/9 F **Age:** 18-35 yr	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 yr	Ultrafine EC **Particle Number/Count:** 10.8 ± 1.7 × 10^5 /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 yr	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 wk. 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-α 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 yr	Fine CAPs (Toronto) **Concentration:** 150 µg/m^3 (range 101-257 µg/m^3) with 120 ppb O_3	Exposures conducted through a facemask which covered the subject's nose and mouth. Subjects were exposed to CAPs + O_3 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 + O_3 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 EC fractions of the CAPs.

Study	Pollutant	Exposure	Findings
Reference: Urch et al. (2005, 081080) **Subjects:** 23 healthy adults **Gender:** 13 M/10 F **Age:** 32 ± 10 yr	Fine CAPs (Toronto); **Concentration:** 150 µg/m^3 (range 102-214 µg/m^3) with 120 ppb O_3	Exposures conducted through a facemask which covered the subject's nose and mouth. Subjects were exposed to CAPs + O_3 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 + O_3 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 $PM_{2.5}$.
Reference: Zareba et al. **(2009, 190101)** **Subjects:** 24 healthy adults **Gender:** 12 M/12 F **Age:** 18-40 yr	Ultrafine EC **Particle Size:** Count median diameter 25 nm **Particle Number/Count:** 2×10^6/cm^3 (10 µg/m^3), 7×10^6/cm^3 (25 µg/m^3) **Concentration:** 10 µg/m^3; 25 µg/m^3	Protocol 1 (n=12, 6 M/6 F): Subjects exposed to 10 µg/m^3 UF carbon and filtered air for 2 h at rest in a randomized crossover design. Exposures were separated by at least 2 wk. Protocol 2 (n=12, 6 M/6 F): Subjects exposed to 10 µg/m^3, 25 µg/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 µg/m3 before 25 µg/m3). Exposures were separated by at least 2 wk. **Time to analysis (both protocols):** Immediately following exposure and 3.5 and 21 h post-exposure.	Exposure to 10 µg/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 µg/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 µg/m^3 at rest, none of which were statistically significant. In Protocol 2, exposure to 10 µg/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 µg/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, 154323) **Subjects:** 9 healthy adults **Gender:** 3 M/6 F **Age:** 18-35 yr	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 $PM_{10-2.5}$, non-heated $PM_{10-2.5}$, 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 yr	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 µg/m^3; Session 2: median 243 µg/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 µg/m^3), acetaldehyde (75 µg/m^3), benzene (30 µg/m^3), 1,3-butadiene (6.3 µg/m^3); Session 2: NO_2 (0.09 ppm), CO (9.1 ppm), formaldehyde (64 µg/m^3), acetaldehyde (40 µg/m^3), benzene (20 µg/m^3), 1,3-butadiene (3.9 µg/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 yr	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: Beckett et al. (2005, 156261) **Subjects:** 12 healthy adults **Gender:** 6 M/6 F **Age:** 23-52 yr	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 wk. **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 yr	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 wk. 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 yr	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	Pollutant	Exposure	Findings
Reference: Bosson et al. (2007, 156286) **Subjects:** 16 healthy adults **Gender:** 7 M/9 F **Age:** 20-28 yr	DE Idling Volvo diesel engine **Concentration:** PM 300 µg/m^3 followed by exposure to filtered air or 0.2 ppm O_3	Subjects exposed to DE for 1 h followed 5 h later by a 2-h exposure to either filtered air or O_3 (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 O_3).	The percentage of neutrophils and concentration of myeloperoxidase in induced sputum (18 h post-O_3/air exposure) was significantly higher following diesel + O_3 than diesel + air.
Reference: Bosson et al. (2008, 196659) **Subjects:** 14 healthy adults **Gender:** 9 M/5 F **Age:** 21-29 yr	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 O_3	Subjects exposed to DE or filtered air for 1h followed 5 h later by a 2-h exposure to O_3 (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 O_3 exposure when preceded by exposure to diesel, compared to O_3 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 yr, F avg 26 yr	Urban traffic particles **Particle Size:** $PM_{2.5}$, $PM_{10-2.5}$ **Particle Number/Count:** 6-700 nm: 10,067/cm^3 **Concentration:** $PM_{2.5}$: 9.7 µg/m^3, $PM_{10-2.5}$: 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 O_3 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 99mTc-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 yr	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 wk. **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: 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 yr, Healthy: avg 73 yr	Fine CAPs (Los Angeles) **Particle Size:** 85% of mass between 0.1 and 2.5 µm **Concentration:** Mean: 194 µg/m^3, Range: 135-229 µg/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 wk. **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 immediately following 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 yr, Healthy: avg 32 yr	Coarse CAPs (Los Angeles) **Particle Size:** 80% of mass between 2.5 and 10 µm, 20% of mass <2.5 µm **Concentration:** Mean: 157 µg/m^3; Range: 56-218 µg/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 wk. **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	Pollutant	Exposure	Findings
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 yr, Healthy: avg 68 yr	Fine CAPs (Los Angeles) **Concentration:** CAPs: 200 µg/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 wk. **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 yr, Healthy:24 ± 8 yr	Ultrafine CAPs (Los Angeles) **Particle Number/Count:** 145,000/cm^3, Range 39,000-312,000/cm^3 **Concentration:** Mean: 100 µg/m^3, Range: 13-277 µg/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 wk. **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 yr	Coarse CAPs (Chapel Hill, NC) **Concentration:** 89 ± 49.5 µg/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 µg/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 µg/m^3 CAPs; p = 0.05). Total protein in BAL fluid was also observed to decrease following CAPs exposure (1.8% decrease per 10 µg/m^3 CAPs). Markers of inflammation in BAL and BL fluids including IL-6, IL-8, and PGE2 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 yr	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 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: 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 yr, Healthy: avg 26 yr	DEP NIST 1650, heavy duty diesel engine **Concentration:** Untreated and treated with 0.1 ppm O_3 (48 h); 300 µg per nostril	DEP (with and without O_3 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, 091375) **Subjects:** 16 healthy adults **Gender:** 10 M/6 F **Age:** 19-59 yr	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 × 10^5/cm^3, < 100 nm: 0.85 × 10^5/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 bronchoalveolar 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	Pollutant	Exposure	Findings
Reference: Mudway et al. (2004, 180208) **Subjects:** 25 healthy adults **Gender:** 16 M/9 F **Age:** 19-42 yr	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 wk. 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 yr	Ultrafine EC **Particle Size:** CMD ~25 nm **Particle Number/Count:** 10 µg/m^3: ~2.0 × 10^6/cm^3; 25 µg/m^3: ~7.0 × 10^6/cm^3; 50 µg/m^3: ~10.8 × 10^6/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 yr	DE Idling Volvo diesel engine **Particle Number/Count:** 4.3 × 10^6/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 yr	DE Idling Volvo diesel engine **Particle Number/Count:** 4.3 × 10^6/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 yr	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: 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 yr	CAPs (Chapel Hill, NC) **Particle Size:** Ultrafine: 0.049 ± 0.009 µm, Coarse: 3.59 ± 0.58 µm **Concentration:** Ultrafine: 47.0 ± 20.2 µg/m^3, Coarse: 89.0 ± 49.5 µg/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 yr	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.	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	Pollutant	Exposure	Findings
Reference: Schaumann et al. (2004, 087966) **Subjects:** 12 healthy adults **Gender:** 4 M/8 F **Age:** Avg 27 ± 2.5 yr	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 yr, Healthy:19-42 yr	DE Volvo diesel engine **Concentration:** PM_{10} 108 µg/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 yr, Healthy: avg 34.5 yr	Aerosols of ammonium bisulfate and sulfuric acid **Particle Size:** MMD 0.3 µm **Concentration:** 200, 2,000 µg/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 wk 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 observed to affect lung function or respiratory systems following exposures to 200 or 2,000 µg/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 yr	DE Idling Volvo diesel engine (TD45, 4.5 L, 4 cylinders, 680 rpm) **Particle Number/Count:** 1.2 × 10^6/cm^3 **Concentration:** 300 µg/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.

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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 Kyoto **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 mo after L coronary artery ligation. ECG started at t0 and the DE exposure at t30 min for a 3-h 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 4-5 h 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:** 13-15 wk	**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; 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:** 13-15 wk	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:** 13-15 wk	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 and SH **Age:** 13-15 wk	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 wk **Weight:** 250-350 g	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; LPS: 350 EU/animal (0.5 mL) **Time to Analysis:** 4 h 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: 44 nm	**Route:** In Vitro **Dose/Concentration:** PM: 1, 50, 100 µg/mL; $ZnSO_4$: 50 µmol **Time to Analysis:** 30 min incubation	**Effect of EHC-93 filtrate on Ca^{2+} Uptake in Cardiomyocytes:** Both PMF and Zn^{2+} inhibited ATP or ionophore-stimulated Ca^{2+} influx in cardiomyocytes.

Study	Pollutant	Exposure	Effects
Reference: Bartoli et al. (2009, 156256) **Species:** Dog **Gender:** Female **Strain:** Mixed breed **Age:** 2-12 yr **Weight:** Average: 15.7 kg, Range: 13.6-18.2 kg	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) µg/m^3, BC: 1.3-32(7.5 ± 6.1) µg/m^3, Particle count: 3000-69300(18230 ± 13.151) particles/cm^3 **Time to Analysis:** Preanesthetized. Tracheostomy. 5 h exposures separated by minimum 1wk. Prazosin administered in 8 of 13 dogs 30-60 min 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:** 14-18 kg	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) µg/m^3, BC: 1.3-32 (7.5 ± 5.6) µg/m^3, Particle number: 3000-69300 (20381 ± 13075) particles/cm^3 **Time to Analysis:** Tracheostomy. Minimum 3 wk recovery. Acclimatized. Exposed 5 h. 2 5 min occlusions of LAD coronary artery separated by 20 min 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:** 10-12 wk	High Whole DE (HWDE); Low Whole DE (LWDE); High PM Filtered (HPMF); Low PM Filtered (LPMF) **Particle Size:** NR	**Route:** Whole-body Inhalation 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 6 h/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 mo	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 µg/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:** 10 wk	Road dust from paved surfaces (Reno, NV) Gasoline engine emissions, containing PM, NOX, 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 µg/m^3 (at dilutions of 10:1, 15:1, and 90:1) Mean concentrations of PM: 61 µg/m^3; NO_X: 18.8 ppm; CO: 80 ppm. **Time to Analysis:** 6 h/days for 3 days. Sacrificed 18 h 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:** 6-10 wk	UFPM: Ultra fine PM, EPA Chapel Hill, NC **Particle Size:** <0.1 µm	**Route:** IT Instillation **Dose/Concentration:** 100 µg in 100 µl **Time to Analysis:** 24 h 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:** 60 days	UfCB: Ultra fine carbon black Ferric 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 72 h post- exposure.	Both high/low-dose UfCB decreased ANN (normal-to-normal intervals) slightly around the 30th h, concurrent increases of LnSDNN. LnRMSSD returned to baseline levels after small initial increases. Minor effects observed after low-dose Fe and Ni instillation; 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:** 10 wk	CAPs: collected during a dust storm from Chung-Li, Taipei **Particle Size:** $PM_{2.5}$	**Route:** Nose-only Inhalation **Dose/Concentration:** 315.55 µg/m^3 **Time to Analysis:** 6 h	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:** 60 days	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 ± 68,8 µg/m^3; Mean number concentration: 2.30 × 10^5 particles/cm^3 (range: 7.12 ×10^3 - 8.26 ×10^5) Summer exposure: 141.0 ± 54.9 µg/m^3; Mean number concentration: 2.78 ×10^5 particles/cm^3 (range: 7.76 ×10^3 - 8.87 × 10^5) **Time to Analysis:** 4 days of spring exposure and days of summer exposure for 5 h 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**: 200 g	CAPs collected in Chung-Li, Taipei **Particle Size:** $PM_{2.5}$ (0.1-2.5 µm)	**Route:** Nose-only Inhalation **Dose/Concentration:** 202.0 ± 68.8 µg/m^3 **Time to Analysis:** 5 h/days for 4 days	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 24 h. After 24 h 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:** C57 and ApoE$^{-/-}$	CAPs (NYU, NY) **Particle Size:** $PM_{2.5}$	**Route:** Whole-body Inhalation **Dose/Concentration:** 19.7 µg/m^3 average concentration over 5 mo (daily average exposure concentration was 110 µg/m^3) **Time to Analysis:** 6 h/days, 5 days/wk, for 5 mo. 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 wk, 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 wk for the overnight period.
Reference: Chen and Nadziejkov(2005, 087219)**(Chen and Nadziejko, 2005, 087219)** **Species:** Mouse **Strain:** C57, ApoE$^{-/-}$ **Age:** 26-28 wk (C57), 39-41 wk (ApoE$^{-/-}$), and 18-20 wk (LDLr$^{-/-}$ [DK])	CAPs (NYU, NY) **Particle Size:** $PM_{2.5}$	**Route:** Whole-body Inhalation **Dose/Concentration:** Mean exposure concentration: 110 µg/m^3 **Time to Analysis:** 6 h/days, 5 days/wk for up to 5mo. Sacrificed 3-6 days 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) **(2006, 156366)**(Corey et al., 2006, 156366) **Species:** Mouse **Gender:** Male **Strain:** ApoE$^{-/-}$ **Age:** 11-12 mo **Weight**: 32.84 g (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: 50 ul; Silica: Min-u-Sil 5 in 50 µl saline **Time to Analysis:** Mice monitored for 1 day baseline prior to and for 4 days 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:** 6-10 wk	Ultrafine PM (collected continuously over 7-day periods in Oct 2002 in Chapel Hill, NC) **Particle Size:** <150 nm	**Route:** IT Instillation **Dose/Concentration:** 100 µg of PM in vehicle **Time to Analysis:** 24 h 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 **Dose/Concentration:** Average concentration 354 µg/m^3 **Time to Analysis:** 8 h/days for 3 consecutive days; plasma samples collected 24 h 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:** 23 mo (Fischer); 11-14 mo (SH) **Weight:** NR	UFP - Ultrafine carbon particles; LPS (Sigma) **Particle Size:** UFP: 36 nm (median size)	**Route:** Whole-body inhalation; intraperitoneal injection (ip) for saline and LPS **Dose/Concentration:** Particles: 150 µg/m^3; LPS: 2 mg/kg bw **Time to Analysis:** Single 6-h exposure to particles. Sacrificed 24 h 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 activities 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 wk **Weight**: 22-2 7g	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 2.5 µm	**Route:** IT Instillation **Dose/Concentration:** PM: 200 µg; 200 µg PM+10 µg LPS; 200 µg PM+100 µg LPS **Time to Analysis:** 18 h 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:** 20 wk	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:** 6 h/days × 5 days/wk × 5 mo	**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: Folkmann et al. (2007, 097344) **Species:** Mouse **Gender:** Female **Strain:** Wild type and $ApoE^{-/-}$ **Age:** 11-13 wk **Weight**: 21 g (avg)	DEP: SRM2975 (particulate fraction of exhaust from a filtering system designed for diesel-powered forklifts). **Particle Size:** DEP: NR	**Route:** Intraperitoneal Iinjection **Dose/Concentration:** 0, 50, 500, 5,000 µg DEP/kg **Time to Analysis:** 6 or 24 h post-ip injection	The expression of inducible nitric oxide synthase (iNOS) mRNA was increased in the liver 6 h post-ip injection. The level of oxidized purine bases, determined by formamidopyrimidine DNA glycosylase sites increased significantly in the liver after 24 h in mice injected w/ 50µg/kg. 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 36 h	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 1 ng/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:** 12 wk **Weight:** 200-300 g	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:** 7 mg /kg **Time to Analysis:** DTPA added to some PM samples preinstillation. Instilled with PM. Necropsy 24 h post-exposure.	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: Gerlofs-Nijland et al. (2007, 097840) **Species:** Rat **Gender:** Male **Strain:** SH/NHsd **Age:** 13 wk **Weight:** 250-350 g	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, 10 mg/kg **Time to Analysis:** 24 h	**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 LYC lowered fibrinogen values more than PM from location MOB, HIA, and MGH. Fine PM showed less differences among the various sites.
Reference: Gerlofs-Nijland et al. (2005, 088652) **Species:** Rat **Gender:** Male **Strain:** SH/NHsd **Age:** 11-12 wk **Weight:** 250-350 g	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:** 4, 24, 48 h	**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 4 h for both PM types.
Reference: Ghelfi et al. (2008, 156468) **Species:** Rat **Strain:** SD **Age:** Adult	CAPs CPZ (Capsazepine) (Axxora LLC, San Diego, CA) **Particle Size:** $PM_{2.5}$	**Route:** CAPs: Whole-body Inhalation; CPZ: IP Injection or Aerosol **Dose/Concentration:** CAPs: mean mass concentration: 218 ± 23 µg/m^3; CPZ: 10 mg/kg (ip), 500 µmol (aerosol) **Time to Analysis:** Experiment 1: CPZ ip or 20 min aerosol pretreatment immediately prior to CAPs exposure. Single CAPs exposure for 5 h. Parameters measured immediately following exposure. Experiment 2: CPZ ip pretreatment prior to CAPs exposure. Exposed to CAPs for 5 h/day for 4 mo. 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 5-h period of CAPs inhalation. Changes in cardiac rhythm and ECG morphology were prevented by CPZ.

Study	Pollutant	Exposure	Effects
Reference: Gilmour et al. (2004, 054175) **Species:** Rat **Gender:** Male **Strain:** Wistar	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: 1.40 mg/m^3 **Time to Analysis:** Single exposure for 7 h. Sacrificed and samples taken at 0, 16, and 48 h 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 et al. (2005, 087410) **Species:** Human **Cell Types:** Primary Human Monocyte Derived Macrophages (MP); Human Umbilical Vein Endothelial Cells (HUVEC); A549 cells; 16HBE	PM_{10}: (Carbon Black from Degussa Ltd, Frankfurt, Germany) **Particle Size:** PM_{10}	**Route:** Cell Culture **Dose/Concentration:** PM_{10}: 50 and 100 µg/mL **Time to Analysis:** 6 and 20 h	The culture media from MPs and 16HBE cells but not A549 cells, exposed to PM_{10} had an enhanced ability to cause clotting. H_2O_2 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.
Reference: Gilmour et al. (2006, 156472) **Species:** Rat **Gender:** Male **Strain:** Wistar Kyoto **Age:** 12-14 wk **Weight:** 280-340 g	Zinc Sulfate ($ZnSO_4$ in saline solution) **Particle Size:** NR	**Route:** IT Instillation **Dose/Concentration:** 131 µg/kg (2 µmol/kg) **Time to Analysis:** 1, 4, 24, 48 h	**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:** Mouse **Cell Type:** Human Microvascular Endothelial Cells (HMEC) **Strain:** C57BL/6J **Gender:** Male **Age:** 2 mo	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 Validation: Ufp: 3.2 4×10^5/cm^3; fp: 2.7 $\times10^5$/cm^3 In vivo validation: Ufp: <0.18 µm; fp: <2.5 µm **Time to Analysis:** 4 h In vivo validation: Exposed to CAPs for 5 h/day, 3 days/wk for 8 wk. Sacrificed 24 h after last CAPs exposure.	Gene-expression profiling showed that both DEP extract and ox-PAPC co-regulated a large number of genes. 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.

Study	Pollutant	Exposure	Effects
Reference: Goto et al. (2004, 088100) **Species:** Rabbit **Gender:** Female **Strains:** New Zealand White **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 4-168 h after BrdU injection. Sacrificed 7days 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 12 h after instillation. Monocyte count was not affected. Labeled monocytes peaked more quickly after DEP exposure (12 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.
Reference: Gottipolu et al. (2009, 190360) **Species:** Rat **Gender:** Male **Strain:** Wistar Kyoto, SH **Age:** 14-16 wk **Weight:** NR	DE (30-kW (40hp) 4-cylinder indirect injection Deutz diesel engine) (O_2- 20%, CO- 1.3-4.8 ppm, NO- <2.5-5.9 ppm, NO_2- <0.25-1.2ppm, SO_2 0.2-0.3 ppm, OC/EC- 0.3 ± 0.03) **Particle Size:** Number Median Diameter: Low- 83 ± 2 nm, High- 88. 2 nm; Volume Median Diameter: Low- 207 ± 2 nm, High- 225 ± 2 nm	**Route:** Inhalation **Dose/Concentration:** Low: 507 ± 4 µg/m^3; High: 2201 ± 14 µg/m^3 **Time to Analysis:** Exposed 4 h/days, 5 days/wk, 4 wk. Necropsied 1day post-exposure.	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	Zn; V **Particle Size:** NR	**Route:** Cell Culture **Dose/Concentration:** 0, 6.25, 12.5, 25, or 50 µm **Time to Analysis:** Toxicity: 24 h post- exposure **Beat Rate:** 0.5, 1, 2, 4, and 24 h PE **PCR:** 6 and 24 h 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 6 h 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:** 18-20 wk	CAPs (Tuxedo, NY) Copollutants measured: O_3 and NO_2. **Particle Size:** 389 ± 2 nm	**Route:** Whole-body Inhalation **Dose/Concentration:** CAPs: 131 ± 99 µg/m^3 (range 13-441 µg/m^3) O_3: 10 ppb NO_2: 4.4 ppb **Time to Analysis:** 6 h/days, 5 days/wk for approximately 4 mo. Tissue collection was performed 3-4 days 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.

Study	Pollutant	Exposure	Effects
Reference: Gurgueira et al. (2002, 036535) **Species:** Rat **Gender:** Male **Strain:** SD **Weight:** 250-300 g	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 Inhalation **Dose/Concentration:** CAPs: average mass concentration: 300 ± 60 µg/m^3; ROFA: 1.7 mg/m^3; CB: 170 µg/m^3 **Time to Analysis:** CAPs: 1, 3, and 5 h; ROFA: 30 min; CB: 5 h	**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 5 h 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 µg/mL **Time to Analysis:** 0, 5, 10, 30, 60, 120 min	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.
Reference: Hansen et al. (2007, 090703) **Species:** Mouse **Gender:** Female **Strain:** $ApoE^{-/-}$ and C57BL/6J $ApoE^{+/+}$ **Age:** 11-13 wk	DEP: SRM-2975 (NIST) **Particle Size:** DEP: 215 nm (geometric mean diameter)	**Route:** Intraperitoneal Injection **Dose/Concentration:** DEP: 0, 0.5 and 5 mg/kg; Aorta segments incubated with 0, 10 and 100 µg DEP/mL **Time to Analysis:** Sacrificed 1 h 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: 215 nm (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:** 12-15 wk	Carbon UFPs ((generated by Electric Spark Generator GFG 1000; Palas, Karlsruhe, Germany) **Particle Size:** 37.6 ± 0.7 nm (mean)	**Route:** Whole-body Inhalation **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 24 h then sacrificed immediately following exposure period OR Sacrificed following 1-3 days recovery period.	**Cardiovascular Performance:** Mild but consistent increase in 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.

Study	Pollutant	Exposure	Effects
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 6 h incubation with DEP or UFP. Other parameters measured after 24 h.	**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.
Reference: Hwang et al. (2005, 089454) **Species:** Mouse **Strain:** C57 and ApoE$^{-/-}$	CAPs (Tuxedo, NY) **Particle Size:** 389 ± 2 nm	**Route:** Whole-body Inhalation **Dose/Concentration:** CAPs Range: 5-627 µg/m^3. Mean CAPs Concentration: 133µg/m^3. Mean Concentrations of O_3 and NO_2 in CAPs: 10 and 4.4 ppb respectively. **Time to Analysis:** 6 h/day, 5 days/wk for 5 mo.	**Long-term Analysis:** Significant decreasing patterns of HR, body temperature, and physical activity in ApoE$^{-/-}$ mice. Nonsignificant changes for C57 mice. The chronic effect changes for ApoE$^{-/-}$ mice were maximal in the last three wk. **Short-term Analysis:** Dose-dependent relationship for HR variations in ApoE$^{-/-}$ mice. **Heart Rate Fluctuation:** HR fluctuations in ApoE$^{-/-}$ 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- 7 wk	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: 4 mg/kg. DEP-OC: 4 mg/kg. LPS: 2.5 mg/kg. Washed DEP+LPS and DEP-OC+LPS: respective additions of LPS to each component prior-experimentation. **Time to Analysis:** Sacrificed 24 h 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: Inoue et al. (2006, 097815) **Species:** Mouse **Gender:** Male **Strains:** C3H/HeJ (TLR-4 point mutant) and C3H/HeN (Control) **Age:** 6 wk	DEP (derived from 4 cyl, 2.74l light duty diesel engine) **Particle Size:** NR	**Route:** IT Instillation **Dose/Concentration:** 12 mg/kg **Time to Analysis:** 24 h	**Hematology:** DEP increased plasma fibrinogen in both strains but with a greater increase in the knockout mice than the wild type.
Reference: Ito et al. (2008, 096823) **Species:** Rat **Gender:** Male **Strain:** Wistar Kyoto (Specific pathogen-free) **Age:** 13-14 wk	CAPs (f-PM), 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 4 days, (2) filtered air for 3 days and CAPs for 1 day or (3) CAPs for 4 days. All groups exposed for a maximum of 4.5 h/days 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 (ETA) 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 ETA receptor mRNA was significantly correlated with the increase in HO-1 mRNA and weakly with the increase in MBP.

Study	Pollutant	Exposure	Effects
Reference: Khandoga A et al. (2004, 087928) **Species:** Mouse **Gender:** Female **Strain:** C57B1/6 **Age:** 5-7 wk	UFPs: Ultra fine carbon black particles (Printex 90) **Particle Size:** 14 nm diameter (60% <100 nm)	**Route:** Aortic Infusion **Dose/Concentration:** 1×10^7 and 5×10^7 total particles infused 300 m^2/g surface area **Time to Analysis:** Single exposure, analysis 2 h 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 GD19) **Strain** SD **Age:** 60-90 days **Weight:** 300 g **Use:** RMCs were harvest from 1 day-old neonatal pups	ROFA-L: Leachate **Particle Size:** <0.2 µm	**Route:** Cell Culture **Dose/Concentration:** 3.5 µg/mL **Time to Analysis:** 1 h	**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.
Reference: Knuckles et al. (2008, 191987) **Species:** Mouse **Gender:** Male **Strain:** C57BL/6 **Age:** 8-10 wk **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 Inhalation. Ex Vivo. **Dose/Concentration:** In vivo: 350 µg/m^3; Ex vivo: $PM_{2.5}$ concentration 2-3 mg/m^3 flow rate 500 mL/min **Time to Analysis:** Exposed 4 h. 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 **Age:** 12-14 wk	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: zinc sulfate **Particle Size:** $PM_{2.5}$	**Route:** IT Instillation **Dose/Concentration:** Doses (mg/kg/wk) are for 8 and 16 wk (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:** 1 x/wk for 8 or 16 wk; analyzed 48 h 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: Kooter et al. (2006, 097547) **Species:** Rat **Gender:** Male **Strain:** SH **Age:** 12-14 wk	CAP-F = fine (Site I) CAP-UF = fine + ultrafine (Site II) (Netherlands) Some measured components: Ammonium, nitrate, sulfate 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 µg/m^3 CAP-UF 269-556 µg/m^3 **Time to Analysis:** 6 h/days for 2 days consecutive, 18 h	**Hematology:** WBC and lymphocytes decreased with both CAP-F and CAP-UF. MPV and MPC (mean platelet volume and component) increased with CAP-UF.

Study	Pollutant	Exposure	Effects
Reference: Kyoso et al. (2005, 186998) **Species:** Rat **Gender:** NR **Strain:** NR **Age:** 15 mo	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 16 h/days (from 5pm-9am) for 7 mo	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 et al. (2004, 087884) **Species:** Rat **Gender:** Male **Strain:** SD **Weight:** 300-350 g	CAPs from Asian dust storm (Taiwan) Measured Components: Si, Al, S, Ca, K, Mg, Fe, As, Ni, W, V, OC, EC, SO_2, NO_2, nitrate, sulfate **Particle Size:** 0.01- 2.5 µm	**Route:** Nose-only Inhalation **Dose/Concentration:** 315.6 $µg/m^3$ (Low) or 684.5 $µg/m^3$ (High) **Time to Analysis:** Low: Exposed for 6 h. Sacrificed 36 h post-exposure High: Exposed for 4.5 h. Sacrificed 36 h post-exposure Pulmonary hypertension induced 2 wk pre-exposure.	**Hematology:** PM induced a dose-dependent increase in WBCs. No change was seen in RBCs. Platelet results were highly variable.
Reference: Lei et al. (2005, 088660) **Species:** Rat **Gender:** Male **Strain:** SD **Weight:** 200-250 g **Use:** ip STZ (60 mg/kg) 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 ($µg/m^3$): (9.8-SD 2.4)), EC (3.6-SD 3.2), Sulfate (4.8-SD 1.2), Nitrate (6.3-SD 3.4) **Time to Analysis:** Single dose. Animals sacrificed 24 h post instillation.	**Effects of Diabetes:** Body weight (bw) of diabetic (D) rats (397.5 g) was lower than non-diabetic (ND) rats (483.1 g). Mean plasma glucose level was 163 mg/daysL in ND rats and 448.2 mg/daysL 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 et al. (2006, 088594) **Species:** Mouse **Gender:** NR **Strain:** BALB/c **Age:** 1day (neonatal) **n:** 10 **Weight:** 4-6 g	PM_{10}, CO, NO_2, and SO_2 from Universidade de Sao Paulo, Brazil. **Particle Size:** PM_{10}	**Route:** Whole-body Inhalation **Dose/Concentration:** Mean (± SD) concentrations were: CO_2: 2.06 ± 0.08 ppm (8h mean); NO_2: 104.75 ± 42.62 $µg/m^3$ (24 h mean); SO_2: 11.07 ± 5.32 $µg/m^3$ (24 h mean); PM_{10}: 35.52 ± 12.84 $µg/m^3$ (24 h mean) **Time to Analysis:** 24 h/days, 7 days/wk for 4 mo	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.
Reference: Li et al. (2005, 088647) **Species:** Rat **Strain:** SD **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 5 min after each UPs dose. HPAECs: exposed to UPs from 1 to 100 µg/mL for up to 2 min	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 (CuSO4) and V (VOSO4), 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.

Study	Pollutant	Exposure	Effects
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:** SD Rats, New Zealand White Rabbits **Weight:** Rat: 200-350 g; Rabbit: 2.5-3.0 kg	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 15 min prior to exposure. Exposed to increasing doses of UPs from 1 to 100 µg/mL. Maximum tension was recorded within 5 min after each UPs dose. HPAECs: exposed to UPs from 1 to 100 µg/mL for 20 and 120 min.	**Effects of UP on H_20_2 Release:** Within minutes after UPs treatment, HPAEC increased H_2O_2 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 H_2O_2 production. NaN_3 inhibited H_2O_2 production stimulated by Cu and V, whereas DPI and APO inhibited only Cu-stimulated H_2O_2 production. Inhibitors of other H_2O_2-producing enzymes, including N-methyl-L-arginine, indomethacin, allopurinol, cimetidine, rotenone, and antimycin, had no effects. **Effects of UP-induced H_20_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 H_2O_2 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:** 6 h/days, 5 days/wk for 5 mo. 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 between 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.
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 µg/m^3, Long-term average: 19.7 µg/m^3 **Time to Analysis:** Exposed 6 h/days, 5 days/wk, 5 or 6 mo. 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:** 6 wk	CAPs from Tuxedo, NY. Component of interest: Ni. **Particle Size:** $PM_{2.5}$	**Route:** Whole-body Inhalation **Dose/Concentration:** Average daily CAPs: 85.6 µg/^{m3}; Average daily Ni: 43 ng/m^3 **Time to Analysis:** 6 h/day, 5 days/wk, for 6 mo (July 2004-January 2005). 10-s ECG, HR, activity, and body temperature data were sampled every 5 min 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.

Study	Pollutant	Exposure	Effects
Reference: Lund et al. (2007, 125741) **Species:** Mouse **Gender:** Male **Strain:** ApoE$^{-/-}$ **Age:** 10 wk **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, fueled 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 µg/m^3), NO_X (0 ppm), CO (0.1 ppm), HC (0.1 ppm); Low (1: 90 dilution of exhaust): PM (8 µg/m^3), NO_X (2 ppm), CO (9 ppm), HC (0.9 ppm); Mid (1: 20): PM (39 µg/m^3), NO_X (12 ppm), CO (50 ppm), HC (8.4 ppm); High (1: 12): PM (61 µg/m^3), NO_X (19 ppm), CO (80 ppm), HC (12 ppm); High-filtered (1:12): PM (2 µg/m^3), NO_X (18 ppm), CO (80 ppm), HC (12.7 ppm). **Time to Analysis:** 6 h/day, 7 days/wk for 7 wk. Mice were sacrificed within 16 h PE. During the study period all animals concurrently exposed to the following: FA: 8 µg/m^3 and 40 µg/m^3; PM Whole Exhaust: 60 µg/m^3; or Filtered Exhaust w/ gases matching the 60 µg/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 et al. (2007, 125741) **Species:** Mouse **Gender:** Male **Strain:** ApoE$^{-/-}$ **Age:** 10 wk **Weight:** NR	GEE (conventional unleaded, nonoxygenated, nonreformulated gasoline- ChevronPhillips Specialty Fuels Division) **Particle Size:** 0.150 µm (MMAD)	**Route:** Inhalation **Dose/Concentration:** PM: 60 µg/m^3, NO_2: 2 ppm, NO: 16 ppm, CO: 80 ppm, THC: 12.7 ppm **Time to Analysis:** Mice fed high-fat diet 30 days before exposure. Exposed 6 h/day, 1 or 7 days. Some groups dosed with Tempol or BQ-123. Killed within 18 h of last exposure.	Aorta gelatinase activity increased with GEE exposure time. MMP-2/9 activity spread throughout the vasculature by day 7. 7 day 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: McQueen et al. (2007, 096266) **Species:** Rat **Gender:** Male **Strain:** Wistar Kyoto **Weight:** 228-500 g	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:** 6 h. Pre-exposure: Vagotomy (sectioning of vagus nerve) or atropine, 1mg/kg i.p. administered 30 min prior, 2 and 4 h post.	**Cardiovascular Response:** Blood pressure and heart rate were unaffected. Average arterial O_2 increased after DEP, but not when compared for each animal. CO_2 and pH were not affected
Reference: Medeiros et al. (2004, 096012) **Species:** Mouse **Gender:** Male **Strain:** BALB/c **Age:** 60 days **Weight:** 20-30 g	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, 24 h	**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).

Study	Pollutant	Exposure	Effects
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 μg/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 μg/cm^2. This adhesion was associated with 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 with 20 μg/cm^2; however PM_{10}-induced expression was observed from 5 μg/cm^2. E-selectin and ICAM-1 had the strongest expression in response to particles.
Reference: Moyer et al. (2002, 052222) **Species:** Mouse **Gender:** Male and Female **Strain:** B6C3F1	In phosphide (InP), Co sulfate heptahydrate ($CoSO_4 \cdot 7H_2O$), 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), $CoSO_4 \cdot 7H_2O$ (1.5-1.8), V_2O_5: (1.0), GaAs: (1.0)	**Route:** Inhalation **Dose/Concentration:** High-Dose Concentration in Chronic Studies, Male (μg/m^3): InP: 0.3, $CoSO_4 \cdot 7H_2O$: 3.0, V_2O_5: 4.0, GaAs: 1.0 High-Dose Concentration in Sub-Chronic Studies, Male or Female (μg/m^3): InP: 100, $CoSO_4 \cdot 7H_2O$: 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 90-day studies of the 4-compounds demonstrating arteritis after a 2-yr period.	**Phase One:** High-dose males developed significantly increased incidences of arteritis over controls in 2 of the 9 studies (InP and $CoSO_4 \cdot 7H_2O$), 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 et al. (2007, 121441) **Species:** Mouse **Gender:** Male **Strain:** 57BL/6 (IL-6$^{+/+}$ and IL-6$^{-/-}$) **Age:** 6-8 wk **Weight:** 20-25 g	PM_{10} from ambient air in Düsseldorf, Germany **Particle Size:** PM_{10}	**Route:** IT Instillation **Dose/Concentration:** PM_{10}: 10 μg; Clodronate: 120 mg **Time to Analysis:** For alveolar macrophage depletion, clodronate instilled into mice lungs following endotracheal intubation 48 h prior to instillation of PM. Parameters measured 24 h 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, 087460) **Species:** Rat **Gender:** Male **Strain:** Wistar Kyoto **Age:** 16 wk	CAPs ($PM_{2.5}$) from Tuxedo, NY. (SO_2, NO_2 O_3 and NH_3 were removed prior to exposure). H_2SO_4 (fine and ultrafine) **Particle Size:** Ultrafine H_2SO_4 50-75 nm (MMAD)	**Route:** Nose-only Inhalation **Dose/Concentration:** CAPs: 80 and 66 μg/m^3 (avg 73); Fine H_2SO_4: 299, 280, 119, and 203 μg/m^3 (avg 225); Ultrafine H_2SO_4: 140, 565, 416, 750 μg/m^3 (avg 468) **Time to Analysis:** 4 h/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 H_2SO_4 aerosol also caused a significant decrease in respiratory rate similar to the effect of CAPs. Ultrafine H_2SO_4 had the opposite effect on respiratory rate compared to CAPs.
Reference: Nadziejko et al. (2004, 055632) **Species:** Rat **Gender:** Male **Strain:** F344 **Age:** 18 mo	PM/CAPs (Tuxedo, NY) UFC (lab generated) SO_2 **Particle Size:** PM (Size Range): 0.5-2.5μm; UFC (MMAD): 30-50 nm	**Route:** Nose-only Inhalation **Dose/Concentration:** PM (μg/m^3): 161-200, avg. 180; UFC (μg/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 4 h 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.

Study	Pollutant	Exposure	Effects
Reference: Nemmar et al. (2008, 096566) **Species:** Rat **Gender:** Male **Strain:** Wistar Kyoto **Weight:** 440 ± 14 g	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:** 48 h 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 RBCs after 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.
Reference: Nemmar et al. (2007, 156800) **Species:** Rat **Gender:** Male **Strain:** Wistar Kyoto **Age:** 16 wk **Weight:** 424 ± 8 g	DEP (SRM 2975) **Particle Size:** NR	**Route:** Tail Vein Injection **Dose/Concentration:** 8, 42, or 212 µg DEP/rat (150µl of 0.02, 0.1, or 0.5 mg/kg) **Time to Analysis:** 24 h	**Effect of DEP on Blood Pressure:** Significant decrease on BP in DEP-exposed rats at doses of 0.02 mg/kg, compared with mean BP observed in controls. **Effect of DEP on HR**: Doses of 0.02, 0.1, and 0.5 mg/kg 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. 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:** 100-110 g	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 40 min. Ex-vivo: animals were ITly instilled w/ DEPs (0 or 50 µg per animal), and blood was collected 5, 15, 30, and 60 min 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 5 min/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:** 100-110 g	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 (5 mg/kg) or IT (0.1 or 0.5 mg/kg) or Sodium Cromoglycate given IP (40 mg/kg), 1 h before DEP or vehicle instillation. Thrombosis: In-vivo thrombogenesis assessed 24 h 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:** 100-110 g	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: 60 nm; NCC-MPSPs: 60 nm; PCA-MPSPs: 60 or 400 nm	**Route:** IT Instillation **Dose/Concentration:** 5, 50, and 500 µg/animal in 120 µl saline **Time to Analysis:** 1 h 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.

Study	Pollutant	Exposure	Effects
Reference: Nemmar et al. (2003, 087931) **Species:** Hamster **Weight:** 100-110 g	DEP (SRM 1650) **Particle Size:** NR	**Route:** IT Instillation **Dose/Concentration:** 50 µg/animal in 120 µl saline **Time to Analysis:** 1, 3, 6, and 24 h	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.
Reference: Niwa et al. (2007, 091309) **Species:** Mouse **Gender:** Male **Strain:** LDLr/KO **Age,:** 6 wk (n = 20) **Use:** IT CB dispersion; 10-14 wk acute effect of CB dispersion on circulating CRP	Carbon Black **Particle Size:** 23-470 nm (mean size 120.7 nm)	**Route:** IT Dispersion **Dose/Concentration:** IT CB Dispersion Study: 1 mg per animal/wk; Acute Effect of CB Dispersion on Circulating CRP Study: 1mg/animal (single administration) **Time to Analysis:** IT CB Dispersion Study: 1x/wk for 10 wk Acute Effect of CB Dispersion on Circulating CRP Study: Single CB administration, blood samples collected 24 h 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:** RAW264.7	Carbon Black (CB); Water-Soluble Fullerene ($C_{60}(OH)_{24}$); Fluoresbrite Carboxylate Microspheres; Ox-LDL; Acetylated-LDL **Particle Size:** Carbon Black and $C_{60}(OH)_{24}$: 7.1 nm (SD 2.4); Fluoresbrite Carboxylate Microspheres: 6 nm	**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 24 h, 13 days, and 50 days; RAW264.7+ $C_{60}(OH)_{24}$ for 24 h or 10 days; RAW264.7+ $C_{60}(OH)_{24}$ for 8 days, then co-treated w/ Ox-LDL for an additional 48 h; RAW264.7+Ox-LDL for 5 days, and then co-cultured w/ C60(OH)24 for an additional 48 h; RAW264.7+ 6 nm beads: 3 days, the Ox-LDL or acetylated-LDL added for 24 h	CB alone had no significant effects on RAW264.7 cell growth. $C_{60}(OH)_{24}$ alone or CB and $C_{60}(OH)_{24}$ 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. $C_{60}(OH)_{24}$ induced LOX-1 protein expression, pro-matrix metalloprotease-9 protein secretion, and tissue factor mRNA expression in lipid-laden macrophages. Although CB or $C_{60}(OH)_{24}$ alone did not induce platelet aggregation, $C_{60}(OH)_{24}$ facilitated ADP-induced platelet aggregation. $C_{60}(OH)_{24}$ also acted as a competitive inhibitor of ADP receptor antagonists in ADP-mediated platelet aggregation.
Reference: Niwa et al. (2008, 156812) **Species:** Rat **Strain:** SD **Age:** 6 wk	CB from Kyoto, Japan **Particle Size:** Mean size (nm) ± SD determined at 1, 8, 15, 22, and 29 day 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 ± 3.5 mg/m^3 **Time to Analysis:** 6 h/day, 5 days/wk, for a total of 4 wk. BP and HR were measured by tail-cuff plethysmography at 1, 14, and 28 day post -exposure. Sacrificed At 1, 7, 14, 28, and 30 day 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, and CRP, were higher in the CB treated groups than in control groups.
Reference: Nurkiewicz et al. (2004, 087968) **Species:** Rat **Gender:** Male **Strain:** SD **Age:** 7-8 wk	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:** 2.2 µm (ROFA mean count diameter)	**Route:** IT Instillation **Dose/Concentration:** ROFA group: 0.1, 0.25, 1, or 2 mg/rat. Vehicle control group: 300 µl saline. Particle control group: TiO_2 0.25 mg/rat. **Time to Analysis:** After single IT instillation of a particular dose, all rats recovered for 24 h.	**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 leukocyte-adhesion and rolling observed in ROFA exposed rats.

Study	Pollutant	Exposure	Effects
Reference: Nurkiewicz et al. (2006, 088611) **Species:** Rat **Gender:** Male **Strain:** SD **Age:** 7-8 wk	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 µl 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 24 h.	**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 et al. (2008, 156816) **Species:** Rat **Gender:** Male **Strain:** SD **Age:** 6-7wk **Weight:** NR	TiO_2 (DeGussa, Sigma-Aldrich) **Particle Size:** Fine- 1 µm, UF- 21 nm	**Route:** Whole-body Inhalation **Dose/Concentration:** Concentrations: Fine- 3-16 mg/m^3; UF- 1.5-12 mg/m^3; Dose: Fine- 8, 20, 36, 67, 90 µg; UF- 4, 6, 10, 19, 30 µg **Time to Analysis:** Acclimated 5 days. Exposed 4-12 h. Sacrificed 24 h post-exposure.	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:** 7-8 wk **Weight:** NR	Fine TiO_2 (Sigma-Aldrich, St. Louis, MO) (~99% rutile) TiO_2 nanoparticles (DeGussa-Aeroxide TiO_2 P25, Parsippany, NJ) (80% anatase, 20% rutile) **Particle Size:** Fine TiO_2- MMAD: 402 nm, Primary size: <5 µm, ,CMD: 710 nm; Nano-TiO_2- MMAD: 138 nm, Primary size: 21 nm, , CMD: 100 nm	**Route:** Aerosol Inhalation **Dose/Concentration:** 1.5-16mg/m^3 **Time to Analysis:** Acclimated 5 days. Exposed 240-720 min. Anesthetized 24 h post-exposure. 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 3 days 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:** cells were incubated for 1, 2, 4, or 8, 24 or 48 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 24 h. Medium then replaced w/serum-free & cells incubated for another 24 h 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:** 12 wk **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 ± 0.18 µm (aerodynamic diameter)	**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 12.5 µg/mL ROFA-L (at 10 mg/mL). Protocol 2: exposed to 1.56, 3.25, 6.26, 12.5 µg/mL ROFA-L (at 10 mg/mL). **Time to Analysis:** Protocol 1: Cells treated with 12.5 µg/mL ROFA-L and/or 104mol/L L-NAME for 20 min 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 et al. (2005, 091377) **Species:** Rat **Strain:** Wistar Kyoto	Carbon Nano Particles (CNPs) (purchased from SES Research, Houston, TX): Multiplewall Nanotubes (MWNT); Single wall 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 s.	**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 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 (2.5×10^8 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 for 1 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 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:** 6-12 wk	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:** ~0.25 µm	**Route:** Whole-body Inhalation **Dose/Concentration:** Low: 30 µg/m^3 Mid-low: 100 µg/m^3 Mid-high: 300 µg/m^3 High: 1000 µg/m^3 **Time to Analysis:** 6 h/day, 7days /wk for 1 wk or 6 mo. Immediate post-exposure analysis.	**Organ Weights:** Liver declined in rats of both genders at 1 wk and female rats at 6 mo. Lung volume increased and lung weight decreased in female rats at 6 mo. 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 6 mo and increased at mid-low and mid-high doses for female rats at 6 mo. ALP decreased for rats of both genders at 1wk and 6 mo 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 6 mo. BUN/Cre ratio decreased in females at 1wk (25%) and both genders at 6 mo at mid-high and high dose (18-19%). **Hematology:** Hemoglobin and hematocrit increased in 6 mo male rats. Bilirubin increased in female rats at 6 mo 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.
Reference: Reed et al. (2004, 055625) **Species:** Rat, Mouse **Gender:** Male and Female **Strain:** CDF (F344)/CrlBR (rat), A/J (mouse) **Age:** 12 wk	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 - 0.15 µm	**Route:** Whole-body Inhalation **Dose/Concentration:** Low: 30 µg/m^3 Mid-low: 100 µg/m^3 Mid-high: 300 µg/m^3 High: 1000 µg/m^3 **Time to Analysis:** 6 h/day, 7 days/wk for 1wk or 6 mo. Analyzed 1 day 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 6 mo. Lung weight increased at high dose at 6mo 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 1 wk and a smaller decrease for male rats at 6 mo. GGT significantly increased at 6 mo 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 6 mo. BUN and BUN/Creatine declined (19%, 17%) in female rats at mid-high and high doses after 6 mo. BUN increased by 21% at mid-low, mid-high and high doses in male rats at 1wk. **Hematology:** WBC decreased in high females after 6 mo. Factor VII (blood clotting) decreased in MH and HR males after 1wk and male and female HR after 6 mo. Thrombin-antithrombin complex declined massively but only in males after 1wk.
Reference: Reed et al. (2008, 156903) **Species:** Rat **Gender:** Male, Female **Strain:** CDF (F344)/CrlBR, SH **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:** 150 nm (MMAD)	**Route:** Whole-body Inhalation **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:** 2 wk quarantine period in chamber. Exposed 6 h/day, 7 days/wk, 3 day-6 mo. SH- surgery to implant telemeter in peritoneal cavity. 4 wk recovery. ECG data obtained every 15 min beginning 3 day pre-exposure, 7 day exposure, 4 day post-exposure.	**Organ Weight:** At 6 mo exposure, the heart weights of male and female rats increased and male rats' seminal vesicle weight decreased. **Clinical Chemistry:** Serum alanine aminotransferase, aspartate aminotransferase, and phosphorus decreased in medium and high-exposure females. **Hematology:** Hematocrit, red blood cell count, and hemoglobin dose-dependently increased for both genders at both time points. Plasma fibrinogen increased at 1wk in males. **CV Effects in SH Rat:** 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: Rhoden et al. (2005, 087878) **Species:** Rat **Gender:** Male **Strain:** SD **Age:** Adult **Weight:** 300 g	Urban Ambient Particles (UAPs): SRM-1649; CAPs (Boston, MA) **Particle Size:** NR	**Route:** UAPs: IT Instillation. CAPs: Inhalation **Dose/Concentration:** UAPs: 750 µg suspended in 300 µl saline; CAPs: 700 ± 180 µg/m^3 **Time to Analysis:** UAPs: 30 min post-instillation. CAPs: immediately after 5 h exposure period	**Oxidative Stress and HR Function:** UAPs instillation led to significant increases in heart oxidants. HR increased 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 Response:** Rats were treated with 50 mg/kg NAC 1 h 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 et al. (2005, 088653) **Species:** Rat **Gender:** Male **Strain:** Wistar Kyoto **Age:** 3 mo **Weight:** ~250 g	$PM_{2.5}$, 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:** 24 h post-instillation	**Hematology:** 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.
Reference: Rivero et al. (2005, 088659) **Species:** Rat **Gender:** Male **Strain:** Wistar Kyoto **Age:** 3 mo **Weight:** ~250 g	$PM_{2.5}$, 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 60 min 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:** 10-12 wk **Weight:** 250-300 g	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: 150 nm (MMAD), 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- 104 ppm, NO- 16.7 ppm, NO_2- 1.1 ppm, SO_2- 1.0 ppm, THC- 12ppm; SDCAs: CO- <1 ppm, NO- 0.19-0.62 ppm, NO_2- 0.10-0.37 ppm, SO_2- 0.07-0.24 ppm, THC- <1 ppm **Time to Analysis:** 6 h exposure. Cannula ligated into trachea and connected to rodent ventilator. Thorax and abdomen opened.	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..
Reference: Simkhovich et al. (2007, 096594) **Species:** Rat **Gender:** Female **Strain:** Fischer 344 × Brown Norway hybrid **Age:** 4, 26 mo	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 min and analysis conducted every 10 min.	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.

Study	Pollutant	Exposure	Effects
Reference: Smith et al. (2006, 110864) **Species:** Rat **Gender:** Male **Strain:** SD **Age:** 8 wk **Weight:** 260-270 g	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 µg/m^3 $PM_{2.5}$ including 600 µg/m^3 PM_1 **Time to Analysis:** 4 h/day for 3 consecutive days. Parameters measured 18 or 36 h post-exposure.	**Hematology:** Plasma protein increased at 18h. Lymphocyte and hematocrit percentage decreased at 36 h.
Reference: Stinn et al. (2005, 088307) **Species:** Rat **Gender:** Male and Female **Strain:** Crl: (WIU BR **Age:** 40 day	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 EC. **Particle Size:** 0.19-0.21 µm (MMAD)	**Route:** Nose-only Inhalation **Dose/Concentration:** 3 and 10 mg/m^3 **Time to Analysis:** 6 h/day, 7 day/wk for 24 mo; 6 mo post-exposure	**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.
Reference: Sun et al. (2005, 087952) **Species:** Mouse **Gender:** Male **Strain:** ApoE$^{-/-}$ **Age:** 16 wk	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 µg/m^3; Daily concentration: 10.6 (SD 3.4) µg/m^3 (mean) Average exposure over 6 mo period: 15.2 µg/m^3. **Time to Analysis:** Study diets fed for at least 10 wk prior to exposure to $PM_{2.5}$ or FA. Exposed for 6 h/day, 5 days/wk for 6 mo. Sacrificed 15-47 days 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.
Reference: Sun et al. (2008, 157033) **Species:** Mouse **Gender:** Male **Strain:** ApoE$^{-/-}$ **Age:** 6 wk	CAPs $PM_{2.5}$ Collected from Sterling Forest State Park, Tuxedo NY (40 miles NW of Manhattan) **Particle Size:** $PM_{2.5}$	**Route: Whole-body** Inhalation **Dose/Concentration:** Average Concentration of: 85 µg/m^3 CAPs in chamber. Average exposure over 6 mo was 15.2 µg/m^3. **Time to Analysis:** 6 h/day, 5 day/wk for 6 mo. 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.

Study	Pollutant	Exposure	Effects
Reference: Sun et al. (2008, 157033) **Species:** Human **Cell Lines:** BEAS-2B; Vascular Smooth Muscle Cells (hSMCs); and Monocytes (THP-1)	Ambient Particles collected from Sterling Forest State Park, Tuxedo, NY (24 h/day for 4 wk) **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 µg/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 µg/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} µg/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:** SD **Age:** 500-650 g	$PM_{2.5}$ or UFP **Particle Size:** $PM_{2.5}$; UFP: <0.1 µm	**Route:** Whole-body Inhalation **Dose/Concentration:** Mean $PM_{2.5}$ concentration: 79.1 ± 7.4 µg/m^3. Normalized $PM_{2.5}$ over 10wk period: 14.1 µg/m^3. **Time to Analysis:** 6 h/day, 5 day/wk random exposure to $PM_{2.5}$, UFP, or FA for a total of 10 wk. At the end of wk 9 exposure, rats were infused w/ 0.75 mg/kg/day 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(P)H oxidase subunits p22phox and p47phox and depletion of tetrahydrobipterin.
Reference: Sun et al. (2008, 157032) **Species:** Rat **Gender:** Male **Strain:** SD **Age:** 500-650 g **Cell Line:** Primary Rat Aortic Smooth Muscle Cells (RASMCs)	$PM_{2.5}$ or UFP **Particle Size:** $PM_{2.5}$; UFP: <0.1 µm	**Route:** Cell Culture **Dose/Concentration:** UFP, $PM_{2.5}$: 10 or 50 µg/mL; AII: 100 nmol/L **Time to Analysis:** Exposed to UFP or $PM_{2.5}$ and parameters measured at 0, 1, 3, 6, and 15 min. 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 et al. (2009, 190487) **Species:** Mouse **Gender:** Male **Strain:** C57BL/6, c-fmsYFP (transgenic, yellow fluorescent protein under monocyte-specific promoter) **Age:** 8, 10 wk **Weight:** NR	PM (concentrated- northeastern regional background; Tuxedo Park, NY) **Particle Size:** 2.5 µm (diameter)	**Route:** Whole-body Inhalation. IT Instillation. **Dose/Concentration:** Exposure chamber (mean): 72.7 µg/m^3, IT: 1.6mg/kg **Time to Analysis:** C57BL/6 mice, fed high-fat chow 10wk. Exposed in vivo 6 h/day, 5 day, 128 days. fmsYFP rendered diabetic or fed normal chow 10 wk. IT instilled with PM 2 times/wk for 10 wk.	**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 ED50 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:** 12 wk **Weight:** Acute (average)- 2.4 ± 0.2 kg, Chronic (average)- 2.7 ± 0.3 kg	PM_{10} (urban; Ottawa, Canada) **Particle Size:**: 0.8 ± 0.4 µm (mean diameter)	**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 2 times/wk for 4 wk.	**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 wk and IL-6 in the first wk. **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 wk 1 in the acute model and wks 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 wk 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, 28 mo **Weight:** NR	Carbon black (CB) (Wright dust feed particle generator-BGI, Waltham, MA) **Particle Size:** 0.1-1.0 µm	**Route:** Whole-body Inhalation **Dose/Concentration:** Average $PM_{2.5}$ concentration- 401 ± 46 µg/m^3, Average PM_{10} concentration- 553 ± 49 µg/m^3 **Time to Analysis:** 3 h/day, 4 days	**Hemodynamics:** CB significantly elevated right atrial and ventricular pressures, pulmonary arterial pressure and vascular resistance, all of which were more pronounced in the 28 mo-old mice. RV contractility (specifically, the ejection fraction and maximum change in pressure over time) reduced in CB-exposed 28 mo-old mice. **Heart Tissue:** CB significantly declined Ca2+-dependent NOS activity and was more pronounced in 28 mo-old mice, who also had NOS2 upregulated. CB enhanced ROS generation and NOS-uncoupling and was greatest in 28 mo-old mice. CB also increased MMP-2, MMP-9, ANP, BNP, which were greatest in 28 mo-old mice. CB also reduced PKG-1 in 28 mo-old mice.

Study	Pollutant	Exposure	Effects
Reference: Tankersley et al. (2007, 097910) **Species:** Mouse **Gender:** Male **Strain:** C3H/HeJ and C57BL/6J **Age:** 10 wk **Weight:** 22-26 g	Carbon Black (CB) **Particle Size:** CB: 2.4 µm (MMAD) (GSD 2.75 µm).	**Route:** CB: Whole-body Inhalation; Sympathetic (S) & Parasympathetic (PS) blockade: IP Injection **Dose/Concentration:** CB: 159 ± 12 µg/m^3; PS (atropine): 0.5 mg/kg; S(propanolol): 1 mg/kg **Time to Analysis:** Successive 3 h CB and FA Exposures: conducted from 9 a.m. to 1 p.m., or at least 3 h 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 et al. (2004, 094378) **Species:** Mice **Strain:** AKR/J **Age:** ~180 days	Carbon Black (CB) and Filtered Air (FA) **Particle Size:** CB: 0.1 to 1 um.	**Route:** Whole-body Inhalation **Dose/Concentration:** CB average concentration: 160 ± 22 µg/m^3 **Time to Analysis:** FA exposure on day 1, CB exposure 3 h/day 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:** F344 **Weight:** 200-250 g	Urban Ambient Particles (EHC-93) from Ottawa, Canada; O_3 **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; O_3: 0, 0.4, 0.8 ppm **Time to Analysis:** 4 h to particles, O_3, or combination of particles and O_3.	Both pollutants individually increased preproET-1, ET-1 and endothelial 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 O_3. Both pollutants transiently increased ET-B receptor mRNA expression, while O_3 decreased ET-A receptor mRNA levels. Coexposure to particles plus O_3 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 et al. (2006, 097483) **Species:** Rat **Gender:** Male **Strain:** F344 **Weight:** 200-250 g	Urban Ambient Particles (EHC-93) from Ottawa, Canada; O_3 **Particle Size:** NR	**Route:** Nose-only Inhalation **Dose/Concentration:** EHC-93: 0, 50 mg/m^3; O_3: 0, 0.8 ppm **Time to Analysis:** 4 h to particles, O_3, or combination of particles and O_3. Sacrificed immediately following exposure or following 24 h recovery.	Circulating levels of both ET-1[1-21] and ET-3[1-21] were increased immediately after exposure to PM and O_3. 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 O_3 and particles, while altering lung preproET-1 and preproET-3 mRNA levels in a fashion similar to O_3 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:** Crl/WI, 250-300 g **Use:** Isolation of Rat Ventricular Cardiomyocytes and Cardiofibroblasts (RVCMs and RVCFBs)	Pigment Black Printex 90 (Frankfurt, Germany); PM: SRM 1648 **Particle Size:** Printex 90: 12-17 nm; 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:** 20 h	**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 et al. (2007, 097883) **Species:** Rat **Strain:** Wistar Kyoto **Cell Type:** Primary Vascular Smooth Muscle Cell Culture (VSMCs): isolated from thoracic aortas from 200-250 g 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:** PM_1, $PM_{2.5}$, PM_{10}	**Route:** In vitro **Dose/Concentration:** 10-100 µg/mL **Time to Analysis:** 3 days	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-κB-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 et al. (2003, 097247) **Species:** Rat **Strain:** Wistar Kyoto **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:** 18 h	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.
Reference: Upadhay et al. (2008, 159345) **Species:** Rat **Gender:** Male **Strain:** SH **Age:** 6 mo **Weight:** NR	Ultrafine Carbon Particles (UFCP) **Particle Size:** Size- 31 ± 0.3 nm, MMAD- 46 nm, Surface area concentration- 0.139 m^2 particles/m^3, Mass specific surface area- 807m^2/g	**Route:** Whole-body Inhalation **Dose/Concentration:** 172 µg/m^3 **Time to Analysis:** Acclimatized 2 day. 1 day baseline. 24 h exposure. 4 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.

Study	Pollutant	Exposure	Effects
Reference: Wallenborn et al. (2008, 191171) **Species:** Rat **Gender:** Male **Strain:** Wistar Kyoto **Age:** 13 wk **Weight:** NR	Zinc Sulfate ($ZnSO_4$, aerosolized) **Particle Size:** NR	**Route:** Nose-only Inhalation **Dose/Concentration:** 9.0 ± 2.1 µg/m^3, 35 ± 8.1 µg /m^3, 123.2 ± 29.6 µg /m^3 **Time to Analysis:** Exposed 5 h/day, 3 days/wk, 16 wk. 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: Wallenborn et al. (2007, 156144) **Species:** Rat **Gender:** Male **Strain:** WKY, SH, and stroke-prone SH (SHRSP) **Age:** 12-15 wk	PM: precipitator unit power plant residual oil combustion **Particle Size:** PM: 3.76 µm (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: Single,** 24 h Note: 4 h post-exposure study done on WKY vs SHRSP but not published.	**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: Wellenius et al. (2003, 055691) **Species:** Dog **Gender:** Female **Strain:** Mixed mongrel **Age:** NR **Weight:** 14-17 kg	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. 5-13 wk recovery. Pairs of subjects: exposed 6 h/day either 2nd or 3rd exposure time and filtered air other days. 5 min preconditioning occlusion. 20 min rest interval. 5 min experimental occlusion. Some dogs exposed 6 h/d, 4 days (consecutive), filtered air on day 4.	CAPs increased the ST-segment elevation and remained elevated 24 h after exposure. This increase was seen in precordial leads V4 and V5. 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 et al. (2004, 087874) **Species:** Rat **Gender:** Male **Strain:** SD **Age:** Adult **Weight:** ~250 g **Use:** Rat Model for Acute Myocardial Infarction (AMI): Left-ventricular MI induced. Animals allowed to recover for at least 12 h after surgery.	CAPs (Boston, MA); exposures during the period of 07/2000 and 01/2003. CO **Particle Size:** $PM_{2.5}$	**Route:** Whole-body Inhalation **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:** 1 h exposure to CAPs or CAPs+CO for 1 h. Exposure to pollutants was preceded and followed by 1 h exposure to FA.	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 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:** SD **Age:** Adult **Weight:** ~250 g **Use:** Rat Model for Acute Myocardial Infarction (AMI): Left-ventricular MI induced. Animals allowed to recover for at least 12 h after surgery.	CAPs: (Boston, MA) **Particle Size:** $PM_{2.5}$	**Route:** Whole-body Inhalation **Dose/Concentration:** CO: 35 ppm; CAPs (median concentration): 645.7 $\mu g.m^3$; CAPs+CO: 37.9 ppm **Time to Analysis:** CAPs or CAPs+CO exposure for 1 h. Exposure to pollutants was preceded and followed by 1 h 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:** 75 day	HP-12 (oil-combustion derived PM obtained from inside wall of a Boston power plant stack burning residual oil number 6). Water-leachable constituents (µg/mg): SO4 (217.3); Zn (11.4); Ni (6.9); Fe (0.0); V (1.3); Cu (0.2); Pb (0.0) 1M HCl-leachable constituents (µg/mg): SO4 (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:** 96 h or 192 h post-instillation.	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-instillation values until 72 h (HR) and 48 h (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:** SD **Use:** Left jugular vein and right carotid artery were cannulated.	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 ≤ 100 nm	**Route:** IV Infusion **Dose/Concentration:** UFDG (50 µg/m) **Time to Analysis:** Infused w/UFAA or UFDG. Monitored continuously for 1 h 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 et al. (2006, 097028) **Species:** Rat **Gender:** Female **Strain:** SD	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 ≤ 100 nm	**Route:** Lagendorff Heart Perfusion **Dose/Concentration:** UFDG (100 µg/2ml); UFID (12.5 µg/l in perfusate); SF-UFID (12.5 µg/l) **Time to Analysis:** Lagendorff 1: Treated w/UFDG. Lagendorff 2: Treated with UFID & SFUFID. Both experiments were monitored continuously for 1 h 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:** 42 wk **Weight:** 3.2 ± 0.1 kg (avg)	EHC-93 from Ottawa, Canada **Particle Size:** NR	**Route:** IT Instillation **Dose/Concentration:** PM_{10} suspension: 5 mg EHC-93 in 1 ml saline **Time to Analysis:** Exposed 2 times/wk for 4 wk. Acute effects observed at 0.5, 1, 2, 4, 8, 12, and 24 h after initial instillation. Subchronic effects observed once/wk for 4 wk.	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 CD49 day, 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.

Study	Pollutant	Exposure	Effects
Reference: Ying et al. (2009, 190111) **Species:** Mice **Gender:** Male **Strain:** ApoE$^{-/-}$ **Age:** 16 wk	CAPs:,New York City (Manhattan), NY; May-Sept 2007 **Particle Size:** $PM_{2.5}$	**Route: Whole-body** Inhalation **Dose/Concentration:** 138.4 ± 83.7 µg/m^3 **Time to Analysis:** 6 h/day, 5 day wk, 4 mo	**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-exposed mice.
Reference: Yokota et al. (2004, 096516) **Species:** Rat **Gender:** Male **Strain:** SD **Weight:** 345-498.2 g	DEP (obtained from the Japan Automobile Research Institute) **Particle Size:** NR	**Route:** IT Instillation **Dose/Concentration:** Group 1: DEP: 1 mg/0.1 ml; Group 2: DEP: 0.2 ml (10, 12.5 or 25 mg/ml); Group 3: DEP 2.5 or 5 mg/0.2 ml **Time to Analysis:** DEP pre-treatment 24-72 h before ischemia/reperfusion.	**DEP Effects on Mmyocardial 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 Rffects 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 72 h.
Reference: Yokota et al. (2005, 096003) **Species:** Rat **Gender:** Male **Strain:** SD **Weight:** 303-472.2 g	DEP from Japan **Particle Size:** NR	**Route:** IT Instillation **Dose/Concentration:** DEP: 5 mg/animal **Time to Analysis:** Single exposure 0.5, 1, 2, 3, 6, 12, 24, 48 h.	At 12 and 24 h post-instillation, circulatory neutrophil counts in the 5 mg DEP group were significantly elevated, and were 2.1-fold (12 h) and 2.3 fold (24 h) in vehicle treated animals. 1 mg DEP caused an increase of approximately 0.4-fold in CNC at 6 h. 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.

Study	Pollutant	Exposure	Effects
Reference: Yokota et al. (2008, 190109) **Species:** Mouse **Gender:** Male **Strain:** ddy **Age:** NR **Weight:** 39.6-46.0 g	DEP (DMSC (dichloromethane soluble-component), RPC (residual particle-component)) **Particle Size:** NR	**Route:** IT Instillation **Dose/Concentration:** 5 mg/kg, 10 mg/kg **Time to Analysis:** DMSC and RPC extracted from DEP. Mice acclimatized 7 day . DEP, DMSC, or RPC instilled. BALF and blood obtained and G-CSF, GM-CSF, IL-6 measured 2, 4, 12, 24 h post-instillation.	**Inflammation:** At 5 mg/kg DEP increased the total cell and macrophage count. DEP or RPC increased neutrophils at 5 and 10 mg/kg. 10 mg/kg DEP or RPC increased macrophages at 4 h and decreased at 12 h. **Hematology:** Compared to 5 mg/kg DEP, RPC increased RBC, WBC, and neutrophils. 10 mg/kg RPC or DEP caused sustained increases in RBC, WBC, and neutrophils. **Cytokines:** 5 mg/kg RPC markedly increased G-CSF and IL-6. Other cytokine increases at this dose were transient. 10 mg/kg DEP increased IL-6 at 4 h, and DEP or RPC increased G-CSF and IL-6 at 12 h. DEP or RPC also increased IL-1β. **Myocardium:** Myocardial MPO activity significantly increased in 5 mg/kg RPC at 12 and 24 h. Myocardial MIP-2 increased the most in 5 mg/kg RPC, while LIX tended to be lowered by RPC.

Table D-2. Respiratory effects: in vitro studies.

Study	Pollutant	Exposure	Effects
Reference: Aam and Fonnum (2007, 155123) **Species:** Human, Rat **Tissues/Cell Types:** Human-Neutrophil Granulocytes (NG); Rat- 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 = 5 µM **Time to Analysis:** 1 h	**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 1 h.
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, 8 h PC10 = 2, 6, 12, 24 h	**Calcium Imaging:** PC10 induced increase of Ca^{2+} 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 6 h, 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 48 h, 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 24-48 h in NHBE and SAEC. BEAS-2B showed slight decrease from 3% to 4% in same time period.

Study	Pollutant	Exposure	Effects
Reference: Agopyan et al. (2004, 156198) **Species:** Human, Mouse **Tissues/Cell Types:** Human-NHBE, SAEC; Mouse-Wildtype and TRPV1(-/-) Terminal Ganglion Neurons (TG)	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 = 6 h	**Calcium Imaging in NHBE and SAEC:** In 100% of reactive cells, ROFA/MSHA induced an increase in Ca^{2+}. Levels remained elevated as long as PM bound to plasma membrane. Washing and disjoining PM from membrane caused Ca^{2+} to slowly decline to baseline. CPZ (or CPZ and amiloride) reversibly inhibited PM-induced rises in Ca^{2+}. **Calcium Imaging in TRPV1(+/+) and (-/-) mice sensory neurons:** All sensitive neurons in TRPV1(+/+) increased Ca^{2+} in response to ROFA. No effect of ROFA in TRPV1(-/-). **cAMP:** ROFA and MSHA induced increases in Ca^{2+} 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 Ca^{2+}. **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 Ca^{2+} free environment.
Reference: Ahn 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:** 24 h	**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.
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 3 h 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 3 h and dephosphorylation after 5 h. In hTrx-1 cells, Akt remained phosphorylated after 5 h. In A549 cells, Akt phosphorylated at 3 h 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.

Study	Pollutant	Exposure	Effects
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, 72 h; Apoptosis: 24 h; DNA Breakage: 72 h; Cytokine Assays: 24 h	**Cytotoxicity:** Cytotoxic effect exhibited dose-dependency after 72 h 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 (10 µg/cm^2) to produce the same effect as other zones (2.5 µg/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.
Reference: Amakawa et al. (2003, 156211) **Species:** Mouse, Human **Strain:** Mouse-ICR **Tissues/Cell Types:** AMs **Gender:** Male **Age:** Mouse 6-7 wk; Human 20-24 yr	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: 5×10^5 cells/mL; Human: 3×10^5 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 24 h post exposure.	**Cells:** For mice, more than 90% of the cells were macrophages and over 90% were viable. For humans, 96% of the cells were macrophages, 3% lymphocytes and 1% neutrophils; over 95% of the human cells were viable. **DEP Cytotoxicity:** None observed **Cytokines:** DEP (10 µg/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 (10 µg/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.

Study	Pollutant	Exposure	Effects
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: 95 nm; 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 24 h	**Inflammatory Markers:** LDH of A549 was unaffected at either time point with DEP or CB. LDH increased with CSC at concentrations high than 10 µg/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 1 h. DEP effect was inhibited by N-acetylcysteine antioxidant pre-treatment. **MAP-Kinase:** DEP induced MMP-1 expression increased ERK1/2 phosphorylation after 10 min, peaking at 30 min, and returning to normal levels at 60 min. 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 mitochondria)	s-ROFA: soluble portion **Particle Size:** 1.95 ± 018 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 one 6-h 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 16 h 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.
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 µg/cm^2 **Time to Analysis:** Cells treated on apical side. Parameters measured 24 h following treatment.	**Cytotoxicity (LDH):** No cytotoxicity for DEP or $PM_{2.5}$ (80 µg/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.

Study	Pollutant	Exposure	Effects
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: 95 nm; TiO_2:150 nm; DEP: NR RER PM_{10}: 79% <0.5 µm, 20% 0.5-1 µm; Metro PM_{10}: 88% <0.5 µm, 11% 0.5-1 µm.	**Route:** Cell Culture (40,000 cells/mL) **Dose/Concentration:** All particles: 0.01, 0.1, 1, 10 µg/cm^2 **Time to Analysis:** 3, 8, 24 h	**Cell Viability:** No effects from any particulate at concentrations up to 10 µg/cm^2 for 24 h. **Inflammatory Effect:** Exposure of cells to 10 µg/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 µg/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 8 h. **Effects on HO-1 Protein Expression:** Exposure to 10 µg/cm^2 of RER or Metro PM_{10} for 24 h induced positive cytoplasmic staining for HO-1.
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 24 h	**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. β-cellulin increased with SC, WUB and WC. No data was available for SUB 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 24 h. DEP had no effect with GRO-α. Removal of metal from particles lowered response of GRO-α.

Study	Pollutant	Exposure	Effects
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, 72 h	**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 48 h. With DEP alone, cell growth was prevented from cell number reduction due to removal of serum at 48 and 72 h. A dose of 10 µg/mL induced a maximum proliferation effect. **Cell Cycle:** DEP increased the percentage of serum-starved cells in S phase at 48 h. 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 48 h. DEP dose-dependently decreased this expression. **NAC:** NAC alone, at 33 mM, induced an increase in cell numbers. DEP-NAC inhibited cell numbers at 48 h. 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. **SP**600125**:** Alone, SP600125 increased cell numbers at 33 mM. DEP-S decreased cell numbers.
Reference: Becher et al. (2007, 097125) **Species:** Rat **Strain:** Crl/WKY **Cell Type:** AM, Alveolar Type II **Gender:** Male **Weight:** 200 g	SPM = suspended PM SRM-1648 **Particle Size:** 6-8 µm	**Route:** Cell Culture (1.5×10^6 cells/well AM; 6×10^6 cells/well Type II) **Dose:** 200 µg/mL = 20 µg/cm^2 **Time to Analysis:** 20 h	**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.
Reference: Becker et al. (2005, 088590) **Species:** Human **Gender:** Male and Female **Age:** 18-35 yr **Cell Types:** Alveolar Macrophages, NHBE	PM (Coarse, Fine, Ultrafine): Chapel Hill, NC **Particle Size:** PM-C: $PM_{2.5}$; PM-F: $PM_{0.1}$; PM UF: <0.1µm	**Route:** Cell Culture ($0.5\text{-}1\times10^5$ cells/well NHBE; $2\text{-}3\times10^5$/mL AM) **Dose/Concentration:** NH BE: 25, 50, 100, 250 µg/mL of PM; AMs: 50 µg/mL of DEP or 10 ng/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.

Study	Pollutant	Exposure	Effects
Reference: Becker et al. (2005, 088592) **Species:** Human **Gender:** Male **Age:** 18-35 yr **Cell Types:** AM, NHBE	PM (Coarse, Fine, Ultrafine): Chapel Hill, NC 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 (3-5×10^5 cells/well NHBE; 2-3×10^5 cells/mL AM) **Dose/Concentration:** NHBE: 11 µg/mL of PM; AM: 50 µg/mL of PM **Time to Analysis:** 18-24 h NHBE; 18 h 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 EC (spark discharge) P90 = Printex 90 (Carbon Black, Degussa) PG = Printex G (Carbon Black, Degussa) **Particle Size:** DEP: 20-40 nm; EC: 5-10 nm; P90: 14 nm; PG: 51 nm	**Route:** Cell Culture (1×10^6 cells/mL AM) **Dose/Concentration:** All particles: 1 (EC only), 3.2, 10, 32, 100 µg/mL **Time to Analysis:** 60 min	**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.
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:** 90 nm (count median mobility diameter)	**Route:** Cell Culture (3×10^7 cells) **Dose/Concentration:** 44 ± 4 ng/cm^2; 87 ± 23 ng/cm^2; 230 ± 70 ng/cm^2 **Time to Analysis:** 6 h	**Cell Viability:** Exposure to clean air resulted in a 93.7 ± 9.1% viability. Exposure to low, mid and 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 cytotoxicity.

Study	Pollutant	Exposure	Effects
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, 30 h	**Amphiregulin Expression:** DEP and $PM_{2.5}$ both increased AR mRNA expression from 6 to 30 h, 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), **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: 95 nm NR **Particle Size:** CB: 95 nm; DEP: NR	**Route:** Cell Culture (3×10^6 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:** 24 h	**Proinflammatory Response:** At 10 µg/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 2-4 h and 1 h respectively. NF-κB DNA binding was enhanced by OE-DEP (15 µg/mL, peak <1 h) 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 (1×10^6 cells/mL J774A.1; 5×10^6 cells/mL PBMC; 5×10^5 cells/well A549) **Dose/Concentration:** PM_{10}: 75 µl (10 µg/mL); CM: 250 µl; tBHP: 12.5 µm (in J774); TNF: 0, 500 pg, 1 ng, 10 ng **Time to Analysis:** tBHP:1, 2, 4 h; PM: 4 h; TNF: 18 h	**Cytokines:** PM_{10} induced release TNF-α protein from PBMCs at 10 µg/mL for 4 h. 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 18 h. 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 (5×10^4 cells/well) **Dose/Concentration:** 30, 60 µg/cm^2 (aliquot of 0.1 µg/µl) **Time to Analysis:** 5, 24, 48, 72 h	**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 72 h 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-dependent manner **ROS:** PM increased ROS at the high dose for 5 h but not at 24 h or with the low dose. **Cytokines:** PM induced TNF-α peaked at 5 h at high dose and 48 h at low dose, both ND at 72 h. PM induced IL-6 starting at 24 h thru 72 h 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 (5×10^5 cells) **Dose/Concentration:** NIST-DEP, C-DEP: 0, 12.5, 25, 50, 100, 200 µg/mL **Time to Analysis:** 1-4 h	**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 H_2O_2. Reaction induced by H_2O_2 was similar to that of DEP. **pStat3 Nuclear Transport:** NIST-DEP induced cytoplasmic pStat3 to move from cytoplasm into nucleus. **pEGFR Dephosphorylation:** After 4 h of NIST-DEP exposure, dephosphorylation was inhibited for up to 90 min.
Reference: Chang et al. (2005, 097776) **Species:** Human **Cell Type:** A540, THP-1	UfCB (Printex 90, Degussa) **Particle Size:** 14 nm	**Route:** Cell Culture (7×10^5 cells) **Dose/Concentration:** 100 µg/mL **Time to Analysis:** 4 h	**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.

Study	Pollutant	Exposure	Effects
Reference: Chauhan et al. (2004, 096682) **Species:** Mouse **Strain:** BALB/c **Cell Type:** RAW 264.7; J774A.1; WR19M.1	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-y: 0-1000 U/mL **Time to Analysis:** Particles added to culture at 0h, LPS and IFN-γ added at 2 h. Parameters measured after 22 h incubation period.	**Stimulation with LPS/IFN-y:** LPS and IFN-y each induced NO release. Combination of LPS and IFN-y produced larger effect in all cell lines. L-NMMA, NOS inhibitor, suppressed most of the NO production with 100 nmol/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.
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:** 24 h	**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: 15.9 nm; DEP-LS: 17.7 nm; GEP: 8.3 nm	**Route:** In Vitro Cellular Exposure (Exhaust flow-through cell culture with air-cell-interface, exhaust diluted 10-15x with 8×10^5 cells/mL) **Dose/Concentration:** DEP (total): 1.5-3.5×10^6 particles/cm^3; GEP (total): 1-2×10^6 particles/cm^3; 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 6 h. 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.

Study	Pollutant	Exposure	Effects
Reference: Chin et al. (2003, 156340) **Species:** Rat, Human **Cell Line/Type:** RAW 264.7, MHS (Alveolar Macrophage Cell Line), A549	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, 4 µg/mL BaP: 2 µg/mL BP-1,6-Q: 1 µM **Time to Analysis:** 1-24 h	**HO-1 mRNA Expression:** In RAW264.7, HO-1 mRNA levels increased with 2 and 4 µg/mL at 2 h. Increases continued to 8 h and declined by 24 h. BaP had no effect. BP-1,6-Q increased HO-1 mRNA after 1 h and was maintained until 8 h. In A549 and MHS, HO-1 mRNA increased after 1 h, peaking at 8 h in A549 and 4 h in MHS. **HO-1 Protein Expression:** An increase of protein was observed from 4-8 h in RAW264.7. **AP-1:** Increases in binding activity were observed in RAW 264.7 cells at 2 h.
Reference: Churg et al. (2005, 088281) **Species:** Rat **Strain:** SD **Weight:** 250 g **Cell Type:** Epithelial Cells of Tracheal Explants	EHC93 (Ottawa Urban Air Particles) TiFe = Iron-loaded fine TiO_2 (obtained from Aldrich Chemicals, Milwaukee, WI) **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, 24 h. Some experiments (referred to as 2 h) explants transferred to different dish and incubated for additional hour. Pre-treated with Inhibitors/Chelators for 2 h.	**Activation of NF-κB:** Both particle types increased nuclear translocation of NF-κB. TiFe and EHC-93 increased NF-κB 1.5 fold at 1 h. TiFe increased NF-κB 3.5 fold at 2 h. EHC-93 increased NF-κB more than 2 fold. TiO_2 by itself did not increase NF-κB at any exposure duration. **Morphological changes in tracheal epithelial cells:** No evidence of dust particles was observed (EHC-93 or TiO_2) in the epithelial cell cytoplasm at 2 h. No evidence of morphologic cell damage from particles was observed. **Colchicine:** Treatment with colchicine did not prevent NF-κB 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% in, 4-7% , 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- 13 nm, P60- 21 nm	**Route:** Cell Culture **Dose/Concentration:** 100, 200 µg/mL **Time to Analysis:** 24 h 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 (3×10^6, 1.5×10^6, 0.75×10^6 cells/20mL) **Dose/Concentration:** LC10: 19 µg/mL; LC50: 75 µg/mL **Time to Analysis:** 24, 48 or 72 h	**p65 Protein:** Phosphorylation of p65 increased in PM-exposed L132 cells in dose-dependent manner. **IκBα Protein:** Phosphorylated IκBα protein concentrations increased in cytoplasm with both particle types at all time points. **p65 and p50 DNA:** p65 DNA binding increased at 24 h with LC10 and LC50, at 48 h with LC10, and at 72 h with LC10 and LC50. p50 DNA binding increased at all time points with LC10 and LC50.

Study	Pollutant	Exposure	Effects
Reference: Dai et al. (2003, 087944) **Species:** Rat **Strain:** SD **Weight:** 250 g **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 1 h. Parameters measured following a 7 day 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 expression could 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.
Reference: Doherty et al. (2007, 096532) **Species:** Rat **Strain:** NR8383 **Cell Types:** AMs	Ratios of: V: Fe; Al: Fe; Mn: Fe V = sodium vanadate ($NaVO_3$) 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 (2×10^5 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:** 20 h	**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. **Cytotoxicity:** 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) 0.5 um **Particle Size:** NR	**Route:** Cell Culture **Dose/Concentration:** DEP and CB: 1-100 µg/mL **Time to Analysis:** Parameters measured 24, 48, 72 h post exposure. 1-HBE Cell Deadhesion Capacity: 24 h, evaluation of detachment performed every 5min for 40 min 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 72 h. **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 100 µg/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 5 min of incubation and onward. **Cell Wound Repair Capacity:** DEP inhibited wound repair/wound closure in a dose-dependent manner.

Study	Pollutant	Exposure	Effects
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, 6 h	**IL-1β:** Increased levels of IL-1β with asbestos and silica were observed in THP1 at 6 h. 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 5 h. Analysis 9 h 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.
Reference: Duvall et al. (2008, 097969) **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-Sterling Forest, NY (PM-SF) **Particle Size:** Coarse: >2.5 µm; Fine: <2.5 µm; UFP: <0.1 µm	**Route:** Cell Culture (100,000 cells/cm^2) **Dose/Concentration:** 5 mg/ml **Time to Analysis:** 1, 24 h 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 EC. **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 EC, 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 EC, caused an increase.
Reference: Dybdahl et al. (2004, 089013) **Species:** Human **Cell Type:** A549	DEP: SRM 1650 (NIST) **Particle Size:** 90 nm (MMAD)	**Route:** Cell Culture (10^5 cells/mL) **Dose/Concentration:** 0, 10, 50, 100, 500 µg/mL **Time to Analysis:** 2, 5, or 24 h	**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 h, 8 fold at 5 h, and 2 fold at 2 h. **Cell Viability:** DEP exposure did not decrease cell viability at any dose tested.

Study	Pollutant	Exposure	Effects
Reference: Fritsch et al. (2006, 156452) **Species:** Mouse **Cell Type:** RAW 264.7	$MAFO_2$: 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:** 165 nm (modal value)	**Route:** Cell Culture (1×10^6 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 2.5 h, 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 5 h. **MAPKs:** Cells pretreated with PD98059, an inhibitor of MEK-1, inhibited AA liberation due to $MAFO_2$ treatment of 13.2 µg/cm^2 **COX-2:** A time-dependent increase of COX-2 protein expression was exhibited at 2.5 and 5 h. **ROS:** A dose-dependent increase in ROS formation was observed at concentrations greater than 31.3 µg/cm^2 after 3 h. **GSH:** There was an observed increase of production at 20 h. Doses greater than 60 µg/cm^2 reduced total glutathione. **HO-1:** There was an observed dose-dependent increase in expression at 4 h.
Reference: Fujii et al. (2002, 036478) **Species:** Human **Cell Type:** HBEC (from current smokers), AMs, Co-Culture: AMs+HBEC **Age:** HBEC: 48-70 yr	PM_{10}: EHC-93 (Ottawa, Canada) **Particle Size:** PM_{10}	**Route:** Cell Culture (HBEC: 2.5-3× 10^6 cells/well); (AMs: 1.0×10^7 total) **Dose/Concentration:** 100, 500 µg/mL **Time to Analysis:** 2, 8, 24 h	**Viability:** Over 90% of HBEC were viable after a 24 h exposure of up to 500 µg/mL of PM. AMs incubated with and without 100 µg/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 8 h. In AMs, TNF-α, IL-1β, IL-6 mRNA expression increased with 100 µg/mL at 2 h. IN HBECs, IL-1β and LIF increased with 100 µg/mL at 2 h. 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 et al.. (2001, 156455) **Species:** Human **Cell Type:** HBEC from current smokers **Age:** 48-70 yr	PM_{10}:EHC93 (Ottawa, Canada)99% <3.0um **Particle Size:** PM_{10}(99% < 3.0 µm)	**Route:** Cell Culture ($2.5\text{-}3\times10^6$ cells/dish) **Dose/Concentration:** 10, 100, 500 µg/mL **Time to Analysis:** 2, 8, 24 h	**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 2 h but declined at 8 and 24 h. GM-CSF increased dose-dependently at 8h and peaked at 24 h. IL-1α increased at 2 h, 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 et al. (2006, 096633) **Species:** Human **Cell Type:** L132	$PM_{2.5}$ (collected in Dunkerque, France for 9mo, Jan-Sept) **Particle Size:** $PM_{2.5}$- 0-0.5 µm (33.63%), 0.5-1.0 µm (30.61%), 1.0-1.5 µm (14.33%), 1.5-2.0 µm (8.69%), 2.0-2.5 µm (4.89%), >2.5 µm (7.87%)	**Route:** Cell Culture: 3×10^6 cells/20ml (24 h); 1.5×10^6 cells/20ml (48h); 0.75×10^6 cells/20ml (72 h) **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 72 h	**Cytotoxicity:** PM induced dose-dependent (R_2 =.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 et al. (2005, 096689) **Species:** Rat **Strain:** Wistar Kyoto **Tissue/Cell Type:** Lung macrophages	BPM: Blowing $PM_{2.5}$; PM collected from Wuwei City, Gansu Province, China (Blowing days correspond to desert storm days) NPM: Non-blowing (normal) $PM_{2.5}$ **Particle Size:** $PM_{2.5}$	**Route:** Cell Culture **Dose/Concentration:** 0, 33, 100, 300 µg/mL **Time to Analysis:** 4 h	**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 100 µg/mL. Overall, NPM induced a slightly higher increase than BPM. **Intracellular Ca2+:** 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 et al. (2006, 097026) **Species:** Rat **Strain:** Wistar Kyoto **Tissue/Cell Type:** Lung macrophages	DPM: dust storm samples; PM collected from Baotou City, Inner Mongolia, China in March 2004 NPM: normal PM **Particle Size:** $PM_{2.5}$	**Route:** Cell Culture **Dose/Concentration:** 0, 33, 100, 300 µg/mL **Time to Analysis:** 4 h	**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. $Ca^{2+}Mg^{2+}$-ATPase were unaffected. **Plasma Membrane Lipid Fluidity**: Results indicate that DPM could increase the surface fluidity of membrane lipid. **Intracellular Ca2+:** 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:** 100 µM FAC - preexposed before metal compounds or oil fly ash 50 µM $VOSO_4$ - preexposed before metal compounds or oil fly ash 100 µg/mL ROFA **Time to Analysis:** 0-1 h, 4 h	**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:** SD **Cell/Tissue Type:** AM	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 µm; Fine: <2.5 µm; UFP: <0.2 µm	**Route:** Cell Culture (2×10^5 cells/mL) **Dose/Concentration:** 125 µg/mL or 250 µg/mL **Time to Analysis:** 4 or 24 h	**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:** 4 h, 6 h, 20 h	**IL-8**: PM_{10} at 50 µg/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 20 h. 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 6 h. 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 20 h.
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 H_2O_2 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; $H2O_2$: 200 µM; NAC and Mannitol: 5 mM **Time to Analysis:** 24 h	**IL-8:** PM_{10}, TSA and $H2O_2$ treatment induced an increase of IL-8. Concomitant exposure of TSA with PM_{10} or H_2O_2 significantly increased IL-8 release when compared to PM_{10} or H_2O_2 alone. IL-8 mRNA expression with PM_{10} or H_2O_2 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 H_2O_2 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 H_2O_2 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-κB:** The activation of the transcription factor NF-κB was enhanced following the inhibition of HDAC with TSA and by treatment with

Study	Pollutant	Exposure	Effects
Reference: Graff 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:** 6 h, 24 h	**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 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, 72 h	**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 increased 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 (1.5×10^6 cells/well) **Dose/Concentration:** 50, 100 µg/mL PM **Time to Analysis:** 20 h	**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 50 µg/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 et al. (2004, 097535) **Species:** Rat, Human **Cell Type:** Alveolar Macrophages (Rat), A549 **Strain:** Wky/NHsd **Gender:** Male **Weight:** 180-230 g	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 (1×10^6 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 100 µg/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 600 µg/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 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: 223 nm (mean diameter); ALI: 122 nm (mean diameter)	**Route:** Suspension (1×10^5 cells/cm^2), Air Liquid Interface (ALI, 1×10^5 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: 2.3×10^7 particles/cm^2) **Time to Analysis:** Exposure for 6 h. Parameters measured 20 h post-exposure.	**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/cm^2 or 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, RAW 264.7	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 (5×10^5 cells/mL) **Dose/Concentration:** All PM: 50, 70, 100 µg/mL **Time to Analysis:** BEAS-2B: 8h; RAW 264.7: 16 h	**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 (R_2 = 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 . **TNF-α:** In RAW264.7, PMSM increased TNF-α production. Polymixin pretreatment significantly reduced TNF-α levels for all 3 PMs which indicates an endotoxin role in macrophage response. TNF-α 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 7 day 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:** 4 h	**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 et al. (2007, 155859) **Species:** Rat **Gender:** Female **Age:** 12-14 wk **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), $H2O_2$ or catalase **Particle Size:** CAPs: ≤2.5 µm; UAP: $PM_{2.5}$; TiO_2: ~1 µm	**Route:** Cell Culture ($2×10^5$ cells/well) **Dose/Concentration:** Caps 100 µg/mL; UAP: 50 or 100 µg/mL; LPS 250 ng/mL; NAC, DMTU: 2, 10, 20 mM; Catalase: 1, 5, 10 mM; H_2O_2 0-50 µm/hr **Time to Analysis:** 18-20h	**TNF-α:** DMTU at 20 mM reduced TNF in LPS-primed cells in control and UAP-treated groups. NAC at 20 mM 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. H_2O_2 increased TNF release in CAPs-exposed cells. TiO_2 had no increased ability to induced cytokine release when mixed with H_2O_2. **Cell Death:** Viability decreased substantially when exposed to H_2O_2 + CAPs. The soluble fraction of CAPs showed to be more effective with H_2O_2 than the insoluble portion. TiO_2 had no significant effect. **NO:** Some CAPs induced slight increases when mixed with H_2O_2. No difference was observed between soluble and insoluble portions of CAPs. **DFO:** DFO at 0.05 mM completely inhibited oxidation induced with soluble CAPs + H_2O_2. Insoluble CAPs + H_2O_2 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 ($1×10^7$ cells) **Dose/Concentration:** 100 µg/mL **Time to Analysis:** 3, 6, 24 h	**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 3 h. Only the MCP-1 levels were statistically significant at 3 h. Levels declined by 6 h. 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 6 h 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.

Study	Pollutant	Exposure	Effects
Reference: Ishii et al. (2005, 096138) **Species:** Human **Cell Type:** AMs (obtained from 10 smokers who stopped smoking 6 wk prior), HBEC	EHC-93: PM_{10} (obtained from Environmental Health Directorate, Ontario, Canada) **Particle Size:** PM_{10}	**Route:** Cell Culture (HBEC: 2.5-3.0×10^6 cells; AM: 1×10^7 cells; co-culture of AM/HBEC: 5×10^6 cells) **Dose/Concentration:** 100 µg/mL **Time to Analysis:** 2, 24 h	**mRNA Expression After 2 h 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 24 h exposure to PM, HBEC exhibited an increase in expression. Expression in AMs were not affected by 2 h PM stimulation. **ICAM-1 Inhibitors:** IgG or anti-CD11b antibody was unaffected in co-culture.
Reference: Jalava 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 (5×10^5 cells/mL) **Dose/Concentration:** 150 µg/mL **Time to Analysis:** Methanol treatment of PM samples: 24 h; Exposure to ambient PM samples: 2, 4, 8, 16, or 24 h.	**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 2 h exposure time was sufficient to induce the typical reductions in cell viability.
Reference: Jalava 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:** $PM_{10-2.5}$; $PM_{2.5-1}$; $PM_{1-0.2}$; $PM_{0.2}$	**Route:** Cell Culture (5×10^6 cells/mL) **Dose/Concentration:** 15, 50, 150 and 300 µg/mL **Time to Analysis:** 24 h	**Particulate Mass Concentrations in HVCL Size Ranges:** The largest increase of PM concentrations was observed in $PM_{1-0.2}$. **NO:** All 12 samples increased NO production when compared to corresponding unexposed controls. Peaks were observed at 150 µg/mL, except in $PM_{1-0.2}$. **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 $PM_{1-0.2}$ and $PM_{0.2}$. IL-6 production for $PM_{0.2}$ 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).

Study	Pollutant	Exposure	Effects
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; $PM_{0.2}$: <0.2 µm	**Route:** Cell Culture (5×10^5 cells/mL) **Dose/Concentration:** 15, 50, 150, 300 µg/mL **Time to Analysis:** 24 h	**PM Characterizations:** The highest mass concentrations of PM_{10} and $PM_{0.2}$ 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 $PM_{0.2}$ 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 $PM_{0.2}$ 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. $PM_{0.2}$-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 $PM_{0.2}$ 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. $PM_{0.2}$ effects on IL-6 response were negligible. **Cytotoxicity:** The average cytotoxicity of PM_{10} and $PM_{2.5}$ were roughly equal, but $PM_{0.2}$ 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: Tioxide Europe (London, UK) and Degussa-Huls (Cheshire, UK) $UFTiO_2$: Tioxide Europe (London, UK) and Degussa-Huls (Cheshire, UK) **Particle Size:** PM_{10}, TiO_2: 200 nm; $UFTiO_2$: 20 nm	**Route:** Cell Culture (110,625 cells/well) **Dose/Concentration:** PM_{10}, TiO_2, $UFTiO_2$: 100 µg/mL; TNF-α: 10 ng/mL **Time to Analysis:** 4 h	**NF-κB and AP-1 DNA Binding**: NF-κB DNA binding increased in PM_{10} and TNF-α exposed macrophages by 9.5 and 12 fold. NF-κB activity remained unaltered in TiO_2 and $UFTiO_2$ 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 $UFTiO_2$ 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 $UFTiO_2$ 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 $UFTiO_2$ media.

Study	Pollutant	Exposure	Effects
Reference: Jung et al. (2006, 132421) **Species:** N/A **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: 185 nm; CB: 25 nm, $PM_{2.5}$	**Route:** Surrogate Lung Fluid **Dose/Concentration:** Soot: 0-30 mg; CB: 5-10 mg; $PM_{2.5}$: 50 or 100 µg/mL **Time to Analysis:** Parameters measured continuously over 2 h.	**OH Radical Formation:** Formation occurred with linear dependence on soot mass. Average response was 0.89 nmol 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 ± 172 nM. 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-κB inhibitor (coexposure) IL-1ß: obtained from Santa Cruz Biotechnology (Santa Cruz, CA) **Particle Size:** DEP: 0.3 µm (mean diameter)	**Route:** Cell Culture (3-4×10^5 cells) **Dose/Concentration:** DEP 25, 100, or 250 µg/mL; IL-1ß: 100 ng/mL **Time to Analysis:** DEP: 4 h pre-treated with BAY11-7082 for 1.5 h; IL-1β: 4 h	**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-κB 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 4 h. 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:** Cell Culture **Dose/Concentration:** Suspension: 40 µg/cm^2; Culture: 100 µg/cm^2 (1 ml/well) **Time to Analysis:** Suspension: 4 h; Culture: 18 h	**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 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 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_2O_3, Fe_2O_3, 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 0.2 mg/mL; Coals 0.7 mg/mL; Diesel 0.01 mg/mL; Al_2O_3 0.5 mg/mL; Fe_2O_3 0.7 mg/mL; SiO_2 0.7 mg/mL; TiO_2 0.7 mg/mL; ZnO 0.05 mg/mL **Time to Analysis:** 24 h	**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 24 h/day for 12 days. 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 interaction **Dose/Concentration:** 5-10 ml of 0.5 M NaCl or BALF **Time to Analysis:** Filters treated with BALF for 4 h	**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	Zn^{2+} **Particle Size:** NA	**Route:** Cell Culture **Dose/Concentration:** 15, 50, 100 µmol **Time to Analysis:** 1-20 h	**Cell Viability:** At 50 µM for 20 h, no apoptosis was induced. **IL-8:** At 12 h, IL-8 increased in dose-dependent manner. At 15 or 50 µM, Zn^{2+} 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 4 h. **EGFP (adenoviral IL-8 promoter):** Levels increased 2.4 fold with 50 µM Zn^{2+}. **Proteases:** With 50 µM Zn^{2+}, phosphorylation of MAPKs ERK, JNK and p38 increased by 15 min and continued increasing up to 2 h. Pre-exposure of inhibitors of MEK, JNK, before Zn^{2+} 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 Zn^{2+}.

Study	Pollutant	Exposure	Effects
Reference: Kleinman et al. (2003, 087938) **Species:** Rat **Strain:** Wistar Kyoto, F344 **Age:** 22-24 mo, 10 wk **Cell Type/Line:** AM	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, 18 h	**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 et al. (2008, 198874) **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 (1×10^6 cells/mL) **Dose/Concentration:** 30-280 µg/mL **Time to Analysis:** 2, 5, 12 h	**Particle Characterization:** PMT+ contained a high mineral particle content. PMT- contained carbon aggregates, 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 significant increases of IL-8. DEP organic extract had no effect.

Study	Pollutant	Exposure	Effects
Reference: Kristovich 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: 150 nm	**Route:** Cell Culture (4×10^6 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 24 h	**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.
Reference: Kubatova et al. (2006, 198835) **Species:** Rat, Human **Cell Type/Line:** RAW 264.7, BEAS-2B	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:** 12 h	**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 µl) **Dose/Concentration:** 0, 50, 100, 150, 200, 250, 300 µg/mL **Time to Analysis:** Cytotoxicity: 24 h; Chomotest: 2 h SOS	**Cytotoxicity:** 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.

Study	Pollutant	Exposure	Effects
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 **Particle Size:** MEP 0.5 µm; MEPE<0.2 µm	**Route:** Cell Culture (1×10^5 cells/well) **Dose/Concentration:** MEP 0.02, 0.2, 0.2, 2, 20 µg/mL; MEPE 20 µg/mL **Time to Analysis:** 24 h	**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. **NF-κB:** MEP (20 µg/l) induced time-dependent activation for 2 h and continued at same level for up to 6 h. 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 6 h.
Reference: Lee and Kang (2002, 198864) **Species:** Mouse **Cell Type/Line:** Peritnoeal Macrophages, RAW 264.7	MEP Yamaha 2-stroke engine using unleaded gas) MEPE(particle-free MEP) **Particle Size:** 0.5 µm	**Route:** Cell Culture (5×10^5 cells/mL (Cytotoxicity), 3×10^5 cells/mL (Apoptosis), 2×10^6 cells (MMP and ROI), 1×10^7 cells (GSH) **Dose/Concentration:** 5, 10, 50, 100, 300, 1000 µg/mL **Time to Analysis:** 6, 12, 18, 24 h	**Cytotoxicity:** Viability decreased dose and time-dependently in all cell types at 24 h. **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. **Ca2+**: 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	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 (2×10^6 cells/well Mouse RAW 264.7 and THP-1; 0.67×10^6 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 264.7 only) 8 h. HO-1, MnSOD Expression: RAW 264.7, THP-1 7h. RAW 264.7 cells exposed to whole DEP 16 h. JNK Activation, IL-8 Production: THP-1 cells 30 min, 16 h. RAW 264.7 cells 90 min. Cytotoxicity: RAW264.7, THP-1 18 h.	**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	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, 120 min	**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 100 µg/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 2 h. 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) **Particle Size:** PM_{10}: Bimodal with peaks around 4-5 um and 7-8 um.	**Route:** Cell Culture (130,000 cells/cm^2) **Dose/Concentration:** 1, 10 or 100 µg/mL **Time to Analysis:** 18, 24 h Analysis of Arachidonic Release (AA): Cells pre-incubated w/ 1 µCI 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 10 µg/mL. PM-Q induced increase of TNF-α at 100 µg/mL.PM-ST induced IL-6 release at 10 µg/mL. PM-G, PM-Q, PM-S induced IL-6 secretion at 100 µg/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 100 µg/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 10 µg/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) **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:** 0-4 h; Mitochondrial Membrane Destabilization: 0-60 min; DNA Defragmentation: 0-6 h; Cytotoxicity: 24 h	**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 O_2- and $H2O_2$ 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 4 h, 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 2 h and returned to basal level at 4 h after exposure. Levels of procaspase-3 and caspase-9 were unaltered by SE exposure after 4 h. Procaspase-3 increased and caspase-9 decreased by $H2O_2$ exposure. SE exposure increased levels of AIF and EndoG (exposure up to 4 h). At 6 h, 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 (5×10^6 cells, 2 mL /well MDMs) **Dose/Concentration:** 5 µg/cm^2 **Time to Analysis:** 2-24 h	**ROS 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 cell free 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, 48 h; NF-κB and AP-1: 12 h Phosphorylation of Akt: 5- 120 min. Effect of LY294002 on DEP: Cells pretreated with LY294002 (0 or 10 µM) for 30 min and then exposed to DEP for 0-60 min.	**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	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 . * 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-κB) (9×10^4 cells/well) **Dose/Concentration:** CAPS 109 ± 178 $\mu g/m^3$ (air exposure); 300 µg PM/ml (culture) **Time to Analysis:** 24 h	**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).

Study	Pollutant	Exposure	Effects
Reference: Madden et al. (2003, 198877) **Species**: Human **Cell Type:** NHBE	DEP(SRM 2975) Diesel Exhaust Extracts from a High load (HL~75% engine load) or Low load (LL 0% engine load): Obtained from Caterpillar diesel engine, 4 cycl, 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:** 24 h (after 2 h of treatment, 0.5 ml of BEGM added to each well and cells incubated for an additional 22 h) .	**Cytotoxicity:** LL, HL and SRM had no effect on LDH release. **51Cr:** 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 51Cr release. **IL-8:** HL induced a 5-fold increase in IL-8 at 500 µg/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.
Reference: Matsuo et al. (2003, 198879) **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:** 0.4 µm (MMAD)	**Route:** Cell Culture (NHBE: 5×10^4 cells/cm^2; NHPAE: 3×10^3 cells/cm^2; TIG-1 and TIG -7: 3×10^3 cells/cm^2; Apoptosis: 2×10^5 cells/cm^2; ROS/NO: 2×10^4 cells/cm^2; Cytotoxicity Modulating Agent: 3×10^4 cells/cm^2; GSH: 3×10^4 cells/cm^2) **Dose/Concentration:** 25, 50, 100, 200, 300, 400, 500 µg/mL **Time to Analysis:** 1 h	**Cytotoxicity 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 LC50 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 increases 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 24 h. 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 et al. (2006, 199517) **Species:** Human **Cell Type:** Peripheral neutrophils **Gender:** Male and Female **Age:** 20-40 yrs	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 (5×10^5 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 H_2O_2: 5, 10, 30, 60 µg/mL MMP-9, LTB-4: 5, 10, 30, 60 µg/mL **Time to Analysis:** f-Actin: 15 min CD11b: 2 h IL-8: 2 or 24 h H_2O_2: 30 min MMP-9, LTB-4: 2 or 24 h	**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 2 h. A significant increase was observed (243%) at 24 h with 30 µg/mL. **LTB-4:** At 2 h, 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%. **H_2O_2:** 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.

Study	Pollutant	Exposure	Effects
Reference: Molinelli et al. (2006, 198949) **Species:** Human **Cell Type:** NHBE, BEAS-2B	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) **Particle Size:** PM_{10}	**Route:** Cell Culture (3×10^3 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:** 48 h	**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.
Reference: Moller et al. (2002, 036589) **Species:** Canine, Mouse **Cell Type:** Beagle-Dog Alveolar Macrophages (BD-AM), J774A.1	$fTiO_2$ (origin NR) $ufTiO_2$ (origin NR) ufP-G: carbon black (Printex-G, Degussa, Frankfurt, Germany) ufP90: carbon black (Printex90, Degussa, Frankfurt, Germany) ufEC90: EC (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: 220 nm; $ufTiO_2$: 20 nm; ufP-G: 51 nm; ufP90: 12 nm; ufEC90: 90 nm; DEP: 120 nm; UrbD: NR	**Route:** Cell Suspension **Dose/Concentration:** 10, 32, 100, 320 µg/mL **Time to Analysis:** 24 h	**Cytoskeleton of J774A.1:** At doses of 32 µg/mL or less, the particles did not significantly influence relaxation and stiffness. $fTiO_2$and ufP90 had no effect at any dose. $ufTiO_2$ 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:** $ufTiO_2$ and $fTiO_2$ both induced some retarded relaxation and increased stiffening at 100 µg/mL dose. $ufTiO_2$ 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, $ufTiO_2$ and $fTiO_2$ significantly reduced phagocytic 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:** $ufTiO_2$ significantly inhibited proliferation compared to the control and $fTiO_2$ 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 $ufTiO_2$ inducing greater apoptosis than $fTiO_2$, ufEC90 than ufP90 and ufEC90 than ufP-G.
Reference: Mutlu et al. (2006, 155994) **Species:** Human, Rat **Cell Type:** A549	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 $\mu g/cm^2$ **Time to Analysis:** 24 h	**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.

Study	Pollutant	Exposure	Effects
Reference: Nam et al. (2004, 198887) **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, 24 h	**NF-κB/IκBα:** 50 µg/cm^2 DEP induced IκBα degradation which peaked at 2 h and recovered after 4 h. Treatment with increasing amount of $PM_{2.5}$ resulted in a dose-dependent decrease in IκBα. $PM_{2.5}$ increased NF-κB in a dose-dependent manner up to 10 µg/cm^2. NF-κB induction peaked at 12 h. **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 NF-κB activity. Antioxidants and iNOS inhibitor reduced PM-induced IκBα degradation.
Reference: Nozaki et al. (2007, 097862) **Species:** Mouse **Cell Line:** LA-4 (Alveolar Epithelial Cells)	PM: Rooftop of 5 story building, 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: 14 nm	**Route:** Cell Culture (1.4×10^4 cells/cm^2) **Dose/Concentration:** 1.1 µg/cm^2 **Time to Analysis:** 24, 28, 72 h	**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).
Reference: Obot et al. (2002, 042370) **Species:** Mouse **Cell Line:** BALB/c **Cell Type:** AM	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 (5×10^5 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:** 4 h	**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, O_2, N, S, Si, Ca, Al, P, Cl. PM-PH mostly contained O_2 and Si. PM-500 had increased O_2, 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 (7-9wk), Human **Cell Line:** Mouse-BALB/c **Cell Type:** AM	PM: SRM 1648 (collected by bag-house 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 PMH_2O: 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 (5×10^5 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-4 h; Human-24 h.	**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 PM2.5:** 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 PM2.5:** Excellent correlation of mouse and human responses for viability and apoptosis was exhibited.

Study	Pollutant	Exposure	Effects
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:** 24 h	**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 cytotoxic 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 6 h. After 12 h 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.
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) **Particle Size:** NR	**Route:** Cell Culture (50000 cells/well) **Dose/Concentration:** 0.001, 0.01, 0.1, 1.0, 10 mM **Time to Analysis:** 24 h	**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 EC50 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 µg/cm^2 **Time to Analysis:** 24-72 h	**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 µg/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.

Study	Pollutant	Exposure	Effects
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: <2.5 µm -P2: 2.5-10 µm -P3: >10 µm BDS-W: solvent washed Graphite Composition: <2.5 µm = 92%, 2.5-10 µm = 5%, >10 µm = 3% **Particle Size:** BDS-P1: <2.5 µm; BDS-P2: 2.5-10 µm; BDS-P3: >10 µm	**Route:** Cell Culture (1-1.5×10^6 cells) **Dose/Concentration:** 3 mg BDS **Time to Analysis:** 5 min-72 h	**Particle Characterization:** By weight, EC 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 60-120 min, BDS was observed in the cells. At 4 h, fluorescence observed in cytoplasmic vesicles and increased during the first 24 h then plateaued for the next 72 h. BDS-W appeared in vesicles sooner than BDS.
Reference: Pozzi et al. (2005, 088610) **Species:** Mouse **Cell Type:** RAW 264.7	PM: Collected continuously for 15 days, 8-10 m 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: 200-250 nm	**Route:** Cell Culture (1.3×10^5 cells/well) **Dose/Concentration:** 30 µg/mL; 14 µg/cm^2 120 µg/mL; 54 µg/cm^2 **Time to Analysis:** 5, 24 h	**Cytotoxicity:** For 24 h, 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 5 h. 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 5 h for both concentrations. PM-C at 30 µg/mL induced a much greater TNF-α release than PM-F at 5 h. **IL-6:** PM-F significantly increased at 5 h for both concentrations. Elevated IL-6 levels were exhibited at both PM-C doses at 24 h. At 5 h, 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 5 h.
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: $MnCl_2•4\ h_20$ Fe: $FeCl_3•6H_20$ **Particle Size:** $PM_{2.5}$	**Route:** Cell Culture (2×10^5 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 × Fe(III) Al and Fe(III) mixtures with Al in molar ratios 0.37, 0.75, 2, 7.5 × Fe(III) **Time to Analysis:** 20 h	**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.

Study	Pollutant	Exposure	Effects
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, 18 h	**CRP:** Treatment with ufCB or PM_{10} produced an increase in CRP expression with similar effects noted after 6 h. 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:** SD **Cell Type:** AMs and cultured lung fibroblasts	DEP: SRM 2975 (NIST) **Particle Size:** 0.5 µm	**Route:** Cell Culture **Dose/Concentration:** 200 µg/mL **Time to Analysis:** 4 h	**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.
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, 18 h	**Cytotoxicity:** After treatment, cells were more than 90% viable. UFPM and FPM caused no gross alterations in cell morphology or adhesion. **MIP3α/CCL20 mRNA (6 h):** 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 (18 h):** 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 (18 h):** 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.

Study	Pollutant	Exposure	Effects
Reference: Riley et al. (2003, 053237) **Species:** Rat **Cell Type:** RLE-6TN (Type II Alveolar Epithelial Cells)	Zn: $ZnCl_2$ Cu: $CuCl_2$ Fe: $FeCl_2$ V: VCl_4 Ni: $NiCl_2$ **Particle Size:** NR	**Route:** Cell Culture **Dose/Concentration:** 0.01, 0.1, 1.0, 10 mM **Time to Analysis:** 2, 4, 24, 72 h	**Cytotoxicity (SDH):** All particles were cytotoxic in a dose-dependent manner. Zn and V were cytotoxic at 0.05 mM, Cu at 0.5 mM, Ni at 0.8 mM and Fe at 2 mM. For Zn, cell death (LDH) had a biphasic response: a slow logslope until approx 0.1 mM at which point it rapidly accelerated to a peak at 5 mM with a small decline at 10 mM. 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 36-48 h followed by a 2-fold recovery by 72 h. Cu showed a faster, steady decline plateauing after 36 h. At low concentrations (0.1 mM), Cu showed a steady slow decline. At 48 h, Zn decreased faster to max activity and returned to control by 72 h. **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.
Reference: Riley et al. (2005, 096452) **Species:** Rat, Human **Cell Type:** RLE-6TN, NR8383 Alveolar Macrophages, A549	Fe: $FeCl_2$ Ni: $NiCl_2$ Cu: $CuCl_2$ V: VCl_2 **Particle Size:** NR	**Route:** Cell Culture (5×10^4 cells/well Alveolar Cells; 1.2×10^5 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:** 2-48 h	**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 24 h. **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 4 h. LDH release maximizes at 12 h and either remains steady or declines.
Reference: Ritz et al. (2007, 198901) **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 µm (diameter)	**Route:** Cell Culture **Dose/Concentration:** 0, 20, 50, 100 µg/mL **Time to Analysis:** 24 h	**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.

Study	Pollutant	Exposure	Effects
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 (1.5×10^4 cells/cm^2) **Dose/Concentration:** 20, 40 or 80 µg/cm^2 **Time to Analysis:** 72 h	**Cytotoxicity:** Responses were dose-dependent; there was no observed site interaction. Cytotoxicity seems to be a result 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 higher TNF-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.
Reference: Sakamoto et al. (2007, 096282) **Species:** Human **Age:** 58-82 yr (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 60 min; cytokines: 6 or 24 h	**Intracellular [Ca2+]:** [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 [Ca2+]:** 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 Ca2+ 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 Ca2+ . 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:** 1 hAEo-	$FeSO_4$ $FeCl_3$ $NiSO_4$ **Particle Size:** NR	**Route:** Cell Culture **Dose/Concentration:** 0.25 and 0.5 mM Fe exposures also contained 60 µg/mL apotransferrin **Time to Analysis:** 24 h	**Cytotoxicity:** Both Fe had no effect. $NiSO_4$ caused marginal cytotoxicity (75%). **Hypoxic Stress:** At 20 h, $NiSO_4$ (at concentrations of 0.25 or 0.5 mM) 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 48 h. At 48 h, 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.

Study	Pollutant	Exposure	Effects
Reference: Salonen et al. (2004, 187053) **Species:** Mouse **Cell Type:** RAW 264.7	PM_{10} (urban traffic) Finland Pooled as winter (W), spring I (SI), or spring II (SII) based on component/time considerations **Particle Size:** PM_{10}: 0.12-10 µm	**Route:** (2×10^6 cells/well) **Dose/Concentration:** 15, 50, 150, 500, 1000 µg/mL **Time to Analysis:** 0, 24 h	Air quality parameters: Winter and spring I did not differ. SII much lower $PM_{2.5}$ Metal data equivocal as well as highly variable resuspension rates. Total PAHs: W=303; SI=233; SII=204 ng/mg **Inflammation (IL-6, TNF-α, NO)/Cytotoxicity:** A dose-dependent increase was observed for TNF-α, IL-6 and NO except for SI. The IL-6 levels, of those particles exposed to SI, decreased at 1000 µg/mL. **TNF-α, IL-6:** SI = SII>>W>control. **NO production:** W≥SI≥SII **Cell Viability:** W=SI=SII toxic at 500 and 1000 µg/mL **Water-soluble vs Insoluble:** TNF-α 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-α or cytotoxicity **LPS inhibitor:** Treatment with the LPS inhibitor eliminated the IL-6 response and, perhaps, slightly reduced the TNF-α response but not cytotoxicity **Hydroxyl radicals:** A dose-dependent induction of hydroxyl radicals and induction of hydroxyl radical lesions (at 500 and 1000 $\mu g/m^3$) in the calf thymus DNA were observed.
Reference: Samet et al. (2003, 113782) **Species:** Human **Cell Type:** A431 (Epidermoid Cells)	As: $NaASO_3$ V: $VOSO_4$ Zn: $ZnSO_4$ **Particle Size:** NR	**Route:** Cell Culture **Dose/Concentration:** 500µM **Time to Analysis:** 20, 30 or 90 min	**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: Collected adjacent to moderate traffic in Rome, Italy **Particle Size:** $PM_{2.5}$	**Route:** Cell Culture (2.5×10^5 cells/mL) **Dose/Concentration:** 0.01, 0.1, 1.0 µg/mL **Time to Analysis:** 24 h	**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.

Study	Pollutant	Exposure	Effects
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 (2.5×10^4 cells/mL) **Dose/Concentration:** DEP, CO 5, 10, 15, 20, 25 µg/mL IFN-y: 10 ng/mL LPS: 1 mg/mL **Time to Analysis:** 1-3 days	**Cytotoxicity:** No cytotoxic effects were observed. **NO:** DEP alone induced NO in a dose-dependent manner which peaked after 1 day and plateaued for days 2 and 3. IFN-y + DEP showed dose- and time-dependency. LPS + DEP showed no effect at 1 day, but dose-dependently reduced NO production on days 2 and 3. Addition of Bacillus Calmette-Guerin (BCG) eliminated the effect of DEP at 2 days but showed a dose-dependent decrease at 3 days. **Effectiveness of Particulate Components:** The carbonaceous core of DEP did not affect BC G-stimulated NO production. CO significantly inhibited BCG-stimulated NO production. Study indicated that the extract of aromatic hydrocarbons and resins caused an inhibitory effect in a dose-dependent manner.
Reference: Seagrave et al. (2007, 097549) **Species:** Human **Gender:** Male (3 donors) **Age:** 16, 23 yr **Cell Type:** 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:** 3 h exposure; 1 or 21 h 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 21 h. **LDH/Cytotoxicity:** DE had a highly variable(donor specific) effect at 1 h and returned to control levels at 21 h **Mitochondrial activity (WST):** DE reduced activity at 1 h and possibly increased activity at 21 h (high donor-to-donor variability) **Mucus Like 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 21 h **Glutathione:** DE caused a large decrease at 1 h but returned to normal at 21 h. **HO-1:** After DE exposure, levels increased but were still lower than air exposed controls **Cytokines:** No differences for IL- 8 or 12, TNF-α, GM-GSF, IL-1α, or IFN-γ 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: 37 nm; Stokes diameter 198 nm	**Route:** Cell Culture (1×10^5 cells/well) **Dose/Concentration:** 0.03 -1,000 $\mu g/cm^2$ **Time to Analysis:** 0, 18 h	**IL-8 release:** DPM increased semi dose-dependently (perhaps steady based on error range) up to 1 $\mu g/cm^2$ after which IL-8 declined dose dependently to zero (control = 100%) at 300 and 1000 $\mu g/cm^2$. LDH release was steady which indicates no cytotoxicity. **DPM interaction with IL-8:** DPM depletes IL-8 from solution in a dose-dependent manner (cell free). 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/Crl BR (mouse) **Age:** 11 wk (mouse) **Weight:** 250 g **Cell Type:** A549, AMs	PM filter collection Collected from diesel or gasoline powered vehicles as follows: BG: BS 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 (1×10^5 cells/well) **Dose/Concentration:** 0.03-10,000 µg/cm^2 **Time to Analysis:** 16-18 h	**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, 198904) **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) **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: 24 h; IL-8: 8 h; Generation of hydroxyl radicals: 8 h	**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 ~17% release of LDH, Hampstead triggered ~ 13% and Oxford Circus ~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. **Hydroxyl 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

Study	Pollutant	Exposure	Effects
Reference: Singal and Finkelstein (2005, 198905) **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: 12 nm surface area ~200 ± 25 m^2/g; CI: ~40 nm	**Route:** Cell Culture (5×10^5 cells/well) **Dose/Concentration:** 18 µg/mL, 36 µg/mL, 72 µg/mL all in 1 mL /well **Time to Analysis:** 24 h	**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 h. **Aerosil 200:** AE2 induced dose- and time-dependent Luc response which peaked at 3 h and decreased thereafter in a similar way as TNF-α. Contrary to TNF-α, AE2 induced much cytotoxicity starting at 6 h. **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 AE2 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 (5×10^5 cells seeded on a 24-well plate) **Dose/Concentration:** 50 µg/mL **Time to Analysis:** 72 h	**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 Type:** AM (rat), Type 2 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:** 20 h	Crustal material (metals and endotoxin but not Ti, As, Cd, Zn, V, Ni, Se) were positively associated with AM IL-6 and TNF-α and Type 2 MIP-2 and IL-6. Sea spray (Na and Cl) was also correlated with AM IL-6.
Reference: Tal et al. (2006, 108588) **Species:** Human **Cell Type:** HAEC	100 mM Zn(II) or V(IV) stock solutions **Particle Size:** NR	**Route:** Cell Culture **Dose/Concentration:** 500 µmol **Time to Analysis:** 5, 20 min	**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 ± 16 nm (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 72 h	**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 after which 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.

Study	Pollutant	Exposure	Effects
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 SiO_2 ROFA **Particle Size:** TiO_2: ~1 µm; SiO_2: ~1 µm; ROFA: NR	**Route:** Cell Culture (1×10^5 cells AM 1.4×10^5 cells RLE/RFL) **Dose/Concentration:** 1-50 µg/mL **Time to Analysis:** 24 h	**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_2O_3 M-Al: Micro Al_2O_3 N-Ce: nano CeO_2 M-Ce: micro CeO_2 N-Fe: nano Fe_2O_3 M-Fe: micro Fe_2O_3 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) **Particle Size:** (Surface mean diameter) N-Al: 6 nm (261 m^2/g) M-Al: 210 nm (7.7 m^2/g) N-Ce: 14 nm (71 m^2/g) M-Ce: 1500 nm (0.6 m^2/g) N-Fe: 5 nm (221 m^2/g) M-Fe: 100 nm (12 m^2/g) N-Ni: 6 nm (145 m^2/g) M-Ni: 16 nm (57 m^2/g) N-Si: 19 nm (127 m^2/g) M-Si: 440 nm (5.4 m^2/g) N-Ti: 6 nm (242 m^2/g) M-Ti: 410 nm (3.5 m^2/g) KLN: 100 nm (24.3 m^2/g) MUS: (NOS <5 µm) DD: 400 nm (6.2 m^2/g) JE: (NOS <3 µm) MNC: 200 nm (13.0 m^2/g)	**Route:** Cell Culture (35,000 cells/cm^2 BEAS; 2500 cells/cm^2 NHBE; 20,000 cells/cm^2 A549) **Dose/Concentration:** 0.53, 5.3 and 53 µg/cm^2 (= 1, 10, 100 µg/mL) **Time to Analysis:** 24 h	**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 concentration 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 BSr 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 µg/cm^2 V = 100 µg/cm^2 C = 100 µg/cm^2 D = 32 µg/cm^2 L = 1000 EU/mL T = 100 µg/cm^2 K = 100 µg/cm^2 **Time to Analysis:** 24 h	**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. **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) **Dose/Concentration:** 10, 20, 40, 80 µg/cm^2 **Time to Analysis:** 24 h	**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 µg/cm^2:** Correlation was medial for OC-1(OC) and P at $0.001<p<0.01$. **IL-6, 80 µg/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 µg/cm^2:** Correlation was weak for EU (Endotoxin), CO_3, Si, and Br. **IL-8, 80 µg/cm^2:** Correlation was medial for CO_3, 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	$PM_{2.5}$ 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:** 0.4-3 µm	**Route:** Cell Culture (20,000/cm^2) **Dose/Concentration:** 10, 20, 40, 80, 160 µg/cm^2 **Time to Analysis:** 24 h	**Elemental Analysis of PM**: 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, 550° F) 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. power plant -OFA: Oil fly ash from Niagara, NY - A: Total Fractions - B: Soluble Fractions - C: Washed Fractions **Particle Size:** PM >2.5 µm; PM: 2-10 µm; PM >10 µm	**Route:** Cell Culture **Dose/Concentration:** 50 µg/mL; 30 µg/cm^2 100µg/mL; 60 µg/cm^2 **Time to Analysis:** 4, 16 h	**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 16 h. Overall, fraction -A was the most potent. **Surface charge:** Surface charge correlated strongly with increases in both Ca^{2+} 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: 95 nm	**Route:** Cell Culture (2×10^5 - 2×10^6 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:** 24 h	**Effect On mRNA Expression (COX-2, TNF-α, 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, TNF-α, 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 (1×10^5 cells/100 mm dish; 3.2×10^4 cells/cm^2) **Dose/Concentration:** 400 µm **Time to Analysis:** 0.5, 1, 4, 24 h	**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 24 h 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:** 5 h	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) $FeCl_3$ $ZnCl_2$ **Particle Size:** CB: 14 nm	**Route:** Cell Culture (4×10^5 cells/mL at 1mL/well) **Dose/Concentration:** CB 1.9 -31 µg/mL; $FeCl_3$, $ZnCl_2$ 0.01-100 µmol **Time to Analysis:** 4 h	**ROS Production in Cells:** CB alone increased ROS. Coexposure with $ZnCl_2$ did not affect ROS. **ROS Production - Cell Free:** CB induced a significant increase in ROS. $ZnCl_2$ had no effect. Coexposure CB/Zn also had no effect. **TNF-α 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_2O_3 Si60: silicasol (SiO_2, amorphous silica) Si100: silicasol Q: crystalline quartz DQ12 **Particle Size:** Fe: 50-90 nm; Si60: 60 nm; Si100: 80-110 nm; Q <5 µm	**Route:** Cell Culture (2×10^4 cells/well. Co-culture: 2×10^4 A549 and 2×10^3 Macrophages) **Dose/Concentration:** A549 light microscopy hematite 100µg/mL (23 µg/cm^2) TEM hematite 50 µg/mL (16 µg/cm^2) Cytotoxicity 10, 50, 100 and 200 µg/mL (6.1, 30, 61 and 121 µg/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: 100 ng/mL **Time to Analysis:** 20 min	**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 et al. (2003, 199749) **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:** 0-8 h	**Cytotoxicity:** Exposure to 50 µmol Zn^{2+} for 8 h did not result in significant alterations in cell viability. **PTEN Protein Levels:** 50 µmol Zn^{2+} for 4 and 8 h significantly decreased levels in a dose-dependent manner. Exposure to 50 µM vanadyl sulfate (tyrosine phosphatase inhibitor) had minimal effects on PTEN. 100 ng/mL of non-specified EGF receptor ligand for 1-8 h 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 Zn^{2+} for 8 h whereas PTEN protein levels declined as early as 4 h. **Proteasome-mediated PTEN Degradation:** Use of MG132 (proteasome inhibitor) had no significant effect on Zn^{2+} 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 Zn^{2+} 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:** 2 h	**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 Zn^{2+} 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 Zn^{2+} for 1-4 h induced phosphorylation of EGFR in NHBE cells. EGFR kinase inhibitor PD153035 (to determine if phosphorylation of EGFR was the result of autophosphorylation of activated EGFR tyrosine kinase activity) caused Zn^{2+} -induced phosphorylation to subside. Zn^{2+} activity requires tyrosine kinase activity. **EGFR Phosphorylation Pathway:** To test whether Zn^{2+} exposure results in ligand release, which in turn can activate phosphorylation, NHBE cells were pretreated with LA1 blocking antibody. Results showed significant suppression of Zn^{2+} induced phosphorylation, therefore Zn^{2+} 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 Zn^{2+} 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 **Time to Analysis:** 4 or 8 h; EGFR phosphorylation: 30, 60, 120, 240 min	**Cell Viability:** Exposure to 50 µmol Zn^{2+} 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 Zn^{2+} induced expression, LY-294002 (a P13 inhibitor) and another unnamed P13 inhibitor were used. Exposed cells indicated suppressed levels of Zn^{2+} induced COX-2. To determine Akt role, ad-DN-Akt (AAA) was used. Infected cells indicated over-expression of Akt and significant reduction of Zn^{2+} induced GSK-3α/β phosphorylation. Over expression of DN-Akt(AAA) blocked Zn^{2+} 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 Zn^{2+}-induced mRNA COX-2 expression, suggesting PTEN inhibits PIP3 signal transduction to Akt. **Analysis of the Src/EGFR Signaling Pathway:** Zn^{2+} induced a time-dependent increase in Src and EGFR phosphorylation in cells. Blockage of Src activity via PP2 (Src inhibitor) decreased Zn^{2+} induced EGFR phosphorylation. The EGFR tyrosine inhibitor completely blocked Zn^{2+}-induced EGFR phosphorylation. EGF (a ligand of EGFR signaling) induced COX-2 expression, suggesting that EGFR regulated Zn^{2+} -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 Zn^{2+}-induced Akt phosphorylation of Src., EGFR and p38. It is thought that p38 is a critical kinase in regulation of Zn^{2+} -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 % OC) **Particle Size:** PNP20: 20 nm; PNP100: 100 µm; SWCNT: 0.8-1.2 nm (diameter); SWCNT: 100-1000 nm; QD: 30 nm; UAPS: <150 nm	**Route:** Cell Culture (1.2×10^6 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 1440 min. Intermediate measurements at 15, 30, 60, 120, 240 and 1440 min.	**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 Leq (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 4 h 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 1 h (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.

Study	Pollutant	Exposure	Effects
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 (3×10^4 cells/well) **Dose/Concentration:** 1, 10, 100, 250, 500 and 1000 µg/mL; main testing 250 µg/mL **Time to Analysis:** 12 h	**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.
Reference: Zhang et al. (2007, 156179) **Species:** Human, Rat **Cell Type:** A549, RLE-6TN	$PM_{2.5}$: 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 µg/cm^2 **Time to Analysis:** 24 h	**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:** 30-360 min	**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.

Table D-3. Respiratory effects: in vivo studies.

Reference	Pollutant	Exposure	Effects
Reference: Adamson et al. (2003, 087943) **Species:** Rat **Gender:** Male **Strain:** SD **Weight:** 150 g	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:** 4 h, 1 day, 3 day, 7 days, 14 days	**BALF Cells:** The greatest increase in cell numbers was observed with EHC-93W. Activity peaked at 1 day with a return to normal levels by 7 days. 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 1 day and 4 h 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:** 6 wk **Weight:** 19-24 g	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) 1 h prior **Time to Analysis:** 5 consecutive days; 72 h 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 5 day 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 1 mg/kg DEP - 6.26 ± 0.87 cells 10 mg/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:** 8-8.5 wk	DEP: Obtained from Dr. Masaru Sagai (Amori, Japan) **Particle Size:** NR	**Route:** IT Instillation **Dose/Concentration:** Lung Damage: 0.1 mg/mouse; Survival Analysis: 0.2 mg/mouse; ESR: 0.05 mg/mouse **Time to Analysis:** 24 h	**ESR:** hTrx-1 induced 0.05 mg generation of hydroxyl radicals in the lungs (mid thorax ESR spectra) compared to control. **BAL 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 wk	UFCP: Ultra Fine Carbon Particles (electric spark generator, Model GFG 1000; Palas, Karlsruhe, Germany) Measured Component: UFCP>96% EC **Particle Size:** 49 nm	**Route:** Whole-body Inhalation **Dose/Concentration:** 380 µg/m^3 **Time to Analysis:** 4 and 24 h; 0 and 24 h 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. **BAL Inflammatory/Injury Markers:** Total protein concentration significantly increased post 24 h inhalation. Post 4 h, 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:** SD **Weight:** ~250 g	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:** < 3 µm (mean diameter)	**Route:** IT Instillation **Dose/Concentration:** 1mg/100g bw in 300 µl saline; 60 mg/kg **Time to Analysis:** 24 h; 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. **BAL 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:** 6 wk **Weight:** 29-33 g	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: 4 mg/kg; LPS: 2.5 mg/kg; DL or DOL: NR **Time to Analysis:** 24 h	**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:** 7 wk **Weight:** 22.3 ± 073 g	RER: PM_{10} Paris, France subway CB TiO_2 DEP **Particle Size:** RER: 79% < 0.5 µm; 20%: 0.5-1 µm CB: 95 nm 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 24 h	**BALF Cells:** 100 µg RER and 100 µg DEP increased total cell count and neutrophil influx after 8 h and returned to normal by 24 h. Smaller doses of RER and DEP induced no effect. CB induced no effect. **BAL 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 8 h 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: Batalha et al. (2002, 088109) **Species:** Rat **Gender:** Male **Strain:** SD **Age:** NR **Weight:** 200-250 g	CAPs (Harvard Ambient Particle Concentrator) **Particle Size:** Mean: 2.7 µm	**Route:** Whole-body Inhalation **Dose/Concentration:** Range: 73.5-733 µg/m^3 **Time to Analysis:** CAPs exposure 5 h/day, 3 days (consecutive). SO_2 exposure to induce CB 5 h/day, 5 days/wk, 6 wk. Killed 24 h 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, SO42-, EC and OC increased. Univariate analyses showed significant negative correlations between the L/W ratio and Si and SO42- in normal rats and Si and OC in CB rats. Multivariate analysis showed only Si to be significant in both groups.
Reference: Becher et al. (2007, 097125) **Species:** Mouse **Strain:** Crl/Wky (iNOS(-/-)) and C57Bl/6 **Gender:** Male **Age:** 8-14 wk **Weight:** 25 g	Suspended PM: SRM-1648 **Particle Size:** NR	**Route:** IT Instillation **Dose/Concentration:** 1.6 µg/lung; 64 mg/kg **Time to Analysis:** 20 h	**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:** SD **Weight:** 200-250 g	Douglas Fir Wood Smoke (generated by burning wood at 400˚C in crucible oven) **Particle Size:** NR	**Route:** Nose-only Inhalation **Dose/Concentration:** 25 g/mouse **Time to Analysis:** Various exposure periods (0, 5, 1 0, 15, 20 min). Parameters measured after 24 h recovery period.	**Biochemical Parameters:** Lipid peroxidation increased after 20 min of wood smoke 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	Pollutant	Exposure	Effects
Reference: Cao et al. (2007, 097491) **Species:** Rat **Strain:** SH and WKY **Age:** 12 wk	$PM_{2.5}$ (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/day for 3 days, sacrificed 24 h following last exposure	**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. **BAL 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. **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: Carter et al. (2006, 095936) **Species:** Rat, Mouse, Hamster **Gender:** Female (all) **Strain:** F-344 (rat), B6C3F1 (mouse), Syrian Golden (hamster) **Age:** 7-10 wk	CB: Printex 90 **Particle Size:** primary size: 17 nm; 1.2-1.6 µm (aerosol aerodynamic diameter)	**Route:** Whole-body Inhalation **Dose/Concentration:** 1, 7, 50 mg/m^3 **Time to Analysis:** 6 h/day for 5 days/wk for 13 wk; 1 day, 3 m, 11 m post-exposure	**Superoxide:** Levels rose in all species at 50 mg dose. Hamsters had no increase at 7 and 1 mg doses. Mice also increased at 7 mg. Rats significantly increased at all dose levels. Rats maintained elevation except for the 50 mg dose at 11 mo postexposure; it declined but was still higher than control. Mice maintained elevation at 50 mg while 7 mg returned to control levels by 3 mo postexposure. **H_2O_2:** At 50 mg, increased levels in all species, with the highest in rat, were observed. At 7 mg, increased levels in rats and mice were initially seen but levels returned to baseline by 11 mo. Hamster levels were not significant. At 1 mg, no significant changes were observed. **NO:** Induced similar reactions as H_2O_2. Rat response continued through the study while mice and hamsters returned to baseline by 11 mo postexposure. Rats produced significantly higher levels at all times than other species. **BALF Cells:** CB induced significant increases in neutrophils at 7 and 50 mg 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 similar in all three species at 50 mg, 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 1 day. **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	Pollutant	Exposure	Effects
Reference: Cassee et al. (2005, 087962) **Species:** Rat **Gender:** Male **Strain:** Wistar Kyoto and SH/NHsd **Age:** 7 wk and 8-12 wk	CAPs: $PM_{2.5}$ Netherland suburban, industrial and freeway tunnel site collections Wistar rats pre-exposed to O_3 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:** 8 h O_3 pre-exposure; 6 h CAPS exposure; 48 h post-exposure	**BALF Cells:** Wistar exhibited increased protein, albumin, NAG and decreased ALP activity and macrophage numbers. Wistar showed increased PMNs due to O_3, but was not significantly increased with additional CAPs exposure. SH showed no effect of CAPS except for the increased PMNs. **BAL 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 O_3-only rats. Both O_3-only and O_3+CAPS showed bronchial epithelium hypertrophy and perivascular influx of PMNs. **BrdU Labeling Index of Terminal Bronchiolar Epithelium:** No CAPs effects were observed.
Reference: Chang et al. (2005, 097776) **Species:** Mouse **Gender:** Male **Strain:** ICR **Age:** 5 wk **Weight:** 25-30 g	UFCB: Ultrafine Carbon Black - Printex 90 (Degussa) **Particle Size:** 14 nm	**Route:** IT Instillation **Dose/Concentration:** 200 µg/100 uµ/mouse **Time to Analysis:** Parameters measured 4, 16, 21, 42 h post single exposure	**BALF Cells:** Neutrophil number was at control level at 4 h, increased after 16 h, peaked at 21 h and returned to normal at 42 h. No effect was observed for the macrophage count. **BAL Inflammatory/Injury Markers:** UfCB increased total protein with peak at 21 h. TNF-α increased at 4 h and returned to normal at 16 h. **VEGF (Vascular Endothelial Growth Factor):** Increased at 4 h and peaked at 16 h but remained elevated at 21 and 42 h. VEGF and total protein in BALF were correlated (R_2 = 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 16 h post-IT was observed.
Reference: Chang et al. (2007, 097475) **Species:** Mouse **Gender:** Male **Strain:** ICR **Age:** 5 wk **Weight:** 25-30 g	UFCB: Ultrafine Carbon Black - Printex 90 (Degussa) **Particle Size:** 14 nm diameter	**Route:** IT Instillation **Dose/Concentration:** 200 µg/mouse; 8 mg/kg **Time to Analysis:** Pretreatment with NAC (N-acetylcysteine) ip 320 mg/kg, 2 h before UFCB IT instillation. Parameters measured 24 h 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. **BAL 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 (R_2 = 0.796) and VEGF and α2M (R_2 = 0.7331) in BAL.

Reference	Pollutant	Exposure	Effects
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:** 6-8 wk	ROFA: Obtained from Power unit 4, Boston, MA Absent of LPS **Particle Size:** NR	**Route:** IT Instillation **Dose/Concentration:** 6 mg/kg bw (150 µg in 50µl/ 25 g) **Time to Analysis:** 24 h; Additional HeJ and OuJ mice: single: 1.5, 3 and 6 h (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. **BAL 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 1.5 h. Post 6 h, TLR4 levels were greater than the control levels. OuJ expression increased beginning 1.5 h post exposure. **TLR4 Protein Level:** Protein level of OuJ mice significantly exceeded (~2-3 fold) HeJ mice at 1.5, 3 and 6 h. **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: Churg et al. (2003, 087899) **Species:** Human **Gender:** Female (Mexico City); Male, Female (Vancouver) **Age:** 66 ± 9yr (Mexico City); 76 ± 11yr (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 - >1000×10^6 g 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 >20 yr. 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:** SD **Age:** 60 day	ROFA FP&L plant #6 oil, 1% sulfur **Particle Size:** ~1.95 µm	**Route:** IT Instillation, Nose-only Inhalation (IH) **Dose/Concentration:** IT instillation = 110 µg/rat IH = 12 mg/m^3 **Time to Analysis:** IT instillation: single; IH: 6 h 24, 48, 96 h (histopathology 24 and 48 only)	**ROFA distribution:** IH and IT instillation resulted in equivocal distribution (µg/g lung tissue) in 5 different lung lobes. **Airway Hyperactivity:** IT instillation resulted in doubled airway hyperreactivity at 24 h which was sustained for 96 h. IH hyperreactivity did not reach statistically significant level. **BALF Cells:** Neutrophils peaked at 24 h and slowly declined at 48 and 96 h. **BAL Inflammatory/Injury Markers:** IH and IT instillation showed very similar responses (R_2 = 0.98). Time-dependent increases were observed for protein and LDH. **Lung Pathology:** IT instillation showed more alveolitis, bronchial inflammatory and fibrinous fluid infiltrate. IH showed relatively more congestion of small airways and alveolar hemorrhage.

Reference	Pollutant	Exposure	Effects
Reference: Courtois et al. (2008, 156369) **Species:** Rat **Gender:** Male **Strain:** Wistar Kyoto **Age:** 12-14 wk **Weight:** NR	PM (SRM 1648; 63% inOC, 4-7% OC, >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- 13 nm, P60- 21 nm; TiO_2 mean diameter: 0.14 µm	**Route:** IT Instillation **Dose/Concentration:** 5 mg PM or TiO_2 **Time to Analysis:** 6-72 h	Particles were present in lung parenchyma that was removed 12 and 72 h post-instillation.
Reference: Dick et al. (2003, 036605) **Species:** Mouse **Gender:** Female **Strain:** CD1 **Age:** 8-10 wk **Weight:** 20-25 g	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, 5.0 mg/kg **Time to Analysis:** DMTU 500 mg/kg bw 30 min pre-exposure for some mice. Parameters measured 18 h post-exposure.	**Particle Characteristics:** S increased (CO-33.20 µg/mg, FI- 49.44 µg/mg FU- 122.79 µg/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. **BAL 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: Dybdahl et al. (2004, 089013) **Species:** Mouse **Gender:** Female **Strain:** BALB/CJ or trans-genic (MutaMouse) **Age:** 9-10 wk **Weight:** ~20 g	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 90 min; II: 90 min/day for 4 days; I & II: parameters measured 1, 3, or 22 h 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 22 h post exposure. For 20 mg/m^3 DEP, a significantly higher IL-6 level was observed at 1 h post exposure but normalized at 3 h. **BALF Cells:** Inhalation of DEP did not decrease viability of BALF cells. 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:** F344, SH **Age:** 23 m (Fisher), 11-14 m (SH)	UFP: argon-filled chamber with electric arc discharge (TSI, Inc., St. Paul, MN) **Particle Size:** 36 nm	**Route:** Whole-body Inhalation. **Dose/Concentration:** UFP: 150 µg/m^3 bw; LPS: 2 mg/kg **Time to Analysis:** 6 h, 18 h	**BALF Cells:** Neither inhaled UFP nor LPS cause a significant increase in BALF 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. **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	Pollutant	Exposure	Effects
Reference: Elder et al. (2004, 087354) **Species:** Rat **Gender:** Male **Strain:** F344 **Age:** 21 mo	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: 0.95-3.13×10^5 particles/cm^3 Endotoxin: 84 EU Influenza (IV): 10, 000 EID 50 in 250 µl **Time to Analysis:** 1×6 h, 3×6 h or both. Parameters measured 18 h post-exposure. 48 h 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 6 h, 3×6 h 3: IV + PM 6 h 4: IV + PM 6 h, 3×6 h	No departures from normal baseline cellular or biochemical values were observed, suggesting that on-road exposures were well tolerated by the rats. **BAL 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 1 day. After 3 days, no statistically significant changes were noted. **Experiment 3:** Influenza virus significantly increased ROS release in BALF cells. **Experiment 4:** Influenza virus significantly increased both percentage of PMNs in BALF and BALF cell ROS release.
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, Deguss-Huels (Trostberg, Germany). LSCb: Sterling V, low surface area carbon black, Cabot (Boston, MA) **Particle Size:** HSCb = 14 nm, LSCb = 70 nm	**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/day, 5 daus/wk for 13 wk. Parameters measured 1 day, 3 mo, 11 mo 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:** SD	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: 30 nm; Cabosil: 7 nm	**Route:** IT Instillation **Dose/Concentration:** 1 mg/rat DEP; 1 mg/rat Cabosil **Time to Analysis:** Pretreatment with 0.5 unit of bleomycin; IT 3 or 7 days 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	Pollutant	Exposure	Effects
Reference: Finnerty et al. (2007, 156434) **Species:** Mouse **Gender:** Male **Strain:** C57BL/61 **Age:** 12 wk **Weight:** 24.3 ± 0.3 g	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: 200 mg/mouse; 9.1 mg/kg PM+LPS10: 200 mg PM+10 mg LPS PM+LPS100: 200 mg PM+100 mg LPS LPS: 100 µg **Time to Analysis:** 18 h	**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:** 5-6 wk	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/dayfor 4 wk. Parameters measured 1 day 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: Gerlofs-Nijland et al. (2005, 088652) **Species:** Rat **Gender:** Male **Strain:** SH/NHsd **Age:** 11-12 wk **Weight:** 250-350 g	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:** 4, 24, 48 h	**BALF Cells:** PMN significantly increased in RTD (3 and 10 mg/kg dose) and EHC-93 exposed animals at 24 h and decreased by 48 h but remained statistically significant. AM numbers decreased for 3 mg/kg RTD group at 4 h. **BAL 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 48 h, although these increases were less than EHC-93 values at the same time points. Alkaline phosphatase increased dose-dependently for RTD at 48 h. 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 48 h. A dose-dependent increase in TNF-α at 4 h 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 48 h. A dose-dependent increase in MIP-2 for all RTD dose groups were observed and remained elevated through 48 h for both PM types (although values were returning to control levels). **Pulmonary Histopathology:** A dose-dependent increase in the number of inflammatory foci at 24 and 48 h 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:** 13 wk **Weight:** 250-350 g	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, 10 mg/kg **Time to Analysis:** 24 h	**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 alveolitis. Fine PM from LYC (10 mg/kg dose) also caused some bronchiolitis. **BAL 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. **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. **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:** 12 wk **Weight:** 200-300 g	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/kg **Time to Analysis:** 24 h	Cytotoxicity (LDH, protein, albumin) and inflammation (NAG, MPO, TNF-α were increased by PM, and were greatest in the coarse PM fraction. Metal-rich PM had greater inflammatory and cytotoxic effects. PAH content influenced greater inflammation (including neutrophils), and cytotoxicity. 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:** 500 µg/rat; 2 mg/kg **Time to Analysis:** 24 h	**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. **BAL Inflammatory/Injury Markers:** Increased protein and LDH concentrations in the homozygous strain were observed when compared to control
Reference: Ghio et al. (2005, 088275) **Species:** Rat **Gender:** Male **Strain:** SD **Age:** 60 day **Weight:** 250-300 g	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, 24 h 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	Pollutant	Exposure	Effects
Reference: Gilmour et al. (2007, 096433) **Species:** Mouse **Gender:** Female **Strain:** BALB/c **Age:** 10-12 wk **Weight:** 20-22 g	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: 2.5-10 µm; FI: ≤ 2.5 µm; UF: ≤ 0.1 µm	**Route:** Oropharyngeal Aspiration **Dose/Concentration:** 25 µg or 100 µg PM; 1.25 or 5 mg/kg **Time to Analysis:** 18 h	**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. **BAL 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. **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:** 8-10 wk **Weight:** 20-25 g	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:** 25 ug or 100 µg/mouse **Time to Analysis:** 18 h	**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. **BAL 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: Gilmour et al. (2004, 087948) **Species:** Rat **Gender:** Male **Strain:** SH/NQIBR, WKY **Age:** 12 wk **Weight:** 280-340 g	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:** 24 h	**BALF Cells:** No increase in macrophage number was observed in either rat strain following saline or PM exposure at 24 h. **BAL 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. **Cytokines:** MIP-2 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-κB:** A significant increase in NF-κB 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	Pollutant	Exposure	Effects
Reference: Gilmour et al. (2004, 054175) **Species:** Rat **Gender:** Male **Strain:** Wistar Kyoto **Age:** 12 wk	ufCB: Ultrafine carbon black (Printex 90 (Degussa) CB: (Huber 990, HR. Haeffner and Co) **Particle Size:** ufCB: 14 nm; CB: 260 nm (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 48 h post-exposure.	**BALF 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:** 200-250 g	CAPs (Boston; Harvard Ambient Particle Concentrator) **Particle Size:** 0.27 ± 2.3 µm (diameter)	**Route:** Whole-body Inhalation **Dose/Concentration:** 73.5-733 µg/m^3 **Time to Analysis:** Exposed 5 h/days, 3 days (consecutive). BAL 24 h post-exposure	**BALF Cells and Inflammatory Markers:** 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, SH **Age:** 14-16 wk **Weight:** NR	DE (30-kW (40hp) 4-cylinder indirect injection Deutz diesel engine) (O_2- 20%, CO- 1.3-4.8 ppm, NO- <2.5-5.9 ppm, NO_2- <0.25-1.2 ppm, SO_2- 0.2-0.3 ppm, OC/EC- 0.3 ± 0.03) **Particle Size:** Number Median Diameter: Low- 83 ± 2 nm, High- 88.2 nm; Volume Median Diameter: Low- 207 ± 2 nm, High- 225 ± 2 nm	**Route:** Inhalation **Dose/Concentration:** Low- 507 ± 4 µg/m^3, High- 2201 ± 14 µg/m^3 **Time to Analysis:** Exposed 4 h/day, 5 days/wk, 4 wk. Necropsied 1 day post-exposure.	DE increased neutrophils in a concentration-dependent manner, and GGT activity at the high dose. Particle-laden macrophages were found in DE-exposed rats.
Reference: Gunnison and Chen (2005, 087956) **Species:** Mouse **Gender:** Male **Strain:** DK (ApoE$^{-/-}$, LDLr$^{-/-}$) **Age:** 18-20 wk	CAPS (Northeastern regional background) Ambient air copollutants measured O_3, NO_2 **Particle Size:** 389 ± 2 nm	**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:** 6 h/day, 5 days/wk for 4 mo (5/12/03-9/5/03). Sacrificed 3-4 days 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: Gurgueira et al. (2002, 036535) **Species:** Rat **Gender:** Male **Strain:** SD **Weight:** 250-300 g	CAPs (Harvard Ambient Particle Concentrator) CB (C198 Fischer Scientific, Pittsburg, PA USA) Composed of 85.9 ± 0.2% Carbon, 13.0 ± 0.2% O_2, 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 µg/m^3 **Time to Analysis:** 1, 3, 5 h CAPs Exposure followed by immediate post-exposure analysis. 5 h CB, immediate analysis. 30min ROFA, Immediate analysis.	**In situ Chemiluminescence(CL):** Data show a significant increase in lung and heart CL at 5 h. 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. **BAL 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	Pollutant	Exposure	Effects
Reference: Hamoir et al. (2003, 096664) **Species:** Rabbit **Strain:** New Zealand **Age:** 12-16 wk **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 190 nm (PSC24, PSC110, PSC190); PSA: 190 nm	**Route:** IT Instillation **Dose/Concentration:** PSC24: 0.04 or 4 mg/rabbit PSC110, PSC190, PSA190: 4 mg/rabbit **Time to Analysis:** 0, 30, 60, 90, 120 min	**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: Happo et al. (2007, 096630) **Species:** Mouse **Gender:** Male **Strain:** C57BL/6J **Weight:** 19-30 g **Age:** 10-11 wk	PMC (Coarse) PMF (Fine) PMUF (Ultrafine) Collected in 6 European cities: Duisburg, Prague, Amsterdam, Helsinki, Barcelona, Athens **Particle Size:** PMC: PM_{10}-2.5; PMF: $PM_{2.5}$-0.2; PMUF: PM0.2	**Route:** IT Instillation **Dose/Concentration:** 1, 3, 10 mg/kg Time course: 10 mg/kg **Time to Analysis:** 1. Dose-Response study: parameters measured 24 h post exposure. 2. Time course study: parameters measured 4, 12, 24 h post single exposure (at 10 mg/kg).	**BALF Cells:** 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. **BAL Injury Markers:** 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. **Cytokines:** 1. Only PMC induced dose-dependent responses that reached statistical significance at 10 mg/kg. PMF and PMUF induced minimal and inconsistent responses. 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. 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. 2. All PMC samples induced the highest levels of KC production 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:** 14-17 wk **Weight:** NR	Carbon UFP **Particle Size:** 37.6 ± 0.7 nm (diameter)	**Route:** Inhalation **Dose/Concentration:** 180 µg/m^3 **Time to Analysis:** 24 h exposure. 3 day recovery.	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:** 10-12 wk **Weight:** NR	CAPs (Detroit; July-Sept. 2000; Harvard Ambient Fine Particle Concentrator) **Particle Size:** 2.5 µm (diameter)	**Route:** Inhalation, IT Instillation. **Dose/Concentration:** 4 day concentration: 676 ± 288 µg/m^3, 5 day 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 10 h/day 1, 4, 5 days (consecutive). Another group of rats IT instilled. Both groups killed 24 h post-exposure.	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:** 8 wk **Weight:** 17-22 g	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: 100 µg/m^3 or 3 mg/m^3; SO_2<0.01 ppm; 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, 5 days/wk for 4 or 12 wk, Immediate	**BALF Cells:** Alveolar macrophages (AMs) increased dose-dependently at 30 and 90 day. 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. Lymphocytes and neutrophils increased time- and dose-dependently with a greater 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 mo (20.3%) vs. low dose group (5.3 and 7% respectively). **Cytokines:** At 30 days, TNF-α, IL-12p40, IL-4 and IL-10 mRNA increased, IL1b and iNOS decreased. IFN-γ increased in BALB/c but decreased in C57BL/c. IL-6 mRNA was not affected. At 90 day, 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//6TLR$^{+/+}$, C57BL//6TLR$^{-/-}$ **Age:** 8-9 wk	ROFA **Particle Size:** NR	**Route:** Oropharyngeal Aspiration **Dose/Concentration:** 50 µl of 1µg/mL suspension per mouse **Time to Analysis:** Parameters measured post single exposure of 6 and 24 h.	**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 Kyoto **Age:** 3 m **Weight:** 250-300 g	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 µl; 0.44-0.72 mg/kg **Time to Analysis:** 18 h	**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 TGF-ß 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 wk	DEP (derived from 4 cyl, 2.74l light duty diesel engine) **Particle Size:** NR	**Route:** IT Instillation **Dose/Concentration:** 12 mg/kg **Time to Analysis:** 24 h	**BALF Cells:** DEP induced an increase in total cells, neutrophils, and mononuclear cells. 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.

Reference	Pollutant	Exposure	Effects
Reference: Inoue et al. (2005, 097481) **Species:** Mouse **Gender:** Male **Strain:** NC/Nga **Age:** 10 wk	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 6 wk. Parameters measured 24 h 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 Kyoto **Age:** 5 wk	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, O_2 **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:** 16 h/day, 6 days/wk, for 6, 12, 18 & 24 mo. 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 Cells:** The HR group showed a significant increase in total cell count from 6 to 18 mo. The percentage of PMN increased at 6mo in M, MG and HR group. M group lymphocytes significantly increased at 6, 12, and 24 mo of exposure. Macrophages decreased at 6 mo for the M and HR groups. **BAL 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. **Mucus and Surfactant:** The HR group showed a significant increase from 12 to 18 mo.
Reference: Jones et al. (2005, 198883) **Species:** Rabbit **Strain:** New Zealand **Weight:** 2.5- 3.5 kg	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 0.5 mL saline **Time to Analysis:** Parameters measured at varying times from 6 h to 91 days post treatment.	**MCSP:** At 6 h, neutrophils increased. Macrophages increased 3 fold. At 60 h, neutrophils were pyknotic and the lungs displayed a thickened interstitium containing silica particles. At 5 days, collagen deposition appeared. At 8 days, fibroblastic activity and necrosis were observed. At 15 days, aggregation of silica particles and necrotic debris were apparent. At 8 wk, fibroblasts were still present. At 13 wk, active scarring and raised neutrophil macrophage counts were still present. **ASP:** At 15 h, neutrophils increased. Macrophages tripled and remained increased for 3wk. At 4 day, macrophages bore particles. At 13 day, neutrophils decreased significantly. By 25 day, silica spheres were gradually removed from lungs. **PET Scanning:** 18F-fluoroproline showed increased activity beginning at 14 days and peaking at 41-54 days. **Microautoradiography:** 3 h-proline at 13 wk 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:** 5 wk	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: 62.7 µg/m^3 PM, 55.7 ppb NO_2,; Control group: 14.3 µg/m^3 PM, 5.1 ppb NO_2 **Time to Analysis:** Exposed for 24, 48, 60 wk. Parameters measured immediately following exposure.	**Respiratory Tissue:** Post 24 wk, the lung surface was light gray with some BC particle deposits. Post 48-60 wk, however, the surface was scattered with particle deposits in addition to its light gray color. **Airway Changes:** After 60 wk, 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 ciliated cells post 48 wk. Clara cells were unaffected. **Lymph Nodes:** Deposition of carbon particles were noted in the trachea and bronchiole-associated lymph nodes post 24 wk. **Alveolar Changes:** No changes in morphology of broncho-alveolar junctions were noted. Anthracosis observed within alveolar walls and pleura post 24 wk and became progressively marked with increased exposure. No change in the number of alveolar holes between exposure and control groups were observed.
Reference: Kato et al. (2003, 198882) **Species:** Rat **Gender:** Male **Strain:** SD **Age:** 7 wk **Weight:** 190-220 g	Polystyrene latex suspension of latex beads (Japan Synthetic Rubber Co.), uncoated or coated with lecithin **Particle Size:** 240 nm	**Route:** IT Instillation with nebulizer **Dose/Concentration:** 5 ml of 0.2% suspension administered over 20 min at flow rate of 0.25 ml/min **Time to Analysis:** Exposed for 20 min. Parameters measured 30 min 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:** 22-24 m	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: 0.2 ppm CCL: 50 µg/m^3 EC + 70 µg/m^3 ABS + 0.2 ppm O_3 CCH: 100 µg/m^3 EC + 140 µg/m^3 ABS + 0.2 ppm O_3 **Time to Analysis:** 4 h/days, 3 consecutive days/wk for 4 wk	**BALF Cells:** CCL and CCH induced macrophage respiratory burst activity. The effect induced by O_3 was not significant. **BAL 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. **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.
Reference: Kleinman and Phalen (2006, 088596) **Species:** Rat **Gender:** Male **Strain:** SD **Age:** 6 wk **Weight:** 200 g	LO_3: Low O_3 HO_3: 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:** LO_3 = 0.30 ppm HO_3 = 0.61 ppm LS = 0.48 mg/m^3 HS = 1.00 mg/m^3 LOLS = 0.31 ppm + 0.41 mg/m^3 LOHS = 0.31 ppm +1.04 mg/m^3 HOLS = 0.60 ppm + 0.52 mg/m^3 HOHS = 0.60 ppm + 0.86 mg/m^3 **Time to Analysis:** Exposed for 4 h. Parameters measured 42 h post-exposure.	**Inflammatory Lesions in Lung Parenchyma:** Neither Type 1 or 2 lung lesions were affected by sulfuric acid alone. HO_3 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 LO_3 and HO_3). **Macrophage Phagocytosis:** All levels of exposure (no data for LO_3 and HO_3) decreased phagocytosis.

Reference	Pollutant	Exposure	Effects
Reference: Kodavanti et al. (2005, 087946) **Species:** Rat **Gender:** Male **Strain:** WKY and SH/NCrlBR **Age:** 11-14 wk	CAPs (EPA, NC) Measured components included Al, Be, Ba, Co, Cu, Zn, Pb, Mn, Ni, Ag, Ti, As. **Particle Size:** 1 day: 1.07-1.19 µm; 2 days: 1.27-1.48 µm	**Route:** Whole-body Inhalation **Dose/Concentration:** 1 day study: 1138-1765 µg/m^3 2 day study: 144-2758 µg/m^3 **Time to Analysis:** 4 hr (SH only); 4 hr/day, 2 day (WKY and SH) Post-exposure: 1 day: 3 h except study #4, 18-20 h; 2 day: 18-20 h	**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 2 day study, WKY rats showed decreases in total cells; this decrease was associated with decreased macrophages. WKY showed an increase in neutrophils. **BAL 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:** 12-14 wk	CAP-F = fine (Site I) CAP-UF = fine + ultrafine (Site II) (Netherlands) Some measured components: Ammonium, nitrate, sulfate 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 µg/m^3 CAP-UF 269-556 µg/m^3 **Time to Analysis:** 6 h/day for 2 days consecutive, 18 h	**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. **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 457µg/m^3 of CAP-F and increased at 3613 µg/m^3 of CAP-F. **Pathology.** No changes were observed.
Reference: Kumar et al. (2004, 096655) **Species:** Rat **Gender:** Male **Strain:** Wistar Kyoto **Weight:** 150 ± 20 g	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:** 4 h/day for 28 day. 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 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. **BAL Inflammatory/Injury Markers:** LDH, GGT, ALP and lavagable protein increased by 140, 450, 160 and 50%, respectively.
Reference: Lei et al. (2004, 087999) **Species:** Rat **Gender:** Male **Strain:** SD **Weight:** 318 ± 8 g	CAPs (Yaipei, Taiwan) **Particle Size:** 0.01- 2.5 µm	**Reference:** Nose-only Inhalation **Dose/Concentration:** 371 ± 208 µg/m^3 **Time to Analysis:** 6 h/day for 3 day, 5 h post-exposure pulmonary function. 2 day post-exposure for BALF collection Pulmonary hypertension induced 2 wk 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. **BAL 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:** SD **Weight:** 300-350 g	CAPs from Asian dust storm (Taiwan) Measured Components: Si, Al, S, Ca, K, Mg, Fe, As, Ni, W, V, OC, EC, SO_2, NO_2, nitrate, sulfate **Particle Size:** 0.01- 2.5 µm	**Route:** Nose-only Inhalation **Dose/Concentration:** 315.6 µg/m^3 (Low) or 684.5 µg/m^3 (High) **Time to Analysis:** Low: Exposed for 6 h. Sacrificed 36 h post-exposure High: Exposed for 4.5 h. Sacrificed 36 h post-exposure Pulmonary hypertension induced 2 wk pre-exposure.	**BALF Cells:** PM induced dose-dependent increases in total cells and percentage of neutrophils. No change in macrophages, lymphocytes or eosinophils 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:** 9 wk **Weight:** NR	DEP (2369-cc diesel engine manufactured by Isuzu Motor, operated at 1050 rpm, 80% load, commercial light oil) **Particle Size:** NR	**Route:** Inhalation **Dose/Concentration:** DEP: 103.1 ± 9.2 µg/m^3, CO: 3.5 ± 0.1 ppm, NO_2: 2.2 ± 0.3 ppm, SO_2: <0.01 ppm **Time to Analysis:** Protocol 1: Exposed 7h/day, 5 days/wk. Sacrificed at day 0, week 1, 4, 8. Protocol 2: DE alone or DE+NAC 7h/day, 1-5 days.	**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 1 wk compared to the control but returned to control levels at 8 wk. **BALF:** Compared to the other strain, the total number of cells and macrophages increased significantly at 1 wk in C57BL/6 mice and at 8 wk 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 1 wk and C57BL/6 mice at 4 and 8 wk. HO-1 protein percentage changes from the control were greater in BALB/c mice at 1 wk and C57BL/c mice at 8 wk. **NAC:** NAC inhibited the increased Penh values, total number of cells and macrophages in C57BL/6 mice at 1 wk and neutrophils and lymphocytes in both strains.
Reference: Liu et al. (2008, 156709) **Species:** Mouse **Gender:** Female **Strain:** BALB/c **Age:** 11 wk **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 **Dose/Concentration:** Average particle concentration: 1.28 mg/m^3 **Time to Analysis:** Four groups: saline+air control, saline+DEP, A. fumigatus+air, A.fumigatus+DEP. A. fumigatus exposure every 4 day for 6 doses. DEP exposure 5 h/day for 3 wk 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:** 6-8 wk **Weight:** NR	PM (high density traffic; winter 2004; São Paulo, Brazil) (NO_2, CO, SO_2) **Particle Size:** 10 µm (diameter)	**Route:** Open-Top Exposure Chamber **Dose/Concentration:** 33.86 ± 2.09 µg/m^3 **Time to Analysis:** Some rats pretreated with papain. Exposed to UAP or filtered air 24 h/day, 7 days/wk, 2 mo.	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:** 7 wk	TiO_2 (DuPont) **Particle Size:** NR	**Route:** Whole-body Inhalation **Dose/Concentration:** 10, 50 or 250 mg/m^3 **Time to Analysis:** 6 h/day, 5 days/wk, 13 wk. Parameters measured 0, 4, 13, 26, 52 wk post-exposure.	**OPN (osteopontin) Expression:** At 0 wk, 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 wk, the mid-dose and high-dose elevated OPN mRNA levels. At 13 wk, the high dose elevated OPN mRNA levels. No significant elevation with mid dose level was observed. At 26 wk, the mid and high dose induced elevated OPN mRNA levels. At 52 wk, 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 wk. At 13 wk, protein increased 9-fold (~800 pg/mL OPN) at mid dose and 100 -fold (~8000 pg/mL OPn) at high dose. At 26 wk, the mid and high dose groups remained elevated. At 52 wk, protein increased by 2.5 fold in low dose, 7-fold in mid dose and 166-fold in high dose group. **Histopathology:** At 52 wk, 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 µm	**Route:** Intranasal Installation **Dose/Concentration:** 0.17 mg/kg **Time to Analysis:** 3×day, 3 days/wk, 2 days apart (1, 4, 7 day). Parameters measured 1 h post-exposure.	**Particle Characteristics:** 3 types, ultrafines <0.2 um (inorganics ND), bunched agglomerates of ultrafines and <40 um with aluminum silicates, ions and trace metals. **BALF Cells:** Increased amount of phagocytes in alveolar area, reducing airspace percentage (control 52.9% ± 1.39, UAP-BA 24.7% ± 2.87). Increased number of PAS positive cells. **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.
Reference: Mauad et al. (2008, 156743) **Species:** Mouse **Gender:** Male, Female **Strain:** BALB/c **Age:** 10 day **Weight:** Parental: 21.4 ± 4.0 - 26.3 ± 2.8 g; 15 day-old offspring: 7.8 ± 1.1 - 9.0 ± 1.0 g; 90 day-old offspring: 20.3 ± 2.3 - 27.4 ± 1.8 g	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 ± 3.0 µg/m^3, nonfiltered chamber- 16.9 ± 8.3 µg/m^3; Outdoor concentration: PM_{10}- 36.3 ± 15.8 µg/m^3, CO- 1.7 ± 0.7ppm, NO- 89.4 ± 31.9 µg/m^3, SO_2- 8.1 ± 4.8 µg/m^3 **Time to Analysis:** Nonfiltered exposure 24 h/day for 4 mo. Mated at 120 days exposure. After birth, 30 females and offspring transferred to filtered or nonfiltered chamber. Killed 15 or 90 day of age.	Mild foci of macrophage accumulations containing black dots of carbon pigment occurred in the alveolar areas on 90 day-old mice. Surface-to-volume ratio decreased from 15 to 90 days 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:** 8-10 wk	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: 110 nm; DEE-ER: NR	**Route:** Whole-body inhalation **Dose/Concentration:** DEE PM: 236 µg/m^3 DEE-ER PM: 7 µg/m^3 **Time to Analysis:** DEE: 6 h/day for 7 days. DEE-ER: 6 h/day for 7 days. RSV administered post-exposure for some: single, 4 days. Those not infected with RSV sacrificed immediately upon last exposure.	**Differences in Exposure Conditions:** CO, PM, EC, OC, 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 Kyoto **Weight:** 228-500 g	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:** 6 h. Pre-exposure: Vagotomy (sectioning of vagus nerve) or atropine, 1 mg/kg i.p. administered 30 min prior, 2 and 4 h 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.
Reference: Medeiros et al. (2004, 096012) **Species:** Mouse **Gender:** Male **Strain:** BALB/c **Age:** 60 days **Weight:** 20-30 g	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; 0.5 mg/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, 24 h	**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:** 6-8 wk **Weight:** 20-25 g	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:** 1-7 days	**Alveolar Fluid Clearance:** At 100 µg/mouse, decreased clearance peaked at 24 h and recovered at 7 days. **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 100 µg/mouse, decreased activity of Na, K-ATPase in basolateral membranes was observed.
Reference: Nadziejko, et al. (2002, 087460) **Species:** Rat **Gender:** Male **Strain:** SH **Age:** 16 wk	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: 160 nm; UFA: 50-75 nm	**Route:** Nose-only Inhalation **Dose/Concentration:** CAPS 80, 66 µg/m^3; avg 73 µg/m^3 FA: 299, 280, 119, 203 µg/m^3; avg 225 µg/m^3 UFA: 140, 565, 416, 750 µg/m^3; avg 468 µg/m^3 **Time to Analysis:** 10 exposures of 4 h 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.
Reference: Nemmar et al. (2007, 156800) **Species:** Rat **Gender:** Male **Strain:** Wistar Kyoto **Age:** 16 wk **Weight:** 424 ± 8g	DEP: SRM 2975 **Particle Size:** <1 µm	**Route:** Intravenous Injection **Dose/Concentration:** 0.02, 0.1 or 0.5 mg/kg **Time to Analysis:** single, 24 h	**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	Pollutant	Exposure	Effects
Reference: Nemmar et al. (2003, 087931) **Species:** Hamster **Gender:** Male and Female **Weight:** 100-110 g	PS: Polystyrene particles PSC: Polystyrene particles, Carboxylate modified PSA: Polystyrene particles, Amine modified **Particle Size:** PS, PSC, PSA-60: 60 nm; PSA-400: 400 nm	**Route:** IT Instillation **Dose/Concentration:** 5, 50 or 500 µg/animal; 0.05, 0.5, 5 mg/kg **Time to Analysis:** Single, 10 min post-exposure Rose Bengal administered to induce thrombosis, immediate study thereafter	**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 500 ug 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.
Reference: Nemmar et al. (2003, 097487) **Species:** Hamster **Gender:** NR **Weight:** 100-110 g	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 24 h 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%). **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. No effect on histamine in BALF or plasma was observed.
Reference: Pereira et al. (2007, 156019) **Species:** Rat **Gender:** Male **Strain:** Wistar Kyoto **Age:** 3 m	Ambient Particles (Porto Allegre, Brazil) **Particle Size:** <10µm	**Route:** Whole-body Inhalation **Dose/Concentration:** P-6: 34, 22 or 225 µg/m^3 P-20: 139 or 112 µg/m^3 P-I: 99 µg/m^3 **Time to Analysis:** P-6: single/continuous for 6 h P-20: single/continuous for 20h P-I: intermittent (5 h) periods per day for 4 days consecutively Parameters measured 0 or 24 h post-exposure	**BAL 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:** 10 days (pups), Pregnant females- 10-14 days of gestation **Weight:** NR	PM (Fe and soot from combustion of acetylene and ethylene in a laminar diffusion flame system) **Particle Size:** Median diameter: 72-74 nm; size range: 10-50 nm	**Route:** Inhalation **Dose/Concentration:** Mean mass concentration: 243 ± 34 µg/m^3; Average Fe concentration: 96 µg/m^3 **Time to Analysis:** Exposed 10 days postnatal age, 6 h/day, 3 days (consecutive).	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:** 11-13 wk (adult male), 10-12 days (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) 40 nm; Soot (primary particles, diameter) 20-40 nm	**Route:** Whole-body Inhalation **Dose/Concentration:** Adult males: Fe- 57, 90 µg/m^3, Soot- 250 µg/m^3, Fe+Soot- Fe: 45 µg/m^3, Total PM: 250 µg/m^3; Neonates: Fe+Soot- Low: Fe- 30 µg/m^3, Total PM: 250 µg/m^3, High: Fe- 100 µg/m^3, Total PM: 250 µg/m^3 **Time to Analysis:** Adult males exposed to Fe, soot, Fe+Soot, or filtered air. Exposed 6 h/d, 3 days (consecutive). BAL, 2 h postexposure, lung tissue, 24 h postexposure. Neonatal rats exposed to Fe+Soot 10-12 day-old and 23-25 day-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	Pollutant	Exposure	Effects
Reference: Pires-Neto et al. (2006, 096734) **Species:** Mouse **Gender:** Male **Strain:** Swiss **Age:** 6 days	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 µg/m^3 Control: 18.62 µg/m^3 NO_2: 59.52 µg/m^3 Control: 37.08 µg/m^3 CB: 12.52 µg/m^3 Control: 0 µg/m^3 **Time to Analysis:** 24 h/day, 7 days/wk for 5 mo (weaned at 21 days 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:** 21-28 yr	DEP: generated from idling Volvo diesel engine DEP 300 µg/m^3 comprised of: NO_2 1.6 ppm NO 4.5 ppm CO 7.5 ppm Hydrocarbons 4.3 ppm Formaldehyde 0.26 mg/m^3 Suspended particulates 4.3×10^6/cm^3 **Particle Size:** <10 µm	**Route:** Whole-body Inhalation **Dose/Concentration:** DEP 300 µg/m^3 **Time to Analysis:** Single exposure for 1 h. Parameters measured 6 h 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: Pradhan et al. (2005, 096128) **Species:** Rat **Gender:** Female **Strain:** Wistar Albino **Weight:** 120-180 g	RSPM: Respirable Suspended PM (Lucknow, India) Quartz dust (positive control) **Particle Size:** < 5 µm	**Route:** IT Instillation **Dose/Concentration:** 2.5, 5.0, or 10.0 mg/ 0.05 ml; 20, 42, 83 mg/kg **Time to Analysis:** 15 days.	**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. **BAL 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:** NR **Age:** NR **Weight:** 330-370 g	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 **Dose/Concentration:** WS: 60 g, $PM_{2.5}$: 363 ± 23 µg/m^3, PM_{10}: 502 ± 34 µg/m^3 **Time to Analysis:** Exposed 3 h, 5 days/wk for 1, 2, 3, 4, 6, 7 mo.	WS significantly decreased body weight between 4 and 7 m exposure. The concentration of blood carboxyhemoglobin increased. Recovered BALF 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	Pollutant	Exposure	Effects
Reference: Rao et al. (2005, 095756) **Species:** Rat **Strain:** SD **Weight:** 175 g	DEP: SRM 2975 **Particle Size:** 0.5 µm	**Route:** IT Instillation **Dose/Concentration:** 5, 35, 50 mg/kg bw **Time to Analysis:** Sacrificed 1, 7, 30 days post single exposure. Cytokines measured after 24 h incubation (in vitro).	**BALF Cells:** Macrophages unaffected. Increased PMNs at 1 day for all dose levels, sustained elevation at 7days for mid and high dose and at 30 days for all dose levels. **BAL Inflammatory/Injury Markers:** Increased albumin at 1 and 30 days at all dose levels. Increased LDH except at low dose at 7 days. **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 7 days for mid and high dose and at 30 days 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 1 day for mid and high dose, returning to basal levels at 7 days. MIP-2 increased for all dose levels at all time points. NO level unaffected.
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:** 6-12 wk	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:** ~0.25 µm	**Route:** Whole-body Inhalation **Dose/Concentration:** Low: 30 µg/m^3 Mid-low: 100 µg/m^3 Mid-high: 300 µg/m^3 High: 1000 µg/m^3 **Time to Analysis:** 6 hr/day, 7 days/wk for 1wk or 6 mo. Immediate post-exposure analysis.	**Organ Weights:** Liver declined in rats of both genders at 1 wk and female rats at 6 m. Lung volume increased and lung weight decreased in female rats at 6 m. Spleen weight increased in female mice and rats at 1 wk. Thymus weight decreased in male rats at 1 wk. **Cells:** Eosinophils 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:** 12 wk	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 - 0.15 µm	**Route:** Whole-body Inhalation **Dose/Concentration:** Low: 30 µg/m^3 Mid-low: 100 µg/m^3 Mid-high: 300 µg/m^3 High: 1000 µg/m^3 **Time to Analysis:** 6 h/day, 7 days/wk for 1wk or 6 mo. Analyzed 1 day 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 6 mo. 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. **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: 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:** 150 nm (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 6 h/day, 7 days/wk, 3 days-6 mo.	**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	Pollutant	Exposure	Effects
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:** 150 nm (MMAD)	**Route:** Whole-body Inhalation **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 - exposed 6 h/days, 7 days/wk, 3 days-6 mo. C57BL/6- 1wk exposure. Instillation of P. aeruginosa. Killed 18 h postinstillation. BALB/c- Conditioned to exposure chambers and mated. Pregnant females exposed GD 1 and throughout gestation. Offspring exposures continued until 4 wk-old. Half of offspring sensitized to OVA. Tested for airway reactivity by methacholine challenge 48 h post-instillation 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 6 mo. Hypomethylation occurred in females at 1 wk. 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:** 150 nm (MMAD)	**Route:** Whole-body Inhalation **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:** 6 h/day, 7 days/wk, 3 days-6 mo.	**Organ Weight:** At 6 mo. exposure, the heart weights of male and female rats increased and male rats' seminal vesicle weight decreased. **Histopathology:** PM-containing macrophages increased by 6 mo. **Lung DNA Damage:** Hypermethylation occurred in medium- and high-exposure male rats at 6 mo. **BAL:** For both genders in the high-exposure group, LDH and MIP-2 significantly increased at 6 mo. ROS decreased at 1wk and 6 mo. Generally, the production of hydrogen peroxide and superoxide decreased in the high-exposure group and medium- and high-exposure groups, respectively. **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:** SD **Weight:** ~200 g	DEP: SRM1650 CB Elftex-12 furnace black, Cabot, Boston, MA **Particle Size:** NR	**Route:** IT Instillation **Dose/Concentration:** 5, 15, or 35 µg/kg **Time to Analysis:** Single; 1, 3, 5, 7 days post exposure	**CYP1A1:** DEP at all doses significantly increased CYP1A1 protein, was maximal at 1 day, and normalized at 5 days. CB had no effect. **CYP2B1:** DEP and CB at 15 and 35 mg/kg inhibited activity at 1 day. Protein level significantly decreased at 1 day with 5, 15 and 35 mg/kg DEP and at 15 and 35 mg/kg CB. A time dependent decrease was shown at 35 mg/kg for both DEP and CB.
Reference: Renwick et al. (2004, 056067) **Species:** Rat **Gender:** Male **Strain:** Wistar Kyoto **Weight:** 370-470 g	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: 260 nm; UCB: 14 nm; FTO: 250 nm; UTO: 29 nm	**Route:** IT Instillation **Dose/Concentration:** 125 or 500 µg/rat **Time to Analysis:** Single, 24 h	**BALF Cells:** UTO and UCB induced a large dose-dependent increase in percent neutrophils (only statistically significant at 500 µg for UTO). **BAL 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	Pollutant	Exposure	Effects
Reference: Rhoden et al. (2004, 087969) **Species:** Rat **Gender:** Male **Strain:** SD **Weight:** 250-300 g	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 5 h. Analyzed 24 h post-exposure. (CAPS-NAC = CAPS with 50 mg/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. **BALF Cells:** CAPS increased PMN 4 fold. NAS treatment reduced this increase to control levels. **BAL Inflammatory/Injury Markers:** LDH and total protein not affected. Histology confirms slight inflammation with CAPS and no inflammation with CAPs-NAC. **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.
Reference: Rhoden et al. (2008, 190475) **Species:** Rat **Gender:** Male **Strain:** SD **Age:** NR **Weight:** 300 g	Urban Air Particles (UAP) (SRM 1649) **Particle Size:** NR	**Route:** IT Instillation **Dose/Concentration:** 1mg in 100 µL saline **Time to Analysis:** Instilled with UAP. CL analysis:15 min post-exposure. BAL measurements: 4 h post-exposure. Some rats pre-treated with MnTBAP 2 h prior to UAP exposure.	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 Kyoto **Age:** 3 mo. **Weight:** 250 g	Ambient air (Sao Paulo, Brazil) **Particle Size:** <2.5 µm	**Route:** IT Instillation **Dose/Concentration:** 100 or 500 µg/rat; 0.4 or 2 mg/kg **Time to Analysis:** Single, 24 h	**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:** Lung wet/dry ratios were unaffected.
Reference: Roberts et al. (2004, 198903) **Species:** Rat **Gender:** Male **Strain:** SD **Age:** 60-90 days **Weight:** 300-350 g	ROFA: SRI (cyclone power plant) **Particle Size:** NR	**Route:** IT Instillation **Dose/Concentration:** 0.5 mg/rat; 1.67 mg/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 6 h and 80% at 24 h. NF-κB activity increased at 6 h but was not statistically significant.
Reference: Saber et al. (2005, 097865) **Species:** Mouse **Gender:** Female **Strain:** TNF(-/-) (B6, 129S-Tnftm1Gk1), C57/BL **Age:** 9-10 wk	DEP: SRM 2975 CB: Printex 90 (Degussa) **Particle Size:** DEP: 215 nm; CB: 90 nm	**Route:** Nose-only Inhalation **Dose/Concentration:** DEP: 20 mg/m^3; CB: 20 mg/m^3 **Time to Analysis:** 90 min/day for 4 days consecutively, 1 h	**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	Pollutant	Exposure	Effects
Reference: Schins et al. (2004, 054173) **Species:** Rat **Gender:** Female **Strain:** Wistar Kyoto **Weight:** 350-550 g	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, 18 h	**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. **BAL 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. **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. **Radical Formation:** Formation of hydroxyl radicals increased with exposure. Relative intensity was: PMC-D, PMF-D, PMC-B, PMF-B, and control.
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:** Single, 24 h	**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. **BAL Inflammatory/Injury Markers:** LDH showed dose-dependent increases with HE 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:** Single, 24 h	**BALF 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. **Histopathology:** All the winter and summer samples, excepting PNS, significantly induced inflammation. **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:** 10-12 wk	DE: (Two 6 cyl Cummins ISB turb0) HWS = hardwood smoke (mixed black/white oak, uncertified conventional wood stove) DE: EC = 557 µg/m^3 OC = 269 µg/m^3 NO = 45 ppm NO_2 = 4 ppm CO = 30 ppm THV = 2 ppm HWS: EC = 43 µg/m^3 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; HWS: 0.36 ± 2.1 µm	**Route:** Whole-body Inhalation **Dose/Concentration:** 30, 100, 300, 1000 µg/m^3 TPM **Time to Analysis:** 6 h/day, 7 days/wk for 6 mo. 1 day post-exposure	**Particle Characteristics:** Major differences K: HWS>>DE; Ca DE>>HWS; Zn: DE>>HWS. **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. **BAL 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 1000 µg/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.
Reference: Seagrave et al. (2008, 191990) **Species:** Rat **Gender:** Male **Strain:** SD **Age:** 10-12 wk **Weight:** 250-300 g	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, SO4, NH4, 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- 150 nm, 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-104 ppm, NO- 16.7 ppm, NO_2- 1.1 ppm, SO_2-1.0ppm, THC- 12 ppm; SDCAs: CO- <1 ppm, NO-0.19-0.62 ppm, NO_2- 0.10-0.37 ppm, SO_2- 0.07-0.24 ppm, THC- <1 ppm **Time to Analysis:** 6 h exposure, immediately post-exposure	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. GEE did not affect the amount of macrophages or PMN. SDCAs increased macrophages. The RD low dose increased macrophages and PMN. SDCAs increased Penh values and tidal volumes.
Reference: Singh et al. (2004, 087472) **Species:** Mouse **Gender:** Female **Strain:** CD-1 **Age:** 6-8 wk	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 100 µg/mouse **Time to Analysis:** single, 4 h (18 h 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 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 100 µg dose level. Similarly, DEP only increased IL-6 at the 100 µg dose level. **BAL Inflammatory/Injury Markers:** Microalbumin increased for both pollutants except DEP induced increases only at 100 µg. Endotoxin increased microal-bumin. NAG increased with 100 µg A-DEP.

Reference	Pollutant	Exposure	Effects
Reference: Smith et al. (2003, 042107) **Species:** Rat **Gender:** Male **Strain:** SD **Age:** 11-12 wk	CAPs (Fresno, CA) **Particle Size:** <2.5 µm	**Route:** Whole-body Inhalation **Dose/Concentration:** 6 exp in 2 sets of 3: Fall1 = 847 µg/m^3 Fall2 = 260 µg/m^3 Fall3 = 369 µg/m^3 Winter1 = 815 µg/m^3 Winter2 = 190 µg/m^3 Winter3 = 371 µg/m^3 **Time to Analysis:** 4 h/days 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:** SD **Age:** 8 wk **Weight:** 260-270 g	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 µg/m^3 $PM_{2.5}$ including 600 µg/m^3 PM_1 **Time to Analysis:** 4 h/days for 3 consecutive days. Parameters measured 18 or 36 h post-exposure.	**BALF Cells:** Percent and total number of neutrophils in BALF and blood increased significantly at both 18 and 36 h. 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 18 h. IL-1β increased at 36 h. **Other:** Gamma glutamyl transferase decreased at 36 h. Lung antioxidant increased at 18 h.
Reference: Song et al. (2008, 156093) **Species:** Mouse, **Gender:** Female **Strain:** BALB/c **Age:** 5-6 wk	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 Inhalation (days 6-8) **Dose/Concentration:** 0.6 mg/mL in 50 µL of saline (days 1-5), 6mg/m^3 for 1 h/day for 3 days (days 6-8). **Time to Analysis:** Enhanced Pause (Penh), measured on day 9. BAL and lung tissues collected on day 10.	**Airway Hyperresponsiveness:** Intranasal exposure plus aerosolized DEP caused a significant increase in methacholine-induced Penh over the control. **BAL 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. **Histology:** 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 **Age:** 6-8 wk	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-, SO4 **Particle Size:** PMC: 2.35-8.5 µm; PMF: 0.12-2.35 µm	**Route:** IT Instillation **Dose/Concentration:** 1 and 2.5 mg/animal **Time to Analysis:** Single, 24 h	**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 Cells:** PMNs increased. **Cytokines:** MIP-2 increased dose-dependently. TNF-α also increased. **BAL 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.

Reference	Pollutant	Exposure	Effects
Reference: Stinn et al. (2005, 088307) **Species:** Rat **Gender:** Male and Female **Strain:** Crl: (WIU BR **Age:** 40 days	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 EC. **Particle Size:** 0.19-0.21 µm (MMAD)	**Route:** Nose-only Inhalation **Dose/Concentration:** 3 and 10 mg/m^3 **Time to Analysis:** 6 h/day, 7 days/wk for 24 mo; 6 mo. post-exposure	**Body Weight:** Mean weight increased substantially during the first few weeks in all groups. Food consumption decreased in 1-24 mo but was recovered in 24-30 mo. Body weight decreased at 23 mo in all categories, but recovered except in high dose males at 30 mo. **Organ Weight:** Absolute weight of lungs, larynx and trachea increased from 0 to 12 to 24 mo 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 24 mo. EC 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 24 mo. 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, 30 mo in both genders at all dose levels. **BAL Inflammatory/Injury Markers:** LDH increased in 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 24 mo in both genders. Squamous metaplasia of respiratory epithelium increased in high dose females (12, 24 mo). **Larynx Histopathology:** No effects were observed. **Lung Histopathology:** Alveolar region hyperplasia of alveolar epithelium increased at 12, 24, 30 mo in both genders at all dose levels except for 12 mo 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 respiratory volume/bw for females.

Reference	Pollutant	Exposure	Effects
Reference: Sureshkumar et al. (2005, 088306) **Species:** Mouse **Gender:** Male **Strain:** Swiss **Age:** 10-12 wk **Weight:** 20-25 g	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:** 15 min/day 7, 14 or 21 days; ; <1 h post-exposure	**BALF Cells:** Neutrophils (%) increased at 7, 14 and 21 days (stable). Total cell count, macrophages and eosinophils were unaffected. Leukocytes and lymphocytes increased, though not significantly. **Cytokines:** GE caused time-dependent increases in TNF-α and IL-6. IL-10 and IL-1β were unaffected. **BAL Inflammatory/Injury Markers:** γ-GGT, ALP and LDH increased after 2 wk of GE exposure and stayed stable at 21 days. Total protein slightly increased on 14 and 21 days, though these increases were not statistically significant. **Histopathology:** Minor changes at 7 days, mild edema in alveolar region at 14 days and sloughing of epithelial cells in bronchiolar region and focal accumulation of inflammatory cells in alveolar region at 21 days were observed in a time-dependent manner.
Reference: Tesfaigzi et al. (2002, 025575) **Species:** Rat **Gender:** NR **Strain:** Brown-Norway **Age:** 7-8 wk **Weight:** 310-330 g	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 Inhalation **Dose/Concentration:** Target concentration (low, high exposure): 1, 10 mg/m^3; CO- 15-106.4 ppm, NO- 2.2-18.9 ppm, NO_X- 2.4-19.7 ppm, total hydrocarbon- 3.5-13.8 ppm **Time to Analysis:** 3 h/day, 5 days/wk, 4 or 12 wk.	**Respiratory Function:** 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. **BALF Cells:** 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. **Histopathology:** WS caused minimal to mild chronic inflammation in the epiglottis of the larynx. PAS-positive cells increased in the 30 day high-exposure group. AMs increased with time and concentration. Particle-laden macrophages were seen after 90 days. AB- and PAS-positive epithelial cells increased for the 90 day low exposure group.
Reference: Tin-Tin-Win-Shwe et al. (2006, 088415) **Species:** Mouse **Gender:** Male **Strain:** BALB/c **Age:** 7wk	CB14: Printex 90 (Degussa) CB90: Flammruss 101 (Degussa) **Particle Size:** CB14: 14 nm CB95: 95 nm	**Route:** IT Instillation **Dose/Concentration:** 25, 125, 625 µg/mouse; approx. 1, 5, 25 mg/kg **Time to Analysis:** 1/wk for 4wk. 4 h post-exposure	**Body Weight, Thymus, Spleen, Splenic Cell Count:** No effects were observed. **BALF Cells:** Increased total cell numbers were observed for 125, 625 µg CB14 (dose-dependent) and 625 µg 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 125 µg. Lymphocyte numbers increased at 125 and 625 µg for CB14 and 625 µg for CB95. PMN numbers increased at 125 and 625 µg for CB14 and CB95, but the response was greater with CB14. PMN numbers were proportional to dose surface area for both PM sizes. **BAL Cytokines:** CB14 and CB95 induced dose-dependent increases in IL-1β. TNF-α increased at 125 and 625 µg dose in CB14 with the 125 dose inducing a slightly greater increase. CB14 and CB95 induced CCL-3 increases 125 and 625 µg. **Chemokine mRNA in lung and lymph nodes:** CCL-3 mRNA increased for CB14 but not CB95 4 h 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:** 22-26 g	$PM_{2.5}$ (collected from stacked filter air sampler in Shanghai, China) Fe: $FeSO_4$ Zn: $ZnSO_4$ PMF: $PM_{2.5}$ + $FeSO_4$ PMFZ: $PM_{2.5}$ + $FeSO_4$ + $ZnSO_4$ Major Measured Components: Fe 26 ppm, Zn 9 ppm, S 61 ppm **Particle Size:** $PM_{2.5}$	**Route:** IT Instillation **Dose/Concentration:** PM: 25 mg/mL, 1mg/mouse Fe: 15mg/mL, 0.6 mg/mouse Zn: 15mg/mL, 0.6 mg/mouse PMF: PM 25 mg/mL + Fe 15 mg/mL, 1.6 mg/mouse PMFZ: PM 25 mg/mL + Fe 15 mg/mL, 1.6 mg/mouse **Time to Analysis:** Instilled twice at 0 and 24 h. Parameters measured 24 h following last exposure (at 48 h).	**Synchrotron X-ray imaging:** 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:** SH **Age:** 6 mo. **Weight:** NR	Ultrafine Carbon Particles (UFCP) **Particle Size:** Size- 31 ± 0.3 nm, MMAD- 46 nm, Surface area concentration- 0.139 m^2 particles/m^3, Mass specific surface area- 807 m^2/g	**Route:** Whole-body Inhalation **Dose/Concentration:** 172 µg/m^3 **Time to Analysis:** 24 h exposure. 4 days recovery. Sacrificed 1st or 3rd day of recovery.	**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.
Reference: Wallenborn et al. (2007, 156144) **Species:** Rat **Gender:** Male **Strain:** WKY, SH,, and stroke-prone SH (SHRSP) **Age:** 12-15 wk	PM: precipitator unit power plant residual oil combustion **Particle Size:** PM: 3.76 µm (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:** Single, 24 h Note: 4 h 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. **BAL 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. **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: Wallenborn et al. (2008, 191171) **Species:** Rat **Gender:** Male **Strain:** Wistar Kyoto **Age:** 13 wk **Weight:** NR	Zinc Sulfate ($ZnSO_4$, aerosolized) **Particle Size:** NR	**Route:** Nose-only Inhalation **Dose/Concentration:** 9.0 ± 2.1 µg/m^3, 35 ± 8.1 µg/m^3, 123.2 ± 29.6 µg/m^3 **Time to Analysis:** Exposed 5 h/days, 3 days/wk, 16 wk. Half of the rats used for plasma/serum analysis, other half for isolation of cardiac mitochondria.	A trend toward increased BALF protein was seen. No pulmonary-related effects were seen.

Reference	Pollutant	Exposure	Effects
Reference: Wegesser and Last (2008, 190506) **Species:** Mouse **Gender:** Male **Strain:** BALB/c **Age:** 8-10 wk	Ambient $PM_{2.5-10}$ 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, 72 h 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 6 h, 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. At 50 µg/mouse, MIP-2 concentrations increased, peaking at 3 h, though not statistically significant and returned to basal levels by 6 h. 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:** 6-8 wk **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 1 h/day,10 days. Animals receiving OVA had 20 min 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:** 75 days	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, 6 h for Whole-body plethysmographs (WBP) and repeated daily for 4-7 days, 96 or 192 h post-exposure non-WBP animals: single, 24, 96, 192 h post-exposure	**Tidal Volume:** A dose-dependent decrease in tidal volume (45 % at high dose) was sustained for 1 day with very slow recovery over 7 days. **Breathing Frequency:** Dose-dependent increase (100 % at high dose) with recovery at 7 days was observed. **Minute Ventilation:** Small dose-dependent increases were observed with a return to normal ventilation in 2 days. **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 192 h. Neutrophils increased significantly (10 fold) at 24 h in the mid and high dose groups and showed declined, but still elevated, levels at 192 h. Macrophages slowly increased in a dose-dependent manner at 192 h. **BAL Inflammatory/Injury Markers:** Protein and albumin increased at 24 h, returned to relative basal level at 192 h at the mid and high dose levels. NAG exhibited dose-dependent increases at 24 h and sustained these levels through 192 h.
Reference: Wichers et al. (2006, 103806) **Species:** Rat **Gender:** Male **Strain:** SH **Age:** 71-73 days **Weight:** 255-278 g	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, 6 h of PM Phase II: 1st day filtered air, 4 days of 6 h 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 in juvenile rats over 4 days. **Lung lobe to Body Weight Ratio:** No effects at 1 or 4 days were observed. **Deposition calculations:** V and Co were used to estimate deposition rates (good correlation between two metals at R_2 = 0.94). Total HP-12 deposition using Co was 26 and 99 µg (for 1 day and 4 day experiments) and using V was 31 and 116 µg. 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 4 days studies except for a possible decrease in frequency for the 1 day study.

Reference	Pollutant	Exposure	Effects
Reference: Witten et al. (2005, 087485) **Species:** Rat **Gender:** Female **Strain:** F344 **Age:** 8 wk **Weight:** ~175 g	DEP (heavy-duty Cummins N14 research engine operated at 75% throttle) **Particle Size:** 7.234-294.27 nm	**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 4 h/day, 5 days/wk, 3 wk. 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-exposure 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:** ~4 wk **Weight:** ~175 g	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:** 7.5-294.3 nm	**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 4 h/day, 5 days/wk, 3 wk. 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 et al. (2003, 199749) **Species:** Rat **Gender:** Male **Strain:** SD **Age:** 60 days	Zn^{2+} **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: Yamamoto et al. (2006, 096671) **Species:** Mouse **Gender:** Male **Strain:** BALB/c **Age:** 7 wk **Weight:** 23 g	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%, N0.13%, 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: 14 nm; 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 24 h	**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 24 h higher than at 4 h. 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 TLR_2 mRNA expression with 95CL and 14CL inducing higher increases than LTA. No effect on TLR4 mRNA expression was observed.

Reference	Pollutant	Exposure	Effects
Reference: Yanagisawa et al. (2003, 087487) **Species:** Mouse **Gender:** Male **Strain:** ICR **Age:** 6 wk **Weight:** 29-33 g	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. **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. **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. **mRNA Expression of TLRs:** DEP-OC, DL, DOL and LPS increased TLR_2. DEP had no effect. All particles increased TLR4 mRNA expression.
Reference: Yokohira et al. (2007, 097976) **Species:** Rat **Gender:** Male **Strain:** F344/DuCrj **Age:** 10 wk	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 µm HT: 7.8 ± 1.5 µm POF: <50 um length; <2 µm width PdO: 0.54 ± 1.11 µm CB: 28 nm	**Route:** IT Instillation **Dose/Concentration:** 4 mg/rat in 0.2 ml saline **Time to Analysis:** Single, 1 and 28 days	**Lung Weight/Body Weight Ratio:** DQ-12, HT and POF induced increases after 1 day. After 28 days, all samples induced increases in lung weight. **BALF Cells:** Neutrophils increased significantly in walls and alveolar spaces in all groups on 1 day except at HT. At 28 days, 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 28 days. PdO and CB induced edema at 28 days. Fibrosis was observed after 28 days with the most significant increase, in decreasing order, induced by DQ-12,PdO, POF, HT, CB, and the control. Histiocyte infiltration was observed after 1 day for DQ-12, POF and PdO. At 28 days, 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 28 days with DQ 12, PdO, HT, POF, CB and control. **Immunohistochemistry:** BrdU: At 1 day all 5 particles elevated in both area and number. Activity declined after 28 days but was still higher than the control. **iNOS:** At 1day DQ-12, POF and PdO induced increases. At 28 days, DQ-12 and HT induced increases. **MMP-3:** DQ-12 induced increases at both 1 and 28 days and PdO at 28 days. **Toxicity scoring:** The levels of toxicity are, in decreasing order, as follows: DQ-12, HT/PdO/POF, and CB.

Reference	Pollutant	Exposure	Effects
Reference: Zhao et al. (2006, 100996) **Species:** Rat **Strain:** SD **Age:** NR **Weight:** 200 g	DEP: SRM 2975 DEPE: SRM 1975 **Particle Size:** NR	**Route:** IT Instillation **Dose/Concentration:** 35 mg/kg **Time to Analysis:** Single, 1 day AG group coexposed 30 pre and 3, 6, 9 h 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 and DEP, 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: Zhou et al. (2003, 087940) **Species:** Rat **Gender:** Male **Strain:** SD **Age:** 10-12 wk	UFe: Ultrafine Fe particles **Particle Size:** 72 nm	**Route:** Whole-body Inhalation **Dose/Concentration:** 57 or 90 μg/m^3 **Time to Analysis:** 6 h/days for 3 days, parameters measured within 2 h post-exposure.	**BALF Cells:** No significant changes observed in total cell number, cell viability or cell differentials. **Cytokines:** Only at the high dose was an increase in IL-1β observed. No effect on TNF-α or NF-κB-DNA binding activity was observed. **BAL Inflammatory/Injury Markers:** At the high dose, total protein increased. No significant changes were observed in LDH. **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.

Table D-4. Effects related to immunity and allergy.

Study	Pollutant	Exposure	Effects
Reference: Apicella 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 **TNF-α:** 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:** 6 wk **Weight:** NR	ROFA (solid waste incinerator powered by combustible oil; São Paulo, Brazil) **Particle Size:** NR	**Route:** Intranasal Instillation **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 1-3 h after OVA challenge or saline. Pulmonary responsiveness measured day 30 then sacrificed. Lungs removed, fixed for 48 h.	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.
Reference: Archer et al. (2004, 088097) **Species:** Mouse **Strain:** BALB/c DO11.10+/+ transgenic - ova specific receptor for OVA peptide 323-339 **Age:** 4 wk	PM = SRM 1648 (NIST) TiO_2 **Particle Size:** SRM1648: avg 1.4 µm TiO_2: avg 0.3 µg (sic)	**Route:** Intranasal instillation **Dose/Concentration:** 500 µg/30 µl sterile saline, initial 0-750 µg range finding **Time to Analysis:** Ova challenge at 68 h, Methacholine aerosolization/AR at 72 h	**Airway responsiveness (WBP):** AR induced by Ova/Mch challenge was significantly and dose-dependently increased at doses of SRM1648 ≥500 µg . 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:** 8-10 wk	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, 13 ppm TVH: 0.3, 0.6, 1.3, 3.1 ppm **Time to Analysis:** Pretreatment: ip 10 µg OVA and 2 mg aluminum hydroxide post-OVA. OVA aerosol challenge on day 14, followed by 3 days of HWS. Pre-OVA received aerosol OVA challenge on day 14, then 3 days of HWS on days 26-28 and an immediate (second) OVA challenge HWS 6 h/day for 3 days. Sacrificed 18 h 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-γ 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-γ, OVA IgE, total IgE or OVA IgG2a.

Study	Pollutant	Exposure	Effects
Reference: Burchiel et al. (2005, 088090) **Species:** Mouse **Gender:** Female **Strain:** A/J **Age:** 12-14 wk	HWS (black/white oak) HWS particle Mass BC 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 **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/day for 6 mo.	**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.
Reference: Burchiel et al. (2004, 055557) **Species:** Mouse **Strain:** AJ **Age:** 10-12 wk	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 **Dose/Concentration:** 30, 100, 300, 1000 mg/m^3 diesel PM **Time to Analysis:** 6 h/day, 7 days/wk for 6 mo.	**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: Chan et al. (2006, 097468) **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:** NR	**Route:** Cell Culture **Dose/Concentration:** DEP: 10 µg/mL LPS: 5 ng/mL **Time to Analysis:** 24 h	**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- y 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- y 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:** 10-12 wk **Weight:** 17-20 g	DE: generated from a 30-kW (40 hp), 4-cylinder Deutz BF4M1008 diesel engine Influenza 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 H_3N_2: NR **Particle Size:** NR	**Route:** Inhalation; Oropharyngeal aspiration (virus) **Dose/Concentration:** DE: 529 or 2070 µg/m^3 **Time to Analysis:** 4 h/day for 5 days. Virus exposure immediately after last DE exposure. Analyzed 18 h 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:** 8-10 wk **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 Inhalation **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 7 days. OVA challenge day 14. GEE or air exposed 6 h/day on days 26-28. Immediately after exposure on day 28 challenged with OVA. Tested for MCh-induced changes 24 h post-exposure then sacrificed. Post-OVA protocol: OVA or saline sensitized 7 days. OVA challenge day 14. GEE or air exposed days 15-17. Tested for MCh-induced changes 24 h post-exposure then sacrificed.	**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 IgG2a dose-dependently increased. In OVA-sensitized mice, OVA-specific IgG1 increased in the M group. Airway hyperresponsiveness was lower in the M and HF groups. **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-γ decreased at all exposures. In OVA-sensitized mice, IL-13 dose-dependently decreased.

Study	Pollutant	Exposure	Effects
Reference: de Haar et al. (2005, 097872) **Species:** Mouse **Gender:** Female **Strain:** BALB/cANNCrl **Age:** 6-8 wk **Weight:** NR	CBP: Carbon black particles in phosphate buffered saline, 1: 10 & 1: 100 dilutions (Brunschwich Chemicals, Amsterdam, The Netherlands) OVA: Ovalbumin **Particle Size:** CBP: 30-50 nm	**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 goblet 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:** 6-8 wk **Weight:** NR	CBP: fine (F) and ultrafine (UF) carbon black particles (Ken Donaldson Group) TiO_2: fine and ultrafine OVA: Ovalbumin **Particle Size:** F CBP: 260.0 nm UF CBP: 14.0 nm F TiO_2: 250.0 nm UF TiO_2: 29.0 nm	**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 **Time to Analysis:** Days 0,1,2: Exposed to OVA or CBP+OVA. Sacrificed on day 8 & analyzed after 2 h, or continued to second group. Second group: days 25, 26, 27 given OVA challenge day 28: sacrificed , analyzed 24 h 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. IFN-y production was low, but significantly higher than OVA exposures. **Ultrafine TiO_2 Increase OVA-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 OVA:** 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. IFN-y levels were significantly increased in ultrafine CBP+OVA treated animals. F TiO_2 had low, but significant, increases in IL-4 and IFN-y 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:** 6-8 wk **Weight:** NR	Ultrafine Carbon Black (UFCB) (Brunschwich Chemicals; Amsterdam, The Netherlands) **Particle Size:** Diameter: 30-50 nm	**Route:** Intranasal Exposure **Dose/Concentration:** 20 µg/mL **Time to Analysis:** Exposed days 1, 2, 3. 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 4 day.	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: 30-50 nm	**Route:** Cell Culture **Dose/Concentration:** 25 µg/mL **Time to Analysis:** 18 h	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:** 200-225 g	DEP: SRM 2975 (NIST, Gaithersburg, MD) OVA: Ovalbumin **Particle Size:** 0.5 µm (MMAD)	**Route:** Inhalation **Dose/Concentration:** DEP: 20.6 ± 2.7 mg/m^3 OVA 40.5 ± 6.3 mg/m^3 **Time to Analysis:** 4 h/dayfor 5 days + OVA 30 min/day1 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-y 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 tw0 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:** 200-225 g	DEP: SRM 2975 Diesel Exhaust Particles (NIST) OVA: Ovalbumin **Particle Size:** 0.5 µm (MMAD)	**Route:** Nose-only Inhalation **Dose/Concentration:** DEP 22.7 ± 2.5 mg/m^3 OVA 42.3 ± 5.7 mg/m^3 **Time to Analysis:** Day 1, 8, 15: OVA exposure 30 min/day Days 24-28: DEP exposure 4 h/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 wk **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:** 170 mg/kg **Time to Analysis:** Single, 72 h	**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	UP: Urban ambient particles collected in 5 different sites (Amsterdam, Lodz, Oslo, Rome, Dutch seaside) during four-wk 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- 200 µg DEP: 50 µg OVA: 50 µg **Time to Analysis:** 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:** Type 2 cells, AM	UP: Urban ambient particles collected in 5 different sites (Amsterdam, Lodz, Oslo, Rome, Dutch seaside) during four-wk 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:** 20 h	**Inflammation:** 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 wk **Weight:** NR	DEP: SRM 2975 NIST OVA: Ovalbumin Anti-p75: Rabbit anti-mouse p75 neurotrophin 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: 20 µg MCH: 0, 16, 32, 64 mg/ml **Time to Analysis:** On day 0: ip injection of 20 µg OVA Day 14: intranasal instillation of 50 µl anti-p75 or anti-trkA, 1 h after 1st exposure challenged with OVA aerosol for 1 h 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 (Rn) and anti-trkA did not significantly alter Rn. though Rn 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, 088469) **Species:** Mouse **Gender:** Male **Strains:** C57/Bl6 **Age:** 6 wk	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 1 h post anti-p75 exposure, OVA aerosol challenge for1 h 1 h post OVA challenge: DEP exposure for 5 h 48 h 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. **BALF 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 . **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, C57BL/6 **Age:** 2-4 mo	DEP: 4JB1 type; Isuzu Automobile, Tokyo, Japan **Particle Size**: 2 µm (MMAD)	**Route:** Group1: 1 ip injection of 2 mg of DEP. Group 2: daily ip injections of 2 mg of DEP **Dose/Concentration:** 2 mg **Time to Analysis:** 2-96 h	**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-γ 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-γ and IL-10. DEP LPS-induced increase in INF-γ mRNA responses in spleen cells. DEP caused a dose related suppression of LPS stimulated INF-γ. DEP had little or no effect on the percentage of NK or NKT cells in the spleen and inhibited LPS-induced IFN-γ production by NK and NKT. DEP failed to inhibit the IFN-γ response by anti-CD3 mAb-activated NKT cells. Oxidant activity was not responsible for DEP inhibition of LPS-induced IFN-γ production.
Reference: Fujimaki and Kurokawa (2004, 096575) **Species:** Mouse **Gender:** Male **Strains:** BALB/c **Age:** 4 wk **Cell Types:** Cervical lymph-node (CLN) cells	DE ± particles: Comparison of exposure to DE including particles and exposure to particle-filtered DE DE: 12.09 ± 0.15 NO_X, 1.99 ± 0.02 NO_2, 10.02 ± 0.12 NO, 0.18 ± 0.002 SO_2 and 1769.2 ± 13.2 CO_2 (all in ppm). DE gas: 11.93 ± 0.13 NO_X, 2.93 ± 0.06 NO_2, 8.91 ± 0.09 NO, 0.11 ± 0.003 SO_2 and 1838.8 ± 15.3 CO_2 (all in ppm) **Particle Size:** 0.4 µm (MMAD)	**Route:** Whole-body Inhalation **Dose/Concentration:** Exposure to; 0, 1.0 mg/m^3 or 1.0 mg/m^3 DE gas only (0.04 mg/m^3 PM) **Time to Analysis:** Exposure for 12 h daily for 5 wk. Days 14 and 35 challenge with sugi basic protein (SBP), a cedar pollen allergen, intranasally. Evaluation is 24 and 48 h 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.

Study	Pollutant	Exposure	Effects
Reference: Fujimaki et al. (2005, 156456) **Species:** Mouse **Gender:** Male **Strains:** C57BL/6 **Age:** 4 wk	DE generated by 4 cyl 2.74 l Isuzu diesel DE gas = DE filtered to remove particles Composition of Diesel Exhaust**:** DE DEP: 1.01 mg/m^3 1796 ppm CO_2 12.09 ppm 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:** 0.4 µm (average diameter)	**Route:** Whole-body Inhalation **Dose/Concentration:** 1.0 mg DEP/m^3 or 1.0 mg DEP/m^3 DE gas **Time to Analysis:** 12 h daily, 5 wk. 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 1 day 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 levels. Cell 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 48 h. 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 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	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 **Dose/Concentration:** 0.3, 1.0 and 3.0 mg DEP/m^3 (Groups 1,2,3) **Time to Analysis:** Exposure began at 2 days postcoitum and was continued until 13 days postcoitum. Exposure time was 12 h daily for 7 days/wk. Pregnant females were sacrificed 14 days postcoitum.	**mRNA Expression in Placentas**: In groups exposed to DE, the expression of CYP1A1 mRNA decreased to undetectable levels during placental absorption and INF-γ 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) $NiSO_4$, $CuSO_4$, $VOSO_4$, Na_3VO_4 **Particle Size:** NR	**Route:** Cell Culture; seeded into 6-well plates (3-4.5×10^5 cells/3 mL/well) or 24-well plates (0.6-1×10^5 cells/1.0 mL/well) **Dose/Concentration:** PM: 3, 10, 20, 40, 50 µg/ml Metallic salts: 2, 20, 200 µM **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 sources 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:** 7wk	$PM_{2.5}$ from the German cities of Hettstedt or Zerbst PM Composition: samples from Hettstedt have several-fold higher levels of Zn, Mg, Pb, Cu and Cd than samples from Zerbst. **Particle Size:** $PM_{2.5}$	**Route:** Oropharyngeal Aspirations **Dose/Concentration:** 50-100 µg **Time to Analysis:** Single, 18 h. Sensitization Model: Mice were exposed to 50 µg PM 2 h before being sensitized with 10 µg OVA, repeated two days later. On day 14 all mice were challenged with 20 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. 14 days later mice were exposed to 100 µg $PM_{2.5}$ followed 2 h 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. **BALF Cells**: In non-allergic mice both Hettstedt and Zerbst PM increased 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 Injury Markers:** 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-γ (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:** ~12-14 wk **Weight:** 17-20 g	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: ~240 nm	**Route:** Inhalation **Dose/Concentration:** Low- 514 ± 3 µg/m^3, High-2026 ± 38 µg/m^3 **Time to Analysis:** 4 h/day, 1 or 5 days (consecutive). Necropsied immediately or 18 h postexposure.	**BAL Analysis:** Neutrophils and lung injury dose-dependently increased. ICAM-1 increased immediately after both exposures and after 18h postexposure in the low dose. **Cytokines:** After 1 day exposure, IFN-γ and TNF-α increased immediately at both doses and the high dose, respectively. Immediately after 5 days exposure TNF-α and IFN-γ increased at both concentrations and IL-6 increased at the low dose. At 18 h postexposure IL-6 and IFN-γ 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 5 days exposure, 18 h post-exposure.

Study	Pollutant	Exposure	Effects
Reference: Hamada et al. (2007, 091235) **Species:** Mouse **Gender:** Female (Pregnant close to partruition) **Strain:** BALB/c	ROFA (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 leachate **Dose/Concentration:** 50 mg/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 (1mg) at day 0 followed by exposure to: 1. aerosolized OVA days 12, 13 and 14 (2-wk old protocol) OR 2. aerosolized OVA days 32, 33 and 34 (5 wk old protocol) Analysis 48 h after final allergen exposure	**Susceptibility to Asthma**: Exposure of mother to PBS aerosols during pregnancy did not result in prominent asthma features in young. The offspring of the ROFA mothers revealed increasing AHR and elevated numbers of eosinophils in the BAL fluid. Similar results were seen in both the 2-wk and 5-wk 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-wk group. **Maternal Influence**: Breast milk was not shown to be responsible for the increased susceptibility to allergy seen in offspring. **IL-4 and IFN-γ**: IL-4 and IFN-γ 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-γ causing an increase in the ratio of IL-4/IFN-γ 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 wk	DEP (4-cylinder diesel engine under a 10-torque load) **Particle Size:** NR	**Route:** Nebulization **Dose/Concentration:** 2 mg DEP m^3 **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 day 5.	**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:** 10-12 wk **Weight:** NR	CAPs (Detroit; July-Sept. 2000; Harvard Ambient Fine Particle Concentrator) **Particle Size:** 2.5 µm (diameter)	**Route:** Inhalation; IT Instillation. **Dose/Concentration:** 4 day concentration: 676 ± 288 µg/m^3, 5 day 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:** 10 h/day 1, 4, 5 day (consecutive); F344 rats sensitized to endotoxin, BN rats to OVA. Both groups killed 24 h post-exposure.	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, BAL 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:** 8-10 wk	DEE: Diesel Engine Emissions generated from a 5.9-liter turbo diesel engine fueled by Number 2 fuel. DEE Composition: NO_X: 2.0-43.3 ppm CO: 0.94-29.0 ppm SO_2: 8.3-364.9 ppb **Particle Size:** 0.1-0.2 µm (MMAD)	**Route:** Whole-body Inhalation RSV: IT administration **Dose/Concentration:** DEE: 38.8 µg/m^3 (low level) or 10027 µg/m^3 (high level) RSV: 100 µl **Time to Analysis:** 6 h/day, 7 days After the final 6 h exposure period mice were infected with RSV. Parameters measured 4 days 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. **BALF Cells:** 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 pseudo-stratified, 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. **Cytokines:** TNF-α and IFN-γ 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-γ 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, mucous 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 and 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:** 10-12 wk **Weight:** NR	DEE (2 2000 model 5.9-1 Cummins ISB turbo diesel engines, No. 2 certification diesel fuel) **Particle Size:** NR	**Route:** Inhalation **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:** 6 h/d, 7 days/wk, 1 wk or 6 mo. 1 wk exposure repeated on separate occasion. Immediately after exposure, mice anesthetized, 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:** 10-12 wk **Weight:** NR	CAPs (Grand Rapids, MI; July) **Particle Size:** Diameter: 0.1-2.5 µm	**Route:** Whole-body Inhalation **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 3 day. Challenged with OVA or saline 2wk later for 3 day. Exposed to CAPs 8h/d, 13d. OVA or saline challenge 9 day after first challenge. Sacrificed 24 h 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:** 8 wk **Weight:** 17-22 g	DE -DE (generated by diesel engine and diluted with filtered clean air) **Particle Size:** NR	**Route:** Inhalation **Dose/Concentration:** Low -0.1 mg/m^3 High - 3 mg/m^3 **Time to Analysis:** 7 h/day, 5 days/wk, 1 or 3 mo	**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 mo, and in the low dose group at 1 mo. 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-γ mRNA expression increased in BALB/c mice and decreased in C57BL/6 mice. Similar results were seen at 3 mo, except IL-4 and IFN-γ 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-κB activation occurred after 1 wk 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:** 8 wk **Weight:** 17-22 g	DE (generated by diesel engine and diluted with filtered clean air.) Mycobacterial Infection -M.tuberculosis (ATCC35812) Kurono strain **Particle Size:** NR	**Route:** Inhalation **Dose/Concentration:** Low - 0.1 mg/m^3 High - 3 mg/m^3 Mycobaterial infection: 5 mL (nebulized) of a 106 colony-forming units (CFU) suspension **Time to Analysis:** 7 h/day, 5 day/wk, 1, 2 or 6 mo. Subset infected on last day of DE exposure. CFU evaluation 7 wk 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-γ 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, C3H/HeN **Age:** 6 wk **Weight:** NR	DE: DE generated by 3059cc 4-cylinder diesel engine Der f: Crude extract of D. farinae **Particle Size:** 0.4 µm (MMAD)	**Route:** Inhalation **Dose/Concentration:** 1. Air 2. DE only: 3.0 mg/m^3 3. Air + Der f: 1 mg Der f 4. DE 3.0 mg/m^3 + 1 mg Der f **Time to Analysis:** DE: 12 h/day, 7 days/wk, 8 wk Der f: 2 wk intervals, 6 wk Analyzed 3 days 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 interstitium 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 eosinophil infiltration in the submucosal layer. DE+Der f treated mice in all strains had a slight to moderate eosinophil 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:** 5 wk **Weight:** NR	DEP: 2740cc 4-cylinder engine D. farinae: crude extract **Particle Size:** 0.4 µm (MMAD)	**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 2 wk intervals. Mice examined 3 wk 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. (2007, 096724) **Species:** Mouse **Gender:** Male **Strain:** ICR **Age:** 6 wk **Weight:** 29-33 g	PM-OC: Urban PM, collected for 1 month during early summer, 2001 in Urawa city Saitama, Japan LPS **Particle Size:** <2.0 µm	**Route:** IT Instillation **Dose/Concentration:** Vehicle group: PBS PM-OC group: 4 mg/kg of PM-OC LPS group: 2.5 mg/kg of LPS PM-OC+LPS group: combined administration of PM-OC +LPS **Time to Analysis:** Single, 24 h	**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, 090951) **Species:** Mouse **Gender:** Male **Strain:** ICR **Age:** 6 wk **Weight:** 29-33 g	Carbon black (14 nm PrinteX 90; PrinteX 25; Degussa, Dusseldorf, Germany) **Particle Size:** 14 nm - 300 m^2/g 56 nm - 45 m^2/g	**Route:** IT Instillation **Dose/Concentration:** Vehicle group: PBS at pH7.4 LPS group: 2.5 mg/kg of LPS in vehicle Nanoparticle groups: 4 mg/kg carbon black nanoparticles (14 nm or 56 nm) in vehicle LPS + nanoparticle group: combined administration of carbon black and LPS in vehicle **Time to Analysis:** Single, 24 h	**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. **Histology**: Treatment with LPS+14 nm nanoparticles markedly enhanced neutrophil sequestration into the lung parenchyma compared to LPS alone. LPS+56 nm nanoparticles did not. **Cytokines**: IL-1B level significantly greater for both LPS+ nanoparticles groups. TNF-α was not significantly altered among the experimental groups. **Chemokines**: Challenge with 14 nm 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 nanoparticles resulted in intensive expression 8-OHdG, strongest in LPS+14 nm 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+14 nm 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:** 6 wk **Weight:** 29-33 g	DEPs [4JB-1 type light-duty, four-cylinder, 2.74 liter 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 administration of washed DEP +LPS; DEP-OC+ LPS group: combined administration of DEP-OC + LPS **Time to Analysis:** 4 h	**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 Edema:** Washed DEP + LPS group showed a synergistic enhancement of pulmonary edema and local expression of proinflammatory chemokines (MCP-1, MIP-1α, KC, IL-1B).
Reference: Inoue et al. (2006, 096720) **Species:** Mouse **Gender:** Male **Strain:** ICR **Age:** 6-7 wk **Weight:** 29-33 g	Carbon black (PrinteX 90; PrinteX 25; Degussa, Dusseldorf, Germany) **Particle Size:** 14 nm - 300 m^2/g 56 nm - 45 m^2/g	**Route:** IT instillation **Dose/Concentration:** Vehicle group: PBS Ovalbumin (OVA) group: 1mg OVA; Nanoparticle groups: 50 mg carbon black nanoparticles (14 nm or 56 nm);;OVA + nanoparticle group: combined administration of nanoparticles and OVA **Time to Analysis:** Vehicle group - weekly for 6wk OVA group - biweekly for 6 wk Nanoparticle groups - weekly for 6 wk OVA+Nanoparticle group (same protocol as OVA and Nanoparticle) studied 24 h after last administration	**Nanoparticles:** Exposure to carbon nanoparticles resulted in the lung expression of TARC, GM-CSF and MIP-1α. The levels were higher in the 14 nm group compared to the 56 nm 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, 088625) **Species:** Mouse **Gender:** Male **Strain:** ICR **Age:** 6-7wk **Weight:** 29-33 g	Carbon black (PrinteX 90; PrinteX 25; Degussa, Dusseldorf, Germany) **Particle Size:** 14 nm - 300 m^2/g 56 nm - 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 administration of nanoparticles and OVA **Time to Analysis:** Vehicle group - weekly for 6 wk OVA group - biweekly for 6 wk Nanoparticle groups - weekly for 6 wk OVA+Nanoparticle group: same protocol as OVA and Nanoparticle studied 24 h after last administration	**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. **14 nm Nanoparticles:** All these effects were more prominent when 14 nm nanoparticles were used. The 14 nm 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:** 6 wk **Weight:** 29-33 g	Whole DE (generated by 4-cylinder, 3.059l, Isuzu diesel engine, Isuzu automobile, Tokyo, Japan) LPS **Particle Size:** 110 nm (peak particle size)	**Route:** Whole-body Inhalation **Dose/Concentration:** 0.3 mgsoot/m^3 1.0 mgsoot/m^3 3.0 mg soot/m^3 LPS: 125 mg/kg **Time to Analysis:** LPS prior to 12 h exposure to exhaust	BAL fluid, total cells, neutrophils, protein and gene levels (MCP-1 and KC) decreased 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:** 6 wk **Weight:** 29-33 g **Cell Type** Splenocytes	DEPs [4JB-1 type light-duty, four-cylinder, 2.74 liter Isuzu diesel engine (Isuzu Automobile Co., Tokyo Japan)] LPS **Particle Size:** $PM_{2.5}$	**Route:** Cell Culture (Splenocytes resuspended to cell density of 1×10^6/mL and 1000 mL applied into each of 12-well plate) **Dose/Concentration:** DEP: 100 mg/mL; LPS: 1 mg/mL; LPS(1mg/mL) + DEP (1, 10 or 100 mg/mL) **Time to Analysis:** 72 h	**Cell viability:** No effect. **Mononuclear cell response:** Incubation with DEP alone inhibited basal cytokine production. LPS significantly increased protein levels of IFN-γ, 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, 198885) **Species:** Mouse **Gender:** Male **Strain:** ICR **Age:** 6-7 wk **Weight:** 20-30 g	Carbon nanoparticles (PrinteX 90, PrinteX 25; Dusseldorf, Germany) OVA **Particle Size:** CB14 = 14 nm, CB56 = 56 nm	**Route:** IT Instillation **Dose/Concentration:** 50 µg and/or 1 µg OVA in PBS **Time to Analysis:** 1×/wk for 6 wk; sacrifice 24 h 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, 096692) **Species:** Mouse **Gender:** Male **Strain:** ICR **Age:** 6-7 wk **Weight:** 29-34 g	DEP-OC collected from 4JB1 type, light duty, 4 cylinder, 2.74 liter Isuzu diesel engine, Isuzu Automobile Company, Tokyo, Japan) OVA **Particle Size:** 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 2 wk for 6 wk; DEP components or vehicle 1×/wk for 6 wk; sacrifice 24 h 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.

Study	Pollutant	Exposure	Effects
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 exhaust gas recirculation system. **Particle Size:** $PM_{2.5}$	**Route:** Cell Culture **Dose/Concentration:** 1×10^6 1,10 or 30 mg/mL **Time to Analysis:** 3 h	**ICAM-1 and LDL Receptor mRNA**: Up-regulation in a dose-dependent manner. Statistically significant at 30 mg/mL compared to control. **HO-1 and PAF 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.
Reference: Jang et al. (2005, 155313) **Species:** Mouse **Gender:** Female **Strains:** BALB/c **Age:** 5-6 wk	DEP -generated from 4JB1 type, light duty, four-cylinder diesel engine (Isuzu Automobile, Co, Tokyo, Japan) O_3 - (generated with Sander Model 50 ozonizers, Sander, Eltze Germany) OVA **Particle Size:** NR	**Route:** Whole-body Inhalation **Dose/Concentration:** DEP: 2,000 µg/µL (sic) O_3: 2 ppm (avg 1.98 ± 0.08 ppm) OVA sensitization: 10 mg **Time to Analysis:** OVA sensitization, DEP, O_3 and OVA Challenge on d21- 23 Exposed to O_3 for 3 h and DEP for 1 h AH and BAL measured 1 day after last challenge	**Airway Responsiveness**: OVA + O_3 + DEP exposure group had significantly higher methacholine-induce Penh than sham group or OVA group. **Total cells, proportion of eosinophils and neutrophils**: The OVA + O_3 + DEP group was significantly higher than OVA group and OVA+ O_3 group. **IL-4:** OVA + O_3, OVA + DEP and OVA + O_3 + DEP IL-4 level increased compare to OVA group. **IFN-γ:** Levels significantly decreased in OVA + DEP and OVA + O_3 + DEP compared to OVA + O_3.
Reference: Jaspers et al. (2005, 088115) **Species:** Human **Cell Lines:** A549 cells, primary human bronchial and nasal epithelial cells	DEas: aqueous-trapped solution of DE (emissions from Caterpillar diesel engine, model 3304) Influenza: A/Bangkok/1/79 (H3N2 serotype) **Particle Size:** NR	**Route:** Cell Culture **Dose/Concentration:** Influenza: 3×10^5 cells infected with 320 hemagglutination units (HAU) DEas**:** 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:** 2 h incubation with DEas then virus added. HA RNA levels analyzed at 0, 15, 30, 60 or 120 min post infection. IFN and MxA responses: analyzed 24 h post infection. Fluorescence: some cells treated with GSH-ET 30 min before DEas exposure. Measured 2 h post-influenza infection.	**A549 Cells Increased Susceptibility:** DEas enhances HA RNA levels in A549 cells in a dose-dependent manner. 25 µg/cm^2 significantly enhanced levels in A549 cells compared to the influenza-infected controls. Viral protein levels were increased in A549 cells. Exposure to DEas increased the number of influenza-infected epithelial cells in A549 cells. **Human Nasal and Bronchial Cells Susceptibility:** Exposure to DEas increased HA RNA levels in the nasal and bronchial cells. Statistically significant at 22 µg/cm^2 for nasal cells and approaching significance at 44 µg/cm^2 for bronchial cells. Exposure of both types to 44 µg/cm^2 enhanced viral protein levels. **Influenza Induced IFN Response in A549:** Exposure to DEas does not suppress but enhances IFN-ß 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 IFN-ß or IFN-ß-inducible genes. **Influenza Induced IFN Response in Human Nasal and Bronchial Cells:** Exposure to DEas increased IFN-ß and MxA levels. **Oxidative Stress in A549:** DEas 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 DEas 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 DEas on HA RNA levels.

Study	Pollutant	Exposure	Effects
Reference: Kaan and Hegele (2003, 095753) **Species:** Guinea pig **Gender:** Female **Strain:** Cam Hartley **Age:** 22-29 days **Weight:** 250-300 g **Cell Types:** AM	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} (0.35 µm MMAD)	**Route:** Cell Culture **Dose/Concentration:** PM_{10}: 500 µl/well (100 µg/ml MEM) RSV exposure:: 1 ml/well (6×10^6 pfu/ml MEM) Groups: PM_{10}+RSV RSV+PM_{10} RSV only PM_{10} only negative control **Time to Analysis:** PM_{10} - 60 min; RSV - 90 min Parameters measured 24 h 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-α) 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}). IL-8: PM_{10} significantly decreases RSV-induced IL-8 production and baseline. No affect on sequence of exposure. TNF-α: 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:** 8-19 wk **Weight:** NR	CAPS: fine (F) and ultrafine (UF) using VACES system; performed a 2 sites in Los Angeles, CA, one 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 **Particle Size:** UF: dp ≤ 150 nm F: dp ≤ 2.5 µm	**Route:** Whole-body Inhalation OVA sensitization: nasal instillation OVA challenge: inhalation **Dose/Concentration:** UF at 50 m: 433 µg/m^3 -UF at 150 m: 283 µg/m^3 F at 50 m 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/day, 5 days/wk for 2 wk Sensitization: On morning of each exposure 1st Challenge: week after 10 days of treatment 2nd Challenge: one week following 1st challenge Sacrificed: 24 h 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:** 6-8 wk	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: PM0.15	**Route:** Whole-body Chamber **Dose/Concentration:** 50 m - F: 394 ± 94 µg/m^3 50 m - UF: 297 ± 189 µg/m^3 150 m - F: 387 ± 68 µg/m^3 150 m - UF: 213 ± 95 µg/m^3 OVA - 50 mg in 5 mL saline **Time to Analysis:** 3, 4 h/day, 5 days/wk, 2wk 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 50 m 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 A549	ROFA V_2O_5, $VOSO_4$, SiO_2 TiO_2, $Fe_2(SO_4)_3$, $NiSO_4$, LPS **Particle Size:** 1.95 µm (MMAD)	**Route:** Cell Culture **Dose/Concentration:** ROFA: 0, 2.5, 5, 10, 15, 20 µg/cm^2 LPS: 100 ng/mL V_2O_5: 0, 0.15, 0.3, 0.61, 1.25, 2.5, 5, 10, 20 µg/cm^2 $NiSO_4$, $Fe_2(SO_4)_3$, TiO_2, SiO_2: 0, 1.23, 2.5, 5, 10, 20 µg/cm^2 $VOSO_4$: 0, 0.145, 0.29, 0.58, 1.16, 2.32 µg/cm^2 **Time to Analysis:** LPS: 0, 6, or 18 h ROFA: 0, 2,4,6 h V_2O_5: 0, 0.25, 0.5, 1, 2, 4, 6, 8h $NiSO_4$, $Fe_2(SO_4)_3$, TiO_2, SiO_2, $VOSO_4$: 6 h	**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-1β induced TAP gene expression increases with exposure time and dose. $NiSO_4$ exhibits non-significant dose dependent suppression of inducible TAP gene expression. $Fe_2(SO_4)_3$, 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-1β 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 Kyoto **Age:** 8-10 wk **Weight:** 280-350 g **Cell Types:** AM, 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 **Particle Size:** NR	**Route:** Cell Culture (1×10^6 cells/ml) **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:** 24 h 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. Organic extract-induced expression of Ia antigen in PBM was reduced by treatment with NAC. **AP Activity:** 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 µg/mL organic extract, although higher concentrations suppressed the activity of PBM. **Cytokine Production:** Organic extract treatment of PBM decrease IFN-γ production from T-cells stimulated by PBM. No significant effect on IL-4 observed. **HO-1 Protein Level:** Levels in PBM were 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:** 6 wk **Weight:** 16-20 g	PM - aerosol of soot and iron oxide OVA **Particle Size:** $PM_{0.1}$ - $PM_{2.5}$	**Route:** Inhalation OVA - Intraperitoneal Injections; Aerosol Exposure **Dose/Concentration:** PM - 235-256 µg/m^3 OVA - 10 µg/0.1 mL injection OVA aerosol - 10 mL of 10 mg/mL (1%) solution **Time to Analysis:** PM: 4 h/day, 3 days/wk; OVA: 2 ip injections days 1 and 15. Aerosol on day 28 after first ip; 60 min 3x/wk	**2 Wk PM Exposure/4 Wk 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. **4 Wk OVA Aerosol/ 2 Wk PM Treatment:** The OVA treatment had significantly more goblet cells than the PM alone group. **6 Wk 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 macrophages 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:** 9 wk **Weight:** NR	DEP (2369-cc diesel engine manufactured by Isuzu Motor, operated at 1050 rpm, 80% load, commercial light oil) **Particle Size:** NR	**Route:** Inhalation **Dose/Concentration:** DEP: 103.1 ± 9.2 µg/m^3, CO: 3.5 ± 0.1 ppm, NO_2: 2.2 ± 0.3 ppm, SO_2: <0.01 ppm **Time to Analysis:** Protocol 1: Exposed 7h/day, 5days/wk. Sacrificed at day 0, week 1, 4, 8. Protocol 2: DE alone or DE+NAC 7h/d, 1-5 days.	**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 8 wk. **BALF:** Compared to the other strain, the total number of cells and macrophages increased significantly at 1wk in C57BL/6 mice and at 8wk 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-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 8 wk. HO-1 protein percentage changes from the control were greater in BALB/c mice at 1wk and C57BL/c mice at 8 wk. **NAC:** NAC inhibited the increased Penh values, total number of cells and macrophages in C57BL/6 mice at 1 wk and neutrophils and lymphocytes in both strains.
Reference: Li et al. (2009, 190457) **Species:** Mouse **Gender:** Female **Strain:** BALB/c **Age:** 6-8 wk **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:** Fine- <2.5 µm (diameter), UF- <0.15 µm (diameter)	**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 4 h pre-instillation on days 1, 2, 4, 7, 9. All animals rested and OVA aerosol challenged 30 min 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, 190457) **Species:** Mouse **Cell Line:** 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) **Particle Size:** Fine- <2.5 µm (diameter), UF- <0.15 µm (diameter)	**Route:** Cell Culture **Dose/Concentration:** 1, 5, 8.3, 10 µg/mL **Time to Analysis:** NR	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:** 11wk **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: 1.28 mg/m^3 **Time to Analysis:** Four groups: saline+air control, saline+DEP, A. fumigatus+air, A.fumigatus+DEP. A. fumigatus exposure every 4 days for 6 doses. DEP exposure 5 h/dayfor 3 wk 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: Liu et al. (2007, 093093) **Species:** Mouse **Gender:** Female **Strain:** BALB/c **Age:** 11wk	DEP: 5500-watt single-cylinder diesel engine. **Particle Size:** NR	**Route:** Inhalation **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 4 d. DEP exposure 5 h/day 3wk concurrent with A. fumigatus.	**IgE Production:** IgE production increased with the A.fumigatus treatment 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-γ 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:** SD **Age:** NR **Weight:** 300-400 g **Cell Line:** AM	Carbon-Black Particles (93% C) DEPs (97% C) - toluene-extracted 10-fold Cr, Mn, N; 50-100 fold Al, Cd, Cu, Fe, Mg, Pb, Zn more in DEP aggregates **Particle Size:** Carbon aggregates: 0.17 ± 0.08 µm (mean diameter) Diesel Particles: 0.69 ± 0.46 µm (mean diameter) Primary particles: 0.044 ± 0.01 µm (mean diameter)	**Route:** Cell Culture (0.5×10^6 AM/well) **Dose/Concentration:** 20 µg/mL 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. Bacteria 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 control 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, 098017) **Species:** Mouse **Gender:** Female **Strains:** BALB/c **Age:** 6 wk **Weight:** 15-20 g	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 ± 0.1 ppm CO, 2.2 ± 0.3 ppm NO_2, <0.01 ppm SO_2 and 103.1 ± 9.2 µg/m^3 DEP. **Particle Size:** NR	**Route:** Whole-body Inhalation **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 wks 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 8 wk (at 7 h/day for 5 days/wk).	**Airway Hyperresponsiveness:** Exposure to DE significantly increased airway reactivity to methacholine after 1 wk in both 24 and 48 mg/mL Mch and after 4 wk in the 48 mg/mL. DE exposure caused an increase in airway sensitivity after 1 wk of exposure, 4 wk and 8 wk of exposure did not result in a significant increase. **BAL Cells:** The total cell count was increased after 1 wk of DE exposure. This increase was mostly due to an increase in eosinophils. After 1 wk 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 wk of exposure (compared to control) but also decreased at time periods after. mRNA levels of RANTES were increased at 2 and 3 wk after exposure and remained elevated at 4 wk but not significantly. The level of RANTES protein increased as the weeks went along, but increased significantly only at 8 wk. **Histopathology:** OVA sensitization caused an increase peribronchial and perivascular infiltration of inflammatory cells which peaked at 1 wk 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:** 10-12 wk	CAPs (generated from ambient air in an urban Detroit community). **Particle Size:** 0.1-2.5 µm	**Route:** Whole-body Inhalation **Dose/Concentration:** 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 OVA 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. 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 wk	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) **Particle Size:** Fine PM 0.1-2.5 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- 50 ug 3. OVA (50 µg)+ Amsterdam Coarse PM (100 µg) 4. OVA (50 µg)+ Amsterdam Fine PM (100 µg) 5. OVA (50 µg)+ Lodz Coarse PM (100 µg) 6. OVA (50 µg)+ Lodz Fine PM (100 µg) 7. 5. OVA (50 µg)+ Oslo Coarse PM (100 µg) 8. OVA (50 µg)+ Oslo Fine PM (100 µg) Analysis 5 days 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 decrease 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-γ. 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-γ levels were not significantly affected by most of the groups, but the fine fractions of PM consistently produced higher levels of IFN-γ. **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:** 6-8 wk	Polysterene Particles (PSP) **Particle Size:** 0.1 µm (diameter)	**Route:** Subcutaneous Injection into footpads. **Dose/Concentration:** 40 µg PSP (5.94×10^{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 21 days 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:** 6-7 wk **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:** 5 days after OVA injection	**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-γ. 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 parameters (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:** 150 nm (MMAD)	**Route:** Whole-body Inhalation **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- 6 h/day, 7days/wk, 3 days-6 mo. C57BL/6- 1wk exposure. Instillation of P. aeruginosa. Killed 18 h postinstillation. BALB/c- Pregnant females exposed GD 1 and throughout gestation. Offspring exposures continued until 4wk-old. Half of offspring sensitized to OVA. Tested for airway reactivity by methacholine challenge 48 h post-instillation and euthanized.	The kidney weight of female A/J mice decreased at 6 mo. and was strongly related to PM by the removal of emission PM. PM-containing macrophages increased by 6 mo. Hypomethylation occurred in females at 1 wk. 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: Roberts et al. (2007, 097623) **Species:** Rat **Gender:** Male **Strain:** SD **Age:** 10 wk **Weight:** 250-300 g	R-Total = ROFA (Residual oily fish ash) R-Soluble = Soluble fraction of ROFA R-Chelex = R-Soluble+Chelex (insoluble resin) **Particle Size:** 2.2 µm (mean diameter)	**Route:** IT Instillation **Dose/Concentration:** 10 mg/kg (2.5-3 mg) **Time to Analysis:** Pre-exposure to ROFA samples on Day 0. Inoculation with 5×10^4 L. Monocytogenes or saline on day 3. Sacrifice on days 6, 8, 10.	**Uninfected Groups:** Compared to the controls, 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, BALF cells, NK cells, PMA-stimulated and zyomason-stimulated CL compared to all other groups at various time points. NOX 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 NOX 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:** 18-30 wk **Weight:** NR	DEPs (standard) **Particle Size:** NR	**Route:** Intrapulmonary Instillation **Dose/Concentration:** 100 µg/mouse **Time to Analysis:** Pre-exposure to 2.5×10^4 bacillus Calmette-Guerin bacteria (BC G) with or without coadministration of DEP. Sacrifice 5 wk 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-γ. Except for CD8 cells, no increase in IFN-γ was seen in the BC G + DEP group.

Study	Pollutant	Exposure	Effects
Reference: Schneider et al. (2005, 088368) **Species:** Mouse **Strain:** BALB/c **Cell Line:** RAW 264.7	SRM 1648 (greater than 63% inOC; 4-7% OC; 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%) TiO_2 **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:** 0, 7.8, 15.6, 31.2, and 62.5 µg/cm^2 **Time to Analysis:** 1, 3, 6, and 12 h	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 PGE2 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:** 21-39 yr treatment group; 23-32 yr control group **Tissue Type:** Whole blood samples	PM - organic extracts of airborne sample AERex1d - urban aerosol 1 day sample (total air volume - 1270m^3) AERex5d - 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 10 min. Incubated with rBet v 1 alone or with AERex for 20 min. Ice bath 5 min. Incubated with antibody reagent 20 min.	Nine organic compound classes were identified in AERex1d and AERex5d, with AERex1d having 20 times more PAHs. Basophil activation increased in all treatment groups up to 90%, with AERex1d being the most pronounced. 5-50 fold lower concentrations of AERex1d 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.
Reference: Shwe et al. (2005, 111553) **Species:** Mouse **Gender:** Male **Strains:** BALB/c **Age:** 8 wk **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 = 14 nm (primary particle size); CB95 = 95 nm (primary particle size)	**Route:** IT instillation **Dose/Concentration:** 25, 125, or 625 µg in 1 mL saline solution **Time to Analysis:** 1/wk for 4 wk; 4 or 24 h after last instillation	**BALF 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 625 µg 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 TNF-α 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 125 µg, 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:** 8-10 wk **Weight:** NR	CAPs: Concentrated Ambient Particles (Collected from ambient Boston air on Teflon filters.) TiO_2 IFN-γ S. pneumoniae (ATCC 6303, American Type Culture Collection, Manassas, VA) **Particle Size:** CAPs: <2.5 µm	**Route:** IFN-γ priming: aerosol Particle exposure and infection: Intranasal Instillation **Dose/Concentration:** CAPS or TiO_2: 50 µg/50 µL PBS S. pneumoniae: 105CFU/25 µl saline **Time to Analysis:** Primed for 15 min One time particle exposure 3 h post priming with lung RNA analyzed 3, 6, 24 h after exposure Sacrificed 24 h after exposure or one time 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-γ 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-γ 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 IFN-y 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-y 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. IFN-y+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 IFN-y+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. IFN-y+CAPs showed a 2.5-fold increase in bacterial numbers. **Histopathology:** Indicated moderate pneumonia in PBS+CAPs and severe pneumonia in IFN-y+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 IFN-y+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 IFN-y+CAPs group, approximately 50% greater than controls. In PMNs, iROS increased 100% in the IFN-y+CAPs groups as compared to the controls.

Study	Pollutant	Exposure	Effects
Reference: Steerenberg et al. (2004, 087474) **Species:** Rat **Gender:** Male **Strain:** Wistar Kyoto **Age:** 6-8 wk	DEP:SRM1650a (NIST, Gaithersburg, MD EHC-93: ambient PM (Ottawa, Canada) O_3 (positive control) L. mono: Listeria monocytogenes (strain L242/73 type 4B) **Particle Size:** DEP, EHC-93: NR	**Route:** DEP/EHC-93: intranasal droplet; O_3: Whole-body inhalation **Dose/Concentration:** DEP/EHC-93: 50 µg (1.0 mg/ml) O_3: 2mg/m^3 L. mono: 0.2 or 0.3 ml (5x106 PFU/ml) *I have emailed author regarding correct dose **Time to Analysis:** DEP/EHC-93: 1/day for 7 days (-7 days to -1 days) O_3 24 h/day for 7 days (-7 days to -1 daysR All rats infected on day 0. Sacrificed on days 3, 4, or 5.	**Body weight:** Growth declined for O_3 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 O_3 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 O_3 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 O_3 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:** 6-8 wk	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 allergens. EHC-93: used as a positive control OVA: Ovalbumin **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 20 min/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 ≥ Oslo. **Histopathology:** 9/13 of the coarse PM samples and 5/13 of the fine PM samples induced an inflammatory response. **BALF 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-γ 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-γ levels. TNF-α 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 wk **Treatment:** 1. C.D2-Vil6: Nramp1S and Nramp1R deficient 2. B6.129P2: Nos2tmLau: iNOS deficient 3. BALB/clL4 (tm2Nnt): deficient in IL-4 4. BALB/c (wild type) pretreated with N-Acetylcysteine (NAC)	EHC-93 OVA **Particle Size:** NR	**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 OVA, Nramp1S evoked less of an antibody responses (IgE, IgG1 and IgG2a) compared to Nramp1RHowever 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-γ, 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-γ, 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 wk **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 OVA immunization and challenge: intranasal **Dose/Concentration:** High = 2000 µg/m^3 Low = 500 µg/m^3 **Time to Analysis:** DE exposure for 4 h/day on days 0-4. OVA immunization 40 min after DE exposure on days 0-2 Challenged on days 18 and 28. Sacrificed 4 h after last exposure of day 4 for gene set analysis or 18, 48, or 96 h after the last challenge	**IgE Antibody Production:** In the absence OVA, IgE antibodies were not detected. 18, 48 and 96 h 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 Cells:** Cell counts at 18 and 96 h after OVA treatment did not differ among treatment groups. At 48 h 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 Cytokines:** IL-6 production showed a dose-dependent and time-dependent increase, but was significantly increase in the high dose group at 96 h. 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 h 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 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 exposure): collected using a 2,300-ml Isuzu diesel engine at 1,050 rpm. Composition: Fine particles: 1 mg/m^3 CO: 10.6 ppm NO_2: 7.3 ppm SO_2: 3.3 ppm **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): 100 µg/m^3 **Time to Analysis:** Cells were exposed to varying concentrations of suspended DEP for up to 24 h. NF-κB: analyzed at 6 h after suspended DEP exposure Air exposure at 0, 2, 4, 8 or 14 h	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 25 ng/mL IL-13 and DEP depicted 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 12 h 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-κB / STAT6 Activation:** (it has been suggested that NF-κB plays a role in the trans-criptional regulation of eotaxin gene expression) Cells exposed to 1-25 µg/mL DEP for 6 h increased NF-κB. 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-κB 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-κB 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-κB dependent, but STAT6-independent, pathways.

Study	Pollutant	Exposure	Effects
Reference: Tesfaigzi et al. (2005, 156116) **Species:** Rat **Gender:** Male **Strain:** Brown-Norway **Age:** 6 wk	PM: Wood smoke generated from a conventional wood stove that has a 0.5m^3 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 EC 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 6 h/day for 70 consecutive days OVA IP injection immunization on days 2, 9 OVA aerosol exposure 2 h/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 wood smoke exposed group had a 45% lower dynamic lung compliance when compared to those exposed to the filtered air group before 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 Cells 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-γ 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:** 7 wk **Weight:** 20 g	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 fueled 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 µl of olive oil **Time to Analysis:** Single, sacrificed 3 days 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:** 24 h	**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, 188803) **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:** 10 min - 4 h	**B-Hex Mediator Release in Mast Cells:** Incubation with $SrCl_2$, $NiSO_4$, $CdCl_2$ 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 % T cells (CD3+)) Human lymphocyte cell lines -- DG75 NQO1 wild type	DEP from 4 cyl Isuzu diesel methanol extracts **Particle Size:** NR	**Route:** Cell Culture, PMBC = 1×10^6 cell DG 75 = 3×10^6 cells IgE PMBC 1×10^6/mL B-cells 0.5×10^6/mL **Dose /Concentration:** 2.5, 5, 10, 20 µg DEPX/ plate (20 µg/mL) IgE DEPX 100 ng/mL sulfurophane at 0 - 30 µmol **Time to Analysis:** 6 h mRNA; 16 h protein assay. IgE 14 days.	**Induction of NQO1 by DEPX**: In PBMCs and DG75DEPX dose-dependently induced NQO1 mRNA expression NQO1 ARE was increased NAC inhibited NQO1 gene expression dose 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:** Exposed to antioxidants 5 h. 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:** 6-8 wk **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 1 h/day, 10 days. Animals receiving OVA had 20 min 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:** 8 wk **Weight:** ~175 g	DEP (heavy-duty Cummins N14 research engine operated at 75% throttle) **Particle Size:** 7.234-294.27 nm	**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 4 h/day, 5 days/wk, 3 wk. 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-exposure 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:** ~4 wk **Weight:** ~175 g	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:** 7.5-294.3 nm	**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 4 h/day, 5 days/wk, 3 wk. 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:** 5 wk **Weight:** 25-28 g	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 200 g under a load of 10 torques. **Particle Size:** 0.4 µm (MMAD)	**Route:** IT Instillation **Dose/Concentration:** 50 µg/0.1L 1. Control- 0.1mL PBS 2. DEP-OC- 50 µg 3. Washed DEP- 50 ug 4. Whole DEP- 50 ug DEP-OC + 50 ug 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 **Time to Analysis:** All groups received OVA or PBS every 2 wk for 6 wk and the PM component or PBS once a week for 6 wk.	**BALF Cells**: DEP-OC + OVA caused a significant increase in PMN infiltration in the BALF compared to the control. Exposure to Whole DEP+ OVA caused PMN count to rise further. OVA alone DEP-OC +OVA, Washed DEP + OVA and Whole DEP + OVA all 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-γ 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:** 6-8 wk	DEP- SRM 1650 **Particle Size:** 0.5 µm (MMAD)	**Route:** IT Aspiration **Dose/Concentration:** 1, 5, or 15 mg /kg **Time to Analysis:** 3 times in 2 wk or 6 times in 4 wk.	**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 2 wk; in the 4 wk group there was a significant decrease in platelet counts in mice exposed to 15 mg/kg. **Exposure on Spleen IgM AFC:** DEP exposure for 2 wk 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 wk, the decrease in AFC was not significantly different than the control. **DEP Exposure on Spleen Cell Number/Lymphocyte Counts:** Exposure for 2 or 4 wk 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 wk 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-γ production was decreased by exposure to DEP. IL-4 production was not measured.

Study	Pollutant	Exposure	Effects
Reference: Yin et al. (2005, 088133) **Species:** Rat **Gender:** Male **Strain:** Brown-Norway (BN/CrlBR) **Age:** NR **Weight:** 200-250 g	DEP = SRM 2975 (NIST) Listeria **Particle Size:** NR	**Route:** Nose-only inhalation (DEP), IT instillation (Listeria) **Dose/Concentration:** 100,000 CFU (Listeria); 21.2 ± 2.3 mg/m^3 (DEP) **Time to Analysis:** DEP exposure for 4 h/day for 5 days; infection with Listeria 7 days post-exposure; sacrifice 3 and 7 days 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-1β secretion at day 7 and TNF-α and IL-12 at both day 3 and day 7 IL-10 production was enhanced at day 7. **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 day 7, but significantly diminished levels of IL-10, IL-2 and IFN-γ.
Reference: Yin et al. (2004, 097685) **Species:** Rat **Gender:** Male **Strain:** Brown-Norway (BN/CrlBR) **Age:** NR **Weight:** 200-250 g	DEP = SRM 2975 (NIST) Listeria **Particle Size:** NR	**Route:** Inhalation (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/day for 5 days; inoculation with bacteria 2 h postexposure; sacrifice 3, 7, 10 days postinfection	**Lung Deposit:** Estimated mean lung deposit of DEP = 389 ± 25 µg/rat **Pulmonary Responses and Bacterial Clearance:** DEP significantly augmented Listeria-induced PMN infiltration, lung CFU and recoverable AM at all times post-infection. LDH activity was increased 3 days post-infection. Bacterial count in DEP exposed rats remained significantly higher through day 7. **Cytokine Production by AM:** DEP exposure significantly lowered Listeria-induced production of IL-1β, 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-γ secretion. DEP and Listeria exposure showed and increased production of IL-6 at day 3 and day 7 post-exposure.
Reference: Yin et al. (2007, 198980) **Species:** Rat **Gender:** Male **Strain:** Brown-Norway (BN/CrlBR) **Age:** NR **Weight:** 225-250 g **Cell Line:** AM	DEP = SRM 2975 eDEP = organic DEP extract wDEP = washed DEP CB = carbon black **Particle Size:** DEP: median diameter- 19.4 µm, surface area- 91 m^2/g; CB: 0.1-0.6 µm	**Route:** IT Instillation of Listeria; Cell Culture (2.5×10^5 cells/well) **Dose/Concentration:** DEP: 10, 50, 100 µg/mL; CB: 50 µg/mL **Time to Analysis:** Sacrifice 7 days postinfection or no infection. Cell culture: 1, 4, 16, 24 h.	**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 inhibited 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-γ. wDEP and CB had little effect, except high concentrations of wDEP decreased IFN-γ.

Study	Pollutant	Exposure	Effects
Reference: Yin et al. (2004, 087983) **Species:** Rat **Gender:** Male **Strain:** Brown-Norway (BN/CrlBR) **Age:** NR **Weight:** 225-250 g **Cell Line:** AM	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; Cell Culture **Dose/Concentration:** 50 µg/mL (DEP or CB) **Time to Analysis:** Killed 7 days postinstillation. AM isolated then incubated. DEP treatments for up to 24 h.	**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 inhibited 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:** 200-250 g	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 500 µL sterile saline (Listeria) **Time to Analysis:** DEP exposure for 4 h. Bacterial inoculation. Sacrificed 3, 7 days post-exposure.	**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 CD8+/CD4+ ratio. **IL-2:** DEP exposure in noninfected rats at both doses increased IL-2 in the 24 h culture and decreased IL-2 in the 48 h 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 CD4+/IL-2Rα+ and CD8+/ 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-γ:** DEP decreased IFN-γ at 3 days post-exposure, but increased at 7 days post-exposure in a dose-dependent manner. Uninfected DEP-treated rats did not substantially respond to HKLM. HKLM-induced IFN-γ production is strongly inhibited at all tested DEP doses.
Reference: Zelikoff et al. (2003, 039009) **Species:** Rat **Gender:** Male **Strain:** F344 **Age:** 7-8 mo **Weight:** NR	CAPS (concentrated ambient $PM_{2.5}$ from New York City) S.pneumoniae **Particle Size:** 0.4 µm (MMAD)	**Route:** Nose-only Inhalation (CAPS); IT Instillation (S.pneumoniae) **Dose/Concentration:** CAPS: Study 1- Mean- 345 µg/m^3; 60-600 µg/m^3. ,Study 2- Mean-107 µg/m^3; 65-150 µg/m^3 (S.pneumoniae 2-4×10^7) **Time to Analysis:** Study 1: Uninfected rats exposed to air or CAPS for 3 h. Sacrificed 3, 24, or 72 h post-exposure or IT instilled 4, 24, 72, 120 h and sacrificed 4, 24, 72 h postinfection Study 2: Infection with bacteria. Exposed 48 h later to CAPS or filtered air for 5 h. Sacrifice 9, 18, 24, 72, 120 h post-exposure.	**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 control levels by 4 h postinfection. CAPS had no effect on circulating monocyte values. CAPS significantly increased bacterial burdens at 24 h, 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 controls at 9 h and >300% greater at 18 h. 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:** 7-9 mo. **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:** Nose-only Inhalation, IT instillation (S. pneumoniae) **Dose/Concentration:** Single metals/NYC PM: 65-90 µg/m^3; 15-20×10^6 (S.pneumoniae) **Time to Analysis:** Infection/no infection followed by 5 h exposure to NYC PM or single transition metal. Sacrifice 4, 5, 9, 18, 24, and 120 h after exposure.	CAPs exposure to infected rats significantly increased pulmonary bacterial burdens of S. pneumo in a time-dependent manner. At 9 h, 18 h, 24 h, and 5 days 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 1 h post-exposure. 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 18 h, 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:** 6-8 wk **Weight:** NR **Cell Line:** J774A.1, IFN-y-primed AMs, unprimed AMs	CAPs: Concentrated Air Particles (Boston, MA) Urban air particles (UAP) SRM1649 (Washington, DC) TiO_2 Carbon Black (CB) (Sigma, St. Louis, MO) Streptococcus pneumoniae: strain ATCC6303 **Particle Size:** UAP = NR; TiO_2/CB = NR; CAPs: ≤$PM_{2.5}$	**Route:** Cell Culture **Dose/Concentration:** NR, 100 µg/mL **Time to Analysis:** CAPs for 1 h; bacteria for 1 h. Binding measured 15 h after bacteria exposure. Ingestion measured 2 h after bacteria exposure. Rate and number of killed bacteria measured 2 h after bacteria exposure.	**Binding, Internalization and Killing of Bacteria:** CAPS significantly increased binding of bacteria by IFN-y-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-10 yr **Weight:** 349 ± 116g - 20 kg	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 µg/m^3, $PM_{2.5}$: 21.6 µg/m^3, Pb in TSP: <0.4 µg/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-κB, iNOS, cyclooxygenase-2, glial fibrillatory acidic protein, ApoE, amyloid precursor product and B-amyloid.

Reference	Pollutant	Exposure	Results
Reference: Campbell et al. (2005, 087217) **Species:** Mouse **Strain:** BALB/c **Age:** 7 wk	CAPs from Los Angeles, lacking reactive organic and H_2O soluble gases, O_3, NO_X, SO_X **Particle Size:** F+UF: <2.5 µm; UF: <0.18 µm	**Route:** Whole-body Inhalation **Dose/Concentration:** 20-fold concentration of near highway ambient air, avg UF concentration: 282.5 µg/m^3, avg F concentration: 441.7 µg/m^3 **Time to Analysis:** 4 h/day, 5 days/wk for 2 wk	Mice were challenged with OVA prior to exposure and 1 and 2 wk following exposure, and then brains were assayed. F+UF and UF exposure increased NF-κB DNA binding in brain. TNF-α 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:** SD **Gender:** Male and Female **Age:** 9 wk **Weight:** 190-220 g	Gasoline exhaust (collected from 1996 Guangzhou passenger car with Dongfeng Gasoline Series 155 kw engine and no exhaust catalytic converter fuelled with 90-octane Pb-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 4 wk; 24 h post-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:** 6 wk **Weight:** NR	CAPs (Los Angeles, CA) (OC, EC = ~50%; sulfate, nitrate ~11%) **Particle Size:** NR	**Route:** Whole-body Inhalation **Dose/Concentration:** High dose: Mass concentration- 114.2 µg/m^3, Low dose: Mass concentration: 30.4 µg/m^3 **Time to Analysis:** 5 h/day, 3days/wk, 6 wk; 24 h postexposure.	Activated AP-1 dose-dependently increased. Activated NF-κB 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:** 8 wk	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:** 24 h	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:** 12-13 wk	CAPs from Grand Rapids, MI **Particle Size:** <2.5 µm	**Route:** Whole-body Inhalation **Dose/Concentration:** 500 µg/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: Veronesi et al. (2005, 087481) **Species:** Mouse **Strain:** ApoE$^{-/-}$ or C57Bl/6 **Age:** Young adults	CAPs from Tuxedo, NY **Particle Size:** <2.5 µm	**Route:** Whole-body Inhalation **Dose/Concentration:** Average daily concentration 113 µg/m^3 **Time to Analysis:** 6 h/day, 5days/wk for 4 mo	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	Pollutant	Exposure	Results
Reference: Win-Shwe et al. (2008, 190146) **Species:** Mouse **Gender:** Male **Strain:** BALB/c **Age:** 7 wk **Weight:** NR	DEP (Nanoparticle-rich - NPDE; 81-diesel engine, steady-state condition, 5 h/d, 2000rpm, 0 Nm) (CO, CO_2, NO, NO_2, SO_2) **Particle Size:** 26.21 ± 1.50 nm (diameter)	**Route:** Whole-body Inhalation **Dose/Concentration:** 148.86 ± 8.44 μg/m^3 **Time to Analysis:** 5 h/day, 5 days/wk, 4 wk. Some mice ip injected with lipoteichoic acid (LTA) 1×/wk, 4 wk. Morris water maze behavioral test: 3 days acquisition, 2 day 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-1β, however LTA was primarily responsible for the increases.
Reference: Zanchi et al. (2008, 157173) **Species:** Rat **Gender:** Male **Strain:** Wistar **Age:** 45 days	ROFA from Universidade de São Paulo, Brazil **Particle Size:** 1.2 ± 2.24 μm (MMAD)	**Route:** Intranasal Instillation **Dose/Concentration:** 20 μg/10 μl saline **Time to Analysis:** 30 days	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 Instillation **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 3 h. Protocol 1a: Pregnant mice treated with DEP or TiO_2. Analyzed 19 or 48 h later. Protocol 1b: Pregnant mice DEP, TiO_2 or CB treated day 14 of pregnancy. 4 day-old offspring i.p. injected with OVA+alum. 12-14 days-old exposed aerosolized OVA.	DEP increased BAL PMN counts in normal and pregnant mice. In pregnant mice, DEP and TiO_2 increased IL-1β, 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), 14 days 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 **Dose/Concentration:** 0.3, 1.0 or 3.0 mg DEP/m^3 **Time to Analysis:** 12 h/day, 7 days/wk from 2 day post coitum to 13 dpc. Sacrificed 14 dpc. 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: Hougaard et al. (2008, 156570) **Species:** Mouse **Strain:** C57Bl/6 **Gender:** Pregnant females, male and female offspring **Age:** 12 ,16 wk (female offspring), 13, 17 wk (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 1 h/dayfrom gestation days 7-19. Mice separated for behavioral testing on PND 22 (day of delivery is PND 0). Behavioral testing at 12, 16 wk for female offspring and 13, 17 wk for male offspring.	Body weight of exposed unchanged at birth. Body weight decreased at weaning. Unchanged dams & pups at weaning. At 2 mo, exposed female pups required less time to locate platform in spatial Reversal task of Morris Water maze.

Reference	Pollutant	Exposure	Effects
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:** 240 nm (MMAD); surface area 90 mg^2/g, density 2.1 g/cm^3	**Route:** Inhalation **Dose/Concentration:** 19.1 ± 1.13 mg DEP/m^3 **Time to Analysis:** Pregnant dams exposed GD 7-19, 1 h/day. 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 wk (male adults), 20 days of gestation (fetuses)	ME: Motorcycle Exhaust (generated from 1992 Yamaha cabin motorcycle with two-stroke 50 cc engine). **Particle Size:** NR	**Route:** Nose-only Inhalation **Dose/Concentration:** 1: 10 and 1: 50 dilutions **Time to Analysis:** 2 h/day (1 h in morning and 1 h in afternoon), for 5 consecutive days/wk, for 4 wk (1:50, 1:10 dilutions) and 2 wk (1:10 dilution). Male mated with untreated females. Pregnant females sacrificed on 20 days of gestation. Male and female fetuses observed.	After exposure, decreased body weight and testicular spermatid number were observed. 1: 10 ME exposure for 4 wk (no recovery) decreased testicular weight and increased the inflammatory cytokine mRNA. Glutathione system and lipid peroxidation 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 24 h/day, everyday for 4 mo, beginning 10 days after birth. Males mated to non-exposed females immediately following exposure. Males sacrificed immediately following mating. Pregnant females remain in chamber and sacrificed on 19 days 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:** 10 days **Weight:** Parental: 21.4 ± 4.0 - 26.3 ± 2.8 g; 15 day-old offspring: 7.8 ± 1.1 - 9.0 ± 1.0 g; 90 days-old offspring: 20.3 ± 2.3 - 27.4 ± 1.8 g	PM (busy traffic street São Paulo, Brazil; Aug. 2005-April 2006) (NO_2, SO_2, CO) **Particle Size:** 2.5, 10 µm (diameter)	**Route:** Ambient Air Exposure **Dose/Concentration:** $PM_{2.5}$: filtered chamber- 2.9 ± 3.0 $µg/m^3$, nonfiltered chamber- 16.9 ± 8.3 $µg/m^3$; Outdoor concentration: PM_{10}- 36.3 ± 15.8 $µg/m^3$, CO- 1.7 ± 0.7 ppm, NO- 89.4 ± 31.9 $µg/m^3$, SO_2- 8.1 ± 4.8 $µg/m^3$ **Time to Analysis:** Nonfiltered exposure 24 h/day for 4 mo. Mated at 120 days exposure. After birth, 30 females and offspring transferred to filtered or nonfiltered chamber. Killed 15 or 90 days of age.	Mild foci of macrophage accumulations containing black dots of carbon pigment occurred in the alveolar areas on 90 day-old mice. Surface-to-volume ratio decreased from 15 to 90 days 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: Mohallem et al. (2005, 088657) **Species:** Mouse **Strain:** BALB/c **Gender:** Female **Age:** 10 wk, 10 days	Filtered or ambient air in downtown Sao Paulo situated at crossroads with high traffic density (predominant source of air pollution is automotive). **Particle Size:** NR	**Route:** Whole-body Inhalation **Dose/Concentration:** PM_{10}: 35.5 ± 12.8 $µg/m^3$; CO: 2.2 ± 1.0 ppm; NO_2: 107.8 ± 42.3 $µg/m^3$; SO_2: 11.2 ± 5.3 $µg/m^3$ **Time to Analysis:** Exposed for 24 h/7days/wk for 4 mo. Newborns mated after reaching reproductive age of 12 wk. 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	Pollutant	Exposure	Effects
Reference: Mori et al. (2007, 096564) **Species:** Mouse **Strain:** C57/BL **Gender:** Male **Age:** 6 wk	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:** 2×/wk for 10 wk; 1 wk post last instillation.	cDNA library screen after sub-cutaneous injection identified activated clones related to prostanoids and arachadonic acid (Platg2c2c, Acsl6) and sperm production (Stk35). However, the route of exposure was unconventional.
Reference: Ono et al. (Ono et al., 2007, 156007) **Species:** Mouse **Strain:** ICR **Gender:** Pregnant females, male offspring **Age:** NR (pregnant females), 12 wk (offspring)	DE: generated from 4-cyl diesel Isuzu engine at 1500 rpm using standard diesel fuel. **Particle Size:** NR	**Route:** Inhalation **Dose/Concentration:** NR **Time to Analysis:** Exposed from 2 day post coitum to 16 dpc. Parameters for male offspring measured on days 8, 16, 21, 35, 84 and sacrificed at 84 days.	PND 8 and 16 male reproductive accessory gland weight decreased. PND 21 decreased serum testosterone (T); PND 84 increased serum T. FSHr, sTAR mRNA decreased PND 35 and 84. Relative testis and epididymal weight unchanged. Sertoli cell degeneration observed.
Reference: Ono et al. (Ono et al., 2007, 156007) **Species:** Mouse **Strain:** ICR **Gender:** Male offspring, Pregnant females **Age:** 12 wk (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 2 day postcoitum- 16 dpc. Without undergoing further exposure, male offspring sacrificed at 12 wk.	Dose-dependent increase in seminiferous tubule degeneration and decreased DSP. After 1 mo recovery, DSP recovered at the lowest dose.
Reference: Pinkerton et al. (2004, 087465) **Species:** Rat **Gender:** Female (pregnant), Offspring- NR **Strain:** SD **Age:** 10 days (pups), Pregnant females- 10-14 days of gestation **Weight:** NR	PM (Fe and soot from combustion of acetylene and ethylene in a laminar diffusion flame system) **Particle Size:** Median diameter: 72-74 nm; size range: 10-50 nm	**Route:** Inhalation **Dose/Concentration:** Mean mass concentration: 243 ± 34 µg/m^3; Average Fe concentration: 96 µg/m^3 **Time to Analysis:** Exposed 10 days postnatal age, 6 h/day, 3 days (consecutive). Bromodeoxyuridine injected 2 h 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: Silva et al. **(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:** NR	**Route:** Ambient Air Exposure **Dose/Concentration:** NR **Time to Analysis:** 1st wk, 2nd wk, 3rd wk or combo of exposure during pregnancy.	Decreased fetal weight with exposure in 1st wk of pregnancy. Decreased placental weight with exposure in any of the 3 wk of pregnancy.

Reference	Pollutant	Exposure	Effects
Reference: Somers et al. (2002, 078100) **Species:** Mouse **Strain:** Swiss Webster **Gender:** Male and Female **Age:** 6-8 wk (adult male and females), 5 days (pups)	Ambient air: 2 sites in Canada (polluted industrial area 1km downwind from two integrated steel mills & rural location 30 km away) **Particle Size:** NR	**Route:** Ambient Air Exposure **Dose/Concentration:** NR **Time to Analysis:** Exposed 24 h/day, 7 days/wk for 10 wk from September 10, 1999- November 21, 1999. Exposed to clean air for 6 wk post-treatment. Paired with mice within exposure group. 5d old pups measured.	ESTR germ line mutations following exposure. Heritable mutation rate increased 1.5 to 2 fold in urban vs. rural site. Increased frequency 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 µg/m^3, Urban-Industrial- 38.9 ± 10.5 - 115.3 ± 25.3 µg/m^3 **Time to Analysis:** Exposed 10 wk. Bred 9 wk 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:** 11 wk (offspring), NR (pregnant females)	DE **Particle Size:** NR	**Route:** Inhalation **Dose/Concentration:** 0.3 mg DEP/m^3 **Time to Analysis:** Pregnant females exposed from 2 day post coitum to 16 dpc. Offspring sacrificed 11 wk after birth.	Exposed pups had increased caspase 3 positive cells and decreased purkinjie cell number (cerebellum), similar to human Autism brain phenotype.
Reference: Tozuka et al. (2004, 090864) **Species:** Rat **Strain:** F344 **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 6 h/day 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 6 h/day from GD 7-14 with no exposure on Saturdays or Sundays. Breast milk collected PND14.	Gestational and 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/day from 2 day postcoitum to 13 dpc (with no exposure on days 4, 5, 11, 12). Sacrificed 14 dpc. Only female fetuses studied.	SF-1 & MIS mRNA did not change. Other steroidogenic genes were also unchanged. BMP-15 and 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:** 6 wk, 70 days post natal (offspring)	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 12 h/day, 7 days/wk for 4 mo. Some females sacrificed immediately following exposure. Remainder mated with unexposed males. Parameters measured in offspring at postnatal day 70.	DE-exposed females had decreased uterine weight at 4 mo. Offspring had decreased body weight at 6 and 8 wk of age. Decreased rate of good nesting construction (3 mg/m^3). AGD decreased In males (30 and 70 days old). Organ weight decreased in females and female crown to rump length decreased.
Reference: Ueng et al. (2004, 096199) **Species:** Mouse **Gender:** Female **Strain:** Wistar **Age:** 21days **Cell Line:** MCF-7	ME: generated from a Yamaha Cabin motorcycle 2-stroke 50-cc engine and variable venture carburetor **Particle Size:** NR	**Route:** Intraperitoneal 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/day for 3 days and sacrificed on 24 day. Cell Culture: 3, 24, 30, 48 h and 2 days.	10 mg/kg +E2 induced anti-estrogenic uterine effects and antiestrogenic with in vitro (MCF-7 cells) E2 screen.
Reference: Veras et al. (2008, 190493) **Species:** Mouse **Gender:** Male, Female **Strain:** BALB/c **Age:** 20 days, newborns **Weight:** NR	PM (downtown São Paulo, Brazil near crossroads with high traffic density, 67% $PM_{2.5}$ comprises air pollution) **Particle Size:** 2.5 µm (diameter)	**Route:** Open-Top Chamber **Dose/Concentration:** $PM_{2.5}$- 27.5 µg/m^3; NO_2- 101 µg/m^3; CO- 1.81 µg/m^3; SO_2- 7.66 ppm **Time to Analysis:** 20 days-old mice maintained in filtered or nonfiltered chamber until 60 days-old. Offspring maintained in respective chambers until 21 days-old. Offspring mate at 60 days-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:** 20 days **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:** 2.5 µm (diameter)	**Route:** Open-Top Chamber **Dose/Concentration:** Mean: Non-filtered- 27.5 µg/m^3, Filtered- 6.5 µg/m^3 **Time to Analysis:** 20 days-old mice maintained in filtered or non-filtered chamber. Allowed to mate at 60 days. 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:** 7 days of gestation - parturition (females), 96 days (offspring) **Weight:** 240-262 g (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 **Dose/Concentration:** High dose total group: PM- 1.71 µg/m^3, NO_2- 0.79 ppm; Low dose total group: PM- 0.17 µg/m^3, NO_2- 0.10 ppm **Time to Analysis:** Pregnant rats exposed gestational day 7 to delivery 6 h/day. 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:** 7-9 wk	HEPA-Filtered air (PM removed) and ambient air at 2 sites: -2 km from two integrated steel mills -1 km 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:** NR **Time to Analysis:** Parameters measured 3, 10 wk, or 10 + 6 wk recovery following exposure.	10+6 wk exposure induced increased ESTR mutations in sperm DNA of exposed v filtered. No testicular DNA adducts seen in exposed males. At 3 wk DNA increased adducts seen in lungs of exposed males, not in filtered males. Mutations were PM dependent, and 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; 1050 rpm, 80% load, commercial light oil) **Particle Size:** NR	**Route:** Inhalation. Pre-natal Exposure **Dose/Concentration:** DE: 1.0 mg/m^3; CO: 2.67 ppm, NO_2: 0.23 ppm, SO_2: <0.01 ppm **Time to Analysis:** Pregnant mice exposed 8 h for 5 days 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 12 h light/dark cycle. Activity monitor with infrared ray sensor measured spontaneous motor activity (SMA), 10 min intervals 2 days. 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:** 14 days of gestation (fetuses), 2-13 days 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 2-13 days of gestation. Parameters measured on 14 days 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:** 2-16 days postcoitum (pregnant females), 28 days (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 12 h/days, 7 days/wk from 2-16 days postcoitum. Offspring sacrificed on postnatal day 28.	NOAEL 0.3 mg DEP/m^3. DE exposure induced increased reproductive gland weight (two higher doses) in male mice. mRNA decreases in aromatase and 3 µ-hD (3.0 mg DEP/m^3). No change in sex ratio. Two higher doses induced significant increased reproductive organ weights. Male pup weight Increased at PND 28. Increased serum T was observed in pups exposed to 1.0mg DEP/m^3. Serum T positively correlated with DSP, testis weight, steroid enzyme mRNA.
Reference: Yoshida et al. 2004 (2004, 097760) **Species:** Mouse **Gender:** Female (pregnant), Offspring- male **Strain:** ICR **Age:** 4, 6 wk **Weight:** NR	DE **Particle Size:** NR	**Route:** Inhalation **Dose/Concentration:** 6wk-old males, embryos: 0.3, 1.0, 3.0 mg DEP/m^3, Pregnant mice: 0.1, 3.0 mg DEP/m^3 **Time to Analysis:** 6 wk-old males: Exposed 12 h/day, 6 mo. 1 mo clean air exposure. Pregnant mice: Exposed 2-13 p.c. 8 h/day. Male embryos: Exposed 2-16 p.c. Examined at 4 wk-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 1.0 mg DEP/m^3. No significant differences occurred for testosterone synthetase mRNA.

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 24 h daily 3 mo, extracted. 24 h incubation BaP, c-PAH, EOM, with or without S9. 32P-Postlabeling 4 h. Autoradiography 1-24 h.	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. (2004, 087397) **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:** 10 µm (diameter)	**Route:** Cell Culture **Dose/Concentration:** 2.5, 5, 10, 20m^3 air equivalents/mL **Time to Analysis:** Air samples extracted. Ames assay 48 h. Vivotox test. Comet assay 24 h. 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:** 10 µm (diameter)	**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 15 days, extracted. Ames test: Bacteria growth 12 h, incubated 24 h. Hepatoma bioassay: 24 h 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 12 h. Plates incubated 48 h.	**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/Concentration:** Log 2 dilutions of extracts: 1.0, 0.5, 0.25, 0.125 **Time to Analysis:** SOF extracted 12 h. Plates incubated 48 h.	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	Pollutant	Exposure	Effects
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 1 h with extracts. DNA adduct assay: DNA incubated 4 h 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: 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:** 0.4 µm (mean diameter)	**Route:** Cell Culture **Dose/Concentration:** 0, 0.25, 0.5, 1.0, 2.0 EOM µg/plate **Time to Analysis:** DEPs sonicated 20min. Centrifuged 10 min. Organic material extracted and concentrated. Ames assay. Incubated 3 days.	A-DEPs were more mutagenic in both TA98 and TA100 than SRM 2975. There was 22× more PAH-related and 8-45× 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:** 10 µm (diameter)	**Route:** Cell Culture **Dose/Concentration:** 2.5, 50, 100 µg/plate; EOM range: 6-40 µg/m^3 **Time to Analysis:** 24 h air samples, extracted. Refluxed 18-24 h. GC-MS. Comet assay. 48 h 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. 90 min pre-incubation. 48 h 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 NO_X.
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 25 min. Agar added. 48 h incubation. Rat lung explants incubated 18 h. 12 h 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: 0.554 mg extract (mg PM)-1; Diesel PM: 0.363 mg extract (mg PM)-1	**Route:** Cell Culture **Dose/Concentration:** 1.48, 4.44, 13.3, 40, 120, 360, 1080 µg/plate **Time to Analysis:** 30 min preincubation. 48 h (YG1029). 66 h (YG1024). Overnight preincubation 20 h.	**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	Pollutant	Exposure	Effects
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)), 21 mg/m^3 air (CALUX TCDD Equivalent (TCDDEq)); Cleaned APE: 7.87 mg/m^3 air (CALUX BaPEq), 0.614 mg/m^3 air (CALUX TCDDEq) **Time to Analysis:** Air samples collected, extracted. Preincubation with S. typhimuriam. 3, 24 h exposure in CALUX assay. RNA extracted from mice 6 days 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 mutagenicity in both strains.
Reference: Pastorkova et al. (2004, 087431) **Species:** S. typhimuriam **Strain:** TA98, YG1041 (±S9)	PM (EOM) (Plzeň, Prague, Ústí, Zďár - Czech Republic) **Particle Size:** 10 µm (diameter)	**Route:** Cell Culture **Dose/Concentration:** TA98 (4 doses): 20-200 µg/plate, YG1041 (4 doses): 4-20 µg/plate **Time to Analysis:** Collected 24 h every 18th day, Oct-Mar, 1999-2003. Extracted. Ames assay. 70 h 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 mutagenicity 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 16 h. Fractionated. Ames assay. Gap junction intracellular communication: exposed 1-6 h. 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/day 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:** 2.5 µm (diameter)	**Route:** Cell Culture **Dose/Concentration:** 0.25 mg/ml **Time to Analysis:** Samples taken over 7-16 days. A549 cells incubated 24 h. 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/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, 0.1 mg/plate; Comet Assay: 0.125, 0.25, 0.5, 1.0 mg/mL **Time to Analysis:** Samples extracted 24 h. 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	Pollutant	Exposure	Effects
Reference: Zhang et al. (2007, 157186) **Species:** S. typhimurium **Strain:** TA98, TA100 **Cell Line:** A549	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- 24 h incubation, followed by 2 or 24 h incubation, followed by 4 h incubation. MN assay- 24 h incubation. Comet assay. Ames assay- 72 h 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: Zhao et al. (2004, 100972) **Species:** Rat **Gender:** Male **Strain:** SD **Age:** NR **Weight:** ~200 g **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; S9: 25, 50, 100, 200 µg/plate; Cytosolic protein: 20, 40, 80, 160 µg/plate; Microsomal protein: 5, 10, 20, 40 µg/plate **Time to Analysis:** Rats instilled. Sacrificed 1, 3, 7 days post-exposure. S9, cytosolic, microsomal fractions prepared from lung homogenates. Ames assay: 72 h 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 and in vivo.

Reference	Particle	Exposure	Effects
Reference: Abou Chakra et al. (2007, 098819) **Species:** Human **Gender:** Male, Female **Age:** 6-13 yr 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:** 2.5, 10 µm (diameter)	**Route:** Cell Culture **Dose/Concentration:** 200 µL organic extract; 20 µL aphidicoline **Time to Analysis:** 24 h	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 H1l1.1c2	PM (El Paso, Texas; Juarez, Chihuahua, Mexico; Sunland Park, New Mexico) (organic extracts) **Particle Size:** 10 µm (diameter)	**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:** 24 h	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	Particle	Exposure	Effects
Reference: Bao et al. (2007, 097258) **Cell Line:** Human-hamster hybrid (AL)	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 24 h with or without cytochalasin B or ammonium chloride. Cytotoxicity: 24, 48 h incubation. Mutations: Exposed 24 h. 5-7 days culture. Incubated additional 7-8 days.	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:** 2.5 µm (diameter)	**Route:** Cell Culture **Dose/Concentration:** Strike day: 47.32 µg/m^3; Non-strike day: 43.01 µg/m^3 **Time to Analysis:** 8 h. 24 h recovery. A. cepa roots induced 5 days. Exposed 30 h. Fixed 24 h.	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:** A549	DEP (SRM 1650) **Particle Size:** NR	**Route:** Cell Culture **Dose/Concentration:** 10, 50, 100, 500 µg DEP/mL **Time to Analysis:** 2, 5, 24 h 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, 24 h.
Reference: Gabelova et al. (2007, 156458) **Species:** Human **Cell Line:** Hepatoma Hep G2	PM (PRG-SM, PRG-LB, Košice, Sofia; winter, summer) (organic extracts) **Particle Size:** 10 µm (diameter)	**Route:** Cell Culture **Dose/Concentration:** 5-150 µg/mL **Time to Analysis:** 2, 24, 48 h	Cell viability significantly decreased in the 24, 48 h exposure groups compared to the 2 h 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, 156457) **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:** 10 µm (diameter)	**Route:** Cell Culture **Dose/Concentration:** 5 -150 µg/ml **Time to Analysis:** 24 h DNA adduct formation. 2 h Comet assay. Oxidative DNA damage measured by Fpg-sensitive sites.	Total DNA adducts ranged from ~60 to 200 adducts per 108 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 4JB1-type LD,274 1,4-cylinder Isuzu diesel engine, 10 torque load, cyclone impactor, dilution tunnel constant volume sampler) **Particle Size:** <1 µm (diameter)	**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	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 ± 4.7 mg/day; Industrial mean: 22.6 ± 2.5 mg/day **Time to Analysis:** 24 h air samples 4-11 days. Substrates vortexed 1 h, suspended 4 h, centrifuged 1 h. 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, 195923) **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: 24 h incubation. Treated 24 h. Incubated again 24 h. MN assay: 24 h treatment. Gene mutation: 24 h treatment. Cells replated. 7 days expression times. Staining at 8, 10 days.	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	Particle	Exposure	Effects
Reference: Gualtieri et al. (2005, 097841) **Species:** Human **Cell Line:** A549	TD (Tire debris, generated by rotating new vehicle wheel against a steel brush, significant component of PM_{10}) (organic extracts) **Particle Size:** 10-80 µm (diameter)	**Route:** Cell Culture **Dose/Concentration:** 50, 60, 75 µg/mL **Time to Analysis:** Particles extracted 6 h. Cells subcultured every 3-4 days. After 24 h, TD treatments 24, 48, 72 h.	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	$PM_{2.5}$ 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:** 2.5 or 10 µm (diameter)	**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:** 48 h	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; 1500 rpm, 10 kg/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 2 h for dioxin toxicity measurement. Absorbance at 405 nm measured. Food, polyphenol inhibitory effects: food extract or polyphenol solution added to cytosol solution, shaken 5 min. DEPE added, shaken 5 min. 2 h incubation. Absorbance at 405 nm measured.	The dioxin toxicity equivalent was 6,479 ± 58 ng 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-MutaTM lung epithelial cells	DEP (SRM 1650b) Carbon black (CB) (Printex 90) **Particle Size:** DEP: 18-30 nm; CB: 14 nm; Agglomerates in suspensions: DEP Peaks- 249 nm, CB Peaks- 476 nm	**Route:** Cell Culture **Dose/Concentration:** 37.5, 75 µg/mL **Time to Analysis:** 8 repeated 72 h 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 3 h of exposure. CB-treated cells showed a dose-dependent increase.
Reference: Karlsson et al. (2004, 198976) **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:** <10 µm (mean diameter)	**Route:** Cell Culture **Dose/Concentration:** 0.1, 1.0, 10, 100 µg/cm^2 **Time to Analysis:** Fibroblasts exposed 24 h. Comet assay. Calf thymus incubated 2 h with microsomes or S9. 32P-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:** A549	PM (subway station, urban street) Subway particles: O_2, Fe (Fe from Fe_3O_4) Street particles: Fe from Fe_2O_3 **Particle Size:** 10 µm (diameter)	**Route:** Cell Culture **Dose/Concentration:** Comet: 5, 10, 20, 40 µg/cm^2; 8-oxodG: 10 µg/cm^2 **Time to Analysis:** 4 h.	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:** 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:** 2.5, 10 µm (diameter)	**Route:** Cell Culture **Dose/Concentration:** 40 µg/cm^2 **Time to Analysis:** Cells grown 24 h. Comet assay. Monocytes incubated 10 days. Macrophages incubated 18 h.	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	Particle	Exposure	Effects
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:** 24 h cytotoxicity. 2 h 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:** 24 h	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:** A549	PM (SRM 1949a) **Particle Size:** ≤ 0.18 µm (diameter)	**Route:** Cell Culture **Dose/Concentration:** 0, 50, 100, 200, 400 µg/mL **Time to Analysis:** Cell culture and cell viability assay: PM treatment 24 h. 10 days incubation. Host cell reactivation assay: pGL3-luciferase plasmid UV irradiated 20 min. PM treatment 24 h. 16 h transfection. 24 h PM incubation. DNA repair synthesis assay: PM treatment 24 h. Proteinase K treatment 30 min. supf mutagenesis assay: PM treatment 24 h. PM culture 60 h. 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 and Zhang (2007, 198963) **Species:** Rat **Gender:** Male **Strain:** Wistar Kyoto **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:** 24 h; cultures 4 h.	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, 198953) **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:** 24 h	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- 24 h incubation, CE, DEP exposed 24 h. MN assay- cultured 24 h, 4 h treatment, growth medium incubation 20 h. EROD-microbioassay- 48 h.	**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	Particle	Exposure	Effects
Reference: Poma et al. (2006, 096903) **Species:** Mouse **Cell Line:** RAW 264.7	PM (L'Aquila, Italy; urban); air samples collected weekly basis Jan-Mar 2004. Carbon black (CB) **Particle Size:** 2.1-0.43 µm (diameter)	**Route:** Cell Culture **Dose/Concentration:** 1, 3, 10 µg/cm^2 **Time to Analysis:** Cells cultured 48 h. Treatment 48 h. MN assay: 44 h incubation, 28 h 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: Roubicek et al. (2007, 156929) **Species:** Human **Cell Line:** A549	PM (Mexico City from an industrial area with high-traffic and a medium-traffic residential area) (aqueous or organic extracts) **Particle Size:** 10 µm (diameter)	**Route:** Cell Culture **Dose/Concentration:** 1.25, 1.63, 2.5 m^3/ml equivalents of PM_{10} **Time to Analysis:** Cells treated 24 h followed by 48 h 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:** RAW 264.7	PM (Vallila, Finland; busy traffic site; spring, winter) **Particle Size:** <10 µm (diameter)	**Route:** Cell Culture **Dose/Concentration:** 15, 50, 150, 500, 1000 µg/mL of RPMI **Time to Analysis:** 24 h	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, 198904) **Species:** Human **Cell Line:** 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:** Assays: 1, 10, 50, 100 µg/mL **Time to Analysis:** 8, 24 h.	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:** 10 µm (diameter)	**Route:** Cell Culture **Dose/Concentration:** 10-100 µg/ml **Time to Analysis:** 24 h	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:** A549	PM (Düsseldorf, Germany, July-Dec.) Weekly samplings July-Dec 1999. **Particle Size:** Fine diameter: <2.5 µm; Coarse diameter: 10-2.5 µm	**Route:** Cell Culture **Dose/Concentration:** Fine: 0.57-2.49 mg; Coarse: 0.66-1.89 mg; Concentration: 0.57 mg/mL **Time to Analysis:** NR	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 and Laben, Karviná; background: Červenohorské sedlo, Košetice - Czech Republic; July) (organic extracts, TSP); GP (gas phase). 24 h samples July 2002 **Particle Size:** <2.5 µm (diameter)	**Route:** Cell Culture **Dose/Concentration:** SOS: 8, 4, 2, 1 m^3/ml; Dioxin: TSP+GP- 8, 1.33, 0.22, 0.04 m^3/ml, $PM_{2.5}$+GP: 4, 0.66, 0.11, 0.02 m^3 ml-1 **Time to Analysis:**. SOS chromotest: 22 h incubation. Dioxin toxicity test: 24 h 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, 0.1 mg/plate; Comet Assay: 0.125, 0.25, 0.5, 1.0 mg/mL **Time to Analysis:** 24 h	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), BEAS-2B, 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:** microarray analysis. RT-PCR: 2 h. ELISA: 12 h incubation. Centrifuged 24 h post-treatment. Bioactivity: 12 h incubation. Centrifuged 24 h post-treatment. Medium replaced 48 h post-incubation. Fibroblasts determined 96 h post-incubation. Time response studies: 3-48 h treatment. Concentration response studies: 6 h 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 6 h 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- 57.7 mg/total UPM; Ibirapuera Park: UPM- 32 µg/m^3, EOM- 41.7 mg/total UPM; Salmonella assay- 0.5, 1, 5, 10, 50, 100 UPM equiv/plate (µg) **Time to Analysis:** Organic extraction 20 h. PAH fractionation.	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** 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:** 1, 4, 8, 12, 24 h.	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. ΔΨm 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 ΔΨm 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:** >10.2 - <0.41 µm (diameter)	**Route:** Incubation **Dose/Concentration:** 20, 40 mg/5mL **Time to Analysis:** PM incubated with H_2O_2 and 2'-deoxyguanosine (dG). Stored 3-7 days at -20°C.	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:** A549	PM (Taiyuan, Beijing; Nov-Feb) (Taiyuan: coal-fume pollution; Beijing: coal-fume and vehicle exhaust) **Particle Size:** 2.5 µm (diameter)	**Route:** Cell Culture **Dose/Concentration:** 5, 50, 200 µg/mL **Time to Analysis:** 12-24 h	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 yr **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 yr, 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:** 1-day, 7-day ma.	**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 25.7m3 increase in 7-day ma 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)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
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 yr **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 yr, 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 lags of same day, 7-day, 30-day, and h 0-6 **Mean (SD):** NR **Percentiles:** Sep-Nov: 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 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	**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 ma 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) 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 0-2 day	**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 avg 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) day-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) Avg HR: 0.321 (-0.197, 0.838) 24 h SDNN: 1.040 (-0.415, 2.494) 24 h SDANN: 1.195 (-0.473, 2.863) 24 h RMSSD: 0.321 (-0.197, 0.838) 24 h PNN: 2.837 (-3.791, 9.465) 24 h LF power: 0583 (-3.622, 4.787) 24 h LF normalized: -3.137 (-5.540, -0.733)* 24 h HF power: 0.872 (-4.649, 6.392) 24 h HF normalized: -2.223 (-4.952, 0.505) 24 h 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 yr **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:** 0-5 day	**Pollutant:** PM_{10} **Averaging Time:** 24 h **5 day 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, R2:** 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 yr **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 avg over 1- to 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-day 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-day: 135.8 (1.8, 269.7) 2-day: 108.2 (-10.9, 227.3) 3-day: 109.6 (2.5, 216.7) **8-OHdG:** 1-day: -9.2 (-21.5, 3.2) 2-day: -6.1 (-17.0, 4.8) 3-day: -5.6 (-13.8, 2.6) **PAI-1:** 1-day: 30.0 (12.4, 47.7) 2-day: 19.1 (3.6, 34.7) 3-day: 21.2 (9.7, 32.8) **tPA:** 1-day: 16.0 (-4.1, 36.2) 2-day: 10.4 (-6.3, 27.2) 3-day: 8.8 (-2.8, 20.5) **Fibrinogen:** 1-day: 5.3 (1.5, 15.2) 2-day: 1.5 (-4.4, 7.5) 3-day: 3.3 (-1.1, 7.7) **Heart Rate Variability** **SDNN:** 1-day: -4.9 (-7.8, -2.1) 2-day: -4.0 (-6.6, -1.4) 3-day: -4.1 (-6.1, -2.2) **r-MSSD:** 1-day: -4.8 (-12.3, 2.7) 2-day: -2.2 (-9.0, 4.7) 3-day: -4.0 (-9.0, 0.9) **LF:** 1-day: -6.1 (-10.1, -2.1) 2-day: -3.0 (-7.2, 1.2) 3-day: -4.3 (-7.0, -1.6) **HF:** 1-day: -5.5 (-13.0, 2.1) 2-day: -2.7 (-9.5, 4.1) 3-day: -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 yr mean age= 74 yr **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: C10: IQR = 7 μg/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: A10: IQR = 6.5μg/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 yr **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:** 24 h **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 μg/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+ yr **Study Design: C**ross-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 μg/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 yr **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- to 7-day avg	**Pollutant:** PM_{10} **Averaging Time:** 24 h **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 ms2 (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 U.S. (Forsyth County, NC suburbs of Minneapolis, MN black residents of Jackson, MS)	**Outcome:** Fibrinogen, factor VIII co-agulant activity (VIII-C), von Willebrand factor (vWF), white blood cell count (WBC), and serum albumin **Age Groups:** 45-64 yr **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 avg (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 µg/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 U.S. states	**Outcome:** Ectopy **Age Groups:** Women 50-79 yr **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 0-365 day ‡ 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 µg/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 2005-Jul 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 yr **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-wk 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 h: 25.5 (9.8-133.0) 0-6 h: 15.3 (5.3-83.2) 7-12 h: 17.0 (7.1-186.3) 13-18 h: 28.5 (11.4-167.0) 19-24 h: 30.5 (10.1-148.2) **Monitoring Stations:** Personal monitoring **Copollutant (correlation):** Ambient $PM_{2.5}$ (r = 0.34)	**PM Increment:** 10 µg/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/m2 (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/m2 (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/m2 (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/m2 (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/m2 (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/m2 (n=14): 0.15 (0.12) **Diastolic blood pressure (mm Hg):** All subjects (n=24): 0.19 (0.16) subjects not taking vasoactive meds

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			(n=17): 0.40 (0.18)** subjects w/BMI ≤ 29kg/m2 (n=14): 0.27 (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/m2 (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/m2 (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/m2 (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/m2 (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/m2 (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/m2 (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)**

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			13-18 h: 0.01 (0.09) 19-24 h: -0.08 (0.05) **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:** Feb-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 wk), using Holter monitors, were made. Subjects' residences were within 5 miles of 1 of 2 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 yr **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 2-24 h	**Pollutant:** PM_{10} **Averaging Time:** Hourly Gothenburg, Stockholm **Median:** 2h: 18.95, 14.62 24 h: 19.92, 15.23 **Min:** 2h: 0.00, 0.33 24 h: 2.13, 3.96 **Max:** 2h: 203.75, 159.79 24 h: 78.01, 90.50 **IQR:** 2h: 14.16, 11.59 24 h: 11.49, 9.59 **Monitoring Stations:** 2 **Copollutant:** $PM_{2.5}$, NO_2 **Co-pollutant Correlation** 2 h NO_2: 0.36 24 h NO_2: 0.29	**PM Increment:** Interquartile Range **Odds Ratio (Lower CI, Upper CI):** 2 h: 1.31 (1.00, 1.72) 24 h: 1.24 (0.87, 1.76) **Notes:** OR of ventricular arrhythmia for an IQR increase of air pollutants in different subgroups (Fig 2)
Reference: Ljungman et al. (2009, 191983) **Period of Study:** May 2003-Jul 2004 **Location:** Athens, Greece Helsinki, Finland Ausburg, Germany Barcelona, Spain Rome, Italy Stokholm, Sweeden	**Outcome:** Interleukin-6 Response **Age Groups:** 35-80 yr **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:** 1 day	**Pollutant:** PM_{10} **Averaging Time:** 24 h **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 µg/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 yr **Study Design:** Panel study **N:** 88 elderly subjects **Statistical Analysis:** GEE	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (SD):** Indoor: 12.6 (7.8) Outdoor: 14.5 (7.0)	**PM Increment:** 10 µg/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:** Jan 1993-Dec 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: A**ge, sex, BMI, smoking **Dose-response Investigated?** No **Statistical Package:** SAS, R **Lags Considered:** 0-72 h	**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 µg/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)*** 24-h-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)*** 24-h-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) 24-h-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 (Fig 2) Percent change in HRV for PM_{10} exposure in all ages (Fig 3)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Peters et al. (2009, 191992) **Period of Study:** May 2003-Jul 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- to 5-day avg	**Pollutant:** PM_{10} **Averaging Time:** 24 h **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 µg/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)
Reference: Rosenlund et al. (2007, 114679) **Period of Study:** 1985-1996 **Location:** Stockholm County	**Outcome:** Myocardial Infarction **Age Groups:** 15-79 yr **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 yr, SES **Dose-response Investigated?** No **Statistical Package:** Stata **Lags Considered:** 5 yr	**Pollutant:** PM_{10} **Averaging Time:** 5 yr **Median:** 2.4 **5th-95th:** 0.3-6.2 **Median:** 2.2 **5th-95th:** 0.3-6.0 **Monitoring Stations:** NR **Copollutant:** NO_2, CO **Co-pollutant Correlation:** HNR	**PM Increment:** 5th-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)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
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 yr **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 lags 2-3, 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-day 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-day 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-day 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+ yr **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 AP $PM_{2.5}$ PM_{10} OC EC NO_2 CO	**PM Increment:** IQR (15.2 5-day 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: 0-23 h: 1.2 (0.8, 1.9) 24-47 h: 2.0 (1.1, 3.6) 48-71 h: 2.2 (1.2, 3.8) 5-day mean: 2.0 (1.2, 3.7) **ICAM-1:** Time before draw: 0-23 h: 1.3 (0.9, 1.8) 24-47 h: 3.1 (2.0, 4.8) 48-71 h: 3.4 (2.2, 5.2) 5-day 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: 0-23 h: 4.0 (-0.6, 8.5) 24-47 h: 6.0 (0.6, 11.5) 48-71 h: 1.1 (-4.9, 7.0) 5-day mean: 6.1 (-0.6, 12.8) **FVII:** Time before draw: 0-23 h: -6.6 (-10.4--2.5) 24-47 h: -8.4 (-12.3--4.3) 48-71 h: -5.9 (-9.6, -2.0) 5-day 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+ yr **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_{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 AP $PM_{2.5}$ PM_{10} NO	**PM Increment:** IQR (15.2 5-day 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) lag 1: 1.1 (-5.4, 7.9) lag 2: -3.5 (-8.9, 2.2) lag 3: -1.4 (-6.0, 3.4) 5-day mean: -1.2 (-7.8, 5.8) **Platelets, % change mean (103/µl):** lag 0: -0.4 (-1.9, 1.0) lag 1: 0.4 (-1.4, 2.3) lag 2: 0.5 (-1.4, 2.3) lag 3: -0.1 (-1.6, 1.4) 5-day mean: 0.0 (2.1, 0.0) **Leukocytes, % change in mean (103/µl):** lag0: -1.1 (-2.8, 0.7) lag 1: -0.5 (-2.6, 1.5) lag 2: 0.1 (-2.1, 2.4) lag 3: -0.7 (-2.6, 1.2) 5-day mean: -1.1 (-3.6, 1.4) **Erythrocytes, % change mean (106/µl):** lag0: 0.0 (-0.4, 0.5) lag 1: -0.4 (-1.0, 0.1) lag 2: -0.7 (-1.2, -0.2) lag 3: -0.4 (-0.8, 0.0) 5-day mean: -0.6 (-1.2, -0.1) **Hemoglobin, % change mean (g/dl):** lag 0: -0.1 (-0.7, 0.6) lag 1: -0.4 (-1.2, 0.3) lag 2: -0.7 (-1.3, 0.0) lag 3: -0.3 (-0.9, 0.2) 5-day 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) yr **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, & yr **Dose-response Investigated?** No **Statistical Package:** SPSS **Lags Considered:** 0-7 days	**Pollutant:** PM_{10} **Averaging Time:** 24 h **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 µg/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:** Feb-Apr 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 yr **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: 00-18: 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 day	**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 µg/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 yr **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, and 3 days	**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 Fig 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 yr.

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Whitsel et al. (2009, 191980) **Period of Study:** 1993-2004 **Location:** U.S.	**Outcome:** Heart Rate Variability **Age Groups:** 50-79 yr **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:** 24 h 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 µg/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) 5-day 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) 5-day 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) 5-day 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)* 5-day 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) 5-day 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) 5-day 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) 5-day 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) 5-day avg: -0.72 (-1.57, 0.14) *p < 0.1
Reference: Yeatts et al. (2007, 091266) **Period of Study:** 12-wk period b/t Sep 2003-Jul 2004 **Location:** Chapel Hill, NC	**Outcome:** Heart Rate Variability **Age Groups:** 21-50 yr **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:** 24 h **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 µg/m^3 **Beta, SE, p-value (Lower CI, Upper CI):** NR

[1]All units expressed in µg/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 yr **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- to 4-h ma	**Pollutant:** $PM_{10-2.5}$ **Averaging Time:** 1 h 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 yr mean age= 74 yr **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 ≥ 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: C10-2.5: IQR = 4.5 μg/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: A10-2.5: IQR = 2.4 μg/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:** Feb-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 wk for up to 12 wk), using Holter monitors, were made. Subjects' residences were within 5 mi of 1 of 2 PM monitoring sites. Regressed HRV parameters against 18: 00-20: 00 mean particulate pollution
Reference: Metzger et al. (2007, 092856) **Period of Study:** Aug 1998-Dec 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 h **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 1998-1999 **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, $PM_{2.5}$, PM_1, 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 2 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 μg/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 mo 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 Sep 2003-Jul 2004 **Location:** Chapel Hill, NC	**Outcome:** Heart Rate Variability **Age Groups:** 21-50 yr **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:** 24 h **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 µg/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_24hr: -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 µg/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 yr **Study Design:** Panel (4 planned repeated measures surrounding bus	**Pollutant:** $PM_{2.5}$ (µg/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-min, 4-h, 24-h ma **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 Increment:** IQR **Effect Estimate [Lower CI, Upper CI]:** % change (95%CI) in HRV per IQR in the 24-h ma of the microenvironmental pollutant (IQr = 4.5 µg/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**

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
	trips 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	$PM_{2.5}$ BC Fine particle counts coarse particle counts **Correlation notes:** 24-h mean $PM_{2.5}$, BC, and fine particle count concentrations ranged from 0.80-0.98 r = 0.76-0.97 when limited to time spent on the bus r = 0.55-0.86 when comparing bus concentrations to 24-h ma r = -0.003-0.51 when comparing 5-min avg and 24-h ma Poor correlations found between coarse particle count concentrations and all fine particulate measures during all times periods	**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: -10.7(-12.4, -9.1) HF: -14.9(-16.5, -13.3); LF/HF: 4.9 (3.6, 6.2)' H: 0.9 (0.7,1.1) Independent short- and medium-term associations with HRV across all time periods % change per IQR (95%CI) IQR 5-min means = 6.8 $\mu g/m^3$ and 23: 55-h means = 4.2 $\mu g/m^3$ 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 avg (5-min means) of PM with HRV by bus and nonbus periods IQR for bus = 10 $\mu g/m^3$) and nonbus = 5.6 $\mu g/m^3$) % 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) 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 Fig 2 (magnitude of associations increased with averaging period, with the largest associations consistently found for 24-h ma)

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 yr **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-min, 4-h, 24-h ma **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 ma r = -0.003 to 0.51 when comparing 5-min avg and 24-h ma 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 ma 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 avg (5-min means) of PM with HRV by bus and nonbus periods IQR for bus = 2.6 $\mu g/m^3$) and nonbus = 0.27 $\mu g/m^3$) % 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 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 Fig 2 (magnitude of associations increased with averaging period, with the largest associations consistently found for 24-h ma)
Reference: Auchincloss et al. (2008, 156234)	**Outcome:** Blood pressure: Systolic (SBP), diastolic (DBP), mean arterial	**Pollutant:** $PM_{2.5}$	**PM Increment:** 10 $\mu g/m^3$ (approx. equivalent to difference between 90th

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Period of Study: Jul 2000-Aug 2002 **Location:** 6 U.S. 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)	(MAP), pulse pressure (PP) Avg of 2nd and 3rd BP measurement used for analyses **Age Groups:** 45-84 yr **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 **Dose-response Investigated?** Assessed nonlinear relationships-no evidence of strong threshold/nonlinear effects for $PM_{2.5}$ **Statistical Package:** NR	**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 PM2.5 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 Warm: r = -0.11 NO_2 Cool: r = 0.55 Moderate: r = 0.66 Warm: 0.32	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 $PM_{2.5}$ (avgd for the prior 30 days) **Pulse Pressure** ($PM_{2.5}$ avgd 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 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

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			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 ma **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 0-2 day	**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 avg 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) day-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) Avg HR: 0.617 (-0.782, 2.016) 24 h SDNN: 3.645 (-0.227, 7.517) 24 h SDANN: 4.437 (0.030, 8.844)* 24 h RMSSD: 0.617 (-0.782, 2.016) 24 h PNN 50%: 11.247 (-6.228, 28.722) 24 h LF power: 4.439 (-6.823, 15.701) 24 h LF normalized: -5.659 (-11.815, 0.497) 24 h HF power: 3.800 (-10.863, 18.464) 24 h HF normalized: -6.597 (-13.724, 0.531) 24 h 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 yr **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:** 0-5 days	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **5 day 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, R2:** 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 yr **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 0-14 h	**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 Lag 1 h: -1.44 (-7.75, 4.87) Lag 2 h: -5.33 (-10.97, 0.31)* Lag 3 h: -6.86 (-11.91, -1.81)‡ Lag 4 h: -2.17 (-9.33, 4.99) Lag 5 h: -4.73 (-11.99, 2.53) Lag 6 h: -3.52 (-9.89, 2.84) Lag 7 h: -1.59 (-7.53, 4.35) Lag 8 h: -0.72 (-7.63, 6.20) Lag 9 h: -5.55 (-10.65, -0.45)‡ Lag 10 h: -3.66 (-8.85, 1.53) Lag 11 h: -8.60 (-17.45, 0.24)* Lag 12 h: -5.98 (-14.67, 2.70) Lag 13 h: -8.27 (-17.00, 0.46)* Lag 14 h: -4.19 (-12.71, 4.33) Model 2 Lag 1 h: 4.10 (-0.39, 8.60)* Lag 2 h: -3.21, (-8.78, 2.37) Lag 3 h: -6.45 (-11.59, -1.31)‡ Lag 4 h: -0.01 (-6.96, 6.94) Lag 5 h: -2.03 (-8.27, 4.22) Lag 6 h: -1.99 (-8.46, 4.48) Lag 7 h: -0.34 (-6.22, 5.54) Lag 8 h: 0.72 (-6.35, 7.78) Lag 9 h: -5.26 (-10.62, 0.11)* Lag 10 h: -3.68 (-9.17, 1.80) Lag 11 h: -9.41 (-18.60, -0.23)‡ Lag 12 h: -6.45 (-15.62, 2.72) Lag 13 h: -7.33 (-16.55, 1.89) Lag 14 h: -4.75 (-13.81, 4.32) *p < 0.05, ‡p < 0.10 **Notes:** Model 1 adjusted for smoking status and age only. Model 2 adjusted for smoking status, age, and 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: M**ean 72.8(6.6) yr **Study Design:** Panel **N:** 539 white males **Statistical Analyses:** Mixed Effects Model **Covariates: A**ge, 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- to 2-day ma	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 1 h **Mean (SD):** 11.7 (7.8) **Monitoring Stations:** 1 **Copollutant:** $PM_{1.0}$ **Co-pollutant Correlation:** N/A	**PM Increment:** 10 µg/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:** U.S.	**Outcome:** White Blood Cell count **Age Groups:** 20-89 yr **Study Design:** Panel **N:** 2,978 participants **Statistical Analyses:** Mixed Effects Models **Covariates: A**ge, 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:** 24 h **Mean (SD):** 36.8 (13.0) **Median(range) for** **Q1:** 23.1(14.6-27.8) **Q2:** 31.2 (27.9-34.3) **Q3:** 38.8 (34.3-43.3) **Q4:** 53.7 (43.3-78.5) **Monitoring Stations:** NR **Copollutant:** NR **Co-pollutant Correlation:** N/A	**PM Increment:** Quartile, 1yr avg (36.8 µg/m^3) **Avg WBC count(SE) by PM quartile:** **Q1:** 6760 (79) **Q2:** 6942 (99) **Q3:** 6895 (84) **Q4:** 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. (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 yr **Study Design:** Panel (cross-sectional) **N:** 76 students **Statistical Analyses: L**inear 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 avg over 1- to 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 1 site only) **Copollutant:** PM_{10} Sulfate Nitrate OC EC NO_2 CO SO_2 O_3	**$PM_{2.5}$ Increment:** IQR (1-day 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-day: 90.2 (-10.2, 190.1) 2-day: 99.1 (-26.1, 224.3) 3-day: 100.4 (-2.9, 203.7) 8-OHdG: 1-day: -5.0 (-14.3, 4.4) 2-day: -5.5 (-15.6, 4.6) 3-day: -5.6 (-13.8, 2.6) PAI-1: 1-day: 20.4 (17.3, 33.5) 2-day: 16.2 (1.9, 30.5) 3-day: 20.0 (18.5, 31.5) tPA: 1-day: 12.0 (-2.4, 26.3) 2-day: 12.0 (-2.9, 26.9); 3-day: 12.0 (-2.7, 26.6) Fibrinogen: 1-day: 2.6 (-2.7, 7.8) 2-day: 1.5 (-4.1, 7.1); 3-day: 3.6 (-0.8, 8.1) **Heart Rate Variability** SDNN: 1-day: -4.0 (-6.1 to -1.9) 2-day: -2.5 (-4.6 to -0.4) 3-day: -3.0 (-5.0 to -1.1) r-MSSD: 1-day: -3.0 (-8.7, 2.7) 2-day: -2.0 (-8.4, 4.4); 3-day: -3.6 (-8.8, 1.6) LF: 1-day: -3.1 (-6.1 to -0.1) 2-day: -3.2 (-4.6, 0.1); 3-day: -3.4 (-6.1 to -0.6) HF: 1-day: -3.7 (-9.4, 2.1) 2-day: -2.1 (-8.4, 4.3); 3-day: -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 yr **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 avg 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 1 site only) **Copollutant:** PM_{10} Sulfate $PM_{2.5}$ OC EC NO_2 CO SO_2 O_3	**Nitrate Increment:** IQR (1-day 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-day: -2.1 (-21.9, 17.8) 2-day: -11.6 (-58.6, 35.5) 3-day: -18.7 (-69.9, 32.5) 8-OHdG: 1-day: 9.0 (4.0, 14.1) 2-day: 15.1 (5.9, 24.3) 3-day: 15.0 (4.9, 25.0) PAI-1: 1-day: 4.0 (-2.5, 10.4) 2-day: 11.6 (0.1, 23.1) 3-day: 16.9 (4.3, 29.4) tPA: 1-day: 2.0 (-6.2, 10.3) 2-day: 12.9 (-1.6, 27.5) 3-day: 10.0 (-5.8, 25.8) Fibrinogen: 1-day: 1.6 (-1.3, 4.5) 2-day: 1.3 (-3.9, 6.5) 3-day: 1.0 (-4.6, 6.6) **Heart Rate Variability** SDNN: 1-day: -1.5 (-2.6 to -0.3) 2-day: -2.6 (-4.7 to -0.5) 3-day: -3.0 (-5.3 to -0.7) r-MSSD: 1-day: -5.5 (-8.7 to -2.2) 2-day: -7.1 (-14.0 to -0.2) 3-day: -8.1 (-14.5 to -1.8) LF: 1-day: -1.0 (-1.6 to -0.5) 2-day: -2.0 (-5.6, 1.6) 3-day: -2.0 (-5.2, 1.2) HF: 1-day: -2.0 (-5.3, 14[potential typo, possibly 1.4]) 2-day: -4.9 (-10.9, 0.9) 3-day: -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 yr **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 avg over 1- to 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 1 site only) **Copollutant:** PM_{10} $PM_{2.5}$ Nitrate OC EC NO_2 CO SO_2 O_3	**Sulfate Increment:** IQR (1-day 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-day: 80.0 (9.8, 150.2) 2-day: 87.1 (14.9, 159.4) 3-day: 71.1 (13.0, 129.2) 8-OHdG: 1-day: 1.0 (0.3, 1.3) 2-day: -0.4 (-5.4, 4.7) 3-day: -0.3 (-4.3, 3.7) PAI-1: 1-day: 12.0 (5.4, 18.7) 2-day: 13.3 (6.6, 19.9) 3-day: 11.2 (5.7, 16.6) tPA: 1-day: 2.0 (-4.6, 8.7) 2-day: 3.8 (-2.8, 10.3) 3-day: 3.0 (-2.3, 8.2) Fibrinogen: 1-day: 2.9 (0.2, 5.5) 2-day: 2.8 (0.1, 5.5) 3-day: 2.2 (0.4, 4.7) **Heart Rate Variability** SDNN: 1-day: -3.1 (-4.1 to -2.1) 2-day: -4.1 (-5.2 to -3.1) 3-day: -2.0 (-2.9 to -1.2) r-MSSD: 1-day: -5.0 (-8.0 to -2.0) 2-day: -6.0 (-8.9 to -2.9) 3-day: -5.7 (-8.2 to -3.2) LF: 1-day: -3.4 (-4.9 to -1.8) 2-day: -3.0 (-4.5 to -1.5) 3-day: -3.0 (-4.3 to -1.7) HF: 1-day: -3.5 (-6.5 to -0.4) 2-day: -3.9 (-7.0 to -0.8) 3-day: -3.0 (-5.5 to -0.5)

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Chuang et al. (2007, 098629) **Period of Study:** NR **Location:** Boston, MA	**Outcome:** ST Segment Depression **Age Groups:** 43-75 yr **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 1-72 h	**Pollutant:** $PM_{2.5}$ **Averaging Time:** Hourly **25th, 50th, 75th percentile:** 12-h avg: 6.18, 8.91, 13.18 24-h avg: 6.38, 9.20, 13.31 **Max:** 12-h avg: 37.13 24-h 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-h 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-h 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-h 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-h 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-h mean Myocardial Infarction: -0.042 (-0.057, -0.026) No Myocardial Infarction: -0.012 (-0.023, 0.00) 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-h 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-h St segment levels (Fig 1)

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Dales et al. (2007, 155743) **Period of Study:** NR **Location:** Ottawa, Canada	**Outcome:** Vascular Reactivity **Age Groups:** 18-50 yr **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:** 2 h **Mean (SD):** Downtown: 40 (20) Tunney's Pasture: 10 (10) p-value 0.000 **Monitoring Stations:** NR **Copollutant:** PM1.0 **Co-pollutant Correlation** N/A	**PM Increment:** Interquartile Range (27.02 µg/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: de Hartog et al. (2009, 191904) **Period of Study:** 1998-1999 **Location:** Amsterdam, The 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 0-3 days	**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, PM0.1-1.0, NO_2, SO_2 **Co-pollutant Correlation** NR **Note:** Correlations are provided for source-specific $PM_{2.5}$ & elements	**PM Increment:** 1 µg/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:** Jul-Aug 1999 **Location:** Boston, MA	**Outcome:** Oxygen saturation **Age Groups**: 60.4-89.2 yr **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 µg/m^3) increase 6 h: 13.42 µg/m^3 12 h: 10.81 µg/m^3 24 h: 10.26µg/m^3 48: 10.57 µg/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:** Fig of the variation in oxygen saturation during the first rest period vs.. individual hourly lag measurements for $PM_{2.5}$
Reference: Diez-Roux et al. (2006, 156400) **Period of Study:** Baseline data collected Jun 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 yr **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 wk, medications, infections in last 2 wk (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 wk, 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 U.S. EPA **Copollutant:** SO_2 NO_2 CO O_3	**PM Increment:** 10 µg/m^3 **Effect Estimate [Lower CI, Upper CI]:** Adjusted (all personal-level covariates) relative difference in CRP (mg/L) per 10 µg/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 µg/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	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Dubowsky et al. (2006, 088750) **Period of Study:** Mar-Jun 2002 **Location:** St. Louis, Missouri	**Outcome:** White blood cells (WBC), C-reactive protein (CRP), interleukin-6 (IL-6) **Age Groups:** ≥ 60 yr **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 avgd 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 µg/m^3 (5-day 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 µg/m^3) of $PM_{2.5}$ avgd 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 (x109/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: Dubowsky et al. (2006, 088750) **Period of Study:** Mar-Jun 2002 **Location:** St. Louis, Missouri	**Outcome:** White blood cells (WBC), C-reactive protein (CRP), interleukin-6 (IL-6) **Age Groups:** ≥ 60 yr **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 avgd 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-day 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 (x109/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	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 yr mean age= 74 yr **Study Design: E**xtended 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	**PM Increment:** Increment: C2.5: 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_C2.5: 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_C2.5: 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: A2.5: 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_A2.5: IQR = 3.4 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_T2.5: 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: T2.5: 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: N2.5: 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) **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.

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
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 µg/m^3 **Beta (SE), p-value:** **∆SDNN** 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 **∆ 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 ∆ FEV_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 **∆ FVC** Morning, $\Delta PM_{2.5}$ avg: -0.10 (0.09), 0.31 Morning, $\Delta PM_{2.5}$ peak: -0.12 (0.17), 0.51 **∆ 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 avg 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 yr **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:** 24 h **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 µg/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	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Folino et al. (2009, 191902) **Period of Study:** Jun 2006-May 2007 **Location:** Padua, Italy	**Outcome:** HRV & Inflammatory Markers **Age Groups:** 45-65 yr **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:** 24 h **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 µg/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 yr **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 1 day; 0- to 2-day avg	**Pollutant:** $PM_{2.5}$ **Averaging Time: D**aily **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 µg/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 0-2-day 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 0-2-day 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 0-2-day 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 0-2-day 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 yr **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 0-4 days; 0- to 2-day 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 µg/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 0-2-day 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 0-2-day 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 0-2-day 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 0-2-day avg
Reference: Ibald-Mulli et al. (2004, 087415) **Period of Study: W**inter 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: T**rend, day of week, temperature, barometric pressure, relative humidity, medication use **Dose-response Investigated?** No **Statistical Package:** SAS **Lags Considered:** 0-2, 5-day avg	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **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 µg/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:** Aug 2008 **Location:** Beijing, China	**Outcome:** Cardiovascular Effects **Age Groups:** Median 28 yr **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 h, night, before walk, and 24 h after walk.

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Lanki et al. (2006, 088412) **Period of Study:** Fall 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) yr **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** Lag 0: 0.80 (0.47, 1.36) Lag 1: 0.66 (0.40, 1.10) Lag 2: 1.18 (0.68, 2.06) Lag 3: 1.87 (0.85, 4.09) **Long-range transport** Lag 0: 0.94 (0.84, 1.05) Lag 1: 1.00 (0.92, 1.08) Lag 2: 1.11 (1.02, 1.20) Lag 3: 1.06 (0.95, 1.18) **Oil combustion** Lag 0: 0.87 (0.57, 1.32) Lag 1: 1.04 (0.75, 1.45) Lag 2: 1.10 (0.83, 1.46) Lag 3: 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** Lag 0: 0.91 (0.69, 1.21) Lag 1: 1.22 (0.88, 1.69) Lag 2: 1.53 (1.19, 1.97) Lag 3: 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** Lag 0: 0.98 (0.86, 1.10) Lag 1: 1.03 (0.95, 1.12) Lag 2: 1.11 (1.02, 1.21) Lag 3: 1.02 (0.95, 1.10) **Oil combustion** Lag 0: 0.95 (0.61, 1.49) Lag 1: 1.13 (0.76, 1.68) Lag 2: 1.33 (0.98, 1.80) Lag 3: 1.29 (0.90, 1.86) **Salt** Lag 0: 1.15 (0.56, 2.38) Lag 1: 0.90 (0.44, 1.81) Lag 2: 1.39 (0.63, 3.08) Lag 3: 1.93 (1.00, 3.72) **Local traffic** Lag 0: 0.89 (0.64, 1.23) Lag 1: 1.21 (0.86, 1.71) Lag 2: 1.37 (1.03, 1.83) Lag 3: 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)** Lag0: 0.73 (0.39, 1.38)

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			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 0-24 h	**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 24 h: 9.0, 12.5, 17.7, 30.5 **Monitoring Stations:** 1 **Co-pollutant:** PM<0.1 **Co-pollutant Correlation** Personal & Outdoor $PM_{2.5}$ 1 h & 1 h: 0.70 4 h & 4 h: 0.54 8 h & 8 h: 0.60 12 h & 12 h: 0.50 22 h & 24 h: 0.80 **Notes:** 1-22 h pollutant averaging times. Correlations also available for personal-personal and outdoor-outdoor.	**PM Increment:** 10 µg/m^3 **Odds Ratio (Lower CI, Upper CI):** Personal $PM_{2.5}$ 1-h avg: 3.26 (1.07, 9.99)* 4-h avg: 2.42 (0.75, 7.83) 8-h avg: 1.57 (0.49, 5.09) 12-h avg: 1.96 (0.44, 8.64) 22-h avg: 2.06 (0.30, 14.10) Outdoor $PM_{2.5}$ 1-h avg: 1.77 (0.87, 3.58) 4-h avg: 2.47 (1.05, 5.85)* 8-h avg: 1.83 (0.80, 4.20) 12-h avg: 1.90 (0.77, 4.65) 24-h avg: 1.60 (0.59, 4.39) *p < 0.05
Reference: Liao et al. (2007, 180272) **Period of Study:** 1999-2004 **Location:** 24 U.S. States	**Outcome:** Ectopy **Age Groups:** women 50-79 yr **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 0-365 days	**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 µg/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:** Feb-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 wk), using Holter monitors, were made. Subjects' residences were within 5 mi of 1 of 2 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 yr **Study Design: C**ase-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 2-24 h	**Pollutant:** $PM_{2.5}$ **Averaging Time:** Hourly **Median:** 2 h: 9.17 24 h: 9.49 **Min:** 2 h: 0.15 24 h: 2.97 **Max:** 2 h: 99.25 24 h: 47.07 **IQR:** 2 h: 6.69 24 h: 5.27 **Monitoring Stations:** 1 **Copollutant:** PM_{10}, NO_2 **Co-pollutant Correlation:** NR	**PM Increment:** Interquartile Range **Odds Ratio (Lower CI, Upper CI):** 2 h: 1.23 (0.84, 1.80) 24 h: 1.28 (0.90, 1.84) **Notes:** OR of ventricular arrhythmia for an IQR increase of air pollutants in different subgroups (Fig 2)
Reference: Ljungman et al. **(2009, 191983)** **Period of Study:** May 2003-Jul 2004 **Location:** Athens, Greece Helsinki, Finland Ausburg, Germany Barcelona, Spain Rome, Italy Stokholm, Sweeden	**Outcome:** Interleukin-6 Response **Age Groups:** 35-80 yr **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:** 1 day	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **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 µg/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:** Jun-Dec 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 µg/m^3 increase in 24 h 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 µg/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 yr **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 to 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 to 3.87) DBP: 0.38 mmHg (95% CI: -1.43 to 2.20) H: 0.22 beats/min (95% CI: -0.71 to 1.16) Each increase in personal same day $PM_{2.5}$ was associated with: SBP: 0.37 mmHg (95% CI: -0.93 to 1.67) DBP: -0.20 mmHg (95% CI: -0.85 to 0.46) H: 0.44 beats/min (95% CI: 0.04 to 0.84) **Notes:** Results by health status presented in Fig 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:** Aug 1998-Dec 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)	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h (lagged ma 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)	**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)

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
	Statistical Analyses: linear regression **Covariates:** Apparent temperature, season, age, race, sex, glycosylated hemoglobin, cholesterol, smoking history, BMI **Dose-response Investigated?** No **Statistical Package:** NR	**Monitoring Stations:** 1 site **Copollutant:** $PM_{2.5}$ BC SO_4^{2-}	**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) **Subjects who report smoking in the past (but not within 6 mo)** 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 mo)** 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**

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			past (but not within 6 mo) 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** 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 ma 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 mo)** 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 mo)** 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)

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			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) 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 mo)** 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-Jan 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 ma 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 ma, similar patterns and quantitatively similar results appear in the other lags.

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
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:** SO_4^{2-} **Averaging Time:** 24 h (lagged ma 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 past (but not within 6 mo)** 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 mo)** 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	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 yr **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 yr 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:** $PM_{2.5}$ **Averaging Time:** 24 h (ma 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 ma. 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 ma. 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) 4-day ma. 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 ma. 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 ma. 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 ma. 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	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 yr **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 yr 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 (ma 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 ma. 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 ma. 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 ma. 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 ma. 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 ma 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 ma. 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 yr **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 yr 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 (ma 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 ma. 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 ma. 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 ma. 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 ma, 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 ma. 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 ma. 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-day 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:** Nov 2000-Oct 2003 **Location:** Greater Boston area, MA	**Outcome:** Change in HRV (SDNN, HF, LF, LFHFR) **Mean age:** 72.7 yr **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:** Nov 2000-Dec 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 2 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 1998-1999 **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:** PM1 **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 PM_1: 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 2 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 yr **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 yr 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:** SO_4^{2-} **Averaging Time:** 24 h (ma 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 ma: 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 ma: 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 ma: 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 ma: 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 ma; ' 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 ma 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 SO_4^{2-} among those with high and low levels of vitamins
Reference: Peters et al. (2005, 095747) Also Peters et al. (2005, 156859) **Period of Study:** Feb 1999-Jul 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 µg/m^3 **Effect Estimate:** Each 100 µg/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 1 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 µg/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 yr **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 µg/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:** Jul 1995-Jul 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 µg/m^3 24-h avg: Median = 9.8 µg/m^3 IQr = 7.8 **Copollutant:** O_3, BC, CO, NO_2, SO_2	**PM Increment:** 7.8 µg/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:** Jul 1995-Jul 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 µg/m^3 24-h avg: Median = 9.8 µg/m^3 IQr = 7.8 **Copollutant:** O_3, BC, CO, NO_2, SO_2	**PM Increment:** 9.4 µg/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:** Jul 1995-Jul 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: 0.91µg/m^3 **Copollutant:** O_3, $PM_{2.5}$, CO, NO_2, SO_2	**PM Increment:** 0.91µg/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-Dec 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 µg/m^3 (IQr = 9.7) **Copollutant:** NO_2, SO_2, CO, O_3, EC, OC	**PM Increment:** 9.7 µg/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:** Feb-Dec 2000 **Location:** Vancouver, British Columbia, Canada	**Outcome:** ICD discharges (as a proxy for VT/VF) **Age Groups:** 15-85 yr **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 µg/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 µg/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**: 24 h **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), endo-vthzelin-1, protein C, and interleukin-6 **Age Groups:** 23-30 yr **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 3 p.m. to 12 a.m. 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 PM2.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:** Dec 2001-Apr 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 min **Mean (SD), Range:** 46.8 µg/m^3 (SD = 1.82) **Range:** 0-483 µg/m^3 **Copollutant:** CO	**PM Increment:** 10 µg/m^3 **Effect Estimate:** Each 20 µg/m^3 increase in 5 min $PM_{2.5}$ 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 mo]) Each 10 µg/m^3 increase in 5-min 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 yr 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 µg/m^3 **Effect Estimate:** In the group receiving the fish oil supplement, each 8 µg/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 vs.. 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 yr **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 µg/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 yr **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 lags 2-3, 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-day 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-day 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-day 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:** 25th: 9.7 50th: 14.9 75th: 26.1 **Range (Min, Max):** 2.6, 83.7 **Monitoring Stations:** 1 site **Copollutant:** UFPs AP $PM_{2.5}$ PM_{10} OC EC NO_2 CO	**PM Increment:** IQR (16.4 5-day 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) 24-47 h: 1.5 (0.9, 2.5) 48-71 h: 1.2 (0.8, 1.9) 5-day mean: 1.4 (0.9, 2.3) **ICAM-1** Time before draw: 0-23 h: 0.7 (0.4, 0.9) 24-47 h: 1.3 (0.8, 1.8) 48-71 h: 1.8 (1.2, 2.7) 5-day 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: 0-23 h: 3.9 (-0.3, 8.1) 24-47 h: 3.1 (-1.6, 7.8) 48-71 h: 3.6 (-1.1, 8.3) 5-day mean: 5.6 (0.5, 10.8) **FVII** Time before draw: 0-23 h: -2.5 (-6.2 to 1.4) 24-47 h: -2.8 (-6.1 to 0.6) 48-71 h: -2.3 (-5.0 to 0.6) 5-day 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+ yr **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 AP $PM_{2.5}$ PM_{10} OC EC NO_2 CO	**PM Increment:** IQR (2.3 5-day 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: 0-23 h: 1.2 (0.7, 2.0) 24-47 h: 1.3 (0.7, 2.4) 48-71 h: 1.6 (0.9, 2.7) 5-day mean: 1.2 (0.7, 2.1) **ICAM-1** Time before draw: 0-23 h: 1.0 (0.7, 1.6) 24-47 h: 2.6 (1.7, 3.8) 48-71 h: 4.0 (2.5, 6.1) 5-day 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: 0-23 h: 5.0 (0.0, 10.1) 24-47 h: 7.6 (1.4, 13.7) 48-71 h: 1.1 (-5.2, 7.4) 5-day mean: 5.7 (-0.5, 12.0) **FVII** Time before draw: 0-23 h: -5.7 (-10.5 to -0.7) 24-47 h: -6.9 (-11.2 to -2.3) 48-71 h: -4.2 (-8.4, 0.2) 5-day 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+ yr **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 AP $PM_{2.5}$ PM_{10} OC EC NO_2 CO	**PM Increment:** IQR (0.7 5-day 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: 0-23 h: 1.2 (0.7, 1.9) 24-47 h: 1.3 (0.8, 2.1) 48-71 h: 1.4 (0.8, 2.4) 5-day mean: 1.2 (0.7, 1.8) **ICAM-1** Time before draw: 0-23 h: 0.9 (0.6, 1.3) 24-47 h: 2.0 (1.3, 3.2) 48-71 h: 3.0 (1.8, 4.8) 5-day 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: 0-23 h: 5.5 (0.2, 10.8) 24-47 h: 8.0 (2.1, 13.9) 48-71 h: 3.5 (-2.6, 9.6) 5-day mean: 7.4 (2.0, 12.8) **FVII** Time before draw: 0-23 h: -6.1 (-10.6 to -1.4) 24-47 h: -7.2 (-11.4 to -2.8) 48-71 h: -3.8 (-8.2, 0.9) 5-day 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+ yr **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 AP $PM_{2.5}$ PM_{10} NO	**PM Increment:** IQR (16.4 5-day 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-day mean: 0.2 (-5.4, 6.2) **Platelets, % change mean (103/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-day mean: -0.4 (-1.9, 1.2) **Leukocytes, % change in mean (103/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-day mean: -1.6 (-3.5, 0.3) **Erythrocytes, % change mean (106/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-day 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-day mean: -0.5 (-1.0, 0.1)
Reference: Sarnat et al. (2006, 090489) **Period of Study:** Summer and fall 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 and fall **Dose-response Investigated?** No	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 5 days **Median (IQR):** $PM_{2.5}$: Median: 19.0 µg/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 wk. 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 yr **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:** 0-4 days; 5-day 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 µg/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), 5-day 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), 5-day 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), 5-day 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), 5-day 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), 5-day ma *p < 0.05, ** p < 0.01 **Notes:** Percent change (95% CI) per 10 µg/m^3 $PM_{2.5}$ by GSTM1 genotype (Fig 3)
Reference: Schwartz et al. (2005, 074317) **Period of Study:** 12 wk during the summer of 1999 **Location:** Boston, MA	**Outcome:** Heart rate variability (HRV), (SDNN, r-MSSD, PNN50, LFHFR) **Age Groups:** 61-89 yr **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-h: 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	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Schwartz et al. (2005, 074317) **Period of Study:** 12 wk during the summer of 1999 **Location:** Boston, MA	**Outcome:** Heart rate variability (HRV), (SDNN, r-MSSD, PNN50, LFHFR) **Age Groups:** 61-89 yr **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:** 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: 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 yr **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):** Fall: 20.7 Summer: 12.6 **Percentiles:** IQR Fall: 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	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 (γ-glutamyl semialdehyde in hemoglobin), HBAAS (2-aminoadipic semialdehyde in hemoglobin), MDA (malondialdehyde) **Age Groups:** 20-33 yr **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:** Season, 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 106/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: 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 (γ-glutamyl semi-aldehyde in hemoglobin), HBAAS (2-aminoadipic semialdehyde in hemoglobin), MDA (malondialdehyde) **Age Groups:** 20-33 yr **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 109/g protein): 0.0003 (0.75) Hemoglobin (μmol/g protein): 0.0004 (0.65) Platelet count (x 106/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	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 (γ-glutamyl semialdehyde in hemoglobin), HBAAS (2-aminoadipic semialdehyde in hemoglobin), MDA (malondialdehyde) **Age Groups:** 20-33 yr **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 109/g protein): 0.0008 (0.36) Hemoglobin (μmol/g protein): 0.0005 (0.53) Platelet count (x 106/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: Sullivan et al. (2007, 100083) **Period of Study:** Feb 2000-Mar 2002 **Location:** Seattle, Washington, USA	**Outcome:** Blood CRP, fibrinogen, D-dimer **Age Groups:** >55 yr of age **Study Design:** Panel study **N:** 47 elderly subjects	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Median (IQR):** 7.7 μg/m^3 (6.4) **Monitoring Stations:** 1 **Copollutant:** Indoor $PM_{2.5}$	**PM Increment:** 10 μg/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	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Sullivan et al. (2005, 109418) **Period of Study:** Feb 2000-Mar 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 µg/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 min HRV measures were made in the homes of study subjects over a 10-day period.

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Sullivan et al. (2005, 109418) **Period of Study:** Feb 2000-Mar 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-α (TNF-8- α) Tumor necrosis factor-receptors (p55, p75) Monocyte chemoattractant protein-1 (MCP-1) **Age Groups:** ≥ 55 yr **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 **Statistical Package:** SAS v8.02	**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 μg/m^3 **Effect Estimate [Lower CI, Upper CI]:** Multiplicative change in mean outcome associated with 10 μg/m^3 increase in PM **Among those with different disease status.** **CRP Fold-rise (95%CI)** CV 0-day lag: 1.21 (0.86, 1.70) CV 1-day lag: 1.25 (0.97, 1.58); COPD 0-day lag: 0.93 (0.48, 1.80) COPD 1-day lag: 0.69 (0.33, 1.46) Healthy 0-day lag: 0.98 (0.88, 1.08) Healthy 1-day lag: 1.01 (0.84 1.21) **Fibrinogen Fold-rise (95%CI)** CV 0-day lag: 1.02 (0.98, 1.06) CV 1-day lag: 1.0 (0.97, 1.03); COPD 0-day lag: 1.0 (0.91, 1.09) COPD 1-day lag: 1.08 (0.99, 1.17) Healthy 0-day lag: 0.94 (0.87, 1.01) Healthy 1-day lag: 0.99 (0.88, 1.17) **D-dimer Fold-rise (95%CI)** CV 0-day lag: 1.02 (0.88, 1.17) CV 1-day lag: 1.03 (0.93, 1.15); COPD 0-day lag: 1.04 (0.93, 1.16) COPD 1-day lag: 1.09 (0.94, 1.27) Healthy 0-day lag: 0.95 (0.79, 1.14) Healthy 1-day lag: 0.97 (0.71, 1.31) **Among those with cardiovascular disease** **MCP-1 Fold-rise (95%CI)** 0-day lag: 1.3 (1.1, 1.7) 1-day lag: 1.0 (0.9, 1.3) **ET-1 Fold-rise (95%CI)** 0-day lag: 1.1 (0.8, 1.2) 1-day 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 μg/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 mo 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:** Apr-Aug 2002 **Location:** Mexico City metropolitan area	**Outcome:** Heart rate variability measures (SDNN, pNN50) **Age Groups:** Mean age 27 yr **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 µg/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 yr **Study Design: C**ross-sectional **N:** 3,827 participants **Statistical Analyses:** Linear Regression Models **Covariates: A**ge, 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):** Fig only **Monitoring Stations:** N/A Interpolation used **Copollutant:** NR **Co-pollutant Correlation:** N/A	**PM Increment:** 10 µg/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: Wellenius et al. (2007, 092830) **Period of Study:** Feb 2002-Mar 2003 **Location:** Boston, Massachusetts, USA	**Outcome:** Circulating levels of B-type natriuretic peptide (BNP Measured in whole blood at 0, 6, 12 wk) **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 µg/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	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Wellenius et al. (2007, 092830) **Period of Study:** Feb 2002-Mar 2003 **Location:** Boston, Massachusetts	**Outcome (ICD9 and ICD10):** B-type natriuretic peptide (BNP) (natural-log transformed) **Age Groups:** 33-88 yr **Study Design:** Panel (blood collected at 0, 6, and 12 wk) **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 µg/m^3 **Range (Min, Max):** 0.7-50.9 µg/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 µg/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 yr **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-h: 17.8 µg/m^3 **Copollutant:** O_3, CO, SO_2, NO_2	**PM Increment:** 11.65 µg/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 1-h 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) **Period of Study:** 12-wk period b/t Sep 2003-Jul 2004 **Location:** Chapel Hill, NC	**Outcome:** Heart Rate Variability **Age Groups:** 21-50 yr **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:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean (SD):** 12.5 (6.0) **Min:** 0.6 **Max:** 37.1 **Monitoring Stations:** 1 **Copollutant:** $PM_{10-2.5}$, PM_{10} **Co-pollutant Correlation:** $PM_{10-2.5}$ = 0.46* PM_{10} = NR *p < 0.01	**PM Increment:** 1 µg/m^3 **Beta, SE (Lower CI, Upper CI), p-value:** HRV Max Heart Rate: 0.40, 0.43 (-0.45, 1.24), 0.36 ASDNN5: -0.07, 0.15 (-0.37, 0.22), 0.63 SDANN5: 1.66, 0.65 (0.39, 2.93), 0.02 SDNN24HR(mesc): 1.16, 0.58 (0.02, 2.29), 0.06 rMSSD: 0.53, 0.20 (0.14, 0.91), 0.01 pNN50_24hr: -0.06, 0.11 (-0.27, 0.15), 0.58 pNN50_7min: 0.47, 0.42 (-0.35, 1.29), 0.27 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:** Oct 2000-Apr 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:** $PM_{2.5}$, 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: 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 yr **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	**PM Increment:** IQR **Effect Estimate [Lower CI, Upper CI]: QT interval, % change (95%CI)** **Factor 1:** 0-5 h: -0.1 (-0.6, 0.6) 6-11 h: -0.5 (-1.1, 0.2) 12-17 h: 0.1 (-0.4, 0.4) 18-23 h: -0.2 (-0.7, 0.2) 0-23 h: -0.2 (-0.9, 0.4) 1 day: -0.1 (-0.7, 0.6) 2 day: -0.3 (-0.9, 0.4) 3 day: -0.7 (-1.4, 0.1) 4 day: -0.2 (-0.9, 0.5) 0-4 day avg: -0.7 (-1.8, 0.3) **Factor 2:** 0-5 h: 0.2 (-0.4, 0.8) 6-11h: 0.8 (-0.0, 1.7) 12-17 h: 0.6 (-0.2, 1.4) 18-23 h: 0.5 (-0.4, 1.4) 0-23 h: 0.9 (-0.1, 2.0) 1 day: 1.5 (0.3, 2.7) 2 day: -0.4 (-1.7, 1.0) 3 day: 0.5 (-0.9, 1.9) 4 day: 0.1 (-1.2, 1.4) 0-4 day avg: 1.6 (-0.1, 3.3) **Factor 3:** 0-5 h: 0.1 (-0.3, 0.5) 6-11 h: 0.2 (-0.3, 0.6) 12-17 h: 0.2 (-0.3, 0.6) 18-23 h: 0.1 (-0.3, 0.4) 0-23 h: 0.1 (-0.3, 0.6) 1 day: 0.1 (-0.3, 0.4) 2 day: -0.1 (-0.4, 0.3) 3 day: -0.2 (-0.5, 0.2) 4 day: -0.1 (-0.5, 0.2) 0-4 day avg: -0.1 (-0.7, 0.6) **Factor 4:** 0-5 h: 0.2 (-0.4, 0.8) 6-11 h: 0.8 (0.0, 1.6) 12-17 h: 0.5 (-0.2, 1.3) 18-23 h: 0.5 (-0.2, 1.2) 0-23 h: 0.6 (-0.3, 1.4) 1 day: -0.4 (-1.5, 0.7) 2 day: -0.9 (-2.0, 0.1) 3 day: -0.5 (-1.4, 0.5)

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
		Copollutant: NA	4 day: -0.5 (-1.3, 0.2) 0-4 day avg: -0.3 (-1.7, 1.1) **Factor 5**: n0-5 h: 1.0 (-0.1, 2.1) 6-11 h: 0.9 (-0.2, 2.0) 12-17 h: 0.3 (-0.7, 1.4) 18-23 h: -0.1 (-1.2, 1.0) 0-23h: 0.7 (-0.6, 1.9) 1 day: 0.1 (-1.1, 1.3) 2 day: -0.2 (-1.5, 1.1) 3 day: -0.6 (-1.9, 0.8) 4 day: -0.9 (-2.0, 0.2) 0-4 day avg: -0.4 (-1.9, 1.2) **T wave amplitude, % change (95%CI)** **Factor 1**: 0-5 h: -0.3 (-1.5, 0.9) 6-11 h: -0.6 (-1.9, 0.7) 12-17 h: 0.1 (-0.8, 0.9) 18-23 h: -0.6 (-1.5, 0.4) 0-23 h: -0.5 (-1.8, 0.9) 1 day: 0.4 (-0.9, 1.7) 2 day: 1.2 (-0.3, 2.7) 3 day: 0.2 (-1.2, 1.7) 4 day: -0.2 (-1.3, 1.0) 0-4 day avg: 0.8 (-1.1, 2.6) **Factor 2**: 0-5 h: -1.7 (-3.0 to -0.4) 6-11 h: -2.6 (-4.5 to -0.6) 12-17 h: -1.0 (-2.6, 0.7) 18-23 h: -1.1 (-2.8, 0.7) 0-23 h: -3.1 (-5.3 to -0.9) 1 day: -0.3 (-2.9, 2.2) 2 day: -1.2 (-4.1, 1.7) 3 day: -0.5 (-3.2, 2.1) 4 day: -3.4 (-9.9, 3.1) 0-4 day avg: -1.5 (-4.4, 1.5) **Factor 3**: 0-5 h: -0.3 (-1.1, 0.6) 6-11 h: -0.1 (-0.9, 0.9) 12-17 h: 0.1 (-0.9, 1.0) 18-23 h: -0.4 (-1.2, 0.4) 0-23 h: -0.2 (-1.2, 0.7) 1 day: 0.1 (-0.7, 0.8) 2 day: -0.1 (-0.7, 0.7) 3 day: 0.4 (-0.3, 1.1) 4 day: 0.1 (-0.7, 0.7) 0-4 day avg: 0.3 (-0.9, 1.5) **Factor 4**: 0-5 h: -1.5 (-2.8 to -0.2) 6-11 h: -1.3 (-3.0, 0.3) 12-17 h: -1.1 (-2.7, 0.4) 18-23 h: -0.9 (-2.4, 0.6) 0-23 h: -1.6 (-3.3, 0.1) 1 day: -1.2 (-3.3, 0.9) 2 day: -1.0 (-3.2, 1.2) 3 day: 0.2 (-1.5, 1.9) 4 day: 0.5 (-1.0, 2.0) 0-4 day avg: -1.7 (-4.1, 0.7) **Factor 5**: 0-5 h: -1.6 (-3.6, 0.4) 6-11 h: -0.1 (-2.1, 2.0) 12-17 h: -0.2 (-2.2, 1.8) 18-23 h: -1.8 (-3.8, 0.2) 0-23 h: -1.2 (-3.4, 1.0) 1 day: -1.8 (-4.2, 0.6) 2 day: -0.7 (-3.5, 2.1) 3 day: 0.8 (-1.5, 3.2) 4 day: 0.5 (-1.5, 2.5) 0-4 day avg: -1.4 (-4.0, 1.2) **vWF, % change (95%CI)Factor 1:** 0-5 h: 1.1 (-1.5, 3.6) 6-11 h: 1.6 (-1.2, 4.5) 12-17 h: 0.4 (-1.4, 2.1) 18-23 h: 1.4 (-0.6, 3.5) 0-23 h: 1.6 (-1.3, 4.4) 1 day: -1.0 (-3.9, 1.9) 2 day: -1.8 (-4.8, 1.2)

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			3 day: -2.5 (-5.8, 0.9) 4 day: 0.5 (-2.9, 3.9) 0-4 day avg: -2.5 (-7.1, 2.2) **Factor 2**: 0-5 h: 0.4 (-2.4, 3.2) 6-11 h: -0.4 (-4.3, 3.4) 12-17 h: 2.1 (-1.4, 5.7) 18-23 h: 2.3 (-1.4, 5.9) 0-23 h: 1.9 (-2.8, 6.6) 1 day: 2.8 (-2.8, 8.3) 2 day: 5.1 (-0.8, 11.1) 3 day: 11.4 (5.3, 17.6) 4 day: 6.6 (0.0, 13.1) 0-4 day avg: 11.4 (3.7, 19.1) **Factor 3**: 0-5 h: 1.8 (0.1, 3.6) 6-11 h: 1.7 (-0.3, 3.7) 12-17 h: 2.2 (0.3, 4.2) 18-23 h: 2.8 (1.1, 4.5) 0-23 h: 2.8 (0.8, 4.8) 1 day: 2.7 (1.0, 4.4) 2 day: 3.4 (1.8, 5.0) 3 day: 2.3 (0.8, 3.8) 4 day: 1.4 (-0.2, 2.9) 0-4 day avg: 4.8 (2.0, 7.6) **Factor 4**: 0-5h: 1.5 (-1.4, 4.3) 6-11h: 2.0 (-1.7, 5.6) 12-17h: 2.6 (-0.8, 5.9) 18-23h: 3.5 (0.4, 6.6) 0-23h: 3.2 (-0.5, 7.0) 1 day: 5.4 (0.6, 10.2) 2 day: 4.5 (-0.6, 9.5) 3 day: 3.8 (-0.6, 8.1) 4 day: 3.0 (-0.6, 6.6) 0-4d avg: 11.3 (5.0, 17.6) **Factor 5**: 0-5 h: 1.9 (-2.8, 6.6) 6-11 h: 3.2 (-1.6, 8.0) 12-17 h: 2.4 (-2.3, 7.1) 18-23 h: 1.6 (-3.1, 6.2) 0-23 h: 2.9 (-2.5, 8.2) 1 day: -2.2 (-7.6, 3.2) 2 day: -1.3 (-7.4, 4.9) 3 day: 1.1 (-4.8, 7.1) 4 day: 1.3 (-4.2, 6.7) 0-4 day avg: 3.3 (-4.1, 10.6) **CRP, Odds Ratio (95%CI)** **Factor 1** 0-5 h: 0.9 (0.7, 1.1) 6-11 h: 1.4 (1.1, 1.8) 12-17 h: 1.2 (1.0, 1.4) 18-23 h: 1.0 (0.8, 1.3) 0-23 h: 1.1 (0.9, 1.5) 1 day: 1.4 (1.1, 1.8) 2 day: 1.3 (1.0, 1.7) 3 day: 1.0 (0.7, 1.4) 4 day: 1.1 (0.9, 1.5) 0-4 day avg: 1.6 (1.1, 2.2) **Factor 2** 0-5h: 0.8 (0.6, 1.0) 6-11h: 1.0 (0.7, 1.4) 12-17h: 1.1 (0.8, 1.5) 18-23h: 1.0 (0.8, 1.4) 0-23h: 0.9 (0.6, 1.4) 1 day: 0.9 (0.6, 1.5) 2 day: 2.1 (1.3, 3.3) 3 day: 1.9 (1.0, 3.6) 4 day: 1.4 (0.8, 2.3) 0-4d avg: 1.4 (0.8, 2.6) **Factor 3** 0-5 h: 1.0 (0.8, 1.1) 6-11 h: 0.9 (0.8, 1.1) 12-17 h: 1.0 (0.9, 1.2) 18-23 h: 1.0 (0.8, 1.2) 0-23 h: 1.0 (0.8, 1.2) 1 day: 1.1 (1.0, 1.3)

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			2 day: 1.0 (0.9, 1.2) 3 day: 1.2 (1.1, 1.4) 4 day: 1.1 (1.0, 1.3) 0-4 dY avg: 1.2 (1.0, 1.5) **Factor 4** 0-5 h: 0.8 (0.6, 1.1) 6-11 h: 0.8 (0.6, 1.1) 12-17 h: 1.3 (1.0, 1.8) 18-23 h: 1.1 (0.8, 1.5) 0-23 h: 1.0 (0.7, 1.4) 1 day: 1.5 (1.0, 2.3) 2 day: 2.0 (1.3, 3.2) 3 day: 1.5 (0.9, 2.3) 4 day: 1.3 (0.9, 1.8) 0-4 day avg: 1.7 (1.0, 2.9) **Factor 5** 0-5 h: 0.7 (0.5, 1.1) 6-11 h: 1.4 (0.9, 2.1) 12-17 h: 1.9 (1.3, 2.8) 18-23 h: 1.4 (1.0, 2.0) 0-23 h: 1.4 (0.9, 2.2) 1 day: 1.6 (1.0, 2.6) 2 day: 1.6 (0.9, 2.6) 3 day: 2.3 (1.3, 4.1) 4 day: 1.6 (0.9, 2.8) 0-4 day avg: 2.1 (1.2, 3.8)
Reference: Zanobetti et al. (2004, 087489) **Period of Study:** 1999-2001 **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 µg/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 µg/m^3 for 5-day mean, 13.9 µg/m^3 for 2-day mean **Effect Estimate:** Each 10.4 µg/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 µg/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 (SO_4^{2-}) measurements used to create 48-h, 1-wk, and 4-wk ma **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) SO_4^{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 yr) 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: 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:** SO_4^{2-} **Averaging Time:** Hourly (PN, BC, $PM_{2.5}$) and 24-h (SO_4^{2-}) measurements used to create 48-h, 1-wk, and 4-wk ma **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) SO_4^{2-}	**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	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 (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 (SO_4^{2-}) measurements used to create 48-h, 1-wk, and 4-wk ma **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) SO_4^{2-} (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:** U.S.	**Outcome:** Myocardial Ischemia **Age Groups:** 52-90 **Study Design:** Panel **N:** 55,529 **Statistical Analyses:** Logistic & Linear Regression **Covariates: A**ge, race/ethnicity, education, exam site, BMI, current smoking status, history of CHD, diabetes, hypertension, SBP, chronic lung disease, or hypercholesterolemia, day of week, time of day, temperature, dew point, pressure, season **Dose-response Investigated?** No **Statistical Package:** SAS **Lags Considered:** 0-5-day	**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) Lag 4: 13.9 (8) Lag 5: 14.1 (8) Lag 0-2: 13.9 (7) **Monitoring Stations:** NR‡ **Co-pollutant:** NR ‡ Monitors used in model for spatial interpolation of daily PM values.	**PM Increment:** 10 µg/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 MC 4 or 5: 1.04 (1.00, 1.09), lag 0-2 MC 4 or 5: 1.03 (0.99, 1.07), lag 3-5 **Change (Lower CI, Upper CI), lag:** ST-segment amplitude Lead I: -0.07 (-0.36, 0.21), lag 0-2 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)

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			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 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 μg/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:** 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 yr **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-min, 4-h, 24-h ma **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 ma r = -0.003 to 0.51 when comparing 5-min avg and 24-h ma. 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 ma 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 Fig 2 (magnitude of associations increased with averaging period, with the largest associations consistently found for 24-h ma)

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Adar et al. (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 yr **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 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-min, 4-h, and 24-h ma **Median (IQR):** All: 0.02 (0.11) Facility: 0.01 (0.04) 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 ma r = -0.003 to 0.51 when comparing 5-min avg and 24-h ma. 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 ma 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) 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 Fig 2 (magnitude of associations increased with averaging period, with the largest associations consistently found for 24-h ma)
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-α (TNF-α) 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 yr **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 wk of data during warm period and 6 wk of data during	**Pollutant:** PM (multiple size fractions and components) **Averaging Time:** 24-h avg preceding the blood draw (lag 0) and cumulative avg 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 OCpri: 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 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	**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, OCpri, BC, PN, CO, and nitrogen dioxide from the current-day and multiday avg. There were consistent positive but largely nonsignificant coefficients for TNF-α, 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, OCpri, 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)
	cool period **Dose-response Investigated?** No **Statistical Package:** NR	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 OCpri 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** PM0.25: Mean (SD): 9.47 (2.97) Median: 9.4 IQR: 4.2 Min-Max: 3.31, 18.75 PM0.25-2.5: Mean (SD): 13.53 (10.67) Median: 11.7 IQR: 11.5 Min-Max: 1.29, 66.77 $PM_{10-2.5}$: Mean (SD): 10.04 (4.07) Median: 9.9 IQR: 5.9 Min-Max: 1.76, 22.38 **Indoor PM mass** PM0.25: Mean (SD): 10.45 (6.77) Median: 9.5 IQR: 4.5 Min-Max: 1.42, 69.86 PM0.25-2.5 (µg/m^3): Mean (SD): 7.36 (4.57) Median: 6.5 IQR: 5.7 Min-Max: 0.77, 30.86 $PM_{10-2.5}$: 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 1998-1999 **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, $PM_{2.5}$, $PM_{10-2.5}$, PM_1, 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 2 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:** Feb 1999-Jul 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+ yr **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:** 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. µg/m^3):** n/cm^3 **Monitoring Stations:** 1 site **Copollutant:** UFPs AP $PM_{2.5}$ PM_{10} OC EC NO_2 CO	**PM Increment:** IQR (1299 5-day 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-day 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) 5-day 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-day 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-day 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	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:** Soluble CD40 ligand (sCD40L), platelets, leukocytes, erythrocytes, hemoglobin **Age Groups:** 50+ yr **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 AP $PM_{2.5}$ PM_{10} NO	**PM Increment:** IQR (1299 5-day 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-day mean: -1.3 (-9.9, 8.1) **Platelets, % change mean (103/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-day mean: -0.9 (-3.0, 1.3) **Leukocytes, % change in mean (103/µl)** lag0: -1.9 (-3.8 to -0.1) lag1: -0.6 (-2.9, 1.6) lag2: -0.6 (-3.2, 2.0) lag3: -2.3 (-4.6, 0.1) 5-day mean: -2.7 (-5.5, 0.1) **Erythrocytes, % change mean (106/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-day 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-day 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 yr **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-day 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-day 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-day avg (11003): -0.08 (-3.78, 3.75)

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Pekkanen et al. (2002, 035050) **Period of Study:** Winter 1998-1999 **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, $PM_{2.5}$, $PM_{10-2.5}$, PM_1, 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 2 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:** Feb 1999-Jul 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+ yr **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-day 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-day mean: -0.7 (-7.6, 6.8) **Platelets, % change mean (103/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-day mean: -2.2 (-4.0, -0.3) **Leukocytes, [103/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-day mean: -1.6 (-4.1, 0.8)

[1]All units expressed in µg/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, U.K.	**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 yr: NR 15-64 yr: 2.6 [0.3,5] 65-74 yr: 2.5 [0.1,4.9] 75+ yr: 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-Dec 2004 **Location:** Copenhagen, Denmark	**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 yr (CVD and RD), 5-18 yr (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):** 24(14) **Median:** 21 **IQR:** 16-28 **99th percentile):** 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 $\mu g/m^3$ (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 NCa212: 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 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 Fig form (see notes): **Notes:** Fig 2: Relative risks and 95% confidence intervals per IQR in single day concentration (0- to 5-day lag). Summary of Fig 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:** January 1999−December 2004 **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 yr **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 yr **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 yr, 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 ma and 7-day ma **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 µg/m^3 increase in 24-h ma 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 ma 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:** Dily 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 Fig 2.** Fig 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 2 pollutant models are expressed in Fig 3:** Fig 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 µg/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: Chan et al. (2007, 147787) **Period of Study:** Apr 1997-Dec 2002 **Location:** Boston, MA	**Outcome:** Cerebrovascular Emergency Admissions **Age Groups:** 50+ yr **Study Design:** Tme series **Statistical Analyses:** GAM Poisosn Regression **Covariates:** Yr, mo, day of wk, temperature, dew point **Dose-response Investigated?** No **Statistical Package:** NR **Lags Considered:** 0-3 days	**Pollutant:** PM_{10} Averaging Time: 24 h **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 µg/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	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
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:** Yr, mo, day of wk, 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 µg/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: 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:** 0-2 days	**Pollutant:** PM_{10} **Averaging Time:** 24 h **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 µg/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+ yr **Study Design:** Case-crossover **Statistical Analyses:** Conditional Logistic Regression **Covariates:** Temperature, humidity **Dose-response Investigated?** No **Statistical Package:** NR **Lags Considered:** 0-4 days	**Pollutant:** TSP **Averaging Time:** 24 h **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 0-2-day 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	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Fung et al., (2005, 093262) **Period of Study:** Nov 1995-Dec 2000 **Location:** London, Ontario	**Outcome (ICD-9):** Cardiovascular diseases (410-414, 427-428) **Age Groups:** <65 yr, 65+ yr **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 µg/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: Hanigan et al. (2008, 156518) **Period of Study:** 1996-2005 (Apr-Nov of each yr) **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, yrly population **Season:** Apr-Nov (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 µg/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 Fig (see notes). **Notes:** Fig 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	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Hanigan et al. (2008, 156518) **Period of Study:** 1996-2005 (Apr-Nov of each yr) **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:** 24 h **Mean (SD):** 21.2 (8.2) **Range:** 55.2 **Monitoring Stations:** 2 (monitored & modeled) **Copollutant:** NR **Co-pollutant Correlation:** N/A	**PM Increment:** 10 µg/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 *Fig 3. percent change in hospital admissions per 10 µg/m^3 increase in PM_{10}
Reference: Henrotin et al. (2007, 093270) **Period of Study:** Mar 1994-Dec 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 µg/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: Issever et al. (2005, 097736) **Period of Study:** Jan 1997-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	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Jalaludin et al. (2006, 189416) **Period of Study:** Jan 1997-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+ yr **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 µg/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: Johnston et al. (2007, 155882) **Period of Study:** 2000, 2004, 2005 (Apr-Nov of each yr) **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:** Apr-Nov (dry season) **Dose-response Investigated?** No **Statistical Package:** NR **Lags Considered:** 0-3	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Median:** 17.4 **IQR:** 13.6-22.3 **10-90th Percentile:** 10.3-27.7 **Range:** 1.1-70.0 **Monitoring Stations:** 1 **Copollutant:** NR	**PM Increment:** 10 µg/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:** **Fig 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. **Fig 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	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Koken et al. (2003, 049466) **Period of Study:** Jul and Aug, 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+ yr **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 µg/m^3 (IQR) **Percent Change Estimate [CI]:** No PM data reported
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+ yr, <75 yr, 75+ yr **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:** 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 µg/m^3 **Pooled Rate Ratio [CI]:** All 5 cities (35+ yr) 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+ yr) 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+ yr) 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+ yr) 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:** Dec 1997-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+ yr **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 yr 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 µg/m^3 (IQR) **RR Estimate [CI]: All yr** All ages: 0.99 [0.96,1.01] 64+ yr: 1.05 [1.01,1.10] **Summer** All ages: 1.03 [0.97,1.09] 64+ yr: 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:** 24 h **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 µg/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: 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 –to 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 µg/m^3 **ERR [95% CI]:** CVD: All ages: 0.7 [0.1, 1.2] 65+ yr: 1.1 [0.5, 1.7] Cardiac diseases: All ages: 0.8 [0.2, 1.4] 65+ yr: 1.5 [0.7, 2.2] Ischemic heart diseases: All ages: 1.9 [0.8, 3.0] 65+ yr: 2.9 [1.5, 4.3] Strokes: All ages: 0.2 [-1.6, 1.9] 65+ yr: 0.8 [-0.9, 2.5]

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
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 yr, 65+ yr **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 µg/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: 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 yr, >60 yr **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 µg/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:** Aug 1993-Aug 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 wk, 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 ma, 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 $PM_{2.5}$ OC: r = 0.69 $PM_{2.5}$ EC: r = 0.56 oxygenated hydrocarbon: r = 0.58 **Other variables:** Temperature: r = 0.58 Dew point: r = 0.44	**PM Increment:** 10 µg/m^3 (approximately 1 SD) **RR [95% CI]:** For 3-day ma: 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 Fig 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 yr) **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 µg/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 yr (Lag 0):** All admissions: 0.47 (-0.13, 1.08) **Aged >15 yr (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:** Jan 1993-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 avg: 9.1 (7.5) **Monitoring Stations:** 1 **Copollutant:** NR	**PM Increment:** 10 µg/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: 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 ma	**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 µg/m^3 **Percent increase in risk [95% CI]:** Results summarized in Fig (see notes). **Notes:** Fig 1: Percent increase in risk (and 95% CI) of acute coronary events associated with 10 µg/m^3 of PM_{10} for different lag structures. Summary of Fig 1: Positive, statistically significant or marginally significant associations between association seen for Lag 0, Lag 1 and 2-, 3-, and 4-day ma. Non-statistically significant associations

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
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+ yr **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:** 24 h **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 µg/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) Fig 1. PM_{10} effects, reported as percent increase, on arrhythmia ER visits caused by interquartile range increases, lags 0-6. Fig 2. Relative risks and 95% CI for arrhythmia ER visits according to the division of air pollutant daily concentrations in quintiles.
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 ma (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 µg/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 µg/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 >20ºC PIH: 1.54 [1.31,1.81] IS: 1.46 [1.32,1.61] Temp <20ºC 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:** Nov 1994-Mar 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 (Oct-Mar) vs.. warm months (Apr-Sep). **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 µg/m^3 , and 24.3 µg/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 µg/m^3 increase in PM10:** 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 µg/m3 increase in PM10 (single pollutant models, CIs not given):** All-age respiratory disease: 8.4 All-age RD/CVD: 7.9 18-64 yr 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:** 24 h **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 µg/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: 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:** 24 h **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 µg/m^3) vs.. Comparison (≤125 µg/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	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Villeneuve et al. (2006, 090191) **Period of Study:** Apr 1992-Mar 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+ yr **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 days	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (SD):** All yr: 24.2 (14.8) Summer: 25.9 (16.4) Winter: 22.6 (12.9) **Monitoring Stations:** 3 **Copollutant (correlation):** All yr 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 yr: 16.0 Summer: 17.5 Winter: 16.0 Adjusted OR Estimate [CI]: Acute ischemic stroke All yr 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 yr 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 yr Same-day lag: 0.96 [0.90,1.02] 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+ yr **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 µg/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: Wellenius et al. (2005, 087483) **Period of Study:** Jan 1987-Nov 1999 **Location:** Pittsburgh, Pennsylvania	**Outcome (ICD-9):** Congestive heart failure (428.0-428.1) **Age Groups:** 65+ yr **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 µg/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 µg/m^3 increment) 1.27 [0.66,1.88]

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Wellenius et al. (2005, 088685) **Period of Study:** Jan 1986-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+ yr **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 days	**Pollutant:** PM_{10} **Averaging Time:** 24 h Mean (SD): 32.69 (19.75) **Monitoring Stations:** NR (data obtained from the U.S. 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 µg/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:** Jan 1986-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+ yr **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 U.S. EPA) Copollutant: NR	**PM Increment:** 10 µg/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: 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 µg/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	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
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 µg/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: 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+ yr with diabetes, 65-74 and 75+ yr 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 µg/m^3 Percent Change [CI]: All 4 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 yr **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**: Fig 1 presents percent change in MI per lag day, showing same-day lag to be significant. Fig 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+ yr **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 days; 5-day (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, PM0.03-0.1, PM<0.1, PM<0.10.29, $PM_{2.5}$, CO, NO_2 **Co-pollutant Correlation** PM<0.03: 0.14 PM0.03-0.1: 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-day 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-day 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-day 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-day 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)(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 yr **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 µg/m3 (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 µg/m^3 , and an 18.8 µg/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 µg/m^3 increase): All ages: 0.5% [-1.2, 2.3] ≥ 65 yr: 1.0% [-1.0, 3.0] For all cardiovascular diseases (18 µg/m^3 increase): All ages: 1.0% [-2.3, 4.3] ≥ 65 yr: 1.9% [-2.0, 5.9] For cardiac diseases (10 µg/m^3 increase): All ages: 0.1% [-1.9, 2.1] ≥ 65 yr: 1.6% [-0.8, 4.1] For cardiac diseases (18.8 µg/m^3 increase): All ages: 0.1% [-3.6, 4.0] ≥ 65 yr: 3.1% [-1.5, 7.9] For ischemic heart diseases (10 µg/m^3 increase): All ages: 2.8% [-0.8, 6.6] ≥ 65 yr: 6.4% [1.6, 11.4] For ischemic heart diseases (18 µg/m^3 increase): All ages: 5.4% [-1.5, 12.8] ≥ 65 yr: 12.4 [3.1, 22.6]
Reference: Metzger et al. (2004, 044222) **Period of Study:** Aug 1998-Aug 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 wk, 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 ma; lags 0 -7	**Pollutant:** PM_{10} -2.5 **Averaging Time:** 24 h **Median µg/m3 (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 $PM_{2.5}$ OC: r = 0.51 $PM_{2.5}$ EC: 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 µg/m^3 (approximately 1 SD) RR [95% CI]: For 3 day ma: 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**: Fig 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 Fig 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:** Jan 1999-Dec 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 + yr, 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 wk, age-specific intercept, temperature, dew point temperature, calendar time, indicator for age of 75 yr 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 µg/m3 (IQR)**: All counties assessed: 9.8 (6.9-15.0) Counties in Eastern U.S.: 9.1 (6.6-13.1) Counties in Western U.S.: 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 µg/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 Fig 2-5. Fig 2: Percentage change in emergency hospital admissions for CVD per 10 µg/m^3 increase in PM (single pollutant model and model adjusted for $PM_{2.5}$ concentration) Fig 4: Percentage change in emergency hospital admissions rate for CVD and RD per a 10 µg/m^3 increase in $PM_{10-2.5}$ (0-2 day lags, Eastern vs.. Western USA) Fig 5: County-specific log relative risks of emergency hospital admissions for CVD per 10 µg/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:** Aug 1998-Dec 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 ma (lag 0-2)	**Pollutant:** $PM_{10-2.5}$ **Averaging Time:** 24 h **Mean (µg/m3) (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 = 0.38SO_2: 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 µg/m^3 (IQR) Risk ratio [95% CI]: CVD: 1.004 (0.990-1.019)

[1]All units expressed in µg/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-Dec 2004 **Location:** Copenhagen, Denmark	**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 pulmonary disease (J44), asthma (J45), and status asthmaticus (J46). Pediatric hospital admissions for asthma (J45) and status asthmaticus (J46). **Age Groups:** > 65 yr (CVD and RD), 5-18 yr (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 yr 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 µgm^3 (SD):** 10(5) **Median:** 9 **IQR:** 7-12 **99th percentile):** 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 NCa212: r = 0.82 PM_{10}: r = 0.80 CO: r = 0.46 NO_2: r = 0.42 NO_X: r = 0.40 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 Fig form (see notes): **Notes**: Fig 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: D**aily 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 µg/m3 (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 3 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 µg/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 Fig 2. Fig 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 2 pollutant models are expressed in Fig 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.
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 µg/m3 (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 µg/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 Fig 2. Fig 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 2 pollutant models are expressed in Fig 3: Fig 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/m3 (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 U.S. 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+ yr, population size **Season:** All, Jun-Aug (Summer), Sep-Nov (Fall), Dec-Feb (Winter), Mar-May (Spring) **Dose-response Investigated:** No **Statistical Package:** NR **Lags Considered:** 0- to 2-day lags	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean (µg/m3):** Descriptive information presented in Fig 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 [0.08-1.02] 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 (fall, national): 0.68 [0.29-1.07] Lag 0 (fall, northeast): 1.03 [0.48-1.58] Lag 0 (fall, southeast): 0.17 [-0.72-1.07] Lag 0 (fall, northwest): -0.67 [-6.96-6.05] Lag 0 (fall, 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 (fall): 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]

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			Lag 2 (summer): -0.12 [-0.50-0.26] Lag 2 (fall): 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] Lag 0 (summer, northwest): 0.25 [-21.46-27.96] Lag 0 (summer, southwest): 0.64 [-5.38-7.04] Lag 0 (fall, national): 0.02 [-0.63-0.67] Lag 0 (fall, northeast): -0.01 [-0.87-0.85] Lag 0 (fall, southeast): -0.58 [-2.06-0.91] Lag 0 (fall, northwest): -1.38 [-11.84-10.32] Lag 0 (fall, 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 (fall): 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 (fall, national): 0.39 [-0.22-1.01] Lag 2 (fall, northeast): 0.12 [-0.82-1.07] Lag 2 (fall, southeast): 0.14 [-1.29-1.59]

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			Lag 2 (fall, northwest): -0.74 [-10.08-9.58] Lag 2 (fall, southwest): 0.97[-1.36-3.36]
Reference: Bell et al. (2009, 191007) **Period of Study:** 1999-2005 **Location:** 168 U.S. Counties	**Outcome:** CVD hospital admissions **Study Design:** Retrospective Cohort **Covariates:** Socio-economic conditions, long term temperature **Statistical Analysis:** Bayesian hierarchical model **Age Groups:** ≥ 65 yr	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **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 to 4.2) Summer health effect: -148 (-327 to 31.1) Winter health effect: -80.0 (-182 to 22.0) Central AC Yearly health effect: -42.5(-63.4-21.6) Summer health effect: -79.5 (-143 to 15.7) Winter health effect: -41.9 (-124 to 40.0)
Reference: Bell et al. (2009, 191997) **Period of Study:** 1999-2005 **Location:** U.S.	**Outcome:** Cardiovascular 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, NH4+, 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 in PM Health Effect (Lower CI, Upper CI), lag** EC: 25.8 (4.4, 47.2), lag 0 EC + Ni: 14.0 (-7.6, 35.5), lag 0 EC + V: 14.9 (-7.8, 37.6), lag 0 EC+ V, HS education: 15.0 (3.3, 26.8), lag 0 EC+ V, median income: 15.8 (4.1, 27.5), lag 0 EC+ V, racial composition: 14.2 (2.8, 25.6), lag 0 EC+ V, percent living in urban area: 14.7 (3.1, 26.3), lag 0 EC+ V, population: 13.6 (2.2, 25.0), lag 0 EC + Ni, V: 11.9 (-10.4, 43.2), lag 0 Ni: 19.0 (9.9, 28.2), lag 0 Ni + EC: 17.3 (7.7, 26.9), lag 0 Ni + V: 15.5 (4.1, 26.9), lag 0 Ni + EC, V: 14.9 (3.4, 26.4), lag 0 V: 27.5 (10.6, 44.4), lag 0 V + EC: 23.1 (4.9, 41.4), lag 0 V+ Ni: 10.9 (-9.6, 31.5), lag 0 V + EC, Ni: 8.1 (-13.3, 29.5), lag 0 EC: 11.8 (-69.2, 92.8), lag 1 EC: 21.0 (-46.6, 88.6), lag 2 Ni: 20.6 (-15.5, 56.7), lag 1 Ni: -2.3 (-32.5, 27.9), lag 2 V: 34.0 (-31.2, 99.1), lag 1 V: 8.0 (-46.8, 62.7), lag 2 Percent HS education: -17.4 (-46.8, 11.9), lag 0 Median income: 21.3 (-20.0, 62.5), lag 0 Percent black: 26.9 (-15.8, 69.6), lag 0 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+ yr **Study Design:** Time series **Statistical Analyses:** GAM Poisosn Regression **Covariates:** Yr, mo, day of wk, temperature, dew point **Dose-response Investigated?** No **Statistical Package:** NR **Lags Considered:** 0-3 days	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **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 µg/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:** Yr, mo, day of wk, 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 µg/m3 (SD):** NR **Monitoring Stations:** 1 **Copollutant (correlation):** NR	**PM Increment:** 19.7 µg/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:** October 2001−2003−November 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) µg/m^3 During Fires: 54.1 (21.0) µg/m^3 After Fires: 15.9 (5.5) µg/m^3 Orange Before Fires: 23.2 (9.6) µg/m^3 During Fires: 64.3 (26.5) µg/m^3 After Fires: 15.5 (10.2) µg/m^3 Riverside Before Fires: 32.7 (14.7) µg/m^3 During Fires: 42.1 (25.5) µg/m^3 After Fires: 16.9 (10.2) µg/m^3 San Bernadino Before Fires: 35.7 (16.6) µg/m^3 During Fires: 45.3 (28.7) µg/m^3 After Fires: 18.5 (8.3) µg/m^3 San Diego Before Fires: 18.5 (6.7) µg/m^3	**Increment:** 10 µg/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 Post-Wildfire: 0.991 (0.964-1.019), p = 0.955 Ischaemic Heart Disease All Periods: 0.991 (0.980-1.003) Pre-Wildfire: 0.990 (0.963-1.017) Wildfire: 0.117 (0.990-1.024), p = 0.313 Post-Wildfire: 0.989 (0.950-1.030), p = 0.976 Congestive Heart Failure All Periods: 0.989 (0.974-1.004) Pre-Wildfire: 0.978 (0.942-1.015) Wildfire: 1.016 (0.933-1.039), p = 0.096 Post-Wildfire: 0.969 (0.914-1.027), p = 0.791

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
		During Fires: 76.1 (66.6) µg/m^3 After Fires: 14.2 (7.2) µg/m^3 Ventura Before Fires: 18.4 (8.3) µg/m^3 During Fires: 50.1 (50.5) µg/m^3 After Fires: 12.9 (4.3) µg/m^3 **Copollutant (correlation):** NR	Cardiac Dysrhythmia All Periods: 0.980 (0.962-0.998) Pre-Wildfire: 0.979 (0.935-1.025) Wildfire: 0.989 (0.961-1.017), p = 0.721 Post-Wildfire: 0.976 (0.912-1.044), p = 0.934 Cerebrovascular Disease and Stroke All Periods: 1.019 (1.004-1.035) Pre-Wildfire: 1.015 (0.980-1.052) Wildfire: 1.016 (0.997-1.036), p = 0.971 Post-Wildfire: 1.044 (0.987-1.104), p = 0.379 **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 (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 U.S. 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,	**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 yr **Study Design:** Time series N: 11.5 million Medicare enrollees **Statistical Analyses:** Bayesian 2-stage	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h Mean (µg/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 µg/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]

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Utah, Virginia, Washington, West Virginia, Wisconsin	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		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 μg/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] 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**: Fig 2: Point estimates and 95% posterior intervals of the % change in admissions rates per 10 μg/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

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			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. Fig 3: Point estimates and 95% posterior intervals of the % change in admission rates per 10 μg/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. Fig 4: Point estimates and 95% posterior intervals of the % change in admission per 10 μg/m^3 (Eastern vs.. Western regions): Summary: All estimates for cardiovascular outcomes were positive in the U.S. Eastern region but not in the U.S. 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+ yr **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 days; 5-day (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, PM0.03-0.1, PM<0.1, PM<0.10.29, $PM_{10-2.5}$, CO, NO_2 **Co-pollutant Correlation** PM<0.03: 0.14 PM0.03-0.1: 0.48 PM<0.1: 0.35 PM<0.10.29: 0.88 $PM_{10-2.5}$: 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-day 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-day 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-day 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-day 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 yr. For all respiratory diseases: 0-14 yr, 15-64 yr, and ≥ 65 yr. 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 wk, 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 µg/m^3 increase, and a 27 µg/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 µg/m^3 increase): All ages: 0.9% [0.1, 1.8] ≥ 65 yr: 1.9% [0.9, 3.0] For all cardiovascular diseases (27 µg/m^3 increase): All ages: 2.5% [0.2, 4.9] ≥ 65 yr: 5.3% [2.6, 8.2] For ischemic heart diseases (27 µg/m^3 increase): All ages: 5.2% [-0.6, 11.3] ≥ 65 yr: 12.7% [6.3, 19.5] For cardiac diseases (10 µg/m^3 increase): All ages: 0.9% [-0.1, 2.0] ≥ 65 yr: 2.4% [1.2, 3.7] For cardiac diseases (27 µg/m^3 increase): All ages: 2.5% [-0.3, 5.4] ≥ 65 yr: 6.8% [3.3, 10.3] For ischemic heart diseases (10 µg/m^3 increase): All ages: 1.9 % [-0.2, 4.0] ≥ 65 yr: 4.5% [2.3, 6.8] For all respiratory diseases (10 µg/m^3 increase): 0-14 yr: 0.4% [-1.2, 2.0] 15-64 yr: 0.8% [-0.7, 2.3]; ≥ 65 yr: 0.5% [-2.0, 3.0] For all respiratory diseases (27 µg/m^3 increase): 0-14 yr: 1.1% [-3.1, 5.5] 15-64 yr: 2.2% [-1.8, 6.4]; ≥ 65 yr: 1.3% [-5.3, 8.2] For respiratory infections (10 µg/m^3 increase): All ages: 2.5% [0.1, 4.8] For respiratory infections (27 µg/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:** Jan 1997-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+ yr **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 µg/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).
Reference: Jalaludin et al. (2006, 189416) **Period of Study:** Jan 1997-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+ yr **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:** BS,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/ 104/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).

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
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+ yr **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: Jun-Sep; Non-summer: Oct-May) **Dose-response Investigated:** No **Statistical Package:** S-plus 7.0 **Lags Considered:** Lags 0-5 days, and avg lag effect (0-5 days)	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Median µg/m3 (IQR):** 7.0 (4.8-10.0) **Monitoring Stations:** 6 **Copollutant (correlation):** NR	**PM Increment:** 5.1 µg/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**: Fig 3: % change in stroke/TIA risk associated with an IQR increase in $PM_{2.5}$
Reference: Metzger et al. (2004, 044222) **Period of Study:** Aug 1998-Aug 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 wk, 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 ma, lags 0 -7	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Median µg/m3 (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) $PM_{2.5}$ OC: 0.010 (-0.001-0.045) $PM_{2.5}$ EC: 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 $PM_{2.5}$ OC: r = 0.51 $PM_{2.5}$ EC: 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 µg/m^3 $PM_{2.5}$ water-sol metals: 0.03 µg/m^3 $PM_{2.5}$ sulfates: 5 µg/m^3 $PM_{2.5}$ acidity: 0.02 µequ/m^3 $PM_{2.5}$ OC: 2 µg/m^3 $PM_{2.5}$ EC: 1 µg/m^3 RR [95% CI]: $PM_{2.5}$ (3-day ma): 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 ma):** 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 ma):** 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 ma):**

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			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] **$PM_{2.5}$ OC (3-day ma)**: 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. **$PM_{2.5}$ OC (3-day ma):** 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 disease: 1.021 [0.994-1.049] Results for Lags 0-7 expressed in figures (see notes). **Notes**: Fig 1: RR (95% CI) for single-day lag models for the association of ER visits for CVD with daily ambient $PM_{2.5}$ and associated components. Summary of Fig 1 results: Statistically significant positive associations at Lag 0 and Lag 1 for $PM_{2.5}$, at Lag 0 for $PM_{2.5}$ water soluble metals (inverse association at Lag 7), at Lag 0, Lag 1, and Lag 3 for organic and EC (inverse association at Lag 7). Fig 2: RR (95%) of multipollutant models for the association of ER visits for CVD with daily ambient air quality measurements. Summary of Fig 2 results: Positive association after adjustment for NO_2, CO, and oxygenated hydrocarbons, but attenuated when adjusted for total carbon and null when adjusted for NO_2, CO, total carbon, and oxygenated

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Peng et al. (2008, 156850) **Period of Study:** Jan 1999-Dec 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 + yr, 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 yr 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 µg/m3 (IQR):** All counties assessed: 13.5 (11.1-15.8) Counties in Eastern U.S.: 13.8 (12.3-15.8) Counties in Western U.S.: 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 µg/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 Fig 2-5. Fig 2: Percentage change in emergency hospital admissions for CVD per 10 µg/m^3 increase in $PM_{2.5}$ (single pollutant model and model adjusted for $PM_{10-2.5}$ concentration) Fig 3: Percentage change in emergency hospital admissions for RD per 10 µg/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:** Feb 1999-Jul 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 ma	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 1 h and 24 h **Mean µg/m3 (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: TNC: r = 0.42 CO: r = 0.52 NO_2: r = 0.58 NO: r = 0.50 SO_2: r = 0.48 O_3: r = -0.35 Other variables: 24-h avg: Temperature: r = 0.05 1-h avg: Temperature: r = -0.01	**PM Increment:** 1-h avg: 9.1 µg/m^3 (IQR) 24-h avg: 7.7 µg/m^3 (IQR) OR [95% CI]: Case-Crossover (control selection method (unidirectional with three control periods): **1-h avg:** 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 avg:** 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 avg:** 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) Lag 5: 0.93 (0.85, 1.02) Lag 0 -4 (IQR = 5.8): 1.03 (0.94, 1.14) Unidirectional: Model 1 (unadjusted): 1.175 (1.033, 1.337) Model 2 (adjusted for day of week using indicator variables): 1.179 (1.035,

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			1.343) Model 3 (adjusted for temperature-quadratic, linear air pressure): 1.170 (1.028, 1.333) Model 4 (adjusted for temperature-quadratic, linear air pressure, day of week): 1.176 (1.031, 1.341) Model 5 (temperature-quadratic, air pressure-quadratic, relative humidity-quadratic, day of week using indicator variables): 1.170 (1.026, 1.336) Model 6 (temperature-penalized spline, 4.4 df, linear air pressure, day of week using indicator variables): 1.175 (1.030, 1.340 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 (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) 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

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			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) 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)

Study	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, <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 ma	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean (µg/m3) (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 µg/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**: Fig 1: Percent increase in risk (and 95% CI) of acute coronary events associated with 10 µg/m^3 of $PM_{2.5}$ for different lag structures. Summary of Fig 1: Positive, statistically significant association seen for Lag 0, Lag 1 and 2, 3, and 4 day ma. Positive but non-statistically significant associations seen for Lags 2 and 3. Fig 2: Percent increase in risk (and 95% CI) of acute coronary events associated with 10 µg/m^3 of $PM_{2.5}$ stratified by various characteristics.
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- to 28-day ma.	**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:** 10 µg/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 yr: 3.5 (-13.5, 23.8) Age ≥65 yr: 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 yr: -3.1 (-26.5, 27.8) Age ≥65 yr: 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:** Nov 1998-Dec 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 (Apr 15-Oct 14), and cool season (Oct 15-Apr 14). **Dose-response Investigated:** No **Statistical Package:** NR **Lags Considered:** 0-day lag	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean (µg/m3) (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) $PM_{2.5}$ EC: 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] $PM_{2.5}$ EC: 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] $PM_{2.5}$ OC: 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] $PM_{2.5}$ OC: 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] $PM_{2.5}$ OC: 1.027 [1.004-1.051]

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Schreuder et al. (2006, 097959) **Period of Study:** Sep 1995-May 2002 **Location:** Spokane, WA	**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:** 0-1 day	**Pollutant:** $PM_{2.5}$ (ng/m^3) **Averaging Time:** 24 h **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)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
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: Nov-Feb nonheating season: Mar-Oct) **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 (µg/m3) (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 µg/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 yr: 1.04 [0.95, 1.14] 50-60 yr: 0.99 [0.94, 1.05] 70+ yr: 1.03 [0.98, 1.08] Age (24-h avg): <50 yr: 1.07 [0.98, 1.19] 50-69 yr: 0.99 [0.93, 1.06] 70+ yr: 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] 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 h & 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 µg/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
Reference: Tolbert et al. (2007, 090316) **Period of Study:** Aug 1998-Dec 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 ma(lag 0 -2)	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean (µg/m3) (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). $PM_{2.5}$ OC: 4.4 (3.8 2.7-5.3 0.4-25.9 2.1-7.2). $PM_{2.5}$ EC: 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 Increment:** $PM_{2.5}$: 10.96 µg/m^3 (IQR) $PM_{2.5}$ sulfate: 3.82 µg/m^3 (IQR) $PM_{2.5}$ total carbon: 3.63 µg/m^3 (IQR) $PM_{2.5}$ OC: 2.61 µg/m^3 (IQR) $PM_{2.5}$ EC: 1.15 µg/m^3 (IQR) $PM_{2.5}$ water-soluble metals: 0.03 µg/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] $PM_{2.5}$ OC: CVD: 1.015 [1.005-1.026] $PM_{2.5}$ EC: 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 Fig 1. Fig 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)
		$PM_{2.5}$ TC: r = 0.34 $PM_{2.5}$ water-sol metals: r = 0.65 OHC: r = 0.47 Between $PM_{2.5}$ EC and: PM_{10}: r = 0.61 O_3: r = 0.40 NO_2: r = 0.64 CO: r = 0.66 SO_2: r = 0.22 $PM_{10-2.5}$: r = 0.49 $PM_{2.5}$: r = 0.65$PM_{2.5}$ SO_4: r = 0.32 $PM_{2.5}$ OC: r = 0.82 $PM_{2.5}$ TC: r = 0.91 $PM_{2.5}$ water-sol metals: r = 0.52 OHC: r = 0.35 Between $PM_{2.5}$ OC and: PM_{10}: r = 0.65 O_3: r = 0.54 NO_2: r = 0.62 CO: r = 0.59 SO_2: r = 0.17 $PM_{10-2.5}$: r = 0.49 $PM_{2.5}$: r = 0.70 $PM_{2.5}$ SO_4: r = 0.33 $PM_{2.5}$ EC: r = 0.82 $PM_{2.5}$ TC: r = 0.98 $PM_{2.5}$ water-sol metals: r = 0.49 OHC: r = 0.37 Between $PM_{2.5}$ total carbon and: PM_{10}: r = 0.67 O_3: r = 0.52 NO_2: r = 0.65 CO: r = 0.63 SO_2: r = 0.19 $PM_{10-2.5}$: r = 0.51 $PM_{2.5}$: r = 0.71 $PM_{2.5}$ SO_4: r = 0.34 $PM_{2.5}$ EC: r = 0.91 $PM_{2.5}$ OC: r = 0.98 $PM_{2.5}$ water-sol metals: r = 0.52 OHC: r = 0.38 Between $PM_{2.5}$ water-soluble metals and: PM_{10}: r = 0.73 O_3: r = 0.43 NO_2: r = 0.32 CO: r = 0.35 SO_2: r = 0.06 $PM_{10-2.5}$: r = 0.50 $PM_{2.5}$: r = 0.69 $PM_{2.5}$ SO_4: r = 0.65 $PM_{2.5}$ EC: r = 0.52 $PM_{2.5}$ OC: r = 0.49 $PM_{2.5}$ TC: r = 0.52	

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Villeneuve et al. (2006, 090191) **Period of Study:** Apr 1992-Mar 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+ yr **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 days	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean µg/m3 (SD):** All yr: 8.5 (6.2) Summer: 8.7 (7.1) Winter: 8.3 (5.2) **Monitoring Stations:** 3 **Copollutant (correlation):** All yr SO_2: r = 0.22 NO_2: r = 0.41 CO: r = 0.43 O_3-mean: r = -0.07 O_3-max: r = 0.07 PM_{10}: r = 0.79 Summer SO_2: r = 0.20 NO_2: r = 0.52 CO: r = 0.42 O_3-mean: r = 0.11 O_3-max: r = 0.34 PM_{10}: r = 0.85 Winter SO_2: r = 0.28 NO_2: r = 0.57 CO: r = 0.71 O_3-mean: r = -0.45 O_3-max: r = -0.35 PM_{10}: r = 0.70	**PM Increment:** µg/m^3 (IQR) All yr: 6.3 Summer: 6.5 Winter: 6.0 Adjusted OR Estimate [CI]: Acute ischemic stroke All yr: Same-day lag: 1.00 [0.96,1.04] 1-day lag: 1.00 [0.96,1.05] 3-day lag: 1.01 [0.96,1.06] Summer: Same-day lag: 0.96 [0.90,1.03] 1-day lag: 1.01 [0.94,1.07] 3-day lag: 0.98 [0.89 [1.07] Winter: Same-day lag: 1.04 [0.99,1.10] 1-day lag: 1.01 [0.96,1.07] 3-day lag: 1.05 [0.98,1.13] Hemorrhagic stroke All yr: Same-day lag: 0.99 [0.90,1.08] 1-day lag: 1.07 [0.98,1.16] 3-day lag: 1.05 [0.93,1.19] Summer: Same-day lag: 0.99 [0.86,1.15] 1-day lag: 1.12 [0.97,1.30] 3-day lag: 1.08 [0.88,1.31] Winter: Same-day lag: 1.04 [0.92,1.18] 1-day lag: 1.08 [0.97,1.20] 3-day lag: 1.11 [0.94,1.31] Transient cerebral ischemic attack All yr: Same-day lag: 0.98 [0.93,1.03] 1-day lag: 0.99 [0.95,1.04] 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.
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+ yr **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 (Apr-Sep) 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 (µg/m3) (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 µg/m^3 Myocardial infarction cohort (Lag 0-1): 16.32 µg/m^3 Pneumonia cohort (Lag 0): 17.14 µg/m^3 Pneumonia cohort (Lag 0): 16.32 µg/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 Fig 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.

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 + yr **Study Design:** Case-crossover **N:** 15,578 patients admitted for MI and 25,857 admitted for pneumonia **Statistical Analyses:** Conditional logistic regression **Covariates: T**emperature, day of the week. **Season:** All, and also assessed for interaction by hot (Apr-Sep) 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 (µg/m3) (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 µg/m^3 Myocardial infarction cohort (Lag 0-1): 1.69 µg/m^3 Pneumonia cohort (Lag 0): 2.05 µg/m^3 Pneumonia cohort (Lag 0 -1): 1.69 µg/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 Fig 2. Summary of results: PM_2.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 µg/m^3 unless otherwise specified.

Table E-8. Short-term exposure-cardiovascular-ED/HA-other size fractions.

Study	Design & Methods	Concentrations1	Effect Estimates (95% CI)
Reference: Andersen et al, (2008, 189651) **Period of Study:** May 2001-Dec 2004 **Location:** Copenhagen, Denmark	**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 pulmonary disease (J44), asthma (J45), and status asthmaticus (J46). Pediatric hospital admissions for asthma (J45) and status asthmaticus (J46). **Age Groups:** >65 yr (CVD and RD), 5-18 yr (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 yr olds), pollen (only for pediatric asthma outcome) **Season:** NR	**Pollutant:** Total number concentration of ultrafine and accumulation mode particles (NCtot) (particles/cm^3) **Averaging Time:** 24 h **NCtotal** **Mean (SD):** 8,116 (3502) **Median:** 7,358 **IQR:** 5,738-9,645 **99th Percentile:** 19,895 **NCa12** **Mean (SD):** 493 (315) **Median:** 463 **IQR:** 308-650 **99th Percentile:** 1,263 **NCa23** **Mean (SD):** 2,253 (1,364) **Median:** 2,057 **IQR:** 1,280-3,066 **99th Percentile:** 6,096 **NCa57** **Mean (SD):** 5,104 (2,687) **Median:** 4,562 **IQR:** 3,248-6,274 **99th Percentile:** 14,410 **NCa100** **Mean (SD):** 6,847 (2,846) **Median:** 6,243 **IQR:** 4,959-8,218 **99th Percentile:** 16,189 **NCa212**	**PM Increment:** IQR increase in pollutant level: Nctot: 3907 particles/cm^3 (IQR) N_{ca12}: 342 particles/cm^3 (IQR) N_{ca23}: 1786 particles/cm^3 (IQR) N_{ca57}: 3026 particles/cm^3 (IQR) N_{C100}: 3259 particles/cm^3 (IQR) N_{ca212}: 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]

Study	Design & Methods	Concentrations1	Effect Estimates (95% CI)
	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.	**Mean (SD):** 392 (441) **Median:** 246 **IQR:** 89-584 **99th Percentile:** 2,248 *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 : r = 0.66 NC_{100}: r = 0.98 NC_{a12}: r = 0.31 NC_{a23}: r = 0.57 NC_{a57}: 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	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 [1.00-1.08] 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**: Fig 2: Relative risks and 95% confidence intervals per IQR in single day concentration (0-5 day lag). Summary of Fig 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	Concentrations1	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+ yr, <75 yr, 75+ yr **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/cm3:** 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+ yr) 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+ yr) 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+ yr) 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+ yr) 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.
Reference: Metzger et al. (2004, 044222) **Period of Study:** Aug 1998-Aug 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 ma, 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; $PM_{2.5}$ OC: r = 0.08; $PM_{2.5}$ EC: 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 ma: 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**: Fig 1: RR (95% CI) for single-day lag models for the association of ER visits for CVD with daily ambient UFP. Summary of Fig 1 results: Null or negative associations.

Study	Design & Methods	Concentrations1	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+ yr **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/cm3 (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 =-0.32 Barcelona 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 µg/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	Concentrations1	Effect Estimates (95% CI)
Reference: Aekplakorn, et al. (2003, 089908) **Period of Study**: 107 days, from Oct 1997-Jan 1998 **Location**: Mae Mo district, Lampang Province, North Thailand	**Outcome**: Upper respiratory symptoms, lower respiratory symptoms, cough **Age Groups**: 6-14 yr 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 µg/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	Concentrations1	Effect Estimates (95% CI)
Reference: Andersen et al. (2008, 189651) **Period of Study:** Dec 1998-Dec 2004 **Location:** Copenhagen, Denmark	**Outcome:** Daily symptoms (prospective daily recording of symptoms via diary) **Age Groups:** 0-3 yr **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 yr of life) born between Aug 2, 1998 and Dec 12, 2001 **Statistical Analyses:** Logistic regression model (GEE) **Covariates: T**emperature, 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 µg/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): 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)

Study	Design & Methods	Concentrations1	Effect Estimates (95% CI)
Reference: Boezen et al. (2005, 087396) **Period of Study:** Two consecutive winters (winter 1993-winter 1995) **Location: R**ural (Meppel, Nunspeet) and urban (Amsterdam) areas in the Netherlands	**Outcome:** FEV_1, airway hyperresponsiveness (AHR), serum total IgE and daily data on lower respiratory symptoms (LRS), upper respiratory symptoms (URS), cough and morning and evening peak expiratory flow **Age Groups:** 50-70 yr **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	**Pollutant:** PM_{10} **Averaging Time:** 24 h **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 BS	**PM Increment:** 10 µg/m^3 Effect Estimate [Lower CI, Upper CI]: AHR-/IgE- 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) 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) **Periods of Study:** 3 Winters (1992-1995) **Location:** Urban and rural areas of the Netherlands	**Outcome:** Respiratory symptoms Lower respiratory symptoms (wheeze, attacks of wheezing, shortness of breath) Upper respiratory symptoms (sore throat, runny or blocked nose)	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** Winter 1992-93 Urban: 54.8 Rural: 44.7 Winter 1993-94	**Increment:** 100 µg/m^3 **Odds Ratio (Lower CI, Upper CI) lag:** OR for respiratory symptoms and exposure to PM_{10} in children with BHR and high serum total IgE Lower Respiratory Symptoms 1.32 (1.07, 1.63) 0

Study	Design & Methods	Concentrations1	Effect Estimates (95% CI)
	Bronchial hyperresponsiveness (BHR) **Study Design:** Time-series **Statistical Analyses:** Logistic regression (PROC model) **Age Groups:** 7-11	Urban: 41.5 3 Rural: 44.1 Winter 1994-95 Urban: 31.1 Rural: 26.6 **Range (Min, Max):** Winter 1992-93 Urban: (4.7, 145.6) Rural: (4.8, 103.8) Winter 1993-94 Urban: (12.1, 112.7) Rural: (7.9, 242.2) Winter 1994-95 Urban: (8.8, 89.9) Rural: (7.1, 96.9) **Copollutants:** BS SO_2 NO_2	1.36 (1.13, 1.64) 1 1.36 (1.13, 1.65) 2 2.39 (1.71, 3.35) 0-5 avg. Upper Respiratory Symptoms 1.13 (0.97, 1.32) 0 1.00 (0.87, 1.16) 1 0.96 (0.84, 1.11) 2 0.91 (0.70, 1.18) 0-5 avg >10% morning peak expiratory flow (PEF) decrease 1.10 (0.92, 1.33) 0 1.08 (0.90, 1.28) 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 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) 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

Study	Design & Methods	Concentrations1	Effect Estimates (95% CI)
			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 **Statistical Analyses:** Frequencies **Covariates:** Meteorologic data (i.e. temperature, wind direction, wind speed, and humidity) **Dose-response Investigated?** No	**Pollutant:** PM_{10} **Averaging Time:** 8 h **Mean (SD):** North Kolkata: 535.9 Central Kolkata: 1114.5 South Kolkata: 909.2 **Monitoring Stations:** 1 **Copollutant:** PM<10-3.3 PM<3.3-0.4	**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.: 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	Concentrations1	Effect Estimates (95% CI)
Reference: Dales et al. (2006, 090744) **Period of Study:** Jan 1986-Dec 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 µg/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 Amsterdam, from Nov 1998 to Jun 1999 Erfurt, from Oct 1998 to Apr 1999 Helsinki, from Nov 1998 to Apr 1999 **Location:** Amsterdam, the Netherlands; Erfurt, Germany; Helsinki, Finland	**Outcome:** Respiratory symptoms **Age Groups:** ≥ 50 yr **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: 36.5 Erfurt: 27.1 Helsinki: 19.6 **Range (Min, Max):** Amsterdam: (13.6-112.0) Erfurt: (5.2-104.2) Helsinki: (6.4-67.4) **Monitoring Stations:** 1 **Copollutant:** $PM_{2.5}$ $N_{C0.01-0.1}$ 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.'
Reference: Delfino et al. (1998, 051406) **Period of Study:** Aug-Oct 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 µg/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	Concentrations1	Effect Estimates (95% CI)
Reference: Delfino et al. (2002, 093740) **Period of Study:** Mar-Apr 1996 **Location:** Alpine, California (a semi-rural area)	**Outcome:** Asthma symptoms that interfere with daily activities **Age Groups:** 9-19 yr **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 Mar-Apr **Dose-response Investigated?** Yes **Statistical Package:** SAS, version 8 **Lags Considered:** 0-, 1-, 2-, 3-, 4-, 5-, and 3-day ma	**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 ma 1 h max PM_{10}: 11.1 (1.10-112) 3-day ma 8 h max PM_{10}: 10.1 (1.42-72.0) 3-day ma 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 ma 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 ma 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 ma 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 Fig 2 and not quantitatively reported elsewhere.
Reference: Delfino et al. (2003, 090941) **Period of Study:** Nov 1999-Jan 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

Study	Design & Methods	Concentrations1	Effect Estimates (95% CI)
Reference: Delfino et al. (2004, 056897) **Period of Study:** Sep-Oct 1999 Apr-Jun 2000 **Location:** Alpine, California	**Outcome:** FEV_1 **Age Groups:** 9-19 yr 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:** Spring, 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 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	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	Concentrations1	Effect Estimates (95% CI)
Reference: Delfino et al. (2006, 090745) **Period of Study:** Region 1: Aug to Mid Dec 2003. Region 2: Jul through Nov 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 ma	**Pollutant:** PM_{10} Central Site **Averaging Time:** 24 h Riverside **Mean (SD):** 70.82 (29.36) 50th(Median): 65.96 **Range (Min, Max):** (30.75,54.05) $\mu g/m^3$ 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 ma 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**: Fig of Estimated lag effect of hourly personal $PM_{2.5}$ on FENO. Fig of the Estimated lag effect of hourly personal $PM_{2.5}$ on FENO by use of medications. Fig of one- and two-pollutant models for change in FENO using 2-day Ma personal and central-site pollutant measurements.

Study	Design & Methods	Concentrations1	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 µg/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:** Sep 2001–Dec 2003 **Location:** East Baltimore, MD	**Outcome:** Asthma in the last 12 mo (493.x) **Age Groups:** 2 to 6 yr 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 h **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	Concentrations1	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 yr mean age = 74 yr **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 PM10: r = 0.71	**PM Increment:** Ambient PM_{10}: 7 (IQR) Exposure to ambient PM_{10}: 6.5 (IQR) **Notes**: Effect estimates are presented in Fig 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 wk (dates not specified) **Location:** The 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 µg/m^3 IQ (25th, 75th): (21, 187) µg/m^3 **Copollutants:** BS NO_2 CO NO	**Increment:** 10 µg/m^3 % Increase in eNO and PM_{10} and change in spirometric lung function lag eNO and PM10 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
Reference: Forsberg et al. (1998, 051714) **Period of Study:** Jan 1994-March 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 µg/m3 Rural: 11.5 µg/m3 **Range (Min, Max):** Urban: (0, 40.5) µg/m3 Rural: (1.6, 29.0) µg/m3 Copollutants (correlation): BS: r = 0.73	**Increment:** 10 µg/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) 2 1.041 (0.742, 1.459) 0-6 avg Upper respiratory symptoms: 1.093 (0.989, 1.208) 0 1.018 (0.918, 1.130) 1

Study	Design & Methods	Concentrations1	Effect Estimates (95% CI)
			1.075 (0.962, 1.201) 2 1.052 (0.786, 1.407) 0-6 avg Lower respiratory symptoms: 1.022 (0.855, 1.180) 0 0.998 (0.855, 1.164) 1 1.000 (0.830, 1.206) 2 0.939 (0.703, 1.253) 0-6 avg
			OR between incidence of acute respiratory symptoms and PM_{10} exposure in urban and rural children lag
			Urban Children: Cough: 1.114 (0.886, 1.401) 0 0.891 (0.703, 1.130) 1 0.766 (0.577, 1.017) 2 0.817 (0.523, 1.276) 0-6 avg Phlegm: 0.954 (0.664, 1.371) 0 1.056 (0.744, 1.501) 1 1.416 (0.969, 2.069) 2 0.808 (0.357, 1.827) 0-6 avg Upper respiratory symptoms: 1.155 (0.965, 1.383) 0 0.788 (0.629, 0.986) 1 0.886 (0.728, 1.077) 2 0.770 (0.549, 1.081) 0-6 avg Lower respiratory symptoms: 1.060 (0.828, 1.356) 0 0.763 (0.584, 0.996) 1 0.652 (0.493, 0.863) 2 0.519 (0.306, 0.882) 0-6 avg Rural Children: Cough: 1.052 (0.767, 1.444) 0 0.753 (0.547, 1.038) 1 0.840 (0.571, 1.235) 2 0.800 (0.409, 1.565) 0-6 avg Phlegm: 1.051 (0.731, 1.509) 0 1.010 (0.693, 1.472) 1 0.998 (0.652, 1.528) 2 0.797 (0.344, 1.847) 0-6 avg Upper respiratory symptoms: 1.044 (0.813, 1.341) 0 0.810 (0.612, 1.072) 1 0.800 (0.611, 1.048) 2 0.714 (0.417, 1.220) 0-6 avg Lower respiratory symptoms: 1.079 (0.756, 1.539) 0 0.888 (0.615, 1.281) 1 0.715 (0.472, 1.083) 2 0.822 (0.395, 1.711) 0-6 avg
			OR between prevalence of medication use and PM10 exposure in urban and rural children lag
			Bronchodilator use - Urban children: 0.998 (0.951, 1.048) 0 0.999 (0.952, 1.049) 1 1.006 (0.953, 1.062) 2 0.919 (0.775, 1.090) 0-6 avg Rural children: 0.970 (0.904, 1.040) 0 0.959 (0.893, 1.030) 1 1.008 (0.927, 1.095) 2 1.087 (0.914, 1.292) 0-6 avg
			OR between incidence of medication use and PM_{10} exposure in urban and rural children lag
			Bronchodilator use - Urban children: 1.498 (0.899, 2.498) 0 1.049 (0.565, 1.947) 1 1.148 (0.674, 1.954) 2 1.787 (0.611, 5.227) 0-6 avg Rural children: 1.275 (0.702, 2.315) 0 0.924 (0.437, 1.956) 1

Study	Design & Methods	Concentrations1	Effect Estimates (95% CI)
			1.005 (0.522, 1.936) 2 1.823 (0.534, 6.277) 0-6 avg
Reference: Goncalves et al. (2005, 089884) **Period of Study:** Dec 1992-Mar 1993. Dec 1992-Mar 1994 **Location:** Sao Paulo	**Outcome:** Respiratory morbidity/admissions **Age Groups:** Children <13 yr **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	Concentrations1	Effect Estimates (95% CI)
Reference: Harre et al. (1997, 095726) **Period of Study:** Jun 994-Aug 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 µg/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 (U.S. military camps)	**Outcome:** Weekly rates of upper respiratory disease (URD), reported by the medical treatment facility in each military camp **Age Groups:** U.S. 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 1 in each of the 5 camps	**PM max Quartiles (combining all camps):** Q1: <58.7 µg/m^3 Q2: 60.1 to <75.54 µg/m^3 Q3: 78.56 to <107.56 µg/m^3 Q4: >107.56 µg/m^3 For dichotomous analysis cutoff = 74.55 µg/m^3 PM avg Quartiles (combining all camps): Q1: <42.19 µg/m3 Q2: 42.19 to 64.17 µg/m3 Q3: 64.17 to 81.75 µg/m3 Q4: >81.75 µg/m3 For dichotomous analysis cutoff = 64.17 µg/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] 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

Study	Design & Methods	Concentrations1	Effect Estimates (95% CI)
			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 percentile 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"

Study	Design & Methods	Concentrations1	Effect Estimates (95% CI)
Reference: Hong et al. (2007, 091347) **Period of Study:** Mar 23-May 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 yr) **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	**Pollutant:** PM_{10} **Averaging Time:** 24 h **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) µg/m^3 Median = 0.112 **Range (Min, Max):** (0.061-0.806) Mn: mean = 0.008 (0.005) µg/m^3 Median = 0.007 **Range (Min, Max):** (0.000-0.019) Pb: mean = 0.051 (0.031) µg/m^3 Median = 0.051 **Range (Min, Max):** (0.011-0.155) Zn: mean = 0.021 (0.021) µg/m^3 Median = 0.013 **Range (Min, Max):** (0.006-0.112) Al: mean = 0.085 (0.100) µg/m^3 Median = 0.031 **Range (Min, Max):** (0.017-0.344) Copollutant: $PM_{2.5}$	**Effect Estimate:** Regression coefficients of morning and daily mean PEFR on PM_{10} and metal components using linear mixed-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 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	Concentrations1	Effect Estimates (95% CI)
Reference: Hwang et al. (2006, 088971) **Period of Study:** 2001 **Location:** Taiwan	**Outcome:** Allergic rhinitis **Study Design:** Cross-sectional **Statistical Analyses:** Two-stage hierarchical models **Age Groups:** 6-15 yr	**Pollutant:** PM_{10} **Averaging Time:** 1-h avg **Mean (SD):** 55.58 (16.57) **Range (Min, Max):** (29.36, 99.58) **Copollutants (correlation):** CO: r = 0.27 NO_X: r = 0.34 O_3: r = 0.28 SO_2: r = 0.58	**Increment:** 10 µg/m^3 **Odds Ratio (Lower CI, Upper CI)** lag: PM_{10} alone: 1.00 (0.99, 1.02) , PM_{10}: 0.99 (0.97, 1.00) CO, PM_{10}: 1.00 (0.99, 1.01) O_3, PM_{10}: 1.00 (0.99, 1.02) Gender Male: 1.02 (0.99, 1.04) 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 yr: 1.05 (0.96, 1.14) 6-8 yr: 1.03 (0.98, 1.07) 9-11 yr: 1.00 (0.98, 1.03) 12+ yr: 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) **Period of Study:** Feb 1994-Dec 1994 **Location:** Western and southwestern Sydney, Australia	**Outcome:** Respiratory symptoms, Wheeze, Dry cough, Wet cough **Study Design:** Longitudinal study panel **Statistical Analyses:** Logistic regression model (GEE) **Age Groups:** 9-11 yr	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** 22.8 (13.8) **IQ Range (25th,75th):** (12.00, 122.8) **Copollutants (correlation):** O_3: r = 0.13 NO_2: r = 0.26	**Increment:** 10 µg/m^3 **Odds Ratio (Lower CI, Upper CI)** **Lag** Wheeze 1.01 (0.99, 1.03) 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) 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 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 PM10 exposure by different groups**

Study	Design & Methods	Concentrations1	Effect Estimates (95% CI)
			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 mo, positive histamine challenge, and doctor diagnosed asthma. **Group 2 consists of children with a history of wheeze in the past 12 mo and doctor diagnosed asthma. ***Group 3 consists of children only with a history y of wheeze in the past 12 mo.

Study	Design & Methods	Concentrations1	Effect Estimates (95% CI)
Reference: Jansen, et al. (2005, 082236) **Period of Study:** 1987-2000 **Location:** Seattle, WA	**Outcome:** FENO: fractional exhaled nitrogen oxide, Spirometry, Blood pressure, SaO_2: oxygen saturation, Pulse rate **Age Groups:** 60-86 yr 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.

Study	Design & Methods	Concentrations1	Effect Estimates (95% CI)
Reference: Johnston, et al. (2006, 091386) **Period of Study:** 7 mo (Apr 7-Nov 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 **Lags Considered:** 0-5 days	**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 µg/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 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	Concentrations1	Effect Estimates (95% CI)
Reference: Just et al. (2002, 035429) **Period of Study:**Apr 1996-Jun 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 yr 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 μg/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) 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 ($p = 0.19$).

Study	Design & Methods	Concentrations1	Effect Estimates (95% CI)
Reference: Kulkarni et al. (2006, 089257) **Period of Study:** Nov 2002-Dec 2003 **Location:** Leicester, United Kingdom	**Outcome:** Lung function by spirometry: FVC, FEV_1, FEV_1: FVC, FEF_{25-75} **Age Groups:** 8-15 yr **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 R2 = 0.06 Single pollutant model: FVC: -1.2 [-5.6, 3.2] p = 0.59 R2 = 0.005 Single pollutant model: FEF_{25-75}: -8.6 [-17.3, 0.1] p = 0.05 R2 = 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
Reference: Kuo, et al. (2002, 036310) **Period of Study:** 1-yr period (yr not specified) **Location:** Central Taiwan	**Outcome:** Asthma (yes/no) **Age Groups:** 13-16 yr **Study Design:** Cohort **N:** 12,926 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 avg 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 $\mu g/m^3$ ≥ 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) ≥ 65.9 $\mu g/m^3$: 0.837 [NR] Adjusted (outcome = asthma, yes/no) <65.9 $\mu g/m^3$: 1 (ref) ≥ 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:** May 1999-Jun 1999 Jan 1999-Dec 1999 **Location:** Rome, Italy	**Outcome:** Lung function of subjects (FVC and FEV_1) with COPD, Asthma **Age Groups:** COPD: 50 to 80 yr Asthma: 18 to 64 yr **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	Concentrations1	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 yr (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 µg/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 yr **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:** Winter 2001 (Feb 10-23), spring 2001 (May 5-18), summer 2001 (Jul 14-27), fall 2001 (Sep 22-Oct 5), winter 2002 (Jan 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 wk **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 µg/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	Concentrations1	Effect Estimates (95% CI)
Reference: Mar et al. (2004, 057309) **Period of Study:** 1997-1999 **Location:** Spokane, Washington	**Outcome:** Respiratory symptoms **Age Groups:** Adults: Ages 20-51 yr Children: Ages 7-12 yr **Study Design:** Time-series **N:** 25 people **Statistical Analyses:** Logistic regression **Covariates:** Temperature, relative humidity, day of-the-wk **Statistical Package:** STATA 6 **Lags Considered:** 0-2 days	**Pollutant:** PM_{10} **Mean (SD):** 1997: 24.5 (18.5) 1998: 20.6 (12.3) 1999: 16.8 (8.0) **Monitoring Stations:** 1 station **Copollutant (correlation):** PM_{10} PM1: r = 0.48 $PM_{2.5}$: r = 0.61 $PM_{10-2.5}$: r = 0.93	**PM Increment:** 10 µg/m^3 **OR Estimate [Lower CI, Upper CI]** **lag:** **Adult Respiratory symptoms:** Wheeze: 1.01[0.93, 1.09] lag 0 0.98[0.91, 1.06] lag 1 0.99[0.92, 1.06] lag 2 Breath: 1.02[0.96, 1.08] lag 0 1.01[0.97, 1.06] lag 1 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 **Children Respiratory symptoms**: Wheeze: 0.92[0.71, 1.18] lag 0 0.89[0.64, 1.24] lag 1 0.95[0.69, 1.31] lag 2 Breath: 1.04[0.95, 1.15] lag 0 1.04[0.95, 1.15] lag 1 1.06[0.95, 1.19] lag 2 Cough: 1.09[1.02, 1.16] lag 0 1.08[1.02, 1.14] lag 1 1.10[1.02, 1.18] lag 2 Sputum: 1.08[0.98, 1.17] lag 0 1.07[0.98, 1.17] lag 1 1.07[0.98, 1.16] lag 2 Runny Nose: 1.08[1.00, 1.16] lag 0 1.08[1.02, 1.15] lag 1 1.08[1.02, 1.14] lag 2 Eye Irritation: 1.06[0.74, 1.51] lag 0 0.94[0.70, 1.26] lag 1 0.99[0.88, 1.12]lag 2 Lower Symptoms: 1.07[1.00, 1.14] lag 0 1.06[0.98, 1.15] lag 1 1.07[0.95, 1.19] lag 2 Any Symptoms: 1.07[1.02, 1.11] lag 0 1.09[1.03, 1.15] lag 1 1.10[1.03, 1.17] lag 2

Study	Design & Methods	Concentrations1	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 µg/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 PM10 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
Reference: McCormack et al. (2009, 199833) **Period of Study:** Sep 2001-Apr 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 yr	**Pollutant:** $PM_{10-2.5}$, $PM_{2.5}$ **Averaging Time:** 3 days **Mean (SD) Unit:** $PM_{10-2.5}$: 17.4 ± 21.2 µg/m^3 $PM_{2.5}$: 40.3 ± 35.4 µg/m^3 **Range (Min, Max):** NR **Copollutant (correlation):** NR	**Increment:** 10 µg/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	Concentrations1	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 yr): 0.0121 (0.0037) FEV_1**:** PM_{10} (age 0-3 yr): 0.0102 (0.0034) **PEF:** PM_{10} (Mother smoked during pregnancy): -0.0102 (0.0039)
Reference: Mortimer et al. (2002, 030281) **Period of Study:** Jun-Aug 1993 **Location:** Eight urban areas of the U.S.: 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 yr **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 O_3: r = 0.51	**PM Increment:** 20 µg/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) O_3+PM_{10}: OR = 1.25 (0.97-1.61) O_3+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, MEF_{50}) of elementary school children and on asthma-like symptoms of children who had been classified as sensitive."
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 µg/m^3 **% change in Lung Function per 10 µg/m3** 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

Study	Design & Methods	Concentrations1	Effect Estimates (95% CI)
Reference: Neuberger et al. (2004, 093249) **Period of Study:** Sep 1999-Mar 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 yr (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
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 yr **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 µg/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:** Apr 2003-Mar 2004 **Location:** Fukuoka, Japan	**Outcome:** 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 yr old	**Pollutant:** PM_{10} **Averaging Time:** 3 h **Mean (SD) Unit:** Warmer months, 5-8 am SPM: 40.7 µg/m^3 NO_2: 15.2 ppb O_3: 17.7 ppb Warmer months, 7-10pm SPM: 41.5 µg/m^3 NO_2: 20.0 ppb O_3: 28.1 ppb Colder months, 5-8am SPM: 32.6 µg/m^3 NO_2: 20.5 ppb O_3: 17.5 ppb Colder months, 7-10pm SPM: 34.7 µg/m^3 NO_2: 28.0 ppb O_3: 19.4 ppb **Range (Min, Max):**	**Increment:** 10 µg/m^3 **Relative Risk (Min CI, Max CI)** **Lag** Apr-Sep, 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, -

Study	Design & Methods	Concentrations1	Effect Estimates (95% CI)
		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) Colder months, 7-10pm SPM: (10.3, 131.0) NO_2: (3.6, 49.0) O_3: (1.0, 60.0) **Copollutant (correlation):** Warmer months (24-h mean): O_3: r = 0.32 NO_2: r = 0.30 Colder months (24-h mean): O_3: r = -0.02 NO_2: r = 0.45	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, 1pm-4pm: -1.086 (-0.888, 0.516) O_3, 10am-1pm: -0.315 (-1.123, 0.493) NO_2, 7pm-10pm: 0.296 (-0.806, 1.397) NO_2, 4pm-7pm: 0.220 (-0.818, 1.258) NO_2, 1pm-4pm: 0.438 (-0.568, 1.444) NO_2, 10am-1pm: 0.536 (-0.546, 1.617) Single-pollutant model: SPM, 7pm-10pm: -0.449 (-0.956, 0.058) SPM, 4pm-7pm: -0.449 (-1.029, 0.131) SPM, 1pm-4pm: -0.414 (-0.943, 0.115) SPM, 10am-1pm: -0.486 (-1.051, 0.079) Oct-Mar, 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, 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	Concentrations1	Effect Estimates (95% CI)
Reference: Peacock et al. (2003, 042026) **Period of Study:** Nov 1996-Feb 1997 **Location:** Southern England	**Outcome:** Reduced peak expiratory flow rate (PEFR) **Age Groups:** 7-13 yr **Study Design:** Time-series **N:** 179 **Statistical Analyses:** GEE, multiple regression **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, 5-day ma	**Pollutant:** PM_{10} **Averaging Time:** Daily **Mean (SD):** Rural (nationally validated) 21.2 (11.3) Rural (locally validated) 18.7 (11.3) Urban 1 18.4 (9.8) Urban 2 22.7 (10.6) **Percentiles:** 10th Rural (nationally validated) 11.0 Rural (locally validated) 9.0 Urban 1 10.5 Urban 2 12.5 90th Rural (nationally validated) 33.0 Rural (locally validated) 32.5 Urban 1 32.0 Urban 2 36.0 **Range (Min, Max):** Rural (nationally validated) 7.0, 82.0 Rural (locally validated) 6.6, 87.9 Urban 1 4.7, 62.8 Urban 2 6.7, 63.7 **Monitoring Stations:** 3 **Copollutants:** NO_2 O_3 SO_2 SO_4^{2-}	**Increment:** 10 µg/m^3 **Odds Ratio (Lower CI, Upper CI)** **Lag** **Change in PEFR** Community -0.04 (-0.11, 0.03) 0 0.03 (-0.04, 0.05) 1 -0.01 (-0.07, 0.05) 2 -0.10 (-0.25, 0.05) 0-4 avg Local -0.01 (-0.06, 0.03) 0 0.04 (0.01, 0.08) 1 0.01 (-0.04, 0.05) 2 0.04 (-0.05, 0.13) 0-4 avg **20% decrease in PEFR** All children 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
Reference: Peled, et al. (2005, 156015) **Period of Study:** 5-6 wk between Mar-Jun 1999 and Sep-Dec 1999. **Location:** Ashdod, Ashkelon and Sderot, Israel	**Outcome:** Reduced peak expiratory flow (PEF) **Age Groups:** 7-10 yr **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, fall **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 µg/m^3 β **coefficient (SE) [95% CI]** Sderot: PM_{10} MAX: -0.34 (0.41) [-1.16, 0.46] PM_{10} MAX x sin(ω2 day): 0.84 (0.22) [0.405, 1.28] PM_{10} MAX x cos (ω1 day): -1.61 (0.41) [-2.43, 0.79] PM_{10} MAX x sin (ω1 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 (Jul 1998-Jun 2000) **Location:** City of Rouen, France	**Outcome:** Respiratory drug sales **Age Groups:** 0-14, 15-64, 65-74, over 75 yr **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 µg/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 yr of age (Lower CI, Upper CI) lag: 9.2 (5.9, 12.6) 10 days

Study	Design & Methods	Concentrations1	Effect Estimates (95% CI)
Reference: Preutthipan et al. (2004, 055598) **Period of Study:** 31 days (school days) from Jan−Feb 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 µg/m^3 Low = <120 µg/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	Concentrations1	Effect Estimates (95% CI)
Reference: Rabinovitch et al. (2004, 096753) **Periods of Study:** Nov 1999-Mar 2000 Nov 2000-Mar 2001 Nov 2001-Mar 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 µg/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, 199834) **Period of Study:** Jun 2006-Jul 2006 **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 yr	**Pollutant:** PM_{10} **Averaging Time:** Daily **Mean (SD) Unit:** Urban: 56.9 ± 13.1 µg/m^3 Rural: 13.8 ± 5.6 µg/m^3 **Copollutant (correlation):** NR	All results are presented in Fig format.

Study	Design & Methods	Concentrations1	Effect Estimates (95% CI)
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 yr 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 **Statistical Package:** SAS **Lags Considered:** 0-1 days	**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) 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	**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] 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] FEF25-75%: -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 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.

Study	Design & Methods	Concentrations1	Effect Estimates (95% CI)
Reference: Sahsuvaroglu et al. (2009, 190983) **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-yr avg **Avg:** All Subjects: 20.90 µg/m^3 Boys: 20.88 µg/m^3 Girls: 20.92 µg/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 PM10Spline and NO2LUR** All Girls: 1.063 (0.969-1.666) Older Girls: 1.058 (0.918-1.219) **Odds Ratio (95%CI) for copollutant model PM10Spline and NO2LUR, SO2Theissen and O3Theissen** 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 PM10Spline 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 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:

Study	Design & Methods	Concentrations1	Effect Estimates (95% CI)
			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) 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)

Study	Design & Methods	Concentrations1	Effect Estimates (95% CI)
Reference: Schildcrout et al. (2006, 089812) **Period of Study:** Nov 1993-Sep 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:** 5-12 yr **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 avg 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 CO r = 0.24-0.88	**PM Increment:** 25 µg/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** 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] 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 µg/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) **Period of Study:** 107 days, Oct 1997-Jan 1998 **Location:** Mae Mo district, Lampang Province, north Thailand	**Outcome:** Upper respiratory symptoms, lower respiratory symptoms, cough **Age Groups**: 6-14 yr **Study Design:** Logistic regression **N:** 98 asthmatic school children **Statistical Analyses:** Generalized Estimating Equations, stratified analysis, PROC GENMOD **Covariates:** Temperature and relative humidity **Season**: Winter **Dose-response Investigated?** No **Statistical Package**: SAS v 8.1	**Pollutant:** $PM_{10-2.5}$ **Averaging Time:** Daily **Mean (SD):** NR **Range (Min, Max):** NR **Monitoring Stations**: 3 **Copollutant**: PM_{10}, SO_2	**PM Increment:** 10 µg/m^3 **Odds Ratios [Lower CI, Upper CI] lag:** Asthmatics: URS: 1.04 (0.93, 1.17) lag 0 LRS: 1.09 (0.95, 1.26) lag 0 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

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Bourotte et al. (2007, 150040) **Period of Study:** May 2002-Jul 2002 **Location:** Sao Paulo, Brazil	**Outcome:** Peak expiratory flow (PEF) **Age Groups**: Avg age 39.8 ± 12.3 yr **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:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean (SD):** 21.7 (12.9) µg/m^3 **Range (Min, Max):** (4.13, 62.0) **Components:** Na^+ K^+ $Mg2^+$ $Ca2^+$ Finf Cl- NO_3^- SO_4^{2-} **Monitoring Stations**: 1	**PM Increment:** NR **Effect [Lower CI, Upper CI] lag:** Morning PEF Na^+ concurrent day = -0.454 (-1.605, 0.697) Na^+ 2-day lag = -0.907 (-2.288, 0.474) Na^+ 3-day lag = -1.361 (-2.972, 0.251) K^+ concurrent day = 1.685 (-0.492, 3.862) K^+ 2-day lag = 1.838 (-1.272, 4.984) K^+ 3-day lag = 2.604 (-0.812, 6.025) Mg_2^+ concurrent day = 2.265* (-0.427, 4.956) Mg_2^+ 2-day lag = 1.271 (-1.869, 4.410) Mg_2^+ 3-day lag = 0.939 (-2.425, 4.303) 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) $NO_{3_2}^-$ 3-day lag = 7.341* (3.083, 11.60) $SO_{4_2}^{2-}$ concurrent day = 3.528 (-0.053, 7.110) $SO_{4_2}^{2-}$ 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) $NO_{3_2}^-$ 3-day lag = 1.049 (-3.209, 5.306) $SO_{4_2}^{2-}$ concurrent day = 1.764 (-1.817, 5.346) $SO_{4_2}^{2-}$ 2-day lag = 2.646 (-0.935, 6.228) SO_4^{2-} 3-day lag = 1.764 (-1.817, 5.346)
Reference: Ebelt et al. (2005, 056907) **Period of Study:** Summer of 1998 **Location:** Vancouver, Canada	**Outcome:** Spirometry **Age Groups**: Range from 54-86 yr Mean age= 74 yr **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 Fig 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)
Reference: Lagorio et al.(2006, 089800) **Period of Study:** May 1999-June 1999 and Nov 1999-Dec 1999 **Location:** Rome, Italy	**Outcome:** Lung function of subjects (FVC and FEV_1) with COPD, Asthma **Age Groups**: COPD 50-80 yr Asthma 18-64 yr **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 µg/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)
Reference: Laurent et al. (2008, 156672) **Period of Study:** Dec 2003-Dec 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 yr	**Pollutant:** PM_{10} **Averaging Time:** NR **Mean (SD) Unit:** 20.8 (10.2) µg/m^3 **Range (Min, Max):** NR **Copollutant (correlation):** NO_2, O_3, correlations NR	**Increment:** 10 µg/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 Fig 1 and 2
Reference: Tang et al. (2007, 091269) **Period of Study:** Dec 2003-Feb 2005 **Location:** Sin-Chung City, Taipei County, Taiwan	**Outcome:** Peak expiratory flow rate (PEFR) of asthmatic children **Age Groups**: 6-12 yr **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 mo, 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 µg/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

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
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-yr-old) healthy & with COPD Asthmatic children 6-13-yr-old **Study Design:** Adult and pediatric panel study over 3 yr with 1 monitoring period ("session") per yr **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 **Copollutant (correlation):** CO NO_2 $PM_{2.5}$	**PM Increment:** 10 µg/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 Lag 0 -63.87 [-199.58: 71.84] Lag 1 -96.48 [-232.48: 39.52] Anti-inflam. Medication 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 µg/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) **Period of Study:** Aug-Dec 2000 **Location:** Steubenville, Ohio	**Outcome:** FENO **Age Groups**: Ranged 53.5-90.6 yr **Study Design:** Prospective cohort **N:** Total of 294 breaths from 29 subjects **Statistical Analyses:** Fixed effect models, ANOVA, GLM procedure **Covariates:** Subject, week of study, day of the week, h of the day, ambient barometric pressure, temperature, and relative humidity **Dose-response Investigated?** No **Statistical Package**: SAS **Lags Considered:** Hourly lags, 0-48 h	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 1 h **Mean (SD):** 19.5 **Percentiles:** 25th: 7.6 75th: 25.5 **Range (Min, Max):** NR, 105.8 **Monitoring Stations**: 1 **Averaging Time:** 24 h **Mean (SD):** 19.7 **Percentiles:** 25th: 9.7 75th: 27.4 **Range (Min, Max):** NR, 57.8 **Monitoring Stations**: 1 **Copollutant (correlation):** Ambient NO Indoor NO NO_2 O_3 SO_2	**PM Increment:** 17.9 µg/m^3 **Effect Estimate [Lower CI, Upper CI]:** 1-h Single pollutant models: 0.36 (0.58-2.14) **PM Increment:** 17.7 Effect Estimate [Lower CI, Upper CI]: 24-h ma: 1.45 (0.33-2.57) Multipollutant models for $PM_{2.5}$, ambient NO and room NO and estimated change in FENO (ppb) for an IQR in pollutant measure Model 1 1.95 (0.47-3.43) Model 2 1.38 (0.26-2.51) Model 4 1.97 (0.48-3.46) **Notes:** Association of FENO with $PM_{2.5}$ at different lags presented in Fig 1 are not presented quantitatively elsewhere.
Reference: Adar et al. (2007, 098635) **Period of Study:** Mar-Jun 2002 **Location:** St. Louis, MO	**Outcome:** FENO **Age Groups**: 60+ **Study Design:** Panel Study **N:** 44 non-smoking seniors **Statistical Analyses:** Mixed models containing random subject effects **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 **Statistical Package**: SAS **Lags Considered:** 0	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean (SD):** Pretrip: 14.8 Post-trip: 16.5 **Percentiles:** 25th (pretrip): 11.2 75th (pretrip): 20.1 25th (post-trip): 11.7 75th (post-trip): 21.6 **Monitoring Stations**: 1 **Copollutant (correlation):** BC CO NO_2 SO_2 O_3	**PM Increment:** 9.8 µg/m^3 **Effect Estimate [Lower CI, Upper CI]:** Pre-trip % change: 21.9 (6.7, 39.4) Post-trip % change: -4.7 (-17.1, 9.6)
Reference: Aekplakorn et al. (2003, 089908) **Period of Study:** 107 days, from Oct 1997-Jan 1998 **Location:** Mae Mo district, Lampang Province, north Thailand	**Outcome:** Upper respiratory symptoms, lower respiratory symptoms, cough **Age Groups**: 6-14 yr old **Study Design:** Logistic regression **N:** 98 asthmatic school children **Statistical Analyses:** Generalized Estimating Equations, stratified analysis, PROC GENMOD **Covariates:** Temperature and relative humidity **Season:** Winter **Dose-response Investigated?** No **Statistical Package**: SAS v 8.1	**Pollutant:** $PM_{2.5}$ **Averaging Time:** Daily **Mean (SD):** Sob Pad station: 24.77 Sob Mo station: 24.89 Hua Fai station: 26.27 **Range (Min, Max):** Sob Pad: 4.52, 24.77 Sob Mo: 3.13, 24.89 Hua Fai: 3.67, 26.27 **Monitoring Stations**: 3 **Copollutant:** PM_{10} SO_2	**PM Increment:** 10 µg/m^3 **Odds Ratios [Lower CI, Upper CI] lag:** Asthmatics: URS: 1.04 (0.99, 1.09) lag 0 LRS: 1.05 (0.98, 1.2) lag 0 Cough: 1.05 (0.99, 1.10) lag 0 Non-Asthmatics: URS: 1.03 (0.96, 1.09) lag 0 LRS: 1.02 (0.93, 1.10) lag 0 Cough: 1.00 (0.93, 1.07) 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) lag 0 Cough: 1.00 (0.93, 1.07) lag 0

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
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 1 second (FEV_1), and forced vital capacity (FVC) **Age Groups:** 6-13 yr **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:** Jun 2003-Jun 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 µg/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 FEV25-75: -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) 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)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
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 yr **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 µg/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)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Bourotte et al. (2007, 150040) **Period of Study:** May 2002-Jul 2002 **Location:** Sao Paulo, Brazil	**Outcome:** Peak expiratory flow (PEF) **Age Groups**: Avg age 39.8 ± 12.3 yr **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^- SO_4^{2-} **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) 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) Fl_{nf} 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} f 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 Nov 1998-Jun 1999; in Erfurt, from Oct 1998-Apr 1999; and in Helsinki, from Nov 1998-Apr 1999.) **Location:** Amsterdam, the Netherlands Erfurt, Germany and Helsinki, Finland	**Outcome:** Respiratory symptoms **Age Groups**: ≥ 50 yr **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} NC0.01-0.1 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)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Delfino et al. (2004, 056897) **Period of Study:** Sep-Oct 1999 Apr-Jun 2000 **Location:** Alpine, California	**Outcome**: FEV_1 **Age Groups**: 9-19 yr old **Study Design:** Panel study **N:** 24 children **Statistical Analyses:** GLM Akaike's information criterion and Bayesian information criterion **Covariates:** Day of wk, 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	**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) 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	Results presented graphically; % 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)
Reference: Delfino et al. (2006, 090745) **Period of Study:** Region 1: Aug-Mid Dec 2003. Region 2: Jul-Nov 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-needed B-agonist inhalers **Dose-response Investigated?** No **Lags Considered:** 0, 1, 2, MA day	**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): 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	**PM Increment:** IQR increase (Riverside: 28.41 µg/m^3, Whittier 21.87 µg/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) Central 0.52 (-0.43, 1.47) Stratified by Medication Use Lag = 2-day ma 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:** Fig of Estimated lag effect of hourly personal $PM_{2.5}$ on FENO. Fig of the Estimated lag effect of hourly personal $PM_{2.5}$ on FENO by use of medications. Fig of one- and two-pollutant models for change in FENO using 2-day Ma 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: Aug-Mid Dec 2003. Region 2: Jul-Nov 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 ma	**Pollutant:** $PM_{2.5}$ **PM Component:** EC **Personal Exposure** **Averaging Time:** 24 h Riverside **Mean (SD):** 0.42 (0.69) 50th(Median): 0.34 µg/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 µg/m^3, Whittier 21.87 µg/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 ma 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:** Fig of Estimated lag effect of hourly personal $PM_{2.5}$ on FENO. Fig of the estimated lag effect of hourly personal $PM_{2.5}$ on FENO by use of medications. Fig of one- and two-pollutant models for change in FENO using 2-day Ma 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: Aug-Mid Dec 2003. Region 2: Jul through Nov 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 ma	**Pollutant:** $PM_{2.5}$ **PM Component:** OC **Personal Exposure** **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	**PM Increment:** IQR increase (Riverside: 28.41 µg/m^3, Whittier 21.87 µg/m^3) Mixed-model estimates of the association between personal and central-site air 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 ma. 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:** Fig of Estimated lag effect of hourly personal $PM_{2.5}$ on FENO. Fig of the Estimated lag effect of hourly personal $PM_{2.5}$ on FENO by use of medications. Fig of one- and two-pollutant models for change in FENO using 2-day Ma personal and central-site pollutant measurements

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Dubowsky et al. (2006, 088750) **Period of Study:** Mar 2002–Jun 2002 **Location:** St. Louis, Missouri	**Outcome:** Chronic inflammation, Diabetes, Obesity, Hypertension, Cardiac Risk **Study Design:** Prospective Cohort **Statistical Analyses:** Poisson, LOESS **Age Groups**: ≥ 60	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-h avg **Mean (SD) unit:** 16 (6.0) **Range (Min, Max):** 6.5, 28 **Copollutants:** BC CO NO_2 SO_2 O_3	**Increment:** 5.4 µg/m^3 **% Increase (Lower CI, Upper CI)** **Lag** % increase in inflammatory response and exposure to $PM_{2.5}$ in people ≥ 60 Inflammatory Marker: IL-6: -8 (-16, 8) 1: -6 (-10, 5) 2: -5 (-11, 6) 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 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 2 of the variables.

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 yr Mean age= 74 yr **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 Fig 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 yr Mean age= 74 yr **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:** 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):** Ambient $PM_{2.5}$: r= 0.82 Nonsulfate Ambient $PM_{2.5}$: r= 0.74 Exposure to Ambient Sulfate: r= 0.82	**PM Increment:** Ambient Sulfate: 1.5 (IQR) Exposure to Ambient Sulfate: 0.9 (IQR) **Notes:** Effect estimates are presented in Fig 2 and Electronic Appendix Table 1 (only available with electronic version of article) and not provided quantitatively elsewhere.
Reference: Ferdinands et al. (2008, 156433) **Period of Study:** Aug 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:** Apr-Sep 2001 **Location:** Connecticut Springfield, MA	**Outcome:** Respiratory symptoms including: Wheeze, persistent cough, chest tightness, shortness of breath **Age Groups**: Infants **Study Design:** 1-yr 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	**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)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
	Covariates: Temperature **Dose-response Investigated?** No **Statistical Package**: SAS **Lags Considered:** 1-day lag		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) 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) **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)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			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) 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 O_3 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	**Pollutant:** $PM_{2.5}$ and components **Averaging Time:** Daily **Mean:** (estimated sources, µg/m^3) Motor Vehicle: 6.6 Road Dust: 2.3 Sulfur: 5.5	**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 EC, 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)
	Age Groups: Children aged 4-12	Biomass Burning: 0.9 Oil: 0.8 Sea Salt: 0.5 **Range (Min, Max):** NR **Copollutant (correlation):** NR	L2: 1.00, p = 0.99 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 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

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			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 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 L1: 1.00, p = 0.78 L2: 1.01, p = 0.60 L02: 1.05, p = 0.02 Shortness of Breat1.04, p = 0.01h L0: 1.04, p = 0.01 L1: 1.01, p = 0.60 L2: 1.01, p = 0.63 L02: 1.08, p = 0.02 Chest Tightness L0: 1.02, p = 0.20 L1: 1.02, p = 0.17 L2: 1.00, p = 0.88 L02: 1.06, p = 0.10 Inhaler Use L0: 1.02, p = 0.004 L1: 0.99, p = 0.18 L2: 1.01, p = 0.45

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			L02: 1.03, p = 0.09 Fe, Increment = 100 ng/m^3 Wheeze L0: 1.04, p = 0.02 L1: 1.00, p = 0.80 L2: 1.00, p = 0.87 L02: 1.07, p = 0.05 Persistent Cough L0: 1.02, p = 0.06 L1: 1.01, p = 0.52 L2: 0.99, p = 0.52 L02: 1.04, p = 0.04 Shortness of Breath L0: 1.06, p = 0.002 L1: 1.01, p = 0.65 L2: 0.98, p = 0.27 L02: 1.08, p = 0.04 Chest Tightness L0: 1.01, p = 0.47 L1: 1.02, p = 0.22 L2: 0.98, p = 0.35 L02: 1.05, p = 0.21 Inhaler Use L0: 1.02, p = 0.004 L1: 0.99, p = 0.44 L2: 1.00, p = 0.91 L02: 1.03, p = 0.08 Al, Increment = 50 ng/m^3 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 L02: 1.14, p = 0.07 Inhaler Use L0: 1.04, p = 0.01

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			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 L1: 1.00, p = 0.56 L2: 1.00, p = 0.60 L02: 1.03, p = 0.05 Chest Tightness L0: 1.00, p = 0.34 L1: 1.00, p = 0.55 L2: 0.99, p = 0.49 L02: 1.01, p = 0.52 Inhaler Use L0: 1.00, p = 0.72 L1: 1.00, p = 0.30 L2: 1.00, p = 0.67 L02: 1.00, p = 0.66 Source: Sulfur S, Increment = 1000 ng/m^3 Wheeze L0: 0.98, p = 0.43 L1: 0.99, p = 0.62 L2: 1.02, p = 0.29 L02: 1.00, p = 0.99 Persistent Cough L0: 1.00, p = 0.84 L1: 1.00, p = 0.69 L2: 1.02, p = 0.21 L02: 1.02, p = 0.27 Shortness of Breath L0: 1.01, p = 0.63 L1: 0.99, p = 0.71 L2: 1.01, p = 0.55 L02: 1.01, p = 0.79 Chest Tightness L0: 0.99, p = 0.80 L1: 1.01, p = 0.62

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			L2: 1.01, p = 0.81 L02: 1.02, p = 0.68 Inhaler Use L0: 0.99, p = 0.13 L1: 1.00, p = 0.81 L2: 1.02, p = 0.04 L02: 1.00, p = 0.81 P, Increment = 50 ng/m^3 Wheeze L0: 0.98, p = 0.39 L1: 0.98, p = 0.48 L2: 1.02, p = 0.38 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 L1: 0.99, p = 0.05 L2: 1.00, p = 0.59 L02: 0.99, p = 0.28 Source: Oil V, Increment = 10 ng/m^3 Wheeze L0: 0.99, p = 0.73 L1: 0.96, p = 0.03 L2: 0.99, p = 0.56 L02: 0.93, p = 0.04 Persistent Cough L0: 1.01, p = 0.56 L1: 0.99, p = 0.24 L2: 0.98, p = 0.01 L02: 0.96, p = 0.05 Shortness of Breath L0: 1.01, p = 0.46 L1: 0.98, p = 0.24

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			L2: 1.00, p = 0.83 L02: 0.98, p = 0.58 Chest Tightness L0: 0.99, p = 0.71 L1: 0.98, p = 0.32 L2: 0.98, p = 0.23 L02: 0.94, p = 0.12 Inhaler Use L0: 0.98, p = 0.12 L1: 1.00, p = 0.68 L2: 0.99, p = 0.22 L02: 0.96, p = 0.03 Ni, Increment = 5 ng/m^3 Wheeze L0: 1.01, p = 0.59 L1: 0.97, p = 0.09 L2: 1.00, p = 0.76 L02: 0.99, p = 0.72 Persistent Cough L0: 1.01, p = 0.21 L1: 0.99, p = 0.57 L2: 0.99, p = 0.23 L02: 1.00, p = 0.99 Shortness of Breath L0: 1.04, p = 0.05 L1: 0.98, p = 0.36 L2: 1.00, p = 0.81 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 L0: 1.00, p = 0.31 L1: 1.00, p = 0.31 L2: 1.00, p = 0.51

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			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 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)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			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) 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 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)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			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:** Aug 2002-Oct 2002 Jun 2003-Aug 2003 **Location:** Charlies Bunion Trail (portion of Appalachia Trail)	**Outcome:** Pulmonary function/spirometry-FVC, FEV_1, PEF, FVC/FEV_1, FEF25-75 **Age Groups**: 18-82 yr **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 µg/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 FEF25-75%: 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 FEF25-75%: - 0.041 ± 0.109 0.707 Spirometry result for each quintile ± CI **Quintile 1 (6.0** µg/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 FEF25-75% (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** µg/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 FEF25-75% (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** µg/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 FEF25-75% (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** µg/m^3**):**

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			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 FEF25-75% (L/sec): Prehike: 3.34 ± 0.15 Posthike: 3.30 ± 0.15 PEF (L/sec): Prehike: 7.75 +/ 0.25 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 FEF25-75% (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 FEF25-75% (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 2-4.5 yr **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 mo: 1.30 [1.08, 1.58] lag 1-30 2-4.5 yr: 1.23 [0.94, 1.62] lag 1-30 **RR Estimate for categories of exposure [Lower CI, Upper CI] lag:** Crude RR: Birth-23 mo: > 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: Referent 2-4.5 yr: > 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: Referent

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
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Reference: Hogervorst, et al. (2006, 156559) **Period of Study:** 2002 **Location:** Maastricht, the Netherlands (six schools selected)	**Outcome**: Decreased lung function **Age Groups**: 8-13 yr **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:** **Location:** Ciudad Juarez, Mexico	**Outcome:** FeNO, FEV_1 **Study Design:** Panel **Covariates:** sex, age, body mass index, day of week, season, yr of maternal and paternal education, passive smoking **Statistical Analysis:** linear and nonlinear mixed effects models **Age Groups:** 6-12 yr	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 48 h **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.

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Hong et al. (2007, 091347) **Period of Study:** Mar 23-May 2004 **Location:** School on the Dukjeok Island near Incheon City, Korea	**Outcome:** Peak expiratory flow rate (PEFR) **Age Groups**: 3rd-6th grade (mean age=9.6 yr) **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 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, SaO_2: oxygen saturation, Pulse rate **Age Groups**: 60-86-yr-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.

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Johnston, et al. (2006, 091386) **Period of Study:** 7 mo (Apr-Nov 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"- note Southern Hemisphere **Dose-response Investigated?** No **Statistical Package**: STATA8 **Lags Considered:** 0-5 days	**Pollutant:** $PM_{2.5}$ **Averaging Time:** Daily **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 µg/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** 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 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)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
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 yr 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 µg/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 yr 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 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	**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)
Reference: Kongtip et al. (2006, 096920) **Period of Study:** Sep-Oct 2004 **Location:** Dindang district, Bangkok metropolitan, Thailand	**Outcome:** respiratory and other Outcomes reported **Age Groups**: Age range 15-55 yr **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 µg/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) 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:** May-Jun1999 and Nov-Dec 1999 **Location:** Rome, Italy	**Outcome:** Lung function (FVC and FEV_1) of subjects with COPD, Asthma **Age Groups**: COPD 50-80 yr Asthma 18-64 yr **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)

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 yr 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 **Lags Considered:** 0-4 days	**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
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 yr old **Study Design:** Longitudinal cohort study **N:** 86 children **Statistical Analyses: D**escriptive 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 (Feb 10-23), Spring 2001 (May 5-18), Summer 2001 (Jul 14-27), Fall 2001 (Sep 22-Oct 5), Winter 2002 (Jan 18-31), and Spring 2002 (May 18-31)]. **Dose-response Investigated?** No **Lags Considered:** 1-2 days, 3-5 days	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 2 wk **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 µg/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

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Liu et al. (2009, 192003) **Period of Study:** 4 wk 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 yr	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 1, 2 & 3 days **Mean (SD) Unit (1d):** 6.5 µg/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 µg/m^3 **Percent Change (Min CI, Max CI)** **Lag** FEV_1 Same Day: -0.5 (-1.3-0.3) Lag 1 Day: -0.5 (-1.1-0.5) 2-Day Avg: -0.6 (-1.5-0.4) 3-Day Avg: -1.1 (-3.1-0.9) FEF 25%-75% Same Day: -1.9 (-3.5--0.3) Lag 1 Day: -1.2 (-2.8-0.3) 2-Day Avg: -2.0 (-3.8--0.2) 3-Day Avg: -3.3 (-7.2-0.8) FeNO Same Day: 5.3 (-3.6-15) Lag 1 Day: 1.7 (-6.3-15) 2-Day Avg: 4.3 (-5.4-15.1) 3-Day Avg: -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 Avg: 22.0 (4.8-42.1) 3-Day Avg: 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 Avg: 0.1 (-9.8-11.1) 3-Day Avg: 5.8 (-15.8-33.0)/

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Mar et al. (2004, 057309) **Period of Study:** 1997-1999 **Location:** Spokane, Washington	**Outcome:** Respiratory Symptoms **Age Groups**: Adults: Ages 20-51 yr Children: Ages 7-12 yr **N:** 25 people **Statistical Analyses:** Logistic regression **Covariates:** Temperature, relative humidity, day of-the-wk **Statistical Package**: STATA 6 **Lags Considered:** 0-2 days	**Pollutant:** $PM_{2.5}$ **Mean (SD):** 1997: 11.0 (5.9) 1998: 10.3 (5.4) 1999: 8.1 (3.8) Unit (i.e. $\mu g/m^3$): **Monitoring Stations**: 1 station **Copollutant (correlation):** $PM_{2.5}$ PM_1 r = 0.92 PM_{10} r = 0.61 $PM_{10-2.5}$ r = 0.28	**PM Increment:** 10 $\mu g/m^3$ **OR Estimate [Lower CI, Upper CI] lag:** **Adult Respiratory symptoms:** **Wheeze:** 1.04[0.86, 1.26] lag 0 1.00[0.83, 1.19] lag 1 0.99[0.84, 1.17] lag 2 **Breath:** 0.97[0.87, 1.08] lag 0 0.98[0.87, 1.10] lag 1 0.95[0.80, 1.13] 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] 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] 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

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 **Statistical Analyses:** Linear logistic regression **Age Groups**: > 57	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-h avg	**Increment:** 10 µg/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) 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 yr **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 Fig 1. **Monitoring Stations**: 3 Stations	**PM Increment:** 10 µg/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 Fig 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 7-10 **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:** $PM_{2.5}$ **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, MEF_{50}) 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:** 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 µg/m^3 **% change in Lung Function per 10 µg/m3** 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 MEF_{50}%: 0.04 MEF_{25}%: -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, 189159) **Period of Study:** Nov 2004 **Location:** Tokyo, Japan	**Outcome:** Exhaled nitric oxide levels, (eNO), a marker of airway inflammation **Age Groups**: 5-10 yr **Study Design:** Cohort/Panel study **N:** 19 schoolchildren* **Statistical Analyses:** Linear regression **Covariates:** None **Season:** Nov (fall) **Dose-response Investigated?** No **Statistical Package**: SAS **Lags Considered:** Lag h 1-24, 8-h ma, 7-h ma, 6-h ma, 24-h ma	**Pollutant:** $PM_{2.5}$ **Averaging Time:** Hourly, 24 h **Mean (SD):** 39.0 (16.9) µg/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 µg/m^3 **Mean [Lower CI, Upper CI] lag:** 0.145 [0.62, 0.228] ppb eNO 8-h ma **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 affected by changes in air pollutants over at least the previous 8-h period" $PM_{2.5}$, black carbon, and NO_X 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

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Neuberger et al. (2004, 093249) **Period of Study:** Jun 1999-Jun 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 yr **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-wk avg (preceding interview)	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Copollutant (correlation):** PM_{10} (r=0.94) in Vienna	**PM Increment:** 10 µg/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:** Sep 1999-Mar 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 yr (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 EC OC **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 EC: -0.657 (0.126) lag 0 OC: -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 yr **Study Design:** Longitudinal prospective cohort **N:** 164 children **Statistical Analyses: M**ixed models linear regression with autoregressive correlation structure **Covariates:** Sex, time and individual **Season:** Oct-May **Dose-response Investigated?** No **Statistical Package**: NR **Lags Considered:** Lag 0-7	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Monitoring Stations**: 1	**PM Increment:** 1 µg/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)
Reference: O'Connor et al. (2008, 156818) **Period of Study:** Aug 1998-Jul 2001 **Location:** Boston, the Bronx, Chicago, Dallas, New York, Seattle, Tucson	**Outcome:** Pulmonary function and respiratory symptoms **Age Groups**: 5-12 yr **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 Fig) **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 µg/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
Reference: Peacock et al. (2003, 042026) **Period of Study:** Nov 1996-Feb 1997 **Location:** northern Kent, UK	**Outcome:** Reduced peak expiratory flow rate (PEFR) **Age Groups**: 7-13 yr 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, 5-day ma	**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 **Monitoring Stations**: 3	Sulfate (SO_4^{2-}) Increment: 1.3 µg/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 wk between Mar-Jun 1999 and Sep-Dec 1999. **Location:** Ashdod, Ashkelon and Sderot, Israel	**Outcome:** Reduced peak expiratory flow (PEF) **Age Groups**: 7-10 yr **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, Fall **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 µg/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 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) **Period of Study:** Nov 1996-Apr 1997 **Location:** Helsinki, Finland	**Outcome:** Decreased lung function and respiratory symptoms **Age Groups**: Adults, mean age 53 yr **Study Design:** Time Series **N:** 78 people **Statistical Analyses:** Generalized least squares autoregressive model **Covariates:** Temperature, relative humidity, day of study, day of study squared, binary dummy variable for weekends **Season:** Winter, Spring **Dose-response Investigated?** NR **Statistical Package**: SAS version 6 **Lags Considered:** 0-3	**Pollutant:** $PM_{2.5}$ **PM Component:** Soil, heavy fuel oil, sea salt **Averaging Time:** 24 h **Percentiles: 25th:** Long range transport: 2.44 Local combustion: 1.75 Soil: 0.14 Heavy fuel oil: -0.13 Sea Salt: 0.22 Unidentifiable: -1.41 All sources: 6.47 **50th(Median):** Long range transport: 4.15 Local combustion: 2.41 Soil: 0.64 Heavy fuel oil: 0.10 Sea Salt: 0.27 Unidentifiable: 0.02 All sources: 8.37 **75th:** Long range transport: 7.33 Local combustion: 3.05 Soil: 1.46 Heavy fuel oil: 0.52 Sea Salt: 0.42 Unidentifiable: 0.74 All sources: 11.15 **Range (Min, Max):** Long range transport: (-0.89, 28.31) Local combustion: (0.83, 6.51) Soil: (-1.13, 6.43) Heavy fuel oil: (-0.67, 4.74) Sea Salt: (0.09, 0.98) Unidentifiable: (-4.40, 4.77) All sources: (4.11, 33.53) **Monitoring Stations**: 1 site	**PM Increment:** 1.3 µg/m^3 **$PM_{2.5}$, long range:** **PEF Morning:** 0.37[-0.59, 1.34] lag 0 -1.04[-1.88 to -0.19] lag 1 -0.82[-1.81, 0.16] lag 2 0.22[-0.64, 1.08] lag 3 -0.24[-1.12, 0.64] 5 day mean. **PEF Afternoon:** 0.20[-0.67, 1.06] lag 0 -0.20[-1.24, 0.83] lag 1 -0.30[-1.14, 0.53] lag 2 0.45[-0.57, 1.47] lag 3 0.03[-0.79, 0.85] 5 day mean. **PEF Evening:** -0.33[-1.30, 0.64] lag 0 -0.29[-1.13, 0.55] lag 1 -0.41[-1.46, 0.64] lag 2 0.39[-0.47, 1.24] lag 3 0.07[-0.81, 0.95] 5 day mean **$PM_{2.5}$, local combustion:** **PEF Morning:** -0.73[-1.69, 0.23] lag 0 -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, 0.45] lag 0 -1.16[-1.93 to -0.39] lag 1 0.23[-1.35, 0.90] lag 2 0.56[-0.21, 1.32] lag 3 -1.14[-1.95 to -0.33] 5 day mean **$PM_{2.5}$, soil:** **PEF Morning:** 0.81[0.05, 1.57] lag 0 0.03 [-0.65, 0.71] lag 1 0.50[-0.34, 1.35] lag 2 -0.07[-0.74, 0.61] lag 3 0.39[-0.46, 1.23] 5 day mean. **PEF Afternoon:** 1.05[0.38, 1.72] lag 0 0.40[-0.38, 1.19] lag 1 0.66 [0.03, 1.30] lag 2 -0.36[-1.12, 0.41] lag 3 0.55 [-0.21, 1.32] 5 day mean. **PEF Evening:** 1.08[0.33, 1.84] lag 0 1.00[0.31, 1.68] lag 1 0.33[-0.56, 1.22] lag 2 -0.84 [-1.53 to -0.15]lag 3 0.90[0.08, 1.73] 5 day mean **$PM_{2.5}$, oil:** **PEF Morning:** -0.22[-1.00, 0.56] lag 0 -0.20[-1.24, 0.84] lag 1 0.66[-0.68, 2.00] lag 2 0.57 [-0.18, 1.32] lag 3 0.10[-0.61, 0.81] 5 day mean. **PEF Afternoon:** -0.04[-0.75, 0.67] lag 0

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			0.29[-0.98, 1.55] lag 1 0.08 [-1.13, 1.28] lag 2 0.62[-0.31, 1.54] lag 3 0.07 [-0.64, 0.78] 5 day mean. **PEF Evening:** 0.57[-0.23, 1.37] lag 0 0.12[-0.92, 1.15] lag 1 -0.97[-2.39, 0.45] lag 2 0.40[-0.31, 1.12] lag 3 0.43[-0.33, 1.19] 5 day mean **$PM_{2.5}$, salt:** **PEF Morning:** 0.76[-0.13, 1.65] lag 0 0.43 [-0.30, 1.16] lag 1 0.13[-0.75, 1.02] lag 2 0.38[-0.47, 1.23] lag 3 0.95[-0.18, 2.09] 5 day mean. **PEF Afternoon:** 0.62[-0.18, 1.41] lag 0 0.80[-0.08, 1.69] lag 1 0.14[-0.62, 0.90] lag 2 0.16[-0.83, 1.15] lag 3 0.88 [-0.18, 1.94] 5 day mean. **PEF Evening:** 1.09[0.19, 1.98] lag 0 0.63[-0.10, 1.35] lag 1 0.32[-0.62, 1.26] lag 2 -0.31[-1.16, 0.54] lag 3 0.88[-0.27, 2.02] 5 day mean **$PM_{2.5}$, unidentified:** **PEF Morning:** 0.38[-0.67, 1.43] lag 0 0.09[-0.83, 1.00] lag 1 0.22[-0.82, 1.26] lag 2 0.78 [-0.10, 1.66] lag 3 0.78[-0.14, 1.69] 5 day mean. **PEF Afternoon:** 0.02[-0.92, 0.96] lag 0 0.65[-0.48, 1.77] lag 1 0.17[-0.71, 1.05] lag 2 0.69[-0.36, 1.75] lag 3 0.17 [-0.72, 1.06] 5 day mean. **PEF Evening:** -0.11[-1.17, 0.95] lag 0 0.19[-0.72, 1.10] lag 1 0.86[-0.25, 1.96] lag 2 0.15[-0.70, 1.01] lag 3 -0.19[-1.15, 0.77] 5 day mean **$PM_{2.5}$, local combustion**: **PEF morning:** Cu: -0.25 [-1.25, 0.75] Zn: -0.45[-1.19, 0.29] Mn: 0.13[-0.83, 1.08] Fe: 0.08[-0.70, 0.85]. **PEF afternoon:** Cu: -0.37[-1.29, 0.55] Zn: -0.19[-0.87, 0.50] Mn: -0.48[-1.37, 0.42] Fe: 0.29[-0.45, 1.04]. **PEF evening:** Cu: -0.48[-1.47, 0.52] Zn: -0.17[-0.92, 0.57] Mn: 0.51[-0.44, 1.47] Fe: 0.34[-0.46, 1.14] **$PM_{2.5}$, long range:** **PEF morning:** S: 0.11[-0.886, 1.07] K: -0.10[-1.00, 0.80] Pb: -0.62[-1.37, 0.13]

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			Br: -0.40 [-1.40, 0.60]. **PEF afternoon:** S: -0.05[-0.92, 0.81] K: 0.26[-0.56, 1.07] Pb: -0.12[-0.84, 0.60] Br: 0.15[-0.81, 1.12]. **PEF evening:** S: 0.08[-0.86, 1.02] K: 0.18[-0.70, 1.07]; Pb: -0.20[-0.97, 0.58] Br: 0.35[-0.71, 1.40] **$PM_{2.5}$, soil:** **PEF morning:** Si: 0.27[-0.43, 0.97] Al: 0.17 [-0.72, 1.05] Ca: 0.13[-1.08, 1.35]. **PEF afternoon:** Si: 0.39[-0.24, 1.01] Al: 0.49[-0.29, 1.27] Ca: 0.15[-0.92, 1.22] **PEF evening:** Si: 0.60[-0.06, 1.26] Al: 0.76[-0.08, 1.60] Ca: 0.90[-0.22, 2.03] **$PM_{2.5}$, Oil combustion:** **PEF morning:** V: -0.01[-0.87, 0.86] Ni: -0.09[-1.08, 0.90]. **PEF afternoon:** V: -0.48[-1.32, 0.35] Ni: 0.26[-0.72, 1.23]. **PEF evening:** V: 0.02[-00.88, 0.92] Ni: 0.50[-0.55, 1.55] **$PM_{2.5}$, Sea salt:** **PEF morning:** Na: 0.92[-0.34, 2.17] Cl: 0.93[0.08, 1.79] **PEF afternoon:** Na: 0.96[-0.24, 2.16] Cl: 0.57[-0.22, 1.36] **PEF evening** Na: 0.87[-0.40, 2.15] Cl: 0.65[-0.19, 1.49]

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Pino et al. (2004, 050220) **Period of Study:** Apr 1995-Oct 1996 **Location:** Santiago, Chile	**Outcome:** Respiratory Symptoms, Wheezing bronchitis **Study Design:** Time-series **Statistical Analyses:** Bayesian hierarchical analysis, cubic spline **Age Groups**: 4 mo-2 yr 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 µg/m^3 **% Increase (Lower CI, Upper CI) lag:** % increase in wheezing bronchitis and $PM_{2.5}$ exposure for infants 4 mo-2 yr 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 mo to 2 yr 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 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 yr 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** 25th: 4.0	**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 yr: 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 yr 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 = 0.006 Difference between severity groups,

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
		50th(Median): 5.9 75th: 9.6 **Year 2** 25th: 5.2 50th (Median): 8.5 75th: 11.6 Morning maximum, TEOM **Year 1** 25th: 8 50th (Median): 13 75th: 20 **Year 2** 25th: 11 50th (Median): 16 75th: 23 **Range (Min, Max):** 24-h mean, TEOM Year 1 (2.1, 23.7) Year 2 (1.7, 20.5) 24-h mean, FRM Year 1 (4.3, 53.5) Year 2 (3.4, 26.3) Morning mean, TEOM Year 1 (1.4, 22.7) Year 2 (1.6, 30.2) Morning maximum, TEOM Year 1 (4, 42) Year 2 (4, 46) **Monitoring Stations**: 2 (1 TEOM and 1 Federal Reference Monitor [FRM])	p = 0.08 Aggregated severity groups: 2.7 [-0.1: 5.4] p= 0.06 **Morning maximum $PM_{2.5}$ TEOM** URI not adjusted Mild/Moderate Asthmatics: 1.9 [-0.2: 4.1] p= 0.07 Severe Asthmatics: 3.9 [1.1: 6.8] p = 0.006 Difference between severity groups, p = 0.29 Aggregated severity groups: 2.6 [0.9: 4.2] p= 0.002 URI adjusted Mild/Moderate Asthmatics: 1.6 [-2.2: 5.4] p = 0.41 Severe Asthmatics: 8.1 [2.9: 13.4] p = 0.003 Difference between severity groups, p = 0.03 Aggregated severity groups: 3.8 [0.2: 7.4] p = 0.04 **Leukotriene E4** **24-h $PM_{2.5}$** **TEOM** Lag 0: 3.3 [-0.7: 7.2] p = 0.09 Lag 1: -1.6[-5.7: 2.5] p = 0.40 Lag 2: 1.1 [-2.8: 5.1] p= 0.64 Lag 0-2 Avg: 2.3 [-4.0: 8.6] p = 0.45 **FRM** Lag 0: 2.7 [1.1: 6.5] p = 0.12 Lag 1: -0.8 [-4.9: 3.3] p = 0.65 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 25percentile: 8.9 [3.0: 14.7] p= 0.004 Height 50percentile: 5.9 [1.4: 10.4] p = 0.01 Height 75percentile: 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 25percentile: 8.3 [3.4: 13.2] p = 0.001 Height 50percentile: 6.1 [2.1: 10.2] p= 0.004 Height 75 percentile: 3.2 [-2.0: 8.4] p= 0.23 Model w/o Height × **Pollutant:** 6.2 [1.9: 10.5] p = 0.006
Reference: Rabinovitch et al. (2004, 096753) **Periods of Study:** Nov 1999-Mar 2000 Nov 2000-Mar 2001 Nov 2001-Mar 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.

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Ranzi et al. (2004, 089500) **Period of Study:** Feb-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 yr) **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 ma	**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 µg/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 yr 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: 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: 1.01 [0.997: 1.006] 1.02 **$PM_{2.5}$, 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]

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Sakai et al. (2004, 087435) **Period of Study:** Nov 1999-Mar 2001 **Location:** Diesel-powered ship from Tokyo, Japan to Showa Station on Ongul Island, Antarctica for 366 days (from Feb 1, 2000) and then heading back to Japan on Feb 1, 2001	**Outcome:** circulating leukocyte counts and serum inflammatory cytokine levels **Age Groups**: 24-57 yr, mean=36.1 ± 4.7 yr **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. µg/m3):** 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 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:** Nov 1999-Mar 28, 2001 **Location:** Diesel-powered ship from Tokyo, Japan to Showa Station on Ongul Island, Antarctica for 366 days (from Feb 1, 2000) and then heading back to Japan on Feb 1, 2001	**Outcome:** circulating leukocyte counts and serum inflammatory cytokine levels **Age Groups**: 24-57 yr, mean=36.1 ± 4.7 yr **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. µg/m3):** 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

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
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 yr-old) with COPD, as well as >10 pack-yr 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, ma 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 analysis run separately for each winter **Season:** Winter **Dose-response Investigated?** No **Statistical Package**: SAS **Lags Considered:** 0-2 days	**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 (1.8, 36.6) Winter 2000-2001 (3.4, 59.6) **Monitoring Stations**: multiple sites **Copollutant (correlation):** CO NO_2 PM_{10}	**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.
Reference: Sivacoumar et al. (2006, 111115) **Period of Study:** Apr 1998-May 1998; Sep 1998-Oct 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**: 5.1-13.1 yr 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 Avg 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 µg/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.

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
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 yr 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:** Fall 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: EC, sulfate, nitrate and ETS. **Monitoring Stations**: 2 fixed monitors and up to 10 personal monitors on a given day. **Copollutant (correlation):** Sulfate (0.63)	**PM Increment:** 10 µg/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 µg/m^3 increase in ambient $PM_{2.5}$ [0.0, 4.3 decrease] 1 day
Reference: Tang et al. (2007, 091269) **Period of Study:** Dec 2003-Feb 2005 **Location:** Sin-Chung City, Taipei County, Taiwan	**Outcome:** Peak expiratory flow rate (PEFR) of asthmatic children **Age Groups**: 6-12 yr **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 mo, 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 µg/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-Apr 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 µg/m^3 Erfurt: 23.1 µg/m^3 Helsinki: 12.7 µg/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 NC0.1-1.0: 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 µg/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

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
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-yr-old) healthy & with COPD Asthmatic children 6-13-yr-old **Study Design:** Adult and pediatric panel study over 3 yr with 1 monitoring period ("session") per yr **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: G**ender, age, ventral site temperature and relative humidity, CO, NO_2 **Dose-response Investigated?** No **Statistical Package**: SAS **Lags Considered:** 0-1 days	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **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. **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)	**PM Increment:** 10 µg/m^3 **ADULT** **Personal** $PM_{2.5}$ **-** FEV_1 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] 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-inflammatory medication: Lag 0 -41.73 [-94.31: 10.84] Lag 1 -30.99 [-82.17: 20.19]. Anti-inflammatory 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-inflammatory medication: Lag 0 -75.92 [-145.16: 6.67] Lag 1 -65.08 [-136.98: 6.82]. Anti-inflammatory medication: Lag 0 -28.50 [-94.72: 37.71]

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			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-inflammatory medication: Lag 0 -24.42 [-81.22: 32.38] Lag 1 16.52 [-45.76: 78.80]. Anti-inflammatory 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-inflammatory medication: Lag 0 -33.59 [-89.99: 22.82] Lag 1 31.30 [-29.91: 92.51] Anti-inflammatory 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-inflammatory medication: Lag 0 0.22 [-8.85: 9.29] Lag 1 -10.48 [-18.68: 2.28] Anti-inflammatory 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-inflammatory medication: Lag 0 -13.34 [-25.90: -0.79] Lag 1 -17.13 [-29.86: 4.41]. Anti-inflammatory 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 -6.27 [-14.07: 1.53] Lag 1 -5.64 [-13.73: 2.44]. No anti-inflammatory medication: Lag 0 -7.52 [-17.56: 2.51] Lag 1 -6.92 [-18.03: 4.19]. Anti-inflammatory 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-inflammatory medication: Lag 0 -6.32 [-16.31: 3.68] Lag 1 -0.83 [-11.60: 9.95] Anti-inflammatory 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-inflammatory medication: Lag 0 -3.32 [-9.52: 2.88] Lag 1 -2.49 [-8.23: 3.25]. Anti-inflammatory 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]**.** Anti-inflammatory medication: Lag 0-12.65 [-20.74: -4.56]' Lag 1 -

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			13.84 [-21.82: 5.85] Anti-inflammatory 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-inflammatory medication: Lag 0 -8.23 [-14.77: 1.69] Lag 1 -1.19 [-8.45: 6.07] Anti-inflammatory 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-inflammatory medication: Lag 0 -8.21 [-14.79: 1.62] Lag 1 -0.22 [-7.34: 6.90] Anti-inflammatory 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-Feb 2005 **Location:** Sin-Chung City, Taipei County, Taiwan	**Outcome:** Peak expiratory flow rate (PEFR) of asthmatic children **Age Groups**: 6-12 yr **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 mo, 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:** $PM_{2.5-1}$ **Averaging Time:** 1 h **Mean (SD):** Personal: 6.2 (4.8) **Range (Min, Max):** Personal: 0.3-86.8 **Monitoring Stations**: 1	No quantitative effects reported.
Reference: Tang et al. (2007, 091269) **Period of Study:** Dec 2003-Feb 2005 **Location:** Sin-Chung City, Taipei County, Taiwan	**Outcome:** Peak expiratory flow rate (PEFR) of asthmatic children **Age Groups**: 6-12 yr **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 mo, 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:** PM1 **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 **Monitoring Stations**: 1	**PM Increment:** 27.6 µg/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

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Timonen et al. (2004, 087915) **Period of Study:** Oct 1998-Apr 1999 **Location:** Amsterdam, The 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. µg/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 Helsinki 0.72 CO: Amsterdam 0.22 Erfurt 0.72 Helsinki 0.35	**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.
Reference: Timonen et al. (2004, 087915) **Period of Study:** Oct 1998-Apr 1999 **Location:** Amsterdam, The 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:** NC10-0.1 **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. µg/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

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: von Klot et al. (2002, 034706) **Period of Study:** Sep 1996-Mar 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 yr and range =37-77 yr **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: W**inter **Dose-response Investigated?** No **Statistical Package**: NR **Lags Considered:** 0, 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, ma calculated from same day and preceding days	**Pollutant:** MC0.5-0.1 **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 NC0.1-0.01: r= 0.45 NC0.5-0.1: r= 0.95 NC2.5-0.5: r= 0.92 MC2.5-0.01: r= 1.00 PM_{10}: r= 0.91 NO_2: r= 0.69 CO: r= 0.66 SO_2: r= 0.60	**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 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:** Sep 1996-Mar 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 yr and range =37-77 yr **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, ma calculated from same day and preceding days	**Pollutant:** MC2.5-0.01 **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)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: von Klot et al. (2002, 034706) **Period of Study:** Sep 1996-Mar 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 yr and range =37-77 yr **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, ma calculated from same day and preceding days	**Pollutant:** NC0.1-0.01 **Averaging Time:** 10-min intervals **Mean (SD):** 17,300 /cm^3 Percentiles: 25th: 9286 50th(Median): 16940 75th: 24484 **Range (Min, Max):** (3272-46195) Unit (i.e. µg/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 $MC_{2.5-0.01}$: r= 0.45 PM_{10}: r= 0.51 NO_2: r= 0.66 CO: r= 0.66 SO_2: r= 0.36	**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 NC0.1-0.01 OR= 1.07 (0.97-1.18) MC0.5-0.1: OR= 1.07 (0.98-1.18) Inhaled corticosteroid use NC0.1-0.01 OR= 1.01 (0.87-1.18) MC0.5-0.1: OR= 1.53 (1.39-1.69) Wheezing NC0.1-0.01 OR= 1.12 (1.01-1.24) MC0.5-0.1: 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 NC0.1-0.01 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 NC0.1-0.01 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)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			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) 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:** Sep 1996-Mar 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 yr and range =37-77 yr **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, ma calculated from same day and preceding days	**Pollutant:** NC0.5-0.1 **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. µg/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 NC0.5-0.1 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 NC0.5-0.1 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 NC0.5-0.1 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)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: von Klot et al. (2002, 034706) **Period of Study:** Sep 1996-Mar 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 yr and range =37-77 yr **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, ma calculated from same day and preceding days	**Pollutant:** NC2.5-0.5 **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. µg/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 NC2.5-0.5 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 NC2.5-0.5 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 NC2.5-0.5 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 (0.96-1.10)
Reference: von Klot et al. (2002, 034706) **Period of Study:** Sep 1996-Mar 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 yr and range =37-77 yr **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, ma 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-wk 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 yr olds **Study Design:**	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean (SD):** Winter: 12.7 µg/m^3 Summer: 12.3 µg/m^3 **Range (Min, Max):**	**PM Increment:** Winter: 12.3 µg/m^3 Summer: 6.3 µg/m^3 Mean (PEF l/min) [Lower CI, Upper CI] lag: **Winter morning:** 0.80 [-1.97, 3.67] lag 0 0.62 [-2.22, 3.54] lag 1

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
	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- Jul 14) **Dose-response Investigated?** No **Statistical Package**: Nr **Lags Considered:** Lag 0, lag 1, lag 2, lag 3, 7-day ma	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)	-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] lag 0 -0.99 [-4.69, 2.72] lag 1 -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] lag 1 2.50 [0.28, 4.72] lag 2 3.41 [1.40, 5.44] lag 3 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] lag 2 -0.314 [-0.846 , 0.216] lag 3 **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 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

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			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 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-wk 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 yr 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- Jul 14) **Dose-response Investigated?** No **Statistical Package**: Nr **Lags Considered:** Lag 0, lag 1, lag 2, lag 3, 7-day ma	**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] lag **Winter morning:** -1.75 [-4.00, 0.50] lag 0 -0.91 [-3.44, 1.62] lag 1 -0.62 [-3.16, 1.91] lag 2 -1.82 [-4.27, 0.64] lag 3 -3.22 [-8.03, 1.58] 7-day mean **Winter afternoon:** 0.99 [-1.58, 3.55] lag 0 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] lag 1 1.35 [-1.27, 3.97] lag 2 3.38 [1.03, 5.72] lag 3 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] lag 3

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			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 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 |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

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			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 yr 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- Jul 14) **Dose-response Investigated?** No **Statistical Package**: Nr **Lags Considered:** Lag 0, lag 1, lag 2, lag 3, 7-day ma	**Pollutant:** NO_3 **Averaging Time:** 24 h **Mean (SD):** Winter: 3.6 µg/m^3 Summer: 3.5 µg/m^3 **Range (Min, Max):** Winter: 0.1, 29.9 Summer: 0.7, 13.2 **Monitoring Stations**: 2 stations	**PM Increment:** Winter: 6.7 µg/m^3 Summer: 3.7 µg/m^3 Mean (PEF l/min) [Lower CI, Upper CI] lag: **Winter morning:** -2.08 [-4.02 to -0.15] lag0 -0.64 [-2.87, 1.59] lag 1 0.71 [-1.69, 3.11] lag 2 -1.38 [-3.61, 0.84] lag 3 -0.92 [-5.32, 3.47] 7-day mean **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 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 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

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			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] 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] 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- Dec 2004 **Location:** Copenhagen, Denmark	**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 yr (RD combined), 5-18 yr (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 yr 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 avg (lag 0-4) for RD, and a 6-day avg (lag 0-5) for asthma.	**Pollutant:** PM_{10} ($\mu g/m^3$) **Averaging Time:** 24 h **Mean (SD):** 24(14) **Median:** 21 **IQR:** 16-29 **99th percentile:** 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 : r = 0.32 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 (IQR) **Relative risk (RR) Estimate [CI]:** **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 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 Fig form (see notes): **Notes:** Fig 2: Relative risks and 95% confidence intervals per IQR in single day concentration (0-5 day lag). Summary of Fig 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:** Jan 1999-Jun 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 yr old **Study Design:** Time series **N:** 42,667 admissions **Statistical Analyses:** GEE for multivariable modeling **Covariates:** Season, serial correlation, yr, 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 h and 1 wk **Mean (min-max):** Daily: 27.6 (2-421) Weekly: 25.3 (5.0-116.0) **Monitoring Stations:** NR **Copollutant:** Daily $PM_{2.5}$ ρ = 0.25 (p < 0.01) Weekly $PM_{2.5}$ ρ = 0.08 (p = 0.21)	**PM Increment:** 10 μg/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:** 24 h **Mean (SD) Unit:** Index Days: 111.68 ± 38.32 μg/m^3 Comparison Days: 55.43 ± 24.66 μg/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:** 24 h **Mean Unit:** 49.47 μg/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 ≥ 23º C: 1.11 (1.08-1.14) Temperature < 23º C: 1.09 (1.07-1.11) Adjusted for SO_2 Temperature ≥ 23º C: 1.10 (1.08-1.13) Temperature < 23º C: 1.19 (1.17-1.22) Adjusted for NO_2 Temperature ≥ 23º C: 0.90 (0.88-0.93) Temperature < 23º C: 1.09 (1.07-1.12) Adjusted for CO Temperature ≥ 23º C: 1.03 (1.00-1.05) Temperature < 23º C: 1.07 (1.05-1.10) Adjusted for O_3 Temperature ≥ 23º C: 1.05 (1.03-1.08) Temperature < 23º 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 yr **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: 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 yr **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 ma 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 ma 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 ma 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:** June 1995-Mar 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 days from case date), Dewanji and Moolgavkar (DM) method **Covariates:** Long-term trends, day-of-the-week effect, weather **Season:** All yr **Dose-response Investigated?** No **Statistical Package:** SPlus, R **Lags Considered:** 0-7 days	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** 13.31(6.13) µg/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 µg/m^3 Rr Estimate (65+ Yr) Dm Method: 1.014[0.998,1.029] Lag 0 1.016[0.998,1.034] 3-day avg 0.988[0.970, 1.006] 5-day avg 0.983[0.963, 1.004] 7-day avg Time Series: 1.016[0.999, 1.033] Lag 0 1.015[0.996, 1.035] 3-day avg 1.009[0.987, 1.032] 5-day avg 1.009[0.983, 1.036] 7-day avg Case-Crossover: 1.017[0.998, 1.036] Lag 0 1.015[0.993, 1.037] 3-day avg 1.008[0.984, 1.033] 5-day avg 1.003[0.976, 1.031] 7-day avg
Reference: Fung al. (2005, 093262) **Period of Study:** Nov 1995-Dec 2000 **Location:** London, Ontario	Hospital Admissions **Outcome (ICD-9):** Asthma (493) and all other respiratory diseases (460-519) **Age Groups:** <65 yr 65+ yr **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 µg/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) **Period of Study:** 1995-1998 **Location:** Madrid, Spain	Hospital Admissions **Outcome (ICD):** Asthma (493) **Age Groups:** All ages **Study Design:** Time series **N:** 555,153 at-risk **Statistical Analyses:** GAM, autoregressive Poisson regression **Covariates:** Temperature, relative humidity, pollen, yr, day of the week, public holiday **Season:** NR **Dose-response Investigated?** No **Statistical Package:** S-Plus **Lags Considered:** 0, 1, 2, 3, and 4 days	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (min-max):** 32.1 (11.2-108.6) SD = 12.1 **Monitoring Stations:** 13 **Copollutant (correlation):** SO_2: r = 0.581 NO_2: r = 0.717 O_3: r = -0.188 **Other variables:** O.europaea: r = -0.066 Plantago sp.: r = -0.202 Poaceae: r = -0.132 Urticaceae: r = -0.104 Temp: r = -0.122 Humidity: r = 0.119	**PM Increment:** 10 µg/m^3 **RR Estimate [CI]:** Single-pollutant Current-day lag: 1.011 (0.980-1.042) 1-day lag: 1.006 (0.976-1.037) 2-day lag: 1.008 (0.978-1.038) 3-day lag: 1.039 (1.010-1.068) 4-day lag: 1.027 (0.999-1.056) Adjustment for pollen (PM_{10} 3-day lag) O. europaea: 1.041 (1.011-1.071) Plantago sp.: 1.046 (1.017-1.076) Poaceae: 1.043 (1.015-1.073) Urticaceae: 1.038 (1.009-1.068) All four: 1.045 (1.016-1.074)
Reference: Hajat et al. (2002, 030358) **Period of Study:** Jan 1992-Dec 1994 **Location:** London, England	Family Practice consultations **Outcome:** Upper Resp Disease (excluding allergic rhinitis) (460-3), (465), (470-5), (478) **Age Groups:** 0-14, 15-64, >65 yr **Study Design:** Time series **N:** 268,718-295,740 registered patients **Statistical Analyses:** Poisson regression, GAM, LOESS smoothers, default convergence criteria **Covariates:** Long term trends, pollen counts, flu, meteorological variables **Season:** All yr **Dose-response Investigated?** No **Statistical Package:** SPLUS **Lags Considered:** 2-3	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (SD):** 28.5 (13.7) µg/m^3 Percentiles: 10th: 15.8 90th: 46.5 **Monitoring Stations:** 1 **Copollutant:** NR	**PM Increment:** All Year: 18 Warm Season: 15 Cold Season: 20 **% Change, Single Pollutant Models:** All Year: Ages 0-14: 2.0[-0.2, 4.2] Lag 3 Ages 15-64: 5.7[2.9, 8.6] Lag 2 Ages >65: 10.2[5.3, 15.3] Lag 2 Warm Season: Ages 0-14: 1.1[-2.4, 4.8] Lag 3 Ages 15-64: 6.0[2.7, 9.4] Lag 2 Ages >65: 0.1[-7.7, 8.5] Lag 2 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 Yr 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 Yr 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 Yr 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 (Apr-Nov of each yr) **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:** 24 h **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 *Fig 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 (Apr-Nov of each yr) **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, yrly population **Season:** Apr-Nov (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	**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:** **Fig 3:** Associations between hospitalizations for non-indigenous and indigenous people with estimated ambient PM_{10}. **Summary of Fig 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 1997-Dec 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, fall, winter, spring **Dose-response Investigated?** No **Statistical Package:** SPSS **Lags Considered:** NR	**Pollutant:** PM_{10} **Averaging Time:** 1 mo **Mean (SD):** NR **Monitoring Stations:** 1 **Copollutant:** NR **Correlation with COPD:** r = 0.28 p = 0.03 Adj for temp: r = 0.16 p = 0.23	**PM Increment:** NR **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 yr, 15-64, 65+ yr **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 µg/m^3 (14.0) **Range (Min, Max):** 33.3, 83.1 µg/m^3 **PM Component:** **Monitoring Stations:** 59 **Notes:** Number Of stations estimated from fig. **Copollutant:** NR	**PM Increment:** 10% Increase In PM_{10} (5.9 µg/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: Jaffe et al. (2003, 041957) **Period of Study:** July 1991-June 1996 **Location:** Cincinnati, Cleveland, Columbus, Ohio	ED visits **Outcome (ICD10):** Asthma (493) **Age Groups:** Age 5-34 yr **Study Design:** Time-series **N:** 4,416 recipients **Statistical Analyses:** Poisson regression, GAM **Covariates:** City, day of week, wk, yr, minimum temperature, dispersion parameter **Season:** Jun-Aug only **Dose-response Investigated?** Yes **Statistical Package:** NR **Lags Considered:** 0-3 days	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (SD):** Cincinnati: 43.0(16.4) Cleveland: 60.8(28.4) Columbus: 37.4(16.3) **Range (Min, Max):** Cincinnati: (16,90) Cleveland: (12,183) Columbus: (7,87) **Monitoring Stations:** 3 **Copollutant (correlation):** Cincinnati: PM_{10} O_3 r = 0.42 NO_2 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	**PM Increment:** 50 µg/m^3 **% Change** Asthma Cincinnati: -22%[-49,-19] Lag 3 Cleveland: 12%[0,27] Lag 2 Columbus: 32%[-6,-85] Lag 3 **Ar Estimate [Lower Ci, Upper Ci]** **Lag:** Asthma Cincinnati: PM_{10}: Nr Cleveland: PM_{10}: 1.32 Columbus: PM_{10}: 3.62 **Notes:** Dose response was investigated by assessing the relationship between odds of ed visit by quintile of PM_{10}. Results are displayed in Fig. "no consistent effects for all three cities were observed for PM_{10}." Rate ratios were also reported for each city.

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Jalaludin et al. (2004, 056595) **Period of Study:** Feb-Dec 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:** Fall (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 (µg/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 (Apr-Nov of each yr) **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:** Apr-Nov (dry season) **Dose-response Investigated?** No **Statistical package:** NR **Lags Considered:** 0-3 days	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Median:** 17.4 **IQR:** 13.6-22.3 **10-90th Percentile:** 10.3-27.7 **Range:** 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: Fig 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 Fig 1 results:** Marginally significant positive association at Lag 0 in overall study population. Larger marginally significant positive association among indigenous people. **Fig 2**: OR and 95% CI for hospital admissions for COPD. **Summary of Fig 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. **Fig 3:** OR and 95% CI for hospital admissions for asthma. **Summary of Fig 3 results:** Positive, non-significant (sometime marginally significant) associations at Lag 0, Lag 2, and Lag 3 for overall population and indigenous status strata. **Fig 4:** OR and 95% CI for hospital admissions for respiratory infections. **Summary of Fig 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) µg/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 µg/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: Jan** 2000-Dec 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 yr, 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) µg/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 µg/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:** Jan 2000–Dec 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 yr, 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) µg/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 µg/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 yr **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 µg/m^3-referent PM_{10} >65.9 µg/m^3 Crude OR: 0.837 Adj OR: 0.947 95% CI: (0.640,1.401)
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 yr, 19+ yr **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	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Lee et al. (2002, 034826) **Period of Study:** Dec 1997-Dec 1999 **Location:** Seoul, Korea	Hospital Admissions **Outcome (ICD10):** Asthma, J45, J46, **Age Groups:** Children <15 yr **Study Design:** Time-Series **N:** 822 days, 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 ma for 1-2, 2-3, and 3-4 days	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (SD):** 64.0 (31.8) µg/m^3 **Percentiles: 25th:** 40.5 µg/m^3 **50th(Median):** 59.1 µg/m^3 **75th:** 80.9 µg/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 µg/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 yr.
Reference: Lee et al. (2006, 090176) **Period of Study:** Jan 1997-Dec 2002 **Location:** Hong Kong, China	**Hospital Admission** **Outcome:** Asthma (493) **Age Groups:** <18 yr **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 h **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 yr **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 avg	**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 µg/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	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Lin et al, (2008, 126812) **Period of Study:** 1991-2001 **Location:** New York State, U.S.	**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 yr	**Pollutant:** O_3 (PM_{10} is secondary) **Averaging Time:** 24 h **Mean (SD) Unit:** 19.56 (10.92) µg/m^3 **Range (Min, Max):** 1.0, 90.00 **Copollutant (correlation):** Given in Fig 3	All PM_{10} results are given in Fig 3
Reference: Lin et al. (2002, 026067) **Period of Study:** Jan 1981-Dec 1993 **Location:** Toronto	Hospital Admissions **Outcome (ICD-9):** Asthma (493) **Age Groups:** 6-12 yr **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 avg	**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 µg/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 yr **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 h **Mean (SD):** 33.4 µg/m^3, (13.7) **Range (Min, Max):** 6, 109 µg/m^3 **Monitoring Stations:** 24 **Notes: Copollutant (correlation):** PM_{10}-SO_2: 0.532 PM_{10}-O_3: -0.289 PM_{10}-: 0.721 PM_{10}-NO_2: 0.711	**PM Increment:** 10 µg/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 µg/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: A**ll, 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 max **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 µg/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 yr **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 day ma	**Pollutant:** PM_{10} **Averaging Time:** Daily **Mean (SD):** 60.0 µg/m^3, (26.3) **Range (Min, Max):** 22.8. 186.5 µg/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 µg/m^3 Regression Coefficients (SE): 0.0024 (0.0023), 6 day ma **Notes:** % Increase (SD) for ER visits per 2435 µg/m^3 (IQR) PM_{10} (lag 6 day 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 yr, 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 µg/m^3 (IQR) **% Increase [CI]:** Respiratory Admissions (2-day lag) 0-14 yr: 3.62 [2.34,4.90] 15-64 yr: 3.39 [1.85,4.93] 65+ yr: 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 U.S. Cities	**Outcome:** 490-496, except 493 (COPD), 480-487 (Pneumonia) **Age Groups:** 65 + (U.S. 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 µg/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 µg/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 mo **Age Groups: >**18 yr **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 µg/m^3 **OR Estimate [CI]:** All Adults: 1.08 [0.82,1.43] 18-64 yr: 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 µg/m^3 (annual concentration) All Adults: 1.56 [0.96,2.52] 18-64 yr: 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 yr) **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 µg/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 yr (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 yr (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: Moore et al. (2008, 196685) **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 yr	**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 24 h NO_2: 0.53 24 h CO: 0.36 24 h SO_2: 0.13	Results given are for O_3

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Nascimento et al. (2006, 093247) **Period of Study:** May 2000-Dec 2001 **Location:** São Jose dos Campos, Brazil	Hospital Admissions **Outcome (ICD-10):** Pneumonia (J12-J18) **Age Groups:** 0-10 yr **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 µg/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 yr 7-10 yr 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 µg/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: 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 h **Mean (SD):** 16.8 µg/m^3, (10.2) 1994-1997 16.5 µg/m^3, (10.3) 1998-2000 16.6 , µg/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	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 day , 3 day ma, 0-13 days unconstrained distributed lag	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** 27.9 (12.3) µg/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 µg/m^3 RR Estimate [Lower CI, Upper CI] All Respiratory Outcomes: 1.013 (1.004-1.021), 3 day ma URI: 1.014 (1.004-1.025) , 3 day ma 1.073 (1.048-1.099) , 14-day dist. lag Asthma: 1.009 (0.996-1.022), 3 day 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 day ma 1.087 (1.044-1.132), 14-day dist. lag COPD: 1.018 (0.994-1.043), 3 day 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: Ren et al. (2006, 092824) **Period of Study:** Jan 1996-Dec 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:** Mar 2006-Mar 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 yr **N:** 1050 adult COPD admissions	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (SD) Unit:** Control: 19.87 (8.51) µg/m^3 Case: 20.47 (9.27) µg/m^3 **Range (Min, Max):** Control: 9.77-34.27 Case: 10.04-35.03 **Copollutant (correlation):** NR	**Increment:** 10 µg/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	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 yr, 18+ yr (asthma) All ages (URI//LRI) **Study Design:** Times series **N:** 25 mo 260,000-275,000 health plan members (Aug 1998-Aug 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-day ma (0-2, 2-5, 6-8)	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** PM_{10} mass-29.03 µg/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 day LRI: 1.074 (S), 3-5 day 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:** Jan 1995-Jun 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+ yr 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 day	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Range (90% of concentrations):** 7.9-41.9 µg/m^3 **Monitoring Stations:** 1 **Notes: Copollutant (correlation):** PM_{10} PM1 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 µg/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	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Sun et al. (2006, 090768) **Period of Study:** Jan 2004-Dec 2004 **Location:** Taichung, Taiwan (Central Taiwan)	ED visits **Outcome:** Asthma (493.xx) **Age Groups:** <55, <16, 16-55 yr **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 µg/m^3 (NR) (estimated from Fig)* **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: Szyszkowicz (2007, 092829) **Period of Study:** Jan 1992-Mar 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:** 24 h **Mean (SD) Unit:** 22.6 (13.1) µg/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 yr Apr-Sep, All: 3.7 (1.5-6.0) Apr-Sep, Female: 4.5 (1.8-7.3) Apr-Sep, Male: 3.3 (0.1-6.7) 2 day lag, < 10 yr Year round, All: 2.7 (0.1-5.4) Apr-Sep, All: 6.3 (2.6-10.2) Apr-Sep, Male: 7.4 (3.1-11.9) 2 day lag, ≥ 10 yr Apr-Sep, All: 2.4 (0.1-4.7) Apr-Sep, Female: 3.9 (1.1-6.7)
Reference: Tecer et al, (2008, 180030) **Period of Study:** Dec 2004-Oct 2005 **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 yr	**Pollutant:** PM_{10} **Averaging Time:** NR **Mean, Unit:** 53.3 µg/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 µg/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) **Period of Study:** 1993-2004 **Location:** Atlanta Metropolitan area, Georgia	Hospital Admissions/ED visits **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 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 ma(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 µg/m^3 (IQR) **Risk ratio [95% CI]:** **Single pollutant models:** RD: 1.015 (1.006-1.024) **Notes:** Results of selected multi-pollutant models for respiratory disease are presented in Fig 2. **Fig 2:** PM_{10} adjusted for CO, O_3, NO_2, or NO_2/O_3 (nonwinter months only) **Summary of results:** PM_{10} remained predictive of RD in non-winter months after adjustment for pollutants.
Reference: Tsai et al. (2006, 089768) **Period of Study:** 1996-2003 **Location:** Kaohsiung City, Taiwan	**Outcome:** Asthma (493) **Age Groups:** All (universal health care covers >96% of the population) **Study Design:** Case crossover **N:** 17,682 admissions 63 hospitals **Statistical Analyses:** Conditional Logistic Regression **Covariates:** Temperature, humidity **Season:** Warm and cool seasons **Dose-response Investigated?** No **Statistical Package:** SAS **Lags Considered:** 0-2 day cumulative	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** 76.62 µg/m^3 (NR) **Percentiles:** 25th: 41.73 50th(Median): 74.40 75th: 104.01 **Range (Min, Max):** (16.70, 232.00) **Monitoring Stations:** 6 **Copollutant:** NR	**PM Increment:** 62.28 µg/m^3 **OR Estimate [Lower CI, Upper CI]** **lag:** **Single-pollutant model, 0-2 day cumulative lag** ≥ 25oC: 1.302 [1.155, 1.467] <25oC: 1.556 [1.398, 1.371] **Two-pollutant models, 0-2 day cumulative lag** PM_{10} w/ SO_2 ≥ 25oC: 1.305 [1.156, 1.473] <25oC: 1.540 [1.374, 1.727] PM_{10} w/ O_3 ≥ 25oC: 0.985 [0.842, 1.152] <25oC: 1.581 [1.402, 1.783] 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:** Nov 1994-Mar 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:** 0-4 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 µg/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 µg/m^3 Single Pollutant Models: TS2: 23.2 µg/m^3 Multipollutant Models: TS1/TS2: 50 µg/m^3 **Mean Percentage Change, lag 0** TS 1: Single Pollutant All-age (all yr): 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 yr): 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 yr) 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: Vegni and Ros (2004, 087448) **Period of Study:** Sep 2001-Sep 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, fall, winter **Dose-response Investigated?** No **Statistical Package:** STATA v. 5 **Lags Considered:** 0, 1, and 2 days	**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 Fall: 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 µg/m^3 summer: 30 µg/m^3 Fall: 93 µg/m^3 Winter: 115 µg/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	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Vigotti et al. (2007, 090711) **Period of Study: Jan** 2000-Dec 2000 **Location:** Pisa, Italy	**ED Visits** **Outcome:** Asthmatic attack (493), dry cough (468), acute bronchitis (466) **Age Groups:** <10 yr; 65+ yr **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 yr **Dose-response Investigated?** No **Statistical Package:** NR **Lags Considered:** 0-5 days	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (SD):** 35.4 (15.8) µg/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 µg/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: Xirasagar et al. (2006, 093267) **Period of Study:** 1998-2001 **Location:** Taiwan	**Hospital Admission/ED:** **Outcome:** Asthma or Asthmatic Bronchitis (493) **Age Groups:** <2 yr old, 2~5 yr old, 6~14 yr old **Study Design:** **N:** 27, 275 pediatric hospitalizations **Statistical Analyses:** ARIMA Modeling Spearman's Correlations **Covariates:** Season, ambient temp., rel. humidity, atmospheric pressure, rainfall, h of sunshine **Season:** Spring: Feb-Apr Summer: May-Jul Fall: Aug-Oct Winter: Nov-Jan **Dose-response Investigated?** No **Statistical Package:** EViews 4 **Lags Considered:** NR	**Pollutant:** PM_{10} **Averaging Time:** Monthly means **Mean (SD):** 24.4 µg/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):** <2 yr old: r = 0.315 2~5 yr old: r = 0.589 6~14 yr 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	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
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 µg/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: 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 µg/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	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
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 yr **Dose-response Investigated?** No **Statistical Package:** SAS **Lags Considered:** 0-2	**Pollutant:** PM_{10} **Averaging Time:** NR **Mean (SD):** 48.99 µg/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 µg/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: 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 µg/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 µg/m^3 **OR Estimate [Lower CI, Upper CI]** Single-Pollutant Model (0-2 day 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	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Yang et al. (2004, 087488) **Period of Study:** Jun 1995-Mar 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 yr **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 yr, 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 µg/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 µg/m^3 unless otherwise specified.

Table E-13. Short-term exposure-respiratory-ED/HA-$PM_{10-2.5}$.

Reference	Design & Methods	Concentrations1	Effect Estimates (95% CI)
Reference: Chen et al. (2005, 087555) **Period of Study:** Jun 1995-Mar 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 yr **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:** $PM_{10-2.5}$ (µg/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 µg/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	Concentrations1	Effect Estimates (95% CI)
Reference: Fung et al. (2006, 089789) **Period of Study:** Jun 1995-Mar 1999 **Location:** Vancouver, Canada	**Hospital Admission/ED:** Hospital Admission **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 days from case date), Dewanji and Moolgavkar (DM) method **Covariates:** Long-term trends, day-of-the-week effect, weather **Season:** All yr **Dose-response Investigated?** No **Statistical Package:** SPlus, R **Lags Considered:** 0-7 days	**Pollutant:** $PM_{10-2.5}$ ($\mu g/m^3$) **Averaging Time:** 24-h avg **Mean (SD)** 5.6(3.88) $\mu g/m^3$ **Range (Min, Max):** (-2.9, 27.07) **Monitoring Stations:** NR **Notes: Copollutant (correlation):** $PM_{10-2.5}$ PM_{10} r = 0.83 $PM_{2.5}$ r = 0.34 CO r = 0.51 CoH r = 0.61 O_3 r = -0.11 NO_2 r = 0.52 SO_2 r = 0.57	**PM Increment: :** 4.3 $\mu g/m^3$ **RR Estimate** (65+ yr) DM method: 1.011[0.998,1.024] lag 0 1.016[1.0,1.032] 3-day avg 1.020[1.001,1.039] 5-day avg 1.020[0.998,1.042] 7-day avg Time series: 1.0168[1.003, 1.031] lag 0 1.020[1.003, 1.037] 3-day avg 1.019[0.999, 1.039] 5-day avg 1.018[0.994, 1.042] 7-day avg Case-crossover: 1.019[1.003, 1.034] lag 0 1.019[1.009, 1.038] 3-day avg 1.020[0.999, 1.042] 5-day avg 1.018[0.994, 1.043] 7-day avg
Reference: Halonen et al. (2009, 180379) **Period of Study:** 1998-2004 **Location:** Helsinki, Finland	**Outcome:** Hospital Admissions **Age Groups:** 65+ yr **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 & 5-day (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, PM0.03-0.1, PM<0.1, PM<0.10.29, $PM_{2.5}$, CO, NO_2 **Co-pollutant Correlation** PM<0.03: 0.14 PM0.03-0.1: 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-day 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-day 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-day 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-day mean: 0.24 (-3.62, 4.26) *p < 0.05, ‡p < 0.10

Reference	Design & Methods	Concentrations1	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 yr, 15-64 yr, and ≥ 65 yr 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 µg/m3 (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 µg/m^3 , and an 18.8 µg/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 µg/m^3 increase): 0-14 yr: 6.2% [0.4, 12.3] 15-64 yr: 2.6% [-0.5, 5.8] ≥ 65 yr: 1.9% [-1.9, 5.9] For all respiratory diseases (18.8 µg/m^3 increase): 0-14 yr: 12.0 [0.8, 24.3] 15-64 yr: 5.0 [-0.9, 11.1] ≥ 65 yr: 3.7 [-3.6, 11.4] For respiratory infections (10 µg/m^3): All ages: 4.4% [0.9, 8.0] For respiratory infections (18 µg/m^3): All ages: 8.4% [1.7, 15.5]
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 yr **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- to 7-day avg	**Pollutant:** $PM_{10-2.5}$ (µg/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 µg/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	Design & Methods	Concentrations1	Effect Estimates (95% CI)
Reference: Lin et al. (2002, 026067) **Period of Study:** Jan 1981-Dec 1993 **Location:** Toronto	Hospital Admissions **Outcome (ICD-9)**: Asthma (493) **Age Groups:** 6-12 yr **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- to 7-day avg	**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 day 3-7, yielding similar results
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 days, 3-day ma, 0-13 days 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	Design & Methods	Concentrations1	Effect Estimates (95% CI)
Reference: Peng et al. (2008, 156850) **Period of Study:** Jan 1999-Dec 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 + yr, 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 yr 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 U.S.: 9.1 (6.6-13.1) Counties in Western U.S.: 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 µg/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: Fig 3: Percentage change in emergency hospital admissions for RD per 10 µg/m^3 increase in PM (single pollutant model and model adjusted for $PM_{2.5}$ concentration) Fig 4: Percentage change in emergency hospital admissions rate for CVD and RD per a 10 µg/m^3 increase in $PM_{10-2.5}$ (0-2 day lags, Eastern vs.. Western USA)
Reference: Slaughter et al. (2005, 073854) **Period of Study:** Jan 1995-Jun 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+ yr 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 days	**Pollutant:** $PM_{10-2.5}$ (µg/m^3) **Averaging Time:** 24 h avg **Range (90% of Concentrations):** Reported for $PM_{2.5}$ and PM_{10} only **Monitoring Stations:** 1 **Copollutant (correlation):** $PM_{10-2.5}$ PM1 r = 0.19 $PM_{2.5}$ r = 0.31 PM_{10} r = 0.94 CO r = 0.32 Temperature r = 0.11	**PM Increment:** 25 µg/m^3 **RR Estimate [Lower CI, Upper CI] lag:** ER visits: $PM_{10-2.5}$ All Respiratory Lag 1: 1.01 [0.98, 1.04] Lag 2: 1.01 [0.98, 1.04] Lag 3: 1.02 [0.99, 1.05] 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] COPD (adult) Lag 1: 1.01 [0.93, 1.09] Lag 2: 0.98 [0.90, 1.06] Lag 3: 1.02 [0.95, 1.10]

Reference	Design & Methods	Concentrations1	Effect Estimates (95% CI)
Reference: Tecer et al. (2008, 180030) **Period of Study:** Dec 2004-Oct 2005 **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 yr	**Pollutant:** $PM_{10-2.5}$ **Averaging Time:** NR **Mean, Unit:** 24.3 µg/m^3 **Range (Min, Max):** 4, 195.8 **Copollutant (correlation):** $PM_{2.5}/PM_{10-2.5}$ Mean: 1.49 Range: 0.21, 7.53	**Increment:** 10 µg/m^3 **Odds Ratio (95% CI)** Asthma Lag 0: 1.18 (1.01-1.39) Lag 1: 0.92 (0.78-1.08) Lag 2: 0.98 (0.84-1.15) Lag 3: 1.11 (0.97-1.27) 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: Yang et al., (2004, 087488) **Period of Study:** Jun 1995-Mar 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 yr **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 yr, 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:** $PM_{10-2.5}$ (µg/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 µg/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 µg/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-Dec 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 yr (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 yr 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/m3 (SD):** 10(5) **Median:** 9 **IQR:** 7-12 **99th percentile:** 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 NCa212: r = 0.82 PM_{10}: r = 0.80 CO: r = 0.46 NO_2: r = 0.42 : 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 Fig 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, 093250) **Period of Study:** Oct 2001-Sep 2004 **Location:** Washington, DC	ED Visit/Admissions **Outcome:** Asthma-493 **Age Groups:** 1-17 yr,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 h **Mean:** "low, never reached code red" **Percentiles:** NR **Range (Min, Max):** NR **Monitoring Stations:** 3 **Copollutant (correlation):** NR	**PM Increment:** 1 µg/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 O_3 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 yr, 1-4 yr, and 5-14 yr **Study Design:** Matched case-crossover **N:** ~2.4 million children <15 yr 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 h **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 µg/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 yr (B,M,P,S): 2.4 [0.1,4.7] Matched Multipollutant Model 1-4 yr with 1-h SO_2 (B,S): 1.9 [-1.7,5.6] 1-4 yr 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 yr (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 yr with PM_{10} (B,M,P,S): 2.9 [0.2,5.6] 1-4 yr with 1-h SO_2 (B,S): 1.3 [-1.8,4.4] 1-4 yr with 1-h NO_2 (B,M,P,S): -1.5 [-3.2,0.2] 1-4 yr 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 µg/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 U.S. 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) **Covariates:** Day of the week, temperature, dew point temperature, temporal trends, indicator for persons 75+ yr, population size **Season:** All, Jun-Aug (Summer), Sep-Nov (Fall), Dec-Feb (Winter), Mar-May (Spring) **Dose-response Investigated:** No	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean (µg/m3):** Descriptive information presented in Fig S2 (boxplots): IQR: 8.7 µg/m^3 **Monitoring Stations:** NR **Copollutant (correlation):** NR	**PM Increment:** 10 µg/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] 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]

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
	Statistical Package: NR **Lags Considered:** 0-2 day lags		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 (fall, national): 0.02 [-0.63-0.67] Lag 0 (fall, northeast): -0.01 [-0.87-0.85] Lag 0 (fall, southeast): -0.58 [-2.06-0.91] Lag 0 (fall, northwest): -1.38 [-11.84-10.32] Lag 0 (fall, 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 (fall): 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 (fall, national): 0.39 [-0.22-1.01] Lag 2 (fall, northeast): 0.12 [-0.82-1.07] Lag 2 (fall, southeast): 0.14 [-1.29-1.59] Lag 2 (fall, northwest): -0.74 [-10.08-9.58] Lag 2 (fall, southwest): 0.97[-1.36-3.36]
Reference: Bell et al. (2009, 191007) **Period of Study:** 1999-2005 **Location:** 168 U.S. 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:** 24 h **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 U.S. 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, NH4+, 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: 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) µg/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 µg/m^3 % Change, lag 0-3-day avg URD 6.0 (3.1, 9.1) LRD 5.8 (2.8, 8.9) Asthma 4.4 (-1.3, 10.4)

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Chen et al. (2005, 087555) **Period of Study:** Jun 1995-Mar 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 yr **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 µg/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: Chimonas and Gessner (2007, 093261) **Period of Study:** Jan 1999-Jun 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 yr old **Study Design:** Time series **N:** 42,667 admissions **Statistical Analyses:** GEE for multivariable modeling **Covariates:** Season, serial correlation, yr, weekend, temperature, precipitation, and wind speed **Season:** NR **Dose-response Investigated?** No **Statistical Package:** SPSS (dataset), SAS (analysis) **Lags Considered:** 1 day and 1 wk	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h and 1 wk **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 µg/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: Delfino et al. (2009, 191994) **Period of Study:** Oct 2003-Nov 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) µg/m^3 During Fires: 54.1 (21.0) µg/m^3 After Fires: 15.9 (5.5) µg/m^3 Orange Before Fires: 23.2 (9.6) µg/m^3 During Fires: 64.3 (26.5) µg/m^3 After Fires: 15.5 (10.2) µg/m^3 Riverside Before Fires: 32.7 (14.7) µg/m^3 During Fires: 42.1 (25.5) µg/m^3 After Fires: 16.9 (10.2) µg/m^3 San Bernadino Before Fires: 35.7 (16.6) µg/m^3 During Fires: 45.3 (28.7) µg/m^3 After Fires: 18.5 (8.3) µg/m^3 San Diego Before Fires: 18.5 (6.7) µg/m^3 During Fires: 76.1 (66.6) µg/m^3 After Fires: 14.2 (7.2) µg/m^3 Ventura	**Increment:** 10 µg/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:

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
		Before Fires: 18.4 (8.3) µg/m^3 During Fires: 50.1 (50.5) µg/m^3 After Fires: 12.9 (4.3) µg/m^3 **Copollutant (correlation):** NR	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) Wildfire: 1.086 (1.016-1.162), p = 0.101 Post-Wildfire: 1.057 (0.839-1.332), p = 0.380 Asthma, Ages 0-4, Females: All Periods: 0.937 (0.845-1.040) Pre-Wildfire: 0.880 (0.706-1.099) Wildfire: 1.073 (0.965-1.194), p = 0.116 Post-Wildfire: 0.699 (0.515-0.949), p = 0.214 Asthma, Ages 5-19, Males and Females: All Periods: 1.006 (0.966-1.048) Pre-Wildfire: 1.045 (0.936-1.167) Wildfire: 0.999 (0.935-1.068), p = 0.492 Post-Wildfire: 0.918 (0.788-1.069), p = 0.198 Asthma, Ages 5-19, Males: All Periods: 0.991 (0.935-1.051) Pre-Wildfire: 1.034 (0.892-1.198) Wildfire: 0.969 (0.883-1.064), p = 0.462 Post-Wildfire: 0.979 (0.806-1.189), p = 0.671 Asthma, Ages 5-19, Females: All Periods: 1.026 (0.964-1.092) Pre-Wildfire: 1.065 (0.901-1.260) Wildfire: 1.033 (0.943-1.132), p = 0.768 Post-Wildfire: 0.831 (0.640-1.079), p = 0.136 Asthma, Ages 20-64, Males and Females: All Periods: 1.043 (1.012-

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			1.076) Pre-Wildfire: 1.037 (0.957-1.123) Wildfire: 1.041 (0.995-1.090), p = 0.931 Post-Wildfire: 1.000 (0.882-1.132), p = 0.624 Asthma, Ages 20-64, Males: All Periods: 1.013 (0.954-1.077) Pre-Wildfire: 1.159 (0.996-1.349) Wildfire: 0.939 (0.837-1.053), p = 0.026 Post-Wildfire: 1.275 (1.020-1.595), p = 0.486 Asthma, Ages 20-64, Females: All Periods: 1.052 (1.015-1.090) Pre-Wildfire: 0.995 (0.904-1.096) Wildfire: 1.064 (1.014-1.116), p = 0.247 Post-Wildfire: 0.908 (0.780-1.056), p = 0.310 Asthma, Ages 65-99, Males and Females: All Periods: 1.027 (0.974-1.082) Pre-Wildfire: 0.951 (0.849-1.064) Wildfire: 1.101 (1.030-1.178), p = 0.032 Post-Wildfire: 1.168 (0.967-1.412), p = 0.072 Asthma, Ages 65-99, Males: All Periods: 1.046 (0.957-1.142) Pre-Wildfire: 0.948 (0.804-1.116) Wildfire: 1.185 (1.077-1.305), p = 0.029 Post-Wildfire: 0.902 (0.629-1.294), p = 0.804 Asthma, Ages 65-99, Females: All Periods: 1.018 (0.958-1.081) Pre-Wildfire: 0.947 (0.813-1.102) Wildfire: 1.065 (0.977-1.162), p = 0.195 Post-Wildfire: 1.263 (1.024-1.557), p = 0.032 Acute Bronchitis and Bronchiolitis, All Ages: All Periods: 1.044 (0.990-1.102) Pre-Wildfire: 1.001 (0.890-1.126) Wildfire: 1.096 (1.018-1.179), p = 0.223 Post-Wildfire: 1.031 (0.870-1.222), p = 0.779 Acute Bronchitis and Bronchiolitis, Ages 0-4: All Periods: 1.017 (0.949-1.089) Pre-Wildfire: 0.987 (0.847-1.149) Wildfire: 1.092 (0.997-1.195), p = 0.276 Post-Wildfire: 0.910 (0.700-1.183), p = 0.588 Acute Bronchitis and Bronchiolitis, Ages 5-19: No Convergence Acute Bronchitis and Bronchiolitis, Ages 20-64: All Periods: 1.039 (0.912-1.183) Pre-Wildfire: 1.001 (0.792-1.266) Wildfire: 1.044 (0.872-1.252), p = 0.778 Post-Wildfire: 1.259 (0.921-1.722), p = 0.275 Acute Bronchitis and Bronchiolitis, Ages 65-99: All Periods: 1.134 (1039-1.238) Pre-Wildfire: 1.073 (0.764-1.505) Wildfire: 1.143 (1.032-1.265), p = 0.730 Post-Wildfire: 1.190 (0.865-1.638), p = 0.652 COPD, Ages 20-99: All Periods: 1.018 (0.994-1.042) Pre-Wildfire: 1.007 (0.958-1.058) Wildfire: 1.038 (1.004-1.075), p = 0.320 Post-Wildfire: 1.024 (0.943-1.112),

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			p = 0.728 COPD, Ages 20-64: All Periods: 1.022 (0.980-1.066) Pre-Wildfire: 0.995 (0.916-1.081) Wildfire: 1.068 (1.009-1.131), p = 0.161 Post-Wildfire: 1.015 (0.893-1.153), p = 0.728 COPD, Ages 65-99: All Periods: 1.019 (0.992-1.048) Pre-Wildfire: 1.014 (0.955-1.077) Wildfire: 1.031 (0.990-1.074), p = 0.660 Post-Wildfire: 1.023 (0.928-1.128), p = 0.878 Pneumonia, All Ages: All Periods: 1.009 (0.994-1.024) Pre-Wildfire: 1.045 (0.931-1.180) Wildfire: 1.028 (1.007-1.050), p = 0.420 Post-Wildfire: 0.980 (0.927-1.035), p = 0.045 Pneumonia, Ages 0-4: All Periods: 0.995 (0.944-1.049) Pre-Wildfire: 1.048 (0.931-1.180) Wildfire: 1.018 (0.948-1.092), p = 0.691 Post-Wildfire: 0.823 (0.649-1.044), p = 0.089 Pneumonia, Ages 5-19: All Periods: 1.031 (0.966-1.098) Pre-Wildfire: 1.017 (0.882-1.172) Wildfire: 1.064 (0.990-1.142), p = 0.586 Post-Wildfire: 1.017 (0.767-1.349), p = 0.998 Pneumonia, Ages 20-64: All Periods: 1.008 (0.982-1.035) Pre-Wildfire: 1.041 (0.982-1.104) Wildfire: 1.032 (0.994-1.072), p = 0.823 Post-Wildfire: 1.013 (0.913-1.124), p = 0.633 Pneumonia, Ages 65-99: All Periods: 1.011 (0.993-1.030) Pre-Wildfire: 1.050 (1.006-1.097) Wildfire: 1.029 (1.002-1.057), p = 0.445 Post-Wildfire: 0.985 (0.920-1.055), p = 0.127 **Relative Rate (Min CI, Max CI) in relation to pre-wildfire period (1)** All Respiratory, All Ages: Wildfire, unadjusted for $PM_{2.5}$: 0.961 (0.916-1.008) Wildfire, adjusted for $PM_{2.5}$: 0.903 (0.850-0.960) Post-wildfire, unadjusted for $PM_{2.5}$: 1.143 (1.072-1.219) Post-wildfire, adjusted for $PM_{2.5}$: 1.173 (1.097-1.253) All Respiratory, Ages 0-4: Wildfire, unadjusted for $PM_{2.5}$: 0.865 (0.757-0.989) Wildfire, adjusted for $PM_{2.5}$: 0.842 (0.717-0.988) Post-wildfire, unadjusted for $PM_{2.5}$: 1.152 (0.957-1.388) Post-wildfire, adjusted for $PM_{2.5}$: 1.162 (0.954-1.415) All Respiratory, Ages 5-19: Wildfire, unadjusted for $PM_{2.5}$: 1.098 (0.901-1.324) Wildfire, adjusted for $PM_{2.5}$: 1.087 (0.863-1.370)

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			Post-wildfire, unadjusted for $PM_{2.5}$: 1.373 (1.089-1.732) Post-wildfire, adjusted for $PM_{2.5}$: 1.467 (1.142-1.883) All Respiratory, Ages 20-64: Wildfire, unadjusted for $PM_{2.5}$: 0.991 (0.922-1.066) Wildfire, adjusted for $PM_{2.5}$: 0.923 (0.843-1.012) Post-wildfire, unadjusted for $PM_{2.5}$: 1.074 (0.971-1.188) Post-wildfire, adjusted for $PM_{2.5}$: 1.104 (0.992-1.228) All Respiratory, Ages 65-99: Wildfire, unadjusted for $PM_{2.5}$: 0.932 (0.867-1.003) Wildfire, adjusted for $PM_{2.5}$: 0.874 (0.795-0.959) Post-wildfire, unadjusted for $PM_{2.5}$: 1.147 (1.045-1.259) Post-wildfire, adjusted for $PM_{2.5}$: 1.193 (1.084-1.313) Asthma, All Ages: Wildfire, unadjusted for $PM_{2.5}$: 1.088 (0.965-1.227) Wildfire, adjusted for $PM_{2.5}$: 0.992 (0.856-1.149) Post-wildfire, unadjusted for $PM_{2.5}$: 1.264 (1.085-1.473) Post-wildfire, adjusted for $PM_{2.5}$: 1.336 (1.134-1.573) Asthma, Ages 0-4: Wildfire, unadjusted for $PM_{2.5}$: 0.806 (0.632-1.029) Wildfire, adjusted for $PM_{2.5}$: 1.282 (0.958-1.716) Post-wildfire, unadjusted for $PM_{2.5}$: 1.092 (1.759-1.572) Post-wildfire, adjusted for $PM_{2.5}$: 1.133 (0.777-1.654) Asthma, Ages 5-19: Wildfire, unadjusted for $PM_{2.5}$: 1.254 (0.999-1.575) Wildfire, adjusted for $PM_{2.5}$: 1.282 (0.958-1.716) Post-wildfire, unadjusted for $PM_{2.5}$: 1.564 (1.160-2.109) Post-wildfire, adjusted for $PM_{2.5}$: 1.629 (1.184-2.243) Asthma, Ages 20-64: Wildfire, unadjusted for $PM_{2.5}$: 1.273 (1.067-1.518) Wildfire, adjusted for $PM_{2.5}$: 1.221 (0.979-1.524) Post-wildfire, unadjusted for $PM_{2.5}$: 1.362 (1.043-1.779) Post-wildfire, adjusted for $PM_{2.5}$: 1.486 (1.111-1.987) Asthma, Ages 65-99: Wildfire, unadjusted for $PM_{2.5}$: 0.869 (0.657-1.151) Wildfire, adjusted for $PM_{2.5}$: 0.645 (0.450-0.925) Post-wildfire, unadjusted for $PM_{2.5}$: 0.924 (0.606-1.408) Post-wildfire, adjusted for $PM_{2.5}$: 1.005 (0.650-1.552) Acute Bronchitis and Bronchiolitis, All Ages: Wildfire, unadjusted for $PM_{2.5}$: 1.143 (0.878-1.490) Wildfire, adjusted for $PM_{2.5}$: 0.959 (0.696-1.321)

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			Post-wildfire, unadjusted for $PM_{2.5}$: 1.482 (1.042-2.109) Post-wildfire, adjusted for $PM_{2.5}$: 1.580 (1.089-2.291) Acute Bronchitis and Bronchiolitis, Ages 0-4: Wildfire, unadjusted for $PM_{2.5}$: 1.128 (0.819-1.555) Wildfire, adjusted for $PM_{2.5}$: 0.899 (0.607-1.333) Post-wildfire, unadjusted for $PM_{2.5}$: 1.520 (0.947-2.440) Post-wildfire, adjusted for $PM_{2.5}$: 1.547 (0.954-2.507) Acute Bronchitis and Bronchiolitis, Ages 5-19 No Correlation Acute Bronchitis and Bronchiolitis, Ages 20-64: Wildfire, unadjusted for $PM_{2.5}$: 1.350 (0.688-2.648) Wildfire, adjusted for $PM_{2.5}$: 1.320 (0.608-2.863) Post-wildfire, unadjusted for $PM_{2.5}$: 2.454 (1.068-5.640) Post-wildfire, adjusted for $PM_{2.5}$: 2.515 (1.055-5.998) Acute Bronchitis and Bronchiolitis, Ages 65-99: Wildfire, unadjusted for $PM_{2.5}$: 1.166 (0.643-2.115) Wildfire, adjusted for $PM_{2.5}$: 0.934 (0.422-20.66) Post-wildfire, unadjusted for $PM_{2.5}$: 0.911 (0.428-1.942) Post-wildfire, adjusted for $PM_{2.5}$: 0.997 (0.439-2.262) COPD, Ages 20-99: Wildfire, unadjusted for $PM_{2.5}$: 0.988 (0.875-1.115) Wildfire, adjusted for $PM_{2.5}$: 0.913 (0.779-1.069) Post-wildfire, unadjusted for $PM_{2.5}$: 1.043 (0.885-1.228) Post-wildfire, adjusted for $PM_{2.5}$: 1.064 (0.897-1.262) COPD, Ages 20-64: Wildfire, unadjusted for $PM_{2.5}$: 0.967 (0.779-1.201) Wildfire, adjusted for $PM_{2.5}$: 0.873 (0.660-1.156) Post-wildfire, unadjusted for $PM_{2.5}$: 1.175 (0.862-1.601) Post-wildfire, adjusted for $PM_{2.5}$: 1.311 (0.954-1.802) COPD, Ages 65-99: Wildfire, unadjusted for $PM_{2.5}$: 1.002 (0.869-1.156) Wildfire, adjusted for $PM_{2.5}$: 0.926 (0.767-1.117) Post-wildfire, unadjusted for $PM_{2.5}$: 0.985 (0.811-1.196) Post-wildfire, adjusted for $PM_{2.5}$: 0.981 (0.798-1.206) Pneumonia, All Ages: Wildfire, unadjusted for $PM_{2.5}$: 0.943 (0.868-1.025) Wildfire, adjusted for $PM_{2.5}$: 0.888 (0.799-0.986) Post-wildfire, unadjusted for $PM_{2.5}$: 1.294 (1.158-1.446) Post-wildfire, adjusted for $PM_{2.5}$: 1.318 (1.174-1.479)

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			Pneumonia, Ages 0-4: Wildfire, unadjusted for $PM_{2.5}$: 0.938 (0.705-1.247) Wildfire, adjusted for $PM_{2.5}$: 0.951 (0.678-1.333) Post-wildfire, unadjusted for $PM_{2.5}$: 1.458 (0.974-20182) Post-wildfire, adjusted for $PM_{2.5}$: 1.374 (0.885-2.133) Pneumonia, Ages 5-19: Wildfire, unadjusted for $PM_{2.5}$: 0.891 (0.604-1.312) Wildfire, adjusted for $PM_{2.5}$: 0.830 (0.541-1.272) Post-wildfire, unadjusted for $PM_{2.5}$: 0.960 (0.588-1.569) Post-wildfire, adjusted for $PM_{2.5}$: 0.969 (0.578-1.624) Pneumonia, Ages 20-64: Wildfire, unadjusted for $PM_{2.5}$: 0.927 (0.795-1.081) Wildfire, adjusted for $PM_{2.5}$: 0.837 (0.690-1.016) Post-wildfire, unadjusted for $PM_{2.5}$: 1.314 (1.064-1.622) Post-wildfire, adjusted for $PM_{2.5}$: 1.300 (1.047-1.615) Pneumonia, Ages 65-99: Wildfire, unadjusted for $PM_{2.5}$: 0.959 (0.861-1.068) Wildfire, adjusted for $PM_{2.5}$: 0.899 (1.782-1.033) Post-wildfire, unadjusted for $PM_{2.5}$: 1.277 (1.102-1.481) Post-wildfire, adjusted for $PM_{2.5}$: 1.331 (1.142-1.552)
Reference: Dominici et al. (2006, 088398) **Period of Study:** 1999-2002 **Location:** 204 U.S. 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 yr **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	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
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 yr 65-74 yr >75 yr **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 yr), 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 µg/m^3 **Percentage Change in Hospital Admission Rates [PI]:** COPD-Same day All >65: 0.91 [0.18,1.64] 65-74 yr: 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 yr: 0.93 [0.04,1.82] >75: 0.92 [0.32,1.53] **Notes:** Other lag data shown in Fig 2-4
Reference: Erbas et al. (2005, 073849) **Period of Study:** Jul 1989-Dec 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:** PM0.1-1 (API) **Averaging Time:** 24 h **Mean (min-max):** NR **Monitoring Stations:** 9 **Copollutant (correlation):** NR	**PM Increment:** Increase from the 10^{th}-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	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Fung et al. (2006, 089789) **Period of Study:** Jun 1995-Mar 1999 **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 days from case date), Dewanji and Moolgavkar (DM) method **Covariates:** Long-term trends, day-of-the-week effect, weather **Season:** All yr **Dose-response Investigated?** No **Statistical Package:** SPlus, R **Lags Considered:** 0-7 days	**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 µg/m^3 **RR Estimate (65+ yr)** 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: Hinwood et al. (2006, 088976) **Period of Study:** Jan 1992-Dec 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 yr **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 µg/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 µg/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 yr with an estimated 0.5% increase in hospitalizations.

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Hirshon et al. (2008, 180375) **Period of Study:** Jun 2002-Nov 2002 **Location:** Baltimore, Maryland	**Outcome:** Hospital admissions for asthma **Study Design:** Time-series **Covariates:** Spatial distance from pollution monitor, demographic variation, long term, seasonal and daily trends, weather and other pollutants **Statistical Analysis:** Overdispersed Poisson regression **Age Groups:** 0-17 yr	**Pollutant:** $PM_{2.5}$ zinc **Averaging Time:** 24 h **Mean (SD) Unit:** 22.42 (25.14) µg/m^3 **Range (Min, Max):** NR **Copollutant (correlation):** Ni: 0.41 Cr: 0.17 Fe: 0.54 Sulfate: 0.01 CO: 0.40 $PM_{2.5}$: 0.39 O_3: 0.01 NO_2: 0.66 EC: 0.48	**Increment:** NR **Relative Risk (95% CI), Best fit Model** Medium = 8.63-20.76 ng/m^3 High = >20.76 ng/m^3 No Lag Medium: 1.12 (0.98-1.28) High: 1.09 (0.91-1.30) 1-day Lag Medium: 1.23 (1.07-1.41) High: 1.16 (0.97-1.39) 2-day Lag Medium: 1.11 (0.94-1.30) High: 1.15 (0.96-1.38) **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: 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 yr, 15-64 yr, and ≥ 65 yr. 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 µg/m^3 increase, and a 27 µg/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 µg/m^3 increase): 0-14 yr: 1.1% [-3.1, 5.5] 15-64 yr: 2.2% [-1.8, 6.4]; ≥ 65 yr: 1.3% [-5.3, 8.2] For respiratory infections (10 µg/m^3 increase): All ages: 2.5% [0.1, 4.8] For respiratory infections (27 µg/m^3 increase): All ages: 7.0% [0.7, 13.6]

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Ko et al. (2007, 091639) **Period of Study:** Jan 2000-Dec 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 yr, 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: Ko et al. (2007, 092844) **Period of Study:** Jan 2000-Dec 2005 **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 yr, evaluated effect of season in analysis **Dose-response Investigated?** No **Statistical Package:** SPLUS 4.0 **Lags Considered:** 0-5 days	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean (SD):** 36.4 (21.1) **Percentiles**: 25th: 20.0 50th(Median): 32.5 75th: 47.7 **Range (Min, Max):** (6, 163) **Monitoring Stations:** 14 **Copollutant (correlation):** $PM_{2.5}$: PM_{10} r = 0.956 NO_2 r = 0.774 O_3 r = 0.585 SO_2 r = 0.482	**PM Increment:** 10.0 µg/m^3 **RR Estimate** Asthma (Single-pollutant model): 1.008[1.004, 1.013] lag 0 1.004[1.000, 1.009] lag 1 1.004[1.000, 1.009] lag 2 1.009[1.005, 1.014] lag 3 1.006[1.001, 1.011] lag 4 1.002[0.998, 1.007] lag 5 1.009[1.004, 1.014] lag 0-1 1.012[1.007, 1.018] 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: Lee et al. (2006, 090176) **Period of Study:** Jan 1997-Dec 2002 **Location:** Hong Kong, China	Hospital Admission **Outcome:** Asthma (493) **Age Groups:** <18 yr **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 h **Mean (SD):** 45.3 µg/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 µg/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	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Letz and Quinn (2005, 088752) **Period of Study:** Oct 2001-Aug 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 yr **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- to 7-day avg	**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 µg/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 1981-Dec 1993 **Location:** Toronto	Hospital Admissions **Outcome (ICD-9):** Asthma (493) **Age Groups:** 6-12 yr **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- to 7-day avg	**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 µg/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 yr **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: Mohr et al. (2008, 180215) **Period of Study:** Jun 2001-May 2003 **Location:** St. Louis, MO	**Outcome:** Asthma ER Visits **Age Groups:** 2-17 yr **Study Design:** Time series **Statistical Analyses:** GEE Poisson models **Covariates:** Season, weekend exposure, allergens **Dose-response Investigated:** No **Statistical Package:** SAS **Lags Considered:** 1 day	**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 µg/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	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	Hospital Admissions **Outcome (ICD-9):** Bronchitis, emphysema, asthma, bronchiectasis, extrinsic allergic alveolitis, and chronic airway obstruction (490-496) **Age Groups:** 3.0-5.9 yr 7-10 yr 65+ yr **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 µg/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: 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 yr	**Pollutant:** $PM_{2.5}$ and components **Averaging Time:** 24 h **Mean (SD) Unit:** 19.4 µg/m^3 **IQR:** 14.6 µg/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-yr respiratory hospital admissions in children <19yrs, 3-day 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, 3 day 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:** Jan 1995-Jun 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+ yr 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 days	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-h avg **Range (90% of Concentrations):** 4.2-20.2 µg/m^3 **Monitoring Stations:** One **Notes: Copollutant (correlation):** $PM_{2.5}$ PM1 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 µg/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: Tecer et al. (2008, 180030) **Period of Study:** Dec 2004-Oct 2005 **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 yr	**Pollutant:** $PM_{2.5}$ **Averaging Time:** NR **Mean, Unit:** 29.1 µg/m^3 **Range (Min, Max):** 4.55, 95.65 **Copollutant (correlation):** $PM_{2.5}/PM_{10}$ Mean: 0.56 Range: 0.17-0.88 $PM_{2.5}/PM_{10-2.5}$ Mean: 1.49 Range: 0.21-7.53	**Increment:** 10 µg/m^3 **Odds Ratio (95% CI)** Asthma Lag 0: 1.15 (0.99-1.34) Lag 1: 0.85 (0.70-1.03) Lag 2: 0.87 (0.73-1.04) Lag 3: 0.93 (0.79-1.10) Lag 4: 1.25 (1.05-1.50) Allergic Rhinitis with Asthma Lag 0: 1.21 (1.10-1.33) 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: Tolbert et al. (2007, 090316) **Period of Study:** Aug 1998-Dec 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	**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	**PM Increment:** $PM_{2.5}$: 10.96 µg/m^3 (IQR) $PM_{2.5}$ sulfate: 3.82 µg/m^3 (IQR) $PM_{2.5}$ total carbon: 3.63 µg/m^3 (IQR) $PM_{2.5}$ OC: 2.61 µg/m^3 (IQR) $PM_{2.5}$ EC: 1.15 µg/m^3 (IQR) $PM_{2.5}$ water-soluble metals: 0.03 µg/m^3

Reference	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
	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 ma(lag 0 -2)	0.5-21.9 1.7-9.5) $PM_{2.5}$ OC: 4.4 (3.8 2.7-5.3 0.4-25.9 2.1-7.2) $PM_{2.5}$ EC: 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 $PM_{2.5}$ EC and: PM_{10}: r = 0.61 O_3: r = 0.40 NO_2: r = 0.64 CO: r = 0.66 SO_2: r = 0.22 $PM_{10-2.5}$: r = 0.49 $PM_{2.5}$: r = 0.65 $PM_{2.5}$ SO_4: r = 0.32 $PM_{2.5}$ OC: r = 0.82 $PM_{2.5}$ TC: r = 0.91 $PM_{2.5}$ water soluble metals: r = 0.52 OHC: r = 0.35 Between $PM_{2.5}$ OC and: PM_{10}: r = 0.65 O_3: r = 0.54 NO_2: r = 0.62 CO: r = 0.59 SO_2: r = 0.17 $PM_{10-2.5}$: r = 0.49 $PM_{2.5}$: r = 0.70 $PM_{2.5}$ SO_4: r = 0.33 $PM_{2.5}$ EC: r = 0.82 $PM_{2.5}$ TC: r = 0.98 $PM_{2.5}$ water-sol metals: r = 0.49 OHC: r = 0.37 Between $PM_{2.5}$ total carbon and: PM_{10}: r = 0.67 O_3: r = 0.52 NO_2: r = 0.65 CO: r = 0.63 SO_2: r = 0.19 $PM_{10-2.5}$: r = 0.51 $PM_{2.5}$: r = 0.71	(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] $PM_{2.5}$ OC: RD: 1.003 [0.995-1.011] $PM_{2.5}$ EC: 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)
		$PM_{2.5}$ SO_4: r = 0.34 $PM_{2.5}$ EC: r = 0.91 $PM_{2.5}$ OC: r = 0.98 $PM_{2.5}$ water-sol metals: r = 0.52 OHC: r = 0.38 Between $PM_{2.5}$ water-soluble metals and: PM_{10}: r = 0.73 O_3: r = 0.43 NO_2: r = 0.32 CO: r = 0.35 SO_2: r = 0.06 $PM_{10-2.5}$: r = 0.50 $PM_{2.5}$: r = 0.69 $PM_{2.5}$ SO_4: r = 0.65 $PM_{2.5}$ EC: r = 0.52 $PM_{2.5}$ OC: r = 0.49 $PM_{2.5}$ TC: r = 0.52	
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 µg/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 1995-Mar 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 yr **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 yr, 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 µg/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 yr **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 yr **Study Design:** Time series **N:** 1254 admissions **Statistical Analyses:** Poisson multiple regression, GAM **Covariates:** Season, temperature, humidity, O_3, 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: 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 yr **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 µg/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 µg/m^3 $PM_{2.5}$ lag 0-1 avg: 16.32 µg/m^3 % change in Pneumonia: 6.48[1.13, 11.43] lag 0 5.56[-0.45, 11.27] mean lag 1

[1]All units expressed in µg/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) **Period of Study:** 2001-2004 **Location:** Copenhagen, Denmark	**Outcome (ICD10):** Respiratory disease (J41-46) Asthma (J45, 46) **Age Groups:** 5-18 and >65 **Study Design:** Time-series **N:** 1327 days ~1.5 million people at-risk **Statistical Analyses:** Poisson regression, GAM. **Covariates:** Influenza epidemics, pollen, temperature, dew point, day-of-week, holiday, season. **Season:** All **Dose-response Investigated?** No **Statistical Package:** R with gam and mgcv packages. **Lags Considered:** 0-5	**Pollutant:** Number concentration (NC) of ultrafine & accumulation mode particles **Averaging Time:** 24 h **Mean particles/cm3 (SD):** NCtot (total): 8116 (3502) 25th: 4959 50th: 6243 75th: 8218 99th: 16189 IQR: 3259 NC100 (<100 nm): 6847 (2864) 25th: 5738 50th (Median): 7358 75th: 9645 99th: 19895 IQR: 3907 Mean particles/cm^3 for 4 size modes (median diameter (nm) noted): NCa12: 493(315) NCa23: 2253 (1364) NCa57: 5104 (2687) NCa212: 6847 (2864) **Monitoring Stations:** 3 (Background, rural Background, urban Curbside, urban) **Notes:** NC exposure data available for n = 578 days. Information on distribution of 4 size modes provided in the paper. **Copollutant (correlation):** NCtot and PM_{10}: r = 0.39 NCtot and $PM_{2.5}$: r = 0.40 NCtot and NO_2: r = 0.68 PM_{10} and $PM_{2.5}$: r = 0.8 "Low or no" correlations between 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	**PM Increment:** Based on the IQR, specific to metric (see below). **RR Estimate:** Single pollutant results, Asthma, (5-18 yr), lag 0-5: $PM_{2.5}$: 1.15 [1, 1.32], IQr = 5 NCtot: 1.07 [0.98, 1.17], IQr = 3907 NC100: 1.06 [0.97, 1.16], IQr = 3259 NCa12: 1.08 [0.99,1.18], IQr = 342 NCa212: 1.08 [1, 1.17], IQr = 495 NCa23: 1.09 [0.98,1.21], IQr = 1786 NCa57: 1.02 [0.94,1.12], IQr = 3026 2-pollutant results: NCa212 w/ PM_{10}: 1.1 [0.96, 1.13], IQr = 495 NCtot w/ PM_{10}: 1.03 [0.92, 1.15] NCtot w/ $PM_{2.5}$: 1.04 [0.85, 1.28] All RD, (>65 yr), lag 0-4, single pollutant results: $PM_{2.5}$: 1 [0.95, 1.05] NCtot: 1.04 [1, 1.07] IQr = 3907 NC100: 1.03 [0.99, 1.07], IQr = 3259 NC12: 1.01 [0.98, 1.05], IQr = 342 NC212: 1.04 [1.01, 1.08], IQr = 495 NCa23: 0.99 [0.94, 1.03], IQr = 1786 NCa57: 1.04 [1, 1.08], IQr = 3026 2-pollutant results: NCa212 w/ PM_{10}: 1.01 [0.96, 1.07], IQr = 495 NCtot w/ $PM_{2.5}$: 0.97 [0.89, 1.05] NCtot w/ PM_{10}: 1 [0.96, 1.05] **Notes:** Multipollutant model results also included for models with 4 size modes.
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** µg/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

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:** RSPM (Respirable Suspended PM <10 µm) **Averaging Time:** 8 h **Mean µg/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
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-day ma 9.090 [3.12-15.40] 3 day ma 10.28 [4.05-16.90] 4-day ma 11.63 [5.46-19.318] 5 day ma 12.61 [5.68-20.00] 6-day ma 12.56 [5.47-20.13] 7-day 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 1992-May 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 ma **Mean µg/m^3 (SD):** NR **Monitoring Stations:** 1 **Copollutant (correlation):** N	**PM Increment:** NR **Correlation with Number of Admissions:** Entire yr 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 1989-Dec 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 h **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+ yr **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 & 5-day (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, PM0.03-0.1, PM<0.1, PM<0.10.29, $PM_{10-2.5}$, CO, NO_2 **Co-pollutant Correlation** PM<0.03: 0.14 PM0.03-0.1: 0.48 PM<0.1: 0.35 PM<0.10.29: 0.88 $PM_{10-2.5}$: 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-day 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-day 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-day 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-day 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 1992-Dec 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** µg/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, 188530) **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, yr, 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:** PM1 **Averaging Time:** 24-h avg **Mean (SD):** 1.91 (2.95) µg/m^3 **Range (Min, Max):** 0.0, 56.6 µg/m^3 **Monitoring Stations:** 2 **Notes: Copollutant (correlation):** NR	**PM Increment:** 10 µg/m^3 **RR Estimate [Lower CI, Upper CI] lag:** Asthma, COPD (499-496): Adjusted for day, month & yr: 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 & yr: 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 & yr: 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 yr 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	**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- to 2-day avg	**Pollutant:** TSP **Averaging Time:** Means of daily total levels at stations **Mean (SD):** 105.3 µg/m^3 , (44.2) **Percentiles: 25th:** NR **50th(Median):** 96.0 µg/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 µg/m^3 increase **% Increase, lag 0-2-day 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-day 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 µg/m^3 increase **Covariates:** Gender, age, daily mean temperature, season, day of week, holidays, solar radiation **Season:** All **Lags Considered:** 1- to 3-day avg	**Pollutant:** Total suspended particulate **Averaging Time:** Mean of admission day and 3 preceding days **Mean (SD):** 114.5 µg/m^3, (42.8) **Percentiles:** 25th: NR 50th(Median): 109.9 µg/m^3 75th: NR **Monitoring Stations:** 10 **Notes: Copollutant (correlation):** TSP-NO: 0.76	**PM Increment:** 10 µg/m^3 % Increase, lag 1-3-day avg <4 yr: 1.8% [0.00, 3.05] 4-15 yr: 3.0% [0.01, 5.08] all: 1.8% [0.03, 3.02] adjusted for all covariates **Notes:** Multipollutant models also used
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 yr 7-10 yr 65+ yr **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_1 **Averaging Time:** 24 h **Mean** µg/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.

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
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 yr Pediatric asthma 2-18 yr Adults >18 yr **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? Y**es **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, EC (EC), OC (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]
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 yr **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 days, 0- to 1-day 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" **Copollutant (correlation):** NR	**PM Increment:** "per unit increase" **RR Estimate [Lower CI, Upper CI]** **lag:** **Single pollutant model** Respiratory >64 yr 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 yr 1.0641 [1.0006, 1.1315] lag2 1.0893 [1.0240, 1.1587] lag3 Asthma + COPD >64 yr 1.0713 [1.0179, 1.1276] lag3 1.0552 [1.0082, 1.1045] lag 0-1 avg Pneumonia & Acute Bronchitis >64 yr 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 yr 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

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Sinclair and Tolsma (2004, 088696) **Period of Study:** 25 mo **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 yr; 18+ yr (asthma); All ages (URI//LRI) **Study Design:** Times series **N:** 25 mo 260,000-275,000 health plan members (Aug 1998-Aug 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-day ma (0-2, 2-5, 6-8)	**Pollutant:** $PM_{2.5-10}$ ($\mu g/m^3$) **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 day lag URI: Course PM = 1.021 (S) 3-5 day lag LRI: Coarse PM = 1.07 (S) 3-5 day 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: Sinclair and Tolsma (2004, 088696) **Period of Study:** 25 mo **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 yr, 18+ yr (asthma) All ages (URI//LRI) **Study Design:** Times series **N:** 25 mo 260,000-275,000 health plan members (Aug 1998-Aug 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-day ma (0-2, 2-5, 6-8)	**Pollutant:** UF (PM_{10}-100 nm) **Averaging Time:** 24 h avg **Mean (SD):** PM_{10}-100 nm area ($\mu m2/cm^3$)- 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).

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Slaughter et al. (2005, 073854) **Period of Study:** Jan 1995-Jun 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+ yr 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 days	**Pollutant:** PM_1 **Averaging Time:** 24-h avg **Range (90% of concentrations):** 3.3-17.6 µg/m^3 **Monitoring Stations: 1** **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 µg/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]
Reference: Slaughter et al. (2005, 073854) **Period of Study:** Jan 1995-Jun 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+ yr 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 days	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-h avg **Range (90% of Concentrations):** 4.2-20.2 µg/m^3 **Monitoring Stations: 1** **Notes: Copollutant (correlation):** $PM_{2.5}$ PM1 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 µg/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 yr **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 µg/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 µg/m^3 $PM_{2.5}$ lag 0-1 avg: 16.32 µg/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 yr **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 µg/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 µg/m^3 BC lag 0-1 avg: 1.69 µg/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 µg/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, 054808) **Period of Study:** ~5 yr for most cities, during the 1990s **Location:** 28 European cities (APHEA2)	**Outcome:** Nonaccidental 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 µg/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)
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 µg/m^3 **Range (Min, Max):** NR **Copollutant:** BS **Note:** PM_{10} only measured in 21 cities.	**Increment:** 10 µg/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: Nonaccidental (<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 µg/m^3 **Relative Risk (Lower CI, Upper CI) lag:** Nonaccidental: 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

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
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 Study population: 65,180 elderly residents with history of hospitalization for heart or lung disease	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SE) unit:** 37.6 (15.5) µg/m^3 **Range (Min, Max):** (3.7, 128) **Copollutant:** NR	**Increment:** 10 µg/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, 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) 0-1 All: 1.1% (0.4, 1.9) 0-1
Reference: Bell et al. (2009, 191007) **Period of Study:** 1987-2000 **Location:** 84 U.S. 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:** 24 h **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:** U.S.	**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, NH4+, 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) **Period of Study:** 1996-2002 **Location:** 15 Italian cities	**Outcome:** Mortality All-cause (nonaccidental) (<800) Cardiovascular (390-459) Respiratory (460-519) **Study Design:** Meta-analysis **Statistical Analyses:** Poisson GLM **Age Groups:** All ages	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** NR **Range (Min, Max):** NR **Copollutant:** SO_2 NO_2 CO O_3	**Increment:** 10 µg/m^3 **% Increase (Lower CI, Upper CI) lag:** All-cause: 0.31% (-0.19, 0.74) 0-1 Winter: 0.08% 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
Reference: Burnett et al. (2004, 086247) **Period of Study:** 1981-1999 **Location:** 12 Canadian cities	**Outcome:** Mortality: Nonaccidental (<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 $PM_{10-2.5}$: 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 $PM_{10-2.5}$ measurements.	**Increment:** 10 µg/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
Reference: Cakmak et al. (2007, 091170) **Period of Study:** Jan 1997-Dec 2003 **Location:** Chile-7 cities	**Outcome:** Mortality: Nonaccidental (<800) Cardiovascular diseases (390-459) Respiratory diseases (460-519) **Study Design:** Time-series **Statistical Analyses:** Poisson Random effects regression model **Age Groups:** All age ≤ 64 yr 65-74 yr 75-84 yr ≥ 85 yr	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** 84.9 **Range (Min, Max):** NR **Copollutant (correlation):** O_3: r = -0.16-0.13 SO_2: r = 0.37-0.77 CO: r = 0.49-0.82 **Note:** Correlations are between pollutants for seven monitoring stations.	**Increment:** 10 µg/m^3 **% Increase (Lower CI, Upper CI) lag:** Nonaccidental: 0.97% (-1.09, 2.76) 0 1.31% (-1.56, 3.68) 0-5 $PM_{10}+O_3+SO_2+CO$: 0.80% (-0.87, 2.28) 0 ≤ 64: 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 Apr-Sep: 1.03% (-1.17, 2.93) 0 1.37% (-1.64, 3.82) 0-5 Oct-Mar: 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) **Period of Study:** 2001-2004 **Location:** Shanghai, China	**Outcome** (ICD9: 2001; ICD10: 2002-2004): Mortality: Nonaccidental causes (ICD9 <800; ICD10 A00-R99) Cardiovascular (ICD9 390-459; I CD10 I00-I99) Respiratory (ICD9 460-519; ICD10 J00-J98) **Study Design:** Time-series **Statistical Analyses:** Poisson GAM **Age Groups:** All ages	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** 102.0 **Range (Min, Max):** (14.0-566.8) **Copollutant (correlation):** SO_2 r = 0.64 NO_2 r = 0.71	**Increment:** 10 µg/m^3 **% Increase (Lower CI, Upper CI) lag:** Nonaccidental Single Pollutant: 0.26% (0.14, 0.37) PM_{10}+SO_2: 0.08% (-0.07, 0.22) PM_{10}+NO_2: 0.01% (-0.14, 0.17) PM_{10}+SO_2+NO_2: 0.00% (-0.16, 0.16) Cardiovascular mortality Single Pollutant: 0.27% (0.10, 0.44) PM_{10}+SO_2: 0.12% (-0.10, 0.34) 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)
Reference: Daniels et al. (2004, 087343) **Period of Study:** 1987-1994 **Location:** 20 Largest U.S. cities	**Outcome:** Mortality: Total (Nonaccidental) mortality Cardiovascular-Respiratory (390-448) (480-486, 487, 490-496, 507) Other-cause mortality **Study Design:** Time-series **Statistical Analyses:** City-Specific Estimates: Poisson GLM, natural cubic splines Combined Estimates: 2-stage Bayesian hierarchical model **Age Groups:** All ages	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** Los Angeles: 46.0 New York: 28.8 Chicago: 35.6 Dallas-Ft. Worth: 23.8 Houston: 30.0 San Diego: 33.6 Santa Ana-Anaheim: 37.4 Phoenix: 39.7 Detroit: 40.9 Miami: 25.7 Philadelphia: 35.4 Minneapolis: 26.9 Seattle: 25.3 San Jose: 30.4 Cleveland: 45.1 San Bernardino: 37.0 Pittsburgh: 31.6 Oakland: 26.3 Atlanta: 34.4 San Antonio: 23.8	**Increment:** 10 µg/m^3 **% Increase (Lower CI, Upper CI) lag:** Total (nonaccidental): 0.17% (0.03, 0.30) 0 0.20% (0.07, 0.33) 1 0.28% (0.16, 0.41) 0-1 avg Cardiovascular-Respiratory: 0.17% (-0.01, 0.35) 0 0.27% (0.09, 0.44) 1 0.30% (0.18, 0.51) 0-1 avg Other-cause: 0.17% (-0.03, 0.37) 0 0.12% (-0.07, 0.31) 1 0.20% (0.01, 0.38) 0-1 avg Threshold Models: Total Mortality Threshold = 15 µg/m^3 0.30% (0.17, 0.42) 0-1 avg Threshold = 0 µg/m^3 0.28% (0.16, 0.41) 0-1 avg Threshold = 20 µg/m^3 0.30% (0.16, 0.43) 0-1 avg

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: De Leon et al. (2003, 055688) **Period of Study:** Jan 1985-Dec 1994 **Location:** New York, New York	**Outcome:** Mortality: Circulatory (390-459) Cancer (140-239) **Study Design:** Time-series **Statistical Analyses:** Poisson GAM **Age Groups:** All ages <75 yr >75 yr	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** 33.27 µg/m^3 IQR (25th, 75th): (22.67, 40.83) **Copollutant (correlation):** O_3 CO SO_2 NO_2	**Increment:** 18.16 µg/m^3 **Relative Risk (Lower CI, Upper CI) lag:** All Ages Cancer: 1.014 (1.000, 1.029) 0-1 -w/out respiratory: 1.011 (0.996, 1.026) 0-1 -w/ respiratory: 1.051 (0.998, 1.107) 0-1 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 yr 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 yr 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) 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 (nonaccidental) (<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 yr; 65-74 yr; ≥ 75 yr	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** NR **Range (Min, Max):** NR **Copollutant (correlation):** NR	**Increment:** 10 µg/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, 059158) **Period of Study:** 1987-1994 **Location:** 90 U.S. cities (NMMAPS)	**Outcome:** Mortality: Total (nonaccidental) **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 µg/m^3 **% Increase (Lower CI, Upper CI) lag:** α = 3 0.2% (0.05, 0.35)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Dominici et al. (2004, 096951) **Period of Study:** 1986-1993 **Location:** 10 U.S. cities	**Outcome:** Mortality: Total (nonaccidental) **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 µg/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 (nonaccidental) 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 µg/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 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) 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.

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Fischer et al. (2003, 043739) **Period of Study:** 1986-1994 **Location:** The Netherlands	**Outcome:** Mortality: Nonaccidental (<800) Pneumonia (480-486) COPD (490-496) Cardiovascular (390-448) **Study Design:** Time-series **Statistical Analyses:** Poisson GAM, LOESS **Age Groups:** <45 yr 45-64 yr 65-74 yr ≥ 75 yr	**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 µg/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) 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:** Jun 2003-Aug 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 µg/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) µg/m^3 IQR (25th, 75th): (36.1, 63.0) **Copollutant (correlation):** NR	**Increment:** 10 µg/m^3 **% Increase (Lower CI, Upper CI)** **lag:** Nonaccidental: 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) **Period of Study:** 1997-2004 **Location:** 9 Italian cities	**Outcome:** Mortality: Nonaccidental (<800) **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:** 35.1-71.5 Range (5th, 95th): Lowest 5th: 14.3 Highest 95th: 147.0 **Copollutant (correlation):** NR	**Increment:** 10 µg/m^3 **% Increase (Lower CI, Upper CI) lag:** Total: 0.60% (0.31, 0.89) 0-1 Age 35-64: -0.20% (-0.77, 0.37) 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 days 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
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) µg/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}
Reference: Kan and Chen (2003, 087372) **Period of Study:** Jan 2000-Dec 2001 **Location:** Shanghai, China	**Outcome:** Mortality: Nonaccidental (<800) Cardiovascular (390-459) COPD (490-496) **Study Design:** Time-series **Statistical Analyses:** Poisson GAM, LOESS **Age Groups:** All ages <65 yr 65-75 yr >75 yr	**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 µg/m^3 **Relative Risk (Lower CI, Upper CI) lag:** Nonaccidental: 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:** Jan 2000-Dec 2001 **Location:** Shanghai, China	**Outcome:** Mortality: Nonaccidental (<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 µg/m^3 **Odds Ratio (Lower CI, Upper CI) lag:** Nonaccidental: Bidirectional referent days: 7 days: 1.000 (0.9988, 1.002) 0-1 ma 7 and 14 days: 1.002 (1.000, 1.004) 0-1 ma 7, 14, and 21 days: 1.003 (1.001, 1.005) 0-1 ma Unidirectional referent days: 7 days: 1.015 (1.012, 1.018) 0-1 ma 7 and 14 days: 1.017 (1.015, 1.019) 0-1 ma 7, 14, and 21 days: 1.019 (1.012, 1.021) 0-1 ma Bidirectional referent days (7, 14, and 21 days): Cardiovascular: 1.004 (1.001, 1.007) 0-1 ma COPD: 1.006 (0.999, 1.013) 0-1 ma Nonaccidental: 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
Reference: Kan et al. (2005, 087561) **Period of Study:** Apr 2003-May 2003 **Location:** Beijing, China	**Outcome:** Mortality: Severe acute respiratory syndrome (SARS) **Study Design:** Time-series **Statistical Analyses:** Poisson, GAM, smoothing spline **Age Groups:** All ages	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** 149.1 (8.1) **Range (Min, Max):** (34, 246) **Copollutant:** SO_2 NO_2	**Increment:** 10 µg/m^3 **Relative Risk (Lower CI, Upper CI) lag:** 0.99 (0.96-1.03) 0 1.00 (0.97-1.04) 1 1.02 (0.98-1.06) 2 1.04 (0.99-1.09) 3 1.06 (1.00-1.11) 4 1.06 (1.00-1.12) 5 1.05 (0.98-1.12) 6
Reference: Kan et al. (2007, 091267) **Period of Study:** Mar 2004-Dec 2005 **Location:** Shanghai, China	**Outcome (ICD10):** Mortality: Total (nonaccidental) (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} **Averaging Time:** 24-h avg **Mean (SD):** 107.9 (2.39) µg/m^3 **Range (Min, Max):** (22.0, 403.0) **Copollutant (correlation):** PM_{10} $PM_{2.5}$: r = 0.84 $PM_{10-2.5}$: r = 0.88 O_3: r = 0.21	**Increment:** 10 µg/m^3 **% Increase (Lower CI, Upper CI) lag:** PM_{10} Total: 0.16% (0.02, 0.30) 0-1 Cardiovascular: 0.31% (0.10, 0.53) 0-1 Respiratory: 0.33% (-0.08, 0.75) 0-1

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Kan et al. (2008, 156621) **Period of Study:** Jan 2001-Dec 2004 **Location:** Shanghai, China	**Outcome:** Mortality: Total (nonaccidental) (A00-R99) Cardiovascular (I00-I99) Respiratory (J00-J98) **Study Design:** Time-series **Statistical Analyses:** Poisson GLM, natural splines **Age Groups:** All ages; 0-4 yr 5-44 yr 45-64 yr ≥ 65 yr	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** Warm season: 87.4 (1.8) Cool season: 116.7 (2.8) Entire period: 102.0 (1.7) **Range (Min, Max):** NR **Copollutant (correlation):** SO_2 NO_2 O_3	**Increment:** 10 µg/m^3 **% Increase (Lower CI, Upper CI) lag:** Nonaccidental Warm season: 0.21 (0.09, 0.3) 0-1 Cool season: 0.26 (0.22, 0.30) 0-1 Entire period: 0.25 (0.14, 0.37) 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) 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
Reference: Keatinge and Donaldson (2006, 087536) **Period of Study:** 1991-2002 **Location:** London, England	**Outcome:** Mortality: Total (nonaccidental) **Study Design:** Time-series **Statistical Analyses:** Poisson GAM **Age Groups:** ≥ 65 yr	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** NR **Range (Min, Max):** NR **Copollutant:** O_3 SO_2	**Increment:** 10 µg/m^3 **Mortality per 106 (Lower CI, Upper CI) lag:** PM_{10}+Temp: 2.1 (0.9, 3.3) 0-2 avg 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
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 µg/m^3 Warm Season: 9.8 µg/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

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Kim et al. (2003, 155899) **Period of Study:** Jan 1995-Dec 1999 **Location:** Seoul, Korea	**Outcome (ICD10):** Mortality: Nonaccidental (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 µg/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: 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) 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) **Period of Study:** Jan 1997-Dec 2001 **Location:** Seoul, Korea	**Outcome:** Mortality: Nonaccidental **Study Design:** Time-series **Statistical Analyses:** Poisson GAM, LOESS **Age Groups:** All ages	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** 68.23 (36.36) µg/m^3 IQR (25th, 75th): (42.56, 84.67) **Copollutant (correlation):** NR	**Increment:** 42.11 µg/m^3 **Relative Risk (Lower CI, Upper CI) lag:** 1.021 (1.009, 1.035)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Le Tertre et al. (2005, 087560) **Period of Study:** NR **Location:** 21 European cities (APHEA-2)	**Outcome:** Mortality: Nonaccidental (<800) **Study Design:** Time-series **Statistical Analyses:** Empirical Bayes **Age Groups:** All ages	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** NR **Range (Min, Max):** NR **Copollutant:** NO_2	**Increment:** 1.0 µg/m^3 **β coefficient (SE) lag:** Athens: 0.001311 (0.0003) 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)
Reference: Lee et al. (2007, 093042) **Period of Study:** Jan 2000-Dec 2004 **Location:** Seoul, Korea	**Outcome (ICD10):** Mortality: Nonaccidental (A00-R99) **Study Design:** Time-series **Statistical Analyses:** Poisson GAM **Age Groups:** All ages	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** w/ Asian dust days: 70.00 (47.80) w/o Asian dust days: 65.77 (33.60) Asian dust days only: 188.49 (142.85) **Copollutant:** CO NO_2 SO_2 O_3	**Increment:** 41.49 µg/m^3 **% Increase (Lower CI, Upper CI) lag:** Model with Asian Dust Days 0.7% (0.2, 1.3) 1-3 Model without Asian dust days 1.0% (0.2, 1.8) 1-3
Reference: Lee and Shaddick (2007, 156685) **Period of Study:** Jan 1993-Dec 1997 **Location:** Cleveland, Ohio Detroit, Michigan Minneapolis, Minnesota Pittsburgh, Pennsylvania	**Outcome (ICD10):** Mortality: Nonaccidental **Study Design:** Time-series **Statistical Analyses:** 1. Bayesian, penalized spline 2. Likelihood, penalized spline **Age Groups:** All ages	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** NR **Range (Min, Max):** NR	**Increment:** 10 µg/m^3 **Relative Risk (Lower CI, Upper CI) lag:** Constant model Cleveland: 1.0049 1 Detroit: 1.0046 1 Minneapolis: 1.0052 1 Pittsburgh: 1.0045 1
Reference: Martins et al. (2004, 087457) **Period of Study:** Jan 1997-Dec 1999 **Location:** São Paulo, Brazil	**Outcome (ICD10):** Mortality: Respiratory (J00-J99) **Study Design:** Time-series **Statistical Analyses:** Poisson GLM, natural cubic splines **Age Groups:** ≥ 60	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** Cerqueira Cesar: 42.5(22.9) 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	The study does not present quantitative results.

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Nawrot et al. (2007, 098619) **Period of Study:** Jan 1997-Dec 2003 **Location:** Flanders, Belgium	**Outcome:** Mortality: Nonaccidental (<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 vs. the lowest season-specific PM_{10} quartile Summer: 7.8% (6.1, 9.6) Spring: 6.3% (4.7, 7.8) Fall: 2.2% (0.58, 3.8) Winter: 1.4% (0.06, 2.9) Warm months (Jun, Jul, Aug): 7.9% (6.2, 9.6) Cold months (Dec, Jan, Feb): 1.5% (0.22, 3.3) Intermediate months (Mar, Apr, May, Sep, Oct, Nov): 4.2% (2.9, 5.6) Warmer Periods (Apr-Sep) Nonaccidental: 1.5% (1.1, 2.0) 0 Respiratory: 2.0% (0.6, 3.7) 0 Cardiovascular: 1.8% (1.1, 2.4) 0

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: O'Neill et al. (2004, 087429) **Period of Study:** 1996-1998 1994-1995 **Location:** Mexico City, Mexico	**Outcome:** Mortality: Nonaccidental **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 Hi-Vol: (15.0, 292.0) TEOM: (13.7, 268.3) Predicted: (11.2, 154.4) Pedregal (1996-1998) Hi-Vol: (5.0, 226.0) TEOM: (7.8, 264.4) Predicted: (-0.5, 86.3) Pedregal (1994-1995) Hi-Vol: (24.0, 114.0) TEOM: (8.7, 152.5) Impactor: (15.0, 154.0) Predicted: (3.9, 75.9)	**Increment:** 10 µg/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 0.09% (-0.16, 0.34) 1 -0.12% (-0.43, 0.20) 2 -0.02% (-0.26, 0.21) 3 -0.14% (-0.37, 0.09) 4 -0.05% (-0.28, 0.18) 5 0.00% (-0.39, 0.38) 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 -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 **Location:** Mexico City and Monterrey, Mexico	**Outcome:** Mortality: Nonaccidental Cardiovascular (390-460) Respiratory (460-520) Other-causes **Study Design:** Time-series **Statistical Analyses:** Poisson, natural cubic splines **Age Groups:** All ages, 0-15, ≥ 65	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** Mexico City: 75.8 (31.4) Monterrey: 50.0 (23.5) **Range (Min, Max):** Mexico City: (18.0, 233.9) Monterrey: (6.2, 230.8) **Copollutant:** O_3	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)
Reference: O'Neill et al. (2008, 192314) **Period of Study:** Jan 1998-Dec 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 yr	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (SD) µg/m³:** 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 µg/m³ **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 yr: 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 yr: 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 yr: 0.75 (-0.49-2.02) All Adults, df (yr) None: 5.4 Primary: 6.0 Secondary: 6.0 ≥ 12 yr: 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 yr: 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 yr: 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 yr: 1.71 (0.61-2.83) Women, df (yr) None: 5.4 Primary: 4.4 Secondary: 4.8 ≥ 12 yr: 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 yr: 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 yr: 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 yr: 1.07 (-0.41-2.57) Men, df (yr) None: 3.8 Primary: 5.6 Secondary: 4.6 ≥ 12 yr: 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 yr: 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 yr: 2.20 (1.27-3.15)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			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 yr: 2.20 (1.27-3.15) All Adults, df (yr) None: 4.0 Primary: 4.0 Secondary: 2.8 ≥ 12 yr: 1.6 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 yr: 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 yr: 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 yr: 3.35 (1.49-5.25) Women, df (yr) None: 2.4 Primary: 3.6 Secondary: 1.4 ≥ 12 yr: 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 yr: 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 yr: 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 yr: 3.18 (1.60-4.79) Men, df (yr) None: 4.4 Primary: 3.2 Secondary: 0.8 ≥ 12 yr: 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) ≥ 12 yr: 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 yr: 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 yr: 2.00 (0.93-3.07) All Adults, df (yr) None: 3.6 Primary: 5.6 Secondary: 4.0 ≥ 12 yr: 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 yr: 0.60 (-0.32-1.52) Women, Lag 1

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			None: 1.58 (0.58-2.58) Primary: 0.79 (0.42-1.17) Secondary: 0.76 (0.25-1.28) ≥ 12 yr: 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 yr: 1.06 (-0.27-2.41) Women, df (yr) None: 2.6 Primary: 4.8 Secondary: 4.4 ≥ 12 yr: 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 yr: 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) ≥ 12 yr: 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 yr: 1.98 (0.76-3.20) Men, df (yr) None: 2.8 Primary: 4.8 Secondary: 4.4 ≥ 12 yr: 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 yr: 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 yr: 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 yr: 1.83 (0.09-3.59) All Adults, df (yr) None: 5.6 Primary: 5.4 Secondary: 6.0 ≥ 12 yr: 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 yr: 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 yr: 1.50 (0.32-2.70) Women, Distributed Lags 0-5 None: 0.75 (-0.56-2.08) Primary: 1.43 (0.29-2.59) Secondary: 0.06 (-1.01-1.15) ≥ 12 yr: 1.48 (0.10-2.87) Women, df (yr) None: 5.4 Primary: 4.2 Secondary: 4.8

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			≥ 12 yr: 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 yr: 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 yr: 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 yr: 1.76 (-0.35-3.91) Men, df (yr) None: 3.8 Primary: 5.6 Secondary: 4.6 ≥ 12 yr: 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 yr: 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 yr: 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 yr: 3.63 (2.01-5.29) All Adults, df (yr) None: 4.0 Primary: 3.8 Secondary: 2.6 ≥ 12 yr: 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 yr: 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 yr: 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 yr: 2.53 (0.70-4.40) Women, df (yr) None: 2.4 Primary: 3.4 Secondary: 1.2 ≥ 12 yr: 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 yr: 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 yr: 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 yr: 1.45 (-0.34-3.29)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			Men, df (yr) None: 4.6 Primary: 3.0 Secondary: 0.8 ≥ 12 yr: 1.0 **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 yr: 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 yr: 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 yr: 4.02 (2.78-5.27) All Adults, df (yr) None: 3.8 Primary: 5.2 Secondary: 4.0 ≥ 12 yr: 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 yr: 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 yr: 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 yr: 0.87 (-0.02-1.78) Women, df (yr) None: 2.4 Primary: 4.8 Secondary: 4.4 ≥ 12 yr: 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) ≥ 12 yr: 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 yr: 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 yr: 2.99 (1.66-4.33) Men, df (yr) None: 2.0 Primary: 4.4 Secondary: 4.4 ≥ 12 yr: 1.8

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Peng et al. (2005, 087463) **Period of Study:** 1987-2000 **Location:** 100 U.S. cities (NMMAPS)	**Outcome:** Mortality: Nonaccidental **Study Design:** Time-series **Statistical Analyses:** Bayesian semiparametric hierarchical models **Age Groups:** All ages	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Median (SD) unit:** 27.1 **Range (Min, Max):** (13.2, 48.7) **Copollutant (correlation):** NR	**Increment:** 10 µg/m^3 **% Increase (Lower CI, Upper CI) lag:** Winter: -0.4% (-0.30, 0.21) 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 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 **PM10 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 **PM10 + O3 (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 **PM10 + O3 (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 **PM10 + NO2 (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
Reference: Penttinen et al. (2004, 087432) **Period of Study:** 1988-1996 **Location:** Helsinki, Finland	**Outcome:** Mortality: Total (nonaccidental) (<800) Cardiovascular (390-459) Respiratory (460-519) **Study Design:** Time-series **Statistical Analyses:** Poisson GAM, LOESS **Age Groups:** 15-64 yr 65-74 yr ≥ 75 yr	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Median (SD) unit:** 21 µg/m^3 **Range (Min, Max):** (0.2, 213) **Copollutant (correlation):** O_3: r = -0.09 NO_2: r = 0.50 CO: r = 0.45 SO_2: r = 0.61 TSP: r = 0.72	**Increment:** 10 µg/m^3 **% Increase (Lower CI, Upper CI) lag:** Total (nonaccidental) -0.23% (-1.47, 1.01) 0 0.88% (-0.32, 2.08) 1 0.11 (-0.51, 0.73) 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
Reference: Qian et al. (2007, 093054) **Period of Study:** 2001-2004 **Location:** Wuhan, China	**Outcome:** Mortality: Total (nonaccidental) (<800) Cardiovascular (390-459) Stroke (430-438) Cardiac Diseases (390-398) Respiratory (460-519) Cardiopulmonary **Study Design:** Time-series **Statistical Analyses:** Poisson GAM, natural splines **Age Groups:** All ages	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** 141.8 3 **Range (Min, Max):** (24.8, 477.8) **Copollutant (correlation):** NO_2 SO_2 O_3	**Increment:** 10 µg/m^3 **% Increase (Lower CI, Upper CI) lag:** Nonaccidental 0.36% (0.19, 0.53) 0 0.28% (0.12, 0.45) 1 0.43% (0.24, 0.62) 0-1 0.08% (-0.15, 0.31) 0-4 <45 yr 0.28% (-0.26, 0.82) 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 yr 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 yr

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
	<45 yr		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
	≥ 45 yr		≥ 65 yr 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
	<65 yr		
	≥ 65 yr		
			Cardiovascular 0.51% (0.28, 0.75) 0 0.35% (0.12, 0.58) 1 0.58% (0.31, 0.84) 0-1 0.35% (0.05, 0.66) 0-4 <45 yr 0.59% (-0.62, 1.82) 0 0.93% (-0.22, 2.08) 1 1.07% (-0.27, 2.42) 0-1 1.15% (-0.40, 2.72) 0-4 ≥ 45 yr 0.51% (0.27, 0.75) 0 0.33% (0.10, 0.56) 1 0.56% (0.30, 0.83) 0-1 0.33% (0.02, 0.63) 0-4 <65 yr 0.27% (-0.23, 0.76) 0 0.30% (-0.16, 0.77) 1 0.42% (-0.12, 0.97) 0-1 0.43% (-0.19, 1.06) 0-4 ≥ 65 yr 0.57% (0.31, 0.83) 0 0.36% (0.11, 0.61) 1 0.61% (0.32, 0.90) 0-1 0.33% (0.00, 0.67) 0-4
			Stroke 0.44% (0.16, 0.72) 0 0.41% (0.14, 0.68) 1 0.58% (0.27, 0.89) 0-1 0.45% (0.09, 0.81) 0-4 <45 yr 1.18% (-0.45, 2.83) 0 1.66% (0.11, 3.24) 1 1.91% (0.10, 3.75) 0-1 2.72% (0.58, 4.89) 0-4 ≥ 45 yr 0.42% (0.14, 0.70) 0 0.37% (0.10, 0.65) 1 0.55% (0.23, 0.86) 0-1 0.39% (0.03, 0.76) 0-4 <65 yr 0.26% (-0.35, 0.87) 0 0.38% (-0.20, 0.96) 1 0.48% (-0.19, 1.16) 0-1 0.57% (-0.21, 1.35) 0-4 ≥ 65 yr 0.49% (0.17, 0.80) 0 0.41% (0.11, 0.72) 1 0.61% (0.26, 0.96) 0-1 0.42% (0.02, 0.83) 0-4
			Cardiac 0.49% (0.08, 0.89) 0 0.28% (-0.11, 0.67) 1 0.49% (0.04, 0.94) 0-1 0.22% (-0.29, 0.74) 0-4 <45 yr 0.25% (-1.64, 2.17) 0 0.56% (-1.22, 2.38) 1 0.61% (-1.47, 2.74) 0-1 -0.42% (-2.80, 2.02) 0-4 ≥ 45 yr 0.49% (0.09, 0.91) 0 0.27% (-0.12, 0.66) 1 0.48% (0.03, 0.94) 0-1 0.25% (-0.27, 0.77) 0-4

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			<65 yr 0.00% (-0.89, 0.90) 0 0.12% (-0.73, 0.98) 1 0.13% (-0.86, 1.13) 0-1 0.05% (-1.08, 1.20) 0-4 ≥ 65 yr 0.60% (0.17, 1.03) 0 0.32% (-0.10, 0.74) 1 0.57% (0.09, 1.06) 0-1 0.26% (-0.29, 0.82) 0-4 Respiratory 0.71% (0.20, 1.23) 0 0.63% (0.13, 1.13) 1 0.86% (0.28, 1.44) 0-1 0.19% (-0.48, 0.87) 0-4 <45 yr 1.74% (-1.28, 4.86) 0 2.52% (-0.30, 5.42) 1 2.95% (-0.41, 6.42) 0-1 3.47% (-0.61, 7.73) 0-4 ≥ 45 yr 0.69% (0.18, 1.21) 0 0.58% (0.09, 1.08) 1 0.81% (0.23, 1.39) 0-1 0.13% (-0.54, 0.80) 0-4 <65 yr 0.06% (-1.30, 1.43) 0 -0.53% (-1.83, 0.79) 1 -0.32% (-1.84, 1.22) 0-1 -0.72% (-2.47, 1.05) 0-4 ≥ 65 yr 0.79% (0.27, 1.31) 0 0.76% (0.26, 1.26) 1 0.99% (0.41, 1.57) 0-1 0.30% (-0.38, 0.98) 0-4 Cardiopulmonary 0.46% (0.23, 0.69) 0 0.35% (0.13, 0.57) 1 0.53% (0.28, 0.79) 0-1 0.11% (-0.19, 0.42) 0-4 <45 yr 0.71% (-0.48, 1.92) 0 1.26% (0.14, 2.4) 1 1.39% (0.06, 2.74) 0-1 1.41% (-0.18, 3.03) 0-4 ≥ 45 yr 0.45% (0.23, 0.68) 0 0.32% (0.10, 0.54) 1 0.51% (0.25, 0.77) 0-1 0.08% (-0.23, 0.38) 0-4 <65 yr 0.14% (-0.34, 0.61) 0 0.15% (-0.30, 0.61) 1 0.23% (-0.30, 0.76) 0-1 0.11% (-0.52, 0.74) 0-4 ≥ 65 yr 0.53% (0.28, 0.78) 0 0.39% (0.15, 0.63) 1 0.60% (0.32, 0.88) 0-1 0.11% (-0.22, 0.45) 0-4 **Two-pollutant Models** Nonaccidental PM_{10}+NO_2: 0.14% (-0.07, 0.36) 0 PM_{10}+SO_2: 0.37% (0.20, 0.55) 0 PM_{10}+O_3: 0.34% (0.17, 0.51) 0 Cardiovascular PM_{10}+NO_2: 0.34% (0.04, 0.63) 0 PM_{10}+SO_2: 0.53% (0.28, 0.77) 0 PM_{10}+O_3: 0.50% (0.26, 0.74) 0 Stroke PM_{10}+NO_2: 0.28% (-0.07, 0.63) 0 PM_{10}+SO_2: 0.49% (0.21, 0.78) 0 PM_{10}+O_3: 0.44 (0.16, 0.72) 0

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			Cardiac PM_{10}+NO_2: 0.24% (-0.27, 0.75) 0 PM_{10}+SO_2: 0.43 (0.01, 0.84) 0 PM_{10}+O_3: 0.44% (0.03, 0.85) 0 Respiratory PM_{10}+NO_2: 0.46% (-0.19, 1.12) 0 PM_{10}+SO_2: 0.64% (0.11, 1.18) 0 PM_{10}+O_3: 0.67% (0.15, 1.20) 0 Cardiopulmonary PM_{10}+NO_2: 0.26% (-0.02, 0.55) 0 PM_{10}+SO_2: 0.46% (0.23, 0.70) 0 PM_{10}+O_3: 0.44% (0.21, 0.67) 0
Reference: Qian et al. (2008, 156894) **Period of Study:** Jul 2001-Jun 2004 **Location:** Wuhan, China	**Outcome:** Mortality: Total (nonaccidental) (<800) Cardiovascular (390-459) Stroke (430-438) Cardiac diseases (390-398, 410-429) Respiratory (460-519) Cardiopulmonary (390-459, 460-519) **Study Design:** Time-series **Statistical Analyses:** Poisson GLM, natural splines and penalized splines **Age Groups:** All ages <65 yr ≥ 65 yr	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** Normal temperature: 145.7 (64.6) Low temperature: 117.3 (49.5) High temperature: 96.3 (27.9) **Range (Min, Max):** NR **Copollutant (correlation):** Normal temperature: NO_2: r = 0/72 SO_2: r = 0.59 O_3: r = 0.06 Low temperature: NO_2: r = 0.83 SO_2: r = 0.74 O_3: r = 0.19 High temperature: NO_2: r = 0.68 SO_2: r = 0.15 O_3: r = 0.65	**Increment:** 10 µg/m^3 **% Increase (Lower CI, Upper CI) lag:** Nonaccidental: Normal: All ages: 0.36 (0.17, 0.56) 0-1 <65: 0.23 (-0.10, 0.56) 0-1 ≥ 65: 0.51 (0.18, 0.64) 0-1 PM_{10}+NO_2: 0.07 (-0.17, 0.30) 0-1 PM_{10}+SO_2: 0.27 (0.06, 0.47) 0-1 PM_{10}+O_3: 0.38 (0.18, 0.58) 0-1 Low: All ages: 0.62 (-0.09, 1.34) 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 PM_{10}+NO_2: 0.11 (-0.23, 0.45) 0-1 PM_{10}+SO_2: 0.27 (-0.02, 0.55) 0-1 PM_{10}+O_3: 0.42 (0.15, 0.70) Low: All ages: 0.72 (-0.25, 1.70) 0-1 <65: 2.63 (0.67, 4.63) 0-1 ≥ 65: 0.24 (-0.84, 1.32) 0-1 PM_{10}+NO_2: 0.37 (-0.62, 1.38) 0-1 PM_{10}+SO_2: 0.50 (-0.47, 1.49) 0-1 PM_{10}+O_3: 0.82 (-0.16, 1.80) 0-1 High: All ages: 3.28 (1.24, 5.37) 0-1 <65: 4.32 (0.10, 8.71) 0-1 ≥ 65: 3.03 (0.77, 5.34) 0-1 PM_{10}+NO_2: 3.00 (0.95, 5.09) 0-1 PM_{10}+SO_2: 3.20 (1.16, 5.29) 0-1 PM_{10}+O_3: 3.71 (1.50, 5.96) 0-1 Stroke: Normal: All ages: 0.38 (0.06, 0.70) <65: 0.17 (-0.53, 0.88) 0-1 ≥ 65: 0.43 (0.07, 0.79) 0-1 PM_{10}+NO_2: 0.09 (-0.31, 0.49) 0-1 PM_{10}+SO_2: 0.31 (-0.03, 0.64) 0-1 PM_{10}+O_3: 0.38 (0.05, 0.71) 0-1 Low: All ages: 0.67 (-0.50, 1.85) 0-1 <65: 2.85 (0.34, 5.42) 0-1 ≥ 65: 0.11 (-1.22, 1.45) 0-1 PM_{10}+NO_2: 0.29 (-0.90, 1.51) 0-1 PM_{10}+SO_2: 0.53 (-0.65, 1.73) 0-1

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			$PM_{10}+O_3$: 0.69 (-0.48, 1.87) 0-1 High: All ages: 2.35 (-0.03, 4.78) 0-1 <65: 4.54 (-0.79, 10.16) 0-1 ≥ 65: 1.83 (-0.83, 4.57) $PM_{10}+NO_2$: 2.05 (-0.34, 4.49) 0-1 $PM_{10}+SO_2$: 2.31 (-0.07, 4.74) 0-1 $PM_{10}+O_3$: 2.77 (0.25, 5.35) 0-1 Cardiac: Normal: All ages: 0.32 (-0.14, 0.79) 0-1 <65: -0.04 (-1.07, 1.01) 0-1 ≥ 65: 0.40 (-0.10, 0.91) 0-1 $PM_{10}+NO_2$: 0.02 (-0.57, 0.60) 0-1 $PM_{10}+SO_2$: 0.11 (-0.38, 0.61) 0-1 $PM_{10}+O_3$: 0.41 (-0.06, 0.89) 0-1 Low: All ages: 0.50 (-1.10, 2.13) 0-1 <65: 1.79 (-1.65, 5.35) 0-1 ≥ 65: 0.19 (-1.55, 1.95) 0-1 $PM_{10}+NO_2$: 0.12 (-1.53, 1.80) 0-1 $PM_{10}+SO_2$: 0.14 (-1.48, 1.78) 0-1 $PM_{10}+O_3$: 0.72 (-0.90, 2.37) 0-1 High: All ages: 3.31 (-0.22, 6.97) 0-1 <65: 2.71 (-4.58, 10.56) 0-1 ≥ 65: 3.45 (-0.41, 7.46) 0-1 $PM_{10}+NO_2$: 3.01 (-0.54, 6.69) 0-1 $PM_{10}+SO_2$: 3.17 (-0.37, 6.84) 0-1 $PM_{10}+O_3$: 4.92 (0.96, 9.03) 0-1 Respiratory: Normal: All ages: 0.80 (0.25, 1.35) 0-1 <65: -0.35 (-1.85, 1.18) 0-1 ≥ 65: 0.93 (0.38, 1.50) 0-1 $PM_{10}+NO_2$: 0.30 (-0.39, 0.99) 0-1 $PM_{10}+SO_2$: 0.64 (0.07, 1.22) 0-1 $PM_{10}+O_3$: 0.84 (0.28, 1.41) 0-1 Low: All ages: 1.07 (-0.76, 2.95) 0-1 <65: -1.13 (-6.33, 4.35) 0-1 ≥ 65: 1.30 (-0.57, 3.20) 0-1 $PM_{10}+NO_2$: 0.44 (-1.46, 2.36) 0-1 $PM_{10}+SO_2$: 0.80 (-1.05, 2.69) 0-1 $PM_{10}+O_3$: 1.11 (-0.73, 2.99) 0-1 High: All ages: 1.15 (-3.54, 6.07) 0-1 <65: -3.42 (-15.82, 10.80) 0-1 ≥ 65: 1.76 (-3.03, 6.78) 0-1 $PM_{10}+NO_2$: 0.63 (-4.07, 5.55) 0-1 $PM_{10}+SO_2$: 1.03 (-3.66, 5.94) 0-1 $PM_{10}+O_3$: 2.66 (-2.44, 8.02) 0-1 Cardiopulmonary: Normal: All ages: 0.45 (0.19, 0.70) 0-1 <65: 0.07 (-0.47, 0.61) 0-1 ≥ 65: 0.53 (0.25, 0.81) 0-1 $PM_{10}+NO_2$: 0.15 (-0.17, 0.47) 0-1 $PM_{10}+SO_2$: 0.34 (0.07, 0.61) 0-1 $PM_{10}+O_3$: 0.43 (0.17, 0.70) 0-1 Low: All ages: 0.69 (-0.22, 1.61) 0-1 <65: 1.95 (0.04, 3.90) 0-1 ≥ 65: 0.43 (-0.57, 1.44) 0-1 $PM_{10}+NO_2$: 0.33 (-0.61, 1.27) 0-1 $PM_{10}+SO_2$: 0.50 (-0.42, 1.43) 0-1 $PM_{10}+O_3$: 0.76 (-0.16, 1.68) 0-1 High: All ages: 3.02 (1.03, 5.04) 0-1 <65: 3.49 (-0.66, 7.81) 0-1 ≥ 65: 2.91 (0.74, 5.12) 0-1 $PM_{10}+NO_2$: 2.70 (0.72, 4.73) 0-1 $PM_{10}+SO_2$: 2.95 (0.96, 4.97) 0-1 $PM_{10}+O_3$: 3.32 (1.16, 5.53) 0-1

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Ren et al. (2006, 092824) **Period of Study:** Jan 1996-Dec 2001 **Location:** Brisbane, Australia	**Outcome:** Mortality: Nonaccidental Cardiovascular (390-448) **Study Design:** Time-series **Statistical Analyses:** Poisson GAM, cubic spline **Age Groups:** All ages	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** 15.84 **Range (Min, Max):** (2.5, 60) **Copollutant:** O_3	The study presents quantitative results associated with an incremental increase in temperature, not PM_{10}.
Reference: Roberts (2004, 087924) **Period of Study:** 1987-1994 **Location:** Cook County, Illinois Allegheny County, Pennsylvania	**Outcome:** Mortality: Nonaccidental (<800) **Study Design:** Time-series **Statistical Analyses:** Poisson GAM, smooth splines Poisson GLM, natural cubic splines **Age Groups:** ≥ 65 yr	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Median (SD) unit:** Cook County Lower Temp.: 29.24 Middle Temp.: 30.03 Upper Temp.: 52.76 Allegheny County Lower Temp.: 16.50 Middle Temp.: 24.97 Upper Temp.: 55.42 Range (10th, 90th): Cook County Lower Tem.: (16.42, 46.42) Middle Temp.: (14.79, 56.33) Upper Temp.: (30.81, 82.81) Allegheny County Lower Temp.: (5.14, 34.54) Middle Temp.: (8.91, 57.91) Upper Temp.: (30.91, 88.99)	**Increment:** 10 µg/m^3 **% Increase (SE) lag:** GLM **Cook** α = 0.5 No Interaction: 0.288% (0.157) 0 Low Temp.: -0.272% (0.380) 0 Middle Temp.: 0.344% (0.165) 0 Upper Temp.: 0.281% (0.239) 0 No Interaction: 0.359% (0.149) 1 Low Temp.: -0.168% (0.372) 1 Middle Temp.: 0.361% (0.156) 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 α = 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 Middle Temp.: 0.161% (0.156) 1 Upper Temp.: 0.301% (0.278) 1 No Interaction: 0.260% (0.181) 0-1 ma Low Temp.: -0.163% (0.431) 0-1 ma Middle Temp.: 0.305% (0.188) 0-1 ma Upper Temp.: 0.207% (0.291) 0-1 ma No Interaction: 0.289% (0.225) 0-3 ma Low Temp.: 0.014% (0.459) 0-3 ma Middle Temp.: 0.311% (0.231) 0-3 ma Upper Temp.: 0.301% (0.334) 0-3 ma α = 2 No Interaction: 0.060% (0.158) 0 0 Low Temp.: -0.464% (0.486) 0 0 Middle Temp.: 0.115% (0.168) 0 0 Upper Temp.: -0.022% (0.319) 0 0 No Interaction: 0.101% (0.152) 1 Low Temp.: -0.432% (0.484) 1 Middle Temp.: 0.089% (0.160) 1 Upper Temp.: 0.455% (0.327) 1 No Interaction: 0.129% (0.184) 0-1 ma Low Temp.: -0.320% (0.546) 0-1 ma Middle Temp.: 0.157% (0.193) 0-1 ma Upper Temp.: 0.130% (0.346) 0-1 ma No Interaction: 0.090% (0.236) 0-3 ma Low Temp.: -0.319% (0.572) 0-3 ma Middle Temp.: 0.105% (0.244) 0-3 ma Upper Temp.: 0.193% (0.412) 0-3 ma

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			Allegheny α = 0.5 No Interaction: 0.078% (0.209) 0 Low Temp.: -0.759% (0.643) 0 Middle Temp.: 0.207% (0.216) 0 High Temp.: -0.367% (0.364) 0 No Interaction: 0.189% (0.206) 1 Low Temp.: -0.335% (0.691) 1 Middle Temp.: 0.293% (0.215) 1 High Temp.: -0.171% (0.349) 1 No Interaction: 0.224% (0.246) 0-1 ma Low Temp.: -0.753% (0.763) 0-1 ma Middle Temp.: 0.353% (0.253) 0-1 ma High Temp.: -0.142% (0.382) 0-1 ma No Interaction: 0.526% (0.300) 0-3 ma Low Temp.: 0.050% (0.733) 0-3 ma Middle Temp.: 0.688% (0.310) 0-3 ma High Temp.: -0.043% (0.436) 0-3 ma α = 1 No Interaction: 0.078% (0.211) 0 Low Temp.: -0.694% (0.656) 0 Middle Temp.: 0.214% (0.219) 0 High Temp.: -0.533% (0.430) 0 No Interaction: 0.179% (0.207) 1 Low Temp.: -0.283% (0.718) 1 Middle Temp.: 0.273% (0.217) 1 High Temp.: -0.221% (0.396) 1 No Interaction: 0.221% (0.249) 0-1 ma Low Temp.: -0.731% (0.794) 0-1 ma Middle Temp.: 0.348% (0.258) 0-1 ma High Temp.: -0.253% (0.447) 0-1 ma No Interaction: 0.464% (0.309) 0-3 ma Low Temp.: 0.056% (0.780) 0-3 ma Middle Temp.: 0.626% (0.319) 0-3 ma High Temp.: -0.356% (0.516) 0-3 ma α = 2 No Interaction: 0.034% (0.217) 0 Low Temp.: -1.059% (0.715) 0 Middle Temp.: 0.162% (0.230) 0 High Temp.: -0.233% (0.489) 0 No Interaction: 0.130% (0.214) 1 Low Temp.: -0.189% (0.800) 1 Middle Temp.: 0.157% (0.226) 1 High Temp.: 0.070% (0.471) 1 No Interaction: 0.183% (0.260) 0-1 ma Low Temp.: -0.918% (0.907) 0-1 ma Middle Temp.: 0.279% (0.273) 0-1 ma High Temp.: -0.001% (0.526) 0-1 ma No Interaction: 0.270% (0.331) 0-3 ma Low Temp.: -0.105% (0.898) 0-3 ma Middle Temp.: 0.394% (0.346) 0-3 ma High Temp.: -0.287% (0.615) 0-3 ma **GAM** **Cook** α = 0.5 No Interaction: 0.438% (0.151) 0 Low Temp.: -0.178% (0.364) 0 Middle Temp.: 0.439% (0.163) 0 Upper Temp.: 0.627% (0.197) 0 No Interaction: 0.495% (0.144) 1 Low Temp.: -0.114% (0.361) 1 Middle Temp.: 0.460% (0.151) 1 Upper Temp.: 0.938% (0.208) 1 No Interaction: 0.710% (0.169) 0-1 ma Low Temp.: 0.151% (0.379) 0-1 ma Middle Temp.: 0.686% (0.180) 0-1 ma Upper Temp.: 0.952% (0.214) 0-1 ma No Interaction: 0.923% (0.203) 0-3 ma Low Temp.: 0.532% (0.402) 0-3 ma Middle Temp.: 0.855% (0.210) 0-3 ma Upper Temp.: 1.289% (0.251) 0-3 ma α = 1 No Interaction: 0.190% (0.154) 0

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			Low Temp.: -0.338% (0.414) 0 Middle Temp.: 0.242% (0.162) 0 Upper Temp.: 0.161% (0.230) 0 No Interaction: 0.239% (0.146) 1 Low Temp.: -0.283% (0.406) 1 Middle Temp.: 0.248% (0.152) 1 Upper Temp.: 0.453% (0.244) 1 No Interaction: 0.353% (0.174) 0-1 ma Low Temp.: -0.074% (0.437) 0-1 ma Middle Temp.: 0.388% (0.182) 0-1 ma Upper Temp.: 0.345% (0.251) 0-1 ma No Interaction: 0.453% (0.213) 0-3 ma Low Temp.: 0.190% (0.460) 0-3 ma Middle Temp.: 0.455% (0.219) 0-3 ma Upper Temp.: 0.557% (0.294) 0-3 ma α = 2 No Interaction: 0.071% (0.157) 0 0 Low Temp.: -0.534% (0.478) 0 0 Middle Temp.: 0.132% (0.165) 0 0 Upper Temp.: 0.011% (0.264) 0 0 No Interaction: 0.099% (0.150) 1 Low Temp.: -0.467% (0.472) 1 Middle Temp.: 0.109% (0.156) 1 Upper Temp.: 0.329% (0.278) 1 No Interaction: 0.168% (0.180) 0-1 ma Low Temp.: -0.371% (0.525) 0-1 ma Middle Temp.: 0.216% (0.188) 0-1 ma Upper Temp.: 0.116% (0.290) 0-1 ma No Interaction: 0.149% (0.227) 0-3 ma Low Temp.: -0.291% (0.557) 0-3 ma Middle Temp.: 0.174% (0.233) 0-3 ma Upper Temp.: 0.210% (0.340) 0-3 ma **Allegheny** α = 0.5 No Interaction: 0.245% (0.203) 0 Low Temp.: -0.727% (0.648) 0 Middle Temp.: 0.314% (0.216) 0 High Temp.: 0.308% (0.287) 0 No Interaction: 0.446% (0.199) 1 Low Temp.: -0.307% (0.701) 1 Middle Temp.: 0.469% (0.211) 1 High Temp.: 0.556% (0.285) 1 No Interaction: 0.522% (0.237) 0-1 ma Low Temp.: -0.646% (0.761) 0-1 ma Middle Temp.: 0.567% (0.251) 0-1 ma High Temp.: 0.640% (0.307) 0-1 ma No Interaction: 0.977% (0.282) 0-3 ma Low Temp.: 0.307% (0.733) 0-3 ma Middle Temp.: 1.027% (0.296) 0-3 ma High Temp.: 1.001% (0.352) 0-3 ma α = 1 No Interaction: 0.107% (0.209) 0 Low Temp.: -0.819% (0.699) 0 Middle Temp.: 0.229% (0.219) 0 High Temp.: -0.214% (0.350) 0 No Interaction: 0.223% (0.205) 1 Low Temp.: -0.316% (0.751) 1 Middle Temp.: 0.295% (0.216) 1 High Temp.: 0.002% (0.341) 1 No Interaction: 0.267% (0.246) 0-1 ma Low Temp.: -0.797% (0.840) 0-1 ma Middle Temp.: 0.372% (0.257) 0-1 ma High Temp.: 0.035% (0.372) 0-1 ma No Interaction: 0.534% (0.302) 0-3 ma Low Temp.: 0.029% (0.810) 0-3 ma Middle Temp.: 0.660% (0.314) 0-3 ma High Temp.: 0.071% (0.431) 0-3 ma

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			α = 2 No Interaction: 0.061% (0.214) 0 Low Temp.: -1.048% (0.749) 0 Middle Temp.: 0.206% (0.226) 0 High Temp.: -0.332% (0.419) 0 No Interaction: 0.145% (0.211) 1 Low Temp.: -0.278% (0.816) 1 Middle Temp.: 0.210% (0.223) 1 High Temp.: -0.105% (0.394) 1 No Interaction: 0.180% (0.256) 0-1 ma Low Temp.: -1.028% (0.931) 0-1 ma Middle Temp.: 0.298% (0.269) 0-1 ma High Temp.: -0.114% (0.441) 0-1 ma No Interaction: 0.275% (0.324) 0-3 ma Low Temp.: -0.384% (0.915) 0-3 ma Middle Temp.: 0.436% (0.338) 0-3 ma High Temp.: -0.366% (0.513) 0-3 ma
Reference: Roberts (2004, 087924) **Period of Study:** 1987-1994 **Location:** Cook County, Illinois Allegheny County, Pennsylvania	**Outcome:** Mortality: Nonaccidental **Study Design:** Time-series **Statistical Analyses:** Poisson GLM **Age Groups:** ≥ 65 yr	**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 (Roberts, 2005, 087992) **Period of Study:** Cook County: 1987-2000. Allegheny County: 1987-1998 **Location:** Cook County, Illinois Allegheny County, Pennsylvania	**Outcome:** Mortality: Nonaccidental **Study Design:** Time-series **Statistical Analyses:** Poisson **Age Groups:** ≥ 65 yr	**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
Reference: Roberts (2006, 089762) **Period of Study:** 1987-2000 **Location:** Cook County, Illinois Suffolk County, Massachusetts (NMMAPS)	**Outcome:** Mortality: Nonaccidental **Study Design:** Time-series **Statistical Analyses:** Poisson GLM **Age Groups:** ≥ 65 yr	**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 µg/m^3 Suffolk County: 14.0 µg/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

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Roberts and Martin (2006, 097799) **Period of Study:** 1987-2000 **Location:** Cook County, Illinois (NMMAPS)	**Outcome:** Mortality: Nonaccidental **Study Design:** Time-series **Statistical Analyses:** Dose-response 1. Piecewise linear relationship (no-threshold) with change point at 25 µg/m^3 and 50 µg/m^3 2. Piecewise linear relationship (threshold), exposure below 25 µg/m^3 no effect, and exposures above 50 µg/m^3 having a different effect then exposures between 25 µg/m^3 and 50 µg/m^3 **Age Groups:** ≥ 65 yr	**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: Nonaccidental 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:** Nonaccidental Model 1 Base df: 0.079 (0.050) 0 Double df: 0.044 (0.046) 0 Half df: 0.107 (0.052) 0 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) 0-2 ma **Notes:** Model 1 uses current day's mortality count, while Model 2 uses a 3-day moving total mortality count.

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
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 (nonaccidental) 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 µg/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 (nonaccidental) 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 Lake City.

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Roberts and Martin (2007, 156916) **Period of Study:** 1987-2000 **Location:** 10 U.S. cities (NMMAPS)	**Outcome:** Mortality: Nonaccidental **Study Design:** Time-series **Statistical Analyses:** Poisson **Age Groups:** ≥ 65 yr	**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 μg/m^3 Slope below: 0.00130 (0.00016) 0-5 Slope above: -0.00163 (0.00026) 0-5 Change-point: 30 μg/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 μg/m^3 Slope below: 0.00044 (0.00011) 3-day ma Slope above: -0.00077 (0.00020) 3- day ma Change-point: 30 μg/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 (nonaccidental) (<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 μg/m^3 to Torino: 65 μg/m^3) Percentile (90th): Range: (Stockholm: 27 μg/m^3 to Torino: 129 μg/m^3) **Copollutant (correlation):** BS	The study does not present quantitative results.
Reference: Schwartz (2004, 078998) **Period of Study:** 1986-1993 **Location:** 14 U.S. cities	**Outcome:** Mortality: Nonaccidental (<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 μg/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

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Schwartz (2004, 053506) **Period of Study:** 1986-1993 **Location:** 14 U.S. cities	**Outcome:** Mortality: Nonaccidental (<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: 23-36 µg/m^3 IQR (25th, 75th): Range 25th: 17-24 µg/m^3 Range 75th: 31-57 µg/m^3 **Copollutant (correlation):** CO SO_2 NO_2 O_3	**Increment:** 10 µg/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:** Jul 1996-Jun 1998 **Location:** São Paulo, Brazil	**Outcome:** Mortality: Myocardial infarction **Study Design:** Time-series **Statistical Analyses:** Poisson GAM **Age Groups:** ≥ 35 yr	**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 µg/m^3 **β (SE) lag:** PM_{10}: 0.001 (0.001) PM_{10}+CO+SO_2: 0.0004 (0.0008)
Reference: Simpson et al. (2005, 087438) **Period of Study:** 1/1996-12/1999 **Location:** 4 Australian cities	**Outcome:** Mortality: Nonaccidental (<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 µg/m^3 **% Increase (Lower CI, Upper CI)** **lag:** 0.2% (-0.8, 1.2)
Reference: Slaughter et al. (2005, 073854) **Period of Study:** Jan 1995-Dec 1999 **Location:** Spokane, Washington	**Outcome:** Mortality: Nonaccidental (<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) µg/m^3 **Copollutant (correlation):** PM_{10} $PM_{10-2.5}$: r = 0.94 CO: r = 0.32	**Increment**: : 25 µg/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: Nonaccidental (<800) **Study Design:** Time-series **Statistical Analyses:** Poisson Principal component analysis (PCA) **Age Groups:** All ages	**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 µg/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
Reference: Stafoggia et al. (2008, 157005) **Period of Study:** 1997-2004	**Outcome:** Mortality: Total (nonaccidental) (<800)	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD) unit:** Bologna: 50.4 (31.7)	**Increment:** 10 µg/m^3 **% Increase (Lower CI, Upper CI) lag:** Cardiovascular All yr: 0.63% (0.31, 1.38) 0-1

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Location: 9 Italian cities	Cardiovascular (390-459) Respiratory (460-519) Other natural causes **Study Design:** Time-stratified case-crossover **Statistical Analyses:** Conditional logistic regression **Age Groups:** ≥ 35 yr	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	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 yr: 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 yr: 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 <50th Percentile: 0.07% (-0.27, 0.41) 0-1 50th-75th Percentile: 1.08% (-0.02, 2.19) 0-1 >75th Percentile: 2.30% (1.06, 3.56) 0-1 Total (nonaccidental) All yr: 0.53% (0.25, 0.80) 0-1 Winter: 0.20% (-0.08, 0.49) 0-1 Spring: 0.62% (0.14, 1.10) 0-1 Summer: 2.54% (1.31, 3.78) 0-1 Fall: 1.21% (0.37, 2.06) 0-1 Apparent Temperature <50th Percentile: 0.21% (-0.06, 0.47) 0-1 50th-75th Percentile: 1.60% (0.64, 2.57) 0-1 >75th Percentile: 2.55% (1.58, 3.52) 0-1 **β coefficient (SE) lag:** Linear interaction PM_{10} and Apparent Temperature Cardiovascular <50th Percentile: -0.000117 (0.000415) 0-1 50th-75th Percentile: 0.003445 (0.001407) 0-1 >75th Percentile: 0.002764 (0.001795) 0-1 Respiratory <50th Percentile: 0.001119 (0.000943) 0-1 50th-75th Percentile: -0.001120 (0.003480) 0-1 >75th Percentile: 0.005306 (0.004350) 0-1 Other natural causes <50th Percentile: 0.000411 (0.000383) 0-1 50th-75th Percentile: -0.001526 (0.001207) 0-1 >75th Percentile:

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			0.002564 (0.001958) 0-1 Total (nonaccidental) <50th Percentile: 0.000246 (0.000269) 0-1 50th-75th Percentile: 0.000584 (0.000880) 0-1 >75th Percentile: 0.002396 (0.001629) 0-1
Reference: Stölzel et al. (2007, 091374) **Period of Study:** Sep 1995-Aug 2001 **Location:** Erfurt, Germany	**Outcome:** Mortality: Total (nonaccidental) (<800) Cardio-respiratory (390-459, 460-519, 785, 786) **Study Design:** Time-series **Statistical Analyses:** Poisson GAM **Age Groups:** All ages	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD) unit:** : 31.9 (23.2) IQR (25th, 75th): (16.5, 39.5) **Copollutant (correlation):** MC0.1-0.5: r = 0.85 MC0.01-2.5: r = 0.84 NO: r = 0.54 NO_2: r = 0.62 CO: r = 0.50	**Increment:** 23 µg/m^3 **Relative Risk (Lower CI, Upper CI) lag:** Total (nonaccidental) 1.004 (0.980 1.029) 0 1.004 (0.981 1.027) 1 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
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_{10} **Averaging Time:** 24-h avg **Median (SD) unit:** Lag 0: 28.05 Lag 1: 27.97 Lag 2: 28.40 **Range (Min, Max):** (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: :** 16.51 µg/m^3 **Odds Ratio (Lower CI, Upper CI) lag:** Overall 1.05 (0.87, 1.27) 0 0.91 (0.75, 1.11) 1 1.03 (0.82, 1.28) 2

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Sunyer et al. (2002, 034835) **Period of Study:** 1985-1995 **Location:** Barcelona, Spain	**Outcome:** Mortality: Respiratory mortality **Study Design:** Case-crossover **Statistical Analyses:** Condition logistic regression **Age Groups:** >14 Study population: Asthmatic individuals: 5,610	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Median (SD) unit:** 61.2 **Range (Min, Max):** (17.3, 240.7) **Copollutant:** BS NO_2 O_3 SO_2 CO	**Increment:** 32.7 µg/m^3 **Odds Ratio (Lower CI, Upper CI) lag:** Asthmatic individuals with 1 ED visit 0.884 (0.672, 1.162) 0-2 avg Asthmatic individuals with >1 ED visit 1.084 (0.661, 1.778) 0-2 avg Asthma/COPD individuals with >1 ED visit 1.011 (0.746, 1.368) 0-2 avg
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 (nonaccidental) (<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 µg/m^3 **β (x 1000) (SE (x 1000)):** Total (nonaccidental) 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.
Reference: Tsai et al. (2003, 050480) **Period of Study:** 1994-2000 **Location:** Kaohsiung, Taiwan	**Outcome:** Mortality: Total (nonaccidental) (<800) Respiratory (460-519) Circulatory (390-459) **Study Design:** Bidirectional case-crossover **Statistical Analyses:** Conditional logistic regression **Age Groups:** All ages	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** 81.45 **Range (Min, Max):** (20.50, 232.00) **Copollutant:** SO_2 NO_2 CO O_3	**Increment:** 67.00 µg/m^3 **Odds Ratio (Lower CI, Upper CI) lag:** Total (nonaccidental) 1.000 (0.947, 1.056) 0-2 avg Respiratory 1.023 (0.829, 1.264) 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 (nonaccidental) (<800) Respiratory (460-519) Cardiovascular (390-459) Other-causes **Study Design:** Time-series **Statistical Analyses:** Poisson GAM, LOESS **Age Groups:** All ages 55-64 yr 65-74 yr ≥ 75 yr	**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 µg/m^3 **% Increase (Lower CI, Upper CI) lag:** Total (nonaccidental) 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
Reference: Vedal et al. (2003, 039044) **Period of Study:** Jan 1994-Dec 1996 **Location:** Vancouver, British Columbia, Canada	**Outcome:** Mortality: Total (nonaccidental) (<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:** Jan 1995-Dec 1995 **Location:** Chongqing, China	**Outcome:** Mortality: Total (nonaccidental) (<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 mo of the study period.	**Increment:** 100 µg/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
Reference: Vichit-Vadakan et al. (2008, 157095) **Period of Study:** Jan 1999-Dec 2003 **Location:** Bangkok, Thailand	**Outcome (ICD10):** Mortality: Nonaccidental (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 yr 5-44 yr 18-50 yr 45-64 yr ≥ 50 yr ≥ 65 yr ≥ 75 yr	**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 µg/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 nonaccidental 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

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			≥ 65: 1.5 (0.9, 2.1) 0-1 ≥ 75: 2.2 (1.3, 3.0) 0-1 Sex-specific for nonaccidental Male: 1.2 (0.7, 1.7) 0-1 Female: 1.3 (0.7, 1.9) 0-1 Nonaccidental 1.2 (0.8, 1.6) 0 0.9 (0.6, 1.3) 1 0.9 (0.5, 1.3) 2 0.8 (0.4, 1.2) 3 0.3 (-0.1, 0.7) 4 1.3 (0.8, 1.7) 0-1 1.4 (0.9, 1.9) 0-4 Cardiovascular 1.5 (0.5, 2.6) 0 1.7 (0.7, 2.7) 1 1.6 (0.6, 2.6) 2 0.8 (-0.1, 1.8) 3 -0.1 (-1.1, 0.9) 4 1.9 (0.8, 3.0) 0-1 1.9 (0.6, 3.2) 0-4 Respiratory 1.0 (-0.3, 2.3) 0 0.8 (-0.5, 2.0) 1 1.1 (-0.1, 2.3) 2 1.3 (0.1, 2.6) 3 0.7 (-0.6, 1.9) 4 1.0 (-0.4, 2.4) 0-1 1.9 (1.2, 2.6) 0-4 ≥ 65 1.5 (0.9, 2.0) 0 1.1 (0.6, 1.7) 1 1.1 (0.6, 1.6) 2 1.2 (0.6, 1.7) 3 0.7 (0.2, 1.2) 4 1.5 (0.9, 2.1) 0-1 1.9 (1.2, 2.6) 0-4 **Sensitivity analysis:** Nonaccidental (df): 3: 1.3 (0.9, 1.8) 4: 1.2 (0.8, 1.7) 6: 1.3 (0.8, 1.7) 6, with SO_2: 1.2 (0.8, 1.7) 6, with NO_2: 1.0 (0.2, 1.8) 6, with O_3: 1.1 (0.6, 1.7) 9: 1.1 (0.7, 1.6) 12: 1.1 (0.6, 1.5) 15: 1.2 (0.7, 1.6) Cardiovascular (df): 3: 1.8 (0.8, 2.7) 4: 1.6 (0.7, 2.6) 6: 1.7 (0.7, 2.7) 6, with SO_2: 2.0 (0.9, 3.3) 6, with NO_2: 2.3 (0.2, 4.3) 6, with O_3: 1.8 (0.5, 3.2) 9: 1.7 (0.6, 2.8) 12: 1.8 (0.7 to 3.0) 15: 2.2 (0.9, 3.4)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Villeneuve et al. (2003, 055051) **Period of Study:** 1986-1999 **Location:** Vancouver, Canada	**Outcome:** Mortality: Nonaccidental (<800) Cardiovascular (401-440) Respiratory (460-519) Cancer (140-239) **Study Design:** Time-series **Statistical Analyses:** Poisson, natural splines **Age Groups:** ≥ 65 yr	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** Daily 14.0 Every 6th Day 19.6 **Range (Min, Max):** Daily (3.8, 52.2) Every 6th Day (3.5, 63.0) **Copollutant:** SO_2 CO NO_2 O_3 $PM_{2.5}$ $PM_{10-2.5}$	**Increment:** 15.4 µg/m^3 **% Increase (Lower CI, Upper CI) lag:** Nonaccidental 3.7% (-0.5, 8.0) 0-2 avg 2.6% (-0.9, 6.1) 0 2.7% (-0.7, 6.2) 1 1.9% (-1.4, 5.3) 2 Cardiovascular 3.4% (-2.7, 9.8) 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
Reference: Welty et al. (2008, 157134) **Period of Study:** 1987-2000 **Location:** Chicago, Illinois	**Outcome:** Mortality: Total (nonaccidental) **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 µg/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 (nonaccidental) (<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 µg/m^3 **% Increase (SE) lag:** Distributed Lag Model: Seasonally-Temporally Varying Temperature variables: 0, 1-2, 1-7, 1-14 S(t, 1 × yr): 0.229 (0.053) 1 S(t, 2 × yr): 0.220 (0.053) 1 S(t, 4 × yr): 0.187 (0.050) 1 S(t, 8 × yr): 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 × yr): 0.195 (0.048) 1 S(t, 2 × yr): 0.200 (0.051) 1 S(t, 4 × yr): 0.176 (0.050) 1 S(t, 8 × yr): 0.149 (0.050) 1 Distributed Lag Model: Nonlinear Temperature variables: 0, 1-2, 1-7, 1-14 S(t, 4 × yr): 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 × yr): 0.172 (0.045) 1 Temperature variables: S(0,2), S(1-2,2), S(1-7,2), S(1-14,2) S(t, 4 × yr): 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 × yr): 0.189 (0.047) 1

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			Temperature variables: S(0,4), S(1-2,4), S(1-7,4), S(1-14,4) S(t, 4 × yr): 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 × yr): 0.190 (0.048) 1 Temperature variables: 0, 1-2, 1-7 S(t, 4 × yr): 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 × yr): 0.186 (0.044) 1 Temperature variables: S(0,2), S(1-2,2), S(1-7,2) S(t, 4 × yr): 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 × yr): 0.201 (0.047) 1 Temperature variables: S(0,4), S(1-2,4), S(1-7,4) S(t, 4 × yr): 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 × yr): 0.205 (0.047) 1 Temperature variables: S(0,4), S(1-2,4) S(t, 4 × yr): 0.250 (0.045) 1 Temperature variables: S(0,4), S(1-2,4), S(0×1-2,4) S(t, 4 × yr): 0.253 (0.044) 1 Temperature variables: S(0,4) S(t, 4 × yr): 0.220 (0.045) 1 **Notes:** 0 indicates current-day temperature 1-r indicates avg of lag 1 through lag r temperature S (, ρ) indicates a natural spline smooth with ρ degrees of freedom. S (t, α x yr) indicates the natural spline smooth of time with degrees of freedom equal to α x (number of yr of data).

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Wong et al. (2007, 098391) **Period of Study:** Jan 1998-Dec 1998 **Location:** Hong Kong, China	**Outcome:** Mortality: Total (nonaccidental) (<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 yr; ≥ 65 yr	**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 µg/m^3 **% Excess Risk (Lower CI, Upper CI) lag:** Main Analysis Nonaccidental 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) 2 ≥ 65: 0.25% (-2.62, 3.19) 0 -0.66% (-3.29, 2.04) 2 Sensitivity Analysis Poisson Regression Nonaccidental ≥ 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 Nonaccidental ≥ 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) 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 Nonaccidental ≥ 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) **Period of Study:** Jan 1998-Dec 1998	**Outcome:** Mortality: Total (nonaccidental) (<800)	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** 48.1 (24.3)	**Increment:** 10 µg/m^3 **% Excess Risk (Lower CI, Upper CI) lag:** Nonaccidental

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Location: Hong Kong, China	Cardiorespiratory (390-519) **Study Design:** Main analysis: Time-series **Sensitivity analysis:** Case-only **Statistical Analyses:** Main analysis: Poisson GAM, natural cubic spline **Sensitivity analysis:** Logistic regression **Age Groups:** ≥ 30 yr; ≥ 65 yr	**Range (Min, Max):** (15.5, 140.5) **Copollutant:** NO_2 SO_2 O_3	Exercise ≥ 30: 0.13% (-1.16, 1.44) 1 ≥ 65: 0.24% (-1.16, 1.67) 1 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) 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) Nonaccidental 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 Nonaccidental 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 ≥ 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

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			Case-only by Exercise Group (Never as Reference) Nonaccidental ≥ 30 Low: -3.34% (-5.77 to -0.85) 1 Moderate: -6.32% (-8.55 to -4.03) 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 yr; ≥ 65 yr	**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 µg/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) 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

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Wong et al. (2008, 157152) **Period of Study:** Bangkok: 1999-2003 Hong Kong: 1996-2002 Shanghai & Wuhan: 2001-2004 **Location:** Bangkok, Thailand Hong Kong, Shanghai, and Wuhan, China	**Outcome (ICD10):** Mortality: Natural causes (A00-R99) Cardiovascular (I00-I99) Respiratory (J00-J98) **Study Design:** Time-series **Statistical Analyses:** Poisson GLM, natural splines **Age Groups:** All ages ≥ 65 yr ≥ 75 yr	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** Bangkok: 52.0 Hong Kong: 51.6 Shanghai: 102.0 Wuhan: 141.8 **Range (Min, Max):** Bangkok: (21.3, 169.2) Hong Kong: (13.7, 189.0) Shanghai: (14.0, 566.8) Wuhan: (24.8, 477.8) **Copollutant:** NO_2 SO_2 O_3	**Increment:** 10 µg/m^3 **% Excess Risk (Lower CI, Upper CI) lag:** Random Effects (4 cities) Natural causes: 0.55% (0.26, 0.85) 0-1 Cardiovascular: 0.58% (0.22, 0.93) 0-1 Respiratory: 0.62% (0.22, 1.02) 0-1 Random Effects (3 Chinese cities) Natural causes: 0.37% (0.21, 0.54) 0-1 Cardiovascular: 0.44% (0.19, 0.68) 0-1 Respiratory: 0.60% (0.16, 1.04) 0-1 Sensitivity Analysis Random Effects (4 cities) Omit PM_{10}>95th: 0.53% (0.27, 0.78) 0-1 Omit PM_{10}>75th: 0.53% (0.29, 0.78) 0-1 Omit PM_{10}>180 µg/m^3: 0.65% (0.24, 1.06) 0-1 Omit stations with high traffic source: 0.55% (0.26, 0.85) 0-1 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}>95th: 0.47% (0.21, 0.73) 0-1 Omit PM_{10}>75th: 0.55% (0.24, 0.85) 0-1 Omit PM_{10}>180 µg/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:** Jan 1996-Dec 2002 **Location:** Hong Kong	**Outcome (ICD10):** Mortality: Nonaccidental (A00-T99 Z00-Z99) Cardiovascular (I00-I99) Respiratory (J00-J98) **Study Design:** Time-series **Statistical Analyses:** Poisson GLM, natural splines **Age Groups:** All ages	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** 51.6 (25.3) **Range (Min, Max):** (13.5, 188.5) **Copollutant:** NO_2 SO_2 O_3	**Increment:** 10 µg/m^3 **% Excess Risk (Lower CI, Upper CI) lag:** Nonaccidental: Low SDI 0.37 (-0.10, 0.84) 0 0.40 (-0.04, 0.84) 1 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

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			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 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 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 Nonaccidental: 0.23 (-0.25, 0.72) 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 Nonaccidental: 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 Nonaccidental: 0.04 (-0.15, 0.22) 0-1 Cardiovascular: 0.27 (-0.07, 0.61) 0-1 Respiratory: -0.04 (-0.46, 0.37)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			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 µg/m^3
Period of Study: 1994-1998	Nonaccidental (<800)	**Averaging Time:** 24-h avg	**Odds Ratio (Lower CI, Upper CI) lag:**
Location: Taipei, Taiwan	Circulatory (390-459)	**Mean (SD):** 51.99	Nonaccidental
	Respiratory (460-519)	**Range (Min, Max):** (13.71, 211.30)	0.995 (0.971, 1.020) 0
	Study Design: Bi-directional case-crossover	**Copollutant:** SO_2 NO_2 CO O_3	Respiratory
	Statistical Analyses: Conditional logistic regression		0.986 (0.906, 1.074) 0
	Age Groups: All ages		Circulatory
			0.988 (0.942, 1.035)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Zanobetti et al. (2003, 042812) **Period of Study:** 1990-1997 **Location:** 10 European cities (APHEA2)	**Outcome:** Mortality: Nonaccidental (<800) Circulatory (390-459) Respiratory (460-519) **Study Design:** Time-series **Statistical Analyses:** Poisson GAM **Age Groups:** 15-64 yr 65-74 yr ≥ 75 yr	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** Athens: 42.7 (12.9) Budapest: 41 (9.1) Lodz: 53.5 (15.5) London: 28.8 (13.7) Madrid: 37.8 (17.7) Paris: 22.5 (11.5) Prague: 76.2 (45.7) Rome: 58.7 (17.4) Stockholm: 15.5 (7.9) Tel Aviv: 50.3 (57.5) **Range (Min, Max):** NR **Copollutant (correlation):** NR	**Increment:** 10 µg/m^3 **% Increase (Lower CI, Upper CI) lag:** Cardiovascular 0.69% (0.31, 1.08) 0-1 avg 40-day distributed lag 1.99% (1.44, 2.54) 4th degree 1.97% (1.38, 2.55) Unrestricted Respiratory 0.74% (-0.17, 1.66) 0-1 avg 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 Nonaccidental 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) 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

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Zeka et al. (2005, 088068) **Period of Study:** Jan 1989-Dec 2000 **Location:** 20 U.S. cities	**Outcome (ICD10):** Mortality: All-cause (nonaccidental) (V01-Y98) Heart Disease (I01-I51) IHD (I20-I25) Myocardial infarction (I21, I22) Dysrhythmias (I46-I49) Heart failure (I50) Stroke (I60-I69) Respiratory (J00-J99) Pneumonia (J12-J18) COPD (J40-J44, J47) **Study Design:** Time-stratified case-crossover **Statistical Analyses:** Conditional logistic regression **Age Groups:** All ages	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** Birmingham: 31.9 (18.0) µg/m^3 Boulder: 22.1 (11.3) Caton: 26.6 (11.5) Chicago: 33.7 (16.4) Cincinnati: 31.4 (13.9) Cleveland: 37.5 (18.7) Colorado Springs: 24.0 (13.2) Columbus: 28.5 (12.5) Denver: 28.5 (12.8) Detroit: 32.1 (17.7) Honolulu: 15.9 (6.8) Minneapolis: 24.7 (12.3) Nashville: 30.1 (12.1) New Haven: 25.4 (14.4) Pittsburgh: 30.2 (18.5) Provo: 33.7 (22.2) Seattle: 26.4 (14.7) Salt lake City: 35.0 (20.8) µ Terra Haute: 29.2 (14.6) µ Youngstown: 30.8 (13.9) **Range (Min, Max):** NR **Copollutant (correlation):** NR	**Increment:** 10 µg/m^3 **% Increase (Lower CI, Upper CI) lag:** Single-lag model All-Cause (nonaccidental) 0.20% (0.08, 0.32) 0 0.35% (0.21, 0.49) 1 0.24% (0.14, 0.34) 2 Respiratory 0.34% (-0.07, 0.75) 0 0.52% (0.15, 0.89) 1 0.51% (0.16, 0.86) 2 COPD -0.06% (-0.63, 0.51) 0 0.43% (-0.14, 1.00) 1 0.39% (-0.16, 0.94) 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) 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 (nonaccidental) 0.45% (0.25, 0.65) 0-3 Respiratory 0.87% (0.38, 1.36) 0-3 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) **Period of Study:** Jan 1989-Dec 2000 **Location:** 20 U.S. cities	**Outcome (ICD10):** Mortality: All-cause (nonaccidental) (V01-Y98) Heart Disease (I01-I51) Myocardial infarction (I21, I22) Stroke (I60-I69) Respiratory (J00-J99) **Study Design:** Time-stratified case-	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** Birmingham: 31.9 (18.0) µg/m^3 Boulder: 22.1 (11.3) Caton: 26.6 (11.5) Chicago: 33.7 (16.4) Cincinnati: 31.4 (13.9) Cleveland: 37.5 (18.7) Colorado Springs: 24.0 (13.2)	**Increment:** 10 µg/m^3 **% Increase (Lower CI, Upper CI) lag: All-cause (nonaccidental)** Male: 0.46% (0.28, 0.64) 1-2 avg Female: 0.37% (0.17, 0.57) 1-2 avg White: 0.40% (0.22, 0.58) 1-2 avg Black: 0.37% (-0.02, 0.76) 1-2 avg **Age:** <65: 0.25% (0.01, 0.49) 1-2 avg 75: 0.23% (-0.06, 0.52) 1-2 avg

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
	crossover **Statistical Analyses:** Conditional logistic regression **Age Groups:** All ages <65 yr 65-75 yr >75 yr	Columbus: 28.5 (12.5) Denver: 28.5 (12.8) Detroit: 32.1 (17.7) Honolulu: 15.9 (6.8) Minneapolis: 24.7 (12.3) Nashville: 30.1 (12.1) New Haven: 25.4 (14.4) Pittsburgh: 30.2 (18.5) Provo: 33.7 (22.2) Seattle: 26.4 (14.7) Salt lake City: 35.0 (20.8) Terra Haute: 29.2 (14.6) Youngstown: 30.8 (13.9) **Range (Min, Max):** NR **Copollutant (correlation):** NR	>75: 0.64% (0.44, 0.84) 1-2 avg **Educational Attainment:** Low (<8 yr): 0.62% (0.29, 0.95) 1-2 avg Medium (8-12 yr): 0.36% (0.12, 0.60) 1-2 avg High (>12 yr): 0.27% (-0.004, 0.54) 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 yr): 0.82% (-0.32, 1.96) 0-3 Medium (8-12 yr): 0.88% (0.12, 1.64) 0-3 High (>12 yr): 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, 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 yr): 0.72% (0.23, 1.21) 2 Medium (8-12 yr): 0.38% (0.07, 0.69) 2 High (>12 yr): 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

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			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) 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 yr): 0.33% (-0.83, 1.49) 0 Medium (8-12 yr): 0.79% (0.28, 1.30) 0 High (>12 yr): -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 yr): 0.07% (-1.44, 1.58) 1 Medium (8-12 yr): 0.29% (-0.32, 0.90) 1 High (>12 yr): 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:

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			0.79% (0.26, 1.32) 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% (-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 μg/m^3 unless otherwise specified.

Table E-17. Short-term exposure-mortality - $PM_{10-2.5}$.

Study	Design & Methods	Concentrations1	Effect Estimates (95% CI)
Reference: Burnett et al. (2004, 086247) **Period of Study:** 1981-1999 **Location:** 12 Canadian cities	**Outcome:** Mortality: Nonaccidental (<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 µg/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:** Mar 2004-Dec 2005 **Location:** Shanghai, China	**Outcome (ICD10):** Mortality: Total (nonaccidental) (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 µg/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
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 yr	**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 µg/m^3 Warm Season: 5.7 µg/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:** Aug 1998-Jul 2000 **Location:** Fulton and DeKalb counties, Georgia (ARIES)	**Outcome:** Mortality: Nonaccidental (<800) Cardiovascular (390-459) Respiratory (460-519) Cancer (140-239) **Study Design:** Time-series **Statistical Analyses:** Poisson GLM, natural cubic splines **Age Groups:** <65 yr, ≥ 65 yr	**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	Concentrations1	Effect Estimates (95% CI)
Reference: Perez et al. (2008, 156020) **Period of Study:** Mar 2003-Dec 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, PM1	**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:** Mar 2003-Dec 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, PM1	**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:** Mar 2003-Dec 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, PM1	**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:** Jan 1995-Dec 1999 **Location:** Spokane, Washington	**Outcome:** Mortality: Nonaccidental (< 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 $PM_{10-2.5}$.
Reference: Stieb et al. (2002, 025205) **Period of Study:** Publication dates of studies: 1985-Dec 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 (nonaccidental) **Study Design:** Meta-analysis **Statistical Analyses:** Random effects model **Age Groups:** All ages	**Pollutant:** $PM_{10-2.5}$ **Averaging Time:** NR **Mean (SD):** NR **Range (Min, Max):** NR **Copollutant:** Varied between studies: $PM_{2.5}$, O_3, SO_2, NO_2, CO	**Increment:** 13.0 $\mu g/m^3$ **% Increase (Lower CI, Upper CI) lag:** Single-pollutant models: 10 studies $PM_{10-2.5}$: 1.2% (0.5, 1.9) Multipollutant models: 6 studies $PM_{10-2.5}$: 0.9% (-0.3, 2.0)

Study	Design & Methods	Concentrations1	Effect Estimates (95% CI)
Reference: Villeneuve et al. (2003, 055051) **Period of Study:** 1986-1999 **Location:** Vancouver, Canada	**Outcome:** Mortality: Nonaccidental (<800) Cardiovascular (401-440) Respiratory (460-519) Cancer (140-239) **Study Design:** Time-series **Statistical Analyses:** Poisson, natural splines **Age Groups:** ≥ 65	**Pollutant:** $PM_{10-2.5}$ **Averaging Time:** 24-h avg **Mean (SD):** Daily: 6.1 Every 6th Day 8.3 **Range (Min, Max):** Daily: (0.0, 72.0) Every 6th Day: (0.7, 35.0) **Copollutant:** $PM_{2.5}$ PM_{10} SO_2 CO NO_2 O_3	**Increment:** 11.0 µg/m^3 **% Increase (Lower CI, Upper CI) lag:** Nonaccidental 1.4% (-2.5, 5.4) 0-2 avg 1.0% (-1.9, 4.0) 0 -1.1% (-4.0, 1.8) 1 2.0% (-1.0, 5.1) 2 Cardiovascular 5.9% (-0.2, 12.4) 0-2 avg 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
Reference: Wilson et al. (2007, 157149) **Period of Study:** 1995-1997 **Location:** Phoenix, Arizona	**Outcome:** Mortality: Cardiovascular **Study Design:** Time-series **Statistical Analyses:** Poisson GAM, nonparametric smoothing spline **Age Groups:** >25	**Pollutant:** $PM_{10-2.5}$ **Averaging Time:** 24-h avg **Mean (SD):** NR **Range (Min, Max):** NR **Copollutant (correlation):** NR	**Increment:** 10 µg/m^3 **% Excess Risk (Lower CI, Upper CI) lag:** Central Phoenix: 2.4% (-1.2, 6.1) 0-5 ma Middle Phoenix: 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 µg/m^3 unless otherwise specified.

Table E-18. Short-term exposure-mortality - $PM_{2.5}$ (including PM components/sources).

Study	Design & Methods	Concentrations1	Effect Estimates (95% CI)
Reference: Basu et al. (2008, 098716) **Period of Study:** May 1999-Sept 2003 **Location:** 9 California counties	**Outcome (ICD10):** Mortality: Nonaccidental (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 (nonaccidental) 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 µg/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
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 (nonaccidental (<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 µg/m^3 **Range (Min, Max):** NR **Copollutant (correlation):** NR	**Increment:** 10 µg/m^3 **% Increase (Lower CI, Upper CI) lag:** All-cause (nonaccidental): 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

Study	Design & Methods	Concentrations1	Effect Estimates (95% CI)
			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 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: ll 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 μg/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 μg/m^3: All cause: 1.41% (-0.49, 3.30) 1 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

Study	Design & Methods	Concentrations1	Effect Estimates (95% CI)
			Respiratory: -2.08% (-4.47, 0.31) 1 Cardiovascular: -1.02% (-2.44, 0.41) 1 Stroke: 0.69% (-1.19, 2.57) 1
Reference: Franklin et al. (2008, 097426) **Period of Study:** 2000-2005 **Location:** 25 U.S. communities	**Outcome (ICD10):** Mortality: Nonaccidental (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: 9.6-34.4 Spring: 6.7-27.6 Summer: 7.6-26.0 Fall: 9.5-32.1 **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 µg/m^3 **% Increase (Lower CI, Upper CI) lag:** Nonaccidental: 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 µg/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% NH4: 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% As: 0.58%
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 µg/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% (2.8-19.8) 2 -1.1% (-7.5 to 5.2) 3

Study	Design & Methods	Concentrations1	Effect Estimates (95% CI)
Reference: Hopke et al. (2006, 088390) **Period of Study:** Washington, DC: Aug 1988-Dec 1997. Phoenix, Arizona: Mar 1995-Jun 1998 **Location:** Washington, DC and surrounding counties Phoenix, Arizona	**Outcome:** Mortality: Total (nonaccidental) 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.
Reference: Ito et al. (2006, 088391) **Period of Study:** Aug 1988-Dec 1997 **Location:** Washington, DC and surrounding counties	**Outcome:** Mortality: Total (nonaccidental) Cardiovascular Cardiovascular-Respiratory **Study Design:** Time-series Source-apportionment **Statistical Analyses:** Poisson GLM, natural splines **Age Groups:** All ages	**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):** 17.8 (8.7) **Range (Min, Max):** NR **Copollutant (correlation):** NR	**Increment:** $PM_{2.5}$ = 28.7 µg/m^3 $PM_{2.5}$ Sources 5-95th = Not reported **% Increase (Lower CI, Upper CI) lag:** Secondary sulfate (variance-weighted mean percent excess mortality) 6.7% (1.7, 11.7) 3 Primary coal-related $PM_{2.5}$ (mean percent excess mortality) 5.0% (1.0, 9.1) 3 Residual oil (mean percent excess mortality) 2.7% (-1.1, 6.5) 2 Traffic-related $PM_{2.5}$ (mean percent excess mortality) 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/yr: 7.9% (3.3, 12.6) 3 4 df/yr: 8.3% (3.7, 13.1) 3 8 df/yr: 8.3% (3.7, 13.2) 3 16 df/yr: 8.1% (3.1, 13.2) 3
Reference: Kan et al. (2007, 091267) **Period of Study:** Mar 2004-Dec 2005 **Location:** Shanghai, China	**Outcome (ICD10):** Mortality: Total (nonaccidental) (A00-R99) Cardiovascular (I00-I99) Respiratory (J00-J98) **Study Design:** Time-series **Statistical Analyses:** Poisson GAM, penalized splines **Age Groups:** All ages	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-h avg **Mean (SD):** 52.3 (1.57) **Range (Min, Max):** (2.0, 330.3) **Copollutant (correlation):** PM_{10}: r = 0.84 $PM_{10-2.5}$: r = 0.48 O_3: r = 0.31	**Increment:** 10 µg/m^3 **% Increase (Lower CI, Upper CI)** **lag:** Total: 0.36% (0.11, 0.61) 0-1 Cardiovascular: 0.41% (0.01, 0.82) 0-1 Respiratory: 0.95% (0.16, 1.73) 0-1

Study	Design & Methods	Concentrations1	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:** $PM_{2.5}$ **Averaging Time:** 24-h avg **Median (SD) unit:** Cold Season: 8.2 Warm Season: 7.8 **Range (Min, Max):** Cold Season: (1.1, 69.5) Warm Season: (1.1, 41.5) **Copollutant:** O_3 CO NO_2 PM_{10} $PM_{10-2.5}$ UFP	**Increment:** Cold Season: 6.7 µg/m^3 Warm Season: 5.7 µg/m^3 **% Increase (Lower CI, Upper CI) lag:** Cold Season -0.19% (-3.77, 3.51) 0 -0.17% (-3.73, 3.52) 1 0.59% (-2.95, 4.26) 2 0.46% (-3.10, 4.15) 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) **Period of Study:** Aug 1998-Jul 2000 **Location:** Fulton and DeKalb counties, Georgia (ARIES)	**Outcome:** Mortality: Nonaccidental (<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:** $PM_{2.5}$ **Averaging Time:** 24-h avg **Mean (SD):** 19.62 (8.32) **Range (Min, Max):** (5.29, 48.01) **Copollutant:** $PM_{10-2.5}$ 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: $PM_{2.5}$: 0.00398 (0.00161) 0-1 Monthly Knots: $PM_{2.5}$: 0.00544 (0.00184) 0-1 Biweekly Knots: $PM_{2.5}$: 0.00369 (0.00201) 0-1
Reference: Lippmann et al. (2006, 091165) **Period of Study:** 2000-2003 **Location:** 60 U.S. cities (NMMAPS)	**Outcome:** Mortality: Nonaccidental (<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: Nonaccidental (<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

Study	Design & Methods	Concentrations1	Effect Estimates (95% CI)
Reference: Ostro et al. (2006, 087991) **Period of Study:** Jan 1999-Dec 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 yr	**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 CO r = 0.60 O_3 (1h) r = -0.14 O_3 (8h) r = -0.22	**Increment:** 10 µg/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) 0-1 Ischemic heart disease: 0.3% (-0.5, 1.0) 0-1 Males: 0.5% (-0.2, 1.2) 0-1 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 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

Study	Design & Methods	Concentrations1	Effect Estimates (95% CI)
Reference: Ostro et al. (2007, 091354) **Period of Study:** $PM_{2.5}$ speciation analysis: Jan 2000-Dec 2003. $PM_{2.5}$ analysis: Jan 1999-Dec 2003 **Location:** 6 California counties (2000-2003). 9 California counties (1999-2003) (CALFINE)	**Outcome (ICD10):** Mortality: Total (nonaccidental) mortality Respiratory (J00-J98) Cardiovascular (I00-I99) **Study Design:** Time-series **Statistical Analyses:** Poisson, natural splines **Age Groups:** >65	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-h avg **Mean (SD):** 2000-2003: 19.28 1999-2003: 18.6 **Range (Min, Max): NR** **Copollutant (correlation):** 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	**Increment:** 14.6 µg/m^3 **% Increase (Lower CI, Upper CI)** **lag:** Cardiovascular 1.6% (0.0, 3.1) 3 **Notes:** The study does not present all estimates quantitatively.
Reference: Ostro et al. (2008, 097971) **Period of Study:** Jan 2000-Dec 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:** Mar 2003-Dec 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) µg/m^3 **Range (Min, Max):** 0.6, 45.5 **Copollutant:** $PM_{10-2.5}$, PM_1	**Increment:** 10 µg/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	Concentrations1	Effect Estimates (95% CI)
Reference: Perez et al. (2008, 156020) **Period of Study:** Mar 2003-Dec 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) µg/m^3 **Range (Min, Max):** 0.6, 45.5 **Copollutant:** $PM_{10-2.5}$, PM_1	**Increment:** 10 µg/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:** Mar 2003-Dec 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) µg/m^3 **Range (Min, Max):** 0.6, 45.5 **Copollutant:** $PM_{10-2.5}$, PM_1	**Increment:** 10 µg/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 (nonaccidental) (<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 yr: 17.0 (8.7) Winters (Dec-Feb): 17.2 (6.8) Summers (Jun-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 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

Study	Design & Methods	Concentrations1	Effect Estimates (95% CI)
			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 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	Concentrations1	Effect Estimates (95% CI)
Reference: Rosenthal et al. (2008, 156925) **Period of Study:** Jul 2002-Jul 2006 **Location:** Indianapolis, Indiana	**Outcome:** Non-Dead on Arrival (DOA) Out-of-Hospital Cardiac Arrests (OHCA) Witnessed non-DOA OHCA **Study Design:** Case-crossover **Statistical Analyses:** Time-stratified conditional logistic regression **Age Groups:** All ages Study Population: Non-DOA OHCA: 1,374 Witnessed non-DOA OHCA: 511	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-h avg Hourly **Mean (SD):** NR IQR (25th, 75th): All non-DOA All heart rhythms: (9.4, 19.5) OHCA: (9.6, 19.5) Referents: (9.3, 19.5) Asystole: (9.2, 19.4) OHCA: (9.2, 19.7) Asystole: (9.2, 19.2) Witnessed non-DOA hourly All heart rhythms: (8.8, 20.7) OHCA: (8.8, 21.9) Referents: (8.8, 20.4) Asystole: (8.5, 19.8) OHCA: (9.4, 21.3) Referents: (8.3, 19.1) **Copollutant (correlation):** NR	**Increment:** 10 µg/m^3 **Hazard Ratio (Lower CI, Upper CI) lag:** Out-of-Hospital non-DOA Cardiac Arrests All 1.02 (0.94, 1.11) 0 1.00 (0.92, 1.08) 1 0.98 (0.90, 1.06) 2 1.00 (0.92, 1.08) 3 1.02 (0.92, 1.12) 0-1 avg 1.01 (0.91, 1.12) 0-2 avg 1.02 (0.91, 1.14) 0-3 avg Asystole 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) 0 0.98 (0.83, 1.15) 1 0.96 (0.82, 1.14) 2 0.95 (0.82, 1.10) 3 0.96 (0.80, 1.17) 0-1 avg 0.98 (0.80, 1.21) 0-2 avg 0.98 (0.78, 1.21) 0-3 avg Witnessed Out-of-Hospital non-DOA Cardiac Arrests (lag represents h in which or h before OHCA occurred) All: 1.12 (1.01, 1.25) 0 White: 1.18 (1.03, 1.35) 0 60-75: 1.25 (1.05, 1.49) 0 Asystole: 1.22 (1.01, 1.59) 0
Reference: Schwartz et al. (2002, 025312) **Period of Study:** 1979-Late 1980's **Location:** 6 U.S. cities	**Outcome:** Mortality: Total (nonaccidental) (<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.

Study	Design & Methods	Concentrations1	Effect Estimates (95% CI)
Reference: Simpson et al. (2005, 087438) **Period of Study:** Jan 1996-Dec 1999 **Location:** 4 Australian cities	**Outcome:** Mortality: Nonaccidental (<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 µg/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 µg/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:** Jan 1995-Dec 1999 **Location:** Spokane, Washington	**Outcome:** Mortality: Nonaccidental (<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 $PM_{10-2.5}$: r = 0.31 CO: r = 0.62	**Increment:** $PM_{2.5}$: 10 µg/m^3 PM_{10}: 25 µg/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
Reference: Stieb et al. (2002, 025205) **Period of Study:** Publication dates of studies: 1985-Dec 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 (nonaccidental) **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 µg/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 µg/m^3 $PM_{2.5}$: 13.8 µg/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 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 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

Study	Design & Methods	Concentrations1	Effect Estimates (95% CI)
			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 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 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) 0 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) 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

Study	Design & Methods	Concentrations1	Effect Estimates (95% CI)
			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) 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: Aug 1988-Dec 1997. Phoenix, Arizona: 1995-1997 **Location:** Washington, DC and surrounding counties Phoenix, Arizona	**Outcome:** Mortality: Total (nonaccidental) (<800) Cardiovascular (390-448) **Study Design:** Time-series Source-apportionment **Statistical Analyses:** Poisson GLM, natural splines **Age Groups:** Washington, DC: All ages Phoenix, Arizona: ≥ 65	**Pollutant:** $PM_{2.5}$, and source apportioned $PM_{2.5}$: Crustal Traffic Secondary SO_4 Secondary NO_3 Wood Oil Salt 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)	**Increment:** 10 µg/m^3 **% Increase:** Total (nonaccidental): Secondary sulfate: Phoenix: 5.2% Washington, DC: 3.8% Motor vehicles: Phoenix: 0.9% Washington, DC: 4.2%

Study	Design & Methods	Concentrations1	Effect Estimates (95% CI)
Reference: Villeneuve et al. (2003, 055051) **Period of Study:** 1986-1999 **Location:** Vancouver, Canada	**Outcome:** Mortality: Nonaccidental (<800) Cardiovascular (401-440) Respiratory (460-519) Cancer (140-239) **Study Design:** Time-series **Statistical Analyses:** Poisson, natural splines **Age Groups:** ≥ 65	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-h avg **Mean (SD):** Daily $PM_{2.5}$: 7.9 Every 6th Day $PM_{2.5}$: 11.6 **Range (Min, Max):** Daily $PM_{2.5}$: (2.0, 32.0) Every 6th Day $PM_{2.5}$: (1.8, 43.0) **Copollutant:** SO_2 CO NO_2 O_3	**Increment:** $PM_{2.5}$ (Daily): 9.0 µg/m^3 $PM_{2.5}$ (6th Day): 15.7 µg/m^3 **% Increase (Lower CI, Upper CI) lag:** Nonaccidental $PM_{2.5}$ (Daily) -0.1% (-5.1, 5.2) 0-2 avg -0.1% (-4.1, 4.1) 0 -0.3% (-4.2, 3.7) 1 0.5% (-3.3, 4.4) 2 $PM_{2.5}$ (6th Day) -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) 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
Reference: Wilson et al. (2007, 157149) **Period of Study:** 1995-1997 **Location:** Phoenix, Arizona	**Outcome:** Cardiovascular **Study Design:** Time-series **Statistical Analyses:** Poisson GAM, nonparametric smoothing spline **Age Groups:** >25	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-h avg **Mean (SD):** NR **Range (Min, Max):** NR **Copollutant (correlation):** NR	**Increment:** 10 µg/m^3 **% Excess Risk (Lower CI, Upper CI) lag:** Central Phoenix: 11.5% (2.8, 20.9) 0-5 ma 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 µg/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) **Period of Study:** Mar 2003-Dec 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_1 **Averaging Time:** 24 h **Mean (SD) Unit:** 20.0 (10.3) µg/m^3 **Range (Min, Max):** 1.9, 80.1 **Copollutant:** $PM_{10-2.5}$, $PM_{2.5-1}$	**Increment:** 10 µg/m^3 **Odds Ratio (95%CI) lag** Avg L0-1: 1.005 (0.960-1.053), p = 0.824 L1: 1.012 (0.969-1.056), p = 0.599 L2: 1.042 (0.998-1.087), p = 0.063 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) **Period of Study:** Mar 2003-Dec 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_1 **Averaging Time:** 24 h **Mean (SD) Unit:** 20.0 (10.3) µg/m^3 **Range (Min, Max):** 1.9, 80.1 **Copollutant:** $PM_{10-2.5}$, $PM_{2.5-1}$	**Increment:** 10 µg/m^3 **Odds Ratio (95%CI) lag** Avg L0-1: 1.028 (1.000-1.057), p = 0.054 L1: 1.029 (1.003-1.056), p = 0.030 L2: 1.023 (0.996-1.050), p = 0.091 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
Reference: Perez et al. (2008, 156020) **Period of Study:** Mar 2003-Dec 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) µg/m^3 **Range (Min, Max):** 1.9, 80.1 **Copollutant:** $PM_{10-2.5}$, $PM_{2.5-1}$	**Increment:** 10 µg/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:** Jan 995-Dec 1999 **Location:** Spokane, Washington	**Outcome:** Mortality: Nonaccidental (<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.
Reference: Stölzel et al. (2007, 091374) **Period of Study:** Sept 1995-Aug 2001 **Location:** Erfurt, Germany	**Outcome:** Mortality: Total (nonaccidental) (<800) Cardio-respiratory (390-459, 460-519, 785, 786) **Study Design:** Time-series **Statistical Analyses:** Poisson GAM **Age Groups:** All ages	**Pollutant:** $MC_{0.1-0.5}$, $MC_{0.01-2.5}$ **Averaging Time:** 24-h avg **Mean (SD):** $MC_{0.1-0.5}$: 17.6 (14.8) $MC_{0.01-2.5}$: 22.3 (19.2) IQR (25th, 75th): $MC_{0.1-0.5}$: (8.4, 21.5) $MC_{0.01-2.5}$: (10.5, 27.3) **Copollutant (correlation):** $MC_{0.1-0.5}$ NO: r = 0.52 NO_2: r = 0.60 CO: r = 0.58 $MC_{0.01-2.5}$ NO: r = 0.51 NO_2: r = 0.58 CO: r = 0.57	**Increment:** $MC_{0.1-0.5}$: 13.1 µg/m^3 $MC_{0.01-2.5}$: 16.8 µg/m^3 **Relative Risk (Lower CI, Upper CI) lag:** Total (nonaccidental) $MC_{0.1-0.5}$ 1.010 (0.986 1.034) 0 1.006 (0.983 1.029) 1 1.007 (0.985 1.029) 2 0.994 (0.973 1.016) 3

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			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 MC0.1-0.5 1.004 (0.977 1.031) 0 1.004 (0.979 1.029) 1 1.001 (0.978 1.026) 2 0.991 (0.967 1.014) 3 1.000 (0.977 1.023) 4 1.000 (0.976 1.023) 5 MC0.01-2.5 1.001 (0.977 1.026) 0 0.999 (0.976 1.022) 1 0.998 (0.976 1.021) 2 0.985 (0.964 1.007) 3 1.001 (0.980 1.022) 4 1.003 (0.981 1.024) 5

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:** PM7 **Averaging Time:** 1-h avg **Mean (SD):** Warmer Months (Apr-Sep): 40.3 Colder Months (Oct-Mar): 39.4 **Range (Min, Max):** NR **Copollutant (correlation):** Warmer Months NO_2: r = 0.46-0.63 Ox: r = -0.14 to 0.20 Colder Months NO_2: 0.42-0.79 Ox: r = -0.36 to -0.14	**Increment:** 30 µg/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 µg/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 µg/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 µg/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 µg/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 µg/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 yr (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 yr (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 yr) **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: 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:**

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			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 yr: 1.47 (1.01, 2.14), p = 0.04 **Risk of DVT associated with PM_{10} (yr 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 yr: 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 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:** Jan 1995-Sept 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

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
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 yr 7.9 ± 1.3 yr **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 mi 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 Jun 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 yr (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-yr 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.

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
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 yr **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: 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)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
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 yr **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 yr **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:** Study used an air pollution model incorporating points, lines, and grids as 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 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: 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) vs. normal levels **Age Groups:** 44-84 yr **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-yr 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} **Averaging Time:** Avg of previous month, avg of previous 2 mo (recent exposures) 20-yr directly monitored PM_{10} avg, 20-yr imputed PM_{10} avg (longer-term exposures) **Mean (SD):** Previous 20 yr: 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-yr imputed PM_{10} was derived using a space-time model) **Copollutant (correlation):** $PM_{2.5}$	**PM Increment:** 10 µg/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 yr or shorter-term PM exposure were not significant ($p < 0.01$) by gender, age, city, race/ethnicity or study site.
Reference: Puett et al, (2008, 156891) **Period of Study:** 1992-2002 **Location:** Northeastern metropolitan U.S.	**Outcome:** Nonfatal myocardial infarction **Study Design:** Cohort **Covariates:** Age in months, state of residence, yr 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-mo ma **Mean (SD) Unit:** NR **Range (Min, Max):** NR **Copollutant (correlation):** NR	**Increment:** 10 µg/m^3 **Hazard Ratio, 95% CI, 12 month ma** 0.94 (0.77-1.15)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
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 yr **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) ($\mu g/m^3$) **Averaging Time:** 30 yr (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 $\mu g/m^3$ (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}.

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Zanobetti & Schwartz (2007, 091247) **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 Columbus, Ohio Denver, 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 and Youngstown, Ohio)	**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 yr **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) **Season:** Assessed as a confounder **Dose-response Investigated?** No **Statistical Package:** NR	**Pollutant:** PM_{10} **Averaging Time:** Yearly avg of pollution for that yr and lags up to the 3 previous yr (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 U.S. 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 yr of failure and for the distributed lag from the yr of failure up to 3 previous yr **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 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 yr 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 (yr 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 Fig 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 µg/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, 156209) **Period of Study:** Oct 2000-Sep 2002 (exposure averaging period) outcome assessed in 2002 **Location:** 5 U.S. 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)	**Outcome (ICD9 and ICD10):** Abdominal aortic calcium (AAC), a marker of systemic atherosclerosis (quantitative measure of interest was the Agatston score) **Age Groups:** 46-88 yr **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-yr 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	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 2-yr 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	**PM Increment:** 10 $\mu g/m^3$ **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) ≥ 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

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			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)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Auchincloss et al. (2008, 156234) **Period of Study:** Jul 2000-Aug 2002 **Location:** 6 U.S. 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 (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 yr **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 **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	**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 (correlation):** SO_2 NO_2 CO Traffic-related exposures (straight-line distance to a highway; total road length around a residence)	**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 $PM_{2.5}$ (avg 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, 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. Rresults 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

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Calderón-Garcidueñas et al. (2009, 192107) **Period of Study:** Sept 2004-Jan 2005 **Location:** Mexico City and Polotitlan, Mexico	**Outcome:** Flow cytometry **Study Design:** Panel **Covariates:** NR **Statistical Analysis:** Pearson's Correlation **Statistical Package:** Stata **Age Groups:** 9.7 ± 1.2 yr	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 1-, 2- and 7-day avg **Mean (SD) Unit:** 35.89 ± 0.93 $\mu g/m^3$ **Range (Min, Max):** NR **Copollutant:** PM_{10}, O_3	**Increment:** NR **Flow cytometry results and their statistical significance in control vs. exposed children** 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 Jun 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 yr **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:** $PM_{2.5}$ **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 U.S. 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) 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) 1.02 **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 yr **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:** $PM_{2.5}$ **Averaging Time:** 1 yr (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 10th-90th 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 avg of $PM_{2.5}$ and measured $PM_{2.5}$: 0.86-0.88, depending on season.	**PM Increment:** 3.91 µg/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 yr: 1.18 (0.83, 1.68) Age >60 yr: 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 (Fig 2) Participants who had not been working full-time during the last 5 yr showed stronger effects, with possible dose-response between $PM_{2.5}$ and CAC (results presented in Fig 3)
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 yr **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:** $PM_{2.5}$ (µg/m^3) **Averaging Time:** Yearly mean estimated with model for yr 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

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
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 yr	**Pollutant:** $PM_{2.5}$ **Averaging Time:** Daily **Mean (SD) Unit:** 22.96 (0.85) **Range (Min, Max):** NR **Copollutant (correlation):** NR	**Increment:** 3.91 µg/m^3 **Odds Ratio (95%CI) for prevalence of peripheral arterial disease** 0.87 (0.57-1.34)
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 yr 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:** $PM_{2.5}$ (µg/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 µg/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 yr:** 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 µg/ 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 µg/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 U.S. 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 yr (median age at	**Pollutant:** $PM_{2.5}$ (µg/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)	**PM Increment: 10** µg/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)
	enrollment: 63) **Study Design:** Cohort (median follow-up of 6 yr) **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 yr 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	Median: 13.4 **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 10th-90th 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 ≥ $50,000: H: 1.20 (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)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			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) 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)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			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) 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 (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)
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) vs. normal levels **Age Groups:** 44-84 yr **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 mo (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-yr $PM_{2.5}$ 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)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Solomon et al. (2003, 156994) **Period of Study:** Exposures measures 1966-1969 Health endpoints assessed via questionnaire, yr not reported but apparently 30 yr 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 yr 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 yr 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 yr **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 µg/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
Reference: Avol et al. (2001, 020552) **Period of Study:** 1993-1998 **Location:** Southern California	**Outcome:** FVC, FEV_1, MMEF, PEFR **Age Groups:** 10 yr **Study Design:** cohort **N:** 110 **Statistical Analyses:** Linear regression **Covariates:** Sex, race, cohort entry yr, annual avg change in height, weight, BMI **Dose-response Investigated?** No	**Pollutant:** PM_{10} **Averaging Time:** 24-h PM_{10} avgd over 1994 **Mean (SD):** 15.0-66.2	**PM Increment:** 10 µg/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)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
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 yr **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-mo avg **Mean (SD):** NR **Range (Min, Max):** NR **Monitoring Stations**: 9 **Copollutant (correlation):** NR	**PM Increment:** 10 µg/m^3 "Fig 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." "Fig 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 wk in Jul and Jan 1997 and 2 wk in Nov 1996 and Apr 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 PM10 in congested streets: 22.7** **Percent change PM10 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 yr **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 yr **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 FEF_{25-75}, FEF_{75}, 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.
Reference: Cavanagh et al. (2007, 189802) **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):** Fall 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 U.S. 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.

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
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 yr **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-yr), 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 $\mu g/m^3$/yr **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-yr 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-yr (equivalent to a reduction of 10 $\mu g/m^3$ in the annual avg during the mean follow-up time of 10.9 yr) 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 FEF25-75 (i.e., by 14.0 mL per second 95% CI, 3.1 to 24.8)
Reference: Gauderman et al. (2000, 012531) **Period of Study:** 1993-1997 **Location:** Southern California	**Outcome:** FVC, FEV_1, MMEF, FEF75 **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) FEF_{75} -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 yr) **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 yr, 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 avg **Mean (SD):** The avg levels were presented in an online data supplement (Fig 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 yr **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 yr 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 µg/m^3 EC: 1.2 µg/m^3 OC: 10.5 µg/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/FEF25.75 **Age Groups:** Children (mean age 10 at recruitment, followed for 8 yr) **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 yr, 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 µg/m^3 Pollutant effect reported as difference in 8 yr 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 avg **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 $\mu g/m^3$ **Odds Ratio Estimate [Lower CI, Upper CI]:** Odds of having 2 or more pulmonary exacerbations as compared to 1 or less in 2000 1.08 (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 (Apr-Nov) 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 $\mu g/m^3$ **Range (Min, Max):** 6.93, 31.12 **Copollutant (correlation):** NR	**Increment:** 10 $\mu g/m^3$ **Percent Increase (95% CI)** *Full results reported visually in Fig 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)
Reference: Ho et al. (2007, 093265) **Period of Study:** Oct 1995-Mar 1996 **Location:** Taiwan, Republic of China	**Outcome:** Asthma **Age Groups:** 10-17 yr **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}.

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Hong et al. (2004, 156565) **Period of Study:** 2001 **Location:** Kerinci, SP7, and Pelalawan, Indonesia	**Outcome:** Respiratory symptoms **Age Groups:** <12 yr **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 wk (cough, cold, phlegm) and the last 12 mo. **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 wk before he field survey to 1 mo after the survey **Mean (SD):** Kerinci 102.9 (49.6) µg/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 mo 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)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
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 **Covariates:** Gender, atopy, ETS exposure, baseline lung function, first height, height difference, school site **Season:** Winter, summer **Dose-response Investigated?** No	**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) 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)	**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) 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 MEF_{25-75} (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: Summer: -0.095 (0.000) Winter: -0.011 (0.474)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Hwang et al. (2006, 088971) **Period of Study:** 2001 **Location:** Taiwan	**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 yr (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):** 55.58 (16.57) Percentiles: 25th: 42.96 50th(Median): 53.81 75th: 70.37 **Range (Min, Max):** 29.36, 99.58 **Monitoring Stations**: 22 **Copollutant (correlation):** NO_X (r = 0.34) SO_2 (r = 0.58) CO (r = 0.27) O_3 (r = 0.28)	**PM Increment:** 10 µg/m^3 RR Estimate [Lower CI, Upper CI] Single pollutant model: 1.00 [0.99, 1.02] Controlling for NO_X: 0.99 [0.97, 1.00] Controlling for CO: 1.00 [0.99, 1.01] Controlling for O_3: 1.00 [0.99, 1.02]
Reference: Hwang et al, (2008, 134420) **Period of Study:** 2001-2003 **Location:** Taiwan	**Outcome:** Oral Cleft **Study Design:** Case-control **Covariates:** Maternal age, plurality, gestational age, population density and season of conception **Statistical Analysis:** Logistic regression **Age Groups:** Infants	**Pollutant:** PM_{10} **Averaging Time:** hourly **Mean (SD) Unit:** Avg: 54.83 ± 13.07 µg/m^3 Spring: 64.44 ± 16.21 µg/m^3 Summer: 39.11 ± 8.31 µg/m^3 Fall: 47.76 ± 11.77 µg/m^3 Winter: 68.00 ± 21.88 µg/m^3 **Range (Min, Max):** Avg: 20.75-78.05 µg/m^3 Spring: 23.33-94.33 µg/m^3 Summer: 17.33-60.00 µg/m^3 Fall: 21.00-72.00 µg/m^3 Winter: 21.33-116.00 µg/m^3 **Copollutant (correlation):** CO: -0.19 NO_X: 0.56 O_3: 0.39 SO_2: 0.50	**Increment:** 10 µg/m^3 **Odds Ratio (Min CI, Max CI);** Single Pollutant Model Month 1: 1.01 (0.96-1.06) Month 2: 1.00 (0.95-1.05) Month 3: 0.99 (0.95-1.05) Two Pollutant Model (O_3 + PM_{10}) Month 1: 0.99 (0.94-1.04) Month 2: 0.99 (0.94-1.04) Month 3: 0.98 (0.93-1.04) Two Pollutant Model (CO + PM_{10}) Month 1: 1.01 (0.96-1.06) Month 2: 1.00 (0.95-1.05) Month 3: 0.99 (0.95-1.05) Two Pollutant Model (NO_X + PM_{10}) Month 1: 1.02 (0.97-1.08) Month 2: 1.01 (0.95-1.07) Month 3: 1.01 (0.95-1.07) 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-Apr 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 yr (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:** Apr 1997-Jul 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 1 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 yr 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 wk preceding measurement, season **Dose-response Investigated?** No **Statistical Package**: MLwiN	**Pollutant:** $PM_{2.5}$ **Averaging Time:** Annual **Mean (SD):** 20.5 µg/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 µg/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:** Fig 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 yr) **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 Mar 64 Dec 54 Ganghwa Mar 64 Dec 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 Dec than in Mar for both male and female students at all academic yr…Because only the level of PM_{10} was significantly higher for Mar than for Dec in both areas, the authors suggest that decrements of pulmonary function in Mar for both areas are associated with the increased level of PM_{10}"
Reference: Kim et al. (2004, 087383) **Period of Study:** Mar-Jun (spring) 2001 Sep-Nov (fall) 2001 **Location:** Alameda County, CA	**Outcome:** Asthma, bronchitis **Age Groups:** Children (in 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_{10} **Averaging Time:** 9 wk **Mean (SD):** Study Avg 30 **Monitoring Stations**: 10 **Copollutant (correlation):** r2 is approximately 0.9 for all copollutants – BC, $PM_{2.5}$, NO_X, NO_2, NO (NO_X-NO_2)	**PM Increment:** 1.4 (IQR) **OR Estimate [Lower CI, Upper CI]:** Bronchitis All subjects: 1.03 [0.99, 1.07] LTR subjects: 1.02 [0.98, 1.07] LTR females: 1.04 [1.01, 1.09] LTR males: 1.01 [0.95, 1.06] Asthma All subjects: 1.02 [0.96, 1.09] 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]

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Kumar et al. (2004, 089873) **Period of Study:** 1999-2001 **Location:** Mandi Gobindgarh and Morinda, Punjab State, northern India	**Outcome:** Chronic respiratory symptoms & Spirometric ventilatory defect **Age Groups:** >15 yr **Study Design:** Cross-sectional **N:** 3603 individuals **Statistical Analyses:** Logistic regression **Covariates:** Age, gender, migration, SES, smoking, type of cooking fuel use **Dose-response Investigated?** No	**Pollutant:** PM_{10} **Mean (SD):** Study town 112.8 (17.9) Reference town 75.8 (2.9)	**PM_{10} Increment:** Low vs. High OR (Lower CI, Upper CI) p-value Chronic respiratory symptoms Low 1.00 (ref) High 1.5 (1.2, 1.8) <0.001 Spirometric ventilatory defect Low 1.00 (ref) High 2.4 (2.0-2.9) <0.001
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:** R2 = 0.7642, P = 0.13
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 FEF50: 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

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
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 µg/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)
Reference: McConnell et al. (2003, 049490) **Period of Study:** 1993-1999 **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-yr avg **Mean (SD):** .30.8(13.4) µg/m^3 **Range (Min, Max):** 15.7-63.5 PM Component: particulate OC and EC **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 EC: r = 0.71 OC: r = 0.70 NO_2: r = 0.20 O_3: r = 0.64	**PM Increment:** Between community range 47.8 µg/m^3 Between community unit 1 µg/m^3 Within community 1 µg/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 yr, 12-13 yr, 15-16 yr **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 yr **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 (yrly). **Age Groups:** 10-15-yr-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 yr (n = 48 = 12 communities · 4 yr) 25th: NR 50th(Median): 3.4 75th: NR **Range (Min, Max):** Community by yr (n = 48 = 12 communities · 4 yr): (0.89, 8.7) **Monitoring Stations**: 12 **Copollutant:** O_3, NO_2, EC, OC, Acid vapor (acetic and formic acid)	**PM Increment:** 6.1 µg/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:** Nov 2000 and Sep 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 h over 1 yr **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 µg/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-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 yr 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 µg/m^3 Odds Ratio [lower CI, Upper CI] Annual PM_{10}: 0.93 [0.67, 1.27] Mar-Aug 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:** Jun 1999-Jun 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 yr **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 µg/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
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 yr **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) FEF25% -77.4 (-133.4 to -21.4) FEF50% -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) FEF25% -61.5 (-110.0 to -13.1) FEF50% -45.6 (-87.7 to -3.5) FEV_1 -7.3 (-22.4, 7.7) FVC -2.1 (-18.6, 14.4)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Parker et al. (2009, 192359) **Period of Study:** 1999-2005 **Location:** U.S.	**Outcome:** Respiratory allergy/hayfever **Study Design:** Cohort **Covariates:** Survey yr, 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 yr	**Pollutant:** PM_{10} **Averaging Time:** NR **Median:** 24.1 µg/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 µg/m^3 **Odds Ratio (95% CI)** Single Pollutant Model, variable N Adjusted: 1.04 (0.99-1.09)
Reference: Penard-Morand et al. (2005, 087951) **Period of Study:** Mar 1999-Oct 2000 Mean concentrations of NO_2, SO_2, PM_{10}, and O_3 were taken from Jan 1998-Dec 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 yr **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 yr **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 µg/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 yr): 1.05 (0.72-1.54) Asthma (past yr): 1.23 (0.77-1.95) Rhinoconjunctivitis (past yr): 1.17 (0.86-1.59) Atopic dermatitis (past yr): 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 yr): 1.31 (0.71-2.36) Asthma (past yr): 1.25 (0.66-2.37) Rhinoconjunctivitis (past yr): 1.22 (0.98-1.68) Atopic dermatitis (past yr): 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) Molds: 1.62 (0.64-4.09)
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 µg/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 yr (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 yr **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 yr 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 µg/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 yr) **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 µg/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
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 yr **Study Design:** Cohort **N:** 4064 students **Statistical Analyses:** Cchi-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

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Rojas-Martinez et al. (2007, 091064) **Period of Study:** 1996-1999 **Location:** Mexico City, Mexico	**Outcome:** Lung function: FEV_1, FVC, FEF25-75% **Age Groups:** Children 8 yr 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 **Statistical Package:** SA	**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] FEF25-75%: -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] FEF25-75%: -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] FEF25-75%: -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] FEF25-75%: -7 [-27: 12] FEV_1/FVC: 0.08 [0.03: 0.13] Boys One-pollutant model FVC: -33 [-41: -25] FEV: -27 [-34: -19] FEF25-75%: -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] FEF25-75%: -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] FEF25-75%: -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] FEF25-75%: -12 [-30: 6] FEV_1/FVC: -0.002 [-0.06: 0.05]
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 5-yr 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 yr 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,	**Outcome:** Respiratory Symptoms	**Pollutant:** PM_{10}	**Increment:** 10 $\mu g/m^3$

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
191950) **Period of Study:** 1991-2002 **Location:** Switzerland	**Study Design:** Prospective Cohort **Statistical Analysis:** Logistic Regression Model **Age Groups:** Adults, 18-60 yr 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 yr, daily number of cigarettes, yr since smoking cessation, passive smoking in general/at work, occupational exposure to airbourne irritants	**Averaging Time:** Annual **Mean (SD) Unit:** **Range (Min, Max):** **Copollutant (correlation):** NR	**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 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 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)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			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) 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:** Nov 2002-Apr 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 yr **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:** 1-day lag & 5-day ma	**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):** ΔPEF = mean daily deviations in PEF PM_{10}-ΔPEF: (-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 μg/m^3 ΔPEF (difference or change in peak expiratory flow) -0.0318 L/min

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Tager et al. (2005, 087538) **Period of Study:** Apr 2000-Jun 2000, Feb 2001-Jun 2001, Feb 2002-Jun 2002 **Location:** Los Angeles, California San Francisco, California	**Outcome:** Lung Function (FEV_1, FVC, PEFR, FEF75, FEF25-75, FEF25-75/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} FEF25-75/FVC) LnFEF75: M: -0.009 (0.0009), 0.009 (0.007) W: -0.010 (0.0007), 0.008 (0.0005)
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)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Wheeler and Ben-Schlomo (2005, 188766) **Period of Study:** 1995-1997 **Location:** England	**Outcome:** FEV_1 **Age Groups:** 16-79 yr **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-h, 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 FEV1 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
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 yr with or apart from colds) Persistent phlegm (brought up phlegm or mucus from the chest for at least 1 month per yr 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, yr of questionnaire administration, season of questionnaire administration, parental asthma prevalence	**Pollutant:** PM_{10} **Averaging Time:** 2 yr **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 yr (1993-1996) Schoolyards of participating children over a period of 2 yr (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 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: 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<10-3.3 **Averaging Time:** 8 h **Mean (SD):** North Kolkata: 269.8 Central Kolkata: 679.2 South Kolkata: 460.1 **Monitoring Stations**: 1 **Copollutant (correlation):** PM_{10} PM<3.3-0.	**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 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: 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

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, FEF75 **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-wk 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 µg/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) FEF75 -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) FEF75 -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) FEF75 -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 yr) **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 yr, 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 avg **Mean (SD):** The avg levels were presented in an online data supplement (Fig 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 µg/m^3 Association Estimate: $PM_{10-2.5}$ was not correlated with any of the pulmonary function tests that were analyzed
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

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: McConnell et al. (2003, 049490) **Period of Study:** 1993-1999 **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-yr 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 µg/m^3 Between community unit 1 µg/m^3 Within community 1 µg/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)
Reference: Millstein et al. (2004, 088629) **Period of Study:** Mar-Aug, 1995, and Sep 1995-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 yr 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 µg/m^3 **Odds Ratio [lower CI, Upper CI]** Annual $PM_{10-2.5}$: 0.96 [0.74, 1.25] Mar-Aug $PM_{10-2.5}$: 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:** U.S.	**Outcome:** Respiratory allergy/hayfever **Study Design:** Cohort **Covariates:** Survey yr, 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 yr	**Pollutant:** $PM_{10-2.5}$ **Averaging Time:** NR **Median:** 11.2 µg/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 µg/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 yr with or apart from colds) Persistent phlegm (brought up phlegm or mucus from the chest for at least 1 month per yr 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, yr of questionnaire administration, season of questionnaire administration, parental asthma prevalence	**Pollutant:** $PM_{10-2.5}$ **Averaging Time:** 2 yr **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 yr (1993-1996) Schoolyards of participating children over a period of 2 yr (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) **Period of Study:** Mar 1999-Oct 2000 **Location:** France (Bordeaux, Clermont-Ferrand, Creteil, Marseille, Strasbourg,, & Reims)	**Outcome:** EIB, Flexural atopic dermatitis, asthma, rhiniconjuctivitis, allergic rhinitis **Age Groups:** Children mean 10.4 ± 0.7 yr **Study Design:** Semi-individual design **N:** 5338 **Statistical Analyses:** Logistic regression **Covariates:** Age, sex, family history of allergy, passive smoking **Season:** NR **Dose-response Investigated?** No **Statistical Package:** SAS	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 5-day mean (Mon.-Fri.) over a 13-wk to 24-wk span Residential Proximity Level **Mean (SD):** Low conc: 8.7 High conc: 20.7 **Range (Min, Max):** Low conc: (1.6, 12.2) High conc: (12.5, 54.0) City Level **Mean (SD):** Low conc: 9.6 High conc: 23.0 **Range (Min, Max):** Low conc: (4.7, 12.7) High conc: (13.0, 54.5)	**PM Increment:** High vs. Low **Allergic and respiratory morbidity OR Estimate (Lower CI, Upper CI)** Proximity Level EIB (C) 1.35 (1.10, 1.67) Fl. Atopic dermatitis (C) 2.51 (2.06, 3.06) Asthma (P) 1.11 (0.88, 1.39) Atopic asthma (P) 1.43 (1.07, 1.91) Non-atopic asthma (P) 0.73 (0.49, 1.07) Rhiniconjunctivitis (P) 0.94 (0.77, 1.15) Atopic dermatitis (P) 1.05 (0.88, 1.27) Asthma (L) 1.00 (0.82, 1.22) Allergic Rhinitis (L) 1.09 (0.93, 1.27) Atopic dermatitis (L) 0.94 (0.82, 1.09) 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 yr 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) Molds 1.00 (0.69, 1.46)
Reference: Bakke et al. (2004, 156246) **Period of Study:** Jan 1989-Jun 2002 **Location:** One of Norway's major construction companies	**Outcome:** Spirometric measurements **Age Groups:** All ages, mean = 39 yr **Study Design:** Cohort **N:** 651 male construction workers **Statistical Analyses:** Multiple linear regression models **Covariates:** Age, yr 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. µg/m^3):** mg·y/m^3 **Monitoring Stations**: 16 tunnel sites visited with sampling equipment **Copollutant (correlation):** Total dust: r = 0.99 α 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

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Bakke et al. (2004, 156246) **Period of Study:** Jan 1989-Jun 2002 **Location:** One of Norway's major construction companies	**Outcome:** Spirometric measurements **Age Groups:** All ages, mean = 39 yr **Study Design:** Cohort **N:** 651 male construction workers **Statistical Analyses:** Multiple linear regression models **Covariates:** Age, yr 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. µg/m^3):** mg·y/m^3 **Monitoring Stations**: 16 tunnel sites visited with sampling equipment **Copollutant (correlation):** Respirable dust: r = 0.99 α 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
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 yr **Study Design:** Cohort **N:** 1446 **Statistical Analyses:** Logistic regression models **Covariates:** Age, gender, use of ß2-agonists, use of inhaled corticosteroids, smoking, yr of data collection, and avg daily exposure to $PM_{2.5}$ in the 12 mo 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 mo 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-yr-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 mo **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 4 yr of life. **Symptom At 4-Yr-Old** Wheeze 4-yr-old: 1.23 [1.00: 1.51] Early-life: 1.20 [0.99: 1.46] Asthma, MD-diagnosed 4-yr-old: 1.15 [0.82: 1.62] Early-life: 1.32 [0.96: 1.83] Dry cough at night 4-yr-old: 1.11 [0.94: 1.31] Early-life: 1.14 [0.98: 1.33] Bronchitis, MD-diagnosed 4-yr-old: 0.88 [0.66: 1.18] Early-life: 0.86 [0.66: 1.11] ENT infection 4-yr-old: 1.13 [0.98: 1.31] Early-life: 1.17 [1.02: 1.34] Flu/serious cold, MD-diagnosed 4-yr-old: 1.21 [1.02: 1.42] Early-life: 1.25 [1.07: 1.46] Itchy rash 4-yr-old: 0.96 [0.82: 1.11] Early-life: 0.98 [0.85: 1.14] Eczema, MD-diagnosed 4-yr-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-Yr-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-yr-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 mo **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. µg/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 4 yr of life. **Symptom At 4-Yr-Old** Wheeze 4-yr-old: 1.18 [0.98: 1.41] Early-life: 1.18 [1.00: 1.40] Asthma, MD-diagnosed 4-yr-old: 1.15 [0.85: 1.55] Early-life: 1.30 [0.98: 1.71] Dry cough at night 4-yr-old: 1.13 [0.97: 1.30] Early-life: 1.14 [1.00: 1.31] Bronchitis, MD-diagnosed 4-yr-old: 0.90 [0.69: 1.16] Early-life: 0.88 [0.69: 1.11] ENT infection 4-yr-old: 1.15 [1.01: 1.31] Early-life: 1.16 [1.03: 1.31] Flu/serious cold, MD-diagnosed 4-yr-old: 1.18 [1.02: 1.36] Early-life: 1.19 [1.04: 1.37] Itchy rash 4-yr-old: 0.94 [0.82: 1.08] Early-life: 0.97 [0.85: 1.10] Eczema, MD-diagnosed 4-yr-old: 0.99 [0.84: 1.17] Early-life: 0.97 [0.83: 1.14] **Allergen Sensitivity At 4-Yr-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-Yr-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]
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-wk periods dispersed throughout 1 yr, 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 µg/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)

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:** $PM_{2.5}$ "soot" **Averaging Time:** 4 2-wk periods dispersed throughout 1 yr, 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. µg/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 µg/m^3 EC) **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]
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 yr) **Age Groups:** 0-2 yr **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:** EC (EC) **Averaging Time:** 8 wk (4 2-week periods dispersed throughout 1 yr, 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 µg/m^3 (~ IQR) EC: ~0.5 µg/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, 087809) **Period of Study:** 3 wk in Jul and Jan 1997 and 2 wk in Nov 1996 and Apr 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 yr **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
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 yr **Study Design:** Cross-sectional **N:** 9,488 subjects who had been residents in same place for at least 3 yr 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

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:** $PM_{2.5}$ **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
Reference: Gauderman et al. (2000, 012531) **Period of Study:** 1993-1997 **Location:** Southern California	**Outcome:** FVC, FEV_1, MMEF, FEF75 **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-wk avg $PM_{2.5}$ **Mean (SD):** $PM_{2.5}$ 25.9 **Copollutant (correlation):** 03: r = -0.32 $PM_{10-2.5}$: r = 0.76 NO_2: r = 0.74 Inorg. Acid: r = 0.79	**Increment:** 25.9 µg/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) FEF75 -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) FEF75 -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) FEF75 -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 yr) **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 yr, 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 avg **Mean (SD):** The avg levels were presented in an online data supplement (Fig E1) **PM Component:** EC and OC. **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 µg/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 yr **Study Design:** Prospective Cohort Study **N:** 12 Communities 2,034 children 24,972 child-mo **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-wk measurements used to create annual avg 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 µg/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/FEF25.75 **Age Groups:** Children (mean age 10 at recruitment, followed for 8 yr) **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 yr, 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 µg/m^3 Pollutant effect reported as difference in 8 yr 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 yr **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 (y/n), home dampness (y/n), indoor moulds	**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: 10th: 1.47 * 10-5 25th: 1.54 * 10-5	**PM Increment:** $PM_{2.5}$ mass: 1.5 µg/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 yr: All: 0.96 (0.83-1.12) Males: 0.93 (0.76-1.14) Females: 1.04 (0.83-1.30) **Cough W/O Infection** ($PM_{2.5}$ mass) Age of 1 yr: All: 1.34 (1.11-1.61) Males: 1.43 (1.14-1.80) Females: 1.19 (0.84-1.70) **Dry Cough At Night** ($PM_{2.5}$ mass) Age of 1 yr: All: 1.31 (1.07-1.60) Males: 1.39 (1.08-1.78) Females: 1.17 (0.81-1.68)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
	(y/n), keeping of dogs (y/n) and cats (y/n) study (GINI or LISA) **Dose-response Investigated?** No	50th(Median): 1.70 * 10-5 75th: 1.88 * 10-5 90th: 2.13 * 10-5 **Range (Min, Max):** $PM_{2.5}$ mass: 11.9, 21.9 $PM_{2.5}$ absorbance: 1.38 to 4.39 * 10-5 $PM_{2.5}$ mass: $PM_{2.5}$ absorbance: 1/m **PM Component:** $PM_{2.5}$ mass $PM_{2.5}$ absorbance (as a marker of diesel soot) **Monitoring Stations**: 40 **Copollutant (correlation):** NO_2: r = 0.99 $PM_{2.5}$ absorbance and NO_2: r = 0.95 $PM_{2.5}$ mass and $PM_{2.5}$ absorbance: r = 0.96	Age of 2 yr: All: 1.20 (1.02-1.42) Males: 1.25 (1.01-1.55) Females: 1.13 (0.86-1.48) **Bronchitis** ($PM_{2.5}$ mass) Age of 1 yr: All: 0.98 (0.80-1.20) Males: 0.97 (0.76-1.25) Females: 0.98 (0.68-1.41) Age of 2 yr: All: 0.92 (0.78-1.09) Males: 0.92 (0.74-1.14) Females: 0.91 (0.68-1.21) **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 yr: 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 yr: 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 yr: 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) Females: 1.16 (0.79-1.71) Age of 2 yr: 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 yr: 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 yr: 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 yr: All: 0.92 (0.78-1.09) Males: 0.83 (0.66-1.05) Females: 1.06 (0.83-1.34))

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:** Children and adults over the age of 6 **Study Design:** Ccohort **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 avg **Mean (SD):** 13.7(4.2) Percentiles: 25th: 11.8 50th(Median): 13.9 75th: 15.9 **Monitoring Stations**: 713	**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 1.21 (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)
Reference: Hertz-Picciotto et al. (2005, 088678) **Period of Study:** May 1994-Mar 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 avg **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 µg/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 Fig 2 and Table 3

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: (Hertz-Picciotto et al., 2007, 135917) **Period of Study:** 1994-98 + follow-ups at up to 4.5 yr 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 yr 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, yr 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 avg **Mean (SD):** Daily mean 22.3 (sd 16 for 3-day avg, 11 for 45-day avg)	**PM Increment:** 25 µg/m^3 **RR Estimate [Lower CI, Upper CI] lag:** Bronchitis, birth-23 mo of age Categorical model High 30-day avg $PM_{2.5}$ (greater than 50 µg/m^3) 2.26(1.81-2.82) Medium 30-day avg $PM_{2.5}$ (between 25 and 50 µg/m^3) 1.48(1.32-1.65) Continuous model 1.30(1.08-1.58) Bronchitis, 2-4.5 yr of age Categorical model High 30-day avg $PM_{2.5}$ (greater than 50 µg/m^3) 3.66(2.07-6.48) Medium 30-day avg $PM_{2.5}$ (between 25 and 50 µg/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.
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 yr 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 µg/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]

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
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 yr **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 yr	**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 OC correlated as a "non-O_3 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) FEF25-75 ≤ 90: 21.1 (8.8, 50.5) FEF25-75 90-110: 11.9 (4.7, 30.0) FEF25-75 >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) FEF25-75 ≤ 90: 23.8 (10.2, 55.6) FEF25-75 90-110: 23.9 (9.9, 57.7) FEF25-75 >110: 15.9 (6.3, 40.5) Overall: 18.4 (9.4, 35.9)
Reference: Karr et al. (2007, 090719) **Period of Study:** 1995-2000 **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 µg/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-Jun (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 wk **Mean (SD):** Study Avg 12 **Monitoring Stations**: 10 **Copollutant (correlation):** r2 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 µg/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)
Reference: McConnell et al. (2003, 049490) **Period of Study:** 1993-1999 **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-yr avg **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 µg/m^3 Between community unit 1 µg/m^3 Within community 1 µg/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)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: McConnell et al. (2003, 049490) **Period of Study:** 1993-1999 **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:** EC **Averaging Time:** 4-yr 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 OC: r = 0.88 NO_2: r = 0.54 O_3: r = 0.68	**PM Increment:** Between community range 1.1 µg/m^3 Between community unit 1 µg/m^3 Within community 1 µg/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)
Reference: McConnell et al. (2003, 049490) **Period of Study:** 1993-1999 **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:** OC **Averaging Time:** 4-yr 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 µg/m^3 Between community unit 1 µg/m^3 Within community 1 µg/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)

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 (yrly). **Age Groups:** 10-15-yr-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 yr (n = 48 = 12 communities · 4 yr) 25th: NR 50th(Median): 3.4 75th: NR **Range (Min, Max):** Community by yr (n = 48 = 12 communities · 4 yr): (0.89, 8.7) **Monitoring Stations**: 12 **Copollutant:** O_3 NO_2 EC OC Acid vapor (acetic and formic acid)	**PM Increment:** 3.4 µg/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.
Reference: (Meng et al., 2007, 093275) **Period of Study:** Nov 2000 and Sep 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 h **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.

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Millstein, J et al. (2004, 088629) **Period of Study:** Mar-Aug 1995, and Sep 1995-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 yr 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 µg/m^3 **Odds Ratio [lower CI, Upper CI]** Annual $PM_{2.5}$: 1.04 [0.83, 1.29] Mar-Aug $PM_{2.5}$: 0.91 [0.64, 1.30] Sep-Feb $PM_{2.5}$: 1.18 [0.89, 1.58]
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 yr **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 yr, 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 µg/m^3 **Odds Ratio [Lower CI, Upper CI]** Adjusted OR for $PM_{2.5}$ and: sneezing, runny/stuffed nose during the first yr 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 yr 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]

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Morgenstern et al. (2007, 090747) **Period of Study:** May 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 yr **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 yr, 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. μg/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 yr 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]
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 yr **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:** U.S.	**Outcome:** Respiratory allergy/hayfever **Study Design:** Cohort **Covariates:** Survey yr, 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 yr	**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 μg/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 yr **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 yr investigated, yr 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:** Nov 2002-Apr 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 yr **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:** 1 day lag & 5-day ma	**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):** ΔPEF = mean daily deviations in PEF $PM_{2.5}$-ΔPEF: -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 μg/m^3 ΔPEF (difference or change in peak expiratory flow) -0.0297 L/min
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

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: (Solomon et al., 2003, 087441) **Period of Study:** 1966-1997 **Location:** United Kingdom: Northern England, North-West Midlands, and Wales.	**Outcome:** Cardio-respiratory morbidity **Age Groups:** 45 yr 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:** Mar 1986-Oct 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, yr, 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) FEF25-75%: -2.97 (-5.8 to -0.2) Current Smokers: FEV_1: 0.62 (-2.1, 3.4) FVC: 0.64 (-2.0, 3.3) FEF25-75%: -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) FEF25-75%: -8.78 (-14.7 to -2.9) Nonsmokers: FEV_1: -0.98 (-2.9, 0.9) FVC: -0.32 (-2.0, 1.4) FEF25-75%: -4.39 (-8.1 to -0.6) Exposure-response relationship presented graphically in Fig 1: the highest BC exposure group had decreases in FEV_1, FVC, and FEF25-75% compared with the lowest tertile group, although these differences were not statistically significant.
Reference: (Sunyer et al., 2006, 089771) **Period of Study:** initial selection: 1991-1993, follow-up Jun 2000-Dec 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 mo **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]

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 yr with or apart from colds) Persistent phlegm (brought up phlegm or mucus from the chest for at least 1 month per yr 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, yr of questionnaire administration, season of questionnaire administration, parental asthma prevalence.	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 2 yr **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 yr (1993-1996) schoolyards of participating children over a period of 2 yr (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) **Period of Study:** 2000-2004 **Location:** Beirut, Lebanon	ED Admissions **Outcome:** Acute respiratory symptoms: asthma, URTI, pneumonia, bronchitis **Age Groups:** <17 **Study Design:** Ecological (natural experiment comparing admissions before and after ban on diesel fuel) **N:** 5 hospitals, 7573 admissions Oct-Feb, 4303 admissions Oct-Dec **Statistical Analyses:** T-test, Poisson regression **Covariates:** Month of Year, temperature, humidity, orthogonalized rainfall **Season:** Oct-Dec (excluding flu season) and Oct-Feb **Dose-response Investigated?** No **Statistical Package:** NR **Lags Considered:** 1-2 yr before the ban compared to 1-2 yr after the ban	**Pollutant:** PM from diesel **Range (Min, Max):** NR **PM Component:** NR **Monitoring Stations:** 1 **Notes:** Did not look at specific exposure data looked at outcome with respect to a timeline that plotted admissions before and after a ban on diesel fuel. **Copollutant:** NR	**PM Increment:** NA β (p-value): **2-yr pre-ban vs. 2-yr post-ban** Oct to Feb All Resp: 0.128 (0.32) Asthma: -0.176 (0.16) Bronchitis: 0.505 (0.02) Pneumonia: 0.287 (0.17) URTI: -0.265 (0.41) Oct to Dec 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-yr pre-ban vs. 1-yr 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-yr pre-ban vs. 1-yr 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)
Reference: Kasamatsu et al. (2006, 156627) **Period of Study:** 2001-2002 **Location:** Shenyang, China	**Outcome:** FVC, FEV_1, PEF, FEF75 **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) FEF75 -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) FEF75 -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) FEF75 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) FEF75 -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, FEF75 **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 µg/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) FEF75 -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) FEF75 -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) FEF75 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) FEF75 -0.033(-0.106,0.040)

[1]All units expressed in µg/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 yr	**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 yr)** IQR: 43 days/yr Males: 2.38 (1.42, 3.97) Females: 1.08 (0.55, 2.13)
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:** Feb 2001 **Location:** Prague, Czech Republic	**Outcome:** Total DNA adducts (bulky aromatic PAH-DNA adducts and … **Age Groups:** 22-50 yr **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[α]P. **Total PAH-DNA adducts**: p = 0.065 Exposed: 0.92 ± 0.28 adducts/108 nucleotids Control: 0.82 ± 0.23 adducts/108 nucleotids **B[α]P-like adducts:** Exposed: 0.122 ± 0.36 adducts/108 nucleotids Control: 0.099 ± 0.035 adducts/108 nucleotids Multiple regression "like" B[α]P-DNA adduct for air pollution exposure group: B = 0.016, p = 0.01

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
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 24-h 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
Reference: (Pope et al., 2002, 024689) **Period of Study:** 1982-1998 **Location:** 50 U.S. states, District of Columbia, and Puerto Rico	**Outcome (ICD9):** Lung cancer mortality (162) **Age Groups:** Ages >30 yr **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 Fig 5 and not presented quantitatively anywhere else
Reference: Sram et al, (2007, 188457) **Period of Study:** Jan and Mar 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 yr, spending 8+ h outdoors	**Pollutant:** PM_{10} **Averaging Time:** NR **Mean (SD) Unit:** Jan: 55.6 µg/m^3 Mar: 36.4 µg/m^3 **Copollutant:** $PM_{2.5}$	Results not given by PM increment.
Reference: Sram et al, (2007, 188457) **Period of Study:** Jan and Mar 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 yr, spending 8+ h outdoors	**Pollutant:** $PM_{2.5}$ **Averaging Time:** NR **Mean (SD) Unit:** Jan: 44.4 µg/m^3 Mar: 24.8 µg/m^3 **Copollutant:** PM_{10}	Results not given by PM increment.

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
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 yr, mean age 44.	**Pollutant:** PM_{10} **Averaging Time:** NR **Mean (SD) Unit:** NR **Individual Exposure Range:** 73.4-1220 µg/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
Reference: (Vineis et al., 2006, 192089) **Period of Study:** 1990-1999 **Location:** 10 European countries	**Outcome:** Lung cancer **Study Design:** Nested case-control **Covariates:** Age, sex, country, smoking status, time since recruitment, education, BMI, physical activity, intake of fruit, vegetables, meat, alcohol and energy **Statistical Analysis:** Conditional logistic regression models **Statistical Package:** NR **Age Groups:** 35-74 at recruitment	**Pollutant:** PM_{10} **Averaging Time:** NR **Mean by Country (µg/m^3):** 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	**Increment:** 10 µg/m^3 **Odds Ratios (Min CI, Max CI) for increase in lung cancer per increment increase in** PM_{10} 0.91 (0.70-1.18)
Reference: (Wei et al., 2009, 192361) **Period of Study:** Nov 2006-Jan 2007 **Location:** Peking, China	**Outcome:** Urinary 8-OHdG increase **Study Design:** Panel **Covariates:** NR **Statistical Analysis:** Analysis of variance model with autoregressive terms **Statistical Package:** SAS **Age Groups:** Two nonsmoking security guards, ages 18 and 20	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Median:** 154.87 µg/m^3 **IQR:** 166.29 **Copollutant (correlation):** NA	**Increment:** 166.29 µg/m^3 **8-OHdG Concentrations, pre and post-work shift, subjects avgd** Pre-work: 1.83 Post-work: 6.92 **Concentration Changes (95%CI) of 8-OHdG per IQR Increase** 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 µg/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) **Period of Study:** Jan 1999-Jun 2007 **Location:** Boston, Massachusetts	**Outcome:** DNA methylation of LINE-1 and Alu **Study Design:** Panel **Covariates:** age, BMI, smoking status, pack-yr, statin use, fasting blood glucose, diabetes mellitus, percent lymphocytes and neutrophils in differential blood count, day of the week, season, temperature **Statistical Analysis:** Mixed effects models **Statistical Package:** SAS **Age Groups:** 719 elderly individuals, mean age 73.3, range 55-100 yr	**Pollutant:** $PM_{2.5}$ **Averaging Time:** NR **Mean (SD) Unit:** 4h: 12.2 (7.7) µg/m^3 1 day: 10.9 (6.3) µg/m^3 2 day: 10.6 (5.2) µg/m^3 3 day: 10.4 (4.8) µg/m^3 4 day: 10.3 (4.3) µg/m^3 5d: 10.2 (3.9) µg/m^3 6d: 10.3 (3.5) µg/m^3 7d: 10.3 (3.3) µg/m^3 **Copollutants:** Black carbon, Sulfate	**Increment:** SD for each lag **Correlation Coefficient (95% CI)** **Lag for LINE-1 Methylation** 4h: -0.07 (-0.13, -0.01), p = 0.03 1 day: -0.09 (-0.16, -0.02), p = 0.008 2 day: -0.10 (-0.17, -0.03), p = 0.003 3 day: -0.10 (-0.17, --0.04), p = 0.003 4 day: -0.10 (-0.16, -0.03), p = 0.004 5d: -0.10 (-0.16, -0.03), p = 0.004 6d: -0.11 (-0.17, -0.04), p = 0.001 7d: -0.13 (-0.19, -0.06), p < 0.001 **Correlation Coefficient (95% CI)** **Lag for Alu Methylation** 4h: 0.03 (-0.03, 0.09), p = 0.28 1 day: -0.01 (-0.07, 0.05), p = 0.74 2 day: -0.01 (-0.07, 0.05), p = 0.82 3 day: -0.01 (-0.07, 0.05), p = 0.78 4 day: -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 ma of pollutant levels** 4h: -0.04 (-0.11, 0.03), p = 0.24 7d: -0.11 (-0.18, -0.05), p = 0.001
Reference: Binkova et al. (2007, 156273) **Period of Study:** Feb 2001 **Location:** Prague, Czech Republic	**Outcome:** Bulky aromatic PAH-DNA adducts **Age Groups:** 22-50 yr **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[α]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[α]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

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
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 yr at enrollment	**Pollutant:** $PM_{2.5}$, estimated from PM_{10} levelsf **Averaging Time:** 24 h **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:** 10 µg/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 yr of age or younger **Study Design:** Matched case-control by sex, yr 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
Reference: Nafstad et al. (2004, 087949) **Period of Study:** 1972-1998 **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 yr	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 and Burnett, 2007, 090928) **Period of Study:** 1982-1998 **Location:** 50 U.S. states, District of Columbia, and Puerto Rico	**Outcome:** Lung cancer mortality (162) **Age Groups:** >30 yr **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) **Avg:** 17.7(3.7) **Averaging time:** 1982-1998	**PM Increment:** 10 µg/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 Fig 2-5. Authors found that $PM_{2.5}$ had the strongest association with increased risk of all-cause, cardiopulmonary, and lung cancer mortality.

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Sram et al, (2007, 188457) **Period of Study:** Feb 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 yr, spending 8+ h outdoors	**Pollutant:** PM_{10} **Averaging Time:** NR **Range:** 32-55µg/m^3 **Copollutant:** $PM_{2.5}$	Results not given by PM increment.
Reference: Sram et al, (2007, 188457) **Period of Study:** Feb 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 yr, spending 8+ h outdoors	**Pollutant:** $PM_{2.5}$ **Averaging Time:** NR **Range:** 27-38µg/m^3 **Copollutant:** PM_{10}	Results not given by PM increment.
Reference: Tovalin et al. (Tovalin et al., 2006, 091322) **Period of Study:** Apr-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 µg/m^3 Indoor Worker: 86.6 µg/m^3 Puebla Outdoor Worker: 122 µg/m^3 Indoor Worker: 78.3 µg/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 µg/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 et al., 2002, 024689) **Period of Study:** 1982-1998 **Location:** 50 U.S. states, District of Columbia, and Puerto Rico	**Outcome:** Lung cancer mortality (162) **Age Groups:** Ages >30 yr who were members of a household with at least 1 individual ≥45yrs. **Study Design:** Longitudinal cohort (Cancer Prevention Study II) **N:** 359,000 CPS II participants with information regarding PM15 and PM15-$PM_{2.5}$ **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: yr smoked, yr smoked squared, cigarettes per day, cigarettes per day squared, number of h per day exposed to passive cigarette smoke.	**Pollutant:** PM_{15} **Mean (SD):** 1979-1983: 40.3(7.7) **Pollutant:** PM15-2.5 **Mean (SD):** 1979-1983: 19.2(6.1) **Averaging Time:** 1979-1983	Relative risks effect estimates were recorded in Fig 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 µg/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 yr 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 µg/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 wk): 1.13 (0.95, 1.35)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Chen et al. (2002, 024945) **Period of Study:** 1991-1999 **Location:** Washoe County, Nevada	**Outcome:** Birth weight **Age Groups:** Sngle births with gestational age between 37-44 wk and maternal all ages **Study Design:** Cross-sectional **N:** 33,859 single births **Statistical Analyses:** multiple linear and logistic regression **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 **Dose-response Investigated?** No **Statistical Package:** SPSS 10.0	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (SD):** 31.53 (22.32) **Percentiles: 25th:** 16.80 **50th(Median):** 26.30 **75th:** 39.35 **Range (Min, Max):** (0.97-157.32) **Monitoring Stations:** 4 **Copollutant:** CO O_3	**PM Increment:** 10 µg/m^3 **Effect Estimate [Lower CI, Upper CI]:** Using continuous pollutant variables Model 1-PM_{10} 1 trimester Crude model: ß = -0.186 (0.225) Adjusted model: ß = -0.082 (0.221) 2 trimester Crude model: ß = 0.045 (0.223) Adjusted model: ß = -0.020 (0.221) 3 trimester 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 2 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 3 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 µg/m^3 Exposure to PM_{10} at 3 trimester at >44.74 µg/m^3: OR = 1.105 (0.714-1.709) Between 19.72-44.74 µg/m^3: OR = 1.050 (0.811-1.360) **Notes:** Crude model: model with air-pollutant variables controlled with gestational age only. Adjusted model: model with air-pollutant variables controlled with confounding variables including gestational age, infant sex, maternal residential city, education, medical risk factors, active tobacco use, drug use, alcohol use, the trimester begins prenatal visits, total prenatal visits, mother's age, race and ethnicity of mother, and weight gain of mother.

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Dales et al. (2004, 087342) **Period of Study:** Jan 1984-Dec 1999 **Location:** Canada (12 cities)	**Outcome:** SIDS (a sudden, unexplained death of a child <1 yr 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 mo **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 1 monitoring site, they were avgd **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).

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Dugandzic et al. (2006, 088681) **Period of Study:** Jan 1988-Dec 2000 **Location:** Nova Scotia, Canada	**Outcome:** Low birth weight (LBW) (<2500 grams) **Age Groups:** Babies born ≥ 37 wk (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, yr 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 3 stations monitored more than 1 pollutant. Daily data were available for gaseous pollutants while particulate levels were measured every sixth day.	**PM Increment:** 1) IQR (5 μg/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 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:** Jan 1996-Dec 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, yr and maternal county of residence	**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 or 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 wk 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 Sepal 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 Sepal 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** **Conotruncal defects:** 25th: 1.13 (0.79, 1.62) 50th: 1.20 (0.84, 1.72) 75th: 1.26 (0.86, 1.84)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
	Season: Covariate in model **Dose-response Investigated?** Yes **Statistical Package:** SAS v 8.2		**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 Sepal 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 Sepal 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) 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 Sepal defects:** 25th: 1.06 (0.90, 1.24) 50th: 1.10 (0.94, 1.29) 75th: 1.08 (0.92, 1.27)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
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 µg/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)
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, ma 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 µg/m^3, 245.4 µg/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 µg/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

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Hansen, et al. (2006, 089818) **Period of Study:** Jul 2000-Jun 2003 **Location:** Brisbane, Australia	**Outcome:** Pre-term birth (<37 wk) **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, avgd 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 µg/m^3 Last 90 days prior to birth 5.7 µg/m^3 **Odds Ratio [Lower CI, Upper CI]:** Trimester 1 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 **Dose-response Investigated?** Yes, assessed exposures as quartiles **Statistical Package:** SAS v8.2	**Pollutant:** PM_{10} **Averaging Time:** Trimester and monthly avg 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_{10} T2: r = 0.12 PM_{10} T3: r = -0.55 O_3 T1: r = 0.77 O_3 T2: r = 0.28 O_3 T3: r = -0.61 NO_2 T1: r = 0.32 NO_2 T2: r = -0.65 NO_2 T3: r = -0.17 visibility reducing particles (bsp) T1: r = 0.82 visibility reducing particles (bsp) T2: r = -.15 visibility reducing particles (bsp) T3: r = -0.50 PM_{10} T1: r = 0.12 PM_{10} T2: PM_{10} T3: r = 0.04 O_3 T1: r = -0.11 O_3 T2: r = 0.80 O_3 T3: r = 0.18 NO_2 T1: r = 0.77 NO_2 T2: r = 0.25 NO_2 T3: r = -0.72 visibility reducing particles (bsp) T1: r = 0.23 visibility reducing particles (bsp) T2: r = 0.80 visibility reducing particles (bsp) T3: r = -0.24 PM_{10} T1: r = -0.55 PM_{10} T2: r = 0.04 PM_{10} T3:	**PM Increment:** IQR (8.1 µg/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) Quartiles of exposure: 1: Ref 2: 12.7 (-2.3, 27.6) 3: 7.6 (-10.6, 25.7) 4: 1.0 (-18.7, 20.7) p-trend: 0.922 Trimester 3: Continuous exposure: 3.6 (-6.9, 14.0) Quartiles of exposure: 1: Ref 2: 2.9 (-12.8, 18.7) 3: 18.5 (0.0, 36.9) 4: 4.3 (-15.8, 24.4) p-trend: 0.524 **ORs for SGA associated with trimester-specific exposures** Trimester 1: Continuous exposure: 1.04 (0.96, 1.12) Quartiles of exposure: 1: Ref 2: 1.23 (1.07, 1.42) 3: 1.12 (0.95, 1.31) 4: 1.12 (0.94, 1.34) p-trend: 0.361 Trimester 2: Continuous exposure: 0.95 (0.88, 1.04) Quartiles of exposure: 1: Ref 2: 0.96 (0.83, 1.11) 3: 1.06 (0.89, 1.25) 4: 0.98 (0.81, 1.18) p-trend: 0.962 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)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
		O_3 T1: r = -0.56 O_3 T2: r = -0.18 O_3 T3: r = 0.81 NO_2 T1: r = -0.20 NO_2 T2: r = 0.75 NO_2 T3: r = 0.22 visibility reducing particles (bsp) T1: r = -0.62 visibility reducing particles (bsp) T2: r = 0.19 visibility reducing particles (bsp) T3: r = 0.79	Quartiles of exposure: 1: Ref Trimester 3: Continuous exposure: 0.93 (0.85, 1.03) Quartiles of exposure: 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) Quartiles of exposure: 1: Ref 2: -0.02 (-0.07, 0.04) 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 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

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: (Hansen et al., 2009, 192362) **Period of Study:** Jan 1997-Dec 2004 **Location:** Brisbane, Australia	**Outcome:** Birth defects- artery and valve, atrial and ventricular Sepal, 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 µg/m^3 **Range (Min, Max):** (4.4, 151.7) **Copollutant (correlation):** NR	**Increment:** 4µg/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 Sepal 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 Sepal 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 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 wk term birth: ≥ 37 wk but <42 wk) **Age Groups:** Infants **Study Design:** Cross-sectional **N:** 123,840 singleton births of >20 wk 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 avg used to calculate the mean concentration over the first trimester, the 3 mo preceding birth, the first month after the estimated date of conception, and the month prior to delivery **Mean (SD):** (24 h avg) All yr: 16.3 (6.38) Summer: 18.2 (7.20) Fall: 17.0 (6.23) Winter: 14.5 (5.57) Spring: 15.7 (5.82) **Monitoring Stations:** 14 stations within the Sydney metropolitan area (levels avgd to provide 1 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 µg/m^3 **Effect Estimate [Lower CI, Upper CI]:** ORs (air pollutant concentration during the 1st trimester and preterm birth by season) Fall: 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 mo 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 fall did not remain in 2 pollutant models (ORs between 0.77 and 1.04)
Reference: Kaiser et al. (2004, 076674) **Period of Study:** 1995-1997 **Location:** 25 U.S. 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 mo **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 yr of birth, avg temperature in the first 2 mo of life **Season:** All adjusted for month/yr 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 µg/m^3	**PM Increment:** Analysis 1: 16.4 µg/m^3 (difference between reference level of 12 µg/m^3 and observed mean level of 28.4 µg/m^3) Analysis 2: 13 µg/m^3 (difference between reference level of 12 µg/m^3 and 25 µg/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 µg/m^3 (i.e., reference level was set at 12 µg/m^3) -Attributable risks are based on the RRs reported by Woodruff et al, 1997 for a 10 µg/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 2001-May 2004	**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	**Pollutant:** PM_{10} **Averaging Time:** Used hourly exposure levels to calculate avg	**PM increment:** 10 µg/m^3 **Preterm:** 1st Trimester Odds Ratios:

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Location: Seoul, Korea	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 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	exposure levels at each trimester, each month of pregnancy, and 6 wk 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	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) **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: 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 µg/m^3 increase in PM concentration:**

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			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)
Reference: Lee et al. (2003, 043202) **Period of Study:** Jan 1996-Dec 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 wk **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 mo using IQR during last 5 mo: 0.94 [0.85, 1.05] Low exposure in first 5 mo using IQR during first 5 mo: 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 yr of age, and low parental education level.
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:** Jan 1998-Dec 2000 **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, "ma 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 µg/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 µg/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 ma from 2-7 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 µg/m^3 increase in PM_{10}
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	**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 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)
	(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	PM_{10} SO_4^{2-} (r = 0.10) $NSPM_{10}$-non-sulfate portion of PM_{10} (r = 0.91) CO (r = 0.27) SO_2 (r = 0.04) **Notes:** TSP-based sulfate was adjusted for compatibility with the PM_{10}-based data	SO_4^{2-}: -0.0002 (0.0061) $NSPM_{10}$: 0.0115 (0.0014) Cause of death: All Birth weight: LBW 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) 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

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			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) $NSPM_{10}$: 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 wk **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, yr 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 **Statistical Package:** STATA	**Pollutant:** PM_{10} **Averaging Time:** Trimester avg 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 ≥ 95th: ≥ 43.929 3rd Trimester <25th: <24.702 25 to <50th: 24.702, 30.162 50 to <75th: 30.163, 35.642 75 to <95th: 35.643, 43.588 ≥ 95th: ≥ 43.589 **Monitoring Stations:** 3-4 per city **Copollutants:** CO, SO_2	**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) Continuous: 0.93 (0.85, 1.00) 2nd Trimester Crude <25th: 1.00 25 to <50th: 1.01 (0.93, 1.10) 50 to <75th: 0.90 (0.66, 1.21) 75 to <95th: 0.92 (0.62, 1.34) ≥ 95th: 0.90 (0.61, 1.33) Continuous: 0.95 (0.78, 1.16) 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

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			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) 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 <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)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			Continuous: 0.96 (0.80, 1.15)
Reference: Mannes et al.(2005, 087895) **Period of Study:** Jan 1998-Dec 2000 **Location:** Metropolitan Sydney, Australia	**Outcome:** Risk of SGA and birth weight **Age Groups:** All singleton births >20 wk 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 µg/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)
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 wk 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 yr **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 h mean **Mean (SD):** 65.04 (27.28) **Range (Min, Max):** (14.80, 192.80) **Monitoring Stations:** 13 (avgd 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)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Ritz et al. (2000, 012068) **Period of Study:** 1989-1993 **Location:** Southern California	**Outcome:** Preterm birth (treated dichotomously as birth at <37 wk gestation Also analyzed continuously) **Age Groups:** Infants (born vaginally between 26-44 wk 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 wk exposure period) **Statistical Package:** NR	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg at 6 day intervals avgd pollutant measures for 1, 2, 4, 6, 8, 12, and 26 wk before birth and the whole pregnancy period **Mean (SD):** 6 wk before birth: 47.5 (15.0) 1st month of pregnancy: 49.3 (16.9) **Range (Min, Max):** 6 wk 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 wk 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:** Avgd 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 wk 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 wk 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 wk before birth Crude: 1.27 (1.12, 1.44) 2 exposure periods: 1.27 (1.11, 1.44) 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 wk 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 wk 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 (Jul 1990-Jul 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 Sepum 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 Sepal defects not included in the conotruncal category. **Age Groups:** All live born infants and fetal deaths diagnosed between 20 wk of gestation and 1 yr 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 O_3 and CO, study found a clear dose-response pattern for aortic Sepum and valve and ventricular Sepal 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 O_3 on these cardiac defects. (Quantitative results not shown).

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
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 yr 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, fall, winter) **Dose-response Investigated?** Yes **Statistical Package:** NR	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (SD):** **2** wk before death: 46.2 1 month before death: 46.3 2 mo before death: 46.3 6 mo before death: 46.3 **Range (Min, Max):** **2** wk before death: (21.0-83.5) 1 month before death: (25.0-77.2) 2 mo before death: (27.6-74.2) 6 mo before death: (31.3-69.5) **Monitoring Stations:** maximum of 31 **Copollutants (correlation):** **2** wk before death CO: r = 0.33 NO_2: r = 0.48 O_3: r = 0.12 1 month before death CO: r = 0.33 NO_2: r = 0.48 O_3: r = 0.12 2 mo before death CO: r = 0.32 NO_2: r = 0.48 O_3: r = 0.12 6 mo before death CO: r = 0.29 NO_2: r = 0.44 O_3: r = 0.16	**PM Increment:** 10 $\mu g/m^3$ **Effect Estimate [Lower CI, Upper CI]:** **All-cause death** **2 mo before death** Single-pollutant model: <25th = 1.04 (1.01-1.06) 25th-75th = 0.96 (0.89-1.04) >75th = 1.14 (1.03-1.27) Multiple-pollutant model: <25th = 1.02 (0.99-1.05) 25th-75th = 0.92 (0.84-1.00) >75th = 1.07 (0.95-1.20) **SIDS** **2 mo before death:** Single-pollutant model: <25th = 1.03 (0.99-1.08) 25th-75th = 0.94 (0.81-1.08) >75th = 1.13 (0.93-1.36) Multiple-pollutant model: <25th = 1.01 (0.95-1.07) 25th-75th = 0.90 (0.76-1.06) >75th = 0.99 (0.80-1.24) **Respiratory death** **2 wk before death** **Postneonatal deaths (28 days to 1 y)** Single-pollutant model: <25th = 1.05 (1.01-1.10) 25th-75th = 1.13 (1.01-1.10) >75th = 1.46 (1.13-1.88) Multiple-pollutant model: <25th = 1.04 (0.98-1.09) 25th-75th = 1.09 (0.86-1.38) >75th = 1.40 (1.03-1.89) **Postneonatal deaths (28 days to 3 mo)** Single-pollutant model: <25th = 1.01 (0.95-1.08) 25th-75th = 1.16 (0.82-1.63) >75th = 1.44 (0.96-2.17) Multiple-pollutant model: <25th = 1.00 (0.92-1.09) 25th-75th = 0.97 (0.67-1.42) >75th = 1.23 (0.76-2.00) **Post neonatal deaths (4-12 mo)** Single-pollutant model: <25th = 1.12 (1.02-1.23) 25th-75th = 1.08 (0.81-1.44) >75th = 1.41 (1.02-1.96) Multiple-pollutant model: <25th = 1.07 (1.00-1.15) 25th-75th = 1.02 (0.75-1.40) >75th = 1.36 (0.92-2.01)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Rogers et al. (2006, 091232) **Period of Study:** 1986-1988 **Location:** Georgia, USA	**Outcome**: VLBW Term, AGA, Preterm AGA, Preterm, SGA **Age Groups:** Newborns and their mothers (<19 to ≥ 35-yr-old) **Study Design:** Case-control **N:** 325 infants (69 preterm SGA 59 preterm AGA 197 term AGA) and their mothers **Statistical Analyses:** Logistic regression **Covariates:** Maternal age, maternal race, maternal education, active and passive smoking, birth season, prepregnancy weight, pregnancy weight gain, maternal toxemia, anemia, asthma **Dose-response Investigated?** Yes, used **Statistical Package**: SUDAAN Cochran-Armitage test for trend to determine whether the observed proportions of cases and controls differed in a linear manner across exposure categories.	**Pollutant:** PM_{10} **Averaging Time:** annual Preterm SGA: **50th(Median)**: 3.38 Preterm AGA: **50th(Median)**: 7.84 Term AGA: **50th(Median)**: 3.23 **Monitoring Stations**: NR **Percent Mothers Residing In County With Industrial Point Source** Preterm SGA: 60.9% Preterm AGA: 79.7% 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%	**PM Increment:** Quartile **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. 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) **Period of Study:** 1997-2001 **Location:** Ciudad Juarez, Mexico	**Outcome:** Respiratory-related infant mortality ICD9 (460-519) ICD10 (J00-J99) **Age Groups:** 1 month to 1 yr **Study Design:** Case crossover **N:** 216 respiratory-related deaths N = 412 other causes and N = 628 total deaths **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 2- and 3-day avg 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. **Covariate**: Temperature, season **Dose-response Investigated?** Yes **Statistical Package:** STATA 7.0 **Lags Considered:** 1-15 days	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (SD):** 1997: 33.04 (20.67) µg/m^3 1998: 35.25 (17.32) µg/m^3 1999: 45.92 (28.69) µg/m^3 2000: 43.38 (23.77) µg/m^3 2001: 39.46 (29.43) µg/m^3 **Monitoring Stations:** 5 stations in Ciudad Juarez 2 stations in El Paso (close to U.S.-Mexico border) **Copollutant (correlation):** O_3: r = 0.01 **Notes:** Ciudad Juarez monitors measured PM_{10} every 6 days while El Paso monitors measured on a daily basis.	**PM Increment:** 20 µg/m^3 **RR Estimate [Lower CI, Upper CI] lag:** **Total mortality:** OR = 1.02 (0.94-1.11) lag 1 OR = 1.03 (0.95-1.12) lag 2 OR = 1.03 (0.94-1.13 ac2 OR = 1.04 (0.95-1.15) ac3 **Respiratory mortality** OR = 0.95 (0.83-1.09) lag 1 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 2 or 3 days before death.
Reference: Sagiv et al. (2005, 087468) **Period of Study:** Jan 1997-Dec 2001 **Location:** Allegheny county, Beaver county, Lackawanna county, Philadelphia county, Pennsylvania, U.S.A.	**Outcome:** Preterm birth (<36 wk) **Age Groups:** Babies born between 20 and 44 wk **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 µg/m^3 2) Quartiles (first quartile is the reference) **Exposure period: 6 wk before birth** Per 50 µg/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 µg/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 Fig 1 displaying a graph of the relative risk (RR) and 95% confidence i ntervals (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 Fig shows the approximate RRs per 50 µg/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) **Period of Study:** 1975-1987 **Location:** Southern California	**Outcome:** Birth weight Low birth weight (LBW <2500 g) Intrauterine growth retardation (IUGR) **Age Groups:** Children born full-term (between 37 and 44 wk) **Study Design:** Cohort study **N:** 3901 children **Statistical Analyses:** Linear mixed-effects Logistic regression **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 **Season:** All **Dose-response Investigated?** Yes **Statistical Package:** SAS	**Pollutant:** PM_{10} **Averaging Time:** Monthly **Mean (SD):** Entire pregnancy: 45.8 (12.9) First trimester: 46.6 (15.9) Second trimester: 45.4 (14.8) Third trimester: 45.4 (15.5) **Monitoring Stations:** 1 or 3 (See notes) **Copollutant (correlation):** Entire pregnancy PM_{10}-O_3[10-6]: r = 0.54 PM_{10}-O_3[24 h): r = 0.20 PM_{10}-NO_2: r = 0.55 PM_{10}-CO: r = 0.41 First trimester PM_{10}-O_3[10-6]: r = 0.54 PM_{10}-O_3[24 h]: r = 0.34 PM_{10}-NO_2: r = 0.48 PM_{10}-CO: r = 0.29 Second trimester PM_{10}-O_3[10-6): r = 0.50 PM_{10}-O_3(24 h): r = 0.27 PM_{10}-NO_2: r = 0.53 PM_{10}-CO: r = 0.35 Third trimester PM_{10}-O_3[10-6]: r = 0.52 PM_{10}-O_3[24 h]: r = 0.31 PM_{10}-NO_2: r = 0.52 PM_{10}-CO: r = 0.37 **Notes:** Exposure estimates were calculated by spatially interpolated monthly avg which were based off of three monitoring stations located within 50 km of the ZIP code region of maternal birth residences.	**PM Increment:** IQR (interquartile range) **Outcome: birth weight (g)** Single-pollutant model Entire pregnancy 18 µg/m^3: -19.9 (-43.6, 3.8) First trimester 20 µg/m^3: -3.0 (-22.7, 16.7) Second trimester 19 µg/m^3: -15.7 (-36.1, 4.7) Third trimester 20 µg/m^3: -21.7 (-42.2 to -1.1) Multipollutant model (Included O_3 (24 h) in model Third trimester exposure) 20 µg/m^3: -10.8 (-31.8, 10.2) **Outcome: IUGR (ORs)** Single-pollutant model Entire pregnancy 18 µg/m^3: 1.1 (0.9, 1.3) First trimester 20 µg/m^3: 1.0 (0.9, 1.2) Second trimester 19 µg/m^3: 1.0 (0.9, 1.2) Third trimester 20 µg/m^3: 1.1 (0.9, 1.3) **Outcome: LBW** Single-pollutant model Entire pregnancy 18 µg/m^3: 1.3 (0.8, 2.2) First trimester 20 µg/m^3: 1.0 (0.7, 1.5) Second trimester 19 µg/m^3: 1.2 (0.8, 1.7) Third trimester 20 µg/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).
Reference: (Sokol et al., 2006, 098539) **Period of Study:** Jan 1996-Dec 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 µg/m^3 **Copollutant (correlation):** O_3, NO_2, CO	PM_{10} specific results are given in Fig 3-. 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)	**PM Increment:** Trimester ≥ 90th percentile compared to <90thpercentile Least-square (ANCOVA) mean (SE) **All Genotypes** 1st trimester <90th , N(%): 158 (90.3%): 3253 (37) ≥ 90th percentile, N(%): 17 (9.7%): 2841 (145) P-Value for mean birth weight for ≥ 90th percentile PM_{10} vs. for <90th percentile 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 percentile, N(%): 153 (89.5%): 3253 (39) ≥ 90th percentile, N(%): 18 (10.5%): 3026 (157) P-Value for mean birth weight for ≥ 90th

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
		3rd trimester (23.45, 172.75) **Monitoring Stations**: 27 **Copollutant:** CO SO_2 NO_2	percentile PM_{10} vs. for <90th percentile 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 percentile, N(%): 162 (90.5%): 3226 (38) ≥ 90th percentile, N(%): 17 (9.5%): 3122 (140) P-Value for mean birth weight for ≥ 90th percentile PM_{10} vs. for <90th percentile 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 percentile, N(%): 60 (34.3%): 3350 (64) ≥ 90th percentile, N(%): 5 (2.9%): 3001 (229) P-Value for mean birth weight for ≥ 90th percentile PM_{10} vs. for <90th percentile 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 percentile, N(%): 59 (34.5%): 3335 (66) ≥ 90th percentile, N(%): 6 (3.5%): 3281 (249) P-Value for mean birth weight for ≥ 90th percentile PM_{10} vs. for <90th percentile 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 percentile, N(%): 61 (34.1%): 3327 (65) ≥ 90th percentile, N(%): 6 (3.4%): 3227 (300) p-Value for mean birth weight for ≥ 90th percentile PM_{10} vs. for <90th percentile 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 percentile, N(%): 98 (56.0%): 3193 (48) ≥ 90th percentile, N(%): 12 (6.9%): 2799 (169) P-Value for mean birth weight for ≥ 90th percentile PM_{10} vs. for <90th percentile 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 percentile, N(%): 94 (55.0%): 3200 (52) ≥ 90th percentile, N(%): 12 (7.0%): 2933 (176) P-Value for mean birth weight for ≥ 90th percentile PM_{10} vs. for <90th percentile PM_{10} Adjusted: 0.161 Adjusted, with CO: 0.172

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			Adjusted, with NO_2: 0.152 Adjusted, with SO_2: 0.158 3rd trimester <90th percentile, N(%): 101 (56.4%): 3165 (49) ≥ 90th percentile, N(%): 11 (6.2%): 3087 (147) P-Value for mean birth weight for ≥ 90th percentile PM_{10} vs. for <90th percentile 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 percentile, N(%): 87 (49.7%): 3244 (52) ≥ 90th percentile, N(%): 7 (4.0%): 2983 (232) P-Value for mean birth weight for ≥ 90th percentile PM_{10} vs. for <90th percentile 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 percentile, N(%): 82 (48.0%): 3243 (55) ≥ 90th percentile, N(%): 11 (6.4%): 3185 (207) p-Value for mean birth weight for ≥ 90th percentile PM_{10} vs. for <90th percentile 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 percentile, N(%): 90 (50.3%): 3239 (53) ≥ 90th percentile, N(%): 9 (5.0%): 2944 (198) P-Value for mean birth weight for ≥ 90th percentile PM_{10} vs. for <90th percentile 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 percentile, N(%): 71 (40.6%): 3262 (56) ≥ 90th percentile, N(%): 10 (5.7%): 2773 (171) P-Value for mean birth weight for ≥ 90th percentile PM_{10} vs. for <90th percentile 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 percentile, N(%): 71 (41.5%): 3264 (61) ≥ 90th percentile, N(%): 7 (4.1%): 2862 (208) P-Value for mean birth weight for ≥ 90th percentile PM_{10} vs. for <90th percentile 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 percentile, N(%): 72 (40.2%): 3207

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			(58) ≥ 90th percentile, N(%): 8 (4.5%): 3262 (180) P-Value for mean birth weight for ≥ 90th percentile PM_{10} vs. for <90th percentile 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, 090709) **Period of Study:** 1994-2000 **Location:** Kaohsiung, Taiwan	**Outcome:** Post neonatal mortality **Age Groups:** Infants more than 27 days and less than 1 yr **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 µg/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 µg/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, 088668) **Period of Study:** 1994-2000 **Location:** Los Angeles County, California, U.S.	**Outcome:** Term low birth weight (LBW) (<2500 g at ≥ 37 completed wk gestation), Vaginal birth <37 completed wk gestation **Age Groups:** LBW: ≥ 37 completed wk Preterm births: <37 completed wk **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 h (every 6 days) Entire pregnancy Trimesters of pregnancy Months of pregnancy 6 wk before birth **Mean (SD):** First trimester: 42.2 Third trimester: 41.5 6 wk before birth: 39.1 **Range (Min, Max):** First trimester: 26.3, 77.4 Third trimester: 25.7, 74.6 6 wk 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 wk 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 µg/m^3 2) 3 levels: a) <25 percentile (reference) b) 25%-75 percentile c) ≥ 75 percentile **Incidence of LBW (third trimester exposure)** <32.8 µg/m^3: 2.0 (1.8, 2.2) 32.8 to <43.4 µg/m^3: 2.0 (1.9, 2.1) ≥ 43.4 µg/m^3: 2.2 (2.0, 2.4) **Incidence of preterm birth (first trimester exposure)** <32.9 µg/m^3: 8.7 (8.3, 9.2) 32.9 to <43.9 µg/m^3: 8.8 (8.5, 9.1) ≥ 43.9 µg/m^3: 8.6 (8.1, 9.0) **Incidence of preterm birth (6 wk before birth exposure)** <31.8 µg/m^3: 8.8 (8.4, 9.3) 31.8 to <44.1 µg/m^3: 8.6 (8.3, 8.9) ≥ 44.1 µg/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 µg/m^3: 1.22 (1.05, 1.41) 33.4 to <44.7 µg/m^3: 1.08 (0.76, 1.52) ≥ 44.7 µg/m^3: 1.48 (1.00, 2.19) Multipollutant model: Distance ≤ 1 mile Per 10 µg/m^3: 1.36 (1.12, 1.65) 33.4 to <44.7 µg/m^3: 1.16 (0.77, 1.74) ≥ 44.7 µg/m^3: 1.58 (0.95, 2.62) Single-pollutant model: 1 <distance ≤ 2 mile Per 10 µg/m^3: 0.98 (0.90, 1.06) 33.4 to <44.7 µg/m^3: 0.95 (0.80, 1.13) ≥ 44.7 µg/m^3: 0.96 (0.78, 1.18) Multipollutant model: 1 <distance ≤ 2 mile Per 10 µg/m^3: 1.02 (0.92, 1.14) 33.4 to <44.7 µg/m^3: 0.93 (0.77, 1.12) ≥ 44.7 µg/m^3: 1.02 (0.79, 1.32) Single-pollutant model:

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			2 <distance ≤ 4 mile Per 10 µg/m^3: 1.03 (0.99, 1.08) 33.9 to <45.0 µg/m^3: 1.04 (0.96, 1.14) ≥ 45.0 µg/m^3: 1.08 (0.97, 1.20) Multipollutant model: 2 <distance ≤ 4 mile Per 10 µg/m^3: 1.04 (0.98, 1.09) 33.9 to <45.0 µg/m^3: 1.02 (0.92, 1.12) ≥ 45.0 µg/m^3: 1.06 (0.93, 1.21) **Zip-code-level analysis** Single-pollutant model: Per 10 µg/m^3: 1.03 (0.97, 1.09) 33.2 to <43.6 µg/m^3: 0.98 (0.86, 1.11) ≥ 43.6 µg/m^3: 1.03 (0.88, 1.21) Multipollutant model: Per 10 µg/m^3: 1.07 (0.99, 1.15) 33.2 to <43.6 µg/m^3: 0.97 (0.85, 1.12) ≥ 43.6 µg/m^3: 1.09 (0.90, 1.31) **Outcome: LBW** **Exposure Period: Entire pregnancy period** **Address-level analysis:** Multipollutant model: Per 10 µg/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 µg/m^3: 1.00 (0.93, 1.09) 33.3 to <45.1 µg/m^3: 1.07 (0.90, 1.26) ≥ 45.1 µg/m^3: 1.12 (0.91, 1.38) Multipollutant model: Distance ≤ 1 mile Per 10 µg/m^3: 1.00 (0.90, 1.12) 33.3 to <45.1 µg/m^3: 1.12 (0.92, 1.36) ≥ 45.1 µg/m^3: 1.17 (0.90, 1.50) Single-pollutant model: 1 <distance ≤ 2 mile Per 10 µg/m^3: 1.01 (0.97, 1.05) 33.7 to <45.3 µg/m^3: 1.03 (0.95, 1.12) ≥ 45.3 µg/m^3: 1.07 (0.97, 1.19) Multipollutant model: 1 <distance ≤ 2 mile Per 10 µg/m^3: 1.04 (0.99, 1.10) 33.7 to <45.3 µg/m^3: 1.07 (0.98, 1.17) ≥ 45.3 µg/m^3: 1.13 (1.00, 1.27) Single-pollutant model: 2 <distance ≤ 4 mile Per 10 µg/m^3: 1.01 (0.99, 1.03) 34.1 to <45.5 µg/m^3: 1.03 (0.99, 1.08) ≥ 45.5 µg/m^3: 1.02 (0.96, 1.07) Multipollutant model: 2 <distance ≤ 4 mile Per 10 µg/m^3: 0.99 (0.97, 1.02) 34.1 to <45.5 µg/m^3: 0.99 (0.95, 1.04) ≥ 45.5 µg/m^3: 0.94 (0.89, 1.01) **Zip-code-level analysis** Single-pollutant model: Per 10 µg/m^3: 0.99 (0.96, 1.01) 33.3 to <44.2 µg/m^3: 1.01 (0.95, 1.08) ≥ 44.2 µg/m^3: 0.98 (0.90, 1.05) Multipollutant model: Per 10 µg/m^3: 0.99 (0.96, 1.03) 33.3 to <44.2 µg/m^3: 1.03 (0.97, 1.11) ≥ 44.2 µg/m^3: 1.01 (0.92, 1.11) **Outcome: Preterm birth** **Exposure Period: 6 wk before birth** **Address-level analysis:** Single-pollutant model: Distance ≤ 1 mile Per 10 µg/m^3: 1.02 (0.95, 1.10)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			32.5 to <44.8 µg/m^3: 1.09 (0.92, 1.29) ≥ 44.8 µg/m^3: 1.12 (0.92, 1.37) Multipollutant model: Distance ≤ 1 mile Per 10 µg/m^3: 1.06 (0.97, 1.16) 32.5 to <44.8 µg/m^3: 1.09 (0.90, 1.31) ≥ 44.8 µg/m^3: 1.17 (0.91, 1.49) Single-pollutant model: 1 <distance ≤ 2 mile Per 10 µg/m^3: 1.00 (0.96, 1.03) 32.3 to <45.3 µg/m^3: 0.99 (0.91, 1.07) ≥ 45.3 µg/m^3: 0.99 (0.89, 1.10) Multipollutant model: 1 <distance ≤ 2 mile Per 10 µg/m^3: 1.01 (0.97, 1.06) 32.3 to <45.3 µg/m^3: 1.00 (0.92, 1.10) ≥ 45.3 µg/m^3: 1.02 (0.91, 1.16) Single-pollutant model: 2 <distance ≤ 4 mile Per 10 µg/m^3: 0.99 (0.98, 1.01) 33.1 to <45.3 µg/m^3: 1.00 (0.96, 1.05) ≥ 45.3 µg/m^3: 0.98 (0.93, 1.03) Multipollutant model: 2 <distance ≤ 4 mile Per 10 µg/m^3: 1.00 (0.98, 1.02) 33.1 to <45.3 µg/m^3: 1.01 (0.96, 1.05) ≥ 45.3 µg/m^3: 0.98 (0.92, 1.04) **Zip-code-level analysis** Single-pollutant model: Per 10 µg/m^3: 1.02 (0.99, 1.04) 32.1 to <44.3 µg/m^3: 1.01 (0.95, 1.07) ≥ 44.3 µg/m^3: 1.04 (0.96, 1.12) Multipollutant model: Per 10 µg/m^3: 1.02 (0.99, 1.06) 32.1 to <44.3 µg/m^3: 1.02 (0.95, 1.09) ≥ 44.3 µg/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}.
Reference: Woodruff et al. (1997, 084271) **Period of Study:** 1989-1991 **Location:** 86 Metropolitan Statistical Areas in the U.S. (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 mo of life Infant's month and yr 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 mo 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 µg/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 µg/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:** U.S. 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, yr 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 yr 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 mo 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 avg 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 µg/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 U.S. 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 mo 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) 2nd trimester (31.45, 139.13) 3rd trimester (23.45, 172.75) **Monitoring Stations**: 27 **Copollutant:** CO SO_2	**PM Increment:** Trimester ≥ 90th percentile compared to <90th percentile Least-square (ANCOVA) mean (SE) **All Genotypes** 1st trimester <90th percentile, N(%): 158 (90.3%): 3253 (37) ≥ 90th percentile, N(%): 17 (9.7%): 2841 (145) P-Value for mean birth weight for ≥ 90th percentile PM_{10} vs. for <90th percentile 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 percentile, N(%): 153 (89.5%): 3253 (39) ≥ 90th percentile, N(%): 18 (10.5%): 3026 (157) p-Value for mean birth weight for ≥ 90th percentile PM_{10} vs. for <90th percentile 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 percentile, N(%):

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
		NO_2	162 (90.5%): 3226 (38) ≥ 90th percentile, N(%): 17 (9.5%): 3122 (140) P-Value for mean birth weight for ≥ 90th percentile PM_{10} vs. for <90th percentile 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 percentile, N(%): 60 (34.3%): 3350 (64) ≥ 90th percentile, N(%): 5 (2.9%): 3001 (229) P-Value for mean birth weight for ≥ 90th percentile PM_{10} vs. for <90th percentile 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 percentile, N(%): 59 (34.5%): 3335 (66) ≥ 90th percentile, N(%): 6 (3.5%): 3281 (249) p-Value for mean birth weight for ≥ 90th percentile PM_{10} vs. for <90th percentile 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 percentile, N(%): 61 (34.1%): 3327 (65) ≥ 90th percentile, N(%): 6 (3.4%): 3227 (300) P-Value for mean birth weight for ≥ 90th percentile PM_{10} vs. for <90th percentile 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 percentile, N(%): 98 (56.0%): 3193 (48) ≥ 90th percentile, N(%): 12 (6.9%): 2799 (169) P-Value for mean birth weight for ≥ 90th percentile PM_{10} vs. for <90th percentile 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 percentile, N(%): 94 (55.0%): 3200 (52) ≥ 90th percentile, N(%): 12 (7.0%): 2933 (176) P-Value for mean birth weight for ≥ 90th percentile PM_{10} vs. for <90th percentile 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 percentile, N(%): 101 (56.4%): 3165 (49) ≥ 90th percentile, N(%): 11 (6.2%): 3087 (147) P-Value for mean birth weight for

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			≥ 90th percentile PM_{10} vs. for <90th percentile 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 percentile, N(%): 87 (49.7%): 3244 (52) ≥ 90th percentile, N(%): 7 (4.0%): 2983 (232) P-Value for mean birth weight for ≥ 90th percentile PM_{10} vs. for <90th percentile 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 percentile, N(%): 82 (48.0%): 3243 (55) ≥ 90th percentile, N(%): 11 (6.4%): 3185 (207) P-Value for mean birth weight for ≥ 90th percentile PM_{10} vs. for <90th percentile 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 percentile, N(%): 90 (50.3%): 3239 (53) ≥ 90th percentile, N(%): 9 (5.0%): 2944 (198) P-Value for mean birth weight for ≥ 90th percentile PM_{10} vs. for <90th percentile 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 percentile, N(%): 71 (40.6%): 3262 (56) ≥ 90th percentile, N(%): 10 (5.7%): 2773 (171) P-Value for mean birth weight for ≥ 90th percentile PM_{10} vs. for <90th percentile 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 percentile, N(%): 71 (41.5%): 3264 (61) ≥ 90th percentile, N(%): 7 (4.1%): 2862 (208) P-Value for mean birth weight for ≥ 90th percentile PM_{10} vs. for <90th percentile 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 percentile, N(%): 72 (40.2%): 3207 (58) ≥ 90th percentile, N(%): 8 (4.5%): 3262 (180) P-Value for mean birth weight for ≥ 90th percentile PM_{10} vs. for <90th percentile PM_{10} Adjusted: 0.777 Adjusted, with CO: 0.607

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			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 yr **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 µg/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 µg/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, 088668) **Period of Study:** 1994-2000 **Location:** Los Angeles County, California, U.S.	**Outcome:** Term low birth weight (LBW) (<2500 g at ≥ 37 completed wk gestation), Vaginal birth <37 completed wk gestation **Age Groups:** LBW: ≥ 37 completed wk Preterm births: <37 completed wk **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 h (every 6 days) Entire pregnancy Trimesters of pregnancy Months of pregnancy 6 wk before birth **Mean (SD):** First trimester: 42.2 Third trimester: 41.5 6 wk before birth: 39.1 **Range (Min, Max):** First trimester: 26.3, 77.4 Third trimester: 25.7, 74.6 6 wk 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 wk 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 µg/m^3 2) 3 levels: a) <25 percentile (reference) b) 25%-75 percentile c) ≥ 75 percentile **Incidence of LBW (third trimester exposure)** <32.8 µg/m^3: 2.0 (1.8, 2.2) 32.8 to <43.4 µg/m^3: 2.0 (1.9, 2.1) ≥ 43.4 µg/m^3: 2.2 (2.0, 2.4) **Incidence of preterm birth (first trimester exposure)** <32.9 µg/m^3: 8.7 (8.3, 9.2) 32.9 to <43.9 µg/m^3: 8.8 (8.5, 9.1) ≥ 43.9 µg/m^3: 8.6 (8.1, 9.0) **Incidence of preterm birth (6 wk before birth exposure)** <31.8 µg/m^3: 8.8 (8.4, 9.3) 31.8 to <44.1 µg/m^3: 8.6 (8.3, 8.9) ≥ 44.1 µg/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 µg/m^3: 1.22 (1.05, 1.41) 33.4 to <44.7 µg/m^3: 1.08 (0.76, 1.52) ≥ 44.7 µg/m^3: 1.48 (1.00, 2.19) Multipollutant model: Distance ≤ 1 mile Per 10 µg/m^3: 1.36 (1.12, 1.65) 33.4 to <44.7 µg/m^3: 1.16 (0.77, 1.74) ≥ 44.7 µg/m^3: 1.58 (0.95, 2.62) Single-pollutant model: 1 <distance ≤ 2 mile Per 10 µg/m^3: 0.98 (0.90, 1.06) 33.4 to <44.7 µg/m^3: 0.95 (0.80, 1.13) ≥ 44.7 µg/m^3: 0.96 (0.78, 1.18) Multipollutant model: 1 <distance ≤ 2 mile Per 10 µg/m^3: 1.02 (0.92, 1.14) 33.4 to <44.7 µg/m^3: 0.93 (0.77, 1.12) ≥ 44.7 µg/m^3: 1.02 (0.79, 1.32) Single-pollutant model: 2 <distance ≤ 4 mile Per 10 µg/m^3: 1.03 (0.99, 1.08) 33.9 to <45.0 µg/m^3: 1.04 (0.96, 1.14) ≥ 45.0 µg/m^3: 1.08 (0.97, 1.20) Multipollutant model: 2 <distance ≤ 4 mile Per 10 µg/m^3: 1.04 (0.98, 1.09) 33.9 to <45.0 µg/m^3: 1.02 (0.92, 1.12) ≥ 45.0 µg/m^3: 1.06 (0.93, 1.21) **Zip-code-level analysis** Single-pollutant model:

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			Per 10 µg/m^3: 1.03 (0.97, 1.09) 33.2 to <43.6 µg/m^3: 0.98 (0.86, 1.11) ≥ 43.6 µg/m^3: 1.03 (0.88, 1.21) Multipollutant model: Per 10 µg/m^3: 1.07 (0.99, 1.15) 33.2 to <43.6 µg/m^3: 0.97 (0.85, 1.12) ≥ 43.6 µg/m^3: 1.09 (0.90, 1.31) **Outcome: LBW** **Exposure Period: Entire pregnancy period** **Address-level analysis:** Multipollutant model: Per 10 µg/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 µg/m^3: 1.00 (0.93, 1.09) 33.3 to <45.1 µg/m^3: 1.07 (0.90, 1.26) ≥ 45.1 µg/m^3: 1.12 (0.91, 1.38) Multipollutant model: Distance ≤ 1 mile Per 10 µg/m^3: 1.00 (0.90, 1.12) 33.3 to <45.1 µg/m^3: 1.12 (0.92, 1.36) ≥ 45.1 µg/m^3: 1.17 (0.90, 1.50) Single-pollutant model: 1 <distance ≤ 2 mile Per 10 µg/m^3: 1.01 (0.97, 1.05) 33.7 to <45.3 µg/m^3: 1.03 (0.95, 1.12) ≥ 45.3 µg/m^3: 1.07 (0.97, 1.19) Multipollutant model: 1 <distance ≤ 2 mile Per 10 µg/m^3: 1.04 (0.99, 1.10) 33.7 to <45.3 µg/m^3: 1.07 (0.98, 1.17) ≥ 45.3 µg/m^3: 1.13 (1.00, 1.27) Single-pollutant model: 2 <distance ≤ 4 mile Per 10 µg/m^3: 1.01 (0.99, 1.03) 34.1 to <45.5 µg/m^3: 1.03 (0.99, 1.08) ≥ 45.5 µg/m^3: 1.02 (0.96, 1.07) Multipollutant model: 2 <distance ≤ 4 mile Per 10 µg/m^3: 0.99 (0.97, 1.02) 34.1 to <45.5 µg/m^3: 0.99 (0.95, 1.04) ≥ 45.5 µg/m^3: 0.94 (0.89, 1.01) **Zip-code-level analysis** Single-pollutant model: Per 10 µg/m^3: 0.99 (0.96, 1.01) 33.3 to <44.2 µg/m^3: 1.01 (0.95, 1.08) ≥ 44.2 µg/m^3: 0.98 (0.90, 1.05) Multipollutant model: Per 10 µg/m^3: 0.99 (0.96, 1.03) 33.3 to <44.2 µg/m^3: 1.03 (0.97, 1.11) ≥ 44.2 µg/m^3: 1.01 (0.92, 1.11) **Outcome: Preterm birth** **Exposure Period: 6 wk before birth** **Address-level analysis:** Single-pollutant model: Distance ≤ 1 mile Per 10 µg/m^3: 1.02 (0.95, 1.10) 32.5 to <44.8 µg/m^3: 1.09 (0.92, 1.29) ≥ 44.8 µg/m^3: 1.12 (0.92, 1.37) Multipollutant model: Distance ≤ 1 mile Per 10 µg/m^3: 1.06 (0.97, 1.16) 32.5 to <44.8 µg/m^3: 1.09 (0.90, 1.31) ≥ 44.8 µg/m^3: 1.17 (0.91, 1.49) Single-pollutant model: 1 <distance ≤ 2 mile Per 10 µg/m^3: 1.00 (0.96, 1.03) 32.3 to <45.3 µg/m^3: 0.99 (0.91, 1.07) ≥ 45.3 µg/m^3: 0.99 (0.89, 1.10) Multipollutant model:

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			1 <distance ≤ 2 mile Per 10 µg/m^3: 1.01 (0.97, 1.06) 32.3 to <45.3 µg/m^3: 1.00 (0.92, 1.10) ≥ 45.3 µg/m^3: 1.02 (0.91, 1.16) Single-pollutant model: 2 <distance ≤ 4 mile Per 10 µg/m^3: 0.99 (0.98, 1.01) 33.1 to <45.3 µg/m^3: 1.00 (0.96, 1.05) ≥ 45.3 µg/m^3: 0.98 (0.93, 1.03) Multipollutant model: 2 <distance ≤ 4 mile Per 10 µg/m^3: 1.00 (0.98, 1.02) 33.1 to <45.3 µg/m^3: 1.01 (0.96, 1.05) ≥ 45.3 µg/m^3: 0.98 (0.92, 1.04) **Zip-code-level analysis** Single-pollutant model: Per 10 µg/m^3: 1.02 (0.99, 1.04) 32.1 to <44.3 µg/m^3: 1.01 (0.95, 1.07) ≥ 44.3 µg/m^3: 1.04 (0.96, 1.12) Multipollutant model: Per 10 µg/m^3: 1.02 (0.99, 1.06) 32.1 to <44.3 µg/m^3: 1.02 (0.95, 1.09) ≥ 44.3 µg/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}.
Reference: Woodruff et al. (1997, 084271) **Period of Study:** 1989-1991 **Location:** 86 Metropolitan Statistical Areas in the U.S. (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 mo of life Infant's month and yr 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 mo 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 µg/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 µg/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:** U.S. 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, yr 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 yr 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 mo 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 avg 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 µg/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 U.S. 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 mo 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 yr **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 2 day period in the second trimester by Personal Environmental Monitoring Sampler (PEMS)	**PM Increment:** in 1 µg/m^3 and tertiles T1: <27.0 µg/m^3 T2: 27.0-46.2 µg/m^3 T3: ≥ 46.2 µg/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]
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)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			$NSPM_{10}$: 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) 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 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 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 SO_4^{2-}: -0.0177 (0.0509) $NSPM_{10}$: 0.0128 (0.0119) Associations with SIDS by smoking status Smoking status: Yes Birth weight: Normal SO_4^{2-}: -0.0722 (0.0284) $NSPM_{10}$: 0.0206 (0.0071) Smoking status: No Birth weight: Normal SO_4^{2-}: -0.114 (0.021) $NSPM_{10}$: 0.0117 (0.005) Smoking status: Yes Birth weight: LBW SO_4^{2-}: -0.0958 (0.0483) $NSPM_{10}$: 0.0345 (0.0125) Smoking status: No Birth weight: LBW 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 SO_4^{2-}: 0.43 (0.37, 0.51) $NSPM_{10}$: 1.33 (1.18, 1.50)

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 µg/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 Fig. 6 and 7
Reference: Loomis et al. (1999, 087288) **Period of Study:** Jan 1993-Jul 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:** 1 **Copollutant:** O_3 NO_2 NO NO_X SO_2 **Notes:** Pearson correlation coefficients ranging from 0.52 to 0.71	**PM Increment:** 10 µg/m^3 **Effect Estimate [Lower CI, Upper CI]:** %Change in infant mortality Lags 0-5 (single day) presented in Fig 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 2 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) 3 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) 4 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:** Jan 1998-Dec 2000 **Location:** metropolitan Sydney, Australia	**Outcome:** Risk of small for gestational age (SGA) and birth weight **Age Groups:** All singleton births >20 wk 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 µg/m^3 Risk of SGA All births 1 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 1 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 1 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 1 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)
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 wk 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 µg/m^3| Referent **PM Increment:** 11.9-13.9 µg/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 µg/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 µg/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, 156846) **Period of Study:** 2001-2003 **Location:** U.S.	**Outcome:** Low birth weight **Study Design:** Cohort **N:** 785,965 Singleton births delivered at 40 wk 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 mo **Mean (SD):** 14.5 25th: 12.1 75th: 17.6 **Copollutant (correlation):** SO_2, NO_2 CO O_3	**PM Increment:** 10 µg/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)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
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 yr 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:** 24 h **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 µg/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 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 2003-Dec 2003 **Location:** Los Angeles, California	**Outcome:** Preterm births (infants delivered before 37 wk) **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 wk 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:** Jan 1998-Jan 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 $PM_{2.5}$ (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 Increment:** 1) 1 μg/m^3 2) Quartiles: a) 1st (reference) (7.2-13.5 μg/m^3) b) 2nd (13.5-14.4 μg/m^3) c) 3rd (14.4-15.4 μg/m^3) day) 4th (15.41-17.5 μg/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 μg/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 μg/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 μg/m^3: 1.07 (0.91, 1.26) **Single-pollutant models** (restricted analysis to $PM_{2.5}$ absorbance below the median) Per 1 μg/m^3: 1.15 (0.89, 1.52) **Prevalence ratios (PRs) of birth weight <3000 g** **Multipollutant models (simultaneous**

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
		$PM_{2.5}$ (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	**Adjustment of 3rd trimester $PM_{2.5}$ and whole pregnancy $PM_{2.5}$)** $PM_{2.5}$ (whole pregnancy) Per 1 µg/m^3: 0.96 (0.75, 1.19) $PM_{2.5}$ (3rd trimester) Per 1 µg/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 µg/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 µg/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 µg/m^3: 1.03 (0.90, 1.17) **Prevalence ratios (PRs) of birth weight <3000 g during exposure over second trimester of pregnancy** 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 mo after birth** Per 1 µg/m^3: 7% (-7%, 22%)
Reference: (Slama et al., 2007, 093216) **Period of Study:** Jan 1998-Jan 1999	**Outcome:** Birth weight offspring at term **Study Design:** Cohort study	**Pollutant:** $PM_{2.5}$ absorbance (estimated) **Averaging Time:** Entire pregnancy period and trimesters	**PM Increment:** 1) 0.5 * 10-5/m 2) Quartiles: a) 1st (reference) (1.29-1.61) b) 2nd (1.61-1.72)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Location: Munich, Germany	**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	**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.): -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	c) 3rd (1.72-1.89) day) 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) **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);

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			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 mo 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 wk gestation) Vaginal birth <37 completed wk gestation **Age Groups:** LBW: ≥ 37 completed wk Preterm births: <37 completed wk **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:** 24 h (every 3 days) Entire pregnancy Trimesters of pregnancy Months of pregnancy 6 wk before birth **Mean (SD):** First trimester: 21.9 Third trimester: 21.0 6 wk before birth: 21.0 **Range (Min, Max):** First trimester: 11.8-38.9 Third trimester: 11.8-.38.9 6 wk 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 wk 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	**PM Increment:** 1) 10 µg/m^3 2) 3 levels: a) <25 percentile (reference) b) 25%-75 percentile c) ≥ 75 percentile **Incidence of LBW (third trimester exposure)** <17.1 µg/m^3: 2.4 (2.0, 2.8) 17.1 to <24.0 µg/m^3: 2.2 (2.0, 2.5) ≥ 24.0 µg/m^3: 2.1 (1.7, 2.4) **Incidence of preterm birth (first trimester exposure)** <18.0 µg/m^3: 10.6 (9.6, 11.7) 18.0 to <25.4 µg/m^3: 8.8 (8.1, 9.5) ≥ 25.4 µg/m^3: 9.0 (8.1, 10.0) **Incidence of preterm birth (6 wk before birth exposure)** <16.5 µg/m^3: 8.2 (7.4, 9.1) 16.5 to <24.7 µg/m^3: 8.8 (8.2, 9.4) ≥ 24.7 µg/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 µg/m^3: 0.85 (0.70, 1.02) 18.1 to <25.2 µg/m^3: 0.91 (0.72, 1.16) ≥ 25.2 µg/m^3: 0.83 (0.60, 1.14) Single-pollutant model: 1 <distance ≤ 2 mile Per 10 µg/m^3: 0.85 (0.74, 0.99) 18.3 to <25.2 µg/m^3: 0.81 (0.69, 0.94)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			≥ 25.2 µg/m^3: 0.79 (0.65, 0.97) Multipollutant model1 <distance ≤ 2 mile Per 10 µg/m^3: 1.18 (0.84, 1.65) Single-pollutant model: 2 <distance ≤ 4 mile Per 10 µg/m^3: 0.83 (0.78, 0.88) 18.5 to <24.9 µg/m^3: 0.79 (0.74, 0.85) ≥ 24.9 µg/m^3: 0.76 (0.70, 0.84) **Zip-code-level analysis:** Single-pollutant model: Per 10 µg/m^3: 0.73 (0.67, 0.80) 18.0 to <25.4 µg/m^3: 0.70 (0.61, 0.80) ≥ 25.4 µg/m^3: 0.64 (0.53, 0.76) **Outcome: Preterm birth** **Exposure Period: 6 wk before birth** **Address-level analysis:** Single-pollutant model: Distance ≤ 1 mile Per 10 µg/m^3: 1.09 (0.91, 1.30) 16.8 to <24.1 µg/m^3: 1.21 (0.97, 1.51) ≥ 24.1 µg/m^3: 1.25 (0.93, 1.68) Single-pollutant model: 1 <distance ≤ 2 mile Per 10 µg/m^3: 1.08 (0.97, 1.21) 17.2 to <24.5 µg/m^3: 0.94 (0.82, 1.08) ≥ 24.5 µg/m^3: 1.04 (0.87, 1.24) Single-pollutant model: 2 <distance ≤ 4 mile Per 10 µg/m^3: 1.05 (0.99, 1.10) 17.3 to <24.6 µg/m^3: 1.06 (1.00, 1.13) ≥ 24.6 µg/m^3: 1.08 (0.99, 1.17) **Zip-code-level analysis** Single-pollutant model: Per 10 µg/m^3: 1.10 (1.00, 1.21) 16.5 to <24.7 µg/m^3: 1.06 (0.94, 1.20) ≥ 24.7 µg/m^3: 1.19 (1.02, 1.40) **(See Notes)** Multipollutant model Per 10 µg/m^3: 1.12 (0.90, 1.40) ≥ 24.6 µg/m^3: 1.12 (0.82, 1.52) **Notes:** In the table, the 75 percentile is noted as 24.7 µg/m^3. However, the text notes the 75 percentile as 24.3 µg/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 µg/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 $PM_{2.5}$ avgd over all monitors in county) Adjusted: 2.28 (0.94, 5.52) Respiratory (averaging all $PM_{2.5}$ measurements in county over the 2-yr 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:** U.S. 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, yr 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 yr 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 mo 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 avg 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:** Oct 1991-Mar 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 µg/m^3 2 (9/1/1995-2/28/1998): 41.1 ± 28.4 µg/m^3 3 (3/1/1998-3/31/2002): 24.3 ± 15.4 µg/m^3 Total: 38.0 ±28.3 µg/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 Ma, 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 Ma, 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:** Jan 1998-Jan 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 Increment:** 1) 1 µg/m^3 2) Quartiles: a) 1st (reference) (7.2-13.5 µg/m^3) b) 2nd (13.5-14.4 µg/m^3) c) 3rd (14.4-15.4 µg/m^3) day) 4th (15.41-17.5 µg/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 µg/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 µg/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 µg/m^3: 1.07 (0.91, 1.26) **Single-pollutant models** (restricted

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
		$PM_{2.5}$ (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	Analysis to $PM_{2.5}$ absorbance below the median) Per 1 µg/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 µg/m^3: 0.96 (0.75, 1.19) $PM_{2.5}$ (3rd trimester) Per 1 µg/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 µg/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 µg/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 µg/m^3: 1.03 (0.90, 1.17) **Prevalence ratios (PRs) of birth weight <3000 g during exposure over second trimester of pregnancy** 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 mo after birth**

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			Per 1 µg/m^3: 7% (-7%, 22%)
Reference: (Slama et al., 2007, 093216) **Period of Study:** Jan 1998-Jan 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.): -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.): -	**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) day) 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) **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);

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
		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	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 mo 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 wk gestation) Vaginal birth <37 completed wk gestation **Age Groups:** LBW: ≥ 37 completed wk Preterm births: <37 completed wk **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:** 24 h (every 3 days) Entire pregnancy Trimesters of pregnancy Months of pregnancy 6 wk before birth **Mean (SD):** First trimester: 21.9 Third trimester: 21.0 6 wk before birth: 21.0 **Range (Min, Max):** First trimester: 11.8-38.9 Third trimester: 11.8-.38.9 6 wk 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 wk before birth:	**PM Increment:** 1) 10 µg/m^3 2) 3 levels: a) <25 percentile (reference) b) 25%-75 percentile c) ≥ 75 percentile **Incidence of LBW (third trimester exposure)** <17.1 µg/m^3: 2.4 (2.0, 2.8) 17.1 to <24.0 µg/m^3: 2.2 (2.0, 2.5) ≥ 24.0 µg/m^3: 2.1 (1.7, 2.4) **Incidence of preterm birth (first trimester exposure)** <18.0 µg/m^3: 10.6 (9.6, 11.7) 18.0 to <25.4 µg/m^3: 8.8 (8.1, 9.5) ≥ 25.4 µg/m^3: 9.0 (8.1, 10.0) **Incidence of preterm birth (6 wk before birth exposure)** <16.5 µg/m^3: 8.2 (7.4, 9.1) 16.5 to <24.7 µg/m^3: 8.8 (8.2, 9.4) ≥ 24.7 µg/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 µg/m^3: 0.85 (0.70, 1.02) 18.1 to <25.2 µg/m^3:

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
		$PM_{2.5}$-CO: 0.63 $PM_{2.5}$-NO_2: 0.74 $PM_{2.5}$-O_3: -0.60 $PM_{2.5}$-PM_{10}: 0.60	0.91 (0.72, 1.16) ≥ 25.2 µg/m^3: 0.83 (0.60, 1.14) Single-pollutant model: 1 <distance ≤ 2 mile Per 10 µg/m^3: 0.85 (0.74, 0.99) 18.3 to <25.2 µg/m^3: 0.81 (0.69, 0.94) ≥ 25.2 µg/m^3: 0.79 (0.65, 0.97) Multipollutant model1 <distance ≤ 2 mile Per 10 µg/m^3: 1.18 (0.84, 1.65) Single-pollutant model: 2 <distance ≤ 4 mile Per 10 µg/m^3: 0.83 (0.78, 0.88) 18.5 to <24.9 µg/m^3: 0.79 (0.74, 0.85) ≥ 24.9 µg/m^3: 0.76 (0.70, 0.84) **Zip-code-level analysis:** Single-pollutant model: Per 10 µg/m^3: 0.73 (0.67, 0.80) 18.0 to <25.4 µg/m^3: 0.70 (0.61, 0.80) ≥ 25.4 µg/m^3: 0.64 (0.53, 0.76) **Outcome: Preterm birth** **Exposure Period: 6 wk before birth** **Address-level analysis:** Single-pollutant model: Distance ≤ 1 mile Per 10 µg/m^3: 1.09 (0.91, 1.30) 16.8 to <24.1 µg/m^3: 1.21 (0.97, 1.51) ≥ 24.1 µg/m^3: 1.25 (0.93, 1.68) Single-pollutant model: 1 <distance ≤ 2 mile Per 10 µg/m^3: 1.08 (0.97, 1.21) 17.2 to <24.5 µg/m^3: 0.94 (0.82, 1.08) ≥ 24.5 µg/m^3: 1.04 (0.87, 1.24) Single-pollutant model: 2 <distance ≤ 4 mile Per 10 µg/m^3: 1.05 (0.99, 1.10) 17.3 to <24.6 µg/m^3: 1.06 (1.00, 1.13) ≥ 24.6 µg/m^3: 1.08 (0.99, 1.17) **Zip-code-level analysis** Single-pollutant model: Per 10 µg/m^3: 1.10 (1.00, 1.21) 16.5 to <24.7 µg/m^3: 1.06 (0.94, 1.20) ≥ 24.7 µg/m^3: 1.19 (1.02, 1.40) **(See Notes)** Multipollutant model Per 10 µg/m^3: 1.12 (0.90, 1.40) ≥ 24.6 µg/m^3: 1.12 (0.82, 1.52) **Notes:** In the table, the 75 percentile is noted as 24.7 µg/m^3. However, the text notes the 75 percentile as 24.3 µg/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 h (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 $PM_{2.5}$ avgd over all monitors in county) Adjusted: 2.28 (0.94, 5.52) Respiratory (averaging all $PM_{2.5}$ measurements in county over the 2-yr 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:** U.S. 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, yr 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 yr 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 mo 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 avg 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 µg/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 µg/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:** Oct 1991-Mar 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 µg/m^3 2 (9/1/1995-2/28/1998): 41.1 ± 28.4 µg/m^3 3 (3/1/1998-3/31/2002): 24.3 ± 15.4 µg/m^3 Total: 38.0 ±28.3 µg/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 Ma, 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 Ma, 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)

[1]All units expressed in µg/m^3 unless otherwise specified.

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) **Period of Study:** 1973-1998 **Location:** San Francisco, San Diego, Los Angeles, CA	**Outcome:** Mortality: CHD **Study Design:** Cohort **Statistical Analyses:** Cox proportion hazards model **Age Groups:** >25	**Pollutant:** $PM_{10-2.5}$ **Averaging Time:** 25 yr **Mean (SD):** 25.4 **Range (Min, Max):** NR **Copollutant:** NO_2 O_3 SO_2	**Increment:** 10 µg/m^3 **Relative Risk (Lower CI, Upper CI) lag:** Males $PM_{10-2.5}$: 0.93 (0.68, 1.29) 0-1 $PM_{10-2.5}$+NO_2: 0.86 (0.62, 1.20) 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) **Period of Study:** 1999-2000 **Location:** United States	**Outcome:** Mortality **Study Design:** Cohort Study (Cystic Fibrosis Cohort) **Statistical Analyses:** Logistic Regression **Age Groups:** >6 yr	**Pollutant:** $PM_{2.5}$ **Averaging Time:** Annual avg **Mean (SD) unit:** $PM_{2.5}$: 13.7 (4.2) **IQR:** $PM_{2.5}$: 11.8-15.9 **Copollutant:** O_3 NO_2 SO_2 CO	**Increment:** 10 µg/m^3 $PM_{2.5}$: 1.32 (0.91-1.93)
Reference: Lipert et al**. (2009, 190271)** **Period of Study:** 1989-1996 **Location:** Various parts of the Untied States	**Outcome:** Mortality **Study Design:** Retrospective Cohort **Statistical Analyses:** Cox proportional hazards regression **Age Groups:** Male U.S. veterans between ages of 39 and 63 (Avg. age: 51)	**Pollutant:** $PM_{10-2.5}$ **Mean (SD):** 16.0 (5.1)	**Increment:** 12 1.07 (1.01, 1.13)
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 yr+	**Pollutant:** $PM_{10-2.5}$ **Averaging Time:** Monthly avg **Mean (SD):** $PM_{10-2.5}$: 27.3 (8.6) **IQR:** 9.7 **Copollutant:** O_3: 0.70 SO_2: 0.31 NO_2: 0.23 SO_4: 0.47	**Increment:** IQR All Cause: 1.05 (0.92-1.20) Resp: 1.19 (0.88, 1.62) Lung Cancer: 1.25 (0.63-2.49)

[1]All units expressed in µg/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 (Fig 1):** log HR for a 24.5 µg/m^3 increase in $PM_{2.5}$ over time Yr 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 (Fig 1):** Log HR for a 19.9 µg/m^3 increase in Sulfates over time Yr 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 µg/m^3 - 0.4 (0.1, 0.8) Reduction to 20 µg/m^3 - 0.8 (0.2, 1.6) Reduction to 15 µg/m^3 - 1.6 (0.4, 3.1) Reduction to 10 µg/m^3 - 3.0 (0.8, 5.8)
Reference: Beelen et al. (2008, 156263) **Period of Study:** 1987-1996 **Location:** Netherlands	**Outcome:** Mortality: Total (nonaccidental) (<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) µg/m^3 **Range (Min, Max):** (23.0, 36.8) **Copollutant (correlation):** NO_2: (>0.8) BS: (>0.8) SO_2: (>0.6)	**Increment:** 11 µg/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: F Ull 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:** Oct 1991-Mar 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 µg/m^3 2 (9/1/1995-2/28/1998): 41.1 ± 28.4 µg/m^3 3 (3/1/1998-3/31/2002): 24.3 ± 15.4 µg/m^3 Total: 38.0 ±28.3 µg/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 Ma, 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 Ma, 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) **Period of Study:** 1987-1996 **Location:** The Netherlands	**Outcome:** All cause mortality (ICD-9 400-440, 460-519, > 800) **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, saturated 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 yr at enrollment	**Pollutant:** $PM_{2.5}$, estimated from PM_{10} levelsf **Averaging Time:** 24 h **50th Percentile:** 28 µg/m^3 **Range (Min, Max):** 23-37 **Copollutant (correlation):** NO_2: 0.75 BS: 0.84 NO: 0.69 SO_2: 0.43	**Increment:** 10 µg/m^3 **Relative Risk (95 % CI) for $PM_{2.5}$ concentrations and cause specific mortality** Case Cohort Natural Cause: 0.86 (0.66-1.13) Cardiovascular: 0.83 (0.60-1.15) Respiratory: 1.02 (0.56-1.88) Lung Cancer: 0.87 (0.52-1.47) Noncardiopulmonary, non-lung cancer: 0.85 (0.65-1.23) Full Cohort Natural Cause: 1.06 (0.97-1.16) Cardiovascular: 1.04 (0.90-1.21) 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)
Reference: Chen et al. (2005, 087942) **Period of Study:** 1973-1998 **Location:** San Francisco, San Diego, Los Angeles, CA	**Outcome:** Mortality: CHD **Study Design:** Cohort **Statistical Analyses:** Cox proportion hazards model **Age Groups:** >25	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 25 yr **Mean (SD):** 29.0 **Range (Min, Max):** NR **Copollutant:** NO_2, O_3, SO_2	**Increment:** 10 µg/m^3 **Relative Risk (Lower CI, Upper CI) lag:** Males $PM_{2.5}$: 0.89 (0.69, 1.17) 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

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Eftim et al. (2008, 099104) **Period of Study:** 2000-2002 **Location:** USA, Same cities as six cities and ACS cohorts	**Outcome (ICD-9):** All nonaccidental causes (<800) **Study Design:** Cross-sectional **Statistical Analyses:** Log-linear regression, Poisson **Age Groups:** >65	**Pollutant:** $PM_{2.5}$ **Averaging Time:** Annual avg **Mean (SD): |** ACS: 13.6 (2.8) SCS: 14.1 (3.1) **Range (Min, Max):** ACS: (6.0, 25.1); SCS: (9.6, 19.1)	**Increment:** 10 µg/m^3 **% Increase in Mortality for overall exposure period and individual yr (95%CI Min, 95%CI Max):** ACS (adjusted for age, sex) Overall: 10.8 (8.6, 13.0) 2000: 10.9 (7.3, 14.6) 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)
Reference: Enstrom et al. (2005, 087356) **Period of Study:** 1973-2002 **Location:** 25 California Colonies 11 California Colonies (EPA IPN study)	**Outcome:** Mortality: Cardiovascular-respiratory (390-448) (480-486, 487, 490-496, 507) **Study Design:** Retrospective cohort **Statistical Analyses:** Cox proportional hazards regression model, SAS PHREG **Age Groups:** 35 or older	**Pollutant:** $PM_{2.5}$ **Averaging Time:** Annual **Mean (SD):** 23.4 **Range (Min, Max):** (13.1 µg/m^3, 36.1)	**Relative Risk (Lower CI, Upper CI)** **RR from causes for both sexes by county from 1973-2002** Alameda: 0.962 (0.926,0.999) Butte: 0.999 (0.910,1.096) 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)
Reference: Filleul et al. (2005, 087357) **Period of Study:** 1974-1976 **Location:** 7 cities in France	**Outcome:** Nonaccidental causes (<800), cardiopulmonary disease (401-440 and 460-519), lung cancer (162) **Age Groups:** 25-59 yr **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 µg/m^3 Adjusted mortality rate ratios: 24 areas: All nonaccidental 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 nonaccidental causes: 1.05[1.02, 1.08] Lung cancer: 1.00[0.92, 1.10] Cardiopulmonary disease: 1.06[1.01, 1.12]

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Fuentes et al. (2006, 097647) **Period of Study:** Jun 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 µg/m^3 $PM_{2.5}$: 1.066 (1.064, 1.069) PM_{10}: 1.030 (1.028, 1.032)
Reference: Janes et al. (2007, 090927) **Period of Study:** 2000-2002 **Location:** 113 U.S. counties	**Outcome:** Mortality: **Study Design:** Time-series **Statistical Analyses:** Cox proportional hazards model **Age Groups:** 65-74 75-84 85+	**Pollutant:** $PM_{2.5}$ **Averaging Time:** Annual avg **Mean (SD):** NR **Range (Min, Max):** NR	**Increment:** 1 µg/m^3 **% Increase (Lower CI, Upper CI) lag:** Overall % Increase by age-sex stratum Age Category 65-74: Male: 1.48 (0.93,2.03) Female: 0.83 (0.24,1.43) 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) **Period of Study:** 1982 **Location:** 151 cities from ACS	**Outcome:** Mortality **Study Design:** Multilevel, individual-ecologic analysis **Statistical Analysis:** Cox proportional hazards model **Covariates:** Smoking, education, occupational exposures, BMI, marital status, alcohol consumption, gender	**Pollutant:** Sulfates **Mean (SD):** 10.6 **Range (Min, Max):** 3.6,23.5	**Increment:** 19.9 (Range) All Cause: SO_4: 1.17 (1.07, 1.27) SO_4 + CO: 1.16 (1.10, 1.23) SO_4 + NO_2: 1.16 (1.08, 1.24) 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 µg/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) **Period of Study:** 1974-1998 Period 1: 1974-1989 Period 2: 1990-1998 **Location:** Nine U.S. Cities Watertown, MA Kingston, TN Harriman, TN St. Louis, MO Steubenville, OH Portage, WI |Wyocena, WI Pardeeville, WI Topeka, KS	**Outcome:** Total mortality Nonaccidental (<800) Cardiovascular (400-440) Respiratory (485-496) Lung Cancer (162) Other **Study Design:** Prospective Cohort **Statistical Analyses:** Cox proportional hazards regression **Age Groups:** 25-74	**Pollutant:** $PM_{2.5}$ **Averaging Time:** Annual avg **Mean (SD):** Period 1 Portage: 11.4 Topeka: 12.4 Watertown: 15.4 Harriman: 20.9 St Louis: 19.2 Steubenville: 29.0 Period 2 Portage: 10.2 Topeka: 13.1 Watertown: 12.1 Harriman: 18.1 St. Louis: 13.4 Steubenville: 22.0	**Increment:** 10 µg/m^3 **Relative Risk (Lower CI, Upper CI) lag:** **Period 1:** Portage: 1.00 Topeka: 1.06 (0.86, 1.31) Watertown: 1.06 (0.87, 1.28) Harriman: 1.19 (0.98, 1.44) St Louis: 1.15 (0.96, 1.38) Steubenville: 1.31 (1.10, 1.57) **Period 2:** Portage: NR Topeka: 1.01 (0.83, 1.22) Watertown: 0.82 (0.67, 1.00) Harriman: 1.10 (0.91, 1.33) St Louis: 0.96 (0.80, 1.15) Steubenville: 1.06 (0.89, 1.27) **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 1 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 2 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) **Period of Study:** 1989-1996 **Location:** Various parts of the Untied States	**Outcome:** Mortality **Study Design:** Retrospective Cohort **Statistical Analyses:** Cox proportional hazards regression **Age Groups:** Male U.S. veterans between ages of 39 and 63 (Avg. age: 51)	**Pollutant:** Sulfate **Mean (SD) from 1976-81:** 10.7 (3.6)	**Increment:** 8 1.045 (0.944, 1.157)
Reference: Lipfert et al. (2006, 088756) **Period of Study:** 1989-1996 **Location:** Various parts of the Untied States	**Outcome:** Mortality **Study Design:** Retrospective Cohort **Statistical Analyses:** Cox proportional hazards regression **Age Groups:** Male U.S. veterans between ages of 39 and 63 (Avg age 51)	**Pollutant:** $PM_{2.5}$ **Mean (SD):** 14.3 (3.2)	**Increment:** 8 1.118 (1.038, 1.203)

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 U.S. veterans between ages of 39 and 63 (Avg. age: 51)	**Pollutant:** $PM_{2.5}$ **Averaging Time:** Annual avg **Mean (SD):** 15.02 (4.80) µg/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 µg/m^3 **% Increase per 10 µg/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 U.S.	**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 yr	**Pollutant:** $PM_{2.5}$ **Averaging Time:** NR **Mean Unit:** 1979-1983: 21.20 µg/m^3 1999-2000: 14.02 µg/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 µg/m^3 **Hazard Ratio (95% CI)** **MSA & DIFF** Increment Change: 10.78 (1.043-1.115) Change 5-15 µg/m^3: 1.128 (1.077-1.183) Change 10-20 µg/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-yr 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 µg/m^3 **HR (95% CI)** **28 County, 3-yr model** All 7 ecologic covariates: 0.977 (0.932-1.025)
Reference: Krewski et al. (2009, 191193) **Period of Study:** 1979-2000 **Location:** 48 contiguous states U.S.	**Outcome:** Death from cardiopulmonary 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 yr	**Pollutant:** $PM_{2.5}$ **Averaging Time:** NR **Mean Unit:** 1979-1983: 21.20 µg/m^3 1999-2000: 14.02 µg/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 µg/m^3 **Hazard Ratio (95% CI)** **MSA & DIFF** Increment Change: 1.078 (1.077-1.182) Change 5-15 µg/m^3: 1.208 (1.132-1.290) Change 10-20 µg/m^3: 1.127 (1.081-1.174) **HR (95% CI)** **Los Angeles** Parsimonious ecologic covariates: 1.086 (0.939-1.285) **HR (95% CI)** **15-yr 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 µg/m^3 **HR (95% CI)** **28 County, 3-yr 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) **Period of Study:** 1979-2000 **Location:** 48 contiguous states U.S.	**Outcome:** Death from ischemic heart 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 yr	**Pollutant:** $PM_{2.5}$ **Averaging Time:** NR **Mean Unit:** 1979-1983: 21.20 µg/m^3 1999-2000: 14.02 µg/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 µg/m^3 **Hazard Ratio (95% CI)** **MSA & DIFF** Increment Change: 1.196 (1.177-1.407) Change 5-15 µg/m^3: 1.484 (1.311-1.680) Change 10-20 µg/m^3: 1.283 (1.186-1.387) **HR (95% CI)** **Los Angeles** 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 µg/m^3 **HR (95% CI)** **28 County, 3 yr model** All 7 ecologic covariates: 1.072 (0.980-1.172)
Reference: Krewski et al. (2009, 191193) **Period of Study:** 1979-2000 **Location:** 48 contiguous states U.S.	**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 yr	**Pollutant:** $PM_{2.5}$ **Averaging Time:** NR **Mean Unit:** 1979-1983: 21.20 µg/m^3 1999-2000: 14.02 µg/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 µg/m^3 **Hazard Ratio (95% CI)** **MSA & DIFF** Increment Change: 1.142 (1.057-1.234) Change 5-15 µg/m^3: 1.236 (1.114-1.372) Change 10-20 µg/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-yr 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 µg/m^3 **HR (95% CI)** **28 County, 3-yr 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 U.S.	**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 yr	**Pollutant:** $PM_{2.5}$ **Averaging Time:** NR **Mean Unit:** 1979-1983: 21.20 µg/m^3 1999-2000: 14.02 µg/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 µg/m^3 **HR (95% CI)** **28 County, 3 yr 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 U.S.	**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 yr	**Pollutant:** $PM_{2.5}$ **Averaging Time:** NR **Mean Unit:** 1979-1983: 21.20 µg/m^3 1999-2000: 14.02 µg/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 µg/m^3 **HR (95% CI)** **28 County, 3-yr 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 U.S.	**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 yr	**Pollutant:** $PM_{2.5}$ **Averaging Time:** NR **Mean Unit:** 1979-1983: 21.20 µg/m^3 1999-2000: 14.02 µg/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 µg/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 U.S.	**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 yr	**Pollutant:** $PM_{2.5}$ **Averaging Time:** NR **Mean Unit:** 1979-1983: 21.20 µg/m^3 1999-2000: 14.02 µg/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 µg/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 U.S.	**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 yr	**Pollutant:** $PM_{2.5}$ **Averaging Time:** NR **Mean Unit:** 1979-1983: 21.20 µg/m^3 1999-2000: 14.02 µg/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 µg/m^3 **Hazard Ratio (95% CI)** **MSA & DIFF** Increment Change: 1.010 (0.968-1.055) Change 5-15 µg/m^3: 1.026 (0.970-1.085) Change 10-20 µg/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)
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 yr+	**Pollutant:** $PM_{2.5}$ **Averaging Time:** Monthly avg **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)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Miller et al. (2007, 090130) **Period of Study:** 1994-1998 **Location:** 36 U.S. 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 µg/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)
Reference: Naess et al. (2007, 090736) **Period of Study:** 1992-1998 **Location:** Oslo, Norway	**Outcome:** Mortality: Nonaccidental (<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-yr 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 µg/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 µg/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 µg/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 µg/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 µg/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 **Location:** Oslo, Norway	**Outcome:** Mortality: Lung cancer (162) COPD (490-496) 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	**Pollutant:** $PM_{2.5}$ **Averaging Time:** (Mo-yr) avg **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	**Relative Risk (CI min, CI max)** **RR on All-cause mortality of $PM_{2.5}$ in 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:

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			$PM_{2.5}$: 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: $M_{2.5}$: 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 $PM_{2.5}$ 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)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			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: 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 yr old nonsmoking adults	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 48-h avg **Mean Range:** 17 to 49 µg/m^3	**Increment:** 10 µg/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."
Reference: Pope et al. (2004, 055880) **Period of Study:** 1982-2000 **Location:** Metropolitan areas in all 50 states in the U.S.	**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 µg/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)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
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),
Reference: Pope et al. (2009, 190107) **Period of Study:** 1978-1982, 1997-2001 **Location:** 211 U.S. counties and 51 metropolitan areas	**Outcome:** Increased life expectancy **Study Design:** Cross-sectional **Statistical Analysis:** Cross-sectional regression **Age Groups:** Adults ≥45 yr	**Pollutant:** $PM_{2.5}$ **Averaging Time:** Daily, quarterly and annual **Mean (SD) Unit:** 1979-1983: 20.61 ± 4.36 µg/m^3 1999-2000: 14.10 ± 2.86 µg/m^3 **Range (Min, Max):** NR **Copollutant (correlation):** NR	**Increment:** 10 µg/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

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
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 (Dec-Feb) Summer (Jun-Aug) **Statistical Package:** S-Plus 6.1	**Pollutant:** $PM_{2.5}$ **Averaging Time:** NR **Mean (SD):** All yr: 17.0 (8.7) µg/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] 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) **Period of Study:** 2006 **Location:** U.S.	**Outcome:** Mortality **Study Design:** Expert Judgment Study **Statistical Analyses:** Standard best practices for expert elicitation	**Pollutant:** $PM_{2.5}$ **Averaging Time:** Annual avg **Mean (SD):** 4-30	Quantitative results are not presented in the text, but can be found graphically in Fig 3. "Most of the experts' central estimates fall at or above the 2002 ACS median (0.6% per µg/m^3) and below the original Six Cities median (1.2% per µg/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: Nonaccidental (<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 Fig 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 Fig 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 Fig 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
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)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Villeneuve et al. (2002, 042576) **Period of Study:** 1974-1991 **Location:** Six U.S. Cities: Steubenville, OH, St. Louis, MO, Portage, WI, Topeka, KS, Watertown, MA, Kingston/ Harriman, TN	**Outcome (ICD10):** Mortality: Nonaccidental (<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 µg/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 2 yr <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 yr before current yr <60: 1.35 (1.08, 1.67) >60: 1.08 (0.95, 1.22) Total: 1.14 (1.02, 1.27) Time dependent estimate of $PM_{2.5}$ received >5 yr before current yr <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:** U.S. 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 µg/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 U.S. Cities	**Outcome:** Mortality, all causes, excluding ICD codes S00-U99 **Study Design:** Time-series **Covariates:** Region, season	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean (SD)** Birmingham AL - 16.5 Phoenix AZ - 11.4 LittleRock AR - 14.3	**Increment:** 10 µg/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)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
	Statistical Analysis: Poisson regression **Age Groups:** All	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 Denver CO - 10.3 Hartford CT - 11.6 New Haven CT - 13.7 Wilmington DE - 15.1 Davie FL - 8.4 Miami FL - 9.4 Jacksonville FL - 10.6 Pensacola FL - 12.4 Tampa FL - 11.9 Orlando FL - 10.3 Palm beach FL - 7.9 Pinellas FL - 10.4 Atlanta GA - 17.6 Chicago IL - 15.9 Gary IN - 15.3 Indianapolis IN - 16.3 Cedar Rapids IA - 11.0 Des Moines IA - 10.5 Davenport IA - 12.3 Louisville KY - 15.9 Baton Rouge LA - 13.4 Avondale LA - 12.3 New Orleans LA - 12.6 Baltimore MD - 15.6 Springfield MA - 12.3 Boston MA - 12.4 Worcester MA - 11.3 Holland MI - 12.1 Grand Rapids MI - 13.6 Detroit MI - 16.2 Minneapolis MN - 11.1 Kansas MO - 12.0 St Louis MO - 14.5 Omaha NE - 10.4 Elizabeth NJ - 14.7 Albuquerque NM - 6.7 New York NY - 14.8 Bath NY - 9.6 Durham NC - 14.3 Winston NC - 14.7 Greensborough NC - 14.2 Charlotte NC - 15.3 Raleigh NC - 14.3 Middletown OH - 16.4 Youngstown OH - 15.6 Cleveland OH - 16.4 Columbus OH - 16.2 Cincinnati OH - 17.1 Steubenville OH - 17.0 Toledo OH - 14.9 Dayton OH - 16.2 Akron OH - 16.0 Warren OH - 15.3 Oklahoma OK - 9.9 Tulsa OK - 11.1 Bend OR - 7.8 Medford OR - 9.9 Klamath OR - 10.6 Eugene OR - 8.0 Portland OR - 8.8 Gettysburg PA - 13.4 Pittsburgh PA - 15.7 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	Winter: 0.56 (0.17-0.94) Spring: 2.57 (1.96-3.19) Summer: 0.25 (-0.13-0.63) Fall: 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) Summer: -0.03 (-0.75-0.69) Fall: 0.92 (0.17-1.68) MI Overall: 1.18 (0.48-1.89) Winter: 1.29 (-0.14-2.75) Spring: 2.12 (0.53-3.74) Summer: -0.03 (-1.46-1.42) Fall: 1.24 (0.12-2.36) Stroke Overall: 1.78 (0.96-2.62) Winter: 1.93 (0.34-3.54) Spring: 2.04 (-0.02-4.13) Summer: 1.64 (0.05-3.26) Fall: 1.69 (0.06-3.35) Respiratory Overall: 1.68 (1.04-2.33) Winter: 0.86 (-0.16-1.88) Spring: 4.62 (3.08-6.18) Summer: 0.78 (-0.49-2.06) Fall: 1.45 (0.19-2.72) **Percent Increase (95% CI) in mortality by increment in $PM_{2.5}$ combined by region** All Cause Mortality Humid Subtropical and Maritime: 1.02 (0.65-1.38) Warm Summer Continental: 1.19 (0.73-1.64) Hot Summer Continental: 1.14 (0.55-1.73) Dry: 1.18 (-0.70-3.10) Dry, Continental: 1.26 (-0.21-2.76) Mediterranean: 0.50 (0.00-1.01) CVD Humid Subtropical and Maritime: 0.78 (0.05-1.51) Warm Summer Continental: 1.43 (0.67-2.19) Hot Summer Continental: 0.43 (-0.53-1.40) Dry: 3.11 (-0.02-6.33) Dry, Continental: 1.67 (-0.75-4.16) Mediterranean: 0.16 (-0.46-0.79) MI Humid Subtropical and Maritime: 0.97 (-0.29-2.26) Warm Summer Continental: 1.50 (0.05-2.97) Hot Summer Continental: 0.64 (-0.96-2.28) Dry: 4.25 (-2.38-11.33) Dry, Continental: 0.60 (-7.42-9.32) Mediterranean: 1.85 (-0.66-4.41) Stroke Humid Subtropical and Maritime: 2.94 (1.59-4.32) Warm Summer Continental: 1.85 (0.04-3.69) Hot Summer Continental: 0.77 (-1.77-3.38) Dry: 1.82 (-6.98-11.45) Dry, Continental: 2.49 (-2.32-7.53) Mediterranean: 0.95 (-0.66-2.59) Respiratory Humid Subtropical and Maritime: 0.91 (-0.25-2.08) Warm Summer Continental: 2.12 (0.89-3.36) Hot Summer Continental:

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
		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 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	3.36 (1.95-4.79) Dry: 5.81 (-0.04-12.00) Dry, Continental: -0.31 (-5.89-5.61) Mediterranean: 1.06 (-0.36-2.50)
Reference: Zeger et al. (2007, 157176) **Period of Study:** 2000-2002 **Location:** 250 largest U.S. counties	**Outcome:** Mortality **Study Design:** Retrospective Cohort (MCAPS) **Statistical Analyses:** Log-linear regression models (GAM) **Covariates:** Age, gender, race, county-level SES, education and COPD SMR **Age Groups:** 65+ 65-74, 75-84, 85+	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 3-yr avg	**Increment:** 10 µg/m^3 65+: 1.076 (1.044, 1.108) Eastern U.S.: 1.125 (1.091, 1.159) Central U.S.: 1.196 (1.115, 1.277) Western U.S.: 1.029 (0.994, 1.064) 65-74: 1.156 (1.117, 1.196) 75-84: 1.081 (1.042, 1.121) 85+: 0.995 (0.956, 1.035)

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
Reference: Zeger et al. (2008, 191951)	**Outcome:** Mortality	**Pollutant:** $PM_{2.5}$	**Increment:** 10 µg/m^3
Period of Study: 2000-2005	**Study Design:** Retrospective Cohort	**Averaging Time:** Annual	**Relative Risk (Min CI, Max CI) lag**
Location: 4568 zip codes in urban areas	**Statistical Analysis:** Log-linear regression model	**Median (SD) Unit:**	Risk estimate for increase in mortality per increase in $PM_{2.5}$, all ages
	Age Groups: ≥65	Eastern: 14.0 µg/m^3	Eastern Region Age: 1.155 (1.130-1.180) Age + SES: 1.105 (1.084-1.125) Age + SES + COPD: 1.068 (1.049-1.087)
		Central: 10.7 µg/m^3	Central Region Age: 1.178 (1.133-1.222) Age + SES: 1.089 (1.052-1.125) Age + SES + COPD: 1.132 (1.095-1.169)
		Western: 13.1 µg/m^3	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)
		All: 13.2 µg/m^3	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) 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)
		Range (IQR):	
		Eastern: 12.3-15.3	
		Central: 9.8-12.2	
		Western: 10.4-18.5	
		All: 11.1-14.9	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)
		Copollutant (correlation): NR	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 µg/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 Tlaxcala and Veracruz (control cities), Mexico	**Outcome (ICD9 and ICD10):** COX2 (cyclooxygenase), IL-1β, CD14 in lungs, OB (olfactory bulb), frontal cortex, hippocampus, substantia nigrae, periaqueductal gray and vagus nerves **Age Groups Analyzed:** Subjects 2-45 yr of age mean=25.1 ± 1.5 yr **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	**PM Size:** No measure of PM used Mexico City as the "polluted city" and Tlaxcala and Veracruz as the "control cities" **Averaging Time:** NA **Mean (SD):** NA **Percentiles:** NA **Range (Min, Max):** NA **Unit (i.e. μg/m^3):** NA **Number of Monitoring Stations:** NA **Co-pollutant (correlation):** NA	**PM Increment:** NA **Effect Estimate [Lower CI, Upper CI]:** RT-PCR sample results from Control and Mexico City (MC) lung, CNS, PNS (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± 6.7 x 106 MC residents: 42.3± 7.4 x 106 P-value: 0.015 IL-1β lung Controls: 3.08±1.87 x 106 MC residents: 4.51± 2.6 x 106 P-value: 0.60 COX2 OB (olfactory bulb) Controls: 12.9± .0 x 105 MC residents: 38.7± 5.5 x 105 P-value: 0.0002 IL-1β OB Controls: 3.4± 0.8 x 104 MC residents: 7.7± 1.0 x 104 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± 0.4 x 105 MC residents: 5.0± 0.7 x 105 P-value: 0.008 IL-1β frontal Controls: 0.6± 0.2 x 104 MC residents: 6.2± 1.3 x 104 P-value: 0.0002 COX2 hippocampus Controls: 1.9± 0.5 x 105 MC residents: 1.6± 8.7 x 105 P-value: 0.1 IL-1β hippocampus Controls: 1.8±0.2 x 104 MC residents: 3.0±0.5 x 104 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 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

Study	Design & Methods	Concentrations[1]	Effect Estimates (95% CI)
			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) 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) **Period of Study:** 1989-1991 **Location:** U.S.	**Outcome:** Change in central nervous system function **Study Design:** Panel **Covariates:** Age, sex, race/ethnicity, individual socioeconomic position, lifestyle factors, household and neighborhood characteristics, conventional CVD risk factors **Statistical Analysis:** Pearson Chi-square tests and t-tests, as appropriate **Statistical Package:** STATA **Age Groups:** 20-59 yr	**Pollutant:** PM_{10} **Averaging Time:** 1 yr **Mean (SD) Unit:** 37.2 ± 12.8 μg/m^3 **Copollutant:** O_3	**Increment:** 10 μg/m^3 **Regression Coefficient β (95% CI)** Crude 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) Model 1: adjusted for age, sex, race/ethnicity SRTT: 2.03 (-0.15-4.20) 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 yr 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 avg of two seasons as measure of avg lifetime BC exposure **Dose-response Investigated? (Yes/No):** No **Statistical package:** SAS (v9.0)	**PM Size:** Black carbon (BC) **Averaging Time:** Lifetime exposure Estimated 24 h 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 avg 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 avg of the cold and warm seasons as the measure of avg 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: Adj for demographic factors: -3.7 (-7.2, -0.2) Adj for above factors + secondhand smoke: -3.7 (-7.3, -0.1) Adj for above factors + birth weight: -3.8 (-7.4, -0.2) Adj for above factors + blood lead level: -3.9 (-7.5, -0.3)

[1]All units expressed in μg/m^3 unless otherwise specified.

Annex E References

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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 yr, but apportionment done for 1.5 yr 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 ma). Biomass and secondary components significantly associated with respiratory HA (5-day ma). No significant effects for asthma HA in children (6-day ma). **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 yr)/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 New York 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 yr)	**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.					
Reference: Franklin et al. (2008, 097426) **Location:** STN/25 communities/2000-2005 (6 yr) **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.					

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: Gent et al. (2009, 180399) **Location:** 2 monitors in New Haven, CT/ 3.5 yr **Particle Size:** $PM_{2.5}$	**Subjects:** Children with physician diagnosed asthma and symptoms or medication use in previous 12 mo, 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, 088391) **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 (Washington, DC only), residual oil (Washington, DC only), Wood smoke/ biomass combustion, Sea salt, incinerator (Washington, DC only), primary coal (Washington, 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 (nonaccidental 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 Study) **Particle Size:** NR	**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
	Results: Lag 0-1 avg for all results. Overall 6 cities, mobile source factor (using Pb as tracer) had greatest association with daily mortality (3.4%) with 10 µg/m3 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, 042811)** 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, (Washington, DC only), residual oil (Washington, DC only), woodsmoke/ biomass combustion, sea salt, incinerator (Washington, DC only); primary coal (Washington, 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 (nonaccidental) 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 yr **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 yr **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 HAs: EC associated with same-day CVD HAs 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 HAs:** OCM associated with same-day respiratory HAs 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 mo) **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 avg 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, PNN_{50}, 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 yr **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, secondary 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 yr **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 µg/m3 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 mo 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 yr/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 µg/m3) CAPs (3-day mean CAPs concentration range: 126.1-481.0 µg/m3)	**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., 2000, 013252) 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 sampling for 1 yr **Particle Size:** PM_{10}	**Subjects:** Normal human bronchial epithelial and human AM **Exposure:** (2-3X105 cells/mL; 11 or 50 µg/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
	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 (avg for all studies, paired: 203.4, crossover: 360.8 µg/m3) 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 µg/m3)	**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 µg/m3); 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 (r^2>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 µg/m3)	**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 (µg/mg). Zn correlated with plasma fibrinogen in SH rats (p = 0.0023).					
Reference: Lippmann et al. (2005, 087453) **Location:** Rural location upwind from New York City **Particle Size:** $PM_{2.5}$	**Subjects:** Mice (C57 and ApoE) **Exposure:** CAPs (avg. mass concentration 113 µg/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 New York City **Particle Size:** $PM_{2.5}$	**Subjects:** Mice ($ApoE^{-/-}$) **Exposure:** CAPs (avg. mass concentration 85.6 µg/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 New York City **Particle Size:** $PM_{2.5}$	**Subjects:** NR **Exposure:** CAPs (90,000/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-κB. Oil combustion correlated the greatest with NF-κB (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 2001-2002	**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 µg/m3) 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 µg/m3)	**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 elements 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 mo	**Constituents considered for grouping:** 13 elements, TC, 5 OC variables, 4 EC variables, 2 ions, EU, one ratio (Ca: Al), OP, CO_3	**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 (R^2 = 0.50) and pyrolyzed OC (R^2 = 0.46), then Ca/Al (R^2 = 0.21). Carbonate carbon, EC3, and Sr correlated with IL-8 (R^2 = 0.27, 0.13, and 0.25, respectively). EC and Ni correlated with IL-8 trend over the range of 10-80 µg/cm2 (R^2 = 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 µg/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 yr **Particle Size:** 2.5; filtered to 0.22 um	**Subjects:** Alveolar macrophage cell line (NR8383); 1 ×106 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, pyrotechnics, platinum	**PM variables used:** Mass contribution of sources
	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 R^2 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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