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June 2001
Preliminary Draft

Review of the National Ambient Air Quality Standards for Particulate Matter:

Policy Assessment of Scientific and Technical Information

OAQPS Staff Paper

Notice

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1. INTRODUCTION

1.1 PURPOSE

The purpose of this preliminary draft Staff Paper, prepared by the Office of Air Quality Planning and Standards (OAQPS), is to identify the key policy-relevant scientific information contained in the EPA draft document, *Air Quality Criteria for Particulate Matter – Second External Review Draft* (EPA, 2001; henceforth referred to as draft CD and cited as CD), recognizing that this information is still provisional at this time. Preliminary and planned staff analyses (e.g., analyses of air quality and visibility data, human health risk assessment) are also presented for public and peer review prior to completing and incorporating results of such analyses into a subsequent draft of this document.

When final, this Staff Paper will evaluate the policy implications of the key studies and scientific information contained in the final *Air Quality Criteria for Particulate Matter* (henceforth the CD), and identify the critical elements that EPA staff believe should be considered in the review of the national ambient air quality standards (NAAQS) for particulate matter (PM). This assessment is intended to help "bridge the gap" between the scientific review contained in the CD and the judgments required of the Administrator in setting NAAQS for PM (*Natural Resources Defense Council v. Administrator*, 902 F.2d 962, 967 (D.C. Cir. 1990)). Thus, emphasis will be placed on identifying those conclusions and uncertainties in the available scientific literature that the staff believes should be considered in selecting PM indicators, forms, averaging times, and levels for the primary (health-based) and secondary (welfare-based) standards, which must be considered collectively in evaluating the health and welfare protection afforded by PM standards. The final Staff Paper will present factors relevant to the evaluation of current primary and secondary NAAQS, as well as staff conclusions and recommendations of options for the Administrator to consider.

While this preliminary draft Staff Paper should be of use to all parties interested in the NAAQS review, it is written for those decision makers, scientists, and staff who have some familiarity with the technical discussions contained in the draft CD.

1.2 BACKGROUND

1.2.1 Legislative Requirements

Two sections of the Clean Air Act govern the establishment and revision of the NAAQS (42 U.S.C. 7401 to 7671q, as amended). Section 108 (42 U.S.C. 7408) directs the Administrator to identify pollutants that "may reasonably be anticipated to endanger public health and welfare" and to issue air quality criteria for them. These air quality criteria are intended to "accurately reflect the latest scientific knowledge useful in indicating the kind and extent of identifiable effects on public health or welfare which may be expected from the presence of [a] pollutant in ambient air . . . ."

Section 109 (42 U.S.C. 7409) directs the Administrator to propose and promulgate "primary" and "secondary" NAAQS for pollutants identified under section 108. Section 109(b)(1) defines a primary standard as one "the attainment and maintenance of which in the judgment of the Administrator, based on such criteria and allowing an adequate margin of safety, are requisite to protect the public health."[1] A secondary standard, as defined in Section 109(b)(2), must "specify a level of air quality the attainment and maintenance of which, in the judgment of the Administrator, based on such criteria, is requisite to protect the public welfare from any known or anticipated adverse effects associated with the presence of [the] pollutant in the ambient air." Welfare effects as defined in section 302(h) [42 U.S.C. 7602(h)] include, but are not limited to, "effects on soils, water, crops, vegetation, man-made materials, animals, wildlife, weather, visibility and climate, damage to and deterioration of property, and hazards to transportation, as well as effects on economic values and on personal comfort and well-being."

Section 109(d)(1) of the Act requires that "not later than December 31, 1980, and at 5-year intervals thereafter, the Administrator shall complete a thorough review of the criteria published under section 108 and the national ambient air quality standards . . . and shall make such revisions in such criteria and standards . . . as may be appropriate . . . ." Section 109(d)(2)

[1]The legislative history of section 109 indicates that a primary standard is to be set at "the maximum permissible ambient air level . . . which will protect the health of any [sensitive] group of the population," and that for this purpose "reference should be made to a representative sample of persons comprising the sensitive group rather than to a single person in such a group" (S. Rep. No. 91-1196, 91st Cong., 2d Sess. 10 (1970)).

requires that an independent scientific review committee "shall complete a review of the criteria . . . and the national primary and secondary ambient air quality standards . . . and shall recommend to the Administrator any . . . revisions of existing criteria and standards as may be appropriate . . . ." Since the early 1980's, this independent review function has been performed by the Clean Air Scientific Advisory Committee (CASAC) of EPA's Science Advisory Board.

The U.S. Court of Appeals for the District of Columbia Circuit has held that the requirement for an adequate margin of safety for primary standards was intended to address uncertainties associated with inconclusive scientific and technical information available at the time of standard setting. It was also intended to provide a reasonable degree of protection against hazards that research has not yet identified (*Lead Industries Association v. EPA*, 647 F.2d 1130, 1154 (D.C. Cir 1980), cert. denied, 101 S. Ct. 621 (1980); *American Petroleum Institute v. Costle*, 665 F.2d 1176, 1177 (D.C. Cir. 1981), cert. denied, 102 S.Ct. 1737 (1982)). Both kinds of uncertainties are components of the risk associated with pollution at levels below those at which human health effects can be said to occur with reasonable scientific certainty. Thus, by selecting primary standards that provide an adequate margin of safety, the Administrator is seeking not only to prevent pollution levels that have been demonstrated to be harmful but also to prevent lower pollutant levels that may pose an unacceptable risk of harm, even if the risk is not precisely identified as to nature or degree.

In selecting a margin of safety, the EPA considers such factors as the nature and severity of the health effects involved, the size of the sensitive population(s) as risk, and the kind and degree of the uncertainties that must be addressed. The selection of any particular approach to providing an adequate margin of safety is a policy choice left specifically to the Administrator's judgment *(Lead Industries Association v. EPA*, supra, 647 F.2d at 1161-62).

1.2.2 History of PM NAAQS Reviews

National ambient air quality standards for PM were first established in 1971, based on the original criteria document (DHEW, 1969). Particulate matter is the generic term for a broad class of chemically and physically diverse substances that exist as discrete particles (liquid droplets or solids) over a wide range of sizes. Particles originate from a variety of anthropogenic

stationary and mobile sources as well as natural sources. Particles may be emitted directly or formed in the atmosphere by transformations of gaseous emissions such as sulfur oxides, nitrogen oxides, and volatile organic compounds. The chemical and physical properties of PM vary greatly with time, region, meteorology, and source category, thus complicating the assessment of health and welfare effects.

The reference method specified for determining attainment of the original standards was the high-volume sampler, which collects PM up to a nominal size of 25 to 45 micrometers (μm) (referred to as total suspended particles or TSP). The primary standards (measured by the indicator TSP) were 260 $\mu g/m^3$, 24-hour average, not to be exceeded more than once per year, and 75 $\mu g/m^3$, annual geometric mean. The secondary standard was 150 $\mu g/m^3$, 24-hour average, not to be exceeded more than once per year.

In October 1979 (44 FR 56731), EPA announced the first periodic review of the criteria and NAAQS for PM, and significant revisions to the original standards were promulgated in 1987 (52 FR 24854, July 1, 1987). In that decision, EPA changed the indicator for particles from TSP to PM_{10}, the latter referring to particles with a mean aerodynamic diameter[2] less than or equal to 10 μm. EPA also revised the level and form of the primary standards by: (1) replacing the 24-hour TSP standard with a 24-hour PM_{10} standard of 150 $\mu g/m^3$ with no more than one expected exceedance per year; and (2) replacing the annual TSP standard with a PM_{10} standard of 50 $\mu g/m^3$, annual arithmetic mean. The secondary standard was revised by replacing it with 24-hour and annual standards identical in all respects to the primary standards. The revisions also included a new reference method for the measurement of PM_{10} in the ambient air and rules for determining attainment of the new standards. On judicial review, the revised standards were upheld in all respects *(Natural Resources Defense Council v. Administrator*, 902 F. 2d 962 (D.C. Cir. 1990), cert. denied, 111 S. Ct. 952 (1991)).

[2]The more precise term is 50 percent cut point or 50 percent diameter (D_{50}). This is the aerodynamic particle diameter for which the efficiency of particle collection is 50 percent. Larger particles are not excluded altogether, but are collected with substantially decreasing efficiency and smaller particles are collected with increasing (up to 100 percent) efficiency.

In December 1994, EPA presented its plan for the second periodic review of the criteria and NAAQS for PM to the CASAC, and significant revisions to the NAAQS were promulgated in 1997 (62 FR 38652, July 18, 1997). In that decision, the PM NAAQS were revised in several respects. While it was determined that the PM NAAQS should continue to focus on particles less than or equal to 10 μm in diameter, it was also determined that the fine and coarse fractions of PM_{10} should be considered separately. New standards were added, using $PM_{2.5}$, referring to particles with a mean aerodynamic diameter less than or equal to 2.5 μm, as the indicator for fine particles, with PM_{10} standards retained for the purpose of regulating coarse-fraction particles. Two new $PM_{2.5}$ standards were set: an annual standard of 15 $\mu g/m^3$, based on the 3-year average of annual arithmetic mean $PM_{2.5}$ concentrations from single or multiple community-oriented monitors; and a 24-hour standard of 65 $\mu g/m^3$, based on the 3-year average of the 98th percentile of 24-hour $PM_{2.5}$ concentrations at each population-oriented monitor within an area. To continue to address coarse-fraction particles, the annual PM_{10} standard was retained, while the 24-hour PM_{10} standard was revised to be based on the 99th percentile of 24-hour PM_{10} concentrations at each monitor in an area. The secondary standards were revised by making them identical in all respects to the primary standards.

In May 1998, in response to challenges filed by industry and others, a three-judge panel of the U.S. Court of Appeals for the District of Columbia Circuit issued a split opinion regarding the NAAQS for PM. The Panel recognized the scientific basis for the PM NAAQS revisions, stating that "the growing empirical evidence demonstrating a relationship between fine particle pollution and adverse health effects amply justifies establishment of new fine particle standards." Further, the Panel found "ample support" for EPA's decision to regulate coarse particle pollution, although it vacated the revised coarse particle standards on the basis of PM_{10} being a "poorly matched indicator for coarse particulate pollution" because PM_{10} includes fine particles.[3] More generally, the Panel held (with one dissenting opinion) that the Clean Air Act, as applied and absent further clarification, is unconstitutional because it "effects an unconstitutional delegation of legislative power." Although the Panel stated that "the factors EPA uses in determining the

[3] The 1987 PM_{10} standards remain in effect.

degree of public health concern associated with different levels of ozone and PM are reasonable," it remanded the NAAQS to the EPA, stating that when EPA considers these factors for potential non-threshold pollutants "what EPA lacks is any determinate criterion for drawing lines" to determine where the standards should be set. Also, consistent with EPA's long-standing interpretation, the Panel unanimously held that in setting NAAQS EPA is "not permitted to consider the cost of implementing those standards."

These two general rulings were appealed to the U.S. Supreme Court, and in February 2001, the Supreme Court issued a unanimous decision that reversed the Court of Appeals' ruling on the constitutional issue and upheld its ruling on the cost issue. In so doing, the Supreme Court upheld EPA's position on both issues. Because the Court of Appeals had not rendered decisions on all issues related to the 1997 PM NAAQS that had originally been before that court, the case was sent back for resolution of any remaining issues. The Court of Appeals has scheduled further briefing on those issues this summer and fall. Although the litigation has not yet been fully resolved, the $PM_{2.5}$ standards have not been revoked and thus remain in place.

On October 23, 1997, EPA published its plans for the current periodic review of the PM NAAQS (62 FR 55201). As part of the process of preparing the PM CD, on April 6-9, 1999, the EPA's National Center for Environmental Assessment (NCEA) hosted a peer review workshop on drafts of key chapters of the CD. The first external review draft CD was reviewed by CASAC and the public at a meeting held on December 2, 1999. Based on CASAC and public comment, NCEA revised the CD and released the second external review draft in April 2001 for review by CASAC and the public at a meeting to be held July 23-24, 2001.

This preliminary draft Staff Paper is being provided to the CASAC and the public for comment at that same public meeting. Subsequently, EPA intends to complete staff analyses and to address CASAC and public comments on this draft in a second draft that will then be made available for further review and comment by CASAC and the public.

1.3 APPROACH

The final Staff Paper will rely on the scientific evidence reviewed in the final CD in evaluating the adequacy of the existing PM NAAQS for protection of public health and welfare. The results of comparative air quality and human health risk analyses, as well as analyses examining visibility impairment, will also be presented in the final Staff Paper. The final Staff Paper will include the staff's overall evaluation of the primary and secondary NAAQS and conclusions and recommendations as to whether any revisions are appropriate to address public health and welfare effects associated with fine- and coarse-fraction particles. In so doing, the staff will assess and integrate new scientific and technical findings with information gained in previous reviews in the context of those critical elements that the staff believes should be considered.

In conducting various technical analyses, the staff intends to focus separately on fine- and coarse-fraction particles, building upon the conclusions reached in the last review, and taking into account any new information that has become available. More specifically, sufficient data now exist to conduct air quality analyses to characterize spatial and temporal air quality patterns, for example, primarily in terms of $PM_{2.5}$ and $PM_{10-2.5}$ as the indicators for fine- and coarse-fraction particles, respectively, the later referring to particles with a mean aerodynamic diameter between 2.5 and 10 μm. Similarly, the current draft plan for human health risk analyses focuses on analyzing various health effects associated with $PM_{2.5}$, and identifies for further consideration the possibility of also analyzing certain health effects associated with $PM_{10-2.5}$.

Beyond this introductory chapter, this preliminary draft Staff Paper is organized into four chapters, with an additional chapter to be added in the next draft presenting staff conclusions and recommendations on the primary and secondary standards. More specifically, Chapter 2 focuses on air quality characterizations, including information on atmospheric concentrations, chemistry, and sources of PM, including, to the extent possible, evaluation of newly available air quality monitoring data, as well as information on the relationship between ambient air quality and human exposure. Chapter 3 presents key information on PM-associated health effects, relying primarily on the review of recent epidemiological and toxicological studies in the draft CD and integrating the new information with findings from previous criteria and NAAQS reviews. Draft

plans for a quantitative human health risk analysis are presented for comment in Chapter 4. Information on welfare effects of ambient PM is presented in Chapter 5, together with analyses of data on visibility and draft plans for conducting a focus-group-based assessment of urban visibility impairment.

REFERENCES

Environmental Protection Agency. (2001) Air Quality Criteria for Particulate Matter. Research Triangle Park, NC: Office of Research and Development; report no. EPA/600/P-99/002. March.

U.S. Department of Health, Education and Welfare. (1969) Air Quality Criteria for Particulate Matter. U.S. Government Printing Office, Washington DC, AP-49.

2. AIR QUALITY CHARACTERIZATION

2.1 INTRODUCTION

This chapter defines the various subclasses of particulate matter (PM) and then briefly discusses the physical and chemical properties of PM in the atmosphere, sources of PM, PM measurement methods, and recent PM concentrations and trends. This information is useful for interpreting the available health and welfare effects information and in making recommendations for appropriate indicators for PM. Section 2.2 presents information on the basic physical and chemical properties of classes of PM, and is not substantially different from information contained in the 1996 Criteria Document (EPA, 1996a) and Staff Paper (EPA, 1996b). Section 2.3 presents information on the methods used to measure PM and some of the important considerations in designing these methods. Section 2.4 presents data on PM concentrations, trends, and spatial patterns. Section 2.5 provides information on the temporal variability of PM across daily and monthly time scales. Much of the information in Sections 2.4 and 2.5 is derived from analyses of new data collected by the recently deployed nationwide network of $PM_{2.5}$ monitors. Section 2.6 defines and discusses background levels of PM. Section 2.7 provides national estimates of source emissions. Section 2.8 addresses the relationship between ambient PM levels and human exposure to PM. Finally, Section 2.9 summarizes relevant information on the optical and radiative effects of particles.

2.2 CHARACTERIZATION OF U.S. AMBIENT PARTICULATE MATTER

PM represents a broad class of chemically and physically diverse substances that exist as discrete particles in the condensed (liquid or solid) phase. Particles can be described by size, formation mechanism, origin, chemical composition, atmospheric behavior, and by what is measured by a specific sampling technique. Fine-mode and coarse-mode particles, which are defined in Section 2.2.1.1, are distinct entities with fundamentally different sources and formation processes, chemical composition, atmospheric residence times and behaviors, and transport distances. The 1996 Criteria Document concluded that these differences alone justified consideration of fine-mode and coarse-mode particles as separate pollutants (EPA 1996a, p. 13-

3), and this conclusion is reiterated in the new draft Criteria Document (CD, p. 9-1). The fundamental differences between fine-mode and coarse-mode particles are also important considerations in assessing the available health effects and exposure information.

2.2.1 Particle Size Distributions

Particle properties, including their associated health and welfare effects, differ by size. The diameters of atmospheric particles span 5 orders of magnitude, ranging from 0.001 micrometers to 100 micrometers (μm).[1] The size and associated composition of particles determine their behavior in the respiratory system (i.e., how far the particles are able to penetrate, where particles are deposited, and how effective the body's clearance mechanisms are in removing them). Furthermore, a particle's size is one of the most important parameters in determining its residence time in ambient air, which is a key consideration in assessing exposure. Particle size is also a determinant of visibility impairment, a welfare effect linked to ambient particles. Particle surface area, number, chemical composition, water solubility, formation processes, and emissions sources all vary with particle size.

Two common conventions for classifying particles by size include: (1) modes, based on observed particle size distributions; and (2) cut points, based on the inlet restriction of a specific PM sampling device.

2.2.1.1 Modes

Based on extensive examinations of particle size distributions in several U.S. locations in the 1970's, Whitby (1978) found that particles display a consistent multi-modal distribution over several physical metrics, such as mass and volume (CD, p. 2-9). These modes are apparent in Figure 2-1, which shows average ambient distributions of particle number, surface area, and volume by particle size. Panel (a) illustrates that most ambient particles are very small, below 0.1 μm, while panel (c) indicates most of the particle volume, and therefore most of the mass,

[1] In this Staff Paper, particle size or diameter usually refers to a normalized measure called aerodynamic diameter. Most ambient particles are irregularly shaped rather than perfect spheres. The aerodynamic diameter of any irregular shaped particle is defined as the diameter of a spherical particle with a material density of 1 g/cm^3 and the same settling velocity as the irregular shaped particle. Particles with the same physical size and shape but different densities will have different aerodynamic diameters (CD, p. 2-3).

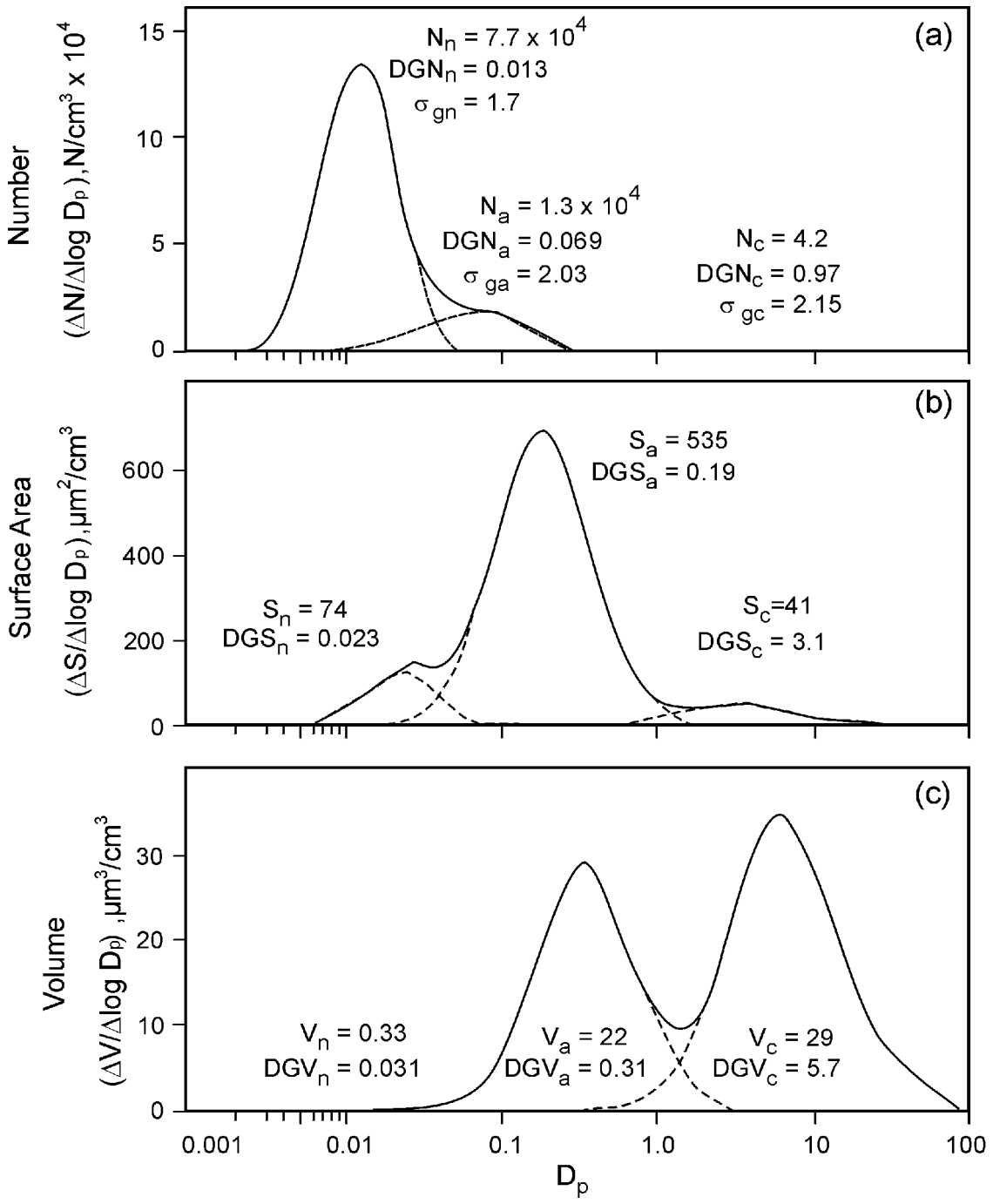


Figure 2-1. Distribution of coarse [c], accumulation [a], and nuclei or ultrafine [n], mode particles by three characteristics: Panel (a) number [N], Panel (b) surface area [S], and Panel (c) volume [V] for the grand average continental size distribution. D_p = geometric diameter; DGN = geometric mean diameter by number; DGS = geometric mean diameter by surface area; DGV = geometric mean diameter by volume.

Source: Whitby (1978); CD, page 2-7.

is found in particles larger than 0.1 µm. The surface area distribution in panel (b) peaks around 0.2 µm (CD, p. 2-5). Distributions may vary across locations, conditions, and time due to differences in sources, atmospheric conditions, and topography.

As illustrated in panel (c) of Figure 2-1, volume distributions measured in ambient air in the United States are almost always found naturally to be bimodal, with an intermodal minimum between 1 and 3 µm (CD, p. 2-6). The distribution of particles that are mostly larger than this minimum is termed "coarse mode," and the distribution of particles that are mostly smaller than the minimum is termed "fine mode." Fine-mode particles are separated into two sub-modes: "accumulation mode" and "nuclei mode" (also known as "ultrafines"). The accumulation mode and the nuclei mode are apparent as the leftmost peaks in the number and surface area distributions in Figure 2-1, whereas the accumulation mode is apparent as the leftmost peak in the volume distribution. Since nuclei-mode particles have relatively low mass and grow rapidly into accumulation-mode particles, they are not commonly observed as a separate mode in volume or mass distributions. Exceptions include clean or remote areas with low PM concentrations, and areas near freshly generated fine-mode particles such as freeways and intersections with heavy automobile traffic (CD, pp. 2-10 and 2-17).

2.2.1.2 Sampler Cut Points

Another set of particle size classifications is derived from the characteristics of ambient particle samplers. Particle samplers typically use size-selective air inlets that are defined by their 50 percent cut point, which is the cut point at which 50 percent of particles of a specified diameter are captured by the inlet. The usual notation for these definitions is "PM_x", where *x* refers to measurements with a cut point of *x* µm aerodynamic diameter. Because of the overlap in the distributions of ambient particles, no single cut point can precisely separate fine-mode and coarse-mode particles. The objective of size-selective sampling is usually to measure particle size fractions with some special relationship to human health impacts, visibility impairment, or emissions sources.

The EPA has historically defined indicators of PM for national ambient air quality standards (NAAQS) using various cut points. Figure 2-2 presents an idealized distribution of ambient PM showing the fractions collected by size-selective samplers. Prior to 1987, the

indicator for the PM NAAQS was total suspended particulate matter (TSP), and was defined by the design of the High Volume Sampler (hivol).[2] As shown in Figure 2-2, TSP includes particle diameters less than 40 μm. When EPA established new PM standards in 1987, the selection of PM_{10} as an indicator was intended to focus regulatory concern on particles small enough to enter the thoracic region of the lungs. In 1997, EPA established a new standard for a fraction of fine-mode particles based in part on epidemiological studies that used $PM_{2.5}$ concentrations as an exposure index. Figure 2-2 shows the distribution of particles captured by the PM_{10} Federal Reference Method (FRM) sampler[3] and the $PM_{2.5}$ FRM sampler[4].

The common PM measurement indicators used in this Staff Paper are summarized in Table 2-1. Note that the terms "fine fraction" and "coarse fraction" are used interchangeably with $PM_{2.5}$ and $PM_{10-2.5}$, respectively, to refer to specific portions of the fine and coarse modes collected by size selective samplers.

2.2.2 Sources and Formation Processes

In most locations, a variety of activities contribute to PM concentrations. Fine-mode and coarse-mode particles generally have distinct sources and formation mechanisms although there is some overlap. Coarse-mode particles are primary particles, meaning they are emitted directly as particles. Most coarse-mode particles result from mechanical disruption such as crushing, grinding, evaporation of sprays, or dust resuspension. Specific sources include construction and demolition activities, sea spray, and resuspension of settled dust from soil surfaces and roads (CD, p. 3-34). The amount of energy required to break down primary particles into smaller particles normally limits coarse-mode particle sizes to greater than 1.0 μm diameter (EPA 1996a, p. 13-7).

[2] 40 CFR Part 50, Appendix B.

[3] 40 CFR Part 50, Appendix J.

[4] 40 CFR Part 50, Appendix L.

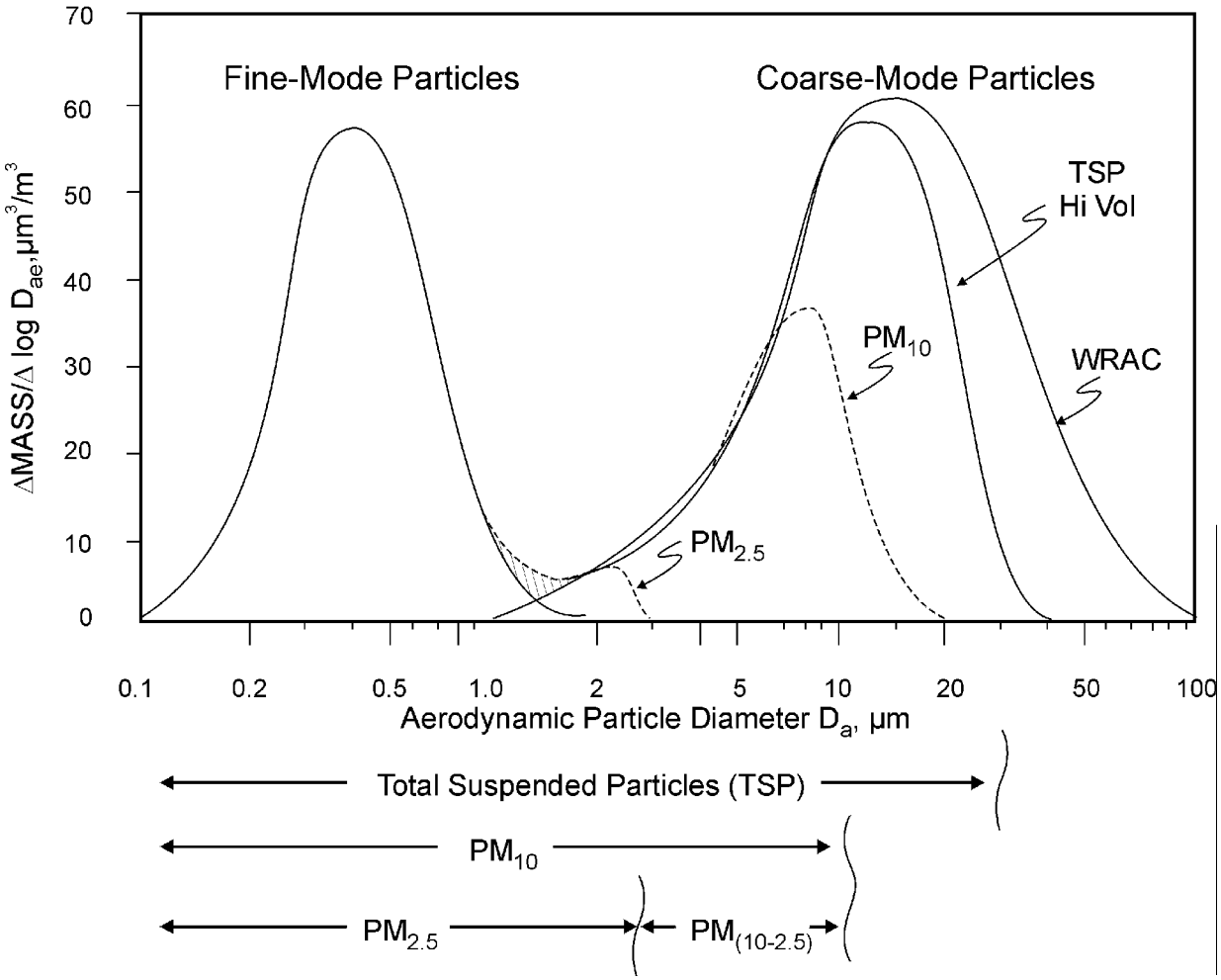


Figure 2-2. An idealized distribution of ambient particulate matter showing fine-mode particles and coarse-mode particles and the fractions collected by size-selective samplers. (WRAC is the Wide Range Aerosol Classifier which collects the entire coarse mode.) Note that this idealized distribution is truncated at a diameter of 0.1 μm, such that it does not include the ultrafine fraction.

Source: Adapted from Wilson and Suh (1997); CD, page 2-11.

Some combustion-generated particles such as fly ash are also found in the coarse mode.

Table 2-1. Particle Size Fraction Terminology Used in Staff Paper

Term	Description
Size Distribution Modes	
Coarse-Mode Particles	The distribution of particles larger than the intermodal minimum in volume or mass distributions, which generally occurs between 1 and 3 μm.
Fine-Mode Particles	The distribution of particles smaller than the intermodal minimum in volume or mass distributions, which generally occurs between 1 and 3 μm. Particles in this mode are the most numerous and represent the most surface area.
Accumulation-Mode Particles	A subset of fine-mode particles with diameters above about 0.1 μm.
Nuclei-Mode Particles ("ultrafines")	A subset of fine-mode particles with diameters below about 0.1 μm.
Sampling Measurements	
Total Suspended Particles (TSP)	Particles measured by a high volume sampler as described in 40 CFR Part 50, Appendix B. This sampler has a cut point of aerodynamic diameters that varies between 25 and 40 μm depending on wind speed and direction.
PM_{10}	Particles measured by a sampler that contains a size fractionator (classifier) designed with an effective cut point of 10 μm aerodynamic diameter. This measurement includes the fine mode and part of the general coarse mode and is an indicator for thoracic particles (i.e., particles that penetrate to the tracheo-bronchial and the gas-exchange regions of the lung).
$PM_{2.5}$ "fine fraction"	Particles measured by a sampler that contains a size fractionator (classifier) designed with an effective cut point of 2.5 μm aerodynamic diameter. The collected particles include most of the fine mode. A small portion of the coarse mode may be included depending on the sharpness of the sampler efficiency curve and the size of coarse mode particles present.
$PM_{(10-2.5)}$ "coarse fraction"	Particles measured directly using a dichotomous sampler or subtraction of particles measured by a $PM_{2.5}$ sampler from those measured by a PM_{10} sampler. This measurement is an indicator for the fraction of coarse-mode thoracic particles (i.e., particles that penetrate to the tracheo-bronchial and the gas-exchange regions of the lung).

Directly emitted particles are also found in the fine mode, the most common being nuclei-mode particles emitted as combustion-related vapors that rapidly condense. They originate from fuel combustion (from vehicles, power generation, and industrial facilities), residential wood burning, and agricultural and silvicultural burning. However, the majority of fine-mode mass is attributable to secondary particles, formed in the atmosphere from gases (CD, p. 2-20). Fine-mode particles are usually formed from gases in three ways: (1) nucleation (i.e., gas molecules coming together to form a new particle); (2) condensation of gases onto existing particles; and (3) coagulation of particles (CD, p. 2-2). Gas phase material condenses preferentially on smaller particles, and the rate constant for coagulation of two particles decreases as the particle size increases. Therefore, nuclei-mode particles grow into the accumulation mode, but accumulation-mode particles do not grow into the coarse mode (CD, p. 2-16). Examples of secondary particle formation include: (1) the conversion of sulfur dioxide (SO_2) to sulfuric acid (H_2SO_4) droplets that further react with ammonia (NH_3) to form sulfate (ammonium sulfate ($(NH_4)_2SO_4$) or ammonium acid sulfate (NH_4HSO_4)) particles; (2) the conversion of nitrogen dioxide (NO_2) to nitric acid (HNO_3) which reacts further with ammonia to form ammonium nitrate (NH_4NO_3) particles; and (3) reactions involving volatile organic compounds (VOC) yielding organic compounds with low ambient temperature vapor pressures that nucleate or condense on existing particles to form secondary organic particles (CD, p. 2-21).

2.2.3 Chemical Composition

Based on studies conducted in most parts of the U.S., the draft CD reports that coarse-mode particles are composed primarily of crustal materials such as calcium, aluminum, silicon, magnesium, and iron. Some organic materials such as pollen, spores, and plant and animal debris are also found predominantly in the coarse mode (CD, p. 2-19). Fine-mode particles are composed primarily of sulfate, nitrate, ammonium, and hydrogen ions; elemental carbon, secondary organic compounds and some primary organic compounds; and certain transition metals deriving primarily from combustion processes..

Some components, such as potassium and nitrate, may be found in both the fine and coarse particle modes, but different sources or mechanisms contribute to their existence in each

mode. Potassium in coarse-mode particles comes from soil. Potassium in fine-mode particles comes from emissions of burning wood or cooking meat. Nitrate in fine-mode particles comes primarily from the reaction of gas-phase nitric acid with gas-phase ammonia to form ammonium nitrate particles. Nitrate in coarse-mode particles comes primarily from the reaction of gas-phase nitric acid with pre-existing coarse-mode particles (CD, p. 2-19).

Many ambient particles also contain water (particle-bound water) as a result of equilibrium of water vapor with water bound to hygroscopic particles (CD, p. 2-28). Particle-bound water influences the size of particles and in turn their aerodynamic and light scattering properties. Studies of the change in particle size with changes in relative humidity (RH) suggest that a small fraction of accumulation-mode particles (with a dry diameter smaller than 1 μm) will be larger than 1 μm in diameter at RH below 60%, but a larger fraction will grow above 1 μm for RH above 80% (CD, p. 2-39). The amount of the increase in particle size with increasing RH is dependent on the particle's chemical composition (CD, p. 4-91). Particles containing inorganic salts and acids are more hygroscopic than particles composed primarily of organic species.

2.2.4 Fate and Transport

Fine-mode and coarse-mode particles typically exhibit different behavior in the atmosphere. These differences affect several exposure considerations including the representativeness of central-site monitored values and the behavior indoors of particles that were formed outdoors. The ambient residence time of atmospheric particles varies with size. Coarse-mode particles can settle rapidly from the atmosphere with lifetimes from a few seconds to hours, and their spatial impact is limited because they tend to fall out of the air in the downwind area near their emission point. Larger coarse-mode particles are not readily transported across urban or broader areas, because they are generally too large to follow air streams, and they tend to be easily removed by impaction on surfaces. Smaller-sized coarse-mode particles can have longer lives and longer travel distances, especially in extreme circumstances, such as dust storms (CD, p. 2-30).

Fine-mode particles are kept suspended by normal air motions and have low surface deposition rates. Because they grow rapidly into the accumulation mode, the subset of nuclei-

mode particles have a very short life, on the order of minutes to hours. Nuclei-mode particles are also small enough to be removed through diffusion to falling rain drops (CD, p. 2-32). Accumulation-mode particles, which do not grow into the coarse mode, can be transported thousands of kilometers and remain in the atmosphere for days to weeks. Accumulation-mode particles are removed from the atmosphere primarily by cloud processes. They serve as condensation nuclei for cloud droplet formation and eventually fall as rain drops. However, accumulation-mode particles are not effectively removed from the atmosphere by falling rain (CD, p. 2-30).

Because fine-mode particles remain suspended for days to weeks, and travel much farther than coarse-mode particles, fine-mode particles are theoretically likely to be more uniformly dispersed at urban scales than coarse particles. In contrast, coarse-mode particles tend to exhibit more elevated concentrations near sources (EPA 1996a, p. 13-15).

The characteristics of nuclei-mode, accumulation-mode, and coarse-mode particles that were discussed in the preceding sections are summarized in Table 2-2.

2.3 PM MEASUREMENT METHODS

The draft CD indicates that the methods used to measure PM are important to understanding population exposure to PM, evaluating health risks, and developing risk management strategies. Because PM is not a homogeneous pollutant, measuring and characterizing particles suspended in the atmosphere is a significant challenge, and there is no perfect method for every application.[5] Measurements include particle mass, composition, and particle number. Most instruments collect PM by drawing a controlled volume of ambient air through a size-selective inlet, usually defined by the inlet's 50 percent cut point. Often used measurements or indicators of fine-mode particles include $PM_{2.5}$, $PM_{1.0}$, British or black smoke (BS), coefficient of haze (COH), sulfates, acids, and PM_{10} (in areas dominated by fine-mode particles). Measurements of coarse-mode particles include $PM_{10-2.5}$, $PM_{15-2.5}$, and PM_{10} (in areas dominated by coarse-mode particles).

[5] Refer to EPA 1996a, Chapter 4 and draft CD Chapter 2 for more comprehensive assessments of particle measurement methods.

Table 2-2. Comparison of Ambient Particles: Fine Mode (Nuclei Mode plus Accumulation Mode) and Coarse Mode

	Fine-Mode Particles		Coarse-Mode Particles
	Nuclei Mode	Accumulation Mode	
Aerometric Diameter	< 0.1 μm	0.1 – 3.0 μm	> 1.0 μm
Formed from:	Combustion, high temperature processes and atmospheric reactions		Break-up of large solids/droplets
Formed by:	Nucleation Condensation Coagulation	Condensation Coagulation Evaporation of fog and cloud droplets in which gases have dissolved and reacted	Mechanical disruption (crushing, grinding, abrasion of surfaces) Evaporation of sprays Suspension of dusts Reactions of gases in or on particles
Composed of:	Sulfate, $SO_4^=$ Elemental carbon Metals compounds (Pb, Cd, V, Ni, Cu, Zn, Mn, Fe, K, etc.) Organic compounds with very low, saturation vapor pressure at ambient temperature	Sulfate Nitrate, NO_3^- Ammonium, NH_4^+ Hydrogen ion, H^+ Elemental carbon, Large variety of organic compounds Metal compounds Particle-bound water	Suspended soil or street dust Fly ash from uncontrolled combustion of coal, oil, wood Nitrates/chlorides from HNO_3/HCl Oxides of crustal elements (Si, Al, Ti, Fe, Mg) $CaCO_3$, NaCl, sea salt Pollen, mold, fungal spores Plant/animal fragments Tire, brake pad, and road wear debris
Solubility:	Probably less soluble than accumulation mode	Largely soluble, hygroscopic and deliquescent	Largely insoluble and non-hygroscopic
Sources:	Combustion of coal, oil, gasoline, diesel fuel, wood Atmospheric transformation of SO_2 and some organic compounds High temperature processes, smelters, steel mills, etc.	Combustion Atmospheric transformation products of NO_x, SO_2, and organic compounds including biogenic organic species (e.g., terpenes) High temperature processes Volcanic activity Wildfires	Resuspension of industrial dust and soil tracked onto roads and streets Suspension from disturbed soil (e.g., farming, mining, unpaved roads) Construction and demolition Uncontrolled coal and oil combustion Ocean spray Biological sources
Atmospheric half-life:	Minutes to hours	Days to weeks	Minutes to hours
Removal Processes:	Grows into accumulation mode Scavenging by falling rain drops	Forms cloud droplets and rains out Dry deposition	Dry deposition by fallout Scavenging by falling rain drops
Travel distance:	<1 to 10s of km	100s to 1000s of km	<1 to 10s of km (100s to 1000s in dust storms)

Source: Adapted from Wilson and Suh (1997); CD, p. 2-35.

PM mass can be measured directly, by gravimetric methods, or indirectly using methods that rely on the physical properties of particles. The most common direct measurement methods include filter-based methods where ambient aerosols are collected for a specified period of time (e.g., 24 hours) on filters that are weighed to determine mass. Examples include the Federal Reference Method monitors for $PM_{2.5}$ and PM_{10}. Dichotomous samplers contain a separator that splits the air stream from a PM_{10} inlet into two streams so that both fine and coarse fraction particles can be collected on separate filters. With this approach a fraction of the fine-mode particles are collected with the coarse-mode particles.

Another widely used gravimetric method is the Tapered Element Oscillating Microbalance (TEOM®) sensor, consisting of a replaceable filter mounted on the narrow end of a hollow tapered quartz tube. The air flow passes through the filter, and the aerosol mass collected on the filter causes the characteristic oscillation frequency of the tapered tube to change in direct relation to particle mass. This approach allows mass measurements on a near-continuous basis (every few minutes).

Other methods that produce near-continuous PM measurements include beta attenuation sampler and the Continuous Ambient Mass Monitor (CAMM). Beta attenuation (or beta gauge) samplers determine the mass of particles deposited on a filter by measuring the absorption of electrons generated by a radioactive isotope. The absorption varies with the mass of the particles. The CAMM measures the pressure drop increase that occurs in relation to particle loading on a membrane filter.

PM has also been characterized in the U.S. and abroad by indirect filter-based optical methods that rely on the light scattering or absorbing properties of both suspended PM and PM collected on a filter.[6] These include BS and COH, as well as estimates derived from visibility measurements. In locations where they are calibrated to standard mass units, these indirect measurements can be useful surrogates for particle mass. The BS method typically involves impacting samples from a 4.5 μm inlet onto white filter paper where blackness of the stain is measured by light absorption. Smoke particles composed of elemental carbon (EC) typically

[6] See Section 2.8 of this chapter for a discussion of the optical properties of PM.

make the largest contribution to stain darkness. Since the mix of ambient particles varies widely by location and time of year, the correlation between BS measurements and PM mass are highly site- and time-specific. COH is determined using a light transmittance method. This involves impacting samples from a 5.0 μm inlet onto filter tape where the opacity of the resulting stain is determined. This technique is somewhat more responsive to non-carbon particles than the BS method. Nephelometers measure the light scattered by ambient aerosols in order to calculate light extinction. This method results in measurements that can correlate well with the mass of fine-mode particles below 2 μm diameter.

There are a variety of methods used to identify and describe the characteristic components of ambient PM. X-ray fluorescence (XRF) is a commonly used laboratory technique for analyzing the elemental composition of primary particles deposited on filters Wet chemical analysis methods, such as ion chromatography (IC) and automated colorimetry (AC) are used to measure ions such as nitrate (NO_3^-), sulfate ($SO_4^=$), chloride (Cl^-), ammonium (NH^+), sodium (Na^+), and phosphate (PO_4^{3-}).

There are several methods for separating organic carbon (OC) and elemental carbon (EC) in ambient samples. Thermal/optical reflectance (TOR) and thermal manganese oxidation (TMO) have been commonly applied in aerosol studies in the United States. Still another method is the thermal/optical transmission (TOT) method. This method is similar to TOR and yields comparable estimates of total carbon, but gives a different split between OC and EC. Monitoring methods capable of separately measuring sulfate, nitrate, and carbon particles on a near-continuous basis are currently under development..

The presence of semi-volatile PM components and sampling in extreme climate conditions present special challenges for designing measurement methods. Accurate measurement of fine-mode particles is particularly difficult when the relative humidity is high, or when winds cause high ambient concentrations of wind-blown soil. In these conditions, a significant amount of either fine-mode or coarse-mode material may be found in the inter-modal region between 1.0 and 3 μm diameter. The draft CD suggests that under these conditions a better measurement of fine-mode particles could be obtained by removing all or most particle-bound water, measuring PM at a constant relative humidity, and using a cut point of 1.0 μm rather than 2.5 μm diameter (CD, p.

2-40). All continuous monitoring methods require removal of particle-bound water prior to mass measurement. However, heating the inlet stream to a constant temperature to keep moisture in the vapor phase can have the negative effect of removing a portion of the PM compounds that have equilibrium vapor pressures that are higher than typical ambient temperatures, and can chemically degrade some organic compounds. Newer techniques use diffusion drying to remove water vapor, leading to vaporization of particle-bound water without heating.

In addition to particle mass and composition, the number of ambient particles can also be measured. Recently there has been increasing interest in examining the relationship between the number of ambient particles and health effects. A nano-scanning mobility particle sizer (NSMPS) counts particles in the 0.003 to 0.15 μm range. A standard scanning mobility particle sizer (SMPS) counts particles in the 0.01 to 1 μm range, and a laser particle counter (LPC) counts particles in the 0.1 to 2 μm range. An aerodynamic particle sizer measures particles in the 0.7 to 10 μm range. These techniques have not yet been widely used in health effects studies.

2.4 PM CONCENTRATIONS, TRENDS, AND SPATIAL PATTERNS

This section provides analysis of the latest available PM air quality data, including PM levels, composition, spatial patterns, and temporal patterns. Only recently has a full year of mass concentration data from a nationwide network of $PM_{2.5}$ Federal Reference Method (FRM) monitors been available, and analyses of those data are presented here. Readers should be cautioned not to draw conclusions regarding the attainment or nonattainment status from a single year of PM monitoring data. EPA regulations, in 40 CFR Part 50, Appendix N, require 3 years of monitoring data and specify minimum data completeness requirements for data used to make decisions regarding attainment status. Not all PM FRM monitors that were operated in 1999 recorded valid PM measurements for all four calendar quarters. In the figures that follow, data completeness is illustrated by the size of the circles on the map, with smaller circles indicating relatively incomplete data for the year. Additional $PM_{2.5}$ data are presented from other long-term monitoring efforts, including data from the network for Interagency Monitoring of Protected Visual Environments (IMPROVE) and from the California Air Resources Board, which are not directly comparable to the FRM monitor data.

2.4.1 PM_{10}

State and local air pollution control agencies have been collecting PM_{10} mass concentration data using EPA-approved FRM samplers and reporting these data to EPA's publicly available Aerometric Information Retrieval System (AIRS) data base since mid-1987.[7] PM_{10} data from 1999 are shown in Figures 2-3a and 2-3b. Figure 2-3a shows the PM_{10} annual mean concentrations, and Figure 2-3b shows the second highest 24-hour average concentrations. Most areas of the country had concentrations below the level of the annual mean PM_{10} standard (50 $\mu g/m^3$). Exceptions include central South Carolina, Puerto Rico, and several places in the southwestern U.S. and central California. Most areas of the country also had concentrations below the level of the 24-hour standard (150 $\mu g/m^3$), with exceptions mostly in the western U.S.

In the 1998 National Air Quality and Emissions Trends Report (EPA 2000b), EPA examined national and regional PM_{10} trends for the 10-year period from 1989 to 1998. Figure 2-4 shows the national trend and the trend in each EPA region. The figure shows approximately a 25 percent decline in concentrations over the 10 year period with regional declines in the eastern U.S. ranging from 18 to 21 percent, and declines in the western U.S. ranging from 31 to 38 percent. In the national trend and in several regions, the declines appearing to level off in more recent years. Figure 2-5 shows the national 10-year trend in annual mean PM_{10} concentrations for 906 sites broken down into rural, suburban, and urban locations. Rural levels are significantly lower than suburban and urban levels, but all three classifications show a similar decline of about 25 percent.

[7] Based in part on this data, EPA has designated areas of the country that are not attaining PM_{10} standards. As of July 2000 there were a total of 66 areas classified as moderate or serious nonattainment areas, mostly in the western U.S., with fewer in heavily populated or industrialized eastern areas. See designated nonattainment areas at www.epa.gov/oar/oaqps/greenbook.

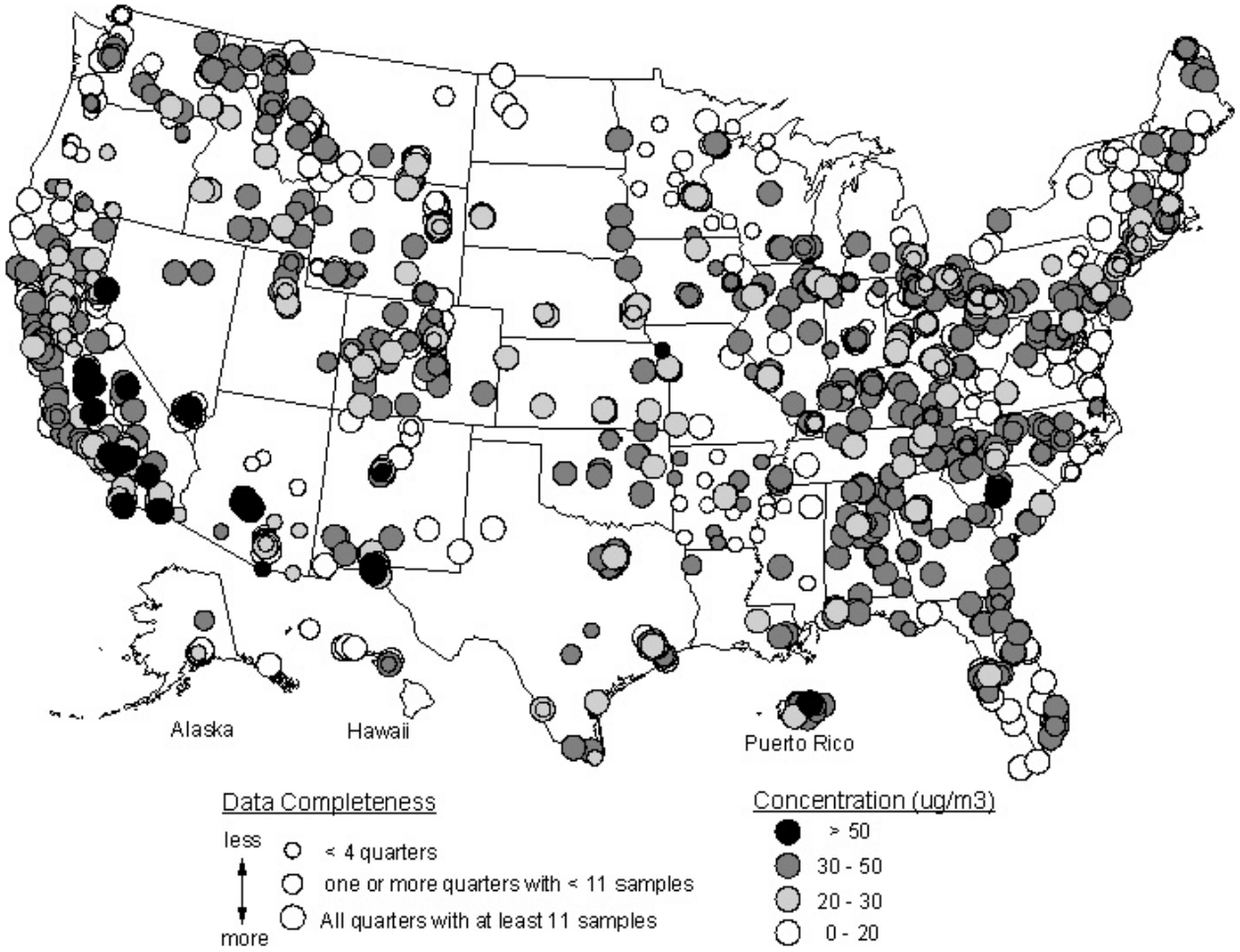


Figure 2-3a. 1999 annual mean PM_{10} concentrations (µg/m^3)

Source: Fitz-Simons et al. (2000)

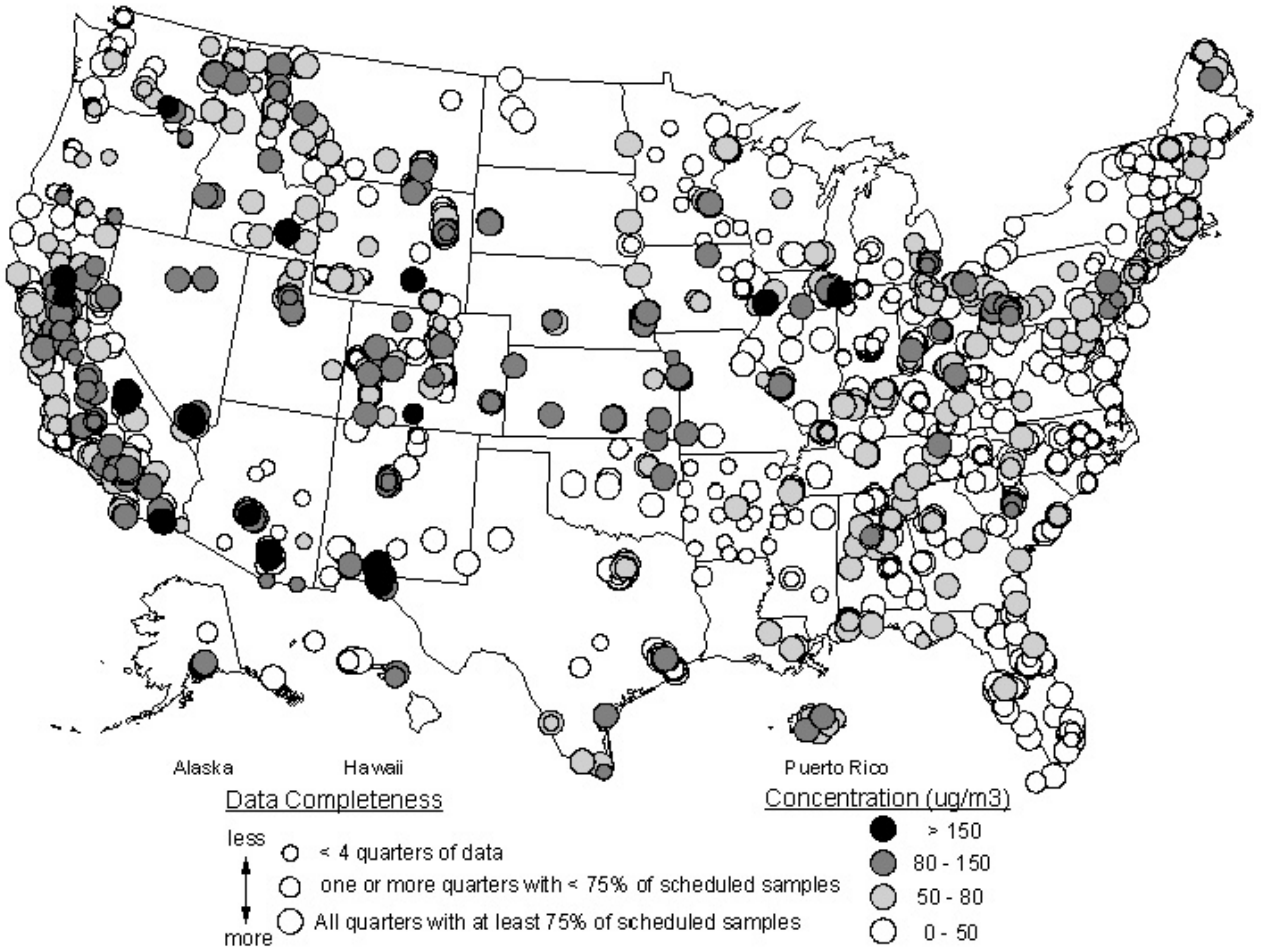
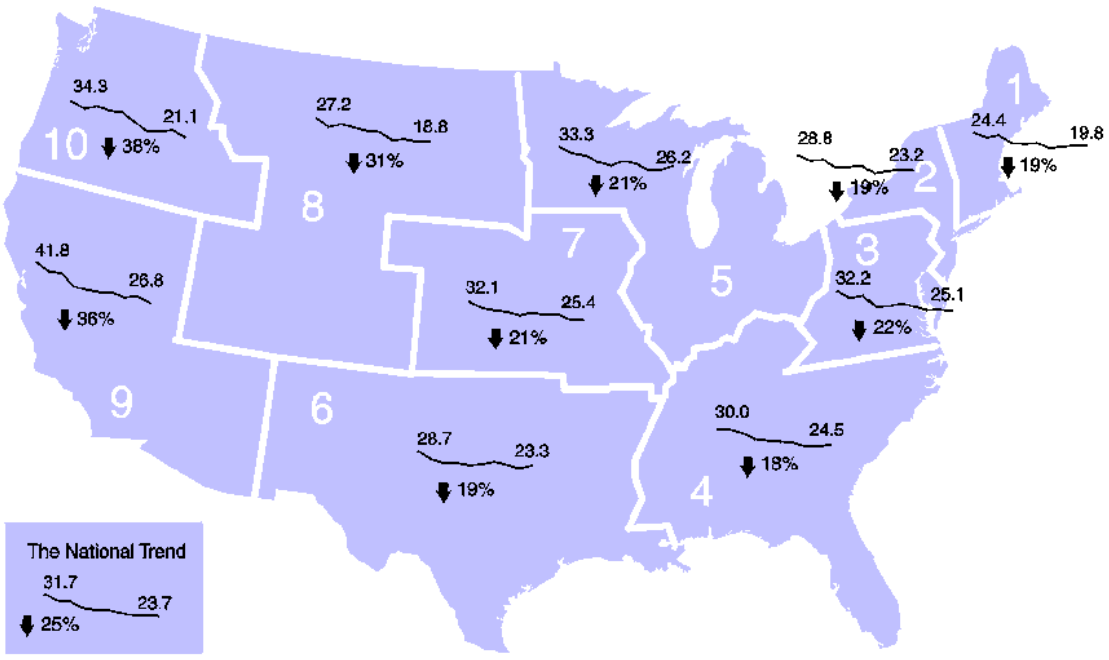


Figure 2-3b. 1999 2nd highest 24-hour average PM_{10} concentrations (µg/m^3)

Source: Fitz-Simons et al. (2000)



Alaska is in EPA Region 10; Hawaii, EPA Region 9; and Puerto Rico, EPA Region 2.
Concentrations are µg/m3.

Figure 2-4. Trend in annual mean PM_{10} concentrations by EPA region, 1989-1998 (µg/m^3).

Source: Environmental Protection Agency (2000b)

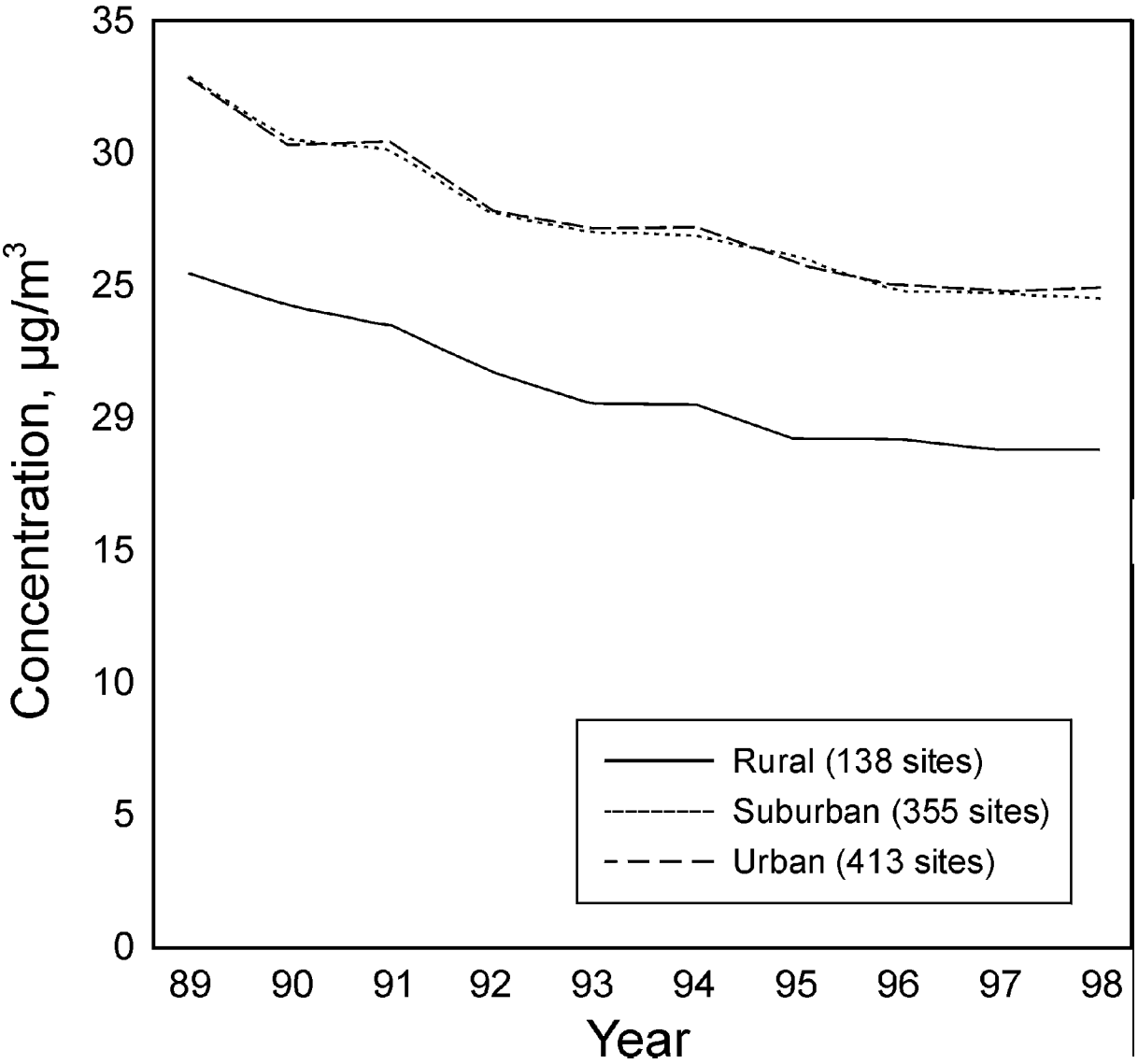


Figure 2-5. Nationwide trend in annual mean PM_{10} concentrations for rural, suburban, and urban locations from 1989 through 1998.

Source: Environmental Protection Agency (2000b)

2.4.2 $PM_{2.5}$

Following the 1997 PM NAAQS revisions, which set a new NAAQS for $PM_{2.5}$, EPA led a nationwide effort to deploy and operate over 1000 $PM_{2.5}$ monitors. These monitors use the Federal Reference Method (FRM), which if followed assures that PM data are collected using standard equipment, operating procedures, and data handling techniques.[8] The first year of data collected by that network has been analyzed by Fitz-Simons et al. (2000). About 54 percent of the monitors had fewer than 11 valid samples recorded in every quarter, the minimum number generally required for calculating quarterly means.[9]

Figure 2-6a depicts nationwide annual mean $PM_{2.5}$ concentrations from the FRM network. Many locations in the eastern U.S. and in California were above 15 $\mu g/m^3$. Annual mean concentrations were above 20 $\mu g/m^3$ in several major urban areas throughout the eastern U.S., including Pittsburgh, Cleveland, Atlanta, Chicago, St. Louis, and in Los Angeles and the central valley of California. Sites in the central and western mountain regions of the U.S. had generally low annual mean concentrations, most below 10 $\mu g/m^3$.

Figure 2-6b depicts nationwide 98^{th} percentile 24-hour average $PM_{2.5}$ concentrations from the FRM monitor network. Concentrations above 65 $\mu g/m^3$ were relatively rare in the eastern U.S., but more prevalent in California. Values in the 40 - 65 $\mu g/m^3$ range were more common in the eastern U.S. and on the west coast, but relatively rare in the central and western mountain regions. In these regions, the 98^{th} percentile 24-hour average concentrations were more typically below 40 $\mu g/m^3$, with many below 30 $\mu g/m^3$.

There are limited data available on longer-term trends in $PM_{2.5}$ concentrations. Long-term $PM_{2.5}$ data collected by the California Air Resources Board show that from 1990 to 1995 annual average $PM_{2.5}$ concentrations decreased about 50% in the South Coast Air Basin, 35% in the San Joaquin Valley, 30% in the San Francisco Bay Area, and 35% in the Sacramento Valley (Dolislager and Motallebi, 1999). $PM_{2.5}$ data also have been collected continuously since 1994 as part of a children's health study in twelve communities in southern California (Taylor et al.,

[8] See 40 CFR Parts 50 and 58 for monitoring program requirements.

[9] See 40 CFR Part 50, Appendix N, Section 2.0 Comparisons with the $PM_{2.5}$ standards.

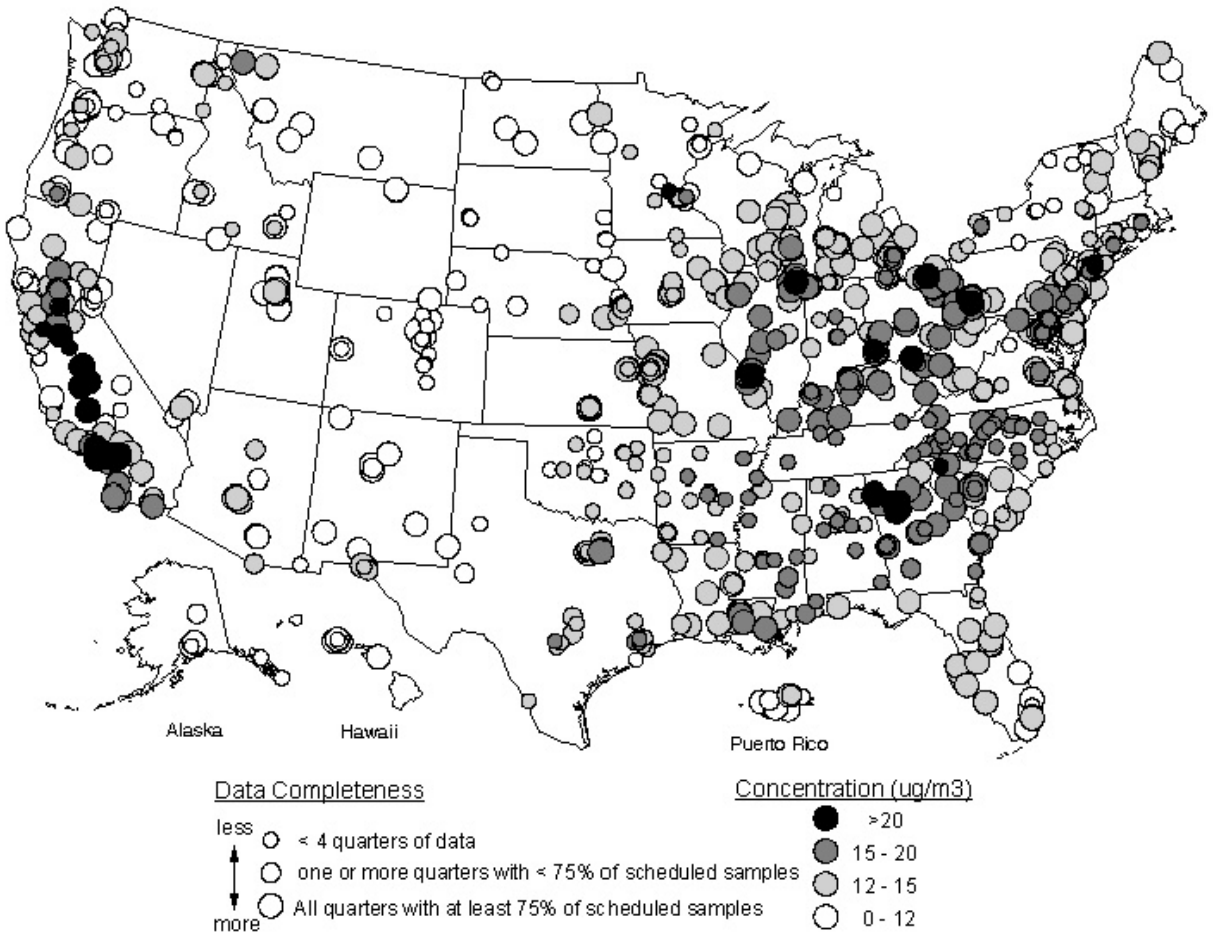


Figure 2-6a. 1999 annual mean $PM_{2.5}$ concentrations (µg/m^3)

Source: Fitz-Simons et al. (2000)

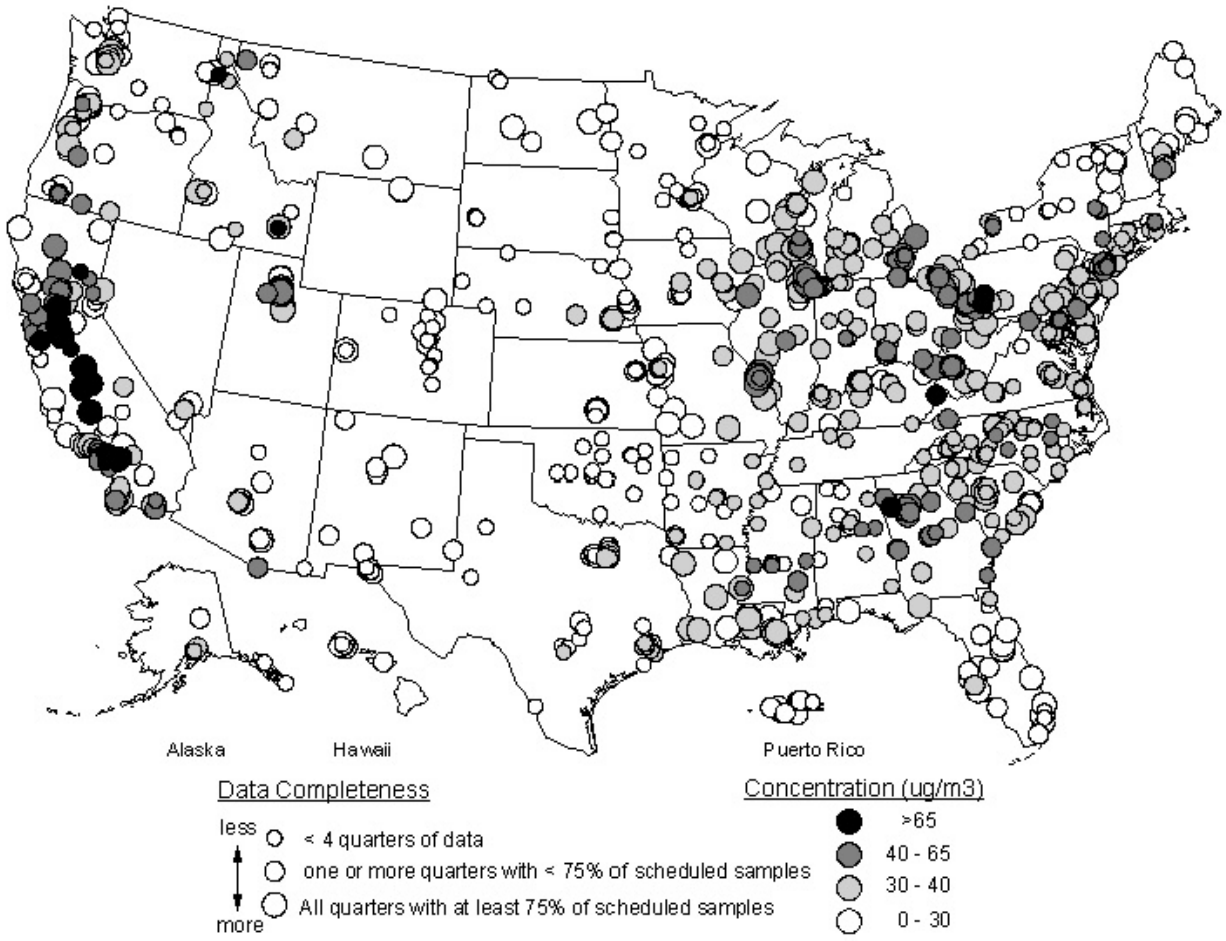


Figure 2-6b. 1999 98th percentile 24-hour average $PM_{2.5}$ concentrations (µg/m^3)

Source: Fitz-Simons et al. (2000)

1998). Data collected in this study from 1994 to 1998 at all sites show decreases in $PM_{2.5}$ ranging from 2% at Santa Maria to 37% at San Dimas/Glendora.

The IMPROVE monitoring network, which consists of sites located primarily in national parks and wilderness areas throughout the U.S., provides $PM_{2.5}$ trends for generally rural areas. Figures 2-7a and 2-7b show the 10 year trend from 1989-1998 at 10 eastern and 24 western IMPROVE sites.[10] At the eastern sites, measured $PM_{2.5}$ decreased about 9 percent from 1992 to 1995, but increased about 12 percent from 1995 to 1998. At the western sites $PM_{2.5}$ decreased 11 percent from 1989 to 1998. The trend for a single urban IMPROVE site located in Washington, D.C. is shown in Figure 2-7c. At that site, $PM_{2.5}$ concentrations increased about 26 percent from 1990 to 1993, then decreased about 23 percent from 1993 to 1995. The 1997 concentration was about 5 percent lower than the 1989 level.

As discussed in Section 2.2.4, fine-mode particles are likely to be more uniformly dispersed at urban scales than coarse-mode particles. Analyses of 1999 $PM_{2.5}$ FRM monitoring data from four large metropolitan areas indicate that multiple sites in these urban areas were highly correlated throughout the year. More than 75 percent of the between-site correlation coefficients in Atlanta, Detroit, Phoenix, and Seattle were greater than 0.85 (CD, p. 3-29). In separate studies, similar results were found in Philadelphia during the summers of 1993 and 1994 (CD, p. 3-28).

2.4.3 $PM_{10-2.5}$

$PM_{10-2.5}$ is a measure of the coarse-mode fraction of PM_{10}, and can be measured by a dichotomous sampler, or by using a difference method with collocated monitors under the same sampling protocol. A nationwide network of samplers using these methods is not available. However, an approximation of $PM_{10-2.5}$ can be made using a difference method on same-day data collected in 1999 from PM_{10} and $PM_{2.5}$ FRM monitors in the same physical location. Since the protocol for each monitor is not identical, the results should be viewed with caution. A more complete and accurate view of $PM_{10-2.5}$ values can be obtained by nationwide deployment of

[10] The lines on these figures showing the trend in PM components is discussed in Section 2.4.5.

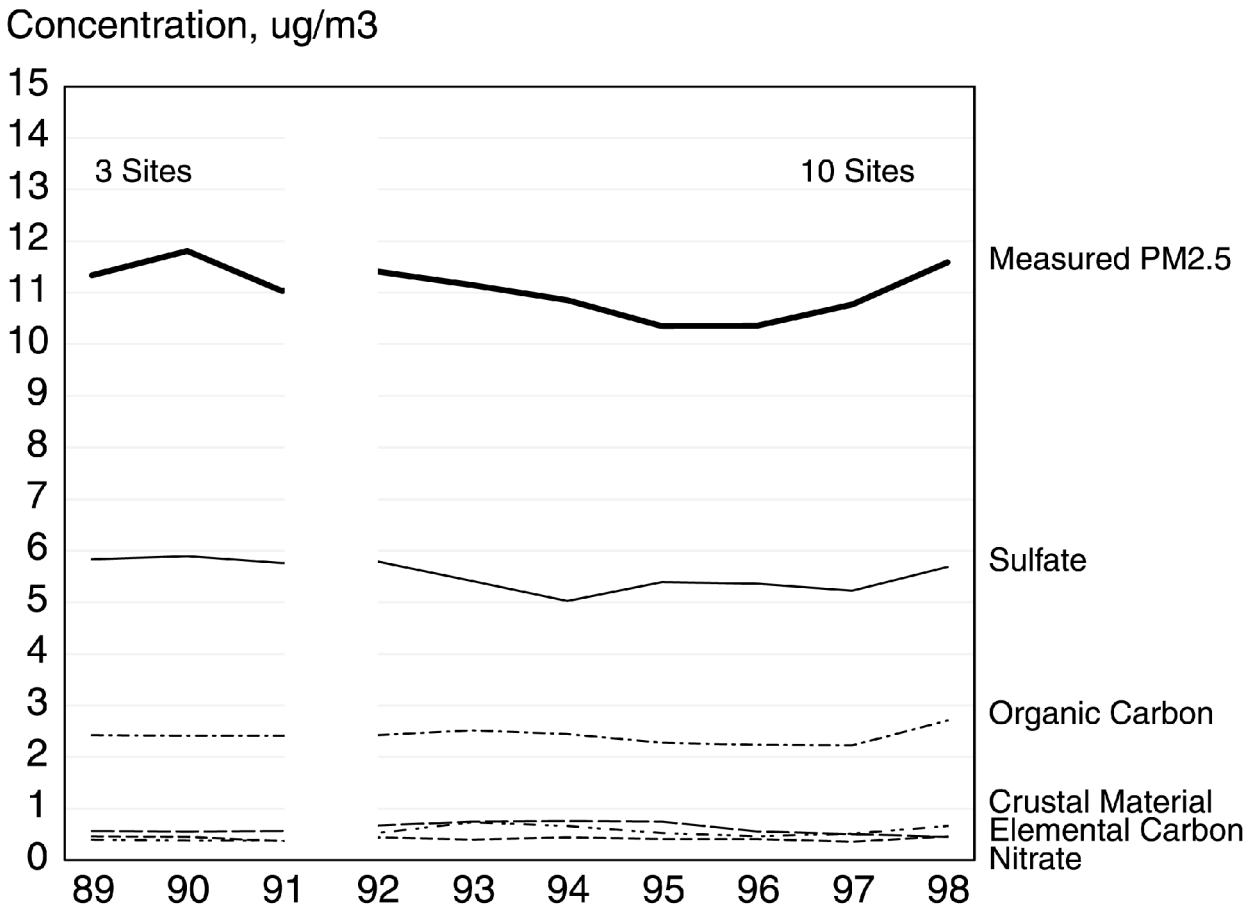


Figure 2-7a. $PM_{2.5}$ Concentrations, 1989-1998 at eastern IMPROVE sites

Source: U.S. Environmental Protection Agency (2000b)

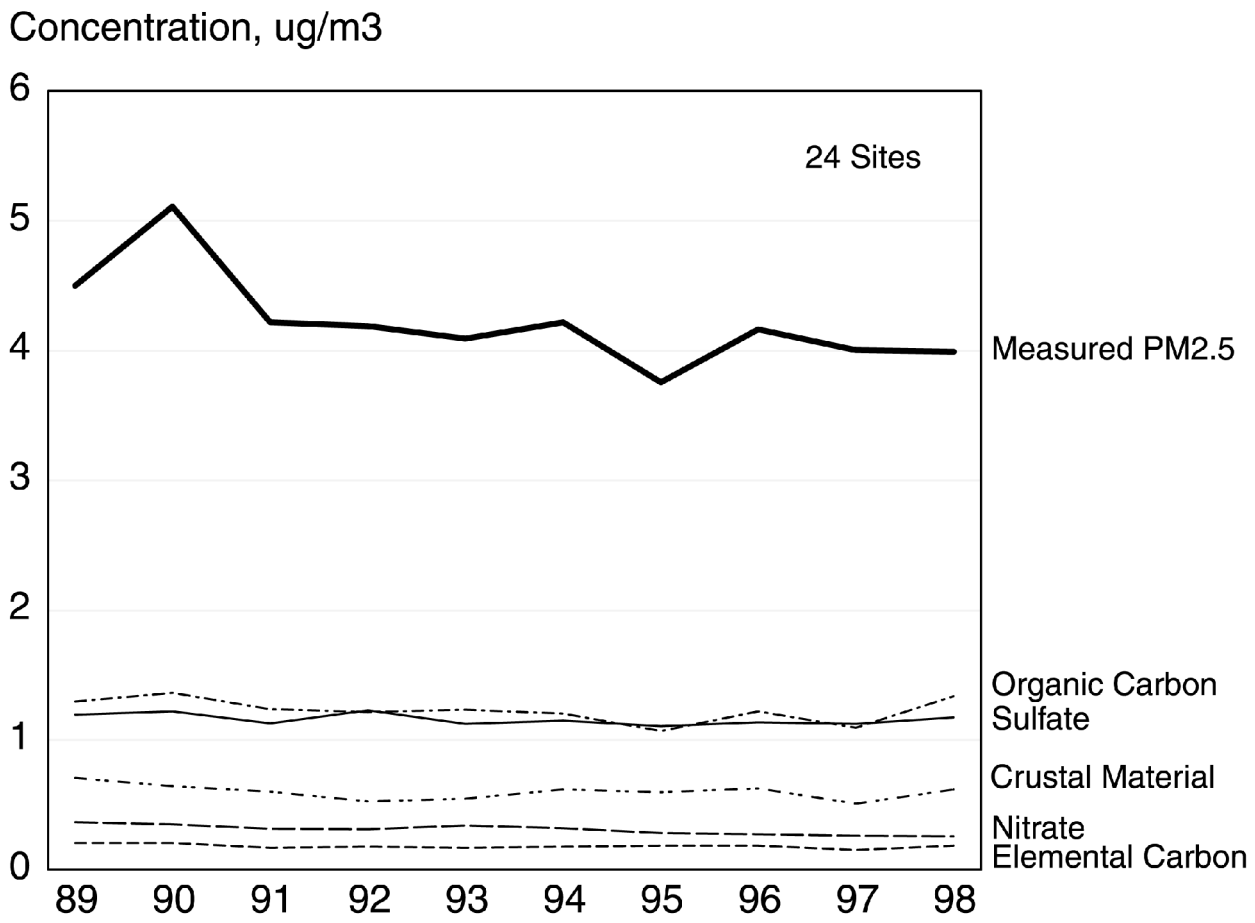


Figure 2-7b. $PM_{2.5}$ Concentrations, 1989-1998 at western IMPROVE sites

Source: U.S. Environmental Protection Agency, (2000b)

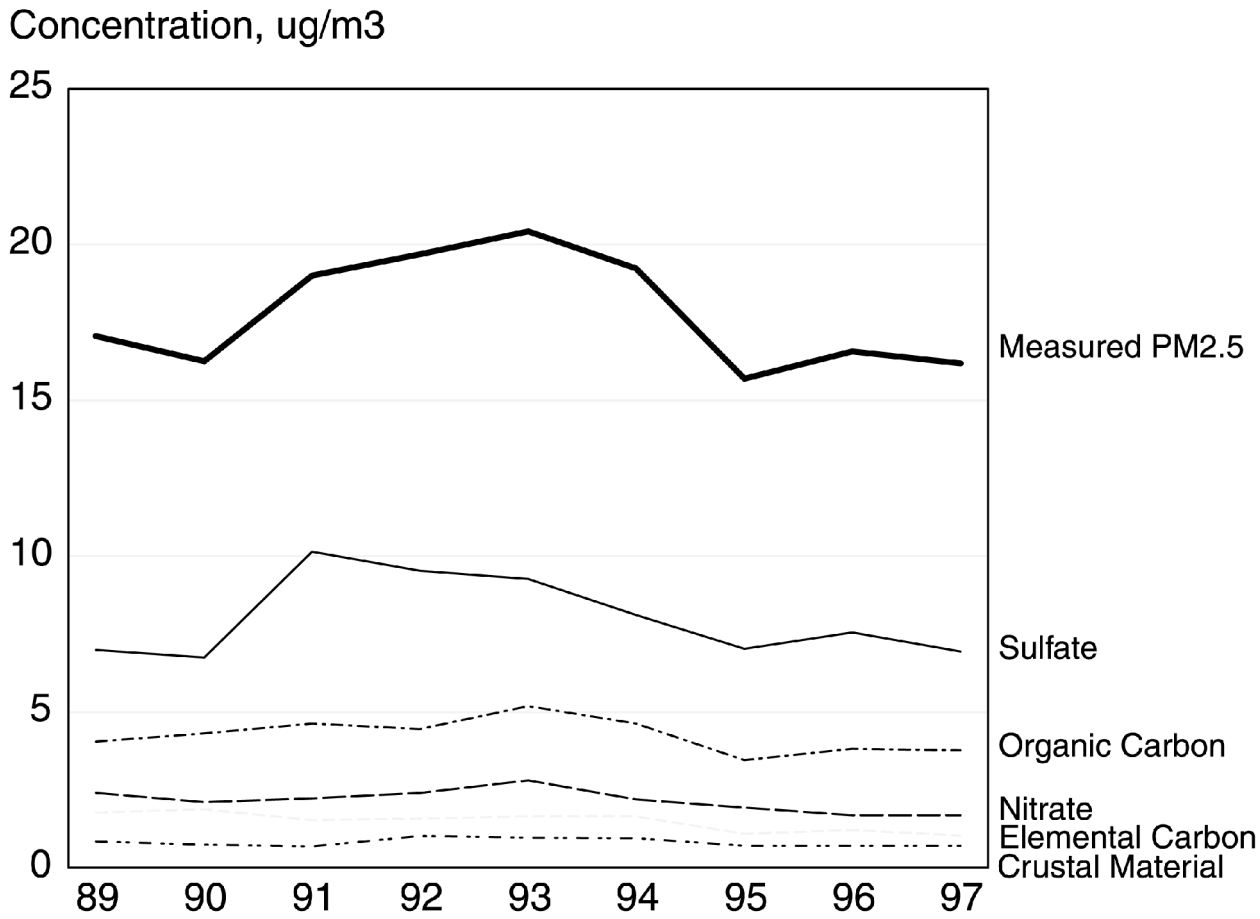


Figure 2-7c. $PM_{2.5}$ Concentrations, 1989-1997 at the Washington, D.C. IMPROVE site

Source: U.S. Environmental Protection Agency (2000b)

collocated PM_{10} and $PM_{2.5}$ monitors that use an equivalent monitoring protocol.

Figure 2-8a shows estimated annual mean $PM_{10-2.5}$ and Figure 2-8b shows the estimated 98th percentile 24-hour average $PM_{10-2.5}$ developed from 1999 FRM monitor data. Since there are currently no data completeness requirements for $PM_{10-2.5}$, the completeness criteria shown in these figures was chosen simply to be consistent with the previous PM_{10} and $PM_{2.5}$ maps. Similarly, since there is no standard for $PM_{10-2.5}$, the annual mean and 98th percentile 24-hour average values were chosen for consistency with the $PM_{2.5}$ maps. The limited data show that annual mean concentrations vary widely, with higher concentrations in several areas of the midwestern U.S. and southern California. A similar pattern emerges for the estimated 98th percentile 24-hour average $PM_{10-2.5}$ concentrations. The southeastern U.S. data are relatively incomplete, but preliminary estimates suggest relatively low $PM_{10-2.5}$ levels throughout that region.

2.4.4 Ultrafine Particles

There are no nationwide monitoring networks for ultrafine particles (< 0.1 μm), and only a few recent published studies of ultrafine particle counts in the U.S. At an urban site in Atlanta, Georgia, particles in three size classes were measured on a continuous basis between August 1998 and August 1999. The classes included ultrafine particles in two size ranges, 0.003 to 0.01 μm and 0.01 to 0.1 μm, and a subset of accumulation-mode particles in the range of 0.1 to 2 μm (Woo et al., 2000). Figure 2-9 shows the annual average number and volume concentrations for these three size classes. The vast majority, 89%, of the number of particles were in the ultrafine mode (smaller than 0.1 μm), but 83% of the particle volume was in the subset of accumulation-mode particles. The researchers found that for particles up to 2 μm there was little evidence of any correlation between number concentration and either volume or surface area. This suggests that fine-mode particle mass, which arises primarily from particles larger than ultrafines, does not correlate well with particle number, which is dominated by particles in the ultrafine mode.

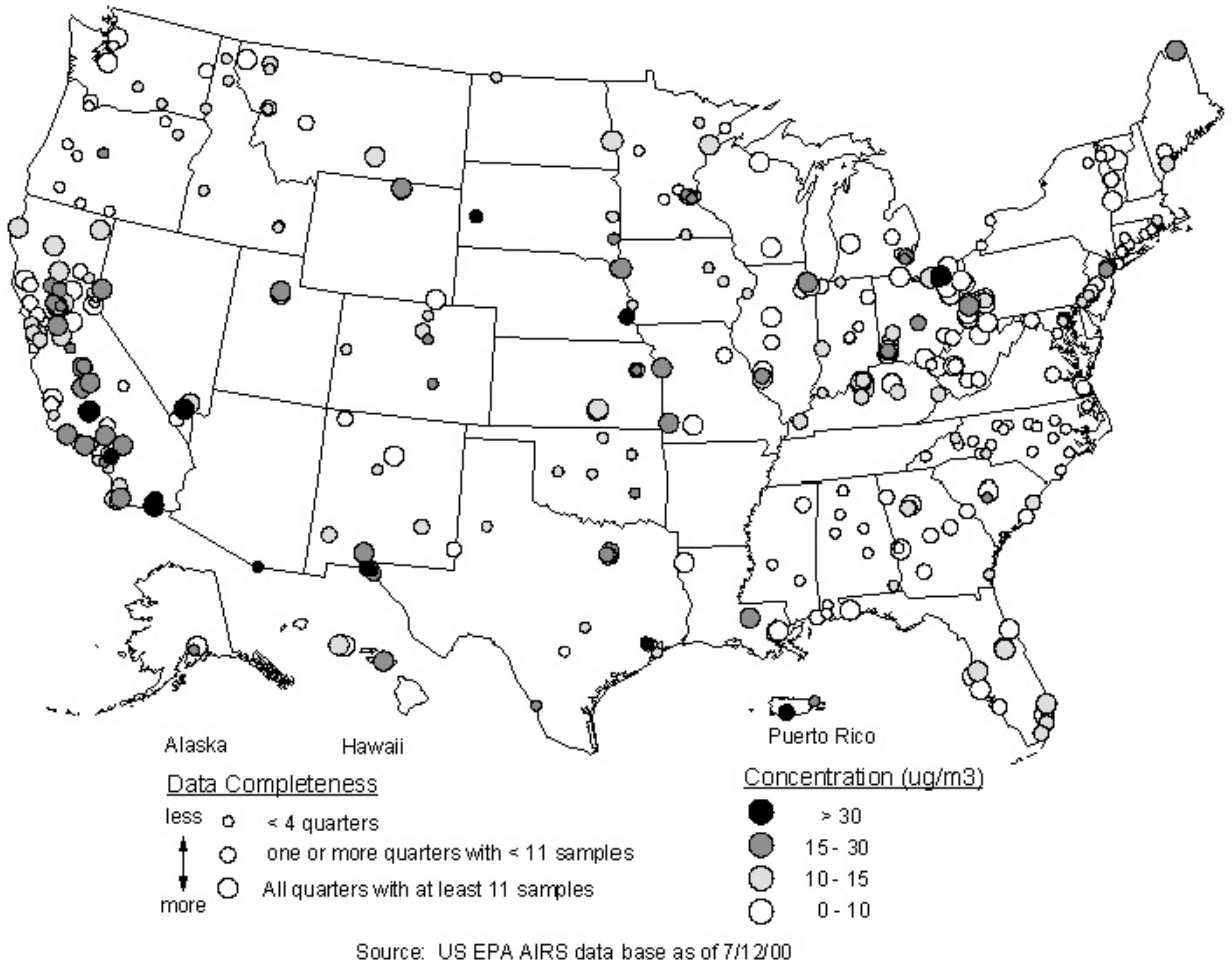


Figure 2-8a. 1999 estimated annual mean $PM_{10-2.5}$ concentrations ($\mu g/m^3$)

Source: Fitz-Simons et al. (2000)

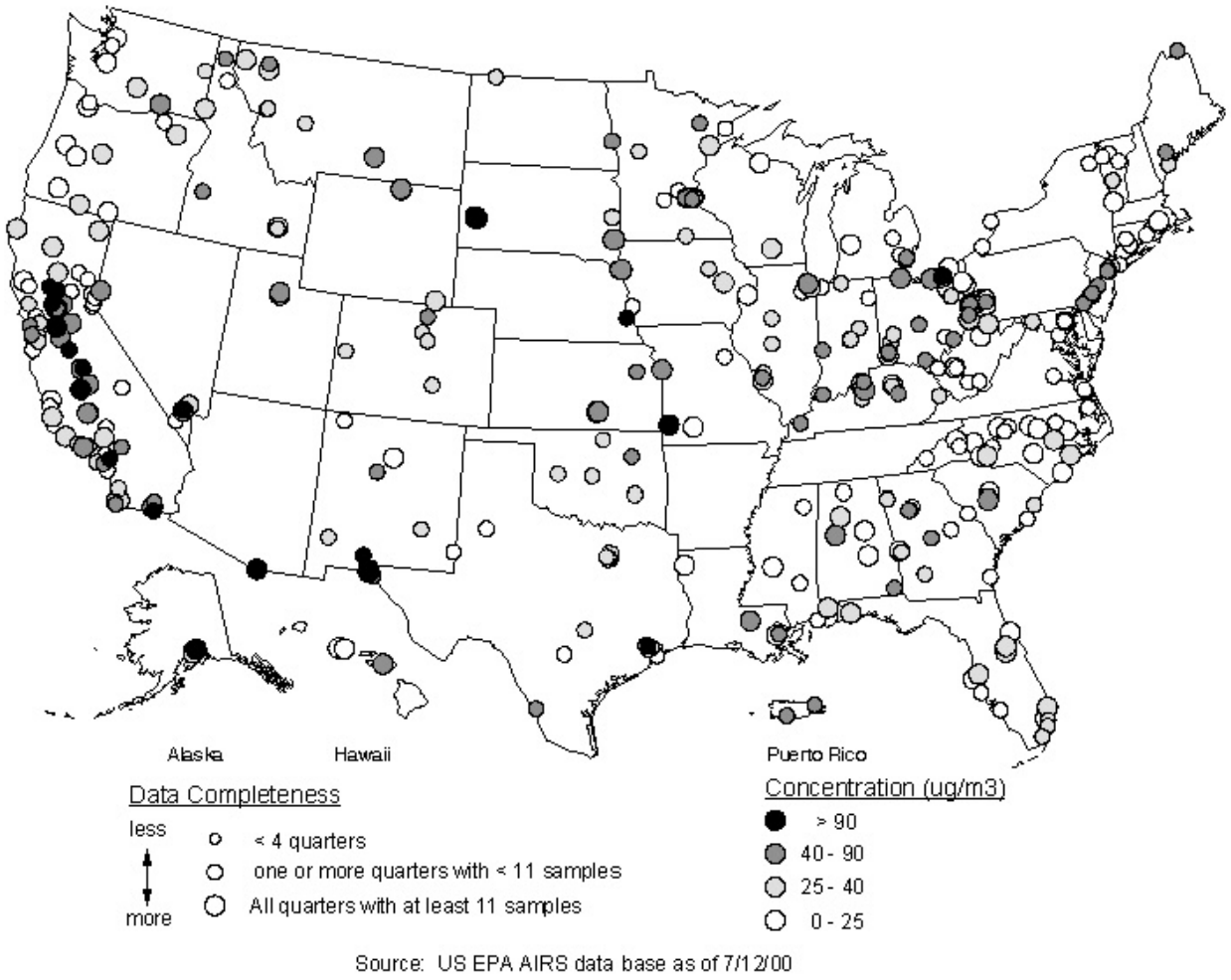


Figure 2-8b. 1999 estimated 98th percentile 24-hour average $PM_{10-2.5}$ concentrations (µg/m³)

Note: The circle sizes on this map indicating the relative number of data points used to generate the estimates are not entirely accurate. The values, however, are accurate. A new map with revised completeness indicators is being generated.

Source: Fitz-Simons et al. (2000)

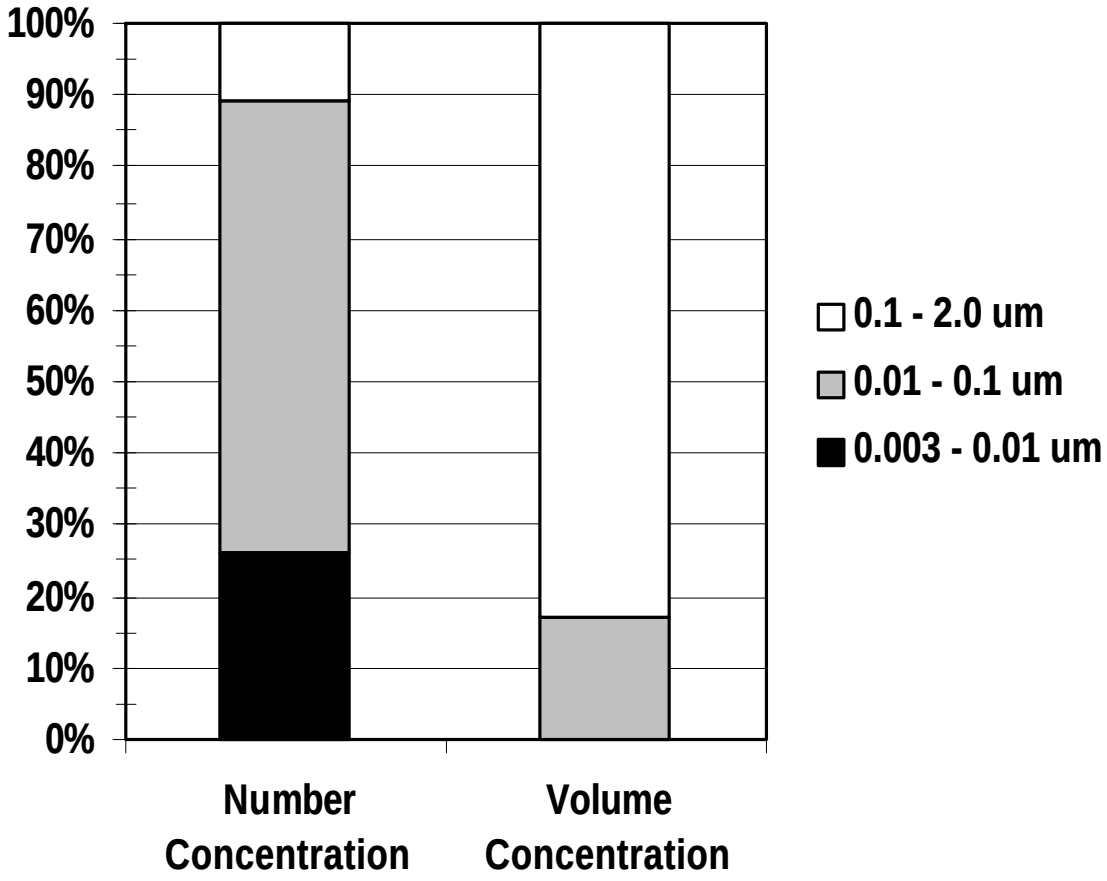


Figure 2-9. Yearly average fractions of fine (0.1–2.0 µm) and ultrafine (0.003–0.01 µm) particle number and volume concentrations in Atlanta

2.4.5 Components of PM

Atmospheric PM contains many different chemical components that vary by location, time of day, and time of year. The 1996 CD and Staff Paper provided indications of regional composition differences based on data from short-term urban studies and the predominantly rural IMPROVE network. More recent data appears consistent with earlier findings. Table 2-3 shows typical annual average fine fraction mass apportionment among chemical components in the eastern and western U.S. In general, eastern U.S. fine-mode particles are dominated by sulfate, and to a lesser extent by organic carbon. Western U.S. fine-mode particles appear to have a greater proportion of organic carbon, nitrate, and crustal material.

Table 2-3. Gross Annual Average Chemical Composition of $PM_{2.5}$ Particles Obtained in Rural Areas of the Eastern and Western U.S. by the IMPROVE Network and in Mixed Rural, Suburban, and Urban Areas Obtained by Studies Summarized in the 1996 PM Criteria Document

	IMPROVE		1996 PM AQCD	
	Eastern US	Western US	Eastern US	Western US
	% Contribution		% Contribution [a]	
$SO_4^=$	56	33	44	11
EC	5	6	5	14
OC	27	36	27	38
NO_3	5	8	1	15
Crustal	7	17	6	14
	Reconstructed $PM_{2.5}$ Concentration ($\mu g/m^3$)		$PM_{2.5}$ Concentration ($\mu g/m^3$)	
$PM_{2.5}$	11.0	3.9	31.0	37.3

[a] Note that contributions do not add to 100% due because a portion of the measured total mass was not chemically characterized.
Sources: IMPROVE network – EPA (2000a), 1996 PM Criteria Document – EPA (1996a)

Trends in remote area concentrations of PM components, generated with data from the IMPROVE network, are shown in Figures 2-7a and 2-7b. All of the components have shown variability of less than 1 $\mu g/m^3$ over the ten year period from 1989 to 1998. At the eastern sites sulfate appeared to be declining until 1994, but has risen again in recent years. In 1998 organic

carbon was at its highest level over the 10 year period.[11] Data from the urban IMPROVE site in Washington, D.C., shown in Figure 2-7c, indicates that all the components were lower in 1997 than at the their peaks during the preceding 8 years. In 1997 sulfate is about 3 $\mu g/m^3$ lower than its 1991 peak of just over 10 $\mu g/m^3$.

Data collected from 1994 to 1998 as part of a children's health study in twelve communities in southern California also indicate decreases in major identified components such as nitrate, sulfate, ammonium, and acids (Taylor et al., 1998). However, the undefined components indicated a mixed pattern of increases and decreases at the same sites. A similar downward trend was observed from 1978 to 1995 in nitrate and sulfate concentrations at sites in North Long Beach and Riverside, California (Dolislager and Motallebi, 1999).

2.4.6 Relationships Among $PM_{2.5}$, PM_{10}, and $PM_{10-2.5}$

In this section, new information from the nationwide $PM_{2.5}$ FRM monitoring network on the relationship among PM indicators in different regions is presented. Figure 2-10 shows the distribution of 1999 ratios of $PM_{2.5}$ to PM_{10} at sites in different geographic regions. The ratios are highest in the eastern U.S. regions with median ratios from 0.64 to 0.69, and lowest in the Southwest region, with a median ratio of 0.39. These data appear to be generally consistent with earlier findings from a more limited set of sites reported in the 1996 CD.

Correlations among pollutant indicators can provide insights into how well one indicator can represent the variability in another indicator. For instance, in some areas PM_{10} may serve as a good indicator of $PM_{2.5}$. Figure 2-11 shows the results of a nationwide analysis of the urban area correlations among PM size fractions using 1999 24-hour average data from the FRM monitoring networks. PM_{10} and $PM_{2.5}$ measured on the same days at collocated sites are fairly well correlated in most parts of the country with the lowest correlations in the Upper Midwest and Southwest. As might be expected from their differences in origin, composition, and behavior, fine-fraction mass ($PM_{2.5}$) is generally not well correlated with coarse-fraction mass

[11] Unidentified PM components are an important part of total measured PM mass, and affect the year to year variability in the mass trend. For example, in Figure 2-7b, the upward spike in 1990 and the downward spike in 1995 are dominated by changes in the unidentified fraction.

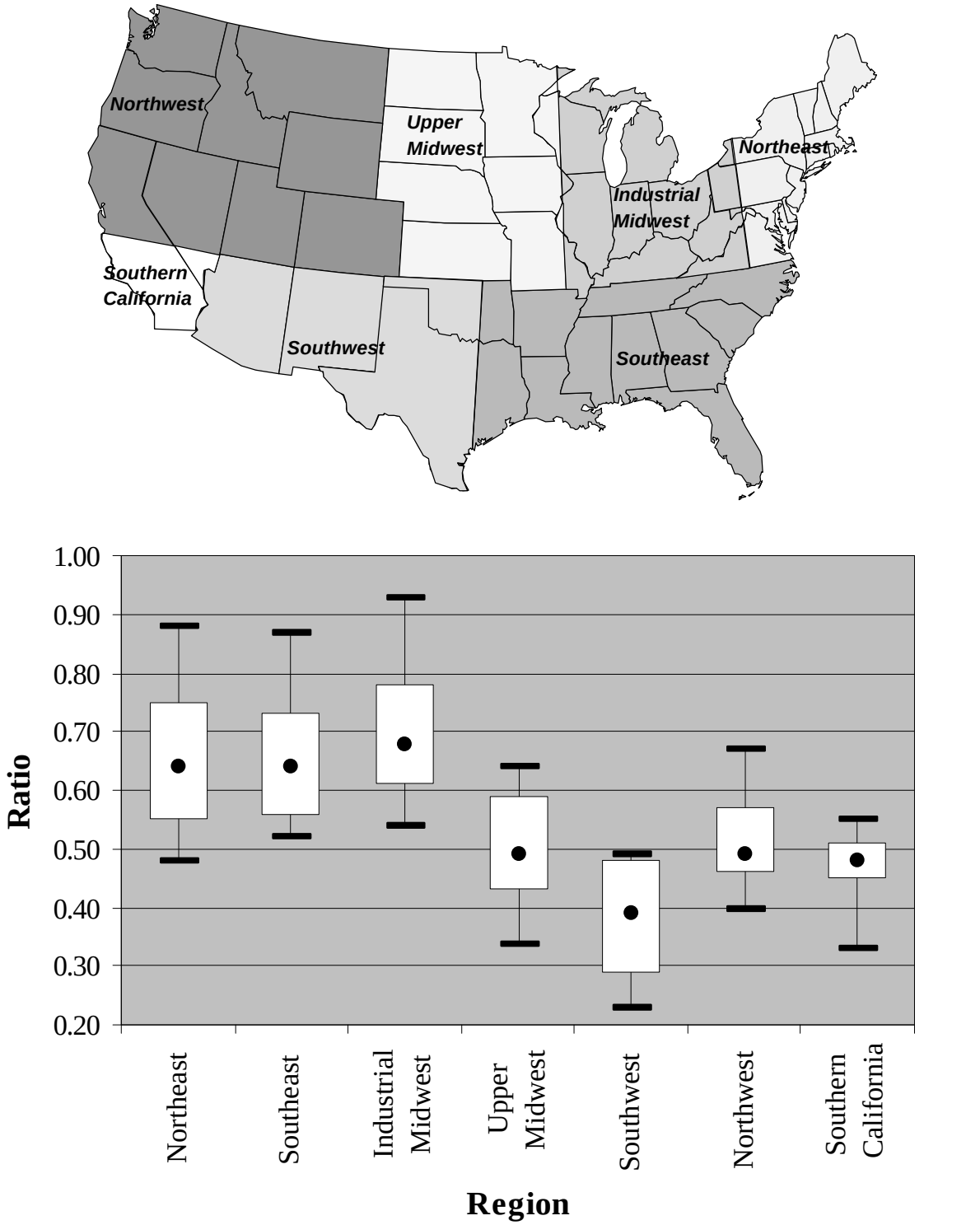


Figure 2-10. Distribution of Ratios of $PM_{2.5}$ to PM_{10} by Region. Box represents upper and lower quartiles of the distribution; whiskers represent 10th and 90th percentiles; black dot represents median.

Source: Adapted from Fitz-Simons et al. (2000), Attachment E

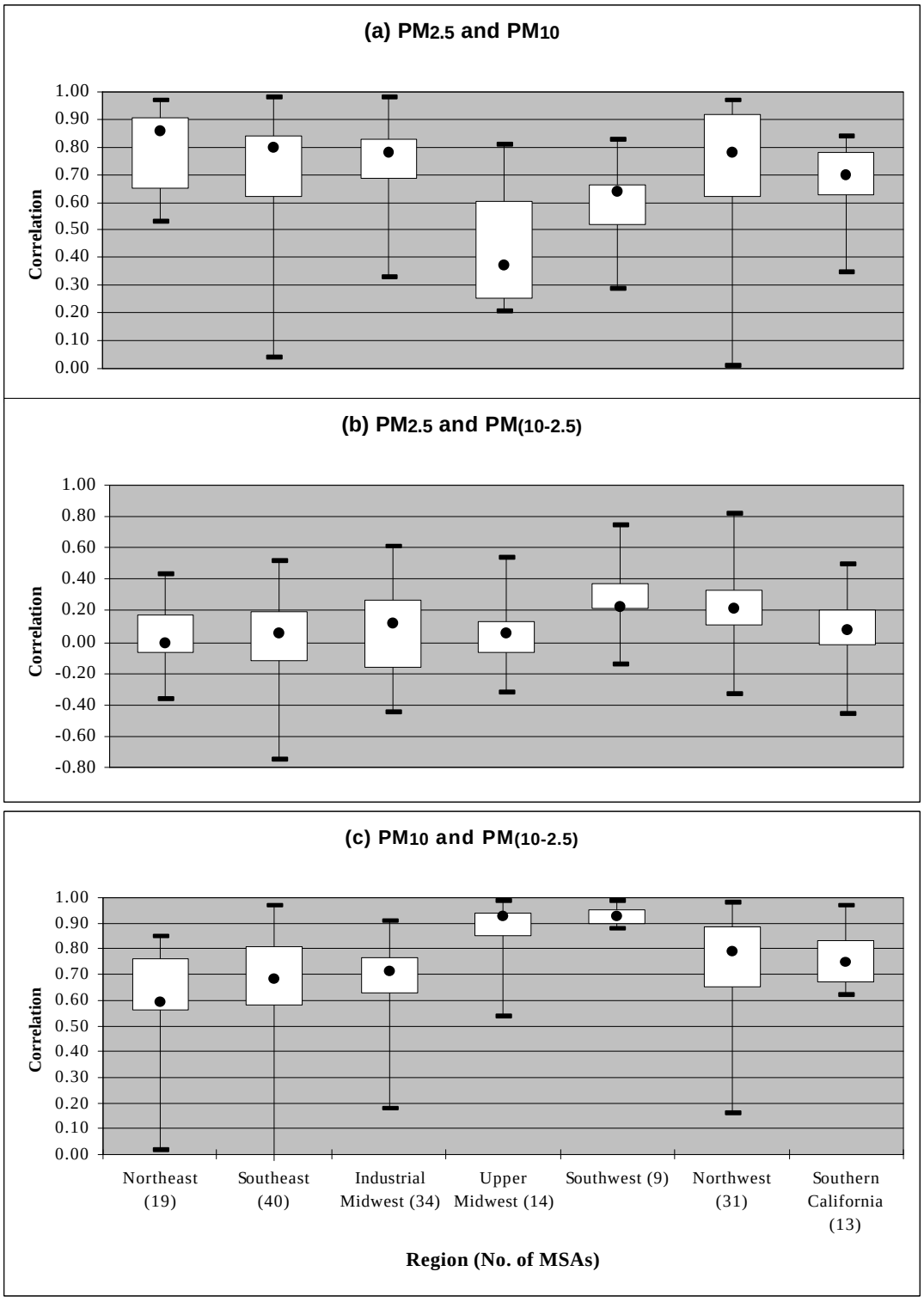


Figure 2-11. Distribution of Urban Area Correlations of 24-hour Average PM by Region. Box represents upper and lower quartiles of the distribution; whiskers represent minimum and maximum; black dot represents median.

Source: Adapted from Fitz-Simons et al. (2000), Attachment I

($PM_{10-2.5}$). In many cases the correlations are negative. The most consistently high positive correlations of $PM_{2.5}$ to $PM_{10-2.5}$ are in the Southwest, where the low ratio of $PM_{2.5}$ to PM_{10} suggests that crustal material makes a more significant contribution to $PM_{2.5}$ than in other regions. Finally, the correlation between $PM_{10-2.5}$ and PM_{10} is relatively high in all regions, ranging from 0.59 in the Northeast to 0.93 in the Upper Midwest and Southwest. The highest correlations appear in regions with low correlations between $PM_{2.5}$ and PM_{10}.

2.5 TEMPORAL PATTERNS IN PM CONCENTRATIONS

2.5.1 $PM_{2.5}$ Patterns

Data from the 1999 $PM_{2.5}$ FRM network analyzed by Fitz-Simons, et al. (2000) show distinct seasonal variation in average $PM_{2.5}$ concentrations. Readers should be cautioned that this analysis represents a single year of data, and that patterns may vary from year to year. The summaries in Figure 2-12a (urban) and Figure 2-12b (rural) show the distributions of monthly average concentrations in different geographic regions. The months with peak urban $PM_{2.5}$ concentrations vary by region. The urban areas in the eastern regions all show peaks in the summer months (June-August), and the western regions all show peaks in the late fall and winter months (November-January). In most regions the urban and rural patterns are similar, with $PM_{2.5}$ concentrations generally lower in rural areas. However, Southern California urban and rural monitors show different seasonal patterns, with urban winter peaks not present in rural areas. Also, in the Northwest the rural winter peak is not as pronounced as it is in urban areas.

Using data from a limited number (31) of continuous non-FRM $PM_{2.5}$ monitors, Fitz-Simons et al. (2000) summarized diurnal patterns in $PM_{2.5}$ concentrations. Caution should be used in interpreting data from continuous methods, which can produce significant artifacts related to semi-volatile components (CD, p. 3-22). Figure 2-13 shows the 1999 annual hourly average distribution summary for monitors in each region. In most regions the figure shows a cycle of elevated $PM_{2.5}$ levels between 6:00 a.m. and 9:00 a.m., and again in the evening hours

Figure 2-12a.
1999 Monthly Average Urban $PM_{2.5}$ Distributions by Region. Box represents interquartile range; plus sign is the mean; box line is the median.

Source: Fitz-Simons et al. (2000)

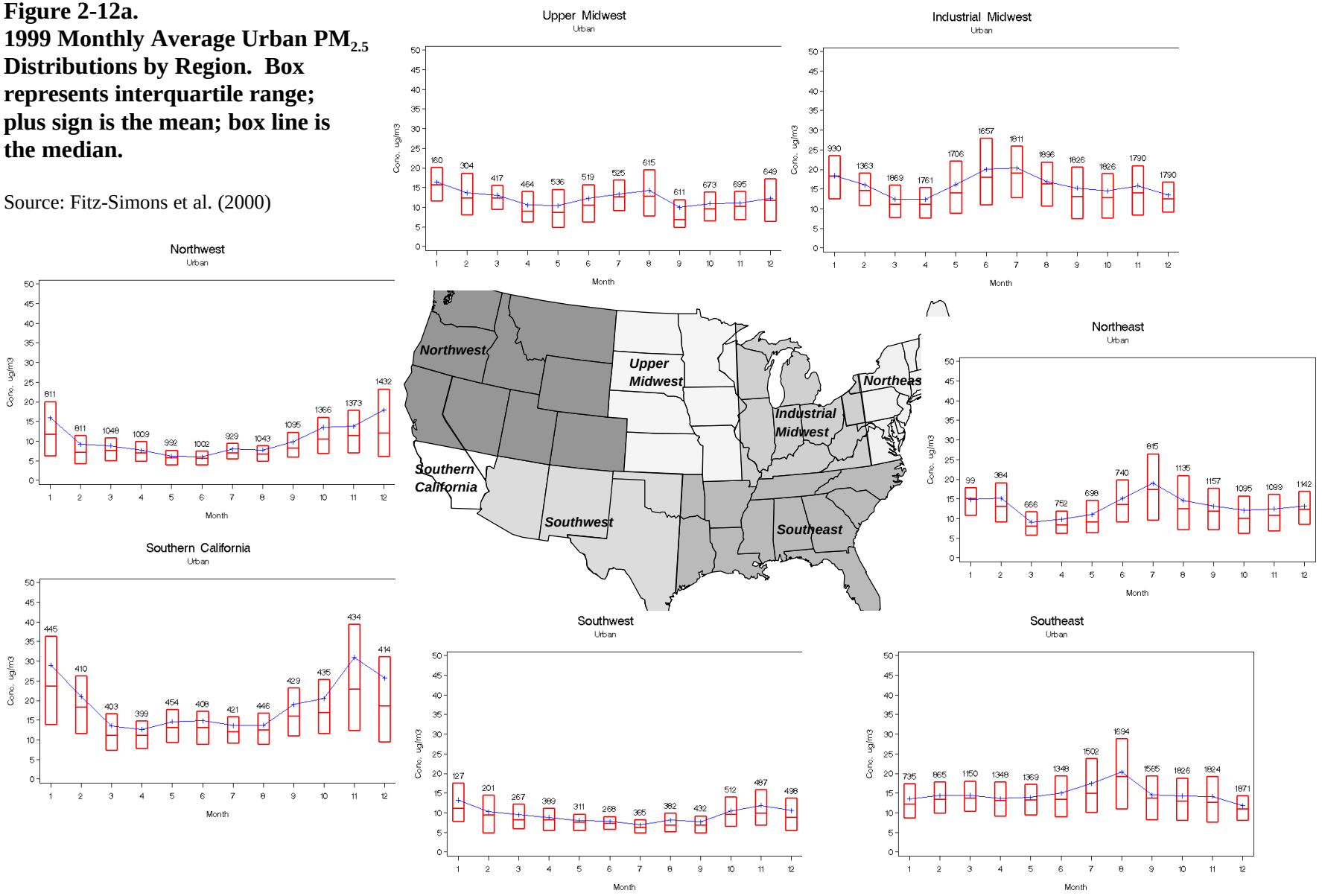


Figure 2-12b.
1999 Monthly Average Rural $PM_{2.5}$ Distributions by Region. Box represents interquartile range; plus sign is the mean; box line is the median.

Source: Fitz-Simons et al. (2000)

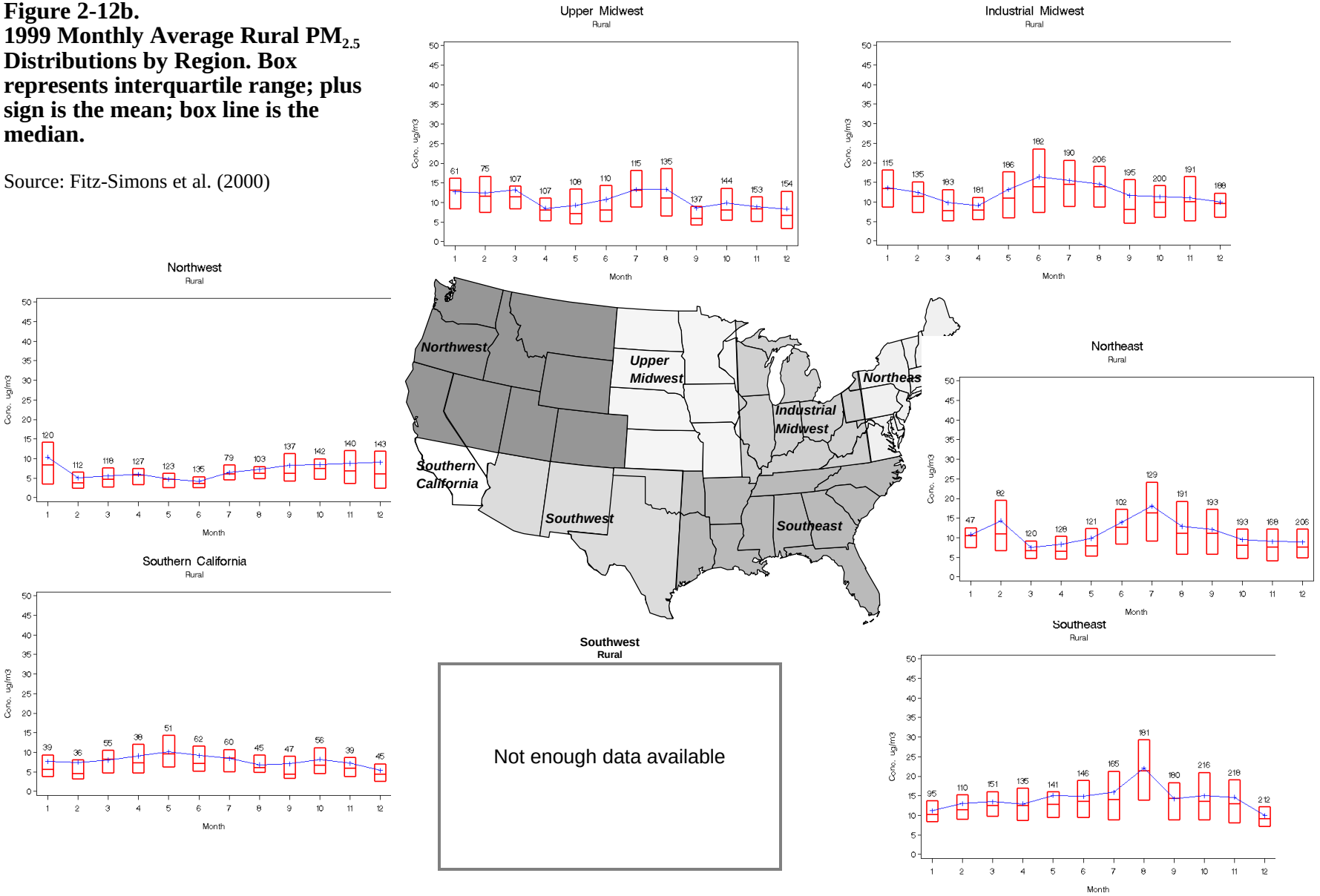
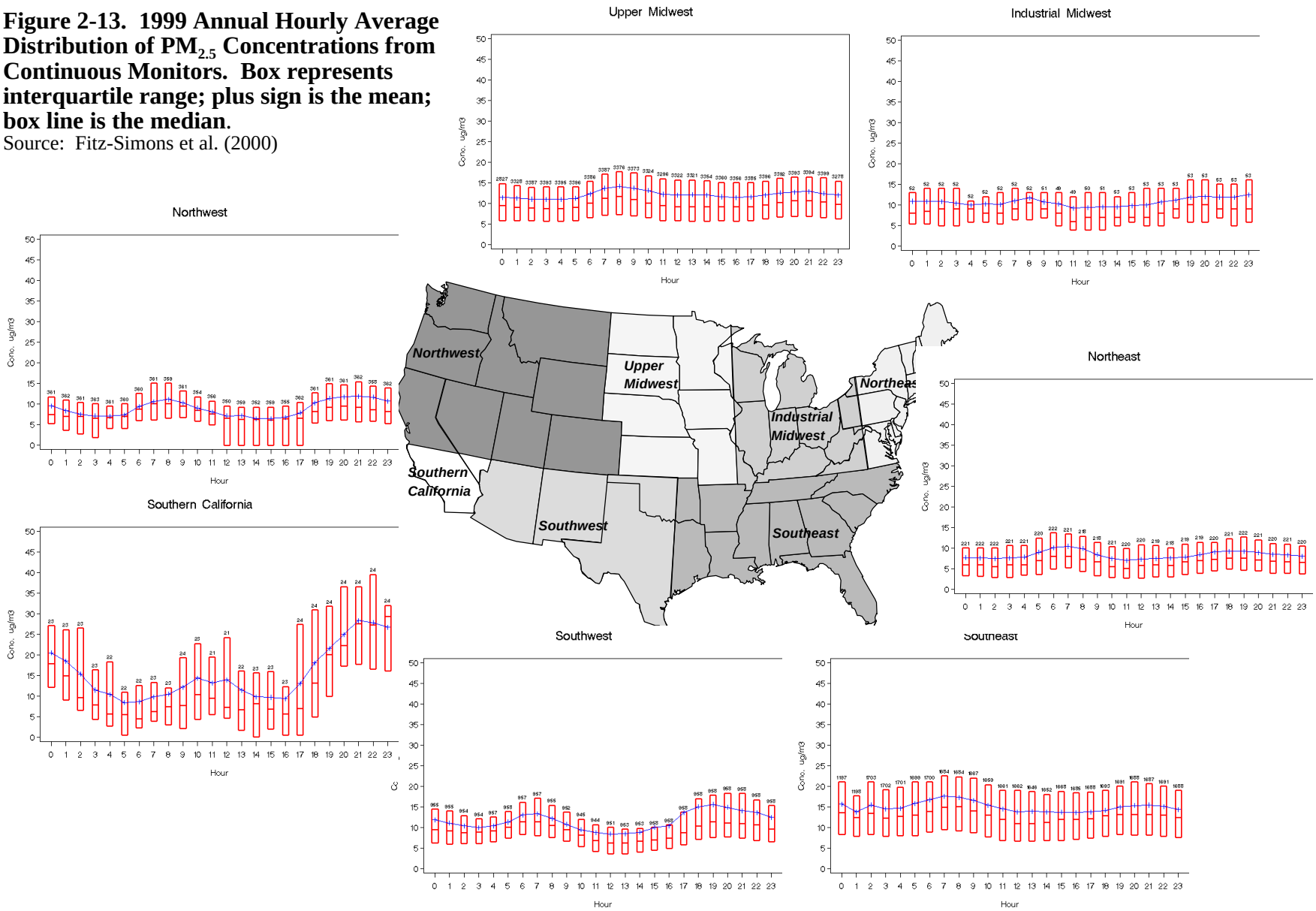


Figure 2-13. 1999 Annual Hourly Average Distribution of $PM_{2.5}$ Concentrations from Continuous Monitors. Box represents interquartile range; plus sign is the mean; box line is the median.
Source: Fitz-Simons et al. (2000)



starting around 6:00 p.m. However, there is significant variation in day-to-day profiles, as suggested in the box plots by the relatively large ratio of the interquartile range to the median. These cycles vary by location and by calendar quarter, and possibly by the type of monitor and monitor operating procedures.

The continuous monitors also provide some insight into short-term (e.g., hourly) increases in $PM_{2.5}$, which might be important to understanding associations between elevated PM levels and adverse health effects. The 1999 data in Figure 2-14 show the distribution of increases from one hour to the next in hourly average $PM_{2.5}$ concentrations. Typical increases (median) range from 0.8 $\mu g/m^3$ to 3.0 $\mu g/m^3$, and more atypical increases (95th percentile) range from 4.0 $\mu g/m^3$ to 16.4 $\mu g/m^3$. However, rare increases were observed to be an order of magnitude higher than this range.

2.5.2 Ultrafine Patterns

Few U.S. studies have extensively examined diurnal or seasonal patterns for ultrafine particles. At an urban site in Atlanta, Georgia, Woo et al. (2000) found that ultrafine particle number concentrations tend to be higher on weekdays than on weekends. Concentrations of particles in the range of 0.01 to 0.1 µm are higher at night than during the daytime, and tend to reach their highest values during morning rush hour. Smaller particles in the range of 0.004 to 0.01 µm were elevated during rush hour when temperatures were below 50°F. Several periods of relatively high ultrafine particle levels were observed during the year-long study period from August 1998 to August 1999, and SO_2 measurements show corresponding peaks during these periods.

2.6 PM BACKGROUND LEVELS

For the purposes of this document, background PM is defined as the distribution of PM concentrations that would be observed in the U.S. in the absence of anthropogenic, or man-made, emissions of primary PM and precursor emissions of VOC, NO_x, SO_2, and NH_3 in North America. Thus, background includes PM from natural sources and transport of PM from outside of North America. Estimating background concentrations is important for the health risk

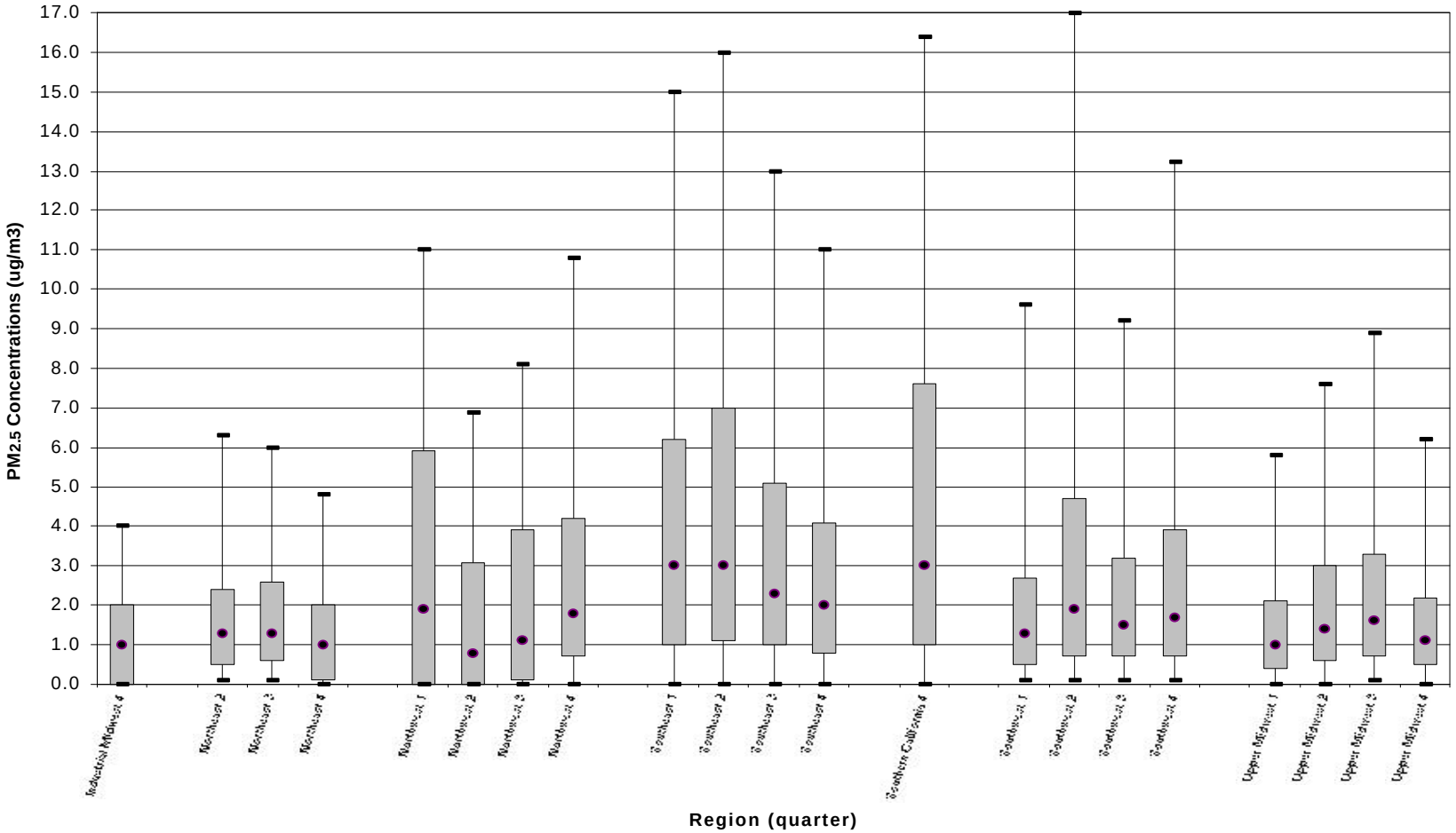


Figure 2-14. 1999 Quarterly Distribution of Hour-to-Hour Increases in Hourly Average $PM_{2.5}$ Concentrations at Continuous Monitors. Bar represents interquartile range; whiskers represent 5th and 95th percentiles; black dot represents the median.

Source: Adapted from Fitz-Simons et al. (2000), Appendix N

analyses presented in Chapter 5 and the assessment of ecosystem and visibility effects in Chapter 7. The draft CD does not provide any new conclusions about background concentration levels. However, it does discuss the increasing recognition and understanding of the long-range transport of PM from outside the U.S.

Background levels of PM vary by geographic location and season, and have a natural component and a human-made (anthropogenic) component. The natural background arises from: (1) physical processes of the atmosphere that entrain small particles (e.g., crustal material, sea salt spray); (2) volcanic eruptions (e.g., sulfates); natural combustion such as wildfires (e.g., elemental and organic carbon, and inorganic and organic PM precursors); and (4) the activities of wild animals and plants (e.g., fine organic aerosols, inorganic and organic PM precursors). The exact magnitude of the natural portion of PM for a given geographic location can not be precisely determined because it is difficult to distinguish local sources of PM from the long-range transport of anthropogenic particles and precursors.

PM can be transported long distances from natural events occurring outside the continental United States (CD, p. 3-44). The occurrence and location of these long-range transport events are highly variable and their impacts on the United States are equally variable. Several recent studies have focused on identifying the origin, sources, and impacts of recent transnational transport events.

- The transport of PM from biomass burning in Central America and southern Mexico in 1998 has been shown to contribute to elevated PM levels in southern Texas and throughout the entire central and southeastern United States (CD, p. 3-45).
- Wildfires in the boreal forests of northwestern Canada may impact large portions of the eastern United States. Wotowa and Trainer (2000) estimate that a July 1995 Canadian wildfire episode resulted in excess $PM_{2.5}$ concentrations ranging from 5 $\mu g/m^3$ in the Southeast, to nearly 100 $\mu g/m^3$ in the northern Plains States (CD, p. 3-47).
- Windblown dust from dust storms in the North African Sahara desert has been observed in satellite images as plumes crossing the Atlantic Ocean and reaching the southeast coast of the United States, primarily in Florida, and North African dust has also been tracked as far

as Illinois and Maine. These events have been estimated to contribute 6 to 11 μg/m^3 to 24-hour average $PM_{2.5}$ levels during the events in affected areas (CD, p. 3-45).

- Dust transport from the deserts of Asia (e.g., Gobi, Taklimakan) across the Pacific Ocean to the northwestern U.S. also occurs. Husar et al. (2000) report that the average PM_{10} level at over 150 reporting stations throughout the northwestern U.S. was 65 μg/m^3 during an episode in the last week in April 1998, compared to an average of about 20 μg/m^3 during the rest of April and May (CD, p. 3-45).

The draft CD provides the broad estimates of annual average background PM levels shown in Table 2-4. The lower bounds of the ranges are based on compilations of natural versus human-made emissions levels, ambient measurements in remote areas, and regression studies using human-made and/or natural tracers (NAPAP, 1991; Trijonis, 1982). The upper bounds are derived from the multi-year annual averages of the "clean" remote monitoring sites in the IMPROVE network (Malm et al., 1994). Since the IMPROVE data reflect the effects of anthropogenic emissions from within North America, they provide conservative estimates of the upper bounds. There is a definite geographic difference in background levels with lower levels in the western U.S. and higher levels in the eastern U.S. The eastern U.S. is estimated to have more natural organic fine-mode particles and more water associated with hygroscopic fine-mode particles than the western U.S. due to generally higher humidity levels.

Table 2-4. Estimated Range of Annual Average PM_{10} and $PM_{2.5}$ Regional Background Levels

	Western U.S. (μg/m^3)	Eastern U.S. (μg/m^3)
PM_{10}	4 - 8	5 - 11
$PM_{2.5}$	1 - 4	2 - 5

Source: CD, p. 3-10

Over shorter periods of time (e.g., days or weeks), the range of expected background concentrations is much broader. Specific natural events such as wildfires, volcanic eruptions, and

dust storms can lead to very high levels of PM comparable to, or greater than, those driven by man-made emissions in polluted urban atmospheres.

2.7 PM-RELATED SOURCE EMISSIONS AND TRENDS

Insights into what is driving ambient levels of PM can be gained by examining the emissions levels of pollutants that contribute to ambient PM. There is an indirect link between source emissions and ambient concentrations of PM that is affected by complex atmospheric processes, including gaseous chemical reactions and pollution transport.

EPA publishes estimates of annual source emissions of pollutants related to ambient criteria pollutant concentrations. The most recent EPA report contains a national inventory of 1998 emissions (EPA, 2000a). National emissions estimates are uncertain, and there have been few field studies to test emission inventories observationally. The draft CD concludes that uncertainties in national emissions estimates could be as low as 10 percent for the best characterized source categories (e.g., SO_2 from electric utilities), while emissions estimates from fugitive dust sources should be regarded as order-of-magnitude (CD, p. 3-59). However, recent advances in developing fugitive dust emission factors and emissions algorithms using those factors, and a better understanding of the fate and transport characteristics of fugitive dust emissions released at ground level will reduce the uncertainty of estimates now being developed.

2.7.1 Primary PM Emissions

Estimates of directly emitted, or primary, PM are dominated by fugitive dust emissions. Fugitive dust sources include paved and unpaved road dust, dust from construction and agricultural activities, and natural sources like geogenic wind erosion. The majority of directly emitted PM is estimated to be coarse-mode crustal material. Though highly uncertain, estimates of PM_{10} fugitive dust-related emissions are more than 5 times higher than estimates of $PM_{2.5}$ fugitive dust-related emissions – 30.9 million short tons compared to 5.5 million short tons (EPA 2000a). Recent research has found that about 75 percent of these emissions are within 2 meters of the ground at the point they are measured, and a significant portion are likely to be removed or deposited within a few kilometers of their release point due to turbulence associated with surface

topography, or the presence of vegetation or structures (DRI, 2000). This is consistent with the generally small amount of crustal material found in ambient samples in most locations. Estimated annual emissions of directly emitted PM_{10} and $PM_{2.5}$ from the subset of non-fugitive sources in the U.S. are summarized in Figure 2-15. The direct emissions profiles for both $PM_{2.5}$ and PM_{10} are similar, with nearly half of emissions originating from stationary (point and area) source fuel combustion and motor vehicles. A large portion is also attributed to a variety of area source combustion processes, such as open burning. Area source emissions are often more difficult to characterize and are more uncertain than point source emissions.

Because total direct emissions of PM are dominated by highly uncertain estimates for fugitive dust sources, the long-term emissions trend for total PM is highly uncertain. Table 2-5 shows the 10 year change in primary PM emissions from the subset of non-fugitive dust sources and from all sources. Direct PM_{10} emissions from non-fugitive dust sources were estimated to decline 15 percent from 1990 to 1998 due to reductions from diesel engines, residential wood combustion, and assorted industrial processes, particularly in mineral processing industries. Over the same period primary $PM_{2.5}$ emissions from non-fugitive dust sources were estimated to decline 15 percent. However, not all categories of non-fugitive dust sources experienced declines. Emissions of direct $PM_{2.5}$ from coal-based fuel combustion at electric utilities, which comprise nearly 5 percent of the non-fugitive dust total, increased by over 36 percent (EPA 2000a, Table A-6). Due primarily to estimated increases in fugitive dust emissions, primary PM_{10} and $PM_{2.5}$ emissions from all sources were estimated to increase by 16 percent and 5 percent respectively.

2.7.2 PM Precursor Gas Emissions

Major precursors of secondarily formed fine fraction particles include SO_2, nitrogen oxides (NO_x), which encompasses NO and NO_2, and certain organic compounds. Figures 2-16 and 2-17 presents the relative contribution of various sources to nationwide SO_2, NO_x, VOC, and NH_3 emissions estimates. Fuel combustion in the electric utility and industrial sectors dominate nationwide estimates of SO_2 emissions. Emissions from motor vehicles make up the greatest

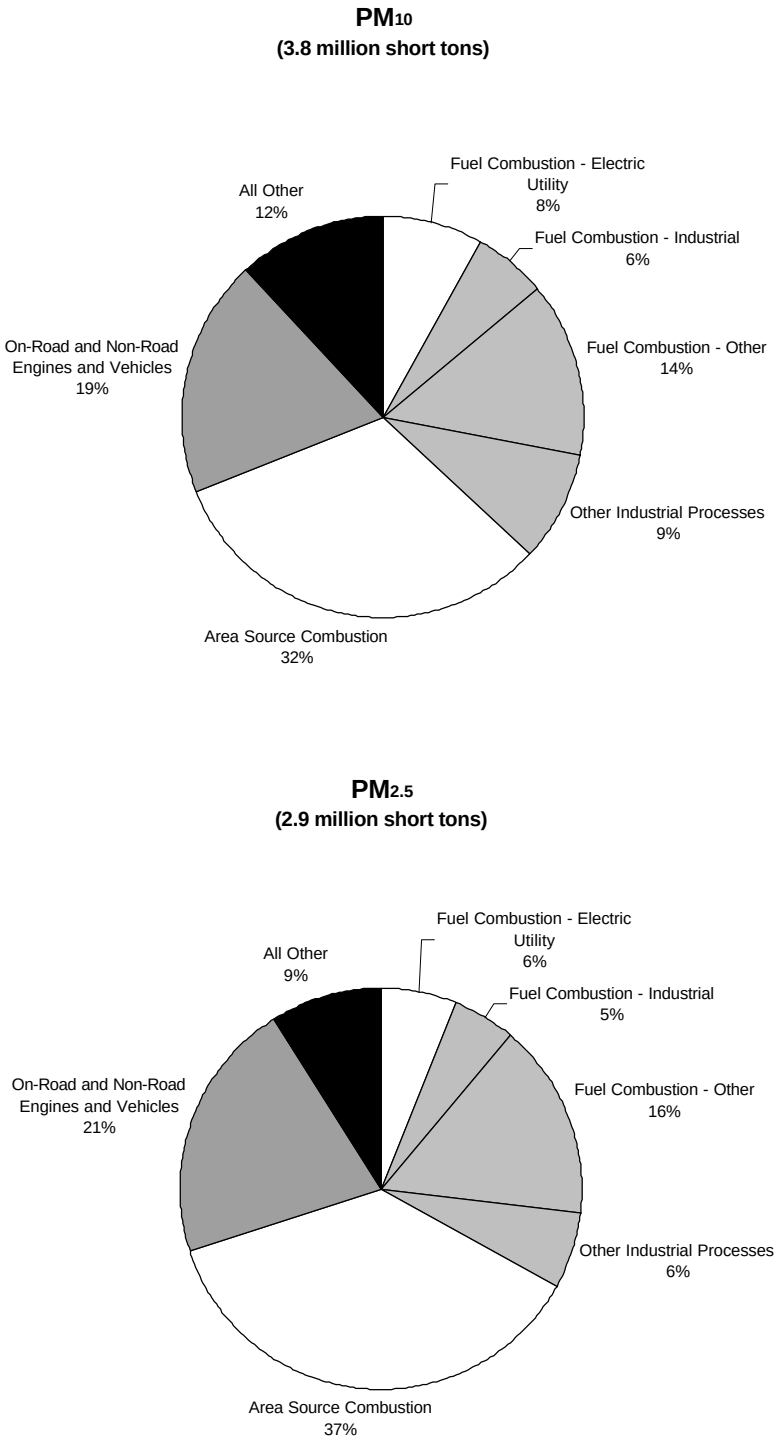


Figure 2-15. 1998 national direct emissions of PM by principal source categories for non-fugitive dust sources

Source: U.S. Environmental Protection Agency (2000a)

Table 2-5. Nationwide Changes in Estimated Annual Emissions of Primary PM and Gaseous Precursors to Secondary PM, 1989 to 1998

	1990 Emissions (million short tons)	1998 Emissions (million short tons)	% Change 1990-1998
Primary PM_{10}			
non-fugitive dust sources	4.5	3.8	-15%
all sources	30.0	34.7	16%
Primary $PM_{2.5}$			
non-fugitive dust sources	3.4	2.9	-15%
all sources	8.0	8.4	5%
SO_2	23.7	19.6	-17%
NO_x	24.0	24.5	2%
VOC	20.9	17.9	-14%
NH_3	4.3	4.9	14%

Source: Environmental Protection Agency (2000a), Tables A-2 through A-8

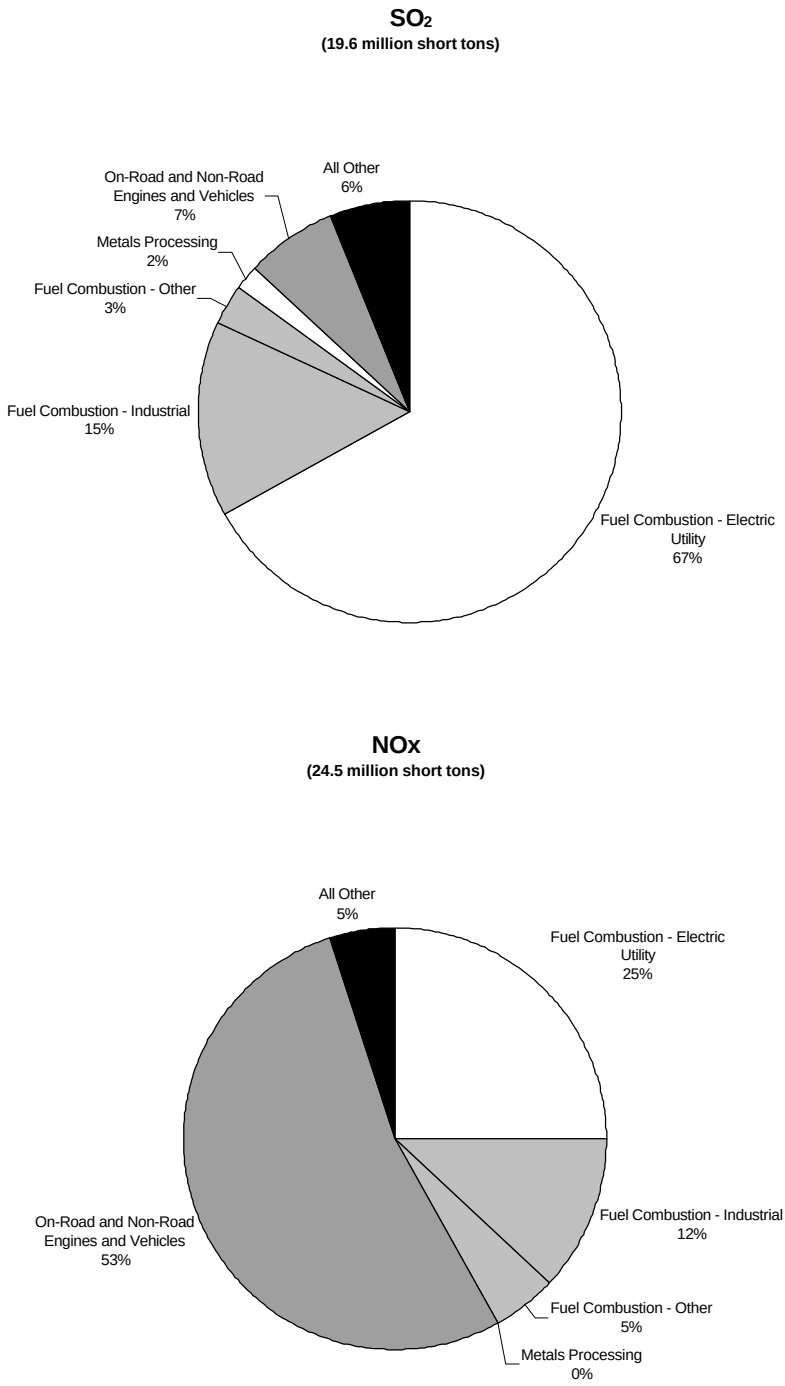


Figure 2-16. 1998 nationwide emissions of SO_2 and NO_x by principal source categories

Source: U.S. Environment Protection Agency (2000a)

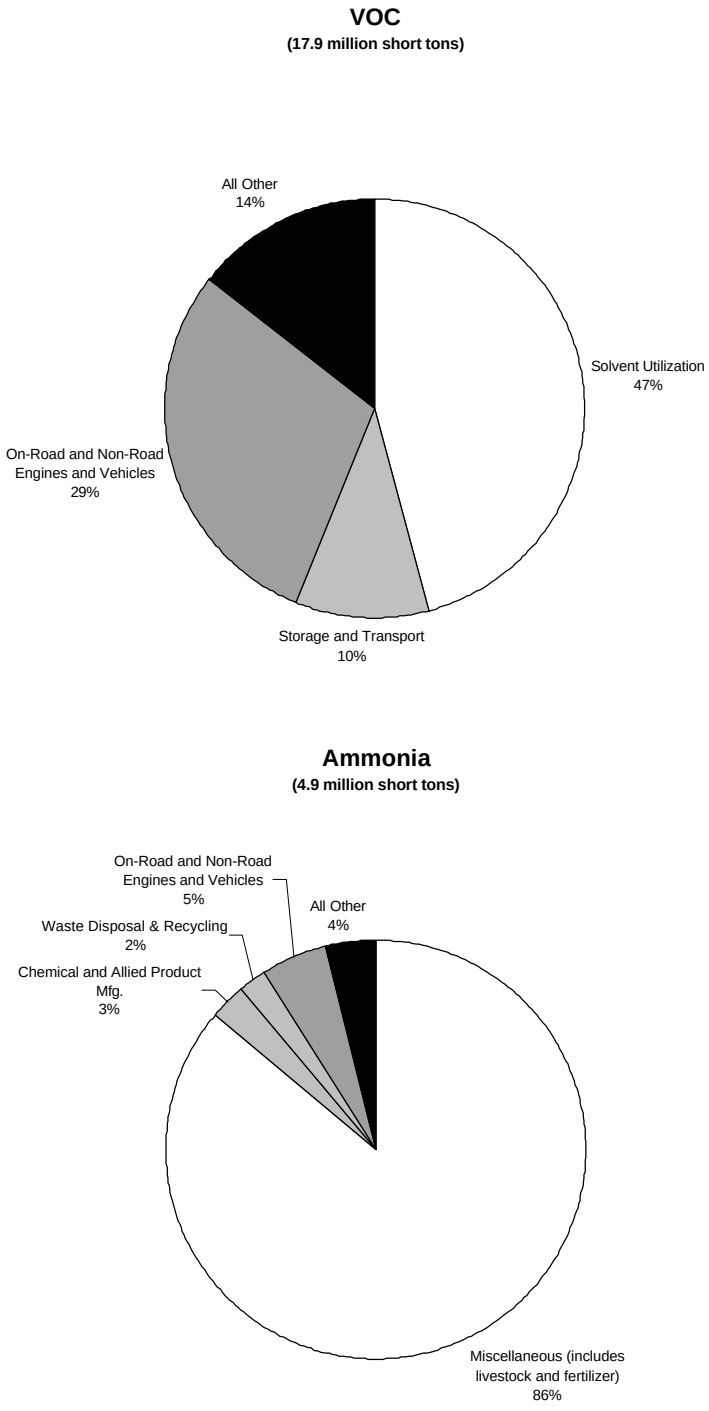


Figure 2-17. 1998 nationwide emissions of VOC and Ammonia by principal source categories
Source: U.S. Environmental Protection Agency (2000a)

portion of nationwide NO_x emissions. Motor vehicle emissions also comprise a substantial portion of nationwide VOC emissions, though the greatest contribution comes from the use of various solvents. The vast majority of nationwide NH_3 emissions are estimated to come from livestock operations and fertilizer application, but in urban areas there is a significant contribution from light-duty cars and trucks, as well as certain industrial processes.

The relationship between changes in precursor emissions and resulting changes in ambient $PM_{2.5}$ is nonlinear. Thus, it is difficult to project the impact on $PM_{2.5}$ arising from expected changes in PM precursor emissions without air quality simulation models that incorporate treatment of complex chemical transformation processes. While generally SO_2 emissions reductions lead to reductions in sulfate aerosol, and NO_x emissions reductions lead to reductions in nitrate aerosol, the direction and extent of changes will vary by location and season, depending on fluctuations in NH_3 emissions and changes in prevailing meteorology and photochemistry.

Table 2-5 shows the 10-year change in estimated national annual PM precursor emissions. Reductions in SO_2 emissions have occurred largely because of CAA programs such as SO_2 NAAQS implementation, the Acid Deposition Program, the prevention of significant deterioration (PSD) program, and the new source performance standards (NSPS) program. Despite significant economic growth, NO_x emissions increases have been limited due to PSD, NSPS, the Acid Deposition Program, and mobile source control programs. Future reductions in NO_x are projected for the eastern U.S. from electric utilities as a result of both the Acid Deposition Program and ozone NAAQS implementation. Also, substantial NO_x controls will also be required from motor vehicles in the form of new “Tier 2" standards for light-duty highway vehicles, and new standards for heavy-duty (mostly diesel) highway vehicles. EPA estimates that VOC emissions have declined about 20 percent from 1989 to 1998 due to ozone-related programs and tighter motor vehicle standards. NH_3 emissions were estimated to increase 14 percent due primarily to motor vehicles, fertilizer application and livestock operations.

2.8 RELATIONSHIP BETWEEN HUMAN EXPOSURE TO AMBIENT PM AND CENTRAL MONITOR MEASUREMENTS OF PM

The statutory focus of the primary PM NAAQS is on providing protection from adverse effects to public health associated with the presence of PM in the *ambient* air – that is, the focus is on particles that are emitted by sources to the outdoors (i.e., ambient PM). An understanding of human exposure to ambient PM helps inform the evaluation of underlying assumptions and interpretation of results of epidemiological studies that characterize relationships between monitored ambient PM concentrations and observed health effects (discussed in Chapter 3). Further, epidemiological studies of long term exposure raise more complex issues, which are noted in Chapter 3.

An important exposure-related issue for this PM NAAQS review is the characterization of the relationships between ambient fixed-site PM concentrations and personal exposure to ambient PM, as characterized by particle size, composition, or other factors. The focus here is on particle size distinctions; the draft CD in Section 5.5 discusses in more detail the exposure relationships related to compositional differences. Information on the type and strength of these relationships, discussed below, is relevant to the evaluation and interpretation of associations found in epidemiological studies using ambient PM concentrations as a surrogate for exposure.[12]

2.8.1 Definitions

An individual's exposure to PM results from breathing air containing PM in different types of microenvironments (e.g., outdoors near home, outdoors away from home, indoors at home, indoors at office or school, commuting, restaurants, malls, other public places, etc.) These microenvironments may have different concentrations of PM with particles originating from a wide variety of sources. Exposure is defined as the contact by an individual with a pollutant for a specific duration of time at a visible external boundary (CD, p. 5-1). Average exposure of an individual to PM, averaged over any given time period of length T, can further be expressed as $E = C_i t_i / T$, the sum of the concentration (C_i) of PM in each microenvironment a person spends his or

[12] Consideration of exposure measurement error and the effects of exposure misclassification on the interpretation of the epidemiological studies are addressed in Chapter 3.

her time in during the course of a day, times the time (t_i) spent in each microenvironment, divided by the total time (T) in all of the microenvironments. Total exposure to an individual is $C_i t_i$, the sum of all exposures during the period T.

As discussed in Section 2.7, outdoor concentrations of PM are the result of anthropogenic and natural emissions sources of PM, and are affected by meteorology, atmospheric chemistry, and removal processes. Indoor concentrations of PM are affected by several factors, including ambient outdoor concentrations and processes that result in infiltration of ambient PM into building (e.g., indoor/outdoor air exchange, particle penetration across the building envelope), indoor sources of PM, aerosol dynamics and indoor chemistry, and removal mechanisms such as particle deposition, exfiltration, and air-conditioning and air cleaning devices (CD, p. 5-96). Concentrations of PM inside vehicles are subject to essentially the same factors as indoor concentrations of PM inside the buildings. Total personal exposure to PM has an additional component, the personal cloud, which results specifically from the activities of an individual that typically generate particles affecting only the individual or a small localized area surrounding the person (e.g., walking on a carpet). Personal cloud is assumed to be predominantly due to non-ambient PM sources.

In characterizing human exposure to PM concentrations relevant to the NAAQS, the draft CD conceptually separates *total exposure* to PM into exposure to *ambient*[13] PM (*ambient exposure*) and exposure to all other sources of PM (*non-ambient exposure*). The draft CD describes PM according to both the source (i.e., ambient or non-ambient) and the microenvironments where the exposure occurs (e.g., outdoors near home, indoors in various rooms, within vehicles). Ambient PM can be differentiated as *ambient-outdoor PM*, outdoor concentrations of ambient PM generally measured at a centrally located fixed site or at specific outdoor locations, including outdoors near home, offices, etc. and *ambient-indoor PM*, ambient PM that has penetrated indoors, entering buildings by infiltration (e.g., through cracks) and bulk flow (e.g., through open windows). *Non-ambient PM* is comprised of PM generated from indoor

[13] Ambient PM includes not only emissions that are generated outdoors, but also emissions generated indoors and directly vented to the outdoors, such as emissions from wood-stoves, fire places, and some manufacturing processes.

sources and the indoor personal cloud. *Indoor-generated* PM is that which is due to indoor sources of particles, which include smoking, cooking, other sources of combustion, cleaning, resuspension, mechanical processes, and chemical reactions. Thus, *indoor PM* is the concentration of PM indoors, and includes ambient-indoor PM, indoor-generated PM, and the personal cloud.

2.8.2 Ambient Concentration as a Surrogate for Particle Exposure

The 1996 Criteria Document (EPA, 1996a) presented a thorough review of PM exposure-related studies up to that time. The previous Staff Paper (EPA, 1996b) drew upon the studies, analyses, and conclusions presented in the 1996 Criteria Document and discussed two interconnected PM exposure issues: (1) the ability of central fixed-site PM monitors to represent population exposure to ambient PM, and (2) how differences between fine and coarse mode particles affect population exposures. Distinctions between PM size classes and components were found to be important considerations in addressing representativeness of central monitors. For example, fine-mode particles have a longer residence time and are more uniformly distributed in the atmosphere than coarse-mode particles. The 1996 Staff Paper (EPA, 1996b) concluded that central measurements of daily variations of PM have a plausible linkage to daily variations of human exposures to ambient PM, that this linkage is stronger for fine-mode particles than for coarse-mode or fine-mode plus coarse-mode particles, and within the fine mode stronger for sulfates than for H^+. The 1996 Staff Paper further concluded that "central monitoring can be a useful, if imprecise, index for representing the average exposure of people in a community to PM of outdoor origin." (EPA, 1996b, p. IV-15,16).

Exposure studies published since 1996 and reanalyses of studies that appeared in the 1996 Criteria Document are reviewed in the draft CD, and provide additional support for the findings made in the 1996 Criteria Document and 1996 Staff Paper. As discussed in the draft CD (CD, p. 9-24, 25) and in the discussion that follows, an individual's total personal exposure to PM generally differs from the ambient concentration measured at the central site monitor because of: (1) spatial differences in ambient PM concentrations across a city or region; (2) generally only a fraction of the ambient PM penetrates to indoor or in-vehicle microenvironments; and (3) a

variety of indoor sources that produce predominantly ultrafine and coarse-mode particles will contribute to total personal exposure. Thus, the amount of time spent outdoors, indoors, and in vehicles and the types of activities engaged in (e.g., smoking, cooking, vacuuming) also will heavily influence personal exposure to PM.

With regard to the first factor that influences the relationship between total personal exposure and concentrations measured at central site monitors, fine-mode particles are more likely to be more uniformly dispersed across urban scales than coarse-mode particles. Analyses of 1999 $PM_{2.5}$ FRM monitoring data from four large metropolitan areas indicates that, in general, multiple sites in these urban areas are highly correlated throughout the year, although there are exceptions to this rule (CD, p. 3-57). It is likely that $PM_{2.5}$ concentrations are distributed evenly enough so that one site, or the average of several sites, provides an adequate measure of the community average concentration for $PM_{2.5}$. Where $PM_{2.5}$ is a major fraction of PM_{10} this may also be true for PM_{10}, in other cases, however, there is the potential for large PM_{10} spatial variability in some communities. In some instances the average ambient concentration and the average exposure to ambient PM may differ, but the levels tend to move up and down together. The draft CD acknowledges that this spatial uniformity may not be the case for $PM_{10-2.5}$, for specific chemical components, or for sites located near sources (CD, p. 9-24). At this time there are not sufficient data to assess the spatial variability of ultrafine PM or PM components, except for sulfate, which tends to be regionally uniformly distributed (CD, p. 5-97).

The second factor influencing the relationship between ambient PM concentrations and total personal exposure to PM is the extent to which ambient PM penetrates indoors and remains suspended in the air. PM penetration is heavily dependent on the air exchange rate, and also on penetration efficiency and deposition or removal rate, both of which vary with particle aerodynamic size. Air exchange rates (the rates at which the indoor air in a building is replaced by outdoor air) are influenced by building structure, the use of air conditioning and heating, opening and closing of doors and windows, and meteorological factors (e.g., difference in temperature between indoors and outdoors). Based on physical mass-balance considerations, usually the higher the air exchange rate the greater the personal exposure to ambient PM in the indoor and in-vehicle microenvironments. Rates of infiltration of outdoor PM into homes are higher for PM_1 and $PM_{2.5}$ than for PM_{10}, $PM_{10-2.5}$, or ultrafine particles (CD, p. 5-97). Since $PM_{10-2.5}$ infiltrates

indoors less readily than $PM_{2.5}$ and settles out more rapidly than $PM_{2.5}$, the ambient indoor/outdoor concentration ratios for $PM_{10-2.5}$ are smaller than for $PM_{2.5}$. These considerations suggest that central-site ambient measurements are expected to be more representative of ambient $PM_{2.5}$ personal exposure than ambient PM_{10} or $PM_{10-2.5}$ exposures.

The third factor influencing the relationship between ambient concentrations and total personal exposure is the contribution of indoor sources to total personal exposure. Several studies have shown that the contribution of indoor sources to total personal exposure is independent of ambient PM. Indoor PM concentrations are often higher than outdoor concentrations due to the additional PM generated from indoor sources. Indoor sources such as cooking, and smoking generate fine-mode particles, and dusting, vacuuming, and resuspension generate coarse-mode particles. Indoor sources tend to produce coarse-mode and nuclei-mode particles more than accumulation-mode particles (CD, p. 9-25).

An important finding is that ambient PM concentrations have been demonstrated to be correlated with ambient exposure but independent of nonambient exposure (CD, p. 5-99). This is illustrated in Figures 2-18a,b,c, which show the empirical relationships between ambient PM_{10} concentrations and (a) total exposure, (b) ambient exposure, and (c) nonambient exposure. The data for these figures are from the PTEAM study[14], which was considered in the previous PM NAAQS review (EPA, 1996a, p. 7-24, 7-88) and has provided more data than any other study for this type of analysis. The regression figures were developed according to models described in Mage et al. (1999) and Wilson et al. (2000) and used parameters estimated by Özkaynak et al., 1996a. Figure 2-18(a) shows the weak relationship between total personal exposure and ambient concentrations. Figure 2-18(b) shows that ambient exposure and ambient concentrations are well correlated (correlation 0.86). Figure 2-18(c) illustrates the independence of nonambient exposure and ambient concentrations and also the high variability of nonambient exposure due to differences found in indoor sources across the study homes.

[14] EPA's Particle Total Exposure Assessment Methodology (PTEAM) field study (Clayton et al., 1993; Özkaynak et al., 1996a;b) is one of only two large-scale probability sample based field studies conducted in the U.S. or Canada. The study measured indoor, outdoor, personal PM, the air exchange rate for each home, and time spent in various indoor residential and outdoor microenvironments for 147 subjects/households, 12-hr time periods in Riverside, California.

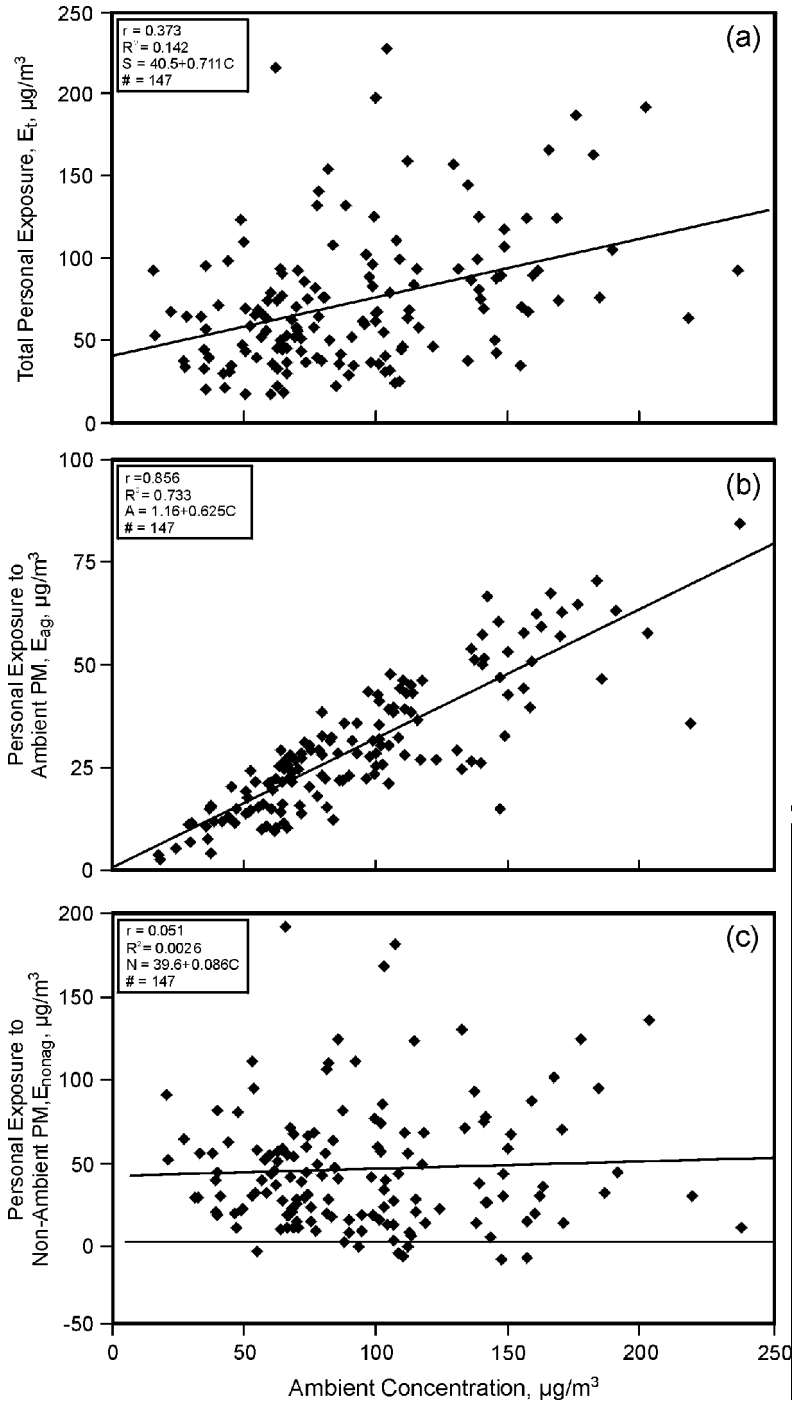


Figure 2-18. Regression analyses of aspects of daytime personal exposure to PM_{10} estimated using data from the PTEAM study. (a) Total personal exposure to PM regressed on ambient concentration, C_a. (b) Personal exposure to ambient PM regressed on C_a. (c) Personal exposure to nonambient PM regressed on C_a.

Source: Draft CD (EPA, 2000a). Data from Clayton et al. (1993).

Cross-sectional correlations were reported to be near zero in some exposure studies comparing ambient PM concentrations and total personal exposure to PM across different individuals for the same day. Poor correlations that were found were mainly due to the fact that some subjects lived in homes with low or relatively constant indoor sources and others had many different types of indoor sources. The indoor-generated concentrations are essentially considered a source of random measurement noise on top of the more predictable relationship between ambient PM and exposure to ambient PM. When short-term fluctuations of indoor-generated PM are minimized by taking daily averages and following specific individuals over time (i.e., a longitudinal correlation), the reported correlations between ambient PM and exposure to ambient PM become much stronger. This is probably because the non-ambient contribution for any given individual tends to remain fairly similar over time (e.g., people living with a smoker or using a wood stove in the winter).

Furthermore, studies with subjects experiencing small indoor source contributions to their personal exposures (e.g. the elderly in retirement homes), such that total exposure is mostly from ambient PM, generally exhibit both high cross-sectional and high longitudinal correlations between total personal exposure and ambient PM. Correlations between personal and ambient measurements of PM, using a predominantly outdoor component of PM, have shown that indeed the correlations can be quite high when indoor generated PM mass contributions are excluded. In particular, central-site measurements of sulfate (which is primarily fine-mode PM) have also been found to be highly correlated with total personal exposure to sulfate (CD, p. 5-97).

The draft CD discusses the finding by some researchers that epidemiology yields statistically significant associations between ambient concentrations and health effects even though there is a near zero correlation between ambient concentrations and [total] personal exposures in many studies (CD, p. 9-85, 86). This has been described by some exposure analysts as an “exposure paradox.” The explanation of this seemingly counterintuitive finding is that, as discussed above, total personal exposure includes both ambient and non-ambient generated components. However, community time series epidemiology only addresses the ambient component of exposure. Thus, the appropriate correlation to focus on, for these types of epidemiologic studies, is the correlation between ambient concentration as measured at a central-site monitor or average of several

monitors and personal exposure to ambient PM. Also, the appropriate correlation (of ambient concentrations and exposure to ambient PM) is not the pooled correlation of different days and different people, but rather the correlation between daily ambient concentrations and community average daily personal exposure to ambient PM. Based on the review of the available exposure-related studies, the draft CD concludes that for time-series epidemiology, ambient PM concentrations are a useful surrogate for exposure to ambient PM (CD, p. 9-86).

2.9 OPTICAL AND RADIATIVE PROPERTIES OF PARTICLES

By scattering and absorbing electromagnetic radiation, ambient particles can impair visibility, affect the amount of ultraviolet radiation that reaches the earth, and affect global climate processes. Electromagnetic radiation is emitted by the sun at ultraviolet (0.015 to 0.4 μm) and visible (0.4 to 0.8 μm) wavelengths, and by the earth at infrared (0.75 to 1000 μm) wavelengths. The effects of ambient particles on the transmission of these segments of the electromagnetic spectrum depend on the radiative properties of the particles, which in turn are dependent on the size and shape of the particles, their composition, the distribution of components within individual particles, and on their vertical and horizontal distribution in the lower atmosphere. In general, radiative effects of particles tend to be at their maximum when the particle radius is similar to the wavelength of the incident radiation (CD, p. 4-129).

2.9.1 PM Properties Affecting Visibility

Visibility is affected by scattering and absorption of light in visible wavelengths by particles and gases in the atmosphere (CD, p. 4-88). The efficiency of particles in causing visibility impairment depends on particle size, shape, and composition. Fine-mode particles, especially those in the accumulation mode, are generally most effective in impairing visibility. The fine-mode particle components principally responsible for visibility impairment are sulfates, nitrates, organic matter, elemental carbon, and soil dust. All such particles scatter light to some degree, but only elemental carbon plays a significant role in light absorption. Since elemental carbon, which is a product of incomplete combustion from activities such as the burning of wood or diesel

fuel, is a relatively small component of PM in most areas, impairment is generally dominated by scattering rather than absorption.

Because humidity causes hygroscopic particles to grow in size, humidity plays a significant role in particle-related impairment. The amount of increase in particle size with increasing relative humidity depends on particle composition (CD, p. 4-91). Humidity-related particle growth is a more important factor in the eastern U.S., where annual average relative humidity levels are 70 to 80 percent compared to 50 to 60 percent in the western U.S. Due to relative humidity differences, the same ambient mass concentration of particles would likely cause greater visibility impairment in an eastern location than a western one.

2.9.2 PM Properties Affecting Transmission of Ultraviolet Radiation

The transmission of solar radiation in the ultraviolet (UV) range through the earth's atmosphere is affected by ozone, clouds and particles. Of particular interest is the effect of particles on radiation in the ultraviolet-B (UV-B) range (generally from 0.280 to 0.320 μm), which has been associated with various biological effects. Relative to ozone, the effects of ambient particles on the transmission of UV-B radiation are more complex (CD, p. 4-134). The draft CD notes that even the sign of the effect can reverse as the composition of the particle mix in an air mass changes from scattering to absorbing types (e.g., from sulfate to elemental carbon and/or PAH's), and that there is an interaction in the radiative effects of scattering particles and absorbing molecules, such as ozone, in the lower atmosphere.

The effects of particles in the lower atmosphere on the transmission of solar UV-B radiation have been examined both by field measurements and by radiative transfer model calculations (CD, pp. 4-134 to 4-137). The draft CD cites several studies that reinforce the idea that particles can play an important role in modulating the attenuation of solar UV-B radiation, although none included measurements of ambient PM concentrations, so that direct relationships between PM levels and UV-B radiation transmission could not be determined. While ambient particles are generally expected to decrease the flux of solar UV-B radiation reaching the surface, any comprehensive assessment of the radiative effects of particles would be location-specific and complicated by the role of particles in photochemical activity in the lower atmosphere. Whether

the photochemical production of ozone is enhanced, neutralized, or even reversed by the presence of ambient particles will be location-specific and dependent on particle composition. Also complicating any assessment of solar UV-B radiation penetration to specific areas of the earth's surface are the influences of clouds, which in turn are affected by the presence of ambient particles. The available studies, conducted in diverse locations around the world, demonstrate that relationships between particles and solar UV-B radiation transmission can vary considerably over location, conditions, and time.

2.9.3 PM Properties Affecting Climate

The effects of PM on the transfer of radiation in the visible and infrared spectral regions also play a role in global or regional climate. Particles can have both direct and indirect effects on climatic processes. The direct effects are the result of the same physical processes responsible for visibility degradation, namely scattering and absorption (CD, p. 4-152). However, while visibility impairment is caused by particle scattering in all directions, climate effects result mainly from scattering light back toward its source. This reflection of solar radiation back to space decreases the transmission of visible radiation to the surface and results in a decrease in the heating rate of the surface and the lower atmosphere. At the same time, absorption of either incoming solar radiation or outgoing terrestrial radiation by particles, primarily organic carbon, results in an increase in the heating rate of the lower atmosphere.

The extent to which ambient particles scatter and absorb radiation is highly dependent on their composition and optical properties and on the wavelength of the radiation. For example, sulfate and nitrate particles effectively scatter solar radiation, and they weakly absorb infrared, but not visible, radiation. The effects of mineral dust particles are complex; they weakly absorb radiation, but their overall effect depends on particle size and reflectivity, and they contribute to atmospheric warming by absorbing infrared radiation. Organic carbon particles mainly reflect radiation, whereas elemental carbon and other black carbon particles (e.g., some PAH's) strongly absorb radiation; however, the optical properties of carbonaceous particles are modified if they become coated with water or sulfuric acid. Upon being deposited onto surfaces, particles can also

either absorb or reflect radiation depending in part on the relative reflectivity of the particles and the surfaces on which they are deposited.

In addition to these direct effects, particles can also have an indirect effect on climate. For example, sulfate particles can serve as condensation nuclei which alter the size distribution of cloud droplets by producing more droplets with smaller sizes (CD, p. 4-153). Because the total surface area of the cloud droplets is increased, the amount of solar radiation that clouds reflect back to space is increased. Also, smaller cloud droplets have a lower probability of precipitating, causing them to have longer atmospheric lifetimes.

The overall radiative effects of particles, both direct and indirect, are not the simple sum of effects caused by individual classes of particles because of interactions between particles and other atmospheric gases. As discussed in Section 4.5.2.2 of the draft CD, the effects of sulfate particles have been the most widely considered, with globally averaged effects of sulfate particles generally estimated to have partially offset the warming effects caused by increases in greenhouse gases. On the other hand, global-scale modeling of mineral dust particles has found that even the sign as well as the magnitude of effects depends on the vertical distribution and effective particle radius.

In general, the draft CD makes clear that the effects of PM on climate are complex and not well understood. In general, on a global scale atmospheric particles likely exert an overall net effect of slowing atmospheric warming. However, deviations from global mean values can be very large even on a regional scale, with any estimation of more localized effects introducing even greater complexity. The draft CD concludes that any estimate of the net effect on global climatic processes, and regional or local meteorology and consequent human health or environmental effects, due to location-specific changes in emissions of particles or their precursors would be highly uncertain (CD, p. 4-155).

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3. CHARACTERIZATION OF PM-RELATED HEALTH EFFECTS

3.1 INTRODUCTION

This chapter summarizes key information relevant to assessment of the known and potential health effects associated with exposure to ambient PM, alone and in combination with other pollutants that are routinely present in ambient air. A comprehensive discussion of this information, focusing on the new scientific information available since the last review, can be found in Chapters 6 - 9 of the draft CD, with Chapter 9 drawing upon the new information to update the integrated assessment provided in the 1996 PM CD.

The presentation here organizes the key health effects information into those elements essential for the evaluation of current and alternative standards for PM. Drawing primarily upon the epidemiological, toxicological, dosimetry, and exposure-related information in the draft CD, this chapter summarizes: (1) information and hypotheses regarding mechanisms by which particles that penetrate to and deposit in various regions of the respiratory tract may exert effects; (2) the nature of effects that have been associated with ambient PM, with a focus on fine- and coarse-fraction PM; (3) the identification of sensitive populations that appear to be at greater risk to the effects of ambient PM; and (4) issues related to interpretation and evaluation of the health effects evidence, including discussion of the role of co-pollutants, evidence for effects of various PM components, and issues regarding assessment of epidemiological evidence. Staff conclusions and recommendations related to primary standards for PM will be incorporated into Chapter 6 of a subsequent draft of this Staff Paper.

In the last review, a variety of health effects had been associated with ambient PM at concentrations extending from those found in the historic London episodes down to levels below the 1987 PM_{10} standards. Of particular importance from the last review were the conclusions that (1) ambient particles smaller than 10 μm that penetrate into the thoracic region of the respiratory tract remain of greatest concern to health, (2) the fine and coarse fractions of PM_{10} should be considered separately for the purposes of setting ambient air quality standards, and (3) the consistency and coherence of the health effects evidence greatly adds to the strength and plausibility of the observed PM associations. Important uncertainties remained, however, such as

issues related to interpreting the role of gaseous co-pollutants in PM associations with health effects, and the lack of accepted biological mechanisms that could explain observed effects.

An unprecedented number of new studies containing further evidence of serious health effects have been published since the last review, with important new information coming from epidemiological, toxicological, controlled human exposure, and dosimetry studies. For example, important new epidemiological studies include:

- Multi-city studies that use uniform methodologies to investigate the effects of PM on health with data from multiple locations with varying climate and air pollution mixes, contributing to increased understanding of the role of various confounders, including gaseous co-pollutants, on observed PM associations.

- Several studies evaluating independent associations between effects and fine- and coarse-fraction particles, as well as specific components (e.g., ultrafines, crustal[1] particles).

- New analyses and approaches to addressing issues related to confounders, possible effects thresholds, and measurement error and exposure misclassification.

- Studies presenting new factor analysis methods to evaluate health effects associated with different PM source types.

Important new toxicological, controlled human exposure, and dosimetry studies include, for example:

- Animal and controlled human exposure studies using concentrated ambient particles (CAPs), new indicators of response (e.g., heart rate variability), as well as animal models representing sensitive subpopulations, that are relevant to the plausibility of the epidemiological evidence and provide insights into potential mechanisms for PM-related effects.

- Dosimetry studies using new modeling methods and controlled exposures that provide increased understanding of the dosimetry of different particle size classes and in members of potentially sensitive subpopulations, such as people with chronic respiratory disease.

Based on an evaluation of the new evidence and consideration of possible alternative explanations for the reported PM effects, the draft CD concludes that fine- and coarse-fraction

[1] "Crustal" is used here to describe particles of geologic origin, which can be found in both fine- and coarse-fraction PM.

particles should continue to be treated as distinct subclasses of PM (CD, p. 9-1); that "the reported associations of PM exposure and effects are valid;" and that the newer evidence

> . . . (a) further substantiates associations of such serious health effects with U.S. ambient PM_{10} levels, (b) also more strongly establishes fine particles . . . as likely being important contributors to the observed human health effects, and (c) now provides additional information on associations between coarse-fraction ($PM_{10-2.5}$) particles and adverse health impacts. The overall coherence . . . strengthens the 1996 PM AQCD evaluation suggesting a likely causal role of ambient PM in contributing to the reported effects. (CD, p. 9-2)

3.2 MECHANISMS

This section briefly summarizes available information concerning the penetration and deposition of particles in the respiratory tract and outlines hypothesized physiological and pathological responses to PM, drawing from information presented in previous PM criteria and standard reviews and in Chapters 7 - 9 of the draft CD. The 1996 staff analysis of this information concluded that the available toxicological and clinical information yields no demonstrated biological mechanism(s) that can explain the associations between ambient PM exposure and mortality and morbidity reported in community epidemiologic studies (EPA, 1996b, p. V-2). While that conclusion still holds true, substantial progress has been made in identifying and understanding a number of potential pathways that were the subject of speculation in the last review. The major purposes of the discussion presented here are to note the available information of greatest relevance in identifying those fractions of PM that are most likely to be of concern to health, to examine possible links between ambient particles deposited in various regions of the respiratory tract and reported effects in humans, to identify factors that may contribute to susceptibility in sensitive populations, and to focus attention on the advances in mechanistic research that are providing evidence in support of a biological basis for a causal link between ambient PM exposures and reported health effects.

As discussed in the 1996 Staff Paper, an evaluation of the ways by which inhaled particles might ultimately affect human health must take account of patterns of deposition and clearance in the respiratory tract. The draft CD stresses that the probability of any biological effect of PM depends on particle deposition and retention, as well as underlying dose-response relationships

(CD, p. 9-32). The major elements of these considerations have been developed in previous reviews and are summarized briefly here. The human respiratory tract can be divided into three main regions: (1) extra-thoracic, (2) tracheobronchial, and (3) alveolar (CD, p. 9-27). The regions differ markedly in structure, function, size, mechanisms of deposition and removal, and sensitivity or reactivity to deposited particles; overall, the concerns related to ambient particles are greater for the two lower regions (EPA, 1982b; CD, Chapter 7). The junction of conducting and respiratory airways appears to be a key anatomic focus; many inhaled particles of critical size are deposited in the respiratory bronchioles that lie just distal to this junction, and many of the changes characteristic of emphysema involve respiratory bronchioles and alveolar ducts (Hogg et al., 1968). Recent modeling work has documented that ultrafine, as well as larger particles show enhanced deposition of particles at airway bifurcations (Heistracher and Hofmann, 1997; Hofmann et al., 1996). The potential effects of deposited particles are influenced by the speed and nature of removal. These clearance and translocation mechanisms that vary with each of the three regions (CD, Table 7-1, Figure 7-2).

Deposition of ambient particles in the three regions of the respiratory tract does not occur at divisions clearly corresponding to the atmospheric aerosol distributions shown above in Chapter 2. The draft CD summarizes simulations of deposition of ambient particle distributions that indicate fine- and coarse-fraction particles are deposited in both the tracheobronchial and alveolar regions (CD, Chapter 7). While fine- ($\leq$2.5 μm) and coarse-fraction (10 - 2.5 μm) particles deposit to about the same extent on a percent particle mass basis in the trachea and upper bronchi, a distinctly higher percent of fine mass (than coarse) deposits in the alveolar region. It follows from the relationships summarized here in Chapter 2 that most of the particle surface area and numbers that deposit are associated with the fine fraction. The draft CD notes that the number dose (particles/cm^2/day) of fine particles to the lung is orders of magnitude higher than that for coarse-fraction particles.

Information from the last review, as well as important new studies discussed in the draft CD, add to evidence from the earlier 1987 review, showing how breathing patterns and respiratory disease status can affect regional particle deposition patterns. The 1996 CD showed that as mouth-breathing or workload increases so does deposition in the bronchial and alveolar

regions. For those individuals considered to be mouth breathers, deposition increases for coarse-fraction particles in the tracheobronchial region (EPA, 1996a, pp. 166-168). Bennett et al. (1997b) found people with chronic obstructive pulmonary disease (COPD) had about 2.5 times the average deposition rates of healthy adults, related both to elevated tidal volume and breathing rate. In such a case, the respiratory condition can enhance sensitivity to inhaled particles by increasing the delivered dose to sensitive regions. Such dosimetry studies are of obvious relevance to identifying sensitive populations, which is discussed more fully in Section 3.4.

As discussed in the 1996 Staff Paper, evidence from epidemiological studies of occupational and historical community exposures and laboratory studies of animal and human responses to simulated ambient particle components suggested that at exposures well above the current PM_{10} standards, particles may produce physiological and ultimately pathological effects by a variety of mechanisms. Previous criteria and standards reviews included an integrated extensive examination of available literature on the potential mechanisms, consequences, and observed responses to particle deposition organized according to major regions of the respiratory tract (EPA, 1982b, 1996a,b). Based on these assessments and considering the composition of typical urban PM, staff concluded, with CASAC concurrence (Friedlander, 1982; Wolff, 1996), that particles that deposit in the thoracic region (tracheobronchial and alveolar regions), i.e. particles smaller than 10 μm diameter, were of greatest concern for standard setting (EPA, 1996b, p. V-3, Figure V-1). Although more recent information has expanded our understanding of these issues, no basis has emerged to change that fundamental conclusion.

In the last two reviews, staff identified a number of *potential* mechanisms and supporting observations by which common components of ambient particles that deposit in the thoracic region, alone or in combination with pollutant gases, might produce health effects (EPA, 1982b, Table 5-2; 1996b, Table V-2). While there has been little doubt in the scientific community that the historical London air pollution episodes had profound effects on daily mortality and morbidity, no combination of the mechanisms/observations advanced in the past reviews has been sufficiently tested or generally accepted as explaining the historical community results. Moreover, the potential mechanisms cited in those previous reviews were based on insights developed from laboratory and occupational/community epidemiological studies that involved concentrations that

were substantially higher than those observed in current U.S. atmospheres, and in many cases using laboratory-generated particles that may be of limited relevance to community exposures (EPA, 1996b, p V-4).

Fully defining the mechanisms of action for PM would involve description of the pathogenesis or origin and development of any related diseases or processes resulting in premature mortality. While the substantial recent progress presented in Chapters 8 and 9 of the draft CD and summarized below has provided important insights that contribute to the plausibility of community study results, this more ambitious goal of understanding fundamental mechanisms has not yet been reached. Some of the more important findings presented therein, including those related to the cardiovascular system, may be more accurately described as intermediate responses potentially caused by PM exposure rather than complete mechanisms. It appears unlikely that the complex mixes of particles that are present in community air pollution would act alone though any single pathway of response. Accordingly, it is plausible that several responses might occur in concert to produce reported health endpoints.

By way of illustration, Mauderly et al. (1998) examined prevalent hypotheses related to PM health effects that have been under consideration, in order to guide PM monitoring programs. They produced an illustrative list of 11 components/characteristics of interest for which some evidence existed. The list included: 1) PM mass concentration, 2) PM particle size/surface area, 3) ultrafine PM, 4) metals, 5) acids, 6) organic compounds, 7) biogenic particles, 8) sulfate and nitrate salts, 9) peroxides, 10) soot, and 11) co-factors, including effects modification or confounding by co-occurring gases and meteorology. The authors stress that this list is neither definitive nor exhaustive, and note that "it is generally accepted as most likely that multiple toxic species act by several mechanistic pathways to cause the range of health effects that have been observed" (Mauderly et al., 1998).

In assessing the more recent animal, controlled human, and epidemiologic information, the draft CD developed a summary of current thinking on pathophysiological mechanisms for the effects of low concentrations of particulate air pollution (CD, pp. 8-72 to 8-77, pp. 9-89 to 9-94). The potential mechanisms discussed in the draft CD, organized by effects category, are reproduced in Table 3-1 below.

Table 3-1. Summary of Current PM Mechanism Hypotheses (CD, pp. 8-72 to 8-77, pp. 9-89 to 9-94)

Effect	Potential Mechanisms
Direct Pulmonary Effects	Lung injury and inflammation
	Increased susceptibility to respiratory infections
	Increased airway reactivity and asthma aggravation
Systemic Effects Secondary to Lung Injury	Impairment of heart function by lowering blood oxygen levels and increasing the work of breathing
	Lung inflamation and cytokine production leading to systemic hemodynamic effects
	Increased risk of heart attacks and strokes because of increased blood coagulability secondary to lung inflamation
	PM/lung interactions potentially affecting hematopoiesis
Direct Effects on the Heart	Heart rate variability
	Autonomic control of the heart and cardiovascular system
	Uptake of particles and/or distribution of soluble components into the systemic circulation

The CD discussion highlights portions of the recent information that serve as support for these effects categories and potential mechanisms. The relative support for these hypotheses/intermediate effects and their relevance to real world inhalation of ambient particles varies significantly. Moreover, some variability of results exist among different approaches, investigators, animal models, and even day-to-day within studies. The list of hypotheses in Table 3-1 was developed mainly in reference to effects from short-term rather than long-term exposure to PM. Repeated occurrences of some short-term insults, such as inflammation, might contribute to long-term effects, but wholly different mechanisms might also be important in the development of chronic responses. Even where clear mechanisms cannot be specified, however, the increasing laboratory evidence of the pathways by which particles apparently affect the respiratory and

cardiovascular systems adds to the plausibility that particles, alone or in combination with pollutant gases, are playing a causal role in the effects observed in epidemiological studies.

Substantial new toxicologic information outlined in the draft CD as supporting these mechanisms relates to evidence for the occurrence of lung injury and inflammation and intermediate effects on the heart with exposure to PM. Numerous animal toxicological studies have provided clear evidence that lung injury and inflammation occur with exposure to residual oil fly ash (ROFA). While this model particle is reflective of a real world combustion product, it is rich in acidic metals, and its occurrence in contemporary U.S. atmospheres is limited. It has been useful in elucidating the importance of metal interactions in producing inflamation. More relevant evidence for inflammation has been reported in some, but not all, studies using CAPs or instilled ambient particles. Most of the CAPs studies reflect the effects of fine particles between 0.2 to 2 um, and exclude both the ultrafine and coarse fractions. Costa and Dreher (1997) summarized evidence from studies showing increased inflammatory cell counts with instillation to ambient particles collected in U.S., Canadian, and German cities, and Brain et al. (1998) showed that similar levels of acute inflammatory injury were caused by urban air particles and Kuwaiti oil fire particles (on an equal mass basis). In one new controlled human exposure study, Ghio et al. (2000) reported increased neutrophil counts and elevated levels of blood fibrinogen in lavage fluid from healthy volunteers after exposure to CAPs.

ROFA administration has caused more severe inflammatory effects in animals, including increased lung permeability which could lead to reduced oxygenation of the blood (CD, p. 9-91). However, the draft CD finds that, based on studies where CAPs were used, severe disturbances of oxygenation or pulmonary function by ambient PM are unlikely (CD, p. 9-91). *In vitro* studies provide support for the observed inflammatory effects on ambient PM and constituent substances, in finding evidence of reactive oxidant species that can damage lung cells. Several studies of ambient particles (e.g. Utah Valley ambient samples) showed that soluble extracts (including metals) are responsible for oxidant generation, release of IL-8 and IL-6, and PMN influx (CD, p 8-48). Inflammatory changes in the lung could lead to systemic effects, in that elevated levels of inflammatory cytokines (e.g., interleukin-8) in the respiratory system result in

cardiovascular effects. To date however, no studies have shown a clear-cut link between changes in cardiovascular function and production of cytokines in the lung (CD, p. 8-75).

Lung inflammation could also lead to increased blood coagulability that increases the risk of heart attacks and strokes. It is widely known that increased coagulability of the blood is linked to increased risk of heart attacks (CD, p. 9-92). Some toxicological and epidemiological studies have shown that ambient PM exposure can result in increased levels of fibrinogen (Ghio et al., 2000; Peters al., 2000) or plasma viscosity (Peters et al., 1997), but Godleski et al. (2000) and Seaton et al. (2000) did not report similar changes in fibrinogen or clotting-related blood factors.

Animal studies have provided initial evidence that high particle concentrations can have systemic, especially cardiovascular, effects (CD, p. 8-34). In response, recent epidemiology studies have begun to include more sensitive measures of cardiovascular responses. An increasingly coherent picture is emerging of linkages between ambient PM and such responses. An integrated discussion of this evidence is presented below in Section 3.3.3.3. Several potential mechanisms of relevance to such effects, involving secondary responses to PM effects on the lung, are noted above in Table 3-1. The draft CD also poses possible mechanisms for direct effects on the heart. Inhaled PM could affect autonomic control of the heart and cardiovascular system, with resulting changes in heart rate or heart rate variability. Also, inhaled PM could affect the heart or other organs if particles or particle constituents are released into the circulatory system from the lungs, although this remains somewhat speculative.

In conclusion, dosimetric information shows that both fine- and coarse-fraction particles (smaller than 10 μm) can penetrate and deposit in the tracheobronchial and alveolar regions of the lung. Particles also may carry other harmful substances with them to these regions, with the smaller particles having the greatest surface area available for such transport (see Chapter 2 above). While a variety of responses to constituents of ambient PM have been hypothesized to contribute to the reported health effects, there is no currently accepted mechanism(s) as to how relatively low concentrations of ambient PM may cause the health effects that have been reported in the epidemiological literature. Nevertheless, a substantial and growing base of recent experimental studies is providing important new insights. The draft CD concludes that "[t]he newer experimental evidence, therefore, adds considerable support for interpreting the

epidemiologic findings discussed below as being indicative of causal relationships between exposures to ambient PM and consequent associated increased morbidity and mortality risks." (CD, p. 9-40). The continued emphasis on these lines of research should provide important insights on mechanisms for the next standards review.

3.3 NATURE OF EFFECTS

The 1996 Staff Paper identified the following key health effects categories associated with PM exposure (EPA, 1996b, pp V-8 and V-9):

- Increased mortality
- Indices of morbidity associated with respiratory and cardiovascular disease
    - Hospital admissions and emergency room visits
    - School absences
    - Work loss days
    - Restricted activity days
    - Effects on lung function and symptoms
    - Morphological changes
    - Altered host defense mechanisms

Additional evidence is now available to identify the following new indices of morbidity:

- Physicians' office or clinic visits
- Effects on cardiovascular function indicators, such as heart rate variability

In considering the nature of effects, it is important to note some key characteristics and limitations of the kinds of studies used to identify them. The general strengths and weaknesses of epidemiology studies were discussed in detail in the 1996 CD (Chapter 12) and are briefly reviewed in Section 6.1 of the draft CD. Epidemiology studies can identify associations between actual community-level air pollution containing PM and population-level health effects, and can provide evidence useful in making inferences with regard to the causality of such relationships, although they cannot alone be used to demonstrate mechanisms of action. Epidemiological studies can also provide information that can help to identify sensitive populations particularly at risk for effects (summarized below in Section 3.4).

A central issue in the analysis of epidemiological evidence considered throughout the discussion of effects in this section (and further in Section 3.5) is the role of co-pollutants as potential confounders or effect modifiers in associations between health effects and PM. In addition, co-pollutants may act as indicators for fine particles derived form specific combustion sources; for example, the CD for CO concluded that ambient CO may be a surrogate for air pollution from combustion sources (EPA, 2000a). Confounding occurs when a health effect that is caused by one risk factor is attributed to another variable that is correlated with the causal risk factor; epidemiological analyses attempt to adjust or control for potential confounders. A gaseous co-pollutant (e.g., O_3, CO, SO_2 and NO_2) meets the criteria for potential confounding in PM-health associations if: (1) it is a potential risk factor for the health effect under study; (2) it is correlated with PM; and (3) it does not act as an intermediate step in the pathway between PM exposure and the health effect under study (CD, p. 6-4). Effect modifiers include variables that may influence the health response to the pollutant exposure (e.g., co-pollutants, individual susceptibility, smoking or age); epidemiological analyses do not attempt to control for effect modifiers, but rather to identify and assess the level of effect modification (CD, p. 6-4). Other important issues and uncertainties involved in evaluating epidemiological studies are related to the role of various components within the fine and coarse fractions, as well as various analytical issues including lag periods, model specification, measurement error, and various exposure periods (summarized below in Section 3.5).

Animal toxicology, controlled human exposure, and dosimetry studies can provide important support to epidemiological studies and can help elucidate biological mechanisms that explain observed effects (discussed above in Section 3.2). Such studies can also provide important information on risk factors for individual or population susceptibility to effects and on characteristics of particles (e.g., constituents and subclasses) that may play key roles in the production of health effects. However, as discussed in more detail in Chapter 8 of the draft CD, the doses used in animal studies are generally much higher than community-level concentrations, and important differences in dosimetry can exist across species. As a result, such studies can result in animal models that may not mirror human health responses. Further, controlled human exposure studies can only address the least severe health endpoints, for obvious ethical reasons,

and the need remains to link effects observed in such studies under simulated exposure conditions (e.g., with regard to chemical composition, particle size, and concentration) to those that would likely occur in real-world environments.

Recognizing the different strengths and limitations of these various kinds of studies, key evidence illustrating these major PM effects categories is outlined below, with an emphasis on the most recent information. Mortality effects are discussed in section 3.3.1, with discussion of indices of morbidity in section 3.3.2, organized into three general categories: increased hospital admissions and emergency room visits, effects on the respiratory system, including all other morbidity indices except those related to the cardiovascular system, which are discussed separately as the third category. Finally, the consistency and coherence of the overall body of evidence showing associations between health effects and exposure to fine- and coarse-fraction PM, alone and in combination with other pollutants, is discussed in section 3.3.3, reflecting an integration of information across effects categories and disciplines, and consideration of the role of gaseous co-pollutants.

3.3.1 Premature Mortality

This section discusses (1) mortality associations with short-term PM exposure, with emphasis on results from newly available multi-city analyses, (2) associations with long-term PM exposure, and (3) issues related to interpreting the results of mortality studies, including mortality displacement and life shortening.

3.3.1.1 Mortality and Short-term PM Exposure

Historical reports of dramatic pollution episodes have provided clear evidence of mortality associated with high levels of PM and other pollutants, as summarized in the 1996 CD (EPA, 1996a, pp. 12-28 to 12-31) and Staff Paper (EPA, 1996b, p. V-11). More recently, associations between increased daily mortality and PM have been reported at much lower PM concentrations in a large number of areas with differing climates, PM composition, and levels of gaseous co-pollutants. The 1996 CD summarized about 35 time-series mortality studies using various PM

indicators; the majority of these studies reported positive, statistically significant[2] associations for PM_{10}, as well as for $PM_{2.5}$ and other indicators of fine-fraction particles (e.g., sulfates and H^+). Significant associations were reported for total mortality[3] for PM_{10} and indicators of fine-fraction particles (EPA, 1996b, Tables V-3, V-11, V-12) and cause-specific mortality (i.e., respiratory- and cardiovascular-related mortality) in the general population and in the elderly for PM_{10} (EPA, 1996b, Table V-4). In the 1996 CD, one daily mortality study addressed coarse-fraction particles ($PM_{10-2.5}$), reporting no statistically significant association across the six cities included in the study, although a significant association was reported in one of the six cities (EPA, 1996b, Table V-14).

In the previous PM NAAQS review, much consideration was given to the effects of PM and co-pollutants, acting alone and in combination, in the associations with adverse health effects reported in epidemiological studies. The 1996 CD evaluated the findings of studies that used single- and multiple-pollutant models to assess the potential for co-pollutant confounding and effects modification. In some studies, PM effect estimate sizes were relatively unchanged when gaseous pollutants were included in the models, and where the estimate was reduced, it typically remained statistically significant (EPA, 1996a, p. 13-57). Much attention was focused on a series of analyses and reanalyses using data from one U.S. city, Philadelphia, the most comprehensive of which was a study funded by the Health Effects Institute (HEI). This study reported associations between mortality and TSP and other pollutants, concluding that it was difficult to distinguish the effects of TSP from one or more gaseous co-pollutants for this single location due in part to the fact that the co-pollutants were generally correlated with TSP. Indeed, the limitations of even the most comprehensive single-city analyses precluded definitive conclusions concerning the role of PM. For this reason, both the 1996 CD and Staff Paper examined the consistency and coherence of effects across studies of individual cities having different pollutant mixtures, climate, and other factors. Based on the consistent positive associations found in such multiple studies, the CD

[2]Unless otherwise noted, statistically significant results are reported at a 95% confidence level.

[3]In these discussions, "total" mortality represents mortality from all causes excluding accidents and suicides, as the term is typically used in epidemiological studies on mortality and air pollution.

concluded that PM effects were not sensitive to other pollutants and the "findings regarding the PM effects are valid" (CD, p 13-57, SP, p V-56).

Taking into account these findings, the HEI Oversight Committee recommended that future research into the role of co-pollutants should improve upon the examination of multiple single city studies by different investigators by conducting multi-city studies, using consistent analytical approaches across cities, noting that "[c]onsistent and repeated observations in locales with different air pollution profiles can provide the most convincing epidemiological evidence to support generalizing the findings from these models" (HEI, 1997, p. 38).

Since the last review, more than 70 new time-series daily PM-mortality studies have been published (Table 6-1 of the draft CD), including several multi-city studies that are responsive to the recommendations from the last review. The draft CD notes that with only a few exceptions, these newly reported associations are generally positive, many are statistically significant (using both single- and multi-pollutant models), and the reported effects estimates are generally consistent with the range of estimates from the last review (CD, p. 9-44). Drawing from the current draft CD and the 1996 CD, Appendix A, Table 1, summarizes increased daily mortality effects estimates for increments of PM_{10}, $PM_{2.5}$, and $PM_{10-2.5}$ from all available multi-city and single-city U.S. and Canadian studies[4] as a consolidated reference for the following discussion of associations between daily PM and increased total and cause-specific mortality.

3.3.1.1.1 Multi-city Studies of Total Daily Mortality

In considering the body of evidence on associations between PM and mortality in this standards review, the multi-city studies are of particular relevance. The multi-city studies combine data from a number of cities that may vary in climate, air pollutant sources or concentrations, and other potential risk factors. The advantages of multi-city analyses include: (1) evaluation of associations in larger data sets can provide more precise effects estimates than pooling results from separate studies; (2) consistency in data handling and model specification can eliminate variation due to study design; (3) effect modification or confounding by co-pollutants

[4] Findings of U.S. and Canadian studies are more directly applicable for the review of the PM NAAQS, though all study results are considered in the overall review of new scientific information. For consistency across studies, the effects estimates summarized in Appendix A, Table 1, are from single-pollutant models.

can be evaluated by combining data from areas with differing air pollutant combinations; (4) regional or geographical variation in effects can be evaluated; and (5) "publication bias" or exclusion of reporting of negative or nonsignificant findings can be avoided (CD, p. 6-39).

In the previous review, a single multi-city study evaluated associations between daily mortality and PM, including fine- and coarse-fraction particles for six U.S. cities (Schwartz et al., 1996). Significant increases in total mortality of 4.0% and 3.8% were reported per 25 $\mu g/m^3$ and 50 $\mu g/m^3$ of $PM_{2.5}$ and PM_{10}, respectively, while $PM_{10-2.5}$ was not significantly associated with mortality. Two new analyses of the six-city data have reported results consistent with the findings reported by Schwartz and colleagues (Klemm and Mason, 2000; Laden et al., 2000). The role of gaseous co-pollutants was not directly addressed in any of these analyses.

Several new multi-city analyses, discussed below, provide valuable new insights on associations between PM and mortality, including more direct evaluation of the role of co-pollutants in PM-mortality associations through the use of multi-pollutant modeling.

The National Morbidity, Mortality and Air Pollution Study (NMMAPS) included analyses of PM_{10} effects on mortality in 90 U.S. cities, with additional, more detailed, analyses being conducted in a subset of the 20 largest U.S. cities (discussed below in sections on cause-specific mortality and morbidity) (Samet et al., 2000a,b,c; Domenici et al., 2000). A uniform methodology was used to evaluate the relationship between mortality and PM_{10} for the different cities, and the results were synthesized to provide a combined estimate of effects across the cities. These analyses are "marked by extremely sophisticated approaches addressing issues of measurement error biases, co-pollutant evaluations, regional spatial correlation, and synthesis of results from multiple cities by hierarchical Bayesian meta-regressions and meta-analyses" (CD, p. 6-39, 6-40).

As seen in Figure 3-1, the overall risk estimate for all cities is a statistically significant increase of 2.3% in total mortality per 50 $\mu g/m^3$ increase in PM_{10} lagged one day[5] (Samet et al., 2000a,b). Further, PM_{10} was also positively associated with mortality at 0-day and 2-day lags. In two additional reports on analyses using data from the 20 largest U.S. cities, reported increases in

[5]Note that Figure 3-1 includes results for 88 cities in the continental U.S.; Anchorage, AK and Honolulu, HI are not included.

total mortality per 50 μg/m^3 increase in PM_{10} were 1.9% (Domenici et al., 2000) and 2.6% (Samet et al., 2000c).

Also seen in Figure 3-1 are the results based on a regional assessment of these cities, using seven U.S. regions. Samet et al. (2000a,b) report that some variability in effects can be seen across cities and between regions. As seen in Figure 3-1, effect estimates for individual cities vary; some are even negative, though not statistically significant. In addition, combined effect estimates for each of the seven U.S. regions varied, with generally higher effects reported in the Northeast States (a 4.5% increase in total mortality per 50 μg/m^3 increase in PM_{10} lagged one day) and in Southern California. Data on some county-specific variables (e.g., mean household income, percent of people not graduating from high school, percent of people using public transportation) were included in analyses to investigate regional differences, but the investigators did not identify any factors that might explain the apparent differences (CD, p. 6-43).

Notable variability in effects estimates across the 90 cities in this study would not be unexpected when taking into account the study design that included many locations for which the sample size (in terms of population and amount of PM_{10} data) was inherently smaller for a given study period. To further examine the observed variability, the draft CD presents the 90-city effect estimates plotted against the natural log of mortality-days (a product of each city's daily mortality rate and the number of days for which PM data were available) as an indicator of the statistical power of the analysis of each individual city (Figure 3-2). Traditionally, sample size is an important factor in assessing the statistical power of a study, and, in time-series studies, the extent of the time series is one measure of sample size, as is the number of health events per day (or alternative time interval). In the multi-stage analyses, the NMMAPS investigators used several weighting methods in combining estimates from the individual cities. As seen in Figure 3-2, cities with the greatest weight or statistical power tended to have more precise effect estimates (with narrower confidence intervals), and these effect estimates were generally positive

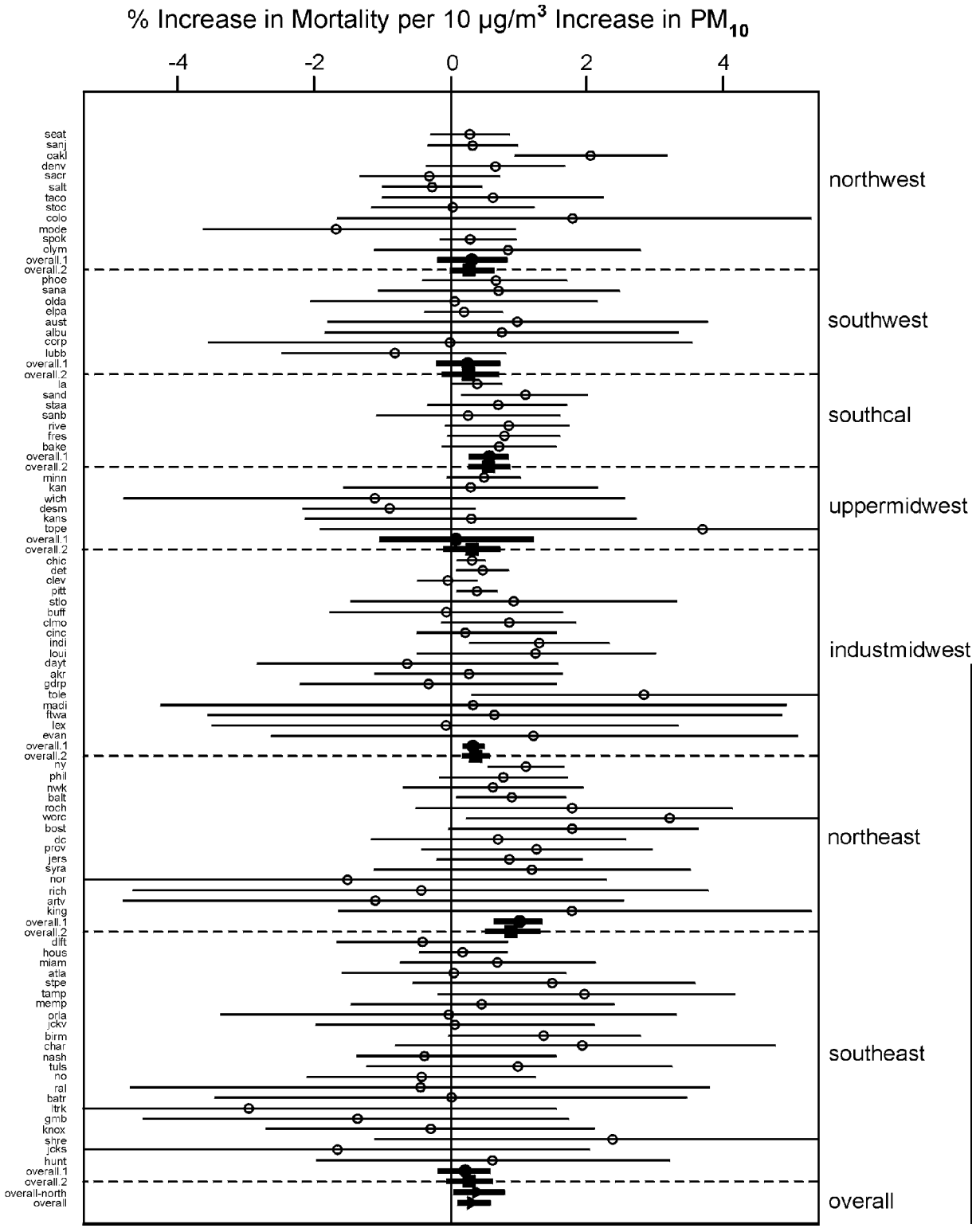


Figure 3-1. PM_{10}-mortality effects estimates for the 88 largest U.S. cities as shown in the original NMMAPS report. From Samet et al. (2000a,b). (CD Figure 6-1).

and statistically significant. The draft CD concludes that this "suggests some relationship between effect size and study weight, overall" (CD, p. 6-212), indicating that variation in study power may be a factor in explaining the apparent variation in effects estimates across cities. The draft CD also presents these relationships on a regional basis (Figure 6-13, p. 6-262), suggesting that further examination of these relationships may reveal interesting new insights into factors that may account for any apparent intra- and inter-regional disparities (CD, p. 263).

One key objective of the NMMAPS analysis was to characterize the effects of PM_{10} and each of the gaseous co-pollutants, alone and in combination. An important result of this assessment is the finding that the associations reported between PM_{10} and mortality in the 90-city analyses were not confounded by the presence of the gaseous co-pollutants (Samet et al., 2000b). As seen in Figure 3-3, the effect of inclusion of other pollutants in this model on the association between PM_{10} and mortality ranges from small to modest, and importantly does not affect the statistical significance of the PM_{10} estimates. Significant single-pollutant associations were reported for mortality for three of the gaseous co-pollutants (CO, NO_2 and SO_2), and a significant association was reported for O_3 in the summer. The effects of the gaseous pollutants were, however, generally diminished in multi-pollutant models that included PM_{10} (CD, p. 6-222). The effects of CO alone were generally positive and significant, but adjustments for other pollutants tended to reduce the effect. The authors concluded that "[t]his figure suggests that the effect of PM_{10} is robust to the inclusion of other pollutants." (Samet et al., 2000b, p. 19).

Schwartz (2000a) conducted a series of multi-city analyses using data from 10 U.S. cities where every-day PM monitoring data were available (in many areas, PM is monitored on a 1-in-3 or 1-in-6 day basis). Using inverse variance weighting methods to combine results across cities, a statistically significant association was reported between PM_{10} and mortality, with an effect estimate of a 3.4% increase per 50 $\mu g/m^3$ PM_{10}, and effect estimate sizes were the same in summer and winter (CD, p. 6-44). This study also included the use of an alternative analytical approach to assess confounding by co-pollutants. This approach uses data from multiple locations and assesses whether there is an association between the PM effect estimate and the PM-gaseous pollutant relationship in each location. A statistical relationship is first developed

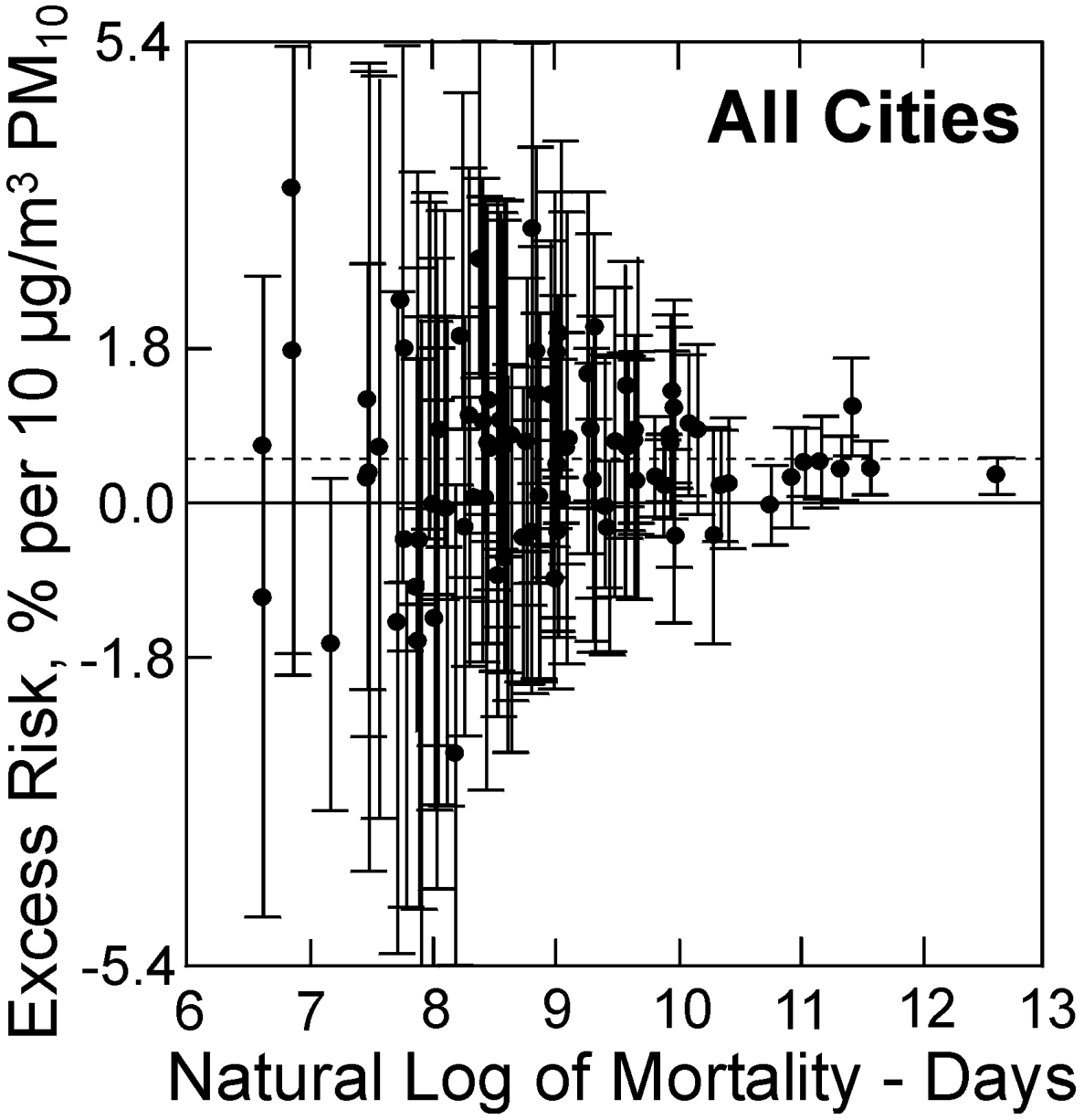


Figure 3-2. The EPA-derived plot showing relationship of PM_{10} total mortality effects estimates and 95% confidence intervals for all cities in the Samet et al. (2000a,b) NMMAPS 90-cities analyses in relation to study size (i.e., the natural logarithm of numbers of deaths times days of PM observations). Note generally narrower confidence intervals for more homogeneously positive effects estimates as study size increases beyond about the log 9 value (i.e., beyond about 8,000 deaths-days of observation). The dashed line depicts the overall nationwide effect estimate (grand mean) of approximately 0.5% per 10 µg/m^3 PM_{10} (CD Figure 6-12).

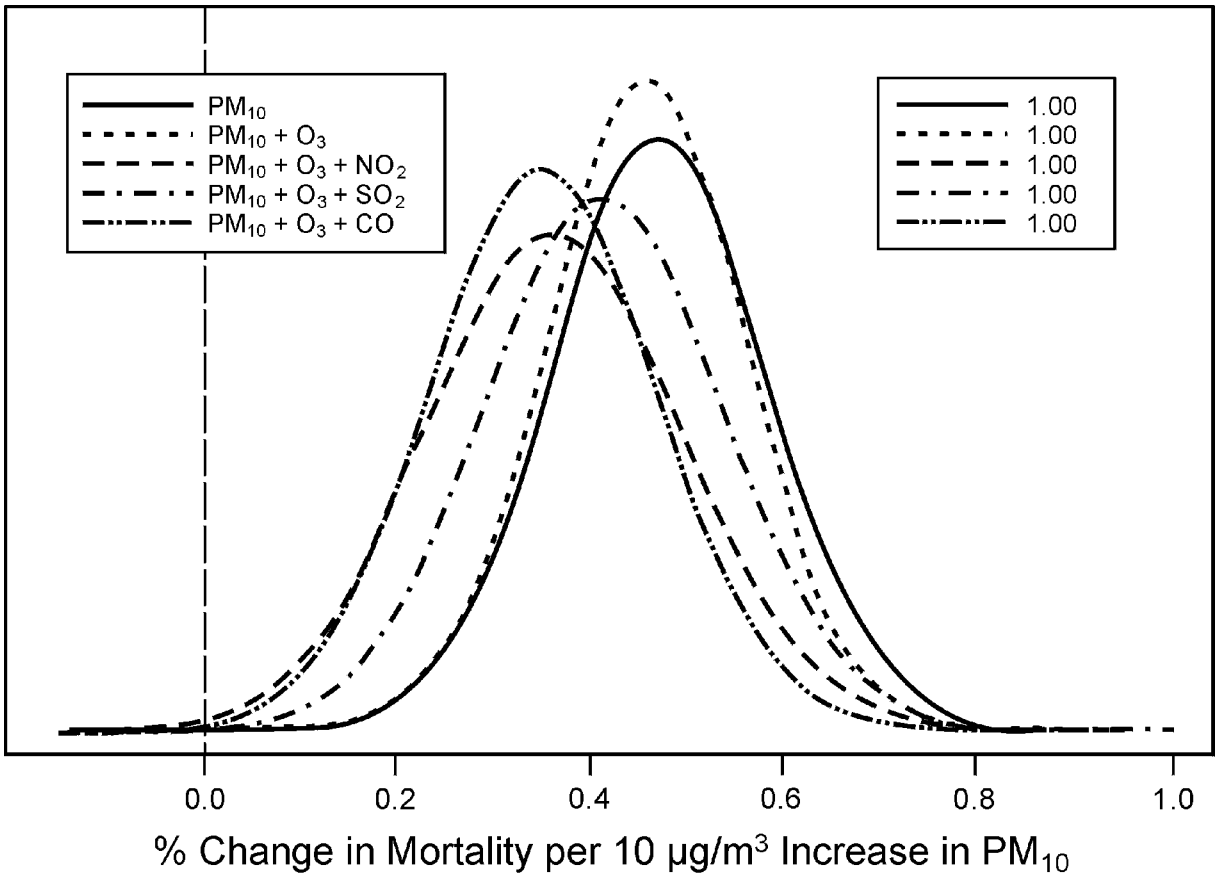


Figure 3-3. Marginal posterior distributions for effect of PM_{10} on total mortality at lag 1 with and without control for other pollutants, for the 90 cities. The numbers in the upper right legend are the posterior probabilities that the overall effects are greater than 0. (From CD Figure 6-10)

Source: Samet et al. (2000a,b).

for PM and the co-pollutant, then in multi-stage modeling, the PM-health model includes adjustment for the PM-co-pollutant correlation. The expectation is that, if an association with PM is really due to confounding by another pollutant, there would be a trend toward larger effects being found in areas where the coefficient between PM and the other pollutant is larger (CD, p. 6-225). No relationship was reported between PM_{10}-mortality associations and coefficients between PM_{10} and O_3, CO, or SO_2, suggesting a lack of confounding by co-pollutants.

Further analyses of subsets of the 10 U.S. cities investigated additional research questions, including the form of the concentration-response function and assessment of possible effect thresholds, and the influence of influenza epidemics on PM-mortality relationships (Schwartz, 2000a,b,d; Schwartz and Zanobetti, 2000; Zanobetti and Schwartz, 2000; and Braga et al., 2000). These findings will be discussed further as each topic is addressed in this chapter.

In a combined analysis of data for the 8 largest Canadian cities, Burnett et al. (2000) reported that mortality was significantly associated with both $PM_{2.5}$ and PM_{10}, but not $PM_{10-2.5}$. Overall effect estimates for increased total mortality of 3.0% and 3.5% were reported per 25 $\mu g/m^3$ and 50 $\mu g/m^3$ increases in $PM_{2.5}$ and PM_{10}, respectively. Additional analyses were conducted using $PM_{2.5}$ components, including sulfates and a number of metals, and these results are discussed further in Section 3.5.2. The Canadian 8-city study also showed that the associations between mortality and $PM_{2.5}$ and PM_{10} generally remained significant in a number of analyses when gaseous co-pollutants and 0- and 1-day lags were included in the models, although in a few instances the effects estimates were reduced and lost statistical significance. The authors conclude that mortality is associated with both PM and gaseous pollutants (Burnett et al., 2000).

In addition, a European multi-city study, Air Pollution and Health: A European Project (APHEA), has resulted in a series of analyses that were summarized in the draft CD (pp. 6-47 to 6-49). Although the studies used consistent analytical methodologies, the PM measurement methods varied between cities, including TSP, BS, PM_{13}, and PM_{10}, thus making the quantitative comparisons with U.S. and Canadian findings more difficult. Significant associations between various measures of PM and mortality were reported in some overall analyses, with differences reported between regions. The effects estimates reported for western cities, approximately 2% increase in mortality per 50 $\mu g/m^3$ PM_{10}, are consistent with those reported in U.S. and Canadian studies, but no significant associations were reported with data from central or eastern European countries. The APHEA investigators postulated a number of potential reasons for variation between regions, such as differences in exposure representativeness, pollution mix, sensitive sub-population proportions, or model fit for seasonal control (CD, p. 6-48).

The results from each of the U.S. and Canadian multi-city studies are summarized in Table 3-2 (including the two reanalyses of data from six U.S. cities used in Schwartz et al., 1996). The

draft CD notes that the combined daily mortality estimates from these multi-city studies are all consistent with the range of PM_{10} effects estimates reported in the last review (CD, p. 6-49) (i.e., 1.5% to 8.5% per 50 µg/m^3 PM_{10}), with the 90-city estimate toward the lower end of the range. Further, similarly sized effect estimates are reported between total mortality and PM_{10} and $PM_{2.5,}$ but no significant associations are reported with $PM_{10-2.5}$.

TABLE 3-2. RESULTS OF U.S. AND CANADIAN MULTI-CITY STUDIES ON ASSOCIATIONS BETWEEN SHORT-TERM PM EXPOSURE AND MORTALITY

Study	% Increase in Mortality per 50 µg/m^3 $PM_{15/10}$	% Increase in Mortality per 25 µg/m^3 $PM_{2.5}$	% Increase in Mortality per 25 µg/m^3 $PM_{10-2.5}$	Range of City PM Mean Levels (µg/m^3)
Six U.S. Cities Schwartz et al., 1996	*4.04 (2.53, 5.62)*	*3.79 (2.77, 4.82)*	*1.00 (-0.37, 2.40)*	*PM_{10} 17.8-45.6 $PM_{2.5}$ 11.2-29.6 $PM_{10-2.5}$ 6.6-16.1*
Six U.S. Cities (reanalysis) Klemm and Mason, 2000	4.08 (2.78, 5.36)	3.28 (2.27, 4.31)	1.00 (-0.37, 2.40)	$PM_{15/10}$ medians 14.4-30.3 $PM_{2.5}$ medians 9.0-23.1 $PM_{10-2.5}$ medians 5.0-13.0
Six U.S. Cities (new analysis) Laden et al., 2000	---	4.05 (2.78, 5.34)	---	$PM_{2.5}$ NR
90 U.S. Cities Samet et al., 2000a,b	2.27 (0.10, 4.48)	---	---	PM_{10} 15.3-52.0
10 U.S. Cities Schwartz et al., 2000	3.40 (2.65, 4.14)	---	---	PM_{10} 27.1-40.6
8 Canadian Cities Burnett et al., 2000	3.51 (1.04, 6.04)	3.03 (1.10, 4.99)	1.82 (-0.72, 4.43)	PM_{10} 20.4-31.0 $PM_{2.5}$ 9.5-17.7 $PM_{10-2.5}$ 8.9-16.8

In summary, the findings of the Six-Cities study that was available during the previous review have been confirmed by new analyses, and powerful new multi-city analyses have provided important new evidence showing associations between daily mortality and changes in PM_{10} and $PM_{2.5,}$ alone and in combination with gaseous co-pollutants routinely present in the ambient air.

3.3.1.1.2 Other Studies of Total Daily Mortality

Numerous studies have been conducted in single cities or locations in the U.S. or Canada (summary of results in Appendix A, Table 1), as well as locations in Europe, Mexico City, South America, Asia or Australia (summary of results in Table 6-1 of the draft CD). As was observed based on the more limited studies available in the last review, the associations reported in the recent studies on PM_{10} and mortality are largely positive, and frequently statistically significant. Similarly, a number of new studies also provide evidence of statistically significant associations with $PM_{2.5}$. In contrast, statistically significant associations were not generally reported for $PM_{10-2.5}$. Using the same approach taken in the CD in presenting the NMMAPS results (Figure 3-2), the results of U.S. and Canadian single-location and multi-city analyses for mortality with PM_{10}, $PM_{2.5}$, and $PM_{10-2.5}$ (using single-pollutant model results) are plotted in Figures 3-4, 3-5 and 3-6, respectively. Effect estimates are plotted in order of increasing study power or weight, and, as seen in Figure 3-2, there is the expected tendency for results of studies with greater power to have more precise effect estimates. Along with the new study findings, each figure includes effect estimates for studies included in the 1996 CD and, for comparison purposes, the range of statistically significant effect estimates from the previous review. Effect estimates for total, cardiovascular and respiratory mortality are included to give an overview of the entire body of mortality studies, though cause-specific findings will be discussed further in the next section.

A number of new single-city analyses have included multi-pollutant modeling for evaluating effects of PM and co-pollutants. As was found in the previous review, some of these analyses report that PM effect sizes are little affected by the inclusion of co-pollutant gases in the models, while others report potential confounding by one or more co-pollutants. In U.S. studies conducted in Coachella Valley and Santa Clara County, California and Detroit, Michigan, investigators concluded that generally positive associations (both significant and non-significant) between PM and mortality were relatively unchanged in multi-pollutant models (Ostro et al.,

1999, 2000; Lippmann et al., 2000; Fairley, 1999). As in the previous review, some of the new single-city studies found evidence of confounding. In the U.S., based on analyses in Cook, Los Angeles, and Maricopa Counties, Moolgavkar (2000a) reported that the inclusion of gaseous co-pollutants resulted in large reductions in PM effect estimates.

As seen in Figures 3-4 and 3-5, associations between total mortality and both PM_{10} and $PM_{2.5}$ are generally positive and many reach statistical significance, especially in those studies with greater study power or weight. For both, the results of the larger studies show quantitative consistency in findings between studies, as well as with the ranges of statistically significant effects estimates from the 1996 CD. The range of findings among the smaller studies is greater with a few fairly large effects estimates, some of which attain statistical significance, but with much larger confidence intervals. In contrast, few significant associations were reported with $PM_{10-2.5}$ (Figure 3-6), with none occurring among the studies with greater power.

While some of the studies conducted in Europe, Mexico or South America use gravimetric PM measurements (e.g., PM_{10}, $PM_{2.5}$, $PM_{10-2.5}$), many of the non-North American studies use PM indicators such as TSP, BS or COH, and the Australian studies use nephelometric measures of PM. As summarized in Table 6-1 of the draft CD, these studies also show largely positive, significant associations between PM and mortality. While effect estimates for different PM indicators may not be quantitatively comparable, the results from all of these studies taken together show qualitative consistency in finding significant associations between changes in PM and daily mortality.

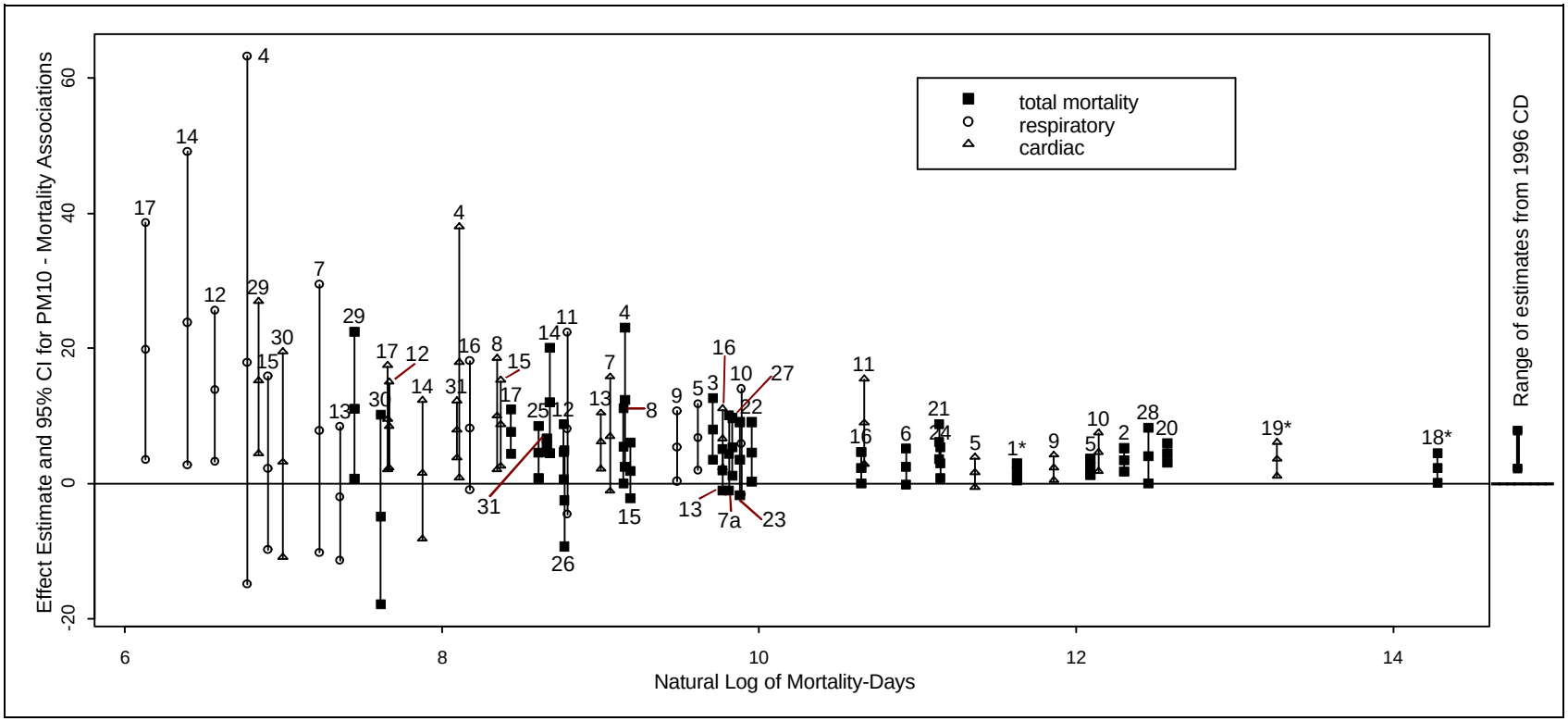


Figure 3-4. Effects estimates for PM_{10} and mortality from total, respiratory and cardiovascular causes from U.S. and Canadian cities in relation to study size, in terms of the natural log of the mortality-days product (the product of study days and the number of deaths per day) as an indicator of study weight, or power. Note that the study findings become more precise and quantitatively consistent as study power increases. Multi-city studies denoted with an asterisk above; study locations are identified below (data in Appendix 3-A, Table 4A)

1. Burnett et al., 2000, 8 Canadian cities
2. Burnett et al., 1998, Toronto
3. Fairley, 1999, Santa Clara
4. Gwynn et al., 2000, Buffalo
5. Ito and Thurston, 1996, Chicago
6. Kinney et al., 1995, LA
7. Lippmann et al., 2000, Detroit
8. Mar et al., 2000, Phoenix
9. Moolgavkar, 2000a, Cook Co
10. Moolgavkar., 2000a, LA
11. Moolgavkar , 2000a, Maricopa
12. Ostro et al., 1999, Coachella Valley
13. Ostro et al., 2000, Coachella Valley
14. Pope et al., 1999, Ogden
15. Pope et al., 1999, Provo/Orem
16. Pope et al., 1999, Salt Lake City
17. Pope et al., 1992, Utah Valley
18. Samet et al., 2000b, 90 U.S. city
19. Samet et al., 2000c, 20 U.S. city
20. Schwartz and Zanobetti, 2000, Chicago
21. Schwartz et al., 1996, Boston
22. Schwartz et al., 1996, Knoxville
23. Schwartz et al., 1996, Portage
24. Schwartz et al., 1996, St. Louis
25. Schwartz et al., 1996, Steubenville
26. Schwartz et al., 1996, Topeka
27. Schwartz., 1993, Birmingham
28. Styer et al., 1995, Chicago
29. Tsai et al., 2000, Camden NJ
30. Tsai et al., 2000, Elizabeth NJ
31. Tsai et al., 2000, Newark NJ

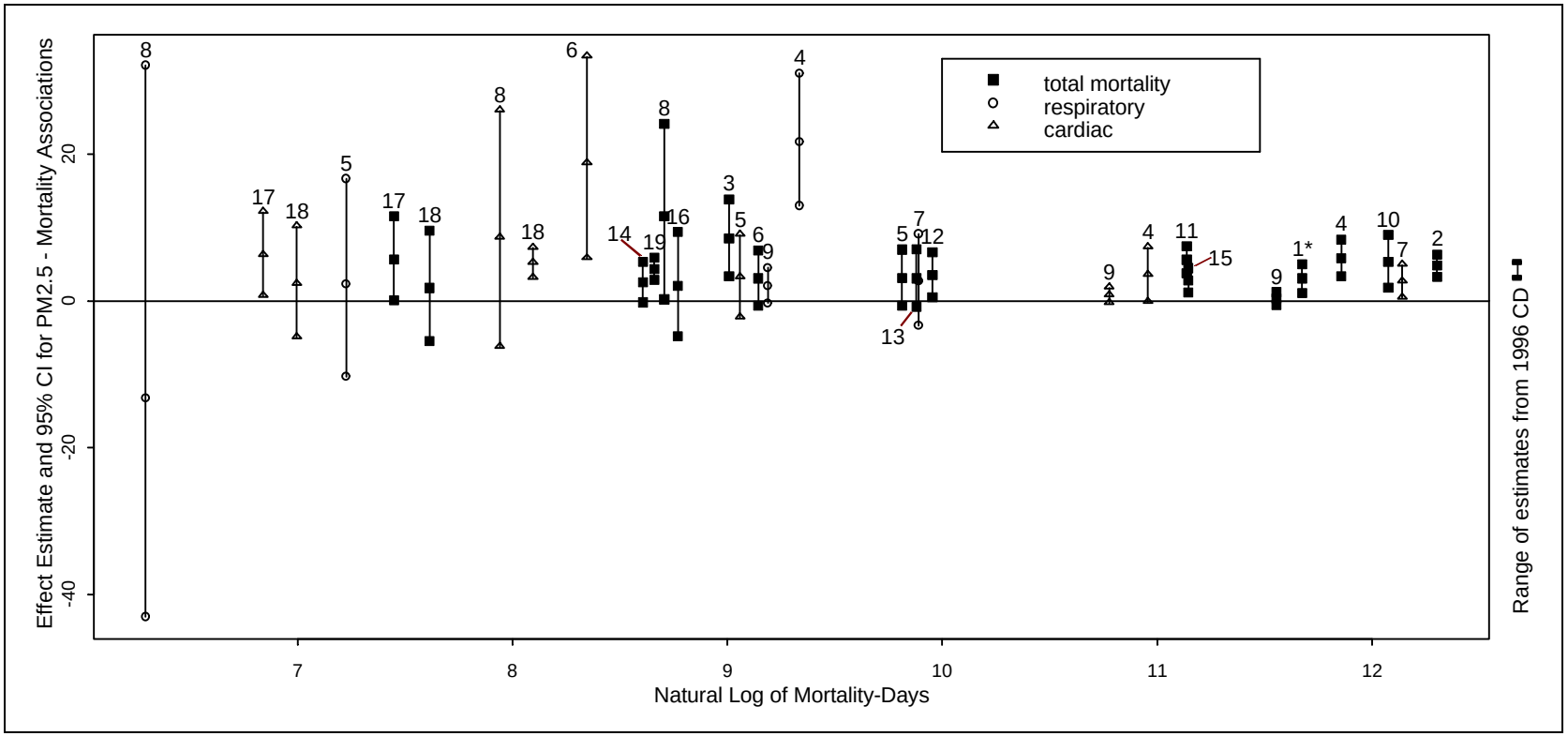


Figure 3-5. Effects estimates for $PM_{2.5}$ and mortality from total, respiratory and cardiovascular causes from U.S. and Canadian cities in relation to study size, in terms of the natural log of the mortality-days product (the product of study days and the number of deaths per day) as an indicator of study weight, or power. Note that the study findings become more precise and quantitatively consistent as study power increases. Multi-city studies denoted with an asterisk above; study locations are identified below (data in Appendix A, Table 4)

1. Burnett et al., 2000, 8 Canadian cities
2. Burnett et al., 1998, Toronto
3. Fairley, 1999, Santa Clara
4. Goldberg et al., 2000, Montreal
5. Lippmann et al., 2000, Detroit
6. Mar et al., 2000, Phoenix
7. Moolgavkar., 2000a, LA
8. Ostro et al., 1995, So. California
9. Ostro et al., 2000, Coachella Valley
10. Schwartz 2000c, Boston
11. Schwartz et al., 1996, Boston
12. Schwartz et al., 1996, Knoxville
13. Schwartz t al., 1996, Portage
14. Schwartz et al., 1996, St. Louis
15. Schwartz et al., 1996, Steubenville
16. Schwartz et al., 1996, Topeka
17. Tsai et al., 2000, Camden NJ
18.Tsai et al., 2000, Elizabeth NJ
19. Tsai et al., 2000, Newark NJ

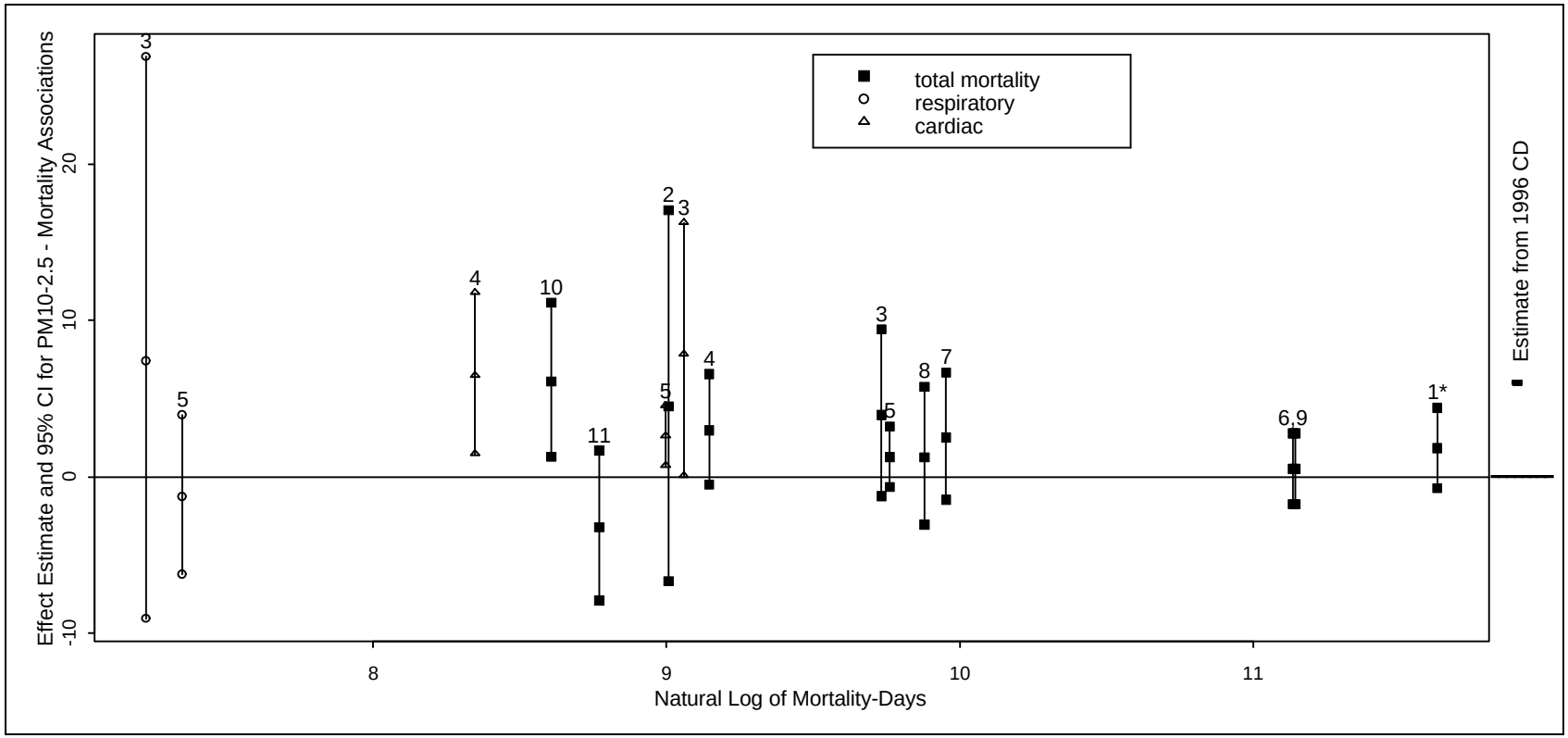


Figure 3-6. Effects estimates for $PM_{10-2.5}$ and mortality from total, respiratory and cardiovascular causes from U.S. and Canadian cities in relation to study size, in terms of the natural log of the mortality-days product (the product of study days and the number of deaths per day) as an indicator of study weight, or power. Note that the study findings become more precise and quantitatively consistent as study power increases. Multi-city studies denoted with an asterisk above; study locations are identified below (data in Appendix 3-A, Table 4C)

1. Burnett et al., 2000, 8 Canadian cities
2. Fairley, 1999, Santa Clara
3. Lippmann et al., 2000, Detroit
4. Mar et al., 2000, Phoenix
5. Ostro et al., 2000, Coachella Valley
6. Schwartz et al., 1996, Boston.
7. Schwartz et al., 1996, Knoxville
8. Schwartz et al., 1996, Portage
9. Schwartz et al., 1996, St. Louis
10. Schwartz et al., 1996, Steubenville
11. Schwartz et al., 1996, Topeka

3.3.1.1.3 Cause-specific Daily Mortality

In the 1996 Staff Paper, several studies also reported associations between PM_{10} and respiratory and cardiovascular mortality (EPA, 1996b, p. V-13). The associations reported with mortality from respiratory or cardiovascular diseases were generally consistent with the results for total mortality, and the CD concluded that this lent support to the biological plausibility of the PM associations (EPA, 1996a, p. 12-69). If particles have effects on the respiratory or cardiovascular systems, it would be expected that associations reported for total mortality reflect the underlying associations with cardiorespiratory[6] mortality and not be influenced by deaths from non-cardiorespiratory causes (EPA, 1996a, p. 12-77).

Figures 3-4, 3-5, and 3-6 shown above present findings for PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$, respectively, from U.S. and Canadian studies, where it can be seen that there is general consistency between effects estimate ranges for mortality from total, respiratory and cardiovascular causes. In general, as was observed in the 1996 CD, some of the effect estimates for respiratory mortality are larger in magnitude but less precise, with large confidence intervals, which is likely because respiratory-related deaths comprise a small proportion of daily mortality rates.

A number of studies have evaluated associations for both total and cause-specific mortality. The recent U.S. multi-city study, NMMAPS, included a comparison of findings for total and cardiorespiratory mortality for the 20 largest U.S. cities. The effect estimate for deaths from cardiorespiratory causes was somewhat larger (3.5% increase per 50 $\mu g/m^3$ increase in PM_{10}) than that for deaths from all causes (2.6% increase per 50 $\mu g/m^3$ increase in PM_{10}) (Samet et al., 2000c). In the results of individual studies, as summarized in Appendix A, Table 1, effects estimates for mortality from respiratory and cardiovascular causes tend to be larger than those for total mortality, though these comparisons are not readily apparent in Figures 3-4 through 3-6 when combined with all study results. For example, Tsai et al. (2000) also report cardiorespiratory mortality effect estimates with $PM_{2.5}$ and PM_{15} that are somewhat larger than those for total mortality. For respiratory and cardiovascular mortality, nearly all of the U.S. and

[6] "Cardiorespiratory" refers to cardiovascular and respiratory diseases, combined, and is used here as an equivalent term to "cardiopulmonary".

Canadian studies show somewhat larger effects estimates than for total mortality associations with PM_{10} and $PM_{2.5}$ (e.g., Gwynn et al., 2000; Ostro et al., 1999; Pope et al., 1999; Fairley, 1999; Lippmann et al., 2000; Mar et al., 2000; Goldberg et al., 2000) (results in Appendix A, Table 1). As was found with total mortality, few significant associations were reported with $PM_{10-2.5}$ for cause-specific mortality; however, in those few studies, the effects estimates for cardiovascular mortality tended to be greater than those for total mortality (Mar et al., 2000; Ostro et al., 2000).

In NMMAPS analyses, a positive, but not statistically significant, association was also reported with "other" or non-cardiorespiratory deaths (Samet et al., 2000c). In some analyses where "other" causes of death were evaluated, no associations with PM were reported (Ostro et al., 1999, 2000). Some associations between PM and "other" mortality were reported in a Detroit study (Lippmann et al., 2000), but the draft CD observes "that the 'other' mortality showed seasonal cycles and apparent influenza peaks, suggesting that this series may have also been influenced by respiratory contributing causes" (CD, p. 6-72). In Montreal, fine PM was associated with "other nonaccidental causes" of death, but when analyses included more specific "other" causes, significant associations were reported only for diabetes, which typically also involves cardiovascular complications as it progresses (Goldberg et al., 2000). The draft CD concludes, "at least some of these 'other' associations may also be due to seasonal cycles that include relationships to peaks in influenza epidemics that may imply respiratory complications as a 'contributing' cause to the 'other' deaths. Or, the 'other' category may include sufficient numbers of deaths due to diabetes or other diseases which may also involve cardiovascular complications as contributing causes." (CD, p. 6-75).

In addition to the evidence from epidemiology studies, new, though limited, information is available from toxicology studies that offers insight into PM-related mortality. In some of the toxicology studies summarized in Chapter 8 of the draft CD, animals died after exposure to PM or PM surrogates, though none of these studies was designed to assess lethality. For example, some studies have used monocrotaline-treated rats as a model for individuals with cardiorespiratory disease, and "have demonstrated that intratracheal instillation of high levels of ambient particles can increase or accelerate death related to monocrotaline administration in rats" (CD, p. 8-25). Indicators of inflammation or cardiac arrhythmia were also measured in these studies (CD, Table 8-7). While the suitability of this animal model may be questioned, the findings offer some

evidence of plausibility to the associations with cardiorespiratory mortality reported in epidemiology studies. Since the studies were designed to assess effects on cardiovascular or respiratory systems, the toxicological evidence for PM-related effects is more fully discussed in the sections on respiratory and cardiovascular systems effects.

In summary, the new studies continue to report risks for mortality from cardiovascular and respiratory diseases with increasing PM, and the findings suggest that associations reported for total mortality are indicative of associations with deaths from cardiorespiratory-related causes.

3.3.1.2 Mortality and Long-term PM Exposure

The 1996 CD summarized the findings of a number of cross-sectional studies that had been conducted over the past several decades. These studies had identified associations between increased mortality and residence in communities with higher pollution levels, but concern was raised about the lack of information on potentially important covariates and methodological limitations (EPA, 1996a, p. 12-159). Results were also available from three more recent prospective cohort studies (i.e., the Six Cities, American Cancer Society (ACS), and California Seventh Day Adventist (ASHMOG) studies) that included subject-specific information on potential confounders (e.g., smoking history, occupation, health history) and were considered to provide more reliable results (EPA, 1996a, p. 13-33).

The strongest evidence from the prospective cohort studies was reported for associations with fine particles. The ACS study reported significant associations for $PM_{2.5}$ and sulfates (a fine particle surrogate). The Six Cities study evaluated effects of many PM size classes, and significant associations were reported with PM_{15}, $PM_{2.5}$, sulfates and non-sulfate fine particles, but not with TSP or coarse particles (TSP-PM_{15} or PM_{15}-$PM_{2.5}$) (EPA, 1996a, Table 12-18). Both the Six Cities and ACS studies reported associations with mortality from all causes and cardiorespiratory causes, with larger effects estimates for cardiorespiratory causes. The AHSMOG study did not find an association between TSP and mortality. The CD concluded that the chronic exposure studies, taken together, suggested associations between increases in mortality and long-term exposure to PM (EPA, 1996a, p. 13-34).

The new studies that are available for the current review include a comprehensive reanalysis and extended analyses of data from the Six Cities and ACS studies (Krewski et al., 2000) and new analyses using updated data from the AHSMOG study (Abbey et al., 1999).

Findings from the original Six Cities, ACS, and AHSMOG investigations together with those from new studies and reanalyses are summarized in Table 3-3.

The reanalysis of the Six Cities and ACS studies included two major components, a replication and validation study, and a sensitivity analysis, where alternative risk models and analytic approaches were used to test the robustness of the original analyses. In the first phase, the Investigators reported the data from the two studies to be of generally high quality, and was able to replicate the original results, confirming the original investigators' findings of associations with both total and cardiorespiratory mortality (CD, p. 6-83).

The sensitivity analyses generally reported that the use of alternative models, including variables that had not been used in the original analyses (e.g., physical activity, lung function, marital status), did not materially alter the original findings. The Investigators also obtained data on additional city-level variables that were not available in the original data sets (e.g., population change, measures of income, maximum temperature, number of hospital beds, water hardness) and included these data in the models. The associations between fine particles and mortality were generally unchanged in these new analyses, with the exception of population change, which did somewhat reduce the size of the associations with fine particles or sulfates.

Further analyses were conducted using data for potentially susceptible subgroups, and the results did not show differences in the PM-mortality associations between most subgroups, including gender, smoking status, exposure to occupational dusts and fumes, and marital status. However, the effects of fine particles appeared to be larger in the subgroup without a high school education than with more education; the Investigators postulated that this relationship could be due to some unidentified socioeconomic effect modifier.

TABLE 3-3. EFFECT ESTIMATES PER INCREMENTS[A] IN LONG-TERM MEAN LEVELS OF FINE AND INHALABLE PARTICLE INDICATORS FROM U.S. AND CANADIAN STUDIES

Type of Health Effect & Location	Indicator	Change in Health Indicator per Increment in PM	Range of City PM Levels * Means ($\mu g/m^3$)
Increased total mortality in adults		Relative Risk (95% CI)	
Six City[B]	*$PM_{15/10}$ (20 $\mu g/m^3$)*	*1.18 (1.06-1.32)*	*18-47*
	$PM_{2.5}$ (20 $\mu g/m^3$)	*1.28 (1.09-1.51)*	*11-30*
Six City[C]	*$PM_{15-2.5}$ (20 $\mu g/m^3$)*	*1.43 (0.82-2.47)*	*range = 9.7*
ACS Study[D] *(151 U.S. SMSA)*	*$PM_{2.5}$ (20 $\mu g/m^3$)*	*1.14 (1.07-1.21)*	*9-34*
Six City Reanalysis[E]	$PM_{15/10}$ (20 $\mu g/m^3$)	1.19 (1.06-1.34)	18.2-46.5
	$PM_{2.5}$ (20 $\mu g/m^3$)	1.28 (1.09-1.51)	11.0-29.6
ACS Study Reanalysis[E]	$PM_{15/10}$ (20 $\mu g/m^3$) (SSI)	1.02 (0.99-1.04)	34-101
	$PM_{2.5}$ (20 $\mu g/m^3$)	1.14 (1.08-1.21)	9.0-33.4
	$PM_{15-2.5}$ (20 $\mu g/m^3$)	1.01 (0.97-1.05)	9-42
	$PM_{2.5}$ (20 $\mu g/m^3$)	1.14 (1.08-1.21)	9.0-33.4
Southern California[F]	PM_{10} (20 $\mu g/m^3$)	1.01 (0.92, 1.10)**	51 (±17)
	PM_{10} (cutoff= 30 d/yr >100 $\mu g/m^3$)	0.99 (0.93, 1.06)**	
	$PM_{2.5}$ (24.3 $\mu g/m^3$)	1.22 (0.95, 1.58) (males)	31.9 (17.2-45.2)
	$PM_{10-2.5}$ (9.7 $\mu g/m^3$)	1.05 (0.92, 1.20) (males)	27.3 (3.7, 44.3)

* Range of mean PM levels given unless, as indicated, studies reported overall study mean (min, max), or mean (±SD)

** represents pooled estimates for males and females, using inverse weighted variances

[A]Results calculated using PM increment between the high and low levels in cities, or other PM increments given in parentheses

References:
[B]Dockery et al. (1993)
[C]EPA, (1996a)
[D]Pope et al. (1995)
[E]Krewski et al. (2000)
[F]Abbey et al. (1999)

Adapted from CD Tables 6-11 and 9-6.

It has been recognized that pollution levels have declined over time in many areas. When some key risk factors, including pollution level, were allowed to vary over time in the analyses, it was found that the association between fine particles and mortality was reduced, but remained statistically significant. This might be expected, if the most polluted cities had the greatest decline in pollutant levels as controls were applied (CD, p. 6-85).

The original analyses had not included assessment of co-pollutant confounding, though single-pollutant analyses between mortality and the co-pollutant gases were done in the Six Cities analysis. Significant or borderline significant associations were reported with SO_2 and NO_2, but it was observed that these pollutants were strongly correlated with PM (CD, p. 12-168). The Investigators obtained additional data on gaseous pollutant concentrations and evaluated both the effects of these pollutants alone and with PM in multi-pollutant models. Significant associations were reported between mortality and sulfur dioxide, and in multiple pollutant models, the sulfur dioxide associations often appeared stronger than those for fine particles and sulfates. The authors suggest that it is more likely that sulfur dioxide is acting as a marker for other mortality-associated pollutants, and conclude "Nonetheless, both fine particles and sulfate continued to demonstrate a positive association with mortality even after adjustment for the effects of sulfur dioxide in our spatial regression analyses." (Krewski et al., 2000, p. 233, 234)

Several methods were used to address variation from city to city, or spatial correlation among cities, using the larger sulfate data set. The resulting sulfate associations were sometimes smaller and sometimes larger than the original effect estimate. The Investigators concluded: "it suggests that uncontrolled spatial autocorrelation accounts for 24% to 64% of the observed relation. Nonetheless, all our models continued to show an association between elevated risks of mortality and exposure to airborne sulfate." (Krewski et al., 2000, p. 228).

In summary, the draft CD concluded that the reanalysis generally confirmed the original investigators' findings of associations between mortality and long-term exposure to fine particles. As seen in draft CD Table 6-6, the mortality relative risk estimates reported in the replication analysis were nearly identical to those reported in the original studies (CD, p. 6-84). In the sensitivity analyses, Krewski et al. (2000) reported risk estimates that were "remarkably robust to alternative risk models" (p. 25). While recognizing that increased mortality may be attributable to

more than one component of ambient air pollution, the reanalysis confirmed the association between mortality and fine particle and sulfate exposures (CD, p. 6-87).

Analyses of the AHSMOG cohort available for the 1996 CD reported no significant associations between mortality and PM, measured as TSP (Abbey et al., 1991). In the new studies discussed in the draft CD (pp. 6-87 to 6-99), analyses have used more recent air quality data for PM_{10} and have estimated $PM_{2.5}$ concentrations from visibility data. A significant association was reported for total mortality and PM_{10} (number of days exceeding 100 $\mu g/m^3$) for males (CD, p. 6-88), but no significant associations were reported for other PM_{10} indices (e.g., 30 $\mu g/m^3$ increase), for deaths from contributing respiratory causes, and among females. Additional analyses were conducted using only data from males and estimated $PM_{2.5}$ and $PM_{10-2.5}$ concentrations; larger effects estimates were reported for mortality with $PM_{2.5}$ than with $PM_{10-2.5}$, but again, the estimates were generally not statistically significant (CD, Table 6-10). The draft CD concludes that the "lack of consistent findings in this study does not cast doubt on the findings of the Six Cities and ACS studies, which both had larger study populations (especially the ACS study), were based on measured PM data (in contrast with AHSMOG PM estimates based on TSP or visibility measurements) and have been validated through an exhaustive reanalysis." (CD, p. 6-94).

An additional new long-term exposure study has been recently published (Lipfert et al., 2000b). The study examines a prospective cohort of military men assembled by the Veterans Administration in the 1970s. The investigators report inconsistent and largely nonsignificant associations between PM exposure (including, depending on availability, TSP, PM_{10}, $PM_{2.5}$, PM_{15} and $PM_{15-2.5}$) and mortality. The draft CD finds "it is difficult to assess the methodological soundness of this study or to interpret its preliminary results. The findings may reflect one or more unintentional forms of confounding" (CD, p. 6-101). The final model used by the authors included 233 variables, of which 162 were interaction terms of systolic blood pressure, diastolic blood pressure, and body mass index variables with age. The blood pressure variables may be an important intermediate step in the causal pathway between PM and cardiorespiratory health effects, and it is generally inappropriate to treat factors in the causal pathway as confounders (CD, p. 6-100 and 6-101). In summary, the CD concludes that the results of this study do not cast doubt on the results of the Six Cities, ACS and reanalysis studies.

In addition to the analyses of total and cardiorespiratory mortality described above, the three prospective cohort studies examined PM in relation to lung cancer mortality. None of the three studies (Six Cities, ACS, AHSMOG) reported a significant association between long-term exposure to fine particles and lung cancer mortality (EPA, 1996b, p. V-17). The reanalysis study confirmed these findings for the Six Cities and ACS studies (Krewski et al., 2000). One new study on potential lung cancer associations has used data from the AHSMOG cohort. As summarized in the draft CD, significant associations were reported between long-term PM_{10} exposure and lung cancer mortality for males, but not females; some associations were also reported with other gaseous pollutants. The findings were based on a small number of lung cancer deaths in the cohort, and the effect estimates were quite variable, with some described as "high non-credible RR [relative risk]" (CD, p. 6-91). Further analysis using data for males and estimated $PM_{2.5}$ and $PM_{10-2.5}$ reported no statistically significant associations with lung cancer mortality for either $PM_{2.5}$ or $PM_{10-2.5}$ (CD, p. 6-92). Thus, there remains little evidence for lung cancer associations with ambient PM mass.

A few new studies have linked infant mortality with average ambient PM concentrations over periods of one month or more during gestation or around the time of birth. Each of the studies reviewed in the draft CD (Section 6.2.3.4) reported significant associations between infant mortality and PM exposure. One recent U.S. study reported significant associations between PM_{10} concentrations during the first 2 months of the infant's life and mortality from respiratory causes and sudden infant death syndrome (Woodruff et al., 1997). Studies conducted in the Czech Republic and Mexico City also find associations with infant mortality, and the CD concludes that these findings "suggest that infants may be among sub-populations notably affected by long-term PM exposure" (CD, p. 6-106). Less consistent evidence was reported for an association between PM exposure during gestation and low birth weight for infants (CD, p. 6-102).

In summary, positive, statistically significant associations between mortality from total or cardiorespiratory causes and fine particles were reported in the Six Cities and ACS studies and these results were confirmed in an extensive reanalysis. In considering these results, as well as the other evidence related to long-term exposures discussed above, the draft CD concludes that long-

term PM exposure durations are likely associated with serious human health effects. (CD, p. 6-267).

3.3.1.3 Mortality Displacement and Life-Shortening

The 1996 CD and Staff Paper discussed the issue of mortality displacement, or whether some of the acute mortality associations represent deaths among the weakest individuals who might have died within days even without PM exposure (sometimes referred to as "harvesting"). Limited data were available, and it was concluded that there may be evidence of mortality displacement occurring in some portion of the population, but that further research was needed to more fully address this question (EPA, 1996b, p. V-19). In its assessment of the extent of life-shortening that may occur with long-term exposure to PM, the CD concluded that increased mortality results from both short-term and long-term ambient PM exposure, and that the amount of life shortening could potentially be on the order of years (EPA, 1996a, p. 13-45).

More recently, the extent to which mortality displacement may be occurring was investigated using two new types of analyses. One type of study separated time-series data into three components -- seasonal and longer fluctuations, intermediate fluctuations, and short-term fluctuations -- and varied the cutoff between the intermediate and short-term cycles to test for the presence of harvesting (Schwartz, 2000; Schwartz and Zanobetti, 2000). While there was evidence in the Boston analysis that mortality from chronic obstructive pulmonary disease (COPD) may be displaced by a only few months, effect sizes for deaths from pneumonia, heart attacks, and all causes were reported to increase as longer time scales were included, thus offering no evidence for harvesting effects. (Schwartz, 2000). Similar results were reported in the analysis of data from Chicago; this study also reported that effect size increased more steeply with increasing time scale for deaths outside the hospital than for in-hospital deaths (Schwartz and Zanobetti, 2000). Using data from Milan, Italy, positive associations were reported between TSP and mortality up to 13 days, with no effect reported in the next few days, then positive coefficients from 20 days to 45 days (maximum time scale used in study), possibly providing evidence for an initial "rebound" due to depletion of the susceptible population, but with an overall increase in effect size when considering mortality over the longer time scale (Zanobetti et al., 2000). Using first simulation analyses, then analyses using data from Philadelphia, effects of harvesting were assessed at 3 days, 30 days, and 300 days (Zeger et al., 1999), and larger effect

sizes were reported for the longer frequency ranges. The results of these studies "suggest that the extent of harvesting, if any, is not a matter of a few days" (CD, p. 6-245).

The extent of life-shortening that may be associated with long-term PM exposure has been investigated in a recent analysis using effect estimates from existing studies and life-table analysis methods (Brunekreef, 1997). Chronic exposure to PM, with an exposure difference of 10 $\mu g/m^3$, was associated with a reduction in 1.31 years in the population's life expectancy at age 25. Taking into account the evidence from a few new studies showing associations between infant mortality and PM exposure, the draft CD finds that these data suggest that potential life-shortening associated with long-term PM exposure may be even greater than Brunekreef's (1997) estimate. (CD, p. 6-106).

3.3.3 Indices of Morbidity

As noted in 1996 PM Staff Paper, given the statistically significant positive associations between community PM concentrations and mortality, it is reasonable to anticipate that comparable epidemiological studies should find increased morbidity with elevated levels of PM (EPA, 1996b, p. V-21). This was indeed the case in the past review, where positive associations were reported between PM and morbidity effects ranging from the more severe (e.g., hospitalization for respiratory or cardiovascular diseases) to moderate exacerbation of respiratory conditions or decreases in lung function. Staff noted the logical relationships between the cause specific mortality and hospital admissions results, as well as those across the range of morbidity effects and sensitive populations.

A number of more recent epidemiological studies also find increased hospital admissions or emergency room visits, as well as changes in lung function and respiratory symptoms with PM exposure. Other new epidemiology studies have expanded the range of morbidity indices of morbidity associated with PM, including physicians' office or clinic visits for respiratory disease, and cardiovascular health indicators such as heart rate or heart rate variability. In the previous review, several epidemiology studies also reported increased numbers of school absences, lost work days or restricted activity days with increased PM (EPA, 1996b, p. V-22); little new evidence is provided for these morbidity indices in the draft CD.

The recent literature also shows productive interactions among toxicological, controlled human, and epidemiological studies of morbidity effects. Effects related to some new endpoints measured in the recent epidemiological studies, such as heart rate variability, were first reported in animal toxicology studies. Some toxicology studies have used ambient PM samples from areas in which epidemiological studies were conducted (e.g. Ghio, 1999a,b). In addition, many laboratory studies have measured cellular or physiological changes, such as changes in numbers of immune cell types, levels of cytokines, or measures of pulmonary or cardiovascular function following exposure to CAPs or instilled ambient particles. The more subtle biological responses measured in such studies may provide supporting evidence for morbidity associations reported without being considered separate indices of morbidity.

3.3.3.1 Hospital Admissions or Emergency Room Visits

Hospitalization and emergency room visits are measures of more severe respiratory or cardiovascular morbidity, and associations with these health outcomes have been evaluated in numerous studies. The 1996 Staff Paper observed that epidemiological studies demonstrated associations between hospital admissions and emergency room visits for respiratory and cardiac causes and PM_{10} exposure (EPA, 1996b, p. V-21). Most studies evaluated relationships with admissions/visits for respiratory diseases, including asthma, COPD and pneumonia, and nearly all associations were statistically significant. Where multi-pollutant models were evaluated, associations reported with PM_{10} were not substantially changed with the inclusion of gaseous co-pollutants in the models. Several studies had also reported associations between PM and hospital admissions for cardiovascular diseases. The 1996 CD included results from only one study where $PM_{2.5}$ and $PM_{10-2.5}$ data were available, and associations with total respiratory admissions/visits were reported for both, with the associations with fine particles or fine particle components were larger and less influenced by co-pollutant confounding (Thurston et al., 1994). As noted in the 1996 Staff Paper, the associations reported with hospital admissions and emergency room visits were coherent with the findings of significant associations with mortality, especially mortality from cardiovascular and respiratory causes.

Numerous recent studies have continued to report significant associations between PM and hospital admissions or emergency room visits for respiratory or cardiovascular diseases. The new studies have included multi-city analyses, numerous assessments using cardiovascular

admissions/visits, and evaluation of the effects of fine- and coarse-fraction particles. The findings from U.S. and Canadian studies on associations with PM_{10}, $PM_{2.5}$ or $PM_{10-2.5}$ are presented in Figures 3-7, 3-8 and 3-9, respectively. In these figures, effects estimates are presented by general respiratory or cardiovascular effects categories, separated into more specific subcategories in cases where results from several studies are available (e.g., COPD, asthma). Within each group, the results are presented in order of decreasing study size or power, using the natural log of the product of study days times number of admissions/visits per day. The results for all new cardiovascular and respiratory admissions/visits studies, including those using nongravimetric PM measurements and studies from non-North American locations, are summarized in the draft CD in Tables 6-16 and 6-17, respectively, and the effect estimates for PM_{10}, $PM_{2.5}$ or $PM_{10-2.5}$ from U.S. and Canadian studies are summarized in Appendix A, Tables 2 and 3, respectively.

Effect estimates for PM_{10} presented in Figure 3-7 include findings from multi-city studies, as well as results from studies available for review in the 1996 CD, with the range of statistically significant effect estimates from the 1996 CD indicated at the right-hand margin; for $PM_{2.5}$ or $PM_{10-2.5}$, the effects estimates from the only study on respiratory admissions/visits available in the 1996 CD are indicated in the right-hand margins in Figures 3-8 and 3-9. In general, positive, mostly statistically significant associations for both respiratory and cardiovascular admissions/visits are seen with PM_{10} and $PM_{2.5}$, as well as with $PM_{10-2.5}$.

As discussed previously, the results of multi-city studies are of particular relevance in the review of PM standards. The recent U.S. multi-city study, NMMAPS, reported statistically significant associations between PM_{10} and hospital admissions in the elderly for cardiovascular diseases, pneumonia or COPD in 14 cities (Samet et al., 2000b), with somewhat larger effect

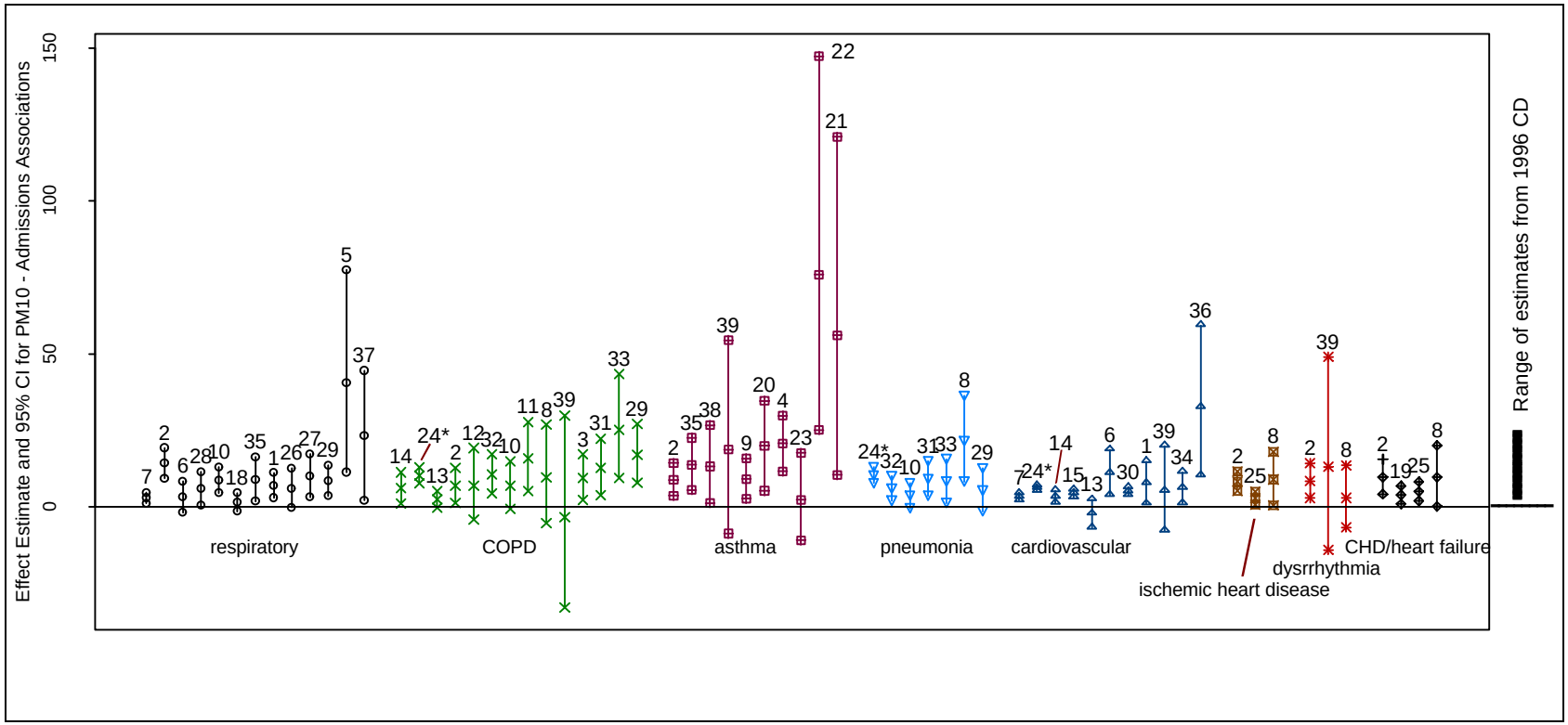


Figure 3-7. Effects estimates for PM_{10} and hospital admissions, emergency room visits (denoted ◊) or physicians office visits (denoted ⊙) for various respiratory and cardiovascular diseases from U.S. and Canadian studies. Within each category, associations are ranked by decreasing natural log of the morbidity-days product (product of study days and number of admissions/visits per day). Multi-city studies denoted with an asterisk above; study locations are identified below (data in Appendix 3-A, Table 4D)

1. Burnett et al., 1997, Toronto
2. Burnett et al., 1999, Toronto
3. Chen et al., 2000, Reno
4. Choudbury et al., 1997, Anchorage ⊙
5. Delfino et al., 1997, Montreal ◊
6. Gwynn et al., 2000, Buffalo
7. Linn et al., 2000, LA
8. Lippmann et al., 2000, Detroit
9. Lipsett et al., 1997, Santa Clara ◊
10. Moolgavkar et al., 1997, Minn/St. Paul
11. Moolgavkar et al., 2000, King Co.
12. Moolgavkar, 2000c, Maricopa Co.
13. Moolgavkar, 2000b, Maricopa Co.
14. Moolgavkar, 2000c, Cook Co,
15. Moolgavkar, 2000b, LA
16. Moolgavkar, 2000c, LA.
17. Moolgavkar, 2000b, Cook Co.
18. Moolgavkar, et al., 1997, Birmingham
19. Morris and Naumova, 1998, Chicago
20. Nauenberg and Basu, 1999, LA
21. Norris et al., 2000, Seattle ◊
22. Norris et al., 1999, Seattle ◊
23. Norris et al., 2000, Spokane ◊
24. Samet et al., 2000b, 14 U.S. cities
25. Schwartz and Morris, 1995, Detroit
26. Schwartz, 1995, New Haven
27. Schwartz., 1995, Tacoma
28. Schwartz et al., 1996, Cleveland
29. Schwartz et al., 1996, Spokane
30. Schwartz., 1999, 8 US Counties
31. Schwartz, 1994b, Birmingham
32. Schwartz, 1994a, Detroit
33. Schwartz, 1994c, Minn/St. Paul
34. Schwartz, 1997, Tucson
35. Sheppard et al., 1999, Seattle
36. Stieb et al., 2000, St. John ◊
37. Thurston et al., 1994 Toronto
38. Tolbert et al., 2000b, Atlanta ◊
39. Tolbert et al., 2000a, Atlanta ◊

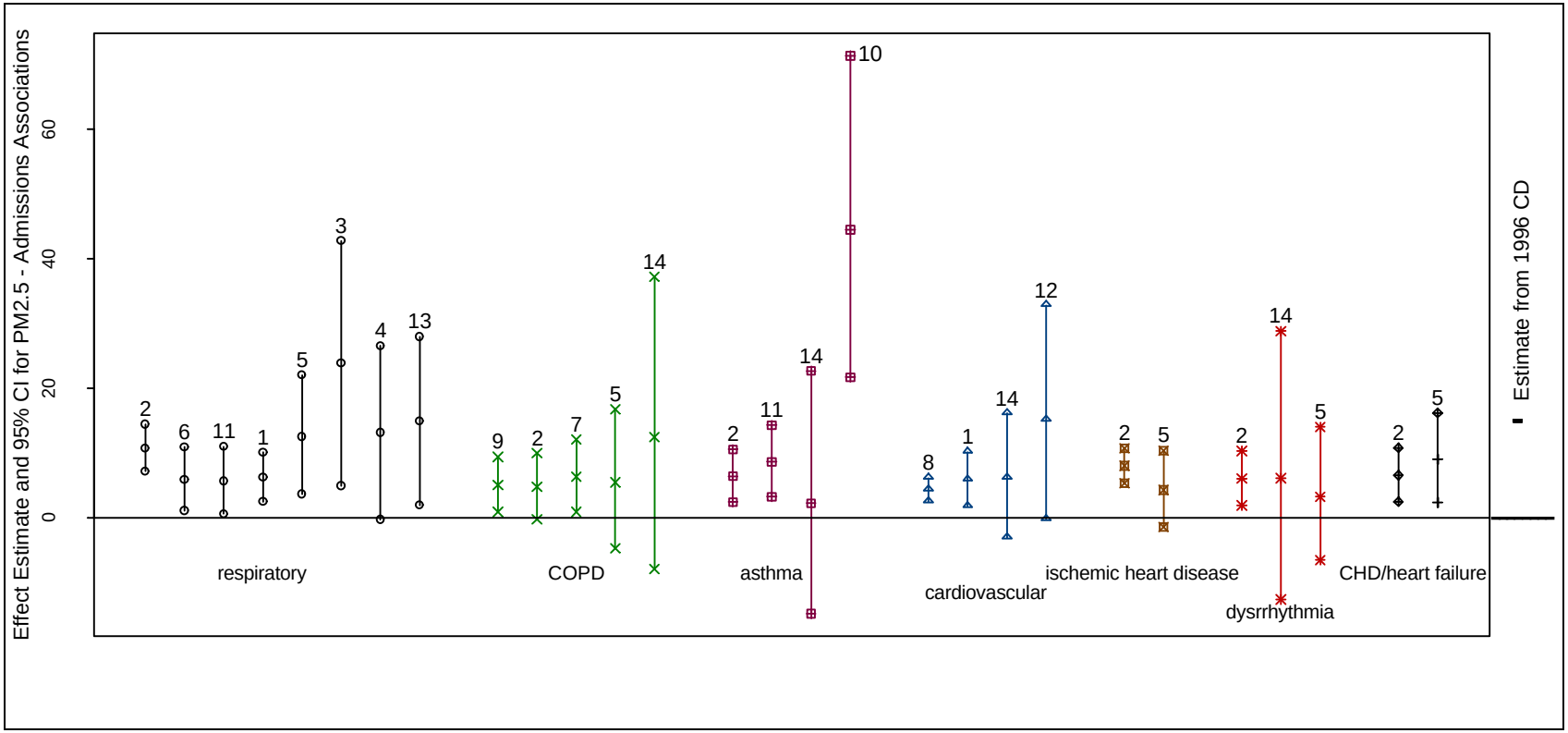


Figure 3-8. Effects estimates for $PM_{2.5}$ and hospital admissions or emergency room visits (denoted ◊) for various respiratory and cardiovascular diseases from U.S. and Canadian studies. Within each category, associations are ranked by decreasing natural log of the morbidity-days product (product of study days and number of admissions/visits per day). Study locations are identified below (data in Appendix 3-A, Table 4E)

1. Burnett et al., 1997, Toronto
2. Burnett et al., 1999, Toronto
3. Delfino et al., 1997, Montreal ◊
4. Delfino et al., 1998, Montreal ◊
5. Lippmann et al., 2000, Detroit
6. Lumley and Heagerty, 1999, King Co
7. Moolgavkar et al., 2000, King Co.
8. Moolgavkar, 2000b, LA
9. Moolgavkar, 2000c, LA
10. Norris et al., 1999, Seattle ◊
11. Sheppard et al., 1999, Seattle
12. Stieb et al., 2000, St. John ◊
13. Thurston et al., 1994, Toronto
14. Tolbert et al., 2000a, Atlanta ◊

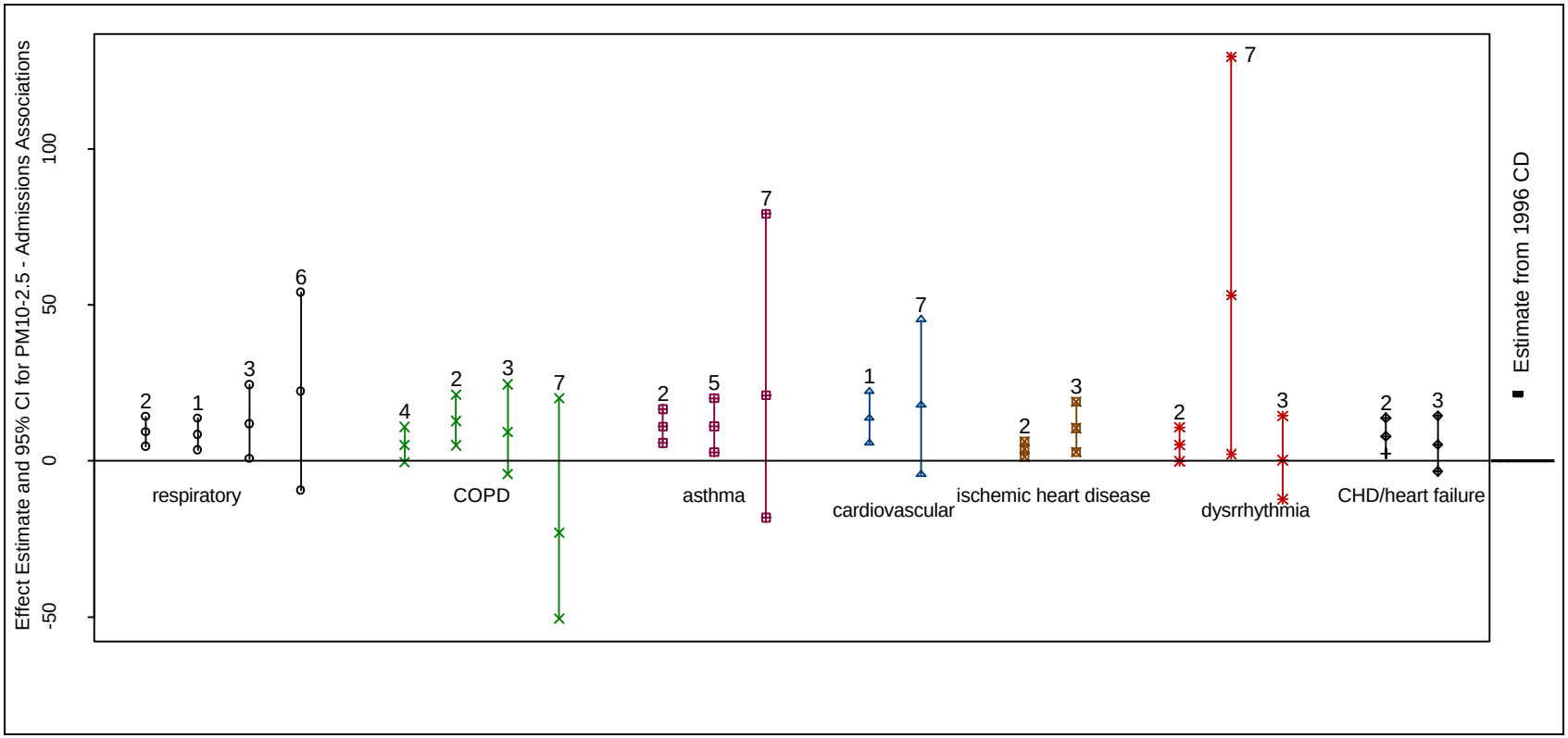


Figure 3-9. Effects estimates for $PM_{10-2.5}$ and hospital admissions or emergency room visits (denoted ◊)for various respiratory and cardiovascular diseases from U.S. and Canadian studies. Within each category, associations are ranked by decreasing natural log of the morbidity-days product (product of study days and number of admissions/visits per day). Study locations are identified below (data in Appendix 3-A, Table 4F)

1. Burnett et al., 1997, Toronto
2. Burnett et al., 1999, Toronto
3. Lippmann et al., 2000, Detroit
4. Moolgavkar, 2000b, LA
5. Sheppard et al., 1999, Seattle
6. Thurston et al., 1994, Toronto
7. Tolbert et al., 2000a, Atlanta ◊

estimates when a distributed lag approach was used (Zanobetti et al., 2000). Increases of 6% in hospital admissions for cardiovascular disease and 10% in hospital admissions for COPD or pneumonia per 50 $\mu g/m^3$ increase in PM_{10} were reported. In addition, the authors used a new approach for evaluating potential confounding by testing for associations between the PM effect estimate and the PM-gaseous pollutant relationship in each location (as was done in multi-city mortality analyses described in Section 3.3.1.1.1). No evidence was found for trends between the coefficients between PM_{10} and O_3 or SO_2 and PM_{10}-respiratory admissions associations, or between the coefficients between PM_{10} and CO, O_3 or SO_2 and PM_{10}-cardiovascular admissions associations, indicating that confounding by co-pollutants is unlikely (Samet et al., 2000b).

A multi-city study analysis for 8 U.S. counties also reported statistically significant associations between PM_{10} and hospital admissions for cardiovascular diseases among the elderly. An increase of 5% in admissions was associated with a 50 $\mu g/m^3$ increase in PM_{10}, with no evidence of confounding with ambient CO (Schwartz, 1999).

In the European multi-city study, APHEA, associations between PM and admissions/visits for all respiratory diseases, asthma or COPD were largely positive, though not always statistically significant. While the APHEA analyses used PM measurements from a variety of methods (e.g., suspended particles, black smoke), which makes quantitative comparisons with North American studies difficult, the draft CD observes that the APHEA results are qualitatively consistent with results of other studies (CD, p. 6-177).

Considering all U.S. and Canadian studies, PM_{10} and $PM_{2.5}$ are associated with admissions/visits for respiratory diseases and specific disease categories, including asthma, COPD, pneumonia, and the findings are generally consistent with those reported in the 1996 CD. In Figure 3-7, it can be seen that most associations between PM_{10} and admissions/visits for respiratory causes are positive and statistically significant. A number of new studies have also reported significant associations between $PM_{2.5}$ and admissions/visits for respiratory diseases (Figure 3-8). The CD concludes that the numerous recent studies provide evidence for associations with PM_{10} and $PM_{2.5}$ at levels lower than had been demonstrated previously for this health outcome (CD, p. 6-179).

Though fewer studies are available, several recent studies show significant associations between admissions/visits for respiratory diseases and $PM_{10-2.5}$ (Figure 3-9). In addition, the draft

CD observes that, as was found in the previous review, significant associations are reported between PM_{10} and hospital admissions or emergency room visits for respiratory diseases in studies that were conducted in areas of the western U.S. where coarse-fraction particles are predominant (CD, p. 6-236), indicating a likely role for coarse-fraction particles in the reported effects. Thus, both fine- and coarse-fraction particles appear to be linked to increases in hospital admissions and emergency room visits for respiratory diseases, though more evidence is available for fine-fraction particles. In addition, where investigators have used two-pollutant models to test the independence of the effects of each size fraction, $PM_{2.5}$ and $PM_{10-2.5}$ were not highly correlated and had independent effects (Lippmann et al., 2000; Moolgavkar, 2000c).

Figures 3-7 through 3-9 present effects estimates from single-pollutant models. As discussed above, the multi-city analyses of hospital admissions have not found evidence of significant confounding by co-pollutant gases. In single-city studies, a number of investigators evaluated the effects of gaseous co-pollutants independently and in multi-pollutant models with PM. As discussed in further detail in Section 3.5.1, some gaseous pollutants have been reported to have independent effects on the respiratory system and might be expected to act as confounders in PM-admissions/visits associations. For example, a number of studies have indicated that O_3 is associated with increased admission/visits for respiratory diseases, such as asthma, and a number of the studies in Table 6-17 of the draft CD report significant associations with O_3. In some of these studies, PM effect estimates were reduced in two-pollutant models with O_3 (e.g., Tolbert et al., 2000b; Delfino et al., 1998), but in others, PM associations were generally reported to be robust to inclusion of O_3 in the models (e.g., Lippmann et al., 2000; Gwynn et al., 2000; Burnett et al., 1997) and less evidence was found for potential confounding by other gaseous pollutants (results summarized in Table 6-17 of the draft CD). In considering studies of cardiovascular admissions/visits, the draft CD focused on CO as a co-pollutant of interest, due to the known effects of CO on the cardiovascular system (EPA, 1999). The draft CD finds that "[t]he above analyses of daily PM_{10} and CO in U.S. cities, overall, suggest that elevated concentrations of both PM_{10} and CO may enhance risk of cardiovascular (CVD)-related morbidity leading to acute hospitalizations" (CD, p. 6-128). In studies of cardiovascular and chronic respiratory disease admissions/visits, Moolgavkar (2000b,c) reports that associations with PM were dramatically reduced with the inclusion of either CO or NO_2 (differs by location and health endpoint) in the

models. For cardiovascular admissions/visits (but equally true for respiratory diseases) the CD concludes: "In some studies, PM clearly carries an independent association after controlling for gaseous co-pollutants. In others, the 'PM effects' are markedly reduced once co-pollutants are added to the model; but this may in part be due to both PM and co-pollutants such as CO and NO_2 being emitted from a common source (motor vehicles) and consequent colinearity between them and/or the gaseous pollutants such as CO having independent effects on cardiovascular function" (CD, p. 6-141).

The CD concludes that the U.S. multi-city studies (Samet et al., 2000a,b; Schwartz, 1999) likely provide the most precise estimates for relationships of U.S. ambient PM_{10} exposure to increased risk for hospitalization (CD, pp. 6-127, 6-172). Taken together, the findings of new studies and those reviewed in the 1996 CD offer consistent evidence for associations between ambient PM concentrations and admissions/visits to the hospital or emergency room for respiratory or cardiovascular diseases.

3.3.3.2 Effects on the Respiratory System

Evidence available in the previous review suggested associations between PM exposure and respiratory effects such as changes in lung function, increases in respiratory symptoms or disease, as well as related morbidity indices such as school absences, lost work days and restricted activity days (EPA, 1996b, pp. V-21 and V-22). From epidemiology or controlled human exposure studies of short-term PM exposure, it was reported that sensitive individuals (especially those with asthma or pre-existing respiratory symptoms) may have increased or aggravated symptoms, with or without reduced lung function (EPA, 1996b, p. V-23). Long-term (months to years) exposure to PM was linked with decreased lung function and increased incidence of respiratory diseases such as bronchitis (EPA, 1996b, p. V-26). The results of studies using long-term and short-term PM exposure data were reported to be consistent with one another. In addition, toxicology studies using surrogate particles or PM components, generally at high concentrations, and autopsy studies of humans and animals reported evidence of pulmonary effects, including morphological damage (e.g., changes in cellular structure of the airways), and changes in resistance to infection.

Recently published studies summarized in the draft CD have included toxicological or controlled human exposure studies of exposures to ambient PM, using inhalation exposures to

CAPs or intratracheal instillation of ambient PM samples. These studies provide additional new evidence linking PM with respiratory effects. Among the many new epidemiology studies are several assessing relationships between PM and additional health endpoints, including physicians' office visits. A number have evaluated effects on lung function or respiratory symptoms, while few new studies have assessed effects such as school absences or work loss days, which are indirect measures that may be linked with respiratory illness.

Acute Respiratory Effects - Epidemiological Studies. Among the new epidemiology studies are several using medical visits for respiratory illness as a measure of health effects. These studies have evaluated effects of pollutant exposure on visits to physician's offices (Anchorage, Alaska, Choudhury et al., 1997; London, UK, Hajat et al., 1999; Santiago, Chile, Ostro et al., 1999), or doctor's visits to patients (Paris, France, Medina et al., 1997). Visits for asthma were significantly increased with PM exposure in children (Medina et al., 1997) and people of all ages (Choudhury et al., 1997), and significant associations were found with visits for lower respiratory diseases in children (Ostro et al., 1999) and adults (Hajat et al., 1999).

The draft CD notes that these studies "provide new insight into the fact that there is a broader scope of severe morbidity associated with PM air pollution exposure than previously documented" (CD, p. 6-180). These studies find associations in a range of 3% to 42% increases in medical visits with a 50 $\mu g/m^3$ change in PM_{10} (CD Table 6-17). The results of these studies offer further support for coherence in effects on the respiratory tract, since they are consistent with findings of increased mortality and hospital admissions or emergency room visits for respiratory diseases. These new studies also indicate the potentially more widespread public health impact of the less severe respiratory health endpoints (CD, p. 6-181).

New epidemiology studies on PM-related effects on respiratory symptoms or lung function are summarized in draft CD Tables 6-19 through 6-23; the studies are grouped by health status of the study subjects (asthmatic or nonasthmatic) and PM exposure (short- and long-term). Only a few recent North American publications are available; the results for U.S. and Canadian studies using gravimetric PM data are included in Appendix A, Table 2. Most U.S. and Canadian studies used gravimetric PM data, generally PM_{10} and sometimes $PM_{2.5}$ and $PM_{10-2.5}$, and most were studies using children.

All studies of effects in children reported significant associations with a range of respiratory symptoms (e.g., cough, wheeze, shortness of breath) (Neas et al., 1995, 1996; Ostro et al., 1995; Pope et al., 1991; Schwartz et al., 1994; Vedal et al., 1998). Some (Neas et al., 1999; Schwartz and Neas, 2000; Vedal et al., 1998), but not all (Neas et al., 1995, 1996; Thurston et al., 1997), of the North American studies also reported significant associations between PM_{10}, $PM_{2.5}$ or $PM_{10-2.5}$ and decreases in lung function measures (e.g., decreased peak expiratory flow rate).

From the limited number of studies using adults, Naeher et al. (1999) found significant associations between $PM_{10,}$ $PM_{2.5}$ and $PM_{10-2.5}$ and decreased lung function in adult women, but no significant associations were found with respiratory symptoms by Ostro et al. (1991) or Pope et al. (1991).

In those studies where $PM_{2.5}$ and $PM_{10-2.5}$ data were available, the findings suggest roles for both fine- and coarse-fraction PM in reduced lung function and increased respiratory symptoms (CD, p. 6-237). For example, using data from the Six Cities study, lower respiratory symptoms were found to be significantly increased for children with $PM_{2.5}$ but not with $PM_{10-2.5}$, while the reverse was true for cough (Schwartz and Neas, 2000). When both $PM_{2.5}$ and $PM_{10-2.5}$ were included in models, the effect estimates were reduced for each, but $PM_{2.5}$ retained significance in the association with lower respiratory symptoms and $PM_{10-2.5}$ retained significance in the association with cough. In the last review, several studies reported significant associations between symptoms or lung function changes with PM_{10} and fine particles or fine particle surrogates, but no data were available for coarse-fraction particles (EPA 1996b, Table V-12). The new studies continue to show effects of short-term exposure to PM_{10} and $PM_{2.5}$ and offer additional evidence for associations between $PM_{10-2.5}$ and respiratory morbidity.

Considering also results from studies conducted outside the U.S. and Canada, the draft CD finds evidence supporting increases in respiratory symptoms associated with short-term exposures to PM for both asthmatic and nonasthmatic subjects, though many associations did not reach statistical significance. Again, considering the full body of literature, short-term PM exposure was associated with decreases in lung function (e.g., peak expiratory flow rate) in studies of asthmatics (CD, p. 6-184) but little evidence was reported for associations between lung function and short-term PM exposure in nonasthmatic subjects (CD, p. 6-194).

Acute Respiratory Effects - Laboratory Studies. Key toxicology or controlled human exposure studies summarized in the draft CD include: (1) exposures of human volunteers in a clinical setting to concentrated ambient PM; (2) animal studies with exposure to ambient PM by inhalation of CAPs or intratracheal installation of ambient PM samples; and (3) *in vitro* exposures to ambient particles using cells from the respiratory system (e.g., bronchial epithelial cells, macrophages). The principal effects studied have been inflammatory response and other indicators of lung injury.

Inflammatory responses in the respiratory system were reported in humans and animals exposed to concentrated ambient fine particles. Although less evidence is available from studies using ambient particle exposures, Costa and Dreher (1997) summarized evidence from studies showing increased inflammatory cell counts with exposure to ambient particles collected in U.S., Canadian, and German cities, and Brain et al. (1998) showed that similar levels of acute inflammatory injury were caused by urban air particles and Kuwaiti oil fire particles (on an equal mass basis). One new controlled human exposure study also reported evidence of inflammatory changes in the lung with exposure to CAPs (Ghio et al., 2000).

The types of effects reported included increases in neutrophils (either total number or percent) in the lungs in humans (Ghio et al., 2000) and in animals (Clarke et al., 1999; Godleski et al., 2000; Gordon et al., 1998; Kodavanti et al., 2000); though changes in immune cell numbers haven't been observed in all studies (Gordon et al., 2000). Increased neutrophil levels have been reported with ROFA exposures in animals or cell cultures (e.g., Costa and Dreher, 1997; Killingsworth et al., 1997). Increases also have been reported in other immune cell types such as eosinophils or alveolar macrophages (CD, Table 8-4). Increases in immune cells, again commonly neutrophils, also were reported with intratracheal exposure to urban particles in animals (Brain et al., 1998; Li et al., 1996, 1997; Ghio et al., 1999, Kennedy et al., 1998).

Other inflammatory changes reported have included changes in levels or increased release of cytokines, or chemicals released as part of the inflammatory process (e.g., interleukins such as IL-8). The draft CD concludes that exposure of lung cells to ambient PM, ROFA or PM surrogates leads to increased production of cytokines and that the effects may be mediated, at least in part, through production of reactive oxygen species (CD, p. 8-57).

A number of animal studies have shown that exposure to diesel exhaust particles could increase the production or release of inflammatory cells, such as eosinophils (CD, p. 8-44). Controlled exposures of humans to diesel exhaust particles also have resulted in increases in inflammatory cells indicative of enhanced response to allergens (CD, p. 8-45). Together, the human and animal studies provide evidence that particle exposures can produce inflammatory changes in the respiratory system.

Animal studies also have reported evidence of general lung injury, including increased protein levels in lung fluids with exposure to ambient particles (CD Table 8-3) or combustion-related particles such as ROFA (CD, Table 8-4). One general cause of lung cell injury is the production of reactive oxidant species that can damage the epithelial cells in the lung; these chemicals can be produced as part of an inflammatory response to particle exposure. In *in vitro* experiments, ambient PM exposures were reported to have effects that included increased release of inflammatory chemicals, evidence of oxidant stress on the cells, and evidence of general cellular toxicity (e.g., release of proteins) (CD Table 8-8). Several *in vitro* studies have reported evidence of increased oxidative stress in lung cell cultures exposed to particles collected in Utah Valley; notably, the particle doses used in these studies were only 2-3-fold greater than generally estimated doses for humans breathing ambient air (Ghio et al., 1999a,b; Soukup et al., 2000). In two of these studies, the transition metal content of the particles appeared to be more closely linked to reported effects than the quantity of particles (Ghio et al., 1999a,b). Soukup and colleagues (2000) also tested the effects of particles collected in Utah Valley, and found evidence of oxidant activity with particles collected at times when a major industrial PM source was in operation, but not when the industrial source was shut down. In this latter study, however, the effects did not appear to be closely correlated with metal content of the particles.

Findings of inflammation and lung injury are generally consistent with epidemiological results showing increases in respiratory symptoms or exacerbation of respiratory diseases. Some epidemiological studies also have reported increased admissions/visits for respiratory infections or pneumonia, and there is some toxicological evidence indicating increased susceptibility to respiratory infections. The 1996 CD observed that impairment of pulmonary host defense mechanisms by acidic particles was consistent with observations of increased prevalence of bronchitis in communities with higher levels of acidic PM (EPA 1996a, p. 13-75). Similarly, the

draft CD finds evidence of altered lung responses to microbial agents, though at high PM concentrations (CD, p. 8-47).

The epidemiology findings are consistent with those of the previous review in showing associations with both respiratory symptom incidence and decreased lung function. As reported previously, the evidence is somewhat stronger for changes in symptoms than lung function. The findings from studies of physicians' office visits for respiratory diseases offer new evidence of acute respiratory effects with exposure to ambient PM that is coherent with evidence of increased respiratory symptoms and admissions/visits to the hospital or emergency room for respiratory disease. While urging caution in interpreting the findings of the high-dose toxicology studies, the draft CD concludes that the findings "have shown clearly that PM obtained from various sources can cause lung inflammation and injury" and that "[t]he fact that instillation of ambient PM collected from different geographical areas and from a variety of emission sources consistently caused pulmonary inflammation and injury tends to corroborate epidemiological studies that report increased respiratory morbidity and mortality associated with PM in many different geographical areas and climates." (CD, pp. 8-19 and 8-20).

Chronic Effects. In the 1996 CD, only a few epidemiology studies had assessed associations between long-term PM exposure and lung function changes or respiratory symptoms. Among U.S. and Canadian studies, the Six Cities and 24-Cities studies had provided data suggesting associations with chronic bronchitis and decreased FEV_1 or FVC in children (CD, p. 6-205). In the 1996 Staff Paper, significant associations were observed between decreased lung function or increased incidence of bronchitis in children with fine particles or fine particle surrogates, with less evidence for associations with PM_{10}, PM_{15} or TSP (EPA, 1996b, Table V-13).

Several new epidemiological analyses have been conducted on long-term pollutant exposure effects on respiratory symptoms or lung function in the U.S.; numerous European, Asian, and Australian studies have also been published. Little new evidence is available from toxicology or controlled human exposure studies regarding long-term effects of PM exposure. The new U.S. epidemiological studies are based on data from two main cohort studies, a study of schoolchildren in 12 Southern California Communities and an adult cohort of Seventh Day Adventists (AHSMOG).

As seen in Table 3-4, initial publications from the 12 Southern California Communities childrens cohort show significant associations between long-term exposure to PM and incidence of bronchitis or phlegm among the subgroup of children with asthma, though no significant associations were found for the subgroups of children without asthma (McConnell et al., 1999). In this study, some significant associations were also found for NO_2 and acid vapor (hydrochloric and nitric acids) with incidence of bronchitis and phlegm and the authors found it difficult to distinguish effects of these pollutants; no significant associations were found with ozone.

In another analysis using the same cohort, children who entered the cohort while in the 4^{th} grade showed, in tests conducted when these children were in the 7^{th} grade, decreases in lung function growth with increasing exposure to PM, including PM_{10}, $PM_{2.5}$, $PM_{10-2.5}$, and acid vapor (hydrochloric and nitric acids) (Gauderman et al., 2000). Again, there was evidence for associations with NO_2 and acid vapor but not with ozone. Two-pollutant models were tested in this study, and the effect estimates for the various PM indices, NO_2 and acid vapor were generally reduced in size. The authors observe that motor vehicle emissions are a major source of ambient particles, NO_2 and inorganic acids and thus they were unable to identify the independent effects of each pollutant (Gauderman et al., 2000, p. 1388).

In this study, significant associations were reported between ambient concentrations of both fine and coarse fraction particles and reductions in mid-maximal expiratory flow (a measure of small airways function); the effect size for $PM_{10-2.5}$ was slightly, but not significantly, larger than that for $PM_{2.5}$. Growth in another lung function measure, forced vital capacity, was significantly reduced with exposure to PM_{10} and acid vapor (hydrochloric and nitric acids), while associations (though not statistically significant) were indicated for both $PM_{2.5}$ and $PM_{10-2.5}$ (Table 3-4; Gauderman et al., 2000). While limited to two childrens' study populations, these findings are consistent with those from short-term exposure studies where respiratory morbidity is associated with both $PM_{2.5}$ and $PM_{10-2.5}$.

For adults, the 1996 CD summarized the results of a several cross-sectional studies as well as one cohort study (AHSMOG), and found evidence for increased incidence of respiratory diseases, especially bronchitis, with long-term PM exposure (EPA, 1996a, p. 12-197). Further analyses have been done in the AHSMOG cohort, and significant decreases in lung function (FEV_1) were reported only for the subgroup of males with a family history of lung disease (Abbey

TABLE 3-4. EFFECT ESTIMATES PER INCREMENTS[A] IN LONG-TERM MEAN LEVELS OF FINE AND INHALABLE PARTICLE INDICATORS FROM U.S. AND CANADIAN STUDIES

Type of Health Effect & Location	Indicator	Change in Health Indicator per Increment in PM[a]	Range of City PM Levels * Means ($\mu g/m^3$)
Increased bronchitis in children		Odds Ratio (95% CI)	
Six City[B]	*$PM_{15/10}$ (50 $\mu g/m^3$)*	*3.26 (1.13, 10.28)*	*20-59*
Six City[C]	*TSP (100 $\mu g/m^3$)*	*2.80 (1.17, 7.03)*	*39-114*
24 City[D]	*H^+ (100 nmol/m^3)*	*2.65 (1.22, 5.74)*	*6.2-41.0*
24 City[D]	*$SO_4^=$ (15 $\mu g/m^3$)*	*3.02 (1.28, 7.03)*	*18.1-67.3*
24 City[D]	*$PM_{2.1}$ (25 $\mu g/m^3$)*	*1.97 (0.85, 4.51)*	*9.1-17.3*
24 City[D]	*PM_{10} (50 $\mu g/m^3$)*	*3.29 (0.81, 13.62)*	*22.0-28.6*
Southern California[E]	*$SO_4^=$ (15 $\mu g/m^3$)*	*1.39 (0.99, 1.92)*	---
12 Southern California communities[F] (all children)	PM_{10} (25 $\mu g/m^3$) acid vapor (1.7 ppb)	0.94 (0.74, 1.19) 1.16 (0.79, 1.68)	28.0-84.9 0.9-3.2 ppb
12 Southern California communities[F] (children with asthma)	PM_{10} (19 $\mu g/m^3$) PM_{25} (15 $\mu g/m^3$) acid vapor (1.8 ppb)	1.4 (1.1, 1.8) 1.4 (0.9, 2.3) 1.1 (0.7, 1.6)	13.0-70.7 6.7-31.5 1.0-5.0 ppb
Increased cough in children		Odds Ratio (95% CI)	
12 Southern California communities[F] (all children)	PM_{10} (25 $\mu g/m^3$) acid vapor (1.7 ppb)	1.06 (0.93, 1.21) 1.13 (0.92, 1.38)	28.0-84.9 0.9-3.2 ppb
12 Southern California communities[G] (children with asthma)	PM_{10} (19 $\mu g/m^3$) PM_{25} (15 $\mu g/m^3$) acid vapor (1.8 ppb)	1.1 (0.0.8, 1.7) 1.3 (0.7, 2.4) 1.4 (0.9, 2.1)	13.0-70.7 6.7-31.5 1.0-5.0 ppb
Increased obstruction in adults			
Southern California[H]	PM_{10} (cutoff of 42 d/yr >100 $\mu g/m^3$)	1.09 (0.92, 1.30)	NR
Decreased lung function in children			
Six City[B]	*$PM_{15/10}$ (50 $\mu g/m^3$)*	*NS Changes*	*20-59*
Six City[C]	*TSP (100 $\mu g/m^3$)*	*NS Changes*	*39-114*
24 City[I]	*H^+ (52 nmoles/m^3)*	*–3.45% (-4.87, -2.01) FVC*	*6.2-41.0*
24 City[I]	*$PM_{2.1}$ (15 $\mu g/m^3$)*	*–3.21% (-4.98, -1.41) FVC*	*18.1-67.3*
24 City[I]	*$SO_4^=$ (7 $\mu g/m^3$)*	*–3.06% (-4.50, -1.60) FVC*	*9.1-17.3*
24 City[I]	*PM_{10} (17 $\mu g/m^3$)*	*–2.42% (-4.30, -.0.51) FVC*	*22.0-28.6*
12 Southern California communities[J] (all children)	PM_{10} (25 $\mu g/m^3$) acid vapor (1.7 ppb)	-24.9 (-47.2, -2.6) FVC -24.9 (-65.08, 15.28) FVC	28.0-84.9 0.9-3.2 ppb

12 Southern California communities[J] (all children)	PM_{10} (25 µg/m^3) acid vapor (1.7 ppb)	-32.0 (-58.9, -5.1) MMEF -7.9 (-60.43, 44.63) MMEF	28.0-84.9 0.9-3.2 ppb
12 Southern California communities[K] (4th grade cohort)	PM_{10} (51.5 µg/m^3) $PM_{2.5}$ (25.9 µg/m^3) $PM_{10-2.5}$ (25.6 µg/m^3) acid vapor (4.3 ppb)	-0.58 (-1.14, -0.02) FVC growth -0.47 (-0.94, 0.01) FVC growth -0.57 (-1.20, 0.06) FVC growth -0.57 (-1.06, -0.07) FVC growth	NR
12 Southern California communities[K] (4th grade cohort)	PM_{10} (51.5 µg/m^3) $PM_{2.5}$ (25.9 µg/m^3) $PM_{10-2.5}$ (25.6 µg/m^3) acid vapor (4.3 ppb)	-1.32 (-2.43, -0.20) MMEF growth -1.03 (-1.95, -0.09) MMEF growth -1.37 (-2.57, -0.15) MMEF growth -1.03 (-2.09, 0.05) MMEF growth	NR
Decreased lung function in adults			
AHSMOG, So. Calif.[L] (% predicted FEV_1, females)	PM_{10} (cutoff of 54.2 d/yr >100 µg/m^3)	+0.9 % (-0.8, 2.5) FEV_1	52.7 (21.3, 80.6)
AHSMOG, So. Calif.[L] (% predicted FEV_1, males)	PM_{10} (cutoff of 54.2 d/yr >100 µg/m^3)	+0.3 % (-2.2, 2.8) FEV_1	54.1 (20.0, 80.6)
AHSMOG, So. Calif.[L] (% predicted FEV_1, males whose parents had asthma, bronchitis, emphysema)	PM_{10} (cutoff of 54.2 d/yr >100 µg/m^3)	-7.2 % (-11.5, -2.7) FEV_1	54.1 (20.0, 80.6)
AHSMOG, So. Calif.[L] (% predicted FEV_1, females)	$SO_4^=$ (1.6 µg/m^3)	NS; Not reported	7.4 (2.7, 10.1)
AHSMOG, So. Calif.[L] (% predicted FEV_1, males)	$SO_4^=$ (1.6 µg/m^3)	-1.5 % (-2.9, -0.1) FEV_1	7.3 (2.0, 10.1)

* Range of mean PM levels given unless, as indicated, studies reported overall study mean (min, max), or mean (±SD); NR=not reported.
[A]Results calculated using PM increment between the high and low levels in cities, or other PM increments given in parentheses; NS Changes = No significant changes.

References:
[B]Dockery et al. (1989)
[C]Ware et al. (1986)
[D]Dockery et al. (1996)
[E]Abbey et al. (1995a,b,c)
[F]Peters et al. (1999a)
[G]McConnell et al. (1999)
[H]Berglund et al. (1999)
[I]Raizenne et al. (1996)
[J]Peters et al. (1999b)
[K]Gauderman et al. (2000)
[L]Abbey et al. (1998)

et al., 1998). Associations were also found with sulfates and O_3, but not SO_2, in males. In two-pollutant models, the coefficients for PM_{10} and sulfates were found to remain unchanged or increase in size, while O_3 and SO_2 were reduced and lost statistical significance.

Numerous long-term studies of respiratory effects have been conducted in non-North American countries, and many report significant associations between indicators of long-term PM exposure and either decreases in lung function or increased respiratory disease prevalence (summarized in Table 6-23 of the draft CD). These new findings are consistent with those of the previous review as well as with findings of associations between short-term PM exposure and increased respiratory symptoms or decreased lung function. Long-term PM exposures (months to years) may be associated with decreased lung function growth or increased incidence of respiratory disease, but there are still few publications for these effects, and the results are not entirely consistent or conclusive. However, the overall results from the non-North American studies lend general support to the coherence of respiratory effects associated with long-term PM exposure reported across disciplines and health studies.

3.3.3.3 Effects on the Cardiovascular System

In the last review, evidence was available from a number of epidemiology studies indicating that PM was associated with increased mortality and hospital admissions for cardiovascular diseases. These findings inspired further research so that an expanded body of evidence is available in this review from toxicology, epidemiology, and controlled human exposure studies. As described above, new epidemiological evidence generally supports the previous findings. In addition, new evidence from controlled human exposure, toxicological and epidemiological studies indicates that exposure to ambient PM, PM from combustion sources, or PM surrogates may be associated with additional cardiovascular health endpoints such as changes in heart rate variability and plasma fibrinogen levels.

PM was first linked with arrhythmia in toxicological studies, notably in an important new series of studies using inhalation exposure to CAPs. Changes in electrocardiogram (ECG) patterns, increased heart rate variability and decreased heart rate have been reported in a toxicology study using dogs exposed to CAPs (Godleski et al., 2000). The CD concludes that the findings for heart rate variability and ECG changes, respectively, suggest both pro- and anti-arrhythmic responses (CD, p. 8-31). The ECG changes included increases in the S-T peak, which suggests that CAPs can augment the ischemia associated with coronary artery blockage in this animal model (CD, p. 8-32).

Similarly, altered ECG pattern was reported in ROFA-treated spontaneously hypertensive rats (Kodavanti et al., 2000). However, Muggenberg et al. (2000) reported no consistent changes in ECG pattern in ROFA-treated beagle dogs. Increased arrhythmia was reported in rats exposed to ROFA and to urban particles collected in Ottawa; no cardiac effects were reported with exposure to Mt. St. Helens volcanic ash, which is one form of crustal material (Watkinson et al., 2000). Watkinson and colleagues used several animal models in this study, and reported exaggerated effects in rats that had been treated with monocrotaline, including premature mortality. Some effects were also reported in healthy rats, though mortality only occurred in the compromised animals. Increased mortality was reported in a previous study using ROFA exposures in monocrotaline-treated rats, and the authors also reported serious arrhythmic events in normal rats exposed to ROFA (Watkinson et al., 1998). The draft CD concludes that "animal studies have provided initial evidence that high concentrations of inhaled or instilled particles can have systemic, especially cardiovascular, effects. In the case of [monocrotaline-treated] rats, these effects may be lethal." (CD, p. 8-34).

In addition, one new epidemiological study used data on discharge frequency from implanted cardiac defibrillators; discharges occur when the patient is experiencing cardiac arrythmia. Peters et al. (2000) reported generally positive associations between increased defibrillator discharges and PM_{10}, $PM_{2.5}$, and particulate black carbon, but the associations were only significant for $PM_{2.5}$.

In several studies, tests of cardiac function (e.g., heart rate, heart rate variability) were done repeatedly for panels of elderly people over a period of several weeks. Generally, increased heart rate and decreased heart rate variability are associated with increased mortality from cardiovascular disease; further discussion of these cardiac health measures is included in Appendix B to Chapter 6 of the draft CD. Most new studies reported decreases in several measures of heart rate variability with increased PM (Liao et al., 1999; Gold et al., 2000; Pope et al., 1999c), though Pope et al. (1999c) reported a significant increase with one measure of short-term heart rate variability for PM_{10}. Significant associations were reported between $PM_{2.5}$ and heart rate variability in panel studies conducted in Baltimore and Boston (Liao et al., 1999; Gold et al., 2000). Gold et al. (2000) did not find associations between heart rate variability and $PM_{10-2.5}$, or with O_3, CO or SO_2.

The findings on changes in heart rate are less consistent than those for heart rate variability. In Utah Valley, Pope et al. (1999b) reported a significant increase in heart rate with ambient PM_{10} concentration, but no association with oxygen saturation, using a larger cohort of elderly subjects than in the first study. An association was also reported between TSP and increased heart rate (Peters et al., 1999) in a European study; significant increases were also found with SO_2, though the authors observe that SO_2 may be acting as an indicator for inhalable particles in this study. However, decreased heart rate was reported in the Boston panel study (Gold et al., 2000); associations were also found with NO_2 and SO_2, but the associations with $PM_{2.5}$ were more stable and retained significance in two-pollutant models. Decreased heart rate was also reported in an animal study using intratracheal installation of urban PM (but not with Mt. St. Helens volcanic ash) (Watkinson et al., 2000). In a study using rats and hamsters, no effects were reported in hamsters, but increased heart rate and blood cell differential counts were reported in rats (Gordon et al., 2000).

Some studies have reported increases in blood components or characteristics. Fibrinogen is a blood clotting factor and it is released in inflammatory processes; it has been reported to be a risk factor for ischemic heart disease and cerebrovascular disease, and it contributes to blood plasma viscosity (Gardner et al., 2000). In humans exposed to concentrated ambient fine PM, fibrinogen levels were increased in blood obtained 18 hours after exposure, and some inflammatory effects were also reported (Ghio et al., 2000). In a European cohort of heart patients, increased fibrinogen levels were a significant risk factor for the occurrence of cardiovascular events, and there was evidence for an interaction between PM (measured as BS) and fibrinogen levels (Prescott et al., 2000). However, fibrinogen level was not associated with PM exposure in another European epidemiology study (Seaton et al., 1999).

Using data from an existing European cohort study, conducted during a time period that included an episode of unusually high pollution levels, associations were reported between TSP and levels of C-reactive protein, which is an indicator of inflammation, tissue damage and infection, and generally related to increased risk of coronary events or ischemic syndromes (Peters, et al., 2000). Associations were also reported with increased plasma viscosity (associated with increased risk of heart attacks) in the blood and levels of TSP, though the associations were not statistically significant (Peters et al., 1997). This study also reported associations with SO_2 and CO that

reached statistical significance for women, but not for men. Increased C-reactive protein was reported to be associated with ambient PM_{10} in one epidemiology study in the United Kingdom study (Seaton et al., 1999).

A number of toxicology studies have also reported such hemolytic effects as changes in blood factors such as hemoglobin levels or platelet counts. Using animals exposed to CAPs, analyses were done with PM components and factor analysis methods were used to assess effects of PM from different sources. None of the PM factors was associated with changes in platelet count, but several factors or components were associated with changes in counts of inflammatory cells, such as white blood cells (Clarke et al., 2000). The sulfur factor was associated with decreases in red blood cell counts and hemoglobin levels, while some inflammatory changes were reported to be associated with the aluminum/silica factor and the vanadium/nickel factor. In this study, no associations were reported with concentrated fine PM mass. One new epidemiology study does not show significant changes in blood factors such as hemoglobin levels or platelet counts, but does find changes in red blood cell count (Seaton et al., 1999).

Though the number of these studies is small, and there are some inconsistencies in findings between studies, these results are generally coherent with findings of increased mortality or hospital admissions for cardiovascular diseases. It should be noted that what appear to be inconsistencies in findings may reflect differing levels of sensitivity and ability to distinguish exposure and temporal features across studies from different disciplines. Regarding the epidemiology studies, the draft CD concludes: "The above findings add support for some intriguing hypotheses regarding possible mechanisms by which PM exposure may be linked with adverse cardiac outcomes. They are especially interesting in terms of implicating both increased blood viscosity and C-reactive protein, a biological marker of inflammatory responses thought to be predictive of increased risk for serious cardiac events" (CD, p. 6-140). Animal toxicology findings were generally consistent with findings of human studies, though as observed previously, there are inconsistencies between studies for a number of individual effects.

The results of new epidemiological studies show PM exposure to be associated with excess risk of mortality or hospital admissions for cardiovascular diseases. The results of panel studies, controlled human exposure studies, and animal toxicology studies generally provide coherence with the findings from community health studies in finding associations with increased heart rate,

decreased heart rate variability, increases in inflammatory substances such as C-reactive protein, and in plasma viscosity or blood fibrinogen levels. It must be recognized that these findings are from only a few studies and there are a few inconsistencies in findings between studies; caution is also urged when comparing studies conducted in differing animal models and using high dose or exposure levels. Nonetheless, these findings shed some light on potential mechanisms for the associations with increased mortality or hospital admissions for cardiovascular diseases observed in epidemiology studies.

3.3.4. Consistency and Coherence of Health Effects Evidence

The 1996 Staff Paper pointed out the inherent limitations in trying to determine the role of PM by examining even the most thorough studies of individual cities that show associations between ambient PM and various health effects. Accordingly, the staff presented a more comprehensive synthesis that considered the consistency and coherence of the available evidence in evaluating the likelihood of PM being causally associated with the observed effects (EPA, 1996b, V-54 to 58). While significantly more evidence of associations between ambient PM and health effects is now available, including multi-city studies that address some of the single-city limitations, it is still important to consider the consistency and coherence of the available evidence as a whole.

As discussed in the last review, consistency of an association is evidenced by repeated observations by different investigators, in different places, circumstances and time; and by the consistency of the association with other known facts (EPA, 1996a, Chapter 13; Bates, 1992). Beyond considering the consistency of associations for individual health endpoints, coherence refers to the logical or systematic interrelationship between different health indices that would be expected to be seen across studies of different endpoints or from different disciplines. The consistency and coherence of the expanded body of evidence now available is discussed and evaluated below.

3.3.4.1 Consistency

The 1996 Criteria Document summarized over 80 community epidemiological studies evaluating associations between short-term PM levels and mortality and morbidity endpoints in a number of locations throughout the world, using a variety of statistical techniques, of which over 60 studies found consistent, positive, significant associations (EPA, 1996a, Tables 12-2 and 12-8

to 12-13). The 1996 Staff Paper displayed the relative risk estimates for mortality and morbidity effects associated with PM_{10} from the U.S. and Canadian studies, concluding that despite the variations in study locations and approaches, the estimates for each health endpoint were relatively consistent among the studies; although, as would be expected, some variation was seen (EPA, 1996b, B-55 and Figure V-2).

As discussed above, since the last review, more than 70 new PM-mortality studies alone have been published, as well as a large number of new morbidity studies, and several major multi-city studies. The draft CD notes that the effects estimates from the new studies in the U.S. and throughout the world are generally consistent with those observed in the last review, not only from PM_{10} multi- and single-city studies (shown above in Figures 3-4 and 3-7 from U.S. and Canadian studies for mortality and hospital/ER admissions, respectively), but also from the significantly expanded body of studies of fine-fraction (e.g., $PM_{2.5}$) particles (similarly shown above in Figures 3-5 and 3-8) (CD, p. 6-266). The evidence from coarse-fraction ($PM_{10-2.5}$) studies (as shown above in Figures 3-6 and 3-9), while somewhat expanded, remains more limited and presents more difficulty in attempting to draw conclusions about the consistency of the reported associations across studies (CD, p. 6-267). Bringing together the findings for $PM_{2.5}$ from all U.S. and Canadian studies for a range of health endpoints from mortality to varying indices of morbidity, Figure 3-10 shows that the effects estimates for each health endpoint are relatively consistent among the studies, very similar to the consistent pattern observed for PM_{10} studies in the last review (EPA, 1996b, Figure V-2).

Looking more closely at the variations for particular endpoints observed across cities within the 90-city NMMAPS study reveals more heterogeneity of city-specific PM_{10}-mortality effects estimates than in the past review (as discussed above in Section 3.3.1.1.1). At least some of the increased variability is to be expected based on a study design that includes areas with more limited PM sampling days and population sizes than is usual for single-city publications. The CD presents some evidence that the inter-city variability may, at least in part, simply reflect imprecise PM effect estimates derived from smaller-sized analyses (of less extensive available air pollution data or numbers of deaths) tending to obscure more precise estimates from larger-size analyses for

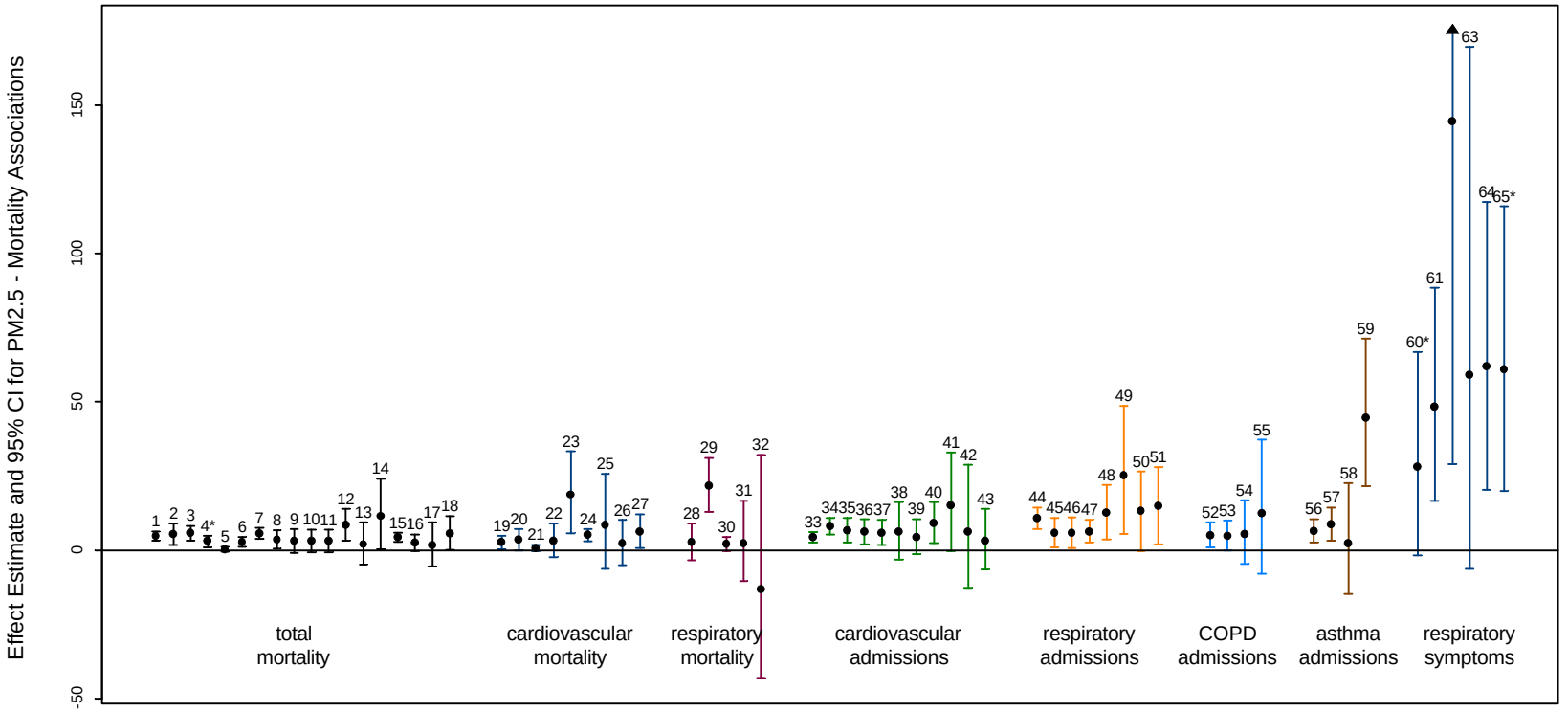


Figure 3-10. Estimated excess mortality and morbidity risks per 25 µg/m^3 $PM_{2.5}$ from U.S. and Canadian studies (listed below), showing consistency and coherence across the different effects categories. Within each category, results are ranked by decreasing natural log of the mortality- or morbidity-days product. Multi-city studies denoted with an asterisk.

Total Mortality:
1. Burnett et al., 1998, Toronto, Canada
2. Schwartz, 2000c Boston, MA
3. Goldberg et al., 2000, Montreal, Canada
4. Burnett et al., 2000, 8 Canadian cities
5. Ostro et al., 1995, So. California
6. Schwartz et al., 1996, St. Louis, MO
7. Schwartz et al., 1996, Boston, MA
8. Schwartz et al., 1996, Knoxville, TN
9. Schwartz et al., 1996 , Portage, WI
10. Lippmann et al., 2000, Detroit, MI
11. Mar et al., 2000, Phoenix, AZ
12. Fairley, 1999, Santa Clara, CA
13. Schwartz et al., 1996, Topeka, KS
14. Ostro et al., 2000, Coachella Valley, CA
15. Tsai et al., 2000, Newark, NJ
16. Schwartz et al., 1996, Steubenville, OH
17. Tsai et al., 2000, Elizabeth, NJ
18. Tsai et al., 2000, Camden, NJ

Cardiovascular Mortality:
19. Moolgavkar et al., 2000, Los Angeles, CA
20. Goldberg et al., 2000, Montreal, Canada
21. Ostro et al., 1995 So. California
22 Lippmann et al., 2000, Detroit, MI
23. Mar et al., 2000, Phoenix, AZ
24. Tsai et al., 2000, Newark, NJ
25. Ostro et al., 2000, Coachella Valley, CA
26. Tsai et al., 2000, Elizabeth, NJ
27. Tsai et al., 2000, Camden, NJ

Respiratory Mortality:
28. Moolgavkar., 2000a, Los Angeles
29. Goldberg et al., 2000, Montreal, Canada
30. Ostro et al., 1995, So. California
31. Lippmann et al., 2000, Detroit, MI
32. Ostro et al., 2000, Coachella Valley, CA

Cardiovascular Admissions:
33. Moolgavkar, 2000b, Los Angeles, CA
34. Burnett et al., 1999, Toronto, Canada (IHD)
35. Burnett et al., 1999, Toronto, Canada (HF)
36. Burnett et al., 1999, Toronto, Canada (dysrhythmia)
37. Burnett et al., 1999, Toronto, Canada
38. Tolbert et al., 2000, Atlanta, GA)
39. Lippmann et al., 2000, Detroit, MI (IHD)
40. Lippmann et al., 2000, Detroit, MI (HF)
41. Stieb et al., 2000, St. John, Canada
42. Tolbert et al., 2000a, Atlanta, GA (dysrrhythmia
43. Lippmann et al., 2000, Detroit (dysrrhythmia,)

Respiratory Admissions:
44. Burnett et al., 1999, Toronto, Canada (resp. infection)
45. Lumley and Heagerty, 1999, Seattle, WA (PM1)
46. Stieb et al., 2000, St. John, Canada
47. Burnett et al., 1999, Toronto, Canada
48. Lippmann et al., 2000, Detroit, MI (pneumonia)
49. Delfino et al., 1997, Montreal, Canada
50. Delfino et al., 1998, Montreal, Canada
51. Thurston et al., 1994, Toronto, Canada

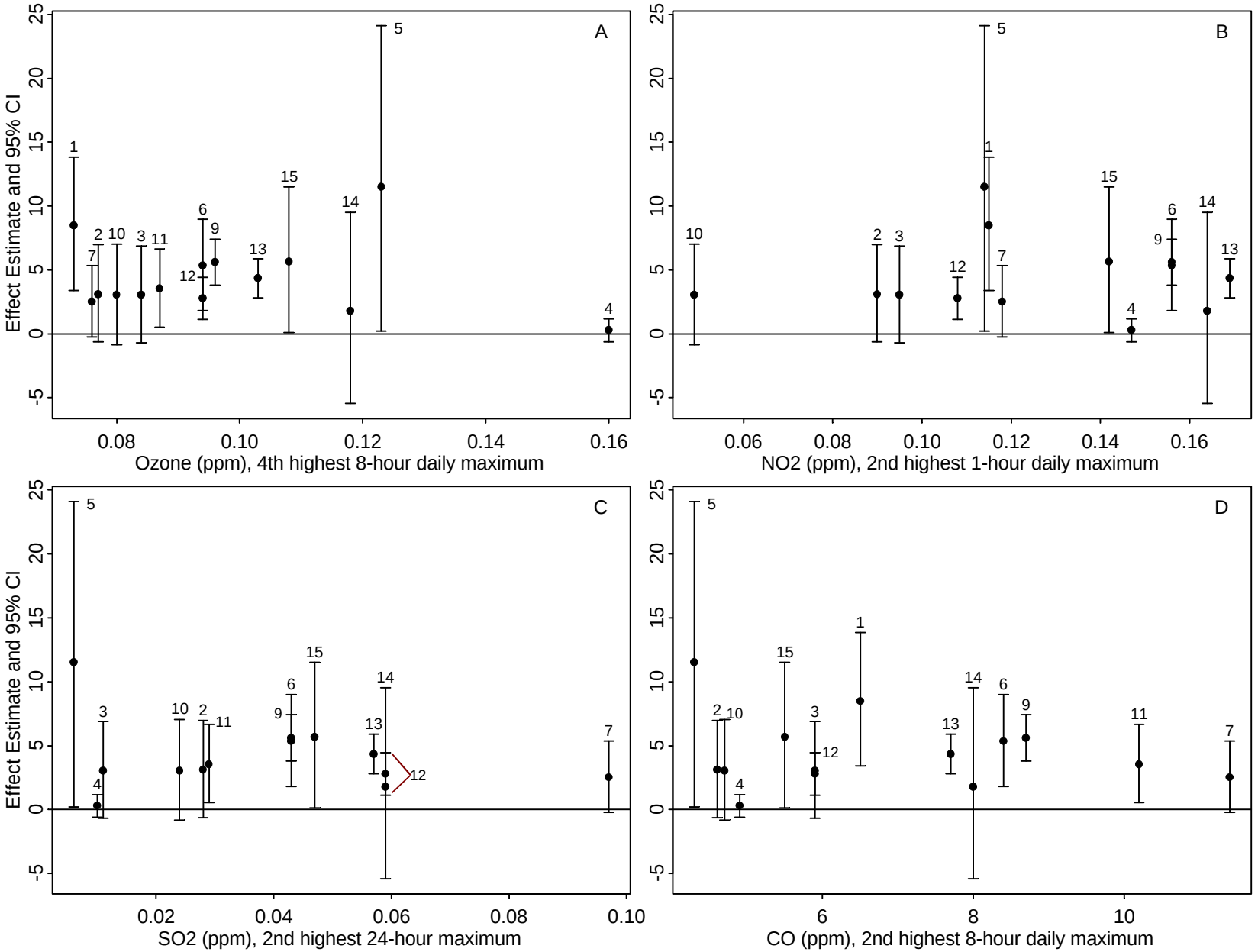
COPD Admissions:
52. Moolgavkar, 2000c, Los Angeles, CA
53. Burnett et al., 1999, Toronto, Canada
54. Lippmann et al., 2000, Detroit, MI
55. Tolbert et al., 2000a, Atlanta, GA

Asthma Admissions:
56. Burnett et al., 1999, Toronto, Canada
57. Sheppard et al., 1999, Seattle, WA
58. Tolbert et al., 2000, Atlanta, GA
59. Norris et al., 1999, Seattle, WA

Respiratory Symptoms:
60. Schwartz and Neas, 1999, 6 U.S. city reanalysis (cough)
61. Neas et al., 1996, State College, PA (cough)
62. Neas et al., 1995, Uniontown, PA (cough)
63. Neas et al., 1996, State College, PA (wheeze)
64. Neas et al., 1996, State College, PA (cold)
65. Schwartz and Neas, 1999, 6 U.S. city reanalysis (lower resp. symptoms)

other locations, which tend to be consistently more positive and statistically significant (CD, p. and 6-260 to 6-263). The variability may also be due to other analytical factors, or reflect an as yet unexplained location-specific difference in exposures or weather and air pollution mixes (CD, p 6-260). The CD also discusses the suggestion of regional heterogeneity in the quantitative estimates, which suggest larger effects estimates for the Northeast Southern California than other regions (CD p 6-263, 6-264). It is as yet unclear whether these are significant and real differences, or whether related to analytical or city/sampling size issues. The CD notes that, if real, such differences would not be inconsistent with potential regional differences in particle size/composition or population exposure patterns (CD, p6--264). While warranting further study, the observed inter-city and regional variations in the NMMAPS do not call into question the qualitative consistency observed across all the available studies, including the combined results from the available multi-city studies.

In further considering the consistency of the reported PM effects, it is important to evaluate the sensitivity of the PM estimates to the differing levels of co-pollutants present in various study locations. Such an evaluation supplements the multi-city and single city analyses discussed in earlier sections. In the last review, this analysis examined PM_{10} effects estimates, to consider whether the reported PM effects can be interpreted appropriately as being likely independent effects attributable to PM, or whether the evidence suggests that the reported PM effects likely result from the influence of other pollutants present in the ambient air in the study locations, either through confounding or effects modification. As discussed in the 1996 Staff Paper, if PM is acting independently, then a consistent association should be observed in a variety of locations of differing levels of co-pollutants. On the other hand, if the reported PM effects are confounded or modified by any of the co-pollutants, then the reported PM effects would be expected to show a trend of being higher in areas with relatively high concentrations of the confounding co-pollutant and lower in areas with relatively low co-pollutant concentrations (EPA, 1996b, V-55). Figure 3-11 shows the reported $PM_{2.5}$ mortality effects estimates (from single-pollutant models) from U.S. and Canadian studies relative to the levels of O_3, NO_2, SO_2, and CO present in the study locations. As was seen in the last review for PM_{10} (EPA, 1996b, Figure V-3a,b), the magnitude and statistical significance of the associations reported between $PM_{2.5}$ and mortality in these studies


A
B
C
D
Effect Estimate and 95% CI
Ozone (ppm), 4th highest 8-hour daily maximum
NO2 (ppm), 2nd highest 1-hour daily maximum
SO2 (ppm), 2nd highest 24-hour maximum
CO (ppm), 2nd highest 8-hour daily maximum

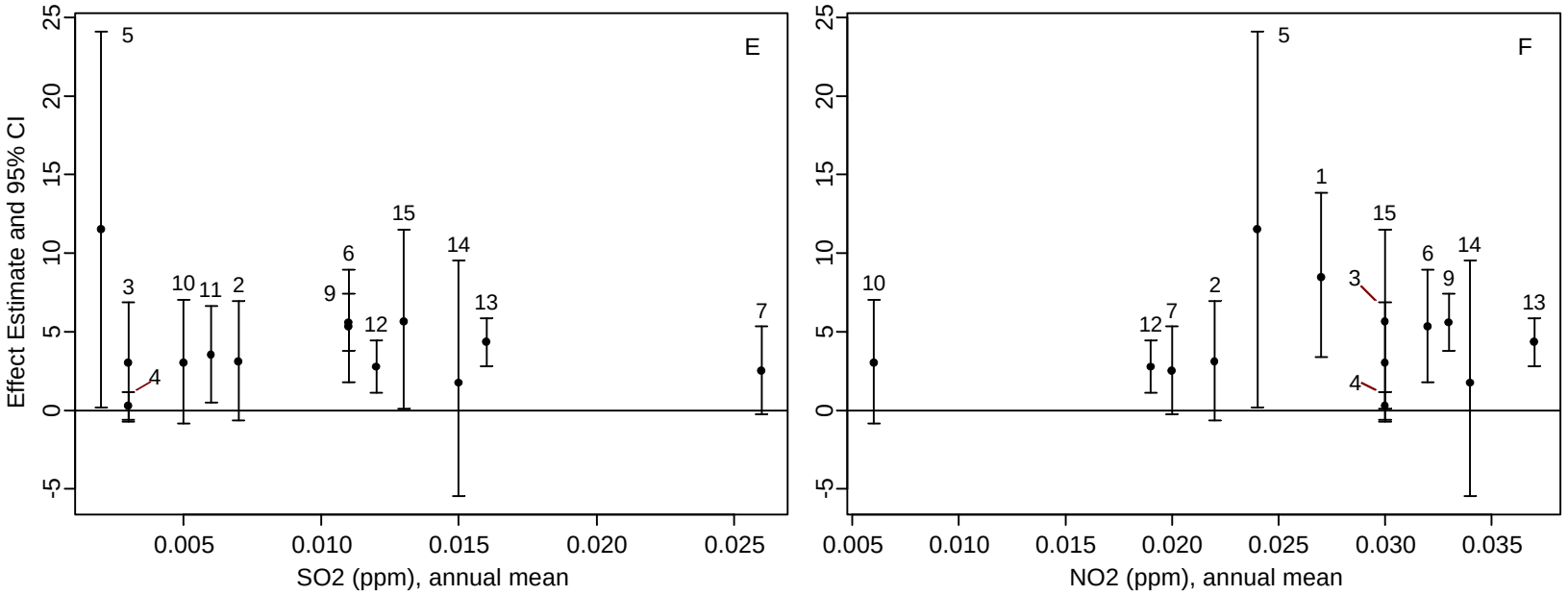


Figure 3-11. Associations between $PM_{2.5}$ and total mortality from U.S. studies, plotted against gaseous pollutant concentrations from the same locations. Air quality data obtained from the Aerometric Information Retrieval System (AIRS) for each study time period: (A) mean of 4th highest 8-hour ozone concentration; (B) mean of 2nd highest 8-hour CO concentration; (C) mean of 2nd highest 1-hour NO_2 concentration; (D) mean of 2nd highest 24-hour SO_2 concentration; (E) annual mean SO_2 concentration; (F) annual mean NO_2 concentration. Study locations are identified below (data in Appendix 3-A, Table 5)

1. Fairley, 1999, Santa Clara
2. Lippmann et al., 2000, Detroit
3. Mar et al., 2000, Phoenix
4. Ostro et al., 1995, So. California
5. Ostro et al., 2000, Coachella Valley
6. Schwartz 2000c, Boston
7. Schwartz et al., 1996, Boston
8. Schwartz et al., 1996, Knoxville
9. Schwartz t al., 1996, Portage
10. Schwartz et al., 1996, St. Louis
11. Schwartz et al., 1996, Steubenville
12. Schwartz et al., 1996, Topeka
13. Tsai et al., 2000, Camden NJ
14. Tsai et al., 2000, Elizabeth NJ
15. Tsai et al., 2000, Newark NJ

show no trends with the levels of any of the four gaseous co-pollutants. While not definitive, these consistent patterns indicate that it is more likely that there is an independent effect of $PM_{2.5}$, as well as PM_{10}, that is not confounded or appreciably modified by the gaseous pollutants.

More specific information relevant to evaluation of potential confounding or effects modification for each of the four major gaseous co-pollutants is discussed below in Section 3.5.1.

3.3.4.2 Coherence

In addition to the consistently observed associations for each of these effects, the newly available epidemiological and toxicological evidence reinforces and adds to the coherence in the kinds of health effects associated with PM exposure noted in the last review (EPA, 1996b, V-56). The 1996 Criteria Document provided a qualitative review of the coherence of the health effects associated with both short- and long-term exposure to PM (EPA, 1996a, Tables 13-6 and 13-7). In that review, it was noted that PM is related to a number of logically linked effects of both the respiratory and cardiovascular systems. Respiratory system effects included premature mortality and increased hospital and emergency room admissions for respiratory-related causes, as well as increased respiratory disease and symptoms and decreased lung function. Cardiovascular system effects included premature mortality and increased hospital and emergency room admissions for cardiovascular-related causes. In addition to this observed qualitative coherence, quantitative coherence was also observed in that the increases in respiratory- and cardiovascular-related hospital admissions were more frequently occurring than the increases in mortality for the same causes, based on reported relative risk estimates and baseline population incidence statistics (EPA, 1996a, Table 13-8).

The newly available evidence of PM-related effects expands upon the previously observed qualitative coherence. New PM-related effects associations have now been reported, including increased physicians' visits for respiratory causes and various new cardiovascular-related endpoints, that serve to fill in the spectrum of observed effects from physiological changes that are linked to more serious health outcomes through premature mortality. The new epidemiologic and toxicologic evidence on cardiovascular-related endpoints discussed in Section 3.3.3.3 above is suggestive of coherence in effects on the cardiovascular system for ambient measured as CAPs, $PM_{2.5}$, or PM_{10}. It is important to note the draft CD cautions that the findings should be viewed

as providing limited or preliminary support for PM-related cardiovascular effects (CD, p. 6-268). Changes in heart rate or heart rate variability are linked with more serious cardiovascular outcomes, including increased risk of heart attacks. The findings of increased levels of fibrinogen or plasma viscosity indicate a potential link between ambient PM exposure and the occurrence of ischemic events, and the increases seen in blood factors such as C-reactive protein provide evidence for inflammatory changes that can be linked with more serious cardiac effects.

The new evidence also continues to support the quantitative coherence observed in the last review. For example, in the NMMAPS studies, 2.6% and 3.5% increases in total and cardiorespiratory mortality, respectively, were reported for a 50 $\mu g/m^3$ increase in daily PM_{10}, while increases in hospital admissions of 6% (for cardiovascular causes, with a range across other studies of approximately 3% to 10%) and 10% (for COPD or pneumonia, with a range across other studies of approximately 5% to 25% for respiratory-related causes) were similarly reported. In addition, several new studies have reported associations with visits to physicians' offices for respiratory disease, ranging from 3% to 42% increases for a 50 $\mu g/m^3$ increase in daily PM_{10}. In the new studies on lung function changes or respiratory symptoms incidence, increases in risk of respiratory-related symptoms range up to over 50% per 50 $\mu g/m^3$ increase in daily PM_{10}. Updated baseline incidence rates for respiratory and heart diseases reported in the draft CD (p. 9-102 to 9-103), considered together with these illustrative ranges of effects estimates (and with the ranges shown above in Figures 3-3 through 3-10), continue to show that the quantitative coherence across all PM-related endpoints, especially for PM_{10} as well as for $PM_{2.5}$, is consistent with expectations (CD, p. 6-267 to 6-268). Further, as noted in the last review (EPA, 1996b, V-57), the larger effects estimates reported in long-term exposure studies are coherent with the smaller effects estimates reported for associations with daily changes in PM concentrations. As noted above in the discussion of consistency, the limited amount of information available on $PM_{10-2.5}$ presents more difficulty in attempting to draw conclusions about coherence of effects of coarse-fraction particles.

As noted in the last review, the coherence of PM-related effects is further strengthened by studies demonstrating associations with a range of effects in the same population, as illustrated by studies in a number of locations (EPA, 1996b, V-57). For example, studies in Utah Valley have shown a number of closely related health outcomes associated with PM exposures, including decreased lung function, increased respiratory symptoms, increased medication use in asthmatics,

and increased elementary school absences (frequently due to upper respiratory illness) (EPA, 1996b, V-57).

In summary, these observations suggest coherence from subtle changes in lung function or heart rate variability to increased mortality from cardiorespiratory diseases reported in epidemiological studies. Taken as a whole, the newly available health studies together with studies available in past reviews show general coherence for PM-related effects in the respiratory and cardiovascular systems. The expanded evidence for coherence in effects, along with previously described observations of marked consistency in the results of recent studies and those available in the last review, support a causal link between PM, especially as indexed by PM_{10} and $PM_{2.5}$, and effects on the cardiovascular and respiratory systems (CD, p. 6-266 to 6-267).

3.4 SENSITIVE GROUPS FOR PM-RELATED HEALTH EFFECTS

In general, subpopulations that have been identified in previous PM NAAQS reviews as being potentially more sensitive to the adverse health effects of PM have included individuals with respiratory and cardiovascular disease, the elderly, children, and asthmatic individuals (EPA 1996b, pp. V-33 to V-36). As summarized in the draft CD, Section 9.7, new studies continue to support consideration of these subpopulations as potentially sensitive to PM.

Individuals with respiratory and cardiovascular disease: Numerous epidemiology studies have identified individuals with cardiorespiratory diseases (e.g., asthma, COPD) as being at greater risk for adverse effects with PM exposure (CD, p. 9-99). Most notably, one recent epidemiology study (Goldberg et al., 2000) linked mortality data with information on preexisting health conditions (e.g., pharmaceutical prescriptions, medical visits) to investigate differences between groups according to health status. The authors reported that associations between $PM_{2.5}$, COH or sulfates and total mortality were increased among individuals with preexisting acute lower respiratory disease, congestive heart failure, and any cardiovascular disease. New information from studies of cardiovascular health measures such as plasma viscosity or changes in heart rate or heart rate variability provides additional support for consideration of persons with cardiovascular disease as being susceptible to the PM-related effects (CD, p. 9-112).

Asthma has been of particular public interest as a respiratory condition that may lead to sensitivity to air pollution effects. Included in Appendix A, Table 2, are numerous epidemiology

studies reporting increased medical visits for asthma with exposure to PM_{10}, $PM_{2.5}$ or $PM_{10-2.5}$, and most studies reported significant associations. In considering asthmatics as a susceptible subpopulation, the results for studies evaluating changes in lung function and respiratory symptoms were evaluated separately for asthmatic and nonasthmatic subjects. The draft CD reported that asthmatic subjects had greater reduction in pulmonary function with PM exposure, but both asthmatic and non-asthmatic subjects had similar responses in respiratory symptom studies (CD Section 6.3.3.1). A number of toxicology studies have evaluated the effects of particles or surrogate particles on allergic diseases, including allergic asthma, and the draft CD finds that "[t]hese studies provide biological plausibility for the exacerbation of allergic asthma associated with episodic exposure to PM" (CD, p. 8-45).

New dosimetry studies have shown that, among people with COPD, airflow may be unevenly distributed due to airway obstruction, resulting in deeper penetration of particles in the better ventilated regions, or increased local deposition of particles. In addition, ventilation rate and rate of air flow is often increased with airway obstruction. The findings of these studies suggest that total lung deposition generally is increased with obstructed airways, regardless of deposition distribution between the tracheobronchial or alveolar regions (CD, p. 7-22).

A number of animal models of susceptible populations have been used in toxicology studies examining PM. These include: monocrotaline treatment of rats as a model of cardiorespiratory disease; SO_2-induced chronic bronchitis in rats; ovalbumin sensitization in rodents as a model of airway hyperresponsiveness; and genetically predisposed animals such as the spontaneously hypertensive rat. The advantages and disadvantages of these animal models are discussed more fully in Section 8.4 of the draft CD. While recognizing that further research is needed, the draft CD concludes that these studies "have consistently shown that animals with compromised health, either genetic or induced, are more susceptible to instilled or inhaled particles, although the increased animal-to-animal variability in these models has caused problems" (CD, p. 8-87).

Age-related subpopulations: In the previous review, numerous studies indicated that the elderly and children are more susceptible to PM-related health effects (EPA, 1996a, p. 12-364). Similarly, in reviewing the recent studies of PM-related medical visits or admissions/visits for respiratory diseases, the draft CD finds that the groups identified as being most strongly affected by PM are older adults and the very young (CD, p. 6-172). Goldberg et al. (2000) also report that

associations between PM and mortality were generally larger among persons greater than 65 years of age, which is consistent with the findings of numerous previous studies. Several new epidemiology studies have reported significant associations between PM exposure and intrauterine growth reduction or low birth weight, known to be infant health risk factors, as well as excess infant mortality (CD, p. 9-106).

In addition, the draft CD highlights findings of a number of new studies that raise the possibility that deposition may be greater in children than adults; it is also noted that children's generally higher activity levels with accompanying higher ventilation rates might contribute to increased particle deposition (CD, p. 7-20). However, dosimetric evidence has not identified elderly adults to be at increased risk due to difference in lung deposition, clearance or retention of inhaled particles associated with aging, per se, though the draft CD concludes that "[p]robably of much more importance in placing elderly adults at increased risk for PM effects is the higher propensity for such individuals to have preexisting cardiovascular or respiratory disease conditions." (CD, p. 9-106).

Other Subpopulations: Other subpopulations have been evaluated as potentially susceptible groups in recent studies. New dosimetry studies have indicated that total lung deposition and deposition peaks may be greater in females than in males (CD Section 7.2.3.1), and one new epidemiology study reported that associations between PM_{10} and mortality were greater in females than males (Zanobetti and Schwartz, 2000). However, the reverse was found in the AHSMOG prospective cohort (described in Section 3.3.1.2) and no gender differences were reported in the largest prospective cohort studies (Six Cities and ACS).

Zanobetti and Schwartz (2000) did not find differences in PM_{10}-mortality associations in analyses stratified by race or education level (an indicator of socioeconomic status). Yet with long-term PM exposure, Krewski et al. (2000) reported greater mortality effects among those with lower levels of education. There is as yet insufficient evidence to identify new subpopulations as being potentially susceptible to PM-related effects. In summary, the findings of new epidemiology, dosimetry and toxicology studies provide support for previous findings that individuals with respiratory and cardiovascular disease, individuals with infections, the elderly, children, and asthmatic individuals are subpopulations that may be more sensitive to the adverse health effects of ambient PM exposure.

3.5 EVALUATION OF PM-RELATED HEALTH EFFECTS EVIDENCE

In the preceding sections, evidence from new health studies has been summarized and integrated with findings from previous reviews. As has been seen in previous reviews, much of the health evidence is taken from epidemiology studies, though critical new insights are offered in the results of toxicology and controlled human exposure studies. The 1996 CD and Staff Paper discussed, at some length, issues related to the interpretation and evaluation of epidemiological evidence. While recognizing that additional research was needed on some issues, the 1996 CD concluded that "the epidemiologic findings cannot be wholly attributed to inappropriate or incorrect statistical methods, misspecification of concentration-effect models, biases in study design or implementation, measurement errors in health endpoint, pollution exposure, weather, or other variables, nor confounding of PM effects with effects of other factors" (EPA, 1996a, p. 13-92). In this section, the new findings relevant to the interpretation of epidemiological information will be discussed.

In the evaluation of the health effects evidence, one important consideration is the evidence for health effects of PM alone or in the presence of co-pollutants. Throughout the preceding discussions on the nature of health effects associated with PM, and the consistency and coherence of the health evidence, consideration of potential confounding by co-pollutants has been discussed. Here, additional considerations relevant to each of the four major gaseous co-pollutants will be discussed in Section 3.5.1.

In addition, new information is available on potential health effects of PM components or source-related PM, as summarized in Section 3.5.2. Several additional key issues are discussed in the draft CD, and the new information that would inform this NAAQS review is summarized in Section 3.5.3 for: (1) the lag period between exposure and occurrence of health effects; (2) the exposure time window for effects, specifically relating acute exposure periods of hours to days with health effects; (3) the influence of model specification on epidemiology findings; and (4) the influence of exposure error or exposure misclassification on reported PM-health associations.

3.5.1 Additional Evidence on the Role of Gaseous Co-pollutants

In the preceding sections, several methods for assessing potential confounding by co-pollutants were discussed (i.e., multi-pollutant modeling in multiple or single locations, assessing

the relationship between PM-mortality associations and the PM-co-pollutant correlation, and observing the relationships between PM-health effect estimates and co-pollutant concentrations). The results of these analyses generally support an independent association between PM and health effects such as mortality or hospital admissions or emergency room visits for cardiorespiratory diseases. In this section, additional information is summarized for each of the major gaseous co-pollutants identified as potential confounding factors or effects modifiers for PM-health associations.

Ozone. As observed in the 1996 Staff Paper, among the gaseous co-pollutants, there is greater potential for O_3 to be a confounder in studies of respiratory effects (EPA, 1996b, p. V-51). Ozone has been found to have independent effects on the respiratory system; for example, increased hospital admissions and emergency room visits for respiratory causes have been associated with ambient O_3 exposures (EPA 1998, p. 25). Among recent studies, the PM effect estimates for COPD (but not pneumonia) hospital admissions were reduced in Lippmann et al. (2000), and Tolbert et al. (2000a) and Delfino et al. (1998) reported reductions in effects estimates for PM_{10} and $PM_{2.5}$ with asthma admissions when O_3 was included in the model. However, associations between PM indices and hospital admissions for respiratory disease remained significant in models containing O_3 in Toronto (Burnett et al., 1997), and in a number of the European and Latin American studies highlighted in Table 6-17 of the draft CD.

The epidemiology studies showed little evidence of confounding by O_3 for associations between PM and cardiovascular mortality or morbidity. In the multi-city epidemiology studies, associations between mortality and PM (including $PM_{2.5}$ or $PM_{10-2.5}$, where available) were relatively unaffected by the addition of O_3 to the models (10 U.S. cities, Schwartz et al., 2000; 8 Canadian cities, Burnett et al., 2000). The draft CD concludes that PM and O_3 can be most clearly separated as having independent effects, compared with other gaseous co-pollutants. (CD, p. 9-81).

Co-pollutants can serve not only as confounders or effect modifiers, but there may be interactive effects reported with co-exposure to multiple pollutants. Recent animal toxicology studies have tested effects of exposure to PM or PM surrogates (e.g., urban PM, carbon particles, acid aerosols) in combination with O_3 (CD, Table 8-10). In two Canadian studies, co-exposure to O_3 and urban particles potentiated the effects reported with O_3 alone (Bouthillier et al., 1998;

Vincent et al., 1997), while mixed results were reported from studies using combinations of acid aerosols and O_3 (CD Table 8-10).

Carbon monoxide. CO reduces oxygen delivery to the body's organs and tissues, and the health threat from CO is most serious for those who suffer from cardiovascular disease, such as angina pectoris (EPA, 1998, p. 10). Thus, CO may be expected to potentially confound associations between PM and cardiovascular mortality or morbidity. It is considered less likely that CO would confound associations with respiratory effects.

New studies have generally reported associations between PM and mortality (especially from total or respiratory causes) to be unaffected when CO was added to two-pollutant models (Lippmann et al., 2000; Burnett et al., 1998). Little evidence of confounding was also reported in two-pollutant models for respiratory admissions/visits. However, in some studies of admissions/visits for cardiovascular diseases, the PM effects sizes were reduced in two-pollutant models with CO. Reflecting also the evidence summarized in the recent CD for CO, the draft CD finds that "[a]mong the gaseous criteria pollutants, CO has emerged as the most consistently associated with cardiovascular (CVD) hospitalizations. The CO effects are generally robust in the multi-pollutant model, sometimes as much so as PM effects. However, the typically low levels of ambient CO concentrations in most such studies and minimal expected impacts on carboxyhemoglobin levels and consequent associated hypoxic effects thought to underlie CO CVD effects complicate interpretation of the CO findings and argue for the possibility that CO may be serving as a general surrogate for combustion products (e.g., PM) in the ambient pollution mix." (CD, p. 9-73).

As observed in the 1996 Staff Paper, exposure misclassification may introduce significant problems in interpreting epidemiological findings on CO-related effects, due to the nature of urban and indoor sources of CO (EPA, 1996b, p. V-52). While CO has been reported to cause cardiac effects in the higher concentrations used in controlled human exposure studies, it is unlikely that CO is confounding the effects associated with ambient PM in the more recent epidemiological studies.

Sulfur dioxide. Potential confounding between PM and SO_2 has been evaluated in some detail in previous reviews. As stated in the 1996 Staff Paper, both PM (measured as TSP or black smoke) and SO_2 were elevated during the historical pollution episodes such as those occurring in London during the 1950's, and the concentrations of SO_2 and PM were highly correlated due to

common emissions sources. A number of epidemiological analyses evaluated potential confounding for PM and SO_2 in associations with mortality, and in some studies it was difficult to distinguish effects of SO_2 and PM. It was observed, however, that SO_2 generally does not penetrate into the deeper portions of the lung, based on evidence from dosimetry and controlled human exposure studies. In addition, SO_2 concentrations are generally low indoors (where people spend the greatest part of their time) due to rapid removal by indoor surfaces. Staff concluded that "it is unlikely that SO_2 is responsible for all or the observed associations between PM and mortality" (EPA, 1996b, p. V-49).

Newly published epidemiological studies generally find no evidence of confounding in associations with mortality or hospital admissions or emergency room visits with short-term PM exposures when SO_2 is included in models. However, in the reanalysis of long-term studies (discussed in Section 3.3.1.2), significant associations were reported between mortality and sulfur dioxide, and in multiple pollutant models the sulfur dioxide associations often appeared stronger than those for fine particles and sulfates. However, the SO_2 associations were also reduced in two-pollutant models, and the correlation between SO_2 and sulfates makes it difficult to distinguish their effects. In the results of toxicology studies with co-exposure to PM and SO_2, there was little evidence for interaction with particles in causing effects (CD Table 8-10).

Nitrogen dioxide. NO_2 exposure has been associated with changes in airway responsiveness and pulmonary function in individuals with preexisting respiratory illnesses and increases in respiratory illnesses in children (Trends report, p. 20). In multi-pollutant models available from the new epidemiology studies, inclusion of NO_2 in the models has varying effects on the effect estimate for PM_{10}. Lippmann et al. (2000), for example, reports results for total, cardiovascular, and respiratory mortality, as well as hospital admissions for a number of specific respiratory or cardiovascular diseases. In two-pollutant models with NO_2, the PM effects are often relatively unaffected, but when substantial changes are noted, the PM effect may be either increased or decreased. Moolgavkar (2000b) finds that NO_2 reduces effect estimates between PM_{10} and cardiovascular admissions in Cook County, IL, but not in Los Angeles County, CA or Maricopa County, AZ. The 1996 Staff Paper recognized that, especially in the western U.S., NO_x emissions can be a major source of fine particles, which makes it difficult to distinguish effects of the two pollutants (EPA, 1996b, p. V-53).

Summary. The CD concludes "Overall, it appears, however, that ambient PM and O_3 can be most clearly separated out as likely having independent effects, their concentrations often not being highly correlated. More difficulty is encountered, at times, in sorting out whether NO_2, CO, or SO_2 are exerting independent effects in cities where they tend to be highly correlated with ambient PM concentrations, possibly because of derivation of important PM constituents from the same source (e.g., NO_2, CO, PM from mobile sources) or a gaseous pollutant (e.g., SO_2) serving as a precursor for a significant PM component (e.g., sulfate)" (CD, p. 9-81).

In interpreting the findings of these multi-pollutant analyses, it is important to recognize that there are issues in co-pollutant confounding that multi-pollutant models may not be able to address. Inclusion of pollutants that are highly correlated with one another can lead to misleading conclusions in identifying a specific causal pollutant. For example, collinearity between pollutants may occur if the gaseous pollutants and PM come from the same sources, or if PM constituents are derived from gaseous pollutants (e.g., sulfates from SO_2) (CD, p. 6-227). Sources of PM constituents include combustion of various fuels, gasoline or diesel engine exhaust, and some industrial processes (CD, Table 9-2); these sources also emit gaseous pollutants. When collinearity exists, multi-pollutant models would be expected to produce unstable and statistically insignificant effect estimates for both PM and the co-pollutants (CD, p. 9-81).

Some investigators have raised the possibility that PM may be a key surrogate or marker for a larger subset of pollutants in the overall ambient air pollution mix (CD, p. 9-39). Given the heterogeneous nature of PM, co-pollutants may also be indicators for fine particles derived from specific combustion sources. For example, when CO is included in a two pollutant model with $PM_{2.5}$, CO may serve as an indicator for that portion of total $PM_{2.5}$ that is derived from mobile source emissions.

It is also important to consider differences in population exposures to the ambient pollutants. The link between ambient PM concentrations, measured at centrally-located monitors, and individuals' exposures to ambient PM is discussed at length in Chapter 5 of the CD and Sections 2.8 and 3.5.3.3 of the Staff Paper. In considering exposure to the gaseous pollutants as well, the CD states, "it is also significant to note that, although ambient concentrations of a number of gaseous pollutants (O_3, NO_2, SO_2) often are found to be highly correlated with various PM parameters, personal exposures to these gases are not correlated highly with personal exposure to

PM indicators. The correlations of the ambient concentrations of these gases also are not correlated highly with the personal exposure to these gases. Therefore, when significant statistical associations are found between these gases and health effects, it could be that these gases may, at times, be serving as surrogates for PM rather than being causal themselves. Pertinent information on CO has not been reported." (CD, p. 9-85)

Taking into consideration the findings of single- and multi-city studies and other evaluations of potential confounding by gaseous co-pollutants described in preceding sections, the evidence generally indicates that PM, alone or in combination with other pollutants, has independent effects on morbidity and mortality. In reviewing the epidemiological evidence, the draft CD concludes that "[o]verall, although such issues may warrant further evaluation, it appears unlikely at this time that such confounding accounts for the vast array of effects attributed to ambient PM . . ." (CD, p. 9-81).

3.5.2 PM Components or Sources

Much of the focus of the preceding discussions on the nature of PM-related effects has been epidemiological studies that use gravimetric PM measurements, with an emphasis on PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$. However, there is a growing body of information on effects associated with PM components, smaller ultrafine particles, or PM associated with specific sources. In the 1996 CD, evidence from toxicological studies on the effects of acid aerosols, metals, ultrafine particles, diesel emission particles, silica, and bioaerosols was available. Among the recent studies are epidemiology analyses on the effects of ultrafine particles or studies using factor analysis to evaluate the effects of PM from different sources. The following sections will discuss, to the extent that information is available, evidence on health associations with ultrafine particles and other PM components or source-related PM.

3.5.2.1 Ultrafine Particles

As described in Chapter 2, ultrafine particles generally include particles smaller than 0.1 μm in diameter and are considered nuclei-mode particles. Ultrafine particles are a portion of fine PM; they predominate in the number of particles, but comprise only a small portion of fine PM mass. It has been suggested, based on toxicological evidence, that ultrafine particles may be more toxic than larger particles. It has also been proposed that particle surfaces, or the chemical composition of particle surfaces, may be responsible for PM toxicity, and ultrafine particles have relatively large surface areas (CD, p. 8-68).

The toxicology studies available to date addressing potential effects of ultrafine particles have used PM surrogates or model particles, such as ultrafine carbon or TiO_2 particles. Several new studies are reviewed in the draft CD with somewhat mixed findings on whether greater effects are reported with ultrafine particles than with fine particles. However, in studies using metal oxide dusts, the health response was increased with increasing total surface area, suggesting that particle surface chemistry is an important component of biological responses (CD, p. 8-71). Overall, the draft CD concludes that there is insufficient toxicological evidence to conclude that ambient ultrafine particle concentrations are more strongly linked to health effects than mass concentrations of fine particles (CD, p. 8-85).

A limited number of epidemiological studies, all conducted in European nations, have evaluated health associations with ultrafine particles. One study reported associations between total mortality and both fine particle mass and ultrafine particle number count data, with effects of about the same magnitude reported for each PM size fraction. The authors concluded that both fine and ultrafine particles showed independent effects on mortality at ambient concentrations (Wichmann et al., 2000). Three studies, using panels of asthmatic children or adults, have reported associations between ultrafine particles and increased symptoms or decreased pulmonary function. All reported associations with both ultrafine particle number concentrations and mass concentrations of BS, $PM_{2.5}$ or PM_{10}. In one study, the authors concluded that health effects associations were greater with fine than with ultrafine particles, though significant associations were reported with both (Peters et al., 1997). The authors of the other two studies concluded that separating the effects of different particle size classes was difficult (Pekkanen et al., 1997; Tiittanen et al., 1999), and

Pekkanen et al. (1997) concluded that stronger associations were found with BS or PM_{10} mass than with ultrafine particle counts.

Finally, some new evidence from human exposure studies has indicated that infiltration rates for ultrafine particles into buildings are lower than those for fine (accumulation mode) particles (CD, p. 9-24). This would suggest that community exposure to PM is greater for fine particles than ambient ultrafine particles, and makes it unlikely that health associations found with ambient $PM_{2.5}$ are truly reflecting underlying associations with ultrafine PM. The results of recent epidemiological and toxicological investigations indicate that health effects may be associated with ultrafine particle number or total particle surface area, but the overall findings do not indicate that exposure to ultrafine particles results in greater health responses than PM mass concentrations.

3.5.2.2 Other PM Components, PM Sources

As briefly discussed above, a number of toxicology studies on effects of PM components or surrogates were available during the previous review. In addition, a substantial body of epidemiological studies had evaluated relationships between mortality and morbidity and ambient sulfate or acid aerosol concentrations. The 1996 CD concluded that the epidemiology studies suggest that strongly acidic PM, including sulfates as an indicator of acid aerosols, was associated with both acute and chronic health effects (EPA, 1996a, p. 12-253).

Recent studies have evaluated the effects of not only numerous PM components (e.g., sulfates, nitrates, acids, metals, elemental carbon, biological components), but also PM from different sources (e.g., motor vehicle or industrial emissions, crustal material). Among epidemiological studies that examined the effects of specific components of PM, most commonly used were sulfates and acids, COH, and elemental carbon or organic carbon (as indicators of motor vehicle emissions). Some evidence is reported for associations with components or PM source indicators in community health studies, as outlined below. A larger body of evidence on effects of specific PM components is available from toxicological studies. Regarding the animal toxicology study results, the draft CD concludes that "[t]o date, toxicology studies on PM have provided only very limited evidence for specific PM components being responsible for observed cardiorespiratory effects of ambient PM" (CD, p. 8-83).

As was reported in the previous review, numerous epidemiology studies have indicated that both mortality and morbidity effects are associated with ambient exposures to sulfates and acid

aerosols (H^+). Similarly, associations reported in recent studies between ambient sulfates and mortality are positive and most are statistically significant (CD, figure 6-5). The draft CD concludes that, in these studies, the relative significance of sulfate and H^+ varied from city to city, and the associations were stronger in cities where the sulfate and H^+ levels were relatively high (CD, p. 6-66). Significant associations were reported using sulfates as the PM indicator in the studies of long-term PM exposure and mortality (CD, Tables 6-14 and 6-15). A number of respiratory medical visit studies included assessment of associations with sulfates or acids and also reported significant associations (CD, pp. 6-166 to 6-168).

One new study with exposures to CAPs in dogs reported an association between the sulfur factor of the particles with changes in red blood cell count and hemoglobin levels (Clarke et al., 2000). However, considering the remaining literature from toxicological and controlled human exposure studies using exposure to acid aerosols (CD, Table 8-1), the draft CD concludes that the new studies are consistent with the findings from the previous review, where it was concluded that effects were reported in toxicological or controlled human exposure studies only when levels were very high, although "acid components should not be ruled out as possible mediators of PM health effects" (CD, p. 9-100). One difference between the epidemiological and toxicological studies is that the epidemiological studies were measuring sulfates or acidity of the ambient aerosol, while toxicological studies were using exposures to acid aerosols alone. The draft CD concludes that interactions between different metals and the acidity of PM were reported to influence the severity and kinetics of lung injury induced by ROFA and its soluble transition metals (CD, p. 8-21). This suggests that interaction between some PM components may be an important factor in some health effects associations.

Elemental carbon and organic carbon concentrations were used in studies conducted in Atlanta (Klemm and Mason, 2000) and Phoenix (Mar et al., 2000). Both were significant predictors of mortality in the Phoenix study, but no PM indicators were reported to be significantly associated with mortality in the Atlanta study, possibly due to its small sample size. The draft CD observes that the correlation between COH, elemental carbon and organic carbon and other mobile source related pollutants (fine PM, NO_2, CO) were high, and concludes that the results reported in these analyses suggest that "PM components from mobile sources are likely associated with mortality" (CD, p. 6-65).

The 1996 CD concluded that effects of bioaerosols (e.g., endotoxin) were reported in toxicological or controlled human exposure studies only when levels were very high. The recent toxicological and controlled human exposure studies on the effects of ambient bioaerosols, primarily endotoxins, are summarized in draft CD Table 8-6. These studies of workers exposed in agricultural settings showed respiratory changes, such as reduced lung function or increased airway responsiveness, with increasing dust or endotoxin exposure levels. These occupational study findings were supported by evidence for inflammatory responses in animal or controlled human exposure studies. However, the endotoxin levels measured in these studies were far greater than levels generally reported in ambient air. The draft CD concludes "although these exposures are massive compared to endotoxin levels in ambient PM in U.S. cities, these studies serve to illustrate the effects of endotoxin and associated bioaerosol material in healthy nonsensitized individuals" (CD, p. 8-25). In addition, a number of epidemiology studies have associations of mold spore concentrations on lung function or asthma symptom severity (Delfino et al., 1996, 1997; Neas et al., 1996). In evaluating the results of new epidemiology studies on the association between mortality and coarse fraction particles, the draft CD suggests that the findings of associations in some areas "hint at possible contributions of biogenic materials (e.g., molds, endotoxins, etc.) to the observed coarse particle effects" but sufficient evidence is not yet available to support or refute this hypothesis (CD, p. 9-57).

From toxicological studies, the most substantive new evidence is provided for effects of metals and diesel exhaust particles. For diesel exhaust particles, the draft CD finds growing evidence from toxicology studies that diesel PM exacerbates the allergic response to inhaled antigens, and indications that the organic constituents of diesel PM may contribute to these effects.[7]

Metals, especially water soluble metals, have been reported to cause cell injury and inflammatory changes in toxicology studies, but it is not clear that these effects are found with the small metal concentrations reported in ambient PM (CD, p. 8-85). The transition metals, such as iron, vanadium or nickel, have been most commonly associated with adverse effects in toxicology studies. As summarized by Costa and Dreher (1997), a number of toxicology studies have shown

[7] Evidence from both epidemiological and toxicological studies is evaluated in detail in the draft Diesel Health Assessment Document (EPA, 2000b).

that effects were more closely linked to the metal content of particles than particle mass, though some studies have not found strong associations with particulate metals (e.g., Soukup et al., 2000). Limited evidence is available from epidemiology studies, though one new study reported associations between mortality and particulate iron, nickel and zinc in 8 Canadian Cities (Burnett et al., 2000).

Four new epidemiological studies and one toxicological study have used factor analysis to investigate health associations with PM ($PM_{2.5}$ and PM_{10} or PM_{15}) from different sources (Laden et al., 2000; Mar et al., 2000; Tsai et al., 2000; Ozkaynak et al., 1996; Clarke et al., 2000). These studies used elements or other PM components as indicators of the emissions sources; for example, Laden et al. (2000) use silicon as an indicator for fine particles of crustal or geologic origin (CD, Table 6-5). In addition to testing associations between PM mass and mortality, the four studies evaluated relationships with the PM source factors. The four epidemiology studies are fairly consistent in finding associations for mortality with indicators of PM (both $PM_{10/15}$ and $PM_{2.5}$) from combustion sources, but not from geologic sources (CD, pp. 6-67 to 6-72). The draft CD concludes that the results of the epidemiology studies generally indicate that a "number of combustion-related source-types were associated with mortality, including motor vehicle emissions, coal combustion, oil burning and vegetative burning" (CD, p. 6-78).

In the toxicological study, dogs were exposed to CAPs and numerous indicators of lung injury or inflammation (e.g., white blood cell counts, protein in lung lavage fluid) and cardiovascular health (e.g., platelet and red blood cell counts, hemoglobin or fibrinogen levels) were measured (Clarke et al., 2000). While little evidence was reported for effects with fine PM mass, the authors also conducted factor analysis and identified four PM factors: aluminum/silicon, sulfur, vanadium/nickel, and bromine/lead. The sulfur factor was linked with decreases in red blood cell counts and hemoglobin levels, while the aluminum/silicon and vanadium/nickel factors were linked with inflammatory changes, such as increases in neutrophils or white blood cell counts. The authors conclude that specific components of particles may be responsible for effects, but do not distinguish PM sources that would be linked to each of the PM factors or components.

The effects of PM of crustal or geologic origin were also investigated in two epidemiological studies that used meteorological data in conjunction with air quality data to identify days where wind-blown crustal particles predominate. Both studies reported no evidence of

associations between mortality and wind-blown crustal particles (Schwartz et al., 1999; Pope et al., 1999). In contrast, another study, conducted in Coachella Valley, CA, where coarse particles of geologic origin predominate PM_{10} concentrations, reported significant associations between mortality and PM_{10} (Ostro et al., 1999). Taken together, the draft CD finds that the results of these studies suggest that particles of crustal origin (whether in the fine or coarse fraction of PM) are not likely associated with acute mortality (CD, pp. 6-56 to 6-58). However, the draft CD observes that "crustal" particles may carry biological components (e.g., endotoxin), pesticides or herbicides (as may occur in agricultural situations), or components of emissions from vehicles, smelters, or other industrial operations (CD, p. 6-274). In addition, the existing studies have assessed only mortality as a health endpoint, and there are numerous morbidity indices of potential concern.

These recent studies provide some new evidence for health effects associations with many different PM components such as sulfates, acids and metals. For mortality, the factor analysis studies appear to implicate ambient PM from combustion-related sources in associations with total mortality, but not particles of crustal or geologic origin (CD, p. 9-61). Recognizing that ambient PM exposure has been associated with increases in numerous health indices, the evidence is still too limited to allow identification of which PM components or sources might be more toxic than others, and growing evidence indicates that there are numerous potentially toxic PM components and there may also be interaction occurring between components.

3.5.3 Issues Regarding Interpretation of Epidemiology Studies

The 1996 CD included extensive discussions of methodological issues for epidemiological studies, including questions about model specification or selection, and measurement error in pollutant measurements and exposure error. As summarized in the 1996 Staff Paper, PM-health effects associations reported in epidemiological studies were not likely an artifact of model specification, since analyses or reanalyses of data using different modeling strategies reported similar results (EPA 1996b, p. V-39). In the 1996 CD, less information was available to quantitatively evaluate the potential influence of measurement or exposure error in interpreting epidemiological study findings. A few new publications have explored these questions, and the findings are summarized here. Finally, little information was available for the 1996 CD to allow

comparison of differing lag periods or exposure time windows for PM-related health effects; the recent studies have provided some new information, as discussed below.

3.5.3.1 Lag Periods

Many epidemiological studies on the health effects of acute PM exposure have tested several lag periods, or time delays between the pollution measurement and the occurrence of the health outcome being measured. Commonly used lags are 0 day (effects occurring on the same day as the pollution measurement), 1 to several days, or average pollution measures over several days preceding the health outcome. Often, several lag periods are tested, and the results for the most statistically significant lag period are reported in the publication. As stated in the draft CD, "While this practice may bias the chance of finding a significant association, without a firm biological reason to establish a fixed pre-determined lag, it appears reasonable" (CD, p. 6-238). An alternative approach, the distributed lag, has been introduced in several new studies; the effect of pollution on health is assessed as the effect of a weighted average pollution variable, recognizing that effects of air pollution can occur on several subsequent days.

In the NMMAPS analysis of PM_{10} associations with total mortality, lag periods of 0, 1 and 2 days were used across all cities. The authors reported associations with all three lags, with the largest association being reported for a 1-day lag period. As stated in the draft CD, "since the cardiovascular, respiratory or other causes of acute mortality usually associated with PM are not at all specific, there is little *a priori* reason to believe that they must have the same relation to current or previous PM exposures at different sites" (CD, p. 6-239). In fact, the most significant lag period varied somewhat between NMMAPS study locations, though the range is only from 0-day to 2-day lag periods (draft CD Table 6-24). Several new studies have shown that lag periods may vary for different causes of death; for example, Rossi et al (1999) reported stronger associations between deaths from respiratory infections or heart failure with same-day TSP concentrations, and between myocardial infarction and COPD with TSP lagged 3-4 days (CD, p. 6-232).

For morbidity effects, the findings are similar. The draft CD reports that time series studies of hospital admissions or emergency room visits for cardiovascular diseases suggest that the strongest effects are reported at lag 0, with some effects seen at lag 1 but little beyond a one-day lag (CD, p. 6-137). But in evaluating admissions for specific disease categories, Lippmann et al. (2000) reported the most significant associations between PM_{10} lagged 0 days and pneumonia,

while the "best" lags for heart failure, ischemic heart disease and COPD were 1 day, 2 days and 3 days, respectively. Burnett et al. (2000) also reported significant associations between PM_{10} and dysrhythmia with a 0-day lag, with asthma and heart failure for an average of PM_{10} concentrations over the 0-2 day lags, and with obstructive lung disease at a 2-day lag. In the NMMAPS evaluation of PM_{10} associations with hospital admissions among the elderly, the distributed lag approach was reported to generally result in stronger associations.

In summary, the draft CD states "It may be possible that different PM components may produce effects which appear at different lags or that different preexisting conditions may lead to different delays between exposure and effect. Thus, although maximum effect sizes for PM effects have often been reported for 0-1 day lags, evidence is also beginning to suggest that more consideration should be given to lags of several days . . . higher overall risks may exist than implied by [the] maximum estimated for any particular single or two-day lags." (CD, p. 6-233).

3.5.3.2 Model Specification

The influence of choices made in statistical model specification on the results of epidemiological analyses was examined extensively during the previous NAAQS review. The 1996 CD evaluated the effect of different modeling strategies, and the methods used to adjust for meteorological variables, seasonal or long-term trends, and co-pollutants on the results of epidemiological studies (adjustment for co-pollutants was addressed above in Section 3.5.1). The 1996 CD reported that health associations reported with PM were relatively insensitive to different methods of weather adjustment, and concluded that the results across studies "are not model specific, nor are they artifactually derived due to misspecification of any specific model. The robustness of the results of different modeling strategies and approaches increases our confidence in their validity" (EPA 1996a, p. 13-54).

Among the new studies reviewed in the draft CD are some that use case-crossover methods. The case-crossover study design has only recently been applied in studies of the health effects of air pollutants. This type of study uses the health event (e.g., hospital admission for heart disease) as the case period, and selects a control period from some specific time before or after the event, and assesses whether there are differences in risk factors (air pollutants and other factors) between the periods. The draft CD in Section 6.4.8 presents the findings of three such studies, and all three

studies report associations between PM and mortality that are consistent with the results of the more numerous time-series analyses.

Along with the review of new case-crossover studies, the draft CD also reviews the new evidence on model specification from time-series studies. While identifying some remaining issues needing further study, the draft CD concludes that "[t]hese analyses suggest that the overall findings are not very sensitive to these analytical choices . . ." (CD, p. 6-249).

The draft CD reviews some new studies that evaluate adjustment for factors other than weather or co-pollutants that have been suggested as potential confounders for PM-related effects. One analysis using a subset of NMMAPS data for 5 cities investigated the influence of respiratory epidemics as a potential confounder for PM_{10}-mortality associations. As summarized in the draft CD (p. 6-44), control for respiratory epidemics only reduced the association between PM_{10} and mortality slightly, from 4.3% to 4.0% with a 50 $\mu g/m^3$ increase in PM_{10}, and the association remained statistically significant (Braga et al., 2000). Schwartz (2000b) evaluated PM_{10}-mortality associations among different socio-economic strata (e.g., race, gender, education level, percent nonwhite) and for deaths in-hospital and outside the hospital. The addition of socioeconomic variables to the models did not modify the PM_{10}-mortality effect estimates, but the effect estimate for deaths occurring outside the hospital was substantially greater than the effect estimate for in-hospital deaths. Pollen count was also examined as a potential confounder for respiratory medical visits, and it was reported that pollen levels did not influence the results (CD, p. 6-181).

Methods used in assessing effects associated with long-term exposure to pollutants were also reviewed as a part of the reanalysis of the long-term mortality studies (Krewski et al., 2000). The authors applied an array of different models and variables to determine whether the original results would remain robust to different analytic assumptions and model specifications. The draft CD concludes "None of these alternative models produced results that materially altered the original findings" (CD, p. 6-83).

3.5.3.3 Measurement Error

In this and previous reviews of the PM NAAQS, much of the health evidence for PM-related effects comes from epidemiological studies where ambient PM measurements are used to represent community PM exposures. One key issue is the use of PM concentrations measured at central locations to represent the community's exposure to ambient PM. As discussed in Section

2.8 above, daily changes in individuals' personal exposure to ambient PM is well correlated with daily changes in ambient PM measured as central monitors. Thus, the draft CD concludes that ambient PM concentrations are a useful surrogate for exposure to ambient PM (CD, p. 9-86).

Another key issue in interpreting epidemiology study findings is related to error in the measurements of the pollutants. Analyses available for the 1996 Staff Paper indicated that random measurement error in pollutant concentration data is not likely to bias the findings of epidemiologic analyses using these data. However, a remaining question was the existence of differential measurement error, where one pollutant was measured with more error than another, and the effect this might have in comparing epidemiologic findings for the two pollutants (EPA, 1996b, p. V-42).

The draft CD summarizes the findings of several new analyses that show the potential influence of differential measurement error on epidemiological analysis results, though the conditions required for the error to substantially influence the epidemiological findings are severe and unlikely to exist in current studies. In simulation analyses of a "causal" pollutant and a "confounder" with differing degrees of measurement error and collinearity between the pollutants it was found that, in some circumstances, a causal variable measured with error may be overlooked and its significance transferred to a surrogate. However, for "transfer of apparent causality" from the causal pollutant to the confounder to occur, there must be high levels of both measurement error in the causal variable and collinearity between the two variables (Zidak et al., 1996; Zeger et al., 1999; Fung and Krewski, 1999). An additional analysis applied measurement error models to data from the Harvard Six Cities study, specifically testing relationships between mortality and either fine or coarse fraction particles. The authors identified several variables that could influence bias in effects estimates for fine- or coarse-fraction particles: the true correlation of fine- and coarse-fraction particles, measurement errors for both, and the underlying true ratio of the toxicity of fine- and coarse-fraction particles. The existence of measurement error and collinearity between pollutants could result in underestimation of the effects of the less well-measured pollutant. However, the authors conclude "it is inadequate to state that differences in measurement error among fine and coarse particles will lead to false negative findings for coarse particles. If the underlying true ratio of the fine and coarse particle toxicities is large (i.e., greater than 3:1), fine particle exposure must be measured significantly more precisely in order not to *underestimate* the ratio of fine particle toxicity versus coarse particle toxicity" (Carrothers and Evans, 2000, p. 72).

Thus, while the potential remains for differential error in pollutant measurements to influence the results of epidemiological studies, it is unlikely that the levels of measurement error and correlation between pollutants reported in existing studies would result in transfer of apparent causality from one pollutant to another.

The influence of exposure misclassification on the results of epidemiological analyses has been further investigated in one major new analysis that was conducted as a part of NMMAPS (Zeger et al., 2000). Using data collected in previous exposure studies, the authors developed a relationship between personal exposure to ambient particles and ambient PM_{10} concentrations. The authors reported that the association between PM_{10} and mortality using ambient PM_{10} concentrations underestimated the association between personal ambient PM_{10} exposure and mortality.

In reviewing these new studies, along with analyses that were available in previous reviews, the draft CD concludes "the studies that examined joint effects of correlation and error suggest that PM effects are likely underestimated, and the spurious PM effects (i.e., qualitative bias such as change in the sign of the coefficient) due to transferring of effects from other covariates require extreme conditions and are, therefore, unlikely." (CD, p. 6-245)

3.5.3.4 Exposure Time Periods for Acute Effects

In the previous PM NAAQS review, epidemiological studies on acute effects of PM exposure primarily used 24-hour average PM concentrations. The newly available epidemiological studies include several where 1-hour or 8-hour average ambient PM concentrations are used in time-series analyses, and some evidence is from panel studies of cardiac patients with average PM concentrations of one to several hours. Toxicology or controlled human exposure studies often use shorter exposure time periods, and a new body of evidence is available from studies using inhalation exposures to ambient particles, including one study of controlled human exposures to CAPs.

As discussed earlier, one controlled human exposure study included exposure to concentrated ambient $PM_{2.5}$ for 2 hours, and reported mild increases in neutrophils in bronchoalveolar lavage samples and increased blood fibrinogen levels after the exposure period (Ghio et al., 2000) . Animal toxicology studies have used inhalation exposures to CAPs or PM surrogates with exposure time periods generally in the range of 1 to 6 hours per day, sometimes for several days (CD, Tables 8-3 and 8-7). A range of effects have been reported in these animal

studies, including evidence for respiratory effects such as lung injury and inflammation and cardiovascular effects such as arrhythmia. Based on the findings of these studies, it is apparent that acute exposure to PM of a few hours' duration can result in physiological or cellular changes.

Several recent epidemiology studies have reported findings for PM averaged over 24 hours and shorter time periods (1-hour and 8-hour) that do not show substantial differences in effects reported for different averaging times. These studies have used data from continuous PM monitors, such as the TEOM or nephelometer (see Chapter 2 for details on monitoring methods), and evaluated associations with total mortality, hospital admissions, heart rate variability and respiratory symptoms. Some studies have reported larger effect estimates for one- or several-hour concentrations than for 24-hour average concentrations, e.g.,1-hour and 8-hour PM_{10} with respiratory symptoms in California (Delfino et al., 1998) and heart rate variability changes with 4-hour $PM_{2.5}$ levels in Boston (Gold et al., 2000). In contrast, larger effect estimate sizes were reported for associations between total mortality and 24-hour $PM_{2.5}$ levels than 1-hour levels in Melbourne and Brisbane, Australia (Simpson et al., 1997, 2000). In two other Australian studies, similar effects were reported for 1-hour and 24-hour $PM_{2.5}$ levels with total mortality in Melbourne (Morgan et al., 1998) and hospital admissions for respiratory disease in Sydney (Morgan et al., 1997).

Thus, the results of the recent epidemiology studies time do not provide substantive evidence that mortality or morbidity are more strongly associated with one short-term exposure interval than another. The results of controlled human exposure and animal toxicology provide some evidence that health effects can be result from PM exposures of a few hours' duration; in fact, it is logical to expect that some health effects would be nearly instantaneous while others might require a longer duration of exposure.

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4. CHARACTERIZATION OF HEALTH RISKS

4.1 INTRODUCTION

This chapter briefly summarizes the PM risk analyses conducted for two urban study areas (Philadelphia and Los Angeles counties) during the previous review of the PM NAAQS and describes the proposed scope of EPA's updated risk analyses to be conducted for the current review of the standards. The updated risk analyses will focus on the risks of mortality, morbidity, and symptoms associated with recent ambient air quality levels and just attaining the current suite of $PM_{2.5}$ NAAQS and any other alternative $PM_{2.5}$ standards that may be identified as appropriate for consideration during the course of the current review of the PM NAAQS. EPA also is considering the appropriateness of conducting risk analyses for respiratory-related hospital admissions and respiratory symptoms associated with coarse-fraction PM (i.e., $PM_{10-2.5}$) for recent air quality levels and upon just meeting potential $PM_{10-2.5}$ standards. Results from the updated risk analyses will be presented in the next draft of this Staff Paper. As discussed in Chapters 2, the fact that the sources and composition of $PM_{2.5}$ and $PM_{10-2.5}$ are largely distinct, along with the new health effects evidence discussed in Chapter 3, supports the recommendation from the previous Staff Paper that fine-and coarse-fraction particles be considered as separate pollutants. At that time, a number of health studies indicated differences in health effects between fine-and coarse-fraction particles, and suggested that serious health effects, such as premature mortality, were more closely associated with fine-fraction particles. The new studies, summarized in the draft CD (CD, Chapter 6), continue to show associations between serious health effects, including premature mortality, and fine-fraction PM, but they also offer new evidence indicating possible associations between coarse-fraction PM and health effects. For coarse-fraction particles the strongest evidence is found relating $PM_{10-2.5}$ ambient concentrations and increased respiratory hospital admissions and respiratory symptoms.

4.1.1 Goals for Updated PM Risk Analyses

The goals of the updated PM risk analyses are: (1) to provide a rough sense of the potential magnitude of PM-associated mortality and morbidity associated with current $PM_{2.5}$

levels and with attaining the current suite of $PM_{2.5}$ NAAQS (as well as any potential alternative $PM_{2.5}$ standards identified as part of this review); (2) to provide a rough sense of the potential magnitude of PM-associated morbidity associated with current $PM_{10-2.5}$ levels and with attaining possible alternative $PM_{10-2.5}$ NAAQS (if the decision is made that there is sufficient evidence to warrant conducting a risk analysis for coarse-fraction PM); (3) to develop a better understanding of the influence of various inputs and assumptions on the risk estimates; and (4) to gain qualitative insights into the nature of the risks associated with exposure to PM. The staff recognizes that due to the many sources of uncertainty inherent in conducting PM risk analyses, any PM risk estimates presented in the next draft Staff Paper should not be interpreted as demonstrated health impacts or precise measures of risk. Further, the staff recognizes the limited role of the risk analyses in this standards review and do not plan to use the risk estimates as a principal basis for recommending selection among alternative standard levels.

4.1.2 Summary of Risk Analyses Conducted During Prior PM NAAQS Review

For the prior review, EPA conducted a number of risk analyses that estimated population risk for two defined urban study areas (i.e., Philadelphia and Los Angeles counties). The PM health risk model combined information about daily PM air quality for these two study areas with estimated concentration-response functions derived from epidemiological studies and baseline health incidence data for specific health endpoints to derive estimates of the annual incidence of specific health effects occurring under "as is" air quality. Since site-specific relative risks were not available for all endpoints in both locations (and in the absence of more information concerning which individual studies might best characterize the health risk in a given location), a form of meta analysis (referred to as a "pooled analysis") was conducted which combined the results of the studies that met specified criteria. The analyses also examined the reduction in estimated incidence that would result upon just attaining the existing PM_{10} standards and several sets of alternative $PM_{2.5}$ standards. The methodological approach followed in conducting the prior risk analyses is described in Section 6 of the 1996 Staff Paper (EPA, 1996b) and in several technical reports (Abt Associates, 1996; Abt Associates, 1997a,b) and articles (Post et al., 2000; Deck et al., 2001).

Summarized below are the key observations resulting from the prior risk analyses which were most pertinent to the decision on the PM NAAQS, as well as several important caveats and limitations associated with these analyses:

- EPA placed greater weight on the overall conclusions derived from the health effect studies – that PM air pollution is likely causing or contributing to significant adverse effects at levels below those permitted by the existing PM_{10} standards – than on the specific concentration-response functions and quantitative risk estimates derived from them. The quantitative risk estimates included significant uncertainty and, therefore, were not viewed as demonstrated health impacts. Nevertheless, EPA did state that it believed the analyses presented reasonable estimates as to the possible extent of risk for these effects given the available information (62 FR 38656).

- Consideration of key uncertainties and alternative assumptions resulted in fairly wide ranges in estimates of the incidence of PM-related mortality and morbidity effects and risk reductions associated with attainment of alternative standards in both locations in the risk analyses. Significantly, the combined results for these two cities alone found that the risk remaining after attaining the current PM_{10} standards was on the order of hundreds of premature deaths each year, hundreds to thousands of respiratory-related hospital admissions, and tens of thousands of additional respiratory-related symptoms in children (62 FR 38656).

- Based on the results from the sensitivity analyses of key uncertainties and the integrated uncertainty analyses, the single most important factor influencing the uncertainty associated with the risk estimates was whether or not a threshold concentration exists below which PM-associated health risks are not likely to occur (62 FR 38656).

- Over the course of a year, the few peak 24-hour $PM_{2.5}$ concentrations appeared to contribute a relatively small amount to the total health risk posed by the entire air quality distribution as compared to the aggregated risks associated with the low to mid-range $PM_{2.5}$ concentrations (62 FR 38656).

- There was greater uncertainty about both the existence and the magnitude of estimated excess mortality and other effects associated with $PM_{2.5}$ exposures as one considered lower concentrations that approach background levels (62 FR 38656).

- Based on the results from the sensitivity analyses of key uncertainties and/or the integrated uncertainty analyses, the following uncertainties had a much more modest impact on the risk estimates: inclusion of individual copollutant species when estimating PM effect sizes; the choice of approach to adjusting the slope in analyzing alternative cutpoints; the value chosen to represent average annual background PM concentrations; and the choice of rollback adjustment approaches for simulating attainment of alternative $PM_{2.5}$ standards (EPA, 1996b).

4.2 GENERAL SCOPE OF PLANNED PM RISK ANALYSES

As discussed in Chapter 3 above, the draft CD (CD, p. 9-40) finds that "[t]he newer experimental evidence, therefore, adds considerable support for interpreting the epidemiologic findings . . . as being indicative of causal relationships between exposures to ambient PM and consequent associated increased morbidity and mortality risks." The risk analyses planned for this NAAQS review are premised on the assumption that $PM_{2.5}$ is causally related to the mortality, morbidity, and symptomatic effects (alone and/or in combination with other pollutants) observed in the epidemiological studies. Since the last review, additional studies have been published which strengthen the basis for concern about mortality and morbidity health endpoints being related to ambient $PM_{2.5}$ exposures. Therefore, EPA plans to conduct risk analyses for $PM_{2.5}$ and several health endpoints, including mortality, hospital admissions, and respiratory symptoms. In addition, there is a growing, but limited data base reporting health effects associated with coarse-fraction PM and which uses $PM_{10-2.5}$ as the air quality indicator. The strongest evidence indicating potential health effects associated with coarse-fraction PM is for respiratory-related hospital admissions and respiratory symptoms. Currently, EPA is considering whether to conduct risk analyses for $PM_{10-2.5}$ for these two categories of effects.

The staff welcomes CASAC and public input on (1) the relevant health studies to include in the $PM_{2.5}$ risk analysis, (2) whether or not to conduct a limited coarse-fraction risk analysis, and (3) if a coarse-fraction risk analysis is conducted, which health endpoints and studies should be considered. The discussion below includes information on studies and concentration-response functions for both $PM_{2.5}$ and $PM_{10-2.5}$ to help inform a decision on whether to proceed with a limited coarse-fraction risk analysis focused on respiratory-related hospital admissions and respiratory symptoms. Similarly, air quality information on $PM_{10-2.5}$ for possible urban counties that could be selected for such analyses also are included in this chapter.

The planned $PM_{2.5}$ risk analyses will focus on selected health endpoints such as increased daily mortality, increased hospital admissions for respiratory and cardiopulmonary causes, and increased respiratory symptoms for children. A consequence of limiting the analyses to selected health endpoints is that the risk estimates may understate the type and extent of potential health

impacts of PM exposures. Although the risk analyses will not address all health effects for which there is some evidence of association with exposure to PM, all such effects are identified and considered in Chapter 3.

The risk assessment to be conducted as part of this review, like the prior risk assessment done as part of the last review (EPA, 1996b), will use concentration-response functions from epidemiological studies based on ambient PM concentrations measured at fixed-site, population-oriented, ambient monitors. As discussed earlier in Chapter 2 (Section 2.8), measurements of daily variations of ambient $PM_{2.5}$ concentrations, as used in the time-series studies that provide the concentration-response relationships for these analyses, have a plausible linkage to the daily variations of exposure from ambient sources for the populations represented by ambient monitoring stations. The draft CD concludes that this linkage is better for indicators of fine particles (e.g., $PM_{2.5}$) and PM_{10} but that this may not be the case for $PM_{10-2.5}$, for specific chemical components, for source contributions, or for sites located near sources (CD, p. 9-24). A more detailed discussion of the possible impact of exposure misclassification on the estimated concentration-response relationships derived from the community epidemiological studies is presented above in Chapter 3 (see Section 3.5.3.3).

While quantitative estimates of personal or population exposure do not enter into derivations of the risk estimates, an understanding of the nature of the relationships between ambient PM and its various components and human exposure underlies the conceptual basis for the risk assessment. Unlike recent reviews for ozone and carbon monoxide, where exposure analyses played an important role, a quantitative exposure analysis will not be conducted as part of this review since the currently available epidemiology health effects evidence relates ambient PM concentrations, not exposures, to health effects. As discussed in Chapter 4 of the draft CD, EPA and the exposure analysis community are working to improve exposure models designed specifically to address PM. Both EPA and the broader scientific community also are in the process of collecting new information in PM exposure measurement field studies that will improve the scientific basis for exposure analyses that may be considered in future reviews.

While the NAAQS are intended to provide protection from exposure to ambient PM, EPA recognizes that exposures to PM from other sources (i.e., non-ambient PM) also have the

potential to affect health. The EPA's Office of Radiation and Indoor Air and other Federal Agencies, such as the Consumer Product Safety Commission (CPSC) and the Occupational Safety and Health Administration (OSHA), address potential health effects related to indoor, occupational, environmental tobacco smoke, and other non-ambient sources of PM exposure. Like the prior risk analysis, contributions to health risk from non-ambient sources are beyond the scope of the proposed risk analyses for the NAAQS review.

This proposed PM health risk analysis is similar in many respects to the prior risk analysis conducted for the last PM NAAQS review. Both the prior and the current proposed PM risk analyses:

- estimate risks for the urban centers of example cities, rather than attempt a nationwide analysis.

- analyze risks under a recent 12-month period of air quality (labeled "as is") and under a situation where air quality just attains the current set of standards. (The risk analyses also will include any potential alternative $PM_{2.5}$ and $PM_{10-2.5}$ standards that are identified as part of this review).

- estimate risks only for concentrations exceeding estimated background levels.

- present qualitative and quantitative considerations of uncertainty, including sensitivity analyses of key individual uncertainties and integrated sensitivity analyses combining key parameters.

Both the prior and the current planned PM risk analyses focus on health endpoints for which concentration-response functions have been estimated in epidemiological studies. Since these studies estimate concentration-response functions using air quality from fixed-site, population-oriented monitors, the appropriate application of these functions in a PM risk analysis similarly requires the use of air quality data from fixed-site, population-oriented, ambient monitors. This is identical to the approach taken in the last PM NAAQS review.

The scope of the planned $PM_{2.5}$ risk analyses is to develop risk estimates for at least two selected urban areas: Philadelphia County, and a portion (roughly the southeastern third) of Los Angeles County (hereafter referred to as "Los Angeles County"). The staff is soliciting comment on whether it should also include Salt Lake County in the $PM_{2.5}$ risk analyses, if it proceeds to conduct a coarse fraction PM analysis for this county. The scope of the potential $PM_{10-2.5}$ risk

analyses is to develop risk estimates for Los Angeles County and Salt Lake County. These areas have been chosen based on availability of $PM_{2.5}$ and $PM_{10-2.5}$ air quality data. There also is a desire to include areas from the eastern and western parts of the United States to reflect regional differences in the composition of $PM_{2..5}$. Because elevated $PM_{10-2.5}$ levels are primarily a problem in the western parts of the United States and because of the lack of eastern sites with adequate $PM_{10-2.5}$ data, EPA is considering conducting the potential coarse-fraction risk analyses only in the two western areas (i.e., Salt Lake County and Los Angeles County). Finally, estimates of risks above background PM concentrations are judged to be more relevant to policy decisions about the NAAQS than estimates that include risks potentially attributable to uncontrollable background PM concentrations.

The following sections summarize the planned scope of the risk analyses and key components of the risk model. A separate draft "Scoping Plan" (EPA, 2001c) is also available which provides a more detailed discussion. EPA plans to include and discuss the results from the risk analyses in the next draft of this Staff Paper.

4.2.1 Overview of Components of the Risk Model

In order to estimate the incidence of a particular health effect associated with "as is" conditions in a specific county attributable to ambient $PM_{2.5}$ or $PM_{10-2.5}$ exposures and the change in incidence of the health effect in that county corresponding to a given change in $PM_{2.5}$ and $PM_{10-2.5}$ levels resulting from just attaining a specified set of $PM_{2.5}$ and $PM_{10-2.5}$ standards, the following three elements are required:

- air quality information including: (1) "as is" air quality data for $PM_{2.5}$ and $PM_{10-2.5}$ from population-oriented monitors for the selected county, (2) estimates of background $PM_{2.5}$ and $PM_{10-2.5}$ concentrations appropriate for that location, and (3) a method for adjusting the "as is" data to reflect patterns of air quality estimated to occur when the county attains a given set of standards.

- relative-risk basedconcentration-response functions which provide an estimate of the relationship between the health endpoints of interest and ambient $PM_{2.5}$ and $PM_{10-2.5}$ concentrations.

- baseline health effects incidence or incidence rates which provide an estimate of the incidence or incidence rate of health effects corresponding to "as is" $PM_{2.5}$ and $PM_{10-2.5}$ levels.

Figure 4-1 provides a broad schematic depicting the role of these components in the risk analyses. Those points where EPA proposes to conduct analyses of alternative assumptions, procedures, or data are indicated by a circle with S_x in it. A fuller description of the type of sensitivity analyses planned is included in Table 4-1.

Most epidemiological studies estimating relationships between PM and health effects assume an exponential concentration-response function.[1] In this model,

$$y = B\, e^{\beta x} \quad , \qquad \text{(Equation 4-1)}$$

where x is the ambient PM level, y is the incidence of the health endpoint of interest at PM level x, β is the coefficient of ambient PM concentration, and B is the incidence at x=0, i.e., when there is no ambient PM. The change in health effects incidence from the baseline incidence, y (the incidence at "as is" PM concentration, x) to y_0 (the incidence at PM concentration x_0, attaining the alternative standards) corresponding to a given change in ambient PM levels, $\Delta x = x_0 - x$, is then

$$\Delta y = y[e^{\beta \Delta x} - 1] \qquad \text{(Equation 4-2)}$$

or, alternatively,

$$\Delta y = y(RR_{\Delta x} - 1) \qquad \text{(Equation 4-3)}$$

where $RR_{\Delta x}$ is the relative risk associated with the change in ambient PM levels, Δx.

[1]For some studies on respiratory hospital admissions used in the risk analysis a linear concentration-response function was estimated.

Figure 1. Major Components of Particulate Matter Health Risk Analysis

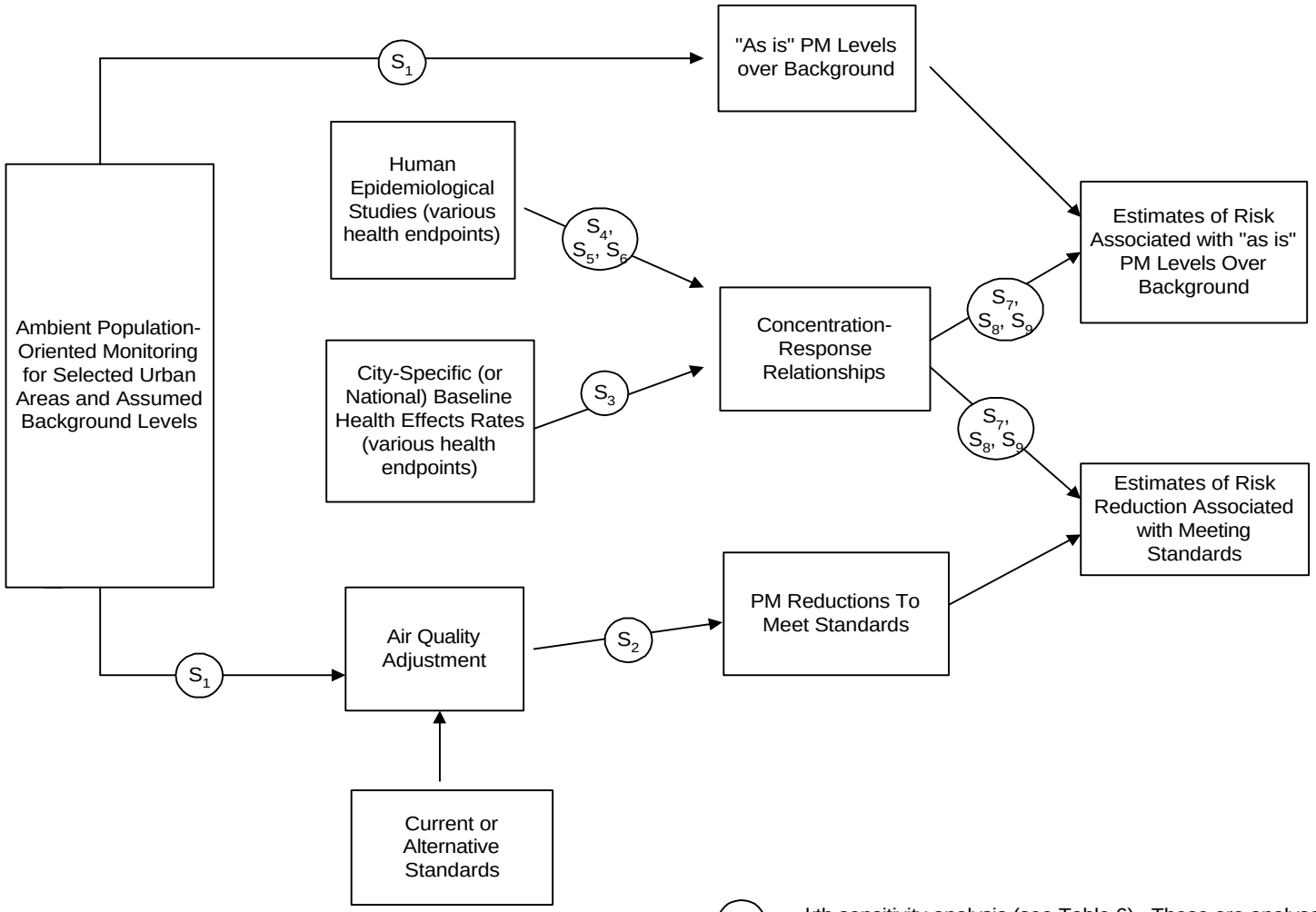

Table 4-1. Planned Sensitivity Analyses

Analysis Number (Figure 1)	Component of the Risk Analysis	Sensitivity Analysis or Comparison
1	Air Quality	A sensitivity analysis of the effect of different assumptions about background PM levels
2	Air Quality	A sensitivity analysis of the effect of different air quality adjustment procedures on the estimated risk reductions resulting from just meeting alternative 24-hr and annual standards
3	Baseline Incidence	A comparison of using more aggregate incidence data (national, state, etc) versus county-specific information in the county with the best local incidence data
4	Concentration-Response	A comparison or sensitivity analysis of methods of combining averaging times of from 1 to 5 days in the short-term mortality and hospital admissions studies
5	Concentration-Response	A sensitivity analysis or comparison of the effects of including or excluding individual studies from pooled functions to show the sensitivity of the function to inclusion of specific studies
6	Concentration-Response	A comparison or sensitivity analysis of the impact on mortality associated with long-term exposure of different assumptions about the role of historical air quality concentrations in contributing to the reported effects.
7	Concentration-Response	A sensitivity analysis comparing the risks estimated by using concentration-response functions derived for the specific county in question versus pooled functions for endpoints
8	Concentration-Response	A sensitivity analysis using concentration-response functions for PM from multi-pollutant regressions with co-pollutants versus single pollutant regressions
9	Concentration-Response	A sensitivity analysis assuming alternative minimum concentration levels for the occurrence of PM response at concentrations above those for background

Estimates of risk (i.e., incidences or incidence rates of health effects attributable to $PM_{2.5}$ or $PM_{10-2.5}$) will be quantified for $PM_{2.5}$ and $PM_{10-2.5}$ concentrations above background except for those studies in which the background concentration was not within the range of observable $PM_{2.5}$ or $PM_{10-2.5}$ concentrations used for the study (e.g., the prospective cohort mortality studies). For studies that do not evaluate risk at background levels, the effects will be quantified only down to the lowest concentrations observed in the study. Each of these key components is discussed below, highlighting those points at which judgments have been made that will determine the nature and scope of the risk analysis.

4.2.2 Air Quality Considerations

The air quality information required to conduct the PM risk analyses includes: (1) "as is" air quality data for both $PM_{2.5}$ and $PM_{10-2.5}$ from population-oriented monitors for the selected cities, (2) estimates of background $PM_{2.5}$ and $PM_{10-2.5}$ concentrations appropriate to each location, and (3) a method for adjusting the "as is" data to reflect patterns of air quality change estimated to occur when each location attains the current suite of $PM_{2.5}$ standards (as well as any potential alternative $PM_{2.5}$ standards identified as part of this review) or alternative $PM_{10-2.5}$ standards. Table 4-2 provides a summary of the $PM_{2.5}$ and $PM_{10-2.5}$ air quality data for the areas under consideration for inclusion in the risk analyses. The $PM_{10-2.5}$ observations are based on subtracting $PM_{2.5}$ concentrations from the PM_{10} concentration at a co-located monitoring site. Additional discussion of the available PM air quality data for these three locations is presented in the draft Scoping Plan (EPA, 2001c).

Table 4-2. Summary of PM Air Quality Data for Areas to Be Examined in PM Risk Analyses

Area	Population (millions)	Year	Number (%) of Days on Which Air Quality Data are Available		$PM_{2.5}$[b]		$PM_{10-2.5}$[b]	
			$PM_{2.5}$	$PM_{10-2.5}$	Annual Avg. ($\mu g/m^3$)	98^{th} percentile 24-hr Avg.[a] ($\mu g/m^3$)	Annual Avg. ($\mu g/m3$)	98^{th} Percentile 24-hr Avg.[b] ($\mu g/m^3$)
Philadelphia County, PA	1.4	1999	276 (75.6)	-	14.8	35.9	-	-
Los Angeles County, CA[c]	3.8	1998/1999	197 (54.0)	130 (35.6)	24.2	59.5	26.2	54
Salt Lake County, UT	0.85	1999	315 (86.0)	285 (78.0)	9.9	47	15.8	44

[a]The values shown in this column are the 98^{th} percentile values at the "composite monitors" in Philadelphia and Los Angeles. The actual risk analyses will be based on the current form of the standard which requires the 98^{th} percentile value at each monitor not exceed the standard.

[b]The value shown for Los Angeles is the 98^{th} percentile value at the "composite monitor", while the 98^{th} percentile value for Salt Lake County is the 98^{th} percentile value at a specific monitor.

[c]The information in this row is for Southeast Los Angeles County which makes up a little over a third of Los Angeles County.

Background PM concentrations proposed to be used in the risk analyses are defined in Chapter 2 of this Staff Paper as the distribution of PM concentrations that would be observed in the U.S. in the absence of anthropogenic emissions of PM and its precursors in North America. For the proposed risk analyses, an estimate of the annual average background level is desired, rather than a daily average (e.g., the maximum 24-hour level), since accumulated risks will be aggregated for each day throughout the year. The staff have chosen to use the midpoint of the appropriate ranges of annual average estimates for PM background presented in Chapter 2 for the base case risk estimates (i.e., eastern values will be used for Philadelphia County and western values will be used for Los Angeles and Salt Lake Counties).

- For $PM_{2.5}$: 2 to 5 $\mu g/m^3$ for Philadelphia and 1 to 4 $\mu g/m^3$ for Los Angeles and Salt Lake Counties

- For $PM_{10-2.5}$: 3 to 4 $\mu g/m^3$ for Los Angeles and Salt Lake Counties

Sensitivity analyses will be done using the appropriate lower and upper ends of the above ranges to characterize the impact of this model input choice on the risk estimates. OAQPS also recognizes that the estimated ranges for regional background levels of $PM_{10-2.5}$ due to natural sources and transport from outside of North America are more uncertain than the estimates for $PM_{2.5}$.

To estimate the health risks associated with just attaining the current $PM_{2.5}$ standards and alternative $PM_{10-2.5}$ standards, it is necessary to estimate PM concentrations that would occur under each specified standard (or sets of standards). When assessing the risks associated with long-term exposures, using epidemiological studies that use an annual average concentration, the annual mean is simply set equal to the standard level. In contrast, when assessing the risks associated with short-term exposures using epidemiological studies which consider daily average concentrations, the distribution of 24-hour values that would occur upon just attaining a given 24-hour PM standard has to be simulated. While there are many different methods of reducing daily PM levels, prior analyses conducted during the last NAAQS review found that PM levels have in general historically decreased in a proportional manner (i.e., concentrations at different points in the distribution of 24-hour PM values have decreased by approximately the same percentage) (Abt Associates, 1996b). Therefore, attainment of the current $PM_{2.5}$ daily standard and alternative daily $PM_{10-2.5}$ standards will be simulated by adjusting the "as is" air quality data using a proportional rollback approach (i.e., concentrations across the distribution are reduced by the same percentage) for concentrations exceeding the estimated background level. Sensitivity analyses will be conducted to examine alternative air quality adjustment procedures (e.g., a method that reduces the top 10% of daily PM concentrations more than the lower 90%).

4.2.3 Estimating Concentration-Response Functions

The second key component in the risk model is the set of concentration-response functions which provide estimates of the relationship between each health endpoint of interest and ambient

PM concentrations. The staff has selected the most significant health effect endpoints for which the weight of the evidence is supportive of an effect occurring. In cases where all of the available studies failed to find a statistically significant relationship, the effect endpoint was excluded. In situations where there is a mixture of statistically significant and non-significant findings for a given health effect endpoint and PM indicator (e.g., hospital admisisons for COPD patients and $PM_{2.5}$), staff also considered evidence from available PM_{10} studies in making a judgment on whether effects are likely related to PM.

The health endpoints that are proposed to be included in the $PM_{2.5}$ analyses include mortality (due to short- and long-term exposure), hospital admissions, emergency room visits, and respiratory illnesses and/or symptoms not requiring hospitalization. (Lung function studies will not be included.) Inclusion of a health endpoint in the analysis will be based on the weight of the evidence overall. Once it has been determined that a health endpoint will be included in the analysis, inclusion of a study on that health endpoint will not be based on the existence of a statistically significant result. That is, consistent with the approach taken in the prior PM risk analyses, no credible study on an included health endpoint will be excluded from the analysis on the basis of lack of statistically significant findings.

For the potential $PM_{10-2.5}$ risk analyses, EPA is considering including increased respiratory-related hospital admissions and increased respiratory symptoms as health endpoints. As discussed in Chapter 3 of this Staff Paper, these are the two health effect categories with the strongest evidence for effects being associated with $PM_{10-2.5}$ exposure. While there is evidence for other effects being associated with $PM_{10-2.5}$, the staff believes that the evidence is insufficient to justify conducting a quantitative risk analysis for other health endpoints. These other effects are addressed qualitatively in Chapter 3 of this Staff Paper.

Since the 1996 PM risk analyses were carried out, several new studies have investigated the relationship between PM and a health endpoint (e.g., short-term exposure mortality) in multiple cities using consistent methodological approaches in all locations examined. As noted in the draft CD (see, in particular, CD, Section 9.6.2.1.2), such multi-location studies are preferable, all else equal, to meta-analyses (i.e., pooling) of the results of multiple independent single-location studies carried out in different locations. The primary advantage of such multi-location studies is

the consistency in methodology used in all locations, eliminating the possibility that inter-locational differences might be due to differences in study design. In addition, multi-location studies are not subject to the omission of negative results due to publication bias that could affect a meta-analysis of the results of published single-location studies. Finally, any geographical variability in air pollution effects can be systematically evaluated in a multi-location study. For these reasons, such multi-location studies, if available, are preferred to meta-analyses of independent single-location studies.

Consistent with the approach taken in the prior PM risk analyses, if there is no multi-location study for a health endpoint, and if several single-location studies have been identified as appropriate for inclusion in the PM risk analyses, EPA proposes to combine the C-R functions from these studies to form a "pooled" estimate of the risk of that health effect attributable to $PM_{2.5}$ (or $PM_{10-2.5}$) and the risk reductions that would result from meeting current or alternative standards. The relationship between a pollutant and a health effect in a population may vary from one location to another due, for instance, to inter-locational differences in the composition of PM and/or the populations exposed. Pooling the estimates from several studies provides a central tendency estimate of the effect in any randomly selected location, as well as a characterization of the uncertainty about the effect in that location. The staff recognizes that caution is required in deciding which studies should be pooled for any given health endpoint and the draft Scoping Plan (EPA, 2001c) addresses in more detail the proposed principles that would be followed in selecting studies to be pooled.

In selecting studies to be considered for use in the PM risk analyses, the staff set forth several criteria, all of which have to be met to be included for consideration for the proposed risk analyses for this review. These include: (1) only studies cited in the draft CD tables (see CD, Tables 9-3, 9-4, and 9-6) or included in the prior 1996 risk analyses are included, (2) only studies conducted in the United States or Canada are included, (3) only studies that measured $PM_{2.5}$ (or $PM_{2.1}$) and/or $PM_{10-2.5}$ are included, and (4) only studies that are judged to be credible from a methodological standpoint are included. The staff recognizes that the draft CD is currently under review by both the CASAC and general public, and, thus, the final group of studies to be included in the analyses may change based on the review of the draft CD. Table 4-3 summarizes the

available epidemiological studies cited in the draft CD that may be useful in estimating total non-accidental and cause-specific mortality associated with short-term $PM_{2.5}$ exposures. Table 4-4 summarizes the available epidemiological studies cited in the draft CD that may be useful in estimating total and specific kinds of cardiovascular morbidity effects associated with $PM_{2.5}$ exposures. Table 4-5 summarizes the available epidemiological studies cited in the draft CD that may be useful in estimating total and specific kinds of respiratory morbidity effects associated with both $PM_{2.5}$ and $PM_{10-2.5}$ exposures.

In assessing or interpreting public health risk associated with exposure to PM, the form of the concentration-response function is an important component. The 1996 Criteria Document (EPA, 1996a) evaluated evidence from epidemiological studies regarding both functional form and whether a threshold for effects could be identified; this evaluation raised some key questions, but there was not sufficient evidence to draw conclusions (EPA, 1996a, Section 13.6.5).

Among the new epidemiological analyses are several studies that use different modeling methods to investigate potential threshold levels and concentration-response forms. As summarized in the draft CD, two of these studies presented no evidence of the existence of a threshold for associations between PM and acute mortality. Cakmak et al. (1999) tested different methods for detecting the presence of a threshold for the PM-mortality relationship, using Toronto pollution and mortality data. The authors concluded that "if threshold exists, it is highly unlikely that standard statistical analysis can detect it." (CD, p. 6-246). Similarly, Schwartz and Zanobetti (2000) used simulation methods with air quality data from 10 U.S. cities to investigate the presence of a threshold. No evidence was found for the existence of a threshold in the association between PM_{10} and short-term exposure mortality (CD, pp. 6-246, 247).

In addition, using data from 20 U.S. cities to analyze the PM_{10} and short-term exposure mortality relationship, roughly linear associations were found for total and cardiorespiratory mortality, consistent with the lack of a threshold.(CD, p. 6-238; Daniels et al., 2000). Some evidence for thresholds in the relationship between $PM_{2.5}$, but not $PM_{10-2.5}$, and mortality was found using data from Phoenix. Smith et al. (2000) found evidence suggesting a potential threshold level of 20-25 $\mu g/m^3$ for mortality associations with $PM_{2.5}$ but no evidence of a threshold in the relationship between $PM_{10-2.5}$ and mortality. The draft CD (CD, p. 6-247)

observes that the data set used in this analysis is small but the findings warrant further analysis. Overall, considering the results of these new studies, the draft CD concludes that "linear models without a threshold may well be appropriate for estimating the effects of PM_{10} on . . . mortality" (CD, p. 6-248), which is consistent with the conclusions of the previous Criteria Document (EPA, 1996a).

4.2.4 Baseline Health Effects Incidence Rates

The most common health risk model expresses the reduction in health risk (Δy) associated with a given reduction in PM concentrations (Δx) as a percentage of the baseline incidence (y). To accurately assess the impact of PM air quality on health risk in the selected cities, information on the baseline incidence of health effects (i.e., the incidence under "as is" air quality conditions) in each location is therefore needed. Where possible, county-specific incidences or incidence rates will be used. County-specific mortality incidences are available from the National Center for Health Statistics.

Table 4-3. Estimated Increased Mortality per Increments in 24-hr Concentrations of $PM_{2.5}$ from U.S. and Canadian Studies

Study Location (population studied and reference)*	RR (± CI) per 25 µg/m³ $PM_{2.5}$ Increase	Reported $PM_{2.5}$ Levels, Mean (µg/m³) (Min, Max) **
Total (nonaccidental) Mortality		
Six Cities (All ages) (Schwartz et al., 1996a)		
Portage, WI	*1.030 (0.993, 1.071)*	*11.2 (± 7.8)*
Topeka, KS	*1.020 (0.951, 1.092)*	*12.2 (± 7.4)*
Boston, MA	*1.056 (1.038, 1.074)*	*15.7 (± 9.2)*
St. Louis, MO	*1.028 (1.010, 1.043)*	*18.7 (± 10.5)*
Kingston/Knoxville, TN	*1.035 (1.005, 1.066)*	*20.8 (±- 9.6)*
Steubenville, OH	*1.025 (0.998, 1.053)*	*29.6 (± 21.9)*
Overall Six-City results	*1.038 (1.028, 1.048)*	*median 14.7*
Overall Six-City results (Age 65+)	*1.043 (1.03, 1.056)*	*median 14.7*
Detroit, MI (All ages) (Lippmann et al., 2000)	1.031 (0.004, 1.069)	18 (6, 86)
Los Angeles, CA (All ages) (Moolgavkar et al., 2000)	1.4 (-0.1, 2.9)	22 (4, 86)
Montreal, Canada (Goldberg et al., 2000) (All ages) (Age 65+)	 1.029 (0.99, 1.06) 1.033 (0.98, 1.069)	 3.3 (0, 30)
3 New Jersey Cities: Newark Camden Elizabeth (All ages) (Tsai et al., 2000)	 1.043 (1.028, 1.059) 1.057 (1.001, 1.115) 1.018 (0.946, 1.095)	 42.1 (± 22.0) 39.9 (± 18.0) 37.1 (± 19.8)
Philadelphia, PA (All ages) (Lipfert et al., 2000)	1.042 (p<0.055)	17.3 (-0.6, 72.6)
Phoenix, AZ (All ages) (Mar et al., 2000)	1.060 (1.00, 1.154)	13.0 (0, 42)
Phoenix, AZ (Age 65+) (Smith et al., 2000)	(>25 µg/m³) 2.868 (1.126, 7.250) (<25 µg/m³) 0.779 (0.610, 0.995)	NR
Santa Clara County, CA (All ages) (Fairley, 1999)	1.085 (1.032, 1.138)	13 (2, 105)
8 Canadian Cities (All ages) (Burnett et al., 2000)	1.030 (1.011, 1.050)	13.3 (max 86)

Study Location (population studied and reference)*	RR (± CI) per 25 μg/m^3 $PM_{2.5}$ Increase	Reported $PM_{2.5}$ Levels, Mean (μg/m^3) (Min, Max) **
Cause-Specific Mortality		
Cardiorespiratory:		
3 New Jersey Cities:		
Newark	1.051 (1.031, 1.072)	42.1 (±22.0)
Camden	1.062 (1.006, 1.121)	39.9 (±18.0)
Elizabeth	1.023 (0.95, 1.101)	37.1 (± 19.8)
(All ages) (Tsai et al., 2000)		
Total Cardiovascular:		
Six Cities (same as above) (All ages) (Schwartz et al., 1996)	*1.053 (1.035, 1.071)*	*median 14.7*
Detroit, MI (All ages) (Lippmann et al., 2000)	1.032 (0.977, 1.089)	18 (6, 86)
Los Angeles, CA (All ages) (Moolgavkar et al., 2000)	1.027 (1.004, 1.049)	22 (4, 86)
Montreal, Canada (All ages) (Goldberg et al, 2000)	1.034 (0.988, 1.081)	17.4 (2.2, 72.0)
Philadelphia, PA (7-county area) (All ages) (Lipfert et al., 2000)	1.043 (p<0.055)	17.3 (-0.6, 72.6)
Phoenix, AZ (All ages) (Mar et al., 2000)	1.187 (1.057, 1.332)	13.0 (0, 42)
Santa Clara County, CA (All ages) (Fairley, 1999)	1.07 (p>0.05)	13 (2, 105)
Total Respiratory:		
Six Cities (same as above) (All ages) (Schwartz et al., 1996)	*1.085 to 1.103*	*median 14.7*
Detroit, MI (All ages) (Lippmann et al., 2000)	1.023 (0.897, 1.166)	18 (6, 86)
Los Angeles, CA (All ages) (Moolgavkar et al., 2000)	1.027 (0.966, 1.091)	22 (4, 86)
Montreal, Canada (Goldberg et al., 2000) All ages Age 65+	 1.119 (1.015, 1.234)) 1.131 (1.019, 1.255)	 3.3 (0, 30)
Philadelphia, PA (7-county area) (All ages) (Lipfert et al., 2000)	1.022 (p>0.055)	17.3 (-.6, 72.6)
Santa Clara County, CA (All ages) (Fairley, 1999)	1.12 (p>0.05)	13 (2, 105)

* Studies included in the prior 1996 risk analyses are in italics; new studies are in plain text.
** Relative risk (95% confidence interval), except for Fairley (1999) and Lipfert et al. (2000) where insufficient data are available to calculate confidence intervals so p-value is given in parentheses.
*** Min/Max 24-h PM indicator level shown in parentheses unless otherwise noted.

Table 4-4. Estimated Cardiovascular Morbidity Effects per Increments in 24-hr Concentrations of $PM_{2.5}$ from U.S. and Canadian Studies

Health Effect and Study Location (population studied and reference)*	RR (± CI) per 25 µg/m³ $PM_{2.5}$ Increase	Reported $PM_{2.5}$ Levels, Mean (µg/m³) (Min, Max) **
Increased Hospitalization		
Cardiovascular:		
Los Angeles, CA (Age 65+) Los Angeles, CA (Age 20-64) (Moolgavkar et al., 2000)	(age 65+) 1.043 (1.025, 1.061) (age 20-64) 1.035 (1.018, 1.053)	median 22 (4, 86)
Toronto, Canada (All ages) (Burnett et al., 1997)	1.072 (0.994, 1.156)	16.8 (1, 66)
Heart Failure:		
Detroit, MI *** (Lippmann et al., 2000)	1.091 (1.023, 1.162)	18 (6, 86)
Increased emergency department visits		
St. John, Canada (All ages) (Stieb et al., 2000)	1.151 (0.998, 1.328)	Summer 1993 8.5 (max 53.2)

Table 4-5. Estimated Respiratory Morbidity Effects per Increments in 24-hr Concentrations of $PM_{2.5}$ and $PM_{10-2.5}$ from U.S. and Canadian Studies

Study Location (population studied and reference)*	RR (± CI) per 25 µg/m^3 $PM_{2.5}$ Increase	RR (± CI) per 25 µg/m^3 $PM_{10-2.5}$ Increase	Reported $PM_{2.5}$ Levels, Mean (µg/m^3) (Min, Max) **
Increased Admission to Hospital			
Total Respiratory:			
Toronto, Canada (All ages) (Burnett et al., 1997)	1.086 (1.034, 1.141)	1.127 (1.052, 1.207)	$PM_{2.5}$ 16.8 (1, 66) PM_{10} 28.1 (4, 102) $PM_{10-2.5}$ 11.6 (1, 56)
Toronto, Canada (Age >64 years) (Thurston et al., 1994)	*1.15 (1.02, 1.28)*		*$PM_{2.5}$ 18.6 (NR, 66)*
Pneumonia:			
Detroit, MI (Age >65 years) (Lippmann et al., 2000)	1.125 (1.037, 1.220)	1.119 (1.007, 1.244)	$PM_{2.5}$ 18 (6, 86) PM_{10} 31 (max 105) $PM_{10-2.5}$ 13 (4, 50)
Respiratory infections:			
Toronto, Canada (All ages) (Burnett et al., 1997)	1.108 (1.072, 1.145)	1.093 (1.046, 1.142)	$PM_{2.5}$ 18.0 (max 90) PM_{10} 30.2 (max 116) $PM_{10-2.5}$ 12.2 (max 68)
COPD:			
Detroit, MI (All ages)(Lippmann et al., 2000)	1.055 (0.953, 1.168)		18 (6, 86)
King County, WA (All ages) (Moolgavkar et al., 2000)	1.065 (1.3, 1.118)	---	$PM_{2.5}$ 18.1 (3, 96) PM_{10}
Los Angeles, CA (Age >65 years) (Moolgavkar et al., 2000)	1.051 (1.009, 1.094)	---	$PM_{2.5}$ median 224, 86) PM_{10} median 44 (7, 166)
Increased respiratory emergency department visits			
Montreal, Canada (Age 65+) (Delfino et al., 1997)	1.239 (1.049, 1.428)	---	summer 1993 $PM_{2.5}$ 12.2 (max 31) PM_{10} 21.7 (max 51)
St. John, Canada (All ages) (Stieb et al., 2000)	1.057 (1.006, 1.110)	---	summer 1993 $PM_{2.5}$ 8.5 (max 53.2) PM_{10}14.0 (max 70.3)

Study Location (population studied and reference)*	RR (± CI) per 25 µg/m^3 $PM_{2.5}$ Increase	RR (± CI) per 25 µg/m^3 $PM_{10-2.5}$ Increase	Reported $PM_{2.5}$ Levels, Mean (µg/m^3) (Min, Max) **
Asthma:			
Increased Respiratory Symptoms			
Uniontown, PA (evening cough) (Neas et al., 1995)	*1.45 (1.07, 1.97)*		*24.5 (max 88.1)*
Southwest Virginia (Runny or Stuffy nose) (Zhang et al., 2000)		2.62 (1.16, 5.87)	$PM_{2.5}$ NR $PM_{10-2.5}$ NR
State College, PA Cough Cold (Neas et al., 1996)	 1.61 (1.21,2.17) 1.45 (1.29, 4.64)	 ---	$PM_{2.1}$ 23.5 (max 85.8) $PM_{10-2.5}$ ---
Six Cities reanalysis : Cough Lower respiratory symptoms (Children grades 2-5) (Schwartz and Neas, 2000)		 1.77 (1.23, 2.54) 1.51 (0.94, 4.87)	 $PM_{2.5}$ (same as Six Cities) $PM_{10-2.5}$ NR
Six Cities: *Cough* *Lower respiratory symptoms (LRS)* *(Children grades 2-5) (Schwartz et al., 1994)*	 *1.24 (1.00, 1.54)* *1.58 (1.18, 2.10)*		*18.0 (max 86.0)*

Table 4-6. Effect Estimates per Increments in Long-term Mean Levels of Fine Particle Indicators from U.S. and Canadian Studies

Type of Health Effect and Study Location (population studied and reference)	RR (± CI) per 25 μg/m^3 $PM_{2.5}$ Increase	Range of City $PM_{2.5}$ Levels, Means (μg/m^3)
Increased total mortality in adults		
Six Cities Reanalysis (Age 25+) (Krewski et al., 2000)	1.39	11-30
ACS Study Reanalysis (Age 30+) (Krewski et al., 2000)	1.18	9-33
Increased cardiopulmonary mortality in adults		
Six Cities Reanalysis (Age 25+) (Krewski et al., 2000)	1.45	11-30
ACS Study Reanalysis (Age 30+) (Krewski et al., 2000)	1.31	9-33

For many of the morbidity endpoints, however, county-specific incidence rates are difficult to obtain. County-specific rates for hospital admissions are in the process of being obtained for Philadelphia, Los Angeles, and Salt Lake counties. For other morbidity endpoints, such as respiratory symptoms in children, incidence information aggregated at a higher level may be all that is available. The level of aggregation closest to county-specific will be used; however, for some morbidity endpoints, it may be necessary to estimate county-specific incidence using national-level incidence rates. For some health endpoints, there may be no information on incidence other than the information provided for the city in which the concentration-response function was estimated. A discussion will be presented of the rationale for the choice of incidence data used for each location. The lack of city- or county-specific incidence data will increase uncertainty concerning the estimates of risk for the specific cities selected for the risk analysis.

To the extent possible, a quantitative comparison will be provided to help assess the accuracy of using incidence rates at a higher level of aggregation (e.g., national incidence rates) by comparing these rates to city- or county-specific incidence rates where these are available.

4.2.5 Uncertainties in Risk Analyses and Plans for Conducting Sensitivity Analyses

There are considerable uncertainties in risk analyses for any air pollutant. These are compounded in the case of a pollutant such as PM (as opposed to, for example, O_3), given the diversity of composition in this generally defined pollutant. Among the major sources of uncertainty in the planned risk analyses are:

- The statistical uncertainty surrounding estimates of PM coefficients in concentration-response functions used in the analysis.

- The transferability of PM concentration-response functions from study locations to the locations selected for the risk analysis due to variations in PM composition across cities; the possible role of associated copollutants in influencing PM risk; and variations in the relation of ambient exposure to ambient monitoring in different locations. There is also uncertainty concerning the transferability of health functions to future PM aerosol mixes. In addition, cities may have different population sensitivity to PM effects (with some sensitive populations likely still to be defined).

- The air quality adjustment procedure that will be used to simulate just meeting alternative PM standards, and uncertainty about the extent to which reductions in PM will consist of reductions in fine versus coarse particles.

- Use of baseline health effects incidence information that is not specific to the county in question.

- Applying pooled concentration-response functions to represent the overall effect of particles on a particular health endpoint from studies in several locations.

- The impact of historical air quality on estimates of health risk from long-term PM exposures – the duration of time that a reduction in particle concentrations must be maintained in a given location in order to experience the predicted reduction in health risk and/or the possibility of lags between exposure and health effect.

- The effect of normalizing to different degrees the amounts of health risk experienced or reduced in different locations because of differences in the completeness of the air quality data sets.

- Estimated background concentrations for each location.

- The effect of measurement uncertainty in the original health studies used to develop the concentration-response relationships.

The uncertainties from some of these sources – in particular, the statistical uncertainty surrounding estimates of pollutant coefficients – can be characterized quantitatively. It will be possible, for example, to calculate confidence intervals around risk estimates based on the statistical uncertainty associated with the estimates of pollutant coefficients used in the risk analyses. These confidence intervals will express the range within which the true risks are likely to fall *if the statistical uncertainty surrounding pollutant coefficient estimates were the only uncertainty in the analyses*. There are, of course, several other uncertainties in the risk analyses, as noted above. If there were sufficient information to quantitatively characterize these sources of uncertainty, they could be included in a Monte Carlo analysis to produce confidence intervals that more accurately reflect all sources of uncertainty.

Uncertainties in the risk analysis are proposed to be handled in the following ways:

- Limitations and assumptions in the quantification process will be clearly stated and explained.

- For any endpoint for which only a single concentration-response function has been estimated, the uncertainty resulting from the statistical uncertainty associated with the estimate of the pollutant coefficient will be characterized by confidence intervals around the point estimate of risk. As noted above, such a confidence interval will express the range within which the true risk is likely to fall *if the statistical uncertainty surrounding the pollutant coefficient estimate were the only uncertainty in the analysis*. It will not, for example, reflect the uncertainty concerning whether the pollutant coefficients in the study location and the assessment location are the same.[2]

- For any endpoint for which a pooled function has been derived from two or more studies, a credible interval will be presented along with the point estimate of risk. Credible intervals will reflect not only the within-study statistical uncertainty, but the between-study variability in pollutant coefficients as well. These credible intervals will therefore, to some extent, also reflect the uncertainty associated with applying functions estimated in locations other than the assessment location.

[2] This is not an uncertainty, of course, if the concentration-response function has been estimated in the assessment location.

- Sensitivity analyses will be conducted to illustrate the effects of changing key default assumptions on the mean results of the assessment, and quantitative comparisons[3] presented to inform other analytic choices.

Possible additional or alternative approaches to characterizing uncertainty that are being considered include the following:

- To include in an overall assessment of uncertainty those sources of uncertainty that cannot readily be quantified, "integrated sensitivity analyses" may be presented. These analyses rely on staff judgment to assign probabilities to possible alternatives. For example, staff judgment would be used to assess the likelihood that each of several possible alternative assumptions is the correct one. This procedure allows sources of uncertainty that are otherwise not quantifiable to be included in a Monte Carlo analysis of overall sensitivity to various alternative values.

- Different sets of plausible assumptions that would result in "low end," "middle," and "high end" estimates of incidence could be identified, and the estimates resulting under each set of assumptions could be presented as alternatives.

4.3 $PM_{2.5}$ Risk Estimates for Philadelphia and Los Angeles Counties

The next draft of the Staff Paper will include presentation of base case risk estimates for "as is" air quality, air quality levels associated with just attaining the current $PM_{2.5}$ standards, and air quality associated with attaining any potential alternative $PM_{2.5}$ standards that are identified as part of this review. In addition, results of sensitivity analyses of individual uncertainties and assumptions as well as integrated uncertainty analyses examining the impact of several key uncertainties will be presented. This section will then conclude with key observations from the $PM_{2.5}$ risk analyses.

4.4 $PM_{10-2.5}$ Risk Estimates for Example Counties

If the Agency decides to conduct $PM_{10-2.5}$ risk analyses, this section will include base case risk estimates for as is air quality, air quality levels associated with just attaining the current $PM_{2.5}$ standards, and air quality associated with attaining any alternative $PM_{10-2.5}$ standards that are

[3] "Sensitivity analyses" refers to assessing the effects of uncertainty on some of the final risk estimates; "quantitative comparisons" refer to numerical comparisons (e.g. comparisons of monitor values) that are not carried that far.

identified as part of this review. In addition, results of sensitivity analyses of individual uncertainties and assumptions as well as integrated uncertainty analyses examining the impact of several key uncertainties will be presented. This section will then conclude with key observations from the $PM_{10-2.5}$ risk analyses.

REFERENCES

Most Chapter 4 references are available at the end of Chapter 3. References not listed at the end of Chapter 3 are listed here.

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5. CHARACTERIZATION OF PM-RELATED ENVIRONMENTAL EFFECTS

5.1 INTRODUCTION

This chapter summarizes key information relevant to assessing the environmental effects associated with ambient PM, alone and in combination with other pollutants commonly present in the ambient air, drawing upon the most relevant information contained in the draft CD and other significant reports referenced therein. The chapter is organized into a discussion of the effects on public welfare to be considered in this review of the secondary standards for PM. Specifically, this chapter addresses PM-related effects on visibility (Section 5.2), materials (Section 5.3), vegetation and ecosystems (Section 5.4), and solar radiation and global climate change (Section 5.5). For each category of PM-related effects, this preliminary draft chapter presents a brief summary of the relevant scientific information and a preliminary staff assessment of whether the available information is sufficient to be considered as the basis for secondary standards distinct from primary standards for PM. In addition, in assessing information on PM-related effects on solar radiation and global climate change, consideration is given to potential indirect impacts on human health and the environment that may be a consequence of radiative and climatic changes attributable to changes in ambient PM. Staff conclusions and recommendations related to secondary standards for PM will be incorporated into Chapter 6 of a subsequent draft of this Staff Paper.

It is important to note that the discussion of PM-related effects on visibility, vegetation and ecosystems, and solar radiation and global climate change in Chapter 4 of the draft CD builds upon and includes by reference extensive information from several other significant reviews of these areas. Most notably, these reports include the Recommendations of the Grand Canyon Visibility Transport Commission (1996), the National Research Council's *Protecting Visibility in National Parks and Wilderness Areas* (1993), reports of the National Acid Precipitation Assessment Program (1991), previous EPA Criteria Documents, including *Air Quality Criteria for Particulate Matter and Sulfur Oxides* (EPA, 1982) and *Air Quality Criteria for Oxides of Nitrogen* (EPA, 1993), and numerous U.S. and international assessments of stratospheric ozone depletion and global climate change carried out under U.S. Federal interagency programs (e.g.,

the U.S. Global Climate Change Research Program) and the World Meteorological Organization (WMO) and the United Nations Environment Programme (UNEP).

5.2 EFFECTS ON VISIBILITY

Visibility impairment has long been considered the "best understood and most easily measured effect of air pollution" (Council on Environmental Quality, 1978). It is caused by the scattering and absorption of light by particles and gases in the atmosphere. It is the most noticeable effect of fine particles present in the atmosphere. Air pollution degrades the visual appearance of distant objects to an observer, and reduces the range at which they can be distinguished from the background. Ambient particles affect the perceived color of distant objects depending upon particle size and composition, the scattering angle between the observer and illumination, the properties of the atmosphere, and the optical properties of the target being viewed.

This section discusses the role of ambient PM in the impairment of visibility, building upon the information present in the last Staff Paper (EPA, 1996b) and drawing upon the most relevant information contained in the draft CD and significant reports on the science of visibility referenced therein. In particular, this section includes new information on the following topics:

- Planned data analyses to characterize visibility impairment in urban and suburban areas based on 1999 visibility data from 60+ Automated Surface Observation System (ASOS) installations from around the country, and to explore the degree to which the ASOS data correlates with 1999 daily $PM_{2.5}$ measurements.

- An overview of existing and planned visibility programs, goals, and methods for the evaluation of visibility impairment as a basis for standard setting, in the U.S. and abroad, illustrating the significant value placed on efforts to improve visibility outside of national parks and wilderness areas.

- A pilot survey project conducted by EPA in November 2000 in Washington DC to elicit public input on the acceptability of varying levels of visual air quality in urban areas, and plans for conducting a broader survey using the methodology developed and refined as part of the pilot project, using new techniques for photographic representation of visibility impairment.

The presentation here organizes the available information on visibility impairment into elements related to the evaluation of current and alternative standards for PM. Beyond providing an overview of visibility impairment, this section summarizes: (1) the effects of PM on visibility (building upon information presented above in Section 2.9); (2) conditions in Class I and non-urban areas, as well as in urban areas; (3) information on the significance of visibility to public welfare; and (4) approaches to evaluating public perceptions of visibility impairment and judgments about the acceptability of varying degrees of impairment.

5.2.1 Overview of Visibility Impairment

Visibility can be defined as the degree to which the atmosphere is transparent to visible light (NRC, 1993; CD, 4-86). Visibility effects are manifested in two principal ways: (1) as local impairment (e.g., localized hazes and plumes); and (2) as regional haze. These distinctions are significant both to the ways in which visibility goals may be set and air quality management strategies may be devised.

Local-scale visibility degradation is commonly in the form of either a plume resulting from the emissions of a specific source or small group of sources, or it is in the form of a localized haze, such as an urban "brown cloud." Impairment caused by a specific source or small group of sources has been generally termed as "reasonably attributable" impairment. Plumes are comprised of smoke, dust, or colored gas that obscure the sky or horizon relatively near sources. Sources of locally visible plumes, such as the plume from an industrial facility or a burning field, are often easy to identify. "Reasonably attributable" impairment may include contributions to local hazes by individual sources or several identified sources. There have been a limited number of cases in which Federal land managers have certified the existence of visibility impairment in a Class I area (i.e., 156 national parks, wilderness areas, and international parks identified for visibility protection in section 162(a) of the Clean Air Act) that is considered “reasonably attributable” to a particular source.[1]

[1]Two of the most notable cases leading to emissions controls involved the Navajo Generating Station in Arizona and the Mohave power plant in Nevada, for which it was found that sulfur dioxide emissions were contributing to visibility impairment in Grand Canyon National Park.

A localized or layered haze often results from emissions from many sources located across an urban or metropolitan area. This type of impairment may be seen as a band or layer of discoloration appearing well above the terrain. A common manifestation of this type of visibility impairment is the "brown cloud" situation experienced in some cities particularly in the winter months, when cooler temperatures limit vertical mixing of the atmosphere. Urban visibility impairment often results from the combined effect of stationary, mobile, and area source emissions, and complex local meteorological conditions may contribute to such impairment as well. The long-range transport of emissions from sources outside the urban area may also contribute to urban haze levels. A number of studies have been conducted in the past in cities like Denver, Dallas, and Seattle to characterize urban visibility problems.

The second type of impairment, regional haze, results from pollutant emissions from a multitude of sources located across a broad geographic region. It impairs visibility in every direction over a large area, in some cases over multi-state regions. Regional haze masks objects on the horizon and reduces the contrast of nearby objects. The formation, extent, and intensity of regional haze is a function of meteorological and chemical processes, which sometimes cause fine particle loadings to remain suspended in the atmosphere for several days and to be transported hundreds of kilometers from their sources (NRC, 1993). It is this second type of visibility degradation that is principally responsible for impairment in national parks and wilderness areas across the country (NRC, 1993). Visibility in urban areas at times may be dominated by local sources, but often may be significantly affected by long-range transport of haze due to the multi-day residence times of fine particles in the atmosphere. Fine particles transported from urban areas in turn may be significant contributors to regional-scale impairment in Class I and other rural areas.

5.2.2 Effects of PM on Visibility

The efficiency at which a unit mass of particles causes visibility impairment depends on a number of factors, including particle size, composition, and humidity. These basic concepts are discussed above in Section 2.9.1. Building on this information, this section discusses common

measures of visibility impairment, estimated natural visibility conditions, and other important factors in the relationship between PM and visibility impairment.

5.2.2.1 Measures of Visibility Impairment

Several atmospheric optical indices and approaches can be used for characterizing visibility impairment. As summarized below and discussed in more detail in the draft CD, there are several indicators that could be used in regulating air quality for visibility protection, including: (1) human observation of visual range; (2) light extinction (and related parameters of visual range and deciview); (3) light scattering by particles; and (4) fine particle mass concentration (CD, page 4-94).

Human Observation. For many decades, the National Weather Service has recorded hourly visibility at major airports based on human observations of distant targets. This approach has provided a historical record of visibility across the U.S. and has allowed a general interpretation of regional visibility trends. Airport visibility monitoring has been automated in recent years, however, through deployment of the Automated Surface Observing System (ASOS) at more than 900 airports across the country (discussed below in Section 5.2.5). While human observations have been very effective for the purposes of air safety, these data are not as well correlated to air quality levels as data obtained from other automated monitoring methods.

Light Extinction and Related Measures. The light extinction coefficient has been widely used in the U.S. for many years as a metric to describe the effect of pollutant concentrations on visibility. It can be defined as the fraction of light lost or redirected per unit distance through interactions with gases and suspended particles in the atmosphere. The light extinction coefficient represents the summation of light scattering and light absorption due to particles and gases in the atmosphere. Both anthropogenic and non-anthropogenic sources contribute to light extinction. The light extinction coefficient (σ_{ext}) is represented by the following equation (CD, 4-89):

$$\sigma_{ext} = \sigma_{sg} + \sigma_{ag} + \sigma_{sp} + \sigma_{ap}$$

where σ_{sg} = light scattering by gases (also known as Rayleigh scattering)

σ_{ag} = light absorption by gases

σ_{sp} = light scattering by particles

σ_{ap} = light absorption by particles.

Light extinction is commonly expressed in terms of inverse kilometers (km^{-1}) or inverse megameters (Mm^{-1}), where increasing values indicate increasing impairment.

Total light extinction can be measured directly by a transmissometer or it can be calculated from ambient pollutant concentrations. Transmissometers measure the light transmitted through the atmosphere over a distance of 1 to 15 km. The light transmitted between the light source (transmitter) and the light-monitoring component (receiver) is converted to the path-averaged light extinction coefficient. Transmissometers operate continuously, and data is often reported in terms of hourly averages.

Direct relationships exist between measured ambient pollutant concentrations and their contributions to the extinction coefficient. The contribution of each aerosol constituent to total light extinction is derived by multiplying the aerosol concentration by the extinction efficiency for that aerosol constituent. Extinction efficiencies vary by type of aerosol constituent and have been obtained through empirical studies. For certain aerosol constituents, extinction efficiencies increase significantly with increases in relative humidity.

In addition to the optical effects of atmospheric constituents as characterized by the extinction coefficient, lighting conditions and scene characteristics play an important role in determining how well we see objects at a distance. Some of the conditions that influence visibility include whether a scene is viewed towards the sun or away from it, whether the scene is shaded or not, and the color and reflectance of the scene (NAPAP, 1991). For example, a mountain peak in bright sun can be seen from a much greater distance when covered with snow than when it is not.

One's ability to clearly see an object is degraded both by the reduction of image forming light from the object caused by scattering and absorption, and by the addition of non-image forming light that is scattered into the viewer's sight path. This non-image forming light is called path radiance (EPA, 1996a, p. 8-23). A common example of this effect is our inability to see stars in the daytime due to the brightness of the sky caused by Rayleigh scattering. At night, when the sunlight is not being scattered, the stars are readily seen. This same effect causes a haze to appear

bright when looking at scenes that are generally towards the direction of the sun and dark when looking away from the sun.

Though these non-air quality related influences on visibility can sometimes be significant, they cannot be accounted for in any practical sense in formulation of national or regional measures to minimize haze. Lighting conditions change continuously as the sun moves across the sky and as cloud conditions vary. Non-air quality influences on visibility also change when a viewer of a scene simply turns their head. Regardless of the lighting and scene conditions, however, sufficient changes in ambient concentrations of PM will lead to changes in visibility (and the extinction coefficient). The extinction coefficient integrates the effects of aerosols on visibility, yet is not dependent on scene-specific characteristics. It measures the changes in visibility linked to emissions of gases and particles that are subject to some form of human control and potential regulation, and therefore can be useful in comparing visibility impact potential of various air quality management strategies over time and space (NAPAP, 1991).

By apportioning the extinction coefficient to different aerosol constituents, one can estimate changes in visibility due to changes in constituent concentrations (Pitchford and Malm, 1994). The National Research Council's 1993 report *Protecting Visibility in National Parks and Wilderness Areas* states that "[p]rogress toward the visibility goal should be measured in terms of the extinction coefficient, and extinction measurements should be routine and systematic." Thus, it is reasonable to use the change in the light extinction coefficient, determined in multiple ways, as the primary indicator of changes in visibility for regulatory purposes.

Visual range is a measure of visibility that is inversely related to the extinction coefficient. Visual range can be defined as the maximum distance at which one can identify a black object against the horizon sky. The colors and fine detail of many objects will be lost at a distance much less than the visual range, however. Visual range has been widely used in air transportation and military operations in addition to its use in characterizing air quality. Conversion from the extinction coefficient to visual range can be made with the following equation (NAPAP, 1991):

$$\text{Visual Range } (km^{-1}) = 3.91/\sigma_{ext}$$

Another important visibility metric is the deciview, a unitless metric which describes changes in uniform atmospheric extinction that can be perceived by a human observer. It is designed to be linear with respect to perceived visual changes over its entire range in a way that is analogous to the decibel scale for sound (Pitchford and Malm, 1994). Neither visual range nor the extinction coefficient has this property. For example, a 5 km change in visual range or 0.01 km^{-1} change in extinction coefficient can result in a change that is either imperceptible or very apparent depending on baseline visibility conditions. The deciview metric allows one to more effectively express perceptible changes in visibility, regardless of baseline conditions. A one deciview change is a small but perceptible scenic change under many conditions, approximately equal to a 10% change in the extinction coefficient. The deciview metric also may be useful in defining goals for perceptible changes in visibility conditions under future regulatory programs. Deciview can be calculated from the light extinction coefficient (σ_{ext}) by the equation:

$$dv = 10 \log_{10}(\sigma_{ext}/10\ Mm^{-1})$$

Figure 5-1 graphically illustrates the relationships among light extinction, visual range, and deciview.

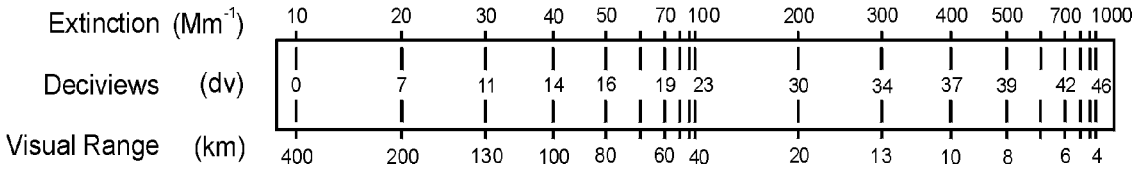

Figure 5-1. Relationship Between Light Extinction, Deciview, and Visual Range.

Light Scattering Coefficient. Across the U.S., light scattering is typically a much larger contributor to total light extinction than light absorption. Of the main categories of particles, only elemental carbon is a key contributor to light absorption and commonly represents only 5-10% of total light extinction (Malm et al., 2000). Light scattering data taken by a nephelometer can be

correlated fairly well with total light extinction measurements using certain assumptions for light absorption. Nephelometers measure the scattering of light by particles contained in a small volume of air, and thus provide a point measurement of scattering.

Fine Particle Mass Concentration. Fine particle (e.g., $PM_{2.5}$) mass concentrations can be used as a general surrogate for visibility impairment. However, as described in many reviews of the science of visibility, the different constituents of $PM_{2.5}$ have variable effects on visibility impairment. For example, crustal material in general accounts for less light scattering per unit mass than other constituents, and sulfates and nitrates contribute greater amounts of light scattering as relative humidity levels exceed 70%. Thus, while higher $PM_{2.5}$ mass concentrations generally indicate higher levels of visibility impairment, it is not as precise a metric as the light extinction coefficient. By using historic averages or regional estimates of the component-specific percentage of total mass, however, one can develop reasonable estimates of light extinction from PM mass concentrations.

5.2.2.2 Rayleigh Scattering and Natural Background Conditions

Rayleigh scattering represents the degree of natural light scattering found in a particle-free atmosphere, caused by the gas molecules that make up "blue sky" (e.g., N_2, O_2). It accounts for a relatively constant level of light extinction nationally, between 10 to 12 Mm^{-1} (NAPAP, 1991; EPA, 1979). The concept of Rayleigh scattering can be used to establish a theoretical maximum horizontal visual range in the earth's atmosphere. At sea level, this maximum visual range is approximately 330 kilometers. Since certain meteorological circumstances can reduce pollution that can result in visibility conditions that are close to "Rayleigh," it is analogous to a baseline or boundary condition against which other extinction components can be compared.

Light extinction caused by PM from natural sources can vary significantly from day to day and location to location due to natural events such as wildfire, dust storms, and volcanic eruptions. It is useful to consider estimates of natural background concentrations of PM on an annual average basis, however, when evaluating the relative contributions of anthropogenic (man-made) and non-anthropogenic sources to total light extinction.

As discussed in Chapter 2, for the purpose of this document, background PM is defined as the distribution of PM concentrations that would be observed in the U.S. in the absence of

anthropogenic emissions of primary PM and precursor emissions of VOC, NO_x, SO_2, and NH_3 in North America. Table 2-4 describes the range for annual average regional background $PM_{2.5}$ mass in the eastern U.S. as 2 to 5 $\mu g/m^3$, and in the western U.S. as 1 to 4 $\mu g/m^3$. For PM_{10}, the estimated annual average background concentrations range from 5 to 11 $\mu g/m^3$ in the eastern U.S., and 4 to 8 $\mu g/m^3$ in the western U.S.

The NAPAP report provides estimates of extinction contributions from Rayleigh scattering plus background levels of fine and coarse particles. In the absence of anthropogenic emissions of visibility-impairing particles, these estimates are 26 $\pm$ 7 Mm^{-1} in the East, and 17 $\pm$ 2.5 Mm^{-1} in the West. These equate to a naturally-occurring visual range in the East of 150 $\pm$ 45 km, and 230 $\pm$ 40 km in the West. Excluding light extinction due to Rayleigh scatter, annual average background levels of fine and coarse particles are estimated to account for 14 Mm^{-1} in the East and about 6 Mm^{-1} in the West. Major contributors that reduce visibility from the Rayleigh maximum to the ranges noted above are naturally-occurring organics, suspended dust (including coarse particles), and water. In these ranges of fine particle concentrations, small changes have a large effect on total extinction. Thus, higher levels of background fine particles and associated humidity in the East result in a fairly significant difference between naturally-occurring visual range in the rural East and West.

5.2.2.3 Contribution of PM to Visibility Conditions

On an annual average basis, the concentrations of background fine particles are generally small when compared with concentrations of fine particles from anthropogenic sources (NRC, 1993). The same relationship holds true when one compares annual average light extinction due to background fine particles with light extinction due to background plus anthropogenic sources. Table VIII-4 in the 1996 Staff Paper (EPA 1996b, p. VIII-10b) makes this comparison for several locations across the country by using background estimates from Table VIII-2 (EPA 1996b, p. VIII-6a) and light extinction values derived from monitored data from the IMPROVE network. These data indicate that anthropogenic emissions make a significant contribution to average light extinction in most parts of the country, as compared to the contribution from background fine particle levels. Man-made contributions account for about one-third of the average extinction coefficient in the rural West and more than 80% in the rural East (NAPAP, 1991).

It is important to note that even in those areas with relatively low concentrations of anthropogenic fine particles, such as the Colorado plateau, small increases in anthropogenic fine particle concentrations can lead to significant decreases in visual range. This is one reason why Class I areas have been given special consideration under the Clean Air Act. This relationship is illustrated by Figure VIII-9 in the 1996 Staff Paper (EPA, 1996b, p. VIII-10c) which relates changes in fine particle concentrations to changes in visibility (represented by the deciview metric). The graph shows that the visibility in an area with lower concentrations of air pollutants (such as many western Class I areas) will be more sensitive to a given increase in fine particle concentration than a more polluted atmosphere will be. Conversely, to achieve a given amount of visibility improvement, a larger reduction in fine particle concentration is required in areas with higher existing concentrations, such as the East, than would be required in areas with lower concentrations.

This relationship also illustrates the relative importance of the overall extinction efficiency of the pollutant mix at particular locations. At a given ambient concentration, areas having higher average extinction efficiencies due to the mix of pollutants would have higher levels of impairment (EPA, 1996b, p. VIII-10c, Figure VIII-9). In the East, the combination of higher humidity levels and a greater percentage of sulfate as compared to the West causes the average extinction efficiency for fine particles to be almost twice that for sites on the Colorado Plateau.

5.2.3 Visibility Conditions in Class I and Non-Urban Areas

5.2.3.1 IMPROVE Visibility Monitoring Network

In conjunction with the National Park Service, other Federal land managers, and State organizations, EPA has supported monitoring in national parks and wilderness areas since 1988. The network was originally established at 30 sites, but it has now been expanded to 110 of the 156 mandatory Federal Class I areas across the country. This long-term visibility monitoring network is known as IMPROVE (Interagency Monitoring of PROtected Visual Environments. The following discussion briefly describes the IMPROVE protocol and provides rationale supporting use of the light extinction coefficient, derived from both direct optical measurements

and measurements of aerosol constituents, for purposes of implementing air quality management programs to improve visibility.

IMPROVE provides direct measurement of fine particles and precursors that contribute to visibility impairment. The IMPROVE network employs aerosol, optical, and scene measurements. Aerosol measurements are taken for PM_{10} and $PM_{2.5}$ mass, and for key constituents of $PM_{2.5}$, such as sulfate, nitrate, organic and elemental carbon, soil dust, and several other elements. Measurements for specific aerosol constituents are used to calculate "reconstructed" aerosol light extinction by multiplying the mass for each constituent by its empirically-derived scattering and/or absorption efficiency. Knowledge of the main constituents of a site's light extinction "budget" is critical for source apportionment and control strategy development. Optical measurements are used to directly measure light extinction or its components. Such measurements are taken principally with either a transmissometer, which measures total light extinction, or a nephelometer, which measures particle scattering (the largest human-caused component of total extinction). Scene characteristics are recorded 3 times daily with 35 millimeter photography and are used to determine the quality of visibility conditions (such as effects on color and contrast) associated with specific levels of light extinction as measured under both direct and aerosol-related methods. Because light extinction levels are derived in two ways under the IMPROVE protocol, this overall approach provides a cross-check in establishing current visibility conditions and trends and in determining how proposed changes in atmospheric constituents would affect future visibility conditions.

5.2.3.2 Current Conditions Based on IMPROVE Data

Annual average visibility conditions (i.e., total light extinction due to anthropogenic and non-anthropogenic sources) vary regionally across the U.S. The rural East generally has higher levels of impairment than remote sites in the West, with the exception of the San Gorgonio Wilderness (CA), Point Reyes National Seashore (CA), and Mount Rainier National Park (WA), which have annual average levels comparable to certain sites in the Northeast. Higher averages in the East are due to generally higher concentrations of anthropogenic fine particles and higher average relative humidity levels. Visibility conditions also vary significantly by season of the year. With the exception of remote sites in the northwestern U.S., visibility is typically worse in the

summer months. This is particularly true in the Appalachian region, where average extinction in the summer exceeds the annual average by 40% (Sisler et al., 1996).

At this time, the 1996 Staff Paper serves as a general reference for understanding rural visibility conditions based on IMPROVE data. The next draft of this Staff Paper will include updated visibility trends and information on current conditions based on the latest available data.

5.2.4 Urban Visibility Conditions

For many years, urban visibility has been characterized using data describing airport visibility conditions. Until the mid-1990's, airport visibility was typically reported on an hourly basis by human observers. An extensive database of these assessments has been maintained and analyzed to characterize visibility trends from the late-1940's to mid-1990's (Schichtel et al., 2000).

As noted earlier, visibility impairment has been studied in several major cities in the past decades because of concerns about fine particles and their potentially significant impacts (e.g., health-related and aesthetic) on the residents of large metropolitan areas (e.g,. Middleton, 1993). Urban areas generally have higher loadings of fine particles and higher visibility impairment levels than monitored Class I areas. Urban area annual mean and 98^{th} percentile 24-hour average $PM_{2.5}$ levels for 1999 are presented above in Chapter 2. These levels are generally higher than those found in the IMPROVE database for rural Class I areas. In general, nitrates are responsible for a greater contribution to urban fine particle mass than in non-urban areas. In addition, some urban areas have higher concentrations of organic carbon and elemental carbon than rural areas due to a higher density of fuel combustion and diesel emissions.

5.2.4.1 Urban Visibility and $PM_{2.5}$ Monitoring Data

In the next draft of the Staff Paper, we intend to include information characterizing urban visibility for several cities around the country. Urban visibility data is available from the IMPROVE network for Washington, DC and South Lake Tahoe. Other cities with available visibility data include Denver, Phoenix, Seattle, and Tucson. In addition, as monitoring data become available from $PM_{2.5}$ speciation sites, we anticipate being able to calculate visibility for these sites in much the same way that is done for IMPROVE network sites.

5.2.4.2 ASOS Airport Visibility Monitoring Network

In 1992, the National Weather Service, Federal Aviation Administration, and Department of Defense began deployment of the Automated Surface Observing System (ASOS). ASOS is now the largest instrument-based visibility monitoring network in the U.S. (CD, 4-99). The ASOS visibility monitoring instrument is a forward scatter meter that has been found to correlate well with light extinction measurements from the Optec transmissometer (NWS, 1998). It is designed to provide consistent, real-time visibility and meteorological measurements to assist with air traffic control operations. More than 500 instruments have been commissioned and another 500 are planned for deployment in the coming years. ASOS visibility data is typically reported for aviation use in small increments up to a maximum of 10 miles visibility. While these truncated data are not useful for characterizing actual visibility levels, the raw, non-truncated data from the 1-minute light extinction and meteorological readings are now archived and available for analysis.

5.2.4.3 ASOS Data: Urban Visibility and Correlation to $PM_{2.5}$ Mass

To improve characterizations of current visibility conditions in non-class I areas, particularly in urban areas, EPA has obtained archived 1999 ASOS data for 63 cities across the country. Staff is in the process of analyzing the ASOS data to determine annual average, seasonal, monthly, and daily visibility conditions; best (10^{th} percentile) and worst (90^{th} percentile) day conditions; and diurnal and day of week conditions. Staff also plans to evaluate correlations between daily ASOS visibility data and 1999 24-hour $PM_{2.5}$ ambient monitoring data for a number of cities. Figure 5-2 is shown here as an illustrative example of such correlations. This information is expected to provide a better understanding of the average amount of light extinction per microgram of $PM_{2.5}$ in different parts of the country. Staff intends to include the results from these analyses in the next draft of this Staff Paper.

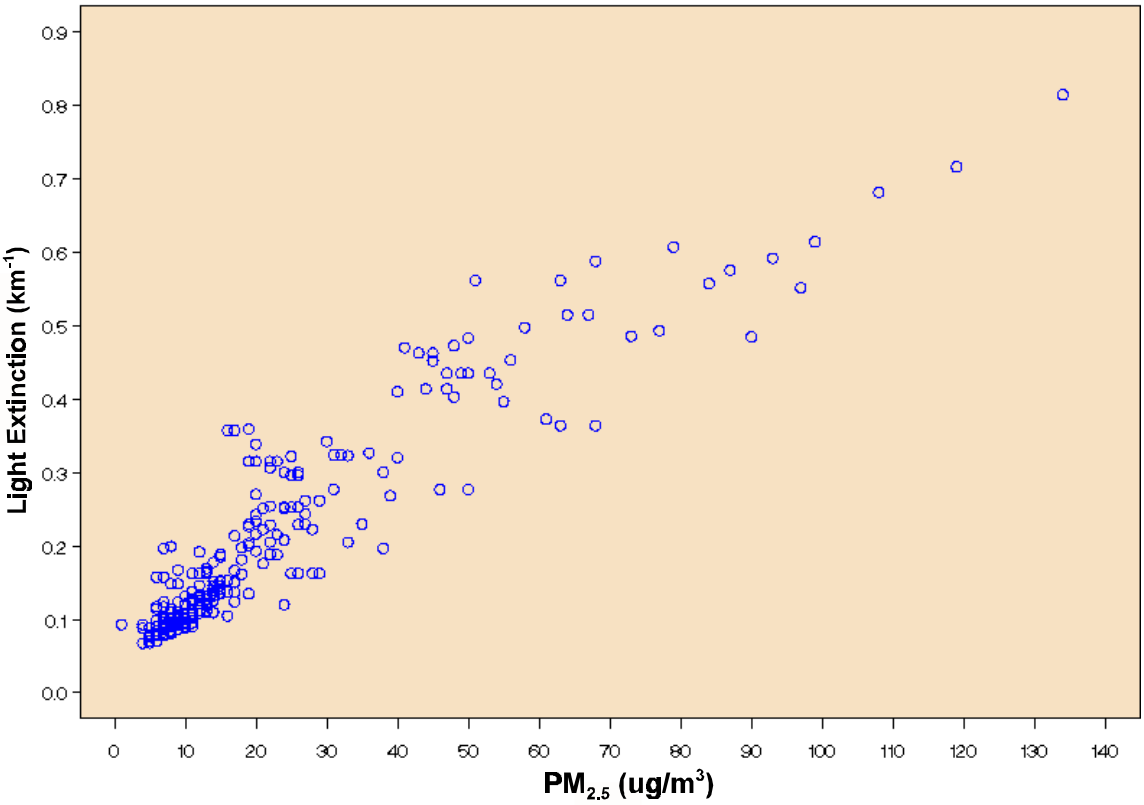

Figure 5-2. Correlation Between 1999 ASOS Airport Visibility Data (km^{-1}) and 24-Hour $PM_{2.5}$ Mass for Fresno, CA

5.2.5 Significance of Visibility to Public Welfare

Visibility is an air quality-related value having direct significance to people's enjoyment of daily activities in all parts of the country. Survey research on public awareness of visual air quality using direct questioning typically reveals that 80% or more of the respondents are aware of poor visual air quality (Cohen et al., 1986). The importance of visual air quality to public welfare across the country has been demonstrated by a number of studies designed to quantify the benefits (or willingness to pay) associated with potential improvements in visibility. More recently, the importance of visual air quality to the policymakers and the general public alike has also been demonstrated by a number of regional, state, and local efforts to address visibility impairment in urban and non-urban areas.

5.2.5.1 The Value of Improving Visual Air Quality

Individuals value good visibility for the well-being it provides them directly, both in the places where they live and work, and in the places where they enjoy recreational opportunities. Millions of Americans appreciate the scenic vistas in national parks and wilderness areas each year. Visitors consistently rate “clean, clear air” as one of the most important features desired in visiting these areas (Department of Interior, 1998). A 1998 survey of 590 representative households by researchers at Colorado State University found that 88% of the respondents believed that "preserving America's most significant places for future generations" is very important, and 87% of the respondents supported efforts to clean up air pollution that impacts national parks (Hass, 1998).

Economists have performed many studies in an attempt to quantify the economic benefits associated with improvements in current visibility conditions both in national parks and in urban areas. Economists distinguish between use values and non-use values. Use values are those aspects of environmental quality that directly affect an individual’s welfare. These include the aesthetic benefits of better visibility, improved road and air safety, and enhanced recreation in activities like hunting and hiking.

Non-use values are those for which an individual is willing to pay for reasons that do not relate to the direct use or enjoyment of any environmental benefit. The component of non-use value that is related to the use of the resource by others in the future is referred to as the bequest value. This value is typically thought of as altruistic in nature. Another potential component of non-use value is the value that is related to preservation of the resource for its own sake, even if there is no human use of the resource. This component of non-use value is sometimes referred to as existence value or preservation value. Non-use values are not traded, directly or indirectly, in markets. For this reason, the measurement of non-use values has proved to be significantly more difficult than the measurement of use values. Non-use values may be related to the desire that a clean environment be available for the use of others now and in the future, or may be related to the desire to know that the resource is being preserved for its own sake, regardless of human use. Non-use values may be a more important component of value for recreational areas, particularly national parks and monuments.

It is well recognized in the U.S. and abroad that there is an important relationship between good air quality and economic benefits due to tourism. A 1998 study by the Department of Interior study found that travel-related expenditures by national park visitors alone average $14.5 billion annually (1996 dollars) and support 210,000 jobs (Peacock, 1998). A similar estimate of economic benefits resulting from visitation to national forests and other public lands could increase this estimate significantly.

McNeill and Roberge (2000) studied the impact of poor visibility episodes on tourism revenues in Greater Vancouver and the Lower Fraser Valley in British Columbia as part of the Georgia Basin Ecosystem Initiative of Environment Canada. Through this analysis a model was developed that predicts future tourist revenue losses that would result from a single extreme visibility episode. They found that such an episode would result in a $7.45 million loss in the Greater Vancouver area and $1.32 million loss in the Fraser Valley.

The results of several valuation studies addressing both urban and rural visibility are presented in the 1996 Criteria Document (EPA, 1996a, p. 8-83, Table 8-5; p. 8-85, Table 8-6) and in the 1996 Staff Paper (EPA, 1996b, p. VIII-3a, Table VIII-1; Chestnut et al., 1994). Past studies by Schulze (1983) and Chestnut and Rowe (1990b) have estimated the preservation values associated with improving the visibility in national parks in the Southwest to be in the range of approximately $2-6 billion annually (CD, 8-84). An analysis of the residential visibility benefits in the eastern U.S. due to reduced sulfur dioxide emissions under the acid rain program suggests an annual value of $2.3 billion (in 1994 dollars) in the year 2010 (Chestnut and Dennis, 1997). The authors suggest that these results could be as much as $1-2 billion more because the above estimate does not include any value placed on eastern air quality improvements by households in the western U.S.

Estimating benefits for visibility can be difficult because visibility is not directly or indirectly valued in markets. The studies cited above are based on a valuation method known as contingent valuation. Concerns have been identified about the reliability of value estimates from contingent valuation studies because research has shown that bias can be introduced easily into these studies if they are not carefully conducted. Accurately measuring willingness-to-pay for avoided health and welfare losses depends on the reliability and validity of the data collected.

However, there is an extensive scientific literature and body of practice on both the theory and technique of contingent valuation. EPA believes that well-designed and well-executed contingent valuation studies are useful for estimating the benefits of environmental effects such as improved visibility (EPA, 2000).

Society also values visibility because of the significant role it plays in transportation safety. Serious episodes of visibility impairment can increase the risk of unsafe air transportation, particularly in urban areas with high air traffic levels (EPA, 1982b). In some cases, extreme haze episodes have led to flight delays or the shutdown of major airports, resulting in economic impacts on air carriers, related businesses, and air travelers. For example, 24-hour $PM_{2.5}$ levels reached 68 $\mu g/m^3$ in St. Louis on May 15, 1998 during a haze episode attributed to wildfires in central America. This event resulted in a reduction in landing rates and significant flight delays at Lambert International Airport. In other cases, high $PM_{2.5}$ and haze levels, such as those experienced during the July 1999 air pollution episode in the northeastern U.S., have played a role in air transportation accidents and loss of life. (NTSB, 2000). During this episode, 24-hour levels of $PM_{2.5}$ ranged from 35-52 $\mu g/m^3$ in the New England states.

5.2.5.2 Visibility Goals and Programs

The value placed on protecting visual air quality is further demonstrated by the existence of a number of programs, goals, standards, and planning efforts that have been established in the U.S. and abroad to address visibility concerns in urban and non-urban areas. These regulatory and planning activities are of particular interest here to the extent that they are illustrative of the significant value that the public places on improving visibility, and because they have developed approaches and methods for evaluating public perceptions and judgments about the acceptability of varying degrees of visibility impairment that can be applied to develop additional information to help inform this review of the secondary PM NAAQS. Specific discussion is provided below on the statutory focus on visibility impairment in the U.S. Clean Air Act (CAA) and on the methods for evaluating public perceptions and judgments developed in conjunction with the establishment of a visibility standard in Denver.

Other examples of regulatory and planning activities in the U.S. include the establishment of visibility standards by the State of California (California Code of Regulations) and the Lake

Tahoe Regional Planning Agency (Molenar, 2000), and the initiative known as the Governor's Brown Cloud Summit in Phoenix, Arizona, for the future establishment of citizen-defined visibility goals using a citizen survey process similar to the Denver approach (Arizona Department of Environmental Quality, 2001).[2] International activities include the establishment of a visibility objective in the Australian state of Victoria (State Government of Victoria, 2000a and 2000b), the ongoing development of a visibility guideline in New Zealand (New Zealand National Institute of Water & Atmospheric Research, 2000a and 2000b; New Zealand Ministry of Environment, 2000), and field studies undertaken to characterize visibility and ambient aerosol loadings in southwestern British Columbia (Pryor, 1996), based on the methodology used by Ely et al. (1991) in setting the Denver visibility standard.

Sections 169A and 169B of the CAA. In addition to the recognition in sections 109 and 302(h) of the CAA that visibility impairment is a welfare effect that is to be protected by secondary NAAQS, additional protection of visibility impairment was outlined in sections 169A and 169B of the Act. Section 169A of the 1977 CAA Amendments established a national visibility goal to "remedy existing impairment and prevent future impairment" in 156 national parks and wilderness areas (Class I areas). The Amendments also called for EPA to issue regulations requiring States to develop long-term strategies to make "reasonable progress" toward the national goal. EPA issued initial regulations in 1980 focusing on visibility problems that could be linked to a single source or small group of sources. At this time, EPA deferred action on regional haze until monitoring, modeling, and source apportionment methods could be improved.

The 1990 CAA Amendments placed additional emphasis on regional haze issues through the addition of section 169B. In accordance with this section, EPA established the Grand Canyon Visibility Transport Commission (GCVTC) in 1991 to address adverse visibility impacts on 16 Class I national parks and wilderness areas on the Colorado Plateau. The GCVTC was comprised of the Governors of nine western states and leaders from a number of Tribal nations. The

[2]For illustrative purposes, Figures 27 to 34 in Appendix B show visual air quality in Phoenix under a range of visibility conditions. The images were generated using the WinHaze program, version 2.8.0, a state-of-the-art image modeling program developed by Air Resource Specialists, Inc.

GCVTC issued its recommendations to EPA in 1996, triggering a requirement in section 169B for EPA issuance of regional haze regulations.

EPA promulgated the final regional haze rule in 1999. The rule was developed with the benefit of many years of visibility research. Two key reports providing a technical basis for the rule were the 1991 NAPAP report and the 1993 National Academy of Sciences report on visibility in national parks and wilderness areas. The latter report concluded that "current scientific knowledge is adequate and control technologies are available for taking regulatory action to improve and protect visibility" (National Research Council, 1993).

Under the regional haze program, States are required to establish goals for improving visibility on the 20% most impaired days in each class I area, and for allowing no degradation on the 20% least impaired days. Each state must also adopt emission reduction strategies which, in combination with the strategies of contributing States, assure that class I area visibility improvement goals are met. The first State implementation plans are to be adopted in the 2003-2008 time period, with the first implementation period extending until 2018. Five multistate planning organizations are evaluating the sources of $PM_{2.5}$ contributing to Class I area visibility impairment to lay the technical foundation for developing strategies coordinated among many States in order to make reasonable progress in Class I areas across the country.

Denver Visibility Program and Standard-Setting Methodology. The State of Colorado adopted a visibility standard for the city of Denver in 1990.[3] Of particular interest here is the process by which the Denver visibility standard was developed, which relied on citizen judgments of acceptable and unacceptable levels of visual air quality (Ely et al., 1991).

Representatives from the Colorado Department of Public Health and Environment (CDPHE) conducted a series of meetings with 17 civic and community groups in which a total of 214 individuals were asked to rate slides having varying levels of visual air quality for a well-known vista in Denver. The CDPHE representatives asked the participants to base their judgments on three factors: 1) the standard was for an urban area, not a pristine national park area

[3] The Denver standard is violated when the four-hour average light extinction exceeds 76 Mm-1 (equivalent to approximately 32 miles visual range and 20 deciviews) during the hours between 8 a.m. and 4 p.m. Transmissometer readings taken when relative humidity is greater than 70% are excluded.

where the standards might be more strict; 2) standard violations should be at visual air quality levels considered to be unreasonable, objectionable, and unacceptable visually; and 3) judgments of standards violations should be based on visual air quality only, not on health effects.

The participants were shown slides in 3 stages. First, they were shown seven warm-up slides describing the range of conditions to be presented. Second, they rated 25 randomly-ordered slides based on a scale of 1 (poor) to 7 (excellent), with 5 duplicates included. Third, they were asked to judge whether the slide would violate what they would consider to be an appropriate urban visibility standard (i.e. whether the level of impairment was "acceptable" or "unacceptable").

The Denver visibility standard-setting process produced the following findings:

- Individuals' judgments of a slide's visual air quality and whether the slide violated a visibility standard are highly correlated (Pearson correlation coefficient greater than 80%) with the group average.

- When participants judged duplicate slides, group averages of the first and second ratings were highly correlated.

- Group averages of visual air quality ratings and "standard violations" were highly correlated. The strong relationship of standard violation judgments with the visual air quality ratings is cited as the best evidence available from this study for the validity of standard violation judgments (Ely et al., 1991).

The ratings for each slide were sorted by increasing order of light extinction, and the percentage of participants that judged each slide to violate the "standard" was calculated. The Denver visibility standard was then established based on a 50% acceptability criterion. Under this approach, the standard was identified as the light extinction level that divides the slides into two groups: those found to be acceptable and those found to be unacceptable by a majority of study participants. For illustrative purposes, Figures 19 to 26 in Appendix B show visual air quality in Denver under a range of visibility conditions (generally corresponding to 10^{th}, 20^{th}, 30^{th}, 40^{th}, 50^{th}, 60^{th} 80^{th}, and 90^{th} percentile values). These images were generated using the WinHaze program, version 2.8.0, a state-of-the-art image modeling program developed by Air Resource Specialists, Inc.

5.2.6 Evaluating Public Perceptions of Visibility Impairment

New tools and methods are now available to communicate and evaluate public perceptions of varying visual effects associated with alternative levels of visibility impairment relative to varying pollution levels and environmental conditions. As described above in Section 5.2.5.2, these tools and methods have been used by others as a basis for developing goals and standards for visibility. Building upon this work, EPA has initiated a project to evaluate public perceptions of visibility impairment in urban areas, and intends to consider using the information developed in this project to help inform the review of the secondary PM NAAQS. In particular, new techniques for photographic representation of visibility impairment are discussed below, followed by a discussion of the survey approach used in the pilot phase of this project and the plans for the continuation of this project.

Staff welcomes CASAC and public input on the information presented below, including the photographic techniques and survey methods planned for use in this project, and the appropriateness of using the results from this project to help inform our review of the secondary PM NAAQS.

5.2.6.1 Photographic Representations of Visibility Impairment

In the past, the principal method for recording and describing visual air quality has been through 35 millimeter photographs. Under the IMPROVE program, EPA and its optical monitoring contractor Air Resource Specialists, Inc. (ARS) have developed an extensive archive of visual air quality photos for national parks and wilderness areas. In comparison, we have only a limited archive of photos of urban areas.

The draft CD discusses some of the methods that are now available to represent different levels of visual air quality (CD, p. 4-107). In 1994, Molenar described a sophisticated visual air quality simulation technique in Atmospheric Environment (Molenar, 1994). This technique, a combination of modeling systems under development for the past 20 years, was developed by ARS.

The technique relies on first obtaining an original base image slide of the scene of interest. The slide should be of a cloudless sky under the cleanest air quality conditions possible. The light extinction represented by the scene should be derived from aerosol and optical data associated

with the day the image was taken, or it should be estimated from contrast measurements of features in the image. The image is then digitized to assign an optical density to each pixel. At this point, the radiance level for each pixel is estimated. Using a detailed topographic map, technicians identify the specific location from which the photo was taken, and they determine the distances to various landmarks and objects in the scene. With this information, a specific distance and elevation is assigned to each pixel.

Using the digital imaging information above, the system then computes the physical and optical properties of an assumed aerosol mix. These properties are input into a radiative transfer model in order to simulate the optical properties of varying pollutant concentrations on the scene. ARS now provides WinHaze, version 2.8.0, an image modeling program for personal computers that employs simplified algorithms based on the sophisticated modeling technique developed by Molenar.

An alternative technique would be to obtain actual photographs of the site of interest at different ambient pollution levels. However, long-term photo archives of this type exist for only a few cities. In addition, studies have shown that observers will perceive an image with a cloud-filled sky as having a higher degree of visibility impairment than one without clouds, even though the PM concentration on both days is the same. The simulation technique has the advantage that it can be done for any location as long as one has a very clear base photo. In addition, the lack of clouds and consistent sun angle in all images in effect standardizes the perception of the images and enables researchers to avoid potentially biased responses due to these factors.

5.2.6.2 Pilot Project: Assessing Public Opinions on Air Pollution-Related Visibility Impairment

The pilot project described here uses the latest techniques for photographic representation of visibility impairment and survey techniques applied by others as a basis for setting visibility goals and standards. Staff developed this project to provide information that may be useful in the EPA's review of the secondary PM NAAQS. The project is premised on the view that public perceptions of and judgments about the acceptability of visibility impairment in urban areas are relevant factors in assessing what constitutes an adverse level of visibility impairment in the context of this NAAQS review.

With this in mind, staff considered various approaches for obtaining public input on visibility impairment. Potential options included a mail survey, a web-based computer survey, a computer-based survey in a public location, and face-to-face meetings with survey participants. As discussed below, one important issue that staff considered in selecting a preferred option involved how to develop images that graphically represent subtle differences in pollutant concentrations and air quality, and selecting the appropriate media for communicating these images to public citizens. Another issue was how to ensure consistency in the way in which participants in any such survey would receive and process this information, recognizing that the method used to conduct the survey (e.g., mail delivery, presentations to small groups) could affect this consistency since the methods differ in the extent of control that the researchers have of the survey process.

Developing Images. The options for presenting images include web-based digital images viewed on computer monitors, print photos, video or DVD, and 35 millimeter slides. Thirty-five millimeter slides generally provide the highest resolution, and the researcher can have a high level of control in how they are presented. As discussed above, this approach was used by Colorado Department of Public Health and Environment staff in its research leading to development of the Denver visibility standard. Large format print photos also have high resolution, but are more costly than slides. The best quality computer monitors can also provide high resolution, but resolution varies greatly from monitor to monitor if the images were provided on the internet. Creating multiple copies of print photos to accompany a mail survey would be quite expensive,

and there would be little control in how the photos would be presented. Taking all of this into account, staff decided to use high resolution 35 mm slides presented to a small group of people at a time.

Having made this decision on image media, staff decided to pursue a pilot project similar to the Denver study that used the ARS visual air quality modeling technique to communicate different levels of visibility impairment to members of the general public. EPA contracted with ARS to develop a series of 27 images of a scene in Washington, DC, consistent with the approach described above. ARS developed this slide series for a vista of Washington, DC as viewed from across the Potomac River near Arlington Cemetery. The vista includes the Mall in downtown Washington, DC and several well-known landmarks, including the Lincoln Memorial, Washington Monument, Capitol Building, Union Station, and Library of Congress. The sight path to the farthest landmark in the scene (the Anacostia neighborhood) is fairly short – approximately 8 km. The base image was taken on a clear day with no cloud cover.

The slides illustrate visual air quality associated with $PM_{2.5}$ concentrations across a broad range of possible conditions, ranging from 2.3 $\mu g/m^3$ to 65 $\mu g/m^3$. Figures 6 and 10 in Appendix B show Washington, DC at 15 $\mu g/m^3$ and 65 $\mu g/m^3$ levels, respectively. The same pollutant mix was used to make each slide so that changes in visual air quality from slide to slide could be attributed solely to changes in PM mass concentrations. For each image, the percent of total $PM_{2.5}$ mass assigned to each component was chosen based on annual average values derived from data collected at the Washington, DC IMPROVE monitoring site from 1988 to 1999. For each $PM_{2.5}$ level, the assumed pollutant mix was as follows: sulfate = 50%; nitrate = 10%; organic carbon = 25%; elemental carbon = 10%; fine soil = 5%.

Coarse-fraction particles also cause light scattering, but are less efficient per unit mass. Based on the relationship of PM_{10} and $PM_{2.5}$ values from Washington, DC IMPROVE data (1988-99), a standard mass value was assigned to PM_{10} for each image equal to 30 % of the $PM_{2.5}$ mass. A standard value of 10 Mm-1 was assumed for Rayleigh scattering. Light absorption by gases is commonly attributed to NO_2, which gives a brownish cast to the sky color, particularly in urban areas. Based on a review of recent AIRS data for Washington, DC, an annual average value of 16 ppb was assumed for NO_2 and taken into account in the image modeling process. Finally, the

images were generated using an assumed annual average relative humidity of 68% (corresponding to an f(RH) factor of 2.98 for calculating light extinction due to sulfates and nitrates). This annual average relative humidity value was derived from National Weather Service data from nearby airports.

Appendix B includes the specific data and the photographic images used in the pilot survey. In particular, Tables 1 and 2 in Appendix B provide the pollutant concentrations and the calculated visibility parameters (i.e., light extinction, visual range, and deciviews), respectively, used to create each slide. Figures 3 through 10 in Appendix B display images of Washington, DC representing 24-hour $PM_{2.5}$ levels of 2.5, 5, 10,15, 20, 30, 40, and 65 $\mu g/m^3$, respectively. Series of images are also provided in Appendix B for Chicago, Illinois (Figures 11-16), Denver, Colorado (Figures 19-26), and Phoenix, Arizona (Figures 27-34).

Focus Group Process and Pilot Survey. EPA contracted with Abt Associates to coordinate the implementation of a pilot focus group session, held on November 16, 2000 in Bethesda, Maryland. The session was designed based on the approach used for the Denver study (see Section 5.2.2.2 above and Ely et al., 1991).[4] This same approach has been successfully implemented by other researchers as well (Pryor, 1996; Hill et al., 2000). The purpose of the pilot focus group session was to evaluate the initial survey process and survey questions so as to refine the approach for future sessions to be held in different cities around the country. Abt Associates summarized the conduct and results from the pilot focus group session in a January 2001 report (Abt Associates, 2001). This report is available for review.

More specifically, six female and three male participants from Maryland, Virginia, and the District of Columbia were invited to participate in the session. Demographically, the group represented a balanced range of ages, races, education levels, and income levels. The session was held in a large meeting room with a one-way mirror for observation by EPA and Abt representatives. Two representatives from Abt Associates facilitated the session. The 35 mm

[4] Methods for the Denver study were based on previous research conducted by the National Park Service (Malm et al., 1981) and National Center for Atmospheric Research (Stewart et al., 1983). The results from these studies have shown that judgments of visual air quality by private citizens are valid and reliable. They also have shown that judgments made from one group to another are highly correlated, and that judgments made from slides are highly correlated to those made in the field (Ely et al., 1991).

slides were displayed on an eight-foot matte screen using a Kodak AMT Ektagraphic projector with a high quality projection lens (f2.8). The participants were located approximately 9 to 13 feet from the projection screen.

The session involved viewing slides in three steps as discussed in the overview of the Denver study. In designing the session, representatives from EPA and Abt Associates decided that to address time constraints and the subtlety of changes between some of the slides with higher $PM_{2.5}$ concentrations, a subset of the 25 slides should be shown. Accordingly, a set of 20 of the 25 original slides were selected for the pilot session. Five duplicates were selected at random and added to the set of 20 originals, resulting in a total set of 25 slides.

The participants were first shown a series of four "warm-up" slides representing the full range of visual air quality conditions they were about to view. Next, the participants were shown the 25 slides in random order and asked to rate the visual air quality of each slide on a seven-point scale, ranging from "Very Poor" to "Very Good." A cumulative score was calculated for each slide by assigning 1 (very poor) to 7 (very good) points to each participant's response, with 63 being the highest cumulative score a slide could receive from the group. Based on the results, it appears that the participants were able to perceive subtle differences between slides in a consistent manner. The cumulative scores for each slide are shown in Figure 17 in Appendix B.

In the final step of the rating process, the participants viewed the slides in a random order again, and were asked to rate the slide as "acceptable" or "unacceptable." They were asked to consider only the visual air quality of the scene, not any assumed public health consequences, nor the potential costs of improving conditions to an "acceptable" level. The results showed three distinct "zones" resulting from the rating process:

- "Acceptable" zone: the set of slides found to be "acceptable" by most participants. (In this case, the acceptable zone generally included slides for 15 $\mu g/m^3$ and less.)

- "Unacceptable" zone: the set of slides found to be "unacceptable" by most participants. (In this case, the unacceptable zone generally included slides for 40 $\mu g/m^3$ and above.)

- "Intermediate" zone: the remaining set of slides, for which there were varying degrees of "acceptable" and "unacceptable" ratings.

Figure 18 in Appendix B illustrates the number of respondents who rated each slide as acceptable or unacceptable. This basic pattern of responses is similar to that found in the Denver study. Staff expects that the results from future meetings to obtain citizen input will also show three basic rating "zones." One objective of a broader survey of citizens will be to see if the $PM_{2.5}$ levels shaping these zones are relatively consistent or highly variable from one region of the country to another.

After the slide rating portion of the session, EPA staff joined the group for a discussion to evaluate the session design. In this part of the session, staff reviewed the survey questions with the participants to determine whether some questions were difficult to understand and needed clarification. We also asked the participants to comment on whether they took health effects or weather effects into account in the rating process. Regarding health effects, staff purposefully designed the survey questions to emphasize that the visual air quality (VAQ) ratings should be based only on the participant's judgment of the visibility level, and should not involve any assumptions about negative health effects that might be experienced from such a VAQ level. The respondents agreed that the survey should not take health effects into account since this could lead to biased responses. Regarding weather effects, some participants stated that some of the hazier images looked like there was a heavy fog present. It was recommended that in future sessions, the facilitator should emphasize that the weather condition in each slide is the same (e.g. a cloudless day), with no fog or precipitation in the air. The summary report for the pilot session includes discussion of a number of other questions asked during the session and potential design improvements (Abt Associates, 2001).

Planned Focus Group Survey. During 2001-2002, staff is planning to conduct additional survey sessions to obtain citizen input on visual air quality in New York City; Asheville, NC; Chicago; Seattle; San Francisco; and at least one other western city to be determined. EPA has contracted with ARS for the development of a high quality slide series for each of these cities. EPA intends to contract with a consulting firm to coordinate the sessions, as was done for the pilot session. The purpose of these additional citizen input sessions will be to evaluate the consistency of citizen responses from one region of the country to another.

5.3 EFFECTS ON MATERIALS

The effects of the deposition of atmospheric pollution, including ambient PM, on materials are related to both physical damage and aesthetic qualities. The deposition of PM (especially sulfates and nitrates) can physically affect materials, adding to the effects of natural weathering processes, by potentially promoting or accelerating the corrosion of metals, by degrading paints, and by deteriorating building materials such as concrete and limestone. Particles contribute to these physical effects because of their electrolytic, hygroscopic and acidic properties, and their ability to sorb corrosive gases (principally SO_2). As noted in the last review, only chemically active fine-mode or hygroscopic coarse-mode particles contribute to these physical effects (EPA 1996b, p. VIII-16).

In addition, the deposition of ambient PM can reduce the aesthetic appeal of buildings and culturally important articles through soiling. Particles consisting primarily of carbonaceous compounds cause soiling of commonly used building materials and culturally important items such as statues and works of art (CD, p. 4-114). Soiling is the deposition of particles on surfaces by impingement, and the accumulation of particles on the surface of an exposed material results in degradation of its appearance. Soiling can be remedied by cleaning or washing, and depending on the soiled material, repainting (EPA, 1996b, p. VIII-19).

Building upon the information presented in the last Staff Paper (EPA, 1996b), and including the limited new information presented in Chapter 4 of the draft CD, the following sections summarize the physical damage and aesthetic soiling effects of PM on materials including metals, paint finishes, and stone and concrete.

5.3.1 Materials Damage Effects

Physical damage such as corrosion, degradation, and deterioration occurs in metals, paint finishes, and building materials such as stone and concrete, respectively. Metals are affected by natural weathering processes even in the absence of atmospheric pollutants. Atmospheric pollutants, most notably SO_2 and particulate sulfates, can have an additive effect, by promoting and accelerating the corrosion of metals. The rate of metal corrosion depends on a number of factors, including the deposition rate and nature of the pollutants; the influence of the protective

corrosion film that forms on metals, slowing corrosion; the amount of moisture present; variability in electrochemical reactions; the presence and concentration of other surface electrolytes; and the orientation of the metal surface. Historically, studies have shown that the rate of metal corrosion decreases in the absence of moisture, since surface moisture facilitates the deposition of pollutants and promotes corrosive electrochemical reactions on metals.

The draft CD (p. 4-117, Table 4-8) summarizes the results of a number of studies investigating the roles of particles (e.g., particulate sulfates) and SO_2 on the corrosion of metals. The draft CD concludes that the role of particles in the corrosion of metals is not clear (CD, p. 4-116). While several studies suggest that particles can promote the corrosion of metals, others have not demonstrated a correlation between particle exposure and metal corrosion. Although the corrosive effects of SO_2 exposure in particular have received much study, there remains insufficient evidence to relate corrosive effects to specific particulate sulfate levels or to establish a quantitative relationship between ambient particulate sulfate and corrosion.

Similar to metals, paints also undergo natural weathering processes, mainly from exposure to environmental factors such as sunlight, moisture, fungi, and varying temperatures. Beyond these natural processes, atmospheric pollutants can affect the durability of paint finishes by promoting discoloration, chalking, loss of gloss, erosion, blistering, and peeling. Historical evidence indicates that particles can damage painted surfaces by serving as carriers of more corrosive pollutants, most notably SO_2, allowing the pollutants to reach the underlying surface, or by serving as concentration sites for other pollutants. A number of studies available in the last review showed some correlation between PM exposure and damage to automobile finishes. In particular, Wolff et al. (1990) concluded that damage to automobile finishes resulted from calcium sulfate forming on painted surfaces by the reaction of calcium from dust particles and sulfuric acid contained in rain or dew. In addition, paint films permeable to water are also susceptible to penetration by acid forming aerosols (EPA 1996b, p. VIII-18). The erosion rate of oil-based house paint has been reported to be enhanced by exposure to SO_2 and humidity; several studies have suggested that the effect of SO_2 is caused by its reaction with extender pigments such as calcium carbonate and zinc oxide, although Miller et al. (1992) suggests that calcium carbonate acts to protect paint substrates (CD, p. 4-119).

With respect to damage to building stone, numerous studies discussed in the draft CD (p. 4-120, Table 4-9) suggest that air pollutants, including sulfur-containing pollutants and atmospheric particles including gypsum, can enhance natural weathering processes. Exposure-related damage to building stone results from the formation of salts in the stone that are subsequently washed away by rain, leaving the surface more susceptible to the effects of air pollutants. Dry deposition of sulfur-containing pollutants and carbonaceous particles promotes the formation of gypsum on the stone's surfaces. Gypsum is a black crusty material that occupies a larger volume than the original stone, causing the stone's surface to become cracked and pitted, leaving rough surfaces that serve as sites for the deposition of airborne particles (CD, page 4-124).

The rate of deterioration of building stone is determined by the pollutant mix and concentration, the stone's permeability and moisture content, and the pollutant deposition velocity. Dry deposition of SO_2 between rain events has been reported to be a major causative factor in pollutant-related erosion of calcareous stones (e.g., limestone, marble, and carbonated cement). While it is clear from the available information that gaseous air pollutants, in particular SO_2, will promote the decay of some types of stones under specific conditions, carboneous particles (non-carbonate carbon) and particles containing metal oxides may help to promote the decay process (CD, p. 4-125).

5.3.2 Soiling Effects

Soiling affects the aesthetic appeal of painted surfaces, including culturally important articles, and stone surfaces. In addition to natural factors, exposure to PM may give painted surfaces a dirty appearance, although few studies are available that evaluate the soiling effects of particles (CD, p. 4-127). Early studies demonstrated an association between particle exposure and increased frequency of cleaning painted surfaces. More recently, Haynie and Lemmons (1990) conducted a study to determine how various environmental factors contribute to the rate of soiling on white painted surfaces. They reported that coarse-mode particles initially contribute more to soiling of horizontal and vertical surfaces than do fine-mode particles, but are more easily removed by rain, leaving stains on the painted surface. The authors concluded that the

accumulation of fine-mode particles, rather than coarse-mode particles, more likely promotes the need for cleaning of the painted surfaces (EPA 1996b, p. VIII-21-22). Creighton et al. (1990) reported that horizontal surfaces soiled faster than vertical surfaces and that large particles were primarily responsible for the soiling of horizontal surfaces not exposed to rainfall. Additionally, a study was conducted to determine the potential soiling of artwork in five Southern California museums (Ligocki, et al., 1993). Findings were that a significant fraction of fine elemental carbon and soil dust particles in the ambient air had penetrated to the indoor environment and may constitute a soiling hazard to displayed artwork (EPA 1996b, p. VIII-22).

As for stone structures, the presence of gypsum is related to soiling of the stone surface by providing sites for particles of dirt to concentrate. Lorusso et al. (1997) attributed the need for frequent cleaning and restoration of historic monuments in Rome to exposure to total suspended particles (TSP). Further, Davidson et al. (2000) evaluated the effects of air pollution exposure on a limestone structure on the University of Pittsburgh campus using estimated average TSP levels in the 1930s and 1940s and actual values for the years 1957 to 1997. Monitored levels of SO_2 were available for the years 1980 to 1998. Based on the available data on pollutant levels and photographs, it was thought that soiling began while the structure was under construction. With decreasing levels of pollution, the soiled areas have been slowly washed away, the process taking several decades, leaving a white, eroded surface (CD, pages 4-126 to 4-127).

5.3.4 Summary

Damage to building materials results from natural weathering processes that are enhanced by exposure to airborne pollution, most notably sulfur-containing pollutants. While ambient PM has been associated with contributing to pollution-related damage to materials, the draft CD concludes that insufficient data exist to relate such effects to specific particle pollution levels, particle size, or chemical composition (CD, p. 4-163). In addition to contributing to physical damage, particle pollution can cause significant detrimental effects by soiling painted surfaces and other building materials. Available data indicate that particle-related soiling can result in increased cleaning frequency and repainting, and may reduce the useful life of the soiled materials.

However, again the draft CD concludes that insufficient data are available to relate soiling effects to specific particle pollutant levels, particle size, or chemical composition (CD, p.4-163).

5.4 EFFECTS ON VEGETATION AND ECOSYSTEMS

Environmental impacts of ambient PM are considered here in relation to effects on vegetation and other components of the environment, such as soils, water, and wildlife, that make up ecosystems. Observed effects can result from the physical and chemical properties of PM and may be caused directly by particle deposition onto the affected vegetation or indirectly through deposition to soils or water. However, the draft CD notes that particle deposition to vegetation and ecosystems is not well understood at this time (CD, p. 4-2). Available evidence does suggest that all modes of deposition must be considered in determining potential impacts to vegetation and ecosystems including: 1) wet deposition in which particles are deposited in rain and snow; 2) occult deposition in which particles are deposited in fog, cloud-water and mists; and 3) dry deposition in which particles are deposited onto surfaces (CD, p. 4-3). Wet deposition is generally more effective for removing fine-mode PM from the atmosphere, whereas dry deposition is more effective for coarse-mode particles.

Based on information contained and referenced in Chapter 4 of the draft CD, the effects of ambient PM alone and in combination with other pollutants are summarized below, focusing first on direct effects on vegetation, then more broadly and importantly on direct and indirect effects on ecosystems.

5.4.1 Direct Effects on Vegetation

Particulate matter that deposits directly from the atmosphere onto above-ground plant surfaces may (1) reside on the leaf, twig, or bark surface for an extended period; (2) be taken up through the leaf surface; or 3) be removed from the plant via resuspension to the atmosphere, washing off by rainfall, or litter-fall with subsequent transfer to the soil (CD, p.4-6). The following discussion focuses on those particles that are intercepted by and remain on the leaves. Most information currently available on plant effects focuses on nitrate particle deposition, in particular, and more generally on acidic deposition, primarily from nitrogen- and sulfur-

containing particles and gaseous pollutants. Depending on the amount and composition of the deposited PM, effects can be either physical, chemical, or both.

Physical effects of PM occur mainly in areas where deposition rates for particles in the coarse mode are high, in some cases leading to crust formation on plant leaves, such as near roadways, agricultural areas and industrial sites. Physical effects that have been observed in vegetation in such areas include reduced photosynthesis and subsequent reductions in carbohydrate formation, root and plant growth; blockage of the stomata preventing adequate gas exchange; changes in leaf temperature (e.g., heat stress); destruction of leaf tissue (e.g., chlorosis, necrosis, and/or abscission); and premature leaf-fall. (CD, pp. 4-7 to 4-8).

In most areas, however, where deposition rates are not high enough for significant physical effects from PM to occur, the chemical composition of PM becomes the key phytotoxic factor leading to plant injury. Often, it is the chemical composition or class of PM in the fine mode that produces phytotoxic effects when deposited onto plant surfaces, as discussed below first for nitrates and other acidic particles, and then for trace metals and organics. However, studies of the direct effects of chemical additions to foliage through particle deposition have found little or no effects of PM on foliar processes unless exposure levels were significantly higher than typically would be experienced in the ambient environment. Further, only a few studies have been completed on the direct effects of fine-mode particles on vegetation, and the conclusion that was reached in the 1982 PM Criteria Document (EPA, 1982), that sufficient data were not available for adequate quantification of dose-response functions, continues to be true today (CD, pp. 4-6 to 4-9).

Acidic Deposition. Nitrogen has long been recognized as the nutrient most important for plant growth. For instance, approximately 75% of the nitrogen in a plant leaf is used during the process of photosynthesis, and to a large extent, it governs the utilization of phosphorus, potassium, and other nutrients. Particle deposition of nitrate, together with other nitrogen-containing gaseous and precipitation-derived sources, represent a substantial fraction of total nitrogen reaching vegetation. However, much of this nitrogen is contributed by gaseous nitric acid vapor, and a considerable amount of the particulate nitrate is taken up indirectly through the soil (CD, p. 4-9). Though plants usually absorb nitrogen (as NH_4^+ or NO_3^-) through their roots,

it is known that foliar uptake of nitrate can occur. However, the mechanism of foliar uptake is not well established, plants vary in their ability to absorb ammonium and nitrate, and it is not currently possible to distinguish sources of chemicals deposited as gases or particles using foliar extraction. Since it has proven difficult to quantify the percentage of nitrogen uptake by leaves that is contributed by ambient particles, direct foliar effects of nitrogen-containing particles have not been documented. (CD, pp. 4-10 to 4-11; 4-41 to 4-42).

Similar to nitrogen, sulfur is an essential plant nutrient that can deposit on vegetation in the form of sulfate particles, or be taken up by plants in gaseous form. Greater than 90% of anthropogenic sulfur emissions are as sulfur dioxide (SO_2), with most of the remaining emissions in the form of sulfate. However, sulfur dioxide is rapidly transformed in the atmosphere to sulfate, which is approximately 30-fold less phytotoxic than SO_2. Low dosages of sulfur can serve as a fertilizer, particularly for plants growing in sulfur-deficient soils. There are only a few field demonstrations of foliar sulfate uptake, however, and the relative importance of foliar leachate and prior dry-deposited sulfate particles remains difficult to quantify. Though current levels of sulfate deposition reportedly exceed the capacity of most vegetative canopies to immobilize the sulfur, sulfate additions in excess of needs do not typically lead to plant injury. Additional studies are needed, however, on the effects of sulfate particles on physiological characteristics of plants following chronic exposures (CD, pp. 4-11 to 4-12).

Though dry deposition of nitrate and sulfate particles does not appear to induce foliar injury at current ambient exposures, when found in acidic precipitation, they do have the potential to cause direct foliar injury. This is especially true when the acidic precipitation is in the form of fog and clouds, which may contain solute concentrations up to 10 times those found in rain. In experiments on seedling and sapling trees, both coniferous and deciduous species showed significant effects on leaf surface structures after exposure to simulated acid rain or acid mist at pH 3.5, while some species have shown subtle effects at pH 4 and above. Epicuticular waxes, which function to prevent water loss from plant leaves, can be destroyed by acid rain in a few weeks which suggests links between acidic precipitation and aging. Due to their longevity and evergreen foliage, the function of epicuticular wax is more crucial in conifers. For example, red spruce seedlings, which have been extensively studied, appear to be more sensitive to acid

precipitation (mist and fog) when compared with other species (CD, pp. 4-13 to 4-14). In addition to accelerated weathering of leaf cuticular surfaces, other direct responses of forest trees to acidic precipitation include increased permeability of leaf surfaces to toxic materials, water, and disease agents; increased leaching of nutrients from foliage; and altered reproductive processes (CD, p. 4-29). All of these effects serve to weaken trees so that they are more susceptible to other stresses (e.g., extreme weather, pests, pathogens).

Trace elements. Of the 90 elements that make up the inorganic fraction of the soil, 80 exist in concentrations of less than 0.1% and are known as "trace elements". Trace elements with a density greater than 6 g/cm^3 are referred to as "heavy metals". Although some trace metals are essential for vegetative growth or animal health, in large quantities, they are all toxic. Most trace metals found in the atmosphere are produced by industrial combustion processes and exist predominantly as metal chloride particles, which tend to be volatile, or as metal oxides, which tend to be nonvolatile and in the vapor phase. Heavy metals introduced into the atmosphere from human activities include antimony, cadmium, chromium, copper, lead, molybdenum, mercury, nickel, silver, tin, vanadium, and zinc (CD, p. 4-15).

Investigations of trace elements present along roadsides and in industrial and urban environments have indicated that impressive burdens of particulate heavy metal can accumulate on vegetative surfaces. Once on the surface, these metals can potentially impact either the metabolism of above-ground plant tissues or the activity of populations of organisms resident on and in the leaf surface (e.g., bacteria, fungi and arthropods). In the first scenario, a trace metal must be brought into solution before it can enter into the leaves or bark of vascular plants. Since the solubility of most trace metals is low, foliar uptake and direct heavy metal toxicity is limited. In those instances when trace metals are absorbed, they are frequently bound in leaf tissue and are lost when the leaf later drops off. Only a few metals have been documented to cause direct phytotoxicity in field conditions, with copper, zinc and nickel toxicities observed most frequently. It is unlikely, therefore, that deposition of trace metals to vegetative surfaces at ambient levels is causing wide spread acute plant toxicity. In the second scenario, little experimental data exists on the effects of trace metals on leaf surface organisms, though trace metal toxicity of lichens has been demonstrated in a few cases (CD, pp. 4-16 to 4-17).

On the other hand, the effects of chronic low-level metal deposition on perennial plant species may be more significant than the acute effects referred to above. When trees are exposed to sub-lethal concentrations of heavy metals, levels of intracellular metal-binding peptides, phytochelatins, increase. In studies designed to test the relationship between heavy metals and the decline of forest tree species in certain areas in the U.S., the data showed a systematic and significant increase in phytochelatin concentrations associated with the extent of tree injury. Though there has been no direct evidence of a physiological association between tree injury and exposure to metals, metals have been implicated because their deposition pattern has been correlated with the decline of certain tree species. (CD, pp. 4-16 to 4-17).

Organics. Many different chemical compounds can fall under the generic classification of "organics". These compounds may also be referred to as toxic substances, pesticides, hazardous air pollutants (HAPs), air toxics, semivolatile organic compounds (SOCs), and persistent organic pollutants (POPs). While these substances are not criteria pollutants, they are discussed here because many of these compounds partition between gas and particle phases and are removed from the atmosphere by both wet and dry deposition.. As particles they can become airborne, be distributed over wide areas, and impact remote ecosystems. Some notable organics include such compounds as DDT, polychlorinated biphenyls (PCBs), and polynuclear aromatic hydrocarbons (PAHs). These substances may enter plants via the roots, be deposited as particles onto the waxy cuticle of leaves or be taken up through the stomata. Which pathway is followed is a function of the chemical and physical properties of the pollutant, environmental conditions, and the plant species. However, the direct uptake of organic contaminants through the cuticle or in the vapor phase through the stomates are poorly characterized for most trace organics. Additionally, the toxicity of organic contaminants to plants and soil microorganisms is not well studied (CD, pp. 4-18 to 4-19).

5.4.2 Ecosystem Effects

As discussed in the draft CD, human existence on this planet depends on the life-support services ecosystems provide. Both ecosystem structure and function play essential roles in providing societal benefits, including products with market value (e.g., fish, minerals, forest

products, biomass fuels, natural fibers, pharmaceuticals) as well as the use and appreciation of natural areas for recreation, aesthetic enjoyment, and study. In addition, ecosystem functions play a major role in maintaining necessary atmospheric, climatic, and radiative balances within our environment (e.g., absorbing pollution, cycling nutrients, degrading wastes) (CD, p. 4-156). The draft CD provides a detailed discussion of the nature of ecosystems, the services they provide, and their response to stress (CD, pp. 4-20 to 4-25).

Ecosystem-level responses occur when the effects of particulate deposition on the biological and physical components of ecosystems become sufficiently widespread as to impact essential processes such as cycling of nutrients and materials. Such responses can be a result of physical effects caused by high levels of PM dust being deposited directly onto vegetative surfaces over a large portion of a plant community, or more importantly, from the chemical effects resulting from the chemical constituents of PM deposited directly onto vegetative surfaces or indirectly through deposition into soil and water environments.

Plant community structure is determined by sampling the various strata within the community (e.g., herbs, seedlings, saplings, trees). Long-term changes in the structure and composition of the strata within plant communities exposed to chronic dust accumulation have been observed, demonstrating that the physical effects of dust accumulation favors the growth of some species and limits others. Specifically, at an experimental site near limestone quarries and processing plants in southwestern Virginia, where dust accumulation occurred for at least 30 years, red maple was more abundant in all strata when compared with the control site where it was present only as a seedling. The growth of tulip poplar, dogwood, hop-hornbeam, black haw and red bud appeared to be favored by the dust, while the growth of conifers and other acid tolerant species such as rhododendron, was limited. It can be assumed that changes in soil alkalinity also occurred at the site due to the heavy deposition of limestone dust, but in the absence of soil analyses, no conclusion was reached as to the role that chemical changes to the soils may have played in these plant community changes. This site exemplifies how the direct physical effects of PM can impact ecosystems (CD, pp. 4-27 to 4-29).

Aside from its physical effects, the impact of PM on ecosystems is determined chiefly by its chemical constituents and their ability to affect the nutrient status of the ecosystem, either by

direct foliar uptake or by directly or indirectly changing soil chemistry, populations of bacteria involved in nutrient cycling, and/or populations of fungi involved in plant nutrient uptake (CD, p. 4-34).

Acidic Deposition. As discussed above, several of the chemical components of PM (e.g., nitrogen, sulfur, calcium) are essential plant nutrients. Additions of any of these nutrients, most importantly particulate nitrogen (nitrates), can affect plant succession patterns and biodiversity. Nitrogen has long been recognized as the nutrient most important for plant growth. In soils low in nitrogen, atmospherically deposited nitrogen can act as a fertilizer. However, not all plants are capable of utilizing extra nitrogen. Inputs of nitrogen to natural ecosystems that alleviate deficiencies and increase growth of some plants can impact competitive relationships and alter species composition and diversity. Plants growing in low resource environments (e.g., infertile soil, shaded understory, deserts, tundra) have been observed to have certain similar characteristics: 1) a slow growth rate, 2) low photosynthetic rate, and 3) low capacity for nutrient uptake (e.g., they tend to respond less than other plant species even when provided with an optimal supply and balance of resources). Since not all plants are equally capable of utilizing extra nitrogen, as nitrogen becomes more readily available, some plants will gain a competitive advantage and will replace those adapted to living in lower nitrogen environments (CD, pp. 4-45 to 4-46). For example, Fenn et al. (1998) report that long-term nitrogen fertilization studies in both New England and Europe suggest that some forests receiving chronic inputs of nitrogen may decline in productivity and experience greater mortality. Long-term fertilization experiments at Mount Ascutney, Vermont, suggest that declining coniferous forest stands with slow nitrogen cycling may be replaced by deciduous fast-growing forest species that cycle nitrogen rapidly (Fenn et al., 1998; CD, p. 4-47).

In some cases, additions of nitrogen above soil background levels can exceed the capacity of plants and soil microorganisms to utilize and retain it, resulting in a condition known as "nitrogen saturation." Specific ecosystem processes affected by nitrogen saturation include: 1) increased plant uptake and allocation, (i.e., a permanent increase in foliar nitrogen and reduced foliar phosphorus and lignin due to the lower availability of carbon, phosphorus, and water); 2) increased litter production, 3) increased ammonification (the release of ammonia) and trace gas

emissions, 4) decreased root biomass, 5) reduced soil fertility (the results of increased cation leaching), 6) increased nitrification (conversion of ammonia to nitrate during decay of litter and soil organic matter), and 7) nitrate leaching resulting in increased nitrate and aluminum concentrations in streams, and decreased water quality (Aber et al., 1989). In addition, studies suggest that during nitrogen saturation, soil microbial communities change from predominantly fungal (mycorrhizal) communities to those dominated by bacteria (Aber et al., 1998). Though the growth of most forests in the U.S. has been and continues to be limited by the nitrogen supply, some U.S. forests are now showing severe symptoms of nitrogen saturation, including high-elevation, non-aggrading spruce-fir ecosystems in the Appalachian Mountains, as well as in the eastern hardwood watersheds at Fernow Experimental Forest near Parsons, West Virginia. Mixed conifer forests and chaparral watersheds with high smog exposure in the Los Angeles Air Basin also are nitrogen saturated and exhibit the highest stream water NO_3^- concentrations for wildlands in North America (Bytnerowicz and Fenn, 1996; Fenn et al., 1998; CD, pp. 4-42 to 4-43). The impact of increasing nitrogen inputs on the nitrogen cycle and forests, wetlands, and aquatic ecosystems is discussed in detail elsewhere (EPA,1993, 1997a; Garner, 1994; World Health Organization, 1997). Understanding the variability in forest ecosystem response to nitrogen input is essential in assessing pollution-related impacts (CD, p. 4-49).

As noted above, sulfur is another essential plant nutrient, the most important source of which for plants is sulfate taken up by the roots, even though plants can also utilize atmospheric SO_2. Atmospheric deposition of sulfate to the soils, therefore, is an important component of the sulfur cycle. The biochemical relationship between sulfur and nitrogen in plant proteins indicates that neither element can be assessed adequately without reference to the other. Nitrogen uptake in forests may be loosely regulated by sulfur availability, but sulfate additions in excess of needs do not necessarily lead to injury. (CD, pp. 4-51 to 4-52).

The nutritional needs of plants also include a suite of other essential minerals such as calcium (Ca), magnesium (Mg) and potassium (K). Soil acidification and its effects result from the deposition of nitrate ($N0_3^-$) and sulfate (SO_4^{-2}) and the associated hydrogen (H^+) ion. The introduction of H^+ by atmospheric deposition or by internal processes will directly impact the fluxes of base cations such as Ca, K, and Mg via cation exchange or weathering processes.

Therefore, soil leaching is often of major importance in cation cycles, and many forest ecosystems show a net loss of base cations. In aluminum-rich soils, acid deposition, by lowering the pH, can increase aluminum concentrations in soil water through dissolution and ion-exchange processes. There is abundant evidence that aluminum is toxic to plants, and it is believed that the toxic effect of aluminum on forest trees could be due to its interference with Ca uptake. Once it enters the forest tree roots, aluminum accumulates in root tissue. Because calcium plays a major role in cell membrane integrity and cell wall structure, reductions in Ca uptake suppresses cambial growth, reduces the rate of wood formation, decreases the amount of functional sapwood and live crown and predisposes trees to disease and injury from stress agents when the functional sapwood becomes less than 25% of cross sectional stem area. There are large variations in Al sensitivity among ecotypes, between and within species due to differences in nutritional demands and physiological status, which are related to age and climate, which change over time (CD, pp. 4-53 to 4-60).

The Integrated Forest Study (IFS) (Johnson and Lindberg, 1992) has characterized the complexity and variability of ecosystem response to atmospheric inputs and provided the most extensive data set available on the effects of atmospheric deposition, including particle deposition, on the cycling of elements in forest ecosystems. The IFS project concluded that acidic deposition is having a significant, often overwhelming effect on both nutrient cycling and cation leaching from the soils in most of the forest ecosystems studied, though the nature of the effects varies from one location to another. It appears that particle deposition has a greater effect on base cation inputs to soils than on base cation losses associated with inputs of sulfur, nitrogen, and H^+. These inputs of base cations have considerable significance, not only to the base cation status of these ecosystems, but also to the potential of incoming precipitation to acidify or alkalize the soils in these ecosystems. However, these net losses or gains of base cations must be placed in the context of the existing soil pool size of exchangeable base cations. The actual rates, directions, and magnitudes of changes that may occur in soils (if any) will depend on rates of inputs from weathering, vegetation outputs, as well as deposition and leaching. In some cases, sites identified as sensitive have large stores of weatherable minerals, while other soils, with smaller stores of weatherable minerals but larger exchangeable cation reserves, are considered less sensitive. In

addition, atmospheric deposition may have significantly affected the nutrient status of some IFS sites through the mobilization of Al. However, the connection between Al mobilization and forest response is still not clear and warrants further study (CD, pp. 4-62 to 4-72).

Trace Elements. Some trace elements deposited directly onto vegetative surfaces can be toxic to the populations of fungi and other microorganisms living on the leaves. Since these organisms play an important role in leaf decomposition after litterfall, changes in these communities can affect the rate of litter decomposition and subsequently nutrient availability for vegetation. Alternatively, trace elements can be absorbed and bound in the leaf tissue, which has also been shown to have a depressing effect on the rates of litter decomposition. Heavy metals deposited from the atmosphere to forests accumulate either in the top, richly organic layer of the forest floor or in the soil layers immediately beneath it, areas where the activity of plant roots and soil organisms is greatest. Because copper, nickel, zinc, cadmium, cobalt and lead compounds can all be toxic to roots and soil organisms, these heavy metals change the litter decomposition processes which influence the availability of essential soil nutrients, ultimately interfering with ecosystem nutrient cycling. Therefore, any effects on structure and function of an ecosystem are likely to occur through the soil and litter. A number of toxic effects of metals on soil microbes have been documented. For example, cadmium was observed to decrease and prolong logarithmic rates of microbial increase, to reduce microbial respiration and fungal spore formation and germination, to inhibit bacterial transformation, and to induce abnormal morphologies. Additionally, the effects of metals on the symbiotic activity of fungi, bacteria, and actinomycetes to plant roots can vary from host to host (Gildon and Tinker, 1983). Alternately, symbiotic associations of mycorrhizal fungi with plants may also provide some additional degree of tolerance to metals (CD, pp. 4-77 to 4-81).

There is some evidence that invertebrates inhabiting soil litter do accumulate metals. Earthworms from roadsides were shown to contain elevated concentrations of cadmium, nickel, lead, and zinc, though interference with earthworm activity was not cited. A study of the accumulation of these same metals in earthworms suggested that cadmium and zinc were concentrated, but not lead. It has further been shown that when soils are acidic, earthworm abundance decreases and bioaccumulation of metals from the soil may increase exponentially with

decreasing pH. Thus, organisms that feed on earthworms from soils with elevated concentrations of lead and zinc for extended periods would be expected to accumulate these metals to toxic levels. Biological accumulation of metals through the plant-herbivore and litter-detritivore chains can occur. Studies indicate that heavy metal deposition onto the soil, via food chain accumulation, can cause excess levels and toxic effects in certain animals (CD, pp. 4-78 to 4-81).

Organics. At the ecosystem level, some organic chemicals are of concern because they may reach toxic levels in both animal and human food chains. Of particular ecological and public concern are the polychlorinated hydrocarbons, such as the dioxins. As discussed above, wet and dry particle deposition are the most important pathways for the accumulation of these more highly chlorinated congeners in vegetation. Though not studied extensively, biodegradation probably does not occur since these compounds are found primarily in the lipophilic cuticle and are very resistant to microbial degradation. Therefore, the grass-cattle-milk/beef pathway is a critical one for humans since exposure often comes from ingestion of animal fat from fish, meat and dairy products. Alternatively, feed contaminated with soil containing the pollutant can be another source of exposure of beef and dairy cattle as well as chickens. Likewise in natural ecosystems, these chemicals tend to bioaccumulate up the food chain. Actions taken by EPA (under the authority of Section 112 of the CAA) and others to evaluate and control sources of Great Waters pollutants of concern appear to have positively affected trends in pollutant concentrations measured in air, sediment, and biota. (CD, pp. 4-30 to 4-32).

5.4.3 Summary

The draft CD presents evidence of effects on vegetation and ecosystems from ambient PM, both in the U.S. and Europe, including in particular effects related to nitrate and acidic deposition. Based on available evidence, the draft CD concludes that "atmospheric PM at levels currently found in the United States has the potential to alter ecosystem structure and function in ways that may reduce their ability to meet societal needs." (CD, p. 4-84). However, the available information does not yet provide the basis to characterize quantitatively the complex relationships between observed adverse effects on vegetation and ecosystems in various locations across the U.S. and levels of PM in the ambient air, due in part to the role that location-specific

environmental factors play, even in determining whether PM deposition occurring in a given location represents a beneficial or an adverse effect. Thus, while evidence of PM-related effects clearly exists, there is insufficient information available at this time to serve as a basis for a national PM air quality standard, defined in terms of concentrations of fine- and/or coarse-fraction particles in the ambient air, specifically selected to protect against adverse effects on vegetation and ecosystems.

5.5 EFFECTS ON SOLAR RADIATION AND GLOBAL CLIMATE CHANGE

The extensive international research and assessment efforts into stratospheric ozone depletion and global climate change provide evidence that atmospheric particles play important roles in two key types of atmospheric processes: 1) alterations in the amount of solar radiation in the ultraviolet range (especially UV-B radiation) penetrating through the earth's atmosphere and reaching its surface, where it can exert a variety of effects on human health, plant and animal biota, and other environmental components; and 2) alterations in the amount of solar radiation in the visible range being transmitted through the earth's atmosphere and either being reflected back into space or absorbed (as well as a lessor role in absorbing infrared radiation emitted by the earth's surface), which enhance heating of the earth's surface and lower atmosphere and lead to consequent "global warming" impacts on human health and the environment (CD, p. 4-129). Information on the role of atmospheric particles in these atmospheric processes is summarized above in Chapter 2 (Section 2.9). Based on information in Chapter 4 of the draft CD, the effects on human health and the environment associated with such atmospheric processes are summarized below, in conjunction with consideration of the potential indirect impacts on human health and the environment that may be a consequence of radiative and climatic changes attributable to changes in ambient PM.

5.5.1 Alterations in Solar UV-B Radiation and Potential Human Health and Environmental Impacts

This section briefly summarizes information on the health and environmental effects associated with UV-B radiation exposure and considers the potential impacts that may result from

changes in UV-B radiation penetration to the earth's surface attributable to changes in ambient PM. The main types of effects associated with exposure to UV-B radiation include direct effects on human health and agricultural and ecological systems, indirect effects on human health and ecosystems, and effects on materials. The study of these effects has been driven by international concern over potentially serious increases in the amount of solar UV-B radiation reaching the earth's surface due to the depletion of the stratospheric ozone layer by the release of various man-made ozone-depleting substances. Extensive qualitative and quantitative characterizations of these global effects attributable to projections of stratospheric ozone depletion have been periodically assessed in studies carried out under WMO and UNEP auspices, with the most recent projections being published by UNEP (1998).

Direct human health effects of UV-B radiation exposure include: skin damage (sunburn) leading to more rapid aging and increased incidence of skin cancer; effects on the eyes, including retinal damage and increased cataract formation possibly leading to blindness; and suppression of some immune system components, contributing to skin cancer induction and possibly increasing susceptibility to certain infectious diseases and/or decreasing effectiveness of vaccinations. Direct environmental effects include damage to terrestrial plants, leading to possible reduced yields of some major food crops and commercially important tress, as well as to biodiversity shifts in natural terrestrial ecosystems; and adverse effects on aquatic life, including reductions in important components of marine food chains as well as other aquatic ecosystem shifts. Indirect health and environmental effects are primarily those mediated through increased tropospheric ozone formation and consequent ozone-related health and environmental impacts. Effects on materials include accelerated polymer weathering and other effects on man-made materials and cultural artifacts. In addition, there are emerging complex issues regarding interactions and feedbacks between climate change and changes in terrestrial and marine biogeochemical cycles due to increased UV-B radiation penetration.

The various assessments of these effects that have been conducted consistently note that the modeled projections quantitatively relating changes in UV-B radiation (attributable to stratospheric ozone depletion) to changes in health and environmental effects are subject to considerable uncertainty, with the role of atmospheric particles being one of numerous

complicating factors. Taking into account the complex interactions between ambient particles and UV-B radiation transmission through the lower atmosphere, the CD concludes that any effort to quantify projected indirect effects of variations in atmospheric PM on human health or the environment due to particle impacts on transmission of solar UV-B radiation would require location-specific evaluations that take into account the composition, concentration, and internal structure of the particles; temporal variations in atmospheric mixing heights and depths of layers containing the particles; and consequent impacts on surface level exposures of humans, ecosystem constituents, or man-made materials (CD, page 4-137).

At present, models are not available to take such complex factors into account, nor is sufficient data available to characterize input variables that would be necessary for any such modeling. The CD concludes, however, that the outcome of such modeling efforts would likely vary from location to location, even as to the direction of changes in the levels of exposures to UV-B radiation, due to location-specific changes in ambient PM concentrations and/or composition (CD, p. 4-137). Beyond considering just average levels of exposures to UV-B radiation in general, the CD notes that ambient PM can affect the directional characteristics of UV-B radiation scattering at ground-level, and thus its biological effectiveness. Also, ambient PM can affect not only biologically damaging UV-B radiation, but can also reduce the ground-level ratio of photorepairing UV-A radiation to UV-B radiation. Further, the CD notes that ambient PM deposition is a major source of PAH in certain water bodies, which can enhance the adverse effects of solar UV-B radiation on aquatic organisms, such that the net effect of ambient PM in some locations may be to increase UV-B radiation-related biological damage to certain aquatic and terrestrial organisms.

5.5.2 Global Climate Change and Potential Human Health and Environmental Impacts

This section briefly summarizes information on the health and environmental vulnerabilities associated with global warming and climate change, and considers the potential impacts that may result from such climatic changes attributable to changes in ambient PM. In general, a number of sectors are seen as vulnerable to climatic change resulting from global warming, including terrestrial and aquatic ecosystems, hydrology and water resources, food and fiber production,

coastal systems, and human health (Intergovernmental Panel on Climate Change, 1998). The study of these vulnerabilities has been driven by international concern over increases in emissions due to man's activities of "greenhouse gases," or their precursors, leading to consequent global warming and climate change. These gases include especially carbon dioxide, nitrous oxide, methane, chlorofluorocarbons, and tropospheric ozone. The presence of ambient PM is one of numerous factors that plays a role in the extremely complex assessment of such climatic changes. The processes involved in global warming and its likely consequent effects have been extensively reviewed, with all assessments and summaries emphasizing the extreme complexity associated with such assessment. Despite the inherent complexity and uncertainties in these global-scale assessments, all typically agree that some global warming has occurred and will continue to occur during the coming decades. Further, the impacts are generally projected to be highly variable across geographic regions, with the potential for both substantial damage in some sectors, or, conversely, the potential for some beneficial outcomes. The most recent report on possible global climate change impacts on various areas in the U.S. is based on assessments now being conducted by the U.S. Global Change Research Program (USGCRG, 2000), summarized in the CD (Appendix 4D).

Potential effects of global warming and climate change on both the environment and human health in the U.S. are summarized in the CD (Section 4.5.2). The most vulnerable environmental sectors and regions in the continental U.S. include long-lived natural forest ecosystems in the East and interior West; water resources in the southern plains; agriculture in the Southeast and southern plains; northern ecosystems and habitats; estuary beaches in developed areas; and low-latitude cool and cold water fisheries. On the other hand, other sectors or subregions may benefit, including west coast coniferous forests; some western rangelands; reduced energy costs for heating in northern latitudes; reduced road salting and snow-clearance costs; longer open-water seasons in northern channels and ports; and agriculture in northern latitudes, the interior West, and the west coast. Both adverse and beneficial environmental effects are projected for Alaska, with possible major declines or loss of some sensitive species occurring in parallel with possible opening of ice-bound transportation routes or expanded agriculture.

With regard to effects on human health, mainly deleterious direct and indirect effects are projected to be associated with global warming and climate change. Such direct health effects include increased mortality linked to temperature extremes (both high and low) and increases in the incidence and spread of vector-borne infectious diseases (e.g., Lyme disease, malaria). Indirect health effects include effects secondary to sea-level rise (e.g., changes in the habitats of mosquitos and other disease vectors) and those secondary to increased tropospheric air pollution (e.g., respiratory effects associated with exposure to ground-level ozone).

The CD (p. 4-154) notes that observational evidence for the climatic effects of ambient particles is sparse. Further, any effort to model the relationship between changes in ambient PM and direct climatic effects would be hindered by a lack of knowledge of ambient particle characteristics including vertical and horizontal variability, size distribution, chemical composition and the distribution of components within individual particles. The CD stresses that the overall radiative effect of particles at a given location is not simply determined by the sum of effects caused by individual classes of particles because of interactions between particles and atmospheric gases. Further, estimation of indirect particle effects are subject to even much greater uncertainties. The CD concludes that, although on a global scale atmospheric particles likely exert an overall net effect of slowing global warming, much uncertainty would be associated with any future efforts aimed at projecting the net effect on global warming processes, resulting climate change, and any consequent human health or environmental effects, due to location-specific changes in emissions of particles or their gaseous precursors (CD, page 4-155).

5.5.3 Summary

A number of assessments of the factors affecting the penetration of solar UV-B radiation to the earth's surface and of the factors affecting global warming and climate change clearly recognize ambient PM as playing various roles in these processes. These assessments, however, have focused on global- and regional-scale impacts, allowing for generalized assumptions to take the place of specific, but unavailable, information on local-scale atmospheric parameters and characteristics of the distribution of particles present in the ambient air. As such, the available information provides no basis for estimating how localized changes in the temporal, spatial, and

composition patterns of ambient PM, likely to occur as a result of expected future emissions of particles and their precursor gases across the U.S., would affect local, regional, or global changes in UV-B radiation penetration and scattering or global warming – even the direction of such effects on a local scale remains uncertain. Moreover, similar concentrations of different particle components can produce opposite net effects. It follows, therefore, that there is insufficient information available to project the extent to which, or even whether, such location-specific changes in ambient PM would indirectly affect human health or the environment secondary to potential changes in UV-B radiation and global warming.

Based on currently available information, the indirect effects of ambient PM, secondary to potential changes in UV-B radiation and global warming, can play no quantitative role in considering whether any revisions of the primary or secondary PM NAAQS are appropriate at this time. Even qualitatively, the available information is very limited in the extent to which it can help inform an assessment of the overall weight of evidence in an assessment of the net health and environmental effects of PM in the ambient air, considering both its direct effects (e.g., inhalation-related health effects) and indirect effects mediated by other routes of exposure and environmental factors (e.g., dermal exposure to UV-B radiation).

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APPENDIX A TABLE 1. ESTIMATED INCREASED MORTALITY PER INCREMENTS IN 24-h CONCENTRATIONS OF PM_{10}, $PM_{2.5}$ AND $PM_{10-2.5}$ FROM U.S. AND CANADIAN STUDIES

Reference, Study Location *	% increase (95% CI) per 50 µg/m^3 PM_{10} Increase	% increase (95% CI) per 25 µg/m^3 $PM_{2.5}$ Increase	% increase (95% CI) per 25 µg/m^3 $PM_{10-2.5}$ Increase	PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ Mean (Range) Levels Reported**
Total (nonaccidental) Mortality				
Ito and Thurston, 1996 Chicago, IL	*2.47 (1.26, 3.69)*	---	---	*PM_{10} 38 (max 128)*
Kinney et al., 1995 Los Angeles, CA	*2.47 (-0.17, 5.18)*	---	---	*PM_{10} 58 (15, 177)*
Pope et al., 1992 Utah Valley, UT	*7.63 (4.41, 10.95)*	---	---	*PM_{10} 47 (11, 297)*
Schwartz, 1993 Birmingham, AL	*5.36 (1.16, 9.73)*	---	---	*PM_{10} 48 (21, 80)*
Schwartz et al., 1996 Boston, MA	*6.15 (3.56, 8.80)*	*5.59 (3.80, 7.42)*	*0.51 (-1.73, 2.78)*	*PM_{10} 24.5 (SD 12.8) $PM_{2.5}$ 15.7 (SD 9.2) $PM_{10-2.5}$ 8.8 (SD 7.0)*
Schwartz et al., 1996 Knoxville, TN	*4.58 (0.27, 9.08)*	*3.54 (0.52, 6.65)*	*2.52 (-1.46, 6.66)*	*PM_{10} 32.0 (SD 14.5) $PM_{2.5}$ 20.8 (SD 9.6) $PM_{10-2.5}$ 11.2 (SD 7.4)*
Schwartz et al., 1996 St. Louis, MO	*3.04 (0.76, 5.37)*	*2.77 (1.13, 4.44)*	*0.50 (-1.73, 2.78)*	*PM_{10} 30.6 (SD 16.2) $PM_{2.5}$ 18.7 (SD 10.5) $PM_{10-2.5}$ 11.9 (SD 8.5)*
Schwartz et al., 1996 Steubenville, OH	*4.58 (0.76, 8.54)*	*2.52 (-0.24, 5.35)*	*6.11 (1.30, 11.15)*	*PM_{10} 45.6 (SD 32.3) $PM_{2.5}$ 29.6 (SD 21.9) $PM_{10-2.5}$ 16.1 (SD 13.0)*
Schwartz et al., 1996 Portage, WI	*3.55 (-1.71, 9.09)*	*3.03 (-0.84, 7.05)*	*1.25 (-3.06, 5.76)*	*PM_{10} 17.8 (SD 11.7) $PM_{2.5}$ 11.2 (SD 7.8) $PM_{10-2.5}$ 6.6 (SD 6.8)*

Reference, Study Location *	% increase (95% CI) per 50 µg/m^3 PM_{10} Increase	% increase (95% CI) per 25 µg/m^3 $PM_{2.5}$ Increase	% increase (95% CI) per 25 µg/m^3 $PM_{10-2.5}$ Increase	PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ Mean (Range) Levels Reported**
Schwartz et al., 1996 Topeka, KS	*-2.48 (-9.33, 4.90)*	*2.01 (-4.83, 9.35)*	*-3.22 (-7.89, 1.69)*	*PM_{10} 26.7 (SD 16.1)* *$PM_{2.5}$ 12.2 (SD 7.4)* *$PM_{10-2.5}$ 14.5 (SD 12.2)*
Schwartz et al., 1996 6 Cities, Overall	*4.06 (2.53, 5.62)*	*3.79 (2.77, 4.82)*	*1.00 (-0.37, 2.40)*	*PM_{10} means 17.8-45.6* *$PM_{2.5}$ means 11.2-29.6* *$PM_{10-2.5}$ means 6.6-16.1*
Styer et al., 1995 Chicago, IL	*4.08 (0.08, 8.24)*	---	---	*PM_{10} 37 (4, 365)*
Samet et al., 2000a,b 90 Largest U.S. Cities	2.27 (0.10, 4.48)	---	---	PM_{10} mean range 15.3-52.0
Samet et al., 2000c 20 Largest U.S. Cities	2.58 (0.41, 4.79)	---	---	PM_{10} mean range 23.8-46.0
Dominici et al., 2000 20 Largest U.S. Cities	1.91 (-0.41, 4.30)	---	---	PM_{10} mean range 23.8-52.0
Schwartz, 2000a 10 U.S. cities	3.40 (2.65, 4.14)	---	---	PM_{10} mean range 27.1-40.6
Braga et al., 2000 5 U.S. cities	4.3 (3.0, 5.6)	---	---	PM_{10} mean range 28-37
Burnett et al., 1998 Toronto, CAN	3.46 (1.74, 5.21)	4.79 (3.26, 6.34)	---	PM_{10} 30.2 (max 116) $PM_{2.5}$ 18.0 (8, 90)
Burnett et al., 2000 8 Canadian Cities	3.51 (1.04, 6.04)	3.03 (1.10, 4.99)	1.82 (-0.72, 4.43)	PM_{10} 25.9 (max 121) $PM_{2.5}$ 13.3 (max 86) $PM_{10-2.5}$ 12.9 (max 99)
Chock et al., 2000 Pittsburgh, PA		<75 years 2.6 (2.0, 7.3) >75 years 1.5 (-3.0, 6.3)	<75 years 0.7 (-1.7, 3.7) >75 years 1.3 (-1.3, 3.8)	NR
Clyde et al., 2000 Phoenix, AZ	6 (>0, 11)	---	---	PM_{10} mean 45.4

Reference, Study Location *	% increase (95% CI) per 50 µg/m^3 PM_{10} Increase	% increase (95% CI) per 25 µg/m^3 $PM_{2.5}$ Increase	% increase (95% CI) per 25 µg/m^3 $PM_{10-2.5}$ Increase	PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ Mean (Range) Levels Reported**
Fairley, 1999 Santa Clara County, CA	8 ($p<0.05$)	8 ($p<0.01$)	2 ($p>0.05$)	PM_{10} 34 (6, 165) $PM_{2.5}$ 13 (2, 105) $PM_{10-2.5}$ 11 (0, 45)
Gamble, 1998 Dallas, TX	-3.56 (-12.73, 6.58)	---	---	PM_{10} 24.5 (11, 86)
Goldberg et al., 2000 Montreal, CAN	---	5.81 (3.36, 8.32)	---	$PM_{2.5}$ 17.6 (4.6, 71.7)
Gwynn et al., 2000 Buffalo, NY	12.33 (2.50, 23.11)	1.54 (0.3, 2.74) (15 ug/m^3 $SO_4^=$)	---	PM_{10} 24.1 (6.8, 90.8) $SO_4^=$61.7 (0.78, 390.5) nmol/m^3
Klemm and Mason, 2000 Atlanta, GA	---	4.8 (-3.2, 13.4)	1.4 (-11.3, 15.9)	$PM_{2.5}$ 19.9 (1.0, 54.8) $PM_{10-2.5}$ 10.1 (0.2, 39.5)
Klemm et al., 2000 Six City reanalysis - St. Louis	2.02 (-0.24, 4.33)	2.01 (0.51, 3.54)	0.25 (-1.98, 2.53)	PM_{10} 30.6 (SD 16.2) $PM_{2.5}$ 18.7 (SD 10.5) $PM_{10-2.5}$ 11.9 (SD 8.5)
Klemm et al., 2000 Six City reanalysis - Steubenville	3.04 (-1.23, 7.48)	1.51 (-1.60, 4.71)	4.82 (4.04, 5.61)	PM_{10} 45.6 (SD 32.3) $PM_{2.5}$ 29.6 (SD 21.9) $PM_{10-2.5}$ 16.1 (SD 13.0)
Klemm et al., 2000 Six City reanalysis - Topeka	-3.45 (-11.37, 5.17)	1.51 (-6.48, 10.18)	-3.71 (-9.17, 2.08)	PM_{10} 26.7 (SD 16.1) $PM_{2.5}$ 12.2 (SD 7.4) $PM_{10-2.5}$ 14.5 (SD 12.2)
Klemm et al., 2000 Six City reanalysis - overall	4.06 (2.78, 5.36)	3.28 (2.27, 4.31)	1.00 (-0.37, 2.40)	PM_{10} means 17.8-45.6 $PM_{2.5}$ means 11.2-29.6 $PM_{10-2.5}$ means 6.6-16.1
Klemm et al., 2000 Six City reanalysis - Knoxville	7.20 (2.29, 12.34)	4.82 (1.40, 8.35)	4.05 (-0.46, 8.76)	PM_{10} 32.0 (SD 14.5) $PM_{2.5}$ 20.8 (SD 9.6) $PM_{10-2.5}$ 11.2 (SD 7.4)

Reference, Study Location *	% increase (95% CI) per 50 µg/m^3 PM_{10} Increase	% increase (95% CI) per 25 µg/m^3 $PM_{2.5}$ Increase	% increase (95% CI) per 25 µg/m^3 $PM_{10-2.5}$ Increase	PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ Mean (Range) Levels Reported**
Klemm et al., 2000 Six City reanalysis - Boston	6.15 (3.56, 8.80)	5.33 (3.54, 7.15)	1.25 (-1.11, 3.68)	PM_{10} 24.5 (SD 12.8) $PM_{2.5}$ 15.7 (SD 9.2) $PM_{10-2.5}$ 8.8 (SD 7.0)
Klemm et al., 2000 Six City reanalysis - Madison	2.02 (-3.42, 7.76)	2.27 (-1.83, 6.54)	0.25 (-4.51, 5.25)	PM_{10} 17.8 (SD 11.7) $PM_{2.5}$ 11.2 (SD 7.8) $PM_{10-2.5}$ 6.6 (SD 6.8)
Laden et al., 2000 Six City reanalysis	---	4.05 (2.78, 5.34) overall -5.65 (-13.74, 3.19) crustal 8.72 (4.22, 13.41) mobile 2.77 (0.64, 4.95) coal	---	$PM_{2.5}$ same as Six City
Levy et al., 1998 King Co., WA	7.2 (-6.3, 22.8)	1.76 (-3.53, 7.34)	---	PM_{10} 29.8 (6.0, 123.0) PM_1 28.7 (16.3, 92.2)
Lipfert et al., 2000 Philadelphia, PA	5.99 (p>0.055)	4.21 (p<0.055)	5.07 (p>0.055)	PM_{10} 32.20 (7.0, 95.0) $PM_{2.5}$ 17.28 (-0.6, 72.6) $PM_{10-2.5}$ 6.80 (-20.0, 28.3)
Lippmann et al., 2000 Detroit, MI	4.41 (-0.98, 10.10)	3.10 (-0.63, 6.98)	3.96 (-1.22, 9.42)	PM_{10} 31 (12, 105) $PM_{2.5}$ 18 (6, 86) $PM_{10-2.5}$ 13 (4, 50) mean (5%, 95%)
Mar et al., 2000 Phoenix, AZ	5.44 (0.06, 11.12)	5.98 (-1.34, 13.85)	2.97 (-0.50, 6.56)	PM_{10} 46.5 (5, 213) $PM_{2.5}$ 13.0 (0, 42) $PM_{10-2.5}$ 33.5 (5, 187)
Moolgavkar, 2000a Los Angeles, CA	1.25 (p<0.05, from figure)	0.6 (p>0.05, from figure)	---	PM_{10} median 44 (7, 166) $PM_{2.5}$ 22 (4, 86)
Moolgavkar, 2000a Cook Co., IL	1.25 (p<0.05, from figure)	---	---	PM_{10} median 35 (3, 365)
Moolgavkar, 2000a Maricopa Co., AZ	3 (p<0.05, from figure)	---	---	PM_{10} median 41 (9, 252)

Reference, Study Location *	% increase (95% CI) per 50 µg/m^3 PM_{10} Increase	% increase (95% CI) per 25 µg/m^3 $PM_{2.5}$ Increase	% increase (95% CI) per 25 µg/m^3 $PM_{10-2.5}$ Increase	PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ Mean (Range) Levels Reported**
Ostro, 1995 San Bernadino and Riverside Counties, CA	---	0.28 (-0.61, 1.17)	---	$PM_{2.5}$ 32.5 (9.3, 190.1) (estimated from visibility)
Ostro et al., 1999 Coachella Valley, CA	4.60 (0.58, 8.79)	---	---	PM_{10} 56.8 (38, 417)
Ostro et al., 2000 Coachella Valley, CA	2.01 (-0.99, 5.10)	11.8 (1.3, 23.4)	0.7 (-0.8, 2.3)	PM_{10} 47.4 (3, 417) $PM_{2.5}$ 16.8 (5, 48) $PM_{10-2.5}$ 17.9 (0, 149)
Pope et al., 1999 Ogden, UT	12.02 (4.49, 20.99)	---	---	PM_{10} 32.1 (4, 182)
Pope et al., 1999 Salt Lake City, UT	2.33 (0.05, 4.66)	---	---	PM_{10} 41.2 (7, 441)
Pope et al., 1999 Provo/Orem, UT	1.87 (-2.15, 6.04)	---	---	PM_{10} 38.4 (1, 317)
Schwartz, 2000c Boston, MA	---	5.33 (1.81, 8.98)	---	$PM_{2.5}$ 15.6 (±9.2)
Schwartz and Zanobetti, 2000 Chicago, IL	4.53 (3.11, 5.96)	---	---	PM_{10} median 36
Tsai et al., 2000 Newark, NJ	5.65 (4.62, 6.70)	4.34 (2.82, 5.89)	---	PM_{15} 55 (SD 6.5) $PM_{2.5}$ 42.1 (SD 22.0)
Tsai et al., 2000 Camden, NJ	11.07 (0.70, 22.51)	5.65 (0.11, 11.51)	---	PM_{15} 47.0 (SD 20.9) $PM_{2.5}$ 39.9 (SD 18.0)
Tsai et al., 2000 Elizabeth, NJ	-4.88 (-17.88, 10.19)	1.77 (-5.44, 9.53)	---	PM_{15} 47.5 (SD 18.8) $PM_{2.5}$ 37.1 (SD 19.8)
Cause-Specific Mortality				

Cardiorespiratory:

Reference, Study Location *	% increase (95% CI) per 50 µg/m^3 PM_{10} Increase	% increase (95% CI) per 25 µg/m^3 $PM_{2.5}$ Increase	% increase (95% CI) per 25 µg/m^3 $PM_{10-2.5}$ Increase	PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ Mean (Range) Levels Reported**
Samet et al., 2000c 20 Largest U.S. Cities	3.45 (1.01, 5.94)	---	---	PM_{10} means 15.3-46.0
Tsai et al., 2000 Newark, NJ	7.79 (3.65, 12.10)	5.13 (3.09, 7.21)	---	PM_{15} 55 (SD 6.5) $PM_{2.5}$ 42.1 (SD 22.0)
Tsai et al., 2000 Camden, NJ	15.03 (4.29, 26.87)	6.18 (0.61, 12.06)	---	PM_{15} 47.0 (SD 20.9) $PM_{2.5}$ 39.9 (SD 18.0)
Tsai et al., 2000 Elizabeth, NJ	3.05 (-11.04, 19.36)	2.28 (-4.97, 10.07)	---	PM_{15} 47.5 (SD 18.8) $PM_{2.5}$ 37.1 (SD 19.8)
Total Cardiovascular:				
Ito and Thurston, 1996 *Chicago, IL*	*1.49 (-0.72, 3.74)*	---	---	*PM_{10} 38 (max 128)*
Pope et al., 1992 *Utah Valley, UT*	*9.36 (1.91, 17.36)*	---	---	*PM_{10} 47 (11, 297)*
Fairley, 1999 Santa Clara County, CA	9 ($p<0.05$)	6.2 ($p>0.05$)	3 ($p>0.05$)	PM_{10} 34 (6, 165) $PM_{2.5}$ 13 (2, 105) $PM_{10-2.5}$ 11 (0, 45)
Goldberg et al., 2000 Montreal, CAN	---	3.48 (-0.16, 7.26)	---	$PM_{2.5}$ 17.6 (4.6, 71.7)
Gwynn et al., 2000 Buffalo, NY	6.86 (-1.28, 15.66)	1.54 (-1.14, 4.28) (15 ug/m^3 $SO_4^=$)	---	PM_{10} 24.1 (6.8, 90.8) $SO_4^=$61.7 (0.78, 390.5) nmol/m^3
Lipfert et al., 2000 Philadelphia, PA (7-county area)	6.92 ($p<0.055$)	10.26 ($p<0.055$)	7.57 ($p>0.055$)	PM_{10} 32.20 (7.0, 95.0) $PM_{2.5}$ 17.28 (-0.6, 72.6) $PM_{10-2.5}$ 6.80 (-20.0, 28.3)
Lippmann et al., 2000 Detroit, MI	6.86 (-1.28, 15.66)	3.17 (-2.29, 8.94)	7.82 (0.03, 16.23)	PM_{10} 31 (12, 105) $PM_{2.5}$ 18 (6, 86) $PM_{10-2.5}$ 13 (4, 50) mean (10%, 90%)

Reference, Study Location *	% increase (95% CI) per 50 µg/m^3 PM_{10} Increase	% increase (95% CI) per 25 µg/m^3 $PM_{2.5}$ Increase	% increase (95% CI) per 25 µg/m^3 $PM_{10-2.5}$ Increase	PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ Mean (Range) Levels Reported**
Mar et al., 2000 Phoenix, AZ	9.86 (1.91, 18.42)	18.68 (5.72, 33.23)	6.45 (1.42, 11.73)	PM_{10} 46.5 (5, 213) $PM_{2.5}$ 13.0 (0, 42) $PM_{10-2.5}$ 33.5 (5, 187)
Moolgavkar, 2000a Los Angeles, CA	4.47 (1.65, 7.37)	2.59 (0.38, 4.85)	---	PM_{10} median 44 (7, 166) $PM_{2.5}$ median 22 (4, 86)
Moolgavkar, 2000a Cook Co., IL	2.21 (0.37, 4.09)	---	---	PM_{10} median 35 (3, 365)
Moolgavkar, 2000a Maricopa Co., AZ	8.85 (2.67, 15.39)	---	---	PM_{10} median 41 (9, 252)
Ostro et al., 2000 Coachella Valley, CA	6.09 (2.05, 10.29)	8.56 (-6.35, 25.84)	2.56 (0.60, 4.49)	PM_{10} 47.4 (3, 417) $PM_{2.5}$ 16.8 (5, 48) $PM_{10-2.5}$ 17.9 (0, 149)
Ostro et al., 1999 Coachella Valley, CA	8.33 (2.14, 14.9)	---	---	PM_{10} 56.8 (38, 417)
Ostro, 1995 San Bernadino and Riverside Counties, CA	---	0.69 (-0.34, 1.74)	---	$PM_{2.5}$ 32.5 (9.3, 190.1) (estimated from visibility)
Pope et al., 1999 Salt Lake City, UT	6.50 (2.21, 10.98)	---	---	PM_{10} 41.2 (7, 441)
Pope et al., 1999 Provo/Orem, UT	8.60 (2.40, 15.18)	---	---	PM_{10} 38.4 (1, 317)
Pope et al., 1999 Ogden, UT	1.41 (-8.33, 12.18)	---	---	PM_{10} 32.1 (4, 182)
Coronary Artery Disease:				
Goldberg et al., 2000 Montreal, CAN	---	4.48 (-0.31, 9.51)	---	$PM_{2.5}$ 17.6 (4.6, 71.7)
Cerebrovascular:				

Reference, Study Location *	% increase (95% CI) per 50 µg/m^3 PM_{10} Increase	% increase (95% CI) per 25 µg/m^3 $PM_{2.5}$ Increase	% increase (95% CI) per 25 µg/m^3 $PM_{10-2.5}$ Increase	PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ Mean (Range) Levels Reported**
Moolgavkar, 2000a Cook Co., IL	3.27 (-0.12, 6.77)	---	---	PM_{10} median 35 (3, 365)
Moolgavkar, 2000a Los Angeles, CA	2.92 (-2.27, 8.39)	3.61 (-0.57, 7.97)	---	PM_{10} median 44 (7, 166) $PM_{2.5}$ 22 (4, 86)
Moolgavkar, 2000a Maricopa Co., AZ	11.09 (0.54, 22.75)	---	---	PM_{10} median 41 (9, 252)
Total Respiratory:				
Ito and Thurston, 1996 *Chicago, IL*	*6.77 (1.97, 11.79)*	---	---	*PM_{10} 38 (max 128)*
Pope et al., 1992 *Utah Valley, UT*	*19.78 (3.51, 38.61)*	---	---	*PM_{10} 47 (11, 297)*
Fairley, 1999 Santa Clara County, CA	11 (p>0.05)	11.5 (p>0.05)	16 (p>0.05)	PM_{10} 34 (6, 165) $PM_{2.5}$ 13 (2, 105) $PM_{10-2.5}$ 11 (0, 45)
Goldberg et al., 2000 Montreal, CAN	---	21.6 (13.0, 31.0)	---	$PM_{2.5}$ 17.6 (4.6, 71.7)
Gwynn et al., 2000 Buffalo, NY	17.89 (-14.87, 63.25)	8.16 (4.18, 12.30) (15 ug/m^3 $SO_4^=$)	---	PM_{10} 24.1 (6.8, 90.8) $SO_4^=$61.7 (0.78, 390.5) nmol/m^3
Lipfert et al., 2000 Philadelphia, PA (7-county area)	-3.17 (p>0.055)	0.66 (p>0.055)	-12.72 (p>0.055)	PM_{10} 32.20 (7.0, 95.0) $PM_{2.5}$ 17.28 (-0.6, 72.6) $PM_{10-2.5}$ 6.80 (-20.0, 28.3)
Lippmann et al., 2000 Detroit, MI	7.84 (-10.18, 29.47)	2.28 (-10.31, 16.63)	7.41 (-9.07, 26.87)	PM_{10} 31 (12, 105) $PM_{2.5}$ 18 (6, 86) $PM_{10-2.5}$ 13 (4, 50) mean (10%, 90%)
Ostro et al., 1999 Coachella Valley, CA	13.88 (3.25, 25.61)	---	---	PM_{10} 56.8 (38, 417)

Reference, Study Location *	% increase (95% CI) per 50 μg/m^3 PM_{10} Increase	% increase (95% CI) per 25 μg/m^3 $PM_{2.5}$ Increase	% increase (95% CI) per 25 μg/m^3 $PM_{10-2.5}$ Increase	PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ Mean (Range) Levels Reported**
Ostro et al., 2000 Coachella Valley, CA	-1.99 (-11.41, 8.44)	-13.28 (-43.05, 32.06)	-1.27 (-6.24, 3.95)	PM_{10} 47.4 (3, 417) $PM_{2.5}$ 16.8 (5, 48) $PM_{10-2.5}$ 17.9 (0, 149)
Ostro, 1995 San Bernadino and Riverside Counties, CA	---	2.08 (-0.35, 4.51)	---	$PM_{2.5}$ 32.5 (9.3, 190.1) (estimated from visibility)
Pope et al., 1999 Ogden, UT	23.80 (2.77, 49.14)	---	---	PM_{10} 32.1 (4, 182)
Pope et al., 1999 Provo/Orem, UT	2.22 (-9.83, 15.89)	---	---	PM_{10} 38.4 (1, 317)
Pope et al., 1999 Salt Lake City, UT	8.17 (-0.97, 18.14)	---	---	PM_{10} 41.2 (7, 441)
COPD:				
Moolgavkar, 2000a Cook Co., IL	5.39 (0.30, 10.74)	---	---	PM_{10} median 35 (3, 365)
Moolgavkar, 2000a Los Angeles, CA	5.91 (-1.64, 14.03)	2.67 (-3.38, 9.10)	---	PM_{10} median 44 (7, 166) $PM_{2.5}$ 22 (4, 86)
Moolgavkar, 2000a Maricopa Co., AZ	8.08 (-4.58, 22.41)	---	---	PM_{10} median 41 (9, 252)

* Studies in italics available in 1996 CD

** mean (minimum, maximum) 24-h PM level shown in parentheses unless otherwise noted.

APPENDIX A, TABLE 2. ESTIMATED RESPIRATORY MORBIDITY EFFECTS PER INCREMENTS IN 24-h CONCENTRATIONS OF PM_{10}, $PM_{2.5}$ AND $PM_{10-2.5}$ FROM U.S. AND CANADIAN STUDIES

Reference, Study Location*	% increase (95% CI) per 50 µg/m^3 PM_{10} Increase	% increase (95% CI) per 25 µg/m^3 $PM_{2.5}$ Increase	% increase (95% CI) per 25 µg/m^3 $PM_{10-2.5}$ Increase	PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ Mean (Range) Levels Reported**
Increased Admission to Hospital or Emergency Room				
Total Respiratory:				
Thurston et al., 1994 Toronto, Canada	*23.26 (2.03, 44.49)*	*15.00 (1.97, 28.03)*	*22.25 (-9.53, 54.03)*	*PM_{10} 29.5-38.8 (max 96.0) $PM_{2.5}$ 15.8-22.3 (max 66.0) $PM_{10-2.5}$ 12.7-16.5 (max 33.0)*
Schwartz, 1995 New Haven, CT	*6.00 (-0.28, 12.68)*	---	---	*PM_{10} 41 (19-67)****
Schwartz, 1995 Tacoma, WA	*10.00 (3.21, 17.23)*	---	---	*PM_{10} 37 (14-67)****
Schwartz et al., 1996 Spokane, WA	*8.50 (3.61, 13.62)*	---	---	*PM_{10} 46 (16-83)****
Schwartz et al., 1996 Cleveland, OH	*5.83 (0.54, 11.40)*	---	---	*PM_{10} 43 (19-72)****
Gwynn et al., 2000 Buffalo, NY	17.27 (0.61, 36.68)	8.16 (4.18, 12.30) (15 µg/m^3 $SO_4^=$)	---	PM_{10} 24.1 (6.8, 90.8) $SO_4^=$ 61.7 (0.78, 390.5) nmol/m^3
Linn et al., 2000 Los Angeles, CA (>29 years)	2.89 (1.09, 4.72)	---	---	PM_{10} 45.5 (5, 132)
Moolgavkar et al., 1997 Minneapolis-St. Paul, MN (>65 years)	8.72 (4.59, 13.01) (COPD + pneumonia)	---	---	PM_{10} 34.0 (17, 55)
Moolgavkar et al., 1997 Birmingham, AL (>65 years)	1.51 (-1.43, 4.54) (COPD + pneumonia)	---	---	PM_{10} 43.4 (18.5, 74.1)
Schwartz et al., 1996 Cleveland, OH (>65 years)	5.83 (0.54, 11.40)	---	---	PM_{10} 43

Lumley and Heagerty, 1999 King County, WA (all ages)	---	5.91 (1.10, 10.97)	---	PM_1 NR
Burnett et al., 1997 Toronto, CAN (all ages)	10.93 (4.53, 17.72)	8.61 (3.39, 14.08)	12.71 (5.33, 20.74)	PM_{10} 28.1 (4, 102) $PM_{2.5}$ 16.8 (1, 66) $PM_{10-2.5}$ 11.6 (1, 56)
Delfino et al., 1997 Montreal, CAN (>64 years)	36.62 (10.02, 63.21)	23.88 (4.94, 42.83)	---	summer 93 PM_{10} 21.7 (max 51) $PM_{2.5}$ 12.2 (max 31)
Delfino et al., 1998 Montreal, CAN (>64 years)	---	13.17 (-0.22, 26.57)	---	$PM_{2.5}$ 18.6 (SD 9.3)
Stieb et al., 2000 St. John, CAN (all ages)	8.8 (1.8, 16.4)	5.69 (0.61, 11.03)	---	summer 93 PM_{10}14.0 (max 70.3) $PM_{2.5}$ 8.5 (max 53.2)
Pneumonia:				
Schwartz 1994b *Birmingham, AL*	*9.09 (3.51, 14.97)*	---	---	*PM_{10} 45 (19-77)****
Schwartz 1994a *Detroit, MI*	*5.92 (1.95, 10.05)*	---	---	*PM_{10} 48 (22-82)****
Schwartz 1994c *Minnesota/St. Paul, MN*	*8.17 (1.22, 15.59)*	---	---	*PM_{10} 36 (18-58)****
Schwartz et al., 1996 *Spokane, WA*	*5.30 (-1.51, 12.58)*	---	---	*PM_{10} 46 (16-83)****
Samet et al., 2000 14 U.S. Cities (>65 years)	10.3 (8.5, 12.1)	---	---	PM_{10} means 24.4-45.3
Lippmann et al., 2000 Detroit, MI (>65 years)	21.4 (8.2, 36.3)	12.5 (3.7, 22.1)	11.9 (0.7, 24.4)	PM_{10} 31 (max 105) $PM_{2.5}$ 18 (6, 86) $PM_{10-2.5}$ 13 (4, 50)
Moolgavkar et al., 1997 Minneapolis-St. Paul, MN (>65 years)	3.5 (-0.5, 7.7)	---	---	PM_{10} 34 (17, 55)
Respiratory infections:				

Burnett et al., 1999 Toronto, CAN (all ages)	14.2 (9.3, 19.3)	10.77 (7.18, 14.47)	9.31 (4.64, 14.18)	PM_{10} 30.2 (max 116) $PM_{2.5}$ 18.0 (max 90) $PM_{10-2.5}$ 12.2 (max 68)
COPD:				
Schwartz 1994c *Minnesota/St. Paul, MN*	*25.30 (9.47, 43.42)*	---	---	*PM_{10} 36 (18-58)****
Schwartz 1994b *Birmingham, AL*	*12.69 (3.81, 22.34)*	---	---	*PM_{10} 45 (19-77)****
Schwartz 1994a *Detroit, MI*	*10.63 (4.41, 17.21)*	---	---	*PM_{10} 48 (22-82)****
Schwartz et al., 1996 *Spokane, WA*	*17.10 (7.85, 27.14)*	---	---	*PM_{10} 46 (16-83)****
Samet et al., 2000 14 U.S. Cities (>65 years)	10.3 (7.7, 13.0)	---	---	PM_{10} means 24.4-45.3
Chen et al., 2000 Reno-Sparks, NV(all ages)	9.4 (2.2, 17.1)	---	---	PM_{10} 36.6 (1.7, 201.3)
Linn et al., 2000 Los Angeles, CA (>29 years)	1.5 (-0.5, 3.5)	---	---	PM_{10} 45.5 (5, 132)
Tolbert et al., 2000a Atlanta, GA (all ages)	-3.5 (33.0, -29.9)	12.44 (-7.89, 37.24)	-23.03 (-50.69, 20.15)	PM_{10} 29.1 (SD 12.0) $PM_{2.5}$ 19.4 (SD 9.35) $PM_{10-2.5}$ 9.39 (SD 4.52)
Lippmann et al., 2000 Detroit, MI (>65 years)	9.6 (-5.3, 26.8)	5.49 (-4.72, 16.80)	9.29 (-4.19, 24.66)	PM_{10} 31 (max 105) $PM_{2.5}$ 18 (6, 86) $PM_{10-2.5}$ 13 (4, 50)
Moolgavkar et al., 1997 Minneapolis-St. Paul, MN (>65 years)	6.9 (-0.6, 15.0)	---	---	PM_{10} 34 (17, 55)
Moolgavkar et al., 2000 King County WA (all ages)	5.1 (0, 10.4)	6.4 (0.9, 12.1)	---	PM_{10} $PM_{2.5}$ 18.1 (3, 96)
Moolgavkar, 2000c Cook Co., IL (>65 years)	2.4 (-0.2, 5.1)	---	---	PM_{10} median 35 (3, 365)

Moolgavkar, 2000c Los Angeles, CA (>65 years)	6.1 (1.1, 11.3)	5.1 (0.9, 9.41)	5.07 (-0.44, 10.90)	PM_{10} median 44 (7, 166) $PM_{2.5}$ median 224, 86) $PM_{10-2.5}$ NR
Moolgavkar, 2000c Maricopa Co., AZ (>65 years)	6.9 (-4.2, 19.3)	---	---	PM_{10} median 41 (9, 252)
Burnett et al., 1999 Toronto, CAN (all ages)	6.90 (1.32, 12.78)	4.78 (-0.17, 9.98)	12.83 (4.93, 21.33)	PM_{10} 30.2 (max 116) $PM_{2.5}$ 18.0 (max 90) $PM_{10-2.5}$ 12.2 (max 68)
Asthma:				
Choudbury et al., 1997 Anchorage, AK Medical Visits (all ages)	20.9 (11.8, 30.8)	---	---	PM_{10} 42.5 (1, 565)
Jacobs et al., 1997 Butte County, CA (all ages)	6.11 (p>0.05)	---	---	PM_{10} 34.3 (6.6, 636)
Linn et al., 2000 Los Angeles, CA (>29 years)	1.5 (-2.4, 5.6)	---	---	PM_{10} 45.5 (5, 132)
Lipsett et al., 1997 Santa Clara Co., CA (all ages)	9.1 (2.7, 15.9) (at 41 F and below)	---	---	PM_{10} 61.2 (9, 165)
Los Angeles, CA Nauenberg and Basu, 1999 (all ages)	20.0 (5.3, 35)	---	---	44.8 (SE 17.23)
Norris et al., 1999 Seattle, WA (<18 years)	75.9 (32.9, 132.8)	44.5 (21.7, 71.4)	---	PM_{10} 21.7 (8.0, 69.3) $PM_{2.5}$ (est) 4.8 (1.2, 32.4)
Norris et al., 2000 Seattle, WA (<19 years)	56.2 (10.4, 121.0)			PM_{10} 21.5 (8.0, 69.3)
Norris et al., 2000 Spokane WA (<19 years)	2.4 (-10.9, 17.6)			PM_{10} 27.9 (4.7, 186.4)
Tolbert et al., 2000b Atlanta, GA (<17 years)	13.2 (1.2, 26.7)	---	---	PM_{10} 38.9 (9, 105)

Tolbert et al., 2000a Atlanta, GA (all ages)	18.8 (-8.7, 54.4)	2.27 (-14.79, 22.74)	21.08 (-18.23, 79.29)	PM_{10} 29.1 (SD 12.0) $PM_{2.5}$ 19.4 (SD 9.35) $PM_{10-2.5}$ 9.39 (SD 4.52)
Sheppard et al., 1999 Seattle, WA (<65 years)	13.7 (5.5, 22.6)	8.7 (3.3, 14.3)	11.1 (2.8, 20.1)	PM_{10} 31.5 (90% 55) $PM_{2.5}$ 16.7 (90%` 32) $PM_{10-2.5}$ 16.2 (90% 29)
Burnett et al., 1999 Toronto, CAN (all ages)	8.9 (3.7, 14.4)	6.44 (2.47, 10.57)	11.05 (5.75, 16.62)	PM_{10} 30.2 (max 116) $PM_{2.5}$ 18.0 (max 90) $PM_{10-2.5}$ 12.2 (max 68)

Increased Respiratory Symptoms	Odds Ratio (95% CI) for 50 ug/m^3 increase in PM_{10}	Odds Ratio (95% CI) for 25 ug/m^3 increase in $PM_{2.5}$	Odds Ratio (95% CI) for 25 ug/m^3 increase in $PM_{10-2.5}$	$PM_{10-2.5}$ Mean (Range) Levels Reported**
Schwartz et al., 1994 6 U.S. cities (children, cough)	*1.39 (1.05, 1.85)*	*1.24 (1.00, 1.54)*	---	*PM_{10} median 30.0 (max 117) $PM_{2.5}$ median 18.0 (max 86)*
Schwartz et al., 1994 6 U.S. cities (children, lower respiratory symptoms)	*2.03 (1.36, 3.04)*	*1.58 (1.18, 2.10)*	---	*PM_{10} median 30.0 (max 117) $PM_{2.5}$ median 18.0 (max 86)*
Neas et al., 1995 Uniontown, PA (children, cough)	---	*2.45 (1.29, 4.64)*	---	*$PM_{2.5}$ 24.5 (max 88.1)*
Ostro et al., 1991 Denver, CO (adults, cough)	*1.09 (0.57, 2.10)*	---	---	*PM_{10} 22 (0.5, 73)*
Pope et al., 1991 Utah Valley, UT (lower respiratory symptoms, schoolchildren)	*1.28 (1.06, 1.56)*	---	---	*PM_{10} 44 (11, 195)*

Pope et al., 1991 *Utah Valley, UT* *(lower respiratory symptoms, asthmatic patients)*	*1.01 (0.81, 1.27)*	---	---	*PM_{10} 44 (11, 195)*
Neas et al., 1996 State College, PA (children, cough)	NR	1.48 (1.17, 1.88) (1-d)	---	PM_{10} 31.9 (max 82.7) $PM_{2.1}$ 23.5 (max 85.8)
Neas et al., 1996 State College, PA (children, wheeze)	NR	1.59 (0.93, 2.70) (1-d)	---	PM_{10} 31.9 (max 82.7) $PM_{2.1}$ 23.5 (max 85.8)
Neas et al., 1996 State College, PA (children, cold)	NR	1.61 (1.21, 2.17) (0-d)	---	PM_{10} 31.9 (max 82.7) $PM_{2.1}$ 23.5 (max 85.8)
Ostro et al., 1995 Los Angeles, CA (children, asthma episode)	1.05 (0.64, 1.73)	---	---	PM_{10} 55.87 (19.63, 101.42)
Ostro et al., 1995 Los Angeles, CA (children, shortness of breath)	1.51 (1.04, 2.17)	---	---	PM_{10} 55.87 (19.63, 101.42)
Schwartz and Neas, 2000 Six Cities reanalysis (children, cough)	---	1.28 (0.98, 1.67)	1.77 (1.23, 2.54)	$PM_{2.5}$ (same as Six Cities) $PM_{10-2.5}$ NR
Schwartz and Neas, 2000 Six Cities reanalysis (children, lower respiratory symptoms)	---	1.61 (1.20, 2.16)	1.51 (0.66, 3.43)	$PM_{2.5}$ (same as Six Cities) $PM_{10-2.5}$ NR

Vedal et al., 1998 Port Alberni, CAN (children, cough)	1.40 (1.14, 1.73)	---	---	PM_{10} median 22.1 (0.2, 159.0) (north site)
Vedal et al., 1998 Port Alberni, CAN (children, phlegm)	1.40 (1.03, 1.90)	---	---	PM_{10} median 22.1 (0.2, 159.0) (north site)
Vedal et al., 1998 Port Alberni, CAN (children, nose symptoms)	1.22 (1.00, 1.47)	---	---	PM_{10} median 22.1 (0.2, 159.0) (north site)
Vedal et al., 1998 Port Alberni, CAN (children, sore throat)	1.34 (1.06, 1.69)	---	---	PM_{10} median 22.1 (0.2, 159.0) (north site)
Vedal et al., 1998 Port Alberni, CAN (children, wheeze)	1.16 (0.82, 1.63)	---	---	PM_{10} median 22.1 (0.2, 159.0) (north site)
Vedal et al., 1998 Port Alberni, CAN (children, chest tightness)	1.34 (0.86, 2.09)	---	---	PM_{10} median 22.1 (0.2, 159.0) (north site)
Vedal et al., 1998 Port Alberni, CAN (children, dyspnea)	1.05 (0.74, 1.49)	---	---	PM_{10} median 22.1 (0.2, 159.0) (north site)
Vedal et al., 1998 Port Alberni, CAN (children, any symptom)	1.16 (1.00, 1.34)	---	---	PM_{10} median 22.1 (0.2, 159.0) (north site)

Decreased Lung Function	Lung Function change (L/min) (95% CI) for 50 ug/m^3 increase in PM_{10}	Lung Function change (L/min) (95% CI) for 25 ug/m^3 increase in $PM_{2.5}$	Lung Function change (L/min) (95% CI) for 25 ug/m^3 increase in $PM_{10-2.5}$	$PM_{10-2.5}$ Mean (Range) Levels Reported**
Neas et al., 1995 *Uniontown, PA* *(children)*	---	*-2.58 (-5.33, +0.35)*	---	*$PM_{2.5}$ 24.5 (max 88.1)*

Thurston et al., (1997) Connecticut summer camp (children)	---	PEFR -5.4 (-12.3, 1.5) (15 µg/m^3 $SO_4^=$)	---	$SO_4^=$ 7.0 (1.1, 26.7)
Naeher et al., 1999 Southwest VA (adult women)	am PEFR -3.65 (-6.79, -0.51) pm PEFR -1.8 (-5.03, 1.43)	am PEFR -1.83 (-3.44, -0.21) pm PEFR -1.05 (-2.77, 0.67)	am PEFR -6.33 (-12.50, -0.15) pm PEFR -2.4 (-8.48, 3.68)	PM_{10} 27.07 (4.89, 69.07) $PM_{2.5}$ 21.62 (3.48, 59.65) $PM_{10-2.5}$ 5.72 (0.00, 19.78)
Neas et al., 1996 State College, PA (children)	---	pm PEFR -0.64 (-1.73, 0.44)	---	$PM_{2.5}$ 23.5 (max 85.8)
Neas et al., 1999 Philadelphia, PA (children)	am PEFR -8.17 (-14.81, -1.56) pm PEFR -1.44 (-7.33, 4.44)	am PEFR -3.29 (-6.64, 0.07) pm PEFR -0.91 (-4.04, 2.21)	am PEFR -4.31 (-11.44, 2.75) pm PEFR 1.88 (-4.75, 8.44)	$PM_{2.5}$ 22.2 (IQR 16.2) $PM_{10-2.5}$ 9.5 (IQR 5.1)
Schwartz and Neas, 2000 Uniontown, PA (reanalysis) (children)	---	pm PEFR -1.52, (-2.80, -0.24)	pm PEFR +1.73 (-2.2, 5.67)	$PM_{2.5}$ 24.5 (max 88.1) $PM_{10-2.5}$ NR
Schwartz and Neas, 2000 State College PA (reanalysis) (children)	---	pm PEFR -0.93 (-1.88, 0.01)	pm PEFR -0.28 (-3.45, 2.87)	$PM_{2.5}$ 23.5 (max 85.8) $PM_{10-2.5}$ NR
Vedal et al., 1998 Port Alberni, CAN (children)	PEF -1.35 (-2.7, -0.05)	---	---	PM_{10} median 22.1 (0.2, 159.0) (north site)

* Studies in italics available in 1996 CD
** mean (minimum, maximum) 24-h PM level shown in parentheses unless otherwise noted.

APPENDIX A, TABLE 3. ESTIMATED CARDIOVASCULAR MORBIDITY EFFECTS PER INCREMENTS IN 24-h CONCENTRATIONS OF PM_{10}, $PM_{2.5}$ AND $PM_{10-2.5}$ FROM U.S. AND CANADIAN STUDIES

Study Location*	% increase (95% CI) per 50 µg/m^3 PM_{10} Increase	% increase (95% CI) per 25 µg/m^3 $PM_{2.5}$ Increase	% increase (95% CI) per 25 µg/m^3 $PM_{10-2.5}$ Increase	PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ Mean (Range) Levels Reported**
Increased Hospitalization				
Total Cardiovascular:				
Samet et al., 2000 14 U.S. Cities (>65 years)	6.0 (5.1, 6.8)	---	---	PM_{10} means 24.4-45.3
Schwartz, 1999 8 U.S. Counties (>65 years)	5.0 (3.7, 6.4)	---	---	PM_{10} means 23-37
Linn et al., 2000 Los Angeles, CA (>29 years)	3.25 (2.04, 4.47)	---	---	PM_{10} 45.5 (5, 132)
Moolgavkar, 2000b Cook Co., IL (>65 years)	4.2 (3.0, 5.5)	---	---	PM_{10} median 35 (3, 365)
Moolgavkar, 2000b Los Angeles, CA (>65 years)	3.3 (2.0, 4.5)	(65+) 4.30 (2.52, 6.11) (<65) 3.54 (1.83, 5.27)	---	PM_{10} median 44, 7, 166) $PM_{2.5}$ median 22 (4, 86)
Moolgavkar, 2000b Maricopa Co., AZ (>65 years)	-2.4 (-6.9, 2.3)	---	---	PM_{10} median 41 (9, 252)
Morris and Naumova, 1998 Chicago, IL (>65 years)	3.92 (1.02, 6.90)	---	---	PM_{10} 41 (6, 117)
Schwartz, 1997 Tucson, AZ (>65 years)	6.07 (1.12, 1.27)	---	---	PM_{10} 42 (90% 63)
Gwynn et al., 2000 Buffalo, NY (all ages)	5.69 (-3.29, 15.50)	1.35 (-1.14, 4.28) (15 µg/m^3 $SO_4^=$)	---	PM_{10} 24.1 (6.8, 90.8) $SO_4^=$61.7 (0.78, 390.5) nmol/m^3
Tolbert et al., 2000a Atlanta, GA (all ages)	5.1 (-7.9, 19.9)	6.11 (-3.08, 16.17)	17.63 (-4.63, 45.07)	PM_{10} 29.1 (SD 12.0) $PM_{2.5}$ 19.4 (SD 9.35) $PM_{10-2.5}$ 9.39 (SD 4.52)

Stieb et al., 2000 St. John, CAN (all ages)	39.2 (5.0, 84.4)	15.11 (0.61, 11.03)	---	summer 93 PM_{10} 14.0 (max 70.3) $PM_{2.5}$ 8.5 (max 53.2)
Burnett et al., 1997 Toronto, CAN (all ages)	12.07 (1.43, 23.81)	7.18 (-0.61, 15.60)	20.46 (8.24, 34.06)	PM_{10} 28.4 (4, 102) $PM_{2.5}$ 16.8 (1, 66) $PM_{10-2.5}$ 11.6 (1, 56)
Ischemic Heart Disease:				
Schwartz and Morris 1995 *Detroit, MI*	*2.83 (0.72, 4.98)*	---	---	*PM_{10} 48 (22-82)***
Lippmann et al., 2000 Detroit, MI (>65 years)	8.91 (0.51, 18.03)	4.33 (-1.39, 10.39)	10.54 (2.73, 18.95)	PM_{10} 31 (max 105) $PM_{2.5}$ 18 (6, 86) $PM_{10-2.5}$ 13 (4, 50)
Burnett et al., 1999 Toronto, CAN (all ages)	8.56 (5.33, 11.48)	8.05 (5.38, 10.78)	3.74 (1.30, 6.25)	PM_{10} 30.2 (max 116) $PM_{2.5}$ 18.0 (max 90) $PM_{10-2.5}$ 12.2 (max 68)
Dysrhythmias:				
Tolbert et al., 2000a Atlanta, GA (all ages)	13.41 (-14.08, 48.99)	6.11 (-12.63, 28.86)	53.16 (2.07, 129.81)	$PM_{2.5}$ 19.4 (SD 9.35) $PM_{10-2.5}$ 9.39 (SD 4.52)
Lippmann et al., 2000 Detroit, MI (>65 years)	2.94 (-6.77, 13.65)	3.24 (-6.54, 14.04)	0.21 (-12.25, 14.43)	PM_{10} 31 (max 105) $PM_{2.5}$ 18 (6, 86) $PM_{10-2.5}$ 13 (4, 50)
Burnett et al., 1999 Toronto, CAN (all ages)	8.41 (2.89, 14.23)	6.06 (1.94, 10.35)	5.13 (-0.21, 10.75)	PM_{10} 30.2 (max 116) $PM_{2.5}$ 18.0 (max 90) $PM_{10-2.5}$ 12.2 (max 68)
Heart Failure:				
Schwartz and Morris, 1995 *Detroit, MI*	*5.04 (1.91, 8.27)*	---	---	*PM_{10} 48 (22-82)****
Linn et al., 2000 Los Angeles, CA (>29 years)	2.02 (-0.94, 5.06)	---	---	PM_{10} 45.5 (5, 132)

Lippmann et al., 2000 Detroit, MI (>65 years)	9.70 (0.17, 20.13)	9.06 (2.36, 16.19)	5.21 (-3.29, 14.46)	PM_{10} 31 (max 105) $PM_{2.5}$ 18 (6, 86) $PM_{10-2.5}$ 13 (4, 50)
Burnett et al., 1999 Toronto, CAN (all ages)	9.70 (4.17, 15.52)	6.59 (2.50, 10.83)	7.88 (2.28, 13.78)	PM_{10} 30.2 (max 116) $PM_{2.5}$ 18.0 (max 90) $PM_{10-2.5}$ 12.2 (max 68)
Myocardial Infarction:				
Linn et al., 2000 Los Angeles, CA (>29 years)	3.04 (0.06, 6.12)	---	---	PM_{10} 45.5 (5, 132)
Cardiac arrhythmia:				
Linn et al., 2000 Los Angeles, CA (>29 years)	1.01 (-1.93, 4.02)	---	---	PM_{10} 45.5 (5, 132)
Cerebrovascular:				
Linn et al., 2000 Los Angeles, CA (>29 years)	0.30 (-2.13, 2.79)	---	---	PM_{10} 45.5 (5, 132)
Moolgavkar, 2000b Cook Co., IL (>65 years)	3.22 (1.46, 5.03)	---	---	PM_{10} median 35 (3, 365)
Moolgavkar, 2000b Los Angeles, CA(>65 years)	1.00 (-1.78, 3.86)	1.51 (-0.76, 3.82)	---	$PM_{2.5}$ 22 (4, 86) $PM_{10-2.5}$ ---
Moolgavkar, 2000b Maricopa Co., AZ (>65 years)	1.00 (-8.40, 11.38)	---	---	PM_{10} median 41 (9, 252)
Burnett et al., 1999 Toronto, CAN (all ages)	"NEG" reported	"NEG" reported	"NEG" reported	PM_{10} 30.2 (max 116) $PM_{2.5}$ 18.0 (max 90) $PM_{10-2.5}$ 12.2 (max 68)
Peripheral circulation diseases:				
Burnett et al., 1999 Toronto, CAN (all ages)	2.58 (-2.67, 8.11)	"NEG" reported	5.63 (0.32, 11.23)	PM_{10} 30.2 (max 116) $PM_{2.5}$ 18.0 (max 90) $PM_{10-2.5}$ 12.2 (max 68)
Stroke:				

Linn et al., 2000 Los Angeles, CA (>29 years)	6.72 (3.64, 9.90)	---	---	PM_{10} 45.5 (5, 132)
Lippmann et al., 2000 Detroit, MI (>65 years)	4.80 (-5.47, 16.19)	1.80 (-5.30, 9.43)	4.90 (-4.69, 15.45)	PM_{10} 31 (max 105) $PM_{2.5}$ 18 (6, 86) $PM_{10-2.5}$ 13 (4, 50)

* Studies in italics available in 1996 CD
** mean (minimum, maximum) 24-h PM level shown in parentheses unless otherwise noted.

APPENDIX A, TABLE 4. Data used in creating Figures 3-4 through 3-9. Effect estimates and confidence intervals for PM-mortality and morbidity associations, and data for number of study days, number of health events per day, and the product of the number of days and number of events.

(A) PM_{10}-mortality associations

citation location, mortality category	effect estimate	lower confidence limit	upper confidence limit	number of days	mortality rate	mortality-day product	ln mortality-day
Samet et al., 2000, 90 U.S. city, total	2.30	0.10	4.50	**	**	1588776	14.278474
Samet 20-city total	2.58	0.41	4.79			1051794.5	13.866008311
Samet et al., 2000, 20 U.S. city, cardiorespiratory	3.45	1.01	5.94	**	**	577275.5	13.2660749012
Schwartz 2000, Chicago, total	4.53	3.11	5.96	2190	132	289080	12.574459
Styer et al., 1995, Chicago, total	4.08	0.08	8.24	2190	117	256230	12.453831
Burnett et al., 1998, Toronto, total	3.46	1.74	5.21	5475	40.17	219930.75	12.301068
Moolgavkar et al., 2000, LA, cardiovascular	4.47	1.65	7.37	3285	57	187245	12.140173
Ito and Thurston, 1996, Chicago, total	2.47	1.26	3.69	1529	116.5	178128.5	12.09026
Moolgavkar et al., 2000, Cook Co, cardiovascular	2.21	0.37	4.09	3285	43	141255	11.858322
Burnett et al., 2000, 8 Canadian cities, total	1.74	0.52	2.97	**	**	112102.6	11.62717
Ito and Thurston, 1996, Chicago, circulatory	1.49	-0.72	3.74	1529	56.2	85929.8	11.361286
Schwartz et al., 1996, St. Louis, total	3.04	0.76	5.37	1375	50.3	69162.5	11.144214
Schwartz et al., 1996, Boston, total	6.15	3.56	8.80	1140	60.2	68628	11.136456
Kinney et al., 1995, LA, total	2.47	-0.17	5.18	364	153	55692	10.927592
Moolgavkar et al., 2000, Maricopa, cardiovascular	8.85	2.67	15.39	3285	13	42705	10.662071
Pope et al., 1999, Salt Lake City, total	2.33	0.05	4.66	3700	11.32	41884	10.642659
Schwartz et al., 1996, Knoxville, total	4.58	0.27	9.08	1481	14.2	21030.2	9.9537148
Moolgavkar et al., 2000, LA, COPD	5.90	-1.64	14.03	3285	6	19710	9.8888814
Schwartz et al., 1996, Portage, total	3.55	-1.71	9.09	1436	13.6	19529.6	9.8796865
Schwartz et al., 1993, Birmingham, total	5.36	1.16	9.73	1087	17.1	18587.7	9.8302554
Lippmann et al., 2000, Detroit, total	4.41	-0.98	10.10	344	53	18232	9.8109336
Pope et al., 1999, Salt Lake City, cardiovascular	6.50	2.21	10.98	3700	4.72	17464	9.7678969
Ostro et al., 2000, Coachella Valley, total	2.01	-0.99	5.10	3011	5.8	17463.8	9.7678854
Fairley, 1999, Santa Clara, total	8.00	3.55	12.65	823	20	16460	9.7086885
Ito and Thurston, 1996, Chicago, respiratory	6.77	1.97	11.79	1529	9.8	14984.2	9.6147516
Moolgavkar et al., 2000, Cook Co., COPD	5.39	0.30	10.74	3285	4	13140	9.4834163
Pope et al., 1999, Provo/Orem, total	1.87	-2.14	6.04	3687	2.65	9770.55	9.187128
Gwynn et al., 2000, Buffalo, total	12.33	2.50	23.11	175	54	9450	9.15377
Mar et al., 2000, Phoenix, total	5.44	0.06	11.12	1095	8.55	9362.25	9.1444409
Lippmann et al., 2000, Detroit, circulatory	6.86	-1.28	15.66	344	25	8600	9.0595175

citation location, mortality category	effect estimate	lower confidence limit	upper confidence limit	number of days	mortality rate	mortality-day product	ln mortality-day
Ostro et al., 2000, Coachella Valley, cardiovascular	6.09	2.05	10.29	3011	2.7	8129.7	9.0032793
Moolgavkar et al., 2000, Maricopa, COPD	8.08	-4.58	22.41	3285	2	6570	8.7902691
Schwartz et al., 1996, Topeka, total	-2.48	-9.33	4.90	1432	4.5	6444	8.7709047
Ostro et al., 1999, Coachella Valley, total	4.60	0.58	8.78	1188	5.4	6415.2	8.7664255
Pope et al., 1999, Ogden, total	12.02	4.49	20.09	2308	2.55	5885.4	8.68023
Tsai et al., 2000, Newark NJ, total (PM15)	5.65	4.62	6.69	156	37	5772	8.6607739
Schwartz et al., 1996, Steubenville, total	4.58	0.76	8.54	1520	3.6	5472	8.6073995
Pope et al., 1992, Utah Valley, total	7.63	4.41	10.95	1706	2.7	4606.2	8.4351585
Pope et al., 1999, Provo/Orem, cardiovascular	8.60	2.40	15.18	3687	1.17	4313.79	8.3695721
Mar et al., 2000, Phoenix, cardiovascular	9.86	1.91	18.42	1095	3.85	4215.75	8.3465828
Pope et al., 2000, Salt Lake City, respiratory	8.17	-0.97	18.14	3700	0.96	3552	8.1752661
Gwynn et al., 2000, Buffalo, circulatory	17.83	0.69	37.88	175	19	3325	8.109225
Tsai et al., 2000, Newark NJ, cardiorespiratory	7.79	3.64	12.10	156	21	3276	8.0943784
Pope et al., 1999, Ogden, cardiovascular	1.41	-8.33	12.18	2308	1.14	2631.12	7.8751649
Ostro et al., 1999 , Coachella Valley, cardiovascular	8.33	2.14	14.89	1188	1.8	2138.4	7.6678132
Pope et al., 1992, Utah Valley, cardiovascular	9.36	1.91	17.36	1706	1.24	2115.44	7.6570181
Tsai et al., 2000, Elizabeth NJ, total	-4.88	-17.88	10.19	156	13	2028	7.6148054
Tsai et al., 2000, Camden NJ, total	11.07	0.70	22.51	156	11	1716	7.4477513
Ostro et al., 2000, Coachella Valley, respiratory	-1.99	-11.41	8.44	3011	0.52	1565.72	7.3561011
Lippmann et al., 2000, Detroit, respiratory	7.84	-10.18	29.47	344	4	1376	7.226936
Tsai et al., 2000, Elizabeth NJ, cardiorespiratory	3.05	-11.04	19.36	156	7	1092	6.9957662
Pope et al., 1999, Provo/Orem, respiratory	2.22	-9.83	15.89	3687	0.27	995.49	6.9032351
Tsai et al., 2000, Camden NJ, cardiorespiratory	15.03	4.29	26.87	156	6	936	6.8416155
Gwynn et al., 2000, Buffalo, respiratory	17.89	-14.87	63.25	175	5	875	6.7742239
Ostro et al., 1999, Coachella Valley, respiratory	13.88	3.25	25.61	1188	0.6	712.8	6.5692009
Pope et al., 1999, Ogden, respiratory	23.80	2.77	49.14	2308	0.26	600.08	6.397063
Pope et al., 1992, Utah Valley, respiratory	19.78	3.51	38.61	1706	0.27	460.62	6.1325734

** Data for mortality rate and number of days (respectively) for the multi-city studies were derived from the following tables: Burnett et al., 2000, Tables 2 and 3; Samet et al., 2000b, Tables A.1 and A.4; Schwartz et al., 1996, Tables 4 and 1.

(B) $PM_{2.5}$-Mortality Associations

citation location, mortality category	effect estimate	lower confidence limit	upper confidence limit	number of days	mortality rate	mortality-day product	ln mortality-day
Burnett et al., 1998, Toronto, total	4.79	3.26	6.34	5475	40.17	219930.75	12.301068
Moolgavkar et al., 2000, LA, cardiovascular	2.59	0.38	4.85	3285	57	187245	12.140173
Schwartz 2000, Boston, total	5.33	1.81	8.98	2920	60	175200	12.073683

citation location, mortality category	effect estimate	lower confidence limit	upper confidence limit	number of days	mortality rate	mortality-day product	ln mortality-day
Goldberg et al., 2000, Montreal, total	5.81	3.36	8.32	3653	38.6	141005.8	11.856556
Burnett et al., 2000, 8 Canadian cities, total	3.03	1.10	4.99	**	**	117452	11.673785
Ostro et al., 1995, So. California, total	0.28	-0.61	1.17	2555	40.73	104065.15	11.552772
Schwartz et al., 1996, St. Louis, total	2.77	1.13	4.44	1375	50.3	69162.5	11.144214
Schwartz et al., 1996, Boston, total	5.59	3.80	7.41	1140	60.2	68628	11.136456
Goldberg et al., 2000, Montreal, cardiovascular	3.48	-0.16	7.26	3653	15.7	57352.1	10.956965
Ostro et al., 1995, So. California, circulatory	0.69	-0.35	1.74	2555	18.74	47880.7	10.776468
Schwartz et al., 1996, Knoxville, total	3.54	0.52	6.65	1481	14.2	21030.2	9.9537148
Moolgavkar et al., 2000, LA, COPD	2.67	-3.38	9.10	3285	6	19710	9.8888814
Schwartz t al., 1996, Portage, total	3.03	-0.84	7.05	1436	13.6	19529.6	9.8796865
Lippmann et al., 2000, Detroit, total	3.10	-0.63	6.98	344	53	18232	9.8109336
Goldberg et al., 2000, Montreal, respiratory	21.65	12.95	31.01	3653	3.1	11324.3	9.3347061
Ostro et al., 1995, So. California, respiratory	2.08	-0.35	4.51	2555	3.83	9785.65	9.1886723
Mar et al., 2000, Phoenix, total	3.03	-0.69	6.88	1095	8.55	9362.25	9.1444409
Lippmann et al., 2000, Detroit, circulatory	3.17	-2.29	8.94	344	25	8600	9.0595175
Fairley, 1999, Santa Clara, total	8.48	3.38	13.84	408	20	8160	9.0069994
Schwartz et al., 1996, Topeka, total	2.01	-4.83	9.35	1432	4.5	6444	8.7709047
Ostro et al., 2000, Coachella Valley, total	11.51	0.21	24.09	1041	5.8	6037.8	8.705795
Tsai et al., 2000, Newark NJ, total	4.34	2.82	5.89	156	37	5772	8.6607739
Schwartz et al., 1996, Steubenville, total	2.52	-0.24	5.35	1520	3.6	5472	8.6073995
Mar et al., 2000, Phoenix, cardiovascular	18.68	5.72	33.23	1095	3.85	4215.75	8.3465828
Tsai et al., 2000, Newark NJ, cardiorespiratory	5.13	3.09	7.21	156	21	3276	8.0943784
Ostro et al., 2000, Coachella Valley, cardiovascular	8.56	-6.35	25.84	1041	2.7	2810.7	7.9411888
Tsai et al., 2000, Elizabeth NJ, total	1.77	-5.45	9.53	156	13	2028	7.6148054
Tsai et al., 2000, Camden NJ, total	5.65	0.11	11.51	156	11	1716	7.4477513
Lippmann et al., 2000, Detroit, respiratory	2.28	-10.31	16.63	344	4	1376	7.226936
Tsai et al., 2000, Elizabeth NJ, cardiorespiratory	2.28	-4.97	10.08	156	7	1092	6.9957662
Tsai et al., 2000, Camden NJ, cardiorespiratory	6.18	0.61	12.06	156	6	936	6.8416155
Ostro et al., 2000, Coachella Valley, respiratory	-13.28	-43.05	32.06	1041	0.52	541.32	6.2940106

** Data for mortality rate and number of days (respectively) for the multi-city studies were derived from the following tables: Burnett et al., 2000, Tables 2 and 3; Samet et al., 2000b, Tables A.1 and A.4; Schwartz et al., 1996, Tables 4 and 1.

(C) $PM_{10-2.5}$-Mortality Associations

citation location, mortality category	effect estimate	lower confidence limit	upper confidence limit	number of days	mortality rate	mortality-day product	ln mortality-day
Burnett et al., 2000, 8 Canadian cities, total	1.82	-0.72	4.43	**	**	112186.7	11.62792

citation location, mortality category	effect estimate	lower confidence limit	upper confidence limit	number of days	mortality rate	mortality-day product	ln mortality-day
Schwartz et al., 1996, St. Louis, total	0.50	-1.73	2.78	1375	50.3	69162.5	11.144214
Schwartz et al., 1996, Boston., total	0.50	-1.73	2.78	1140	60.2	68628	11.136456
Schwartz et al., 1996, Knoxville, total	2.52	-1.46	6.66	1481	14.2	21030.2	9.9537148
Schwartz et al., 1996, Portage, total	1.25	-3.06	5.76	1436	13.6	19529.6	9.8796865
Lippmann et al., 2000, Detroit, total	3.96	-1.22	9.42	344	53	18232	9.8109336
Ostro et al., 2000, Coachella Valley, total	1.28	-0.63	3.22	2990	5.8	17342	9.7608866
Lippmann et al., 2000, Detroit, respiratory	7.41	-9.07	26.87	344	25	8600	9.0595175
Fairley, 1999, Santa Clara, total	4.53	-6.66	17.05	408	20	8160	9.00699944796
Ostro et al., 2000, Coachella Valley, circulatory	2.56	0.66	4.49	2990	2.7	8073	8.9962804
Mar et al., 2000, Phoenix, total	2.97	-0.50	6.56	300	22.9	6870	8.8349194
Schwartz et al., 1996, Topeka, total	-3.22	-7.89	1.69	1432	4.5	6444	8.7709047
Schwartz et al., 1996, Steubenville, total	6.11	1.30	11.15	1520	3.6	5472	8.6073995
Mar et al., 2000, Phoenix, cardiovascular	6.45	1.42	11.73	1095	3.85	4215.75	8.3465828
Ostro et al., 2000, Coachella Valley, respiratory	-1.27	-6.24	3.95	2990	0.52	1554.8	7.3491022
Lippmann et al., 2000, Detroit, circulatory	7.82	0.03	16.23	344	4	1376	7.226936

** Data for mortality rate and number of days (respectively) for the multi-city studies were derived from the following tables: Burnett et al., 2000, Tables 2 and 3; Samet et al., 2000b, Tables A.1 and A.4; Schwartz et al., 1996, Tables 4 and 1.

(D) Associations between PM_{10} and admissions to the hospital or emergency room

citation location, admissions category	effect estimate	lower confidence limit	upper confidence limit	number of days	admissions rate	admissions-day product	ln admissions-day
Linn et al., 2000, LA, respiratory	2.89	1.09	4.72	3640	207	753480	13.532458
Burnett et al., 1999, Toronto, respiratory	14.20	9.32	19.30	5475	13	71175	11.172897
Gwynn et al., 2000, Buffalo, respiratory	3.14	-1.78	8.31	812	56.3	45715.6	10.730195
Schwartz et al., 1996, Cleveland, respiratory	5.83	0.54	11.40	1095	22	24090	10.089552
Moolgavkar et al., 1997, Minn/St. Paul, respiratory	8.72	4.59	13.01	1979	10.55	20878.45	9.9464728
Moolgavkar, et al., 1997, Birmingham, respiratory	1.51	-1.43	4.54	2098	8.26	17329.48	9.7601644
Stieb et al., 2000, St. John, respiratory	8.84	1.84	16.32	1260	10.9	13734	9.5276298
Burnett et al., 1997,Toronto, respiratory	6.95	2.91	11.15	388	23.7	9195.6	9.1264804
Schwartz et al., 1995, New Haven, respiratory	6.00	-0.28	12.68	1095	8.1	8869.5	9.0903737
Schwartz et al., 1995, Tacoma, respiratory	10.00	3.21	17.24	1095	4.2	4599	8.4335942
Schwartz et al., 1996, Spokane, respiratory	8.50	3.61	13.62	821	3.9	3201.9	8.0714997
Delfino et al., 1993, Montreal, respiratory	40.49	11.25	77.43	92	20.12	1851.04	7.5235029
Thurston et al., 1994 Toronto, respiratory	23.26	2.03	44.49	**	**	1693	7.43425738213
Moolgavkar, 2000c, LA, COPD	6.09	1.09	11.34	3285	20	65700	11.092854
Samet et al., 2000b, 14 U.S. Cities, COPD	10.30	7.68	12.98	**	**	60683.31	11.013424

citation location, admissions category	effect estimate	lower confidence limit	upper confidence limit	number of days	admissions rate	admissions-day product	ln admissions-day
Moolgavkar, 2000c, Cook Co,, COPD	2.41	-0.21	5.11	3285	12	39420	10.582029
Burnett et al., 1999, Toronto, COPD	6.90	1.32	12.78	5475	5	27375	10.217385
Moolgavkar, 2000c, Maricopa Co., COPD	6.92	-4.15	19.25	3285	4	13140	9.4834163
Schwartz, 1994, Detroit, COPD	10.63	4.41	17.21	1191	5.8	6907.8	8.8404065
Moolgavkar et al., 1997, Minn/St. Paul, COPD	6.89	-0.64	14.99	1979	2.91	5758.89	8.6585
Moolgavkar et al., 2000, King Co., COPD	15.93	5.2	27.75	2022	2.33	4711.26	8.4577107
Lippmann et al., 2000, Detroit, COPD	9.60	-5.28	26.82	490	8	3920	8.2738469
Tolbert et al., 2000, Atlanta, COPD	-3.45	-33.01	29.92	350	9.7	3395	8.130059
Chen et al., 2000, Reno, COPD	9.41	2.20	17.12	1815	1.72	3121.8	8.046165
Schwartz, 1994, Birmingham, COPD	12.69	3.81	22.34	1369	2.2	3011.8	8.0102932
Schwartz, 1994, Minn/St. Paul, COPD	25.30	9.47	43.42	1251	2.2	2752.2	7.9201559
Schwartz, et al., 1996, Spokane, COPD	17.10	7.85	27.14	821	1	821	6.7105231
Burnett et al., 1999, Toronto, asthma	8.88	3.65	14.36	5475	11	60225	11.005843
Sheppard et al., 1999, Seattle, asthma	13.70	5.46	22.58	2920	2.7	7884	8.9725907
Tolbert et al., 2000a, Atlanta, asthma	13.24	1.21	26.70	276	22	6072	8.7114433
Tolbert et al., 2000b, Atlanta, asthma	18.77	-8.65	54.42	350	15.8	5530	8.6179431
Lipsett et al., 1997, Santa Clara, asthma	9.09	2.72	15.85	368	7.6	2796.8	7.9362312
Nauenberg and Basu, 1999, LA, asthma	20.02	5.33	34.71	315	8.74	2753.1	7.9204828
Choudbury et al., 1997, Anchorage, asthma	20.72	11.65	29.79	1095	2.42	2649.9	7.8822772
Norris et al., 2000, Spokane, asthma	2.35	-10.93	17.61	816.7	3.2	2613.44	7.8684226
Norris et al., 1999, Seattle, asthma	75.91	25.08	147.39	468.5	1.9	890.15	6.79139
Norris et al., 1998, Seattle, asthma	56.20	10.38	121.06	487	1.8	876.6	6.7760508
Samet et al., 2000b, 14 U.S. cities, pneumonia	10.30	7.70	13.00	**	**	168894.37	12.037029
Schwartz, 1994, Detroit, pneumonia	5.92	1.95	10.05	1191	15.7	18698.7	9.8362093
Moolgavkar et al., 1997, Minn/St. Paul, pneumonia	3.54	-0.49	7.72	1979	7.64	15119.56	9.6237445
Schwartz, 1994, Birmingham, pneumonia	9.09	3.51	14.97	1369	5.9	8077.1	8.9967882
Schwartz, 1994, Minn/St. Paul, pneumonia	8.17	1.22	15.59	1251	6	7506	8.923458
Lippmann et al., 2000, Detroit, pneumonia	21.43	8.18	36.29	490	12	5880	8.679312
Schwartz et al., 1996, Spokane, pneumonia	5.30	-1.51	12.58	821	1.9	1559.9	7.352377
Linn et al., 2000, LA, cardiovascular	3.25	2.04	4.47	3640	428	1557920	14.258862
Samet et al., 2000b, 14 U.S. cities, cardiovascular	5.99	5.15	6.83	**	**	673571.53	13.420349
Moolgavkar, 2000b, LA, cardiovascular	3.23	1.17	5.32	3285	172	565020	13.244616
Moolgavkar, 2000b, Cook Co., cardiovascular	4.24	3.00	5.50	3285	110	361350	12.797602
Moolgavkar, 2000b, Maricopa Co., cardiovascular	-2.39	-6.90	2.35	3285	33	108405	11.593629
Gwynn et al., 2000, Buffalo, cardiovascular	10.98	3.79	18.66	812	83	67396	11.118341
Schwartz et al., 1999, 8 US Counties, cardiovascular	5.02	3.67	6.39	1095	31.5	34492.5	10.448497

citation location, admissions category	effect estimate	lower confidence limit	upper confidence limit	number of days	admissions rate	admissions-day product	ln admissions-day
Burnett et al., 1997,Toronto, cardiovascular	7.66	0.93	14.84	388	42.6	16528.8	9.7128596
Tolbert et al., 2000, Atlanta, cardiovascular	5.10	-7.88	19.91	350	45.1	15785	9.6668154
Schwartz, 1997, Tucson, cardiovascular	6.07	1.12	11.27	829.9	13.4	11120.66	9.3165599
Stieb et al., 2000, St. John, cardiovascular	32.51	10.20	59.34	1260	3.5	4410	8.39163
Burnett et al., 1999, Toronto, ischemic heart disease	8.36	5.33	11.48	5475	24	131400	11.786001
Schwartz and Morris, 1995, Detroit, ischemic heart disease	2.83	0.72	4.98	1191	44.1	52523.1	10.869008
Lippmann et al., 2000, Detroit, ischemic heart disease	8.91	0.51	18.03	490	22	10780	9.2854478
Burnett et al., 1999, Toronto, dysrhythmia	8.41	2.89	14.23	5475	5	27375	10.217385
Tolbert et al., 2000, Atlanta, dysrhythmia	13.14	-14.08	48.99	350	11.2	3920	8.2738469
Lippmann et al., 2000, Detroit, dysrhythmia	2.94	-6.76	13.65	490	7	3430	8.1403155
Burnett et al., 1999, Toronto, CHD/heart failure	9.70	4.17	15.52	5475	9	49275	10.805172
Morris et al., 1995, Chicago, CHD/heart failure	3.92	1.02	6.90	1168	34	39712	10.589409
Schwartz and Morris, 1995, Detroit, CHD/heart failure	5.04	1.91	8.27	1191	26.2	31204.2	10.348308
Lippmann et al., 2000, Detroit, CHD/heart failure	9.70	0.17	20.13	490	17	8330	9.0276187

** Data for admissions rate and number of days (respectively) were derived from the following tables: Thurston et al., 1994, Samet et al., 2000b, Tables 7 and 9

(E) Associations between $PM_{2.5}$ and admissions to the hospital or emergency room

citation location, admissions category	effect estimate	lower confidence limit	upper confidence limit	number of days	admissions rate	admissions-day product	ln admissions-day
Burnett et al., 1999, Toronto, respiratory	10.77	7.18	14.47	5475	13	71175	11.172897
Lumley and Heagerty, 1999, King Co., respiratory	5.92	1.10	10.97	2920	7.5	21900	9.9942419
Stieb et al., 2000, St. John, respiratory	5.69	0.62	11.02	1260	10.9	13734	9.5276298
Burnett et al., 1997, Toronto, respiratory	6.24	2.48	10.14	388	23.7	9195.6	9.1264804
Lippmann et al., 2000, Detroit , respiratory	12.51	3.69	22.08	490	12	5880	8.679312
Delfino et al., 1997, Montreal, respiratory	23.88	4.94	42.83	95	26.9	2555.5	7.8460032
Delfino et al., 1998, Montreal, respiratory	13.17	-0.22	26.57	92	20.12	1851.04	7.5235029
Thurston et al., 1994, Toronto, respiratory	15	2	28	**	**	1693	7.4342574
Moolgavkar, 2000c, LA, COPD	5.08	0.91	9.41	3285	20	65700	11.092854
Burnett et al., 1999, Toronto, COPD	4.78	-0.17	9.98	5475	5	27375	10.217385
Moolgavkar, et al., 2000, King Co.	6.40	0.90	12.10	3287	7.75	25474.25	10.145442
Lippmann et al., 2000, Detroit, COPD	5.49	-4.72	16.80	490	8	3920	8.2738469
Tolbert et al., 2000, Atlanta, COPD	12.44	-7.88	37.24	350	9.7	3395	8.130059
Burnett et al., 1999, Toronto, asthma	6.45	2.47	10.57	5475	11	60225	11.005843
Sheppard et al., 1999, Seattle, asthma	8.66	3.29	14.32	2920	2.7	7884	8.9725907
Tolbert et al., 2000, Atlanta, asthma	2.27	-14.79	22.73	350	15.8	5530	8.6179431

citation location, admissions category	effect estimate	lower confidence limit	upper confidence limit	number of days	admissions rate	admissions-day product	ln admissions-day
Norris et al., 1999, Seattle, asthma	44.50	21.70	71.40	487	1.8	876.6	6.7760508
Moolgavkar, 2000b, LA, cardiovascular	4.30	2.52	6.11	3285	172	565020	13.244616
Burnett et al., 1997, Toronto, cardiovascular	5.90	1.79	10.18	388	42.6	16528.8	9.7128596
Tolbert et al., 2000, Atlanta, cardiovascular	6.11	-3.07	16.16	350	45.1	15785	9.6668154
Stieb et al., 2000, St. John, cardiovascular	15.11	-0.25	32.82	1260	3.5	4410	8.39163
Burnett et al., 1999, Toronto, ischemic heart disease	8.05	5.38	10.78	5475	24	131400	11.786001
Lippmann et al., 2000, Detroit, ischemic heart disease	4.33	-1.39	10.39	490	22	10780	9.2854478
Burnett et al., 1999, Toronto, dysrhythmia	6.06	1.94	10.35	5475	5	27375	10.217385
Tolbert et al., 2000, Atlanta, dysrhythmia	6.11	-12.62	28.85	350	11.2	3920	8.2738469
Lippmann et al., 2000, Detroit, dysrhythmia	3.24	-6.54	14.04	490	7	3430	8.1403155
Burnett et al., 1999, Toronto, CHD/heart failure	6.59	2.50	10.83	5475	9	49275	10.805172
Lippmann et al., 2000, Detroit, CHD/heart failure	9.06	2.36	16.19	490	17	8330	9.0276187

** Data for admissions rate and number of days (respectively) were derived from the following tables: Thurston et al., 1994, Table 1

(F) Associations between $PM_{10-2.5}$ and admissions to the hospital or emergency room

citation location, admissions category	study number	effect estimate	lower confidence limit	upper confidence limit	number of days	admissions rate	admissions-day product	ln admissions-day
Burnett et al., 1999, Toronto, respiratory	1	9.31	4.64	14.18	5475	13	71175	11.172897
Burnett et al., 1997, Toronto, respiratory	2	8.46	3.51	13.64	388	23.7	9195.6	9.1264804
Lippmann et al., 2000, Detroit, respiratory	3	11.90	0.65	24.41	490	12	5880	8.679312
Thurston et al., 1994, Toronto, respiratory	4	22.25	-9.53	54.03	**	**	1693	7.4342574
Moolgavkar, 2000b, LA, COPD	5	5.08	-0.44	10.90	3285	20	65700	11.092854
Burnett et al., 1999, Toronto, COPD	6	12.83	4.93	21.33	5475	5	27375	10.217385
Lippmann et al., 2000, Detroit, COPD	7	9.29	-4.19	24.66	490	8	3920	8.2738469
Tolbert et al., 2000, Atlanta, COPD	8	-23.03	-50.68	20.12	350	9.7	3395	8.130059
Burnett et al., 1999, Toronto, asthma	9	11.05	5.75	16.62	5475	11	60225	11.005843
Sheppard et al., 1999, Seattle, asthma	10	11.12	2.83	20.08	2920	2.7	7884	8.9725907
Tolbert et al., 2000, Atlanta, asthma	11	21.08	-18.21	79.25	350	15.8	5530	8.6179431
Burnett et al., 1997, Toronto, cardiovascular	12	13.46	5.52	22.01	388	42.6	16528.8	9.7128596
Tolbert et al., 2000, Atlanta, cardiovascular	13	17.63	-4.61	45.05	350	45.1	15785	9.6668154
Burnett et al., 1999, Toronto, ischemic heart disease	14	3.74	1.30	6.25	5475	24	131400	11.786001
Lippmann et al., 2000, Detroit, ischemic heart disease	15	10.54	2.73	18.95	490	22	10780	9.2854478
Burnett et al., 1999, Toronto, dysrhythmia	16	5.13	-0.21	10.75	5475	5	27375	10.217385
Tolbert et al., 2000, Atlanta, dysrhythmia	17	53.16	2.15	129.65	350	11.2	3920	8.2738469

citation location, admissions category	study number	effect estimate	lower confidence limit	upper confidence limit	number of days	admissions rate	admissions-day product	ln admissions-day
Lippmann et al., 2000, Detroit, dysrhythmia	18	0.21	-12.25	14.43	490	7	3430	8.1403155
Burnett et al., 1999, Toronto, CHD/heart failure	19	7.88	2.28	13.78	5475	9	49275	10.805172
Lippmann et al., 2000, Detroit, CHD/heart failure	20	5.21	-3.29	14.46	490	71	34790	10.457085

** Data for admissions rate and number of days (respectively) were derived from the following tables: Thurston et al., 1994, Table 1

APPENDIX B

FIGURES AND TABLES FOR
CHAPTER 5, SECTION 5.2, ON VISIBILITY

FIGURES:

Figure 5-1 and 5-2 – In Staff Paper Text

Washington, DC Images

[See Figures 3 through 10 at the Staff Paper Web Site, www.epa.gov/ttn/oarpg/t1sp.html, in file WASHDC8IMAGES. These images were generated using WinHaze 2.8.0.]

Chicago, IL Images

[See Figures 11 through 16 at the Staff Paper Web Site, www.epa.gov/ttn/oarpg/t1sp.html, in file CHICAGO6IMAGES. These are actual photographs provided by Illinois EPA.]

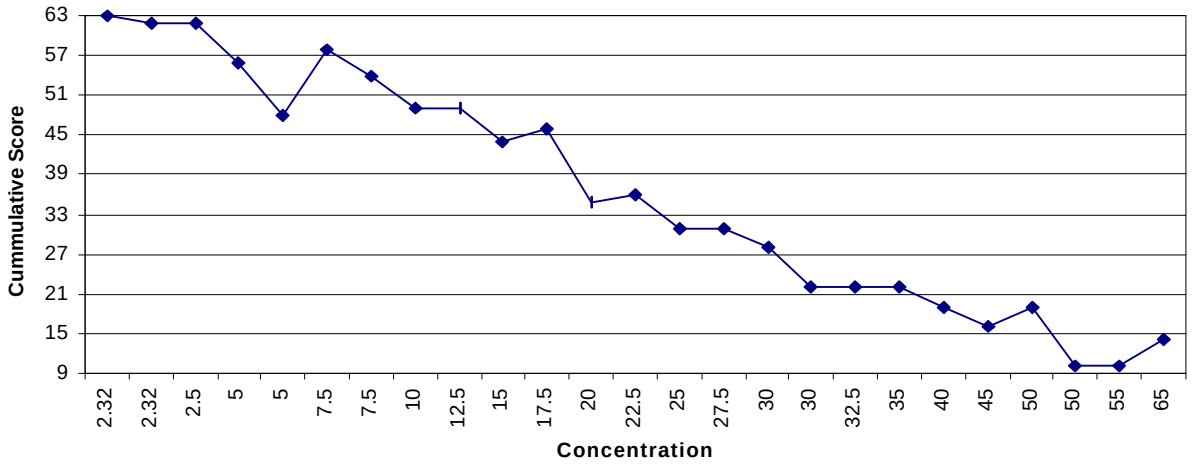

Figure 17. Rating of Visual Air Quality for Washington, DC Images. November 2000 Pilot Project.

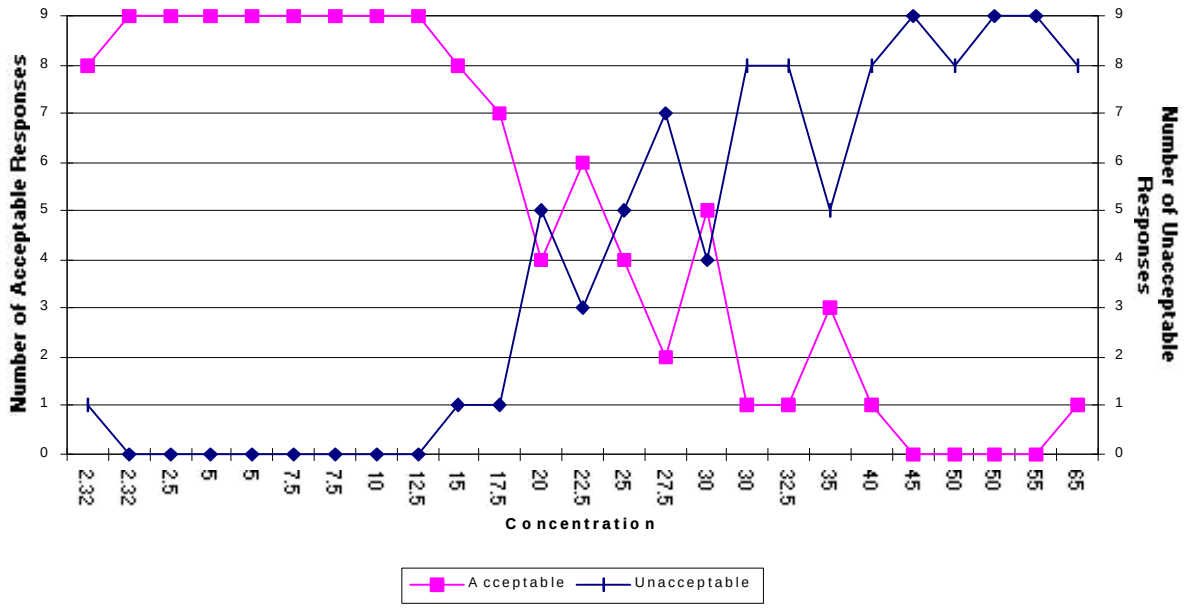

Figure 18. Rating of Acceptability / Unacceptability for Washington, DC Images. November 2000 Pilot Project.

Denver, Colorado Images

[See Figures 19 through 26 at the Staff Paper Web Site, www.epa.gov/ttn/oarpg/t1sp.html, in file DENVER8IMAGES. These images were generated using WinHaze 2.8.0.]

Figure 19. Denver, CO – 35 Mm^{-1}
Figure 20. Denver, CO – 43 Mm^{-1}
Figure 21. Denver, CO – 51 Mm^{-1}
Figure 22. Denver, CO – 61 Mm^{-1}
Figure 23. Denver, CO – 76 Mm^{-1}
Figure 24. Denver, CO – 93 Mm^{-1}
Figure 25. Denver, CO – 167 Mm^{-1}
Figure 26. Denver, CO – 258 Mm^{-1}

Phoenix, Arizona Images

[See Figures 27 through 34 at the Staff Paper Web Site, www.epa.gov/ttn/oarpg/t1sp.html, in file PHOENIX8IMAGES. These images were generated using WinHaze 2.8.0.]

Figure 27. Phoenix, AZ – 2.5 $\mu g/m^3$ $PM_{2.5}$
Figure 28. Phoenix, AZ – 5 $\mu g/m^3$ $PM_{2.5}$
Figure 29. Phoenix, AZ – 10 $\mu g/m^3$ $PM_{2.5}$
Figure 30. Phoenix, AZ – 15 $\mu g/m^3$ $PM_{2.5}$
Figure 31. Phoenix, AZ – 20 $\mu g/m^3$ $PM_{2.5}$
Figure 32. Phoenix, AZ – 30 $\mu g/m^3$ $PM_{2.5}$
Figure 33. Phoenix, AZ – 40 $\mu g/m^3$ $PM_{2.5}$
Figure 34. Phoenix, AZ – 65 $\mu g/m^3$ $PM_{2.5}$

TABLES:

Table 1. Aerosol Concentrations Used to Create Washington, DC Images.

Percent of Fine Mass		Sulfate: 50%	Nitrate: 10%	OC: 25%	EC: 10%	Soil: 5%	Coarse: 30% x fine mass
Slide	**Image (ug/m3)**	**Sulfate (ug/m3)**	**Nitrate (ug/m3)**	**OC (ug/m3)**	**EC (ug/m3)**	**Soil (ug/m3)**	**Coarse (ug/m3)**
1	65.0	32.50	6.50	16.25	6.50	3.25	19.50
2	60.0	30.00	6.00	15.00	6.00	3.00	18.00
3	55.0	27.50	5.50	13.75	5.50	2.75	16.50
4	52.5	26.25	5.25	13.13	5.25	2.63	15.75
5	50.0	25.00	5.00	12.50	5.00	2.50	15.00
6	47.5	23.75	4.75	11.88	4.75	2.38	14.25
7	45.0	22.50	4.50	11.25	4.50	2.25	13.50
8	42.5	21.25	4.25	10.63	4.25	2.13	12.75
9	40.0	20.00	4.00	10.00	4.00	2.00	12.00
10	37.5	18.75	3.75	9.38	3.75	1.88	11.25
11	35.0	17.50	3.50	8.75	3.50	1.75	10.50
12	32.5	16.25	3.25	8.13	3.25	1.63	9.75
13	30.0	15.00	3.00	7.50	3.00	1.50	9.00
14	27.5	13.75	2.75	6.88	2.75	1.38	8.25
15	25.0	12.50	2.50	6.25	2.50	1.25	7.50
16	22.5	11.25	2.25	5.63	2.25	1.13	6.75
17	20.0	10.00	2.00	5.00	2.00	1.00	6.00
18	17.5	8.75	1.75	4.38	1.75	0.88	5.25
19	15.0	7.50	1.50	3.75	1.50	0.75	4.50
20	12.5	6.25	1.25	3.13	1.25	0.63	3.75
21	10.0	5.00	1.00	2.50	1.00	0.50	3.00
22	7.50	3.75	0.75	1.88	0.75	0.38	2.25
23	6.25	3.13	0.63	1.56	0.63	0.31	1.88
24	5.00	2.50	0.50	1.25	0.50	0.25	1.50
25	3.75	1.88	0.38	0.94	0.38	0.19	1.13
26	2.50	1.25	0.25	0.63	0.25	0.13	0.75
27	2.32 (natural) *	0.20	0.10	1.50	0.02	0.50	3.00

* Note: For slide 27, NO2 = 0.0 ppb

Table 2. Visibility Parameters for Washington, DC Images.

Slide	$PM_{2.5}$ (ug/m3)	Light Extinction (Mm-1)	Visual Range (km)	Deciviews
1	65.0	507	7.7	39.3
2	60.0	469	8.3	38.5
3	55.0	431	9.1	37.6
4	52.5	412	9.5	37.2
5	50.0	393	10.0	36.7
6	47.5	374	10.5	36.2
7	45.0	355	11.0	35.7
8	42.5	336	11.6	35.1
9	40.0	317	12.3	34.6
10	37.5	298	13.1	33.9
11	35.0	279	14.0	33.3
12	32.5	260	15.0	32.6
13	30.0	241	16.2	31.8
14	27.5	222	17.6	31.0
15	25.0	203	19	30.1
16	22.5	184	21	29.1
17	20.0	165	24	28.0
18	17.5	146	27	26.8
19	15.0	127	31	25.4
20	12.5	108	36	23.8
21	10.0	89	44	21.9
22	7.50	70	56	19.5
23	6.25	61	64	18.0
24	5.00	51	76	16.3
25	3.75	42	94	14.3
26	2.50	32	122	11.7
27	2.32 (natural)	21	185	7.5