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

Policy Assessment of Scientific and Technical Information

OAQPS Staff Paper – First Draft

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Office of Air Quality Planning and Standards
U.S. Environmental Protection Agency
Research Triangle Park, NC 27711

DISCLAIMER

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http:/www.epa.gov/ttn/naaqs/standards/pm/s_pm_cr_sp.html

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Abbreviations and Acronyms

AC	Automated colorimetry
ACS	American Cancer Society
AHSMOG	Adventist Health and Smoke Study
AIRS	Aerometric Information Retrieval System
APHEA	Air Pollution and Health, a European Approach
AQCD	Air Quality Criteria Document
AQS	Air Quality System
ASOS	Automated Surface Observing System
BC	Black carbon
BS	British or black smoke
CAA	Clean Air Act
CAMM	Continuous Ambient Mass Monitor
CAP	Concentrated ambient particles
CASAC	Clean Air Scientific Advisory Committee
CASTNet	Clean Air Status and Trends Network
C_B	Base cation
CD	Criteria Document
CDC	Centers for Disease Control
CDPHE	Colorado Department of Public Health and Environment
CFR	Code of Federal Regulations
C:N	Carbon-to-nitrogen ratio
CO	Carbon monoxide
COH	Coefficient of haze
COPD	Chronic obstructive pulmonary disease
C-R	Concentration-response
CSS	Coastal sage scrub community
CV	Contingent valuation
EC	Elemental carbon

ECG	Electrocardiogram
EEA	Essential Ecological Attribute
EPA	Environmental Protection Agency
EPEC Framework	Ecological Processes and Effects Subcommittee document, *Framework for Assessing and Reporting on Ecological Condition*
FRM	Federal reference method
GAM	Generalized additive models
GCVTC	Grand Canyon Visibility Transport Commission
GLM	Generalized linear models
HAPs	Hazardous air pollutants
HEI	Health Effects Institute
hosp. adm.	Hospital admissions
IC	Ion chromatography
IFS	Integrated Forest Study
IMPROVE	Interagency Monitoring of Protected Visual Environments
LML	Lowest measured level
LPC	Laser particle counter
LRS	Lower respiratory symptoms
mort.	Mortality
NAAQS	National ambient air quality standards
NADP	National Atmospheric Deposition Program
NAPAP	National Acid Precipitation Assessment Program
NCEA	National Center for Environmental Assessment
NDDN	National Dry Deposition Network
NMMAPS	National Mortality and Morbidity Air Pollution Study
N_2	Nonreactive, molecular nitrogen
NO_2	Nitrogen dioxide
non-accid mort	Non-accidental mortality

Nr	Reactive nitrogen
NSMPS	Nano-scanning mobility particle sizer
NuCM	Nutrient cycling model
O_3	Ozone
OAQPS	Office of Air Quality Planning and Standards
OAR	Office of Air and Radiation
OC	Organic carbon
ORD	Office of Research and Development
PAHs	Polynuclear aromatic hydrocarbons
pneum.	Pneumonia
PTEAM	EPA's Particle Total Exposure Assessment Methodology
PCBs	Polychlorinated biphenyls
PCDD/F	Polychlorinated dibenzo-p-dioxins/dibenzofurans
PM	Particulate matter
$PM_{10-2.5}$	Particles less than or equal to 10 μm in diameter and greater than 2.5 μm in diameter
$PM_{2.5}$	Particles less than or equal to 2.5 μm in diameter
PM_{10}	Particles less than or equal to 10 μm in diameter
POPs	Persistent organic pollutants
RR	Relative risk
SAB	Science Advisory Board
SMPS	Standard scanning mobility particle sizer
SO_2	Sulfur dioxide
SO_4	Sulfate
SOCs	Semivolatile organic compounds
STN	$PM_{2.5}$ Chemical Speciation Trends Network
SP	Staff Paper
TEOM	Tapered Element Oscillating Microbalance sensor
TMO	Thermal manganese oxidation method
TOR	Thermal/optical reflectance method

TOT	Thermal/optical transmission method
TSP	Total suspended particulates
µg	micrograms
µg/m^3	micrograms per cubic meter
URS	Upper respiratory symptoms
U.S.	United States
UV	Ultraviolet
UV-B	Ultraviolet-B
V_d	Deposition velocity
VOCs	Volatile organic compounds
XRF	X-ray fluorescence

1. INTRODUCTION

1.1 PURPOSE

This draft Staff Paper, prepared by the U.S. Environmental Protection Agency's (EPA) Office of Air Quality Planning and Standards (OAQPS), identifies the key policy-relevant scientific information contained in the draft document, *Air Quality Criteria for Particulate Matter – Fourth External Review Draft* (EPA, 2003; henceforth referred to as the draft Criteria Document (CD) and cited as CD), prepared by EPA's National Center for Environmental Assessment (NCEA). Building upon an earlier preliminary draft Staff Paper (EPA, 2001), this document includes results from initial staff analyses (e.g., analyses of air quality and visibility data, and human health risk analyses), identifies additional analyses to be incorporated into a subsequent draft of this document, and presents preliminary staff conclusions and recommendations.

The final version of this Staff Paper will evaluate the policy implications of the key studies and scientific information contained in the final EPA document, *Air Quality Criteria for Particulate Matter* (targeted for completion by the end of 2003), and identify the critical elements that EPA staff believes should be considered in the current review of the national ambient air quality standards (NAAQS) for particulate matter (PM). This policy assessment is intended to help "bridge the gap" between the scientific review contained in the CD and the judgments required of the EPA Administrator in determining whether it is appropriate to revise the NAAQS for PM. 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, averaging times, forms[1], 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 PM NAAQS, as well as staff conclusions and recommendations of options for the Administrator to consider.

[1] The "form" of a standard defines the air quality statistic that is to be compared to the level of the standard in determining whether an area attains the standard.

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

1.2 BACKGROUND

1.2.1 Legislative Requirements

Two sections of the Clean Air Act (Act) govern the establishment and revision of the NAAQS. Section 108 (42 U.S.C. 7408) directs the Administrator to identify "air pollutants" that "in his judgment, may reasonably be anticipated to endanger public health and welfare" and whose "presence . . . in the ambient air results from numerous or diverse mobile or stationary sources" and, if listed, 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."[2] 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."[3]

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[2] 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)].

[3] 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."

In setting standards that are "requisite" to protect public health and welfare, as provided in section 109(b), EPA's task is to establish standards that are neither more nor less stringent than necessary for these purposes. In so doing, EPA may not consider the costs of implementing the standards. See generally *Whitman v. American Trucking Associations*, 531 U.S. 457, 464, 475-76 (2001).

The requirement that primary standards include an adequate margin of safety 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, in selecting primary standards that include 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) at 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.

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 and promulgate such new standards as may be appropriate . . . ." Section 109(d)(2) 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 new . . . standards and

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), which is administered by EPA's Science Advisory Board Staff Office.

1.2.2 History of PM NAAQS Reviews

EPA first established national ambient air quality standards for PM 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[4] 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

[4] 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.

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).

In December 1994, EPA presented its plan for the second periodic review of the criteria and NAAQS for PM to the CASAC, and promulgated significant revisions to the NAAQS in 1997 (62 FR 38652, July 18, 1997). In that decision, EPA revised the PM NAAQS 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. EPA established two new $PM_{2.5}$ standards: 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 98^{th} percentile of 24-hour $PM_{2.5}$ concentrations at each population-oriented monitor within an area. A new reference method for the measurement of $PM_{2.5}$ in the ambient air was also established, as were rules for determining attainment of the new standards. 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 99^{th} percentile of 24-hour PM_{10} concentrations at each monitor in an area. EPA revised the secondary standards by making them identical in all respects to the primary standards.

1.2.3 Litigation Related to 1997 PM Standards

Following promulgation of the revised PM NAAQS, petitions for review were filed by a large number of parties, addressing a broad range of issues. In May 1998, a three-judge panel of

the U.S. Court of Appeals for the District of Columbia Circuit issued an initial decision that upheld EPA's decision to establish fine particle standards, holding that "the growing empirical evidence demonstrating a relationship between fine particle pollution and adverse health effects amply justifies establishment of new fine particle standards." *American Trucking Associations v. EPA* , 175 F. 3d 1027, 1055-56 (D.C. Cir. 1999) (rehearing granted in part and denied in part, 195 F. 3d 4 (D.C. Cir. 1999), affirmed in part and reversed in part, *Whitman v. American Trucking Associations*, 531 U.S. 457 (2001). The Panel also found "ample support" for EPA's decision to regulate coarse particle pollution, but vacated the 1997 revised PM_{10} standards, concluding in part that PM_{10} is a "poorly matched indicator for coarse particulate pollution" because it includes fine particles. Id. at 1053-55. As a result of this aspect of the court's ruling, which EPA did not appeal, the 1987 PM_{10} standards remain in effect. In the current review, EPA is addressing coarse-fraction particles in the current review by considering standards based on an indicator of $PM_{10-2.5}$, referring to particles with a mean aerodynamic diameter greater than 2.5 μm but less than or equal to 10 μm.

More generally, the Panel held (with one dissenting opinion) that EPA's approach to establishing the level of the standards in 1997, both for PM and for ozone NAAQS promulgated on the same day, effected "an unconstitutional delegation of legislative authority." Id. at 1034-40. Although the Panel stated that "the factors EPA uses in determining the degree of public health concern associated with different levels of ozone and PM are reasonable," it remanded the NAAQS to 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. Consistent with EPA's long-standing interpretation, the Panel also reaffirmed prior rulings holding that in setting NAAQS EPA is "not permitted to consider the cost of implementing those standards." Id. at 1040-41.

Both sides filed cross appeals on these issues to the United States Supreme Court, and the Court granted *certiorari*. In February 2001, the Supreme Court issued a unanimous decision upholding EPA's position on both issues. *Whitman v. American Trucking Associations*, 531 U.S. 457, 464, 475-76. On the constitutional issue, the Court held that the statutory requirement that NAAQS be "requisite" to protect public health with an adequate margin of safety sufficiently

guided EPA's discretion, affirming EPA's approach of setting standards that are neither more nor less stringent than necessary. The Supreme Court remanded the case to the Court of Appeals for resolution of any remaining issues that had not been addressed in that court's earlier rulings. Id. at 475-76. In March 2002, the Court of Appeals rejected all remaining challenges to the standards, holding under the traditional standard of review that EPA's $PM_{2.5}$ standards were reasonably supported by the administrative record and were not "arbitrary and capricious." *American Trucking Associations v. EPA*, 283 F. 3d 355, 369-72 (D.C. Cir. 2002). Thus, the 1997 $PM_{2.5}$ standards are in effect.

1.2.4 Current PM NAAQS Review

In October 1997, EPA published its plans for the current periodic review of the PM NAAQS (62 FR 55201, October 23, 1997), including the 1997 $PM_{2.5}$ standards and the 1987 PM_{10} standards. As part of the process of preparing the PM CD, in April 1999, 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 in December 1999. Based on CASAC and public comment, NCEA revised the draft CD and released a second external review draft (March 2001) for review by CASAC and the public at a meeting held in July 2001. A preliminary draft Staff Paper (EPA, 2001) was released in June 2001 for public comment and for consultation with CASAC at the same public meeting. A third external review draft CD was released in May 2002 for review at a meeting held in July 2002.

Shortly after EPA released the third external review draft CD, the Health Effects Institute (HEI)[5] announced that researchers at Johns Hopkins University had discovered problems with applications of statistical software used in a number of important epidemiological studies that had been discussed in that draft CD. In response to this significant issue, EPA took steps in consultation with CASAC to encourage researchers to reanalyze affected studies and to submit them expeditiously for peer review by a special expert panel convened at EPA's request by HEI. EPA subsequently incorporated the results of this reanalysis and peer-review process into a

[5] HEI is an independent research institute, jointly sponsored by EPA and a group of U.S. manufacturers/marketers of motor vehicle and engines, that conducts health effects research on major air pollutants related to motor vehicle emissions.

fourth external review draft CD, which was released in June 2003 for review by CASAC and the public at an August 2003 meeting.

This draft Staff Paper is being provided to CASAC and the public for review at a meeting planned for November 2003. Following that meeting, EPA will complete the staff analyses and take CASAC and public comments into account in preparing a second draft Staff Paper, to be based on the final CD, and will make that draft document available for further review and comment by CASAC and the public.

The schedule for completion of this review is now governed by a consent decree resolving a lawsuit filed in March 2003 by a group of plaintiffs representing national environmental organizations. The lawsuit alleged that EPA had failed to perform its mandatory duty, under section 109(d)(1) [42 U.S.C. 7409(d)(1)], of completing the current review within the period provided by statute. *American Lung Association v. Whitman* (No. 1:03CV00778, D.D.C. 2003). Entered by the court after an opportunity for public comment, the consent decree provides that EPA will issue a final PM CD no later than December 19, 2003, and that EPA will sign for publication notices of proposed and final rulemaking concerning its review of the PM NAAQS no later than March 31, 2005 and December 20, 2005, respectively. These dates are premised on the expectation that a series of interim milestones will be met, including the release of a second draft PM Staff Paper by April 30, 2004, followed by CASAC and public review of that draft by July 31, 2004, with completion of a final PM Staff Paper by September 30, 2004.

1.3 APPROACH

The final Staff Paper will take into account the scientific evidence reviewed in the final CD, and will include: 1) the results of comparative air quality analyses, the final human health risk assessment, and analyses examining visibility impairment; 2) the staff's overall evaluation of the adequacy of the current primary and secondary NAAQS; and 3) staff conclusions and recommendations as to whether any revisions are appropriate to address public health and welfare effects associated with fine- and coarse-fraction particles. For these purposes, 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 the various technical analyses presented in this draft Staff Paper, the staff has focused separately on fine- and coarse-fraction particles, building upon the conclusions reached in the last review, and taking into account the new information that has become available. More specifically, air quality analyses to characterize spatial and temporal air quality patterns have been conducted in terms of $PM_{2.5}$ and $PM_{10-2.5}$ as the indicators for fine- and coarse-fraction particles, respectively. Similarly, the current draft human health risk assessment focuses on analyzing various health effects associated with exposure to ambient $PM_{2.5}$ and $PM_{10-2.5}$, as well as analyses for PM_{10} that provide insight into the health risks associated with exposure to $PM_{2.5}$ and/or $PM_{10-2.5}$.

Following this introductory chapter, this draft Staff Paper is organized into five other chapters. Chapter 2 focuses on air quality characterizations (e.g., information on atmospheric chemistry, sources of PM, measurement methods, and spatial and temporal patterns in ambient PM concentrations based on extensive newly available air quality monitoring data) as well as information on the relationships 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. The results of an interim draft human health risk assessment are presented in Chapter 4, together with information on plans to conduct additional analyses of risk reductions associated with just attaining specified alternative $PM_{2.5}$ and $PM_{10-2.5}$ standards. Information on welfare effects of ambient PM is presented in Chapter 5, together with analyses of data on visibility impairment. Chapter 6 presents preliminary staff conclusions and recommendations on the current PM NAAQS and possible alternative $PM_{2.5}$ and $PM_{10-2.5}$ primary and secondary standards for consideration by the Administrator. The preliminary staff recommendations on possible alternative standards, together with comments received from CASAC and the public on this draft Staff Paper, will help to form the basis for the additional risk analyses to be conducted for inclusion in the second draft Staff Paper.

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Environmental Protection Agency. (2001) Review of the National Ambient Air Quality Standards for Particulate Matter: Policy Assessment of Scientific and Technical Information – Preliminary Draft OAQPS Staff Paper. June.

Environmental Protection Agency. (2003) Air Quality Criteria for Particulate – Fourth External Review Draft . Research Triangle Park, NC: Office of Research and Development; report no. EPA/600/P-99/002. June.

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 classes of 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 for making recommendations on 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 discusses sources of ambient PM and provides a summary of national estimates of source emissions. Section 2.4 presents information on the methods used to measure ambient PM and some important considerations in the design of these methods. Section 2.5 presents data on PM concentrations, trends, and spatial patterns in the United States. Section 2.6 provides information on the temporal variability of PM. Much of the information in Sections 2.5 and 2.6 is derived from analyses of data collected by the nationwide networks of $PM_{2.5}$ and PM_{10} monitors. Section 2.7 defines and discusses background levels of ambient PM. Section 2.8 addresses the relationships between ambient PM levels and human exposure to PM. Finally, Section 2.9 summarizes relevant information on the optical and radiative effects of ambient particles.

2.2 CHARACTERIZATION OF 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, and atmospheric behavior. Fine particles and coarse 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 justified consideration of fine and coarse particles as separate pollutants (EPA 1996a,

p. 13-3), and this conclusion is reiterated in the draft CD (CD, p. 2-36 and 9-4). The fundamental differences between fine and coarse particles are also important considerations in assessing the available health effects and exposure information.

2.2.1 Particle Size Distributions

Particle properties and 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, including how far the particles are able to penetrate, where they deposit, and how effective the body's clearance mechanisms are in removing them. Furthermore, particle size is one of the most important parameters in determining the residence time of particles in ambient air, a key consideration in assessing exposure. Particle size is also a major determinant of visibility impairment, a welfare effect linked to ambient particles. Particle surface area, number, chemical composition, and water solubility all vary with particle size, and are also a variable of the formation processes and emissions sources.

Common conventions for classifying particles by size include: (1) modes, based on observed particle size distributions and formation mechanisms; and (2) "cut points," based on the inlet characteristics of a specific PM sampling device. The terminology used in this draft Staff Paper for describing these classifications is summarized in Table 2-1 and discussed in detail in the following subsections.

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 or volume (CD, p. 2-7). These modes are apparent in Figure 2-1, which shows average ambient distributions of particle number, surface area, and

[1] In this Staff Paper, particle size or diameter refers to a normalized measure called aerodynamic diameter unless otherwise noted. Most ambient particles are irregularly shaped rather than spherical. 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. 2-4).

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

Term	Description
Size Distribution Modes	
Coarse Particles ("Coarse Mode")	The distribution of particles larger than the intermodal minimum in volume or mass distributions; this minimum generally occurs between 1 and 3 μm. These particles usually represent the greatest amount of mass.
Fine Particles	The distribution of particles smaller than the intermodal minimum in volume or mass distributions; this minimum generally occurs between 1 and 3 μm. This includes particles in the nucleation, Aitkin, and accumulation modes.
Accumulation-Mode Particles	A subset of fine particles with diameters above about 0.1 μm. Ultrafine particles grow by coagulation or condensation and "accumulate" in this size range. Particles in this mode usually represent the most surface area.
Ultrafine Particles	A subset of fine particles with diameters below about 0.1 μm, encompassing the Aitkin and nucleation modes. Particles in this size range are usually the most numerous.
Aitkin-Mode Particles	A subset of ultrafine particles with diameters between 0.01 and 0.1 μm.
Nucleation-Mode Particles	Freshly formed particles with diameters below 0.01 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 (50% collection efficiency) of 10 μm aerodynamic diameter. This measurement includes the fine particles and the inhalable part of coarse particles, and is an indicator for 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 (50% collection efficiency) of 2.5 μm aerodynamic diameter. The collected particles include most of the fine particles. A small portion of coarse-mode particles may be included depending on the sharpness of the sampler efficiency curve and the tail of the coarse-mode may extend into the fine fraction.
$PM_{10-2.5}$ ("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 inhalable fraction of coarse-mode particles.

volume by particle size.[2] Panel (a) illustrates that by far, the largest number of ambient particles in a typical distribution are very small, below 0.1 μm in diameter, while panel (c) indicates most of the particle volume, and therefore most of the mass, is found in particles with diameters larger than 0.1 μm.[3] Most of the surface area (panel b) is between 0.1 and 1.0 μm. The surface area distribution in panel (b) peaks around 0.2 μm. Distributions may vary across locations, conditions, and time due to differences in sources, atmospheric conditions, topography, and the age of the aerosol.

As illustrated in panel (c) of Figure 2-1, volume distributions typically measured in ambient air in the U.S. are found to be bimodal, with overlapping tails, and an intermodal minimum between 1 and 3 μm (CD, p. 2-7). The distribution of particles that are mostly larger than this minimum are called "coarse particles," and the distribution of particles that are mostly smaller than the minimum are called "fine particles." Fine particles can be subcategorized into smaller modes: "nucleation mode," "Aitkin mode," and "accumulation mode." Together, nucleation-mode and Aitkin-mode particles make up "ultrafine particles."[4] The nucleation mode is apparent as the largest peak in the number distribution in panel (a), and is also visible in the surface area distribution in panel (b). Nucleation-mode and Aitkin-mode particles have relatively low mass and grow rapidly into accumulation-mode particles, so they are not commonly observed as a separate mode in volume or mass distributions. The accumulation mode is apparent as the leftmost peak in the volume distribution in panel (c) and the largest peak in the surface area distribution in panel (b).

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[2] Particle size distributions, such as those in Figure 2-1, are often expressed in terms of the logarithm of the particle diameter (D_p) on the X-axis and the measured concentration difference on the Y-axis. When the Y-axis concentration difference is plotted on a linear scale, the number of particles, the particle surface area, and the particle volume (per cm^3 air) having diameters in the size range from log D_p to log($D_p + \Delta D_p$) are proportional to the area under that part of the size distribution curve.

[3] Mass is proportional to volume by density.

[4] Whitby (1978) did not identify a separate ultrafine particle mode between 0.01 and 0.1 μm, and therefore the Aitkin mode is not illustrated in Figure 2-1.

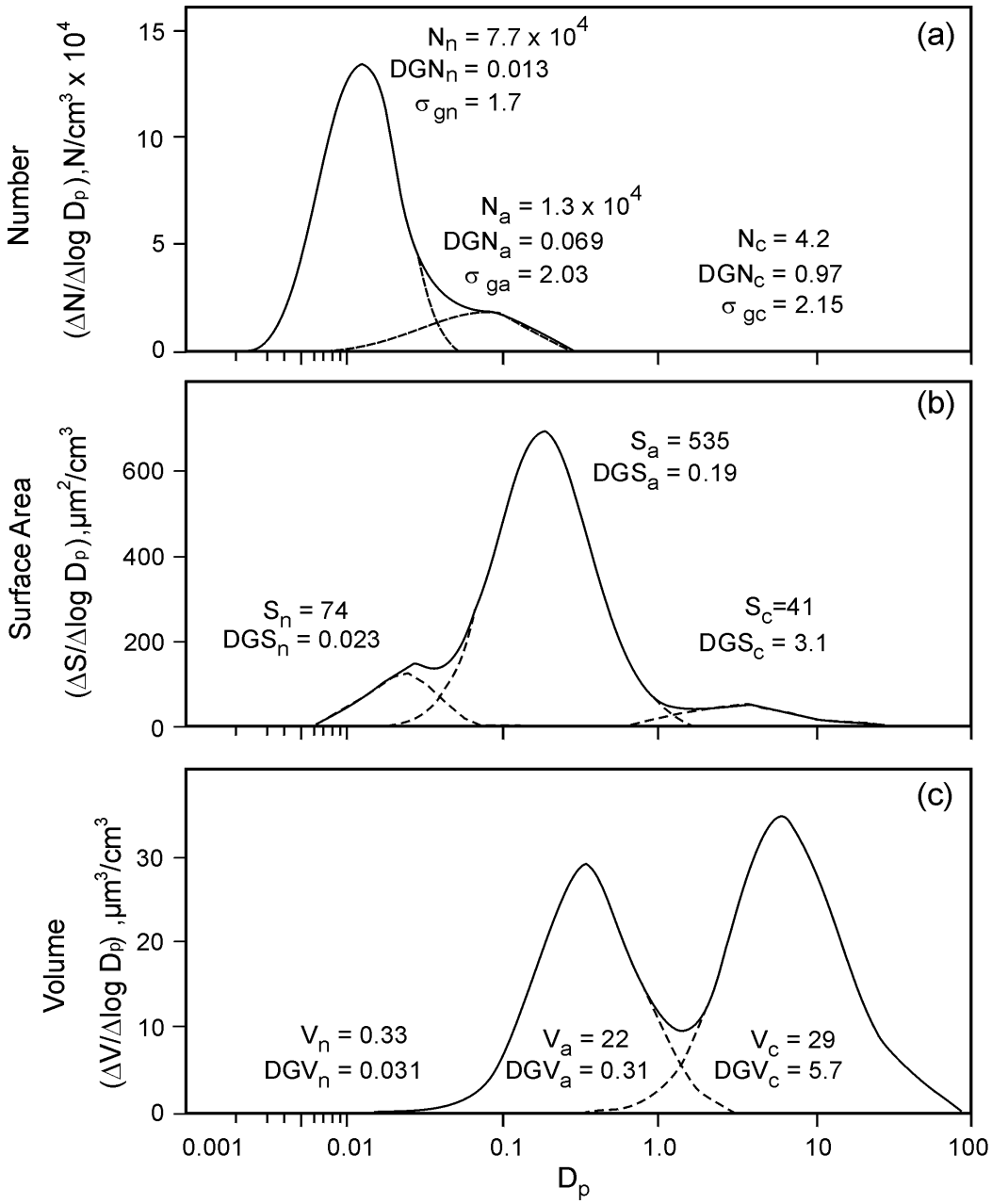

Figure 2-1. Distribution of coarse [subscript c], accumulation [subscript a], and nucleation [subscript n] -mode particles by three characteristics: Panel (a) number [N], Panel (b) surface area [S], and Panel (c) volume [V] for the grand average continental size distribution. D_p = particle diameter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

Source: Whitby (1978); CD, page 2-8.

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 inlets that are defined by their 50 percent cut point, which is the particle aerodynamic diameter at which exactly 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 50% cut point of x μm aerodynamic diameter. Because of the overlap in the distributions of fine- and coarse-mode ambient particles, and the fact that samplers do not have perfect cut points, no single sampler can precisely separate them. 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 NAAQS using cut points of interest. 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 (Hi Vol).[5] As illustrated in Figure 2-2, TSP typically includes particles with diameters less than about 40 μm, but could include even larger particles under certain conditions. 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 pass deeply into the human respiratory tract. In 1997, EPA established new standards for fine particles measured as $PM_{2.5}$. The dashed lines in Figure 2-2 illustrate the distribution of particles captured by the PM_{10} Federal Reference Method (FRM) sampler[6], including all fine and some coarse-mode particles, and the distribution captured by the $PM_{2.5}$ FRM sampler[7], including the potential capture of a small subset of coarse-mode particles.

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[5] 40 CFR Part 50, Appendix B, Reference Method for the Determination of Suspended Particulate Matter in the Atmosphere (High-Volume Method).

[6] 40 CFR Part 50, Appendix J, Reference Method for the Determination of Particulate Matter as PM_{10} in the Atmosphere.

[7] 40 CFR Part 50, Appendix L, Reference Method for the Determination of Fine Particulate Matter as $PM_{2.5}$ in the Atmosphere.

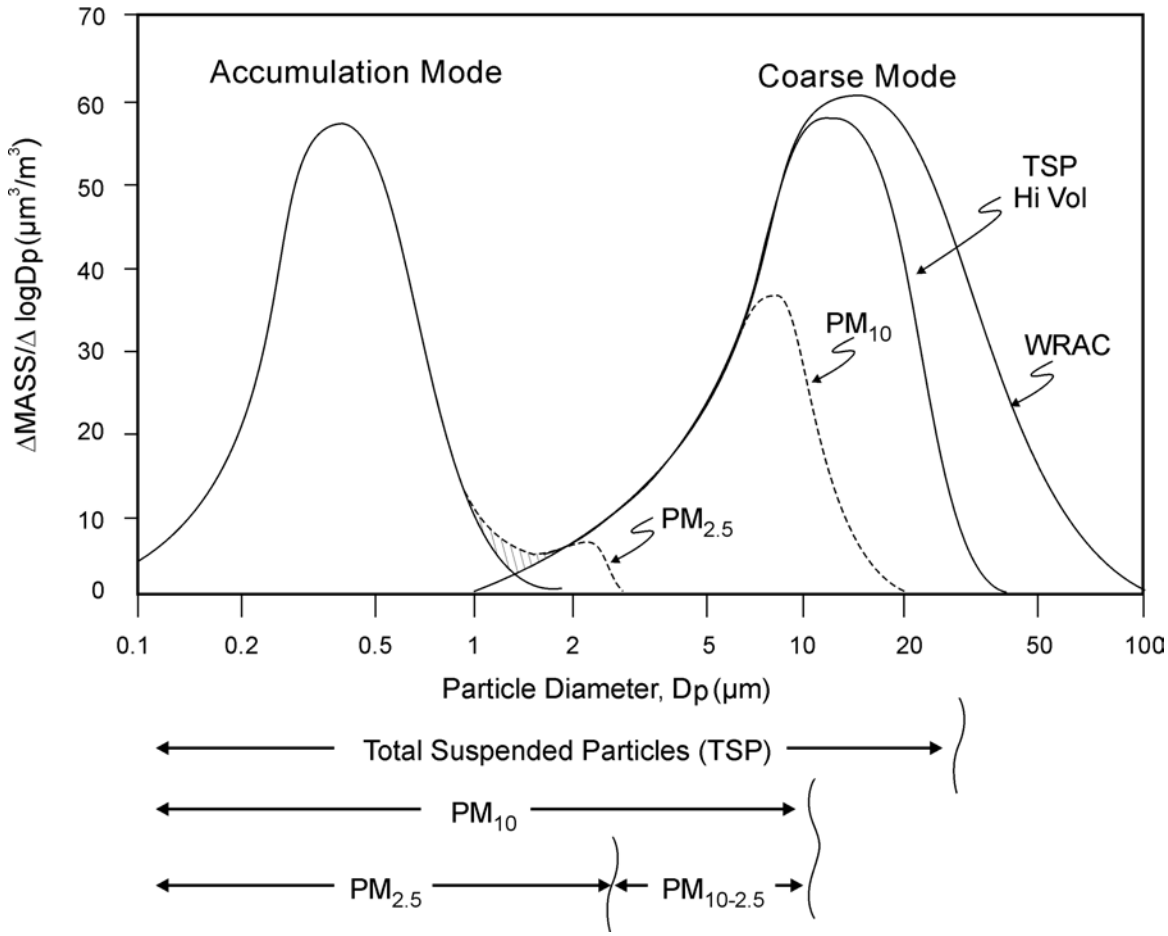

Figure 2-2. An idealized distribution of ambient PM showing fine and coarse particles and the fractions collected by size-selective samplers. (WRAC is the Wide Range Aerosol Classifier which collects the entire coarse mode).

Source: Adapted from Wilson and Suh (1997) and Whitby (1978); CD page 2-16.

The EPA is now considering establishing standards for another PM indicator identified in Table 2-1 as $PM_{10-2.5}$, which represents the inhalable fraction of coarse-mode particles. Section 2.4 discusses measurement methods for this indicator. The reader should 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 ambient PM concentrations. Fine and coarse particles generally have distinct sources and formation mechanisms, although there is some overlap (CD, p. 3-60). Coarse particles are generally primary particles, meaning they are emitted from their source directly as particles. Most coarse particles result from mechanical disruption of large particles by crushing or grinding, from evaporation of sprays, or from dust resuspension. Specific sources include construction and demolition activities, mining and mineral processing, sea spray, wind-blown dust, and resuspension of settled biological material from soil surfaces and roads. The amount of energy required to break down primary particles into smaller particles normally limits coarse particle sizes to greater than 1.0 μm diameter (EPA 1996a, p. 13-7). Some combustion-generated particles, such as fly ash, are also found in the coarse mode.

By contrast, fine particles are produced in high energy processes such as combustion or by atmospheric photochemistry. Common directly emitted fine particles include unburned carbon particles from combustion, and nucleation-mode particles emitted as combustion-related vapors that condense within seconds of being exhausted to ambient air. Fossil-fuel combustion sources include motor vehicles, power generation facilities, industrial facilities, residential wood burning, agricultural burning, and forest fires.

The formation and growth of fine particles are influenced by three processes: (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, the weak bonding of two or more particles into one larger particle. Gas phase material condenses preferentially on smaller particles since they have the greatest surface area, and the rate constant for coagulation of two particles decreases as the particle size increases. Thus, ultrafine particles grow into the accumulation mode, but accumulation-mode particles do not normally grow into the coarse mode (CD, p. 2-20).

Secondary formation processes can result in either new particles or the addition of PM to pre-existing particles. 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 various sulfate particles (e.g., ammonium sulfate $(NH_4)_2SO_4$ or ammonium bisulfate

NH_4HSO_4); (2) the conversion of nitrogen dioxide (NO_2) to nitric acid (HNO_3) droplets which react 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 (saturation) vapor pressures that nucleate or condense on existing particles to form secondary organic aerosol particles (CD, p. 3-62 to 3-72).

2.2.3 Chemical Composition

Based on studies conducted in most parts of the U.S., the draft CD reports that sulfate, ammonium, and hydrogen ions; black carbon, secondary organic compounds, and primary organic species from cooking and combustion; and certain metals, primarily from combustion processes, are found predominately in fine particles. Crustal-related materials such as calcium, aluminum, silicon, magnesium, and iron; and primary organic materials such as pollen, spores, and plant and animal debris are found predominantly in coarse particles (CD, p. 2-29).

Some components, such as potassium and nitrate, may be found in both fine and coarse particles. Potassium in coarse particles comes almost entirely from soil. Potassium in fine particles comes mainly from emissions of burning wood or cooking meat. Nitrate in fine particles comes mainly from the reaction of gas-phase nitric acid with gas-phase ammonia to form ammonium nitrate particles. Nitrate in coarse particles comes primarily from the reaction of gas-phase nitric acid with pre-existing coarse particles (CD, p. 2-29).

Many ambient particles also contain water (i.e., particle-bound water) as a result of an equilibrium between water vapor and hygroscopic particles (CD, p. 2-31). Particle-bound water influences the size of particles and in turn their aerodynamic and light scattering properties (discussed in section 2.9). The amount of particle-bound water will vary with the composition of particles. Sulfates, nitrates, and secondary organic compounds are more hygroscopic than BC, primary OC, and crustal material.

2.2.4 Fate and Transport

Fine- and coarse-mode particles typically exhibit different behavior in the atmosphere. These differences may affect several exposure-related considerations including the representativeness of central-site monitored values and the penetration of particles formed outdoors into indoor spaces. The ambient residence time of atmospheric particles varies with size. Ultrafine particles grow rapidly into the accumulation mode, and have a very short life, on the order of minutes to hours. Ultrafine particles are also small enough to be removed through diffusion to falling rain drops. Accumulation-mode particles remain suspended longer, due to collisions with air molecules, and have relatively low surface deposition rates. They can be transported thousands of kilometers and remain in the atmosphere for days to weeks. Accumulation-mode particles serve as condensation nuclei for cloud droplet formation and are eventually removed from the atmosphere as falling rain drops. Accumulation-mode particles that are not involved in cloud processes are removed from the atmosphere by falling rain, or eventually by gravitational settling and impaction on surfaces.

By contrast, coarse-mode particles can settle rapidly from the atmosphere with lifetimes ranging from minutes to hours depending on their size, atmospheric conditions, and their altitude. 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 gravitational settling and by impaction on surfaces. Smaller-sized coarse-mode particles can have longer lifetimes and longer travel distances, especially in extreme circumstances, such as intercontinental dust storms (CD, p. 2-35). Coarse-mode particles also are readily removed by falling rain drops (CD, p. 2-35).

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

Table 2-2. Comparison of Fine and Coarse Particles

	Fine		Coarse
	Ultrafine	Accumulation	
Aerodynamic Diameter	< 0.1 μm	0.1 to ≤ 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 Reaction of gases in or on particles 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 ions, Black carbon, Metal compounds, Organic compounds with very low saturation vapor pressure at ambient temperatures, particle-bound water.	Sulfate (SO_4^{-2}), Nitrate (NO_3^-), Ammonium (NH_4^+), and Hydrogen (H^+) ions Black carbon, Large variety of organic compounds Metal compounds of Pb, Cd, V, Ni, Cu, Zn, Mn, Fe, K, etc.) Particle-bound water	Suspended soil or street dust Fly ash from uncontrolled combustion of coal, oil, wood Nitrates/chlorides associated with basic metal oxides Oxides of crustal elements (Si, Al, Ti, Fe, Mg) $CaCO_3$, NaCl, sea salt Pollen, mold, fungal spores Plant and 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 except for sea salt
Sources:	Combustion of coal, oil, gasoline, diesel fuel, wood Atmospheric transformation of SO_2 and some organic compounds High temperature industrial processes, smelters, steel mills, etc. Volcanic activity Wildfires	Combustion of coal, oil, gasoline, diesel fuel, wood Atmospheric transformation products of NO_x, SO_2, and organic compounds including biogenic organic species (e.g., terpenes) High temperature industrial processes, smelters, steel mills, etc. 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 Wind-blown dust
Atmospheric half-life:	Minutes to hours	Days to weeks	Minutes to hours Days for particles suspended by dust storms
Removal Processes:	Grows into accumulation mode Diffusion to 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. 2-37.

2.3 SOURCE EMISSIONS

The distribution and amount of emissions of pollutants that contribute to ambient PM can provide insights into observed ambient levels. The links between source emissions and ambient concentrations of PM can include complex, non-linear atmospheric processes, including gaseous chemical reactions and pollution transport.

Source emissions can be measured using monitoring equipment or estimated using emission inventory methods. For most source types, emissions inventory methods are the most practical. The EPA routinely publishes national estimates of annual source emissions of pollutants that contribute to ambient PM concentrations. In general, national emissions estimates are uncertain, and there have been few field studies to test emission inventories against observations. 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 power plants measured by continuous instruments), while fugitive dust sources should be regarded as order-of-magnitude (CD, p. 3-98). The EPA is working to reduce these uncertainties through advances in the understanding of the fate and transport characteristics of fugitive dust emissions released at ground level.

Table 2-3 provides a summary of recent annual estimates of national emissions of primary PM and PM precursors. While reviewing the following discussion on emissions estimates, the reader should keep in mind that national estimates, while instructive, can obscure important distinctions in the relative contributions of different sources across smaller geographic regions, including important differences between urban and rural areas.

2.3.1 Primary PM Emissions

The majority of directly emitted anthropogenic PM is estimated to be coarse particles. Though highly uncertain, recent national estimates of $PM_{10-2.5}$ emissions shown in Table 2-3 are about 2.5 times higher than estimates of $PM_{2.5}$ emissions – 16.9 million short tons compared to 6.8 million short tons. A large portion of primary PM emissions are attributed to a variety of small area-wide sources, which are often more difficult to characterize and are more uncertain than larger point source emissions.

Table 2-3. Annual National Emissions Estimates of Primary PM and PM Precursors (thousand short tons), 1999

Source	NO_x	SO_2	NH_3	VOC [a]	Primary $PM_{2.5}$	Primary $PM_{10-2.5}$
Fuel Combustion	10,026	16,091	48	904	766	263
Industrial Processes	942	1,465	289	7,996	913	350
On-Road Vehicles	8,590	363	260	5,297	229	66
Nonroad Engines and Vehicles	5,515	936	10	3,232	411	47
Agriculture	0	0	769	8	860	3,438
Fire	–	–	–	699	853	133
Livestock	0	0	3,552	0	89	501
Miscellaneous	320	12	–	9	22	2
Fugitive Dust	0	0	0	0	2,631	12,105
Biogenic/Natural	2,315	–	35	48,500	n/e	n/e
TOTAL	27,708	18,867	4,964	66,645	6,773	16,906

n/e = not estimated. Direct PM emissions due to wind erosion on natural surfaces are not estimated because this source is highly sporadic, resulting in emissions too highly uncertain to be included.

[a] VOC emissions have been estimated for compounds known to contribute to ozone formation; the organic compounds that are most important to secondary organic aerosols are a subset of this category, and may include other semi-volatile compounds.

Source: EPA (2001), Tables A-4 through A-10; Guenther et al. (2000).

National estimates of primary $PM_{10-2.5}$ are dominated by fugitive dust and agricultural sources. Fugitive dust sources include paved and unpaved road dust, dust from construction and agricultural activities, and natural sources like geogenic wind erosion (not estimated in Table 2-3). Fugitive dust is also a significant source of primary $PM_{2.5}$. Unlike $PM_{10-2.5}$, where fugitive dust emissions comprise about 75 percent of total emissions, fugitive dust emissions of $PM_{2.5}$ is only about one-third of total emissions. Recent research has found that about 75 percent of these emissions are within 2 meters of the ground when measured. A significant portion of these coarse-mode particles are removed or deposited within a few kilometers of their release point due to turbulence associated with surface topography, and the presence of vegetation or structures (DRI, 2000). This is consistent with the generally small amount of crustal material found in ambient $PM_{2.5}$ samples in most locations. As shown in Table 2-3, direct emissions from fuel combustion, industrial processes, fires, and motor vehicles contribute more to primary $PM_{2.5}$ than to primary $PM_{10-2.5}$.

2.3.2 Secondary PM Precursor Emissions

Major precursors of secondarily formed fine particles include SO_2, nitrogen oxides (NO_x), which encompasses NO and NO_2, and certain organic compounds. Table 2-3 shows the estimated contribution of various sources to nationwide emissions of SO_2, NO_x, VOC, and NH_3. Fuel combustion in the power generation and industrial sectors dominates nationwide estimates of SO_2 emissions and contributes significantly to NO_x emissions. However, emissions from motor vehicles comprise the greatest portion of nationwide NO_x emissions. Motor vehicle emissions also make up a substantial portion of nationwide VOC emissions, with additional contributions from the use of various solvents in industrial processes and commercial products. 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 $PM_{2.5}$ can be nonlinear. Thus, it is difficult to project the impact on ambient $PM_{2.5}$ arising from expected changes in PM precursor emissions without air quality simulation models that incorporate treatment of complex chemical transformation processes and meteorology.

Generally SO_2 emissions reductions lead to reductions in sulfate aerosol, and NO_x emissions reductions lead to reductions in nitrate aerosol. However, 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.

2.4 AMBIENT PM MEASUREMENT METHODS

The methods used to measure ambient PM are important to understanding population exposure to PM, evaluating health risks, and developing and evaluating the effectiveness of risk management strategies. Because PM is not a homogeneous pollutant, measuring and characterizing particles suspended in the atmosphere is a significant challenge.[8] Ambient 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. Measurable indicators of fine particles include $PM_{2.5}$, $PM_{1.0}$, British or black smoke (BS), coefficient of haze (COH), and PM_{10} (in areas dominated by fine particles). Measurable indicators of coarse-mode particles include $PM_{10-2.5}$, $PM_{15-2.5}$, and PM_{10} (in areas dominated by coarse-mode particles).

2.4.1 Particle Mass Measurement Methods

Ambient 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 by difference before and after collection. Examples include the FRM 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.

[8] Refer to EPA 1996a, Chapter 4 and draft CD Chapter 2 for more comprehensive assessments of particle measurement methods. A recent summary of PM measurement methods is also given in Fehsenfeld et al. (2003). Significant improvements and understanding of routine and advanced measurement methods is occurring through EPA's PM Supersites Program (see www.epa.gov/ttn/amtic/supersites.html).

Another widely used 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 to be recorded on a near-continuous basis (i.e., every few minutes).

Other methods that produce near-continuous PM mass measurements include the beta attenuation sampler and the Continuous Ambient Mass Monitor (CAMM). A beta attenuation (or beta gauge) sampler determines the mass of particles deposited on a filter by measuring the absorption of electrons generated by a radioactive isotope, where the absorption is closely related to the mass of the particles. The CAMM measures the pressure drop increase that occurs in relation to particle loading on a membrane filter. Both methods (beta-attenuation and CAMM) require calibration against standard mass measurements as neither measures PM mass directly by gravimetric analysis.

PM has also been characterized in the U.S. and elsewhere by indirect optical methods that rely on the light scattering or absorbing properties of either suspended PM or PM collected on a filter.[9] These include BS, COH, and 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 black carbon, including black carbon (BC), typically make the largest contribution to stain darkness. 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 particles below 2 μm diameter. Since the mix of ambient particles varies

[9] See Section 2.9 of this chapter for a discussion of the optical properties of PM.

widely by location and time of year, the correlation between BS, COH, and nephelometer measurements and PM mass is highly site- and time-specific.

2.4.2 Size-Differentiated Particle Number Concentration Measurement Methods

Recently there has been increasing interest in examining the relationship between the particle number concentration by size and health effects. Several instruments are needed to provide size distribution measurements (number and size) over the 5 orders of magnitude of particle diameters of interest. 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, while widely used in aerosol research, have not yet been widely used in health effects studies.

2.4.3 Chemical Composition Measurement Methods

There are a variety of methods used to identify and describe the characteristic components of ambient PM.[10] 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^+), organic cations (such as acetate), and phosphate (PO_4^{3-}).

There are several methods for separating organic carbon (OC) and black carbon (BC) in ambient filter 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, which uses a different temperature profile than TOR. The two methods yield comparable estimates of total carbon, but give a different split between OC and BC. Monitoring methods capable of separately measuring

[10] The reader is referred to Chapter 2, section 2.2, of the draft CD for a more thorough discussion of sampling and analytical techniques for measuring PM. Methods used in EPA's National PM Speciation Trends Network and other special monitoring programs are summarized in Solomon et al. (2001).

sulfate, nitrate, and carbon particles on a near-continuous basis have been developed recently, but have not yet been widely tested or used.

2.4.4 Measurement Issues

There is no perfect sampler under all conditions, so there are uncertainties between the mass and composition collected and measured by a sampler and the mass and composition of material that exists as suspended PM in ambient air (Fehsenfeld et al., 2003). To date few standard reference materials exist to estimate the accuracy of measured PM mass and chemical composition relative to what is found in air. At best, uncertainty is estimated based on collocated precision and comparability or equivalency to other similar methods, which themselves have unknown uncertainty, or to the FRM, which is defined for regulatory purposes but is not a standard in the classical sense. There are a number of measurement-related issues that can result in positive or negative measurement artifacts which could affect the associations epidemiological researchers find between ambient particles and health effects.

The semivolatile components of PM can create both positive and negative measurement artifacts. Negative artifacts arise from evaporation of the semivolative components of PM during or after collection, which is caused by changes in temperature, relative humidity, or aerosol composition, or due to the pressure drop as collected air moves across the filter. Nitrate losses due to evaporation may represent as much as 10-20% of total $PM_{2.5}$ mass, as shown in southern California studies (CD, p. 2-97). Positive artifacts arise when gas-phase compounds absorb onto or react with filter media or already collected PM, or when particle-bound water is not removed. The chemical interaction of gases being collected with particles already on the filter can also result in negative artifacts. These interactions depend on the compounds contained in collected particles and in the gas phase, and also depend on both location and time.

Particle-bound water can represent a significant fraction of PM mass under conditions where relative humidity is more than 60% (CD, p. 2-46). The amount of particle-bound water will vary with the composition of particles, as discussed in section 2.2.3. The use of heated inlets to remove particle-bound water (e.g. TEOM at 50° C) can result in loss of semi-volatile compounds unless corrective techniques are applied (CD, p. 2-101).

Particle bounce from the filter can result in negative artifacts. This may be more prevalent under lower relative humidity conditions. Impactor coatings can be used to limit particle bounce, but can interfere with mass and chemical composition measurements..

In areas with significant amounts of dust, high wind conditions resulting in blowing dust can interfere with accurate separation of fine- and coarse-fraction particles. In these unique conditions a significant amount of coarse-fraction material can be found in the inter-modal region between 1 and 3 μm. The addition of a $PM_{1.0}$ measurement in these circumstances can provide greater insights into the magnitude of this problem.

2.5 PM CONCENTRATIONS, TRENDS, AND SPATIAL PATTERNS IN THE U.S.

This section provides analysis of the latest available PM air quality data, including PM levels, composition, and spatial patterns. The EPA and the States have been using a national network to measure and collect PM_{10} concentrations since 1987, and $PM_{2.5}$ concentrations since 1999. Summaries through the end of 2002, based on data publically available from EPA's Air Quality System (AQS) as of April 2003, are presented here. $PM_{2.5}$ data from the network for Interagency Monitoring of Protected Visual Environments (IMPROVE) are also presented. Many data summaries are presented by region, as shown in Figure 2-3. These regions were first defined in the 1996 CD, and later revised by researchers in the NMMAPS study, and have proven useful for understanding potential differences in the characteristics of PM in different parts of the U.S..

2.5.1 $PM_{2.5}$

Following the establishment of new standards for $PM_{2.5}$ in 1997, the EPA led a national effort to deploy and operate over 1000 $PM_{2.5}$ monitors. These monitors use the FRM which, when its procedures are followed, assures that PM data are collected using standard equipment,

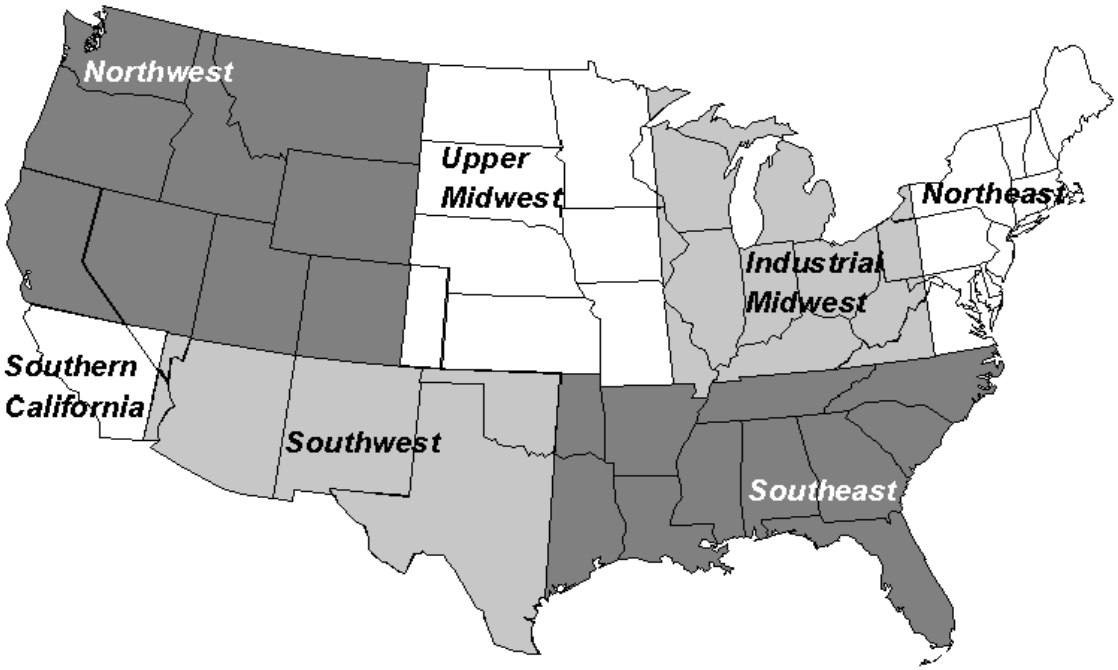

Figure 2-3. Regions used in Staff Paper PM data analysis summaries.

operating procedures, and data handling techniques.[11] Most of these FRM monitors began operation in 1999. The EPA has analyzed the available data collected by this network from 2000-2002. Data from the monitors were screened for completeness with the purpose of avoiding seasonal bias. To be included in these analyses a monitor needed at least a full year of data, defined as either 4, 8, or 12 consecutive quarters with eleven or more observations per quarter. A total of 1142 FRM monitors in the United States met these criteria[12].

The annual $PM_{2.5}$ mean concentrations range from about 2 to 29 $\mu g/m^3$, with a median of about 13 $\mu g/m^3$. The 98th percentiles of the distribution of 24-hour average concentrations range from about 8 to 94 $\mu g/m^3$, with a median of about 33 $\mu g/m^3$. Figures 2-4 and 2-5 depict the

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

[12] 1129 of the 1142 monitors are located in the contiguous continental U.S. covered by the regions shown in Figure 2-3. The remainder are located in Alaska, Hawaii, and U.S. territories.

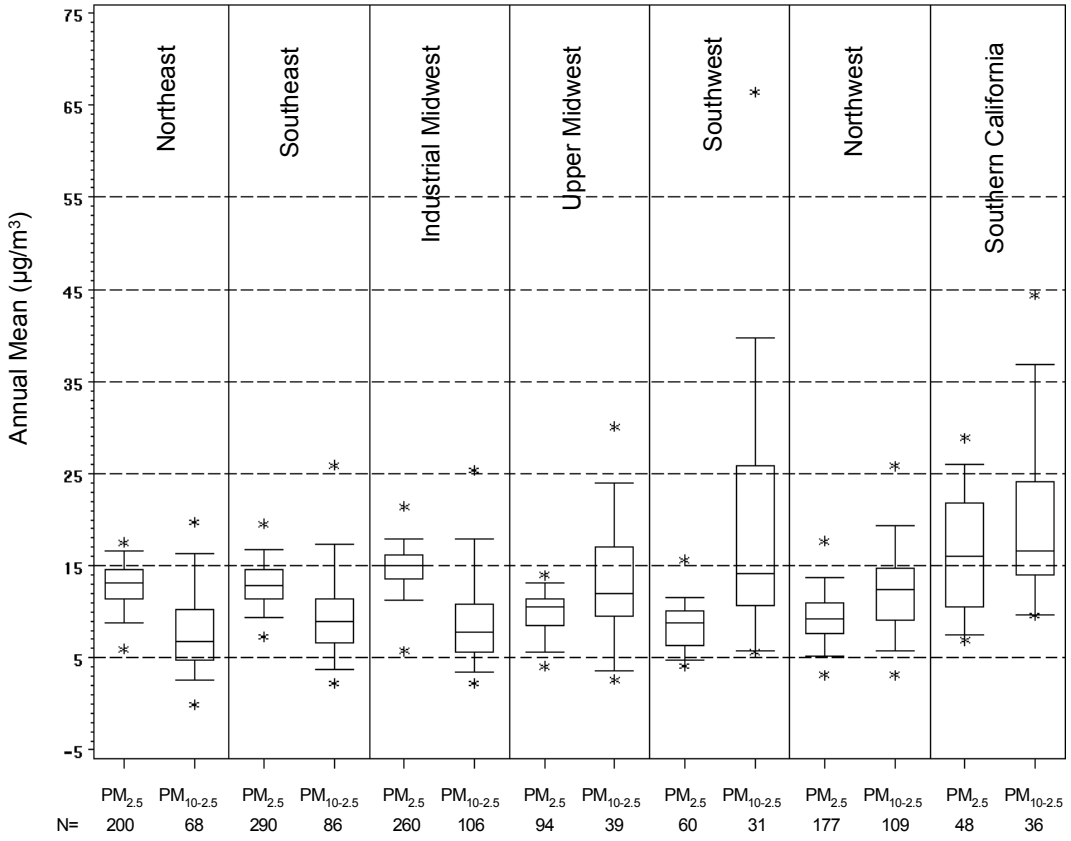

Figure 2-4. Distribution of annual mean $PM_{2.5}$ and estimated annual mean $PM_{10-2.5}$ concentrations by region, 2000-2002. Box depicts interquartile range and median; whiskers depict 5th and 95th percentiles; asterisks depict minimum and maximum. Number below indicates the number of sites in each region.

Source: Schmidt et al. (2003)

regional distribution of site-specific annual mean and 98th percentile 24-hour average $PM_{2.5}$ (and $PM_{10-2.5}$, discussed in section 2.5.3) concentrations, respectively, by geographic region (excluding Alaska, Hawaii, Puerto Rico, and the Virgin Islands). Figures 2-6 and 2-7 are national maps that depict county-level annual mean $PM_{2.5}$ concentrations and 98th percentile 24-hour average

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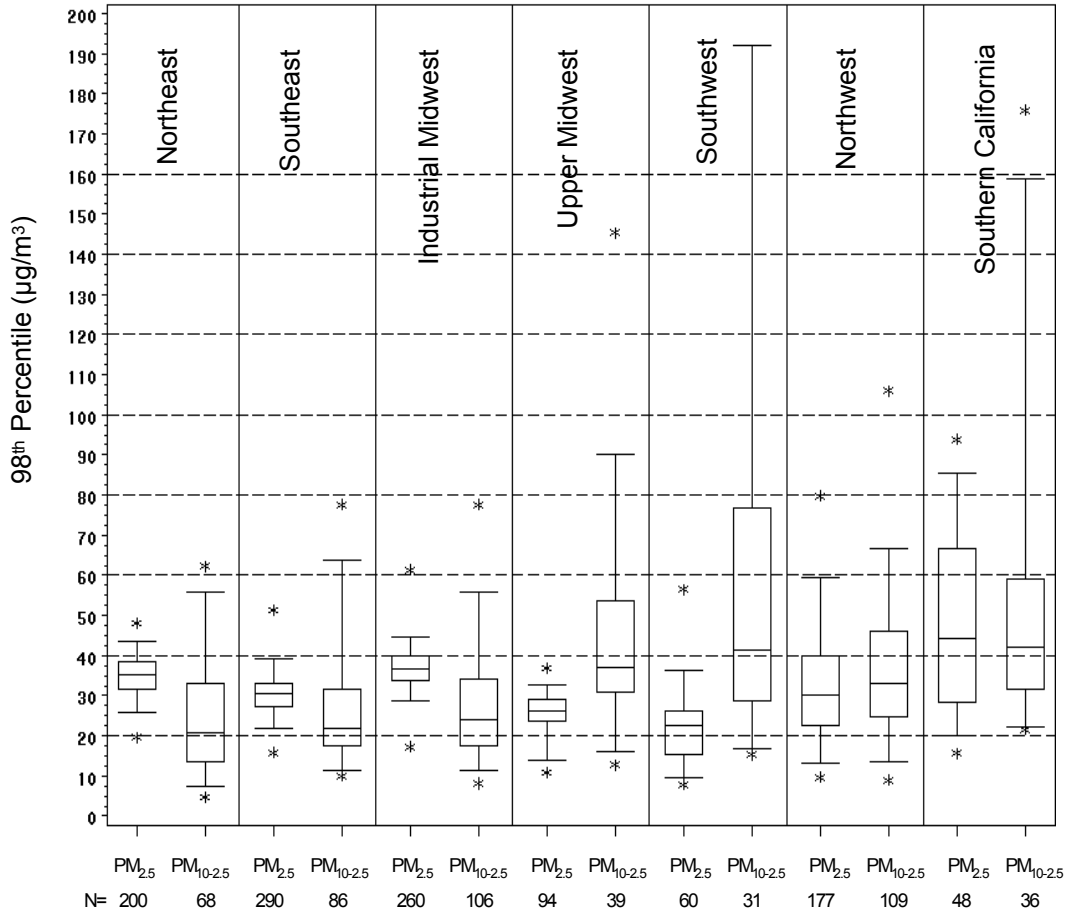


Figure 2-5. Distribution of 98th percentile 24-hour average $PM_{2.5}$ and estimated $PM_{10-2.5}$ concentrations by region, 2000-2002. Box depicts interquartile range and median; whiskers depict 5th and 95th percentiles; asterisks depict minimum and maximum. Number below indicates the number of sites in each region.

Source: Schmidt et al. (2003)

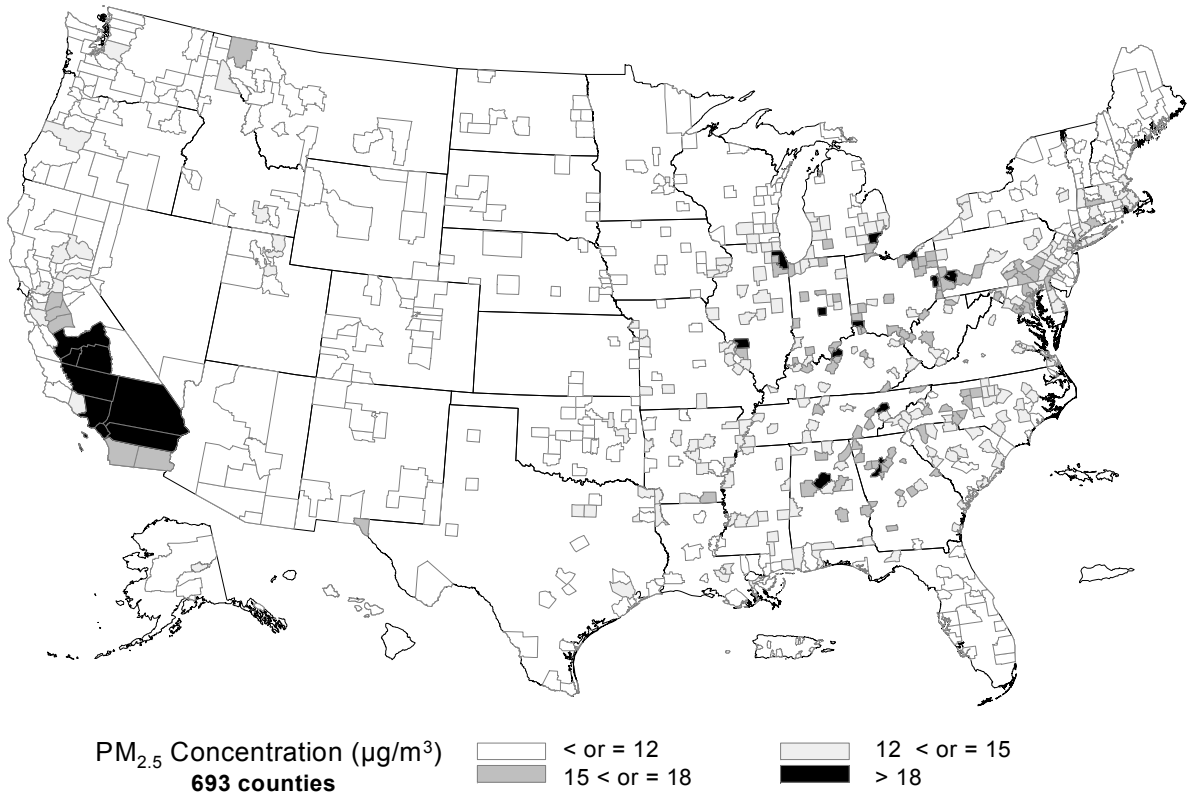


Figure 2-6. County-level maximum annual mean $PM_{2.5}$ concentrations, 2000-2002.

Source: Schmidt et al. (2003)

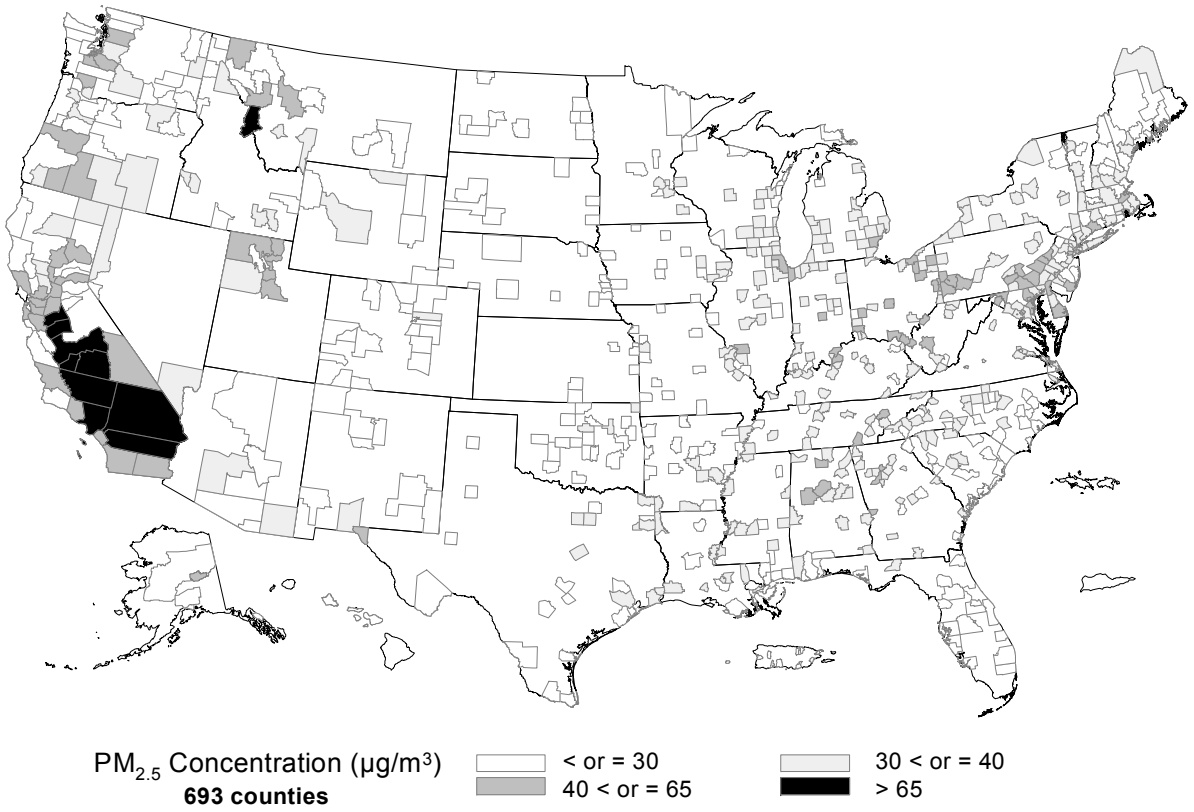


Figure 2-7. County-level maximum 98th percentile 24-hour average $PM_{2.5}$ concentration, 2000-2002.

Source: Schmidt et al. (2003)

concentrations, respectively, from the FRM network.[13] The monitor with the highest concentration in each monitored county is used to represent the value in that county. The map and box plots show that many locations in the eastern U.S. and in California had annual mean $PM_{2.5}$ concentrations above 15 $\mu g/m^3$. Annual mean $PM_{2.5}$ concentrations were above 18 $\mu g/m^3$ in several urban areas throughout the eastern U.S., including Atlanta, Birmingham, Chicago, Cincinnati, Cleveland, Detroit, Indianapolis, Knoxville, Louisville, Pittsburgh, and St. Louis. Los Angeles and the central valley of California also were above 18 $\mu g/m^3$. Sites in the upper midwest, southwest, and northwest regions had generally low annual mean $PM_{2.5}$ concentrations, most below 12 $\mu g/m^3$. The 98th percentile 24-hour average $PM_{2.5}$ concentrations above 65 $\mu g/m^3$ appear only in California and Montana. Values in the 40 to 65 $\mu g/m^3$ range were more common in the eastern U.S. and on the west coast, mostly in or near urban areas, but relatively rare in the upper midwest and southwest regions. In these regions the 98th percentile 24-hour average $PM_{2.5}$ concentrations were more typically below 40 $\mu g/m^3$, with many below 25 $\mu g/m^3$.

The IMPROVE monitoring network, which consists of sites located primarily in national parks and wilderness areas throughout the U.S., provides data for long-term $PM_{2.5}$ trends for generally rural areas.[14] Figure 2-8 shows the composite long-term trend at 9 eastern sites, 23 western sites, and one urban site in Washington, D.C. At the rural eastern sites, measured $PM_{2.5}$ decreased about 16 percent from 1992 to 2001. At the rural western sites $PM_{2.5}$ decreased about 10 percent from 1992 to 2001. At the Washington, D.C. site the annual average $PM_{2.5}$ concentration in 2001 was about 30 percent lower than the 10-year peak in 1994.

[13] Readers should be cautioned not to draw conclusions regarding the potential attainment status of any area from these data summaries. EPA regulations, in 40 CFR Part 50, Appendix N, require 3 consecutive years of monitoring data and specify minimum data completeness requirements for data used to make decisions regarding attainment status. Not all of the PM federal reference method (FRM) monitors that contributed data to the summaries presented here recorded valid PM measurements for all four calendar quarters for each of the 3 years.

[14] Since IMPROVE monitoring instruments and protocols are not identical to FRM monitors, the data are not directly comparable to the FRM monitor data.

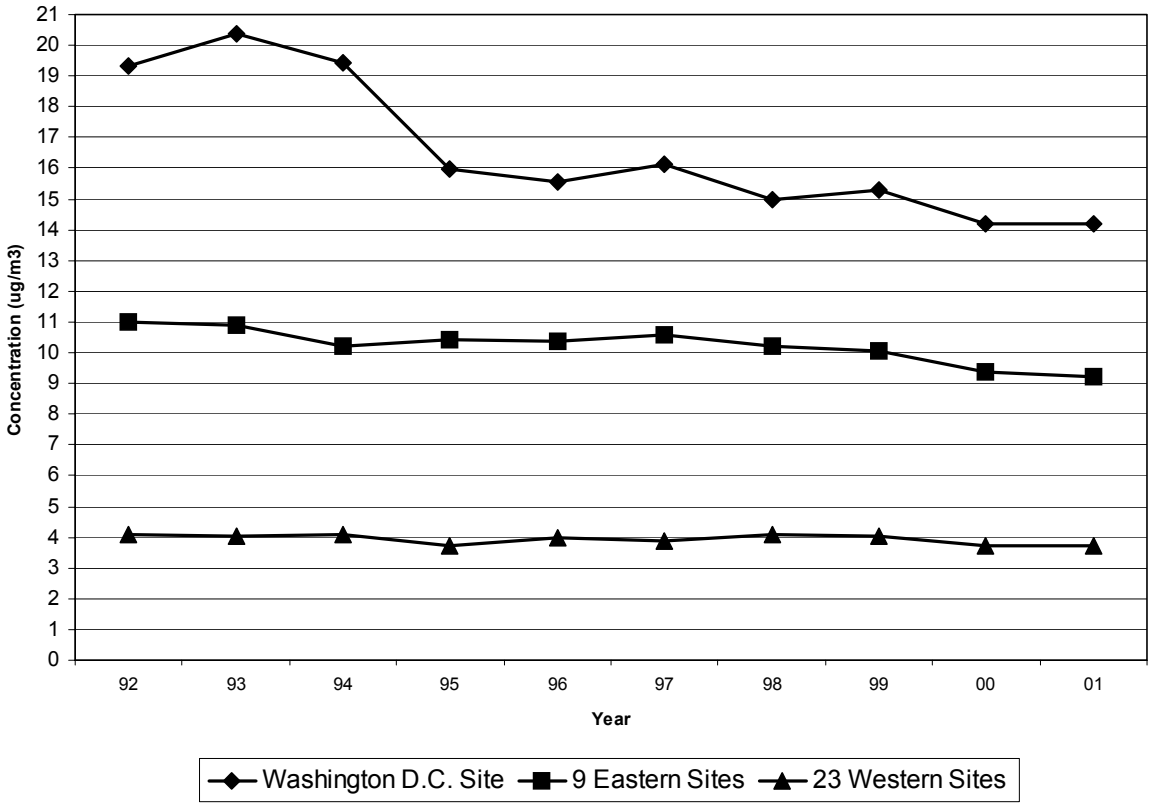


Figure 2-8. Average measured annual mean $PM_{2.5}$ concentration trend at IMPROVE sties, 1992-2001. Included sites must have 8 of 10 valid years of data; missing years are interpolated. Measured mass represents measurement from the filter.

Source: Schmidt et al. (2003)

The relative spatial homogeneity of the ambient air across a specified area can be assessed by examining the values at multiple sites of several indicators, including: (1) site pair correlations, (2) differences in long-term (e.g., annual) average concentrations, and (3) differences in short-term (e.g., daily) average concentrations. An analysis of these indicators for site pairs in 27 urban areas using $PM_{2.5}$ FRM monitoring data from 1999-2001 is included in the draft CD.

An analysis of monitor pairs from each of the 27 urban areas indicates that multiple sites in these areas were highly correlated throughout the period. More than 86 percent (426 out of 491) of the between-site correlation coefficients in all 27 areas were greater than or equal to 0.80, and more than 53 percent (268 out of 491) of the correlations were greater than or equal to

0.90. Further, every area had at least one monitor pair with a correlation coefficient greater than or equal to 0.85 (CD, Appendix 3A).

A summary of the analyses of long-term and short-term concentration differences is shown in Table 2-4. The difference in annual mean $PM_{2.5}$ concentrations between monitor pairs in the 27 cities ranged from less than 1 $\mu g/m^3$ in Baton Rouge to about 8 $\mu g/m^3$ in Pittsburgh. Large differences in annual mean concentrations across a metropolitan area may be due to differences in emissions sources, meteorological, or topography. Small differences may be due only to measurement imprecision (CD, p. 3-46). In urban areas, the site pair with the maximum and minimum annual mean concentration was highly correlated ($r_{(max,min)} \geq 0.70$); the most notable exception was the site pair in Gary, IN ($r_{(max,min)}=0.56$).

The analysis in the draft CD also examined differences in short-term 24-hour average concentrations between the urban site pairs. Small differences throughout the distribution would indicate relatively homogeneous concentration levels between the sites. Table 2-4 presents a summary of the 90th percentile of the distribution (P_{90}) of daily site pair differences in each urban area. The site pair with the largest difference (max pair) and the smallest difference (min pair) are shown. The P_{90} values for the 491 monitor pairs in the 27 urban areas ranged from about 2 to 21 $\mu g/m^3$. Often the site pair with the maximum P_{90} value in each city was also the pair with the largest annual mean difference. The site pair with the highest P_{90} values in each city were generally highly correlated ($r_{max} \geq 0.70$), and in some cases were more highly correlated than the sites with the largest annual mean differences.

Table 2-4. Summary of $PM_{2.5}$ FRM Data Analysis in 27 Metropolitan Areas, 1999-2001.

City	N Sites	Annual Mean (µg/m³)				P_{90} (µg/m³)		
		Max Site	Min Site	% Diff	$r_{(max,min)}$	Max Pair	Min Pair	r_{max}
Pittsburgh, PA	11	22.0	13.8	37%	0.69	21.0	4.2	0.69
Salt Lake City, UT	6	13.6	8.8	35%	0.86	11.4	4.4	0.86
Detroit, MI	10	19.9	13.5	32%	0.89	13.8	5.0	0.84
Cleveland, OH	8	20.2	14.0	31%	0.84	14.3	3.3	0.84
St. Louis, MO	11	20.2	13.9	31%	0.69	15.2	2.8	0.69
Portland, OR	4	9.1	6.3	31%	0.79	6.5	4.1	0.79
Chicago, IL	11	20.6	14.5	30%	0.91	11.3	3.5	0.92
Seattle, WA	4 *	11.9	8.9	25%	0.91	8.5	3.6	0.75
Birmingham, AL	5	21.6	16.6	23%	0.80	15.2	6.6	0.80
Los Angeles, CA	6	23.7	18.3	23%	0.76	18.2	6.2	0.66
Gary, IN	4	17.6	14.0	20%	0.56	11.3	4.2	0.59
Washington, DC	5 *	16.7	13.8	17%	0.84	7.7	3.5	0.84
Kansas City, MO	6	13.8	11.4	17%	0.87	6.5	1.9	0.90
Riverside, CA	5	30.0	25.0	17%	0.93	17.8	3.6	0.81
Dallas, TX	7	13.7	11.5	16%	0.89	6.3	1.9	0.89
Boise, ID	4	10.3	8.7	16%	0.79	8.8	3.8	0.79
Atlanta, GA	6 *	21.2	18.3	14%	0.81	10.8	5.3	0.75
Grand Rapids, MI	4	14.0	12.1	14%	0.93	6.1	3.1	0.93
San Diego, CA	4	17.0	14.6	14%	0.73	11.0	6.3	0.73
Tampa, FL	4	12.7	11.1	13%	0.87	5.0	3.1	0.71
Steubenville, OH	5	18.9	16.5	13%	0.86	10.0	6.2	0.79
Philadelphia, PA	7	16.0	14.1	12%	0.85	7.5	3.3	0.84
Louisville, KY	4	17.4	15.7	10%	0.86	6.0	3.8	0.90
Milwaukee, WI	8	14.4	13.1	9%	0.89	5.3	2.8	0.89
Norfolk, VA	5	13.7	12.6	8%	0.96	5.0	2.6	0.91
Columbia, SC	3	15.7	14.7	6%	0.93	3.3	2.8	0.93
Baton Rouge, LA	3	14.5	14.1	3%	0.97	2.9	2.5	0.93

* Does not include 1 additional site >100 km from the others in the urban area.
P_{90} = 90th percentile of the distribution of differences in 24-hour averages between two sites in the same urban area.
$r_{(max,min)}$ = correlation between intra-urban sites with the largest difference in annual mean concentrations.
$r_{(max)}$ = correlation between intra-urban sites with the largest difference in P_{90} values.

Source: CD, Appendix 3A

2.5.2 PM_{10}

For the purpose of comparison to $PM_{2.5}$ and $PM_{10-2.5}$ concentrations, PM_{10} data from 2000-2002 are presented in Figures 2-9 and 2-10. Figure 2-9 shows the PM_{10} annual mean concentrations, and Figure 2-10 shows the 98th percentile 24-hour average concentrations.[15] As in the earlier $PM_{2.5}$ maps, the monitor with the highest value in each monitored county is used to represent the value in each county. Most areas of the country had concentrations below the level of the annual mean PM_{10} standard of 50 $\mu g/m^3$. Exceptions include several locations in the southwest and in central California. Most areas of the country also had concentrations below the level of the 24-hour standard of 150 $\mu g/m^3$, with exceptions mostly in the western U.S.[16]

In the National Air Quality and Emissions Trends Report (EPA, 2001b), EPA examined national and regional PM_{10} trends for the 10-year period from 1992 to 2001. The EPA found a national average decline in concentrations of approximately 14 percent over the 10 year period, with regional average declines across the eastern U.S. ranging from 7 to 28 percent and declines across the western U.S. ranging from 10 to 31 percent.

2.5.3 $PM_{10-2.5}$

$PM_{10-2.5}$ is a measure of the coarse-mode fraction of PM_{10} being considered in this review. It can be measured by a dichotomous sampler, or by using a difference method with collocated PM_{10} and $PM_{2.5}$ monitors employing the same sampling protocol. A nationwide network of samplers with the specific intent to consistently and accurately measure $PM_{10-2.5}$ does not currently exist. The EPA is currently evaluating a variety of monitoring platforms to establish a FRM for $PM_{10-2.5}$, which would be used in the future to design a national network of monitors to measure coarse-fraction particles. Until such a network is established, estimates of $PM_{10-2.5}$ can be generated for a limited number of locations using a difference method on same-day data

[15] These figures do not depict officially designated PM_{10} nonattainment areas. As of June 23, 2003, there were a total of 64 areas classified as moderate or serious nonattainment areas, mostly in the western U.S. See designated nonattainment areas at www.epa.gov/oar/oaqps/greenbk/pnc.html.

[16] The form of the 1987 PM_{10} standard is based on the annual 2nd highest daily concentration rather than the 98th percentile concentration shown in Figure 2-12. The annual 98th percentile concentration is presented here for consistency with the depictions of $PM_{2.5}$ and $PM_{10-2.5}$ concentrations.

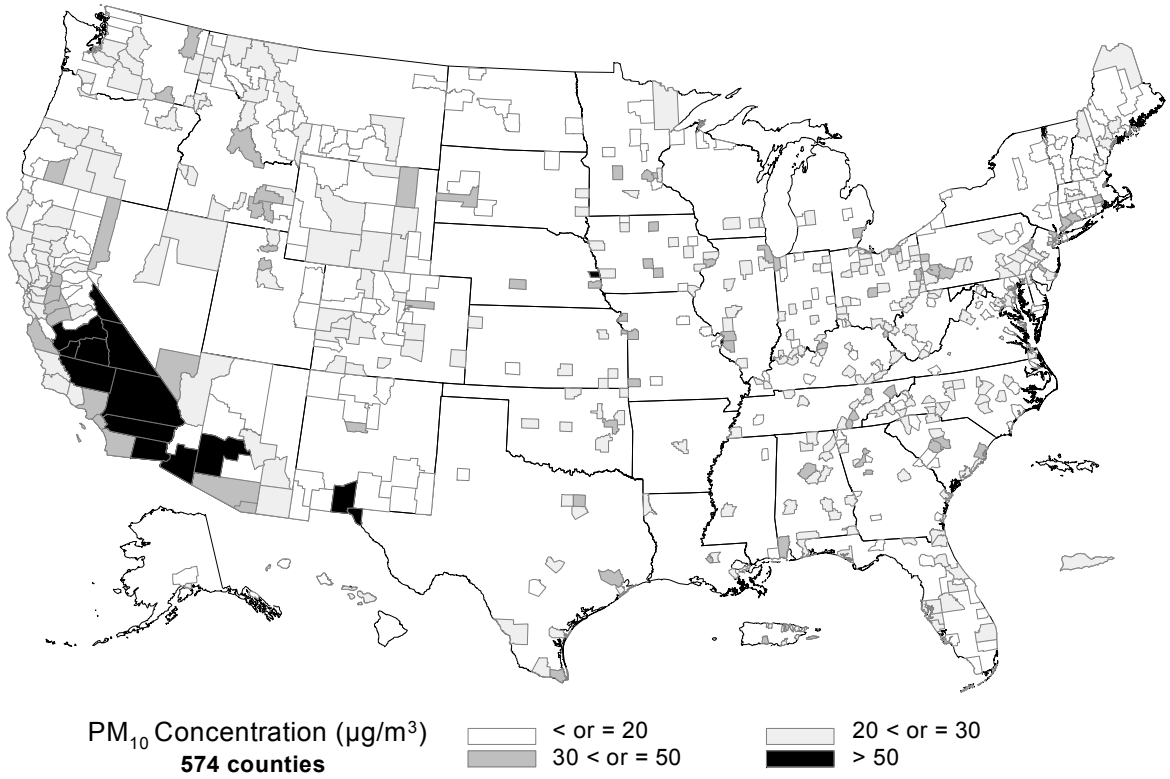


Figure 2-9. County-level maximum annual mean PM_{10} concentrations, 2000-2002.

Source: Schmidt et al. (2003)

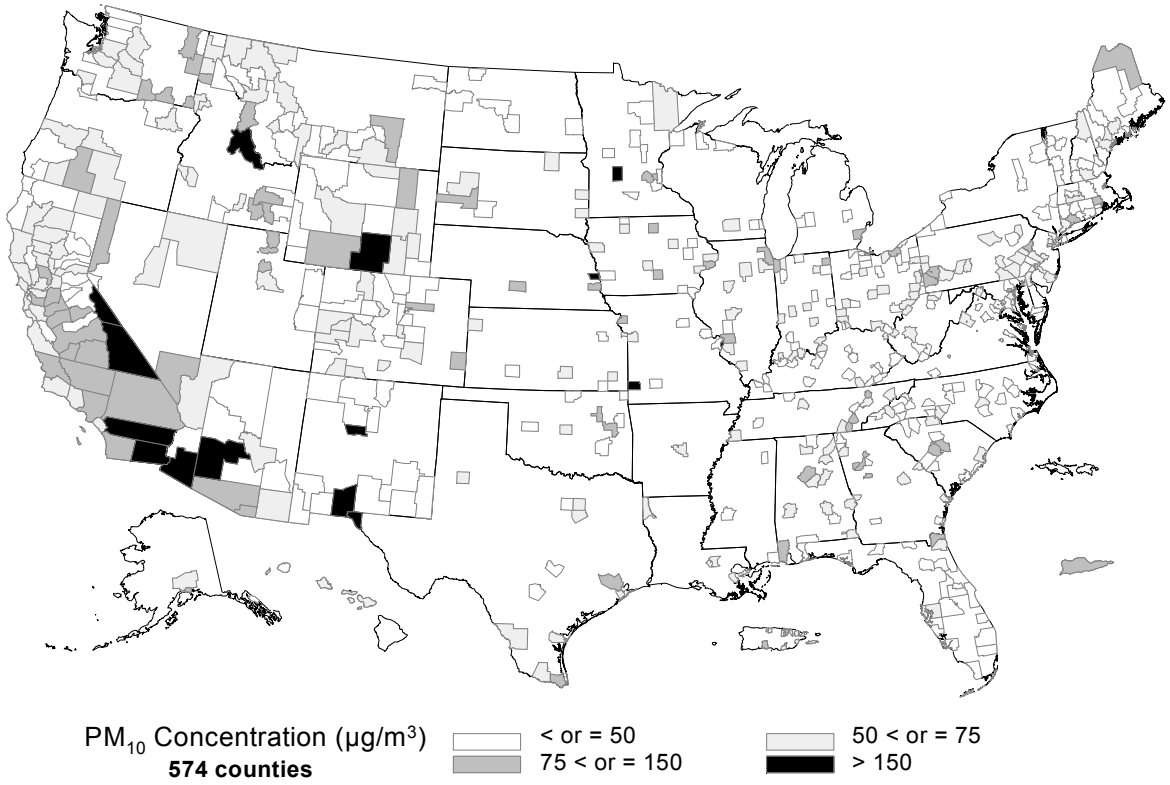


Figure 2-10. County-level maximum 98th percentile 24-hour average PM_{10} concentrations, 2000-2002.

Source: Schmidt et al. (2003)

collected from co-located PM_{10} and $PM_{2.5}$ FRM monitors. Since the protocol for each monitor is not usually identical, the consistency of these $PM_{10-2.5}$ measurements are relatively uncertain, and are referred to as "estimates" in this draft Staff Paper.

The 98th percentiles of the distribution of estimated 24-hour average $PM_{10-2.5}$ concentrations range from about 5 to 315 $\mu g/m^3$, with a median of about 29 $\mu g/m^3$. The box plots in Figures 2-4 and 2-5 (introduced above in section 2.5.1 on $PM_{2.5}$) depict the regional distribution of site-specific estimated annual mean and 98th percentile 24-hour average $PM_{10-2.5}$ concentrations, respectively, by geographic region (excluding Alaska, Hawaii, Puerto Rico, and the Virgin Islands). Figures 2-11 and 2-12 are national maps that depict estimated county-level annual mean $PM_{10-2.5}$ concentrations, and 98th percentile 24-hour average concentrations, respectively. To construct the maps, the site with the highest concentration in each monitored county is used to represent the value in that county. The annual mean $PM_{10-2.5}$ concentrations are generally estimated to be below 45 $\mu g/m^3$, with one maximum value as high as 66 $\mu g/m^3$ (see Figure 2-4), and with a median of about 10 $\mu g/m^3$. Compared to annual mean $PM_{2.5}$ concentrations, annual mean $PM_{10-2.5}$ estimates are more variable, with more distinct regional differences. As shown in Figure 2-4, eastern U.S. estimated annual mean $PM_{10-2.5}$ levels tend to be lower than annual mean $PM_{2.5}$ levels, and in the western U.S. estimated $PM_{10-2.5}$ levels tend to be higher than $PM_{2.5}$ levels. The highest estimated annual mean $PM_{10-2.5}$ concentrations appear in the southwest region and southern California. The estimated 98th percentile 24-hour average $PM_{10-2.5}$ concentrations are generally highest in the southwest, southern California, and upper midwest, where a few sites have estimated concentrations well above 100 $\mu g/m^3$ (see Figure 2-5).

The IMPROVE monitoring network provides data for long-term $PM_{10-2.5}$ trends for generally rural areas. Figure 2-13 presents the composite long-term trend at 9 eastern sites, 23 western sites, and one urban site in Washington D.C. At the rural eastern sites, measured $PM_{10-2.5}$ in 2001 was about 34 percent lower than the 10-year peak in 1994. At the rural western sites, measured $PM_{10-2.5}$ was about 19 percent lower in 2001 than the 10-year peak in 1994. At the Washington, D.C. site the annual average $PM_{10-2.5}$ concentration in 2001 was about 30 percent lower than the 10-year peak in 1994, but nearly 1 $\mu g/m^3$ higher than the 1998 low point.

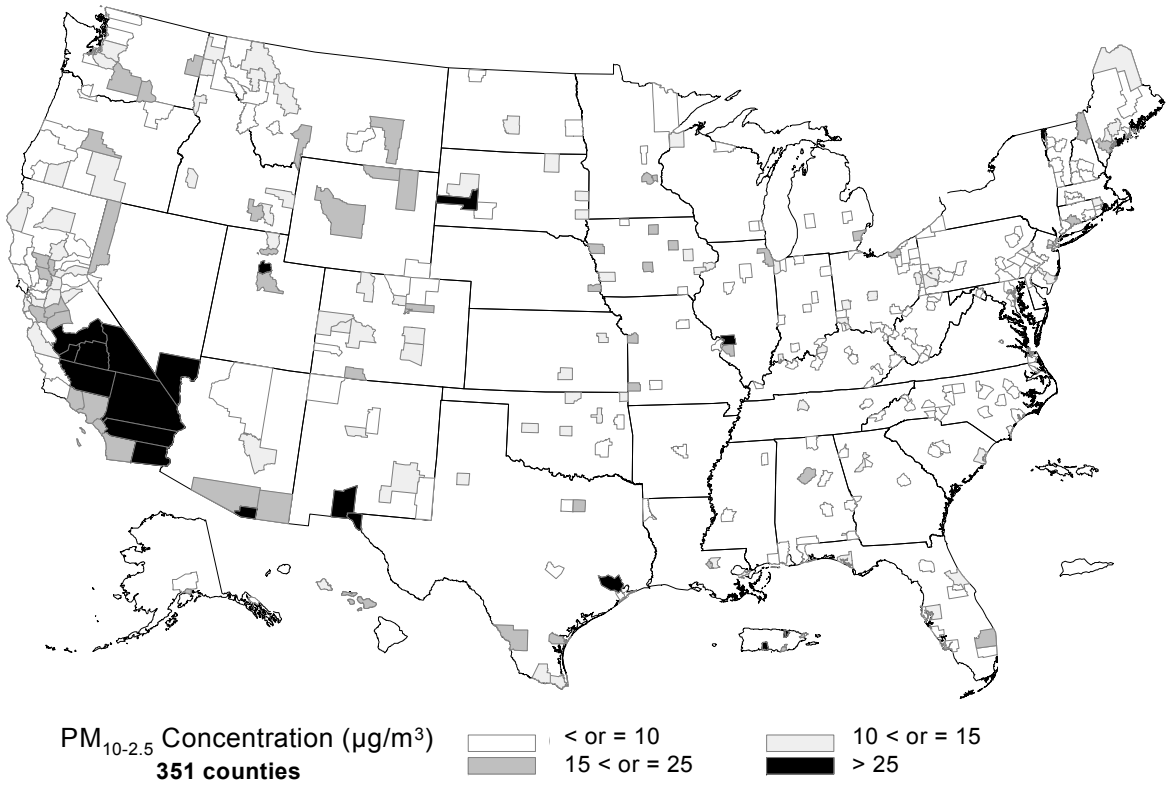


Figure 2-11. Estimated county-level maximum annual mean $PM_{10-2.5}$ concentrations, 2000-2002.

Source: Schmidt et al. (2003)

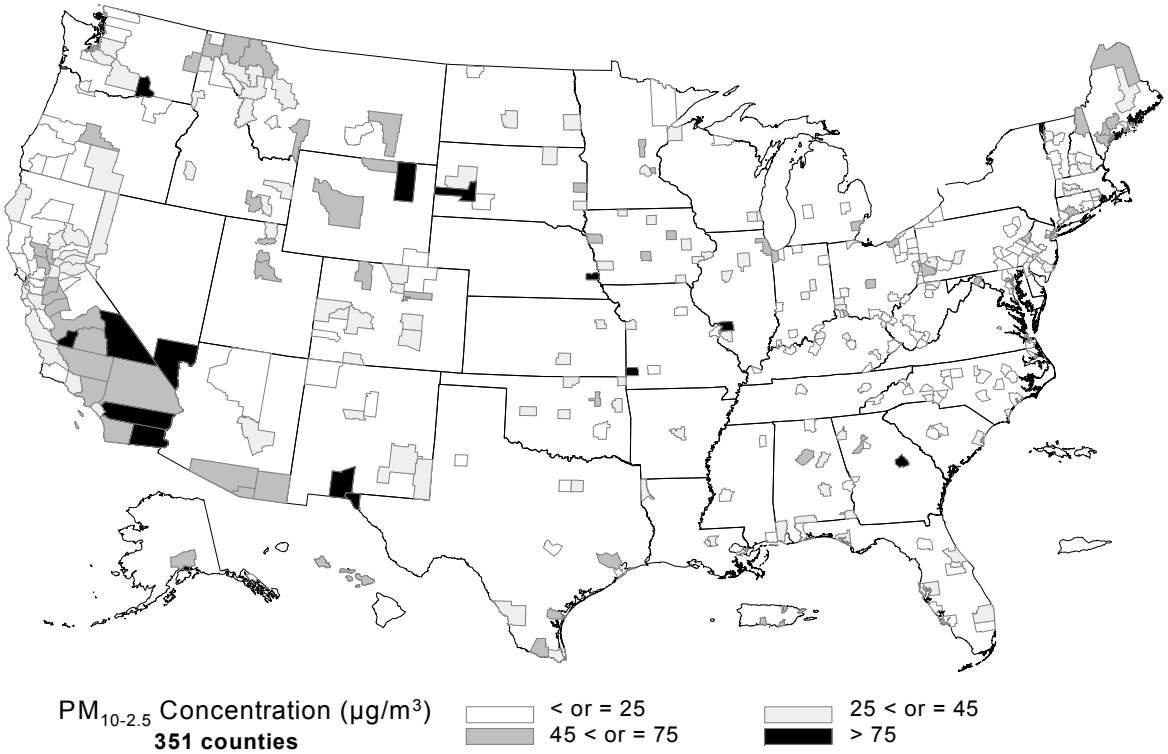


Figure 2-12. Estimated county-level maximum 98th percentile 24-hour average $PM_{10-2.5}$ concentrations, 2000-2002.

Source: Schmidt et al. (2003)

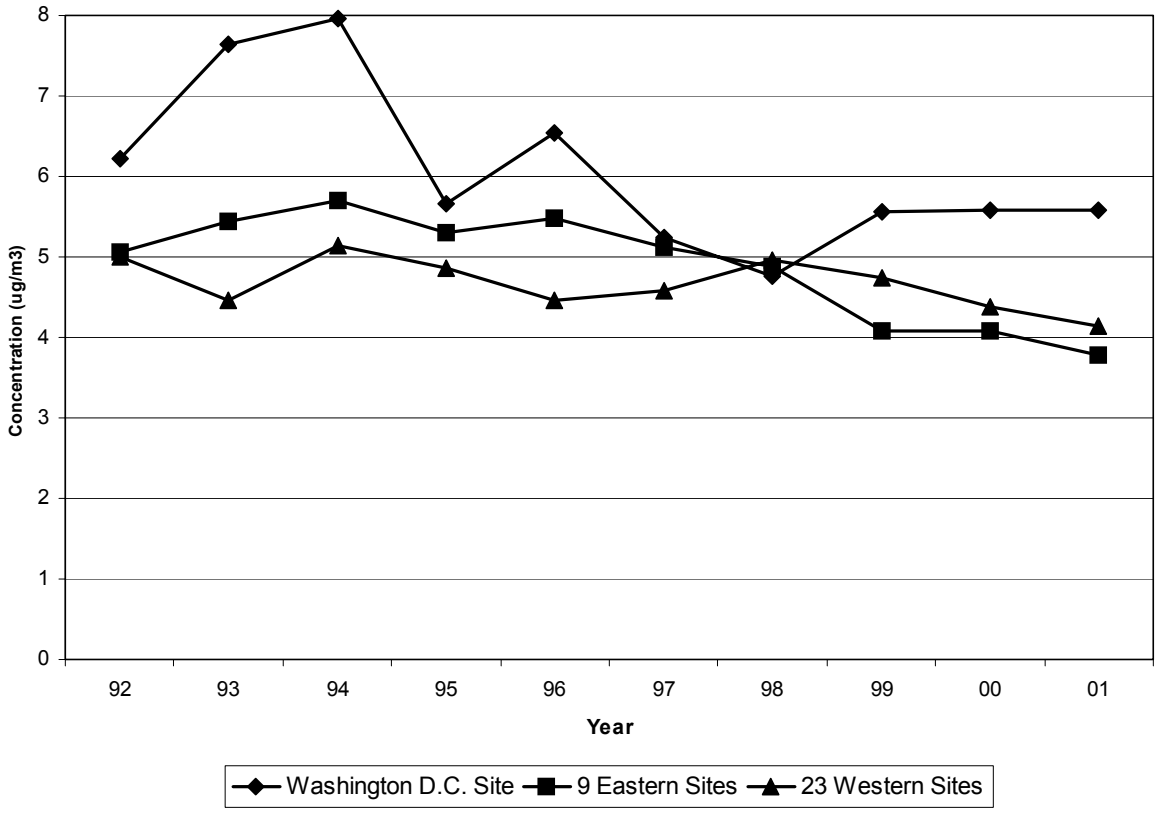


Figure 2-13. Average measured annual average $PM_{10-2.5}$ concentration trend at IMPROVE sties, 1992-2001. Included sites must have 8 of 10 valid years of data; missing years are interpolated. Measured mass represents the measurement from the filter.

Source: Schmidt et al. (2003)

The draft CD contains an analysis of 1999-2001 $PM_{10-2.5}$ estimates in 17 urban areas that is useful for assessing the spatial homogeneity of $PM_{10-2.5}$ across the urban areas (CD, Appendix 3A). This analysis is similar to the 27-city analysis for $PM_{2.5}$ discussed in section 2.5.1 and summarized earlier in Table 2-4. However, since there were fewer site pairings, and fewer urban areas covered, the $PM_{10-2.5}$ results are not as robust as the $PM_{2.5}$ results. The $PM_{10-2.5}$ analysis is summarized in Table 2-5. The analysis reveals generally lower correlations for $PM_{10-2.5}$ compared to the $PM_{2.5}$ correlations in the same city. Of the 65 monitor pairs analyzed, only 4 had correlation coefficients greater than or equal to 0.80, in contrast to more than 86% (426 of 491) of the pairs for $PM_{2.5}$.

Table 2-5. Summary of Estimated $PM_{10-2.5}$ Analysis in 17 Metropolitan Areas, 1999-2001.

City	N Sites	Annual Mean ($\mu g/m^3$)				P_{90} ($\mu g/m^3$)		
		Max Site	Min Site	% Diff	$r_{(max,min)}$	Max Pair	Min Pair	r_{max}
Cleveland, OH	6	26.4	7.2	73%	0.41	40.0	10.6	0.41
Detroit, MI	3	19.4	7.3	62%	0.39	34.9	15.7	0.39
Salt Lake City, UT	3	27.5	14.8	46%	0.72	28.7	9.8	0.72
St. Louis, MO	3	22.5	12.1	46%	0.70	27.2	13.0	0.70
Riverside, CA	4	46.2	25.5	45%	0.32	42.6	13.3	0.36
Dallas, TX	4	19.1	11.2	41%	0.66	16.5	4.5	0.66
San Diego, CA	4	19.4	11.6	40%	0.65	14.7	8.3	0.63
Baton Rouge, LA	2	19.1	12.8	33%	0.40	22.4	22.4	0.40
Los Angeles, CA*	4	24.1	16.1	33%	0.58	17.3	15.5	0.58
Steubenville, OH	4	14.3	10.2	29%	0.54	18.5	10.9	0.48
Gary, IN	3	5.1	3.9	24%	0.79	8.0	6.3	0.60
Columbia, SC	2	9.6	7.4	23%	0.70	8.0	8.0	0.70
Chicago, IL	3	16.1	12.8	20%	0.53	24.6	11.1	0.53
Louisville, KY	2	9.1	7.6	16%	0.65	5.5	5.5	0.65
Portland, OR	2	6.7	5.7	15%	0.69	5.1	5.1	0.69
Milwaukee, WI	2	9.1	7.9	13%	0.65	9.2	9.2	0.65
Tampa, FL	2	11.3	10.1	11%	0.81	5.3	5.3	0.81

* Does not include 1 additional site >100 km from the others in the urban area.
P_{90} = 90th percentile of the distribution of differences in 24-hour averages between two sites in the same urban area.
$r_{(max,min)}$ = correlation between intra-urban sites with the largest difference in annual mean concentrations.
$r_{(max)}$ = correlation between intra-urban sites with the largest difference in P_{90} values.

Source: CD, Appendix 3A

The difference in estimated annual mean $PM_{10-2.5}$ between site pairs in the 17 cities also covered a greater range than was seen for $PM_{2.5}$, with differences up to about 21 $\mu g/m^3$ in Riverside, CA. Similarly, the P_{90} values (described in section 2.5.1) for the 65 site pairs ranged from about 5 $\mu g/m^3$ to about 43 $\mu g/m^3$, which is wider than the range of about 2 $\mu g/m^3$ to 21 $\mu g/m^3$ observed for $PM_{2.5}$.

These indicators provide evidence that $PM_{10-2.5}$ is more heterogenous than $PM_{2.5}$ in some locations (e.g., Cleveland, Detroit, Steubenville), and may be similar in other locations (e.g., Portland, Tampa, St. Louis). Any conclusions should be tempered by the inherent uncertainty in the $PM_{10-2.5}$ estimation method (discussed at the beginning of this section), and the relatively small sample size for $PM_{10-2.5}$ relative to $PM_{2.5}$.

2.5.4 Ultrafine Particles

There are no nationwide monitoring networks for ultrafine particles (i.e., those with diameters < 0.1 μm), and only a few recently published studies of ultrafine particle counts in the U.S. At an urban site in Atlanta, GA, particles in three size classes were measured on a continuous basis between August 1998 and August 1999 (Woo et al., 2001). 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. In Atlanta, the vast majority (89 percent) of the number of particles were in the ultrafine mode (smaller than 0.1 μm), but 83 percent of the particle volume was in the subset of accumulation-mode particles. The researchers found that for particles with diameters up to 2 μm, there was little evidence of any correlation between number concentration and either volume or surface area. Kim et al. (2002) confirmed similarly poor correlations between $PM_{2.5}$ mass and number of ultrafine particles for sites in Los Angeles and nearby Riverside, CA. This suggests that $PM_{2.5}$ cannot be used as a surrogate for ultrafine mass or number, so ultrafine particles need to be measured independently.

Studies of near-roadway particle number and size distributions have shown sharp gradients in ultrafine concentrations around Los Angeles roadways (Zhu et al. 2002a,b). Ultrafine PM concentrations were found to decrease exponentially with distance from the roadway source, and was equal to the upwind "background" location at 300 m downwind.

2.5.5 Components of PM

Atmospheric PM is comprised of many different chemical components that vary by location, time of day, and time of year. Further, as discussed in section 2.3, fine- and coarse-fraction particles have fundamentally different sources and composition. Recent data from the rural IMPROVE network and from the urban speciation network provide indications of regional composition differences for fine-fraction particles. Figure 2-14 shows recent annual average $PM_{2.5}$ mass apportionment among chemical components at several sites in 9 different regions.[17] In general, fine-fraction particles in the eastern U.S. regions are dominated by ammonium sulfate, and carbon compounds. In the western U.S. regions fine-fraction particles have a greater proportion of carbon compounds. With the exception of rural locations in the desert west region, crustal material is a very small portion of fine-fraction particles. The ammonium nitrate component is more prevalent in urban aerosols than in rural aerosols, especially in the California region, and may be an indication of population-driven NO_x sources, such as transportation activity and urban combustion sources.

Trends in rural area and Washington D.C. concentrations of fine-fraction particle components (with the exception of ammonium nitrate) based on data from the IMPROVE network are shown in Figure 2-15. Annual average ammonium sulfate has declined at the urban Washington D.C. site and the rural eastern sites. Both total carbon and crustal material at the Washington D.C. site dropped significantly in 1995, but have not shown significant improvements since then. All other components in all areas have shown small changes over the 10-year period.

[17] Figure 2-14 identifies ammonium as a separate component of $PM_{2.5}$ mass, however, it is associated with either sulfate or nitrate (as ammonium sulfate or ammonium nitrate) roughly in proportion to the amount of sulfate and nitrate indicated.

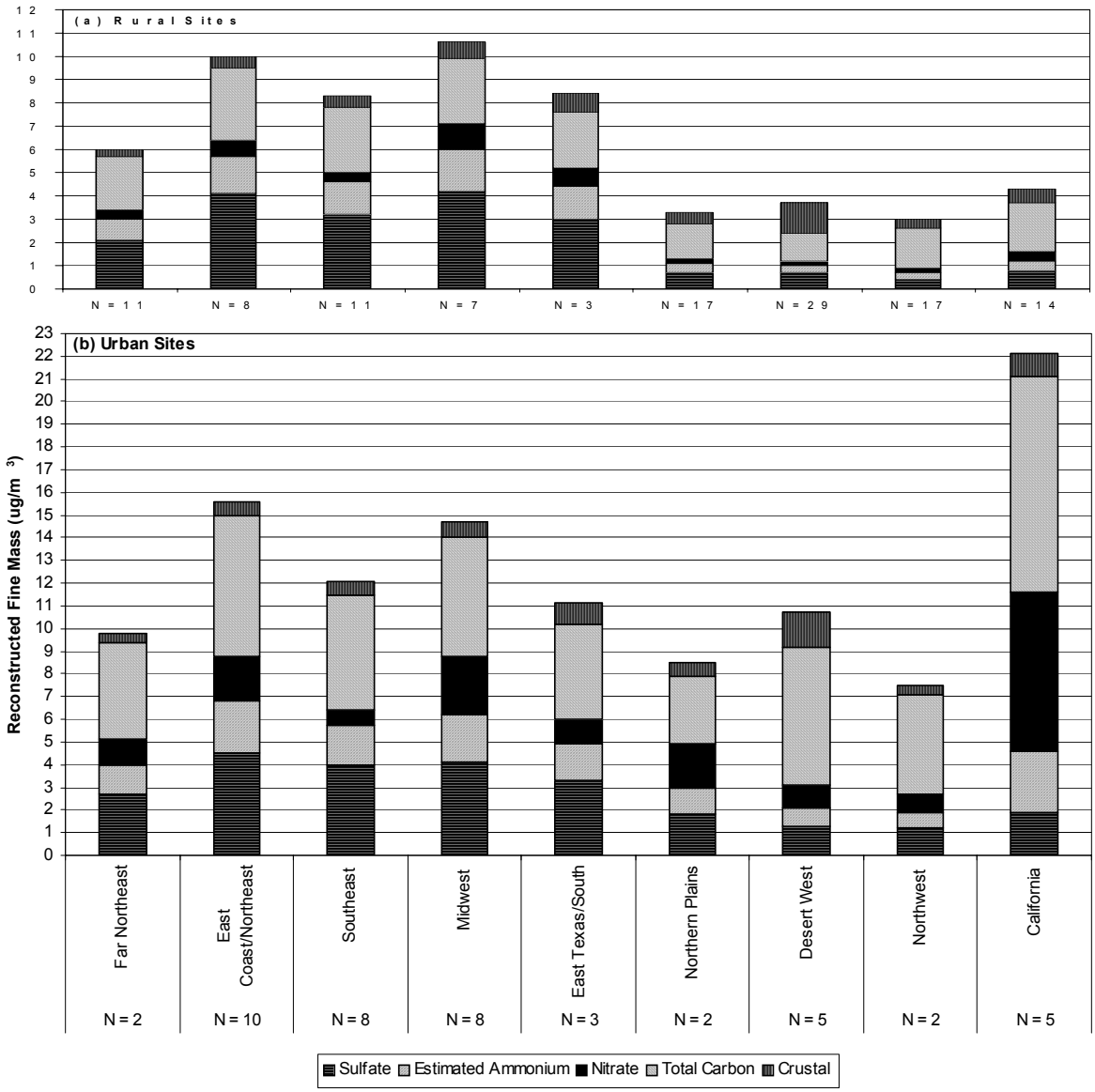


Figure 2-14. Annual average composition of $PM_{2.5}$ by region. Rural data from IMPROVE network, urban data from EPA Speciation Trends Network, September 2001 - August 2002. (components are displayed in the same order as the legend from bottom to top in the stacked bars)

Source: Schmidt et al. (2003)

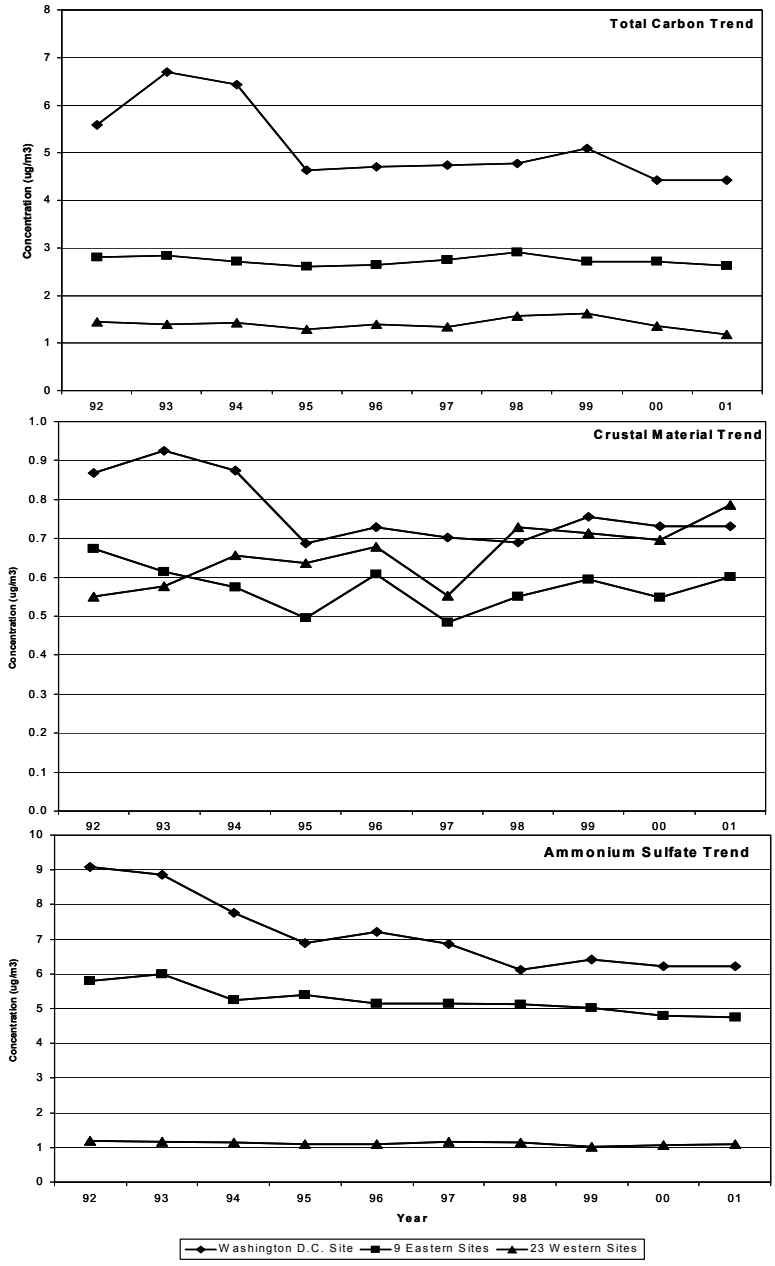


Figure 2-15. Average annual average trend in fine particle ammonium sulfate, total carbon, and crustal material at IMPROVE sites, 1992-2001. Included sites must have 8 of 10 valid years of data; missing years are interpolated. Ammonium nitrate trend is not shown because the methodology changed in 1996.

Source: Schmidt et al. (2003)

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

In this section, information on the relationships among PM indicators in different regions is presented based on data from the nationwide PM FRM monitoring networks. Figure 2-16 shows the distribution of ratios of annual mean $PM_{2.5}$ to PM_{10} at sites in different geographic regions for 2000-2002. The ratios are highest in the eastern U.S. regions with median ratios between 0.6 and 0.7, and lowest in the Southwest region, with a median ratio less than 0.4. These data are generally consistent with earlier findings reported in the 1996 CD from a more limited set of sites. Ratios greater than one are an artifact of the imprecision in the independent PM_{10} and $PM_{2.5}$ measurement methods.

Correlations among pollutant indicators can provide insights into how well one indicator can represent the variability in another indicator. Many earlier epidemiological studies use PM_{10} as an indicator of fine particles. Figure 2-17 shows the results of a nationwide analysis of correlations among PM size fractions using 24-hour average data from the FRM monitoring networks for 2000-2002. $PM_{2.5}$ and PM_{10} measured on the same days at collocated monitors are fairly well correlated, on average, in the eastern regions, and not as well correlated, on average, in the upper midwest and southwest regions. PM_{10} is fairly well correlated with estimated $PM_{10-2.5}$ in most regions, with the highest average correlation in the upper midwest and southwest regions. PM_{10} is more highly correlated, on average, with $PM_{2.5}$ than with estimated $PM_{10-2.5}$ in the northeast and industrial midwest regions. Their correlations are similar in the southeast, and PM_{10} is more highly correlated, on average, with $PM_{10-2.5}$ in the northwest and southern California regions. These data suggest that PM_{10} might be a suitable indicator for either fine-fraction or coarse-fraction particles, depending upon the locational factors. However, in all locations estimated $PM_{10-2.5}$ and $PM_{2.5}$ are very poorly correlated, which should be expected due to their differences in origin, composition, and atmospheric behavior.

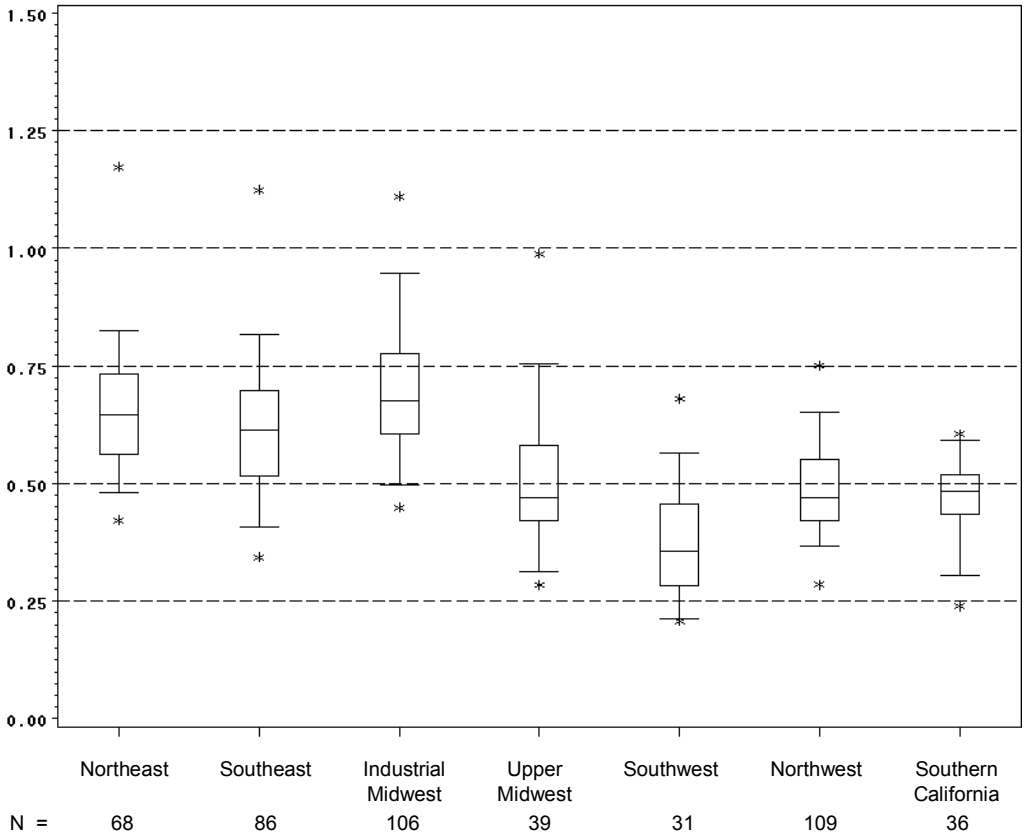


Figure 2-16. Distribution of ratios of annual mean $PM_{2.5}$ to PM_{10} by region, 2000-2002. Box depicts interquartile range and median; whiskers depict 5th and 95th percentiles; asterisks depict minimum and maximum. Number below indicates the number of sites in each region.

Source: Schmidt et al. (2003)

2.6 TEMPORAL PATTERNS IN PM CONCENTRATIONS

2.6.1 $PM_{2.5}$ and $PM_{10-2.5}$ Patterns

Data from the PM FRM network from 2000-2002 show distinct seasonal variations in $PM_{2.5}$ and estimated $PM_{10-2.5}$ concentrations. Figure 2-18 shows the monthly distribution of 24-hour average urban $PM_{2.5}$ concentrations in different geographic regions. The months with peak urban $PM_{2.5}$ concentrations vary by region. The urban areas in the northeast, industrial midwest,

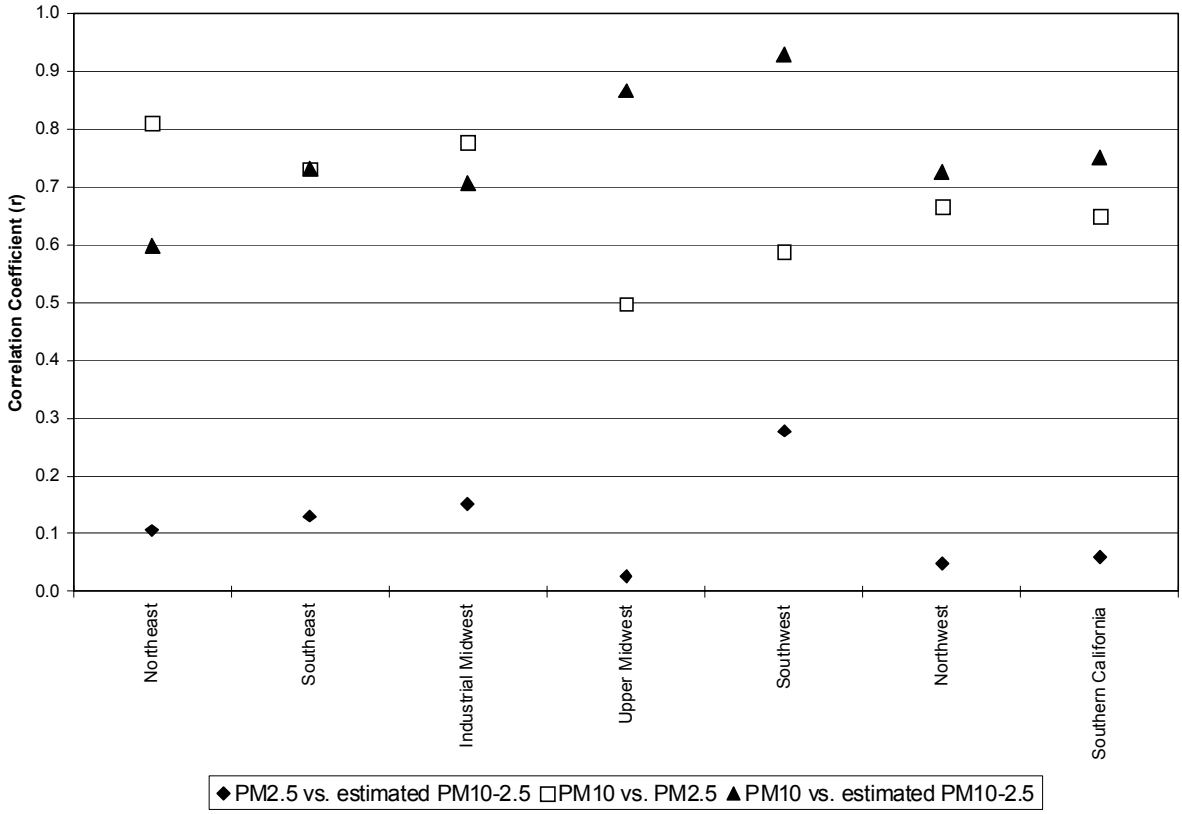


Figure 2-17. Regional average correlations of 24-hour average PM by size fraction.

Source: Schmidt et al. (2003)

and upper midwest regions all exhibit peaks in both the winter and summer months. In the northeast and industrial midwest regions the summer peak is slightly more pronounced than the winter peak, and in the upper midwest region the winter peak is slightly more pronounced than the summer peak. In the southeast a single peak period in the summer is evident. In western regions peaks occur in the late fall and winter months.

Figure 2-19 shows the distributions of estimated 24-hour average urban $PM_{10-2.5}$ concentrations in different geographic regions. In all regions except the southwest, the lowest concentrations occur in the winter months. Elevated levels are apparent in the easternmost regions in either April or May. In the upper midwest, northwest, and southern California regions the highest levels occur in the late summer to early fall. The southwest region exhibits the

Figure 2-18. Urban 24-hour average $PM_{2.5}$ concentration distributions by region and month, 2000-2002. Box depicts interquartile range and median; line connects monthly means.

Source: Schmidt et al. (2003)

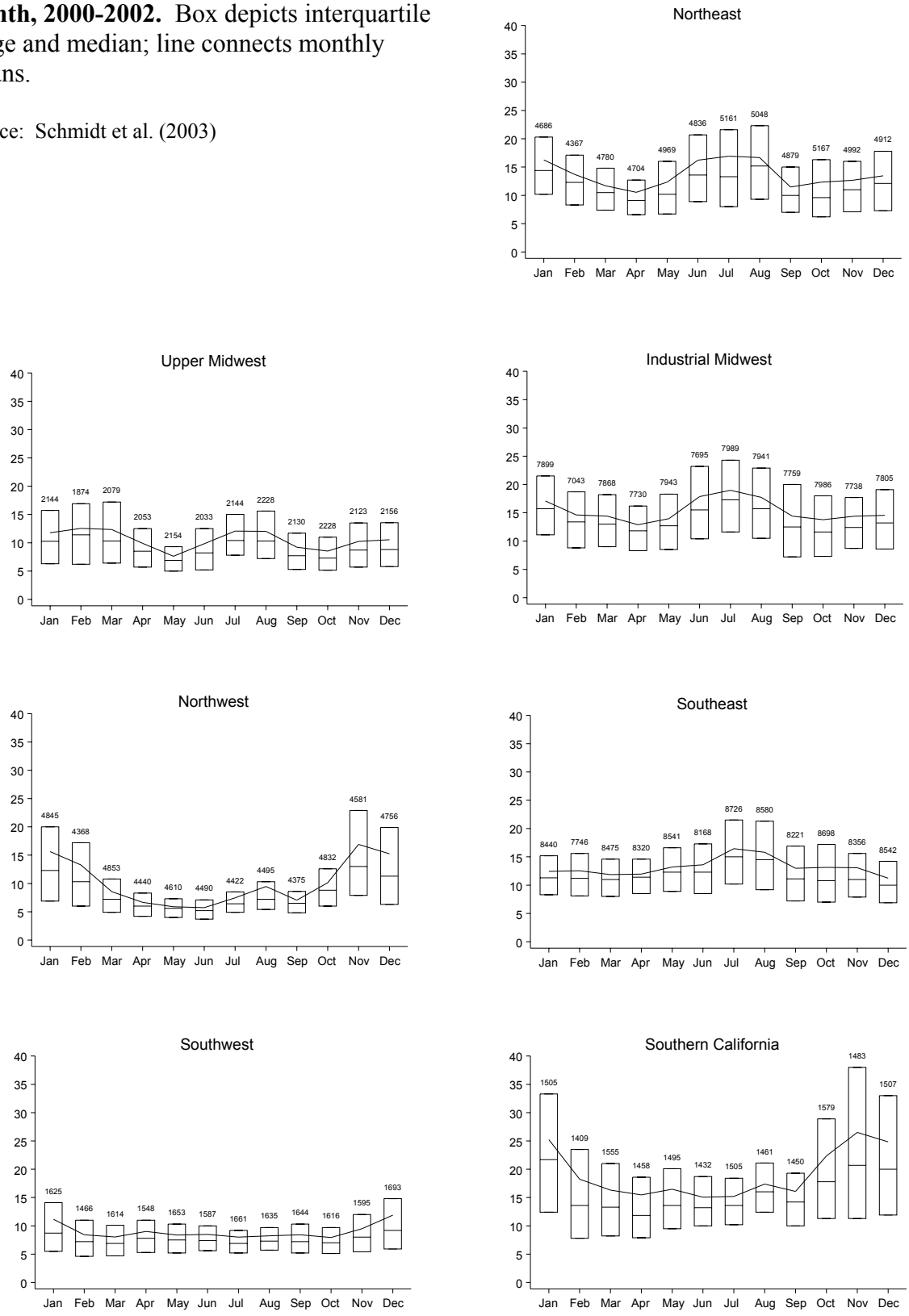
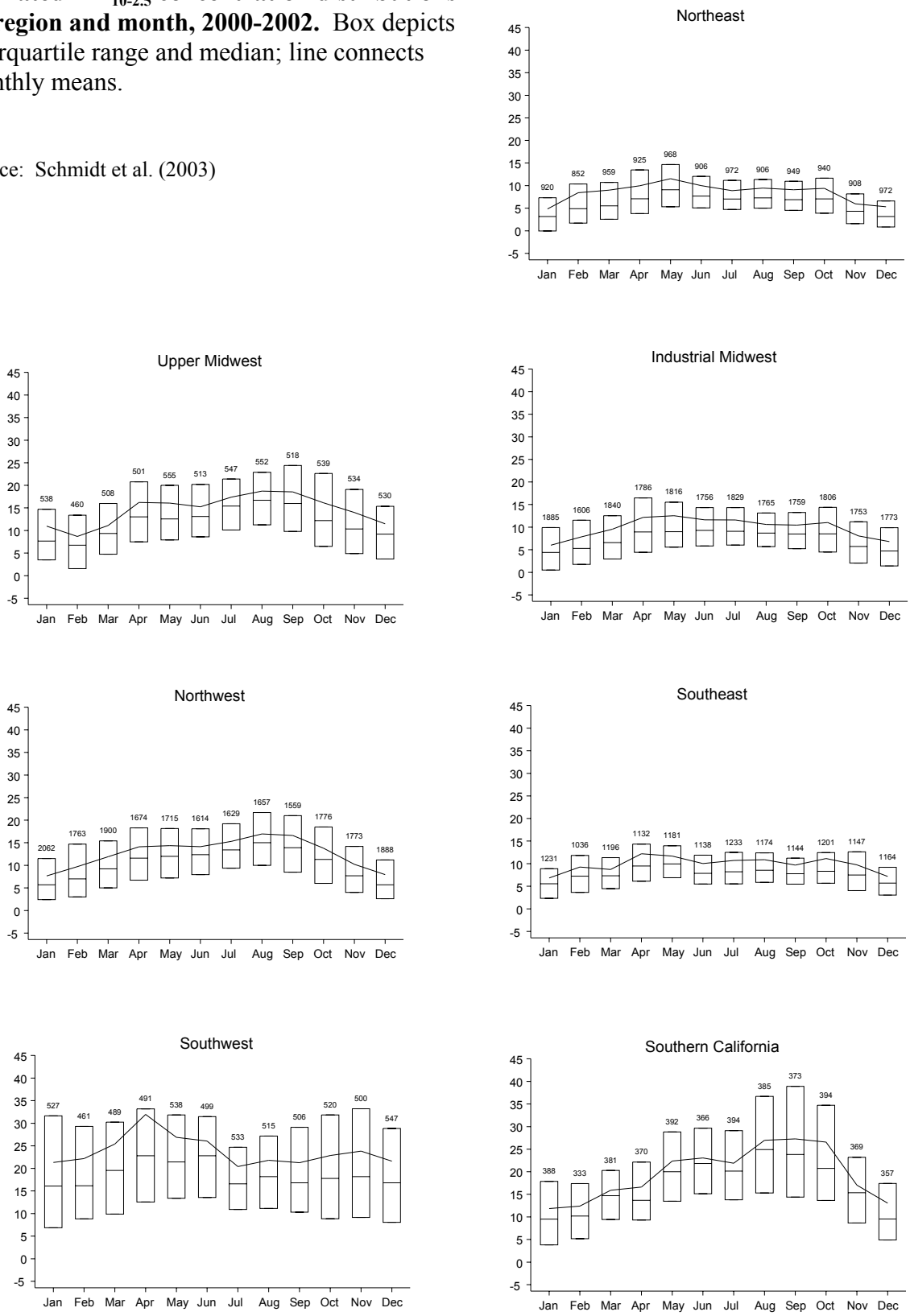


Figure 2-19. Urban 24-hour average estimated $PM_{10-2.5}$ concentration distributions by region and month, 2000-2002. Box depicts interquartile range and median; line connects monthly means.

Source: Schmidt et al. (2003)



greatest range of variability throughout the year. Elevated levels are apparent in the spring, consistent with winds that contribute to blowing dust.

The relationship between the annual mean at a site and the shorter-term 24-hour average peaks is useful for examining the relationships between short- and long-term air quality standards. The box plots in Figures 2-20 and 2-21 show the relationships for $PM_{2.5}$ and estimated $PM_{10-2.5}$, respectively, between annual mean PM concentrations and peak daily concentrations as represented by the 98th percentile of the distribution of daily average concentrations at FRM sites across the U.S. There is considerable variability in peak daily values for sites with similar annual means. For annual mean $PM_{2.5}$ values between 10 and 15 $\mu g/m^3$ the interquartile range spans about 5 to 6 $\mu g/m^3$ for each 1 $\mu g/m^3$ interval. The range between the 5th and 95th percentile values for each interval varies substantially. Estimated $PM_{10-2.5}$ generally exhibits greater variability in 98th percentile values for sites with similar annual means than seen for $PM_{2.5.}$ The maximum estimated $PM_{10-2.5}$ values are quite high relative to the rest of the distribution starting with the annual mean interval over 16 $\mu g/m^3$.

Monitors that provide near-continuous measurements can provide insights into short-term (e.g., hourly average) patterns in PM, which could be important to understanding associations between elevated PM levels and adverse health effects. Examples of average hourly profiles for $PM_{2.5}$ and $PM_{10-2.5}$ from 2000-2002 are shown in Figures 2-22 and 2-23 for a monitoring site in the Cleveland, OH metropolitan area. The $PM_{2.5}$ profile in Figure 2-22 indicates elevated hourly average levels occurred most often between the hours of 6:00 am and 9:00 am, corresponding to the typical morning rush of automobile traffic, with the highest levels in the summer and fall. The 95th percentile concentrations during peak hours can be as high as four times the median level for the same hour. Slightly lower peak levels occur between the hours of 10:00 pm and midnight, corresponding to the onset of the nighttime inversion. This profile of hourly average $PM_{2.5}$ levels is typical of many, but not all, urban areas. The $PM_{10-2.5}$ profile in Figure 2-23 indicates a pattern of hourly average levels similar to the pattern for $PM_{2.5}$.

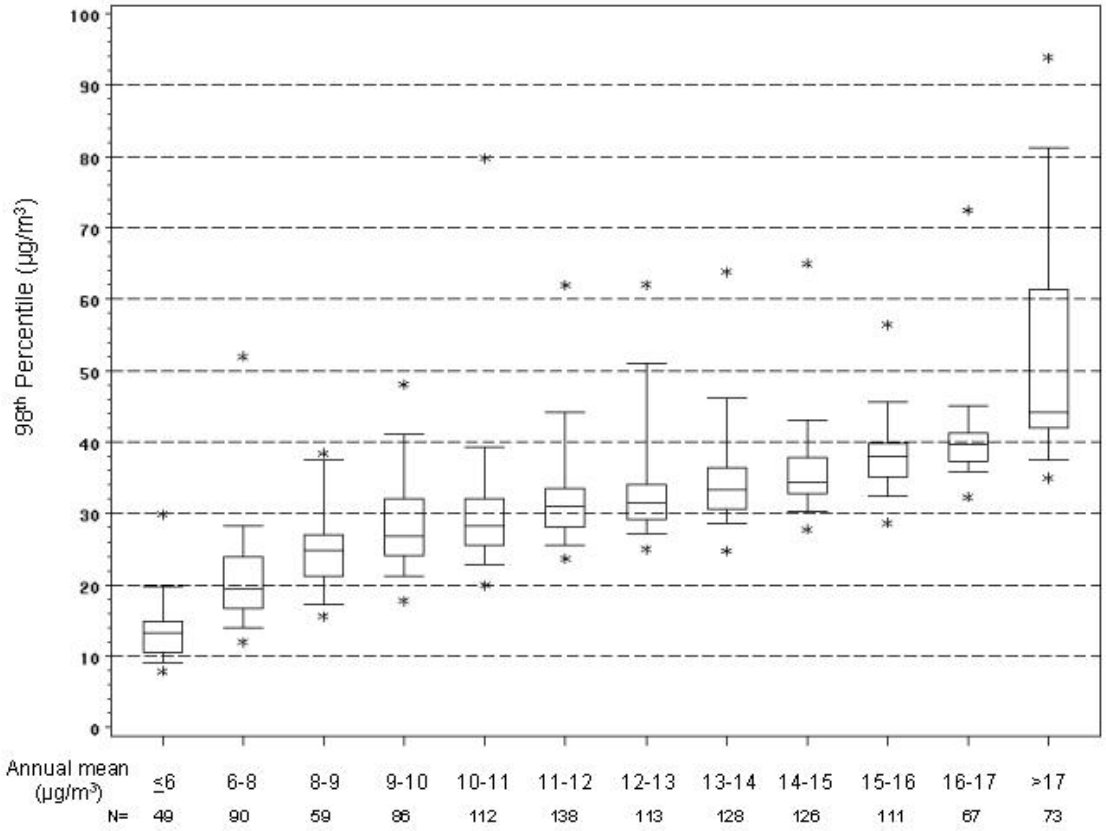


Figure 2-20. Distribution of annual mean vs. 98th percentile 24-hour average $PM_{2.5}$ concentrations, 2000-2002. Box depicts interquartile range and median; whiskers depict 5th and 95th percentiles; asterisks depict maximum and minimum. N = number of sites in each interval.

The hourly ranges shown in Figure 2-22 suggest hour-to-hour changes in $PM_{2.5}$ concentrations encompass several µg/m^3, however, extreme values for hour-to-hour variations can be much larger (Fitz-Simons et al., 2000). An analysis of the distribution of increases in hour-to-hour concentrations at multiple sites across the U.S. for 1999 finds site-level median increases of about 1 µg/m^3 to 30 µg/m^3, and 95th percentile increases ranging from about 4 µg/m^3 to 16 µg/m^3.

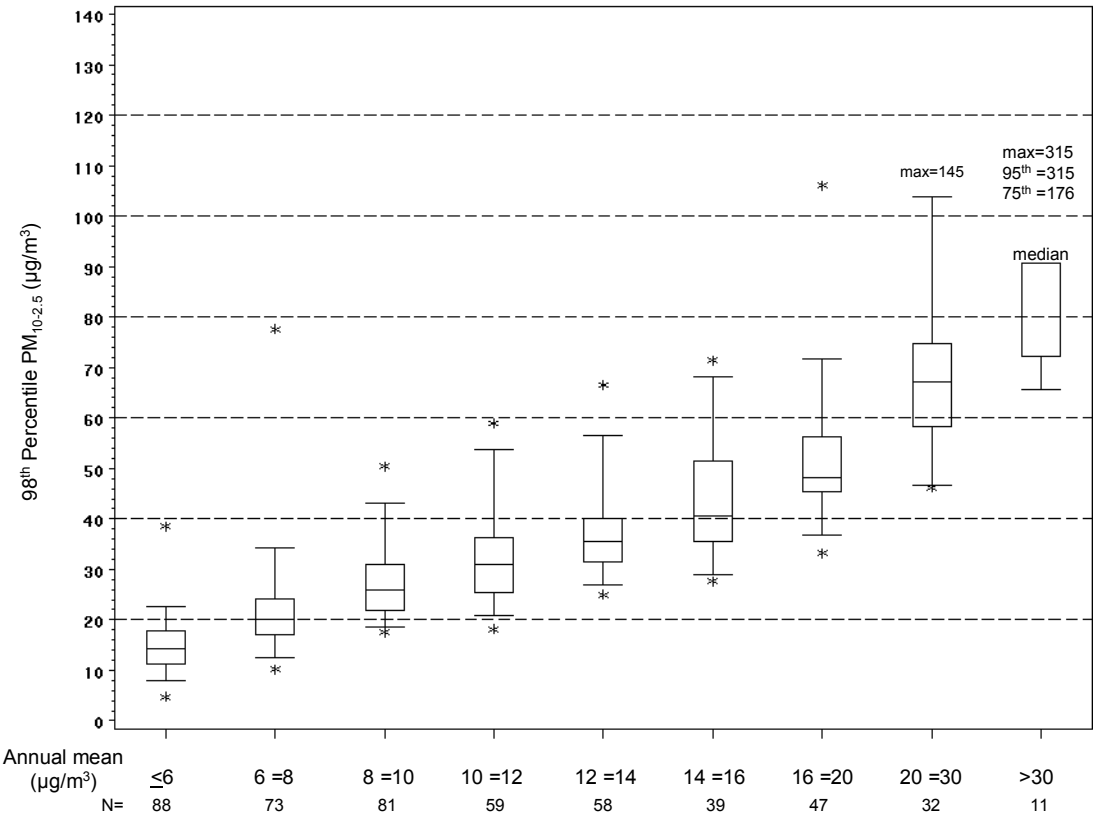


Figure 2-21. Distribution of estimated annual mean vs. 98th percentile 24-hour average $PM_{10-2.5}$ concentrations, 2000-2002. Box depicts interquartile range and median; whiskers depict 5th and 95th percentiles; asterisks depict minimum and maximum. N = number of sites in each interval.

Source: Schmidt et al. (2003)

2.6.2 Ultrafine Patterns

Diurnal or seasonal patterns for ultrafine particles have been extensively studied in relatively few areas of the U.S. At an urban site in Atlanta, GA, ultrafine particle number concentrations were found to be higher on weekdays than on weekends (Woo et al., 2001). Concentrations of particles in the range of 0.01 to 0.1 µm were higher at night than during the daytime, and tended to reach their highest values during the morning period when motor vehicle

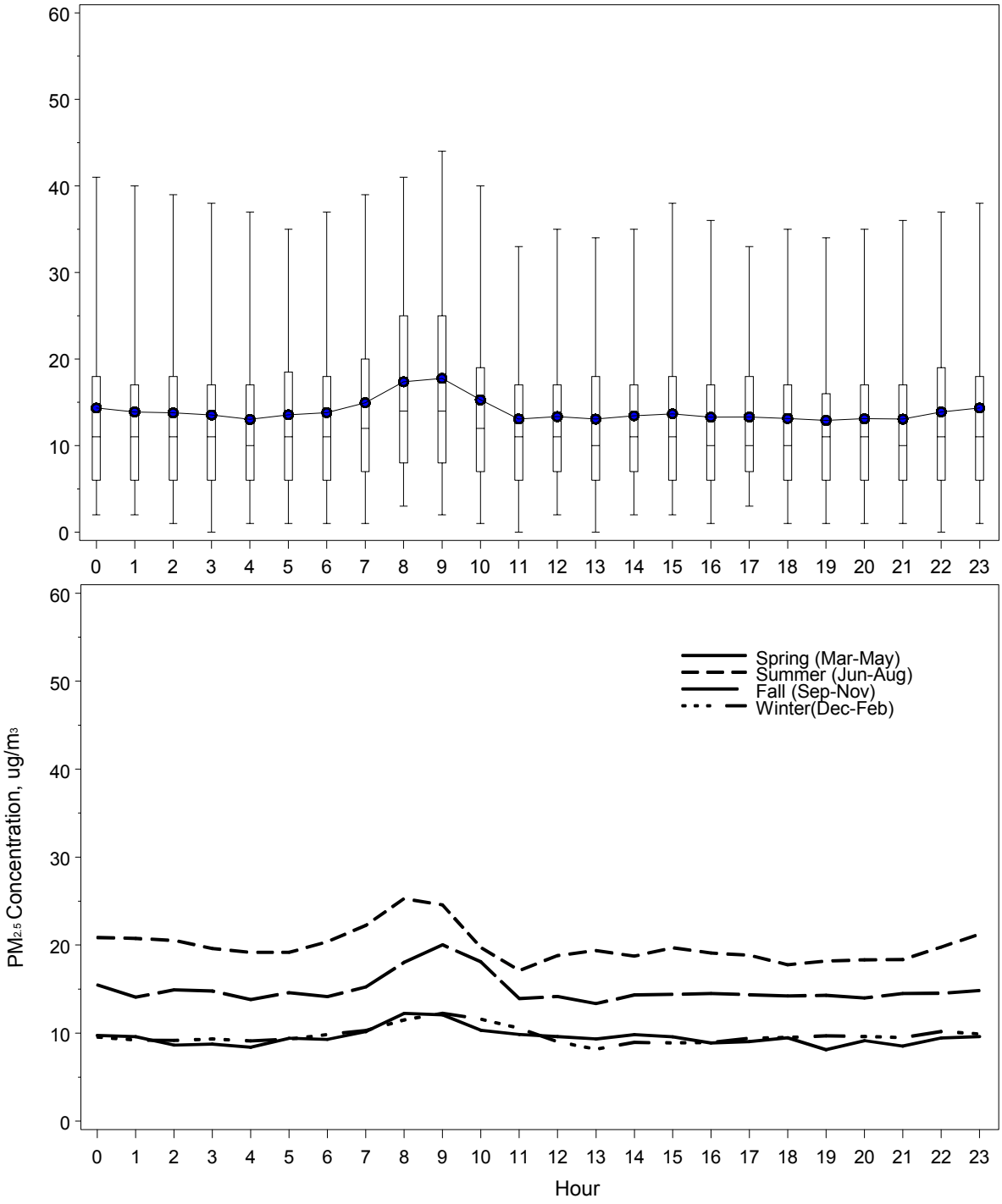


Figure 2-22. Hourly average $PM_{2.5}$ concentrations at a Cleveland, OH monitoring site, 2000-2002. Upper panel shows the distribution of concentrations (box plot of interquartile range, mean, median, 5th and 95th percentiles); lower panel shows seasonal averages by hour.

Source: Schmidt et al. (2003)

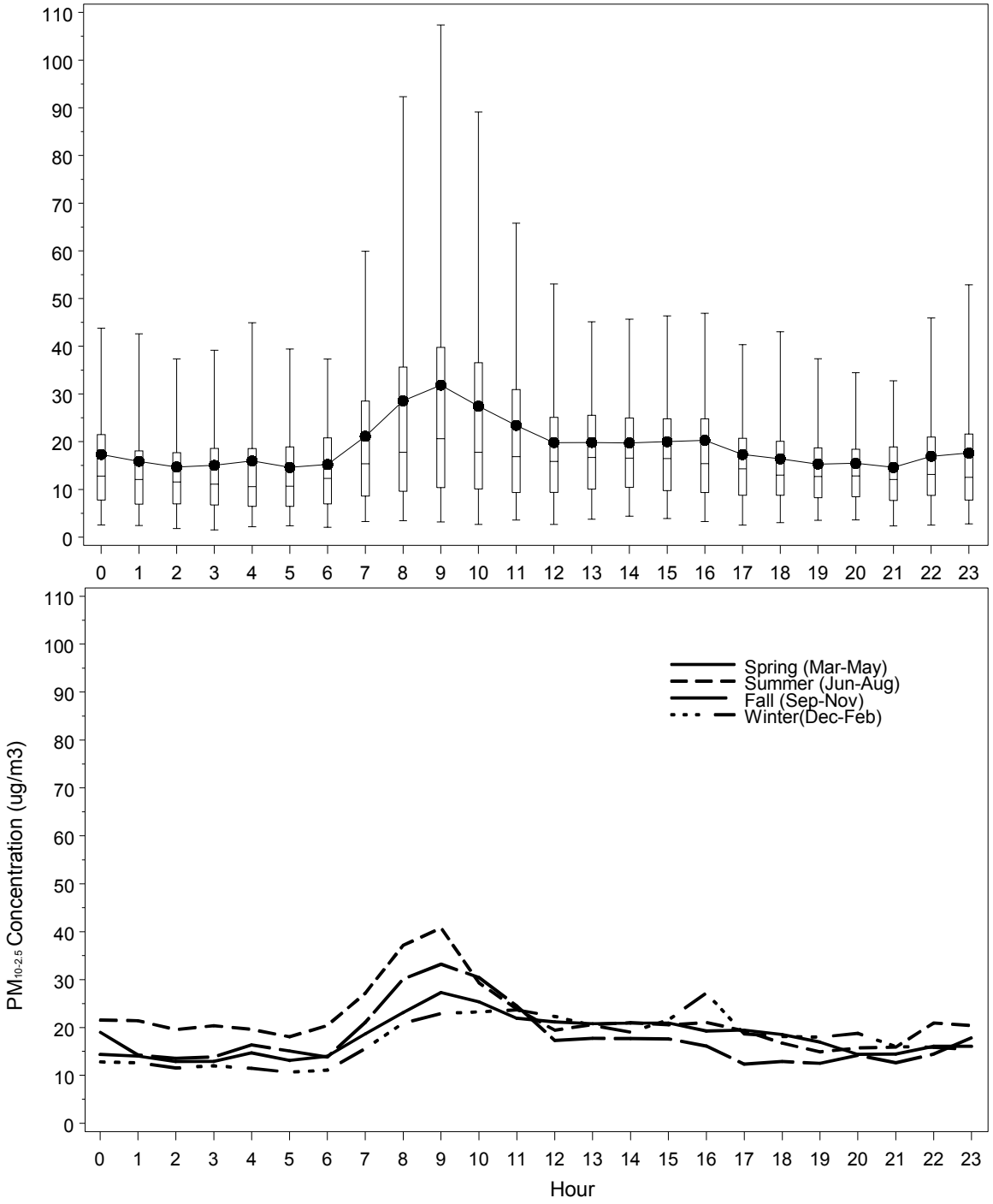


Figure 2-23. Hourly average $PM_{10-2.5}$ concentrations at a Cleveland, OH monitoring site, 2000-2002. Upper panel shows the distribution of concentrations (box plot of interquartile range, mean, median, 5th and 95th percentiles); lower panel shows seasonal averages by hour.

Source: Schmidt et al. (2003)

traffic is heaviest. Smaller particles in the range of 0.004 to 0.01 μm were elevated during the peak traffic period, most notably in cooler temperatures, below 50°F. Kim et al. (2002) report similar results for Riverside, California. At the Atlanta site, episodes of relatively high ultrafine particle levels were observed during corresponding periods of peak SO_2 levels. In El Paso, TX, ultrafine particle concentrations were also shown to vary in patterns similar to traffic volumes (Noble et al., 2003).

2.7 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 both natural and man-made sources outside of North America. Estimating background PM concentrations is important for the health risk analyses presented in Chapter 4and the assessment of ecosystem and visibility effects in Chapter 5. The draft CD states that "recent but limited information about background PM concentrations have not provided sufficient evidence to warrant any changes in estimates of the annual average background concentrations given in the 1996 PM AQCD" (CD, p. 3-104). However, the draft CD 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 an anthropogenic component. The natural background arises from: (1) physical processes of the atmosphere that entrain coarse-mode particles (e.g., windblown crustal material, sea salt spray); (2) volcanic eruptions (e.g., sulfates); (3) natural combustion such as wildfires (e.g., elemental and organic carbon, and inorganic and organic PM precursors); and (4) biogenic sources such as vegetation, microorganisms, and wildlife (e.g., organic PM, inorganic and organic PM precursors). The exact magnitude of the natural portion of PM for a given geographic location cannot 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 or quasi-natural events occurring outside the continental U.S. (CD, p. 3-68). The occurrence and location of these long-range

transport events are highly variable and their impacts on the U.S. are equally variable. The contributions to background from sources outside of North America can be significant on an episodic, but probably not on an annual basis (CD, p. 3-89). Several studies have focused on identifying the origin, sources, and impacts of recent transnational transport events from North America and extra-continental sources.

- 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. 3-71).

- Wildfires in the boreal forests of northwestern Canada may impact large portions of the eastern United States. The CD estimates 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. 3-73).

- 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 U.S., primarily Florida; North African dust has also been tracked as far as Illinois and Maine. These events have been estimated to contribute 6 to 11 $\mu g/m^3$ to 24-hour average $PM_{2.5}$ levels in affected areas during the events (CD, p. 3-69).

- 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 $\mu g/m^3$ during an episode in the last week in April 1998, compared to an average of about 20 $\mu g/m^3$ during the rest of April and May (CD, p. 3-69).

Section 3.3.3 of the draft CD provides broad estimates of annual average background PM levels, as shown in Table 2-6. The lower bounds of the ranges are based on compilations of natural versus anthropogenic emissions levels, ambient measurements in remote areas, and regression studies using anthropogenic and/or natural tracers (the results from 52 publications were used in this effort) (NAPAP, 1991). 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 as well as background, they may overestimate the actual background concentrations. There is a distinct 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. For $PM_{10-2.5}$, the draft CD (p. 3-82) presents a very rough point estimate of 3 $\mu g/m^3$ for an annual average in both the eastern and western U.S.

Table 2-6. Estimated Range of Annual Average PM Regional Background Levels

	Western U.S. ($\mu g/m^3$)	Eastern U.S. ($\mu g/m^3$)
PM_{10}	4 - 8	5 - 11
$PM_{2.5}$	1 - 4	2 - 5
$PM_{10-2.5}$	<1 - 7	<1 - 9

Source: draft CD, p. 3-82

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, both of North American and international origin, can lead to very high levels of PM comparable to, or greater than, those driven by man-made emissions in polluted urban atmospheres. Because such excursions are essentially uncontrollable, EPA has in place a "natural events" policy that removes consideration of them from attainment decisions[18].

Disregarding such large and unique events, an estimate of the range of "typical" background on a daily basis can be obtained from reviewing multi-year data at remote locations. In the last review, EPA staff analyzed $PM_{2.5}$ concentrations from selected IMPROVE sites and found 24-hour peak to annual mean ratios in the range of two to four. Applying these ratios to estimated annual background levels suggested that the highest 24-hour $PM_{2.5}$ background concentrations over the course of a year could be in the range of 15 to 20 $\mu g/m^3$ (EPA, 1996b, p.

[18] Under this policy (Nichols, 1996), EPA will exercise its discretion not to designate areas as nonattainment and/or to discount data in circumstances where an area would attain but for exceedances that result from uncontrollable natural events. Three categories of natural PM events are specified: volcanic or seismic activity, wildland fires, and high wind dust events.

IV-14). Based on current air quality information, EPA staff has conducted an analysis of five recent years (1997-2001) of data at IMPROVE sites across the U.S. (Langstaff, 2003a). Sites with five-year means below the upper end of the ranges for annual mean background in Table 2-6 in the eastern and western U.S. were selected for analysis. There are three such sites in the East and 31 in the West[19]. Staff focused on the 99th percentile concentrations at each of these sites, so as to avoid including excursions that likely reflect exceptional natural events. The 99th percentile concentrations at the eastern sites ranged from 13 to 17 $\mu g/m^3$, and from 6 to 20 $\mu g/m^3$ at the western sites. Even though these monitors are probably among the least likely to be impacted by North American anthropogenic pollution, there will be some impact, and therefore they will still tend to overestimate background concentrations. Considering these factors, the distributions of daily $PM_{2.5}$ concentrations at these sites provide an indication of the ranges for the daily variability of $PM_{2.5}$ background concentrations, and the 99th percentiles of these distributions are a reasonable estimate of the highest daily background concentrations. Staff notes that these recent findings are generally consistent with those from the last review, continuing to suggest a range of about 15 to 20 $\mu g/m^3$ as the upper end of the distribution of daily $PM_{2.5}$ background concentrations in the U.S.

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

The statutory focus of the primary NAAQS for PM is to protect public health from the adverse effects associated with the presence of PM in the ambient air – that is, the focus is on particles in the atmosphere that are emitted by sources to the outdoors or formed by chemical reactions in the atmosphere. We refer to the concentrations of PM in the ambient air as *ambient PM*. An understanding of human exposure to ambient PM helps inform the evaluation of underlying assumptions and interpretation of results of epidemiologic studies that characterize relationships between monitored ambient PM concentrations and observed health effects (discussed in Chapter 3).

[19] One of the western sites (Lasson Volcanic National Park) was removed from the analysis since it had anomalously high concentrations.

An important exposure-related issue for this review is the characterization of the relationships between ambient PM concentrations measured at one or more centrally located monitors and personal exposure to ambient PM, as characterized by particle size, composition, or other factors. Information on the type and strength of these relationships, discussed below, is relevant to the evaluation and interpretation of associations found in epidemiologic studies that use measurements of PM concentrations at centrally located monitors as a surrogate for exposure to ambient PM.[20] The focus here is on particle size distinctions; the draft CD (CD, Section 5.4) also discusses exposure relationships related to compositional differences.

2.8.1 Definitions

Exposure to a contaminant is defined as contact at a boundary between a human and the environment (e.g., the breathing zone) at a specific contaminant concentration for a specific interval of time; it is measured in units of concentration(s) multiplied by time (or time interval) (National Research Council, 1991). An individual's *total personal exposure* to PM results from breathing air containing PM in different types of environments (e.g., outdoors near home, outdoors away from home, indoors at home, indoors at office or school, commuting, restaurants, malls, other public places) These environments may have different concentrations of PM with particles originating from a wide variety of sources.

Ambient PM is comprised of particles emitted by anthropogenic and natural sources to the outdoors and particles formed in the atmosphere from emissions of gaseous precursors. This includes emissions not only from outdoor sources such as smokestacks and automobiles, but also from sources located indoors, such as fireplaces, wood stoves, and some industrial processes, that are vented to the outdoors. Exposure to ambient PM can occur both outdoors and indoors to the extent that ambient PM penetrates into indoor environments – we use the term *PM of ambient origin* to refer to both outdoor and indoor concentrations of ambient PM. We use the term *nonambient PM* to refer to concentrations of PM that are due to other indoor sources of particles such as smoking, cooking, other sources of combustion, cleaning, resuspension of

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

particles, mechanical processes, and chemical interactions occurring indoors. In characterizing human exposure to PM concentrations relevant to setting standards for ambient air quality, the draft CD conceptually separates an individual's total personal exposure to PM into *exposure to PM of ambient origin* and exposure to all other sources of PM (i.e., *nonambient exposure*).

Outdoor concentrations of PM are affected by emissions, meteorology, atmospheric chemistry, and removal processes. Indoor concentrations of PM are affected by several factors, including outdoor concentrations, processes that result in infiltration of ambient PM into buildings, indoor sources of PM, aerosol dynamics and indoor chemistry, and removal mechanisms such as particle deposition, ventilation, and air-conditioning and air cleaning devices (CD, p. 5-119). Concentrations of PM inside vehicles are subject to essentially the same factors as concentrations of PM inside buildings. Personal exposure to PM also includes a component 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, such as walking on a carpet.

Epidemiologic studies use measurements from central monitors to represent the variety of concentrations in an area. We use the term *central site* to mean the site of a PM monitor centrally located with respect to the area being studied. In some cases epidemiologic studies combine the measurements from more than one monitor to obtain a broader representation of area-wide PM concentrations than a single monitor provides.

2.8.2 Centrally Monitored PM 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 particles affect population exposures. Distinctions between PM size classes and components were found to be important considerations in addressing the representativeness of central monitors. For example, fine particles have a longer residence time and generally exhibit less variability in the atmosphere than the coarse fraction. As discussed in the 1996 Staff Paper (EPA, 1996b), the

1996 Criteria Document concluded that measurements of daily variations of PM have a plausible linkage to daily variations of human exposures to PM of ambient origin for the populations represented by the ambient monitoring stations, and that this linkage is stronger for fine particles than for PM_{10} or the coarse fraction of PM_{10}. The 1996 Criteria Document 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 ambient 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.

An individual's total personal exposure to PM generally differs from the ambient concentration measured at the central site monitor because: (1) spatial differences in ambient PM concentrations exist across a city or region; (2) generally only a fraction of the ambient PM is present in indoor or in-vehicle environments, whereas individuals generally spend a large percentage of time indoors; and (3) a variety of indoor sources of PM 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 sites, the spatial variability of PM plays a large role. As discussed in Section 2.5, for many areas $PM_{2.5}$ concentrations are fairly uniform spatially, with higher concentrations near roadways and other direct sources of $PM_{2.5}$. Analyses of $PM_{2.5}$ data for 27 urban areas indicate that differences in annual mean concentrations between monitoring sites in an urban area range from less than 1 $\mu g/m^3$ to as much as 8 $\mu g/m^3$. However, the correlations of daily $PM_{2.5}$ between sites are typically high. Daily mean $PM_{2.5}$ concentrations exhibit much higher spatial variability than annual means, even when the daily concentrations at sites are highly correlated. Although the spatial variability of $PM_{2.5}$ varies for different urban areas, overall, some degree of uniformity results from the widespread formation and long lifetime of the high regional background of secondary $PM_{2.5}$. In summarizing the key findings related to spatial variability in $PM_{2.5}$ concentrations, the draft CD states (p. 3-103):

> Although $PM_{2.5}$ concentrations within an MSA can be highly correlated between sites, there can still be significant differences in their concentrations. The degree of spatial uniformity in $PM_{2.5}$ concentrations and the strength of site to site correlations in urban areas varies across the country. These factors should be considered in using data obtained by the $PM_{2.5}$ FRM network to approximate community-scale human exposures, and caution should be exercised in extrapolating conclusions as to spatial uniformity or correlations obtained in one urban area to another. Limited information also suggests that the spatial variability in urban source contributions is likely to be larger than for regional source contributions to $PM_{2.5}$ and for $PM_{2.5}$, itself.

Relative to fine particles, coarse particles are likely to be more variable across urban scales. Daily mean $PM_{10-2.5}$ concentrations tend to be more variable and have lower inter-site correlations than $PM_{2.5}$, possibly due to their shorter atmospheric lifetime and the more sporadic nature of $PM_{10-2.5}$ sources (CD, Section 3.2.5).

The second factor influencing the relationship between ambient PM concentrations measured at central sites and total personal exposure to PM is the extent to which ambient PM penetrates indoors and remains suspended in the air. If the flow of ambient PM into the home from the outdoors is very restricted, the relationship between ambient PM concentrations measured at a central site and total exposure to PM will be weaker than in a situation where ambient PM flows more readily into the home and is a greater part of the overall indoor PM concentrations. This 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 fraction of PM of ambient origin found in the indoor and in-vehicle environments. Rates of infiltration of outdoor PM into homes through cracks and crevices are higher for $PM_{2.5}$ than for PM_{10}, $PM_{10-2.5}$, or ultrafine particles (CD, p. 5-120). Since $PM_{10-2.5}$ infiltrates indoors less readily than $PM_{2.5}$ and settles out more rapidly than $PM_{2.5}$, a greater proportion of $PM_{2.5}$ of ambient origin is found indoors than $PM_{10-2.5}$, relative to their outdoor

concentrations. Thus, particle size also influences the amounts of PM of ambient origin found indoors.

The third factor influencing the relationship between ambient concentrations measured at central sites and total personal exposure is the contribution of indoor sources to total personal exposure. On average, individuals spend nearly 90 percent of their time indoors. The contribution of indoor sources to indoor concentrations of PM is significant, and can be quite variable on different days and between individuals. Indoor sources such as combustion devices (e.g., stoves and kerosene heaters) generate predominantly fine particles; cooking produces both fine and coarse particles; and resuspension (e.g., dusting, vacuuming, and walking on rugs) generates predominantly coarse particles (CD, p. 5-79).

These three factors related to total personal exposure can give rise to measurement error (CD, p. 5-113 to 5-118), which makes the quantification of relationships between concentrations measured at central site monitors and health effects more difficult due to reduction in statistical power. However, as discussed in the draft CD and below in Chapter 3, exposure measurement errors are not expected to influence the interpretation of findings from either the chronic or time-series epidemiological studies that have used ambient concentration data (CD, p. 5-117).

In looking more specifically at the relationship between personal exposure to PM of ambient origin and concentrations measured at central site monitors, an analysis of data from the PTEAM study[21] provides important findings, as discussed in the draft CD (p. 5-60 to 5-63 and 5-122 to 5-123). The PTEAM study demonstrated that central site ambient PM concentrations are well correlated with personal exposure to PM of ambient origin, while such concentrations are only weakly correlated with total personal exposure. This study also found that estimated exposure to nonambient PM is effectively independent of PM concentrations at central site monitors, and that nonambient exposures are highly variable due to differences in indoor sources across the study homes.

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

In other studies in which subjects experienced small indoor source contributions to their personal exposures, such that total exposure is mostly from PM of ambient origin, high correlations are generally found between total personal exposure and ambient PM measured at a central site (CD, p. 5-51). For example, measurements of ambient sulfate, which is mostly in fine PM, have been found to be highly correlated with total personal exposure to sulfate (CD, p. 5-121). Since in these studies there were minimal indoor sources of sulfate, the relationship between ambient concentrations and total personal exposure to sulfate was not weakened by variability in indoor-generated sulfates, explaining the higher correlations.

The draft CD discusses the finding by some researchers that some epidemiologic studies yield statistically significant associations between ambient concentrations measured at a central site and health effects even though there is a very small correlation between ambient concentrations measured at a central site and total personal exposures (CD, section 5.3.2.4). The explanation of this finding is that, as discussed above, total personal exposure includes both ambient and nonambient generated components, and while the nonambient portion of personal exposure is not generally correlated with ambient concentrations, the exposure to ambient concentrations is correlated with ambient concentrations. Thus, it is not surprising that health effects might correlate with central site PM concentrations, because exposure to ambient PM correlates with these concentrations, and the lack of correlation of total exposure with central site PM concentrations does not alter that relationship. By their statistical design, time-series epidemiologic studies of this type only address the ambient component of exposure.

There is not a clear consensus among exposure analysts at this time as to how well community monitor measurements of ambient air PM concentrations represent a surrogate for personal exposure PM of ambient origin. It is recognized that existing PM exposure measurement errors or uncertainties most likely will reduce the statistical power of PM health effects analyses, thus making it difficult to detect a true underlying association between the exposure metric and the health outcome. However, the use of ambient PM concentrations as a surrogate for exposures is not expected to change the principal conclusions from PM epidemiological studies that use community average health and pollution data. Based on these considerations and on the review of the available exposure-related studies, the draft CD

concludes that for time-series epidemiology, ambient PM concentration as measured at central site monitors is a useful surrogate for exposure to PM of ambient origin (CD, p. 9-117).

2.9 OPTICAL AND RADIATIVE PROPERTIES OF PARTICLES

By scattering and absorbing electromagnetic radiation, ambient particles can impair visibility, affect climate processes, and affect the amount of ultraviolet radiation that reaches the earth. The sun emits electromagnetic radiation across the full spectrum, including at ultraviolet (0.028 to 0.4 μm) and visible (0.4 to 0.7 μm) wavelengths, while the earth emits at thermal infrared (0.7 to 3.2 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 their vertical and horizontal distribution in the lower atmosphere.

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. As discussed in section 4.3 of the draft CD, the efficiency of particles in causing visibility impairment depends on particle size, shape, and composition. Accumulation mode particles, within the fine mode, are generally most effective in impairing visibility. Accumulation-mode particle components principally responsible for visibility impairment are sulfates, nitrates, organic matter, black carbon, and soil dust. All such particles scatter light to some degree, but only black carbon plays a significant role in light absorption. Since black 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. 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, aerosols of a given mass, size distribution, and composition would likely cause greater visibility impairment in an eastern versus a western location.

2.9.2 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 and regional climate. As discussed in section 4.5.1 of the draft CD, particles can have both direct and indirect effects on climatic processes. The direct effects are the result of the same processes responsible for visibility degradation, namely radiative scattering and absorption. However, while visibility impairment is caused by particle scattering in all directions, climate effects result mainly from scattering light away from the earth and into space. 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 black 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; depending on particle size and degree of reflectivity, mineral aerosol can reflect or absorb radiation. Dark minerals absorb across the solar and infrared radiation spectra leading to warming of the atmosphere. Light-colored mineral particles in the appropriate size range can scatter visible radiation, reducing radiation received at the earth's surface. Organic carbon particles mainly reflect radiation, whereas black carbon particles 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 radiative effects, particles can also have a number of indirect effects on climate related to their physical properties. For example, sulfate particles can serve as condensation nuclei which alter the size distribution of cloud droplets by producing more droplets with smaller sizes. 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. An important consequence of this effect on cloud properties is the suppression of rain and potentially major disruption of hydrological cycles downwind of pollution sources, leading to a potentially significant alteration of climate in the affected regions (CD, p. 4-218).

The overall radiative and physical 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 (CD, p. 4-219). As discussed in Section 4.5.1.2 of the draft CD, the effects of sulfate particles have been the most widely considered, with globally averaged radiative 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 suggests that even the sign as well as the magnitude of effects depends on the vertical distribution and effective particle radius.

The draft CD makes clear that atmospheric particles play an important role in climatic processes, but that their role at this time remains poorly quantified. In general, on a global scale, the direct effect of radiative scattering by atmospheric particles is to likely exert an overall net effect of cooling the atmosphere, while particle absorption may lead to warming. The net impact of indirect effects on temperature and rainfall patterns remains difficult to generalize. 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 (CD, p. 217). The draft CD concludes that any effort to model the impacts of local alterations in particle concentrations on projected global climate change or consequent local and regional weather patterns would be subject to considerable uncertainty (CD, p. 4-238).

2.9.2 PM Properties Affecting Transmission of Ultraviolet Radiation

As discussed in section 4.5.2 of the draft CD, 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. 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 that there is an interaction in the radiative effects of scattering particles and absorbing molecules, such as ozone, in the lower atmosphere (CD, p. 4-223).

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. 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 (CD, pp. 4-223 to 4-225). 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. 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, remains the same, or reduced 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 (CD p. 4-226).

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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 studies published since the last review, can be found in Chapters 6 through 9 of the draft 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 epidemiology, toxicological, dosimetry, and exposure-related studies 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 of effects of exposure to ambient PM; and (4) issues related to interpretation and evaluation of the health effects evidence, including discussion of the role of co-pollutants, issues related to epidemiological modeling, evidence for effects of various PM components, and the consistency and coherence of the evidence. Preliminary staff conclusions and recommendations related to primary standards for PM are incorporated into Chapter 6 of this draft Staff Paper.

In the last review, a variety of health effects had been associated with ambient PM at concentrations extending from those elevated levels found in the historic London episodes down to levels below the 1987 PM_{10} standards. Of special 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 demonstrated biological mechanisms that could explain observed effects.

A large number of new studies containing further evidence of serious health effects have been published since the last review, with important new information coming from epidemiologic, toxicological, controlled human exposure, and dosimetry studies. Examples of important new epidemiologic studies include:

- New 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 potential confounders, including gaseous co-pollutants, on observed PM associations. These studies can provide more precise estimates of the magnitude of a PM effect than most smaller-scale individual city studies.

- More studies of various health endpoints evaluating independent associations between effects and fine- and coarse-fraction particles, as well as ultrafine particles or specific components (e.g., sulfates, metals).

- Numerous new studies of cardiovascular endpoints, with particular emphasis on assessment of cardiovascular risk factors as well as symptoms.

- New analyses and approaches to addressing issues related to potential confounding by gaseous co-pollutants, possible thresholds for effects, and measurement error and exposure misclassification.

- Preliminary attempts to evaluate the effects of air pollutant combinations or mixtures including PM components using factor analysis or source apportionment methods to link effects with different PM source types (e.g., combustion, crustal[1] sources).

- Several new "intervention studies" providing evidence for improvements in respiratory or cardiovascular health with reductions in ambient concentrations of particles and gaseous co-pollutants.

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), and animal models representing sensitive subpopulations, that are relevant to the plausibility of the

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

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.

- Studies relating population exposure to PM and other pollutants measured at centrally located monitors to estimates of exposure to ambient pollutants at the individual level have added new insights to our understanding of community health studies.

3.2 MECHANISMS

This section briefly summarizes available information concerning the penetration and deposition of particles in the respiratory tract and outlines potential physiological and pathological responses to PM, drawing from information presented in previous PM criteria and standard reviews and in Chapters 6 - 9 of the draft CD.

The 1996 Staff Paper 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 and reported respiratory and cardiovascular 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 causal links 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 observes that the probability of any biological effect of PM depends on particle deposition and retention, as well as underlying dose-response relationships

(CD, p. 6-42). 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, Figure 6-1). 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, 1996b).

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. Recent studies have indicated that ultrafine, as well as larger particles show enhanced deposition of particles at airway bifurcations (CD, p. 6-20). 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. Peak deposition for ultrafine and coarse-fraction particles is in the upper part of the respiratory tract, while peak deposition for accumulation-mode particles occurs in the alveolar regions (CD, p. 6-23, Figure 6-8). 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; for the smallest particles, ultrafine particles, there are even larger doses in terms of particle number.

The potential effects of deposited particles are influenced by the speed and nature of removal. Clearance and translocation mechanisms include dissolution of particles or particle constituents, mucociliary transport, or endocytosis by cells such as macrophages; the relative importance of the different mechanisms varies with each of the three regions (CD, Table 6-2, Figure 6-11). The presence of respiratory disease is a factor that can alter clearance of particles (CD, p. 6-57). Recent studies have suggested that ultrafine particles may move directly from the lungs into the systemic circulation, providing a pathway by which ambient PM exposure could rapidly affect extrapulmonary organs (CD, p. 6-53).

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). New evidence indicates that people with chronic lung disease can have increased total lung deposition, and can also show increases in local deposition ("hot spots") due to uneven airflow in diseased lungs (CD, p. 6-103). 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 in section 3.4.

As discussed in the 1996 Staff Paper, evidence from epidemiologic 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 then-current PM 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). Evidence from dosimetry studies has indicated 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 hypothesized 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 and observations advanced in the past reviews has been sufficiently tested or generally accepted as explaining the historical community results. Moreover, the hypothesized mechanisms cited in those previous reviews were based on insights developed from laboratory and occupational/community epidemiologic studies that involved concentrations that were substantially higher than those observed in contemporary 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 6, 7 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 fully 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 through 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) discussed particle components or characteristics hypothesized to contribute to PM health, producing an illustrative list of 11 components or 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 PM (CD, pp. 7-120 to 7-127). The potential mechanisms discussed in the draft CD, organized by effect category, are summarized in Table 3-1. The list of potential mechanisms 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. The draft CD highlights emerging evidence for these effects and potential mechanisms, as summarized below. The relative support for these potential mechanisms/intermediate effects and their relevance to real world inhalation

of ambient particles varies significantly. Moreover, the draft CD highlights the variability of results that exist among different approaches, investigators, animal models, and even day-to-day within studies. "Nevertheless, . . . much progress has been made since the 1996 PM AQCD in evaluating pathophysiological mechanisms involved in PM-associated cardiovascular and respiratory health effects" (CD, p. 7-120).

Table 3-1. Summary of Potential Mechanisms Based on Emerging Toxicological Evidence (CD, pp. 7-120 to 7-127)

Effect	Potential Mechanisms
Direct Pulmonary Effects	Lung injury and inflammation
	Increased susceptibility to respiratory infections
	Increased airway reactivity and exacerbation of asthma
Systemic Effects Secondary to Lung Injury	Lung injury leading to impairment of heart function by lowering blood oxygen levels and increasing the work of breathing
	Lung inflamation and cytokine production leading to adverse systemic hemodynamic effects (e.g., arrhythmia)
	Lung inflammation leading to increased risk of heart attacks and strokes due to increased blood coagulability
	PM/lung interactions potentially affecting hematopoiesis (e.g., blood cell formation)
Direct Effects on the Heart	Uptake of particles and/or distribution of soluble components from the lungs into the systemic circulation
	Effects on the autonomic control of the heart and cardiovascular system

Direct Pulmonary Effects. Substantial new toxicologic information summarized in the draft CD provides evidence for the occurrence of direct pulmonary effects; while many of the newer studies use high doses (in mg or hundreds of μg), some have used relatively low doses that are close to ambient concentrations. Evidence that supports hypotheses on direct pulmonary effects includes toxicological and controlled human exposure studies using both sources of

ambient particles and combustion-related particles. Toxicologic studies using intratracheal instillation of ambient particles from various locations (e.g., St. Louis, MO; Washington DC; Dusseldorf, Germany; Ottawa, Canada; Provo and Utah Valley, Utah; Edinburgh, Scotland) have "shown clearly that PM can cause lung inflammation and injury" (CD, p. 7-16). Several studies using filter extracts from Utah Valley ambient samples collected before, during and after the shut-down of a major particle-emitting facility have reported effects such as increases in oxidant generation, release of cytokines such as IL-8, and evidence of pulmonary injury such as increased levels of lactose dehydrogenase (CD, p 7-16 to 7-18). Administration of residual oil fly ash (ROFA, an example of a combustion source particle type) has been shown to produce severe inflammation, with effects including recruitment of neutrophils, eosinophils and monocytes into the airway (CD, p. 7-28). Several new toxicologic or controlled human exposure studies using exposure to CAPs have shown evidence of inflammatory responses that appear to be dependent on particle composition (CD, p. 7-21). *In vitro* studies, summarized in draft CD Table 7-9, also provide support for the observed inflammatory effects of ambient PM and constituent substances (CD, p. 7-68). Evidence from a limited number of toxicologic studies suggests that exposure to particles can increase susceptibility to respiratory infections (CD, p. 7-122). Moreover, a number of epidemiologic studies have linked daily changes in ambient PM concentrations with increased risk of hospitalization for pneumonia or respiratory infections (CD, Table 8-19). Some toxicologic evidence also indicates that PM can aggravate asthmatic symptoms or increase airway reactivity, especially in studies of the effects of diesel exhaust particles (CD, p. 7-122), while a number of epidemiologic studies have reported increased risk of exacerbation of asthma with ambient PM exposure (CD, p. 8-174).

Systemic Effects Secondary to Lung Injury. Secondary systemic effects may also result from lung injury or inflammation. While more limited than for direct pulmonary effects, some new evidence from toxicologic studies suggests that injury or inflammation in the respiratory system can lead to changes in heart rhythm, reduced oxygenation of the blood, changes in blood cell counts, or changes in the blood that can increase the risk of blood clot formation, a risk factor for heart attacks or strokes (CD, pp. 7-123 to 7-125).

Direct Effects on the Heart. Animal studies have also provided initial evidence that particles can have direct cardiovascular effects. As shown in draft CD Figure 9-15, there are

several pathways by which particle deposition in the respiratory system could lead to cardiovascular effects, such as PM-induced pulmonary reflexes resulting in changes in the autonomic nervous system that then could affect heart rhythm (CD, p. 9-62). Also, inhaled PM could affect the heart or other organs if particles or particle constituents are released into the circulatory system from the lungs; some new evidence indicates that the smaller ultrafine particles can move directly from the lungs into the systemic circulation (CD, p. 6-53). Recent epidemiologic studies have begun to include more sensitive measures of cardiovascular responses, linking ambient PM exposure with increased levels of fibrinogen or C-reactive protein in the blood, and with changes in heart function such as decreased heart rate variability or heart rhythm (CD, p. 9-103). The effects observed in epidemiologic studies could result either from direct effects of PM on the cardiovascular system, or secondarily through changes in the pulmonary system.

Summary. 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 (CD, p. 9-50). New studies have identified a variety of responses to constituents of ambient PM that may contribute to the health effects reported in epidemiologic studies. The draft CD observes "A primary causative attribute may not exist but rather many attributes may contribute to a complex mechanism driven by the nature of a given PM and its contributing sources." (CD, p. 9-61). Even though specific mechanisms or pathways for effects have not yet been demonstrated, the increasing laboratory evidence of pathways by which particles could affect the respiratory and cardiovascular systems adds to the plausibility of the conclusion that particles, alone or in combination with other pollutants, are playing a causal role in the effects observed in epidemiologic studies.

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
  - Cardiovascular health indicators, such as heart rate variability or C-reactive protein levels
  - Developmental effects, such as low birth weight, and infant mortality

In considering the nature of effects, it is important to note some key characteristics and limitations of the kinds of studies -- epidemiologic, toxicologic and controlled human exposure studies -- used to identify them.

The general strengths and weaknesses of epidemiologic studies were discussed in detail in the 1996 CD (Chapter 12) and are briefly reviewed in section 8.1.3 of the draft CD. Epidemiologic 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. Epidemiologic 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 from specific combustion sources; for example, the Criteria Document 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 copollutant (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. 8-8). 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. 8-10).

The potential for confounding by co-pollutants is an important question, and throughout the sections that follow, results of epidemiologic studies that consider both PM alone and PM with co-pollutants will be discussed. Testing for confounding using multi-pollutant models has been the most commonly used method to assess potential confounding. As discussed in the draft CD section 8.4.3.3, there are statistical issues to be considered with multi-pollutant models, such as possibly creating mis-fitting models by forcing all pollutants to fit the same lag structure, by adding correlated but non-causal variables, or by omitting important variables. However, where pollutant concentrations are not highly correlated, effect estimates from multi-pollutant models could be more sound than those from single pollutant models (CD, p. 9-69). In addition, it is possible that pollutants may act together, or that the effects of a single pollutant may be mediated by other components of an ambient pollution mix. As stated in the draft CD, "Specific attribution of effects to any single pollutant may therefore be overly simplistic" (CD, p. 9-69).

Several other key issues and considerations related to evaluating epidemiologic studies are also discussed below in section 3.5. For example, issues related to epidemiologic modeling are discussed, including statistical model specification and methods used to adjust for other variables, including weather variables, the lag period between PM exposure and associated effects, and the influence of measurement error on the results of epidemiologic studies. Other considerations related to the evaluation of PM-related health effects evidence discussed in section 3.5 includes the role of various components within the fine and coarse fractions of PM in the different health endpoints that have been associated with PM and the consistency and

coherence of the health effects evidence.

Animal toxicologic, controlled human exposure, and dosimetry studies can provide important support to epidemiologic 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. While animal models are intended to mimic aspects of human disease, the results of animal studies may not directly reflect human health responses. Further, controlled human exposure studies can only address the least severe health endpoints, for obvious ethical reasons, and are limited in their ability to examine health effects in the most sensitive populations. 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 four general categories: increased hospital admissions and emergency room visits for both cardiovascular and respiratory diseases, effects on the respiratory system, effects on the cardiovascular system, and developmental effects.

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) mortality 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 epidemiologic studies. The 1996 CD evaluated the findings of studies that used single- and multiple-pollutant models to assess the potential for copollutant 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

[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 epidemiologic studies on mortality and air pollution.

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 draft CD concluded that PM effects were not sensitive to other pollutants and the "findings regarding the PM effects are valid" (EPA, 1996a, p 13-57, EPA, 1996b, p V-56).

Taking into account these findings, the summary report from the HEI review panel 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. The HEI review panel observed that "[c]onsistent and repeated observations in locales with different air pollution profiles can provide the most convincing epidemiologic evidence to support generalizing the findings from these models" (HEI, 1997, p. 38). In fact, the HEI-sponsored study of PM_{10} and mortality in 90 U.S. cities was designed to use such a multi-city approach (Samet et al., 2000, p. 1).

Since the last review, more than 80 new time-series studies of the relationship between PM and mortality have been published (CD, p. 8-23), including several multi-city studies that are responsive to the recommendations from the last review. 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 effect 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 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. 8-30). While considering results from the full body of studies, staff will place emphasis on the results of multi-city studies in the following discussions.

Regarding the full body of multi-city and single-city studies, 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 effect estimates are generally consistent with the range of estimates from the last review (CD, p. 8-29). Consistent with Table 9-10 in the draft CD (p. 9-91), the Table in Appendix A summarizes daily mortality effect 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. A number of the new time-series epidemiologic studies have used generalized additive models (GAM) in their analyses, and issues have been found with the convergence criteria and the method for determining standard errors when using GAM, as discussed more fully in section 3.5.2.1. In Appendix A and the figures presented in this Chapter, results will be presented from studies that have been reanalyzed to address issues related to GAM use, or where GAM were not used in analysis; results from the full body of studies have been included in the assessment in the CD and are described in the appendices to Chapter 8 of the draft CD.

[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 consideration of consistency in effects across studies, the results presented in figures that follow, and the effect estimates summarized in Appendix A are from single-pollutant models, though results of multi-pollutant models are discussed in the text.

3.3.1.1.1 Multi-city Studies of Total Daily Mortality

The results from the U.S. and Canadian multi-city studies are summarized in Table 3-2. In the previous review, a single multi-city study evaluated associations between daily mortality and PM, including fine- and coarse-fraction particle measurements in six U.S. cities (Schwartz et al., 1996, reanalyzed in Schwartz, 2003a). Reanalyses using the Six City data have reported results consistent with the findings reported by Schwartz and colleagues (Klemm et al., 2000, reanalyzed in Klemm et al., 2003). As shown in Table 3-2, significant increases in total mortality of 2 to over 3% were reported per 25 $\mu g/m^3$ of $PM_{2.5}$ and 50 $\mu g/m^3$ of PM_{10} (using more stringent GAM and GLM)[5] while $PM_{10-2.5}$ was not significantly associated with mortality. The role of gaseous co-pollutants was not directly addressed in these analyses.

Several recent multi-city analyses 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; reanalyzed in Dominici et al., 2003). 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, copollutant evaluations, regional spatial correlation, and synthesis of results from multiple cities by hierarchical Bayesian meta-regressions and meta-analyses" (CD, p. 8-30). Based on the results shown in Figure 3-1, the overall risk estimate for all cities[6] is a statistically significant increase of 1.4% (using more stringent GAM) or 1.1% (using GLM) in total mortality per 50 $\mu g/m^3$ increase in PM_{10} lagged one day (Samet et al., 2000a,b, reanalyzed in

[5]GAM and GLM refer to generalized additive models and generalized linear models, respectively, and are further discussed in section 3.5.2.1 on model specification.

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

Table 3-2. Results of U.S. and Canadian Multi-city Studies on Associations between Short-term PM Exposure and Mortality

Study	% Increase in Total Mortality per:			Range of City PM Mean Levels ($\mu g/m^3$)
	50 $\mu g/m^3$ $PM_{15/10}$	25 $\mu g/m^3$ $PM_{2.5}$	25 $\mu g/m^3$ $PM_{10-2.5}$	
Six U.S. Cities Schwartz et al., 1996, reanalysis in Schwartz, 2003a	*(*)*	*3.5 (2.5, 4.5)*** *3.3 (2.2, 4.3)****	*(*)*	*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 et al., 2003	3.5 (2.0, 5.1)** 2.0 (0.3, 3.8)***	3.0 (2.0, 4.0)** 2.0 (0.9, 3.2)***	0.8 (-0.6, 2.1)** 0.3 (-1.2, 1.8)***	$PM_{15/10}$ medians 14.4-30.3 $PM_{2.5}$ medians 9.0-23.1 $PM_{10-2.5}$ medians 5.0-13.0
90 U.S. Cities Samet et al., 2000a,b; reanalysis in Domenici, 2003	1.4 (0.9, 1.9)** 1.1 (0.5, 1.7)***	---	---	PM_{10} 15.3-52.0
10 U.S. Cities Schwartz et al., 2000, reanalysis Schwartz 2003b	3.3 (2.6, 4.1)** 2.8 (2.0, 3.6)***	---	---	PM_{10} 27.1-40.6
8 Canadian Cities Burnett et al., 2000, reanalysis Burnett and Goldberg, 2003	3.2 (1.1, 5.5) ** 2.7 (-0.1, 5.5)***	2.8 (1.2, 4.4) ** 2.1 (0.1, 4.2)***	1.9 (-0.1, 3.9) ** 1.8 (-0.6, 4.4)***	PM_{10} 20.4-31.0 $PM_{2.5}$ 9.5-17.7 $PM_{10-2.5}$ 8.9-16.8

Studies in italics were available for review in the 1996 PM CD.
* reanalysis results not available
** results from GAM using more stringent convergence criteria
*** results from GLM using natural splines

Domenici, 2003). Further, PM_{10} was also positively associated with mortality at 0-day and 2-day lags (CD Figure 8-5, p. 8-34).

One key objective of the NMMAPS analyses 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 from the 90-city analysis of the "weak influence of gaseous co-pollutants on the PM_{10} effect size estimates" (CD, Figure 8-6, p. 8-35). Less consistent associations were found for the gaseous pollutants than for PM_{10}, though in the overall analyses, weak associations were found with SO_2, NO_2 and CO, and there was a significant association with ozone in the summer months (CD, p. 8-35). The authors concluded that this "suggests that

the effect of PM_{10} is robust to the inclusion of other pollutants." (Samet et al., 2000b, p. 19); this conclusion was affirmed in reanalysis (Domenici, 2003, p. 18). Potential confounding of PM effects by gaseous pollutants is discussed further in section 3.5.1.

As seen in Figure 3-1, effect estimates for many individual cities exhibit wide confidence ranges; the estimates vary and some are negative, though not statistically significant, suggesting potentially more heterogeneity in effect estimates across cities than had been seen with single-city studies. However, the NMMAPS investigators "caution against attempts to interpret estimates for any specific city" (Samet et al., 2000b, p. 43). For many single-city studies, study locations were selected based on the population size and availability of PM measurements. In contrast, NMMAPS, while advantageous in including data from many different locations with different climates and pollutant mixes, included many locations for which the sample size (i.e., population size and PM_{10} data) was inherently smaller for a given study period. This can result in reduced precision for estimated effect estimates for the individual cities. As observed in the draft CD:

> ... increasing scientific weight should be accorded to [studies] in proportion to the precision of their estimate of a health effect. Small studies and studies with an inadequate exposure gradient generally produce less precise estimates than large studies with an adequate exposure gradient. Therefore, the range of exposures (e.g., as indicated by the IQR), the size of the study as indexed by the total number of observations (e.g., days) and total number of events (i.e., total deaths), and the inverse variance for the principal effect estimates are all important indices useful in determining the likely precision of health effect estimates and in according relative scientific weight to the findings of a given study (CD, p. 8-29).

This is illustrated in Figure 3-2, where the individual city effect estimates are 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 precision of the analysis of each individual city. The use of the mortality-day product combines two of these indicators of precision, and it can be seen that as the mortality-day product increases, the effect estimates are generally more precise with narrower confidence intervals. In developing the combined 90-city PM_{10}-mortality effect estimate, the results for individual cities were weighted based on the variance of the city effect estimates, which is another indicator for estimate precision. Thus, staff has placed primary emphasis on the results of the overall multi-city effect estimates.

Also shown in Figure 3-1 are the results based on a grouping of these cities into the same seven U.S. regions depicted in Figure 2-3. Samet et al. (2000a,b, reanalyzed in Domenici, 2003) report that some variability in effects can be seen across cities and between regions. Combined effect estimates for each of the seven U.S. regions varied somewhat, with generally higher effects reported in the Northeast States 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 resulted in significant differences in effect across regions (CD, p. 8-35).

Schwartz (2000, reanalyzed in Schwartz, 2003b) 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 total mortality, with an effect estimate of an increase of 3.3% per 50 $\mu g/m^3$ PM_{10} (using GAM) or 2.8% per 50 $\mu g/m^3$ PM_{10} (using GLM), and effect estimate sizes were the same in summer and winter (CD, p. 8-37).

In a combined analysis of data for the 8 largest Canadian cities, Burnett et al. (2000, reanalyzed in Burnett and Goldberg, 2003) 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 appproximately 2-3% (using more stringent GAM and GLM) 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 3.5.3. 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 effect estimates were reduced and lost statistical significance. The authors conclude that mortality is associated with both PM and gaseous pollutants (Burnett et al., 2000); copollutant effects and multi-pollutant models were not included in the reanalysis of this study's results.

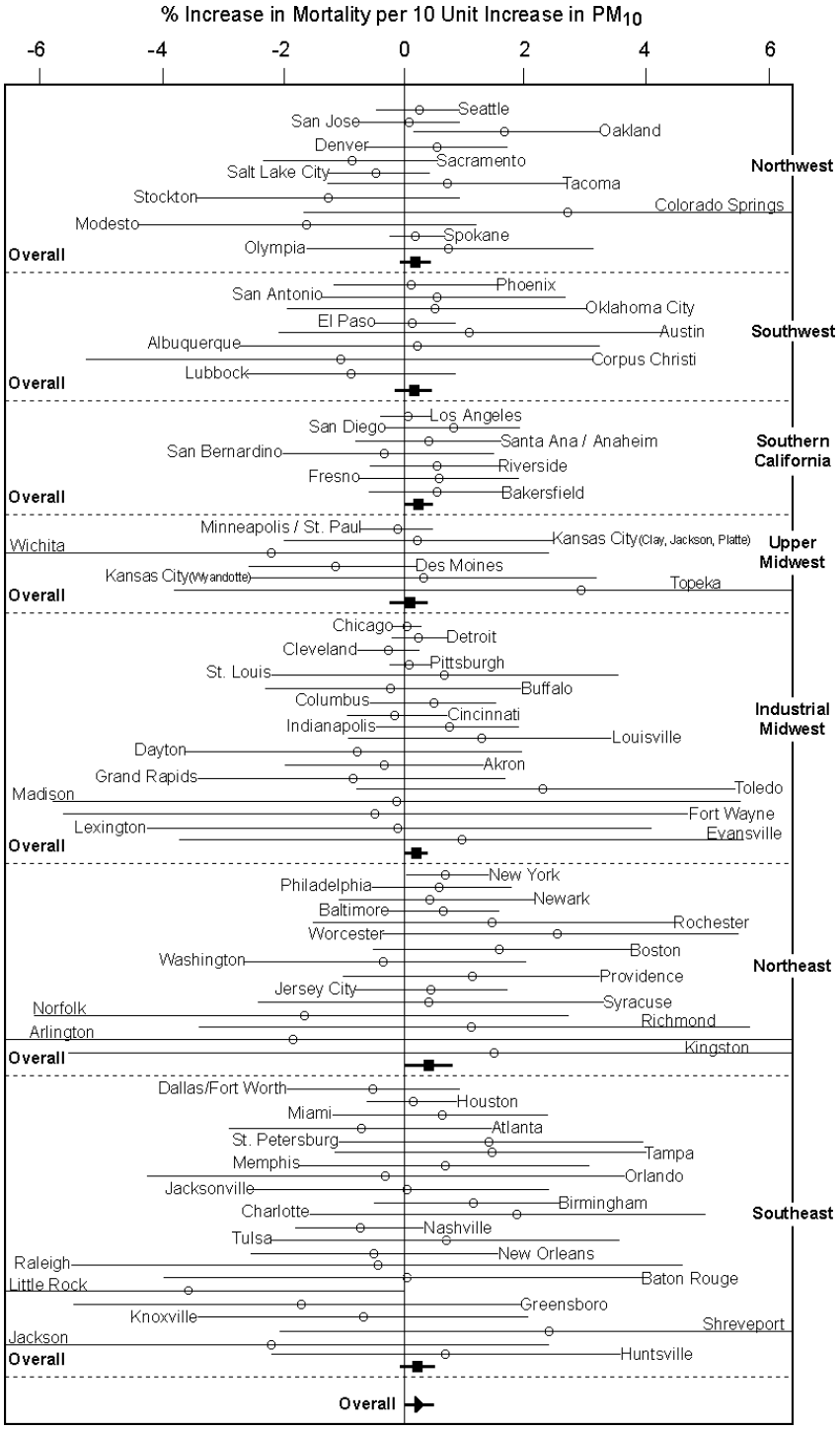


Figure 3-1. Estimated excess risks for PM mortality (1 day lag) for the 88 largest U.S. cities as shown in the revised NMMAPS analysis (CD Figure 8-3)

Source: Dominici et al. (2002; 2003).

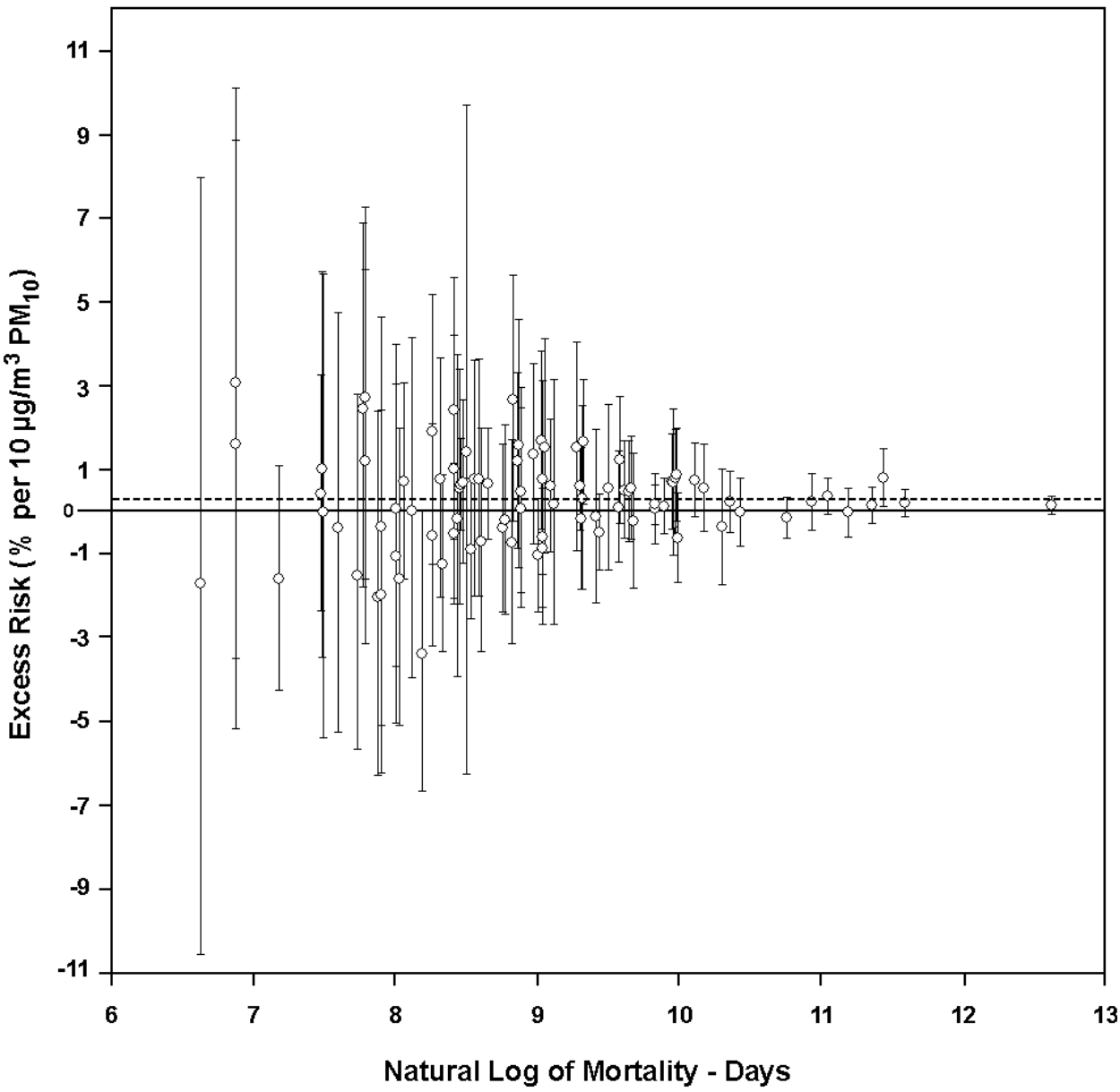


Figure 3-2. An EPA-derived plot showing relationship of PM_{10} total mortality effects estimates and 95% confidence intervals for all cities in the Samet et al. (2000a; Domenici, 2003) NMMAPS 90-cities analyses in relation to study size (i.e., the natural logarithm or numbers of deaths times days of PM observations). The dashed line depicts the overall nationwide effect estimate (grand mean) of approximately 0.28% per 10 µg/m^3 PM_{10} for models with no co-pollutants (from draft CD Figure 8-21).

In addition, a European multi-city study, Air Pollution and Health: A European Project (APHEA), has resulted in a series of analyses that are summarized in the draft CD (pp. 8-34 to 8-37). This study included data from 12 European countries in the initial analyses, and reported effect estimates for western cities of an approximately 2% increase in mortality per 50 $\mu g/m^3$ PM_{10} (CD, p. 8-44). Further analyses were done in a second series of studies (APHEA2) that included data from 29 European cities, where an overall effect estimate of a 3.0% increase in risk of mortality per 50 $\mu g/m^3$ PM_{10} was reported; reanalysis produced essentially identical results (CD, p. 8-47). Using this larger set of cities, little evidence for potential confounding by gaseous pollutants was reported, except for NO_2, which the APHEA investigators considered an indicator of high influence from traffic emissions (CD, p. 8-47). Although the APHEA studies used consistent analytical methodologies, the PM measurement methods varied between cities, including TSP, BS, PM_{13}, and PM_{10}, thus making quantitative comparisons with U.S. and Canadian findings more difficult; however, these PM-mortality effect estimates are generally consistent with the U.S. and Canadian results summarized in Table 3-2.

Analysis of data from the initial 12 APHEA countries was done to investigate factors that may influence the heterogeneity found between western and eastern European cities. In reanalysis of these data to address GAM-related modeling questions, the contrast between western and eastern cities was less clear in the models using more stringent convergence criteria or alternative models, suggesting that the apparent heterogeneity could be sensitive to model specification (CD, p. 8-46). In further investigation of potential heterogeneity across the 29 APHEA cities, the city-specific variables that were found to influence the PM_{10}-mortality associations included climate (larger effects with warmer climates), health status of the population (smaller effects with higher baseline mortality rates) and pollution mix (larger effects with increasing average NO_2 concentration). The investigators suggested that the relationship found with climate may be due to more open windows thus greater exposure to ambient air in warmer areas, and the relationship with NO_2 concentration indicates the importance of traffic as a PM source in the association with mortality (CD, p. 8-47).

In summary, the draft CD concludes that the findings of the Harvard Six-Cities study that were 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. Effect estimates from the multi-city studies range from approximately 1.0 to 3.5% per 50 $\mu g/m^3$ increase in PM_{10} and from 2 to over 3% per 25 $\mu g/m^3$ increase in $PM_{2.5}$ (CD, p. 8-50). The draft CD notes that the combined daily mortality estimates from these multi-city studies are all consistent with the range of PM_{10} effect estimates reported in the last review (CD, p. 8-50), with the 90-city NMMAPS 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 $PM_{10-2.5}$.

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), as well as locations in Europe, Mexico City, South America, Asia or Australia (summary of results in Table 8A-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 positive associations between mortality and both $PM_{2.5}$ and/or $PM_{10-2.5}$, though the associations with $PM_{10-2.5}$ less often reached statistical significance. 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 used nephelometric measures of PM. As summarized in Appendix 8A-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 generally finding significant associations between changes in PM and daily mortality.

From the full body of new studies reviewed and summarized in the draft CD, staff have focused on the results of U.S. and Canadian studies for PM_{10}, $PM_{2.5}$, and $PM_{10-2.5}$. The results of single-city studies are combined with those from the multi-city studies discussed above, and shown in Figures 3-4, 3-5 and 3-6, respectively. Using the same approach taken in the draft CD in presenting the NMMAPS results (Figure 3-2 above), the effect estimates are plotted against the natural log of the mortality-days product, again serving as an indicator of study precision. As

was seen in Figure 3-2, there is the expected tendency of studies to have more precise effect estimates as the mortality-days product increases. Effect estimates from single-pollutant models are presented in these figures for purposes of comparing results across studies; results of multi-pollutant models are briefly discussed below and in section 3.5.1, and discussed more fully in Appendix 8A-1 of the draft CD. Each figure includes effect estimates for both new studies and studies included in the 1996 CD and, for comparison purposes, the range of statistically significant effect estimates from the previous review is indicated in the right-hand margin. Also, for the purpose of comparing effect estimates across the studies, where reanalysis was done, the results from GAM analyses using more stringent criteria are presented. It should be recognized that the standard errors may be larger in models using GLM, but GAM analyses may be more appropriate for model fitting and effect estimate determination (CD, p. 8-196); staff notes that there was little apparent difference seen when comparing figures using GAM stringent or GLM results. 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. Staff notes that, as a subset of the full body of evidence summarized in the draft CD, the range of effect estimates seen in these figures may differ from the effect estimate ranges presented in the draft CD.

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 copollutant 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, reanalyzed in Ostro et al., 2003; Fairley, 1999, reanalyzed in Fairley, 2003; Lippmann et al., 2000, reanalyzed in Ito, 2003). On the other hand, Moolgavkar (2000a, reanalyzed in Moolgavkar, 2003) reported that the inclusion of gaseous co-pollutants resulted in large reductions in PM effect estimates in Cook, Los Angeles, and Maricopa Counties, with general loss of statistical significance.

The CD finds "nearly all of the newly reported analyses with a few exceptions continue to show statistically significant associations between short-term (24 h) PM exposures indexed by a variety of ambient PM measurements and increases in daily mortality in numerous U.S. and Canadian cities, as well as elsewhere around the world" (CD, p. 8-29). For studies conducted in the U.S. and Canada, Figure 3-4 indicates that nearly all of the associations between PM_{10} and total mortality are positive and many reach statistical significance, especially among the more precise effect estimates. Considering the results from all studies, the draft CD reports that effect estimates for associations between PM_{10} and total mortality range from 1.5 to 8.5% per 50 $\mu g/m^3$ PM_{10}.

Studies including both $PM_{2.5}$ and $PM_{10-2.5}$ are summarized in Table 8-2 of the draft CD (p. 8-52) and the effect estimates are presented in Figure 8-7 (CD, p. 8-55); studies from the U.S. as well as other countries are included. The draft CD observes, "All of the studies found positive associations between both the fine and coarse PM indices and increased mortality risk" (CD, p. 8-54). As can be seen in Figure 8-7 of the draft CD, several studies report stronger associations with $PM_{2.5}$ than for $PM_{10-2.5}$, but some new studies suggest effect estimates for $PM_{10-2.5}$ that are similar or slightly larger in size than those for $PM_{2.5}$. The draft CD also observes that there are a number of considerations to take into account in interpreting the results of studies on different PM size fractions, including differences in measurement error for the two size fractions, potential variation from city to city in constituents of each size fraction, and the limited sample sizes for some of the studies (CD, p. 8-57). Focusing on the studies with greater precision, the draft CD reports that effect estimates for total mortality with both $PM_{2.5}$ and $PM_{10-2.5}$ generally range from about 2 to 6% per 25 $\mu g/m^3$ of $PM_{2.5}$ or $PM_{10-2.5}$ (CD, p. 8-57).

In addition to those studies that included both $PM_{2.5}$ and $PM_{10-2.5}$ there are a number of new studies that evaluated associations with $PM_{2.5}$ but not $PM_{10-2.5}$. Figure 3-5 includes effect estimates for $PM_{2.5}$ and mortality from U.S. and Canadian studies, including multi-city studies. All effect estimates for $PM_{2.5}$ and total mortality are positive and, especially for those more precise effect estimates, many are statistically significant. The findings of new studies are

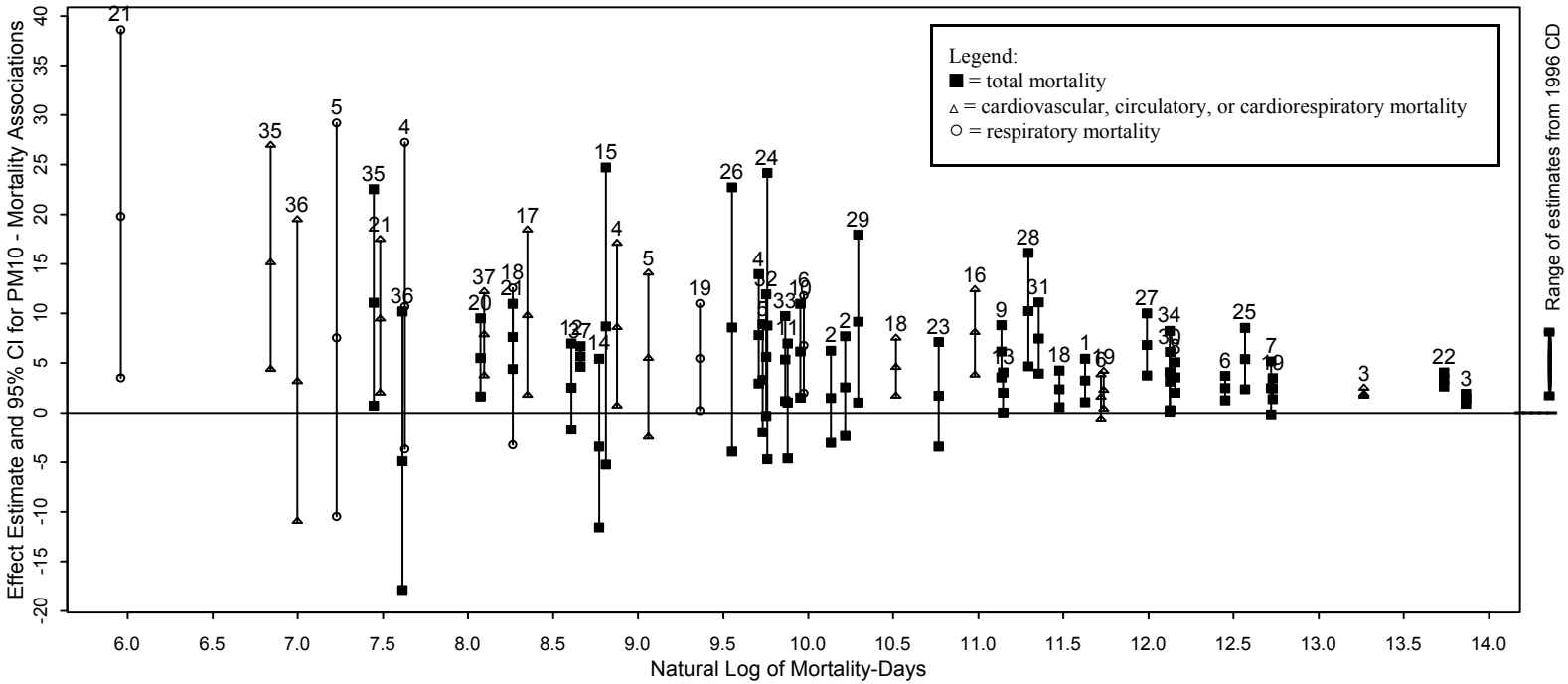


Figure 3-4. Effect estimates for PM_{10} and mortality from total, respiratory and cardiovascular causes from U.S. and Canadian cities in relation to the mortality-days product (the product of study days and the number of deaths per day -- an indicator of study precision). Study locations are identified below; multi-city studies denoted by ★. Results of GAM stringent reanalyses; studies not originally using GAM denoted by • (data in Appendix A).

1. ★ Burnett and Goldberg, 2003, 8 Canadian cities
2. Chock et al., 2000, Pittsburgh •
3. ★ Domenici, 2003, 90 U.S. cities
4. Fairley, 2003, Santa Clara
5. Ito, 2003, Detroit
6. Ito and Thurston, 1996, Chicago •
7. Kinney et al., 1995, LA •
8. ★ Klemm and Mason, 2003, Six Cities overall
9. Klemm and Mason, 2003, Boston
10. Klemm and Mason, 2003, Kinston/Harriman
11. Klemm and Mason, 2003, Portage
12. Klemm and Mason, 2003, Steubenville
13. Klemm and Mason, 2003, St. Louis
14. Klemm and Mason, 2003, Topeka
15. Klemm and Mason, 2000, Atlanta •
16. Lipfert et al., 2000a, Philadelphia •
17. Mar et al., 2003, Phoenix
18. Moolgavkar. 2003, LA County
19. Moolgavkar, 2003, Cook County
20. Ostro et al., 2000, Coachella Valley
21. Pope et al., 1992, Utah Valley •
22. ★ Schwartz, 2003b, 10 Cities overall
23. Schwartz, 2003b, Birmingham
24. Schwartz, 2003b, Canton
25. Schwartz, 2003b, Chicago
26. Schwartz, 2003b, Colorado Springs
27. Schwartz, 2003b, Detroit
28. Schwartz, 2003b, Minneapolis
29. Schwartz, 2003b, New Haven
30. Schwartz, 2003b, Pittsburgh
31. Schwartz, 2003b, Seattle
32. Schwartz, 2003b, Spokane
33. Schwartz, 1993, Birmingham •
34. Styer et al., 1995, Chicago •
35. Tsai et al., 2000, Camden •
36. Tsai et al., 2000, Elizabeth •
37. Tsai et al., 2000, Newark •

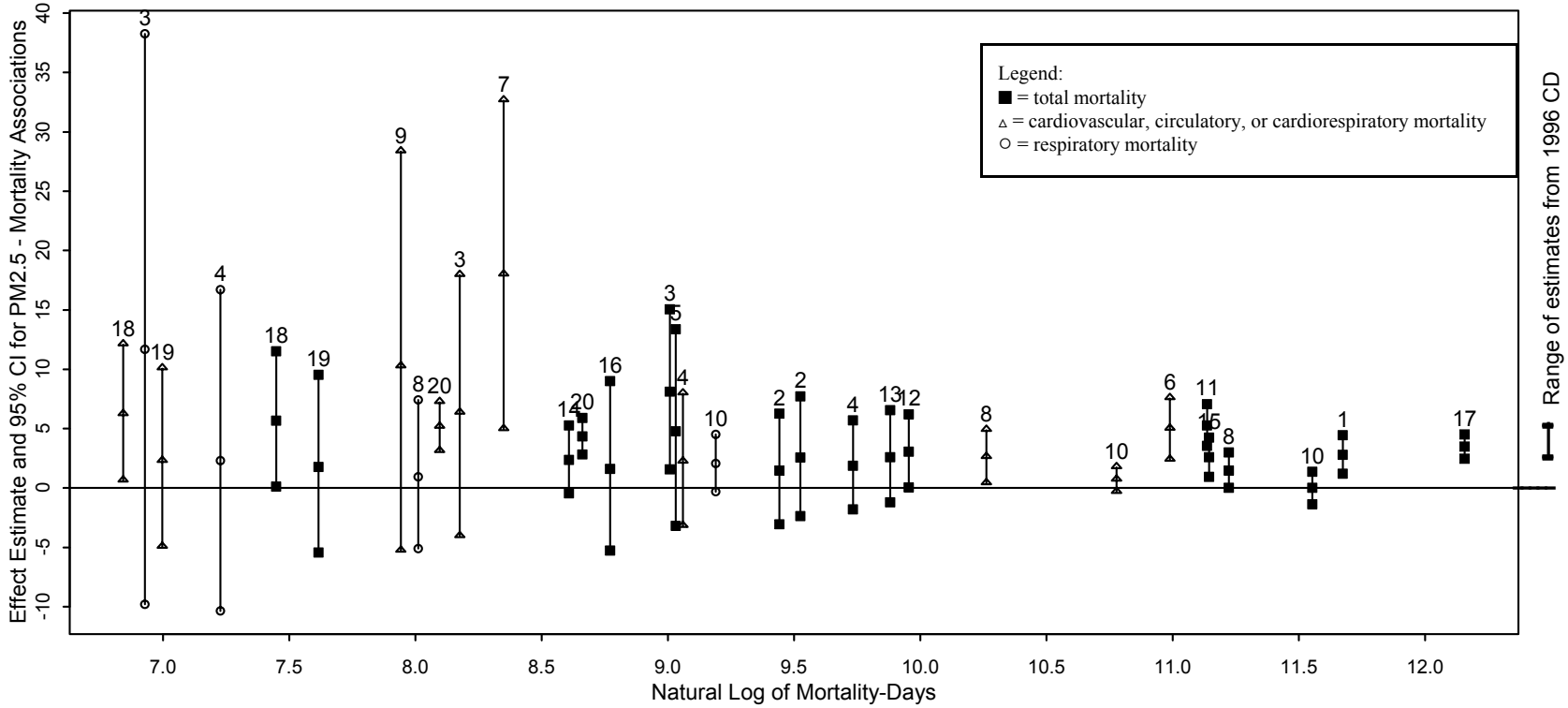


Figure 3-5. Effect estimates for $PM_{2.5}$ and mortality from total, respiratory and cardiovascular causes from U.S. and Canadian cities in relation to the mortality-days product (the product of study days and the number of deaths per day -- an indicator of study precision). Study locations are identified below; multi-city studies denoted by ★. Results of GAM stringent reanalyses; studies not originally using GAM denoted by • (data in Appendix A)

1. ★ Burnett and Goldberg, 2003, 8 Canadian cities
2. Chock et al., 2000, Pittsburgh •
3. Fairley, 2003,, Santa Clara
4. Ito, 2003, Detroit
5. Klemm and Mason, 2000, Atlanta •
6. Lipfert et al., 2000a, Philadelphia •
7. Mar et al., 2003, Phoenix
8. Moolgavkar., 2003, LA
9. Ostro et al., 2003, Coachella Valley
10. Ostro et al., 1995, So. California •
11. Schwartz, 2003a, Boston
12. Schwartz, 2003a, Kingston/ Harriman
13. Schwartz, 2003a, Portage
14. Schwartz, 2003a, Steubenville
15. Schwartz, 2003a, St. Louis
16. Schwartz, 2003a, Topeka
17. ★ Schwartz, 2003a, Six Cities overall
18. Tsai et al., 2000, Camden NJ •
19. Tsai et al., 2000, Elizabeth NJ •
20. Tsai et al., 2000, Newark NJ •

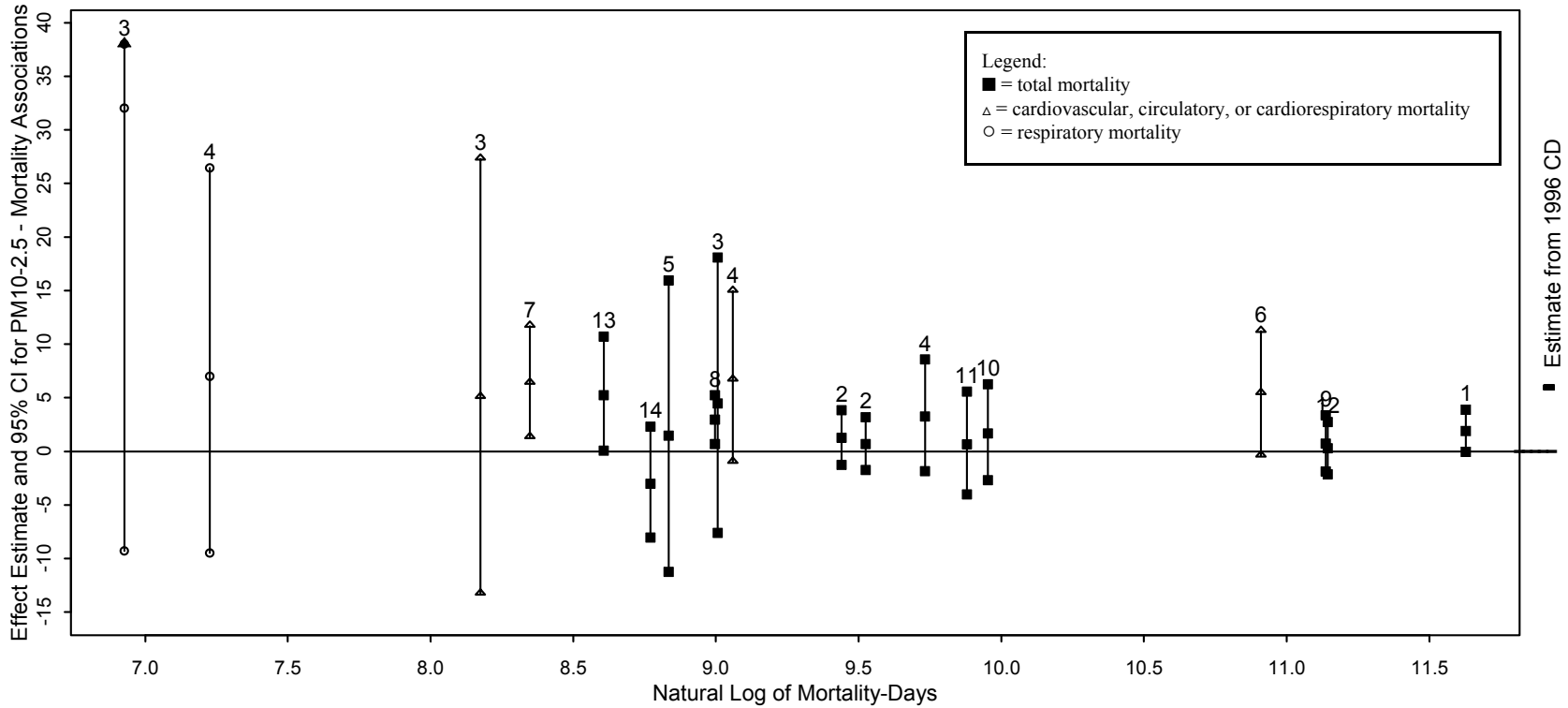


Figure 3-6. Effect estimates for $PM_{10-2.5}$ and mortality from total, respiratory and cardiovascular causes from U.S. and Canadian cities in relation to the mortality-days product (the product of study days and the number of deaths per day -- an indicator of study precision). Study locations are identified below; multi-city studies denoted by ★. Results of GAM stringent reanalyses; studies not originally using GAM denoted by • (data in Appendix A)

1. ★ Burnett and Goldberg, 2003, 8 Canadian cities
2. Chock et al., 2000, Pittsburgh •
3. Fairley, 2003, Santa Clara
4. Ito, 2003, Detroit
5. Klemm and Mason, 2000, Atlanta •
6. Lipfert et al., 2000a, Philadelphia •
7. Mar et al., 2003, Phoenix
8. Ostro et al., 2003, Coachella Valley
9. Schwartz, 2003a, Boston
10. Schwartz, 2003a, Kingston/ Harriman
11. Schwartz, 2003a, Portage
12. Schwartz, 2003a, St. Louis
13. Schwartz, 2003a, Steubenville
14. Schwartz, 2003a, Topeka

consistent with associations found between $PM_{2.5}$ and total mortality in the previous PM NAAQS review (CD, p. 8-76).

As shown in Figure 3-6, effect estimates between $PM_{10-2.5}$ and total mortality are also generally positive, though less likely to reach statistical significance. In the previous PM NAAQS review, very few studies tested associations with $PM_{10-2.5}$, with only the multi-city study using Six Cities data presenting results for $PM_{10-2.5}$ with mortality. In that study, a statistically significant association between total mortality and $PM_{10-2.5}$ was reported for only one of the cities. Some new U.S. and Canadian studies have now reported positive associations with $PM_{10-2.5}$, and some show statistically significant results (Mar et al., 2003; Ostro et al., 2003; results for total mortality not reanalyzed) but most do not. The CD concludes that, while the evidence is not as consistent, recent studies also suggest that $PM_{10-2.5}$ may also be associated with daily mortality (CD, p. 8-77).

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 draft CD concluded that this lent support to the biological plausibility of the PM associations (EPA, 1996a, p. 12-69).

Appendix A and Figures 3-4 through 3-6 include effect estimates for mortality from respiratory and cardiovascular causes, as well as for all nonaccidental causes, from multi-city and single-city studies. For example, 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 (1.6% increase per 50 $\mu g/m^3$ increase in PM_{10}) than that for deaths from all causes (1.1% increase per 50 $\mu g/m^3$ increase in PM_{10}) (GLM results, Samet et al., 2000c, reanalyzed in Domenici, 2003). In general, for respiratory and cardiovascular mortality, the U.S. and Canadian studies tend to show somewhat larger effect estimates than for total mortality associations with PM_{10} (or PM_{15}) and $PM_{2.5}$ (e.g., Gwynn et al., 2000, not reanalyzed; Ostro et al., 1999, reanalyzed in Ostro et al., 2003; Pope et al., 1999a, not reanalyzed; Fairley, 1999, reanalyzed in Fairley, 2003; Lippmann et al., 2000, reanalyzed in Ito, 2003; Mar et al., 2000, reanalyzed in Mar et al., 2003; Goldberg et

al., 2000, reanalyzed in Goldberg and Burnett, 2003; Tsai et al., 2000) (Figures 3-4 and 3-5). 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. 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 effect estimates for cardiovascular mortality tended to be greater than those for total mortality (Mar et al., 2000; Ostro et al., 2000) (both reanalyzed only the cardiovascular mortality results (Mar et al., 2003; Ostro et al., 2003)) (Figure 3-6).

A number of investigators have also tested for associations between PM and "other" or non-cardiorespiratory deaths, and generally no such associations are reported. However, in a few studies, positive, though not always statistically significant, associations were reported for the "other" mortality category. Regarding these findings, 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. 8-75).

In addition to the evidence from epidemiologic studies, new, though limited, information is available from toxicologic studies that offers insight into PM-related mortality. In some of the toxicologic studies using an animal model of cardiopulmonary disease, some animals died after exposure to ROFA, a combustion source related particle sample, though none of these studies was designed to assess lethality (CD, p. 7-45). Indicators of inflammation or cardiac arrhythmia were also measured in these studies (CD, Table 7-7a). While the applicability of effects found for this animal model to human health may be questioned, and the doses were relatively high, the findings lend plausibility to the associations with cardiorespiratory mortality reported in epidemiologic 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 morbidity effects.

In summary, considering studies conducted in the U.S. and other countries, 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. For all studies, as noted above, the draft CD observed that effect estimates for total mortality ranged generally from about 2 to 6% per 25 $\mu g/m^3$ of $PM_{2.5}$ or $PM_{10-2.5}$. Again considering all studies, the draft CD reports that cause-specific effect estimates generally range from about 2 to 20% per 25 $\mu g/m^3$ of $PM_{2.5}$ for cardiovascular or cardiorespiratory mortality and from about 2 to14% per 25 $\mu g/m^3$ of $PM_{2.5}$ for respiratory mortality (CD, p. 9-85). For $PM_{10-2.5}$, cause-specific effect estimates from all studies generally range from about 3 to 7% per 25 $\mu g/m^3$ of $PM_{10-2.5}$ for cardiovascular or cardiorespiratory mortality and from about 3 to 6% per 25 $\mu g/m^3$ of $PM_{10-2.5}$ for respiratory mortality (CD, p. 9-87)

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 several previous decades. The cross-sectional studies had generally identified associations between residence in communities with higher pollution levels and increased mortality in adult populations, but concern had been raised about the lack of information on potentially important covariates and methodological limitations (EPA, 1996a, p. 12-159).

In contrast with cross-sectional studies, prospective cohort studies include subject-specific information on potential confounders (e.g., smoking history, occupation, health history) and have been considered to provide more reliable results (EPA, 1996a, p. 13-33). In the previous review, results were presented for three recent prospective cohort studies of adult populations (i.e., the Six Cities, American Cancer Society (ACS), and California Seventh Day Adventist (AHSMOG) studies). The Six Cities study (Dockery et al., 1993) 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). The ACS study (Pope et al., 1995) reported significant associations for $PM_{2.5}$ and sulfates (a fine particle surrogate). Both the Six Cities and ACS studies reported associations with mortality from all causes and cardiorespiratory causes, with larger effect estimates for cardiorespiratory causes. The AHSMOG study did not find an association between

TSP and mortality (Abbey et al., 1991). The 1996 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 of adult mortality that are available for the current review include a comprehensive reanalysis of data from the Six Cities and ACS studies (Krewski et al., 2000), new analyses using updated data from the AHSMOG study (Abbey et al., 1999) and the ACS study (Pope et al., 2002), and a new analysis using data from a cohort of veterans (Lipfert et al., 2000b). Findings from the original Six Cities, ACS, and AHSMOG investigations together with those from new studies and reanalyses are summarized in Table 3-3. In addition, a new cross-sectional study of adult populations has been published (Lipfert and Morris, 2002) and several recent studies have suggested relationships between long-term PM exposure (e.g., months) and mortality in infants, as discussed later in this section.

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, Krewski and colleagues (2000) reported the data from the two studies to be of generally high quality, and were able to replicate the original results, confirming the original investigators' findings of associations with both total and cardiorespiratory mortality (CD, p. 8-83).

In the second phase, 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. Krewski and colleagues (2000) 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 sensitivity analyses were conducted using data for potentially susceptible subgroups, and the results did not show differences in the PM-mortality associations among most subgroups, including those defined by 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 of persons without a high school education than in one of persons with more education; Krewski and colleagues (2000) postulated that this relationship could be due to some unidentified socioeconomic effect modifier.

The original Six Cities and ACS analyses had not included assessment of potential confounding by co-pollutants. The Six Cities study had included assessment of associations between mortality and the gaseous pollutants individually (Dockery et al., 1993), and 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 (EPA, 1996a, p. 12-168). For the reanalyses, Krewski and colleagues obtained additional data on gaseous pollutant concentrations and evaluated the effects of these pollutants both 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 sulfur dioxide may be acting as a marker for other mortality-associated pollutants, and conclude "[n]onetheless, 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 (approximately 150 cities, as compared with approximately 50 cities for which there were $PM_{2.5}$ data). The resulting sulfate associations were sometimes smaller and sometimes larger than the original effect estimates. The authors concluded that this "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).

Table 3.3 Effect Estimates for Mortality per Increments in Long-term Mean Levels of Fine- and Coarse-fraction Particle Indicators from U.S. and Canadian Studies (from draft CD Table 9-11)

Study	Indicator (Increment)	Relative Risk (95% CI)	Study Concentrations * ($\mu g/m^3$)
Increased Total Mortality in Adults			
Six City[A]	$PM_{15/10}$ (20 $\mu g/m^3$)	1.18 (1.06, 1.32)	NR (18, 47)
	$PM_{2.5}$ (10 $\mu g/m^3$)	1.13 (1.04, 1.23)	NR (11, 30)
	$SO_4^=$ (15 $\mu g/m^3$)	1.54 (1.15, 2.07)	NR (5, 13)
Six City[B]	$PM_{15-2.5}$ (10 $\mu g/m^3$)	1.43 (0.83, 2.48)	
ACS Study[C] (151 U.S. SMSA)	$PM_{2.5}$ (10 $\mu g/m^3$)	1.07 (1.04, 1.10)	18** (9, 34)
	$SO_4^=$ (15 $\mu g/m^3$)	1.11 (1.06, 1.16)	11** (4, 24)
Six City Reanalysis[D]	$PM_{15/10}$ (20 $\mu g/m^3$)	1.19 (1.06, 1.34)	NR (18, 47)
	$PM_{2.5}$ (10 $\mu g/m^3$)	1.14 (1.05, 1.23)	NR (11, 30)
ACS Study Reanalysis[D]	$PM_{15/10}$ (20 $\mu g/m^3$) (dichot)	1.04 (1.01, 1.07)	59 (34, 101)
	$PM_{2.5}$ (10 $\mu g/m^3$)	1.07 (1.04, 1.10)	20 (10, 38)
	$PM_{15-2.5}$ (10 $\mu g/m^3$)	1.00 (0.99, 1.02)	7.1 (9, 42)
ACS Study Extended Analyses[E]	$PM_{2.5}$ (10 $\mu g/m^3$) (1979-83) $PM_{2.5}$ (10 $\mu g/m^3$) (1999-00) $PM_{2.5}$ (10 $\mu g/m^3$) (average)	1.04 (1.01, 1.08) 1.06 (1.02, 1.10) 1.06 (1.02, 1.11)	21 (10, 30)*** 14 (5, 20)*** 18 (±4)
Southern California[F]	PM_{10} (20 $\mu g/m^3$)	1.09 (0.99, 1.21) (males)	51 (0, 84)
	PM_{10} (30 days/year>100 $\mu g/m^3$)	1.08 (1.01, 1.16) (males)	
	PM_{10} (20 $\mu g/m^3$)	0.95 (0.87, 1.03) (females)	51 (0, 84)
	PM_{10} (30 days/year>100 $\mu g/m^3$)	0.96 (0.90, 1.02) (females)	
Southern California[H]	$PM_{2.5}$ (10 $\mu g/m^3$)	1.09 (0.98, 1.21) (males)	32 (17, 45)
	$PM_{10-2.5}$ (10 $\mu g/m^3$)	1.05 (0.92, 1.21) (males)	27 (4, 44)
Veterans Cohort[G]	$PM_{2.5}$ (10 $\mu g/m^3$) (1979-81)	0.90 (0.85, 0.95) (males)	24 (6, 42)
Increased Cardiopulmonary Mortality in Adults			
Six City[A]	$PM_{15/10}$ (20 $\mu g/m^3$)	****	NR (18, 47)
	$PM_{2.5}$ (10 $\mu g/m^3$)	1.18 (1.06, 1.32)	NR (11, 30)
Six City Reanalysis[D]	$PM_{15/10}$ (20 $\mu g/m^3$)	1.20 (1.03, 1.41)	NR (18, 47)
	$PM_{2.5}$ (10 $\mu g/m^3$)	1.19 (1.07, 1.33)	NR (11, 30)
ACS Study[C]	$PM_{2.5}$ (10 $\mu g/m^3$)	1.12 (1.07, 1.17)	18** (9, 34)
ACS Study Reanalysis[D]	$PM_{15/10}$ (20 $\mu g/m^3$) (dichot)	1.07 (1.03, 1.12)	59 (34, 101)
	$PM_{2.5}$ (10 $\mu g/m^3$)	1.12 (1.07, 1.17)	20 (10, 38)
	$PM_{15-2.5}$ (10 $\mu g/m^3$)	1.00 (0.98, 1.03)	7.1 (9, 42)

Study	Indicator (Increment)	Relative Risk (95% CI)	Study Concentrations * ($\mu g/m^3$)
ACS Study Extended Analyses[E]	$PM_{2.5}$ (10 $\mu g/m^3$) (1979-83)	1.06 (1.02, 1.10)	21 (10, 30)***
	$PM_{2.5}$ (10 $\mu g/m^3$) (1999-00)	1.08 (1.02, 1.14)	14 (5, 20)***
	$PM_{2.5}$ (10 $\mu g/m^3$) (average)	1.09 (1.03, 1.16)	18 (±4)
Southern California[F]	PM_{10} (20 $\mu g/m^3$)	1.01 (0.92, 1.10)	51 (0, 84)
Southern California[H]	$PM_{2.5}$ (10 $\mu g/m^3$)	1.23 (0.97, 1.55) (males)	32 (17, 45)
	$PM_{10-2.5}$ (10 $\mu g/m^3$)	1.20 (0.87, 1.64) (males)	27 (4, 44)
Increased Lung Cancer Mortality in Adults			
Six City[A]	$PM_{15/10}$ (20 $\mu g/m^3$)	****	NR (18, 47)
	$PM_{2.5}$ (10 $\mu g/m^3$)	1.18 (0.89, 1.57)	NR (11, 30)
Six City Reanalysis[D]	$PM_{15/10}$ (20 $\mu g/m^3$)	1.14 (0.75, 1.74)	NR (18, 47)
	$PM_{2.5}$ (10 $\mu g/m^3$)	1.21 (0.92, 1.60)	NR (11, 30)
ACS Study[C]	$PM_{2.5}$ (10 $\mu g/m^3$)	1.01 (0.91, 1.12)	18** (9, 34)
ACS Study Reanalysis[D]	$PM_{15/10}$ (20 $\mu g/m^3$) (dichot)	1.01 (0.91, 1.11)	59 (34, 101)
	$PM_{2.5}$ (10 $\mu g/m^3$)	1.01 (0.91, 1.11)	20 (10, 38)
	$PM_{15-2.5}$ (10 $\mu g/m^3$)	0.99 (0.93, 1.05)	7.1 (9, 42)
ACS Study Extended Analyses[E]	$PM_{2.5}$ (10 $\mu g/m^3$) (1979-83)	1.08 (1.01, 1.16)	21 (10, 30)***
	$PM_{2.5}$ (10 $\mu g/m^3$) (1999-00)	1.13 (1.04, 1.22)	14 (5, 20)***
	$PM_{2.5}$ (10 $\mu g/m^3$) (average)	1.14 (1.05, 1.24)	18 (±4)
Southern California[F]	PM_{10} (20 $\mu g/m^3$)	1.81 (1.14, 2.86) (males)	51 (0, 84)
Southern California[H]	$PM_{2.5}$ (10 $\mu g/m^3$)	1.39 (0.79, 2.50) (males)	32 (17, 45)
		1.26 (0.62, 2.55) (males)	27 (4, 44)

* Presented as mean (min, max), or mean (±SD).
** Median
*** Minimum and maximum values estimated from Figure 1 (Pope et al., 2002)
**** Results only for smoking category subgroups.

References:
[A] Dockery et al. (1993)
[B] EPA (1996a)
[C] Pope et al. (1995)
[D] Krewski et al. (2000)
[E] Pope et al. (2002)
[F] Abbey et al. (1999)
[G] Lipfert et al. (2000b)
[H] McDonnell et al. (2000)

In summary, the draft CD concludes that the reanalyses generally confirm the original investigators' findings of associations between mortality and long-term exposure to fine particles with the Six Cities and ACS cohort data. As shown in draft CD Table 8-5, the mortality relative risk estimates reported in the replication analysis were nearly identical to those reported in the original studies (CD, p. 8-84). 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. 8-87).

A new study included extended analyses using the ACS cohort data with additional follow-up health data and air quality data from the new fine particle monitoring network for 1999-2000 (Pope et al., 2002). As shown in Table 8-7 of the draft CD, this new study reports significant associations between long-term exposure to fine particles (using the original air quality data for 1979-1982) and premature mortality from all causes, cardiopulmonary diseases, and lung cancer (p. 8-90). Thus, the relationship persisted with the longer follow-up period for health data, and the effect estimates, while somewhat smaller than those in the original analysis, were consistent with those from the original analysis. Significant associations were also found when using the more recent air quality data, and average concentrations for all air quality data. The authors noted that the $PM_{2.5}$ concentrations from 1999-2000 were highly correlated with $PM_{2.5}$ concentrations from 1979-1983, indicating that "the rank ordering of cities by relative pollution levels remained nearly the same" (Pope et al., 2002, p. 1136). As was seen in the reanalysis study (Krewski et al., 2000), significant associations were reported for fine particles and sulfates, but not coarse fraction particles or TSP.

This extended analysis included the use of data on gaseous pollutant concentrations, more recent data on fine particle concentrations, and evaluated further the influence of other covariates (e.g., dietary intake data, occupational exposure) and model specification in the PM-mortality relationship (e.g., new methods for spatial smoothing and random effects models in the Cox proportional hazards model) (Pope et al., 2002). The investigators reported that the associations found with sulfate and fine particle concentrations were robust to the inclusion of many covariates for socioeconomic factors or personal health variables (e.g., dietary factors, alcohol consumption, body mass index). Their findings in this regard were consistent with those of Krewski et al. (2000). Pope et al. (2002) also found no significant spatial autocorrelation after controlling for fine particles. While Krewski et al. (2000) found that effect estimates change with different methods of control for spatial variability, Pope and colleagues tested new methods for modeling spatial variability, and reported that, after adjusting for spatial correlation, the

estimated $PM_{2.5}$ effects were significant and persistent for cardiopulmonary and lung cancer mortality, and were nearly significant for total mortality (CD, p. 8-96).

Other new analyses using updated data from the AHSMOG cohort included more recent air quality data for PM_{10} and estimated $PM_{2.5}$ concentrations from visibility data, along with new health information from continued follow-up of the Seventh Day Adventist cohort (Abbey et al., 1999; McDonnell et al., 2000). In contrast with the original study in which no statistically significant results were reported with TSP, a significant association was reported between total mortality and PM_{10} (number of days exceeding 100 $\mu g/m^3$) for males (CD, p. 8-98). However, no significant associations were reported for other PM_{10} indices (e.g., 20 $\mu g/m^3$ increase), for deaths from contributing respiratory causes, or for females. Additional analyses were conducted using only data from males and estimated $PM_{2.5}$ and $PM_{10-2.5}$ concentrations; larger effect 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 8-11).

A new long-term exposure study uses data from a cohort of up to 70,000 military men assembled by the Veterans Administration in the 1970s (Lipfert et al., 2000b). The cohort was male, middle-aged (mean age 51 years), included a higher proportion of African-Americans and current or former smokers than the general population, and the participants were selected on the basis of having mild or moderate hypertension (CD, p. 8-101). As such, this cohort represents an "at-risk" population group that is markedly distinct from the general U.S. population.

The authors report that, when methods consistent with those in the other prospective cohort studies were used, similar results were found, stating "[t]he single-mortality-period responses without ecological variables are qualitatively similar to what has been reported before ($SO_4^{=} \geq PM_{2.5} > PM_{15}$)" (CD, p. 8-103). In analyses using shorter PM exposure and mortality time periods, 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. Ecological variables included education, income, population race proportions, altitude, poverty rate, and number of heating-degree days. 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. One hypothesis offered by the authors

is that the "relatively high fraction of mortality within this cohort may have depleted it of susceptible individuals in the later periods of follow-up" (CD, p. 8-103).

Three of the prospective cohort studies described above (Six Cities, ACS, AHSMOG) also examined long-term exposure to PM in relation to lung cancer mortality. In the results available during the previous review, none of the these studies reported a significant association between long-term exposure to fine particles and lung cancer mortality, although the ACS study reported a significant association between lung cancer mortality and sulfates (EPA, 1996b, p. V-17). The reanalysis study confirmed these findings for the Six Cities and ACS studies (Krewski et al., 2000).

One new effect reported in the extended analysis of the ACS study were statistically significant associations between fine particle and sulfate concentrations and lung cancer mortality. Pope et al. (2002) report an 8% increased risk of lung cancer mortality per 10 $\mu g/m^3$ change in $PM_{2.5}$, and this effect estimate is little changed and remains significant with adjustment for covariates, random effects modeling and spatial smoothing methods (CD, Figure 8-10). In new analyses using updated data from the AHSMOG cohort, significant associations were reported between long-term PM_{10} exposure and lung cancer mortality for males, but not females and some associations were also reported with other gaseous pollutants (Beeson et al., 1998). In contrast, in the original analyses (Pope et al., 1995; Abbey et al., 1991) no significant associations were reported with lung cancer mortality. The findings were based on a small number of lung cancer deaths in the cohort, and the effect estimates were quite variable (CD, p. 8-98). Further analysis using data for males and estimated $PM_{2.5}$ and $PM_{10-2.5}$ concentrations reported no statistically significant associations with lung cancer mortality for either $PM_{2.5}$ or $PM_{10-2.5}$ (McDonnell et al., 2000; CD, p. 8-101).

The epidemiologic findings of associations between fine particles and lung cancer mortality are supported by the results of a few recent toxicologic studies that have examined the mutagenic potential of ambient particles and "have shown some degree of evidence that appears to support the biologic plausibility of the long-term lung cancer effects," though it is noted that the studies generally lack adequate information on dose (CD, p. 7-69). In addition, the Health Assessment Document for diesel engine exhaust concludes that diesel engine exhaust, one

source of PM emissions, is a likely human carcinogen (EPA, 2002). On the results of the new epidemiologic studies, the draft CD concluded "[o]verall, these new cohort studies confirm and strengthen the published older ecological and case-control evidence indicating that living in an area that has experienced higher PM exposures can cause a significant increase in RR of lung cancer incidence and associated mortality" (CD, p. 8-228).

The draft CD relies heavily on the results of the ACS and Six Cities study, the reanalysis of these studies, and the extended ACS study in evaluating the results of the prospective cohort studies. The draft CD concludes that the "lack of consistent findings in the AHSMOG study and negative results of the VA study, do not negate the findings of the Six Cities and ACS studies" (CD, p. 8-105). The Six Cities and ACS studies (and reanalyses and new analyses) included measured PM data (in contrast with AHSMOG PM estimates based on TSP or visibility measurements), have study populations more similar to the general population than the VA study cohort, and have been validated through an exhaustive reanalysis (CD, p. 8-108 and 8-109). The draft CD concludes that the recent publications "collectively appear to confirm earlier cross-sectional study indications that the fine mass component of PM_{10} (and usually especially its sulfate constituent) are more strongly correlated with mortality than is the coarse $PM_{10-2.5}$ component" (CD, p. 8-109). The effect estimates for deaths from all causes fall in a range of 4 to 14% increased risk per 10 $\mu g/m^3$ increase in $PM_{2.5}$, with associations for cardiopulmonary mortality (6 to 19% increased risk per 10 $\mu g/m^3$ increase in $PM_{2.5}$) and lung cancer (1 to 39% increased risk per 10 $\mu g/m^3$ increase in $PM_{2.5}$) being generally larger than those for total mortality (Table 3-3).

In addition to the prospective cohort studies, cross-sectional studies have provided information on the relationship between long-term PM exposure and adverse health effects, though emphasis has been placed on the findings of cohort studies due to the availability of individual health data on study participants. One new U.S. cross-sectional study has been conducted that uses pollution and mortality data from five time periods (Lipfert and Morris, 2002). Significant associations were reported for all pollutants considered, though the associations varied by age group and pollutant. The authors emphasized their findings of temporal changes in the pollution-mortality association; there was a trend toward reduced effects

when using more recent data sets, though the associations found using $PM_{2.5}$ data from the new fine particle monitoring network were somewhat larger than those using older data.

One additional new analysis of the Six Cities data has evaluated mortality risk with different estimates of long-term $PM_{2.5}$ exposure. The original study (Dockery et al., 1993) averaged PM concentrations over a period of years to represent long-term PM exposure estimates, while the new analysis includes PM averages over a range of time periods, such as 2 or 3-5 years preceding the individual's death (Villeneuve et al., 2002). The authors reported that effect estimates for mortality were lower with time-dependent $PM_{2.5}$ exposure indicators (e.g., 2 years before individual's death), than with the longer-term average concentrations. They postulate that this is likely due to the "influence of city-specific variations in mortality rates and decreasing levels of air pollution that occurred during follow-up" (CD, p. 8-88). This might be expected, if the most polluted cities had the greatest decline in pollutant levels as controls were applied (CD, p. 8-85). The authors observe that the fixed average concentration window may be more representative of cumulative exposures and thus a more important predictor of mortality than a shorter time period just preceding death (Villeneuva et al., 2002, p. 574).

Summary. Positive, statistically significant associations between mortality from total or cardiorespiratory causes and long-term exposure to particles, especially to fine particles, were reported in the original Six Cities and ACS studies. These results were confirmed in extensive reanalyses and new analyses using updated health and air quality data for the ACS study. In considering these results, as well as the other inconsistent and negative results discussed above, the draft CD concludes that long-term $PM_{2.5}$ exposure durations are likely associated with serious human health effects. (CD, p. 8-277).

3.3.1.3 Infant Mortality

In addition to findings of the prospective cohort studies for adult populations, several new studies have also reported associations between mortality in infants and long-term pollutant concentrations. In these studies, the exposure periods were one month or more during gestation or around the time of birth. Each of the studies reviewed in the draft CD (section 8.2.3.4) reported significant associations between infant mortality and PM exposure. One study, using data from 80 U.S. cities, 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). Lipfert et al. (2000c) conducted a similar analysis using annual average PM_{10} to represent the infants' pollution exposure levels (instead of the 2-month postnatal average), and reported associations similar to those in Woodruff et al. (1997), but also reported that geographic gradients in pollution levels and infant mortality rates make interpreting these results difficult (CD, p. 8-114). In addition, Chay and Greenstone (2001a,b) linked reductions in TSP concentrations with reduced infant mortality in the U.S. (CD, p. 115). Studies conducted in the Czech Republic and Mexico City also reported associations between PM exposure and infant mortality. The draft CD concludes that the findings from this group of studies are "suggestive of a causal relationship between PM exposure and infant mortality" (CD, p. 8-115).

3.3.1.4 Mortality Displacement and Life-Shortening

The 1996 CD and Staff Paper discussed the issue of mortality displacement, or whether the associations reported between mortality and short-term exposure 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 addition, the draft CD assessed potential life-shortening that may occur with long-term exposure to PM, and 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. While there was evidence from the analysis done in Boston 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, 2000c, reanalyzed in Schwartz, 2003). 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 this group of studies "suggest that the extent of harvesting, if any, is not a matter of only a few days" (CD, p. 8-273).

In contrast, two recent studies analyzed data from Chicago and Philadelphia (Smith et al., 1999; Murray and Nelson, 2000, respectively) using methods that included estimation of the size of the "frail" population, and the effect of air quality changes on the life expectancy of those in that population. Findings from both studies "suggest that the frail population is very small and its lifetime short, such that PM or any external stress cannot have more than a few days of life-shortening impacts" (CD, p. 8-273). The draft CD notes that this may be an inherent limitation of the model used in these studies, since it was assumed that mortality was occurring only in the fairly small frail population and PM exposure can also be associated with the development of underlying health problems, thus increasing the size of the frail population (CD, p. 9-138). Consistency is apparent in these studies when comparing results within similar models, but not across the different types of models. Overall, in considering the results of this new body of research, the draft CD concludes that "there appears to be no strong evidence to suggest that PM is shortening life by a few days" (CD, p. 8-273).

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 loss of population life expectancy associated with long-term PM exposure may be even greater than Brunekreef's (1997) estimate (CD, p. 8-274).

3.3.2 Indices of Morbidity

As noted in the 1996 PM Staff Paper, given the statistically significant positive associations between community PM concentrations and mortality, it is reasonable to anticipate that comparable epidemiologic 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 epidemiologic 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 epidemiologic studies have expanded the range of morbidity indices 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 epidemiologic 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 the latter indices in the draft CD.

Recent toxicological and controlled human exposure studies have provided new evidence on morbidity effects that suggests linkages with effects reported in epidemiologic studies. Effects related to some new endpoints measured in the recent epidemiologic studies, such as heart rate variability, were first reported in animal toxicologic studies. Some toxicologic studies have used ambient PM samples from areas in which epidemiologic 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 themselves as separate indices of morbidity.

3.3.2.1 Hospital Admissions and 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 epidemiologic 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. The 1996 CD included some limited information on the relationship between PM_{10} and cardiovascular admissions, but while few in number, the studies did report significant associations for these effects. 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. The 1996 CD included results from only one study where $PM_{2.5}$ and $PM_{10-2.5}$ data were available, and positive associations with total respiratory admissions/visits were reported for both; statistically significant associations were reported with fine particles or fine particle components, and these associations were larger and less influenced by copollutant confounding than those with $PM_{10-2.5}$ (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 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 8-17, 8-19, 8-20, 8-21, and 8-22 (with further descriptive information about the studies in Tables 8B-1 and 8B-2), 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. In general, the recent studies provide evidence that support previously identified associations between PM and admissions/visits for respiratory and cardiovascular diseases (CD pp. 8-136 and 8-167).

As for mortality, staff have focused on the findings from U.S. and Canadian studies for associations with PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$, and the results are presented in Figures 3-7, 3-8 and 3-9, respectively. In these figures, effect estimates are presented by general respiratory or cardiovascular effects categories, separated into more specific subcategories in cases where available (e.g., COPD, asthma). 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 effect estimates from the only study on respiratory admissions/visits available in the 1996 CD are indicated in the right-hand margins in Figures 3-7 and 3-8.

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; reanalyzed in Zanobetti and Schwartz, 2003), with somewhat larger effect estimates when a distributed lag approach was used (Zanobetti et al., 2000; reanalyzed in Zanobetti and Schwartz, 2003). Increases of 5% in hospital admissions for cardiovascular disease and 8% and 6% in hospital admissions for COPD or pneumonia (respectively) per 50 $\mu g/m^3$ increase in PM_{10} were reported (CD Tables 8-16 and 8-17) (in GLM models using penalized splines). 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 relationships 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 PM_{10}-copollutant coefficients (for O_3 or SO_2) and PM_{10}-respiratory admissions associations, or between the PM_{10}-copollutant coefficients (for CO, O_3 or SO_2) and PM_{10}-cardiovascular admissions associations,

indicating that confounding by co-pollutants is unlikely (Samet et al., 2000b; reanalyzed in Zanobetti and Schwartz, 2003).

These data were also used to explore potential factors that might result in variation in effect estimates across cities. The authors reported that PM_{10}-admissions effect estimates were smaller in cities where air conditioning use was greater, which is an indicator of heterogeneity of exposure rather than heterogeneity in biological effects. In addition, there was evidence of larger effect size with greater percentage of PM_{10} emissions from highway vehicles in an area, suggesting the potential importance of PM from motor vehicles in these health associations (Janssen et al., 2001; reanalyzed in Zanobetti and Schwartz, 2003)..

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); this study used GAM in the original analyses and results of reanalyses are not available.

In an analysis of data from 8 European cities from the APHEA study, associations between PM_{10} and admissions/visits for all respiratory diseases, asthma or COPD were all positive, though not always statistically significant. The overall effect estimates were 4.5% per 50 $\mu g/m^3$ increase in PM_{10} for hospital admissions for all respiratory diseases, 5.1% per 50 $\mu g/m^3$ increase in PM_{10} for COPD admissions and 6.1% per 50 $\mu g/m^3$ increase in PM_{10} for asthma hospital admissions (Atkinson et al., 2001). Le Tertre and colleagues (2002; reanalyzed in Le Tertre et al., 2003) reported a 2.5% increase in risk of cardiac hospital admissions per 50 $\mu g/m^3$ increase in PM_{10}. Several previous APHEA publications (summarized in Table 8B-2 of the draft CD) have reported associations between hospital admissions for respiratory disease or asthma and PM measurements from a variety of methods (e.g., suspended particles, black smoke). These findings are generally consistent with those from U.S. multi-city studies.

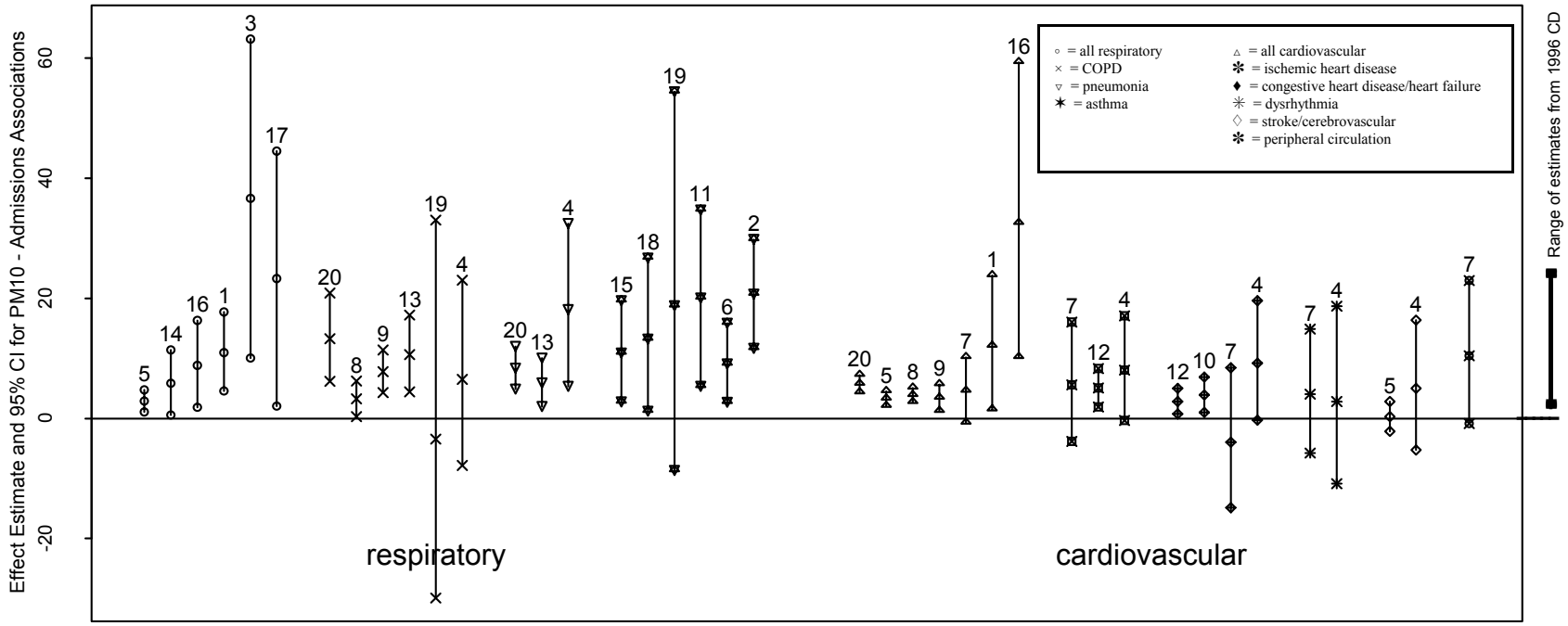


Figure 3-7. Effect estimates for PM_{10} and hospital admissions, emergency room visits (denoted ♢) or physicians office visits (denoted ⊙) for various respiratory and cardiovascular diseases from U.S. and Canadian studies. Study locations are identified below; multi-city studies denoted by ★. Results of GAM stringent reanalyses; studies not using GAM originally denoted • (data in Appendix A)

1. Burnett et al., 1997, Toronto •
2. Choudbury et al., 1997, Anchorage ⊙ •
3. Delfino et al., 1997, Montreal ♢ •
4. Ito, 2003, Detroit
5. Linn et al., 2000, LA •
6. Lipsett et al., 1997, Santa Clara ♢ •
7. Metzger et al., 2003, Atlanta ♢ •
8. Moolgavkar, 2003, Cook County •
9. Moolgavkar, 2003, LA County •
10. Morris and Naumova, 1998, Chicago •
11. Nauenberg and Basu, 1999, LA •
12. Schwartz and Morris, 1995, Detroit •
13. Schwartz et al., 1996, Cleveland •
14. Schwartz, 1994, Detroit •
15. Sheppard, 2003, Seattle
16. Stieb et al., 2000, St. John ♢ •
17. Thurston et al., 1994 Toronto •
18. Tolbert et al., 2000b, Atlanta ♢ •
19. Tolbert et al., 2000a, Atlanta ♢ •
20. ★ Zanobetti and Schwartz, 2003, 14 U.S. cities

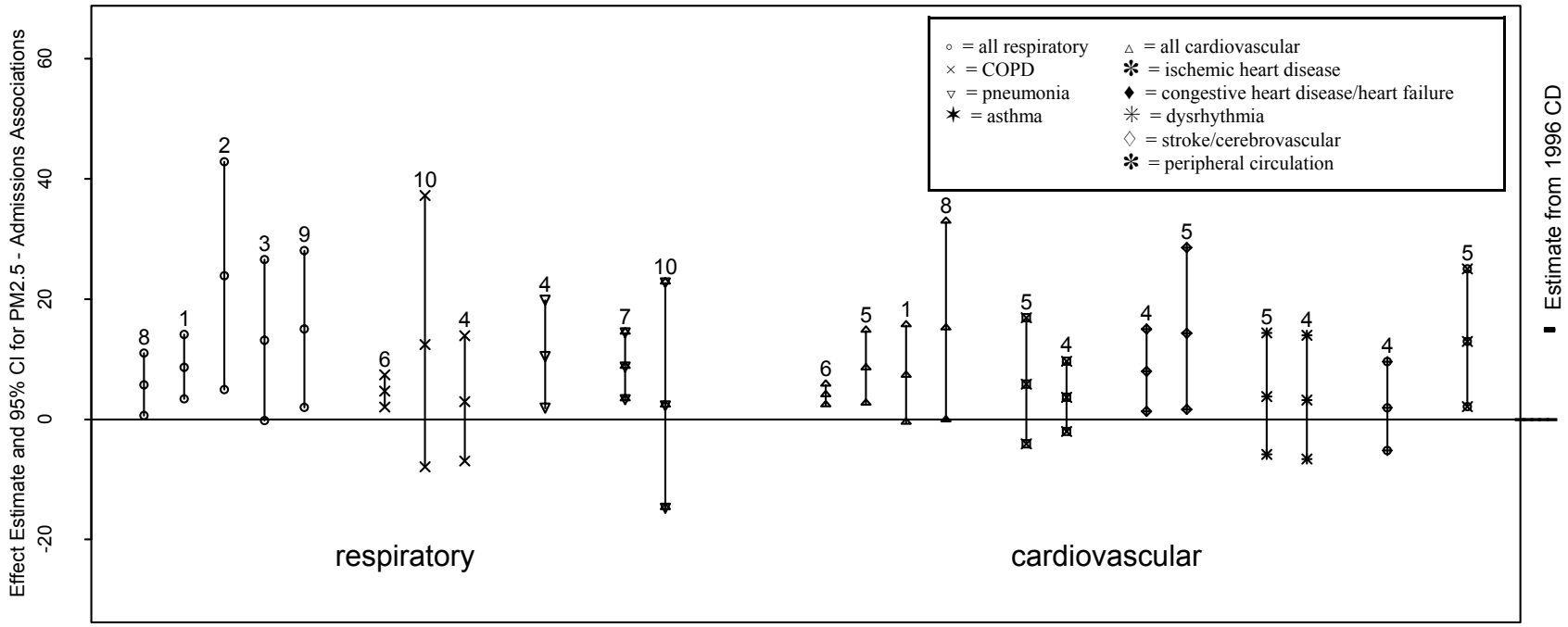


Figure 3-8. Effect estimates for $PM_{2.5}$ and hospital admissions or emergency room visits (denoted ◊) for various respiratory and cardiovascular diseases from U.S. and Canadian studies. Study locations are identified below. Results of GAM stringent reanalyses; studies not using GAM originally denoted • (data in Appendix A)

1. Burnett et al., 1997, Toronto •
2. Delfino et al., 1997, Montreal ◊ •
3. Delfino et al., 1998, Montreal ◊ •
4. Ito, 2003, Detroit
5. Metzger et al., 2003, Atlanta ◊ •
6. Moolgavkar, 2003, LA
7 . Sheppard, 2003, Seattle
8. Stieb et al., 2000, St. John ◊ •
9. Thurston et al., 1994, Toronto •
10. Tolbert et al., 2000a, Atlanta ◊ •

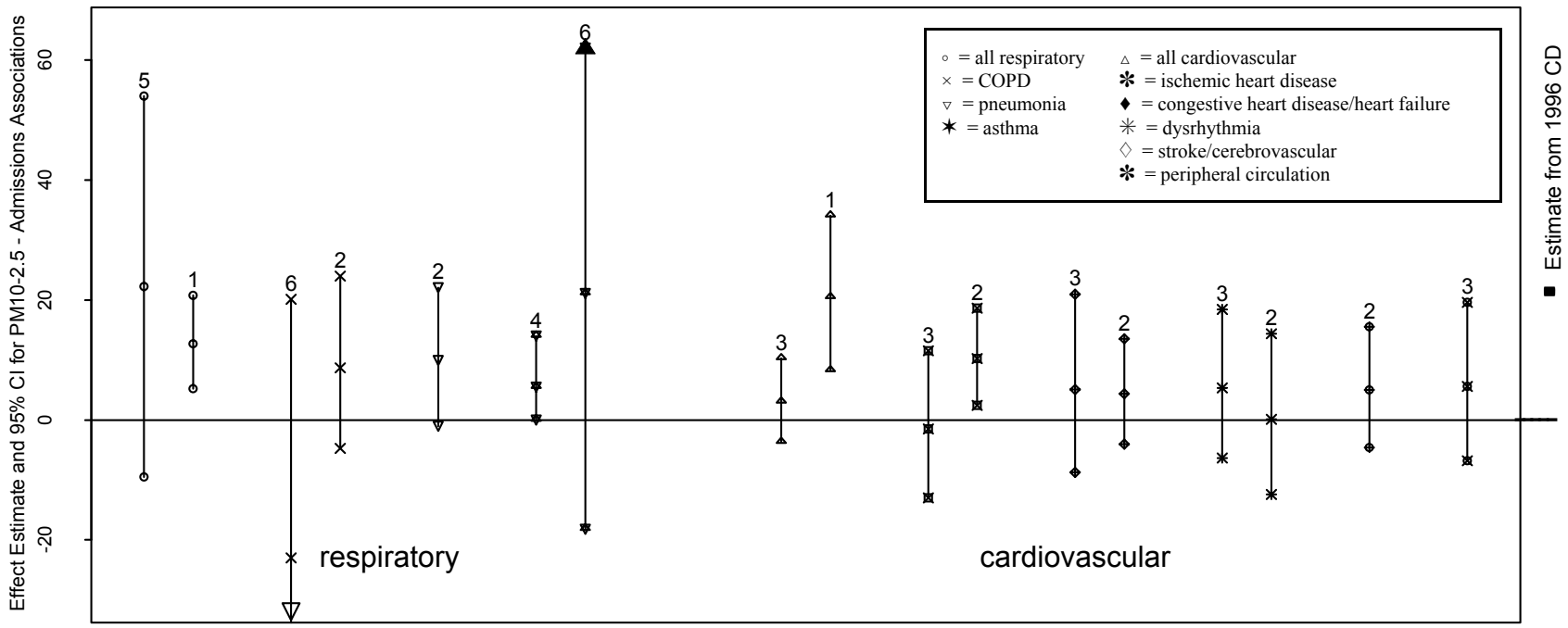


Figure 3-9. Effect estimates for $PM_{10-2.5}$ and hospital admissions or emergency room visits (denoted ◊) for various respiratory and cardiovascular diseases from U.S. and Canadian studies. Results of GAM stringent reanalyses; studies not using GAM originally denoted • (data in Appendix A)

1. Burnett et al., 1997, Toronto •
2. Ito, 2003, Detroit
3. Metzger et al., 2003, Atlanta ◊ •
4. Sheppard, 2003, Seattle
5. Thurston et al., 1994, Toronto •
6. Tolbert et al., 2000a, Atlanta ◊ •

Results for U.S. and Canadian PM_{10} studies are shown in Figure 3-7, where it can be seen that nearly all associations between PM_{10} and admissions/visits for respiratory or cardiovascular causes are positive and many are statistically significant, especially those with greater precision. These results are generally consistent with findings of studies available during the previous review.

Less evidence was available on fine particles during the previous review, but the studies that included fine particles or fine particle indicators consistently reported statistically significant associations with hospital admissions for respiratory diseases (EPA, 1996b, Table V-12, p. V-60b). Recent studies have also reported significant associations between $PM_{2.5}$ and admissions/visits for respiratory diseases (Figure 3-8). In Figure 3-8, it can be seen that associations between $PM_{2.5}$ and hospitalization or emergency room visits for the general category of respiratory diseases are all positive and statistically significant, while the results for individual disease categories (COPD, pneumonia, and asthma) are less consistent, perhaps due to smaller sample sizes for the specific categories. Associations with the general category of cardiovascular diseases are also all positive and statistically significant or nearly so, but again the results for specific diseases (ischemic heart disease, dysrhythmia, congestive heart disease or heart failure, and stroke) are positive but often not statistically significant.

In the previous PM NAAQS review, only one hospital admissions study had included coarse-fraction particles, and it did not report significant associations with hospitalization for respiratory disease (Thurston et al., 1994). However, several new studies show significant associations between $PM_{10-2.5}$ and admissions/visits for both respiratory and cardiovascular diseases (Figure 3-9). As shown in Figure 3-9, the associations reported between $PM_{10-2.5}$ are generally but not always positive, and less often statistically significant than the associations reported with $PM_{2.5}$. In the previous review. staff recognized that information about the effects of coarse-fraction particles can also come from studies linking health effects with PM_{10} in areas where coarse-fraction particles are predominant, and evidence available at that time suggested that aggravation of asthma and respiratory infections and symptoms were associated with daily or episodic increases in PM_{10} dominated by coarse-fraction particles (62 FR 38677). Recent studies conducted in coarse fraction-dominated areas such as Las Vegas, Tucson, Utah Valley, Phoenix, Southern California, and Anchorage have also reported associations between PM_{10} and

adverse health effects. The effects included in these studies include associations between PM_{10} and medical visits for asthma in Anchorage, AK (Choudhury et al., 1997) and hospital admissions for COPD in Las Vegas (Chen et al., 2000) and for cardiovascular disease in Tucson (Schwartz, 1997) (the last two studies were analyzed using GAM and were not reanalyzed). However, in Maricopa County, AZ (Phoenix), Moolgavkar (2000 a,b,c; 2003) did not report significant associations between PM_{10} and hospital admissions for COPD or cardiovascular diseases. Overall, the recent studies continue to show associations between PM_{10} and respiratory morbidity in areas where coarse-fraction particles predominate, and there is evidence suggestive of cardiovascular morbidity as well.

Figures 3-7 through 3-9 present effect estimates from single-pollutant models. As was found for mortality studies, the multi-city analyses of hospital admissions have not found evidence of significant confounding by copollutant 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, several studies have indicated that O_3 is associated with increased admission/visits for respiratory diseases, such as asthma, as has been summarized in the *Air Quality Criteria for Ozone* (EPA, 1996c). A number of the studies included in Tables 8-19 and 8-22 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, reanalyzed in Ito, 2003; Gwynn et al., 2000, not reanalyzed; Burnett et al., 1997) and less evidence was found for potential confounding by other gaseous pollutants (results discussed in more detail in Table 8B-2 of the draft CD). In considering studies of cardiovascular admissions/visits, the draft CD focused on CO as a copollutant 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. 8-130). In studies of cardiovascular and chronic respiratory disease

admissions/visits, Moolgavkar (2000b,c, reanalyzed in Moolgavkar, 2003) reports that associations with PM_{10} and $PM_{2.5}$ 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 draft 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 and/or gaseous pollutant (e.g., CO) having independent effects on cardiovascular function" (CD, p. 8-147).

Summary. PM is associated with admissions/visits for respiratory and cardiovascular diseases and specific disease categories, including ischemic heart disease, asthma, COPD, and pneumonia; the findings are generally consistent with those reported in the 1996 CD. Positive, often statistically significant associations for both respiratory and cardiovascular admissions/visits are seen with PM_{10} and $PM_{2.5}$, and positive, but less frequently statistically significant, associations are found with $PM_{10-2.5}$. As is also the case for mortality studies, U.S. multi-city studies (Samet et al., 2000a,b, reanalyzed in Domenici, 2003; Schwartz, 1999, not reanalyzed) likely provide the most precise estimates for relationships of U.S. ambient PM_{10} exposure to increased risk for hospitalization, and in these studies increases of 5% in hospital admissions for cardiovascular disease and 8% and 6% in hospital admissions for COPD or pneumonia (respectively) per 50 $\mu g/m^3$ increase in PM_{10} were reported (CD Table 8-18, Table 8-19). As stated previously, in single-city studies, effect estimates for cardiovascular admissions generally range from about 1% to 10% per 25 $\mu g/m^3$ $PM_{2.5}$ or $PM_{10-2.5}$ (CD, p. 8-231), and effect estimates for respiratory admissions generally range from about 5% to 25% per 25 $\mu g/m^3$ $PM_{2.5}$ or $PM_{10-2.5}$. Taken together, the findings of new studies and those reviewed in the 1996 CD offer reasonably consistent evidence for associations between ambient PM concentrations, across PM indicators, and admissions/visits to the hospital or emergency room for respiratory or cardiovascular diseases.

3.3.2.2 Effects on the Respiratory System

Evidence available in the previous review suggested associations between PM exposure and effects on the respiratory system 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 epidemiologic 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, toxicologic studies using particles such as acid aerosols, ROFA 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 epidemiologic studies of lung function or symptoms and new morbidity indicators such as physician visits, for both short-term and long-term exposures to PM. Also, recent toxicological or controlled human exposure studies have used exposures to ambient PM (e.g., inhalation exposures to CAPs or intratracheal instillation of ambient PM samples) to assess responses such as lung injury or inflammation. These studies provide additional new evidence linking PM with respiratory effects. Among the many new epidemiologic 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.

3.3.2.2.1 Acute Respiratory Effects

Epidemiologic Studies. Among the new epidemiologic studies are several using medical visits for respiratory illness as a measure of health effects, including Choudhury et al (1997), in Anchorage AK (shown in Figure 3-7) and a number of studies conducted outside the U.S. or

Canada (CD, p. 8-164). These studies have evaluated effects of pollutant exposure on visits to physicians' offices or doctors' visits to patients. Visits for asthma were significantly increased with PM exposure in children and people of all ages, and significant associations were found with visits for lower respiratory diseases in children (CD, p. 8-164).

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. 8-165). These studies find associations that range widely up to 60% increases in medical visits with a 50 $\mu g/m^3$ change in PM_{10} (CD Table 8-22). The results of these studies offer a link between the more severe endpoints, such as increased mortality and hospital admissions or emergency room visits for respiratory diseases, and moderate effects such as respiratory symptoms and decreased lung function. These new studies also indicate the potentially more widespread public health impact of less severe respiratory health endpoints (CD, p. 8-168).

New epidemiologic studies on PM-related effects on respiratory symptoms or lung function are summarized in draft CD Tables 8-23 through 8-33; the studies are grouped by health status of the study subjects (asthmatic or nonasthmatic), type of effects (e.g., symptoms, lung function changes) 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. 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.

For asthmatic subjects, associations were reported between PM_{10} or $PM_{2.5}$ decreases in lung function measures (e.g., decreased peak expiratory flow rate); some but not all of the associations reached statistical significance. In addition, positive associations were reported between PM_{10} or $PM_{2.5}$ and one or more of a range of respiratory symptoms (e.g., cough, wheeze, shortness of breath) but the findings were less consistent than those for lung function (CD, p. 8-170). In studies of nonasthmatic subjects, inconsistent results were reported for changes in lung function, while studies generally reported increases in respiratory symptoms that were often not statistically significant. Generally similar results were found for both PM_{10} or $PM_{2.5}$ (CD, p. 8-180).

Few studies have included both $PM_{2.5}$ and $PM_{10-2.5}$ data. In those studies, the findings suggest roles for both fine- and coarse-fraction PM in reduced lung function and increased respiratory symptoms (CD, p. 9-111). 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.

The newly available epidemiologic studies include some that used ambient PM concentrations averaged over 1- or 8-hours, in addition to 24-hour averages. Two panel studies of symptoms in asthmatic subjects are summarized in the CD, and one reported larger effect estimates for 1- or 8-hour concentrations than for 24-hour PM_{10} concentrations (Delfino et al., 1998) while the other reported larger effect estimates for 24-hour PM_{10} concentrations (Ostro et al., 2001) (CD, p. 8-179).

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 but little evidence was reported for associations between lung function and short-term PM exposure in nonasthmatic subjects.

Laboratory Studies. Key toxicologic or controlled human exposure studies summarized in the draft CD include: (1) exposures of human volunteers in a clinical setting to CAPs; (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 responses and other indicators of lung injury. As has often been the case, many of the new studies have used high doses (e.g., doses in the hundreds of μg to mg), but some new CAPs studies have reported effects with doses that are closer to the range of ambient PM concentrations.

Numerous studies have reported inflammatory changes with exposure to particles, including changes in levels or increased release of cytokines, or chemicals released as part of the inflammatory process (e.g., interleukins such as IL-8) (CD Tables 7-1, 7-2 and 7-3). A variety of particles have been used in these studies, with many involving exposure to ROFA or other combustion-related particles, but some using inhalation of CAPs or instillation of ambient 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). In addition, Ghio and Devlin (2001) exposed human volunteers by intratracheal instillation to ambient PM that had been collected in Utah Valley before, during, and after the time period when a major local PM source, a steel mill had been shut down. A greater inflammatory response was found with exposure to particles collected while the plant was operating than during the period when the steel mill was closed. Similar results were reported in animal toxicologic studies using PM from Utah Valley filters (Dye et al., 2001; Molinelli et al., 2002). The draft CD concludes that these results indicate that the pulmonary effects observed after experimental exposure of humans to the Utah Valley PM are in good accord with epidemiologic reports of adverse respiratory health effects in Utah Valley residents (CD, p. 7-18). The types of effects reported included increases in neutrophils, eosinophils or alveolar macrophages (either total number or percent) and expression of cytokines such as interleukin-6 (CD, Tables 7-2 and 7-3).

A number of animal studies have shown that exposure to diesel exhaust particles can increase the production or release of inflammatory cells, such as eosinophils, and may

exacerbate the allergic response. Controlled exposures of humans to diesel exhaust particles also have resulted in increases in inflammatory cells indicative of enhanced response to allergens. Together, the human and animal studies provide evidence that particle exposures can produce inflammatory changes in the respiratory system (CD, p. 7-120).

Animal studies also have reported evidence of general lung injury, including increased protein levels in lung fluids with exposure to ambient particles (CD Table 7-2) or combustion-related particles such as ROFA (CD, Table 7-3). One general cause of lung cell injury may be 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 7-10). As discussed previously, some toxicological studies have used ambient particles collected in Utah Valley over a time period that included closure of a major PM source, and these studies have reported evidence of general lung injury as well as inflammation (CD, pp. 7-17 and 7-18). Several *in vitro* studies have also 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.

The draft CD concludes that studies using "intratracheal instillation of ambient PM obtained from specific ambient sources have shown clearly that PM can cause lung inflammation and injury" (CD, p. 7-16). Findings of inflammation and lung injury are generally consistent with epidemiologic results showing increases in respiratory symptoms or exacerbation of respiratory diseases. 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).

Summary. The recent epidemiologic findings are consistent with those of the previous review in showing associations with both respiratory symptom incidence and decreased lung function. For asthmatic subjects, PM_{10} or $PM_{2.5}$ were associated with small decreases in lung function and increases in respiratory symptoms, though the associations were not always statistically significant. 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 toxicologic studies where higher doses were used, the draft CD concludes 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 epidemiologic studies that report increased respiratory morbidity and mortality associated with PM in many different geographical areas and climates." (CD, p. 7-19).

3.3.2.2.2 Chronic Respiratory Effects

In the 1996 CD, only a few epidemiologic studies had assessed associations between long-term PM exposure and lung function changes or respiratory symptoms. Among U.S. and Canadian studies, studies using data from the Six Cities and 24-Cities cohorts suggested associations between fine particles and find particle indicators (e.g., acid aerosols) and chronic bronchitis and decreased lung function in children (CD, p. 6-205). For adults, the 1996 CD summarized the results of 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). Overall, 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 $PM_{10-2.5}$, PM_{10}, PM_{15} or TSP (EPA, 1996b, Table V-13).

Several new epidemiologic 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 toxicologic or controlled human exposure studies regarding long-term effects of PM exposure. The new U.S. epidemiologic 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). Results for the new studies, together with the results of the Six Cities and 24-Cities studies, are presented in Table 3-4.

As seen in Table 3-4, initial analysis of data from the 12 Southern California Communities children's cohort show significant associations between long-term exposure to PM_{10} and incidence of bronchitis or increased production of phlegm among the subgroup of children with asthma, though no significant associations were found for the subgroups of children without asthma (Peters et al., 1999a; McConnell et al., 2000). After a four-year follow-up period for 4th grade children, decreases in lung function growth were associated with increasing exposure to PM_{10}, $PM_{2.5}$, and $PM_{10-2.5}$, with generally similar findings for both fine- and coarse-fraction particles. 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). Analysis of data from a second group of children recruited as 4th-graders showed decreases in lung function growth with long-term exposure to PM_{10} and $PM_{2.5}$, but the results were generally not statistically significant (Gauderman et al., 2002). Avol et al. (2001) also reported increased lung function growth among cohort participants who moved from areas with higher to lower PM_{10} concentrations during the study period, compared with participants who remained in high pollution areas. These findings are generally consistent with those from short-term exposure studies where respiratory morbidity is associated with $PM_{10,}$ $PM_{2.5}$ and $PM_{10-2.5}$.

Further analyses have been done for the AHSMOG adult cohort, and significant decreases in lung function were reported only for the subgroup of males with a family history of lung disease, but not for other subgroups of the cohort. 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.

A number of long-term studies of respiratory effects have also 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 8-B8 of the draft CD). The results of U.S. and Canadian studies, presented in Table 3-4, generally show increased symptoms and decreased lung function with increases in PM exposure. These new findings, along with those from non-North American studies, are generally 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.

Table 3.4 Effect Estimates for Morbidity per Increments in Long-term Mean Levels of Fine- and Coarse-fraction Particle Indicators from U.S. and Canadian Studies (from draft CD Table 9-11)

Type of Health Effect Study	Indicator (Increment)	Odds Ratio (95% CI)	Range of City PM Levels ** Means ($\mu g/m^3$)
Increased Bronchitis in Children			
Six City[A]	$PM_{15/10}$ (20 $\mu g/m^3$) $PM_{2.5}$ (10 $\mu g/m^3$)	1.6 (1.1, 2.5) 1.3 (0.9, 2.0)	NR (20, 59) NR (12, 37)
24 City[B]	$SO_4^=$ (15 $\mu g/m^3$) $PM_{2.1}$ (10 $\mu g/m^3$) PM_{10} (20 $\mu g/m^3$)	3.02 (1.28, 7.03) 1.31 (0.94, 1.84) 1.60 (0.92, 2.78)	4.7 (0.7, 7.4) 14.5 (5.8, 20.7) 23.8 (15.4, 32.7)
AHSMOG[C]	$SO_4^=$ (15 $\mu g/m^3$)	1.39 (0.99, 1.92)	—
12 Southern California communities[D] (all children)	PM_{10} (20 $\mu g/m^3$) (1986-1990 data)	0.95 (0.79, 1.15)	NR (28.0, 84.9)
12 Southern California communities[E] (children with asthma)	PM_{10} (20 $\mu g/m^3$) $PM_{2.5}$ (10 $\mu g/m^3$)	1.4 (1.1, 1.8) 1.3 (0.9, 1.7)	34.8 (13.0, 70.7) 15.3 (6.7, 31.5)
Increased Cough in Children			
12 Southern California communities[D] (all children)	PM_{10} (20 $\mu g/m^3$) (1986-1990 data)	1.05 (0.94, 1.16)	NR (28.0, 84.9)
12 Southern California communities[E] (children with asthma)	PM_{10} (20 $\mu g/m^3$) $PM_{2.5}$ (10 $\mu g/m^3$)	1.1 (0.7, 1.8) 1.2 (0.8, 1.8)	13.0-70.7 6.7-31.5
Increased Airway Obstruction in Adults			
AHSMOG[F]	PM_{10} (20 $\mu g/m^3$)	1.19 (0.84, 1.68)	NR
Decreased Lung Function in Children			
Six City[A]	$PM_{15/10}$ (50 $\mu g/m^3$)	NS Changes*	NR (20, 59)

Type of Health Effect Study	Indicator (Increment)	Odds Ratio (95% CI)	Range of City PM Levels ** Means ($\mu g/m^3$)
24 City[G]	$SO_4^=$ (15 $\mu g/m^3$)	−6.56% (-9.64, -3.43) FVC	4.7 (0.7, 7.4)
	$PM_{2.1}$ (10 $\mu g/m^3$)	−2.15% (-3.34, -0.95) FVC	14.5 (5.8, 20.7)
	PM_{10} (20 $\mu g/m^3$)	−2.80% (-4.97, -0.59) FVC	23.8 (15.4, 32.7)
12 Southern California communities[H] (all children)	PM_{10} (20 $\mu g/m^3$) (1986-90 data)	−19.9 (-37.8, -2.6) FVC	NR (28.0, 84.9)
12 Southern California communities[H] (all children)	PM_{10} (20 $\mu g/m^3$) (1986-1990 data)	−25.6 (-47.1, -5.1) MMEF	NR (28.0, 84.9)
12 Southern California communities[I] (4^{th} grade cohort)	PM_{10} (20 $\mu g/m^3$)	−0.23 (-0.44, -0.01) FVC % growth	NR (15, 70)***
	$PM_{2.5}$ (10 $\mu g/m^3$)	−0.18 (-0.36, 0.0) FVC % growth	NR (10, 35) ***
	$PM_{10-2.5}$ (10 $\mu g/m^3$)	−0.22 (-0.47, 0.02) FVC % growth	NR
12 Southern California communities[I] (4^{th} grade cohort)	PM_{10} (20 $\mu g/m^3$)	−0.51 (-0.94, -0.08) MMEF % growth	NR (15, 70)***
	$PM_{2.5}$ (10 $\mu g/m^3$)	−0.4 (-0.75, -0.04) MMEF % growth	NR (10, 35) ***
	$PM_{10-2.5}$ (10 $\mu g/m^3$)	−0.54 (-1.0, -0.06) MMEF % growth	NR
12 Southern California communities[J] (second 4^{th} grade cohort)	PM_{10} (20 $\mu g/m^3$)	−0.12 (-0.26, 0.24) FVC % growth	NR (10, 80)****
	$PM_{2.5}$ (10 $\mu g/m^3$)	−0.06 (-0.30, 0.18) FVC % growth	NR (5, 30) ****
12 Southern California communities[J] (second 4^{th} grade cohort)	PM_{10} (20 $\mu g/m^3$)	−0.26 (-0.75, 0.23) MMEF % growth	NR (10, 80)****
	$PM_{2.5}$ (10 $\mu g/m^3$)	−0.42 (-0.84, 0.0) MMEF % growth	NR (5, 30) ****
12 Southern California communities[J] (second 4^{th} grade cohort)	PM_{10} (20 $\mu g/m^3$)	−0.16 (-0.62, 0.30) PEFR % growth	NR (10, 80)****
	$PM_{2.5}$ (10 $\mu g/m^3$)	−0.20 (-0.64, 0.25) PEFR % growth	NR (5, 30) ****
12 Southern California communities[K]	PM_{10} (20 $\mu g/m^3$)	-3.6 (-18, 11) FVC growth	NR (15.0, 66.2)
12 Southern California communities[K]	PM_{10} (20 $\mu g/m^3$)	-33 (-64, -2.2) MMEF growth	NR (15.0, 66.2)
12 Southern California communities[K]	PM_{10} (20 $\mu g/m^3$)	-70 (-120, -20) PEFR growth	NR (15.0, 66.2)
Lung Function Changes in Adults			
AHSMOG[L] (% predicted FEV_1, females)	PM_{10} (cutoff of 54.2 days/year >100 $\mu g/m^3$)	+0.9 % (-0.8, 2.5) FEV_1	52.7 (21.3, 80.6)
AHSMOG[L] (% predicted FEV_1, males)	PM_{10} (cutoff of 54.2 days/year >100 $\mu g/m^3$)	+0.3 % (-2.2, 2.8) FEV_1	54.1 (20.0, 80.6)

Type of Health Effect Study	Indicator (Increment)	Odds Ratio (95% CI)	Range of City PM Levels ** Means ($\mu g/m^3$)
AHSMOG[L] (% predicted FEV_1, males whose parents had asthma, bronchitis, emphysema)	PM_{10} (cutoff of 54.2 days/year >100 $\mu g/m^3$)	−7.2 % (-11.5, -2.7) FEV_1	54.1 (20.0, 80.6)
AHSMOG[L] (% predicted FEV_1, males)	$SO_4^=$ (1.6 $\mu g/m^3$)	−1.5 % (-2.9, -0.1) FEV_1	7.3 (2.0, 10.1)

* NS Changes = No significant changes (no quantitative results reported).
** Presented as overall study mean (min, max), unless indicated as mean (±SD); NR=not reported.
*** Estimated from Figure 1, Gauderman et al. (2000)
****Estimated from figures available in online data supplement to Gauderman et al. (2002)

References:
[A] Dockery et al. (1989)
[B] Dockery et al. (1996)
[C] Abbey et al. (1995a,b,c)
[D] Peters et al. (1999a)
[E] McConnell et al. (1999)
[F] Berglund et al. (1999)
[G] Raizenne et al. (1996)
[H] Peters et al. (1999b)
[I] Gauderman et al. (2000)
[J] Gauderman et al. (2002)
[K] Avol et al. (2001)
[L] Abbey et al. (1998)

3.3.2.3 Effects on the Cardiovascular System

In the last review, evidence was available from a few epidemiologic studies indicating that PM was associated with increased mortality and hospital admissions for cardiovascular diseases. These findings inspired further research so that a significantly expanded body of evidence is available in this review from epidemiologic, toxicologic, and controlled human exposure studies. As described in the previous section, many new epidemiologic studies have continued to show relationships between mortality and hospital admissions or emergency department visits for cardiovascular diseases. Numerous new controlled human exposure, toxicological and epidemiologic studies have provided evidence on cardiovascular health endpoints, such as changes in cardiac function and changes in blood components or characteristics, that is building new understanding of how exposure to ambient particles could affect the cardiovascular system.

Measures of changes in cardiac function included in these new studies include arrhythmia, alterations in electrocardiogram (ECG) patterns, heart rate or heart rate variability changes, and incidence of myocardial infarction. One recent epidemiologic study reported an

association between the occurrence of nonfatal myocardial infarction (or a heart attack) and ambient fine particle concentrations averaged over the 2 hours preceding the event (Peters et al., 2001). In addition, Peters et al. (2000) used data on discharge frequency from implanted cardiac defibrillators (discharges occur when the patient is experiencing cardiac arrythmia) and reported generally positive associations with several PM indicators, including statistically significant associations with $PM_{2.5}$. As summarized in draft CD Table 7-7, animal studies have also shown evidence of arrythmia or alterations in ECG patterns that included increases in the S-T peak of the heart beat pattern; this type of change indicates effects on repolarization of the heart which is linked with increased risk of sudden death (CD, p. 9-63).

Heart rate variability changes have been reported in a number of epidemiologic studies. 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. Low heart rate variability has been implicated as a predictor of increased cardiovascular morbidity and mortality (CD, p. 9-62). Most, but not all, new studies reported decreases in several measures of heart rate variability with increased PM_{10} and $PM_{2.5}$ (CD, p. 8-143). While many epidemiology studies have used 24-hour average PM measurements, two new studies have linked effects with PM averaged over 4-hours. Gold et al., 2000 (reanalyzed in Gold et al., 2003) reported significant decreases in heart rate variability with 4-hour average $PM_{2.5,}$ but not with $PM_{10-2.5}$; Magari et al. (2001) also reported decreased heart rate variability in 4-hour $PM_{2.5}$ concentrations. The findings on changes in heart rate are less consistent than those for heart rate variability, with some studies reporting significant increases in heart rate with PM exposure, but one study reporting an association with decreased heart rate (CD, p. 8-143).

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). Changes in heart rate or heart rate variability generally indicate effects on the autonomic nervous system, and the draft CD concludes “There is now ample evidence that inhaled particles can affect the heart through the [autonomic nervous system]” (CD, p. 9-61).

Some studies have reported increases in blood components or characteristics. In epidemiologic studies, exposure to particles has resulted in increased levels of C-reactive protein, which is considered an important indicator of inflammation, tissue damage and infection, and related to increased risk of cardiac events (CD, p. 9-65). Fibrinogen is a blood clotting factor 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 PM, fibrinogen levels were increased in some, but not all, studies, and plasma viscosity was increased with PM exposure in one European study (CD, pp. 8-143 to 8-145). One new controlled human exposure study reported mild increases in neutrophils in bronchoalveolar lavage samples and increased blood fibrinogen levels after 2-hour exposure to concentrated ambient $PM_{2.5}$ (Ghio et al., 2000). Finally, changes in blood cell counts, including platelets or red blood cells, was reported in some epidemiologic studies (CD, p. 8-145). A number of toxicologic studies have also reported such hemolytic effects as changes in blood factors such as hemoglobin levels or platelet counts (CD, Table 7-7).

Summary. The draft CD concludes that ". . .animal studies, to date, have provided evidence indicating that high concentrations of inhaled or instilled particles can have systemic, especially cardiovascular, effects." (CD, p. 7-52). A more detailed discussion of the potential mechanisms or pathways by which PM could be linked with effects on the cardiovascular system is included in draft CD section 9.7.3, where the draft CD concludes "[t]aken as a whole, these studies are difficult to interpret but clearly indicate that PM can affect the circulatory system. However, a complete understanding of the pathways by which very small concentrations of inhaled ambient PM can produce vascular changes that can contribute to increased mortality/morbidity remains to be more fully elucidated." (CD, p. 9-66).

Regarding the epidemiologic studies, the draft CD concludes: "[t]he above findings add support for some intriguing hypotheses regarding possible mechanisms by which PM exposure may be linked with adverse cardiac outcomes. They are interesting in 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. 8-145). The results of recent toxicologic studies, epidemiologic panel studies, and controlled human exposure 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 (CD, p. 9-127).

3.3.2.4 Developmental Effects

Some new evidence is available that is suggestive of adverse effects of air pollution on prenatal development. Several recent studies have shown significant associations between PM_{10} concentration averaged over a month or a trimester of gestation and risk of IUGR (intrauterine growth reduction) and low birth weight (CD, pp. 8-112, 8-113). However, when gaseous co-pollutants were included in the studies, it was sometimes difficult to separate the effects of the pollutants. The CD concludes that these effects are emerging as potentially more important than was appreciated in the 1996 CD but the evidence is still limited regarding these effects (CD, pp. 9-136 and 9-137).

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 pre-existing respiratory and cardiovascular disease, the elderly and children (EPA 1996b, pp. V-33 to V-36). The draft CD (sections 9.9.2 and 9.9.3) summarizes the results of new studies as they relate to two major at-risk categories: (1) persons with preexisting disease; and (2) age-related population groups. The new studies continue to support consideration of people with preexisting diseases, the elderly and children as potentially sensitive to PM; new evidence also suggests that infants are a potentially sensitive group, including evidence of development effects during the prenatal period (CD, p. 9-136). Some studies have suggested that other factors, such as socioeconomic status, race, or gender may play a role in susceptibility to PM-related effects, but only limited and inconsistent evidence on such groups is available.

3.4.1 Preexisting disease as a risk factor

Numerous epidemiologic studies have identified individuals with cardiorespiratory diseases (e.g., asthma, COPD) as being at greater risk for adverse effects with PM exposure (CD, p. 9-73). 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. 9-76).

Several new epidemiologic studies have evaluated associations between PM and mortality or hospital admissions for subpopulations with existing heart or lung disease groups, also including diabetes. Associations for PM_{10} with hospital admissions for cardiovascular diseases were larger in the subgroup of people with diabetes (Zanobetti and Schwartz, 2001). In addition, larger effect estimates for hospital admissions for cardiovascular diseases were reported in people with acute respiratory infections, and for hospital admissions for pneumonia in people with asthma, and for hospital admissions for COPD in people with heart failure (Zanobetti et al., 2000). Staff notes that both analyses used GAM and have not been reanalyzed; though in reanalyses of other studies using the same data, there was little effect on the results (Zanobetti and Schwartz, 2003). One new study also indicated in original analyses that risk of mortality for nonaccidental causes was increased in subpopulations with diabetes, heart or lung diseases, but when reanalyzed, the effect estimates generally lost statistical significance (Goldberg et al., 2000; Goldberg and Burnett, 2003).

Asthma has been of particular public interest as a respiratory condition that may lead to sensitivity to air pollution effects. Included in Appendix A are numerous epidemiologic 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. In the studies of lung function or symptoms, asthmatic subjects generally had greater reduction in pulmonary function with PM exposure, but both asthmatic and non-asthmatic subjects had similar responses in respiratory symptom studies. A number of toxicologic 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. 7-58).

New dosimetry studies have shown that, among people with small airways disease such as COPD, airflow may be unevenly distributed due to airway obstruction, and there can be

closure of small airways, 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 are 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, pp. 6-34).

A number of animal models of susceptible populations have been used in toxicologic studies examining PM. These models 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 7.4 of the draft CD. In general, these studies reported greater effects (including lung function changes, evidence of lung injury, or immunological changes) in the animal models of susceptibility. While recognizing that further research is needed, the draft CD concludes that these studies "do provide evidence of enhanced susceptibility to inhaled PM in 'compromised' hosts" (CD, p. 7-50). In addition, a number of new studies, though using animal models and high particle doses, have suggested that genetic susceptibility may play a role in differential responses to inhaled particles across a population (CD, p. 7-52).

3.4.2 Age-related risk factors

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, children and infants (CD, p. 9-135).

Evidence from dosimetry studies 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 (CD, p. 6-103), and limited evidence is available from toxicologic studies on older animal models. However, numerous epidemiologic studies have reported stronger and more consistent associations between mortality or morbidity effects and PM in older age groups (CD, pp.8-146, 8-166). The newer studies of cardiac function have also suggested that elderly persons with preexisting cardiopulmonary disease are susceptible to changes in cardiac function (CD, p.

8-146), indicating a connection between the elderly and preexisting disease status in susceptibility to PM-related effects.

Children have been identified as being particularly susceptible to the effects of PM on the respiratory system (CD, p. 8-152). As summarized above, associations have been found between PM and acute respiratory symptoms or changes in lung function, hospital admissions or emergency room visits for respiratory disease, as well as chronic effects such as decreased lung function growth. The draft CD highlights findings of a number of new studies that raise the possibility that deposition may be greater in children than adults, though the studies "still do not provide unequivocal evidence for significant differences in deposition between children and adults" (CD, p. 6-31). However, children have generally higher activity levels with accompanying higher ventilation rates that might contribute to increased particle deposition. Children also have smaller lungs and higher minute volumes relative to lung size, which would likely result in greater doses of particles per lung surface area compared to adults; they are more likely to spend time outdoors, increasing exposure to ambient particles; and they have the highest rates of respiratory illnesses related to infection (CD, p. 9-135).

Several new epidemiologic 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, suggesting that the prenatal development period may be a time of potential susceptibility to air pollutants (CD, p. 9-106). It is known that the early post-natal period of lung development is a time of high susceptibility for lung damage by pollutants, which is concordant with findings of reduced lung function growth in older children in the Southern California children's cohort (CD, p. 9-136).

3.5 EVALUATION OF PM-RELATED HEALTH EFFECTS EVIDENCE

The preceding sections summarized evidence from new health studies and integrated them with findings from previous PM NAAQS reviews. As in those previous reviews, much of the currently available health evidence is derived from epidemiologic studies, though critical new insights are offered in the results of toxicologic 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. 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 regarding potential confounding by gaseous co-pollutants will be discussed in section 3.5.1. Several additional issues related to analytical modeling in epidemiological studies is discussed in section 3.5.2: (1) the influence of model specification on epidemiologic findings and the related issue of appropriate control for potential confounding by weather and related time-varying factors; (2) the lag period between exposure and occurrence of health effects; and (3) the influence of exposure error or exposure misclassification on reported PM-health associations. In addition, new information is available on potential health effects of PM components or source-related PM, as summarized in section 3.5.3. Finally, evidence regarding the consistency and coherence of PM-related effects is summarized in section 3.5.4.

3.5.1 Role of Gaseous Co-pollutants

The extent to which PM-related effects are confounded or modified by other pollutants was discussed at some length in the 1996 Staff Paper (EPA, 1996b, pp. V-45 to V-54) as one of the important uncertainties considered in making recommendations concerning the PM NAAQS (EPA, 1996b, p. VII-13). As stated previously, another pollutant 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. 8-8). In addition, there may be effects associated with co-exposure to multiple pollutants. The draft CD states, "it is important to continue to recognize that health effects associated statistically with any single pollutant may actually be mediated by multiple components of the complex ambient mix" (CD,

p. 9-69). Recent animal toxicologic studies have tested effects of exposure to PM or PM surrogates (e.g., urban PM, carbon particles, acid aerosols) in combination with O_3. In a series of recent studies, co-exposure to O_3 and urban particles resulted in greater effects than those reported with exposure to O_3 alone, while mixed results were reported from studies using combinations of acid aerosols and O_3 (CD Table 7-12).

In epidemiologic studies, there are a number of methods for assessing potential confounding by co-pollutants, including multi-pollutant modeling in multiple or single locations and assessing the relationship between PM-mortality associations and correlations between PM and copollutant concentrations in multi-city studies. All methods have issues to be considered in interpreting their findings (CD, p. 8-13 to 8-14). It is important to recognize that there are issues relating to potential copollutant confounding that multi-pollutant models may not be able to address. Inclusion of pollutants in a multi-pollutant model that are highly correlated with one another can lead to misleading conclusions in identifying a specific causal pollutant. 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. 8-205). This situation certainly occurs. For example, sources of PM constituents include combustion of various fuels, gasoline or diesel engine exhaust, and some industrial processes (CD, Table 9-1); 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. Methods that use data from multiple cities in hierarchical or two-stage analyses have been used in some more recent studies, but these also may be influenced by model misspecification or heterogeneity across the cities (CD, p. 8-14).

As discussed in the section 3.3, multi-pollutant models have been commonly used to assess potential confounding among pollutants. This approach was used in the NMMAPS mortality analyses for 20 and 90 U.S. cities, in which the authors added first ozone, then ozone and another co-pollutant (e.g., CO, NO_2 or SO_2) to the models (CD, p. 8-35). As shown in Figure 3-10, the relationship between PM_{10} and mortality was little changed in models including control for ozone and other gaseous pollutants. The authors concluded that the PM_{10}-mortality relationship was not confounded by co-pollutant concentrations across 90 U.S. cities (Samet et al., 2000a,b, reanalyzed Domenici 2003). Among the recent single-city studies, a number of

research groups have reported little influence of co-pollutants on PM associations in such models (e.g., Lippmann et al., 2000, reanalyzed Ito, 2003; Fairley 1999, reanalyzed Fairley 2003; Ostro et al., 2000, reanalyzed Ostro et al., 2003), although some have reported substantial reduction in associations with PM when gaseous pollutants are added to the model (e.g., Chock et al., 2000; Moolgavkar et al., 2000a,b,c; reanalyzed Moolgavkar 2003).

An example of the second type of evaluation of multi-pollutant confounding was used in the NMMAPS morbidity analyses in 14 U.S. cities. The authors used an alternative approach, testing for relationships between the coefficients for the PM_{10}-admissions with PM_{10}-co-pollutant correlations for each city. No such relationships were found between the PM_{10} effect estimates and PM_{10}-co-pollutant correlations, leading the authors to conclude that the associations with PM_{10} were not confounded by the gaseous co-pollutants (CD, pp. 8-125, 8-151).

Based on the results of multi-pollutant modeling methods, the draft CD concludes that while there are still difficulties in separating effects of pollutants, "it now appears unlikely that such confounding accounts for the vast array of effects attributed to ambient PM based on the rapidly expanding PM epidemiology database" (CD, p. 9-112). This conclusion is supported not only by statistical approaches, but also by: (1) the biological plausibility of effects of the potentially confounding pollutants; and (2) consideration of exposure relationships.

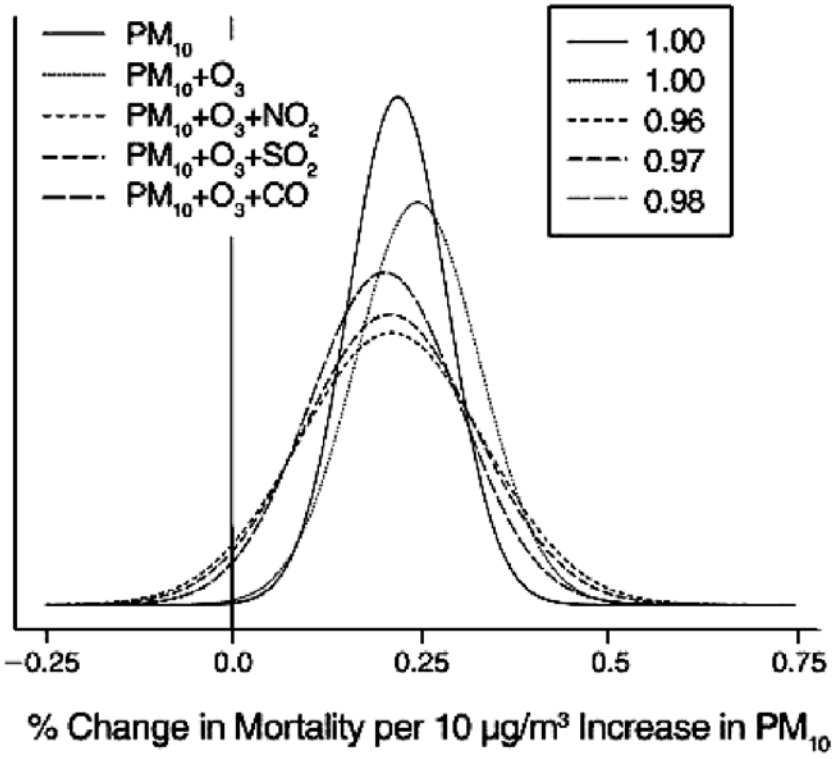


Figure 3-10. Marginal posterior distributions for effect of PM_{10} on total mortality at lag 1 with and without control for other pollutants, for the 90 cities. The numbers in the upper right legend are the posterior probabilities that the overall effects are greater than 0. (CD Figure 8-6)

Source: Dominici et al. (2003).

Biological plausibility. With regard to biological plausibility, the draft CD observes that "It is generally accepted that O_3, NO_2 and SO_2 are associated with diminished pulmonary function and increased respiratory symptoms as well as more serious consequences, and CO exposure has been associated with cardiovascular effects" (CD, p. 8-206). For example, if concentrations of CO and PM are correlated, CO is a potential confounder for PM-related effects on the cardiovascular system, while it is an implausible confounder for effects on the respiratory

system. Reflecting the evidence summarized in the recent Criteria Document 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. 9-37).

Similarly, ozone would plausibly be a potential confounder for respiratory effects, but not cardiovascular effects. In the discussion of studies on respiratory hospital admissions and respiratory symptoms or lung function in asthmatics (CD sections 8.3.2 and 8.3.3), however, where PM and ozone are considered together, they were generally found to have independent effects (CD, pp. 8-174, 8-185).

SO_2 exposure can affect the airways, especially for people with asthma, resulting in breathing difficulties that can be accompanied by symptoms such as wheezing, chest tightness, or shortness of breath. Short-term exposures (e.g., less than 3 hours) to low levels of NO_2 may lead to changes in airway responsiveness and lung function in individuals with preexisting respiratory illnesses and increases in respiratory illnesses in children. Long-term exposures to NO_2 may lead to increased susceptibility to respiratory infection and may cause irreversible alterations in lung structure. Thus, both SO_2 and NO_2 are plausibly linked with effects on the respiratory system, but it is less plausible to consider SO_2 and NO_2 as confounders in associations with effects such as cardiovascular mortality.

Exposure relationships. In addition to considering correlations between ambient concentrations of PM and the gaseous pollutants, several new exposure studies have assessed correlations between ambient pollutant concentrations and individuals' personal exposure to ambient-origin pollutants. Some recent exposure studies have collected personal and ambient monitoring data, collected at a single central site, for $PM_{2.5}$ and gaseous pollutants (e.g., O_3, SO_2 and NO_2), and assessed the degree of day-to-day correlation between the different measures of personal and ambient concentrations. The investigators reported that the personal and ambient

$PM_{2.5}$ measurements were correlated, as were personal exposure to $PM_{2.5}$ and ambient concentrations of the gaseous pollutants. However, the personal and ambient concentrations of each of the gaseous pollutants were not well correlated. The draft CD concludes that ambient concentrations of NO_2, SO_2, and O_3, and likely CO, are unlikely to confound reported associations of ambient PM with health effects, but can possibly serve as surrogates for ambient PM exposure or more likely exposure to source categories with which the gases are correlated, i.e., NO_2 and CO with motor vehicle-associated PM and SO_2 and O_3 with regional sulfate (CD, p. 9-37).

Summary. Several lines of evidence provide information on potential confounding of PM-health relationships by co-pollutants. Among studies using multi-pollutant modeling methods, most but not all report no substantial change in PM effect estimates with control for gaseous co-pollutants. Powerful new evidence is available from multi-city studies, particularly the NMMAPS, where the relationship between PM_{10} and mortality was relatively unchanged with control for gaseous co-pollutants in 90 U.S. cities. Considering biological plausibility, clearly other pollutants such as the gaseous criteria pollutants have been associated with certain health effects; indeed EPA has established NAAQS for the other criteria pollutants based in part on such associations. Where PM and the other pollutants are correlated, it can be difficult to distinguish effects of the various pollutants in multi-pollutant models. However, a number of research groups have found the effects of PM and gases to be independent of one another, for example, in the evidence from asthma symptom studies with PM and ozone. In addition, new evidence on exposure considerations suggests that it is less likely that a relationship found between a health endpoint and ambient PM concentrations is actually representing a relationship with another pollutant. 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 (CD, p. 8-279).

3.5.2 Issues Related to Epidemiological Modeling

The 1996 CD included extensive discussions of methodological issues for epidemiologic studies, including questions about model specification or selection, measurement error in

pollutant measurements and exposure misclassification. As summarized in the 1996 Staff Paper, PM-health effects associations reported in epidemiologic 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). Little information was available for the 1996 CD to allow comparison of differing lag periods for different health effects. Also, few studies quantitatively evaluated the potential influence of measurement or exposure error in interpreting epidemiologic study findings or compared exposure time windows for PM-related health effects.

Recent studies have provided some new information, as discussed below. Broader questions about model selection resurfaced recently in the process of addressing questions about time-series epidemiologic models using GAM. These new questions have been evaluated in a series of reanalyses of some time-series studies, and are discussed in section 3.5.2.1. Some recent studies have evaluated appropriate lag periods between the concentration exposure period and the health outcome being measured, and the results of these studies are discussed in section 3.5.2.2. Finally, section 3.5.2.3 summarizes new information from studies that have assessed the influence of measurement error on epidemiologic analyses.

An additional issue, the form of the concentration-response function, is described in more detail in Chapter 4 (section 4.2.6.1), with the discussion of the risk analyses. To briefly summarize that discussion, a number of short-term exposure studies have evaluated concentration-response functions for mortality, and while one study suggested a potential threshold level for $PM_{2.5}$, overall, the studies do not provide evidence for a discernible threshold level (see p. 4-32). For long-term exposure to PM, the shape of the concentration-response function was also evaluated using data from the ACS cohort, where mean $PM_{2.5}$ concentrations ranged from about 10 to 34 $\mu g/m^3$, and the concentration-response relationships for all-cause and cardiopulmonary mortality could be reasonably approximated by a linear model (CD, p. 8-89). Staff concludes that, while it is reasonable to expect that for individuals or groups of individuals there may be biological thresholds for different effects, discernible population threshold levels have not been detected in available epidemiologic studies.

3.5.2.1 Model Specification

In 2002, questions were raised about the default convergence criteria and standard error calculations made using generalized additive models (GAM), which has been commonly used in

recent time-series epidemiologic studies. As discussed more completely in section 8.4.2 of the draft CD, a number of time-series studies were reanalyzed using alternative methods, typically GAM with more stringent convergence criteria and an alternative model such as generalized linear models (GLM) with natural smoothing splines, and the results of the reanalyses have been compiled and reviewed in a recent HEI publication (HEI, 2003a). In most, but not all, of the reanalyzed studies, it was found that risk estimates were reduced and confidence intervals increased with the use of GAM with more stringent convergence criteria or GLM analyses (CD, p. 8-193). PM_{10} mortality study results are presented in Figure 8-17 of the draft CD, where it can be seen that the reanalyses generally did not substantially change the findings of the original analyses, and the changes in risk estimates with alternative analysis methods were much smaller than the variation in effects across studies. The HEI review committee concluded:

- While the number of studies showing an association of PM with mortality was slightly smaller, the PM association persisted in the majority of studies.
- In some of the large number of studies in which the PM association persisted, the estimates of PM effect were substantially smaller.
- In the few studies in which investigators performed further sensitivity analyses, some showed marked sensitivity of the PM effect estimate to the degree of smoothing and/or the specification of weather. (HEI, 2003b, p. 269)

Thus, these reanalyses indicated that weather continues to be a potential confounder of concern. As summarized in the draft CD, there remains no altogether satisfactory way to select the "best" model to address time and weather variables. If the model does not adequately address daily changes in weather variables, then some effects of temperature on health would be falsely ascribed to the pollution variable. Conversely, if the analyst overcontrols for weather, such that the temperature-health relationship is more "wiggly" than the true dose-response function, then the result will be a much less efficient estimate of the pollutant effect. This would result in incorrectly ascribing some of the true pollution effect to the temperature variable, which would make it difficult to detect a real but small pollution effect. Thus, the HEI review panel recommended "further exploration of these studies to a wider range of alternative degrees of smoothing and to alternative specifications of weather variables in time-series models." (CD, p. 8-202).

Among the new studies included in Appendix 8A of 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. Where both case-crossover and time-series methods have been used to analyze the same or similar data, the results have been similar. For example, Neas et al. (1999) found associations between TSP and daily mortality in Philadelphia similar to those found in previous time-series studies summarized in the 1996 CD (EPA 1996a, pp. 12-283 to 12-302), while Lee et al. (1999) and Lee and Schwartz (1999) used time-series and case-crossover methods with TSP and daily mortality data from Seoul, Korea, and neither reported a statistically significant association.

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 "[n]one of these alternative models produced results that materially altered the original findings" (CD, p. 8-84).

In summary, questions about the analytical methods used in epidemiologic studies, especially the time-series studies, have been raised in this review as well as previous reviews. The recently discovered issues with GAM analyses have raised new questions about addressing time-varying variables such as weather adequately, but the overall conclusion has remained that the original findings are generally supported in reanalyses. In addition, the major reanalysis of the prospective cohort studies reported that the findings were not sensitive to alternative modeling strategies, but additional questions remain about adequately addressing spatial correlation in the data (Krewski et al., 2000).

With the substantial amount of evidence now available on PM-related health effects, EPA has not focused on evaluating the methods used in each individual study, but rather on the full body of evidence, including epidemiologic studies that use a variety of methods and data from

multiple or single locations, as well as information from other scientific disciplines. The approach is summarized in the draft CD:

> Rather, a three-pronged approach is likely to yield useful evaluative information: (1) an overall characterization of evident general commonalities (and/or notable marked differences) among findings from across the body of studies dealing with particular PM exposure indices and types of health outcomes, looking for convergence of evidence regarding types of effects and effect-sizes attributable to ambient PM indices across various methodologically acceptable analyses; (2) thorough, critical assessment of newly published multi-city analyses of PM effects, assuming that greater scientific weight is generally ascribable to their results than those of smaller-sized studies (often of individual cities) yielding presumably less precise effect size estimates; and (3) evaluation of coherence of the findings among different types of effects and across various geographic locations, as well as with other types of pertinent biological information (e.g., exposure, dosimetry, toxicity, etc.). (CD, p. 8-192)

3.5.2.2 Lag Periods

One important model selection question in studies of short-term PM exposures is the selection of a lag period between air pollutant concentration changes and health outcome measures. 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. Many epidemiologic 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, and the results for the most statistically significant lag period are reported in the publication. As stated in the draft CD, "[w]hile 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. 8-234). In some more recent studies, goodness-of-fit criteria have been used to help select the best modeling approach. Some multi-city studies, such as the NMMAPS, have used a fixed lag period for all sites. In addition, an alternative approach, the distributed lag, has been introduced in several new studies. With this approach, 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. 8-235). For morbidity studies, 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. 8-147). Recent studies of hospital admissions for respiratory diseases, summarized in Tables 8-17, 8-19 and 8-20 of the draft CD, report results for varying lag periods for different categories of respiratory disease, ranging from 0 to 3 days. 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 (Samet et al., 2000b). In an analysis using data on cause-specific mortality from 10 U.S. cities, distributed lag estimates were substantially larger than estimates for 1- or 2-day average lag periods (Schwartz, 2003b).

As noted in the draft CD, misspecification of the lag structure can result in modeling biases (CD, p. 8-237). One concern is where PM measurements are available only every other or every sixth day, which makes evaluation of appropriate lag periods difficult, since it isn't possible to test distributed lag or moving average models adequately. Also, as the draft CD states "[o]ne would then expect to see different best-fitting lags for different cause-specific mortality or hospital admissions" (CD, p. 8-238). Based on these potential concerns, and the results of some studies indicating larger effect sizes in distributed lag models, the draft CD concludes that single-day or two-day average lag periods may be underestimating PM effects (CD, p. 8-238).

In summarizing questions about lag periods for the short-term exposure studies, the draft CD states:

> It may be possible that different PM size components or particles with different composition or sources produce effects by different mechanisms manifested 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. Also, if it is considered that all health effects occurring at different lag days are all real effects, so that the risks for each lag day should be additive, then higher overall risks may exist that are higher than

implied by maximum estimates for any particular single or two-day lags. In that case, multi-day averages or distributed lag models should be used. (CD, p. 8-280).

3.5.2.3 Measurement Error

In this and previous reviews of the PM NAAQS, much of the health evidence for PM-related effects comes from epidemiologic 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, daily changes in individuals' personal exposure to ambient PM is well correlated with daily changes in ambient PM measured at central monitors. While particles can be generated from indoor sources, particles from nonambient sources are not correlated temporally with ambient particle concentrations. Thus, the draft CD concludes that ambient PM concentrations measured at central site monitors are a useful surrogate for exposure to ambient PM (CD, p. 9-117, p. 8-252).

Another key issue in interpreting epidemiologic 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 (CD, p. 8-249).

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 effect 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 epidemiologic 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.

Sheppard and Damian (2000) did a simulation study using asthma hospital admissions and PM data from Seattle. As was found for mortality, effect estimates and their standard errors did increase with increasing magnitude of measurement error variance. However, "The change is small and does not influence the relative risk estimate" (Sheppard and Damian, 2000, p. 686).

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 that 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. 8-255)

3.5.3 Consideration of Evidence on PM Size and Composition

Much of the focus of the preceding discussions on the nature of PM-related effects has been epidemiologic studies that use gravimetric PM measurements, with an emphasis on PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$. In the 1996 Staff Paper, evidence from health studies for the two main PM size categories, fine- and coarse-fraction particles, was extensively discussed (EPA, 1996b, p. V-58 to V-76). The body of evidence on fine- and coarse-fraction particles has been greatly expanded, and the results of recent studies have been discussed throughout the discussions of mortality and morbidity effects in section 3.3.

Beyond studies of fine- and coarse-fraction particles, the newly available evidence also includes studies of ultrafine particles, various components in the fine and coarse fractions, and source-related particles. Section 5.3.5.1 will focus on the new information regarding ultrafine particles. Building upon information available in the last review, section 3.5.3.2 discusses reported health associations with different PM components and with various source-related particles.

3.5.3.1 Ultrafine particles

As described in Chapter 2, ultrafine particles generally include particles smaller than 0.1 μm in diameter. Ultrafine particles are a portion of fine PM; they predominate in the number of particles, but comprise only a very small portion of fine PM mass. It has been suggested, based on toxicological evidence, that ultrafine particles may be relatively more toxic than larger particles (e.g., accumulation mode particles). It has also been proposed that particle surfaces, or the chemical composition of particle surfaces, may be related to PM toxicity, and ultrafine particles have relatively large surface area for a given mass (CD, p. 7-84). It is expected that ultrafine particles "because of their small size, are not effectively phagocytized by alveolar macrophages and can easily penetrate the airway epithelium, gaining access to the interstitium" (CD, p. 7-85). In addition, some recent studies indicate that ultrafine particles can be rapidly cleared from the lungs into the systemic circulation and reach other organ systems (CD, p. 6-53).

The toxicologic 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. 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. 7-86). However, 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. 7-118).

A limited number of epidemiologic 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; reanalyzed in Stolzel et al., 2003). 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}, generally reporting significant associations with both ultrafine particle counts and PM mass (Peters et al., 1997; Pekkanen et al., 1997; Tiittanen et al., 1999).

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. 9-32). This would suggest that community exposure to PM is greater for ambient fine particles than ambient ultrafine particles, and makes it unlikely that health associations found with ambient $PM_{2.5}$ mass are truly reflecting underlying associations with ultrafine PM.

3.5.3.2 PM Components and Source-Related Particles

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. In addition, a substantial body of epidemiologic studies had evaluated relationships between

mortality and morbidity and ambient sulfate or acid aerosol concentrations. The 1996 CD concluded that the epidemiologic 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). Many of the components or sources listed above are more likely to be linked with fine-fraction than with coarse-fraction particles, such as sulfates, nitrates, acid aerosols, metals, and motor vehicle emissions. In addition, the CAPs used in toxicology studies has been comprised of concentrated fine-fraction or accumulation mode particles. Biological components (e.g., fragments of pollen grains, mold spores) and crustal material are more likely linked with coarse-fraction particles. Thus, more new information regarding PM components is relevant to fine-fraction than to coarse-fraction particles. In the following discussion, evidence on components that are likely to be found in fine-fraction particles is summarized first, following by components or sources reflecting coarse-fraction particles.

Among epidemiologic 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). Several new epidemiologic studies, and one toxicologic study, have used factor analysis or source apportionment methods in an attempt to distinguish effects of PM from different source types. A larger body of evidence on effects of specific PM components is available from toxicological studies.

As was reported in the previous review, numerous epidemiologic 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 8-8). 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. 8-66). Significant associations were also reported using sulfates as the PM indicator in studies of long-term PM exposure and mortality (CD, p. 8-107).

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, in toxicologic and controlled human exposure studies using exposure to acid aerosols (CD, Table 7-5), limited evidence for effects are reported. The draft CD concludes that the new studies are consistent with the findings from the previous review, where effects were reported in toxicological or controlled human exposure studies only when levels were very high, although "acidic components should not be entirely dismissed as possible mediators of ambient PM health effects, since so little is known about potential cardiovascular impacts or impacts in compromised subjects" (CD, p. 9-57). One difference between the epidemiologic and toxicological studies is that the epidemiologic studies were measuring sulfates or acidity of the ambient aerosol, while toxicological studies were using exposures to acid aerosols alone. The draft CD notes that while the independent toxicities of sulfates and nitrates are found to be low in toxicologic studies, they may influence the toxicity or bioavailability of other PM components, such as the metals (CD, p. 9-69).

Elemental carbon and organic carbon concentrations were used in studies conducted in Atlanta (Klemm and Mason, 2000; Metzger et al., 2003) and Phoenix (Mar et al., 2000, reanalyzed Mar et al., 2003). Both were significant predictors of mortality in the Phoenix study, and of emergency department visits for cardiovascular diseases in Atlanta (Metzger et al., 2003), although no PM indicators were reported to be significantly associated with mortality in Atlanta, possibly due to the small sample size in this preliminary analysis (Klemm and Mason, 2000). The draft CD observes that the correlations between COH, elemental carbon and organic carbon and other mobile source related pollutants (fine PM, NO_2, CO) were high and are likely indicators of particles from motor vehicle emissions, concluding that associations found with these components suggest that "PM components from motor vehicle sources are likely associated with mortality" (CD, p. 8-70).

Recent studies have provided substantial new evidence on effects of particulate metals and diesel exhaust particles. The substantial body of evidence on both cancer and noncancer effects of diesel exhaust emissions (including diesel exhaust particles) is summarized in the *Health Assessment Document for Diesel Engine Exhaust* (EPA, 2002). These effects include respiratory symptoms and exacerbation of the allergic response to inhaled antigens, potentially

contributing to the incidence and severity of allergic rhinitis and asthma (CD, p. 9-60). In addition, long-term exposure to diesel exhaust is likely to pose a lung cancer hazard to humans (EPA, 2002).

Metals, especially water soluble metals, have been reported to cause cell injury and inflammatory changes in toxicologic studies; it is noted that many studies have used relatively high doses (CD, p.7-116). For example, Costa and Dreher (1997) summarized results from a number of toxicologic studies that have reported that effects were more closely linked to the metal content of particles than particle mass. The transition metals, such as iron, vanadium or nickel, have been most commonly associated with effects in toxicologic studies, and autopsy studies have shown that exposure to these metals is widespread as indicated by their presence in the lung tissues of urban residents. The draft CD concludes that combustion particles with high metal content (e.g., ROFA) have been found to cause adverse effects, although it has not yet been established that the same effects are found with the generally lower metal content of ambient particles (CD, p. 9-58). Limited evidence on metals is also available from epidemiologic studies, although one new study reported associations between mortality and particulate iron, nickel and zinc in 8 Canadian Cities (Burnett et al., 2000; these results not reanalyzed). As discussed above, a series of studies used ambient particles or particle extract from Utah Valley PM, where a major source had been closed for a period of time, and reported that effects were greater with particles collected during the period when the steel mill was operating, and that the effects were also correlated with the metal content of the particles (CD, p. 9-63).

In one new toxicologic 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). The authors conducted factor analysis and identified four PM factors: aluminum/silicon, sulfur, vanadium/nickel, and bromine/lead, each of which was found to be associated with different effects. 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.

Four new epidemiologic studies and one toxicologic study have used factor analysis or source apportionment methods to investigate health associations with PM ($PM_{2.5}$ and PM_{10} or PM_{15}) from different sources (Laden et al., 2000, reanalyzed in Schwartz, 2003; Mar et al., 2000, reanalyzed in Mar et al., 2003; 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 8-4). In addition to testing associations between PM mass and mortality, the four studies evaluated relationships with the PM source factors. The four epidemiologic 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. The draft CD concludes that the results of the epidemiologic studies generally indicate that a "number of source types were associated with mortality, including motor vehicle emissions, coal combustion, oil burning and vegetative burning" (CD, p. 8-70).

Bioaerosols are more typically found in coarse-fraction than fine-fraction particles. 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 7-6. 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: "[a]lthough 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. 7-39). In addition, a number of epidemiologic studies have reported 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 epidemiologic 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 in at least some locations" but sufficient evidence is not yet available to support or refute this hypothesis (CD, pp. 9-85).

The effects of PM of crustal or geologic origin were also investigated in two epidemiologic 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). The draft CD finds that the results of these studies, taken together, suggest that particles of crustal origin (whether in the fine or coarse fraction of PM) are not likely associated with acute mortality (CD, pp. 8-59). However, the draft CD observes that "crustal" particles may carry biological components (e.g., endotoxin), toxic trace elements, or components of previously deposited fine PM, such as metals from smelters, steel mills or other industrial operations (CD, p. 8-281). 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. It is also possible that interaction between some PM components may be an important factor in some health effects associations; for example, sulfates may increase the bioavailability of metals, and diesel particles may increase allergic effects of bioaerosols. From the results of toxicologic studies, the draft CD concludes "It appears that many biological responses are produced by PM whether it is composed of a single component or a complex mixture" (CD, p. 7-79). 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. 9-89). The draft CD concludes:

> A primary causative attribute may not exist but rather many attributes may contribute to a complex mechanism driven by the nature of a given PM and its contributing sources. The multiple interactions that may occur in eliciting a response in a host may make the identification of any single causal component difficult and may account for the fact that mass as the most basic metric shows the relationships to health outcomes that it does. (p. 9-61)

3.5.4 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). Substantially more evidence of associations between ambient PM and health effects is now available, including multi-city studies that address some of the limitations of single-city studies, and these studies provide further support for the consistency and coherence of the body of evidence on PM-related effects.

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). 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.

3.5.4.1 Consistency

The 1996 Criteria Document summarized over 80 community epidemiologic 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, V-55 and Figure V-2).

Since the last review, more than 80 new studies have been published on mortality alone

and a large number of new morbidity studies. The draft CD notes that the effect 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, but also from the significantly expanded body of studies using $PM_{2.5}$ (CD, p. 8-276). A number of the new studies have included $PM_{10-2.5}$ and suggest that coarse-fraction particles may also be linked with serious health effects (CD, pp. 8-276, 8-278).

In the 1996 Staff Paper, staff also recognized a number of factors related to addressing why effect estimates might be expected to vary from one location to another. These factors include, for example, variation in ambient PM levels, differing socioeconomic or demographic characteristics, variation in composition of PM across areas, and differing health status (i.e., potential susceptibility) between cities (EPA, 1996b, p. V-55). As discussed above in section 3.3, staff has focused on the results of recent multi-city studies in addressing questions about variation across cities that are difficult to answer in single-city studies. Several multi-city studies have explored potential differences in effect estimates among cities, and have provided some evidence for factors that might explain variation in effect estimates. In addition to the factors recognized in the previous PM NAAQS review, the CD observes that, especially in the NMMAPS analyses for 90 U.S. cities, inclusion of cities with smaller sample sizes (with respect to mortality rate and air quality data availability) may result in greater apparent heterogeneity simply due to more statistical imprecision of results in the smaller cities (CD, p. 8-246). In addition, the CD recognizes that factors such as air conditioner use, as an indicator for reduced exposure to outdoor air pollutants, can potentially explain some variability across areas; this would reflect differences in population exposures, rather than variations in biological response.

While it is reasonable to expect some variation in effects across areas, staff observes that in combining the results for $PM_{2.5}$ from short-term exposure studies conducted across the U.S. and Canada for a range of health endpoints from mortality to varying indices of morbidity (Figure 3-11a), the effect estimates for each health endpoint are relatively consistent among the studies, which is similar to the pattern observed for PM_{10} studies in the last review (EPA, 1996b, Figure V-2). Effect estimates for associations with $PM_{2.5}$ are positive for all health endpoints, and many are statistically significant (Figure 3-11a). For mortality, effect estimates for total mortality fall within the same general range, especially when focusing on the results of studies

with greater precision, and effect estimates for cardiovascular and respiratory diseases are somewhat more variable than those for total mortality but are still generally consistent across the different locations. For morbidity, the effect estimates for hospital admissions or medical visits for both cardiovascular and respiratory diseases vary within a larger range, but that might be expected since results for specific causes (e.g., dysrhythmia, pneumonia) are grouped with results for all cardiovascular or all respiratory diseases in this presentation.

For $PM_{10-2.5,}$ fewer studies are available, and staff observes more evidence for consistency across morbidity effects than for mortality in the results of these studies (Figure 3-11b). The effect estimates for $PM_{10-2.5}$ with cause-specific mortality are generally positive, with several being statistically significant or nearly so, but only one of the effect estimates for $PM_{10-2.5}$ and total mortality is clearly positive and statistically significant. For hospital admissions/medical visits for respiratory and cardiovascular diseases, the associations with $PM_{10-2.5}$ are generally, but not always, positive, and the effect estimates vary somewhat in size, especially those from the preliminary analyses in Atlanta (Tolbert et al., 2000b). In Figures 3-11a and 3-11b, staff observes that the patterns of results for $PM_{2.5}$ and $PM_{10-2.5}$ are generally similar, though the evidence for consistency is greater for $PM_{2.5}$ than for $PM_{10-2.5}$, especially for mortality.

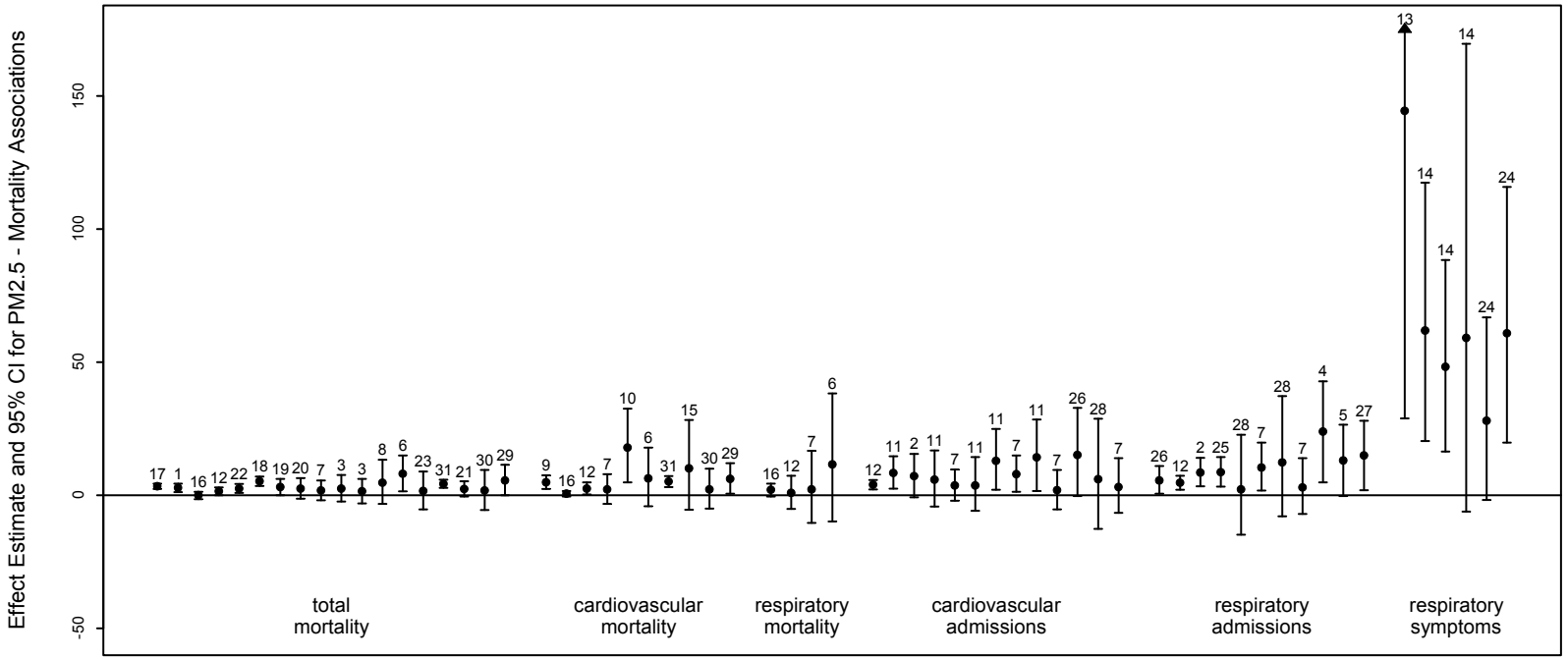


Figure 3-11a. Estimated excess mortality and morbidity risks per 25 µg/m^3 $PM_{2.5}$ from U.S. and Canadian studies (below). Results of GAM stringent reanalyses; studies not originally using GAM denoted by •. Multi-city studies denoted by ★.

1.★ Burnett and Goldberg, 2003, 8 Canadian cities
2. Burnett et al., 1997, Toronto •
3. Chock et al., 2000, Pittsburgh •
4. Delfino et al., 1997, Montreal •
5. Delfino et al., 1998, Montreal •
6. Fairley, 2003,, Santa Clara
7. Ito, 2003, Detroit
8. Klemm and Mason, 2000, Atlanta
7. Lipfert et al., 2000a, Philadelphia •
10. Mar et al., 2003, Phoenix
11. Metzger et al., 2003 •
12. Moolgavkar, 2003, Los Angeles Co.
13. Neas et al., 1995, Uniontown •
14. Neas et al., 1996, State College •
15. Ostro et al., 2003, Coachella Valley
16. Ostro et al., 1995, So. California •
17. ★ Schwartz 2003a, 6 cities overall
18. Schwartz 2003a, Boston
19. Schwartz 2003a, Kingston/Harriman
20. Schwartz 2003a, Portage
21. Schwartz 2003a, Steubenville
22. Schwartz 2003a, St. Louis
23. Schwartz 2003a, Topeka
24. ★ Schwartz and Neas, 2000, 6 cities •
25. Sheppard, 2003, Seattle
26. Stieb et al., 2000, St. John •
27. Thurston et al., 1994, Toronto •
28. Tolbert et al., 2000a, Atlanta •
29. Tsai et al., 2000, Newark •
30. Tsai et al., 2000, Elizabeth •
31. Tsai et al., 2000, Camden •

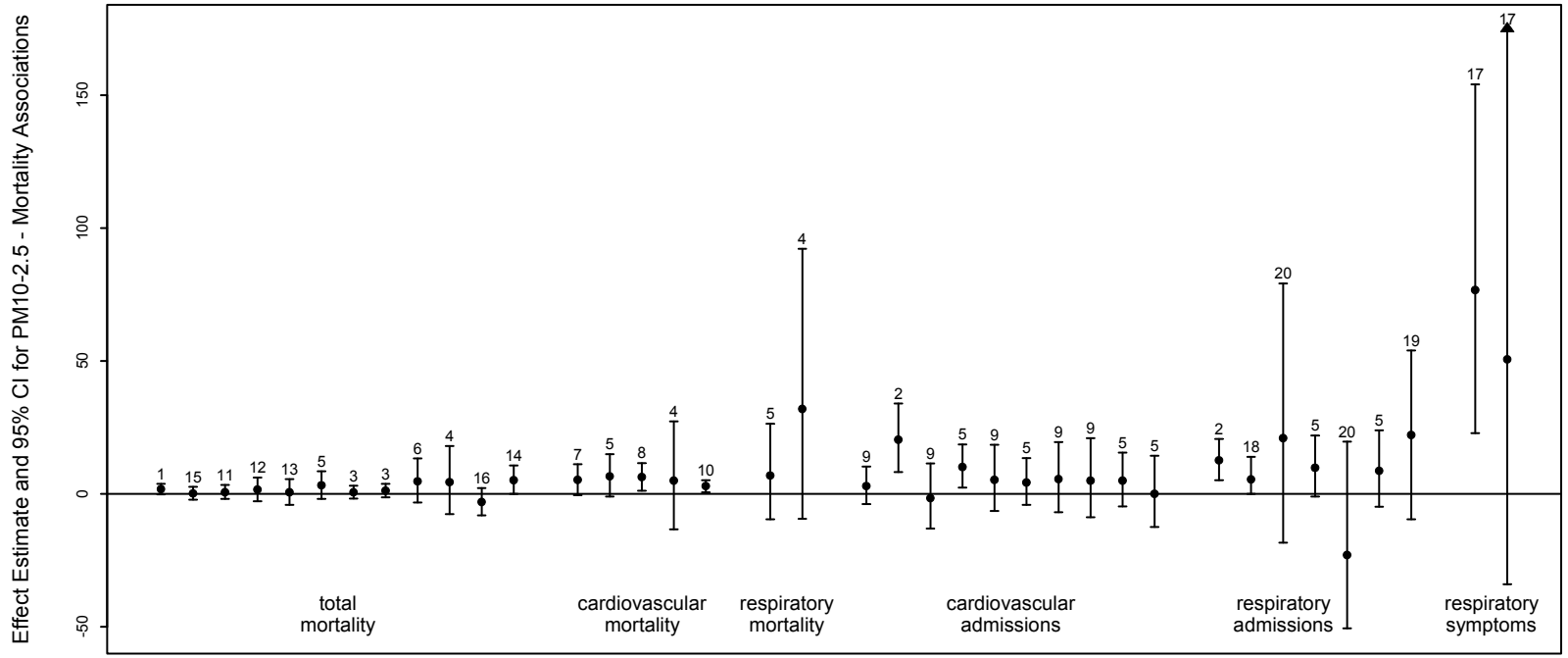


Figure 3-11b. Estimated excess mortality and morbidity risks per 25 µg/m^3 $PM_{10-2.5}$ from U.S. and Canadian studies (below). Results of GAM stringent reanalyses; studies not originally using GAM denoted by •. Multi-city studies denoted by ★.

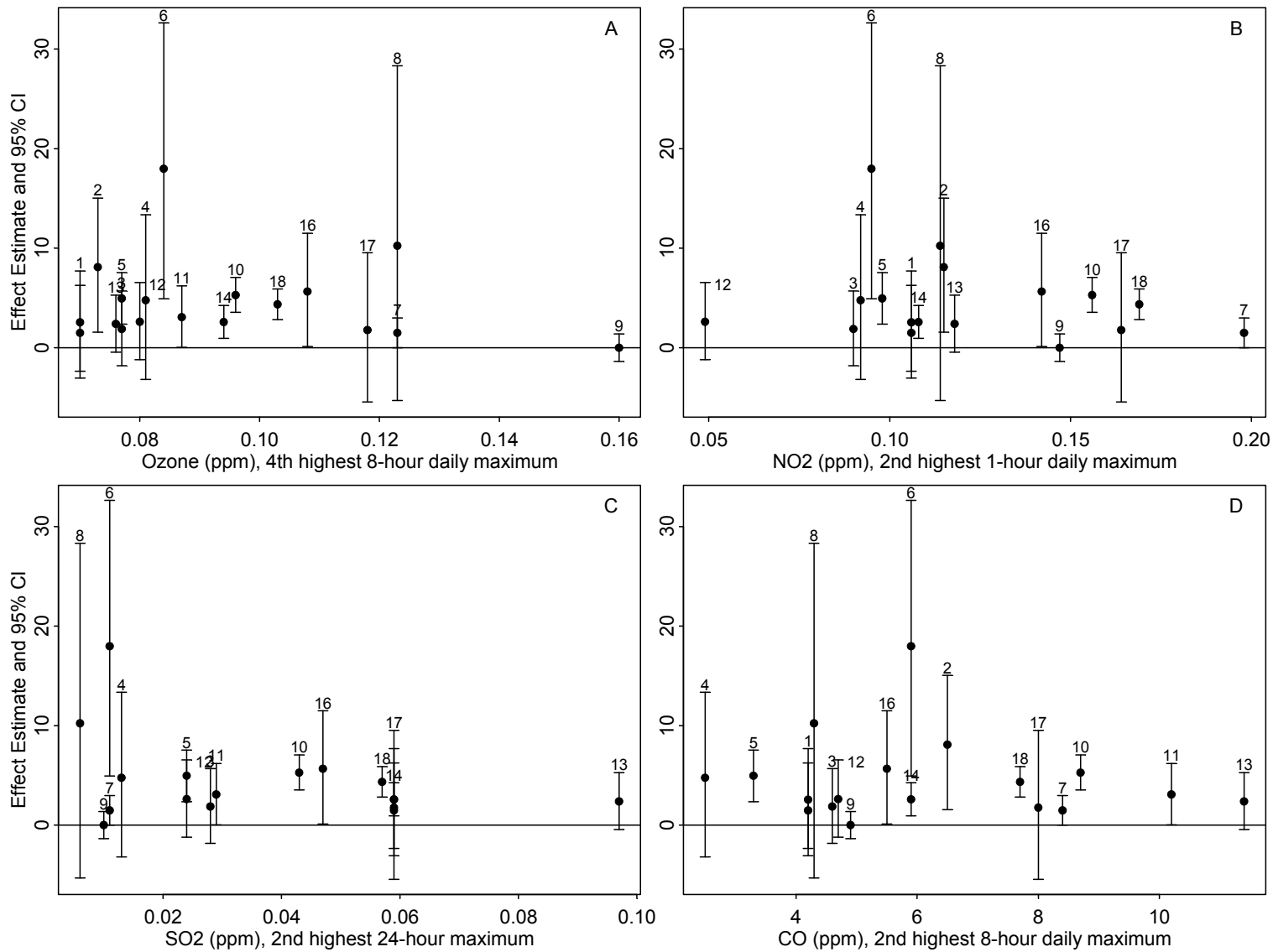
1.★ Burnett and Goldberg, 2003, 8 Canadian cities
2. Burnett et al., 1997, Toronto •
3. Chock et al., 2000, Pittsburgh •
4. Fairley, 2003,, Santa Clara
5. Ito, 2003, Detroit
6. Klemm and Mason, 2000, Atlanta
7. Lipfert et al., 2000a, Philadelphia •
8. Mar et al., 2003, Phoenix
9. Metzger et al., 2003 •
10. Ostro et al., 2003, Coachella Valley
11. Schwartz 2003a, Boston
12. Schwartz 2003a, Kingston/Harriman
13. Schwartz 2003a, Portage
14. Schwartz 2003a, Steubenville
15. Schwartz 2003a, St. Louis
16. Schwartz 2003a, Topeka
17. ★ Schwartz and Neas, 2000, 6 cities •
18. Sheppard, 2003, Seattle
19. Thurston et al., 1994, Toronto •
20. Tolbert et al., 2000a, Atlanta •

Another consideration for the consistency of the reported PM effects is the sensitivity of PM effect 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, the Staff Paper examined PM_{10} effect 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 effect 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. Effect estimates for PM_{10}-mortality associations were plotted against concentrations of gaseous pollutants in the study area, and there was no evidence that associations reported between PM_{10} and mortality were correlated with copollutant concentrations. (EPA, 1996b, Figure V-3a,b). Similarly, Figure 3-12 shows the reported effect estimates for $PM_{2.5}$ and mortality (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}, the magnitude and statistical significance of the associations reported between $PM_{2.5}$ and mortality in these studies 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 seen for PM_{10} in the 1996 Staff Paper, that is not appreciably modified by differing levels of the gaseous pollutants.

In addition, several new "intervention studies" have linked improvements in health with reduction in concentrations of PM and other pollutants. Staff considers these studies to reflect consistency in effect, in that findings of health improvement with reduced pollution is consistent with epidemiological evidence of such associations. Numerous epidemiologic studies in the Utah Valley area reported associations between daily PM_{10} concentrations and a range of health effects, from respiratory symptoms to mortality, and the investigators observed that respiratory hospital admissions and frequency of school absences decreased during a period when a major PM source (a steel mill) was closed (Pope et al., 1992; 1989). The draft CD also summarizes results from studies in Atlanta, Hong Kong, and East Germany, where policy or regulatory changes resulted in reduced air pollutant concentrations, with subsequent observations of

improvements in health, though it was difficult to distinguish effects of reductions in the individual pollutants. The draft CD concludes: "[t]aken together, these epidemiologic intervention studies tend to support the conclusion that reductions in ambient air pollution (especially PM) exposures resulted in decreased respiratory and cardiovascular health effects" (CD, p. 8-218)

Summary. New evidence from multi-city analyses has pointed to several new factors to recognize as potentially resulting in varying effect estimates for different locales. As in the previous review, staff recognizes that there are a number of variables that might be expected to result in different health risk estimates across communities. Overall, however, in evaluating the evidence from the many epidemiology studies, staff observes that there are fairly consistent associations between $PM_{2.5}$ and a range of effects, from respiratory symptoms to mortality from cardiopulmonary diseases. For $PM_{10-2.5}$, there is consistency across findings for morbidity, including respiratory symptoms and hospitalization for cardiopulmonary diseases; while there are some significant associations with mortality for heart or lung diseases, there is less evidence and less consistency in associations with total mortality.


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

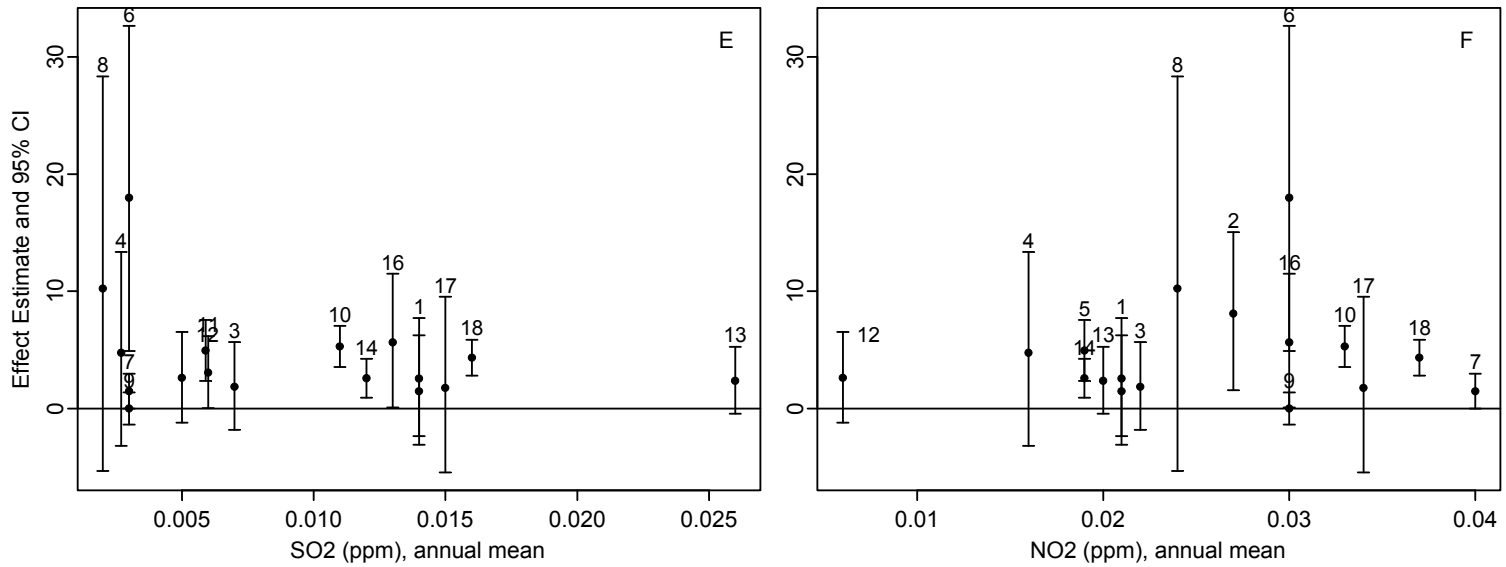


Figure 3-12. Associations between $PM_{2.5}$ and total mortality from U.S. studies, plotted against gaseous pollutant concentrations from the same locations. Air quality data obtained from the Aerometric Information Retrieval System (AIRS) for each study time period: (A) mean of 4th highest 8-hour ozone concentration; (B) mean of 2nd highest 1-hour NO_2 concentration; (C) mean of 2nd highest 24-hour SO_2 concentration; (D) mean of 2nd highest 8-hour CO concentration; (E) annual mean SO_2 concentration; (F) annual mean NO_2 concentration. Study locations are identified below (data in Appendix A)

1. Chock et al., 2000, Pittsburgh
2. Fairley, 2003, Santa Clara
3. Ito, 2003, Detroit
4. Klemm and Mason, 2000, Atlanta
5. Lipfert et al., 2000a
6. Mar et al., 2003, Phoenix
7. Moolgavkar, 2003
8. Ostro et al., 2003, Coachella Valley
9. Ostro et al., 1995, So. California
10. Schwartz, 2003a, Boston
11. Schwartz, 2003a, Knoxville
12. Schwartz, 2003a, Portage
13. Schwartz, 2003a, St. Louis
14. Schwartz, 2003a, Steubenville
15. Schwartz, 2003a, Topeka
16. Tsai et al., 2000, Camden NJ
17. Tsai et al., 2000, Elizabeth NJ
18. Tsai et al., 2000, Newark NJ

3.5.4.2 Coherence

The newly available epidemiologic and toxicological evidence substantially 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 limited evidence for 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).

Qualitative coherence is further supported by the new PM-related effects associations that have now been reported, including increased physicians' visits for respiratory causes and various new cardiovascular-related endpoints. These new findings serve to fill in the spectrum of observed effects from physiological changes that are linked to more serious health outcomes through premature mortality.

For respiratory endpoints, there is evidence of coherence in effects for both $PM_{2.5}$ and $PM_{10\text{-}2.5}$. As seen in Figure 3-11a for $PM_{2.5}$, associations with respiratory mortality are positive, but with wide confidence intervals, generally not reaching statistical significance. Associations between $PM_{2.5}$ and hospital admissions or medical visits for respiratory diseases are all positive, and especially for the more precise findings, a number of effect estimates are statistically significant. Effect estimates for $PM_{2.5}$ with respiratory symptoms are also all positive, with many also statistically significant. For $PM_{10\text{-}2.5}$, Figure 3-11b shows positive associations with respiratory mortality, hospital admissions or medical visits for respiratory disease and respiratory symptoms, except for the preliminary results from Tolbert et al. (2000b). Some associations,

particularly for respiratory admissions/visits, reach statistical significance, but the evidence is less consistent than that for $PM_{2.5}$.

For cardiovascular effects, the new epidemiologic and toxicologic evidence on discussed in section 3.3.2.3 has added important new evidence for coherence in effects on the cardiovascular system for $PM_{2.5}$ or PM_{10}. These studies include evidence on more subtle changes in cardiovascular health, such as levels of fibrinogen or C-reactive protein, or heart rate variability. The draft CD observes that the findings of increased levels of fibrinogen or plasma viscosity indicate a potential link between ambient PM, especially $PM_{2.5}$, 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 (CD, p. 9-128). For $PM_{2.5}$, the results on new cardiovascular health endpoints add to evidence of coherence for cardiovascular effects. Associations between $PM_{2.5}$ and cardiovascular mortality are all positive, and a number are statistically significant, as are associations with hospital admissions or medical visits for cardiovascular diseases (Figure 3-11a).

Less evidence is available for $PM_{10-2.5}$ for these new cardiovascular endpoints. Few studies of the more subtle cardiovascular health endpoints have included $PM_{10-2.5}$, and one study that did reported associations with $PM_{2.5}$ but not $PM_{10-2.5}$ (Peters et al., 2001). Associations between $PM_{10-2.5}$ cardiovascular mortality are positive and some reach statistical significance, and associations with cardiovascular admissions/visits are also generally positive, but the results are less consistent than those for fine-fraction particles (Figure 3-11b). Thus, while there is evidence that suggests associations between coarse-fraction particles and cardiovascular health effects, the evidence is limited and results are not as consistent as those for fine-fraction particles.

The new evidence contributes to the quantitative coherence observed in the last review. In the 1996 Staff Paper, results from studies linking short-term PM_{10} exposure with an array of health endpoints were plotted together, and showed coherence in the findings across the various endpoints. This pattern continues when adding results from the newer studies of short-term PM_{10} exposures. In NMMAPS, 1.1% and 1.6% increases in total and cardiorespiratory mortality, respectively, were reported for a 50 $\mu g/m^3$ increase in daily PM_{10} (in GLM reanalyses) while increases in hospital admissions of 5% (for cardiovascular causes, with a range across other

studies of approximately 3% to 6%) and 8% (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 up to a 75% increase for a 50 $\mu g/m^3$ increase in 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 (Table 9-13), considered together with these illustrative ranges of effect estimates (and with the ranges shown above in Figures 3-4 through 3-9), continue to show that the quantitative coherence across all PM-related endpoints, especially for PM_{10} is consistent with expectations (CD, p. 9-128). Staff observes that these ranges are primarily drawn from single-pollutant model results, and the ranges might differ somewhat if including results of multi-pollutant studies, though the coherence across effect categories would not be expected to change.

Data now also permit an examination of quantitative coherence for effects associated with $PM_{2.5}$ and $PM_{10-2.5}$ (Figures 3-11a and 3-11b). As summarized in the CD, effect estimates for a 25 $\mu g/m^3$ change in $PM_{2.5}$ with mortality range from 2-3% for total mortality from multi-city studies, 2-6% for total mortality in single-city studies, and 2-20% for mortality from cardiovascular or respiratory diseases. From single-city studies, effect estimates generally range from about 1% to 10% per 25 $\mu g/m^3$ $PM_{2.5}$ for cardiovascular admissions and for respiratory admissions, from about 5% to 25% per 25 $\mu g/m^3$ $PM_{2.5}$. Larger effect estimates are reported with respiratory symptoms, ranging up to 150% increase in symptoms per 25 $\mu g/m^3$ $PM_{2.5}$. As described earlier in section 3.3, effect estimates with $PM_{10-2.5}$ with differing health endpoints are of about the same magnitude as those for $PM_{2.5}$, though, as has been stated earlier, the findings are less often statistically significant for $PM_{10-2.5}$ than for $PM_{2.5}$ and less consistent for mortality.

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). As discussed above, 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). Several recent toxicologic and clinical human exposure studies have used particulate matter collected during the same time periods, and in all studies, more health effects were found with exposure to PM collected when the steel mill was operating than with PM collected while the mill was closed (CD, p. 8-215). In addition, the 1996 Staff Paper observed that a series of epidemiologic studies conducted in cities such as Detroit, Birmingham or Utah Valley reported associations with a range of respiratory and cardiovascular effects (EPA, 1996b, p. V-57). These studies generally reported associations with PM_{10}, and a number of recent studies have expanded upon this evidence with findings of associations with a series of morbidity and mortality effects in individual cities, such as Minneapolis or Pittsburgh (see Chapter 4 discussion of city selection for risk assessment). For $PM_{2.5}$ (or PM_1), there have been findings of significant associations with multiple respiratory health endpoints, including hospital admissions and emergency room visits for asthma, and increased asthma symptoms. In Boston, associations have been reported with mortality, respiratory symptoms and several cardiovascular health indicators, ranging from increased risk of myocardial infarction to decreases in heart rate variability.

Summary: These observations strongly underscore coherence in the array of health effects for which associations with exposure to ambient PM have now been reported, from subtle changes in cardiac and lung function to increased aggravation of heart and lung diseases to increases in utilization of health care facilities to increased mortality from cardiorespiratory diseases. $PM_{2.5}$ has been associated with effects on both the respiratory and cardiovascular systems, ranging from the more subtle effects, such as changes in lung function or levels of C-reactive protein, to serious effect such as mortality. For $PM_{10-2.5}$, there is evidence for coherence in effects on the respiratory system, with less consistent evidence for effects on the cardiovascular system. 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, provide increased support for a causal link between PM and effects on the cardiovascular and respiratory systems (CD, p. 9-67).

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4. CHARACTERIZATION OF HEALTH RISKS

4.1 INTRODUCTION

This chapter describes and presents the initial results from an updated PM health risk assessment that is being conducted for EPA's current review of the PM NAAQS. This updated risk assessment builds upon the methodology used in the more limited PM risk assessment (summarized below) that was conducted as part of EPA's prior PM NAAQS review. This updated assessment focuses on (1) the risks of mortality, morbidity, and symptoms associated with recent ambient $PM_{2.5,}$ $PM_{10-2.5}$, and PM_{10} levels, (2) the risk reductions associated with just meeting the current suite of $PM_{2.5}$ NAAQS, and (3) the risk reductions associated with just meeting various alternative $PM_{2.5}$ standards and a range of $PM_{10-2.5}$ standards, consistent with ranges of standards recommended by staff for consideration. The third component of the risk assessment, focusing on alternative $PM_{2.5}$ and $PM_{10-2.5}$ standards, will be included in the next draft of this Staff Paper, based on the preliminary staff recommendations on possible alternative standards contained in Chapter 6 of this draft Staff Paper and taking into account comments received from CASAC and the public on those preliminary recommendations.

As discussed in Chapter 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 in the 1996 Staff Paper that fine- and coarse-fraction particles should 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 Chapter 8 of the draft CD, continue to show associations between serious health effects, including premature mortality, and fine-fraction PM, but they also offer new evidence indicating associations between coarse-fraction PM and health effects.

In June 2001, OAQPS released a draft document, *PM NAAQS Risk Analysis Scoping Plan*, (EPA, 2001c) describing EPA's overall plan for conducting the PM health risk assessment for the current review. The CASAC PM Panel provided feedback on this draft plan in a consultation held July 24, 2001, and the Agency also received comments from the general

public. In January 2002, EPA released a draft document, *Proposed Methodology for Particulate Matter Risk Analyses for Selected Urban Areas*, (Abt Associates, 2002a) for public and CASAC review. This draft document described EPA's plans to conduct a risk assessment for $PM_{2.5}$-related risks for several health endpoints, including mortality, hospital admissions, and respiratory symptoms, and $PM_{10-2.5}$-related risks for hospital admissions and respiratory symptoms. The CASAC PM Panel discussed this draft document in a February 27, 2002 teleconference and provided its comments in a May 23, 2002 Advisory letter to EPA's Administrator (Hopke, 2002). OAQPS also received several comments from the public. In its May 23, 2002 Advisory, the CASAC PM Panel "concluded that the general methodology as described in the report is appropriate. ... Thus, the general framework of the approach is the sensible approach to this risk analysis" (Hopke, 2002). Among its comments, the CASAC Panel suggested extending the risk assessment to include PM_{10} as an indicator in the risk assessment, since many health studies used PM_{10} as an indicator and PM_{10} air quality data are generally available (Hopke, 2002). Risks associated with PM_{10} ambient levels are likely to reflect the contribution of $PM_{2.5}$, $PM_{10-2.5}$, or some combination of both depending on the relative composition of PM in various urban areas. The CASAC Panel also offered a number of comments related to the scope and details of the proposed risk assessment.

In response to a request from CASAC to provide additional details about the proposed scope of the $PM_{10-2.5}$ and PM_{10} components of the planned risk assessment, in April 2003 EPA released a draft memorandum (Abt, 2003a) to the CASAC and the public addressing this topic. On May 1, 2003, the CASAC PM Panel held a consultation with EPA to provide advice on staff plans for conducting the $PM_{10-2.5}$ and PM_{10} components of the health risk assessment. OAQPS has carefully considered these comments in preparing the risk assessments described in this Chapter. These comments also are addressed further in a draft technical report (Abt Associates, 2003b) which provides additional details about the PM risk assessment.[1]

[1]We hereafter refer to the "PM risk assessment" unless reference to a specific PM indicator (e.g., $PM_{2.5}$) is required. The current PM risk assessment considers each of three PM indicators – $PM_{2.5}$, $PM_{10-2.5}$, and PM_{10} – in turn.

4.1.1 Goals for Updated PM Risk Assessment

The goals of the updated PM risk assessment are (1) to develop a better understanding of the influence of various inputs and assumptions on the risk estimates and (2) to gain insights into the nature of the risks associated with exposures to ambient PM. In addition, the risk assessment provides a rough sense of the potential magnitude of PM-associated mortality and morbidity associated with current $PM_{2.5}$, $PM_{10-2.5}$, and PM_{10} levels and with attaining the current suite of $PM_{2.5}$ NAAQS (as well as alternative $PM_{2.5}$ and $PM_{10-2.5}$ standards identified as part of this review) in specific urban areas. The staff recognizes that due to the many sources of uncertainty inherent in conducting the PM risk assessment, the resulting PM risk estimates should not be viewed as precise measures of the health impacts now occurring or anticipated to occur in the future in any given location or nationally. Further, the staff recognizes that the role of the risk assessment in this standards review will necessarily be limited by the significant uncertainties associated with this assessment, discussed in section 4.2.7 below.

4.1.2 Summary of Risk Assessment Conducted During Prior PM NAAQS Review

For the prior review, EPA conducted a health risk assessment that estimated population risk for two defined urban study areas: 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 (C-R) 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.[2] 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 assessment 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. In addition, the assessment

[2] "As is" PM concentrations are defined here as a recent year of air quality.

included sensitivity analyses and integrated uncertainty analyses to better understand the influence of various inputs and assumptions on the risk estimates. The methodological approach followed in conducting the prior risk assessment is described in Chapter 6 of the 1996 Staff Paper (EPA, 1996b) and in several technical reports (Abt Associates, 1996; Abt Associates, 1997a,b) and publications (Post et al., 2000; Deck et al., 2001).

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

- EPA placed greater weight on the overall qualitative 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 C-R 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 assessment presented reasonable estimates as to the possible extent of risk for these effects given the available information (62 FR at 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 assessment. 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 at 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 at 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 at 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 at 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: the use of C-R functions from multi-pollutant, rather than single-pollutant models; the choice of approach to adjusting the slope of the C-R functions in analyzing alternative cutpoints (i.e., hypothesized thresholds); the value chosen to represent average annual background PM concentrations; and the choice of approach to adjusting air quality distributions for simulating attainment of alternative $PM_{2.5}$ standards (EPA, 1996b).

4.2 GENERAL SCOPE OF PM RISK ASSESSMENT

As discussed in Chapter 3 above, the draft CD concludes that "the overall weight of evidence, based on information concerning PM exposure, dosimetry, toxicology, and epidemiology, supports the conclusion that PM, especially fine PM, is the primary contributor to a variety of adverse health effects associated with air pollution. However, difficult technical issues still remain in further separating the effects of fine and coarse particles and in delineating respective contributions of PM acting alone or in combination with gaseous co-pollutants in increasing risks of health effects anticipated to occur in response to exposures to contemporary particle-containing ambient air mixes in the U.S." (CD, p. E-44) For coarse-fraction particles the strongest evidence is found relating $PM_{10-2.5}$ ambient concentrations and increased respiratory hospital admissions and respiratory symptoms. While not as strong as the case for $PM_{2.5}$ and premature mortality, there is sufficient evidence to suggest that $PM_{10-2.5}$ also is likely to be linked with premature mortality. The draft CD finds that the expanded body of evidence substantiates associations between $PM_{2.5}$ and mortality, with some studies suggesting associations between $PM_{10-2.5}$ and mortality as well (CD, p. 8-57). Although mortality effects for $PM_{10-2.5}$ were not included in OAQPS's proposed Risk Analysis Scoping Plan (EPA, 2001c), such effects have been added consistent with advice provided by CASAC PM Panel members in the May 1, 2003 consultation with staff.

The updated risk assessment being conducted for the current NAAQS review is premised on the assumption that each of the ambient PM indicators (i.e., $PM_{2.5}$, $PM_{10-2.5}$, and PM_{10}) are

causally related to the mortality, morbidity, and symptomatic effects (alone and/or in combination with other pollutants) observed in the epidemiological studies. Staff concludes that this assumption is well supported by the evaluation contained in the draft CD and is consistent with the advice provided by the CASAC PM Panel. However, staff recognizes that there are varying degrees of uncertainty associated with whether or not there is a causal relationship for each of the three PM indicators and the various health endpoints and that the degree of uncertainty is directly related to differences in the relative weight of evidence.

This PM risk assessment focuses on selected health endpoints such as increased excess daily mortality and mortality associated with long-term exposure, increased hospital admissions for respiratory and cardiopulmonary causes, and increased respiratory symptoms for children. A consequence of limiting the assessment to selected health endpoints is that the risk estimates likely understate the type and extent of potential health impacts of ambient PM exposures. Although the risk assessment does not address all health effects for which there is some evidence of association with exposure to PM, all such effects are identified and considered previously in Chapter 3.

Like the prior risk assessment done as part of the last review (EPA, 1996b), this current updated risk assessment uses C-R 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) and Chapter 3 (section 3.5.3.3), measurements of daily variations of ambient $PM_{2.5}$ concentrations, as used in the time-series studies that provide the C-R relationships for this assessment, 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 ambient PM concentrations (i.e., those measured at ambient monitoring stations) are a useful surrogate for exposure to ambient PM (CD, p. 9-117, p. 8-252). A more detailed discussion of the possible impact of exposure misclassification on the estimated C-R 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 PM risk estimates for this review, 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 O_3 and CO, 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. As with the prior PM risk assessment, contributions to health risk from non-ambient sources are beyond the scope of the proposed risk assessment for this NAAQS review.

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

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

- analyze risks for a recent 12-month period of air quality (labeled "as is") and for a scenario in which air quality just meets the current set of standards.

- analyze risks for scenarios in which air quality is simulated to just meet potential alternative standards that are recommended by staff for consideration.

- estimate risks only for concentrations exceeding estimated background levels or the lowest measured level (LML) observed in the study, if it is higher than the estimated background level in the assessment location.[3]

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

Both the prior and the current PM risk assessment focus on health endpoints for which C-R functions have been estimated in epidemiological studies. Since these studies estimate C-R functions using air quality data from fixed-site, population-oriented monitors, the appropriate application of these functions in a PM risk assessment 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 current risk assessment includes risk estimates for 9 urban areas for $PM_{2.5}$, 5 urban areas for $PM_{10\text{-}2.5}$, and 12 urban areas for PM_{10}. These areas have been chosen based on availability of PM C-R relationships and adequate PM air quality data. The selection of these areas also reflects a desire to include areas from the various regions of the United States to the extent possible in order to reflect regional differences in the composition of PM and other factors (e.g., different levels of co-pollutants, air-conditioning use).

Finally, estimates of risks above background PM concentrations are provided for short-term exposure health endpoints because they are judged to be more relevant to policy decisions about the NAAQS than estimates that include risks potentially attributable to uncontrollable background PM concentrations. For long-term exposure mortality associated with $PM_{2.5}$, the LMLs for the $PM_{2.5}$ epidemiology studies are 10 and 11 $\mu g/m^3$, which are higher than the range of estimated $PM_{2.5}$ background levels in either the East or West. Estimating risks outside the range of the original epidemiology studies that were the source of the C-R functions would introduce significant additional uncertainties into the risk assessment. Therefore, the risks

[3]A C-R relationship estimated by an epidemiological study may not be representative of the relationship that exists outside the range of concentrations observed during the study. To partially address this problem, risk was not calculated for PM levels below the lowest measured level (LML) in the study, if reported. The LML's for each study that provided a C-R relationship for the current PM risk assessment, where reported, are provided in Appendix C of Abt (2003b).

associated with long-term exposure were only estimated in excess of the LML. Since we do not estimate risks below the LML, the overall long-term exposure mortality risks may be underestimated if annual average $PM_{2.5}$ concentrations below the LMLs contribute to long-term exposure mortality.

The following sections provide an overview of the components of the risk model, describe the selection of urban areas and health endpoints included in the PM risk assessment, discuss each of the major components of the risk model, address characterization of uncertainty and variability associated with the risk estimates, and summarize the currently available PM risk estimates. A separate draft technical report (Abt Associates, 2003b) also is available which provides a more detailed discussion of the risk assessment methodology and risk estimates.

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 or set of counties attributable to ambient $PM_{2.5}$ (or $PM_{10-2.5}$ or PM_{10}) exposures in excess of background and the change in incidence of the health effect in that county or set of counties corresponding to a given change in $PM_{2.5}$ (or $PM_{10-2.5}$) levels resulting from just meeting a specified set of $PM_{2.5}$ (or $PM_{10-2.5}$) standards, the following three elements are required:

- air quality information including: (1) "as is" air quality data for $PM_{2.5}$, $PM_{10-2.5}$, and PM_{10} from population-oriented monitors for the selected location, (2) estimates of background $PM_{2.5}$, $PM_{10-2.5}$, and PM_{10} 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 area just meets a given set of $PM_{2.5}$ (or $PM_{10-2.5}$) standards.

- relative-risk based C-R functions (preferably derived in the assessment location) which provide an estimate of the relationship between the health endpoints of interest and ambient PM concentrations.

- annual or seasonal baseline health effects incidence rates and population data which are needed to provide an estimate of the annual or seasonal baseline incidence of health effects corresponding to "as is" PM levels.

Figure 4-1 provides a broad schematic depicting the role of these components in the risk assessment. 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 summary description of the type of sensitivity analyses performed is included later in section 4.2.7 (See Table 4-9). Each of the three key components (i.e., air quality information, estimated PM-related C-R functions, and baseline incidence) are discussed below, highlighting those points at which judgments have been made.

4.2.2 Criteria for Selection of Health Endpoints and Urban Study Areas

Only two urban counties were included in the risk assessment conducted for the prior PM NAAQS review due to the very limited number of urban areas that had sufficient recent $PM_{2.5}$ ambient air quality monitoring data and because of the limited number of epidemiological studies that directly measured $PM_{2.5}$. As discussed in more detail in Chapter 3, since the last review, a significant number of epidemiological studies have been published examining a variety of health effects associated with ambient $PM_{2.5}$, $PM_{10-2.5}$, and PM_{10} in various urban areas throughout the U.S. and Canada, as well as Europe and other parts of the world. Tables 9-8 and 9-10 in the draft CD summarize the available U.S. and Canadian short-term exposure studies that provide effect estimates for PM (i.e., $PM_{2.5}$, $PM_{10-2.5}$, and PM_{10}) for mortality and morbidity endpoints,
respectively. Table 9-11 summarizes the available U.S. and Canadian studies that provide effect estimates for mortality and other health endpoints for long-term exposure to $PM_{2.5}$ and other PM indicators. While a significant number of new epidemiological studies also have been published since the last review, and are evaluated in the draft CD, the PM risk assessment relies only on U.S. studies given the possible differences in population and characteristics of PM and co-pollutants between the U.S. and these other locations. The approach and criteria that EPA has used to select the health endpoints and urban areas to include in the risk assessment for the three PM indicators are described below.

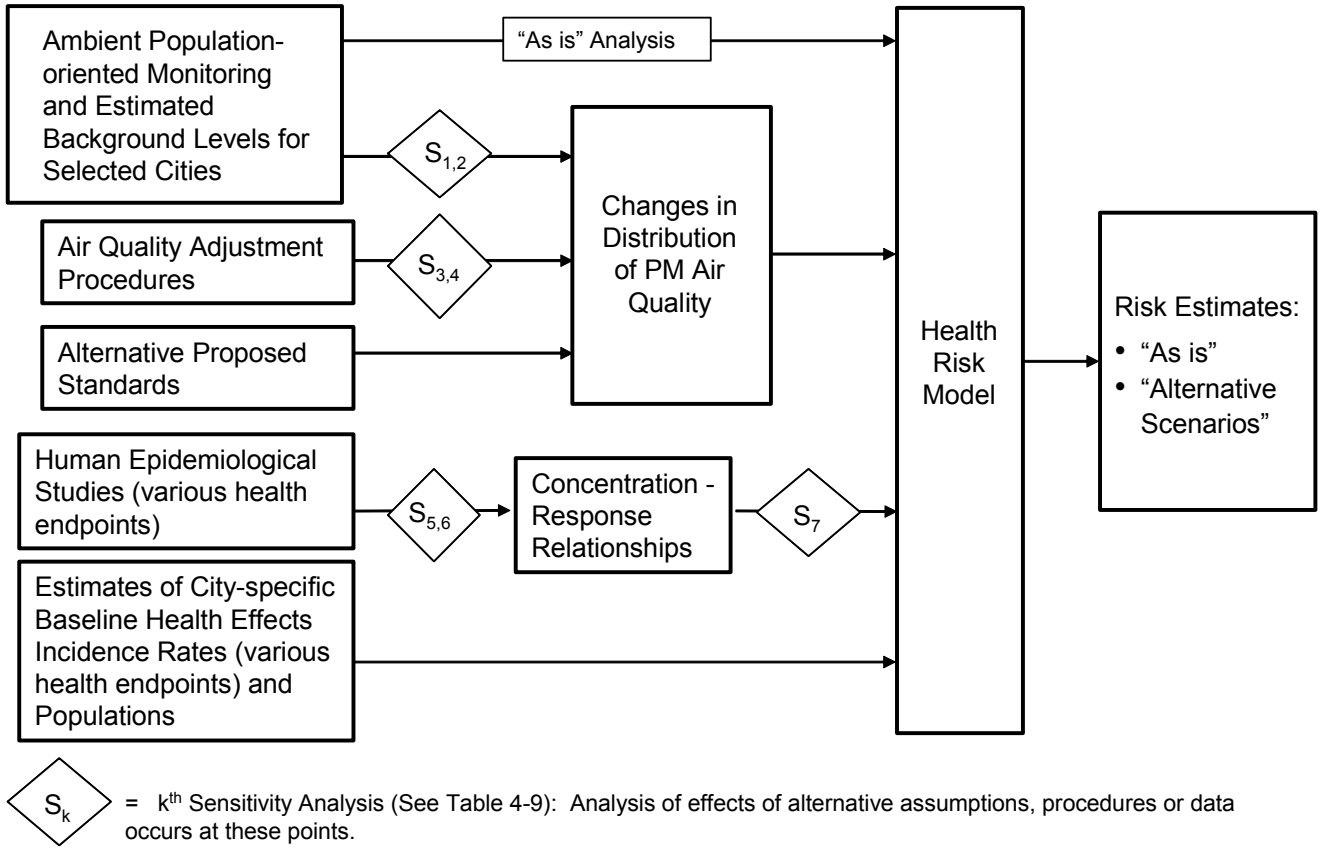

Figure 4-1. Major Components of Particulate Matter Health Risk Assessment

4.2.2.1 Selection of Health Endpoint Categories

OAQPS staff carefully reviewed the health effects evidence evaluated in the draft CD in order to identify potential health effect categories to include in the current PM risk assessment. Given the large number of endpoints and studies addressing PM-related effects, staff recommended for inclusion in the PM risk assessment only the more severe and better understood (in terms of health consequences) health endpoint categories for which the weight of the evidence supports the existence of a relationship between PM and the health effects category.

For the health effect categories included in the current PM risk assessment, the risk assessment assumes that a causal relationship exists. On this topic the draft CD states,

> ... although associations of PM with harmful effects continue to be observed consistently across most new studies, the newer findings do not fully resolve issues concerning relative contributions to the observed epidemiological associations of (a) PM acting along, (b) PM acting in combination with gaseous co-pollutants, (c) the gaseous pollutants per se, and (d) the overall ambient mix. ... However, with considerable new experimental evidence now in hand, it is possible to hypothesize various ways in which ambient exposure to PM acting alone or in combination with other co-pollutants can plausibly be involved in the complex chain of biological events leading to harmful health effects in the human population. This newer experimental evidence, coupled with new exposure analyses results, adds much support for interpreting the epidemiological findings discussed here as likely being indicative of causal relationships between exposures to ambient PM (or specific size or chemical components) and consequent associated increased mortality and morbidity effects.(CD, p. 9-70)

For the three PM indicators ($PM_{2.5}$, PM_{10}, $PM_{10-2.5}$), staff considered only those endpoint categories which provided C-R relationships based on U.S. and Canadian studies that used PM concentrations obtained by one of the following approaches: (1) directly measuring the fine fraction of PM using $PM_{2.5}$ or $PM_{2.1}$, (2) estimating the fine fraction using nepholometry data, (3) directly measuring PM_{10}, and (4) estimating $PM_{10-2.5}$ concentrations based on co-located PM_{10} and $PM_{2.5}$ monitors or based on measurements using dichots.

The staff selected health effect endpoints for each of the three PM indicators based on whether the weight of the evidence available in the draft CD, taking into account advice from CASAC, supports the assumption that the health effects are causally related to the PM indicators. 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 admissions 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 $PM_{2.5}$.

Based on a review of the evidence evaluated in the draft CD and the criteria discussed above, staff included the following broad categories of health endpoints associated with short-term exposures in the risk assessment for all three PM indicators:

- non-accidental total, cardiovascular, and respiratory mortality;
- hospital admissions for cardiovascular and respiratory causes;
- respiratory symptoms not requiring hospitalization

In addition, total, cardiopulmonary, and lung cancer mortality due to long-term exposure are included in the $PM_{2.5}$ risk assessment. Other effects reported to be associated with PM, including, but not limited to, decreased lung function, changes in heart rate variability, and increased emergency room visits are addressed in Chapter 3, but are not included in the quantitative risk assessment.

4.2.2.2 Selection of Study Areas

A primary goal of the current PM risk assessment has been to identify and include urban areas in the U.S. for which epidemiological studies are available that estimate C-R relationships for those locations. This goal is in large part motivated by the evidence from the NMMAPS (Samet et al., 2001) that suggests there is geographic variability in C-R relationships across different urban areas in the U.S. The selection of urban areas to include in the PM risk assessment was based on the following criteria:

- An area had sufficient air quality data for a recent year (1999 or later). Sufficient $PM_{2.5}$ or PM_{10} air quality data is defined as having at least one PM monitor at which there are at least 11 observations per quarter for a one year period.[4] Sufficient air quality data for $PM_{10-2.5}$ is defined as a one year period with at least

[4]For $PM_{2.5,}$ an additional requirement was that a city had to have at least 122 days of data (i.e., equivalent to 1 in 3 day monitoring) for a recent year of air quality to be included.

11 daily values per quarter based on data from co-located $PM_{2.5}$ and PM_{10} monitors.[5,6]

- An area is the same as or close to the location where at least one C-R function for one of the recommended health endpoints has been estimated by a study that satisfies the study selection criteria (see below).

- An area is one in which studies exist that had relatively greater precision, as indicated by a relatively greater number of effect-days observations.

- For the hospital admission effects category relatively recent area-specific baseline incidence data, specific to International Classification of Disease (ICD) codes, are available.

For the $PM_{2.5}$ risk assessment, staff focused on selecting urban areas based primarily on a location's having non-accidental total and cause-specific mortality $PM_{2.5}$ C-R functions since this was the largest data base in terms of number of studies in different locations. Staff then supplemented this by consideration of other morbidity endpoints (e.g,, hospital admissions). Based on a review of studies listed in Tables 9-8 and 9-10 of the draft CD (see also Appendix A of this SP), a candidate pool of 17 urban locations was initially suggested based on short-term exposure mortality studies (16 of the candidate locations); Seattle was added based on a hospital admissions study.[7]

Staff next considered an indicator of study precision for the short-term exposure mortality studies identified in the first step. As discussed above in Chapter 3 (section 3.3.1.1.1) and in Chapter 8 of the draft CD, the natural logarithm of the mortality-days (a product of each city's daily mortality rate and the number of days for which PM data were available) can be used as an indicator of the degree of precision of effect estimates; studies with larger values for this

[5]The criterion of at least 11 observations per quarter is based on EPA guidance on measuring attainment of the daily and annual PM standards outlined in Appendix N of the July 18, 1997 Federal Register notice.

[6]To be consistent with the epidemiological studies which generally used only population-oriented monitors, any monitors where the monitoring objective was listed as "highest concentration monitor" were excluded from consideration. The few monitors excluded were in industrial or commercial areas and are intended to characterize local conditions near major point sources.

[7]The Tolbert et al. (2000) study in Atlanta was excluded from consideration because it presented only preliminary results, and because the draft CD urged caution in interpreting these preliminary results.

indicator should be accorded relatively greater study weight. As discussed previously in Chapter 3, the draft CD notes that based on an analysis of the 90-cities NMMAPS data, there are "generally narrower confidence intervals for more homogeneously positive effects estimates as study size increases beyond about ln (mortality-days) = 9.0" (CD, Figure 8-21, p. 8-244). Based on this observation, staff considered only those urban areas in which studies with relatively greater precision were conducted, specifically including studies that have a natural log of mortality-days greater than or equal to 9.0 for total non-accidental mortality.[8] As a result of applying this criterion, six urban areas were excluded as potential study areas (Camden, NJ; Coachella Valley, CA; Elizabeth, NJ; Newark, NJ; Steubenville, OH; and Topeka, KS).

Finally, staff considered which of the potential study locations identified from steps 1 and 2 above also had sufficient $PM_{2.5}$ ambient monitoring data. A location was considered to have sufficiently complete air quality data if it had at least one monitor at which there were at least 11 observations per quarter and at least 122 observations per year (i.e., equivalent to 1 in 3 day monitoring). This final criterion excluded two of the remaining potential study areas (Knoxville, TN and Portage, WI), leaving eight cities in which epidemiological studies reported C-R relationships for $PM_{2.5}$ and mortality and which had sufficient air quality data in a recent year.

Eight of the nine urban areas, excluding Seattle, are included in the $PM_{2.5}$ risk assessment based on short-term exposure mortality. Since the studies reporting C-R functions for $PM_{2.5}$-related mortality associated with long-term exposure were conducted for multiple cities across the U.S., the issue of matching risk assessment locations with city-specific studies did not arise. Therefore, the $PM_{2.5}$ risk assessment for long-term exposure mortality was conducted for these same eight urban areas.

Most of the morbidity and respiratory symptom studies reporting $PM_{2.5}$-related effects were conducted in the same set of locations as the short-term exposure mortality studies. In considering these other health endpoints, staff applied similar criteria (i.e., studies providing effects estimates with relatively greater precision and availability of recent and adequate $PM_{2.5}$

[8]Most of the epidemiological studies reporting total non-accidental mortality also report on one or more cause specific mortality categories. In such studies, the natural log of mortality days is often less than 9.0 because there are fewer deaths from a specific cause. We included cause-specific mortality C-R functions from such studies, as long as the natural log of total mortality-days was greater than or equal to 9.0.

ambient air quality data). In addition, for the hospital admissions effect category, staff limited selection to those urban areas where the necessary baseline incidence data could be obtained.

Based on applying the above criteria and considerations, the health endpoints and urban locations selected for the $PM_{2.5}$ risk assessment are summarized in Tables 4-1 and 4-2, for mortality and morbidity endpoints, respectively. These tables also list the specific studies that provided the estimated C-R functions used in the $PM_{2.5}$ risk assessment. More detailed information on the studies selected can be found in Exhibit C of the PM risk assessment technical support document (Abt Associates, 2003b).

The selection of urban areas to include for the $PM_{10-2.5}$ risk assessment was based on examining the pool of epidemiological studies reporting associations for $PM_{10-2.5}$ in any of the urban areas already selected for the $PM_{2.5}$ risk assessment. As summarized in Tables 4-3 and 4-4, the $PM_{10-2.5}$ risk assessment is more limited because of the more limited air quality data (requiring co-located $PM_{2.5}$ and PM_{10} monitors or availability of dichot data) as well as the smaller number of studies. As noted previously, consistent with advice from the CASAC PM Panel, short-term exposure mortality was included among the health effects in the $PM_{10-2.5}$ risk assessment. The areas included for short-term exposure mortality in the $PM_{10-2.5}$ risk assessment are Detroit, Philadelphia, Phoenix, and St. Louis.[9] In addition, increased hospital admissions associated with $PM_{10-2.5}$ were estimated for Detroit and Seattle, and increased respiratory symptoms were estimated for St. Louis.

In addition to the criteria listed above, selection of areas to include in the PM_{10} risk assessment was guided by the desire to include urban areas that would further inform comparisons both across the PM indicators (i.e., $PM_{2.5}$, $PM_{10-2.5}$) and across health effect categories. Staff also wanted to include, at a minimum those urban areas already selected for the $PM_{2.5}$ risk assessment, for which city-specific C-R functions for short-term exposure mortality are available from the NMMAPS and/or other studies. Tables 4-5 and 4-6 summarize the urban locations selected for the PM_{10} risk assessment for the mortality and morbidity health endpoints, respectively. Similar to the prior tables, the studies providing the C-R relationships also are identified. Additional details about the epidemiological studies and the C-R functions used in

[9]For Phoenix, there were not sufficient data within AIRS to calculate $PM_{10-2.5}$ concentrations in any of the years from 1999 through 2002. Instead, we used data from 1997, based on co-located TEOM monitors. Because of this, the $PM_{10-2.5}$ risk assessment in Phoenix is treated as a separate special case in the PM risk assessment.

the risk assessment from these studies are provided in Appendix A of Chapter 3 of this draft Staff Paper and in Appendix C of the risk assessment technical support document (Abt, 2003b), respectively.

4.2.3 Air Quality Considerations

As mentioned earlier, air quality information required to conduct the PM risk assessment includes: (1) "as is" air quality data for $PM_{2.5}$, $PM_{10-2.5}$, and PM_{10} from population-oriented monitors for each selected location, (2) estimates of background $PM_{2.5}$, $PM_{10-2.5}$, and PM_{10} concentrations appropriate for each location, and (3) a method for adjusting the "as is" data to reflect patterns of air quality estimated to occur when an area just meets a given set of $PM_{2.5}$ (or $PM_{10-2.5}$) standards. OAQPS retrieved ambient air quality data for $PM_{2.5}$ and PM_{10} for the potential study areas for the years 1999 through 2002 from EPA's Aerometric Information Retrieval System (AIRS). Urban areas were only included in the risk assessment if there was at least one monitor with 11 or more observations per quarter. Staff calculated $PM_{10-2.5}$ concentrations from co-located $PM_{2.5}$ and PM_{10} monitors that met the minimum observation cutoff. The most recent year of PM data was used for each study area and PM indicator subject to meeting this requirement. Table 4-7 provides a summary of the $PM_{2.5}$, $PM_{10-2.5}$, and PM_{10} ambient air quality data for the urban study areas included in the risk assessment. Additional tables providing more detailed information on PM ambient concentrations for these locations, including the number of observations available on a quarterly and annual basis for each monitor, can be found in Appendix A of the technical support document (Abt, 2003b).

Table 4-1. Mortality Health Endpoints, Urban Locations, and Studies Selected for Use in the $PM_{2.5}$ Risk Assessment

Urban Location	Mortality Associated with Short-Term Exposure				Mortality Associated with Long-Term Exposure[G]
	Total (non-accidental)	Cardiovascular	Circulatory	Respiratory	
Boston, MA	Schwartz et al. (1996)[A] *	Klemm et al. (2000)[B] – ischemic heart disease *		Klemm et al. (2000)[B] – COPD *, pneumonia *	Krewski et al. (2000)-6cities Krewski et al. (2000)-ACS Pope et al. (2002)-ACS extended
Detroit, MI	Lippmann et al. (2000)[C]		Lippmann et al. (2000)[C]	Lippmann et al. (2000)[C]	Krewski et al. (2000)-ACS Pope et al. (2002)-ACS extended
Los Angeles, CA	Moolgavkar (2000a)[D]	Moolgavkar (2000a)[D]		Moolgavkar (2000a)[D] – COPD	Krewski et al. (2000)-ACS Pope et al. (2002)-ACS extended
Philadelphia, PA	Lipfert et al. (2000)	Lipfert et al. (2000) *			Krewski et al. (2000)-ACS Pope et al. (2002)-ACS extended
Phoenix, AZ		Mar et al. (2000)[E]			Krewski et al. (2000)-ACS Pope et al. (2002)-ACS extended
Pittsburgh, PA	Chock et al. (2000)				Krewski et al. (2000)-ACS Pope et al. (2002)-ACS extended
San Jose, CA	Fairley (1999)[F]	Fairley (1999)[F]		Fairley (1999)[F]	Krewski et al. (2000)-ACS Pope et al. (2002)-ACS extended
St. Louis, MO	Schwartz et al. (1996)[A]	Klemm et al. (2000)[B] – ischemic heart disease *		Klemm et al. (2000)[B] – COPD *, pneumonia *	Krewski et al. (2000)-6cities Krewski et al. (2000)-ACS Pope et al. (2002)-ACS extended

*Includes a multi-city or multi-county C-R function

[A] Reanalyzed in Schwartz (2003a)

[B] Reanalyzed in Klemm and Mason (2003)

[C] Reanalyzed in Ito (2003)

[D] Reanalyzed in Moolgavkar (2003)

[E] Reanalyzed in Mar et al. (2003)

[F] Reanalyzed in Fairley (2003)

[G]Krewski et al. (2000)-6 cities and ACS provides total and cardiopulmonary mortality and Pope et al. (2002)-ACS extended provides total and lung cancer mortality

Table 4-2. Morbidity Health Endpoints, Urban Locations, and Studies Selected for Use in the $PM_{2.5}$ Risk Assessment

Urban Location	Cardiovascular Hospital Admissions	Respiratory Hospital Admissions	Respiratory Symptoms
Boston, MA			Schwartz and Neas (2000) – cough, lower respiratory symptoms (LRS)
Detroit, MI	Lippmann et al. (2000)[A] – ischemic heart disease, congestive heart failure, dysrhythmias	Lippmann et al. (2000)[A] – pneumonia, COPD	
Los Angeles, CA	Moolgavkar (2000b)[B]	Moolgavkar (2000c)[B] – COPD	
Seattle, WA		Sheppard et al. (1999)[C] – asthma	
St. Louis, MO			Schwartz and Neas (2000) – cough, LRS

[A] Reanalyzed in Ito (2003)
[B] Reanalyzed in Moolgavkar (2003)
[C] Reanalyzed in Sheppard (2003)

Table 4-3. Mortality Health Endpoints, Urban Locations, and Studies Selected for Use in the $PM_{10-2.5}$ Risk Assessment

Urban Location	Total (non-accidental or all cause)	Cardiovascular	Circulatory	Respiratory
Detroit, MI	Lippmann et al. (2000)[A]		Lippmann et al. (2000)[A]	Lippmann et al. (2000)[A]
Philadelphia, PA	Lipfert et al. (2000)	Lipfert et al. (2000)*		
Phoenix, AZ		Mar et al. (2000)[B]		
St. Louis, MO	Schwartz et al. (1996)[C]			

*Includes multi-county, multi-city, regional, or national C-R function
[A] Reanalyzed in Ito (2003)
[B] Reanalyzed in Mar et al. (2003)
[C] Reanalyzed in Schwartz (2003b)

Table 4-4. Morbidity Health Endpoints, Urban Locations, and Studies Selected for Use in the $PM_{10-2.5}$ Risk Assessment

Urban Location	Cardiovascular Hospital Admissions	Respiratory Hospital Admissions	Respiratory Symptoms
Detroit, MI	Lippmann et al. (2000)[A] – Congestive heart disease, Ischemic heart disease Dysrhythmias	Lippmann et al. (2000)[A] – Pneumonia, COPD	
Seattle, WA		Sheppard et al. (1999)[B] – asthma	
St. Louis, MO			Schwartz and Neas (2000) – LRS, cough

*Includes multi-city, regional, or national C-R function
[A] Reanalyzed in Ito (2003)
[B] Reanalyzed in Sheppard (2003)

Table 4-5. Mortality Health Endpoints, Urban Locations, and Studies Selected for Use in the PM_{10} Risk Assessment

Urban Location	Total (non-accidental or all cause)	Cardiovascular	Cardiorespiratory	Circulatory	Respiratory
Boston, MA	Klemm et al. (2000)[A] * Samet et al. (2000)[B] *		Samet et al. (2000)[B] *		
Chicago, IL	Schwartz (2000b)[C]* Samet et al. (2000)[B,] * Styer et al. (1995) Ito and Thurston (1996) Moolgavkar (2000a)[D] Schwartz (2000a)[C]*	Moolgavkar (2000a)[D] Braga et al. (2001)[C] *	Samet et al. (2000)[B] *	Ito and Thurston (1996)	Ito and Thurston (1996) Moolgavkar (2000a)[D] – COPD Braga et al. (2001)[C] – COPD *, pneum. *
Detroit, MI	Schwartz (2000b)[C]* Schwartz (2000a)[C]* Samet et al. (2000)[B,] * Lippmann et al. (2000)[E]	Braga et al. (2001)[C] *	Samet et al. (2000)[B] *	Lippmann et al. (2000)[E]	Lippmann et al. (2000)[E] Braga et al. (2001)[C] – COPD * pneum. *
Los Angeles, CA	Samet et al. (2000)[B] * Kinney et al. (1995) Moolgavkar (2000a)[D]	Moolgavkar (2000a)[D]	Samet et al. (2000)[B] *		Moolgavkar (2000a)[D] – COPD
Minneapolis, MN	Schwartz (2000b)[C] * Schwartz (2000a)[C] * Samet et al. (2000)[B,] *	Braga et al. (2001)[C] *	Samet et al. (2000)[B] *		Braga et al. (2001)[C] – COPD * pneum. *
Philadelphia, PA	Samet et al. (2000)[B] * Lipfert et al. (2000)	Lipfert et al. (2000) *	Samet et al. (2000)[B] *		
Phoenix, AZ	Samet et al. (2000)[B] *	Mar et al. (2000)[F]	Samet et al. (2000)[B] *		
Pittsburgh, PA	Schwartz (2000b)[C] * Schwartz (2000a)[C] * Samet et al. (2000)[B,] * Chock et al. (2000)	Braga et al. (2001)[C] *	Samet et al. (2000)[B] *		Braga et al. (2001)[C] – COPD * pneum. *
Provo, UT	Samet et al. (2000)[B] *				

Urban Location	Total (non-accidental or all cause)	Cardiovascular	Cardiorespiratory	Circulatory	Respiratory
San Jose, CA	Samet et al. (2000)[B] * Fairley (1999)[G]	Fairley (1999)[G]	Samet et al. (2000)[B] *		Fairley (1999)[G]
Seattle, WA	Schwartz (2000b)[C] * Schwartz (2000a)[C] * Samet et al. (2000)[B,] *	Braga et al. (2001)[C] *	Samet et al. (2000)[B] *		Braga et al. (2001)[C] – COPD * pneum. *
St. Louis, MO	Klemm et al. (2000)[A] * Samet et al. (2000)[B] *		Samet et al. (2000)[B] *		

*Includes multi-county, multi-city, regional, or national C-R function

[A] Reanalyzed in Klemm and Mason (2003)

[B] Reanalyzed in Dominici et al. (2003)

[C] Reanalyzed in Schwartz (2003a)

[D] Reanalyzed in Moolgavkar (2003)

[E] Reanalyzed in Ito (2003)

[F] Reanalyzed in Mar et al. (2003)

[G] Reanalyzed in Fairley (2003)

Table 4-6. Morbidity Health Endpoints, Urban Locations, and Studies Selected for Use in the PM_{10} Risk Assessment

Urban Location	Cardiovascular Hospital Admissions	Respiratory Hospital Admissions	Respiratory Symptoms
Boston, MA			Schwartz et al. (1994) – lower respiratory symptoms (LRS), upper respiratory symptoms (URS)
Chicago, IL	Samet et al. (2000)[A] * Moolgavkar (2000b)[B] Morris and Naumova (1998) – congestive heart failure	Samet et al. (2000)[A] – pneumonia, COPD* Moolgavkar (2000c)[B] – COPD	
Detroit, MI	Samet et al. (2000)[A] * Schwartz and Morris (1995) – congestive heart failure, ischemic heart disease, dysrhythmias Lippmann et al. (2000)[C] – congestive heart disease, ischemic heart disease, dysrhythmias	Samet et al. (2000)[A] – pneumonia, COPD* Lippmann et al. (2000)[C] – Pneumonia, COPD Schwartz (1994a) – pneumonia, COPD	
Los Angeles, CA	Moolgavkar (2000b)[B] Linn et al. (2000)	Moolgavkar (2000c)[B] – COPD Linn et al. (2000) – respiratory, COPD, asthma Nauenberg and Basu (1999) – asthma	
Minneapolis, MN	Samet et al. (2000)[A] *	Samet et al. (2000)[A] – pneumonia, COPD* Schwartz (1994b) – pneumonia, COPD	
Pittsburgh, PA	Samet et al. (2000)[A] *	Samet et al. (2000)[A] – pneumonia, COPD*	
Provo, UT	Samet et al. (2000)[A] *	Samet et al. (2000)[A] – pneumonia, COPD*	Pope et al. (1991) – LRS, URS
Seattle, WA	Samet et al. (2000)[A] *	Samet et al. (2000)[A] – pneumonia, COPD* Sheppard et al. (1999)[D] – asthma	Yu et al. (2000) – asthma
St. Louis, MO			Schwartz et al. (1994) – LRS, URS

*Includes multi-city, regional, or national C-R function
[A] Reanalyzed in Zanobetti and Schwartz (2003)
[B] Reanalyzed in Moolgavkar (2003)
[C] Reanalyzed in Ito (2003)
[D] Reanalyzed in Sheppard (2003)

4.2.3.1 Estimating PM Background Levels

Background PM concentrations used in the PM risk assessment are defined above in Chapter 2 as the 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 base case risk estimates, the midpoint of the appropriate ranges of annual average estimates for $PM_{2.5}$ and PM_{10} background presented in Section 2.7 were used (i.e., eastern values were used for eastern study locations and western values were used for western study locations). For $PM_{10-2.5}$ the rough point estimate of 3 $\mu g/m^3$ for both eastern and western portions of the U.S. was used (CD, p.3-82). In addition, sensitivity analyses were also conducted in which the lower- and upper-end of the estimated ranges of background for each PM indicator were used to estimate risk. A sensitivity analysis also was performed examining the potential impact of using a distribution of estimated daily background $PM_{2.5}$ concentrations on the risk estimates. Table 4-8 summarizes the range and midpoint background levels used in the base case and sensitivity analyses.

4.2.3.2 Simulating PM Levels That Just Meet Specified Standards

To estimate the health risks associated with just meeting the current $PM_{2.5}$ standards and alternative $PM_{2.5}$ and $PM_{10-2.5}$ standards it is necessary to estimate the distribution(s) of PM concentrations that would occur under each specified standard (or sets of standards). While compliance with the standards is based on a 3-year average, because of the limited number of years for which $PM_{2.5}$ and $PM_{10-2.5}$ data are available, only a single year of data is used in the risk assessment. 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.

Table 4-7. Summary of PM Ambient Air Quality Data for Risk Assessment Study Areas*

Area	Population (millions)	$PM_{2.5}$**		$PM_{10-2.5}$**		PM_{10}**	
		Annual Avg.	24-hr Avg, 98th%	Annual Avg.	24-hr , 98th%	Annual Avg.	24-hr, 98th%
Boston, MA[a]	2.8	11.4	33.0			24.3	50
Chicago, IL[b]	5.4					25.7	55
Detroit, MI[c]	2.1	15.8	36.7	6.5	23.5	20.2	49
Los Angeles County, CA[d]	9.5	20.4	47.2			36.6	55
Minneapolis, MN[e]	1.6					27.5	61
Philadelphia County, PA[f]	1.5	14.5	40.9	9.2	22.1	25.4	72
Phoenix, AZ[g]	3.1	10.4	28.9	33.3	70.6	47.9	83.9
Pittsburgh, PA[h]	1.3	15.2	41.4			20.5	58
Provo, UT[i]	0.5					28.6	72
San Jose, CA[j]	1.7	12.4	57.5			24.6	77
Seattle, WA[k]	1.7	9.3	24.8	5.7	11.4	18.0	44
St. Louis[l]	2.5	14.7	34.2	3.5	11.4	22.8	69

*Based on air quality data for the year 2002, unless otherwise noted in footnotes below; all concentrations are in $\mu g/m^3$.
**Summary statistics for a "composite monitor" based on average of 24-hour values at the different monitors in urban area that reported on each day.
[a]Includes Middlesex, Norfolk, and Suffolk Counties; PM_{10} data are for 1999.
[b]Includes Cook County.
[c]Includes Wayne County.
[d]Includes Los Angeles County.
[e]Includes Hennepin and Ramsey Counties.
[f]Includes Philadelphia County.
[g]Includes Maricopa County; $PM_{2.5}$ air quality data are for 2001 and $PM_{10-2.5}$ air quality data are based on TEOM data from 1997.
[h]Includes Allegheny County
[i]Includes Utah County
[j]Includes Santa Clara County; $PM_{2.5}$ air quality data are for 2001 and PM_{10} air quality data are for 1999.
[k]Includes King County; $PM_{10-2.5}$ air quality data are for 2000.
[l]Includes St. Louis, Franklin, Jefferson, St. Charles Counties in MO, Clinton, Madison, Monroe, and St. Claire Counties in IL and St. Louis City.

Table 4-8. Summary of Estimated PM Background Concentrations Used in Risk Assessment*

	$PM_{2.5}$		$PM_{10-2.5}$		PM_{10}	
	Range	Midpoint	Range	Point Estimate	Range	Midpoint
Eastern Locations**	2 - 5	3.5	<1 - 9	3	5 - 11	8
Western Locations***	1 - 4	2.5	<1 - 7	3	4 - 8	6

*All concentrations are specified in $\mu g/m^3$

**Eastern locations: Boston, Chicago, Detroit, Minneapolis-St. Paul, Philadelphia, Pittsburgh, St. Louis

***Western locations: Los Angeles, Phoenix, Provo, San Jose, Seattle

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There are many possible ways to create an alternative distribution of daily concentrations that just meets a specified set of PM standards. Both the assessment conducted during the last NAAQS review (see Abt Associates, 1996, section 8.2) and a new analysis of historical air quality (see Abt Associates, 2003b, Appendix B) have found that $PM_{2.5}$ levels in excess of estimated background concentrations have in general historically decreased in a proportional manner (i.e., concentrations at different points in the distribution of 24-hour $PM_{2.5}$ values in excess of an estimated background concentration have decreased by approximately the same percentage). This suggests that, in the absence of detailed air quality modeling, a reasonable method to simulate PM reductions that would result from just meeting a set of standards is to use proportional rollback (i.e., to decrease PM levels on all days by the same percentage) for all concentrations exceeding the background level. A sensitivity analysis also has been conducted to examine the impact on the risk estimates of an alternative air quality adjustment procedure (e.g., a method that reduces the top 10% of daily $PM_{2.5}$ concentrations more than the lower 90%).

In the $PM_{2.5}$ risk assessment, air quality just meeting the current $PM_{2.5}$ standards is simulated by reducing the $PM_{2.5}$ concentrations at the composite monitor by the same percentage

on all days. The percentage reduction is determined by comparing the maximum of the monitor-specific annual averages (or the maximum of the monitor-specific ninety-eighth percentile daily values) adjusted for background with the level of the annual (or daily) standard adjusted for background.[10] Because pollution abatement methods are applied largely to anthropogenic sources of $PM_{2.5}$, rollbacks were applied only to $PM_{2.5}$ concentrations above estimated background levels. The percent reduction is determined by the controlling standard, which for the current suite of $PM_{2.5}$ standards is the existing annual standard for the four urban study areas that do not currently meet the annual average standard. The same basic approach will be applied to simulate just meeting alternative $PM_{2.5}$ and $PM_{10-2.5}$ standards in the assessments that will be included in the next draft of the Staff Paper.

4.2.4 Approach to Estimating PM-Related Health Effects Incidence

The C-R relationships used in the PM risk assessment are empirically estimated relations between average ambient PM concentrations and the health endpoints of interest reported by epidemiological studies for specific urban areas. Most epidemiological studies estimating relationships between PM and health effects used a method referred to as "Poisson regression" to estimate exponential (or log-linear) C-R functions.[11] 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 y_0 concentration, and B is the incidence at x=0, i.e., when there is no ambient $PM_{2.5}$. The difference in health effects incidence, $\Delta y = y_0 - y$, from y_0 to the baseline incidence rate, y, that corresponds to a given difference in ambient $PM_{2.5}$ levels, $\Delta x = x_0 - x$, is then

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

[10]Since an area is allowed, if it meets certain requirements, to determine whether it meets the current annual average standard based on the spatial average of its population-oriented monitors, in section 4.4 the percent rollbacks that would have resulted from using this alternative approach in each study area also are presented.

[11]For some studies on respiratory hospital admissions used in the risk assessment a linear C-R function was estimated.

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 $PM_{2.5}$ levels, Δx. Equations 4-2 and 4-3 are simply alternative ways of expressing the relation between a given difference in ambient $PM_{2.5}$ levels and the corresponding difference in health effects. These equations are the key equations that combine air quality information, C-R information, and baseline health effects incidence information to estimate ambient $PM_{2.5}$ health risk.

The daily time-series epidemiological studies, discussed above in Chapter 3, used models estimating C-R functions in which the PM-related incidence on a given day depends only on same-day PM concentration or previous-day PM concentration (or some variant of those, such as two-day average concentrations). Such models necessarily assume that the longer pattern of PM levels preceding the PM concentration on a given day does not affect mortality on that day. To the extent that PM-related mortality on a given day is affected by PM concentrations over a longer period of time, then these models would be mis-specified, and this mis-specification would affect the predictions of daily incidence based on the model. The extent to which longer-term (i.e., weekly, monthly, seasonal, or annual) $PM_{2.5}$ exposures affect the relationship observed in the daily time-series studies is unknown. However, there is some evidence, based on analyses of PM_{10} data, that mortality on a given day is influenced by prior PM exposures up to more than a month before the date of death (Schwartz, 2000a, reanalyzed in Schwartz, 2003b). Currently, there is insufficient information to adjust for the impact of longer-term exposure on mortality associated with $PM_{2.5}$ exposures and this is an important uncertainty that should be kept in mind as one considers the results from the short-term exposure $PM_{2.5}$ risk assessment. However, for the PM_{10} risk assessment, distributed lag model results will be used where available.

The estimated $PM_{2.5}$-related mortality associated with long-term exposure studies is likely to include mortality related to short-term exposures as well as mortality related to longer-term exposures. As discussed above, estimates of daily mortality based on the time-series studies also are likely to be affected by prior exposures. Therefore, the estimated annual

incidences of mortality calculated based on the short- and long-term exposure studies are not likely to be completely independent and should not simply be added together.

The statistical uncertainty surrounding the estimated $PM_{2.5}$, $PM_{10-2.5}$, and PM_{10} coefficients in the reported C-R functions is reflected in the intervals provided for the risk estimates in sections 4.3 to 4.5. In addition, sensitivity analyses examine how the risk estimates would vary if, instead of the reported C-R relationship, different hypothetical threshold models were applied instead. Another sensitivity analysis addresses how the $PM_{2.5}$ risk estimates would change if a distributed lag model could be applied instead of the single lag models reported in the literature for short-term exposure mortality. Finally, a third sensitivity analysis addresses the possible impact of different assumptions about the role of historical air quality concentrations in contributing to the reported effects associated with long-term exposure. The results of these sensitivity analyses are summarized in section 4.3 .

4.2.5 Baseline Health Effects Incidence Rates and Population Estimates

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 urban study locations, information on the baseline incidence of health effects (i.e., the incidence under "as is" air quality conditions) and population size in each location is therefore needed. Where possible, county-specific incidence or incidence rates have been used. County-specific mortality incidences were available for the year 1998 from CDC Wonder, an interface for public health data dissemination provided by the Centers for Disease Control.[12] The baseline mortality rates for each risk assessment location are provided in Exhibits 5.4, 5.5, and 5.6 of the technical support document (Abt, 2003b). Population sizes, for both total population and various age ranges used in the PM risk assessment were obtained for the year 2000 from the 2000 U.S. Census data[13] and are summarized in Exhibits 5.1, 5.2, and 5.3 of the technical support document (Abt, 2003b).

[12]See http://wonder.cdc.gov/.

[13]See http://factfinder.census.gov/.

For some of the morbidity endpoints, however, county-specific incidence rates were difficult to obtain. County-specific rates for cardiovascular and respiratory hospital discharges, and various subcategories (e.g., pneumonia, asthma), have been obtained, where possible, from state, local, and regional health departments and hospital planning commissions for each of the study areas where there were C-R relationships available for hospital admissions associated with PM concentrations.[14] Baseline hospitalization rates used in each PM risk assessment location are summarized in Exhibits 5.7, 5.8, and 5.9 of the technical support document (Abt, 2003b). For respiratory symptoms in children, the only available estimates of baseline incidence rates were from the studies that estimated the C-R relationships for those endpoints. However, because the risk assessment locations for these endpoints were selected partly on the basis of where studies were carried out, baseline incidence rates reported in these studies should be appropriate for the risk assessment locations to which they were applied.

4.2.6 Concentration-Response Functions Used in Risk Assessment

A key component in the risk model is the set of C-R functions which provide estimates of the relationship between each health endpoint of interest and ambient PM concentrations. As discussed above, the health endpoints that have been included in the $PM_{2.5}$, $PM_{10-2.5}$, and PM_{10} risk assessment for short-term exposure include mortality, hospital admissions, and respiratory symptoms not requiring hospitalization. For $PM_{2.5}$ long-term exposure mortality is also estimated. Health endpoints were included in the risk assessment if the overall weight of the evidence from the collective body of studies supported the conclusion that there was likely to be a causal relationship between PM and the specific health endpoint. Once it had been determined that a health endpoint was to be included in the assessment, inclusion of a study on that health endpoint was not based on the existence of a statistically significant result. Both single-pollutant and, where available, multi-pollutant, C-R functions were used from the studies listed in Tables 9-8, 9-10, and 9-11 of the draft CD (see also Appendix A of this SP).

[14]The data were annual hospital discharge data, which were used as a proxy for hospital admissions. Hospital discharges are issued to all people who are admitted to the hospital, including those who die in the hospital. By using the annual discharge rate, it is assumed that admissions at the end of the year that carry over to the beginning of the next year, and are therefore not included in the discharge data, are offset by the admissions in the previous year that carry over to the beginning of the current year.

As discussed in the draft CD (section 8.4.2) and Chapter 3 (section 3.5.3.1), questions were raised in 2002 about the default convergence criteria (which impact the mean estimate) and standard error calculations (which result in understated standard errors) used in many of the short-term PM epidemiological studies that used generalized additive models (GAMs) in S-Plus. To address these concerns, many of the study authors performed reanalyses of certain of the studies using alternative statistical estimation approaches (e.g., GLM with different degrees of freedom and different types of splines), in addition to using GAMs with a more stringent convergence criterion. To avoid producing a prohibitively large set of results, the PM risk assessment included C-R functions using only GAM with the more stringent convergence criterion (denoted "GAM (stringent)") for all urban locations, except Chicago and Los Angeles. While the GAM (stringent) functions are likely to provide better central tendency estimates, they do not address the issue of understated standard errors of the coefficient estimates. Thus, the confidence intervals included in the risk assessment involving use of the GAM (stringent) C-R functions are somewhat understated. As indicated in the draft CD, "the extent of downward bias in standard error reported in these data (a few percent to ~15%) also appears not to be very substantial, especially when compared to the range of standard errors across studies due to differences in population size and number of days available" (CD, p.8-197). The reanalyzed GLM C-R functions reflect a corrected approach to calculation of the standard errors of the coefficient estimates, but are less likely to provide the best central tendency effect estimate. For Chicago and Los Angeles, a wider array of C-R models was included to illustrate the impact of alternative model specifications on the risk estimates.

More detailed information about the C-R relationships used in the PM risk assessment is provided in Appendix C (Exhibits C.1 through C.27 of the technical report (Abt, 2003b) for each of the study areas. This information includes population characteristics (e.g., age and disease status), form of the model (e.g., log-linear, logistic), whether other pollutants were included in the model, lags used, observed minimum and maximum ambient PM concentrations, and PM coefficients along with lower and upper 5^{th} and 95^{th} confidence intervals.

4.2.6.1 Hypothetical Thresholds

In assessing or interpreting public health risk associated with exposure to PM, the form of the C-R function is a critical 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 studies are several that use different modeling methods to investigate hypothetical threshold levels and C-R forms. As summarized in the draft CD, the results of multi-city studies suggest there is no strong evidence of the existence of a threshold for associations between PM and acute mortality; some single-city studies suggest a hint of a threshold, but not in a statistically significant manner (CD, p. 8-241).

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. 8-239; Daniels et al., 2000). 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 study authors concluded that "if a threshold exists, it is highly likely that standard statistical analysis can detect it" (CD, p. 8-240). 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 $\mu g/m^3$ daily concentrations for short-term exposure 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. 8-241) 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 "more data may need to be examined with alternative approaches ... meanwhile, the use of a linear PM effect model appears to be appropriate" (CD, p. 8-241), which is consistent with the conclusions of the previous Criteria Document (EPA, 1996a).

For long-term exposure to PM, the shape of the C-R function also was evaluated using data from the ACS cohort, where mean $PM_{2.5}$ concentrations ranged from abut 10 to 34 $\mu g/m^3$. As shown in Figure 8-9 of the draft CD, the C-R relationships for all-cause and cardiopulmonary mortality can be reasonably approximated by a linear model. However, for lung cancer, the relationship appears to have a steeper linear slope at lower concentrations, with a flatter linear slope at $PM_{2.5}$ concentrations that exceed about 13 $\mu g/m^3$ (CD, p.8-89). It also is apparent in this figure that the confidence intervals around the estimated C-R functions expand at the low and

high ends of the ranges of data, indicating greater uncertainty in the shape of the C-R relationship across the lowest and highest concentration ranges.

Staff recognizes that it is reasonable to expect that, for individuals or groups of individuals, there may be biological thresholds for different effects. While epidemiological analyses have not identified a threshold level in the range of air quality concentrations in these studies, it is possible that a threshold level exists within these ranges but cannot be detected due to variability in susceptibility across a population. It is also possible that such a level may exist at concentrations lower than the air quality levels observed in current studies. Staff have examined the potential impact on risk estimates of possible threshold levels through sensitivity analyses using various hypothetical threshold levels.

The $PM_{2.5}$ base case risk assessment summarized in section 4.3 and 4.4 below does not include a threshold. Rather risk estimates reflect the potential contribution of $PM_{2.5}$ down to either an estimated background level or the LML in the study, whichever is higher. For a number of studies, including all of the long-term exposure mortality studies, the LML is significantly above the estimated background concentrations and, therefore, there is no contribution to the risk estimates from $PM_{2.5}$ concentrations below the LML in these cases. As noted earlier, sensitivity analyses have been conducted that do include hypothetical alternative thresholds or cutpoints, where risks only are estimated due to $PM_{2.5}$ concentrations exceeding the assumed threshold concentrations. Three hypothetical thresholds (10, 15, and 20 $\mu g/m^3$**)** were used for short-term exposure mortality and three different hypothetical thresholds (10, 12.5, and 15 $\mu g/m^3$) were used for long-term exposure mortality.[15] Results of these sensitivity analyses are discussed below in section 4.3.

4.2.6.2 Single and Multi-Pollutant Models

For several of the epidemiological studies from which C-R relationships for the PM risk assessment were obtained, C-R functions are reported both for the case where only PM levels were entered into the health effects model (i.e., single-pollutant models) and where PM and one or more other measured gaseous co-pollutants (i.e., ozone, nitrogen dioxide, sulfur dioxide,

[15]The groups of hypothetical thresholds (or cutpoints) used for both short- and long-term exposure mortality are similar to those used in the prior $PM_{2.5}$ risk assessment, except that the highest cutpoints (i.e., 30 $\mu g/m^3$ for short-term and 18 $\mu g/m^3$ for long-term) were dropped since there is now more substantial evidence supporting the existence of effects below these levels.

carbon monoxide) were entered into the health effects model (i.e., multi-pollutant models). To the extent that any of the co-pollutants present in the ambient air may have contributed to the health effects attributed to PM in single-pollutant models, risks attributed to PM might be overestimated where C-R functions are based on single-pollutant models. However, as shown in Figure 3-12 (pp. 3-75 - 3-76) for $PM_{2.5}$ and in the last review (EPA, 1996b, p. V-55) for PM_{10}, the magnitude and statistical significance of the associations reported between $PM_{2.5}$ (and PM_{10}) and mortality due to short-term exposure show no trends with the levels of any of the four gaseous co-pollutants examined. As stated earlier, "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 co-pollutants." (SP, p. 3-74)

For some of the gaseous co-pollutants, such as carbon monoxide, nitrogen dioxide, and sulfur dioxide, which tend to be highly correlated with ambient $PM_{2.5}$ concentrations in some cities, it is difficult to sort out whether these pollutants are exerting any independent effect from that attributed to $PM_{2.5}$. As discussed in the draft CD, inclusion of pollutants that are highly correlated with one another can lead to misleading conclusions in identifying a specific causal pollutant. When collinearity exists, multi-pollutant models would be expected to produce unstable and statistically insignificant effects estimates for both PM and the co-pollutants (CD, p. 9- 73). Given that single and multi-pollutant models each have both potential advantages and disadvantages, with neither type clearly preferable over the other, risk estimates based on both single and multi-pollutant models are reported where both are available.

4.2.6.3 Single, Multiple, and Distributed Lag Functions

The question of lags and the problems of correctly specifying the lag structure in a model are discussed extensively in the draft CD (section 8.4.5). The draft CD points out that, in most PM times-series studies, after the basic model is fit (before considering PM), several different PM lags are typically fit in separate single-lag models and the most significant lag is chosen. The draft CD notes that "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" (p. 8-234). There is recent evidence (Schwartz., 2000a, reanalyzed in Schwartz, 2003b), that the relation between PM and health effects may best be described by a distributed

lag (i.e., the incidence of the health effect on day n is influenced by PM concentrations on day n, day n-1, day n-2 and so on). If this is the case, a model that includes only a single lag (e.g., a 0-day lag or a 1-day lag) is likely to understate the total impact of PM. The draft CD makes this point, noting that "if one chooses the most significant single lag day only, and if more than one lag day shows positive (significant or otherwise) associations with mortality, then reporting a RR [relative risk] for only one lag would also underestimate the pollution effects" (p. 8-236).

Because of this, a distributed lag model is considered preferable to a single lag model. However, distributed lag models have been used in only a few cases and only for PM_{10} (e.g., Schwartz, 2000b, reanalyzed in Schwartz, 2003b). Where distributed lag models were available, they were included in the PM risk assessment. When a study reports several single lag models, unless the study authors identify a "best lag", the following lag models were included in the risk assessment based on the assessment in the draft CD:

- both 0- and 1-day lag models for mortality (both total and cause specific),
- both 0- and 1-day lag models for cardiovascular and respiratory hospital admissions, and
- 0-, 1-, and 2-day lag models (if all three were available) for COPD hospital admissions.

In addition, for two urban locations (Chicago and Los Angeles), the risk assessment included all single lag models used in a study to illustrate the effect of lag structure on the risk estimates. A sensitivity analysis was also conducted to examine the potential impact of using a distributed lag approach for short-term exposure mortality associated with $PM_{2.5}$ based on the distributed lag analysis of PM_{10} and mortality (Schwartz, 2000b, reanalyzed in Schwartz, 2003b).

4.2.6.4 Long-term Exposure Mortality $PM_{2.5}$ Concentration-Response Functions

There are far fewer long-term exposure studies than short-term exposure studies in the peer-reviewed literature. The available long-term exposure mortality C-R functions are all based on cohort studies, in which a cohort of individuals is followed over time. Two cohorts that have been studied are particularly relevant for the purposes of this risk assessment. One cohort, in six U.S. cities, was originally followed in a study referred to as the Six Cities study. The other cohort, of participants enrolled by American Cancer Society (ACS) volunteers, was composed of a much larger sample of individuals from many more cities. It was originally followed in a study

referred to as the ACS study. There have recently been reanalyses of both the Six Cities study and the ACS study by Krewski et al. (2000), referred to here as Krewski et al. (2000) – Six Cities reanalysis, and Krewski et al. (2000) – ACS reanalysis. Both of these reanalyses are used in the $PM_{2.5}$ risk assessment. In addition, Pope et al. (2002) extended the follow-up period for the ACS cohort to sixteen years and published findings on the relation of long-term exposure to $PM_{2.5}$ and all-cause mortality as well as cardiopulmonary and lung cancer mortality (referred to here as Pope et al. (2002) - ACS extended. Three different indicators of $PM_{2.5}$ exposure were considered in this extended ACS study. The first indicator, 1979-1983, was the period considered in the original ACS study as well as in the Krewski reanalysis. The second indicator was 1999-2000, and the third was an average of the 1979-1983 and 1999-2000 $PM_{2.5}$ ambient concentrations. The C-R functions based on $PM_{2.5}$ from 1979-1983 are included in the $PM_{2.5}$ risk assessment, because this period is the one used by the study authors in their main presentation of results.

Table 3-3 summarizes the total mortality findings from the long-term exposure mortality studies discussed above, as well as two other PM cohort studies (i.e., the AHSMOG and Veterans' Cohort studies) that are not included in the $PM_{2.5}$ risk assessment. In addition, Table 8-11 from the draft CD presents results for total, cardiovascular and lung cancer mortality. As discussed in Chapter 3 (see section 3.3.1.2), the draft CD concludes that the "lack of consistent findings in the AHSMOG study and negative results of the VA study, do not negate the findings of the Six Cities and ACS studies." (CD, p. 8-105) The Six Cities and ACS studies were based on measured PM data (in contrast with AHSMOG PM estimates based on TSP or visibility measurements), have study populations more similar to the general population than the VA study cohort, and have been validated through an exhaustive reanalysis (CD, p. 8-105). Lacking these advantages, the AHSMOG and VA studies were excluded from the risk assessment. Overall, the draft CD concludes that "there is substantial evidence for a positive association between long-term PM exposure to PM (especially fine particles) and mortality" (CD, p. 8-105).

4.2.7 Characterizing Uncertainty and Variability

An important issue associated with any population health risk assessment is the characterization of uncertainty and variability. *Uncertainty* refers to the lack of knowledge regarding the actual values of model input variables (parameter uncertainty) and of physical

systems or relationships (model uncertainty – e.g., the shapes of C-R functions). In any risk assessment uncertainty is, ideally, reduced to the maximum extent possible. It can be reduced by improved measurement and improved model formulation.

Variability refers to the heterogeneity in a population or parameter. For example, there may be variability among C-R functions describing the relation between $PM_{2.5}$ and mortality across urban areas. This variability may be due to differences in population (e.g., age distribution), population activities that affect exposure to PM (e.g., use of air conditioning), levels of PM and/or co-pollutants, and/or other factors that vary across urban areas.

The current risk assessment incorporates some of the variability in key inputs to the assessment by using location-specific inputs (e.g., location-specific C-R functions, baseline incidence rates, and air quality data). Although spatial variability in these key inputs across all U.S. locations has not been fully characterized, variability across the selected locations is imbedded in the assessment by using, to the extent possible, inputs specific to each urban area. Temporal variability is more difficult to address, because the risk reduction portions of the risk assessment (i.e., estimated risk reduction associated with just meeting specified standards) focus on some unspecified time in the future when specified PM standards are just met. To minimize the degree to which values of inputs to the assessment may be different from the values of those inputs at that unspecified time, we have used the most current inputs available (i.e., year 2002 air quality data for most locations and the most recent available mortality baseline incidence rates (from 1998)). However, we have not tried to predict future changes in inputs (e.g., future population levels or possible changes in baseline incidence rates).

The following are among the major sources of uncertainty in the PM health risk assessment:

- There is uncertainty about whether each of the estimated associations between the three PM indicators ($PM_{2.5}$, $PM_{10-2.5}$, and PM_{10}) and the various health endpoints included in this risk assesment actually reflect a causal relationship. There are varying degrees of uncertainty associated with the various PM indicators and health endpoints related to differences in the weight of evidence supporting judgments about whether an observed association truly reflects a causal relationship.

- There are a number of uncertainties related to estimating the C-R functions, including the following:

- There is uncertainty surrounding estimates of PM coefficients in C-R functions used in the assessment.

- There is uncertainty about the specification of the model (including the shape of the C-R relationship), particularly whether or not there are thresholds below which no response occurs.

- There is uncertainty related to the transferability of PM C-R functions from study locations to the locations selected for the risk assessment and from study location.[16] A C-R function in a study location may not provide an accurate representation of the C-R relationship in the assessment location(s) because of (1) variations in PM composition across cities, (2) the possible role of associated co-pollutants in influencing PM risk, (3) variations in the relation of total ambient exposure (both outdoor and ambient contributions to indoor exposure) to ambient monitoring in different locations (e.g, due to differences in air conditioning use in different regions of the U.S.), (4) differences in population characteristics (e.g., the proportions of members of sensitive subpopulations) and population behavior patterns across locations.

- There is uncertainty related to the extent to which PM C-R functions derived from studies in a given location and time when PM concentrations were higher provide accurate representations of the C-R relationships for the same location with lower annual average concentrations and fewer high daily average concentrations.

- There are a number of uncertainties related to use of baseline incidence rates and population data in the risk assessment, including the following:

  - There is uncertainty related to the extent to which baseline incidence rates, age distribution, and other demographic variables that impact the risk estimates vary for the year(s) when the actual epidemiology studies were conducted, the recent year of air quality used in the assessment, and some unspecified future year when air quality is adjusted to just meet the current or alternative standards.

  - There is uncertainty related to the use of annual incidence rate data to develop daily health effects incidence data.

  - For the respiratory symptoms endpoint, baseline health effects incidence data were only available as a total estimate for all six urban areas combined. This introduces additional uncertainty because the risk assessment applied the overall incidence rate from the six cities to individual cities (i.e., Boston and St. Louis).

[16] The risk assessment locations were selected partly on the basis of where C-R functions were estimated, specifically to avoid this important source of uncertainty. Therefore, this is a source of uncertainty in the risk assessment only when C-R functions from multi-city studies are applied to a risk assessment location.

- There are uncertainties related to the air quality adjustment procedure used to simulate just meeting the current $PM_{2.5}$ standards and alternative $PM_{2.5}$ and $PM_{10-2.5}$ standards and about estimated background concentrations for each location.

- There are uncertainties associated with use of baseline health effects incidence information that is not specific to the assessment locations.[17]

The uncertainties from some of these sources -- in particular, the statistical uncertainty surrounding estimates of the PM coefficients in C-R functions -- were characterized quantitatively. It was possible, for example, to calculate confidence intervals around risk estimates based on the uncertainty associated with the estimates of PM coefficients used in the risk assessment. These confidence intervals express the range within which the risks are likely to fall if the sampling error uncertainty surrounding PM coefficient estimates were the only uncertainty in the assessment.[18] There are, of course, several other significant uncertainties in the risk assessment, as noted above. If there were sufficient information to characterize these sources of uncertainty quantitatively, they could be included in a Monte Carlo analysis to produce confidence intervals that more accurately reflect all sources of uncertainty.

We handled other uncertainties in the risk assessment in several ways:

- Limitations and assumptions in estimating risks and risk reductions were clearly stated and explained.

- Sensitivity analyses were conducted to illustrate the effects of changing key default assumptions on the results of the assessment, and quantitative comparisons were presented to inform other analytic choices.[19] See Table 4-9 for a summary of the sensitivity analyses and quantitative comparisons conducted for the PM risk assessment.

[17] Location-specific baseline incidence rates were obtainable for most health endpoints. The only health endpoints for which this is not the case are cough and lower respiratory symptoms, for which baseline incidence rates were reported in the study (Schwartz and Neas, 2000). These rates were based on six cities combined. Boston and St. Louis, the two assessment locations where these endpoints are evaluated, were two of the six cities.

[18] However, as discussed earlier in section 4.2.6, for the short-term C-R functions based on reanalyzed GAM (stringent) models the confidence intervals are somewhat understated.

[19] "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.

4.3 $PM_{2.5}$, $PM_{10-2.5}$, and PM_{10} RISK ESTIMATES FOR CURRENT ("AS IS") AIR QUALITY

4.3.1 Summary of Risk Estimates

The risk estimates associated with "as is" $PM_{2.5}$, $PM_{10-2.5}$, and PM_{10} concentrations in excess of background levels are summarized in a series of figures in this section. The risk estimates are expressed in terms of percent of total incidence for each health endpoint in these figures. The legend of symbols used for the different health endpoints is presented in Figure 4-2. For each series of estimates, a mean effect estimate is provided along with 95% confidence intervals.[20] Additional detailed tables which present the estimated incidence (both as the number of effects and as a percentage of total incidence) for each risk assessment location are included in the technical support document (see Appendix D). Risk estimates in a given assessment location are presented only for those health endpoints for which there is at least one acceptable C-R function reported for that location. Therefore, the set of health effects shown in the figures varies for the different locations.

Figures 4-3 through 4-8 summarize the $PM_{2.5}$ risk estimates across the various assessment locations. Figure 4-3 shows risk estimates for six urban areas for total non-accidental mortality associated with short-term exposure to $PM_{2.5}$ using single-pollutant, single-city models. Most of the mean effect estimates are in the range 0.5 to 1.5% of total non-accidental mortality, with one location, San Jose, showing the highest risk (about 3%) with 0-day lag and the same area also having the lowest risk (0%) with 1-day lag. Figure 4-4 shows risk estimates for two locations (Boston and St. Louis) for various short-term exposure mortality endpoints using single-city versus multi-city models. Generally, the mean risk estimates for the single- and multi-city models are roughly comparable, with lower risk estimates seen in Boston for the multi-city models compared to the single-city models and the reverse being observed in St. Louis. As expected, the 95% confidence intervals are somewhat smaller for most of the multi-city models compared to the respective single-city model given the greater sample size.

[20]Risk estimates less than zero were truncated at zero since we have no reason or evidence to assume that increasing ambient PM concentrations results in improved health. In cases where the lower confidence bound was ≤ 0, the interval shown does not represent a 95% confidence interval around the mean estimate. In these cases, the upper bound is the 97.5 percentile and the lower confidence bound has been truncated at zero.

Table 4-9. Sensitivity Analyses and Quantitative Comparisons

Analysis Number (Figure 4-1)	PM Indicator	Component of the Risk assessment	Sensitivity Analysis or Comparison
1	$PM_{2.5}$	Air Quality	A sensitivity analysis of the effect of assuming a constant background PM level versus a distribution of daily background levels
2	$PM_{2.5}$, PM_{10}, $PM_{10-2.5}$	Air Quality	A sensitivity analysis of the effect of assuming different (constant) background PM levels
3	$PM_{2.5}$	Air Quality	A sensitivity analysis of the effect of an alternative air quality adjustment procedure on the estimated risk reductions resulting from just meeting the current 24-hr and annual $PM_{2.5}$ standards
4	$PM_{2.5}$	Air Quality	A comparison of the effect of just meeting the current annual $PM_{2.5}$ Standard of 15 $\mu g/m^3$ using the maximum versus the average of monitor-specific averages
5	$PM_{2.5}$, PM_{10}, $PM_{10-2.5}$	Concentration-Response	A sensitivity analysis using an approach to estimate the possible impact of using a distributed lag C-R function
6	$PM_{2.5}$	Concentration-Response	A 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	$PM_{2.5}$, PM_{10}, $PM_{10-2.5}$	Concentration-Response	A sensitivity analysis assuming alternative hypothetical threshold concentration levels for the occurrence of $PM_{2.5}$-related response at concentrations above those for background

Source: Abt Associates (2003b).

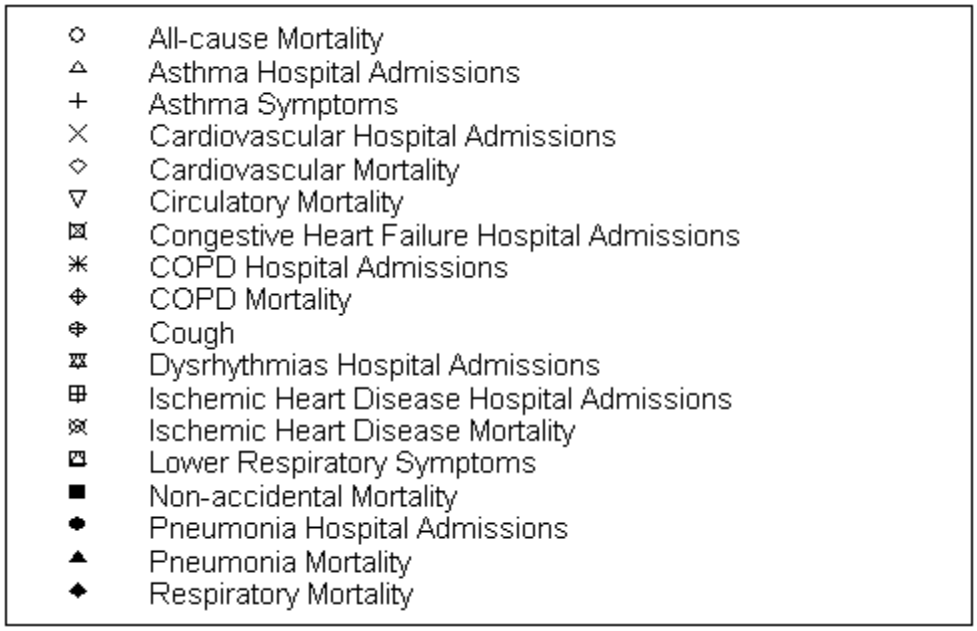

Figure 4-2. Legend for Health Endpoints in Figures

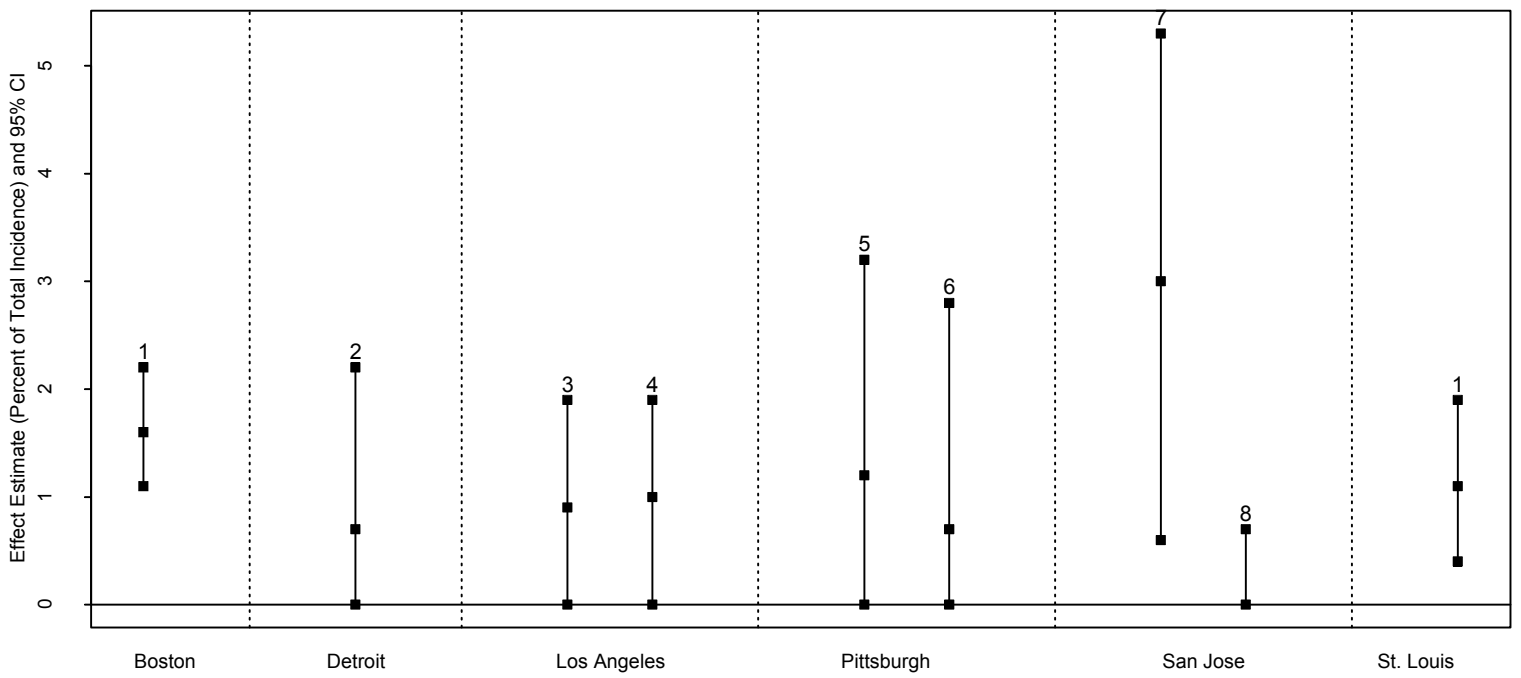

Figure 4-3. Estimated Annual Percent of Total (Non-Accidental) Mortality Associated with Short-Term Exposure to $PM_{2.5}$ (and 95 Percent Confidence Interval): Single-Pollutant, Single-City Models[‡]

Source: Abt (2003b)

1. Schwartz et al. (1996)*
2. Lippmann et al. (2000)**
3. Moolgavkar (2000a)*** – 0-day lag
4. Moolgavkar (2000a)*** – 1-day lag
5. Chock et al. (2000) (age<75)
6. Chock et al. (2000) (age>75)
7. Fairley (1999)**** – 0-day lag
8. Fairley (1999)**** – 1-day lag

*Reanalyzed in Schwartz (2003a)
**Reanalyzed in Ito (2003)
***Reanalyzed in Moolgavkar (2003)
****Reanalyzed in Fairley (2003)

[‡]In cases where the lower bound of the confidence interval was less than zero, the interval does not represent a 95% confidence interval. In these cases, the upper bound is the 97.5th percentile and the lower bound has been truncated at zero.

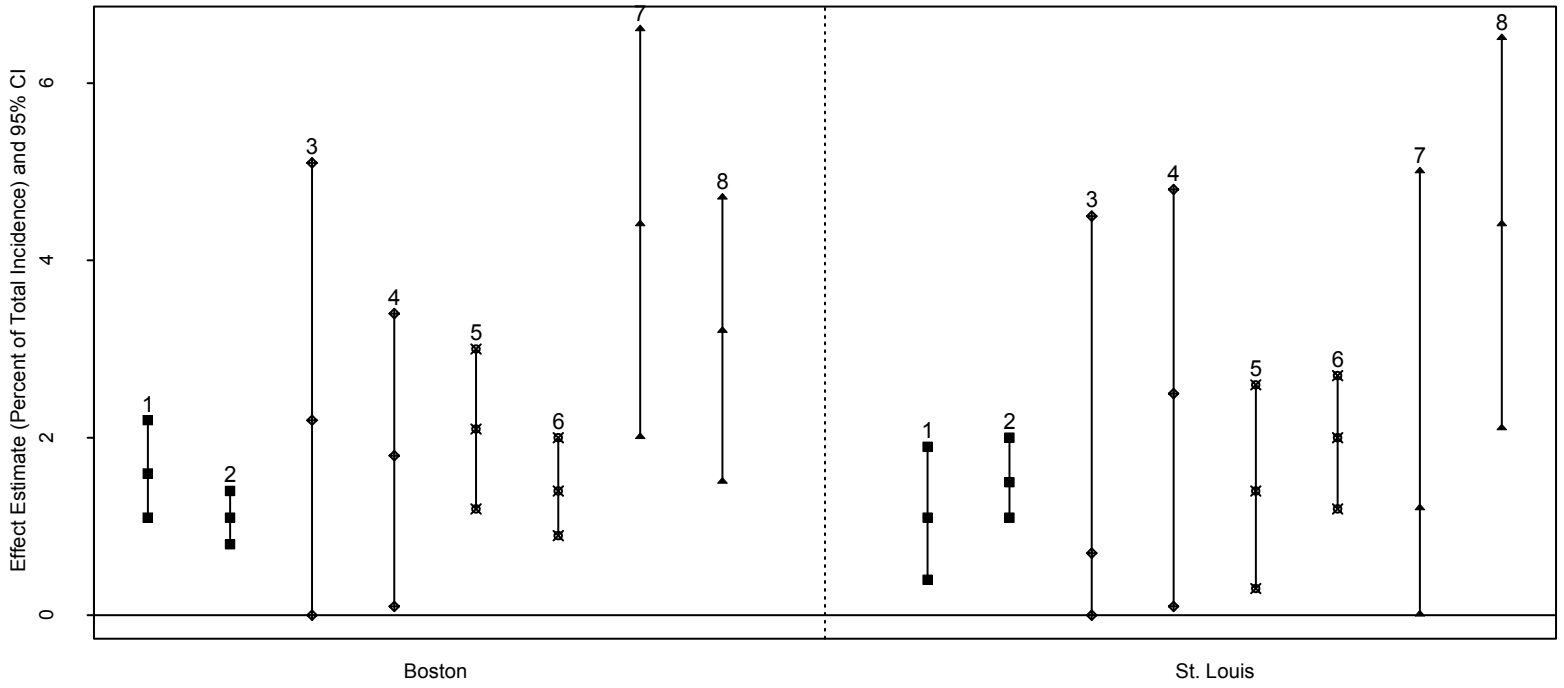

Figure 4-4. Estimated Annual Percent of Health Effects Associated with Short-Term Exposure to $PM_{2.5}$ (and 95 Percent Confidence Interval): Results Based on Single-City versus Multi-City Models‡

(Single-city models are always on the left, followed by the corresponding multi-city models)

Source: Abt (2003b)

1. Schwartz et al. (1996)* – single-city; non-accid. mort.
2. Schwartz et al. (1996)* – 6 cities; non-accid. mort.
3. Klemm et al. (2000)** – single-city; COPD mort.
4. Klemm et al. (2000)** – 6 cities; COPD mort.
5. Klemm et al. (2000)** – single-city; ischemic heart disease mort.
6. Klemm et al. (2000)** – 6 cities; ischemic heart disease mort.
7. Klemm et al. (2000)** – single-city; pneumonia mort.
8. Klemm et al. (2000)** – 6 cities; pneumonia mort.

*Reanalyzed in Schwartz (2003a)

** Reanalyzed in Klemm and Mason (2003)

‡In cases where the lower bound of the confidence interval was less than zero, the interval does not represent a 95% confidence interval. In these cases, the upper bound is the 97.5th percentile and the lower bound has been truncated at zero.

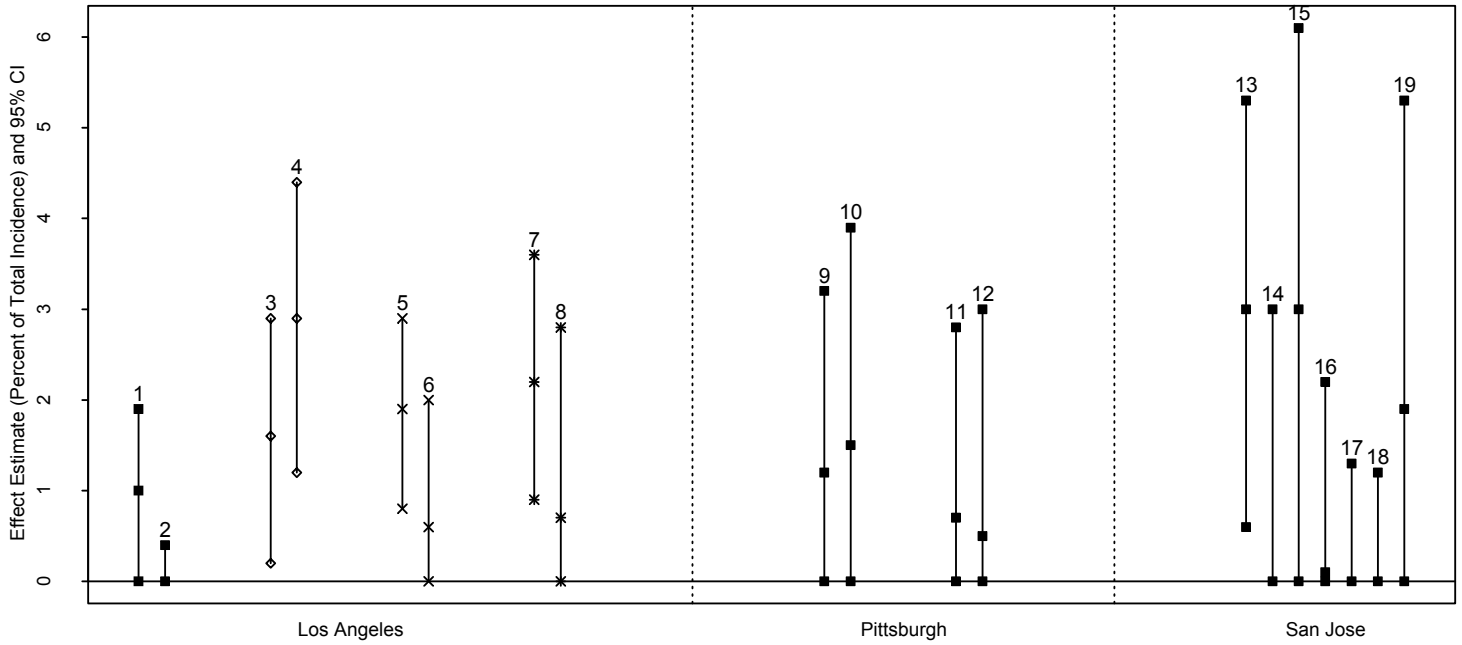

Figure 4-5. Estimated Annual Percent of Health Effects Associated with Short-Term Exposure to $PM_{2.5}$ (and 95 Percent Confidence Interval): Results Based on Single-Pollutant versus Multi-Pollutant Models‡

(Single-pollutant models are always on the left, followed by the corresponding multi-pollutant models)

Source: Abt (2003b)

1. Moolgavkar (2000a)* – non-accid. mort.
2. Moolgavkar (2000a)* – non-accid. mort.; + CO
3. Moolgavkar (2000a)* – cardiovasc. mort.
4. Moolgavkar (2000a)* – cardiovasc. mort.; + CO
5. Moolgavkar (2000b)* – cardiovasc. hosp. adm.
6. Moolgavkar (2000b)* – cardiovasc. hosp. adm.; + CO
7. Moolgavkar (2000c)* – COPD hosp. adm.
8. Moolgavkar (2000c)* – COPD hosp. adm.; + NO2
9. Chock et al. (2000) (age<75) – non-accid. mort.
10. Chock et al. (2000) (age<75) – non-accid. mort.; + CO, O3, SO2, PM10-2.5
11. Chock et al. (2000) (age>75) – non-accid. mort.
12. Chock et al. (2000) (age>75) – non-accid. mort.; + CO, O3, SO2, PM10-2.5
13. Fairley (1999)** – non-accid. mort.;
14. Fairley (1999)** – non-accid. mort.; + COH
15. Fairley (1999)** – non-accid. mort.; + NO3
16. Fairley (1999)** – non-accid. mort.; with SO4
17. Fairley (1999)** – non-accid. mort.; with NO2
18. Fairley (1999)** – non-accid. mort.; + CO
19. Fairley (1999)** – non-accid. mort.; + 8 hr O3

*Reanalyzed in Moolgavkar (2003)

**Reanalyzed in Fairley (2003)

‡In cases where the lower bound of the confidence interval was less than zero, the interval does not represent a 95% confidence interval. In these cases, the upper bound is the 97.5th percentile and the lower bound has been truncated at zero.

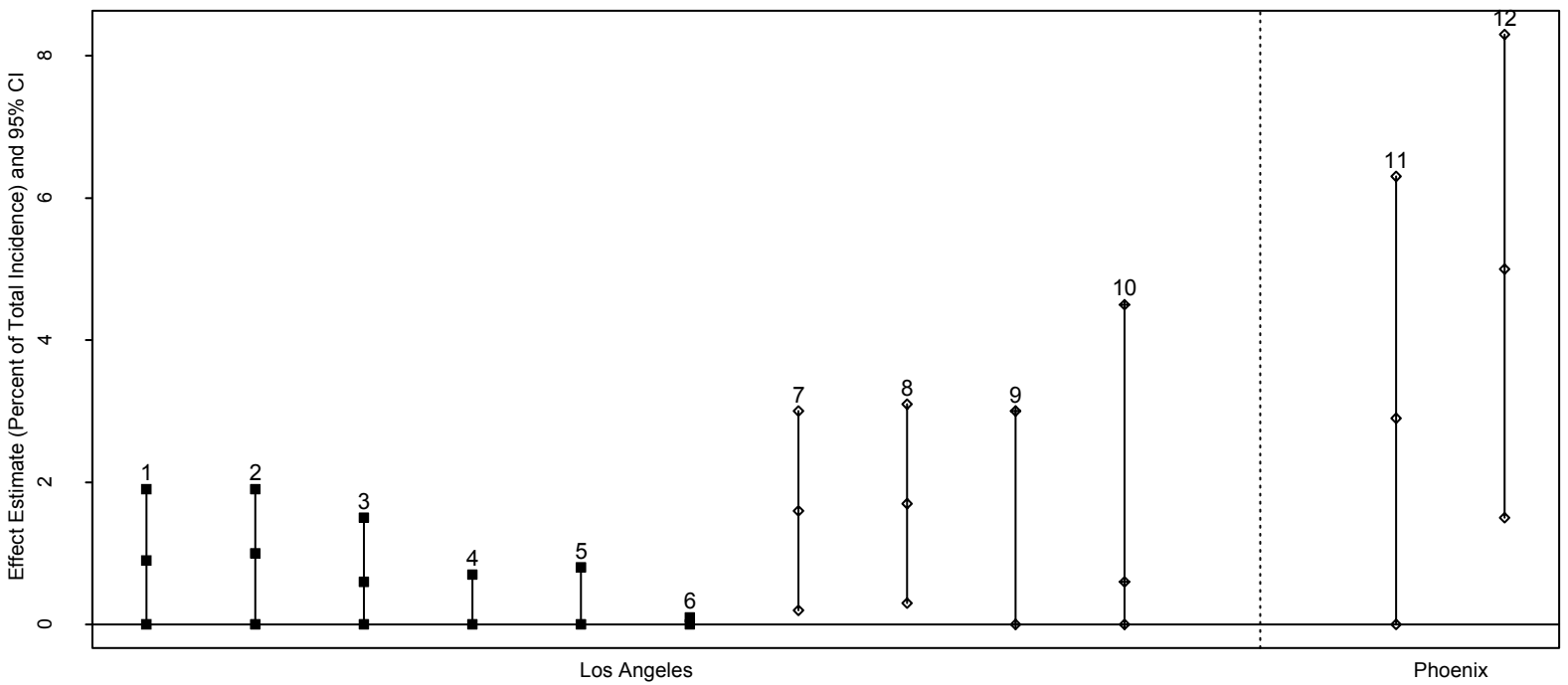

Figure 4-6. Estimated Annual Percent of Mortality Associated with Short-Term Exposure to $PM_{2.5}$ (and 95 Percent Confidence Interval): Effect of Different Lag Models‡

Source: Abt (2003b)

1. Moolgavkar (2000a)* – non-accid. mort. – 0-day lag
2. Moolgavkar (2000a)* – non-accid. mort. – 1-day lag
3. Moolgavkar (2000a)* – non-accid. mort. – 2-day lag
4. Moolgavkar (2000a)* – non-accid. mort. – 3-day lag
5. Moolgavkar (2000a)* – non-accid. mort. – 4-day lag
6. Moolgavkar (2000a)* – non-accid. mort. – 5-day lag
7. Moolgavkar (2000a)* – cardiovascular mort. – 0-day lag
8. Moolgavkar (2000a)* – cardiovascular mort. – 1-day lag
9. Moolgavkar (2000a)* – COPD mort. – 0-day lag
10. Moolgavkar (2000a)* – COPD mort. – 1-day lag
11. Mar et al. (2000)** – cardiovascular mort. – 0-day lag
12. Mar et al. (2000)** – cardiovascular mort. – 1-day lag

*Reanalyzed in Moolgavkar (2003)

**Reanalyzed in Mar et al. (2003)

‡In cases where the lower bound of the confidence interval was less than zero, the interval does not represent a 95% confidence interval. In these cases, the upper bound is the 97.5th percentile and the lower bound has been truncated at zero.

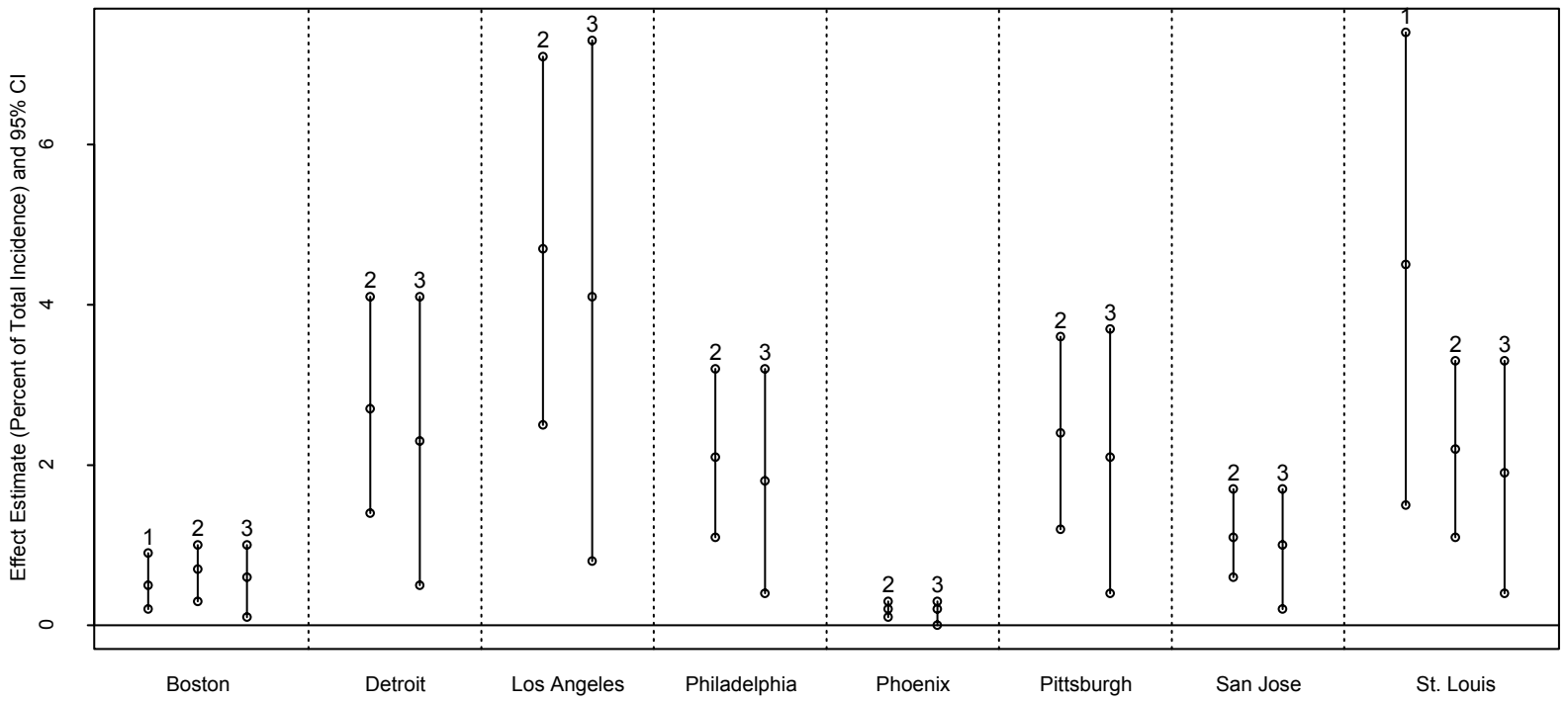

Figure 4-7. Estimated Annual Percent of Mortality Associated with Long-Term Exposure to $PM_{2.5}$ (and 95 Percent Confidence Interval): Single-Pollutant Models‡

Source: Abt (2003b)

1. Krewski et al. (2000) – Six Cities
2. Krewski et al. (2000) – ACS
3. Pope et al. (2002) - ACS extended

‡In cases where the lower bound of the confidence interval was less than zero, the interval does not represent a 95% confidence interval. In these cases, the upper bound is the 97.5th percentile and the lower bound has been truncated at zero.

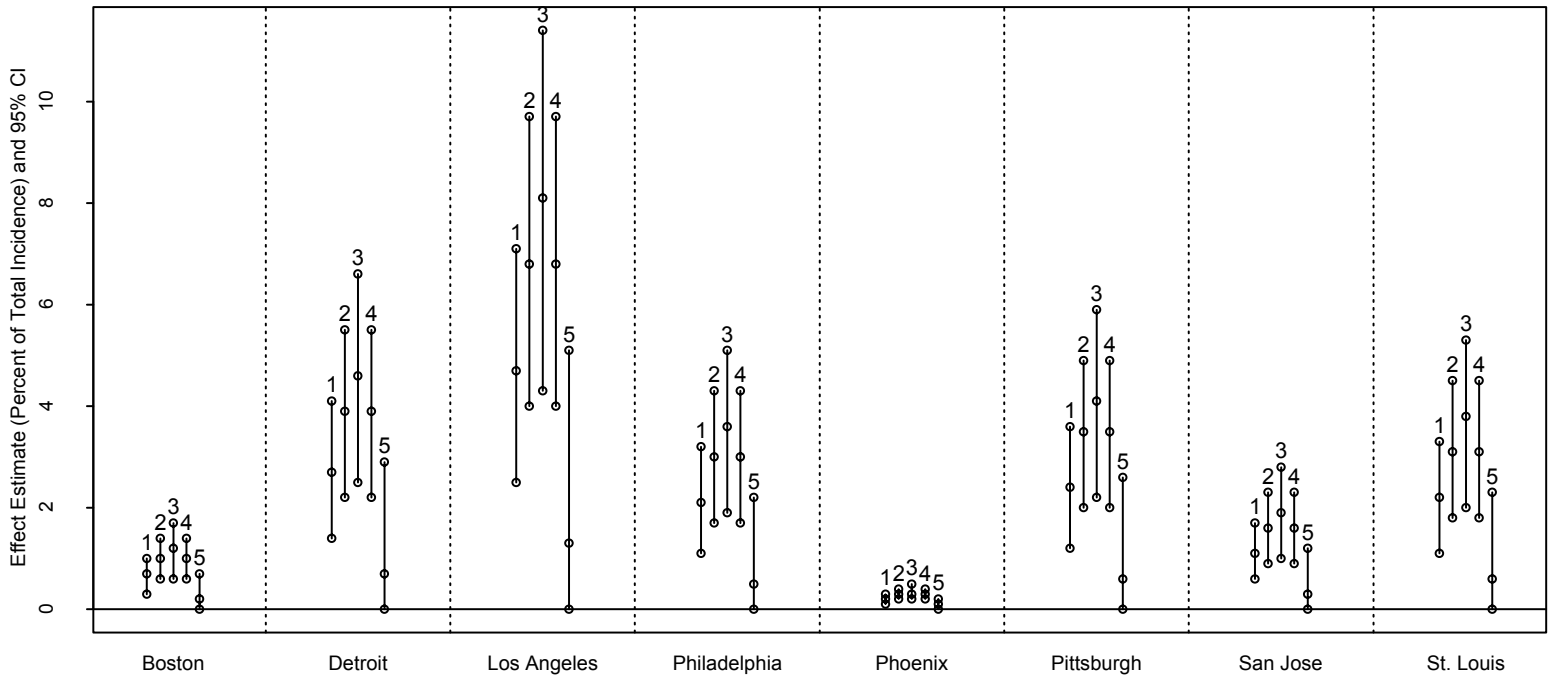

Figure 4-8. Estimated Annual Percent of Mortality Associated with Long-Term Exposure to $PM_{2.5}$ (and 95 Percent Confidence Interval): Single-Pollutant and Multi-Pollutant Models[‡]
(Single-pollutant models are always on the left, followed by the corresponding multi-pollutant models)
Source: Abt (2003b)

1. Krewski et al. (2000) – ACS
2. Krewski et al. (2000) – ACS – with CO
3. Krewski et al. (2000) – ACS – with NO2
4. Krewski et al. (2000) – ACS – with O3
5. Krewski et al. (2000) – ACS – with SO2

[‡]In cases where the lower bound of the confidence interval was less than zero, the interval does not represent a 95% confidence interval. In these cases, the upper bound is the 97.5th percentile and the lower bound has been truncated at zero.

Figure 4-5 compares risk estimates for three locations for various short-term exposure to $PM_{2.5}$ health endpoints examining single-pollutant versus multi-pollutant models. In some cases there is relatively little difference in the risk estimates between the single-pollutant and multi-pollutant models (e.g., Pittsburgh), while in other cases there are larger differences depending on the pollutant(s) added to the model.

Figure 4-6 illustrates the impact of using different lag models for short-term exposure mortality associated with $PM_{2.5}$ in two locations (Los Angeles and Phoenix). Generally, the risk estimates are greatest for the 0- and 1-day lag models. This is consistent with the earlier discussion of lags and short-term exposure mortality in the draft CD and Chapter 3.

Figures 4-7 and 4-8 show risk estimates for mortality related to long-term exposure to $PM_{2.5}$ based on single- and multi-pollutant models, respectively. The mean risk estimates for the single-pollutant models, which include C-R functions based on the Six Cities and ACS studies (Krewski et al., 2000), as well the updated ACS study (Pope et al., 2002), range from less than 0.5% in Boston and Phoenix to as high as roughly 4 to 5% of total mortality in Los Angeles and St. Louis, with most estimates falling in the 1.5 to 2.5% range. As shown in Figure 4-9, the risk estimates based on multi-pollutant models, involving addition of a single co-pollutant in the ACS study, show generally greater risk associated with $PM_{2.5}$ when CO, NO_2, or O_3 were added to the models and lower risk associated with $PM_{2.5}$ when SO_2 was added.[21]

Figure 4-9 shows risk estimates for short-term exposure mortality, hospital admission, and respiratory symptoms associated with $PM_{10-2.5}$ for five urban areas (Detroit, Philadelphia, Phoenix, Seattle, and St. Louis). For Detroit risk estimates are provided for non-accidental mortality, and two types of cause specific mortality, as well as several categories of cardiovascular and respiratory-related hospital admissions. For Philadelphia and Phoenix only cardiovascular mortality risk estimates were able to be developed and only asthma hospital admissions for Seattle. There is significant variability in the effect estimates, expressed as percent of total incidence, for mortality associated with $PM_{10-2.5}$, with three (Detroit, Philadelphia,

[21] The addition of a second pollutant reduced the number of cities available for estimating the C-R function from 50 for $PM_{2.5}$ alone to 44 with addition of CO, to 33 with addition of NO_2, to 45 with addition of O_3 and to 38 with addition of SO_2. The effect of the reduction in the number of cities available for each analysis is to increase the size of the confidence intervals.

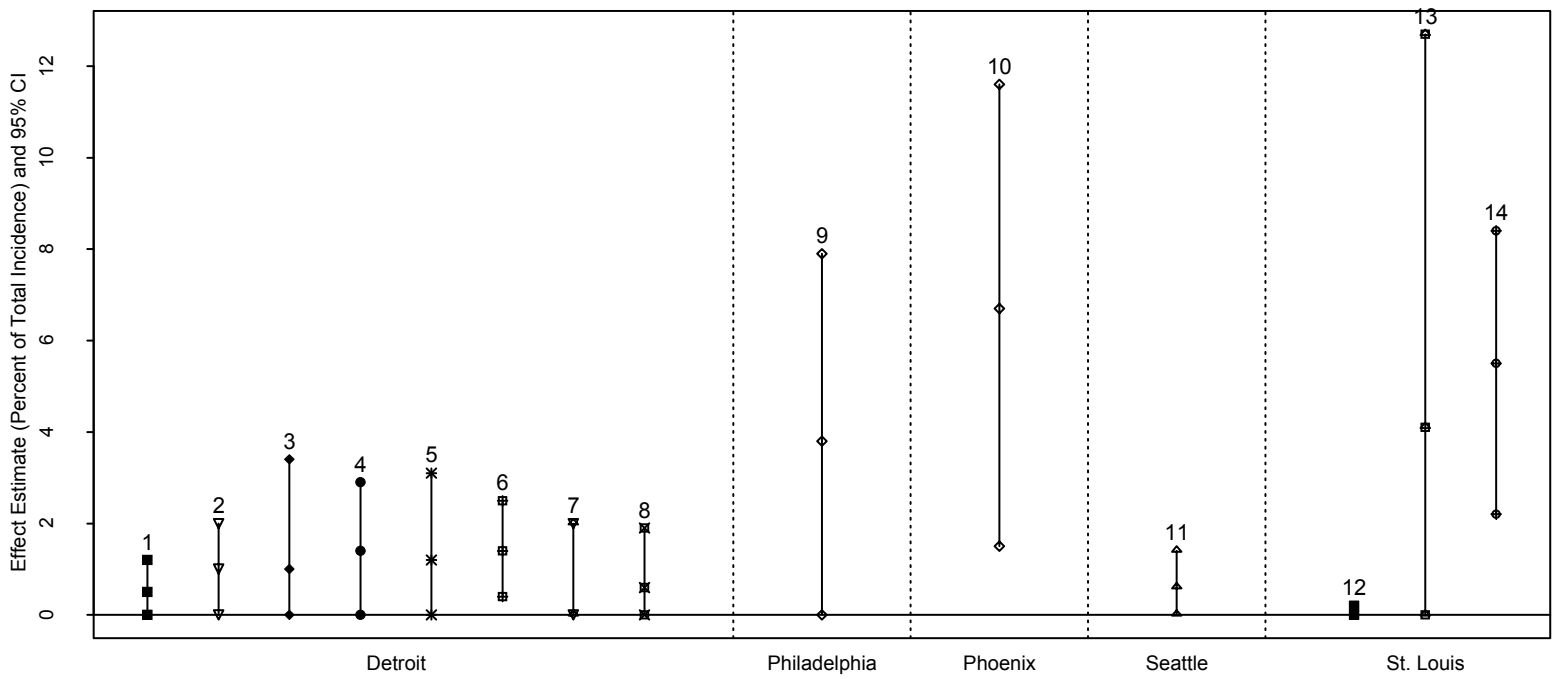

Figure 4-9. Estimated Annual Percent of Health Effects Associated with Short-Term Exposure to $PM_{10-2.5}$ (and 95 Percent Confidence Interval)[‡]

Source: Abt (2003b)

1. Lippmann et al. (2000)* – non-accid. mort.
2. Lippmann et al. (2000)* – circulatory mort.
3. Lippmann et al. (2000)* – respiratory mort.
4. Lippmann et al. (2000)* – pneumonia hosp. adms.
5. Lippmann et al. (2000)* – COPD hosp. adms.
6. Lippmann et al. (2000)* – ischemic heart disease hosp. adms.
7. Lippmann et al. (2000)* – dysrhythmia hosp. adms.
8. Lippmann et al. (2000)* – congestive heart failure hosp. adms.
9. Lipfert et al. (2000) – 7-county model; cardiovasc. mort.
10. Mar et al. (2000)** – cardiovasc. mort.
11. Sheppard et al. (1999)*** – asthma hosp. adms.
12. Schwartz et al. (1996)**** – non-accid. mort.
13. Schwartz and Neas (2000) – 6-city model; lower resp. symptoms
14. Schwartz and Neas (2000) – 6-city model; cough

*Reanalyzed in Ito (2003)
**Reanalyzed in Mar et al. (2003)
****Reanalyzed in Schwartz (2003b)

[‡]In cases where the lower bound of the confidence interval was less than zero, the interval does not represent a 95% confidence interval. In these cases, the upper bound is the 97.5th percentile and the lower bound has been truncated at zero.

and Phoenix) of the four urban areas showing mean effect estimates ranging from about 0.3 to over 6%. In the fourth area, St. Louis, no mean effect on mortality is reported for $PM_{10-2.5}$. However, for St. Louis there were mean effect estimates ranging from roughly 3 to 4% of total incidence for lower respiratory symptoms and cough.

Figures 4-10 through 4-14 summarize the PM_{10} risk estimates across the various assessment locations. Figure 4-10 shows risk estimates for total (non-accidental) mortality associated with short-term exposure to PM_{10} in 11 urban areas across the U.S. for a recent year of air quality. Most, but not all, of the mean effect estimates fall within a range of about 0.5 to 2.5% of total incidence. Only the risk estimates for Minneapolis, which are based on distributed lag models that are expected to yield higher estimates, result in higher mean effect estimates for short-term exposure mortality, showing a range of roughly 3.5 to 4%. The 12 urban areas included in the PM_{10} risk assessment vary considerably in terms of the relative contribution of $PM_{2.5}$ and $PM_{10-2.5}$ to total PM_{10} mass. Several cities are dominated by $PM_{2.5}$ (e.g., Detroit, Pittsburgh, St. Louis), several have roughly equal $PM_{2.5}$ and $PM_{10-2.5}$ (Los Angeles, San Jose, and Seattle) and at least one is dominated by $PM_{10-2.5}$ (Phoenix). There is no apparent pattern in the PM_{10} mortality risk estimates related to the relative ratio of fine- and coarse-fraction PM across the 12 urban areas.

Single- versus multi-city model short-term exposure mortality and morbidity risk estimates are displayed in Figures 4-11 and 4-13, respectively. Single-city models are always on the left, followed by corresponding multi-city models. Generally the mean effect estimates are similar between the single- and multi-city models for short-term mortality, although the 95% confidence intervals are generally smaller for the multi-city models due to larger sample size.

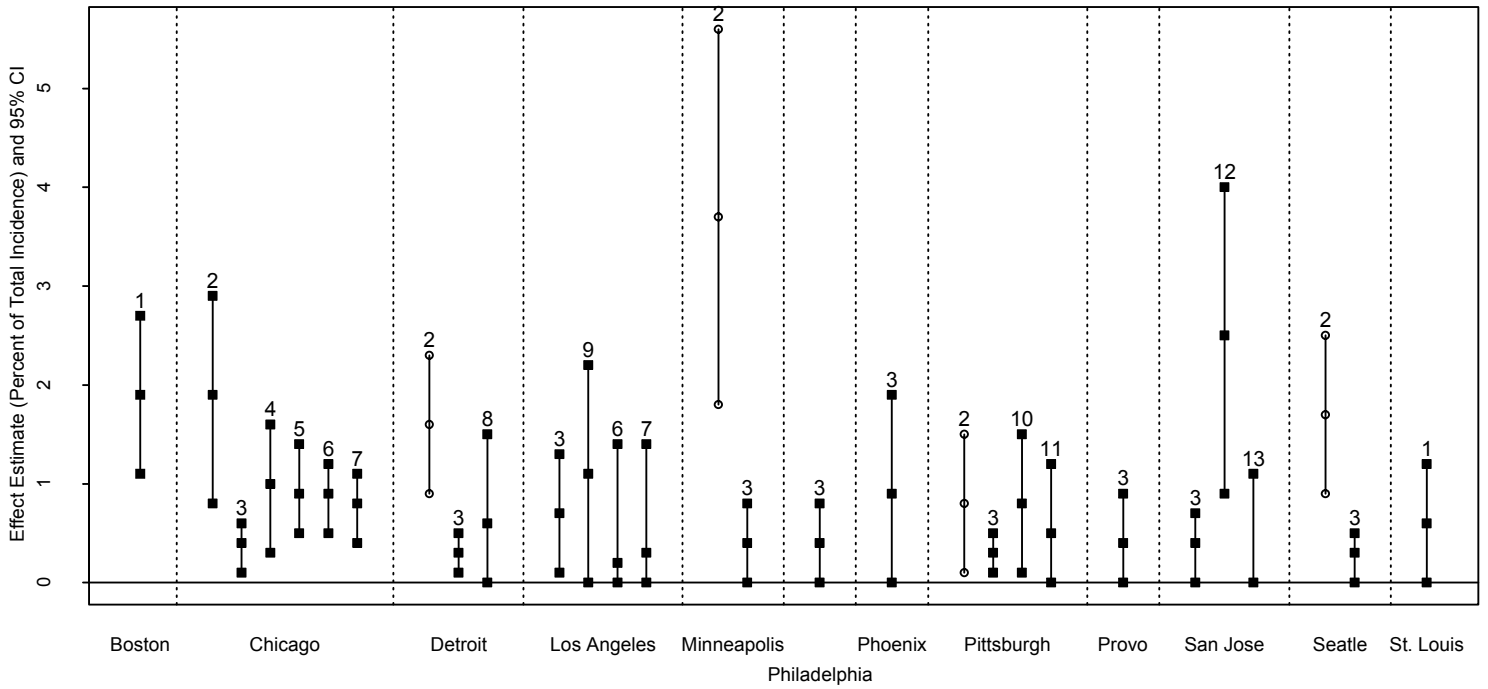

Figure 4-10. Estimated Annual Percent of Total (Non-Accidental) Mortality Associated with Short-Term Exposure to PM_{10} (and 95 Percent Confidence Interval): Single-Pollutant, Single-City Models‡

Source: Abt (2003b)

1. Klemm et al. (2000)*
2. Schwartz (2000b)** — distr. lag
3. Samet et al. (2000)***
4. Styer et al. (1995
5. Ito and Thurston (1996)
6. Moolgavkar (2000a)**** – 0-day lag
7. Moolgavkar (2000a)**** – 1-day lag
8. Lippmann et al. (2000)*****
9. Kinney et al. (1995)
10. Chock et al. (2000) – age<75
11. Chock et al. (2000) – age>75
12. Fairley (1999)****** – 0-day lag
13. Fairley (1999)****** – 1-day lag

*Reanalyzed in Klemm and Mason (2003)
**Reanalyzed in Schwartz (2003a)
***Reanalyzed in Dominici et al. (2002)
****Reanalyzed in Moolgavkar (2002)
*****Reanalyzed in Ito (2003)
******Reanalyzed in Fairley (2003)

‡In cases where the lower bound of the confidence interval was less than zero, the interval does not represent a 95% confidence interval. In these cases, the upper bound is the 97.5th percentile and the lower bound has been truncated at zero.

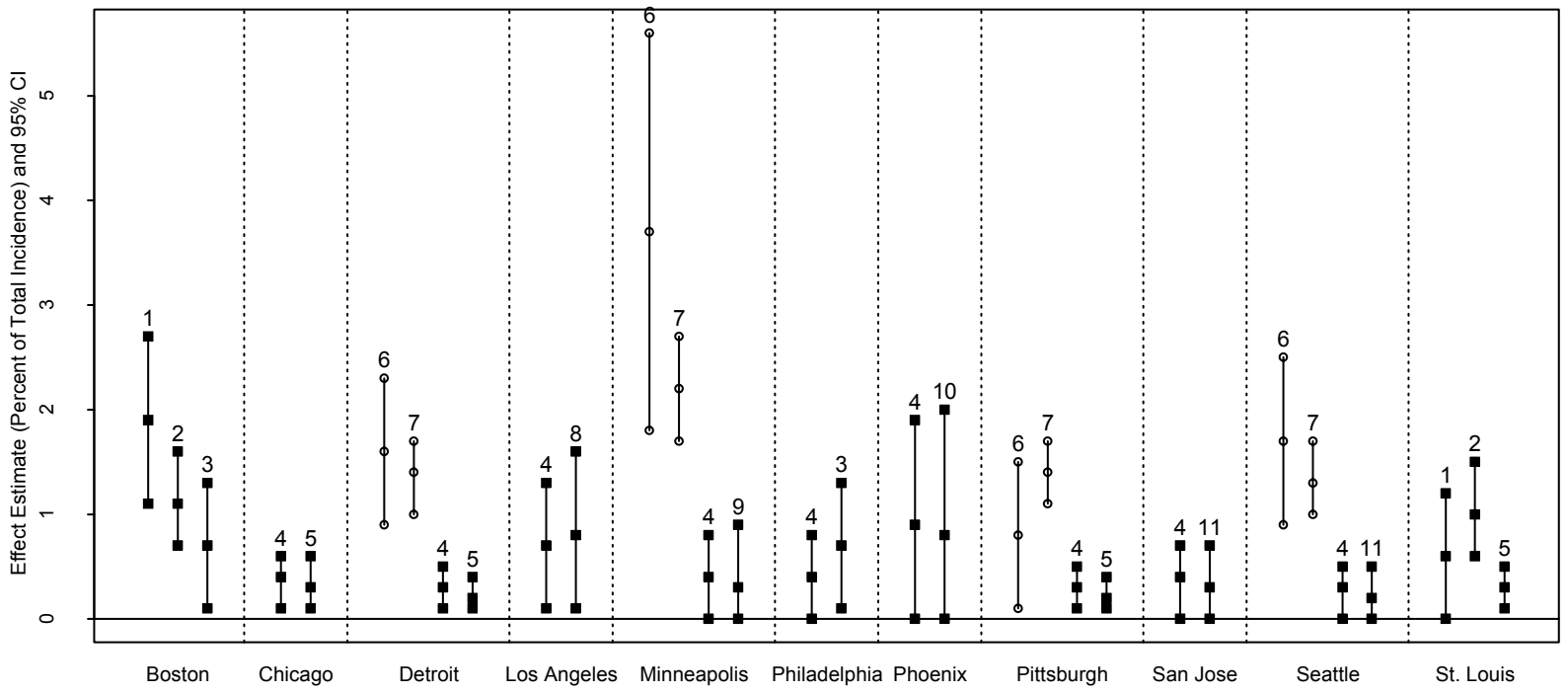

Figure 4-11. Estimated Annual Percent of Health Effects Associated with Short-Term Exposure to PM_{10} (and 95 Percent Confidence Interval): Results Based on Single-City versus Multi-City Models: Mortality (Single-city models are always on the left, followed by the corresponding multi-city models)‡ Source: Abt (2003b)

1. Klemm et al. (2000)* – single-city model
2. Klemm et al. (2000)* – six-city model
3. Samet et al. (2000)** – Northeast regional model
4. Samet et al. (2000)** – single-city model
5. Samet et al. (2000)** – Industrial Midwest regional model
6. Schwartz (2000b)*** – single-city model
7. Schwartz (2000b)*** – 10-city model
8. Samet et al. (2000)** – South California regional model
9. Samet et al. (2000)** – Upper Midwest regional model
10. Samet et al. (2000)** – Southwest regional model
11. Samet et al. (2000)** – Northwest regional model

*Reanalyzed in Klemm and Mason (2003)
**Reanalyzed in Dominici et al. (2002)
***Reanalyzed in Schwartz (2003a)

‡In cases where the lower bound of the confidence interval was less than zero, the interval does not represent a 95% confidence interval. In these cases, the upper bound is the 97.5th percentile and the lower bound has been truncated at zero.

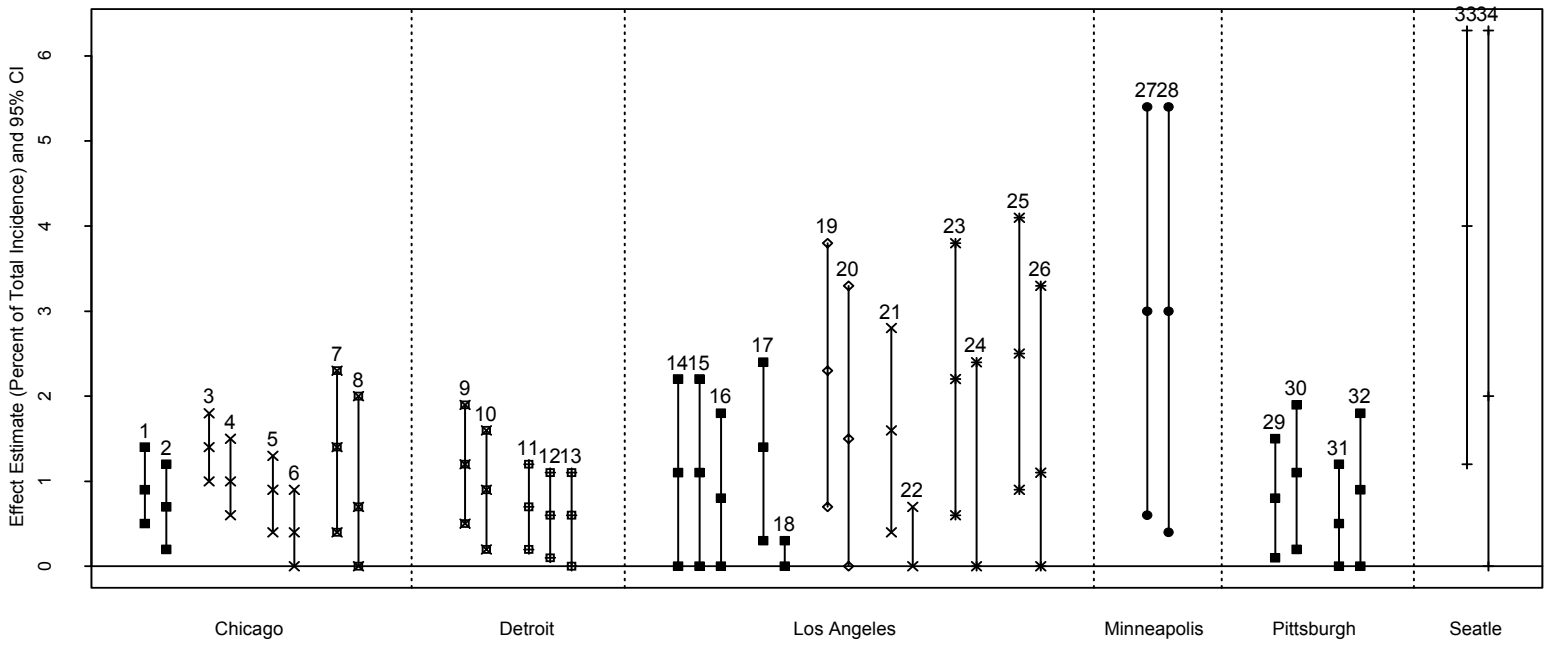

Figure 4-12. Estimated Annual Percent of Health Effects Associated with Short-Term Exposure to PM_{10} (and 95 Percent Confidence Interval): Results Based on Single-Pollutant versus Multi-Pollutant Models‡

(Single-pollutant models are always on the left, followed by the corresponding multi-pollutant models)

Source: Abt (2003b)

1. Ito and Thurston (1996) – non-accid. mort.
2. Ito and Thurston (1996) – non-accid. mort.; +O3
3. Moolgavkar (2000b)* – cardiovasc. hosp. adm.; 0-day lag
4. Moolgavkar (2000b)* – cardiovasc. hosp. adm.; 0-day lag +CO
5. Moolgavkar (2000b)* – cardiovasc. hosp. adm.; 1-day lag
6. Moolgavkar (2000b)* – cardiovasc. hosp. adm.; 1-day lag +CO
7. Morris and Naumova (1998) – cong. heart failure hosp. adm.
8. Morris and Naumova (1998) – cong. heart failure hosp. adm.; +CO, SO2, NO2, O3
9. Schwartz and Morris (1995) – cong. heart failure hosp. adm.
10. Schwartz and Morris (1995) – cong. heart failure hosp. adm.; +CO
11. Schwartz and Morris (1995) – ischemic heart disease hosp. adm.
12. Schwartz and Morris (1995) – ischemic heart disease hosp. adm.; +CO
13. Schwartz and Morris (1995) – ischemic heart disease hosp. adm.; +SO2
14. Kinney et al. (1995) – non-accid. mort.
15. Kinney et al. (1995) – non-accid. mort.; +O3
16. Kinney et al. (1995) – non-accid. mort.; +CO

17. Moolgavkar (2000a)* – non-accid. mort.; 2-day lag*
18. Moolgavkar (2000a)* – non-accid. mort.; 2-day lag; + CO*
19. Moolgavkar (2000a)* – cardiovasc. mort.; 2-day lag*
20. Moolgavkar (2000a)* – cardiovasc. mort.; 2-day lag; + CO*
21. Moolgavkar (2000b)* – cardiovasc. hosp. adm.; 2-day lag*
22. Moolgavkar (2000b)* – cardiovasc. hosp. adm.; 2-day lag; + CO*
23. Moolgavkar (2000c)* – COPD. hosp. adm.; 0-day lag
24. Moolgavkar (2000c)* – COPD. hosp. adm.; 0-day lag; + NO2
25. Moolgavkar (2000c)* – COPD. hosp. adm.; 1-day lag
26. Moolgavkar (2000c)* – COPD. hosp. adm.; 1-day lag; + NO2
27. Schwartz (1994b) – pneumonia hosp. adm.
28. Schwartz (1994b) – pneumonia hosp. adm.; + O3
29. Chock et al. (2000), age<75 – non-accid. mort.
30. Chock et al. (2000), age<75 – non-accid. mort.; + NO2
31. Chock et al. (2000), age>75 – non-accid. mort.
32. Chock et al. (2000), age>75 – non-accid. mort.; + CO
33. Yu et al. (2000) – asthma symptoms**
34. Yu et al. (2000) – asthma symptoms; + CO, SO2

*Reanalyzed in Moolgavkar (2003)

‡In cases where the lower bound of the confidence interval was less than zero, the interval does not represent a 95% confidence interval. In these cases, the upper bound is the 97.5th percentile and the lower bound has been truncated at zero.

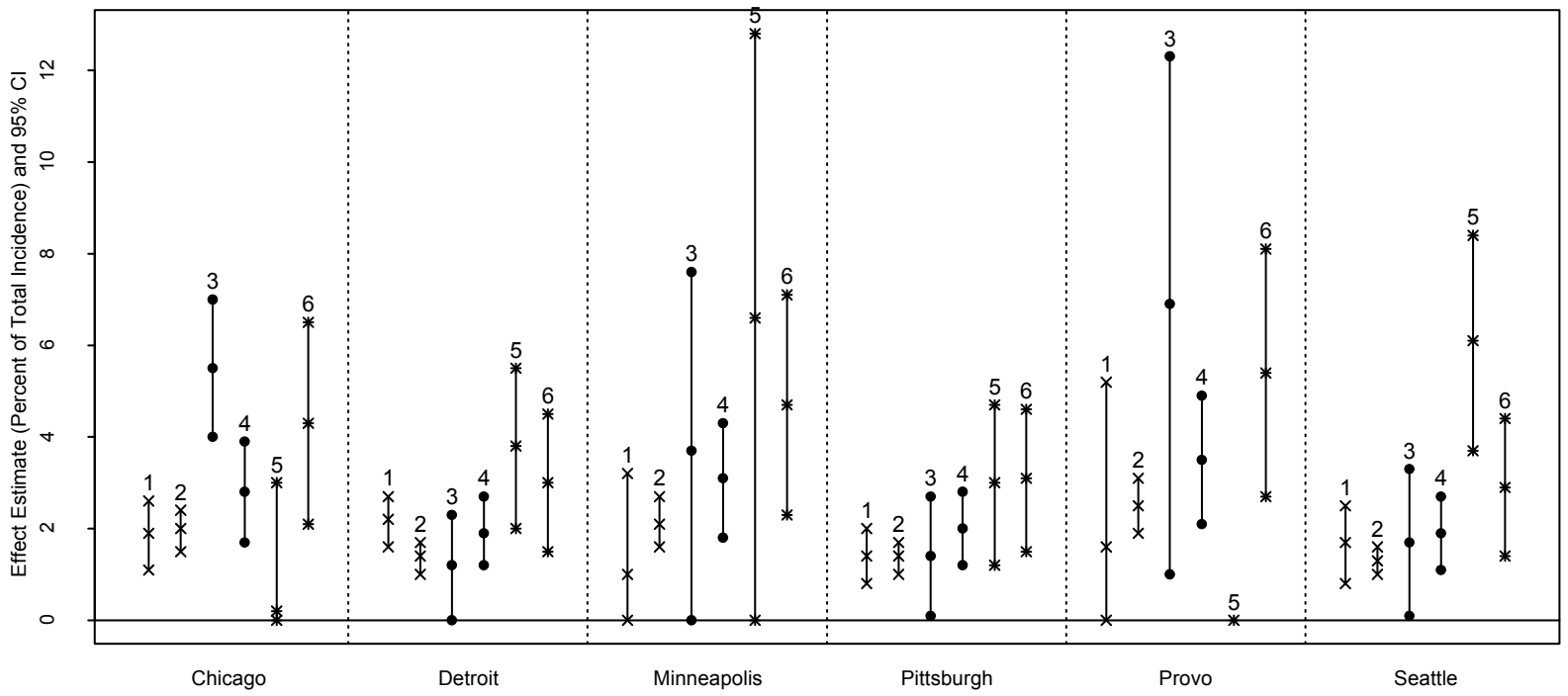

Figure 4-13. Estimated Annual Percent of Health Effects Associated with Short-Term Exposure to PM_{10} (and 95 Percent Confidence Interval): Results Based on Single-City versus Multi-City Models: Morbidity‡

(Single-city models are always on the left, followed by the corresponding multi-city models) Source: Abt (2003b)

1. Samet et al. (2000)* – single-city model; cardiovasc. hosp. adms.
2. Samet et al. (2000)* – 14-city model; cardiovasc. hosp. adms.
3. Samet et al. (2000)* – single-city model; pneumonia hosp. adms.
4. Samet et al. (2000)* – 14-city model; pneumonia hosp. adms.
5. Samet et al. (2000)* – single-city model; COPD hosp. adms.
6. Samet et al. (2000)* – 14-city model; COPD hosp. adms.

*Reanalyzed in Zanobetti and Schwartz (2003)

‡In cases where the lower bound of the confidence interval was less than zero, the interval does not represent a 95% confidence interval. In these cases, the upper bound is the 97.5th percentile and the lower bound has been truncated at zero.

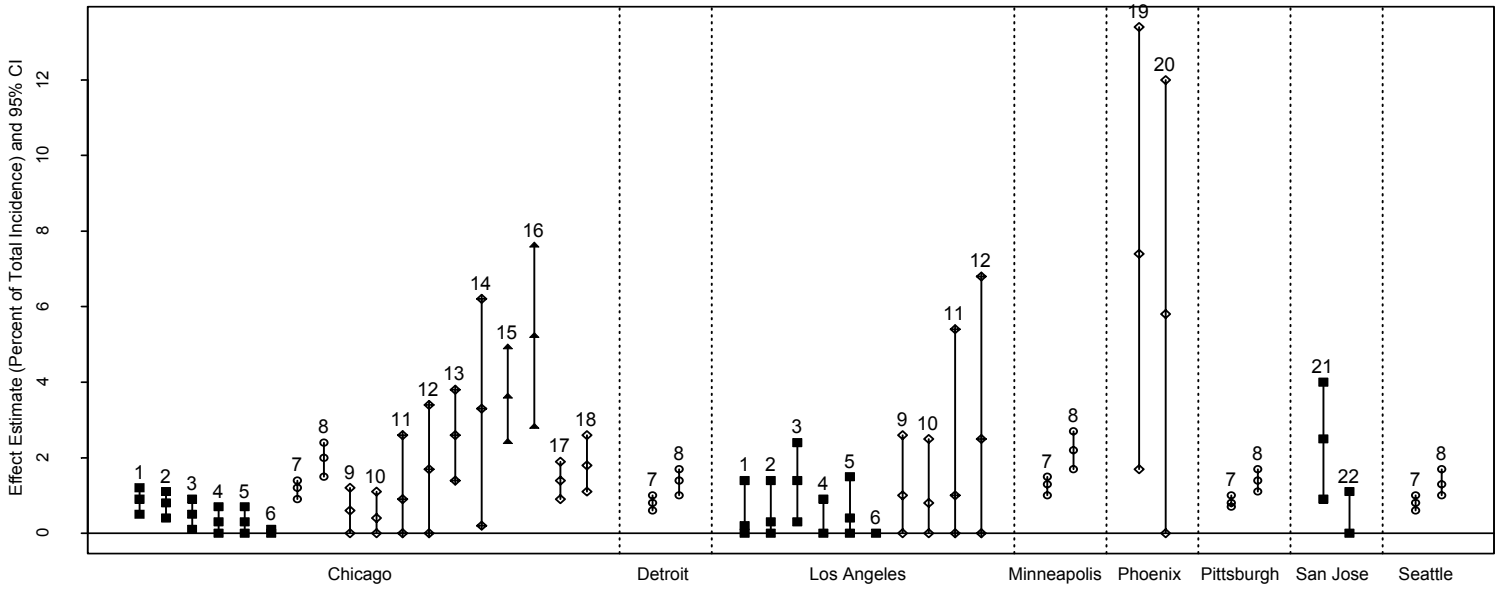

Figure 4-14. Estimated Annual Percent of Mortality Associated with Short-Term Exposure to PM_{10} (and 95 Percent Confidence Interval): Effect of Different Lag Models

Source: Abt (2003b)

1. Moolgavkar (2000a)* – single-city model; non-accid. mort.; 0-day lag
2. Moolgavkar (2000a)* – single-city model; non-accid. mort.; 1-day lag
3. Moolgavkar (2000a)* – single-city model; non-accid. mort.; 2-day lag
4. Moolgavkar (2000a)* – single-city model; non-accid. mort.; 3-day lag
5. Moolgavkar (2000a)* – single-city model; non-accid. mort.; 4-day lag
6. Moolgavkar (2000a)* – single-city model; non-accid. mort.; 5-day lag
7. Schwartz (2000b)** – 10-city model; all cause mort.; mean of 0- and 1-day lags
8. Schwartz (2000b)** – 10-city model; all cause mort.; distr. lag
9. Moolgavkar (2000a)* – single-city model; cardiovasc. mort.; 0-day lag
10. Moolgavkar (2000a)* – single-city model; cardiovasc. mort.; 1-day lag
11. Moolgavkar (2000a)* – single-city model; COPD mort.; 0-day lag
12. Moolgavkar (2000a)* – single-city model; COPD mort.; 1-day lag
13. Braga et al. (2001)** – 10-city model; COPD mort.; mean of 0- and 1-day lags
14. Braga et al. (2001)** – 10-city model; COPD mort.; distr. lag
15. Braga et al. (2001)** – 10-city model; pneumonia mort.; mean of 0- and 1-day lags
16. Braga et al. (2001)** – 10-city model; pneumonia mort.; distr. lag
17. Braga et al. (2001)** – 10-city model; cardiovasc. mort.; mean of 0- and 1-day lags
18. Braga et al. (2001)** – 10-city model; cardiovasc. mort.; distr. lag
19. Mar et al. (2000)*** – cardiovasc. mort.; 0-day lag
20. Mar et al. (2000)*** – cardiovasc. mort.; 1-day lag
21. Fairley (1999)**** – non-accid. mort.; 0-day lag
22. Fairley (1999)**** – non-accid. mort.; 1-day lag

*Reanalyzed in Moolgavkar (2002); **Reanalyzed in Schwartz (2003a);
***Reanalyzed in Mar et al. (2003); ****Reanalyzed in Fairley (2003)

‡In cases where the lower bound of the confidence interval was less than zero, the interval does not represent a 95% confidence interval. In these cases, the upper bound is the 97.5th percentile and the lower bound has been truncated at zero.

In contrast to the mortality figure, there were somewhat larger differences between single- and multi-city models for hospital admissions in four of the six risk assessment locations. Where there were appreciable differences between single- and multi-city models for a given location, there was no consistent pattern in terms of risk estimates being either smaller or larger based on the type of model.

Figure 4-12 compares single- and multi-pollutant model risk estimates for PM_{10} for short-term exposure mortality, hospital admissions, and respiratory symptom health endpoints. In most cases, addition of a single pollutant (e.g., CO, O_3, or NO_2) made relatively little difference in the PM_{10} risk estimates. The exception to this pattern is the set of risk estimates based on Moolgavkar (2000c) (reanalyzed in Moolgavkar, 2003), which show much larger declines in PM_{10} risk estimates in Los Angeles when CO or NO_2 are added to the model.

Risk estimates associated with various single day lag models, as well as a distributed lag model, associated with short-term exposure mortality for PM_{10} are shown in Figure 4-14. Risk estimates associated with the distributed lag model are always larger than the corresponding single-day lag based on the same study. In the two cities where 0- through 5-day lags were examined, the 0- and 1-day lag were associated with the largest risk estimates in Chicago and the 3-day lag was associated with the largest risk estimate in Los Angeles.

4.3.2 Sensitivity Analyses

Several sensitivity analyses were carried out to provide some perspective on the impact of various assumptions and uncertainties on the health risk estimates (see Table 4-9 for summary of different types of sensitivity analyses). These sensitivity analyses were conducted in each of the study areas and the complete results are in the technical support document (Abt, 2003b). The $PM_{2.5}$ risk results for one study area (Detroit), are shown here for some of the sensitivity analyses for illustrative purposes. Detroit has been selected because it provides an opportunity to examine both mortality and morbidity risk estimates and includes both single and multi-pollutant C-R functions.

For purposes of informing decisions about the PM NAAQS, we are interested in PM-related risks due to concentrations over background levels, where background includes PM from natural sources and transport of PM from sources outside of North America. One set of sensitivity analyses examined the impact of using the lower and upper end of the range of estimated

background concentrations cited in the draft CD. For Detroit, the use of alternative estimated $PM_{2.5}$ background levels had no impact on the short-term exposure mortality or hospital admission risk estimates because the LML for $PM_{2.5}$ in Ito (2003) [reanalysis of Lippmann et al. (2000)] was 6 $\mu g/m^3$, which is larger than the range of background levels considered in the sensitivity analysis (i.e., 2 to 5 $\mu g/m^3$). In the other eight $PM_{2.5}$ locations, using the upper and lower end of the range of estimated background generally had a small to modest impact, on the order of +/- 10-20% change in short-term exposure health endpoint risk estimates compared to use of the midpoint of the estimated range of background levels in the base case estimates. Alternative estimated $PM_{2.5}$ background levels had no impact on long-term exposure mortality in Detroit, or any of the other $PM_{2.5}$ locations, because the LML in the long-term studies was 10 or 11 $\mu g/m^3$, which is larger than the range of estimated $PM_{2.5}$ background levels.

A sensitivity analysis also has been conducted that focuses on the impact of using a varying estimated $PM_{2.5}$ background concentration instead of the fixed level used in each study area in the base case assessment. While there are no empirical data that provide the distribution of background $PM_{2.5}$ concentrations, there is information available from a rural monitoring network (IMPROVE) that provides the best available information on the potential variation in background concentrations. Based on an examination of distributions of ambient $PM_{2.5}$ 24-hour data at IMPROVE sites (Langstaff, 2003b), which are mostly located in rural environments near Class I areas, two lognormal distributions were estimated, one for the East (which was applied to Boston, Philadelphia, St. Louis, and Detroit) and one for the West (which was applied to San Jose, Los Angeles, Phoenix, and Seattle). It is important to recognize that all IMPROVE sites measure some $PM_{2.5}$ from anthropogenic sources, and this typically will inflate the standard deviation over what it would be if we were able to measure background concentrations as defined for purposes of this assessment. Because of this, we selected the smallest standard deviation among all IMPROVE sites in the East and in the West. The resulting standard deviation for the East is 3.8 and for the West is 1.5. These are not substantially smaller than the next two smallest standard deviations (3.8 and 4.0 in the East, and 1.6 and 1.6 in the West). The mean value for the Eastern distribution is 3.5 $\mu g/m^3$ and for the Western distribution is 2.5 $\mu g/m^3$ which are based on the midpoint of the estimated $PM_{2.5}$ background concentrations in the draft CD (p. 3-104). It should be noted that the estimated distributions for background may not fully reflect peak 24-h average natural background

concentrations which can be substantially higher than the annual or seasonal average natural background concentrations within areas affected by wildfires and dust storms and long range transport from outside North America (CD, p. 3-103). While the current $PM_{2.5}$ risk assessment, therefore, does not capture these unusual events, it should be noted that there are regulatory provisions to exclude such events for purposes of judging whether an area is meeting the current NAAQS (as noted above in section 2.7).

Background levels are likely to be positively correlated with "as is" levels in a given location. To reflect this likelihood, we assigned a background value to each day that is the same percentile in the distribution of daily background levels as the corresponding "as is" level is in its distribution. Using this approach, the differences in risk estimates of using a constant background versus a varying daily background are so small that they do not show up after rounding.

For $PM_{10-2.5}$, the sensitivity analysis examining the effects of using the lower and upper end of the range of estimated background levels showed no difference in any of the risk estimates for short-term exposure mortality or hospital admissions in Detroit between the base case (which used a value of 3 $\mu g/m^3$ for background) and the lower end where background was estimated to be 0 $\mu g/m^3$. Again there was no effect due to different background levels because the LML for the study providing the C-R relationship (i.e., 5 $\mu g/m^3$) was above the estimated background levels. At the upper end, where background was estimated to be 9 $\mu g/m^3$, the short-term exposure mortality and hospital admission risk estimates were reduced by about 60-70% (see Exhibit 7.14 in Abt (2003b). The effect of different background concentrations for the other $PM_{10-2.5}$ locations can be found in Exhibits D.84 and D.86 through D.89 in Abt (2003b). In addition, the effect of different background concentrations on PM_{10} risk estimates for the 12 PM_{10} locations can be found in Appendix D (Abt, 2003b).

One of the most significant uncertainties continues to be the issue of hypothetical thresholds below which there may be no $PM_{2.5}$ health effects. As discussed above in section 4.2.6.1, there is very limited evidence addressing whether or not thresholds exist for $PM_{2.5}$, with most analyses failing to find evidence that thresholds exist. As a sensitivity analysis, three hypothetical thresholds or cutpoints (10, 15, and 20 $\mu g/m^3$) are used to examine the potential impact on risk estimates for short-term exposure mortality and three different hypothetical thresholds or cutpoints (10, 12.5, and 15) are used to examine the potential impact on risk estimates for long-term

exposure mortality. In conjunction with defining such cutpoints for these sensitivity analyses, the slopes of the C-R functions have been increased to reflect the effect of hypothetical thresholds at the selected levels. A simple slope adjustment method has been used that assumes the slope for the upward-sloping portion of a hockeystick would be approximately a weighted average of the two slopes of a hockeystick - namely, zero and the slope of the upward-sloping portion of the hockeystick (see the technical support document (Abt, 2003b) for additional details). If the data used in a study do not extend down below the cutpoint or extend only slightly below the cutpoint, then the extent of the downward bias of the reported PM coefficient will be minimal or non-existent. This is the case, for example, when the cutpoint is 10 $\mu g/m^3$ or 12.5 $\mu g/m^3$ for long-term exposure mortality, given that the LMLs in the long-term exposure mortality studies were 10 $\mu g/m^3$ and 11 $\mu g/m^3$. The slope adjustment methods used in these sensitivity analyses are intended only to be illustrative of the possible impact on the risk estimates of alternative hypothetical thresholds. A more thorough evaluation of the effect of possible thresholds would require re-analysis of the original health and air quality data.

Table 4-10 shows the results of these sensitivity analyses examining the impact of hypothetical thresholds for short- and long-term exposure mortality risk estimates for the "as is" $PM_{2.5}$ levels in Detroit. From Table 4-10 it is apparent that the short-term exposure risk estimates are particularly sensitive to the application of hypothetical thresholds. For short-term exposure mortality, a hypothetical threshold of 10 $\mu g/m^3$ reduces the percent of total non-accidental mortality incidence associated with $PM_{2.5}$ by about 30% and the highest hypothetical threshold (20 $\mu g/m^3$) reduces it by about 70%. For long-term exposure mortality, a hypothetical threshold of 10 $\mu g/m^3$ has no impact on the risk estimates, because the LML for the epidemiological studies providing the C-R relationships in Detroit is 10 $\mu g/m^3$. A hypothetical threshold of 12.5 $\mu g/m^3$ reduces the percent of total mortality by about 37 to 43% depending on the study and model (i.e., single vs. multi-pollutant) used. A hypothetical threshold of 15 $\mu g/m^3$ reduces the base case long-term exposure mortality incidence associated with $PM_{2.5}$ by about 81 to 86%.

Another sensitivity analysis illustrates how different the risk estimates would be if the C-R functions used for short-term exposure mortality had used distributed lag models instead of single lag models. Schwartz (2000a) has shown in a study of short-term exposure mortality in 10 cities using PM_{10} as the indicator that a distributed lag model predicted the same relative risk that a single

lag model would have predicted if the coefficient was approximately two times what it was estimated to be. To simulate the possible impact of using a distributed lag model, the $PM_{2.5}$ coefficients were multiplied by two. As would be expected, the risk estimates are almost doubled using the distributed lag approximation (see Abt, 2003b; Appendix D).

The influence of using different periods of exposure on the risks estimated in long-term exposure mortality studies also has been examined in a sensitivity analysis. Two alternatives were examined: assuming the relevant $PM_{2.5}$ ambient concentrations were respectively 50% higher than and twice as high as the $PM_{2.5}$ ambient concentrations used in the original epidemiological study. The impact of these varying assumptions about the role of historical air quality on estimates of long-term exposure mortality associated with "as is" $PM_{2.5}$ concentrations is shown for Detroit in Table 4-11. Assuming that $PM_{2.5}$ concentrations were 50% higher than and twice as high as that in the original studies reduces long-term exposure mortality risk estimates by about one-third and one-half, respectively.

Table 4-10. Sensitivity Analysis: Estimated Annual Health risks of Short-Term and Long-Term Exposure Mortality Associated with "As Is" $PM_{2.5}$ Concentrations Using alternative Hypothetical Threshold Models, Detroit, MI, 2000*

Health Effects **	Study	Type	Ages	Lag	Other Pollutants in Model	Percent of total Incidence Associated with $PM_{2.5}$ Above Hypothetical Threshold ***			
						BASE CASE: Background = 3.5 µgm3	Hypothetical Threshold = 10 µgm3	Hypothetical Threshold = 15 µgm3	Hypothetical Threshold = 20 µgm3
Short-Term Exposure Mortality	Single Pollutant Models: Ito (2003) [reanalysis of Lippmann et al. (2000)]	Non-accidental	all	3 day		0.7% (0.0% - 2.2%)	0.5% (0.0% - 1.5%)	0.3% (0.0% - 0.9%)	0.2% (0.0% - 0.5%)
Long-Term Exposure Mortality						BASE CASE: Background = 3.5 µgm3	Hypothetical Threshold = 10 µgm3	Hypothetical Threshold = 12.5 µgm3	Hypothetical Threshold = 15 µgm3
	Single Pollutants Models: Krewski et al. (2000) - ACS	All cause	30+			2.7% (1.4% - 4.1%)	2.7% (1.4% - 4.1%)	1.7% (0.9% - 2.6%)	0.5% (0.2% - 0.7%)
	Pope et al. (2002) - ACS extended	All cause	30+			2.3% (0.5% - 4.1%)	2.3% (0.5% - 4.1%)	1.5% (0.3% - 2.7%)	0.4% (0.1% - 0.8%)
	Multi-Pollutant Models Krewski et al. (2000) - ACS	All cause	30+		CO	3.9% (2.2% - 5.5%)	3.9% (2.2% - 5.5%)	2.4% (1.4% - 3.5%)	0.7% (0.4% - 1.0%)
	Krewski et al. (2000) - ACS	All cause	30+		NO2	4.6% (2.5% - 6.6%)	4.6% (2.5% - 6.6%)	2.9% (1.5% - 4.2%)	0.8% (0.4% - 1.2%)
	Krewski et al. (2000) - ACS	All cause	30+		O3	3.9% (2.2% - 5.5%)	3.9% (2.2% - 5.5%)	2.4% (1.4% - 3.5%)	0.7% (0.4% - 1.0%)
	Krewski et al. (2000) - ACS	All cause	30+		SO2	0.7% (0.0%) - 2.9%)	0.7% (0.0% - 2.9%)	0.4% (0.0% - 1.8%)	0.1% (0.0% - 0.5%)

* This sensitivity analysis was performed only for those studies which reported highest measured levels in the study. See text for an explanation of the slope adjustment method.
** Health effects are associated with short-term exposure to $PM_{2.5}$ unless otherwise specified.
*** Health effects incidence was quantified across the range of PM concentrations observed in each study, when possible, but not below background level. Average background $PM_{2.5}$ is taken to be 3.5 $\mu g/m^3$ in the East and 2.5 $\mu g/m^3$ in the West. Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percentages are rounded to the nearest tenth.
Note: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the $PM_{2.5}$ coefficient.

4.3.3 Key Observations

Sections 4.3.1 and 4.3.2 have summarized the PM health risk estimates and sensitivity analyses associated with "as is" PM air quality levels. Summarized below are key observations resulting from this part of the risk assessment:

- A fairly wide range of risk estimates are observed for PM-related morbidity and mortality health effects across the urban areas analyzed associated with "as is" air quality for the three PM indicators ($PM_{2.5}$, $PM_{10-2.5}$, and PM_{10}).

- Most of the mean effect estimates for $PM_{2.5}$ for the base case analysis are in the range 0.5 to 1.5% for short-term exposure total non-accidental mortality. Generally, the mean risk estimates for the single- and multi-city models are roughly comparable in most of the urban areas analyzed. The impact of adding additional co-pollutants to the models was variable; sometimes there was relatively little difference, while in other cases there were larger differences.

- For long-term exposure mortality associated with $PM_{2.5}$, the mean effect estimates range from about 0.2% to as high as 4 to 5% with most estimates falling in the 1.5 to 2% range for single-pollutant models. Addition of a single co-pollutant resulted in higher risk estimates when CO, NO_2, or O_3 were added to the models for the ACS study and lower risk estimates when SO_2 was added.

- There is significant variability in the mortality effect estimates associated with $PM_{10-2.5}$, with three of the four urban areas showing mean effect estimates ranging from about 0.3 to over 6% of total incidence and a fourth area reporting no mean effect on mortality, but about 4 to nearly 6% of respiratory symptoms in this same area associated with $PM_{10-2.5.}$

- There is no apparent pattern in the PM_{10} mortality risk estimates related to the relative ratio of fine- and coarse-fraction PM across the 12 urban areas analyzed. Most, but not all, of the mean risk estimates for total (non-accidental) mortality associated with short-term exposure to PM_{10} in these 12 urban areas associated with a recent year of air quality range from about 0.5 to 2.5% of total incidence.

Table 4-11. Sensitivity Analysis: The Effect of Assumptions About Historical Air Quality on Estimates of Long-Term Exposure Mortality Associated with "As Is" $PM_{2.5}$ Concentrations, Detroit, MI, 2000

Health Effects	Study	Type	Ages	Other Pollutants in Model	Percent of Total Incidence		
					Base Case:Assuming AQ as Reported	Asuming relevant AQ 50% higher	Assuming relevant AQ twice as high
Long-Term Exposure Mortality	Single Pollutant Models Krewski et al. (2000) - ACS	All cause	30+		2.7% (1.4% - 4.1%)	1.8% (0.9% - 2.7%)	1.3% (0.7% - 2.1%)
	Krewski et al. (2000) - ACS	Cardiopulmonary	30+		5.3% (3.5% - 7.4%)	3.6% (2.3% - 5.0%)	2.7% (1.8% - 3.8%)
	Pope et al. (2002)	All cause	30+		2.3% (0.5% - 4.1%)	1.6% (0.3% - 2.8%)	1.2% (0.2% - 2.1%)
	Pope et al. (2002)	Cardiopulmonary	30+		3.3% (0.9% - 5.7%)	2.2% (0.6% - 3.8%)	1.7% (0.4% - 2.9%)
	Pope et al. (2002)	Lung Cancer	30+		4.5% (0.6% - 8.2%)	3.0% (0.4% - 5.5%)	2.3% (0.3% - 4.2%)
	Multi-Pollutants Models Krewski et al. (2000) - ACS	All cause	30+	CO	3.9% (2.2% - 5.5%)	2.6% (1.5% - 3.7%)	2.0% (1.1% - 2.8%)
	Krewski et al. (2000) - ACS	All cause	30+	NO2	4.6% (2.5% - 4.4%)	3.1% (1.6% - 4.4%)	2.3% (1.2% - 3.3%)
	Krewski et al. (2000) - ACS	All cause	30+	03	3.9% (2.2% - 5.5%)	2.6% (1.5% - 3.7%)	2.0% (1.1% - 2.8%)
	Krewski et al. (2000) - ACS	All cause	30+	SO2	0.7% (0.0% - 2.9%)	0.5% (0.0% - 1.9%)	0.4% (0.0% - 1.4%)

* Health effects incidence was quantified across the range of PM concentration observed in each study, when posible, but not below background level. Average background $PM_{2.5}$ is taken to be 3.5 ug/m^3 in the East and 2.5 ug/m^3 in the West. Estimates less than zero were truncated at zero. Incidences are rounded to the nearest 10; percents are rounded to the nearest tenth.

Note: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM2.5 coefficient.

The wide variability in risk estimates associated with a recent year of air quality for the three different PM indicators is to be expected given the wide range of PM levels across the urban areas analyzed and the variation observed in the C-R relationships obtained from the orginal epidemiology studies. Among other factors, this variability may reflect differences in populations, exposure considerations (e.g., degree of air conditioning use), differences in co-pollutants and/or other stressors, differences in study design, and differences related to exposure and monitor measurement error.

There are considerably fewer $PM_{10-2.5}$ risk estimates available for the five urban areas examined due to the much smaller number of epidemiology studies examining health effects associated with coarse fraction particles. The confidence intervals for the two locations with the highest effect estimates (i.e., Phoenix and Philadelphia), were fairly wide.

- Based on the results from the sensitivity analyses, the single most important factor influencing the risk estimates is whether or not a hypothetical threshold exists below which PM-related health effects do not occur.

- Based on the results from the sensitivity analyses, the following uncertainties have a moderate impact on the risk estimates in some or all of the cities: choice of an alternative estimated background level, use of a distributed lag model, and alternative assumptions about the relevant air quality for long-term exposure mortality. Use of a distribution of daily background concentrations had very little impact on the risk estimates.

During the previous review of the PM NAAQS, EPA provided an illustrative example based on the PM health risk assessment that showed the distribution of mortality risk associated with short-term exposure over a 1-year period. EPA concluded that peak 24-hour $PM_{2.5}$ concentrations appeared "to contribute a relatively small amount to the total health risk posed by an entire air quality distribution as compared to the risks associated with low to mid-range concentrations" (61 FR at 65652, December 13, 1996). Figure 4-15 (a,b) for $PM_{2.5}$ in St. Louis and Figure 4-16 (a,b) for $PM_{10-2.5}$ in Phoenix provide examples of annual distributions of 24-hour PM concentrations and the corresponding distribution of estimated mortality incidence based on two short-term exposure epidemiology studies included in the current PM risk assessment.[22]

[22]The St. Louis $PM_{2.5}$ example uses the C-R function for non-accidental mortality from Schwartz et al. (1996), reanalyzed in Schwartz (2003). The Phoenix $PM_{10-2.5}$ example uses the C-R function for cardiovascular mortality from Mar et al. (2000), reanalyzed in Mar et al. (2003).

- Consistent with the observation made in the previous PM NAAQS review, for both $PM_{2.5}$ and $PM_{10-2.5}$ the peak 24-hour concentrations contribute a relatively small amount to the total health risk associated with short-term exposures on an annual basis.

4.4 RISK ESTIMATES ASSOCIATED WITH JUST MEETING THE CURRENT $PM_{2.5}$ STANDARDS

4.4.1 Summary of Risk Estimates

The second part of the $PM_{2.5}$ risk assessment estimates the risk reductions that would result if the current annual $PM_{2.5}$ standard of 15 $\mu g/m^3$ and the current daily $PM_{2.5}$ standard of 65 $\mu g/m^3$ were just met in the assessment locations. This part of the risk assessment only considers those locations that do not meet the current standards based on 2002 data (i.e., Detroit, Philadelphia, Pittsburgh, Los Angeles, and St. Louis). Again, this section occasionally uses Detroit for illustrative purposes, however, the remaining risk estimates can be found in the technical support document (Abt Associates, 2003b). As noted previously, the 15 $\mu g/m^3$ annual average standard is the controlling standard in all five study areas, consequently, just meeting this standard also results in each of these areas meeting the 65 $\mu g/m^3$, 24-hour standard.

The percent rollback necessary to just meet the annual standards depends on whether the maximum or the spatial average of the monitor-specific annual averages is used. For the current risk assessment, the approach used to simulate just meeting the current annual average standard used the maximum of the monitor-specific annual averages as shown in Table 4-12. Since an area could potentially use the spatial average of the population-oriented monitors to determine whether or not it met the annual average standard, Table 4-12 also presents the percent rollbacks that would have resulted from using this alternative approach in each urban study area.

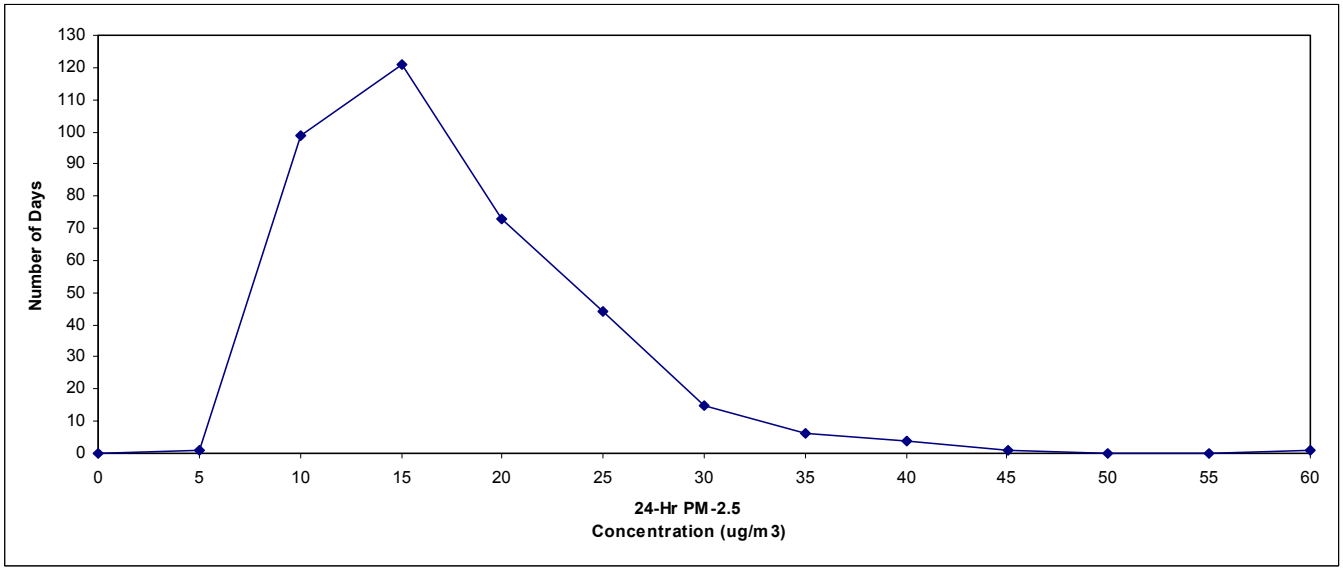

Figure 4-15a. Distribution of 24-Hour $PM_{2.5}$ Concentrations in St. Louis (2002 Air Quality Data)

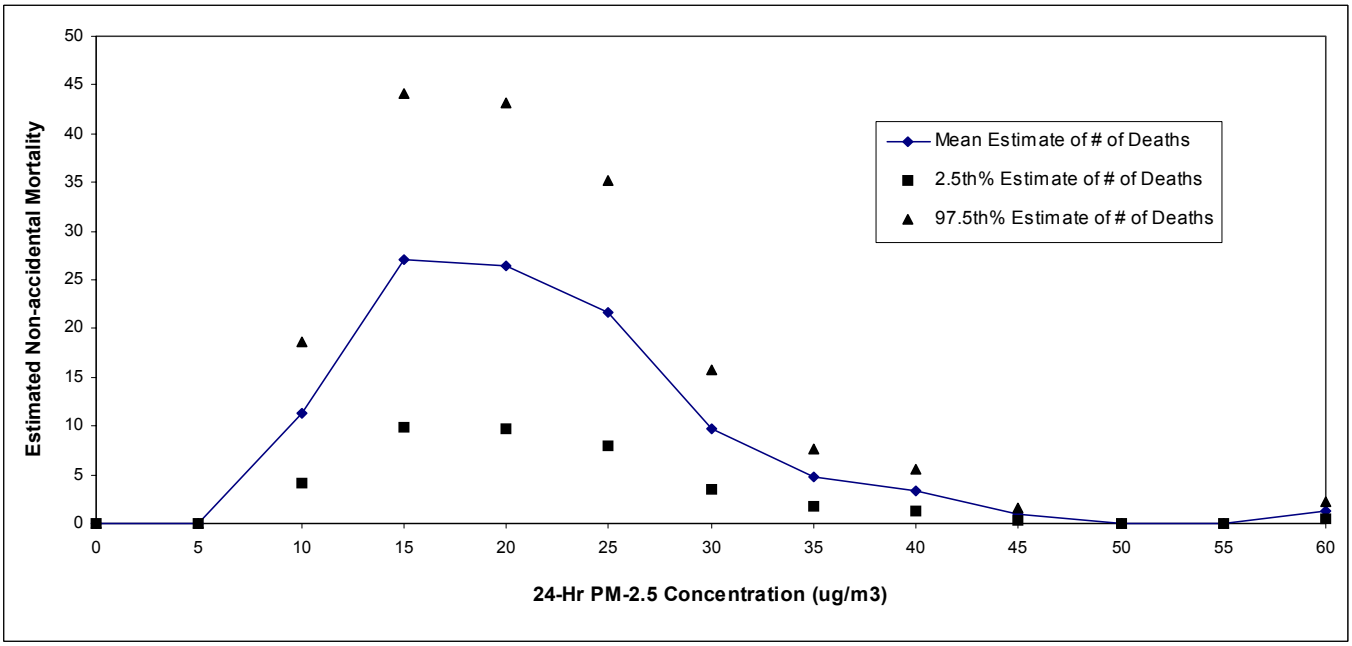

Figure 4-15b. Estimated Non-Accidental Mortality in St. Louis Associated with $PM_{2.5}$ Concentrations (2002 Air Quality Data)

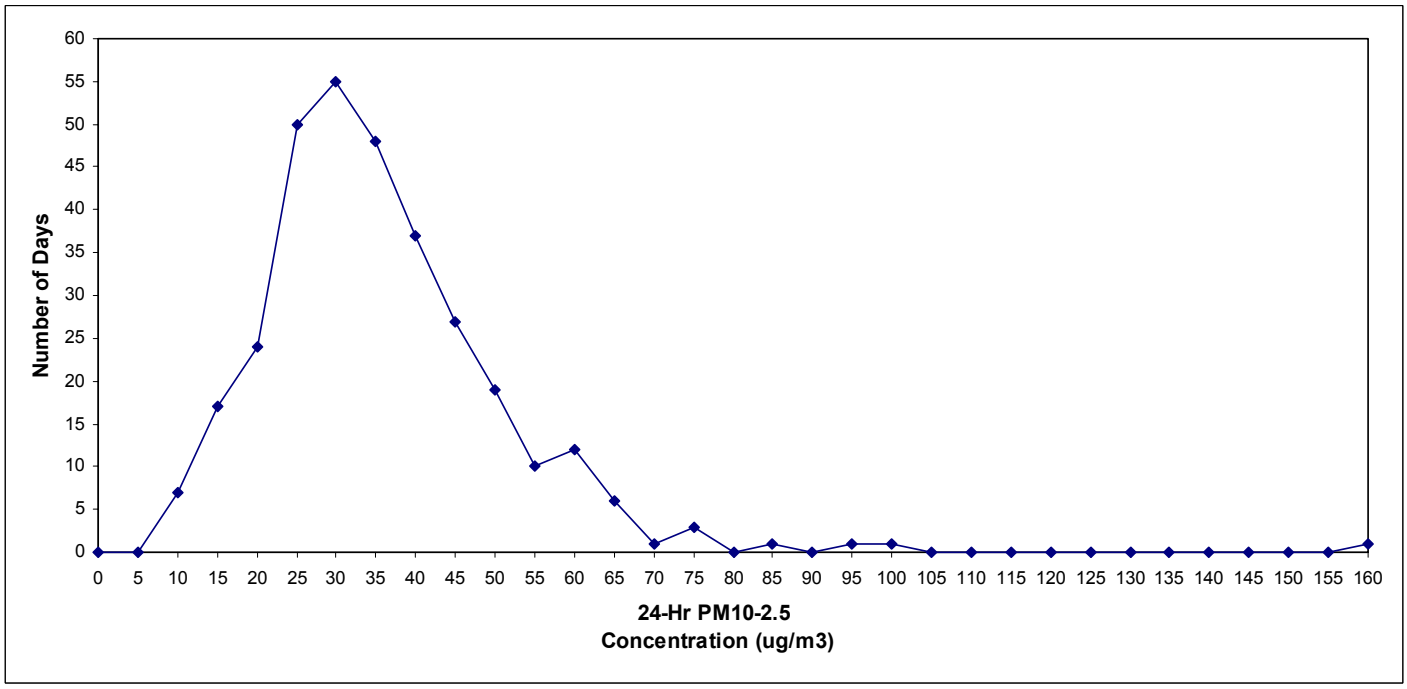

Figure 4-16a. Distribution of 24-Hour $PM_{10-2.5}$ Concentrations in Phoenix (1997 Air Quality Data)

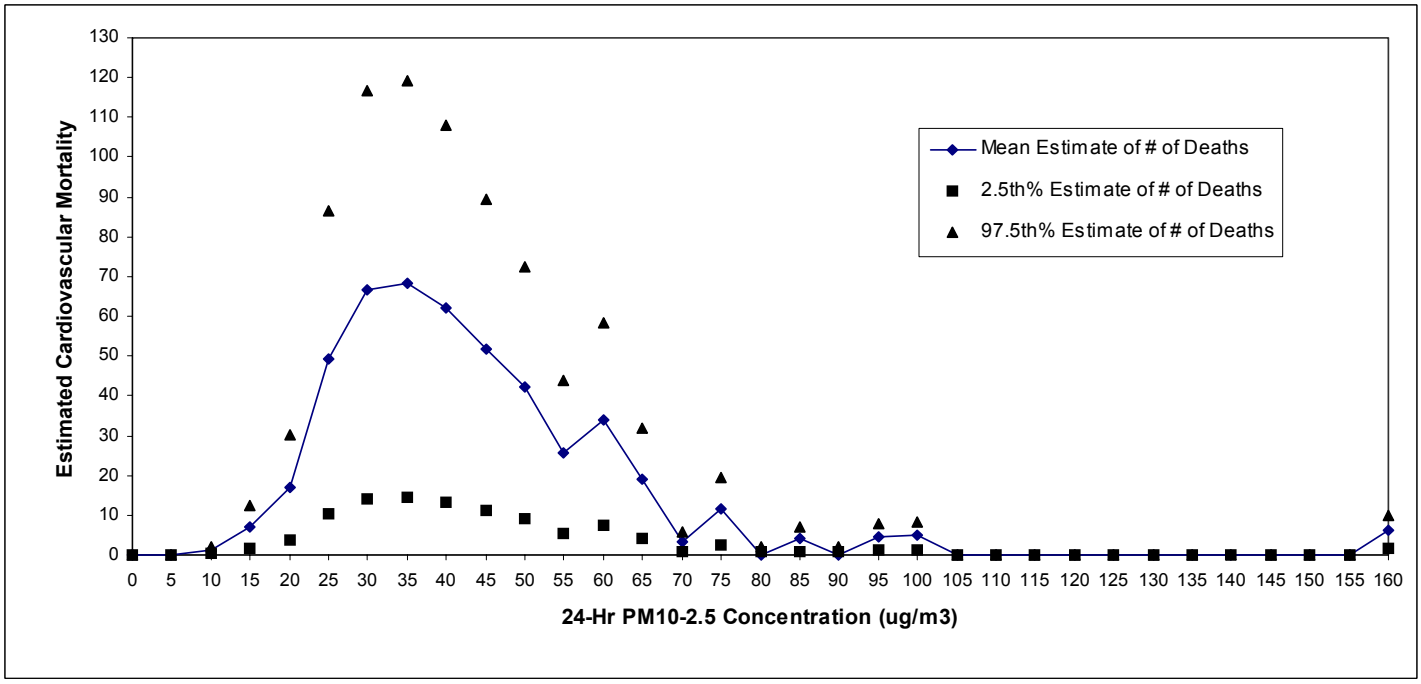

Figure 4-16b. Estimated Cardiovascular Mortality in Phoenix Associated with $PM_{10-2.5}$ Concentrations (1997 Air Quality Data)

Table 4-12. Air Quality Adjustments Required to Just Meet the Current Annual $PM_{2.5}$ Standard of 15 μg/m³ Using the Maximum vs. the Average of Monitor-Specific Averages

Assessment Location	Percent Rollback Necessary to Just Meet the Annual $PM_{2.5}$ Standard	
	Using Maximum of Monitor-Specific Annual Averages	Using Average of Monitor-Specific Annual Averages
Detroit	29.5%	10.9%
Los Angeles	41.9%	30.2%
Philadelphia	9.5%	0.0%
Pittsburgh	8.7%	0.0%
St. Louis	3.4%	0.0%

Source: Abt (2003b)

To gain some perspective on how the risk reductions compare across the four study areas, Table 4-13 shows the reduction in short- and long-term mortality incidence associated with going from "as is" $PM_{2.5}$ air quality (based on 2002 air quality data) to just meeting the current annual $PM_{2.5}$ standard. For short-term exposure mortality, single-pollutant, non-accidental mortality estimates are selected since they are available across the five study areas. For long-term exposure mortality, the ACS single-pollutant model reanalysis estimates for total (all cause) mortality are selected for comparison. Risk reductions for total mortality related to $PM_{2.5}$ long-term exposure associated with the Six City reanalysis are roughly two to three times larger than the ACS reanalysis estimates and risk reductions associated with the ACS extended study (Pope et al., 2002) are slightly smaller than the ACS reanalysis estimates shown in Table 4-14. In Table 4-13 risk reductions are expressed both as a percentage reduction in the $PM_{2.5}$-associated mortality and as a percentage of the total mortality due to $PM_{2.5}$ and other causes. As expected the largest reduction (250 deaths representing a 45.9% reduction in $PM_{2.5}$-related incidence and a 0.4% reduction in total mortality) for short-term exposure mortality is observed for Los Angeles which has the highest $PM_{2.5}$ ambient levels and largest population. The smallest reduction for short-term exposure mortality is observed for St. Louis (10 deaths representing a 3.4% reduction in $PM_{2.5}$-related incidence and a 0.0% reduction (taking into account rounding) in total mortality).

4.4.2 Sensitivity Analyses

The base case risk assessment uses a proportional rollback approach to adjust air quality distributions to simulate the pattern that would occur in an area improving its air quality so that it just meets the current annual average $PM_{2.5}$ standard. The support for this approach is briefly discussed in section 4.2.3 and in more detail in Appendix B of the technical support document (Abt, 2003b). While the available data suggest that this is a reasonable approach, other patterns of change are possible. In a sensitivity analysis an alternative air quality adjustment approach is used which reduces the top 10 percent of the distribution of $PM_{2.5}$ concentrations by 1.6 times as much as the lower 90 percent of concentrations. The result of this alternative hypothetical adjustment which reduces the highest days more than the rest of the distribution showed only a

Table 4-13. Comparison of Annual Estimates of Short- and Long-Term Exposure Mortality Reductions Associated with Just Meeting the Current $PM_{2.5}$ Standards*

Health Effect and Model	Urban Study Area	"As Is" Incidence	Reduction in Incidence	Percent of $PM_{2.5}$-Related Incidence Reduced	Reduction in Incidence Expressed as Percent of Total Incidence
non-accidental mortality, single pollutant, s.t. exposure, 3 day lag	Detroit	130 (0 - 400)	50 (0 - 140)	36.2% (n/a -36.5%)	0.3% (0.0 - 0.8%)
non-accidental mortality, single pollutant, s.t exposure, 1 day lag	Los Angeles	550 (0 - 1060)	250 (0 - 500)	45.9% (45.7 - 46%)	0.4% (0.0 - 0.9%)
non-accidental mortality, single pollutant, s.t. exposure, 0 day lag***	Philadelphia	340	30	9.5%	0.2%
non-accidental mortality (age ≥ 75), single pollutant, s.t. exposure, 0 day lag	Pittsburgh	60 (0 - 250)	10 (0 - 20)	8.8% (n/a - 8.9%)	0.1% (0.0 - 0.3%)
non-accidental mortality, single pollutant, s.t. exposure, lag 0/1day	St. Louis	260 (100 - 420)	10 (0 -10)	3.4% (3.4 - 3.4 %)	0% (0 - 0.1%)
total mortality (age ≥ 30)** long-term exposure	Detroit	500 (260 - 780)	310 (160 - 480)	62.6% (62.5 - 62.8%)	1.7% (0.9 - 2.5%)
total mortality (age ≥ 30)** long-term exposure	Los Angeles	2730 (1420 - 4140)	1980 (1030 -3010)	72.5% (72.2 - 72.7%)	3.4% (1.8 - 5.2%)
total mortality (age ≥ 30)** long-term exposure	Philadelphia	360 (190 - 560)	80 (40 - 130)	23.3% (23.2 - 23.4%)	0.5% (0.3 -0.7%)
total mortality (age ≥ 30)** long-term exposure	Pittsburgh	350 (180 - 540)	70 (40 - 110)	19.8% (19.8 - 19.9%)	0.5% (0.2 - 0.7%)
total mortality (age ≥ 30)** long-term exposure	St. Louis	500 (260 -770)	40 (20 - 60)	8.1% (8.1 - 8.1%)	0.2% (0.1 - 0.3%)

*Risk reductions are relative to the "as is" (year 2002) air quality base case risk estimates.

**These risk reductions are based on the Krewski et al. (2000) ACS reanalysis, single-pollutant model.

n/a = Incidence reduction as a percent of PM-related incidence was calculated by dividing the incidence reduction achieved by rolling back $PM_{2.5}$ above background to just meet the current standards by the incidence associated with "as is" $PM_{2.5}$ above background. In those cases in which the incidence associated with "as is" $PM_{2.5}$ above background was estimated to be zero, this percent could therefore not be calculated.

***The Lipfert et al. (2000) study does not provide the statistical uncertainties surrounding the $PM_{2.5}$ coefficients and, therefore, the 95% confidence intervals could not be calculated.

small difference (less than 1%) in the percent change in PM-associated incidence (see Exhibit 7.8 and Appendix D in Abt, 2003b).

4.4.3 Key Observations

Sections 4.4.1 and 4.4.2 have summarized the PM health risk estimates and sensitivity analyses associated with just meeting the current $PM_{2.5}$ standards. Summarized below are key observations resulting from this part of the risk assessment:

- There is a wide range of reductions in $PM_{2.5}$-related incidence across the five urban areas analyzed which is largely due to the varying amount of reduction in ambient $PM_{2.5}$ concentrations required in these urban areas to just meet the current $PM_{2.5}$ standard. For example, using single-pollutant models the percent of $PM_{2.5}$-related incidence reduced for short-term, non-accidental mortality ranges from about 45% in Los Angeles to about 3% in St. Louis. Similarly, using the ACS study the percent of $PM_{2.5}$-related incidence reduced for long-term exposure mortality ranges from roughly 70% in Los Angeles to about 8% in St. Louis.

- The risk estimates associated with just meeting the current $PM_{2.5}$ standards incorporate several additional sources of uncertainty, including: (1) uncertainty in the pattern of air quality concentration reductions that would be observed across the distribution of PM concentrations in areas attaining the standards ("rollback uncertainty") and (2) uncertainty concerning the degree to which current PM risk coefficients may reflect contributions from other pollutants, or the particular contribution of certain constituents of $PM_{2.5}$, and whether such constituents would be reduced in similar proportion to the reduction in $PM_{2.5}$ as a whole. At least one alternative approach to rolling back the distribution of daily $PM_{2.5}$ concentrations, in which the upper end of the distribution of concentrations was reduced by a greater amount than the rest of the distribution, had little impact on the risk estimates.

4.5 RISK ESTIMATES ASSOCIATED WITH JUST MEETING ALTERNATIVE $PM_{2.5}$ AND $PM_{10-2.5}$ STANDARDS

In the next draft of the PM Staff Paper, staff plans to include risk estimates associated with just meeting alternative $PM_{2.5}$ and alternative $PM_{10-2.5}$ standards. The standards analyzed will be based on the range of standards recommended for consideration in Chapter 6 of this draft Staff Paper, taking into account CASAC and public comments on the proposed ranges received as part of the review of this Staff Paper.

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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 WELFARE EFFECTS

5.1 INTRODUCTION

This chapter summarizes key information relevant to assessing the welfare 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 climate change and solar radiation (section 5.5). For each category of PM-related effects, this chapter presents a brief summary of the relevant scientific information and a 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 climate change and solar radiation, 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. Preliminary staff conclusions and recommendations related to secondary standards for PM are presented in Chapter 6.

It is important to note that the discussion of PM-related effects on visibility, vegetation and ecosystems, and climate change and solar radiation 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, 1998), 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), recent reports of the National Academy of Sciences (NAS, 2001) and the Intergovernmental Panel on Climate Change (IPCC, 1998, 2001a,b), and numerous other 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), the World Meteorological Organization (WMO), and the United Nations Environment Programme (UNEP).

5.2 EFFECTS ON VISIBILITY

Visibility impairment 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 and perceived color of distant objects to an observer and reduces the range at which they can be distinguished from the background. The effect of ambient particles on visibility is dependant upon particle size and composition, atmospheric illumination, the optical 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 presented 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:

- Data analyses for a number of cities in which 1999 daily $PM_{2.5}$ measurements are correlated with visibility data from Automated Surface Observation System (ASOS) installations.

- 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.

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, 4-153). Visibility effects are manifested in two principal ways: (1) as local impairment (e.g., localized hazes and plumes); and (2) as regional haze. This distinction is significant both to the ways in which visibility goals may be set and to how 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 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. 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]

Urban visibility impairment often results from the combined effect of stationary, mobile, and area source emissions. Complex local meteorological conditions may contribute to such impairment as well. 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. 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.

[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 both plants, it was found that sulfur dioxide emissions were contributing to visibility impairment in Grand Canyon National Park.

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 and industrialized 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 with 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 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 4-166).

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 5.2.4). 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 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, 4-155):

$$\sigma_{ext} = \sigma_{sg} + \sigma_{ag} + \sigma_{sp} + \sigma_{ap} \quad (5\text{-}1)$$

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 kilometers. 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 for typical atmospheric aerosols by a combination of empirical approaches and theoretical calculations. For certain aerosol constituents, extinction efficiencies increase significantly with increases in relative humidity.

The IMPROVE visibility monitoring program has developed an algorithm for calculating total light extinction as the sum of aerosol light extinction for each of the five major fine particle components and coarse fraction mass, plus 10 Mm^{-1} for Rayleigh extinction. The mass for each component is multiplied by its dry extinction efficiency, and in the case of sulfate and nitrate, by a relative humidity adjustment factor to account for their hygroscopic behavior (CD, p. 4-170). The relative humidity adjustment factor increases significantly with higher humidity, ranging from about 2 at 70%, to 4 at 90%, and 10 at 95% relative humidity (CD, p. 4-171, Figure 4-38).

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 the sun, whether the scene is shaded, 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 because lighter colored terrain will reflect more light than darker colored terrain.

One's ability to clearly see an object is degraded both by the reduction of image forming light (transmitted radiance) 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 the inability to see stars in the daytime due to the brightness of the sky caused by Rayleigh scattering. At night, in the absence of sunlight, the path radiance is small so the stars are readily seen. More light is generally scattered in the forward direction than back towards the light

source, thus causing a haze to appear bright when looking at scenes that are 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 his 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 the potential impact of various air quality management strategies on visibility (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 large 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):

$$\text{Visual Range (km)} = 3.91/\sigma_{ext}(\text{km}^{-1}) \qquad (5\text{-}2)$$

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. Deciview 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 (Pitchford and Malm, 1994). The deciview metric is also useful in defining goals for perceptible changes in visibility conditions under regulatory programs. Deciview can be calculated from the light extinction coefficient (σ_{ext}) by the equation:

$$\text{Haziness (dv)} = 10 \ln(\sigma_{ext}/10\ Mm^{-1}) \qquad (5\text{-}3)$$

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

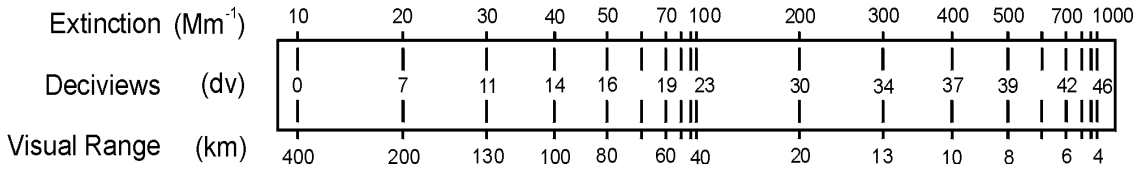

Figure 5-1. Relationship Between Light Extinction, Deciviews, and Visual Range.
Source: Malm, 1999.

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 it 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 (see section 5.2.3.1 for further discussion).

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, 0_2). The magnitude of Rayleigh scattering depends on the wavelength or color of the light being scattered as well as on the density of gas in the atmosphere and varies by site elevation, generally from 9 to 11 Mm^{-1} for green light at about 550 ηm (CD, p. 4-158). A standard value of 10 Mm^{-1} is often used to simplify comparisons of light extinction values across a number of sites with varying elevations (Malm, 2000; CD, p. 4-158). 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 conditions can lead to 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, background PM is defined for the purpose of this document 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 ± 7 Mm^{-1} in the East, and 17 ± 2.5 Mm^{-1} in the West. These equate to a naturally-occurring visual range in the East of 150 ± 45 km, and 230 ± 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. The primary non-anthropogenic substances responsible for natural levels of visibility impairment are naturally-occurring organics, suspended dust (including coarse particles), and water associated with hygroscopic particles. At the ranges of fine particle concentrations associated with natural background conditions noted above, small changes in fine particle mass have a large effect on total light extinction. Thus, higher levels of background fine particles and associated average humidity levels in the East result in a fairly significant difference between naturally occurring visual range in the rural East as compared to the rural 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 makes this comparison for several locations across the country by using background estimates from Table VIII-2 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 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. As discussed in the 1996 Staff Paper, 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 visibility in a more polluted atmosphere (EPA, 1996b, p. VIII-10, Figure VIII-9). 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 between changes in fine particle concentrations and changes in visibility (in deciviews) 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. 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 20 sites, but it has now been expanded to 110 sites that represent all but one (Bering Sea) 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 the rationale supporting use of the light extinction coefficient, derived from either direct optical measurements and/or 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 measurements at all sites, and optical and scene measurements at some of the sites. 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 typically 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. Directly measured light extinction is used under the IMPROVE protocol to cross-check that the aerosol-derived extinction levels are reasonable in establishing current visibility conditions. Aerosol-derived extinction is used to document spatial and temporal trends and to determine how proposed changes in atmospheric constituents would affect future visibility conditions.

5.2.3.2 Current Conditions and Trends 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 urban-influenced sites such as San Gorgonio Wilderness (CA) and Point Reyes National Seashore (CA), which have annual average levels comparable to certain sites in the Northeast. Regional differences are

illustrated by Figures 4-39a and 4-39b in the CD, which show that visibility levels on the 20% haziest days in the west (15 deciviews) are about equal to levels on the 20% best days in the east (CD, p. 4-181).

Higher visibility impairment levels in the East are due to generally higher concentrations of anthropogenic fine particles, particularly sulfates, and higher average relative humidity levels. In fact, sulfates account for 60-86% of the haziness in eastern sites (CD, 4-236). Aerosol light extinction due to sulfate on the 20% haziest days is significantly larger in eastern Class I areas as compared to western areas (CD, p. 4-183; Figures 4-40a and 4-40b). 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).

Regional trends in Class I area visibility are updated annually in the EPA's National Air Quality and Emissions Trends Report (EPA, 2001). Eastern trends for the 20% haziest days from 1992-1999 showed a 1.5 deciview improvement, or about 16%. However, visibility in the east remains significantly impaired, with an average visual range of 14 miles (22 km) on the 20% haziest days. In western Class I areas, aggregate trends showed little change during 1990-1999 for the 20% haziest days, and modest improvements on the 20% mid-range and clearest days. Average visual range on the 20% haziest days in western Class I areas is 80 km.

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 PM and visibility impairment levels than

monitored Class I areas. Urban area annual mean and 98th percentile 24-hour average $PM_{2.5}$ levels for 1999-2001 are presented in Chapter 2. These levels are generally higher than those found in the IMPROVE database for rural Class I areas. Urban areas have higher concentrations of organic carbon, elemental carbon, and particulate nitrate than rural areas due to a higher density of fuel combustion and diesel emissions.

5.2.4.1 ASOS Airport Visibility Monitoring Network

In 1992, the National Weather Service (NWS), Federal Aviation Administration (FAA), 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, p. 4-175). 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. A total of 569 FAA-sponsored and 313 NWS-sponsored automated observing systems are installed at airports throughout the country. ASOS visibility data are typically reported for aviation use in small increments up to a maximum of 10 miles visibility. While this truncated data is 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.

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

EPA obtained archived 1999 ASOS data for 63 cities across the country. The purpose of analyzing the ASOS data was to improve characterizations of current visibility conditions in non-Class I areas, particularly in urban areas, and to evaluate the relationship between ASOS visibility sensor data and $PM_{2.5}$ mass concentrations at ASOS sites.

EPA staff evaluated the 1-minute ASOS data and developed correlations between ASOS measurements and $PM_{2.5}$ concentrations, as well as relationships between quarterly correlation coefficient values and longitude of ASOS sites, monthly ASOS visibility patterns, and diurnal patterns by season, hour of day, and day of week (Szykman and Damberg, 2003). Figure 5-2 shows quarterly distributions of estimated correlation coefficients in 49 cities between ASOS extinction values and $PM_{2.5}$ concentrations at nearby Federal Reference Method sites that were operational in 1999 as a function of the longitude of ASOS sites. Correlations exceeded 0.70 in at least one quarter for 40 of the 49 cities. Correlations were more consistently high in the 3rd quarter, with correlations exceeding 0.70 in 29 (or 59%) of the cities.

Figures 5-3 and 5-4 provide two examples of relatively well-characterized relationships between predicted $PM_{2.5}$ concentrations (based on ASOS extinction values) and measured $PM_{2.5}$ concentrations in Washington, DC and Milwaukee, with correlation coefficients of 0.84 and 0.87, respectively. At least in some areas of the country, as seen in these example cities, the use of ASOS data to understand the linkage between $PM_{2.5}$ concentrations and urban visibility shows a significant amount of promise. EPA intends to conduct additional, more detailed analyses comparing ASOS measurements to $PM_{2.5}$ concentrations from the recently deployed urban speciation network, which may provide more consistently high correlations.

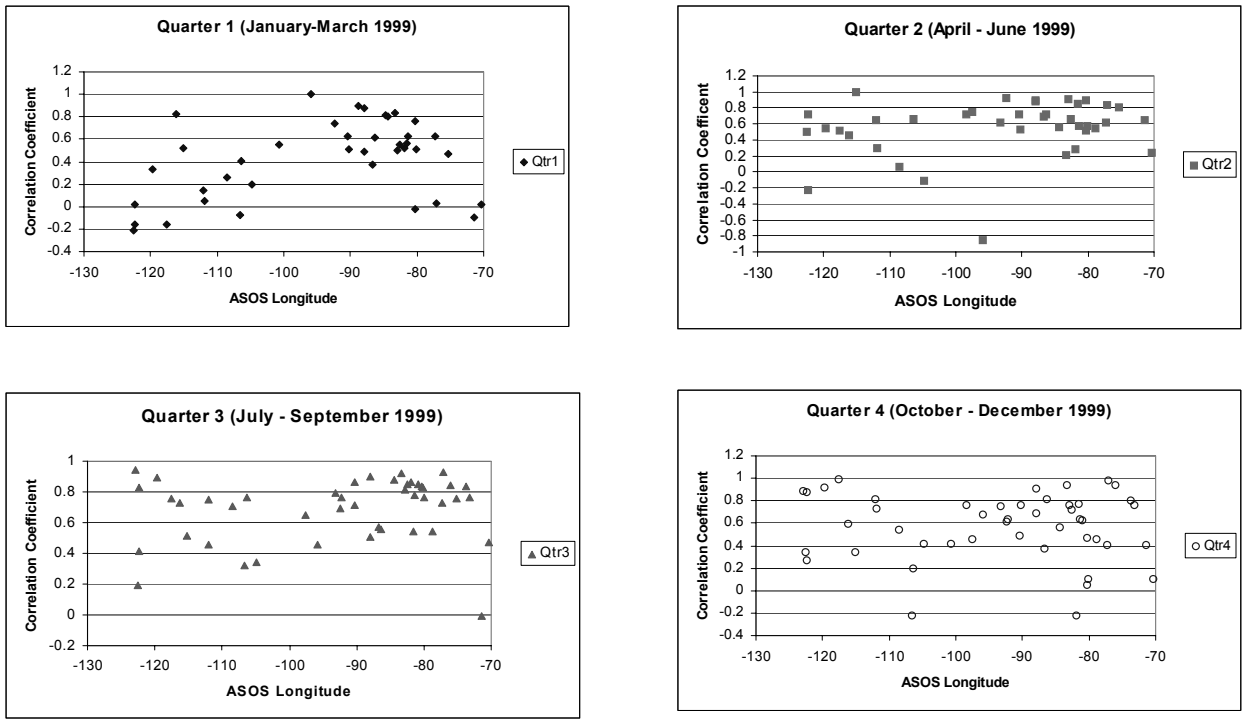

Figure 5-2. Relationship Between Quarterly Distributions of Estimated Correlation Coefficients and Longitude of ASOS Sites.

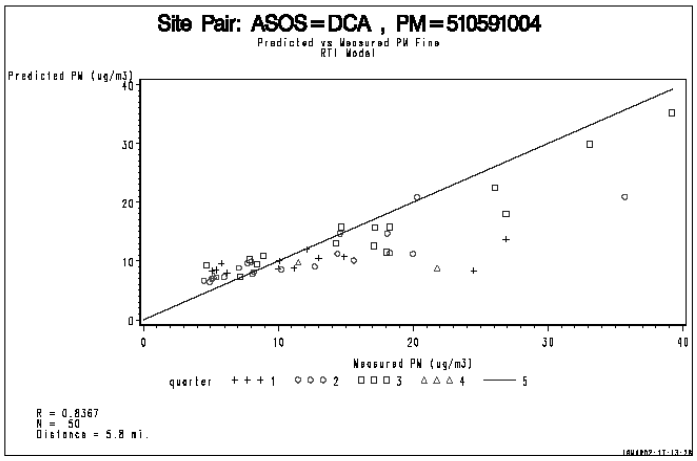

Figure 5-3. Relationship between Predicted and Measured $PM_{2.5}$ Concentrations in Washington, DC.
Diagonal line indicates the regression line.
Source: Szykman and Damberg, 2003.

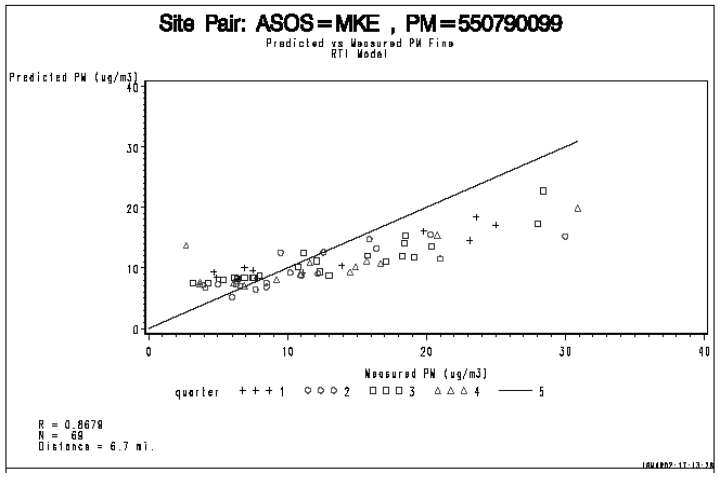

Figure 5-4. Relationship between Predicted and Measured $PM_{2.5}$ Concentrations in Milwaukee, WI.
Diagonal line indicates the regression line.
Source: Szykman and Damberg, 2003.

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 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 and Wakefield, 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 estimation of non-use values has proved to be significantly more difficult than the estimation 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 they 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 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 2003 draft Criteria Document (CD, p 4-187 to 4-191), 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, 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. 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 (CV). 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 estimating 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 CV 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, on May 15, 1998 in St. Louis, Missouri, it was reported that a haze episode attributed to wildfires in central America resulted in a reduction in landing rates and significant flight delays at Lambert International Airport. The 24-hour $PM_{2.5}$ levels reached 68 $\mu g/m^3$ during that episode. In addition, the NTSB has concluded in accident reports that high levels of pollution and haze, 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 as they are illustrative of the significant value that the public places on improving visibility, and because they have developed methods for evaluating public perceptions and judgments about the acceptability of varying degrees of visibility impairment. As discussed below, such methods could be applied to develop additional information that would help to inform EPA's review of the secondary PM NAAQS. Specific discussion is provided below on the Act's special emphasis on protection of visibility in certain areas, the Denver visibility standard, the Phoenix Brown Cloud Summit, and visibility protection efforts in the Canadian province of British Columbia.

In addition to the above visibility protection efforts, staff notes that the State of California (California Code of Regulations) and the Lake Tahoe Regional Planning Agency (Molenar, 2000) have also established visibility standards in the U.S. Internationally, the Australian state of Victoria has established a visibility objective (State Government of Victoria, 2000a and 2000b), and a visibility guideline is under consideration in New Zealand (New Zealand National Institute of Water & Atmospheric Research, 2000a and 2000b; New Zealand Ministry of Environment, 2000).

Sections 169A and 169B of the CAA. In section 302(h) of the Act, visibility impairment is identified as an effect on public welfare to be protected against by secondary NAAQS, among other basic programs under the Act. Additional protection against visibility impairment in special areas is provided for in sections 169A and 169B of the Act. Section 169A, added by 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. Action was deferred 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 GCVTC issued its recommendations to EPA in 1996, triggering a requirement in section 169B for EPA issuance of regional haze regulations.

EPA accordingly promulgated a final regional haze rule in 1999. 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. The Denver standard is violated when the four-hour average light extinction level exceeds 76 Mm^{-1} (equivalent to approximately 50 km 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. The staff note that in setting this standard, the State chose to use a fairly short averaging period (4 hours) in recognition of the fact that visibility conditions can change significantly over the course of the day. Of particular interest 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 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 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 CDPHE researchers sorted the ratings for each slide by increasing order of light extinction and calculated the percent of participants that judged each slide to violate the standard. 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. The CDPHE researchers found this level to be

reasonable because for the slides at this level and above, a majority of the study participants judged the light extinction levels to be unacceptable. In fact, when researchers evaluated all citizen judgments made on all slides at this level and above as a single group, more than 85% of the participants found visibility impairment at and above the level of the selected standard to be unacceptable.

Images are available on an EPA website (http://www.epa.gov/ttn/naaqs/standards/pm/s_pm_cr_sp.html, labeled as Figures 5-5 to 5-12) that illustrate 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 WinHaze, a state-of-the-art image modeling program developed by Air Resource Specialists, Inc. (ARS), discussed below in section 5.2.6.1.

Phoenix, Arizona - Governor's Brown Cloud Summit. On March 15, 2000, Governor Jane Dee Hull established an advisory group process called the Brown Cloud Summit, directing the Summit "to establish options for a visibility standard or other method to track progress in improving visibility in the Phoenix area." The Summit process was formed because of citizen and government concerns about the poor visibility conditions that had been prevalent in the Phoenix metropolitan area over the past several years, particularly in the winter months. The Summit issued its final report in January 2001 (Arizona Department of Environmental Quality, 2001). A key recommendation of the Summit was that citizen-defined visibility goals should be established, based on a citizen survey process, similar to the Denver approach. The final report also recommended a number of short- and long-term emissions reduction strategies to help improve visibility and public health.

In 2002, the Arizona Department of Environmental Quality acted on the recommendation of the Summit and formed the Visibility Index Oversight Committee. The Committee's goal was to coordinate the involvement of Phoenix-area residents in the development of a visibility index. The Phoenix committee patterned its survey process after the process developed by Denver officials to develop a visibility standard there.

The committee contracted with a consulting firm to design a survey instrument and administer the survey. A final report of the survey methods and results is now available (BBC

Research & Consulting, prepared for Arizona Department of Environmental Quality, 2003). The survey included 385 participants in 27 separate sessions. A sample size of 385 was carefully chosen so that responses would be representative of the area's population. Participants were carefully recruited to form a sample group that was demographically representative of the larger Phoenix population. Three sessions were held in Spanish.

Participants were shown a series of 25 images of the same vista of downtown Phoenix, with South Mountain in the background at a distance of about 40 km. Photographic slides of the images were developed using the WinHaze program. The visibility impairment levels ranged from 15 to 35 deciviews. Participants first rated the randomly-shown slides on a scale of 1 (unacceptable) to 7 (excellent). Next, the participants rated slides, again shown in random order, as acceptable or unacceptable. This phase of the survey produced the following findings:

- At least 90 percent of all participants found visible air quality acceptable between 15 deciviews (87 km visual range) and 20 deciviews (53 km);

- At 24 deciviews (36 km), nearly half of all participants thought the visible air quality was unacceptable; and

- By 26 deciviews (29 km), almost three-quarters of participants said it was unacceptable, with nearly all participants considering levels of 31 deciviews (18 km) and higher to be unacceptable.

The information developed in this survey informed the development of future recommendations by the Visibility Index Oversight Committee for a visibility index for the Phoenix Metropolitan Area. Based on the Committee's recommendations, the Arizona Department of Environmental Quality created the Blue Sky Index. Similar to the approach followed for the Denver standard, the Blue Sky Index focuses on a relatively short averaging time (6-hours). The index establishes a target for tracking the number of days each year (250 days in 2001, 260 in 2002, 275 in 2003) during which the 6-hour visual range is 40 km or more, as measured by a transmissometer. The Phoenix survey demonstrates that the rating methodology developed for gathering citizen input for establishing the Denver visibility standard can be reliably transferred to another city while relying on updated imaging technology to simulate a range of visibility impairment levels.

Images are available at the same EPA website (http://www.epa.gov/ttn/naaqs/standards/pm/s_pm_cr_sp.html, labeled as Figures 5-13 to 5-20) that illustrate visual air quality in Phoenix under a range of visibility conditions. These images were also generated using the WinHaze program. The modeled light extinction levels are based on correlations derived from 1994-1997 ambient air quality monitoring data.

Province of British Columbia, Canada. In 1993, the REVEAL (Regional Visibility Experimental Assessment in the Lower Fraser Valley) field study was undertaken to characterize summertime visibility and ambient aerosol loadings in southwestern British Columbia. In 1994, researchers at the University of British Columbia conducted a pilot study on the perception of acceptable visibility conditions in the area, using photographs and optical measurements taken during the summer of 1993 (Pryor, 1996). The study was based on the methodology used in setting the Denver visibility standard (Ely et al., 1991).

Participants in the study were shown slides of two suburban locations in British Columbia: Chilliwack and Abbotsford. After using the same general protocol, Pryor found that responses from this pilot study would indicate a standard in terms of visual range of approximately 40 km for Chilliwack and 60 km for Abbotsford. Pryor (1996) discusses some possible reasons for the variation in standard visibility judgments between the two locations. Factors discussed include the relative complexity of the scenes, different levels of development at each location, potential local source influence on site-specific nephelometer data, and potential bias of the sample population since only students participated. The author observed that the applicable 1995 health objective for PM_{10}, 50 $\mu g/m^3$ for a 24 hour period, would roughly correspond to a visual range of 26 km and therefore would not be protective of a visibility standard set in the range of 40-60 km, as suggested by the pilot study findings. The author expressed the view that the pilot study reinforced the conclusion that the methodology originally developed for the Denver standard-setting process is a sound and effective one for obtaining public participation in a standard-setting process, and that it could be adapted for such use in another geographic location with only minor modifications (Pryor, 1996).

5.2.6 Evaluating Public Perceptions of Visibility Impairment

New methods and tools 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. Survey methods are being refined, applied, and evaluated in various studies, such as those described above in section 5.2.5.2 for Denver, Phoenix, and British Columbia. These methods are intended to assess public perceptions as to the acceptability of varying levels of visual air quality, considered in these studies to be an appropriate basis for developing goals and standards for visibility protection. Staff finds that the survey methods used in the cases discussed above produced reasonably consistent results from location to location, each with a majority of participants finding visual ranges within about 40 to 60 km to be acceptable. As discussed below, new tools for the development of photographic representation of visibility impairment, such as the WinHaze software developed by ARS, are also now available that facilitate the use of such survey methods.

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, federal land management agencies, and 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. 4-174). In particular, Molenar (1994) describes a sophisticated visual air quality simulation technique, incorporated into the WinHaze program developed by ARS, which combined various modeling systems under development for the past 20 years. 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, 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. WinHaze, an image modeling program for personal computers that employs simplified algorithms based on the sophisticated modeling technique, is now available (Air Resource Specialists, 2003).

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 Future Assessment of Public Opinions on Air Pollution-Related Visibility Impairment

In the preliminary draft Staff Paper (EPA, 2001), information was presented on a small pilot project conducted by EPA that applied the survey and photographic techniques discussed above (Abt Associates, 2001). In conjunction with this pilot project, images that illustrate visual air quality in Washington, DC under a range of visibility conditions were prepared and are available at http://www.epa.gov/ttn/naaqs/standards/pm/s_pm_cr_sp.html (labeled as Figures 5-21 to 5-28). Two example images from the set of images found on the website are included below, showing that the U.S. Capitol building, located less than 5 miles from where the base photo was taken, becomes almost entirely obscured at a $PM_{2.5}$ concentration of 65 $\mu g/m^3$.

Figure 5-26. Washington, DC - 30 µg/m^3 $PM_{2.5}$ (16 km visual range)

Figure 5-28. Washington, DC - 65 µg/m^3 $PM_{2.5}$ (8 km visual range)

Information on the pilot project was presented in the preliminary draft Staff Paper to elicit CASAC and public comment on the use of this type of approach to help inform EPA's review of the secondary PM NAAQS, and, more specifically, to elicit comments on various aspects of the survey methodology used in the pilot project. The project was 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.

EPA received general support for the use of this type of approach, and also received advice from members of CASAC as to how the survey methodology could be improved. At that time, EPA staff expressed the intention of refining the approach based on that advice, and preparing a revised methodology document for additional review by CASAC and the public prior to conducting a more extensive survey that could appropriately inform this review. Although resource constraints have since prevented this work from being conducted, EPA hopes to pursue it in the future so the results of a more extensive survey can be used to help inform the next periodic review of the PM secondary standards.

5.2.7 Summary

The draft CD and other reports referenced in section 5.1 provide a significant body of information documenting the effects of PM and its components on atmospheric visibility. The

draft CD provides updated information on studies evaluating particle scattering efficiencies and the relative humidity effects primarily on sulfate and nitrate particle size. Visibility trends in national parks and wilderness areas are presented using data from the IMPROVE visibility monitoring network. While recent emphasis has been placed on characterizing rural visibility conditions for the purposes of implementing the regional haze rule, urban visibility conditions continue to be poorly characterized. In an effort to improve characterization of urban visibility, staff conducted an initial analysis of newly available high-resolution (1-minute) data from the ASOS monitoring network and comparison to nearby $PM_{2.5}$ concentrations, concluding that such analysis appears to be a promising approach. Staff anticipates that additional analyses comparing visibility data from ASOS to data from continuous PM monitors, as well as information from the $PM_{2.5}$ speciation monitoring network and from expanded use of continuous visibility monitoring in urban areas, will allow for better characterization of current conditions and temporal variations in urban visibility conditions across the country in the future.

Information is presented above on the efforts of various urban areas to develop local visibility standards based on a survey methodology that elicits citizen judgments about acceptable and unacceptable levels of visibility as portrayed through photographic slides. In the preliminary Staff Paper, staff discussed a pilot focus group project conducted by EPA in 2000 to evaluate the methodology used in establishing the Denver visibility standard in combination with the WinHaze photographic modeling technique. Similar approaches were used as part of studies to address visibility problems in Phoenix and the Frasier Valley of British Columbia. The common survey approach used in the Denver, Phoenix, and British Columbia studies yielded reasonably consistent results, with each study indicating that a majority of citizens find value in protecting local visibility to within a visual range of about 40 to 60 km. Staff finds this approach to evaluating citizen judgments about acceptable visibility to be promising, and hopes to continue to pursue this approach in the future.

Staff believes that currently available information provides a basis for considering whether any revisions to the $PM_{2.5}$ secondary standards may be appropriate for protecting against PM-related visibility effects. Staff recognizes that until additional monitoring, data analyses, and assessments of citizen judgments of acceptable urban visibility as discussed above can be

conducted, limitations in the available information significantly constrain development of a quantitative basis for a secondary standard(s) based solely on such information. However, staff believes it remains appropriate in this review to consider whether any revisions that may be made to the primary $PM_{2.5}$ standards are also appropriate for revising the $PM_{2.5}$ secondary standards to protect against PM-related effects on visibility. Staff also believes that the consideration of any revisions to the secondary $PM_{2.5}$ standards can be appropriately informed by comparing photographic representations of the degree of visibility impairment allowed under the current 24-hour standard with the degree of visibility impairment that is possible under any alternate 24-hour standard. Photographic representations of visibility conditions associated with a range of $PM_{2.5}$ concentrations are presented in Figures 5-5 through 5-28 for Denver, Phoenix, and Washington, DC. In addition, actual photographs of Chicago illustrating visibility conditions associated with a range of $PM_{2.5}$ concentrations are presented in Figures 5-29 through 5-34. All of these images are available on an EPA website at http://www.epa.gov/ttn/naaqs/standards/pm/s_pm_cr_sp.html.

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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. 4-191). 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 (CD, pp. 4-192 to 4-194).

The draft CD (p. 4-195, Table 4-18) summarizes the results of a number of studies investigating the roles of particles 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. 4-193). 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, or by serving as concentration sites for other pollutants. If sufficient damage to the paint occurs, pollutants may penetrate to the underlying surface. 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 with 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 reportedly been enhanced by exposure to SO_2 and humidity; several studies have suggested that this effect is caused by the reaction of SO_2 with extender pigments such as calcium carbonate and zinc oxide, although Miller et al. (1992) suggest that calcium carbonate acts to protect paint substrates (CD, p. 4-194).

With respect to damage to building stone, numerous studies discussed in the draft CD (pp. 4-197 to 4-202; Table 4-19) suggest that air pollutants, including sulfur-containing pollutants and wet or dry deposition of atmospheric particles and dry deposition of gypsum particles, 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 surface. 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 further deposition of airborne particles (CD, p. 4-197).

The rate of stone deterioration 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, carbonaceous particles (non-carbonate carbon) and particles containing metal oxides may help to promote the decay process (CD, p. 4-302).

5.3.2 Soiling Effects

Soiling affects the aesthetic appeal of painted 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. 4-221). 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; CD, pp. 4-202 to 4-204). Haynie and Lemmons (1990) and 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 penetrates 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 also available for the years 1980 to 1998. Based on the available data on pollutant levels and photographs, the authors concluded 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, pp. 4-203 to 4-204).

5.3.3 Summary

Damage to building materials results from natural weathering processes that are enhanced by exposure to airborne pollution, most notably sulfur-containing pollutants. Ambient PM has been associated with contributing to pollution-related damage to materials, and 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, to date, no quantitative relationships between particle characteristics (e.g., concentrations, particle size, and chemical composition) and the frequency of cleaning or repainting have been established.

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. The following sections discuss the components of PM most relevant for vegetation and ecosystems (5.4.1); the mechanisms by which vegetation or ecosystems come in contact with PM through the processes of deposition (5.4.2); the nature and extent of direct effects of PM to vegetation (5.4.3); the nature and extent of direct and indirect effects of PM on ecosystems (5.4.4); urban ecosystems (5.4.5); PM speciation data and monitoring networks

(5.4.6); and an overall summary of information (5.4.7). These discussions are based on information contained and referenced in Chapter 4 of the draft CD.

This review also introduces the Framework for Assessing and Reporting on Ecological Condition recommended by the Ecological Processes and Effects Committee (EPEC) of EPA's Science Advisory Board (hereafter EPEC Framework; SAB, 2002) and described in subsections 4.2.1 and 4.2.3 of the draft CD. Such a framework can provide a useful approach for organizing discussions of stressor effects on ecosystem components at successive levels of complexity, facilitating elucidation of the linkages between effects at different hierarchical scales and putting them in the broader context of ecological integrity. The vegetation and ecosystem effects of PM identified below encompass the full range, scales, and properties of biological organization listed under the Framework's Essential Ecological Attribute (EEA) "Biotic Condition" which includes effects at the organism, species/population, and community/ecosystem levels (CD, pp. 4-2 to 4-4; 4-55 to 4-60).

5.4.1 PM Characterization

As previously discussed, PM is not a single pollutant, but a heterogeneous mixture of particles differing in size, origin, and chemical composition. Fine PM, which is typically more diverse than coarse PM, is predominantly secondary in nature, having condensed from the vapor phase or been formed by chemical reaction from gaseous precursors in the atmosphere. Sulfur and nitrogen oxides are often oxidized to their respective acids and neutralized with ammonium cations as particulate salts. Fine PM may also contain VOCs, volatilized metals, and products of incomplete combustion. Coarse PM, by contrast, is predominantly primary in nature, having been emitted from area or point sources, and may contain iron, silica, aluminum, and base cations from soil, fragments of plants and insects, pollen, fungal spores, bacteria, and viruses, as well as automobile related debris (e.g., brake linings) (CD, pp. 4-6, 4-7).

Though the chemical constitution of individual particles can be strongly correlated with size, the relationship between particle size and particle composition can also be quite complex, making it difficult in most cases to use particle size as a surrogate for chemistry. For example, nitrogen exhibits a strongly bimodal size distribution, with the peak above 1μm attributed to

HNO_3 adsorption onto coarse alkaline particles, and the peak below 1 μm attributed to gas phase condensation of ammonia with either sulfuric or nitric acid. This heterogeneity of PM exists not only within individual particles or samples from individual sites, but to an even greater extent, between samples from different sites. At this point in time, this heterogeneity of PM across space and time has not been adequately characterized . Since vegetation and other ecosystem components are affected more by particulate chemistry than size fraction, exposure to a given mass concentration of airborne PM may lead to widely differing plant or ecosystem responses, depending on the particular mix of deposited particles. At this time, there has been relatively little research aimed at defining the effects of unspeciated PM mass on plants or ecosystems, though effects of some specific chemical components of PM have been described (CD, pp. 4-6 to 4-8).

5.4.2 PM Deposition

For PM to affect plants and ecosystems, it must first be removed from the atmosphere and deposited onto an ecological surface (e.g. plants, water, soils) through the processes of deposition. There are three major routes of deposition: (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 dry particles are deposited directly onto surfaces. Available evidence suggests that all modes of deposition must be considered in determining potential impacts to vegetation and ecosystems because each mode may dominate over specific intervals of time or space. Wet deposition is generally more effective for removing fine-mode PM from the atmosphere, whereas dry deposition is more effective for coarse-mode particles (CD, p. 4-9).

Several current national monitoring networks (discussed in more detail below in section 5.4.6) have measured both total wet and total dry deposition of a number of ambient air pollutants for the past several decades. However, though much is known about the total deposition of certain chemical species of airborne pollutants, the percentage of that total deposition attributed to particles is often not known or specifically measured.

All forms of particle deposition are affected by many factors (CD, p. 4-10, Table 4-2). For example, dry deposition is influenced by particle size, shape, chemical composition, macro- and micro-surface characteristics (e.g., canopy aerodynamics/roughness, leaf properties such as stickiness/wettability, shape, orientation), and atmospheric characteristics (e.g., wind speed, temperature, humidity) and stability. Wet deposition is influenced by some of the same factors as well as the timing, intensity and duration of the precipitation event, the temperature and humidity conditions following the event, and the amount of dry PM previously deposited on the surface that becomes soluble, potentially increasing foliar uptake, or washed off, transferring it to the soil or other media. The deposition of PM in fog droplets in occult deposition is directly proportional to wind speed, droplet size, concentration, and fog density (liquid water content per volume air). Occult deposition is particularly effective for delivery of dissolved and suspended materials to vegetation because: (1) concentrations of ions are often many-fold higher in clouds or fog than in precipitation or ambient air (e.g., acidic cloud water, which is typically 5-20 times more acid than rainwater, can increase pollutant deposition and exposure to vegetation and soils at high elevation sites by more than 50% of wet and dry deposition levels); (2) PM is delivered in a hydrated and bioavailable form to foliar surfaces and remains hydrated due to conditions of high relative humidity and low radiation; and (3) the mechanisms of sedimentation and impaction for submicron particles that would normally be low in ambient air are increased. High-elevation forests receive larger particulate deposition loadings than equivalent low-elevation sites, due to a number of orographic (mountain related) effects. These orographic effects include higher wind speeds that enhance the rate of aerosol impaction, enhanced rainfall intensity and composition, and increased duration of occult deposition. Additionally, the needle-shaped leaves of the coniferous species often found growing in these high elevation sites, enhance impaction and retention of PM delivered by all three deposition modes (CD, pp. 4-12 to 4-44).

Given all the factors enumerated above and in the CD, direct measurement of PM deposition to the environment can be extremely difficult, and available tabulations of PM deposition velocity (V_d) remain highly variable and suspect (CD, p. 4-9). Due to these complexities, numerous models have been developed to calculate V_d. However, modeling

particle deposition to vegetation is at a relatively early stage of development, and it is not possible to identify a best or most generally applicable modeling approach at this time (CD, p. 4-20). Further, a review by Wesely and Hicks (2000) concluded that a comprehensive understanding of particulate deposition remains a distant goal. It is clear that substantially improved techniques for monitoring and predicting particulate deposition will be required to characterize with some degree of certainty quantitative relationships between ambient PM concentrations, rates and quantities of PM deposition, and associated biological effects on plants and ecosystems (CD, pp. 4-39, 4-54).

5.4.3 Direct Effects on Vegetation

Plant effects can result from either the physical or the chemical properties of PM, or both, and may be caused directly by particle deposition onto the affected vegetation or indirectly through deposition to other media such as soils or water. 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. 4-62). The following discussion focuses on those particles that are intercepted by and remain on the leaves.

Physical effects of PM occur mainly in areas where deposition rates for particles in the coarse mode are high, such as near roadways, agricultural areas and industrial sites. High deposition rates have in some cases led to crust formation on plant leaves. Physical effects that have been observed in vegetation in such areas include reduced photosynthesis and subsequent reductions in carbohydrate formation, and 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, p. 4-63).

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. 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. To a lesser degree, the effects of trace metals and organics are also considered. 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 reached in the 1982 PM/SOx Air Quality Criteria Document (U.S. EPA, 1982), that sufficient data were not available for adequate quantification of dose-response functions, continues to be true today (CD, p. 4-68).

Effects of Nitrogen 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 (CD, p. 4-95). Particle deposition of nitrate, together with other nitrogen-containing gaseous and precipitation-derived sources, represents 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. Though plants usually absorb nitrogen (as NH_4^+ or NO_3^-) through their roots, it is known that foliar uptake of nitrate can also 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. 4-68, 4-71).

Effects of Sulfur Deposition. 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. 4-71, 4-72).

Effects of Acidic Deposition. Though dry deposition of nitrate and sulfate particles does not appear to induce foliar injury at current ambient exposures, when found in acidic precipitation, such particles 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 many 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. 4-72, 4-73). 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. 4-86). 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. Generally, only the heavy metals cadmium, chromium, nickel and mercury are released from stacks (CD, pp. 4-74, 4-75).

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). 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. Only a few studies have been conducted on the effects of trace metals on leaf surface organisms, specifically fungi, and these used soluble compounds containing heavy metals. Results indicated variations in fungal sensitivity and tolerance, with iron, aluminum, nickel, zinc, manganese and lead exhibiting the broadest spectrum of growth suppression. Though trace metals probably occur naturally on leaf surfaces as low-solubility compounds, given sufficient solubility and dose, changes in microbial community structure on leaf surfaces are possible. Because the fungi and bacteria living on and in the surfaces of leaves play an important role in the microbial succession that prepares leaves for decay and litter decomposition, such impacts could affect the larger ecosystem. Trace metal toxicity of lichens has also been demonstrated in a few cases (CD, pp. 4-75, to 4-77).

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. 4-76, 4-77).

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 them 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 is poorly characterized for most trace organics. Additionally, the toxicity of organic contaminants to plants and soil microorganisms is not well studied (CD, pp. 4-77 to 4-79).

5.4.4 Ecosystem Effects

As discussed in the draft CD, human existence on this planet depends on the life-support services ecosystems provide. Both ecosystem structure (biotic condition) and functions (ecological processes) play an essential role in providing ecosystem goods (products) and services. Ecosystem processes maintain clean water, clean air, a vegetated Earth, and a balance of organisms: the functions that enable humans to survive. The benefits they impart include absorption and breakdown of pollutants, cycling of nutrients, binding of soil, degradation of

organic waste, maintenance of a balance of gases in the air, regulation of radiation balance and climate, and the fixation of solar energy (CD, pp. 4-57, 4-58; Table 4-12).

Ecosystem-level responses related to PM occur when the effects of PM deposition on the biological and physical components of ecosystems become sufficiently widespread as to impact essential ecological attributes such as cycling of nutrients and materials or biodiversity. 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 chemical effects resulting from constituents of PM deposited directly onto vegetative surfaces or acting indirectly through deposition into soil and/or aquatic environments.

At an experimental site near limestone quarries and processing plants in southwestern Virginia, where PM dust accumulation occurred for at least 30 years, long-term changes in the structure and composition of the various strata within plant communities (e.g., herbs, seedlings, saplings, trees) were observed. Specifically, 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 chronic PM dust accumulation can impact ecosystems by favoring the growth of some species and limiting others (CD, pp. 4-83 to 4-85).

The most significant PM-related ecosystem-level effects are the result of long-term cumulative deposition of a given chemical species (e.g., nitrate) or mix (e.g., acidic deposition) that exceeds the natural buffering or storage capacity of the ecosystem and/or affects 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, pp. 4-90, 4-91). To understand these effects, long-term,

detailed ecosystem or site-specific data usually are required. The following discussion is organized according to the speciated effects of PM.

Nitrogen Deposition. In the natural environment, nitrogen may be divided into two groups: nonreactive, molecular (N_2) and reactive (Nr). Though N_2 is the most abundant element in the atmosphere, it only becomes available to support the growth of plants and microorganisms after it is converted into a reactive form. Reactive nitrogen includes inorganic reduced forms (e.g., ammonia [NH_3] and ammonium [NH_4^+]), inorganic oxidized forms (e.g., nitrogen oxides [NO_x], nitric acid [HNO_3], nitrous oxide [N_2O], and nitrate [NO_3^-]), and organic compounds (e.g., urea, amine, proteins, and nucleic acids) (CD, pp. 4-95, 4-96).

Evidence shows that Nr is now accumulating in the environment on all spatial scales – local, regional and global. The increase of global Nr is the result of three main causes: (1) widespread cultivation of legumes, rice and other crops that promote the conversion of N_2 to organic nitrogen through biological nitrogen fixation; (2) combustion of fossil fuels, which converts both atmospheric N_2 and fossil nitrogen to reactive NO_x; and (3) the Haber-Bosch process, which converts N_2 to Nr to sustain food production and some industrial activities. The deposition of nitrogen in the U.S. from human activity doubled between 1961 and 1997 due mainly to the use of inorganic nitrogen fertilizers and the emissions of NO_x from fossil fuel emissions with the largest increase occurring in the 1960s and 1970s (CD, pp. 4-96 to 4-98).

Despite the many beneficial effects of Nr on the health and welfare of humans (e.g., the synthetic fertilizers used in cultivation and the cultivation-induced bacterial nitrogen fertilization (BNF) sustain a large portion of the world's population), increased Nr in the global system also contributes to many contemporary environmental problems such as:

- production of tropospheric ozone and aerosols and associated environmental (and human health) problems;

- productivity increases in forests and grasslands, followed by decreases wherever atmospheric Nr deposition increases significantly and critical thresholds are exceeded; Nr additions probably also decrease biodiversity in many natural habitats;

- acidification and loss of biodiversity in lakes and streams in many regions of the world, in association with sulfur deposition;

- eutrophication, hypoxia, loss of biodiversity, and habitat degradation in coastal ecosystems, which is now considered the biggest pollution problem in coastal waters; and

- global climate change and stratospheric ozone depletion, both of which impact the health of ecosystems (and humans) (CD, pp. 4-96 to 4-98).

Unfortunately, large uncertainties regarding the rates of Nr accumulation in the various reservoirs (e.g., atmosphere, soil, water) limits our ability to determine the temporal and spatial distribution of environmental effects. These uncertainties are of great significance because Nr can alter a wide array of biogeochemical processes through exchanges among environmental reservoirs. As depicted in Figure 5-35, this sequence of nitrogen transfers, transformations, and environmental effects is referred to as the nitrogen cascade (CD, p. 4-96).

Nitrogen has long been recognized as the nutrient that is most often limiting (and therefore most important) for plant growth. In soils low in nitrogen, atmospherically deposited nitrogen can act as a fertilizer, though other secondary nutrients may then become limiting (e.g., phosphorus, calcium or magnesium). However, because not all plants are equally capable of utilizing extra nitrogen, some plants will gain a competitive advantage and will replace those that are better adapted to living in lower nitrogen environments, such as those growing in infertile soil, shaded understories, deserts, or tundra. Plants adapted to these low resource environments have been observed to have a slow growth rate, low photosynthetic rate, 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), and low soil microbial activity. The effect of additions of nitrates on plant community succession patterns and biodiversity, has been studied in several long-term nitrogen fertilization studies in both New England and Europe. These studies suggest that some forests receiving chronic inputs of nitrogen may decline in productivity and experience greater mortality. Specifically, the findings 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 (CD, pp. 4-95 to 4-105).

When additions of nitrogen above soil background levels exceed the capacity of plants and soil microorganisms to utilize and retain it, a condition known as “nitrogen saturation”

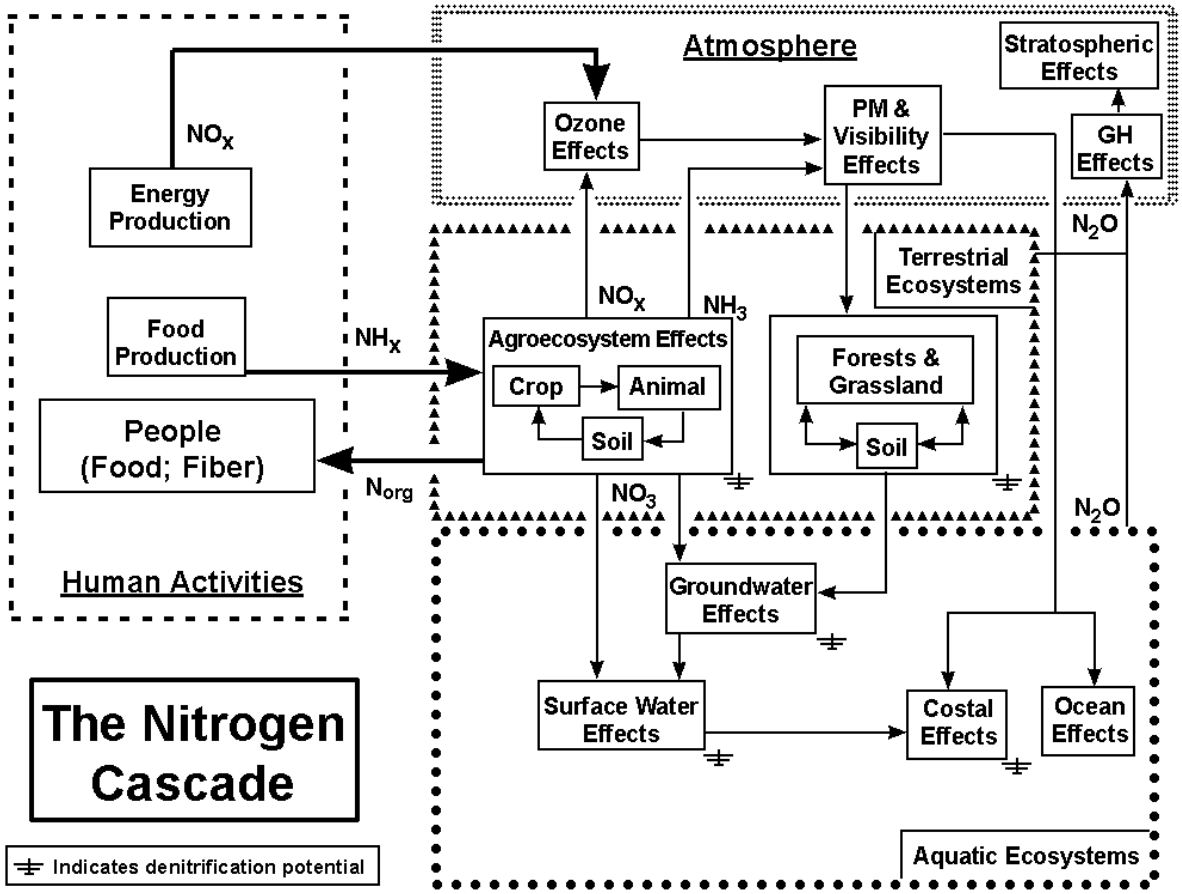

Figure 5-35 Illustration of the nitrogen cascade showing the movement of the human-produced reactive nitrogen (Nr) as it cycles through the various environmental reservoirs in the atmosphere, terrestrial and aquatic ecosystems (Figure 4-15, CD p. 4-97).

results. Specific ecosystem effects associated with nitrogen saturation include: (1) changes in 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. Additionally, studies suggest that during nitrogen saturation, soil microbial communities change from predominantly fungal (mycorrhizal) communities to those dominated by bacteria (Aber et al., 1998; CD, pp. 4-98, 4-99).

Some U.S. forests are now showing severe symptoms of nitrogen saturation, including the northern hardwoods and mixed conifer forests in the Adirondack and Catskill Mountains of New York; the red spruce forests at Whitetop Mountain, Virginia, and Great Smoky Mountains National Park, North Carolina; mixed hardwood watersheds at Fernow Experimental Forest in West Virginia; American beech forests in Great Smoky Mountains National Park, Tennessee; mixed conifer forests and chaparral watersheds in southern California and the southwestern Sierra Nevada in Central California; the alpine tundra/subalpine conifer forests of the Colorado Front Range; and red alder forests in the Cascade Mountains in Washington. All these systems have been exposed to highly elevated nitrogen deposition, and nitrogen saturated watersheds have been reported in the above-mentioned areas. Annual nitrogen additions through deposition in the southwestern Sierra Nevada are similar in magnitude to nitrogen storage in vegetation growth increments of western forests, suggesting that current nitrogen deposition rates may be near the assimilation capacity of the overstory vegetation. Ongoing urban expansion will increase the potential for nitrogen saturation of forests from urban sources (e.g., Salt Lake City, Seattle, Tucson, Denver, central and southern California) unless there are improved emission controls (Fenn et al., 1998).

Not all forest ecosystems react in the same manner to nitrogen deposition. The Harvard Forest in Massachusetts was the site for a nitrogen amendment study over 8 years (1988-96). During the study, nitrate leaching was observed in the pine stand after the first year in the high

nitrogen plots. Further increases in nitrate leaching were observed in the pine stand in 1995 and 1996, while the hardwood stand showed no significant increases in nitrate leaching until 1996. This sharp contrast in the responses of pine and hardwood stands indicates that the mosaic of community types across the landscape must be considered when determining regional scale response to nitrogen deposition (Magill, et al., 2002). Johnson et al. (1991) reported that measurements showing the leaching of nitrates and aluminum (Al^{+3}) from high elevation forests in the Great Smoky Mountains indicate that these forests have reached saturation (CD, p. 4-101).

Plant succession patterns and biodiversity are affected significantly by chronic nitrogen additions in some North American ecosystems. In one example, experimental studies of nitrogen deposition conducted over a 12-year period on Minnesota grasslands observed that plots originally dominated by native warm-season grasses shifted to low-diversity mixtures dominated by cool-season grasses at all but the lowest rates of nitrogen addition. Grasslands with high nitrogen retention and carbon storage rates were the most vulnerable to loss of species and major shifts in nitrogen cycling. The shift to low-diversity mixtures was associated with the decrease in biomass carbon-to-nitrogen (C:N) ratios, increased nitrogen mineralization, increased soil nitrate, high nitrogen losses, and low carbon storage (Weldin and Tilman, 1996). Naeem et al. (1994) experimentally demonstrated under controlled environmental conditions that the loss of biodiversity, genetic resources, productivity, ecosystem buffering against ecological perturbation, and loss of aesthetic and commercially valuable resources also may alter or impair ecosystems services (CD, p. 4-105).

The carbon-to-nitrogen (C:N) ratio of the forest floor can also be changed by nitrogen deposition over time. This change appears to occur when the ecosystem becomes nitrogen saturated. Long-term changes in C:N status have been documented in Central Europe and indicate that nitrogen deposition has changed the forest floor. In Europe, low C:N ratios coincide with high deposition regions. Therefore, to predict the rate of changes in nitrate leaching, it is necessary to be able to predict the rate of changes in the forest floor C:N ratio. Decreased foliar and soil nitrogen and soil C:N ratios, as well as changes in nitrogen mineralization rates, have been observed when comparing responses to nitrogen deposition in forest stands east and west of the Continental Divide in the Colorado Front Range.

Understanding the variability in forest ecosystem response to nitrogen input is essential in assessing pollution risks (CD, p. 4-107).

The mutualistic relationship between plant roots, fungi, and microbes is critical for the growth of the organisms involved, because the plant root zone (rhizosphere) is an important region of nutrient dynamics. The plant roots provide shelter and carbon for the symbiont, whereas the symbiont provides access to limiting nutrients such as nitrogen and phosphorus for the plant. Thus, as indicated above, changes in soil nitrogen that influence below-ground mycorrhizal fungal diversity also impact above-ground plant biodiversity, ecosystem variability, and productivity (CD, pp. 4-107, 4-108).

These types of effects have been observed in the field; for example, iIn studies of the coastal sage scrub (CSS) community in southern California, which is composed of the drought-deciduous shrubs *Artemisia californica*, *Encelia farinosa*, and *Eriogonum fasciculatum*. The CSS in California has been declining in land area and in shrub density over the past 60 years and is being replaced in many areas by Mediterranean annual grasses. At one time, nitrogen deposition was considered as a possible cause, since the deposition rate is high in the Los Angeles Air Basin (Bytnerowicz and Fenn, 1996). However, if increased nitrogen availability were the only variable between the invasive annuals and native shrubs, neither shrubs nor grasses would have a particular advantage. Later studies found that nitrogen enrichment of the soils also induced a shift in the arbuscular mycorrhizal community composition. Larger-spored fungal species (*Scutellospora* and *Gigaspora*), due to a failure to sporulate, decreased in number with a concomitant proliferation of small-spored species of *Glomus aggregatum*, G. *leptotichum*, and G. *geosporum*, indicating a strong selective pressure for the smaller spored species of fungi (Edgerton-Warburton and Allen, 2000). These results demonstrate that nitrogen enrichment of the soil significantly alters the arbuscular mycorrhizal species composition and richness, and markedly decreases the overall diversity of the arbuscular mycorrhizal community. The decline in coastal sage scrub species can, therefore, directly be linked to the decline of the arbuscular mycorrhizal community (Edgerton-Warburton and Allen, 2000) (CD, pp. 4-108, 4-109).

Excessive nitrogen inputs to terrestrial ecosystems also affect aquatic ecosystems. The primary pathways of nitrogen loss from terrestrial ecosystems are hydrological transport beyond

the rooting zone into groundwater or stream water, or surface flows of organic nitrogen as nitrate and nitrogen loss associated with soil erosion. Nitrogen saturation of a high elevation watershed in the southern Appalachian Mountains was observed to affect stream water chemistry. The Great Smoky Mountains National Park in Tennessee and North Carolina receives high total atmospheric deposition of sulfur and nitrogen. A major portion of the atmospheric loading is from dry and cloud deposition. Nitrogen saturation of the watershed resulted in extremely high exports of nitrate and promoted both chronic and episodic stream acidification in streams draining undisturbed watersheds. Significant export of base cations was also observed. Nitrification of the watershed soils resulted in elevations of soil solution aluminum concentrations to levels known to inhibit calcium uptake in red spruce (CD, p. 4-110).

There has been a 3- to 8-fold increase in nitrogen flux from 10 watersheds in the northeastern U. S. since the early 1900s. These increases are associated with nitrogen oxide emissions from combustion which have increased 5-fold. Riverine nitrogen fluxes have been correlated with atmospheric deposition onto their landscapes and also with nitrogen oxides emissions into their airsheds. Data from 10 benchmark watersheds with good historical records indicate that about 36-80% of the riverine total nitrogen export, with an average of 64%, was derived directly or indirectly from nitrogen oxide emissions (CD, pp. 4-110, 4-111).

Direct atmospheric nitrogen deposition and increased nitrogen inputs via runoff into streams, rivers, lakes, and oceans can noticeably affect aquatic ecosystems as well. Estuaries are among the most intensely fertilized ecosystems on Earth, receiving far greater nutrient inputs than other systems. In the Northeast, nitrogen is the element most responsible for eutrophication in coastal waters of the region. An illustrative example is recently reported research (Paerl et al., 2001) characterizing the effects of nitrogen deposition on the Pamlico Sound, NC, estuarine complex, which serves as a key fisheries nursery supporting an estimated 80% of commercial and recreational finfish and shellfish catches in the southeastern U.S. Atlantic coastal region. Such direct atmospheric nitrogen deposition onto waterways feeding into the Pamlico Sound or onto the Sound itself and indirect nitrogen inputs via runoff from upstream watersheds contribute to conditions of severe water oxygen depletion; formation of algae blooms in portions of the Pamlico Sound estuarine complex; altered fish distributions, catches, and physiological states;

and increases in the incidence of disease. Under extreme conditions (e.g., hurricanes) the effects of nitrogen runoff (in combination with excess loadings of metals or other nutrients) can be massive, such as the creation of the widespread "dead-zone" affecting large areas of the estuary (CD, pp. 4-109, 4-110).

The impact of increasing nitrogen inputs on the nitrogen cycle and forests, wetlands, and aquatic ecosystems is discussed in detail elsewhere (EPA, 1993, 1997; 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. 4-98).

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 onto 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. 4-112, 4-113).

Acidic Deposition. Acidic deposition over the past quarter of a century has emerged as a critical environmental stress that affects forested landscapes and aquatic ecosystems in North America, Europe, and Asia. Acidic deposition can originate from transboundary air pollution and affect large geographic areas. It is composed of ions, gases, particles derived from gaseous emissions of sulfur dioxide (SO_2), nitrogen oxides (NO_x), ammonia (NH_3), and particulate emissions of acidifying and neutralizing compounds, and is highly variable across space and time. It links air pollution to diverse terrestrial and aquatic ecosystems (CD, p. 4-114).

Acidic deposition has been firmly implicated as a causal factor in the northeastern high-elevation decline of red spruce (DeHayes et al., 1999). The frequency of freezing injury of red spruce has increased over the past 40 years, a period that coincides with increase emissions of sulfur and nitrogen oxides and increased acidic deposition. DeHayes et al. (1999) indicate that there is a significant positive association between cold tolerance and foliar calcium in trees exhibiting deficiency in foliar calcium, and further state that their studies raise the strong possibility that acid rain alteration of foliar calcium is not unique to red spruce but has been

demonstrated in many other northern temperate forest tree species including yellow birch (*Betula alleghaniensis*), white spruce (*Picea glaucus*), red maple (*Acer rubrum*) eastern white pine (Pinus strobus), and sugar maple (*Acer saccharum*) (CD, pp. 4-120, 4-121).

Although forest ecosystems other than the high-elevation spruce-fir forests are not currently manifesting symptoms of injury directly attributable to acid deposition, less sensitive forests throughout the U.S. are experiencing gradual losses of base cation nutrients, which in many cases will reduce the quality of forest nutrition over the long term (National Science and Technology Council, 1998). In some cases, such effects may not even take decades to occur because these forests have already been receiving sulfur and nitrogen deposition for many years. Soil acidification and its effects result from the deposition of nitrate, sulfate and the associated hydrogen ion. The introduction of the hydrogen ion (H^+) will directly impact the fluxes of base cations such as Ca, K, and Mg via cation exchange or weathering processes and influence the availability of other elements (e.g., aluminum and mercury). Soil leaching is often of major importance in cation cycles, and many forest ecosystems show a net loss of base cations (CD, pp. 4-115, 4-117).

In aluminum-rich soils, acid deposition, by lowering the pH, can increase Al concentrations in soil water through dissolution and ion-exchange processes. There is abundant evidence that Al is toxic to plants, and it is believed that the toxic effect of Al on forest trees could be due to its interference with Ca uptake. Once it enters forest tree roots, Al accumulates in root tissue. Because Ca plays a major role in cell membrane integrity and cell wall structure, reductions in Ca uptake suppress cambial growth, reduce the rate of wood formation, decrease the amount of functional sapwood and live crown, and predispose 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. 4-119, 4-120 and 4-126).

The Integrated Forest Study (IFS) (Johnson and Lindberg, 1992) has characterized the complexity and variability of ecosystem responses 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. For example, at the IFS sites on average, particulate deposition contributes 47% to total Ca deposition (range: 4 to 88%), 49% of total K deposition (range: 7 to 77%), 41% to total Mg deposition (range: 20 to 88%), 36% to total sodium deposition (range: 11 to 63%), and 43% to total base cation deposition (range: 16 to 62%). Of the total particulate deposition, the vast majority (> 90%) is > 2 μm. Given the wide ranges of particulate deposition for each base cation across the IFS sites, however, the unique characteristics of various sites need to be better understood before assumptions are made about the role particulate pollution plays in ecosystem impacts, though it is clear that particulate deposition contributes considerably to the total impact of base cations to most of the IFS sites (CD, pp. 4-127, 4-128).

These inputs of base cations have considerable significance, not only for base cation status, but also for the potential of incoming precipitation to acidify or alkalize the soils in the IFS ecosystems. The actual rates, directions, and magnitudes of changes that may occur in soils (if any), however, will depend on rates of inputs from weathering and vegetation outputs, as well as deposition and leaching. In other words, these net losses or gains of base cations must be placed in the context of the existing soil pool size of exchangeable base cations. For example, some 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. The IFS project further 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^+ (CD, pp. 4-132 to 4-137).

The data collected in the IFS give some idea of the importance of particulate deposition in these forest ecosystems, but they cannot account for the numerous potential feedbacks between vegetation and soils, nor for the dynamics through time that can influence the ultimate

response. One way to examine some of these interactions and dynamics is to use simulation modeling. The nutrient cycling model (NuCM) has been developed specifically for this purpose and has been used to explore the effects of atmospheric deposition, fertilization, and harvesting on some of the IFS sites (Johnson et al., 1993). The NuCM model is a stand-level model that incorporates all major nutrient cycling processes (uptake, translocation, leaching, weathering, organic matter decay, and accumulation) (CD, p. 4-137). Johnson et al. (1999) used the NuCM model to simulate the effects of reduced S, N, and base cation (C_B) deposition on nutrient pools, fluxes, soil, and soil solution chemistry in two contrasting southern Appalachian forest ecosystems: the extremely acidic red spruce sites and the less acidic Coweeta hardwood site from the IFS project. In summary, the authors found that in an extremely acidic system, C_B deposition can have a major effect on C_B leaching through time and S and N deposition had a major effect on Al leaching. At the less acidic Coweeta site, C_B deposition had a minor effect on soils and soil solutions; whereas S and N deposition had delayed but major effects on C_B leaching (CD, pp. 4-137, 4-142).

Seasonal and episodic acidification of surface waters have been observed in the eastern U. S., Canada and Europe. In the Northeast, the Shenandoah National Park in Virginia, and the Great Smoky Mountains, episodic acidification has been associated with the nitrate ion. The acidification of aquatic ecosystems and effects on aquatic biota are discussed in more detail in the EPA document *Air Quality Criteria for Nitrogen Oxides* (EPA, 1993) (CD, p. 4-121). Further, an extensive discussion of the various effects of acidic deposition is presented in the U.S. National Acid Precipitation Assessment Program (NAPAP) Biennial Reports to Congress (National Science and Technology Council, 1998).

Given the significant reductions in sulfur emissions that have occurred in the U. S. and Europe in recent decades, the findings of Driscoll et al. (1989, 2001) and Hedin et al. (1994) are especially relevant. Driscoll et al. (1989, 2001) noted a decline in both SO_4^{-2} and base cations in both atmospheric deposition and stream water over the past two decades at Hubbard Brook Watershed, NH. However, the reductions in SO_2 emissions in Europe and North America in recent years have not been accompanied by equivalent declines in net acidity related to sulfate in precipitation, and may have, to varying degrees, been offset by steep declines in atmospheric

base cation concentrations over the past 10 to 20 years (Hedin et al., 1994). Analysis of the data from the IFS supports the authors' contention that atmospheric base cation inputs may seriously affect ecosystem processes (CD, p. 4-122).

Critical Loads. The critical load has been defined as a "quantitative estimate of an exposure to one or more pollutants below which significant harmful effects on specified sensitive elements of the environment do not occur according to present knowledge" (Lokke et al., 1996). The critical loads framework originated in Europe where the concept has generally been accepted as the basis for abatement strategies to reduce or prevent injury to the functioning and vitality of forest ecosystems caused by long-range transboundary chronic acidic deposition. The concept is useful for estimating the amounts of pollutants that sensitive ecosystems can absorb on a sustained basis without experiencing measurable degradation. The estimation of ecosystem critical loads requires an understanding of how an ecosystem will respond to different loading rates in the long term and is a direct function of the level of sensitivity of the ecosystem to the pollutant and its capability to ameliorate change. In Europe, the elements used in the critical load concept are a biological indicator, a chemical criterion, and a critical value (CD, p. 4-124).

In the U.S. in 1989, a program was designed to develop and evaluate a framework for setting critical loads of nitrogen and sulfur. The flexible six-step approach includes: (1) selection of ecosystem components, indicators, and characterization of the resource; (2) definition of the functional subregions; (3) characterization of deposition within each of the subregions; (4) definition of an assessment endpoint; (5) selection and application of models; and (6) mapping projected ecosystem responses. This approach allows for consideration of variability in ecosystem characteristics and data availability (Strickland et al., 1993; CD, p. 4-124).

In the first step, ecological endpoints or indicators that are measurable characteristics related to the structure, composition, or functioning of ecological systems (i.e., indicators of condition) must be selected. A number of different indicators for monitoring ecosystem status have been proposed. Biogeochemicals (e.g., foliar nitrogen; nutrient ratios (N:P, N:cation); foliar nitrate; foliar δ^{15} N; arginine concentration; soil C:N ratio; NO_3^- in soil extracts or in soil solution; and flux rates of nitrogenous trace gasses from soil) have been discussed in Fenn et al.,

1998. Alternatively, a widely recognized indicator of nitrogen saturation in all ecosystem types, including California forests and chaparral, is increased and prolonged NO_3^- loss below the main rooting zone and in stream water. Seasonal patterns of stream water nitrate concentrations are especially good indicators of watershed nitrogen status. Biological indicators that have been suggested for use in the critical load calculation include mycorrhizal fungi (Lokke et al. 1996) and fine roots, since they are an extremely dynamic component of below-ground ecosystems and can respond rapidly to stress. The physiology of carbon allocation has also been suggested as an indicator of anthropogenic stress (Andersen and Rygiewicz, 1991). It should be kept in mind, however, that the response of a biological indicator is an integration of a number of different stresses. Furthermore, there may be organisms more sensitive to the pollutant(s) than the species selected (Lokke et al., 1996; National Science and Technology Council, 1998) (CD, pp. 4-124 to 126).

Assessment endpoints, on the other hand, selected in step (4) above, represent a formal expression of the environmental value that is to be protected. If the assessment endpoint is to be used as a regulatory limit, it should be socially relevant and the time scales of response appropriate. With regard to the latter, for example, surface water acidification associated with nitrate leaching should respond to decreases in nitrogen loading in a short period of time. However, changes in growth responses of vegetation resulting from soil nutrient imbalances may require years or decades to detect (CD, pp. 4-124, 4-125).

The critical loads concept has significant potential for identifying the level of protection needed to achieve ecosystem sustainability for any specific, well-defined ecosystem over the long-term. However, it is a very data-intensive approach, and, at the present time, there is insufficient data for the vast majority of U.S. ecosystems to allow for practical application of this approach as a basis for developing national standards to protect sensitive U.S. ecosystems from adverse effects related to PM deposition.

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 (CD, p. 4-77). 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. Only toxicity from copper, nickel, and zinc
has been frequently documented, though toxicity from cadmium, cobalt and lead compounds has been seen under unusual situations. Since these metals can all be toxic to roots and soil organisms, they change the litter decomposition processes that influence the availability of essential soil nutrients, ultimately interfering with ecosystem nutrient cycling. Therefore, any heavy metal 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 also 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, it has been suggested that the effect of heavy metals on mycorrhizae fungi can vary from host to host, and in some cases, symbiotic associations of mycorrhizal fungi with certain plants provide some additional degree of tolerance to metals (CD, pp. 4-145 to 4-147).

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 metals in earthworms suggested that cadmium and zinc were concentrated, but not lead. Thus, organisms that feed on earthworms from soils with elevated concentrations of certain metals (e.g. zinc) for extended periods would be expected to accumulate these metals to toxic levels. Increased concentrations of heavy metals have been found in a variety of small mammals living in areas with heavy metal concentrations in the soils. Biological accumulation of metals through the plant-herbivore and litter-detritivore chains also can occur. When soils are acidic, earthworm abundance decreases and bioaccumulation of metals from the soil may increase exponentially with decreasing pH (CD, pp. 4-146, 4-147).

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 (e.g., polychlorinated dibenzo-p-dioxins and dibenzofurans [PCDD/F]). 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 in plants, biodegradation probably does not occur readily since these compounds are found primarily in the lipophilic cuticle and are very resistant to microbial degradation. Therefore, the grass-cattle-milk/beef pathway can be 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 for beef and dairy cattle as well as chickens. Likewise in natural ecosystems, these chemicals tend to bioaccumulate up the food chain. For example, PCDD/Fs have been found in arctic seals and polar bears (CD, pp. 4-87 to 4-89).

Section 112 of the CAA provides the legislative basis for U.S. hazardous air pollutant (HAP) programs. In response to mounting evidence that air pollution contributes to water pollution, Congress included Section 112m (*Atmospheric Deposition to Great Lakes and Coastal Waters*) in the 1990 CAA Amendments that direct the EPA to establish a research program on atmospheric deposition of HAPS to the "Great Waters." Actions taken by EPA 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. Details concerning these effects may be found in "Deposition of Air Pollutants to the Great Waters," Third Report to Congress (EPA, 2000). The Third Report (EPA-453/R-00-005, June 2000), like the First and Second Reports to Congress, focuses on 15 pollutants of concern, including pesticides, metal compounds, chlorinated organic compounds, and nitrogen compounds. The new scientific information in the Third Report supports and builds on three broad conclusions presented in the previous two EPA Reports to Congress:

(1) Atmospheric deposition from human activities can be a significant contributor of toxic chemicals and nitrogen compounds to the Great Waters. The relative importance of atmospheric loading for a particular chemical in a water body depends on many factors (e.g., characteristics

of the water body, properties of the chemical, and the kind and amount of atmospheric deposition versus water discharges).

(2) A plausible link exists between emissions of toxic pollutants of concern into the air above the Great Waters; the deposition of these pollutants (and their transformation products); and the concentrations of these pollutants found in the water, sediments, and biota, especially fish and shellfish. For mercury, fate and transport modeling and exposure assessments predict that the anthropogenic contribution to the total amount of methylmercury in fish is, in part, the result of anthropogenic mercury releases from industrial and combustion sources increasing mercury body burdens (i.e., concentrations) in fish. Also, the consumption of fish is the dominant pathway of exposure to methylmercury for fish-consuming humans and wildlife. However, what is known about each stage of this process varies with each pollutant (for instance, the chemical species of the emissions and its transformation in the atmosphere).

(3) Airborne emissions from local as well as distant sources, from both within and outside the United States, contribute pollutant loadings to waters through atmospheric deposition. Determining the relative roles of particular sources — local, regional, national, and possibly global, as well as anthropogenic, natural, and re-emission of pollutants — contributing to specific water bodies is complex, requiring careful monitoring, atmospheric modeling, and other analytical techniques (CD, pp. 4-89, 4-90).

5.4.5 Urban Ecosystems

Though humans now clearly influence virtually all ecosystems on the planet, nowhere has human activity been more intense than in cities, suburbs, and surrounding areas. Although the study of ecological phenomena in urban environments is not new, the concept of a city as an ecosystem is relatively new for the field of ecology. Urban ecology implicitly recognizes the role humans play in developing unique ecosystems. This new, integrative ecology, which now explicitly incorporates human decisions, culture, institutions, and economic systems, along with primary production, species richness, biogeophysical budgets, ecosystem patterns and processes, and exchanges of materials and influence between cities and surrounding landscapes, is what is

needed if urban ecosystems, and ecosystems in general, are to be understood (CD, pp. 4-149, 4-150).

Though there is a large body of knowledge on concentrations and chemical reactions of air pollutants in cities, there has been little work done on the rates of atmospheric deposition, and especially particle deposition and effects, within urban ecosystems. Existing research indicates that cities are often sources of nitrogen oxides, sulfur oxides, and dust, among many other pollutants. Since some of these pollutants are major plant nutrients, they may be affecting nutrient cycles in plant dominated areas in and around cities. Gases and particles in urban air can increase atmospheric deposition within and downwind of cities. Lovett et al. (2000) observed that concentrations and fluxes of NO_3^-, NH_4^+, Ca^{+2}, Mg^{+2}, SO_4^{-2}, and Cl^- in oak forest throughfall all declined significantly with distance from New York City, while hydrogen ion concentration and flux increased significantly with distance from the city. McDonnell et al. (1997) found that levels of heavy metals in the foliar litter in urban forest soils were higher than those in rural areas, with urban levels approaching or exceeding levels reported to affect soil invertebrates, macrofungi, and soil microbial processes. The urban forests exhibited reduced fungal biomass and arthropod densities when compared to rural stands. In addition to heavy metal stress, urban forests also experience other anthropogenic stress such as poor air quality and low water availability caused by hydrophobic soils. The potential affect of these conditions on ecosystem processes of decomposition and nitrogen cycling appear to be ameliorated by two other anthropogenic factors: increased average temperatures caused by the heat island effect and the introduction and successful colonization of earthworms in the urban forests (McDonnell et al., 1997). Another study (Pouyat and McDonnell, 1991) that looked at heavy metal accumulations in forest soils in southeastern New York found variations in amounts of Zn, Cu, Ni, and Cd that corresponded closely with the urban-rural land use gradient, with pollutants highest near the urban end of the gradient (CD, pp. 4-150 to 4-152).

Though the role of particulate deposition in urban areas has not been explicitly studied, it appears likely to be a significant contributor to the ecosystem effects that have been observed.

5.4.6 Rural PM Air Quality Networks

Atmospheric concentrations of dry particles began to be routinely measured in 1986, with the establishment of EPA's National Dry Deposition Network (NDDN). After new monitoring requirements were added in the 1990 Clean Air Act Amendments, EPA, in cooperation with the National Oceanic and Atmospheric Association, created the Clean Air Status and Trends Network (CASTNet) from the NDDN. CASTNet comprises 85 sites and is considered the nation's primary source for atmospheric data to estimate concentrations for ground-level ozone and the chemical species that make up the dry deposition component of total acid deposition (e.g., sulfate, nitrate, ammonium, sulfur dioxide, and nitric acid), as well as the associated meteorology and site characteristics data that are needed to model dry deposition velocities (CD, pg. 4-21; www.epa.gov/castnet/overview/html).

To provide data on wet deposition levels in the U.S., the National Atmospheric Deposition Program (NADP) was initiated in the late 1970's as a cooperative program between federal, state, and other public and private groups. By the mid-1980's, it had grown to nearly 200 sites and it stands today as the longest running national atmospheric deposition monitoring network (www.epa.gov/castnet/overview/html).

In addition to these deposition monitoring networks, other networks collect data on ambient aerosol concentrations and chemical composition. Such networks include the IMPROVE network, discussed above in sections 2.5 and 5.2.3, and the newly implemented $PM_{2.5}$ chemical Speciation Trends Network (STN) that consists of 54 core National Ambient Monitoring Stations and approximately 250 State and Local Air Monitoring Stations (CD, p. 2-82).

In the future, data from these networks, combined with data from the deposition monitoring networks may allow better understanding of the components of PM that are most strongly influencing PM-related ecological effects.

5.4.7 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 the cascade of Nr in

terrestrial and aquatic ecosystems and those related to nitrate and acidic deposition. Among the observed effects of chronic nitrate deposition and its accumulation in ecosystems are nitrogen saturation, altering of ecosystem structure (i.e., biodiversity), and the functioning of ecosystem processes (e.g., nitrogen cycling). Though nitrogen deposited to ecosystems has not in general been speciated and measured, there is abundant evidence that nitrogen emissions from fossil-fuel burning deposited as nitrate over time has, as cited in the CD and summarized above, affected the functioning of ecosystems. At this time, however, there remains a paucity of widespread, long-term speciated rural PM data (needed for understanding ecosystem level effects); a lack of studies relating unspeciated ambient PM mass/size fraction exposures to vegetation or ecosystem effects; major complexities and regional and meteorological variability associated with the atmospheric deposition processes;and a lack of detailed location-specific environmental data that would be needed to determine whether a given amount of PM deposition occurring in a given location represents a beneficial or an adverse effect. As a result, currently available information does not yet provide a basis for quantitative characterization of the complex relationships between observed PM-related adverse effects on vegetation and ecosystems in various locations across the U.S. and levels of PM in the ambient air. Thus, while evidence of PM-related effects clearly exists, there is insufficient information available at this time to serve as a basis for a secondary national air quality standard for PM, specifically selected to protect against adverse effects on vegetation and ecosystems.

5.5 EFFECTS ON CLIMATE CHANGE AND SOLAR RADIATION

Atmospheric particles alter the amount of solar radiation transmitted through the earth's atmosphere by both scattering and absorbing radiation. More specifically, most components of ambient PM (especially sulfates) scatter and reflect incoming solar radiation back into space, thus offsetting the "greenhouse effect" to some degree by having a cooling effect on climate. In contrast, some components of ambient PM (especially black carbon) also absorb incoming solar radiation and are believed to contribute to some degree to atmospheric warming. Lesser impacts of atmospheric particles are associated with their role in altering the amount of ultraviolet solar radiation (especially UV-B) penetrating through the earth's atmosphere to ground level, where it

can exert a variety of effects on human health, plant and animal biota, and other environmental components (CD, p. 205). The extensive research and assessment efforts into global climate change and stratospheric ozone depletion provide evidence that atmospheric particles play important roles in these two types of atmospheric processes, not only on a global scale, but also on regional and local scales as well.

Information on the role of atmospheric particles in these atmospheric processes is briefly summarized above in Chapter 2 (section 2.9). The effects on human health and the environment associated with these atmospheric processes are briefly summarized below, based on the information in section 4.5 of the draft CD and referenced reports. These effects are discussed below in conjunction with consideration of the potential indirect impacts on human health and the environment that may be a consequence of climatic and radiative changes attributable to local and regional changes in ambient PM.

5.5.1 Climate Change and Potential Human Health and Environmental Impacts

Potential effects of global warming and climate change on both the environment and human health in the U.S. are discussed in section 4.5.1.1 in the draft CD and related references. The draft CD (p. 4-209) notes that while current climate models are successful in simulating present annual mean climate and the seasonal cycle on continental scales, they are lass successful at regional scales. Findings from various referenced assessments illustrate well the considerable uncertainties and difficulties in projecting likely climate change impacts on regional or local scales. For example, uncertainties in calculating the direct radiative effects of atmospheric particles arise from a lack of knowledge of their vertical and horizontal variability, their size distribution, chemical composition, and the distribution of components within individual particles. Any complete assessment of the radiative effects of PM would require computationally intensive calculations that incorporate the spatial and temporal behavior of particles of varying composition that have been emitted from, or formed by precursors emitted from, different sources. In addition, the draft CD (p. 219) notes that calculations of indirect physical effects of particles on climate (e.g., related to alteration of cloud properties and disruption of hydrological cycles, as discussed above in section 2.9) are subject to much larger

uncertainties than those related to the direct radiative effects of particles. The draft CD concludes that at the present time impacts on human health and the environment due to aerosol effects on the climate system can not be calculated with confidence, and notes that the uncertainties associated with such aerosol-related effects will likely remain much larger than those associated with greenhouse gases (CD, p. 4-219). Nevertheless, the draft CD concludes that substantial qualitative information available from observational and modeling studies indicates that different types of atmospheric aerosols (i.e., different components of PM) have both warming and cooling effects on climate, both globally and regionally. Studies also suggest that global and regional climate changes could potentially have both positive and negative effects on human health, human welfare, and the environment.

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

This section briefly summarizes information in section 4.5.2 of the draft CD 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 in UNEP (1998, 2000) and WMO (1999).

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. 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 ground-level 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. (CD, p. 4-221, 4-222).

In contrast to these types of negative impacts associated with increased UV-B penetration to the Earth's surface, the draft CD (p. 4-222, 4-223) summarizes research results that are suggestive of possible beneficial effects of increased UV-B radiation penetration. For example, a number of studies have focused on the protective effects of UV-B radiation with regard to non-skin cancer incidence, which proved suggestive evidence that UV-B radiation, acting through the production of vitamin D, may be a risk-reduction factor for mortality due to several types of cancer, including cancer of the breast, colon, ovary, and prostate, as well as non-Hodgkin lymphoma.

The various assessments of these types of 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 draft 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 the abundance of ozone and other absorbers within the planetary boundary layer and the free troposphere (CD, page 4-227).

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 draft 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. 4-227). Beyond considering just average levels of exposures to UV-B radiation in general, the draft 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 damaging UV-B radiation. Further, the draft 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. (CD, p. 4-227).

5.5.3 Summary

A number of assessments of the factors affecting global warming and climate change as well as those affecting the penetration of solar UV-B radiation to the earth's surface 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 climate or UV-B radiation penetration – 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 climate and UV-B radiation.

Based on currently available information, the potential indirect effects of ambient PM on public health and welfare, secondary to potential PM-related changes in climate and UV-B radiation, 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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6. STAFF CONCLUSIONS AND RECOMMENDATIONS ON PM NAAQS

6.1 INTRODUCTION

This chapter presents preliminary staff conclusions and recommendations for the Administrator to consider in deciding whether the existing PM standards should be revised and, if so, what revised or new standards are appropriate.[1] Drawing from the synthesis of information and analyses contained in both the draft CD and in Chapters 2 through 5 herein, this chapter begins with staff consideration of the overall adequacy of the current primary standards for PM. Subsequent sections address each of the major components needed to define specific standards: pollutant indicator (for both fine- and coarse-fraction particles), averaging time, form and level. Staff conclusions and recommendations for ranges of alternative primary standards are then presented, based on considering how both the major components of an individual standard and a suite of standards operate together to protect public health with an adequate margin of safety. A discussion of secondary PM standards follows, with staff conclusions and recommendations on alternative secondary standards for both fine- and coarse-fraction particles. This chapter concludes with a summary of key uncertainties associated with establishing PM standards and related staff research recommendations.

In recommending a range of primary standard options for the Administrator to consider, the staff notes that the final decision is largely a public health policy judgment. A final decision must draw upon scientific information and analyses about health effects and risks, as well as judgments about how to deal with the range of uncertainties that are inherent in the scientific evidence and analyses. The staff's approach to informing these judgments is based on a recognition that the available health effects evidence generally reflects a continuum consisting of levels at which scientists generally agree that health effects are likely through lower levels at which the likelihood and magnitude of the response become increasingly uncertain. This approach is consistent with the requirements of the NAAQS provisions of the Act and with how EPA and the courts have historically interpreted the Act. These provisions require the

[1] As noted in Chapter 1, staff conclusions and recommendations presented in the next draft Staff Paper will be further informed by consideration of the information and analyses in the final CD, additional staff analyses and the results of the completed human health risk assessment, and CASAC and public comments received on this draft.

Administrator to establish primary standards that are requisite to protect public health with an adequate margin of safety. In so doing, the Administrator seeks to establish standards that are neither more nor less stringent than necessary for this purpose. The provisions do not require that primary standards be set at a zero-risk level, but rather at a level that avoids unacceptable risks.

In recommending secondary standard options, the staff similarly notes that the final decision is largely a public policy judgment that draws upon scientific information and analyses about effects on public welfare, as well as judgments about how to deal with the range of uncertainties that are inherent in the scientific evidence and analyses. In making recommendations, the staff also takes into account the extent to which the recommended ranges of primary standards would provide the requisite protection against adverse PM-related effects on public welfare.

Further, the staff notes that especially where considerable uncertainty exists with regard to appropriate policy choices based on the scientific information and analyses, it is appropriate to consider the risk management implications for public health and welfare of alternative approaches that represent scientifically sound options. Thus, staff has considered risk management implications together with the scientific evidence in assessing whether alternative approaches to establishing PM standards would provide both the level of protection that is requisite and an effective and efficient basis for pollution control strategies that will result in the attainment and maintenance of that level of public health and welfare protection.

6.2 ADEQUACY OF CURRENT PRIMARY PM NAAQS

In 1997, EPA concluded that revision of the PM NAAQS was appropriate, given the body of scientific evidence indicating that health effects were associated with PM at ambient concentrations below the levels of the then effective PM_{10} standards. EPA further concluded that the most effective and efficient approach to revising the PM NAAQS to achieve the requisite level of protection would be to establish separate standards for the fine and coarse fractions of PM_{10} (62 FR at 38,665-66, July 18, 1997). Thus, as summarized in Chapter 1, EPA added $PM_{2.5}$ standards to address effects associated with fine-fraction particles, and retained (with minor

revision) PM_{10} standards, in conjunction with the $PM_{2.5}$ standards, to address effects associated with coarse-fraction particles.

As an initial step in considering the adequacy of the current suite of PM standards, staff first considered whether information now available in this review continues to support the previous conclusion that fine- and coarse-fraction particles should be regulated separately. As discussed in Chapter 2, fine- and coarse-fraction particles are distinct entities with fundamentally different sources and formation processes, chemical composition, atmospheric residence times, behaviors in the atmosphere, and patterns of human exposure. Also, as discussed in Chapter 3, studies have reported associations of increased mortality and morbidity with both fine- and coarse-fraction particles, and the two fractions of PM appear to have independent effects. Further, as was observed in the previous review (EPA, 1996b), the strategies needed for control of fine- and coarse-fraction particles are different. The recent air quality, exposure, and health effects information and analyses presented and evaluated in the draft CD provide further support for and increase staff's confidence in the conclusion that fine- and coarse-fraction particles should be treated as separate pollutants. As the draft CD concludes:

> Fine and thoracic coarse PM, indexed respectively by $PM_{2.5}$ and $PM_{10-2.5}$, should be considered as separate subclasses of PM. Considerations of emissions sources, atmospheric chemistry, physical behavior, exposure relationships, respiratory deposition, toxicologic findings, and epidemiologic observations argue for monitoring fine and thoracic coarse particles separately. (CD, p. E-42)

Thus, staff concludes that it is again appropriate to recommend retaining separate standards for fine- and coarse-fraction particles. Based on this recommendation, the following sections focus separately on the adequacy of the current $PM_{2.5}$ standards for controlling fine-fraction particles and the adequacy of the current PM_{10} standards for controlling coarse-fraction particles.

6.2.1 Adequacy of Current $PM_{2.5}$ Standards

Staff's assessment of the adequacy of the current $PM_{2.5}$ standards draws upon the extensive PM epidemiologic data base, as well as information from toxicologic, dosimetric, and exposure-related research, that has become newly available since the last review, as assessed in the draft CD and Chapter 3. Numerous new epidemiologic studies have shown statistically significant associations between short- and long-term ambient PM exposures (based on

community air quality measures) with a variety of human health endpoints, including total and cardiorespiratory mortality; hospital admissions, emergency department visits, and other medical visits for various respiratory or cardiovascular diseases; respiratory illness and symptoms; and lung function changes.

The draft CD notes that there is considerable coherence across the newly available epidemiologic study findings (CD, p. 9-127), and that the "general internal consistency of the epidemiologic database and available findings demonstrate well that notable human health effects are associated with exposures to ambient PM at concentrations currently found in many geographic locations across the United States" (CD, p. 9-66). These epidemiologic findings are further enhanced by new findings from toxicologic and dosimetric research, which have provided supportive evidence that certain particle attributes can be causally related to the observed health effects and insights into potential mechanisms by which PM may be affecting human health (CD, p. 9-61). The draft CD concludes that newly available epidemiologic studies are important in extending earlier results to many more cities (CD, p. 9-68), and that the relative risk estimates from the new studies generally comport well with previously reported effects estimates (CD, p. 9-75). Further, the draft CD concludes that there is now a large and reasonably convincing body of evidence that confirms earlier associations between short- and long-term ambient PM_{10} exposures and mortality/morbidity effects, as well as a growing body of studies that confirm such associations with ambient $PM_{2.5}$ exposures and suggest that $PM_{2.5}$ is a probable contributing cause of observed PM-associated health effects (CD, p. E-23). In addition, the draft CD highlights new findings that implicate fine-fraction particles as likely contributing to exacerbation of asthma and to increased lung cancer risk (CD, p. E-25, 26).

In evaluating this evidence, staff notes that there are many more epidemiologic studies now available in the current review that provide evidence of associations between $PM_{2.5}$ and serious health effects in areas with air quality at and above the level of the current annual $PM_{2.5}$ standard. In addition, as shown in Chapter 3, Appendix A, and discussed below in section 6.3.3, there are also a few newly available short-term exposure studies that provide evidence of statistically significant associations with $PM_{2.5}$ in areas with air quality below the annual standard level of 15 $\mu g/m^3$. For example, three such studies (Burnett et al., 2000 and Burnett and Goldberg, 2003; Mar et al., 1999, 2003; Fairley, 1999, 2003) report associations between short-

term $PM_{2.5}$ exposure and total and cardiovascular mortality in areas in which long-term average $PM_{2.5}$ concentrations ranged between 13 and 14 $\mu g/m^3$. Other short-term exposure studies with even lower long-term average $PM_{2.5}$ concentrations reported statistically significant associations with morbidity effects, although the weight to be appropriately accorded these studies is far more uncertain. New evidence is also available from long-term exposure studies suggesting that associations extend below the level of the current standard. For example, limited results from the extended ACS long-term exposure study (Pope et al., 2002), are suggestive of an association between mortality and long-term $PM_{2.5}$ exposure across cities in which the average aggregate mean concentration for the most recent period evaluated in the study was approximately 14 $\mu g/m^3$. The entire group of epidemiologic $PM_{2.5}$ studies provides direct and strong support for $PM_{2.5}$ standards that provide at least the level of protection afforded by the current standards, and also clearly calls into question the adequacy of that level of public health protection.

Beyond the epidemiologic studies using $PM_{2.5}$ as an indicator of fine-fraction particles, there is also a large body of newly available evidence from studies that used PM_{10}, as well as other indicators or components of fine-fraction particles (e.g., sulfates, combustion-related components), that provides additional support for the conclusions reached in the last review as to the likely causal role of ambient PM, and the likely importance of fine-fraction particles, in contributing to observed health effects. Such studies notably include new multi-city studies, intervention studies (that relate reductions in ambient PM to observed improvements in respiratory or cardiovascular health), and source-oriented studies (e.g., suggesting associations with combustion- and vehicle-related sources of fine-fraction particles). Further, the draft CD concludes that new epidemiologic studies of ambient PM associations with infant mortality and/or developmental effects, if further substantiated, suggest life shortening that could well be significantly larger than previously estimated, and that new associations with asthma-related increased physicians visits and symptoms suggest likely much larger public health impacts due to ambient PM than just those indexed by the mortality/morbidity effects considered in the last review (CD, p. E-28). Taken together, this group of studies also provides strong support for standards for fine-fraction particles that are at least as protective as the current $PM_{2.5}$ standards. These studies also raise questions as to whether those standards should be revised to provide increased protection to reflect the generally stronger and broader body of evidence now available

relative to the evidence that served as the basis for establishing the current $PM_{2.5}$ standards in the last review.

In considering the adequacy of the current standards, staff recognizes, however, that there are important limitations and uncertainties associated with this expanded body of evidence, as discussed at length in the draft CD and in Chapter 3, that need to be carefully considered in determining the weight to be placed on the newly available studies in the current review. The draft CD notes that several key issues need to be considered when interpreting this body of evidence. For example, while PM-effects associations continue to be observed across most new studies, the newer findings do not fully resolve the extent to which the associations are properly attributed to PM acting alone or in combination with other gaseous co-pollutants, or to the gaseous co-pollutants themselves. The draft CD notes that available statistical methods for assessing potential confounding by gaseous co-pollutants may not yet be fully adequate, although the various approaches that have now been used to evaluate this issue tend to substantiate that reported PM effects are at least partly due to PM acting alone or in the presence of other co-varying gaseous pollutants (CD, p. E-26).

Another issue of particular importance that has recently been highlighted is the sensitivity of various statistical models to the approach used to address potential confounding by weather- and time-related variables in time-series epidemiological studies. This issue resurfaced in the course of reanalyses of a number of the newer studies that were being conducted to address a more narrow issue related to problems associated with the use of commonly used statistical software (CD, 8-201; Chapter 3, section 3.5.3.1 above). These reanalyses suggest that weather continues to be a potential confounder of concern, and that there remains no altogether satisfactory way to select an appropriate model to address such potential confounding. The HEI Special Panel, in reviewing these reanalyses, concluded that this awareness introduces a degree of uncertainty in the time-series epidemiological studies that had not been widely appreciated previously (CD, p. 8-204).

In looking beyond PM mass indicators, a number of newly available studies highlight the issue of the extent to which observed health effects may be associated with various specific chemical components within the mix of fine particles. The potential for various fine particle components to have differing relative toxicities with regard to the various health endpoints being

considered adds complexity to the interpretation of the study results. The draft CD (CD, p. E-28) recognizes that more research is needed to address uncertainties about the extent to which various components may be relatively more or less toxic than others, or than undifferentiated $PM_{2.5}$ mass across the range of health endpoints studied.

These and other issues that introduce uncertainties into the interpretation of the available body of evidence are reflected in the draft CD's recognition that "many challenges still exist with regard to delineating the magnitudes and variabilities of risk estimates for ambient PM, the ability to attribute observed health effects to specific PM constituents, the time intervals over which PM health effects are manifested, the extent to which findings in one location can be generalized to other locations, and the nature and magnitude of the overall public health risk imposed by ambient PM exposure." (CD, p. 9-66).

Staff well recognizes that as the body of available evidence has expanded, it has both added greatly to our knowledge of PM-related effects, as well as to the complexity and uncertainties inherent in efforts to interpret the evidence in a policy-relevant context as a basis for setting appropriate standards. In considering both the available evidence and the inherent uncertainties in the evidence highlighted above, staff concludes that the available evidence sufficiently calls into question the adequacy of the current suite of $PM_{2.5}$ standards that consideration should be given to revising the current $PM_{2.5}$ standards to provide increased public health protection. Staff also concludes that there remain sufficiently important uncertainties in the available body of evidence such that consideration could also be given to retaining the current $PM_{2.5}$ standards at this time. Staff conclusions and recommendations on possible alternative primary standards for fine-fraction particles are discussed below in section 6.3.

6.2.2 Adequacy of Current PM_{10} Standards

EPA's decision in 1997 to continue to use PM_{10} as the indicator for standards to control coarse-fraction particles, rather than a more selective indicator (e.g., $PM_{10-2.5}$), was based in part on the recognition that the only studies of clear quantitative relevance to health effects most likely associated with coarse-fraction particles used undifferentiated PM_{10} as the indicator of coarse-fraction particles (in areas where the coarse fraction was the predominant component of PM_{10}) and on the very limited ambient air quality data then available on $PM_{10-2.5}$, in contrast to

the extensive monitoring network already in place for PM_{10} (62 FR at 38,668). However, as discussed in Chapter 1, in subsequent litigation regarding the 1997 PM NAAQS revisions, the court rejected as arbitrary EPA's reasons for adopting the PM_{10} indicator. In so doing, the court held in part that PM_{10} is a "poorly matched indicator" for coarse-fraction particles in the context of a rule that also includes $PM_{2.5}$ standards because PM_{10} includes $PM_{2.5}$. 175 F. 3d. at 1054. Although the court found "ample support" (id.) for EPA's decision to regulate coarse-fraction particles, the court nonetheless vacated the 1997 revised PM_{10} standards for the control of coarse-fraction particles.

In considering the adequacy of the current PM_{10} standards to control coarse-fraction particles, in conjunction with separate standards for $PM_{2.5}$, staff now concludes, consistent with the court's opinion, that PM_{10} is not an appropriate indicator for such standards. In reaching this conclusion, staff has taken into account information now available in this review, including the body of newly available evidence on health effects associated with coarse-fraction particles from studies that directly used $PM_{10-2.5}$ as the PM indicator. In addition, staff notes that there is now much more information available to characterize air quality in terms of estimated $PM_{10-2.5}$[2] than was available in the last review. Thus, the reasons used in support of the 1997 decision to retain PM_{10} as the indicator for coarse-fraction particles no longer hold. As a consequence of concluding that PM_{10} is no longer an appropriate indicator for coarse-fraction particles, staff also concludes that the current standards defined in terms of a PM_{10} indicator are not adequate or appropriate for the purpose of providing effective and efficient protection from health effects associated with coarse-fraction particles.

Taking into account the newly available information, staff also concludes that it is reasonable and appropriate in this review to consider replacing the current PM_{10} standards with standards based on an indicator of coarse-fraction particles that does not also include the fine fraction (e.g., $PM_{10-2.5}$). Staff conclusions and recommendations on possible alternative

[2] As noted above in section 2.5.3, coarse particle concentrations in EPA's monitoring network are currently estimated, not measured directly, using a difference method in locations with same-day data from co-located PM_{10} and $PM_{2.5}$ FRM monitors, resulting in air quality characterizations that are more uncertain than those available for $PM_{2.5}$ or PM_{10}.

indicators, averaging times, forms and levels for coarse-fraction primary standards are discussed below in section 6.4.

6.3 ALTERNATIVE PRIMARY STANDARDS FOR FINE-FRACTION PARTICLES

6.3.1 Alternative Indicators for Fine-Fraction Particle Standards

In 1997, EPA determined that it was more appropriate to control fine-fraction particles (referred to below as fine particles) as a group, as opposed to singling out particular components or classes of fine particles; thus, an undifferentiated mass-based indicator ($PM_{2.5}$) was selected rather than an indicator based on PM composition. A number of animal toxicologic and controlled human exposure studies had reported health effects associations with high concentrations of numerous fine particle components (e.g., sulfates, nitrates, transition metals, organic compounds), although such associations were not consistently observed. In addition, community health studies had found significant associations between fine particles or PM_{10} and health effects in areas with significant mass contribution of differing components or sources of fine particles, including sulfates, wood smoke, nitrates, secondary organic compounds and acid sulfate aerosols. It was also not possible to rule out any one PM component as contributing to the fine particle effects found in epidemiology studies. Thus, it was determined that total mass of fine particles was the most appropriate indicator for fine particle standards. (62 FR at 38,667).

In considering whether the information available in this review warrants selection of a different indicator for fine particles, staff notes that since the last review many new studies have continued to show associations between various health effects and short- and long-term exposure to fine particles as indexed by $PM_{2.5}$. In addition, an extensive $PM_{2.5}$ monitoring network has been deployed and operated in conjunction with the development of strategies and programs to implement the 1997 $PM_{2.5}$ standards. This network has provided substantial new air quality information, in terms of $PM_{2.5}$, that has been and is being used in ongoing PM research and has provided substantial information on $PM_{2.5}$ air quality that informs this review. Further, staff continues to recognize the importance of an indicator that not only captures all the fine PM components of potential concern (i.e., an effective indicator), but also places greater emphasis

for control on those constituents or fractions that are most likely to result in the largest risk reduction (i.e., an efficient indicator).

While many new studies continue to link adverse health effects with short- and long-term exposure to $PM_{2.5}$, different types of studies also have continued to suggest links between adverse health effects and a range of fine particle components and characteristics. Animal toxicologic and controlled human exposure studies have provided evidence linking a variety of fine particle components or particle types (e.g., sulfates or acid aerosols, metals, organic constituents, bioaerosols, diesel particles) with health effects, although often at high concentrations. In addition, some toxicologic studies have raised questions about the relative toxicity of size-differentiated subsets of fine particles, such as ultrafine particles (at or below 0.1 μm in diameter), focusing in particular on the chemical composition of such particles or their increased surface area. These findings are discussed in detail in Chapter 7 of the draft CD and summarized above in Section 3.5.2.2.

Some epidemiologic studies also have implicated various PM components (e.g., sulfates, nitrates, carbon, organic compounds, and metals) as being associated with adverse effects. For example, the draft CD discusses associations reported in a number of studies between sulfates and mortality, including both short-term (CD, Figure 8-8) and long-term (CD, p. 8-110) exposure studies. In addition, some recent studies have suggested that the ultrafine subset of fine particles may also be associated with adverse effects. For example, some European studies have suggested independent effects of both ultrafine and fine particles. Further, several important new studies have used factor analysis methods to test for associations between mortality and PM from different PM sources. These studies report associations generally with particles from combustion sources, including motor vehicles and burning of coal, wood or vegetation, but not with particles of crustal origin.

Thus, toxicologic and epidemiologic studies summarized above and discussed in the draft CD have provided evidence for effects associated with various fine particle components, size-differentiated subsets of fine particles, and sources of fine particles. The draft CD concludes "the new evidence suggests that exposure to particles from several different source categories, and of different composition and size, may have independent associations with health outcomes"

(CD, p. 9-110). Conversely, the draft CD provides no basis to conclude that any individual fine particle component *cannot* be associated with adverse health effects.

Given the range of health effects with which PM has been associated, and the range of potential mechanisms that may explain how PM may be causally related to such effects, the available evidence provides no basis for expecting that any one component or source of fine particles would be solely responsible for all PM-related effects. As summarized in the draft CD:

> Toxicological studies have provided considerable supportive evidence that certain physicochemical particle attributes can provide elements of 'causality' to observed health effects of ambient PM. A primary causative attribute may not exist but rather many attributes may contribute to a complex mechanism driven by the nature of a given PM and its contributing sources. The multiple interactions that may occur in eliciting a response in a host may make the identification of any single causal component difficult and may account for the fact that mass as the most basic metric shows the relationships to health outcomes that it does. (CD, p. 9-61).

However, it is likely that, for a given health response, some PM components are more closely linked with that response than others. As a consequence, for some specific effects, such as changes in specific immune cell numbers or release of cytokines, there may be stronger correlation with individual PM components than with particle mass. For example, in some toxicologic studies of cardiovascular effects, PM exposures of equal mass did not produce the same effects, indicating that PM composition was important (CD, p. 7-40). Staff recognizes that different PM constituents may have differing biological responses, and that this is an important source of uncertainty in interpreting epidemiologic evidence.

In addition to potential differences in biological responses for different mixes of PM components, there may be differences across communities that can influence the PM-health relationship. As summarized in the CD:

> . . . magnitudes and significance levels of observed air pollution-related effects estimates would be expected to vary somewhat from place to place, if the observed epidemiologic associations denote actual effects, because (a) not only would the complex mixture of PM vary from place to place, but also (b) affected populations may differ in characteristics that could affect susceptibility to air pollution health effects. Such characteristics include sociodemographic factors underlying health status, indoor-outdoor activities, diet, medical care access, exposure to risk factors other than ambient air pollution (such as extreme weather conditions), and variations in factors (e.g., air-conditioning) affecting human exposures to ambient-generated PM. (CD, p. 9-87).

Taking into account the above considerations, staff concludes that it remains appropriate to use undifferentiated particle mass as the basis for the indicator of fine particle standards. Staff further concludes that there is no adequate basis for supplementing mass-based fine particle standards with standards for any specific fine particle component or subset of fine particles. Staff also recognizes, however, that potential differences in PM, arising from different mixes of PM sources across the country, are a source of uncertainty in interpreting the epidemiologic evidence as a basis for establishing national fine particle standards.

In selecting a specific mass-based indicator for fine particles, it is also necessary to specify a specific size fraction, in terms of a sampler cut point, to differentiate the fine and coarse fractions. As recognized in the last review and summarized in Chapter 2, the particle diameter distinguishing fine- and coarse-fraction particles falls between 1 and 3 μm. As relative humidity increases, fine particles grow into this intermodal size range; as relative humidity decreases, more coarse-mode particles may be suspended in this size range (CD, p. 2-119). In its 1997 revisions to the PM NAAQS, EPA recognized that because of the potential overlap between fine and coarse particle mass in this intermodal range, the choice of any specific sampling cut point within this range is largely a policy judgment. In making this judgment, EPA selected 2.5 μm as the cut point for the fine particle indicator, based on considerations of consistency with the epidemiologic studies, the limited potential for intrusion of coarse-fraction particles into the fine fraction, and availability of monitoring technology (62 FR 38668). Further, EPA noted that $PM_{2.5}$ encompasses many specific components of potential concern in the fine fraction, including most sulfates, acids, transition metals, organics, and ultrafine particles.

While additional information is now available on effects associated with various components of fine particles, staff sees no basis in the new information for reaching a different judgment as to the most appropriate indicator of fine particles. Thus, staff concludes that 2.5 μm remains an appropriate cut point for including the larger accumulation-mode particles of the fine fraction while limiting intrusion of coarse fraction particles, and recommends that $PM_{2.5}$ be retained as an effective and efficient indicator for fine-fraction particles.

6.3.2 Alternative Averaging Times for $PM_{2.5}$ Standards

In the last review, EPA established $PM_{2.5}$ standards with annual and 24-hour averaging times (62 FR at 38,668-70). This decision was based in part on evidence of health effects related to both short-term (from less than 1 day to up to several days) and long-term (from a year to several years) measures of PM. EPA recognized that a 24-hour averaging time was consistent with the majority of community epidemiologic studies reporting daily associations. Further, EPA noted that a 24-hour standard could also effectively protect against episodes lasting several days, as well as providing some degree of protection from potential effects associated with shorter duration exposures. EPA also recognized that an annual standard would provide effective protection against both annual and multi-year, cumulative exposures that had been associated with an array of health effects, and that a much longer averaging time would complicate and unnecessarily delay control strategies and attainment decisions. The possibility of seasonal effects also was considered, although the very limited available evidence and the seasonal variability of sources of fine particle emissions across the country did not provide a satisfactory basis for establishing a seasonal averaging time.

In deciding to set both annual and 24-hour standards, EPA concluded that the most effective and efficient approach was to treat the annual standard as the generally controlling standard for lowering the entire distribution of $PM_{2.5}$ concentrations, with the 24-hour standard providing protection against the occurrence of peak 24-hour concentrations, particularly those that present localized or seasonal exposures of concern in areas where the highest 24-hour-to-annual mean $PM_{2.5}$ ratios are appreciably above the national average. This conclusion was supported by results of the PM risk assessment from the last review that indicated that peak 24-hour $PM_{2.5}$ concentrations contributed a relatively small amount to total health risk, such that much if not most of the aggregated annual risk associated with short-term exposures results from the large number of days during which the 24-hour average concentrations are in the low- to mid-range. Further, no evidence suggested that risks associated with long-term exposures are likely to be disproportionately driven by peak 24-hour concentrations. Thus, a generally controlling annual standard was judged to reduce risks associated with both short- and long-term exposures effectively and with more certainty than a 24-hour standard. Further, an annual standard is more stable over time, likely resulting in the development of more consistent risk

reduction strategies, since an area's attainment status would be less likely to change due solely to year-to-year variations in meteorological conditions that affect the atmospheric formation of fine particles.

Information available in this review generally is consistent with and supportive of the conclusions reached in the last review that provided the basis for setting both annual and 24-hour $PM_{2.5}$ standards, and for establishing the annual standard as the generally controlling standard. In considering the new information, staff makes the following observations:

- The conclusion that peak 24-hour $PM_{2.5}$ concentrations contribute a relatively small amount to the total health risk associated with short-term exposures on an annual basis, such that much if not most of the aggregated annual risk results from the large number of days during which the 24-hour average concentrations are in the low- to mid-range, is supported by EPA's updated risk assessment, as discussed in section 4.3.3. Support for this conclusion is also found in studies in which health effect associations remain when high-concentration days are removed from the analysis (Schwartz et al., 1996; Ostro et al., 1999, 2000).

- It continues to be the case, as discussed in section 4.2.6.1, that available short-term exposure studies do not provide evidence of population thresholds, but rather reflect relationships between health effects and ambient PM across a distribution of PM concentrations. Thus, as in the last review, staff recognizes that attempting to identify a "lowest observed effect level" in terms of a 24-hour concentration to serve as a basis for establishing a generally controlling 24-hour $PM_{2.5}$ standard would not be an appropriate approach in this review.

- Some recent studies have used a distributed lag over several days preceding the health event, as discussed in chapter 8 of the draft CD and summarized in section 3.5.2.2. While such studies continue to suggest consideration of a multiple day averaging time, staff notes that limiting 24-hour concentrations of fine particles will also protect against effects found to be associated with PM averaged over several days in health studies. Consistent with the conclusion reached in the last review, staff again concludes that the complexity of a multiple-day averaging time would not provide more effective protection than a 24-hour average.

- There is a growing body of studies that provide additional evidence of effects associated with exposure periods shorter than 24-hours (e.g., one to several hours), as noted in section 3.3. While staff concludes that this information remains too limited to serve as a basis for establishing a shorter-than-24-hour fine particle standard at this time, staff recognizes this as an important area of research that could provide a basis for the consideration of a shorter-term standard in the future.

- While some newer studies have investigated seasonal effects, as noted in section 3.3, staff concludes that currently available evidence of such effects is still too limited to serve as a basis for considering seasonal standards.

Based on the above considerations, staff concludes that the currently available information supports keeping and provides no basis for changing the averaging times of the current $PM_{2.5}$ standards. Staff recommends that both annual and 24-hour alternative $PM_{2.5}$ standards be considered together. Further, staff recommends that the annual standard be established so as to be the generally controlling standard, with the 24-hour standard continuing to provide protection against unusually high peak short-term $PM_{2.5}$ concentrations.

6.3.3 Alternative Annual $PM_{2.5}$ Standards

In the last review, EPA concluded that the level of a generally controlling annual $PM_{2.5}$ standard should be selected so as to limit annual $PM_{2.5}$ concentrations to somewhat below those where the body of epidemiologic evidence is most consistent and coherent (62 FR at 38,675-77). This approach recognized both the strengths and the limitations of the full range of scientific and technical information on the health effects of PM, as well as the associated uncertainties. In so doing, EPA placed the greatest weight on those U.S. and Canadian epidemiologic studies reporting statistically significant health effects associations with direct measures of fine particle mass. Further, EPA placed the greatest emphasis on the short-term exposure studies, while also considering whether the long-term exposure studies suggested the need for a lower level. This approach recognized that while effects may occur over the full range of concentrations observed in the studies, the strongest evidence for short-term $PM_{2.5}$ effects occurs at concentrations near the long-term average across the study period. Based on this approach, EPA set the annual $PM_{2.5}$ standard at a level of 15 $\mu g/m^3$, noting that this standard would provide substantial protection against short-term as well as long-term exposures to fine particles. In so doing, EPA recognized that while the possibility of effects at lower levels cannot be excluded, the evidence for that possibility is highly uncertain and the likelihood of significant health risk, if any, becomes smaller as concentrations approach the lower end of the range of air quality observed in the key epidemiologic studies and/or background levels. In addition, EPA recognized that such a standard could not be expected to offer an adequate margin of safety against the effects of all

potential short-term peak exposures in areas with strong local or seasonal sources of fine particles. Thus, this annual standard was set in conjunction with a 24-hour standard to supplement health protection from unusually high peak levels, as discussed below in section 6.3.4.

Since the last review, many new studies relating ambient $PM_{2.5}$ concentrations to health effects have been published; many of these studies have made progress in addressing some of the key uncertainties identified in the last review; and two or more years of ambient air quality data have been collected from the $PM_{2.5}$ monitoring network. Notwithstanding this extensive body of new information, staff notes that important uncertainties remain that need to be taken into account in weighing the new information as a basis for considering alternative $PM_{2.5}$ annual standards. In considering the newly available information and the associated uncertainties, staff believes that the approach taken in the last review to setting a generally controlling annual standard remains appropriate as a framework for evaluating the currently available information. Staff sees nothing fundamentally different in the currently available information that would invalidate the previous approach or indicate that a different approach would be more appropriate.

In first considering the results of currently available studies of short-term exposure to fine particles, and the associated fine particle levels, staff continues to focus on U.S. and Canadian studies for drawing quantitative conclusions for standard-setting purposes. In so doing, staff believes it is important to take into account the following factors:

- The extent to which reported health effects associations with fine particles are statistically significant, not only in single-pollutant models, but also in multi-pollutant models or other types of analyses that assess the likelihood that the association is robust to the inclusion of potentially confounding co-pollutants.

- The extent to which results modeled using GAM functions were re-analyzed to address statistical problems that may have been present in initially reported results.

- The relative reliability of the air quality data used in the study, including consideration of the type of monitoring used and the extent to which daily air quality was measured or estimated based on less-than-every-day measurements.

- The relative precision of the study results, with greater weight being placed on studies with greater statistical power to produce more precise effects estimates.

- The extent to which the weight of evidence supports an association between fine particles and the health endpoint assessed in a given study.

Effects estimates for mortality and morbidity effects from single-pollutant $PM_{2.5}$ short-term exposure studies are presented in the table in Appendix A of Chapter 3, along with reported mean $PM_{2.5}$ concentrations. Studies included in this table are those in which GAM was not used and those for which GAM results have been reanalyzed. Studies using GAM that were not reanalyzed are not included in this table, but are discussed in the draft CD (Chapter 8) and in section 3.3 above. Similarly, information on the extent to which co-pollutants were assessed in these studies, and the extent to which study results were robust to the inclusion of co-pollutants, is discussed in the draft CD and in section 3.3. Information on the type of monitoring used and the nature of the air quality data used in the study is generally summarized in the draft CD and in section 3.3, and discussed more specifically in the individual studies. The relative precision of mortality and morbidity $PM_{2.5}$ studies is generally shown in Figures 3-5 and 3-8, respectively.

Based on this information, and taking the factors identified above into consideration, staff makes the following observations:

- A number of epidemiologic studies reporting statistically significant associations with mortality and morbidity effects have been conducted in areas in which the long-term (i.e., annual or multi-year) mean 24-hour $PM_{2.5}$ concentrations ranged from over 40 $\mu g/m^3$ down to approximately 8.5 $\mu g/m^3$.

Many of these studies reported associations that remained significant upon reanalysis, were generally robust to the inclusion of co-pollutants in multi-pollutant models, had adequate air quality data for $PM_{2.5}$ mass, and resulted in relatively precise effects estimates of health effects for which the weight of the entire body of evidence supports the likelihood of an association for the health endpoint with fine particles. Other studies not included in the table in Appendix A of Chapter 3 also showed statistically significant associations based on using a GAM function, but because reanalyses have not been done, it is unclear whether the associations would remain significant with the use of more appropriate models. Taken together, these studies greatly extend the evidence of $PM_{2.5}$ associations beyond that available in the last review, in which 15.7 $\mu g/m^3$ was the lowest long-term mean 24-hour $PM_{2.5}$ concentration for which a statistically significant association was reported. Many of the newly available studies shown in

Appendix A in which the mean concentrations were above 15 $\mu g/m^3$ lend additional support for an annual standard at least as protective as the current standard. Further, studies showing statistically significant associations that have been conducted in areas in which the long-term mean 24-hour $PM_{2.5}$ concentrations were lower than 15 $\mu g/m^3$ support consideration of a lower standard level, as discussed below.

- Studies reporting statistically significant associations in areas where the long-term mean 24-hour $PM_{2.5}$ concentrations ranged from approximately 14 $\mu g/m^3$ down to 8.5 $\mu g/m^3$ provide evidence of $PM_{2.5}$-related total and cardiovascular mortality and emergency department visits related to asthma and cardiovascular illness at levels below the current annual standard.

In considering this group of studies, staff has focused first on those studies that included adequate gravimetric $PM_{2.5}$ mass measurements, and where mortality associations with fine particles were generally robust in models including gaseous copollutants. Studies conducted in Phoenix (Mar et al., 1999), Santa Clara County, CA (Fairley, 1999), and in eight Canadian cities (Burnett et al., 2000) reported significant relationships between $PM_{2.5}$ and mortality where mean $PM_{2.5}$ concentrations ranged between 13 and 14 $\mu g/m^3$. These studies were reanalyzed to address questions about the use of GAM, and the study results from Phoenix and Santa Clara County were little changed in alternative models (Mar et al., 2003; Fairley, 2003), although Burnett and Goldberg (2003) reported that their results were sensitive to using different temporal smoothing methods. Beyond these mortality studies, two other studies provided evidence of statistically significant associations with emergency department visits. One such GAM study (Norris et al., 1999) reported a highly significant association with asthma visits where the mean $PM_{2.5}$ concentration was approximately 12 $\mu g/m^3$, primarily based on nephelometry data. However, this study has not been reanalyzed, such that it is not clear if the association would remain significant with a more appropriate model, nor did the study assess the potential for confounding by co-pollutants. The other such study (Stieb et al., 2000) did not use GAM and reported a significant association with cardiovascular visits in a single pollutant model where the mean $PM_{2.5}$ concentration was approximately 8.5 $\mu g/m^3$. However, the $PM_{2.5}$ association was not robust to the inclusion of co-pollutants, such that the authors concluded that the results imply no effects of PM independent of co-pollutants (CD, 8B-15).

Based on consideration of the available $PM_{2.5}$ short-term exposure studies, staff believes that it would be appropriate to consider a range of $PM_{2.5}$ levels for an annual standard that extends down from 15 $\mu g/m^3$ to as low as 12 $\mu g/m^3$. Staff judges that consideration of a level as low as 12 $\mu g/m^3$ would be precautionary and would place much weight on the overall strength and coherence of the evidence, while giving less weight to the uncertainties that add to the difficulties inherent in attempting to interpret the available evidence in a policy-relevant context. Staff notes that while one study provides some suggestion that $PM_{2.5}$ associations may possibly extend to quite low levels beyond this range, it is not possible to distinguish the potential contribution of fine particles from that of the co-pollutants in this study. At the lower end of this range, consideration of air quality data measurement methods, potential confounding by co-pollutants, and uncertainties in appropriate model selection adds considerable uncertainty to the use of study results as a basis for quantitative standard-setting decisions.

Beyond the short-term exposure $PM_{2.5}$ studies, staff also has considered the results of currently available U.S. and Canadian studies of long-term exposure to fine particles. Information on these studies, and the associated mean fine particle levels, is discussed in the draft CD (Chapter 8) and section 3.3 above, especially in Tables 3-3 and 3-4 for mortality and morbidity effects, respectively. Based on this information, staff makes the following observations:

- Since the last review, reanalyses and extensions of previously available long-term exposure studies have confirmed and strengthened the evidence of associations between fine particles and mortality and morbidity effects.

- Newly available long-term mortality and morbidity studies lend support for considering an annual $PM_{2.5}$ standard at a level below the current standard, consistent with the evidence from the short-term exposure studies.

Two of the long-term mortality studies that were available in the previous review used data from the Six Cities and the ACS cohorts, and had aggregate mean $PM_{2.5}$ concentrations of 18 $\mu g/m^3$ (ranging from approximately 11 to 30 $\mu g/m^3$ across cities) and 21 $\mu g/m^3$ (ranging from approximately 9 to 34 $\mu g/m^3$ across cities), respectively. Reanalyses of data from these cohorts continued to report significant associations with $PM_{2.5}$, using essentially the same air quality distributions, and new analyses using the ACS cohort include $PM_{2.5}$ concentrations from 1999-

2000 that range from about 6 to 21 $\mu g/m^3$ (from figure 1, Pope et al., 2002) with a mean of approximately 14 $\mu g/m^3$ (Pope et al., 2002). Significant associations, though with somewhat smaller effect estimates, were reported with $PM_{2.5}$ in the new analysis (Pope et al., 2002).

New reports of associations between morbidity effects and long-term $PM_{2.5}$ exposure were available from the Southern California children's health cohort, where the means of 2-week average $PM_{2.5}$ concentrations, measured in 1994 using an acid/aerosol sampler designed for this study, ranged from approximately 7 to 32 $\mu g/m^3$, with an across-city average of approximately 15 $\mu g/m^3$ (Peters et al., 1999). In figures depicting relationships between lung function growth and average PM concentration, there is no evidence of a threshold in the data (Gauderman et al., 2000, 2002). As summarized in Table 3.4, the series of reports from this cohort study generally show decreases in lung function and lung function growth and increased risk of developing respiratory illness, though the findings do not always reach statistical significance.

Taking into account the above observations from both the short- and long-term exposure studies, staff concludes that the findings of new epidemiologic studies are consistent with the evidence that supported the 1997 decision to set a generally controlling annual $PM_{2.5}$ standard level of 15 $\mu g/m^3$, and that these findings clearly provide a basis for consideration of annual $PM_{2.5}$ standard levels lower than the current standard. In making a preliminary recommendation on a range of annual standard levels for consideration, staff believes that the range should extend down to a level of 12 $\mu g/m^3$. However, staff recognizes that information on the estimated reduction in health risks associated with just attaining alternative standards in this range is not yet available, and staff will consider such information in presenting conclusions and recommendations on this standard in the next draft of this Staff Paper.

In addition to considering the level of an annual standard, consideration must also be given to the form of the standard. In 1997 EPA established the form of the annual $PM_{2.5}$ standard as an annual arithmetic mean, averaged over 3 years, from single or multiple community-oriented monitors. This form was intended to represent a relatively stable measure of air quality and to characterize area-wide $PM_{2.5}$ concentrations. The arithmetic mean serves to represent the broad distribution of daily air quality values, and a 3-year average provides a more stable risk reduction target than a single-year annual average. The annual $PM_{2.5}$ standard level is to be compared to measurements made at either a single representative community-oriented

monitoring site, or the spatial average of measurements from multiple community-oriented monitoring sites (62 FR at 38,672). This approach was judged to be consistent with the epidemiologic studies on which the $PM_{2.5}$ standard was primarily based, in which air quality data were generally averaged from across several monitors in an area or taken from a single monitor that was selected to represent community-wide exposures, not localized "hot spots."

In this review, in conjunction with recommending that consideration be given to alternative standard levels, staff is also reconsidering the appropriateness of allowing for spatial averaging across monitors in an area. There now exists much more $PM_{2.5}$ air quality data than were available in the last review, and consideration of the spatial variability across urban areas that is revealed by this new database (see draft CD, section 3.2.5, and section 2.5 above) raises questions as to whether an annual standard that is based on averaging across monitors in an area will result in sufficient uniformity in public health protection in implementing such a standard in areas across the country. Staff intends to further assess this issue and will consider any such additional analysis in the next draft of this Staff Paper.

6.3.4 Alternative 24-Hour $PM_{2.5}$ Standards

In the last review, EPA considered a number of factors in establishing the level of the 24-hour $PM_{2.5}$ standard, including the significant protection afforded against short-term exposures by the annual $PM_{2.5}$ standard and the role of the 24-hour standard in providing supplemental protection against peak exposures not addressed by the annual standard (62 FR at 38,676-77). Air quality and effects information from the studies that helped to inform the selection of the annual standard also was considered, as were uncertainties in the risks associated with infrequent and isolated peak exposures in areas that meet the annual standard. Having decided to set the annual $PM_{2.5}$ standard at a level of 15 $\mu g/m^3$, EPA set the 24-hour standard at a level of 65 $\mu g/m^3$, which was the upper end of the range under consideration. In so doing, EPA placed much weight on the uncertainties associated with understanding the extent to which infrequent peak 24-hour exposures that could occur in areas that attain the annual standard contribute to the total risk associated with daily exposures over the course of a year.

In setting the level of the 24-hour standard, EPA first selected the form of the standard to be the 98^{th} percentile of 24-hour concentrations at each population-oriented monitor within an

area, averaged over three years (62 FR at 38671-74). EPA selected such a concentration-based form because of its advantages over the previously used expected-exceedance form[3]. A concentration-based form is more reflective of the health risk posed by elevated $PM_{2.5}$ concentrations because it gives proportionally greater weight to days when concentrations are well above the level of the standard than to days when the concentrations are just above the standard. Further, a concentration-based form better compensates for missing data and less-than-every-day monitoring; and, when averaged over 3 years, it has greater stability and, thus, facilitates the development of more stable implementation programs. After considering a range of concentration percentiles from the 95th to the 99th, EPA selected the 98th percentile as an appropriate balance between adequately limiting the occurrence of peak concentrations and providing increased stability and robustness. Further, by basing the form of the standard on concentrations measured at population-oriented monitoring sites (as specified in 40 CFR part 58), EPA intended to provide protection for people residing in or near localized areas of elevated concentrations.

As discussed above in section 6.3.3, since the last review, many new studies relating ambient $PM_{2.5}$ concentrations to health effects provide evidence of associations at air quality levels below those for which statistically significant associations were observed in the last review. In addition, the substantial $PM_{2.5}$ air quality database that is now available allows for better characterization of the relationship between annual concentrations and associated distributions of 24-hour concentrations in a large number of areas across the country. Nevertheless, significant uncertainty remains in understanding the risks associated with peak exposures in areas that meet alternative annual standards.

In considering the newly available information, the associated uncertainties, and the inherent difficulties in using this information as a basis for quantitative standard setting, staff has identified two different approaches to setting a 24-hour $PM_{2.5}$ standard to provide an appropriate degree of supplemental protection relative to the range of staff-recommended alternative annual standards for consideration in this review. One approach would focus on the upper end of the

[3] The form of the 1987 24-hour PM_{10} standard is based on the expected number of days per year (averaged over 3 years) on which the level of the standard is exceeded; thus, attainment with the one-expected exceedance form is determined by comparing the fourth-highest concentration in 3 years with the level of the standard.

distribution of daily $PM_{2.5}$ concentrations from short-term exposure studies reporting statistically significant health effects associations where the annual mean concentrations were within the staff-recommended range of annual standard levels. A second approach would focus on an assessment of recent $PM_{2.5}$ air quality data that presents the distributions of 98th percentile 24-hour average $PM_{2.5}$ concentrations as a function of annual mean $PM_{2.5}$ concentrations (shown in Chapter 2, Figure 2-20). The information presented in this figure provides some indication of the extent to which specific 24-hour standard levels would likely result in some degree of supplemental protection, beyond that provided by an annual standard alone, against peak exposures at sites that would likely attain specific annual standard levels. Staff believes that either approach, or both approaches considered together, can provide an appropriate basis for recommending a range of alternative 24-hour $PM_{2.5}$ standards for consideration in this review. In considering these approaches as a basis for identifying alternative levels for a 24-hour $PM_{2.5}$ standard, staff has continued to focus on standards with the same 98th percentile form as the current standard. While staff notes that alternative forms could also be considered in this review, staff sees nothing in the new information that calls into question the appropriateness of the current form or the basis for the selection of the form in the last review. Thus, staff recommends that consideration be given to retaining the 98th percentile form for a 24-hour $PM_{2.5}$ standard.

Using the first approach to consider alternative levels, staff focused on those short-term exposure studies discussed above in section 6.3.3 that provided the basis for the range of recommended alternative annual standards. The upper end of the $PM_{2.5}$ concentration ranges from these studies as well as other studies reporting health effects associations where annual mean $PM_{2.5}$ concentrations were at or below around 18 $\mu g/m^3$ were obtained from the published study or from the study authors and are documented in Ross (2003). In particular, staff has focused on the upper end of the concentration ranges for studies reporting statistically significant effects where annual mean $PM_{2.5}$ concentrations ranged from around 15 $\mu g/m^3$ down to approximately 12 $\mu g/m^3$. Looking first at the range of 98th percentile concentrations in these studies, the concentrations generally ranged from approximately 32 to 42 $\mu g/m^3$, with one study having a 98th percentile concentration near 60 $\mu g/m^3$. Looking more broadly at the 95th to 99th percentile concentrations in these studies, the concentrations generally ranged from somewhat below 30 $\mu g/m^3$ to approximately 45 $\mu g/m^3$, with one study having a 99th percentile

concentration near 70 μg/m^3. Taken together, this information suggests consideration of 24-hour $PM_{2.5}$ standard levels below the level of the current standard, ranging from approximately 45 μg/m^3 down to about 30 μg/m^3.

Using the second approach, staff looked at the information in Figure 2-20, based on an assessment of air quality data from 2000 to 2003 at over 1000 monitoring sites, to determine what 24-hour standard levels would likely result in some degree of supplemental protection relative to the protection afforded by an annual standard alone, for annual standards within the range of 15 to 12 μg/m^3. As an initial matter, staff observes that the current standard level of 65 μg/m^3 provides essentially no supplemental protection (with only one maximum 98th percentile concentration occurring above that level) in areas where the 3-year average annual means ranged from 16 μg/m^3 down to the lowest levels observed. Staff notes that the maximum values from the 98th percentile concentration distributions in areas with annual means ranging from 15 to 12 μg/m^3 are generally around 60 μg/m^3, whereas the top 5th percentile of these distributions are generally within the range of approximately 50 down to somewhat above 40 μg/m^3.

To assist in understanding the public health implications of various combinations of alternative annual and 24-hour standards, staff assessed (based on the same air quality database) the percentage of counties that would not likely attain various $PM_{2.5}$ annual standards alone in comparison to the percentage of counties that would not likely attain alternative combinations of annual and 24-hour $PM_{2.5}$ standards. This assessment is intended to provide some rough indication of the breadth of supplemental protection potentially afforded by various combinations of alternative standards. The results of such an assessment, based on air quality data from 693 counties (including 1152 monitoring sites), are shown in Table 6-1. From this table it can be seen that for an annual standard set at 15 μg/m^3, 24-hour standard levels ranging from 50 to 40 μg/m^3 would add approximately 2 to 6 % to the percentage of counties nationwide that would not likely attain both standards relative to the number of counties that would not likely attain the annual standard alone. For an annual standard set at 13 μg/m^3, 24-hour standard levels ranging from 50 to 35 μg/m^3 would add approximately 1 to 7 % to the percentage of counties not likely to attain both standards; and for an annual standard set at 12 μg/m^3, 24-hour standard levels in this range would add approximately 0 to 5 % to the percentage of counties.

Staff believes that both approaches provide useful information to frame the range of alternative 24-hour standards that are appropriate for consideration in this review. By combining the ranges suggested by both approaches, staff makes a preliminary recommendation that consideration be given in this review to setting a 24-hour $PM_{2.5}$ standard, with a 98^{th} percentile form, within a range of levels below the level of current standard, extending from approximately 50 $\mu g/m^3$ down to 30 $\mu g/m^3$. As in the last review, staff believes that selecting a 24-hour $PM_{2.5}$ standard from within this range should take into account the degree of protection likely afforded by the selected annual $PM_{2.5}$ standard. However, as noted above in section 6.3.3, staff will consider information not yet available from the human health risk assessment in presenting conclusions and recommendations on this standard in the next draft of this Staff Paper.

TABLE 6-1 PREDICTED PERCENTAGE OF COUNTIES WITH MONITORS NOT LIKELY TO MEET ALTERNATIVE $PM_{2.5}$ STANDARDS

Alternative Standards and Levels ($\mu g/m_3$)	Percent of counties not likely to meet alternative $PM_{2.5}$ standards *								
	Total	Region 1: Northeast	Region 2: Southeast	Region 3: Industrial Midwest	Region 4: Upper Midwest	Region 5: Southwest	Region 6: Northwest	Region 7: Southern CA	Outside Regions**
No. of Counties with Monitors	*693*	*111*	*197*	*145*	*62*	*32*	*114*	*15*	*17*
Annual:									
15	19	20	19	41	0	6	4	60	0
14	31	35	33	63	0	6	7	67	0
13	42	48	49	79	6	6	10	67	6
12	53	67	64	87	13	6	17	67	6
Combined Annual/24-Hour:									
15 / 65	19	20	19	41	0	6	4	60	0
15 / 50	21	20	19	41	0	6	14	67	0
15 / 40	25	26	19	41	0	6	28	80	6
15 / 35	34	48	20	54	2	9	41	80	6
15 / 30	59	84	53	89	11	19	51	80	6
15 / 25	80	97	87	99	50	44	64	80	18
13 / 50	43	48	49	79	6	6	16	73	6
13 / 40	46	50	49	79	6	6	29	80	6
13 / 35	49	54	50	80	8	9	41	80	6
13 / 30	63	84	60	94	16	19	51	80	6
13 / 25	80	97	87	99	50	44	64	80	18
12 / 50	53	67	64	87	13	6	18	73	6
12 / 40	56	68	64	87	13	6	31	80	6
12 / 35	58	69	65	87	15	9	41	80	6
12 / 30	67	86	72	95	21	19	51	80	6
12 / 25	81	97	88	99	50	44	64	80	18

* Based on 4, 8, or 12 consecutive quarters of data with at least 11 samples per quarter from the 2000 to 2002 database. As such, these estimates are not based on the same amount of air quality data that would be used to determine whether an area would attain a given standard or set of standards. These estimates can only approximate the number of counties that are likely not to attain the given standards and should be interpreted with caution.

** "Outside Regions" includes Alaska, Hawaii, Puerto Rico, and the Virgin Islands.

6.4 ALTERNATIVE PRIMARY STANDARDS FOR COARSE-FRACTION PARTICLES

6.4.1 Alternative Indicators for Coarse-Fraction Particle Standards

In 1997, in conjunction with new standards for fine particles, EPA decided to use PM_{10} as an indicator to protect against the effects most likely associated with coarse-fraction particles (referred to below as coarse particles) (62 FR at 38,668). As discussed above in section 6.2.2, in light of currently available information, the reasons for that decision no longer hold, and staff now concludes that PM_{10} is not an appropriate indicator for such standards.

The recent scientific evidence summarized in the draft CD does support, nonetheless, EPA's previous decision generally to use indicators based on PM mass (e.g., $PM_{2.5}$, PM_{10}) for the PM NAAQS, as was discussed above for fine particles. In addition, recent evidence from dosimetric studies supports retaining 10 μm as the appropriate cut point for particles capable of penetrating to the thoracic regions of the lung. Thus, in conjunction with $PM_{2.5}$ standards, staff concludes that an appropriate mass-based indicator for coarse particles would be $PM_{10-2.5}$.

There is limited evidence to support consideration of other indicators for coarse particles, such as individual components within the coarse fraction. In general, less is known about the composition of coarse particles than fine particles. Even less evidence is available from health studies that would allow identification of specific components or groups of components of coarse particles that may be more closely linked with specific health outcomes. Several studies have focused on the crustal, or geological component of particles, and the results suggest that crustal material, either in the fine or coarse fraction, is not associated with mortality (CD, p. 8-59). Studies conducted in Spokane, WA, and Utah Valley, UT reported that mortality was not associated with high PM_{10} concentrations resulting from dust storms, although there were statistically significant associations between PM_{10} and mortality in both (Schwartz et al., 1999 and Pope et al., 1999; studies not reanalyzed). However, particles of crustal origin may be linked with morbidity effects under some conditions, or resuspended coarse particles may serve as carriers for other more toxic components, such as metals from previously deposited fine particles or pesticides applied to agricultural lands. The draft CD concludes, however, that more research is needed to identify conditions under which one or another class of coarse particles may cause little or no adverse health effects, as well as conditions under which such particles

may cause notable effects (CD, p. 8-280). Thus, as for fine particles, there is no evidence that would lead toward the selection of one or more PM components as being primarily responsible for effects associated with coarse particles, nor is there any component that can at this time be eliminated from consideration.

Taking into account the above considerations, staff concludes that an undifferentiated mass-based indicator continues to be the most appropriate indicator for coarse particle standards. Staff recommends that such an indicator would appropriately retain 10 μm as the upper cut point for the coarse fraction, since larger-sized particles are less likely to penetrate into the thoracic regions, and that the lower cut point of 2.5 μm be used so as to most clearly differentiate between coarse ($PM_{10-2.5}$) and fine ($PM_{2.5}$) particles. Staff notes that consideration of a $PM_{10-2.5}$ indicator is supported by EPA's ongoing efforts to establish a FRM for $PM_{10-2.5}$ and to design a national network of such monitors (see section 2.5.3).

6.4.2 Alternative Averaging Times for $PM_{10-2.5}$ Standards

In the last review, EPA retained both annual and 24-hour standards to provide protection against the known and potential effects of short- and long-term exposures to coarse-fraction particles (62 FR at 38,677-79). This decision was based in part on qualitative considerations related to the expectation that deposition of coarse-fraction particles in the respiratory system could aggravate effects in individuals with asthma. In addition, quantitative support came from limited epidemiologic evidence suggesting that aggravation of asthma and respiratory infection and symptoms may be associated with daily or episodic increases in PM_{10}, where dominated by coarse-fraction particles including fugitive dust. Further, potential build-up of insoluble coarse-fraction particles in the lung after long-term exposures to high levels also was considered.

Information available in this review on coarse-fraction particles, while still limited, represents a significant expansion of the evidence available in the last review. A number of epidemiologic studies are now available that report statistically significant associations between short-term exposure to $PM_{10-2.5}$ and both morbidity effects and mortality. In addition, one recent study linked reduced lung function growth with long-term exposure to both $PM_{10-2.5}$ and $PM_{2.5}$. Staff believes that the newly available evidence continues to support the decision made in the last review to maintain both annual and 24-hour standards for control of coarse-fraction particles,

while recognizing that the quantitative evidence related to long-term exposures remains very limited.

In setting such standards, staff believes it is appropriate to use the same approach as discussed above in section 6.3.2 for $PM_{2.5}$ standards; that is, staff recommends that consideration be given to setting a generally controlling annual $PM_{10-2.5}$ standard in conjunction with a 24-hour standard to provide supplemental protection against unusually high peak short-term concentrations. This recommendation is supported by the results of EPA's updated risk assessment, showing that peak 24-hour $PM_{10-2.5}$ concentrations contribute a relatively small amount to the total health risk associated with short-term exposures on an annual basis, such that much if not most of the aggregated annual risk results from the large number of days during which the 24-hour average concentrations are in the low- to mid-range, as discussed in section 4.3.3. In addition, staff recognizes that available short-term exposure studies do not provide evidence of population thresholds, but rather reflect relationships between health effects and ambient $PM_{10-2.5}$ across a distribution of concentrations. Thus, as for $PM_{2.5}$, staff concludes that attempting to identify a "lowest observed effect level" in terms of a 24-hour concentration to serve as a basis for establishing a generally controlling 24-hour $PM_{10-2.5}$ standards would not be an appropriate approach in this review.

On the other hand, staff recognizes that the available evidence is far less certain with regard to potential long-term effects than for short-term effects. Thus, staff believes that consideration also could be given to establishing only a 24-hour $PM_{10-2.5}$ standard, possibly using an alternative approach as discussed below in section 6.4.4..

In considering the available information, staff concludes that there is no basis for considering any averaging times for $PM_{10-2.5}$ standards other than annual and 24-hour averages. While it is possible that peak concentrations of shorter duration may be associated with health effects, there is little published evidence to support a $PM_{10-2.5}$ standard with an averaging time of less than 24 hours. In the following sections, staff provides recommendations regarding alternative annual $PM_{10-2.5}$ standards, followed by recommendations on alternative short-term $PM_{10-2.5}$ standards, either in combination with an annual standard or alone.

6.4.3 Alternative Annual $PM_{10-2.5}$ Standards

In the last review, EPA's decision to retain the annual PM_{10} standard was based on the same approach as was used in 1987 for establishing the standard at a level of 50 $\mu g/m^3$. That approach considered the very limited quantitative evidence of associations linking higher long-term concentrations of various indicators of PM with health effects, together with qualitative evidence of particle deposition in the lung, to conclude that it is possible that cumulative deposition of coarse-fraction particles could be of concern. Other supporting evidence included qualitative information about long-term build-up of silica-containing materials and evidence related to biological aerosols, which supported the need to limit coarse-fraction particles but were not appropriately addressed by traditional air pollution control programs. Thus, this annual standard was set to address effects potentially associated with long-term exposures, in conjunction with a 24-hour standard set to address effects associated with short-term exposures.

In this review, staff is taking into consideration the still limited, but significantly expanded evidence now available that directly links primarily short-term $PM_{10-2.5}$ concentrations with morbidity effects (e.g., respiratory effects and hospital admissions) and mortality. Staff believes it is more appropriate to consider this evidence as a basis for setting a generally controlling annual $PM_{10-2.5}$ standard in conjunction with a supplemental 24-hour standard, as discussed above in section 6.4.2, rather than to use the approach from the last review of looking only at long-term exposure evidence as a basis for an annual standard. In so doing, staff is generally taking into account the same factors as those considered for $PM_{2.5}$, as discussed above in section 6.3.3. Also as discussed above, mortality and morbidity effects estimates and reported mean $PM_{10-2.5}$ concentrations are presented in the table in Appendix A of Chapter 3, or are discussed in the draft CD and in section 3.3 above (for the non-reanalyzed GAM studies). Discussion about the extent to which reported associations are robust to the inclusion of co-pollutants and the nature of the air quality monitoring used in the studies is generally summarized in the draft CD and in section 3.3 above. The relative precision of the mortality and morbidity $PM_{10-2.5}$ studies is depicted above in Figures 3-6 and 3-9, respectively.

Based on this information from short-term exposure studies, and taking the factors outlined in section 6.3.3 into consideration, staff makes the following observation:

- A number of short-term exposure studies reporting statistically significant associations with mortality and morbidity effects have been conducted in areas in which the annual mean 24-hour $PM_{10-2.5}$ concentrations ranged from over 33 $\mu g/m^3$ down to approximately 11 $\mu g/m^3$.

Several of these studies reported significant associations that remained significant upon reanalysis; and some were generally robust to the inclusion of co-pollutants in multi-pollutant models, had adequate air quality data of $PM_{10-2.5}$ mass, and resulted in relatively precise effects estimates of health endpoints for which the weight of the entire body of evidence supports the likelihood of an association with coarse-fraction particles. Other studies not included in the table in Appendix A also showed statistically significant associations based on using a GAM function; but, because reanalyses have not been done, it is unclear whether the associations would remain significant with the use of more appropriate models. Taken together, these studies significantly extend the evidence of $PM_{10-2.5}$ associations beyond that available in the last review.

In considering this group of studies, staff has focused first on those studies where significant mortality associations with $PM_{10-2.5}$ were generally robust in models including gaseous copollutants. Studies conducted in Phoenix (Mar et al., 1999; Mar et al., 2003) and Coachella Valley, CA (Ostro et al., 2000; Ostro et al., 2003) reported relatively precise statistically significant associations between $PM_{10-2.5}$ and total or cardiovascular mortality where mean $PM_{10-2.5}$ concentrations ranged from approximately 33.2 to 30.5 $\mu g/m^3$, respectively. In addition, a significant association was reported with total mortality in Steubenville (Schwartz et al., 1996; Schwartz, 2003), where the annual mean $PM_{10-2.5}$ concentration was approximately 16.1 $\mu g/m^3$, although this particular study did not assess the potential for confounding by co-pollutants.

A few studies also reported significant associations with morbidity effects, including respiratory and cardiovascular hospital admissions and respiratory symptoms. One such reanalyzed study in Detroit (Lippmann et al., 2000; Ito, 2003) reported relatively precise significant associations with hospital admissions for ischemic heart disease and pneumonia that were generally robust to inclusion of co-pollutants, where the annual mean $PM_{10-2.5}$ concentration was approximately 13.3 $\mu g/m^3$. Another study in Seattle (Sheppard et al., 1999; Sheppard et al., 2003) reported a relatively precise significant association with asthma hospital admissions where

the annual mean $PM_{10-2.5}$ concentration was approximately 16.2 $\mu g/m^3$, although the results were not robust to inclusion of co-pollutants, and were based on air quality mass measurements with nephelometry data used to fill in missing days. A few other studies reporting significant morbidity associations were either not reanalyzed or lasted less than a full year, such that annual mean $PM_{10-2.5}$ concentrations were not available.

Based on consideration of the available $PM_{10-2.5}$ short-term exposure studies, staff believes that it would be appropriate to consider a range of $PM_{10-2.5}$ levels for an annual standard that extends down from approximately 30 $\mu g/m^3$ to as low as 13 $\mu g/m^3$. Staff judges that consideration of a level as low as 13 $\mu g/m^3$ would be precautionary and would place much weight on the strength of the more limited and less consistent body of evidence now available on coarse particles as compared to the available evidence on fine particles (discussed above in section 6.3.3). At the lower end of this range, consideration of air quality data measurement methods, potential confounding by co-pollutants, and uncertainties in appropriate model selection adds considerable uncertainty to the use of study results as a basis for quantitative standard-setting decisions. Staff notes that these uncertainties are generally greater with regard to evidence from coarse particle studies than from fine particle studies.

Beyond the short-term exposure $PM_{10-2.5}$ studies, staff has also considered the results of currently available long-term exposure studies, and makes the following observation:

- Since the last review, only one long-term exposure study has become available that has assessed associations between $PM_{10-2.5}$ and morbidity effects, showing limited evidence of an association with reduced lung function growth in children.

This study (Gauderman et al., 2000) reported a significant association between long-term $PM_{10-2.5}$ concentrations (as well as PM_{10} and $PM_{2.5}$) and reduced lung function growth in children across 12 southern California communities, although the study results were mixed and limited and the authors found it difficult to separate the effects of the different PM indicators. Based on the graphical presentation of $PM_{10-2.5}$ concentrations in the study (from which a mean $PM_{10-2.5}$ concentration of roughly 20 $\mu g/m^3$ can be inferred), staff concludes that this study provides suggestive evidence of possible long-term $PM_{10-2.5}$ respiratory effects within the range of annual mean $PM_{10-2.5}$ concentrations recommended for consideration based on the short-term exposure study evidence (13 to 30 $\mu g/m^3$), suggesting that an annual standard at the upper end of that

range may not be protective against possible long-term effects. Staff notes, however, that other long-term exposure studies do not provide evidence of $PM_{10-2.5}$ mortality or morbidity effects, suggesting that it may be appropriate to place little weight on such effects in considering a standard within this range.

Taking into account the above observations from both the short- and long-term exposure studies, staff concludes that the findings of new epidemiologic studies support consideration of a range from approximately 30 $\mu g/m^3$ down to 13 $\mu g/m^3$ in setting a generally controlling annual $PM_{10-2.5}$ standard. As a caution in judging the weight to be placed on the studies discussed above, staff notes that the overall body of evidence for $PM_{10-2.5}$ health effects associations is more limited and less consistent than the evidence for $PM_{2.5}$ associations, especially with regard to mortality effects. In making a preliminary recommendation on a range of annual $PM_{10-2.5}$ standard levels for consideration, staff recognizes that information on the estimated reduction in health risks associated with just attaining alternative standards in this range is not yet available, and staff will consider such information in presenting conclusions and recommendations in the next draft Staff Paper.

In addition to considering the level of an annual standard, consideration must also be given to the form of the standard. Staff sees no basis at this time for recommending any revision to the form of the current annual PM_{10} standard, which is based on an annual arithmetic mean, averaged over 3 years, measured at each monitor within an area. However, as noted above in section 6.3.3, staff is reconsidering the appropriateness of allowing for spatial averaging across monitors as part of the form of an annual $PM_{2.5}$ standard. In further assessing this issue, staff also intends to consider the issue of spatial averaging with regard to the form of a $PM_{10-2.5}$ annual standard.

6.4.4 Alternative 24-Hour $PM_{10-2.5}$ Standards

In the last review, EPA's decision to retain the level of the 24-hour PM_{10} standard of 150 $\mu g/m^3$ (with revision to the form of the standard) was based on two community studies of exposure to fugitive dust that showed health effects only in areas experiencing large exceedances of that standard, as well as on qualitative information regarding the potential for health effects related to short-term exposure to coarse-fraction particles. Because of the very limited nature of

this evidence, staff concluded that while it supported retention of a standard to control coarse-fraction particles, it provided no basis for considering a more protective standard. However, because of concerns about the expected-exceedance-based form of the 1987 PM_{10} standard, primarily related to the stability of the attainment status of an area over time and complex data handling conventions needed in conjunction with less-than-every-day sampling, EPA adopted a concentration-based form for the 24-hour standard, as was done for the 24-hour $PM_{2.5}$ standard, as discussed above in section 6.3.4. In making this change, EPA selected a 99th percentile form, in contrast to the 98th percentile form adopted for the 24-hour $PM_{2.5}$ standard, so as not to allow any relaxation in the level of protection that had been afforded by the previous 1-expected-exceedance form.

As discussed above in section 6.4.3, since the last review, new evidence has become available that directly links primarily short-term $PM_{10-2.5}$ concentrations with morbidity effects and mortality. In considering this evidence as a basis for setting a 24-hour $PM_{10-2.5}$ standard, staff has focused primarily on alternative 24-hour standards that would supplement the protection afforded by the range of recommended annual standards, consistent with the approach used in considering alternative 24-hour $PM_{2.5}$ standards.

In considering alternative levels for a supplemental 24-hour $PM_{10-2.5}$ standard, staff has used the same two approaches that were used to consider alternative levels for the 24-hour $PM_{2.5}$ standard, discussed above in section 6.3.4. In considering these approaches as a basis for identifying alternative levels for a 24-hour $PM_{10-2.5}$ standard, staff has focused on standards with a 98th percentile form, consistent with the form of the 24-hour $PM_{2.5}$ standard. While alternative forms could also be considered in this review, staff believes there is good reason to make the forms consistent across standards, given that the reasoning behind setting different forms in the last review is no longer relevant.

The first approach focuses on the upper end of the distribution of daily $PM_{10-2.5}$ concentrations from short-term exposure studies reporting statistically significant health effects associations where the annual mean concentrations were within the staff-recommended range of annual standard levels. In using this approach, staff has focused on those short-term exposure studies discussed above in section 6.4.3 that provided the basis for the range of recommended alternative annual standards. The upper ends of the $PM_{10-2.5}$ concentration ranges from these and

other studies were obtained from the published study or from the study authors and are documented in Ross (2003). In particular, staff has focused on the upper end of the concentration ranges for studies reporting statistically significant effects where annual mean $PM_{2.5}$ concentrations ranged from approximately 13 $\mu g/m^3$ to over 30 $\mu g/m^3$. Looking first at the range of 98th percentile concentrations in these studies, the concentrations generally ranged from over 30 $\mu g/m^3$ to approximately 70 $\mu g/m^3$, with one study having a 98th percentile concentration over 100 $\mu g/m^3$. Looking more broadly at the 95th to 99th percentile concentrations in these studies, the concentrations generally ranged from somewhat below 30 $\mu g/m^3$ to approximately 75 $\mu g/m^3$, with one study having a 99th percentile concentration over 130 $\mu g/m^3$. Taken together, this information suggests consideration of 24-hour $PM_{10-2.5}$ standard levels ranging from approximately 30 to 75 $\mu g/m^3$ to supplement the protection afforded by the range of recommended annual standards.

The second approach focuses on an assessment of recent $PM_{10-2.5}$ air quality data that presents the distributions of 98th percentile 24-hour average $PM_{10-2.5}$ concentrations as a function of annual mean $PM_{10-2.5}$ concentrations (shown in Chapter 2, Figure 2-21). The information presented in this figure provides some indication of the extent to which specific 24-hour standard levels would likely result in some degree of supplemental protection, beyond that provided by an annual standard alone, against peak exposures at sites that would likely attain specific annual standard levels. In using this approach, staff has looked at the information in Figure 2-21, based on an assessment of air quality data from 2000 to 2002 at almost 500 monitoring sites, to determine what 24-hour standard levels would likely result in some degree of supplemental protection relative to the protection afforded by an annual standard alone, for annual standards within the range of 13 to 30 $\mu g/m^3$. As an initial matter, staff observes that alternative 24-hour standard levels based on this approach are highly dependent upon the level of the annual standard being considered. For example, for an annual standard at the lower end of the range, the top 5th percentile values from the distributions are generally with the range of approximately 40 $\mu g/m^3$ up to about 60 $\mu g/m^3$. For an annual standard around the middle of the range, the top 5th percentile values are generally with the range of approximately 50 $\mu g/m^3$ up to about 70 $\mu g/m^3$. At the upper end of the range of alternative annual standard levels, the top 5th percentile values are generally around 70 $\mu g/m^3$ or above.

To assist in understanding the public health implications of various combinations of alternative annual and 24-hour standards, staff assessed the $PM_{10-2.5}$ air quality database in the same manner as was done for the $PM_{2.5}$ database, as discussed above in section 6.3.4. The results of this assessment, based on air quality data from 351 counties (including 488 monitoring sites), is shown in Table 6-2. (To provide a rough comparison with the current PM_{10} standards, similar information is also provided in this table for the current combined PM_{10} standards.) From this table it can be seen that for an annual $PM_{10-2.5}$ standard set at 13 $\mu g/m^3$, 24-hour standard levels ranging from 60 to 40 $\mu g/m^3$ would add approximately 1 to 5 % to the percentage of counties that would not likely attain both standards relative to the number of counties that would not likely attain the annual standard alone. Similar increments are seen for an annual $PM_{10-2.5}$ standard set at 16 $\mu g/m^3$, with 24-hour standard levels ranging from 70 to 50 $\mu g/m^3$. For an annual $PM_{10-2.5}$ standard set at 30 $\mu g/m^3$, 24-hour standard levels at and above 70 $\mu g/m^3$ would add approximately 4% or less to the percentage of counties that would not likely attain the annual standard alone.

Staff believes that both approaches provide useful information to frame the range of alternative 24-hour standards that are appropriate for consideration in this review. By combining the ranges suggested by both approaches, staff makes a preliminary recommendation that consideration be given in this review to setting a 24-hour $PM_{10-2.5}$ standard, with a 98^{th} percentile form, within a range of levels from 30 $\mu g/m^3$ to 75 $\mu g/m^3$. Staff recognizes that selecting a 24-hour $PM_{10-2.5}$ standard from within this range should take into account the degree of protection afforded by the selected annual $PM_{10-2.5}$ standard.

TABLE 6-2 PREDICTED PERCENTAGE OF COUNTIES WITH MONITORS NOT LIKELY TO MEET ALTERNATIVE $PM_{10-2.5}$ and CURRENT PM_{10} STANDARDS

Alternative Standards and Levels ($\mu g/m_3$)	Percent of counties not likely to meet alternative $PM_{10-2.5}$ standards *								
	Total	Region 1: Northeast	Region 2: Southeast	Region 3: Industrial Midwest	Region 4: Upper Midwest	Region 5: Southwest	Region 6: Northwest	Region 7: Southern CA	Outside Regions**
No. of Counties with $PM_{10-2.5}$ Monitors	*351*	*53*	*70*	*73*	*29*	*19*	*83*	*14*	*10*
$PM_{10-2.5}$: Annual									
30	3	0	0	0	3	21	0	21	10
20	9	0	1	4	14	37	4	64	40
16	19	9	7	7	31	42	22	64	70
13	31	13	14	14	52	53	46	73	80
$PM_{10-2.5}$: 24-Hour									
75	4	0	1	1	10	21	2	21	10
70	6	0	1	4	14	26	5	21	20
60	11	2	7	7	14	37	7	50	50
50	18	13	7	8	31	37	17	57	80
40	29	23	11	15	55	47	35	64	90
35	36	25	20	21	66	53	46	71	90
$PM_{10-2.5}$: Combined Annual/24-Hour:									
30 / 75	5	0	1	1	10	21	2	29	20
30 / 70	7	0	1	4	14	26	5	29	20
30 / 60	11	2	7	7	14	37	7	50	50
20/70	11	0	3	4	17	37	8	60	40
20/60	13	2	7	7	17	37	10	60	50
20/50	19	13	7	8	31	37	18	60	80
16 / 70	20	9	9	7	34	42	23	64	70
16 / 60	21	9	11	8	34	42	24	64	70
16 / 50	24	15	11	10	38	42	29	64	90
13 / 60	32	13	16	14	55	53	46	79	80
13 / 50	33	19	16	15	55	53	47	79	90
13 / 40	36	25	16	18	69	53	47	79	90
No. of Counties with PM_{10} Monitors	*574*	*82*	*118*	*117*	*50*	*30*	*141*	*16*	*20*
PM_{10}: Combined Annual/24-Hour									
50/150	3	0	0	1	6	17	2	50	0

* Based on 4, 8, or 12 consecutive quarters of data with at least 11 samples per quarter from the 2000 to 2002 database. As such, these estimates are not based on the same amount of air quality data that would be used to determine whether an area would attain a given standard or set of standards. These estimates can only approximate the number of counties that are likely not to attain the given standards and should be interpreted with caution.

** "Outside Regions" includes Alaska, Hawaii, Puerto Rico, and the Virgin Islands.

Staff has also considered alternative 24-hour $PM_{10-2.5}$ standards that could serve as the sole means of protecting against known and potential effects associated with short- and long-term exposures to $PM_{10-2.5}$. In so doing, staff recognizes that since currently available short-term exposure studies provide no evidence of population thresholds, it would clearly not be appropriate to attempt to identify a "lowest observed effect level" in terms of a 24-hour concentration to serve as a basis for establishing a 24-hour $PM_{10-2.5}$ standard as the sole standard for coarse-fraction particles. Rather, staff recommends consideration of the same range of 24-hour $PM_{10-2.5}$ levels identified above, on the basis that such standards could potentially provide a roughly equivalent degree of protection on a national scale as would the generally controlling annual standards combined with the supplemental 24-hour standards, although clearly the distribution of counties not likely to attain would be somewhat different across the various regions presented in Table 6-2. In light of the very limited information on potential long-term effects, staff believes that consideration of a sole 24-hour $PM_{10-2.5}$ standard may be appropriate, although staff recognizes that using the currently available evidence to select such a standard may well be more difficult than it would be to select from a range of alternative annual standards in conjunction with a supplemental 24-hour standard.

In making the above preliminary recommendations on a range of 24-hour standards, either in combination with an annual standard or alone, staff recognizes that information on the estimated reduction in health risks associated with just attaining alternative standards in this range is not yet available. Staff will consider such information in presenting conclusions and recommendations in the draft of this Staff Paper.

6.5 SUMMARY OF STAFF RECOMMENDATIONS ON PRIMARY PM NAAQS

The major staff recommendations and supporting conclusions from sections 6.2 through 6.4 are briefly summarized below:

1. Consideration should be given to revising the current $PM_{2.5}$ primary standards to provide increased public health protection from fine particles based primarily on newly available evidence of mortality and morbidity health effects in areas where the annual mean concentrations are below the level of the current annual $PM_{2.5}$ standard.
2. The current PM_{10} primary standards should be revised and replaced with standard(s) that are defined in terms of an indicator that includes the coarse fraction without including the fine fraction. Such standards should be based primarily on evidence now available showing health effects associations with $PM_{10-2.5}$.
3. The suite of primary PM NAAQS should continue to focus on ambient particles capable of penetrating to the thoracic region, with separate standards for the fine and coarse fractions of PM_{10}. Staff concludes that the available information continues to support the use of $PM_{2.5}$ as the indicator for fine particles and supports the use of $PM_{10-2.5}$ as the indicator for coarse particles.
4. Staff recommends that $PM_{2.5}$ primary standards should continue to be based on both annual and 24-hour averaging times.
    a. Staff recommends that the Administrator consider selecting the level of the annual $PM_{2.5}$ primary standard from within a range of 15 $\mu g/m^3$ to approximately 12 $\mu g/m^3$. This recommended range is based primarily on quantitative results from both short- and long-term exposure studies in the U.S. and Canada, as well as taking into account the strength of the entire body of evidence showing health effects associated with exposure to $PM_{2.5}$ and the uncertainties and limitations inherent in evidence and in the methodological approaches available for interpreting the evidence. This recommended range is based on an approach that continues to view the annual standard as the generally controlling standard intended to reduce the entire distribution of $PM_{2.5}$ concentrations so as to provide public health protection by reducing risks associated with both short- and long-term exposures.

b. Staff recommends that the Administrator consider revising the 24-hour $PM_{2.5.}$ primary standard, selecting the level from within a range of approximately 50 $\mu g/m^3$ to 30 $\mu g/m^3$. This recommended range is based on a combination of approaches that focus on the upper end of the $PM_{2.5}$ concentration distributions both in key U.S. and Canadian epidemiologic studies and from the network of current $PM_{2.5}$ monitoring sites. These approaches are consistent with the continuing view that the 24-hour standard should be set so as to provide supplemental protection against peak exposures not addressed by the annual standard. Staff recommends that consideration be given to retaining the current 98^{th} percentile form of the standard.

5. Staff recommends that new $PM_{10-2.5}$ primary standards be established for both annual and 24-hour averaging times, with possible consideration given to establishing just a 24-hour standard.

a. Staff recommends that the Administrator consider selecting the level of the annual $PM_{10-2.5}$ primary standard from within a range of approximately 30 $\mu g/m^3$ to approximately 13 $\mu g/m^3$. This recommended range is based primarily on quantitative results from both short- and long-term exposure studies in the U.S. and Canada, as well as taking into account the strength of the entire body of evidence showing health effects associated with exposure to $PM_{10-2.5}$ and the uncertainties and limitations inherent in evidence and in the methodological approaches available for interpreting the evidence. This recommended range is based on the same approach used for the annual $PM_{2.5}$ standard, in that it views the annual standard as the generally controlling standard intended to reduce the entire distribution of $PM_{10-2.5}$ concentrations so as to provide public health protection by reducing risks associated with short-term and possibly long-term exposures.

b. Staff recommends that the Administrator consider selecting the level of a 24-hour $PM_{10-2.5.}$ primary standard from within a range of approximately 75 $\mu g/m^3$ to 30 $\mu g/m^3$. Consistent with the approach used for the 24-hour $PM_{2.5}$ standard, this recommended range is based on a combination of approaches that focus on the

upper end of the $PM_{10-2.5}$ concentration distributions both in key U.S. and Canadian epidemiologic studies and from the network of current PM_{10} and $PM_{2.5}$ monitoring sites. These approaches are consistent with the continuing view that the 24-hour standard should be set so as to provide supplemental protection against peak exposures not addressed by the annual standard. Alternatively, if consideration is given to establishing a 24-hour standard as the sole standard for $PM_{10-2.5}$, staff recommends that consideration be given to selecting such a standard from within the same range. In either case, staff recommends that consideration be given to setting a 24-hour $PM_{10-2.5}$ standard in terms of a 98^{th} percentile form, consistent with the form of the 24-hour $PM_{2.5}$ standard.

6. In recommending these primary standards and ranges of levels, staff is mindful that the Act requires standards to be set that are requisite to protect public health with an adequate margin of safety, such that the standards are to be neither more nor less stringent than necessary. Thus, the Act does not require that NAAQS be set at zero-risk levels, but rather at levels that avoid unacceptable risks to public health.

6.6 ALTERNATIVE SECONDARY STANDARDS FOR PM

6.6.1 Alternative Standards for Visibility Protection

In 1997, EPA decided to address the effects of PM on visibility by setting secondary standards identical to the suite of $PM_{2.5}$ primary standards, in conjunction with the establishment of a regional haze program under sections 169A and 169B of the Act (62 FR at 38,679-83). In reaching this decision, EPA first concluded that PM, especially in the fine fraction, produces adverse effects on visibility in various locations across the country, including multi-state regions, urban areas, and remote Class I Federal areas (e.g., national parks and wilderness areas). EPA also concluded that addressing visibility impairment solely through setting more stringent national secondary standards would not be an appropriate means to protect the public welfare from adverse impacts of PM on visibility in all parts of the country. As a consequence, EPA determined that an approach that combined national secondary standards with a regional haze program was the most appropriate and effective way to address visibility impairment.

In reaching these conclusions, EPA recognized that the selection of an appropriate level for a national secondary standard to address visibility protection was complicated by regional differences in visibility impairment due to several factors, including background and current levels of PM, the composition of PM, and average relative humidity. As a result of these regional differences, EPA noted that a national standard intended to maintain or improve visibility conditions in many parts of the West would have to be set at or below natural background levels in the East; conversely, a national standard that would improve visibility in the East would permit further degradation in the West. Beyond such problems associated with regional variability, EPA also determined that there was not sufficient information available to establish a standard level that would represent a threshold above which visibility conditions would always be adverse and below which conditions would always be acceptable.

These considerations led EPA to assess whether the protection afforded by the combination of the selected primary $PM_{2.5}$ standards and a regional haze program would provide appropriate protection against the effects of PM on visibility. Based on such an assessment, EPA determined that attainment of the primary $PM_{2.5}$ standards through the implementation of regional control strategies would be expected to result in visibility improvements in the East at both urban and regional scales, but little or no change in the West, except in and near certain urban areas. Further, EPA determined that a regional haze program that would make significant progress toward the national visibility goal in Class I areas would also be expected to improve visibility in many urban and non-Class I areas as well. EPA also noted, however, that the combined effect of the PM NAAQS and regional haze programs may not address all situations in which people living in certain urban areas may place a particularly high value on unique scenic resources in or near these areas. EPA concluded that such situations were more appropriately and effectively addressed by local visibility standards, such as those established by the city of Denver, than by national standards and control programs.

As called for in the last review, EPA promulgated a regional haze program in 1999. That program requires States to establish goals for improving visibility in Class I areas and to adopt control strategies to achieve these goals. More specifically, 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. Since strategies to meet these goals are to reflect a

coordinated approach among States, multistate planning organizations have been formed and are now developing strategies, to be adopted in the 2003 to 2008 time period, that will make reasonable progress in meeting these goals. Thus, although the regional haze program is now moving forward, there is as yet little additional information available as to the visibility improvements likely to result from this program in urban areas and other non-Class I areas.

In considering the information available in this review, as discussed in section 5.2, staff notes that while new research has led to improved understanding of the optical properties of particles and the effects of relative humidity on those properties, it has not changed the fundamental characterization of the role of PM in visibility impairment from the last review. Further, staff notes that regional factors continue to complicate any effort to identify an appropriate level for a national secondary standard to address visibility protection. Thus, staff continues to conclude that PM, especially in the fine fraction, produces adverse effects on visibility in various locations across the country, and that addressing visibility impairment solely through setting more stringent national secondary standards would not be appropriate.

Information now available from visibility and fine particle monitoring networks allows for an updated characterization of visibility trends and current levels in Class I areas and for future analyses to better characterize visibility impairment in urban areas. In considering the most recent monitoring information, as discussed in section 5.2, staff makes the following observations:

- In Class I areas, visibility levels on the 20% haziest days in the West are about equal to levels on the 20% best days in the East. Despite improvement through the 1990's, visibility in the rural East remains significantly impaired, with an average visual range of approximately 20 km on the 20% haziest days (compared to the naturally occurring visual range of about 150 $\pm$ 45 km). In the rural West, the average visual range showed little change over this period, with an average visual range of approximately 100 km on the 20% haziest days (compared to the naturally occurring visual range of about 230 $\pm$ 40 km).

- Urban areas generally have higher visibility impairment than Class I areas. Although urban visibility remains poorly characterized at this time, automated visibility measurements are now available for analysis in the form of high-resolution data from the ASOS network located at major airports to better characterize visibility in urban areas. Initial analysis of ASOS data, as discussed in section 5.2.4.2, shows promise in developing correlations between urban visibility data and $PM_{2.5}$ concentrations, although more analysis is needed.

New information is also now available from local regulatory efforts in the U.S. and abroad to establish standards and programs to address specific visibility concerns in particular urban and non-urban areas, as discussed in section 5.2.5. These efforts have produced new methods and tools to communicate and evaluate public perceptions about varying visual effects associated with alternative levels of visibility impairment relative to varying pollution levels and environmental conditions. Methods involving the use of focus groups to elicit citizen judgments about the acceptability of varying levels of visual air quality in an urban area have been developed by the State of Colorado, and used to develop a visibility standard for Denver. These methods have now been adapted and applied in other areas, including Phoenix, AZ, and the province of British Columbia, Canada, producing reasonably consistent results in terms of the visual ranges found to be generally acceptable by the participants in the various studies. These applications and results lend support to staff's conclusion that this approach holds promise for developing information in the future that will help inform judgments about acceptable urban visibility levels. Although resource constraints have prevented additional development of this approach since staff conducted the initial pilot study that was presented in the preliminary draft Staff Paper (EPA, 2001), EPA hopes to pursue it in the future so the results of a more extensive study can be used to help inform the next periodic review of the PM secondary standards.

Until such methods have been sufficiently developed and more broadly applied, staff believes it is appropriate to make use of photographic representations of visibility impairment to help inform judgments about the acceptability of varying levels of visual air quality in urban areas. As discussed in section 5.2, photographic representations of varying levels of visual air quality have been developed for several urban areas, in conjunction with efforts to develop local programs and with EPA's initial pilot study, and are available on EPA's website (http:/www.epa.gov/ttn/naaqs/standards/pm/s_pm_cr_sp.html) as an attachment to this document. In considering these images for Washington, D.C., Chicago, Denver, and Phoenix, staff makes the following observations:

- At concentrations at or near the level of the current 24-hour $PM_{2.5}$ standard, scenic views (e.g., mountains, historic monuments) as depicted in these images around and within the urban areas are significantly obscured from view.

- Appreciable improvement in the visual clarity of the scenic views depicted in these images occurs at concentrations toward the lower end of the staff-recommended range of consideration for the 24-hour primary $PM_{2.5}$ standards.

In making these observations, staff recognizes the limitations in using such visual representations as a basis for selecting a level for a visibility-based $PM_{2.5}$ secondary standard. Nonetheless, staff believes that these observations, together with the information discussed above and considered in the last review, support consideration of revising the current $PM_{2.5}$ secondary standards to be consistent with any revisions made to the $PM_{2.5}$ primary standards that would afford greater visibility protection, especially toward the lower end of the staff-recommended ranges for the primary standards. As in the last review, staff believes that such standards should be considered in conjunction with the regional haze program now being implemented as an effective approach to providing appropriate protection against PM-related visibility impairment across the country. Staff further believes that the development of local programs continues to be an effective and appropriate approach to provide additional protection for unique scenic resources in and around certain urban areas that are particularly highly valued by people living in those areas. In addition, staff expects that the results of any future local surveys of citizens' judgments as to the acceptability of varying visual air quality levels will provide useful information for consideration in subsequent PM NAAQS reviews.

6.6.2 Alternative Standards for Other Welfare Effects

EPA's decision in 1997 to revise the suite of PM secondary standards took into account not only visibility protection, but also the other PM-related welfare effects considered in the last review (i.e., materials damage and soiling). Based on this broader consideration, EPA established secondary standards for PM identical to the suite of primary standards, including both $PM_{2.5}$ and PM_{10} standards, to provide appropriate protection against the welfare effects associated with fine and coarse particle pollution (62 FR at 38,683). This decision was based on considering both visibility effects associated with fine particles, as discussed above in section 6.6.1, and materials damage and soiling effects associated with both fine and coarse particles. With regard to effects on materials, EPA concluded that both fine and coarse particles can

contribute to materials damage and soiling effects. However, EPA also concluded that the available data did not provide a sufficient basis for establishing a distinct secondary standard based on materials damage or soiling alone. These considerations led EPA to address whether the reductions in fine and coarse particles likely to result from the suite of PM primary standards would provide appropriate protection against the effects of PM on materials. Taking into account the available information and the limitations in that information, EPA judged that setting secondary standards identical to the suite of $PM_{2.5}$ and PM_{10} primary standards would provide increased protection against the effects of fine particles and retain an appropriate degree of control on coarse particles.

In this review, the draft CD and this draft Staff Paper have been broadened in scope to address not only PM-related effects on materials, but also effects on ecosystems and vegetation, as discussed above in section 5.4. In addition, staff has included in this draft Staff Paper consideration of the role of ambient PM in atmospheric process associated with climate change and the transmission of solar radiation, as discussed above in section 5.5. In considering the currently available evidence on each of these types of PM-related welfare effects, staff notes that there is much information linking ambient PM to potentially adverse effects on materials and ecosystems and vegetation, and on characterizing the role of atmospheric particles in climatic and radiative processes. However, based on the limitations in the evidence, especially with regard to the lack of evidence linking various effects to specific levels of ambient PM, staff concludes that the available evidence does not provide a sufficient basis for establishing distinct secondary standards based on any of these effects alone. These considerations lead staff to address whether the reductions in fine and coarse particles likely to result from the current secondary standards or the range of recommended revised $PM_{2.5}$ and $PM_{10-2.5}$ primary standards would provide appropriate protection against these types of PM-related welfare effects.

With regard to PM-related effects on materials, staff notes that the available evidence continues to support the following observations:

- Materials damage and soiling that occur through natural weathering processes are enhanced by exposure to atmospheric pollutants, most notably SO_2 and particulate sulfates.

- While ambient particles play a role in the corrosion of metals and in the weathering of paints and building materials, no quantitative relationships between ambient particle concentrations and rates of damage have been established.

- Similarly, while soiling associated with fine and coarse particles can result in increased cleaning frequency and repainting of surfaces, no quantitative relationships between particle characteristics (e.g., concentrations, particle size, and chemical composition) and the frequency of cleaning or repainting have been established.

Staff believes that these observations and the underlying available evidence continue to support consideration of retaining an appropriate degree of control on both fine and coarse particles. Lacking any specific quantitative basis for establishing distinct standards to protect against PM-related adverse effects on materials, staff recommends consideration be given to (1) retaining the current $PM_{2.5}$ secondary standards or to revising those standards to be consistent with any revisions made to the current $PM_{2.5}$ primary standards, and (2) retaining secondary standards for coarse particles, using a $PM_{10-2.5}$ indicator consistent with the primary standards, at a level that either retains the degree of protection afforded by the current PM_{10} standards or that is consistent with any new $PM_{10-2.5}$ primary standards.

With regard to PM-related effects on ecosystems and vegetation, staff notes that the draft CD presents evidence of such effects, particularly related to nitrate and acidic deposition, and concludes that current PM levels in the U.S. "have the potential to alter ecosystem structure and function in ways that may reduce their ability to meet societal needs" (CD, p. 4-153). Much of the associated uncertainty surrounding the characterization of the relationships between ambient PM levels and ecosystem or vegetation responses is related to the extreme complexity and variability that exist in predicting particle deposition rates, which are affected by particle size and composition, associated atmospheric conditions, and the properties of the surfaces being impacted. Though several national deposition monitoring networks have been successfully measuring wet and dry deposition for several decades, they often do not distinguish the form (e.g., particle, wet, and dry gaseous) in which a given chemical species is deposited, so that it is difficult to know what percentage of total deposition is attributable to ambient PM. Further, data from monitoring sites generally do not address all the variables affecting deposition that come into play in a natural system.

In addition to these uncertainties, many of the documented PM-related ecosystem-level effects only became evident after long-term, chronic exposures to specific chemical constituent(s) of PM eventually exceeded the natural buffering or assimilative capacity of the system. In most cases, PM deposition is not the only source of the chemical species to the affected system and the percentage of the deposition due to ambient PM is often not known. Because ecosystems have different sensitivities and capacities to buffer or assimilate pollutants, it is difficult to predict the rate of deposition that would be likely to lead to the observed adverse effects within any particular ecosystem. Equally difficult is the prediction of recovery rates for already affected areas if deposition of various chemical species were to be reduced.

Despite these uncertainties, a number of significant and adverse environmental effects that either have already occurred or are currently occurring have been linked to chronic deposition of chemical constituents found in ambient PM. Staff notes, for example, that the following effects have been linked with chronic additions of nitrate and its accumulation in ecosystems:

- Productivity increases in forests and grasslands, followed by decreases in productivity and possible decreases in biodiversity in many natural habitats wherever atmospheric reactive nitrogen deposition increases significantly and critical thresholds are exceeded;
- Acidification and loss of biodiversity in lakes and streams in many regions, especially in conjunction with sulfate deposition; and
- Eutrophication, hypoxia, loss of biodiversity, and habitat degradation in coastal ecosystems.

Staff notes that effects of acidic deposition have been extensively documented, as discussed in the draft CD and other reports referenced therein. For example, effects on some species of forest trees linked to acidic deposition include increased permeability of leaf surfaces to toxic materials, water, and disease agents; increased leaching of nutrients from foliage; and altered reproductive processes; all of which serve to weaken trees so that they are more susceptible to other stresses (e.g., extreme weather, pests, pathogens). In particular, acidic deposition has been implicated as a causal factor in the northeastern high-elevation decline of red spruce. Although U.S. forest ecosystems other than the high-elevation spruce-fir forests are not currently manifesting symptoms of injury directly attributable to acid deposition, less sensitive

forests throughout the U.S. are experiencing gradual losses of base cation nutrients, which in many cases will reduce the quality of forest nutrition over the long term.

Further, staff notes that chronic exposures to some metal constituents of ambient particles may be linked to adverse effects on vegetation. For example, although 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.

Taking into account the available evidence linking chemical constituents of both fine and coarse PM to these types of known and potential adverse effects on ecosystems and vegetation, staff believes that further reductions in ambient PM would likely contribute to long-term recovery and prevent further degradation of sensitive ecosystems and vegetation. Staff recognizes, however, that the available evidence does not provide any quantitative basis for establishing distinct national standards for ambient PM. Further, staff recognizes that due to site-specific sensitivities to various components of ambient PM, differing buffering and assimilative capacities, and local and regional differences in the percentage of total deposition that is likely attributable to ambient PM, national standards alone would not be an appropriate means to protect against adverse impacts of ambient PM on ecosystems and vegetation in all parts of the country. Nonetheless, staff believes that reductions in fine and coarse particles likely to result from the current suite of secondary standards or the range of recommended revised $PM_{2.5}$ and new $PM_{10-2.5}$ primary standards would contribute to increased protection against PM-related effects on ecosystems and vegetation. Staff recommends that the potential for increased protection of ecosystems and vegetation be taken into account in considering whether to revise the current PM secondary standards consistent with any revisions that may be made to the PM primary standards. Further, staff believes that any such increased protection should be considered in conjunction with protection afforded by other programs intended to address various aspects of air pollution effects on ecosystems and vegetation, such as the Acid Deposition Program and other regional approaches to reducing pollutants linked to nitrate or acidic deposition.

With regard to the role of ambient PM in climate change processes and in altering the penetration of solar UV-B radiation to the earth's surface, staff notes that information available

in this review derives primarily from broad-scale research and assessments related to the study of global climate change and stratospheric ozone depletion. As such, this information is generally focused on global- and regional-scale processes and impacts and provides essentially no basis for characterizing how differing levels of ambient PM in areas across the U.S. would affect local, regional, or global climatic changes or alter the penetration of UV-B radiation to the earth's surface. As noted in section 4.5.3, even the direction of such effects on a local scale remains uncertain. Moreover, similar concentrations of different particle components can produce opposite net radiative effects. Thus, staff concludes that there is insufficient information available to help inform consideration of whether any revisions of the current PM secondary standards are appropriate at this time based on ambient PM's role in atmospheric processes related to climate or the transmission of solar radiation.

6.6.3 Summary of Staff Recommendations on Secondary PM NAAQS

The major staff recommendations and supporting conclusions from section 6.5 are briefly summarized below:

1. Anthropogenic particles especially in the fine fraction impair visibility in various locations across the country, although the selection of an appropriate level for a national secondary standard to address visibility protection is complicated by regional differences in several factors, including background and current levels of PM, the composition of PM, and average relative humidity. In addition, both fine and coarse particles contribute to materials damage and soiling and to adverse effects on ecosystems and vegetation linked to PM deposition.
2. Staff recommends that consideration be given to revising the current $PM_{2.5}$ secondary standards to be consistent with any revisions made to the $PM_{2.5}$ primary standards that could be expected to afford greater visibility protection.
    a. Such standards, especially if set toward the lower end of the staff-recommended ranges, would be expected to result in appreciable improvements in visual air quality, especially in urban areas, beyond the protection likely to be afforded by the regional haze program now being implemented.

b. Such standards would also be expected to provide increased protection against the effects of fine particles related to materials damage and soiling and potential adverse impacts on ecosystems and vegetation linked to fine-particle deposition.

3. Staff recommends that consideration be given to replacing the current PM_{10} secondary standards with new $PM_{10-2.5}$ secondary standards that either retain the level of protection afforded by the current PM_{10} standards or are identical to any new $PM_{10-2.5}$ primary standards so as to continue control of coarse particles that contribute to materials damage and soiling and potential adverse impacts on ecosystems and vegetation.

4. In making these recommendations, staff has taken into account both the available evidence linking fine and coarse particles with effects on visibility, materials, ecosystems and vegetation, as well as the limitations in the available evidence. In so doing, staff recognizes that the available information does not provide a sufficient basis for the development of distinct national secondary standards to protect against such effects beyond the protection likely to be afforded by the suite of PM primary standards.

6.7 SUMMARY OF KEY UNCERTAINTIES AND RESEARCH RECOMMENDATIONS RELATED TO STANDARD SETTING

Staff believes it is important to continue to highlight the unusually large uncertainties associated with establishing standards for PM relative to other single component pollutants for which NAAQS have been set. Key uncertainties and staff research recommendations on both health-related and welfare-related topics are outlined below. In some cases, research in these areas can go beyond aiding in standard setting to aiding in the development of more efficient and effective control strategies. Staff notes, however, that a full set of research recommendations to meet standards implementation and strategy development needs is beyond the scope of this discussion.

The 1996 PM Staff Paper included a discussion of uncertainties and research recommendations (EPA, 1996b, pp. VII-41-44) that addressed the following issues related to understanding health effects associated with exposure to PM:

- lack of demonstrated biological mechanisms for PM-related effects,
- potential influence of measurement error and exposure error,

- potential confounding by copollutants,
- evaluation of the effects of components or characteristics of particles,
- the shape of concentration-response relationships,
- methodological uncertainties in epidemiological analyses,
- the extent of life span shortening,
- characterization of annual and daily background concentrations, and
- understanding of the effects of coarse fraction particles.

As has been discussed in depth in the draft CD, especially in Chapters 5 through 8, an extensive body of new studies related to understanding health effects associated with exposure to PM is now available that provides important information on many of the topics listed above. For example, regarding the lack of demonstrated biological mechanisms, new evidence from toxicologic and controlled human exposure studies has provided information on an array of potential mechanisms for effects on the cardiac and respiratory systems, as discussed in Chapters 7 and 9 of the draft CD. Still, the draft CD emphasizes that much remains to be learned to fully understand the pathways or mechanisms by which PM is linked with different health endpoints. For each of the issues listed above, new evidence has become available that helps to reduce uncertainties, although uncertainty has been reduced in some areas more than others. Staff has identified the following key uncertainties and research questions that have been highlighted in this review of the health-based primary standards

1. The body of evidence on effects of coarse fraction particles has been greatly expanded, but the uncertainties regarding coarse fraction particles are still greater than those for fine particles. As discussed in Chapter 2, the spatial variability of coarse fraction particles is generally greater than that for fine particles, which will increase uncertainty in the associations between health effects and coarse-fraction particles measured at central site monitors. Additional exposure research is needed to understand the influence of measurement error and exposure error on coarse fraction particle epidemiology results. In addition, little is known about coarse particle composition, and less about the health effects associated with individual components or sources of coarse fraction particles, but

it is possible that there are components of coarse fraction particles (e.g., crustal material) that are less likely to have adverse effects, at least at lower concentrations, than other components.

2. Identification of specific components, properties, and sources of fine particles that are linked with health effects remains an important research need. Available evidence provides no basis for expecting that any one component would be solely responsible for PM-related effects, but it is likely that some components are more closely linked with specific effects than others. Continued source characterization, exposure, epidemiological, and toxicological research is needed to help identify components, characteristics, or sources of particles that may be more closely linked with various specific effects to aid in our understanding of causal agents and in the development of efficient and effective control strategies for reducing health risks. Conducting human exposure research in parallel with such health studies will help reduce the uncertainty associated with interpreting health studies and provide a stronger basis for drawing conclusions regarding observed effects.

3. An important aspect in characterizing risk and making decisions regarding air quality standard levels is the shape of concentration-response functions for PM, including identification of potential threshold levels. Recent studies continue to show no strong evidence for a threshold level in the relationship between PM and mortality, though some studies have suggested potential levels (CD, p. 8-241).

4. The relationship between PM and other air pollutants in causing health effects remains an important question in reducing public health risk from air pollution. Numerous new analyses have indicated that associations found between PM and adverse health effects are not simply reflecting actual associations with some other pollutant. However, effects have been found with the gaseous copollutants, and it is possible that pollutants may interact or modify effects of one another. Further understanding of the sources, exposures, and effects of PM and other air pollutants can assist in the design of effective strategies for public health protection.

5. Methodological issues in epidemiology studies were discussed at length in the previous review, and it appeared at the time that the epidemiology study results were not greatly

affected by selection of differing statistical approaches or methods of controlling for other variables, such as weather. However, investigation of recently discovered questions on the use of generalized additive models in time-series epidemiology studies has again raised model specification issues. While reanalyses of studies using different modeling approaches generally did not generally result in substantial differences in model results, some studies showed marked sensitivity of the PM effect estimate to different methods of adjusting for weather variables. There remains a need for further study on the selection of appropriate modeling strategies and appropriate methods to control for time-varying factors, such as temperature. (see Section 3.5.3.1; CD section 8.4.2)

6. Selection of appropriate averaging times for PM air quality standards is important for public health protection, and available information suggests that effects may be linked to exposures of very short duration (e.g., one or more hours). Data on effects linked with such peak exposures, such as those related to wildfires, agricultural burning, or other episodic events, would be an important aid to public health response and communication programs. Investigation into the PM exposure time periods that are linked with effects will provide valuable information both for the standard-setting process and for risk communication and management efforts.

7. There remain significant uncertainties in the characterization of annual and daily background concentrations for fine particles, and especially for coarse particles. Further analyses of air quality monitoring and modeling that improved these background characterizations would help reduce uncertainties in estimating health risks relevant for standard setting (i.e., those risks associated with exposure to PM in excess of background levels) and would aid in the development and implementation of associated control programs.

With regard to welfare-related effects, discussed in Chapter 4 of the draft CD, staff has identified the following key uncertainties and research questions that have been highlighted in this review of the welfare-based secondary standards:

1. Urban visibility remains poorly characterized. Important information that would improve urban visibility characterization could be obtained from analyses of data from a number of monitoring networks and programs including: ASOS airport visibility data; $PM_{2.5}$ speciation trends network data; State and local continuous visibility monitoring (e.g., transmissometer or nephelometer data); and State and local continuous $PM_{2.5}$ monitoring. High-resolution data and analyses that focus on shorter than 24-hour averaging times would likely be particularly informative.
2. Refinement and broader application of survey methods designed to elicit citizens' judgments about the acceptability of varying levels of local visibility impairment could help inform future decisions on national secondary standards. Such research could appropriately build upon the methodology developed by the State of Colorado and used as a basis for setting a visibility standard for the city of Denver, which has been adapted and applied in other areas in the U.S. and abroad.
3. There remain significant uncertainties associated with the characterization and prediction of particle deposition rates to natural surfaces in general, and most importantly, with respect to particulate nitrate. Reduction in these uncertainties will be key to developing the capability of quantitatively linking ambient PM concentrations with environmental exposures and response. In order to better understand the nature of the role that PM plays in cumulative long-term environmental impacts, more research needs to be conducted on the percentage of total deposition contributed by PM and where necessary, better tools and monitoring methods should be developed.
4. The immense variability in sensitivity to PM deposition across U.S. ecosystems has not yet been adequately characterized, specifically the factors controlling ecosystem sensitivity to and recovery from chronic nitrogen and acid inputs. Data should be collected on a long term basis on a greater variety of ecosystems in conjunction with the development of improved predictive models. Such research could help in future consideration within the U.S. of the "critical loads" concept, which is generally accepted in Europe as the basis for abatement strategies to reduce or prevent injury to the functioning and vitality of forest ecosystems caused by long-range transboundary chronic acidic deposition.

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Appendix A. Estimated Total Cardiovascular and Respiratory Mortality and 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

Original study* Study Location Reanalysis study	Analysis Comments	% increase (95% CI) per 50 µg/m^3 PM_{10}	% increase (95% CI) per 25 µg/m^3 $PM_{2.5}$	% increase (95% CI) per 25 µg/m^3 $PM_{10-2.5}$	PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ Mean (Range) Levels Reported**
MORTALITY: Total (nonaccidental) Mortality					
Ito and Thurston, 1996 Chicago, IL	*GAM not used*	*2.47 (1.26, 3.69)*	---	---	*PM_{10} 38 (max 128)*
Kinney et al., 1995 Los Angeles, CA	*GAM not used*	*2.47 (-0.17, 5.18)*	---	---	*PM_{10} 58 (15, 177)*
Pope et al., 1992 Utah Valley, UT	*GAM not used*	*7.63 (4.41, 10.95)*	---	---	*PM_{10} 47 (11, 297)*
Schwartz, 1993 Birmingham, AL	*GAM not used*	*5.36 (1.16, 9.73)*	---	---	*PM_{10} 48 (21, 80)*
Schwartz et al., 1996 Boston, MA Schwartz, 2003a	*GAM Strict GLM NS GLM BS GLM PS*	---	*5.3 (3.5, 7.1) 5.7 (3.7, 7.6) 5.0 (3.1, 7.0) 4.5 (2.5, 6.5)*	*(GLM PS) 0.7 (-1.9, 3.4)*	*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 Schwartz, 2003a	*GAM Strict GLM NS GLM BS GLM PS*	---	*3.1 (0.0, 6.2) 3.0 (-0.3, 6.6) 2.8 (-0.5, 6.3) 2.6 (-0.8, 6.1)*	*(GLM PS) 1.7 (-2.7, 6.3)*	*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 Schwartz, 2003a	*GAM Strict GLM NS GLM BS GLM PS*	---	*2.6 (0.9, 4.3) 2.4 (0.6, 4.1) 2.6 (0.9, 4.4) 2.3 (0.6, 4.1)*	*(GLM PS) 0.3 (-2.1, 2.7)*	*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 Schwartz, 2003a	*GAM Strict GLM NS GLM BS GLM PS*	---	*2.4 (-0.4, 5.3) 1.7 (-1.3 4.8) 1.5 (-1.5, 4.6) 1.8 (-1.2, 4.9)*	*(GLM PS) 5.2 (0.0, 10.7)*	*PM_{10} 45.6 (SD 32.3) $PM_{2.5}$ 29.6 (SD 21.9) $PM_{10-2.5}$ 16.1 (SD 13.0)*

Original study* Study Location Reanalysis study	Analysis Comments	% increase (95% CI) per 50 µg/m^3 PM_{10}	% increase (95% CI) per 25 µg/m^3 $PM_{2.5}$	% increase (95% CI) per 25 µg/m^3 $PM_{10-2.5}$	PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ Mean (Range) Levels Reported**
Schwartz et al., 1996 *Portage, WI* *Schwartz, 2003a*	*GAM Strict* *GLM NS* *GLM BS* *GLM PS*	---	*2.6 (-1.2, 6.6)* *0.8 (-3.3, 5.1)* *1.5 (-2.7, 5.8)* *1.1 (-3.1, 5.4)*	*(GLM PS)* *0.7 (-4.0, 5.6)*	*PM_{10} 17.8 (SD 11.7)* *$PM_{2.5}$ 11.2 (SD 7.8)* *$PM_{10-2.5}$ 6.6 (SD 6.8)*
Schwartz et al., 1996 *Topeka, KS* *Schwartz, 2003a*	*GAM Strict* *GLM NS* *GLM BS* *GLM PS*	---	*1.6 (-5.3, 9.0)* *2.7 (-5.0, 10.9)* *1.3 (-6.2, 9.3)* *1.4 (-6.3, 9.6)*	*(GLM PS)* *-3.0 (-8.1, 2.3)*	*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* *Schwartz, 2003a*	*GAM Strict* *GLM NS* *GLM BS* *GLM PS*	---	*3.5 (2.5, 4.5)* *3.3 (2.2, 4.3)* *3.0 (2.0, 4.0)* *2.9 (1.8, 4.0)*	---	*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*	*GAM not used*	*4.08 (0.08, 8.24)*	---	---	*PM_{10} 37 (4, 365)*
Samet et al., 2000a,b 90 Largest U.S. Cities Dominici et al. (2003)	GAM strict GLM NS	1.4 (0.9, 1.9) 1.1 (0.5, 1.7)	---	---	PM_{10} mean range 15.3-52.0
Schwartz, 2000a 10 U.S. cities Schwartz, 2003b	GAM Strict GLM NS	3.4 (2.6, 4.1) 2.8 (2.0, 3.6)	---	---	PM_{10} mean range 27.1-40.6
Burnett et al., 2000 8 Canadian Cities Burnett and Goldberg, 2003	GAM Strict GLM NS (6 knots/yr)	3.2 (1.1, 5.5) 2.7 (-0.1, 5.5)	2.8 (1.2, 4.4) 2.1 (0.1, 4.2)	1.9 (-0.1, 3.9) 1.8 (-0.6, 4.4)	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	GAM not used		<75 years 2.6 (-2.0, 7.7) >75 years 1.5 (-3.0, 6.3)	<75 years 0.7 (-1.7, 3.) >75 years 1.3 (-1.3, 3.8)	NR
Clyde et al., 2000 Phoenix, AZ	GAM not used	6 (>0, 11)	---	---	PM_{10} mean 45.4

Original study* Study Location Reanalysis study	Analysis Comments	% increase (95% CI) per 50 μg/m^3 PM_{10}	% increase (95% CI) per 25 μg/m^3 $PM_{2.5}$	% increase (95% CI) per 25 μg/m^3 $PM_{10-2.5}$	PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ Mean (Range) Levels Reported**
Fairley, 1999 Santa Clara County, CA Fairley, 2003	GAM Strict GLM NS	7.8 (2.8, 13.1) 8.3 (2.9, 13.9)	8.1 (1.6, 15.0) 7.0 (1.4, 13.0)	4.5 (-7.6, 18.1) 3.3 (-5.3, 12.6)	PM_{10} 34 (6, 165) $PM_{2.5}$ 13 (2, 105) $PM_{10-2.5}$ 11 (0, 45)
Gamble, 1998 Dallas, TX	GAM not used	-3.56 (-12.73, 6.58)	---	---	PM_{10} 24.5 (11, 86)
Goldberg et al., 2000 Montreal, CAN Goldberg and Burnett, 2003	GAM Strict GLM NS	--- 	4.2 ($p<0.05$) 1.5 ($p>0.05$)	---	$PM_{2.5}$ 17.6 (4.6, 71.7)
Klemm and Mason, 2000 Atlanta, GA	GAM not used	8.7 (-5.2, 24.7)	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 Klemm et al., 2003	GAM Strict GLM NS	2.0 (0.0, 4.1) 1.0 (-1.5, 3.6)	2.0 (0.5, 3.5) 1.3 (-0.5, 3.0)	0.0 (-2.2, 2.3) -0.5 (-3.0, 2.0)	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 Klemm et al., 2003	GAM Strict GLM NS	2.5 (-1.7, 7.0) 1.5 (-1.7, 4.9)	1.5 (-1.6, 4.7) 0.5 (-2.7, 3.8)	4.6 (-0.7, 10.1) 4.0 (-1.6, 10.0)	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 Klemm et al., 2003	GAM Strict GLM NS	-3.5 (-11.6, 5.4) -5.4 (-14.3, 4.4)	1.5 (-6.5, 10.2) -0.5 (-9.5, 9.4)	-3.7 (-9.2, 2.1) -4.7 (-10.8, 1.8)	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 - Knoxville Klemm et al., 2003	GAM Strict GLM NS	6.1 (1.5, 11.0) 5.1 (-0.2, 10.7)	4.3 (0.9, 7.8) 3.8 (-0.1, 7.8)	3.5 (-1.0, 8.2) 3.0 (-1.9, 8.2)	PM_{10} 32.0 (SD 14.5) $PM_{2.5}$ 20.8 (SD 9.6) $PM_{10-2.5}$ 11.2 (SD 7.4)
Klemm et al., 2000 Six City reanalysis - Boston Klemm et al., 2003	GAM Strict GLM NS	6.1 (3.6, 8.8) 5.6 (2.8, 8.5)	5.1 (3.3, 6.9) 4.0 (1.9, 6.2)	1.3 (-1.1, 3.7) 1.8 (-1.0, 4.6)	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 Klemm et al., 2003	GAM Strict GLM NS	1.0 (-4.6, 7.0) -1.5 (-7.7, 5.1)	1.5 (-2.7, 5.9) -1.2 (-5.7, 3.5)	0.0 (-4.8, 5.0) -1.0 (-6.2, 4.5)	PM_{10} 17.8 (SD 11.7) $PM_{2.5}$ 11.2 (SD 7.8) $PM_{10-2.5}$ 6.6 (SD 6.8)

Original study* Study Location Reanalysis study	Analysis Comments	% increase (95% CI) per 50 μg/m^3 PM_{10}	% increase (95% CI) per 25 μg/m^3 $PM_{2.5}$	% increase (95% CI) per 25 μg/m^3 $PM_{10-2.5}$	PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ Mean (Range) Levels Reported**
Klemm et al., 2000 Six City reanalysis - overall Klemm et al., 2003	GAM Strict GLM NS	3.5 (2.0, 5.1) 2.5 (0.8, 4.3)	3.0 (2.0, 4.1) 2.0 (0.9, 3.2)	0.8 (-0.6, 2.1) 0.5(-1.0, 2.0)	PM_{10} means 17.8-45.6 $PM_{2.5}$ means 11.2-29.6 $PM_{10-2.5}$ means 6.6-16.1
Laden et al., 2000 Six City reanalysis Schwartz, 2003a	GLM PS	---	-5.1 (-13.9, 4.6) crustal 9.3 (4.0, 14.9) traffic 2.0 (-0.3, 4.4) coal	---	$PM_{2.5}$ same as Schwartz et al., 1996
Levy et al., 1998 King Co., WA	GAM not used	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	GAM not used	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 Ito, 2003	GAM Strict GLM NS	3.3 (-2.0, 8.9) 3.1 (-2.2, 8.7)	1.9 (-1.8, 5.7) 2.0 (-1.7, 5.8)	3.2 (-1.9, 8.6) 2.8 (-2.2, 8.1)	PM_{10} 31 (12, 105) $PM_{2.5}$ 18 (6, 86) $PM_{10-2.5}$ 13 (4, 50) mean (5%, 95%)
Moolgavkar, 2000a Los Angeles, CA Moolgavkar, 2003	GAM Strict GLM NS	2.4 (0.5, 4.2) 2.3 (0.5, 4.1)	1.5 (0, 3.0) 1.4 (-0.4, 3.2)	---	PM_{10} median 44 (7, 166) $PM_{2.5}$ 22 (4, 86)
Moolgavkar, 2000a Cook Co., IL Moolgavkar, 2003	GAM Strict GLM NS	2.4 (1.4, 3.5) 2.6 (1.6, 3.6)	---	---	PM_{10} median 35 (3, 365)
Ostro, 1995 San Bernadino and Riverside Counties, CA	GAM not used	---	0 (-1.4, 1.4)	---	$PM_{2.5}$ 32.5 (9.3, 190.1) (estimated from visibility)
Schwartz, 2000c Boston, MA Schwartz, 2003a	GLM NS	---	5.8 (4.5, 73) (15-day) 9.7 (8.2, 11.2) (60-day)	---	$PM_{2.5}$ 15.6 (±9.2)

Original study* Study Location Reanalysis study	Analysis Comments	% increase (95% CI) per 50 µg/m^3 PM_{10}	% increase (95% CI) per 25 µg/m^3 $PM_{2.5}$	% increase (95% CI) per 25 µg/m^3 $PM_{10-2.5}$	PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ Mean (Range) Levels Reported**
Schwartz, 2000 Chicago, IL Schwartz, 2003b	Strict GAM (dist. lag)	5.41 (2.36, 8.56)	---	---	PM_{10}
Schwartz, 2000 Pittsburgh, PA Schwartz, 2003b	Strict GAM (dist. lag)	3.14 (0.25, 6.11)	---	---	PM_{10}
Schwartz, 2000 Detroit, MN Schwartz, 2003b	Strict GAM (dist. lag)	6.83 (3.73, 10.02)	---	---	PM_{10}
Schwartz, 2000 Seattle, WA Schwartz, 2003b	Strict GAM (dist. lag)	7.46 (3.94, 11.10)	---	---	PM_{10}
Schwartz, 2000 Minneapolis, MN Schwartz, 2003b	Strict GAM (dist. lag)	10.25 (4.67, 16.12)	---	---	PM_{10}
Schwartz, 2000 Birmingham, AL Schwartz, 2003b	Strict GAM (dist. lag)	1.71 (-3.44, 7.13)	---	---	PM_{10}
Schwartz, 2000 New Haven, CT Schwartz, 2003b	Strict GAM (dist. lag)	9.17 (1.04, 17.96)	---	---	PM_{10}
Schwartz, 2000 Canton, OH Schwartz, 2003b	Strict GAM (dist. lag)	8.79 (-4.69, 24.18)	---	---	PM_{10}
Schwartz, 2000 Spokane, WA Schwartz, 2003b	Strict GAM (dist. lag)	5.62 (-0.31, 11.91)	---	---	PM_{10}

Original study* Study Location Reanalysis study	Analysis Comments	% increase (95% CI) per 50 µg/m^3 PM_{10}	% increase (95% CI) per 25 µg/m^3 $PM_{2.5}$	% increase (95% CI) per 25 µg/m^3 $PM_{10-2.5}$	PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ Mean (Range) Levels Reported**
Schwartz, 2000 Colorado Springs, CO Schwartz, 2003b	Strict GAM (dist. lag)	8.58 (-3.94, 22.73)	---	---	PM_{10}
Tsai et al., 2000 Newark, NJ	GAM not used	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	GAM not used	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	GAM not used	-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 Mortality:					
Samet et al., 2000a,b 90 Largest U.S. Cities Dominici et al. (2002)	GLM NS	1.6 (0.8, 2.4)	---	---	PM_{10} mean range 15.3-52.0
Tsai et al., 2000 Newark, NJ	GAM not used	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	GAM not used	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	GAM not used	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 Mortality					
Ito and Thurston, 1996 *Chicago, IL*	*GAM not used*	*1.49 (-0.72, 3.74)*	---	---	*PM_{10} 38 (max 128)*
Pope et al., 1992 *Utah Valley, UT*	*GAM not used*	*9.36 (1.91, 17.36)*	---	---	*PM_{10} 47 (11, 297)*

Original study* Study Location Reanalysis study	Analysis Comments	% increase (95% CI) per 50 μg/m^3 PM_{10}	% increase (95% CI) per 25 μg/m^3 $PM_{2.5}$	% increase (95% CI) per 25 μg/m^3 $PM_{10-2.5}$	PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ Mean (Range) Levels Reported**
Fairley, 1999 Santa Clara County, CA Fairley, 2003	GAM Strict GLM NS	8.5 (0.6, 17.0) 8.9 (1.3, 17.0)	6.3 (-4.1. 17.9) 6.7 (-2.5, 16.7)	5.0 (-13.3, 27.3)	PM_{10} 34 (6, 165) $PM_{2.5}$ 13 (2, 105) $PM_{10-2.5}$ 11 (0, 45)
Goldberg et al., 2000 Montreal, CAN Goldberg and Burnett, 2003	GAM Strict GLM NS	---	3.48 (-0.16, 7.26)	---	$PM_{2.5}$ 17.6 (4.6, 71.7)
Lipfert et al., 2000 Philadelphia, PA (7-county area)	GAM not used	8.0 (3.7, 12.3)	5.0 (2.4, 7.5)	5.4 (-0.4, 11.2)	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 Ito, 2003	GAM Strict GLM NS	5.4 (-2.6, 14.0) 4.9 (-3.0, 13.5)	2.2 (-3.2, 7.9) 2.0 (-3.4, 7.7)	6.7 (-1.0, 15.0) 6.0 (-1.6, 14.3)	PM_{10} 31 (12, 105) $PM_{2.5}$ 18 (6, 86) $PM_{10-2.5}$ 13 (4, 50) mean (10%, 90%)
Mar et al., 2000 Phoenix, AZ Mar et al., 2003	GAM Strict GLM NS	9.7 (1.7, 18.3) 9.5 (0.6, 19.3)	18.0 (4.9, 32.6) 19.1 (3.9, 36.4)	6.4 (1.3, 11.7) 6.2 (0.8, 12.0)	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 Moolgavkar, 2003	GAM Strict GLM NS	4.5 (1.6, 7.5) 3.9 (0.6, 7.4)	2.6 (0.4, 4.9) 1.7 (-0.8, 4.3)	---	PM_{10} median 44 (7, 166) $PM_{2.5}$ median 22 (4, 86)
Moolgavkar, 2000a Cook Co., IL Moolgavkar, 2003	GAM Strict GLM NS	2.2 (0.3, 4.1) 1.2 (-0.8, 3.1)	---	---	PM_{10} median 35 (3, 365)
Ostro et al., 2000 Coachella Valley, CA Ostro et al., 2003	GAM Strict GLM NS	5.5 (1.6, 9.5) 5.1 (1.2, 9.1)	9.8 (-5.7, 27.9) 10.2 (-5.3, 28.3)	2.9 (0.7, 5.2) 2.7 (0.4, 5.1)	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	GAM not used	---	0.69 (-0.34, 1.74)	---	$PM_{2.5}$ 32.5 (9.3, 190.1) (estimated from visibility)

Original study* Study Location Reanalysis study	Analysis Comments	% increase (95% CI) per 50 µg/m^3 PM_{10}	% increase (95% CI) per 25 µg/m^3 $PM_{2.5}$	% increase (95% CI) per 25 µg/m^3 $PM_{10-2.5}$	PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ Mean (Range) Levels Reported**
Total Respiratory Mortality:					
Ito and Thurston, 1996 *Chicago, IL*	*GAM not used*	*6.77 (1.97, 11.79)*	---	---	*PM_{10} 38 (max 128)*
Pope et al., 1992 *Utah Valley, UT*	*GAM not used*	*19.78 (3.51, 38.61)*	---	---	*PM_{10} 47 (11, 297)*
Fairley, 1999 Santa Clara County, CA Fairley, 2003	GAM Strict GLM NS	10.7 (-3.7, 27.2) 10.8 (-3.4, 27.1)	11.7 (-9.8, 38.3) 13.5 (-3.6, 33.7)	32.1 (-9.1, 92.2)	PM_{10} 34 (6, 165) $PM_{2.5}$ 13 (2, 105) $PM_{10-2.5}$ 11 (0, 45)
Lippmann et al., 2000 Detroit, MI Ito, 2003	GAM Strict GLM NS	7.5 (-10.5, 29.2) 7.9 (-10.2, 29.7)	2.3 (-10.4, 16.7) 3.1 (-9.7, 17.7)	7.0 (-9.5, 26.5) 6.4 (-10.0, 25.7)	PM_{10} 31 (12, 105) $PM_{2.5}$ 18 (6, 86) $PM_{10-2.5}$ 13 (4, 50) mean (10%, 90%)
Ostro, 1995 San Bernadino and Riverside Counties, CA	GAM not used	---	2.08 (-0.35, 4.51)	---	$PM_{2.5}$ 32.5 (9.3, 190.1) (estimated from visibility)
COPD Mortality:					
Moolgavkar, 2000a Cook Co., IL Moolgavkar, 2003	GAM Strict GLM NS	5.5 (0.2, 11.0) 4.5 (-1.6, 11.0)	---	---	PM_{10} median 35 (3, 365)
Moolgavkar, 2000a Los Angeles, CA Moolgavkar, 2003	GAM Strict GLM NS	4.4 (-3.2, 12.6) 6.2 (-3.4, 16.7)	1.0 (-5.1, 7.4) 0.5 (-6.8, 8.4)	---	PM_{10} median 44 (7, 166) $PM_{2.5}$ 22 (4, 86)

Original study* Study Location Reanalysis study	Analysis Comments	% increase (95% CI) per 50 µg/m^3 PM_{10}	% increase (95% CI) per 25 µg/m^3 $PM_{2.5}$	% increase (95% CI) per 25 µg/m^3 $PM_{10-2.5}$	PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ Mean (Range) Levels Reported**
CARDIOVASCULAR MORBIDITY					
Total Cardiovascular Hospital Admissions:					
Samet et al., 2000	strict GAM	4.95 (3.95 ,5.95))	---	---	PM_{10} means 24.4-45.3
14 U.S. Cities (>65 years)	strict GAM	5.73 (4.27, 7.20)			
Zanobetti and Schwartz (2003b)	(dist lag)				
	GLM NS	4.8 (3.55, 6.0)			
	GLM PS	5.0 (4.0, 5.95)			
Linn et al., 2000	GAM not used	3.25 (2.04, 4.47)	---	---	PM_{10} 45.5 (5, 132)
Los Angeles, CA (>29 years)					
Metzger et al., 2003	GAM not used	4.58 (-0.7, 10.2)	8.5 (2.6, 14.7)	3.0 (-3.7, 10.3)	PM_{10} 27.9 (0.5, 90.1)
Atlanta, GA					$PM_{2.5}$ 19.2 (1.8, 54.6)
					$PM_{10-2.5}$ 9.7 (0.5, 34.2)
Moolgavkar, 2000b	strict GAM_{100df}	4.05 (2.9, 5.2)	---	---	PM_{10} median 35 (3, 365)
Cook Co., IL (all ages)	GLM NS_{100df}	4.25 (3.0, 5.5)			
Moolgavkar, 2003					
Moolgavkar, 2000b	GAM_{30df}	3.35 (1.2, 5.5)	3.95 (2.2, 5.7)	---	PM_{10} median 44 (7, 166)
Los Angeles, CA (all ages)	GAM_{100df}	2.7 (0.6, 4.8)	2.9 (1.2, 4.6)		$PM_{2.5}$ median 22 (4, 86)
Moolgavkar, 2003	GLM NS_{100df}	2.75 (0.1, 5.4)	3.15 (1.1, 5.2)		
Tolbert et al., (2000a)	GAM not used	–8.2 (p=0.002)	---	---	PM_{10} 30.1 (SD 12.4)
Atlanta, GA 1993-1998					(Period 1)
Tolbert et al., 2000a	GAM not used	5.1 (-7.9, 19.9)	6.1 (-3.1, 16.2)	17.6 (-4.6, 45.0)	PM_{10} 29.1 (SD 12.0)
Atlanta, GA (all ages)					$PM_{2.5}$ 19.4 (SD 9.35)
					$PM_{10-2.5}$ 9.39 (SD 4.52)
Stieb et al., 2000	GAM not used	39.2 (5.0, 84.4)	15.11 (0.25, 32.8)	---	summer 93
St. John, CAN (all ages)					PM_{10} 14.0 (max 70.3)
					$PM_{2.5}$ 8.5 (max 53.2)

Original study* Study Location Reanalysis study	Analysis Comments	% increase (95% CI) per 50 µg/m^3 PM_{10}	% increase (95% CI) per 25 µg/m^3 $PM_{2.5}$	% increase (95% CI) per 25 µg/m^3 $PM_{10-2.5}$	PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ Mean (Range) Levels Reported**
Burnett et al., 1997 Toronto, CAN (all ages)	GAM not used	12.07 (1.43, 23.81)	7.18 (-0.61, 15.6)	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 Hospital Admissions:					
Schwartz and Morris, 1995 *Detroit (>65 years)*	*GAM not used*	*5.0 (1.9, 8.3)*	---	---	*PM_{10} 48 (22, 82)* *mean (10%, 90%)*
Lippmann et al., 2000 Detroit, MI (>65 years) Ito 2003	Strict GAM GLM NS	8.0 (−0.3, 17.1) 6.2 (−2.0, 15.0)	3.65 (−2.05, 9.7) 3.0 (−2.7, 9.0)	10.2 (2.4, 18.6) 8.1 (0.4, 16.4)	PM_{10} 31 (max 105) $PM_{2.5}$ 18 (6, 86) $PM_{10-2.5}$ 13 (4, 50)
Metzger et al., 2003 Atlanta, GA	GAM not used	5.6 (-3.9, 16.0)	5.8 (-4.1, 16.9)	-4.5 (-13.0, 11.6)	PM_{10} 27.9 (0.5, 90.1) $PM_{2.5}$ 19.2 (1.8, 54.6) $PM_{10-2.5}$ 9.7 (0.5, 34.2)
Dysrhythmias Hospital Admissions:					
Lippmann et al., 2000 Detroit, MI (>65 years) Ito (2003)	Strict GAM GLM NS	2.8 (−10.9-18.7) 2.0 (−11.7-17.7)	3.2 (−6.6-14.0) 2.6 (−7.1-13.3)	0.1 (−12.4-14.4) 0.0 (−12.5-14.3)	PM_{10} 31 (max 105) $PM_{2.5}$ 18 (6, 86) $PM_{10-2.5}$ 13 (4, 50)
Metzger et al., 2003 Atlanta, GA	GAM not used	4.1 (-5.9, 14.9)	3.8 (-5.8, 14.4)	5.3 (-6.3, 18.5)	PM_{10} 27.9 (0.5, 90.1) $PM_{2.5}$ 19.2 (1.8, 54.6) $PM_{10-2.5}$ 9.7 (0.5, 34.2)
Tolbert et al., 2000a Atlanta, GA (all ages)	GAM not used	13.4 (-14.1, 49.0)	6.1 (-12.6, 28.9)	53.2 (2.1, 129.8)	$PM_{2.5}$ 19.4 (SD 9.35) $PM_{10-2.5}$ 9.39 (SD 4.52)

Original study* Study Location Reanalysis study	Analysis Comments	% increase (95% CI) per 50 μg/m^3 PM_{10}	% increase (95% CI) per 25 μg/m^3 $PM_{2.5}$	% increase (95% CI) per 25 μg/m^3 $PM_{10-2.5}$	PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ Mean (Range) Levels Reported**
Heart Failure/Congestive Heart Disease Hospital Admissions:					
Schwartz and Morris, 1995 *Detroit (>65 years)*	*GAM not used*	*2.8 (0.7, 5.0)*	---	---	*PM_{10} 48 (22, 82)* *mean (10%, 90%)*
Linn et al., 2000 Los Angeles, CA (>29 years)	GAM not used	2.02 (-0.94, 5.06)	---	---	PM_{10} 45.5 (5, 132)
Lippmann et al., 2000 Detroit, MI (>65 years) Ito, 2003	Strict GAM GLM NS	9.2 (−0.3-19.6) 8.4 (−1.0-18.7)	8.0 (1.4-15.0) 6.8 (0.3-13.8)	4.4 (−4.0-13.5) 4.9 (−3.55-14.1)	PM_{10} 31 (max 105) $PM_{2.5}$ 18 (6, 86) $PM_{10-2.5}$ 13 (4, 50)
Metzger et al., 2003 Atlanta, GA	GAM not used	-3.9 (-14.9, 8.4)	14.3 (1.7, 28.6)	5.1 (-8.7, 21.0)	PM_{10} 27.9 (0.5, 90.1) $PM_{2.5}$ 19.2 (1.8, 54.6) $PM_{10-2.5}$ 9.7 (0.5, 34.2)
Morris and Naumova, 1998 Chicago, IL (>65 years)	GAM not used	3.92 (1.02, 6.90)	---	---	PM_{10} 41 (6, 117)
Myocardial Infarction Hospital Admissions:					
Linn et al., 2000 Los Angeles, CA (>29 years)	GAM not used	3.04 (0.06, 6.12)	---	---	PM_{10} 45.5 (5, 132)
Cardiac arrhythmia Hospital Admissions:					
Linn et al., 2000 Los Angeles, CA (>29 years)	GAM not used	1.01 (-1.93, 4.02)	---	---	PM_{10} 45.5 (5, 132)
Cerebrovascular Hospital Admissions:					
Linn et al., 2000 Los Angeles, CA (>29 years)	GAM not used	0.30 (-2.13, 2.79)	---	---	PM_{10} 45.5 (5, 132)
Stroke Hospital Admissions:					
Linn et al., 2000 Los Angeles, CA (>29 years)	GAM not used	6.72 (3.64, 9.90)	---	---	PM_{10} 45.5 (5, 132)

Original study* Study Location Reanalysis study	Analysis Comments	% increase (95% CI) per 50 μg/m^3 PM_{10}	% increase (95% CI) per 25 μg/m^3 $PM_{2.5}$	% increase (95% CI) per 25 μg/m^3 $PM_{10-2.5}$	PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ Mean (Range) Levels Reported**
Lippmann et al., 2000 Detroit, MI (>65 years) Ito, 2003	Strict GAM GLM NS	5.00 (-5.27, 16.38) 4.41 (-5.81, 15.74)	1.94 (-5.16, 9.57) 0.97 (-6.06, 8.52)	5.00 (-4.59, 15.56) 5.63 (-4.02, 16.25)	PM_{10} 31 (max 105) $PM_{2.5}$ 18 (6, 86) $PM_{10-2.5}$ 13 (4, 50)
Peripheral Circulation Hospital Admissions:					
Metzger et al., 2003 Atlanta, GA	GAM not used	10.4 (-0.9, 23.0)	13.0 (2.1, 25.0)	5.6 (-7.0, 8.6)	PM_{10} 27.9 (0.5, 90.1) $PM_{2.5}$ 19.2 (1.8, 54.6) $PM_{10-2.5}$ 9.7 (0.5, 34.2)
RESPIRATORY MORBIDITY					
Total Respiratory Hospital Admissions:					
Thurston et al., 1994 *Toronto, Canada*	*GAM not used*	*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)*
Linn et al., 2000 Los Angeles, CA (>29 years)	GAM not used	2.89 (1.09, 4.72)	---	---	PM_{10} 45.5 (5, 132)
Schwartz et al., 1996 Cleveland, OH (>65 years)	GAM not used	5.8 (0.5, 11.4)	---	---	PM_{10} 43
Lumley and Heagerty, 1999 King County, WA (all ages)	GAM not used	---	5.91 (1.10, 10.97)	---	PM_1 NR
Burnett et al., 1997 Toronto, CAN (all ages)	GAM not used	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)	GAM not used	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)

Original study* Study Location Reanalysis study	Analysis Comments	% increase (95% CI) per 50 µg/m^3 PM_{10}	% increase (95% CI) per 25 µg/m^3 $PM_{2.5}$	% increase (95% CI) per 25 µg/m^3 $PM_{10-2.5}$	PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ Mean (Range) Levels Reported**
Delfino et al., 1998 Montreal, CAN (>64 years)	GAM not used	---	13.17 (-0.22, 26.57)	---	$PM_{2.5}$ 18.6 (SD 9.3)
Stieb et al., 2000 St. John, CAN (all ages)	GAM not used	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 Hospital Admissions:					
Schwartz, 1995 *Detroit (>65 years)*	*GAM not used*	*5.9 (1.9, 10.0)*	---	---	*PM_{10} 48 (22, 82)* *mean (10%, 90%)*
Samet et al., 2000 14 U.S. Cities (>65 years) Zanobetti and Schwartz (2003b)	Strict GAM Strict GAM (dist. lag) GLM NS GLM PS	8.8 (5.9, 11.8) 8.3 (4.9, 12.0) 2.9 (0.2, 5.6) 6.3 (2.5, 10.3)	---	---	PM_{10} means 24.4-45.3
Lippmann et al., 2000 Detroit, MI (>65 years) Ito 2003	Strict GAM GLM NS	18.1 (5.3, 32.5) 18.6 (5.6, 33.1)	10.5 (1.8, 19.8) 10.1 (1.5, 19.5)	9.9 (−0.1, 22.0) 11.2 (−0.02, 23.6)	PM_{10} 31 (max 105) $PM_{2.5}$ 18 (6, 86) $PM_{10-2.5}$ 13 (4, 50)
COPD Hospital Admissions:					
Schwartz, 1995 *Detroit (>65 years)*	*GAM not used*	*10.6 (4.4, 17.2)*	---	---	*PM_{10} 48 (22, 82)* *mean (10, 90)*
Samet et al., 2000 14 U.S. Cities (>65 years) Zanobetti and Schwartz (2003b)	Strict GAM Strict GAM (dist. lag) GLM NS GLM PS	8.8 (4.8, 13.0) 13.3 (6.2, 20.9) 6.8 (2.8, 10.8) 8.0 (4.3, 11.9)	---	---	PM_{10} means 24.4-45.3
Linn et al., 2000 Los Angeles, CA (>29 years)	GAM not used	1.5 (-0.5, 3.5)	---	---	PM_{10} 45.5 (5, 132)
Tolbert et al., 2000a Atlanta, GA (all ages)	GAM not used	-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)

Original study* Study Location Reanalysis study	Analysis Comments	% increase (95% CI) per 50 µg/m^3 PM_{10}	% increase (95% CI) per 25 µg/m^3 $PM_{2.5}$	% increase (95% CI) per 25 µg/m^3 $PM_{10-2.5}$	PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ Mean (Range) Levels Reported**
Lippmann et al., 2000 Detroit, MI (>65 years) Ito (2003)	Strict GAM GLM NS	6.5 (−7.8, 23.0) 4.6 (−9.4, 20.8)	3.0(−6.9, 13.9) 0.3(−9.3, 10.9)	8.7 (−4.8, 24.0) 10.8 (−3.1, 26.5)	PM_{10} 31 (max 105) $PM_{2.5}$ 18 (6, 86) $PM_{10-2.5}$ 13 (4, 50)
Moolgavkar, 2000c Cook Co., IL (all ages) Moolgavkar 2003	Strict GAM: 100 df	3.24 (.03, 6.24)	---	---	PM_{10} median 35 (3, 365)
Moolgavkar, 2000c Los Angeles, CA (all ages) Moolgavkar 2003	Strict GAM: 30 df Strict GAM: 100 df GLM NS: 100df	7.78 (4.30, 11.38) 5.52 (2.53-8.59) 5.00 (1.22, 8.91)	4.69 (2.06, 7.39) 2.87 (0.53, 5.27) 2.59 (-0.29, 5.56)		PM_{10} median 44 (7, 166) $PM_{2.5}$ median 22 (4, 86)
Asthma Hospital Admissions:					
Choudbury et al., 1997 Anchorage, AK Medical Visits (all ages)	GAM not used	20.9 (11.8, 30.8)	---	---	PM_{10} 42.5 (1, 565)
Jacobs et al., 1997 Butte County, CA (all ages)	GAM not used	6.11 (p>0.05)	---	---	PM_{10} 34.3 (6.6, 636)
Linn et al., 2000 Los Angeles, CA (>29 years)	GAM not used	1.5 (-2.4, 5.6)	---	---	PM_{10} 45.5 (5, 132)
Lipsett et al., 1997 Santa Clara Co., CA (all ages)	GAM not used	9.1 (2.7, 15.9) (below 40° F)	---	---	PM_{10} 61.2 (9, 165)
Nauenberg and Basu, 1999 Los Angeles, CA (all ages)	GAM not used	20.0 (5.3, 35)	---	---	44.8 (SE 17.23)
Tolbert et al., 2000b Atlanta, GA (<17 years)	GAM not used	13.2 (1.2, 26.7)	---	---	PM_{10} 38.9 (9, 105)
Tolbert et al., 2000a Atlanta, GA (all ages)	GAM not used	18.8 (-8.7, 54.4)	2.3 (-14.8, 22.7)	21.1 (-18.2, 79.3)	PM_{10} 29.1 (SD 12.0) $PM_{2.5}$ 19.4 (SD 9.35) $PM_{10-2.5}$ 9.39 (SD 4.52)

Original study* Study Location Reanalysis study	Analysis Comments	% increase (95% CI) per 50 µg/m^3 PM_{10}	% increase (95% CI) per 25 µg/m^3 $PM_{2.5}$	% increase (95% CI) per 25 µg/m^3 $PM_{10-2.5}$	PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ Mean (Range) Levels Reported**
Sheppard et al., 1999 Seattle, WA (<65 years) Sheppard et al., 2003	Strict GAM GLM NS	10.9 (2.8, 19.6) 8.1 (0.1, 16.7)	8.7 (3.2, 14.4) 6.5 (1.1,12.0)	5.5 (0, 14.0) 5.5 (−2.7, 11.1)	PM_{10} 31.5 (90 55) $PM_{2.5}$ 16.7 (90 32) $PM_{10-2.5}$ 16.2 (90 29)
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)	*GAM not used*	*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)	*GAM not used*	*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)	*GAM not used*	---	*2.45 (1.29, 4.64)*	---	*$PM_{2.5}$ 24.5 (max 88.1)*
Ostro et al., 1991 Denver, CO (adults, cough)	*GAM not used*	*1.09 (0.57, 2.10)*	---	---	*PM_{10} 22 (0.5, 73)*
Pope et al., 1991 Utah Valley, UT (lower respiratory symptoms, schoolchildren)	*GAM not used*	*1.28 (1.06, 1.56)*	---	---	*PM_{10} 44 (11, 195)*
Pope et al., 1991 Utah Valley, UT (lower respiratory symptoms, asthmatic patients)	*GAM not used*	*1.01 (0.81, 1.27)*	---	---	*PM_{10} 44 (11, 195)*

Original study* Study Location Reanalysis study	Analysis Comments	% increase (95% CI) per 50 µg/m^3 PM_{10}	% increase (95% CI) per 25 µg/m^3 $PM_{2.5}$	% increase (95% CI) per 25 µg/m^3 $PM_{10-2.5}$	PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ Mean (Range) Levels Reported**
Neas et al., 1996 State College, PA (children, cough)	GAM not used	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)	GAM not used	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)	GAM not used	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)	GAM not used	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)	GAM not used	1.51 (1.04, 2.17)	---	---	PM_{10} 55.87 (19.63, 101.42)
Schwartz and Neas, 2000 Six Cities reanalysis (children, cough)	GAM not used	---	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)	GAM not used	---	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)	GAM not used	1.40 (1.14, 1.73)	---	---	PM_{10} median 22.1 (0.2, 159.0) (north site)

Original study* Study Location Reanalysis study	Analysis Comments	% increase (95% CI) per 50 µg/m^3 PM_{10}	% increase (95% CI) per 25 µg/m^3 $PM_{2.5}$	% increase (95% CI) per 25 µg/m^3 $PM_{10-2.5}$	PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ Mean (Range) Levels Reported**
Vedal et al., 1998 Port Alberni, CAN (children, phlegm)	GAM not used	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)	GAM not used	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)	GAM not used	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)	GAM not used	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)	GAM not used	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)	GAM not used	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)	GAM not used	1.16 (1.00, 1.34)	---	---	PM_{10} median 22.1 (0.2, 159.0) (north site)

Original study* Study Location Reanalysis study	Analysis Comments	% increase (95% CI) per 50 µg/m^3 PM_{10}	% increase (95% CI) per 25 µg/m^3 $PM_{2.5}$	% increase (95% CI) per 25 µg/m^3 $PM_{10-2.5}$	PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ Mean (Range) Levels Reported**
Lung Function Changes		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)	*GAM not used*	---	*-2.58 (-5.33, +0.35)*	---	*$PM_{2.5}$ 24.5 (max 88.1)*
Thurston et al., (1997) Connecticut summer camp (children)	GAM not used	---	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)	GAM not used	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)	GAM not used	---	pm PEFR -0.64 (-1.73, 0.44)	---	$PM_{2.5}$ 23.5 (max 85.8)
Neas et al., 1999 Philadelphia, PA (children)	GAM not used	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)	GAM not used	---	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)	GAM not used	---	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

Original study* Study Location Reanalysis study	Analysis Comments	% increase (95% CI) per 50 µg/m^3 PM_{10}	% increase (95% CI) per 25 µg/m^3 $PM_{2.5}$	% increase (95% CI) per 25 µg/m^3 $PM_{10-2.5}$	PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ Mean (Range) Levels Reported**
Vedal et al., 1998 Port Alberni, CAN (children)	GAM not used	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.

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15. SUPPLEMENTARY NOTES

16. ABSTRACT

The purpose of the Staff Paper is to evaluate the policy implications of the key scientific and technical information contained in a related EPA document, *Air Quality Criteria for Particulate Matter* (AQCD), and identify critical elements that EPA staff believe should be considered in reviewing the PM National ambient air quality standards (NAAQS). The Staff Paper is intended to "bridge the gap" between the scientific review contained in the AQCD and the public health and welfare policy judgments required of the Administrator in reviewing the NAAQS. A fourth external review draft of the AQCD has recently been made available for public review and comment (66 FR 18929, June 30, 2003), and for review by CASAC at a public meeting on August 25-26, 2003. Building upon an earlier preliminary draft Staff Paper (EPA, 2001), and taking into account the information in the fourth external review draft of the AQCD, this first draft Staff Paper includes results from initial staff analyses (e.g., analyses of air quality and visibility data, and human health risk assessment), plans for additional analyses to be incorporated into a subsequent draft of this document, and preliminary staff conclusions or recommendations on potential revision or retention of the PM NAAQS.

17. KEY WORDS AND DOCUMENT ANALYSIS

a. DESCRIPTORS	b. IDENTIFIERS/OPEN ENDED TERMS	c. COSATI Field/Group
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