United States Environmental Protection Agency

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

Policy Assessment of Scientific and Technical Information

OAQPS Staff Paper – Second Draft

January 2005

EPA-452/D-05-001
January 2005
Second Draft

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Office of Air Quality Planning and Standards
U.S. Environmental Protection Agency
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DISCLAIMER

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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
ANC	Acid neutralizing capacity
APHEA	Air Pollution and Health, a European Approach
AQCD	Air Quality Criteria Document
AQS	Air Quality System
ARS	Air Resource Specialists, Inc.
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
CL	Critical loads
C:N	Carbon-to-nitrogen ratio
CO	Carbon monoxide
COH	Coefficient of haze
COPD	Chronic obstructive pulmonary disease
CPSC	Consumer Product Safety Commission
C-R	Concentration-response
CSS	Coastal sage scrub community
CV	Contingent valuation
EC	Elemental carbon
ECG	Electrocardiogram
ED	Emergency department
EEA	Essential Ecological Attribute

EMAP	Environmental Monitoring and Assessment Program
EPA	Environmental Protection Agency
EPEC	Ecological Processes and Effects Committee
ERP	Episodic Response Project
FDMS	Filter Dynamics Measurement System
FLM	Federal Land Manager
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
HF	Heart failure
hosp. adm.	Hospital admissions
IC	Ion chromatography
IFS	Integrated Forest Study
IHD	Ischemic heart disease
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
NEG/ECP	New England Governors/Eastern Canadian Premiers
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
NWS	National Weather Service

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
OSHA	Occupational Safety and Health Administration
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
PnET-BGC	A forest net productivity model (PnET) linked to a soil model (BGC)
POPs	Persistent organic pollutants
PRB	Policy relevant background
REVEAL	Regional Visibility Experimental Assessment in the Lower Fraser Valley
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
TIME/LTM	Temporally Integrated Monitoring of Ecosystems/Long-Term Monitoring Project
TL	Target load
TMO	Thermal manganese oxidation method
TOR	Thermal/optical reflectance method
TOT	Thermal/optical transmission method
TSD	Technical support document
TSP	Total suspended particulates
μg	micrograms

$\mu g/m^3$	micrograms per cubic meter
UNEP	United Nations Environmental Program
URS	Upper respiratory symptoms
U.S.	United States
UV	Ultraviolet
UV-B	Ultraviolet-B
V_d	Deposition velocity
VOCs	Volatile organic compounds
WMO	World Meteorological Organization
XRF	X-ray fluorescence

1. INTRODUCTION

1.1 PURPOSE

This draft Staff Paper, prepared by staff in the U.S. Environmental Protection Agency's (EPA) Office of Air Quality Planning and Standards (OAQPS), evaluates the policy implications of the key studies and scientific information contained in the document, *Air Quality Criteria for Particulate Matter* (EPA, 2004; henceforth referred to as the Criteria Document (CD) and cited as CD), prepared by EPA's National Center for Environmental Assessment (NCEA). This document, which builds upon an earlier first draft Staff Paper (EPA, 2003), also presents and interprets results from updated and expanded staff analyses (e.g., air quality analyses, human health risk assessments, and visibility analyses) that staff believes should be considered in EPA's current review of the national ambient air quality standards (NAAQS) for particulate matter (PM). This draft Staff Paper presents provisional staff conclusions and recommendations as to potential revisions of the primary (health-based) and secondary (welfare-based) PM NAAQS, based on consideration of the available scientific information and analyses and related limitations and uncertainties. The final version of this document will be informed by comments received through an independent scientific review and public comments on this draft document.

The policy assessment presented in this document 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, and if so, how, it is appropriate to revise the NAAQS for PM. This assessment focuses on the basic elements of PM air quality standards: indicators, averaging times, forms[1], and levels. These elements, which serve to define each standard within the suite of PM NAAQS, must be considered collectively in evaluating the health and welfare protection afforded by the standards.

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.

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[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.

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]

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

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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."

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, 1186 (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), a standing committee of EPA's Science Advisory Board.

1.2.2 History of PM NAAQS Reviews

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.

EPA first established national ambient air quality standards for PM in 1971, based on the original criteria document (DHEW, 1969). 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 including particles with a mean aerodynamic diameter[4] less than or equal to 10 μm, which delineates that subset of inhalable particles small enough to penetrate to the thoracic region (including the tracheobronchial and alveolar regions) of the respiratory tract (referred to as thoracic particles). EPA also revised the level and form of the primary standards by: (1) replacing the 24-hour TSP standard with a 24-hour PM_{10} standard of 150 $\mu g/m^3$ with no more than one expected exceedance per year; and (2) replacing the annual TSP standard with a PM_{10} standard of 50 $\mu g/m^3$, annual arithmetic mean. The secondary standard was revised by

[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.

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 April 1994, EPA announced its plans for the second periodic review of the criteria and NAAQS for PM, 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 nominal 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 the coarse fraction of PM_{10} (referred to as thoracic coarse particles or coarse-fraction particles; generally including particles with a nominal mean aerodynamic diameter greater than 2.5 μm and less than or equal to 10 μm, or $PM_{10-2.5}$). 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 98th 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 thoracic coarse particles, the annual PM_{10} standard was retained, while the 24-hour PM_{10} standard was revised to be based on the 99th percentile of 24-hour PM_{10} concentrations at each monitor in an area. EPA revised the secondary standards by making them identical in all respects to the primary standards.

1.2.3 Litigation Related to the 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 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. Pursuant to the court's decision, EPA deleted 40 CFR section 50.6 (d), the regulatory provision controlling the transition from the pre-existing 1987 PM_{10} standards to the 1997 PM_{10} standards (65 FR 80776, December 22, 2000). The pre-existing 1987 PM_{10} standards remained in place. Id. at 80777. In the current review, EPA is addressing thoracic coarse particles in part by considering standards based on an indicator of $PM_{10-2.5}$.

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 the constitutional and cost 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

elements of the PM standards under review. While the CD focuses on new scientific information available since the last criteria review, it appropriately integrates that information with scientific criteria from previous reviews. The quantitative analyses presented herein (and described in more detail in a number of technical support documents) are based on the most recently available air quality information, so as to provide current characterizations of PM air quality patterns, estimated human health risks related to exposure to ambient PM, and PM-related visibility impairment.

Partly as a consequence of EPA's decision in the last review to consider fine particles and thoracic coarse particles separately, much new information is now available on PM air quality and human health effects directly in terms of $PM_{2.5}$ and, to a much more limited degree, $PM_{10-2.5}$. This information adds to the body of evidence on PM_{10} that has continued to grow since the introduction of that indicator in the first PM NAAQS review. Since the purpose of this review is to evaluate the adequacy of the current standards that separately address fine and thoracic coarse particles, staff has focused this policy assessment and associated quantitative analyses primarily on the evidence related directly to $PM_{2.5}$ and $PM_{10-2.5}$. In so doing, staff has considered PM_{10}-related evidence primarily to help inform our understanding of key issues and to help interpret and provide context for the more limited $PM_{2.5}$ and $PM_{10-2.5}$ evidence.

Following this introductory chapter, this draft Staff Paper is organized into three main parts: the characterization of ambient PM; PM-related health effects and primary PM NAAQS; and PM-related welfare effects and secondary PM NAAQS. The characterization of ambient PM is presented in Chapter 2, which focuses on properties of ambient PM, measurement methods, spatial and temporal patterns in ambient PM concentrations, PM background levels, and ambient PM relationships with human exposure and with visibility impairment. Thus, Chapter 2 provides information relevant to both the health and welfare assessments in the other two main parts of this document.

Chapters 3 through 5 comprise the second main part of this draft Staff Paper dealing with human health and primary standards. Chapter 3 presents a policy-relevant assessment of PM health effects evidence, including an overview of the evidence, key human health-related conclusions from the CD, and an examination of issues related to the quantitative assessment of

the epidemiologic health evidence. Chapter 4 presents a quantitative assessment of PM-related health risks, including risk estimates for current air quality levels as well as those associated with just meeting the current NAAQS and various alternative standards that might be considered in this review. Chapter 5 presents the staff review of the current primary standards for fine and thoracic coarse particles. This chapter begins with a discussion of the broader approach used by staff in this review of the primary PM NAAQS than in the last review, generally reflecting both evidence-based and quantitative risk-based considerations. This review includes consideration of the adequacy of the current standards, conclusions as to alternative indicators, averaging times, levels and forms, and provisional recommendations on ranges of alternative primary standards for consideration by the Administrator.

Chapters 6 and 7 comprise the third main part of this draft Staff Paper dealing with welfare effects and secondary standards. Chapter 6 presents a policy-relevant assessment of PM welfare effects evidence, including evidence related to visibility impairment as well as to effects on vegetation and ecosystems, climate change processes, and man-made materials. This chapter's emphasis is on visibility impairment, reflecting the availability of a significant amount of policy-relevant information and staff analyses which serve as the basis for staff consideration of a secondary standard specifically for visibility protection. Chapter 7 presents the staff review of the current secondary standards, beginning with a discussion of the approach used by staff in this review of the secondary PM NAAQS. This review includes consideration of the adequacy of the current standards, conclusions as to alternative indicators, averaging times, levels and forms, and provisional recommendations on ranges of alternative secondary standards for consideration by the Administrator.

The staff conclusions and recommendations presented herein are provisional; final staff conclusions and recommendations, to be presented in the final version of this document, will be informed by comments received from CASAC and the public in their reviews of this draft document.

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2. CHARACTERIZATION OF AMBIENT PM

2.1 INTRODUCTION

This chapter generally characterizes various classes of ambient PM in terms of physical and chemical properties, measurement methods, recent concentrations and trends, and relationships with human exposure and visibility impairment. This information is useful for interpreting the available health and welfare effects information, and for making recommendations on appropriate indicators for primary and secondary PM standards. The information presented in this chapter was drawn from the CD and additional analyses of data from various PM monitoring networks.

Section 2.2 presents information on the basic physical and chemical properties of classes of PM. Section 2.3 presents information on the methods used to measure ambient PM and some important considerations in the design of these methods. Section 2.4 presents data on PM concentrations, trends, and spatial patterns in the U.S. Section 2.5 provides information on the temporal variability of PM. Much of the information in Sections 2.4 and 2.5 is derived from analyses of data collected by the nationwide networks of $PM_{2.5}$ and PM_{10} monitors through 2003. Section 2.6 defines and discusses background levels of ambient PM. Section 2.7 addresses the relationships between ambient PM levels and human exposure to PM. Section 2.8 addresses the relationship between ambient $PM_{2.5}$ levels and visibility impairment. An appendix to this chapter (Appendix 2-A) discusses sources of ambient PM and provides a summary of national estimates of source emissions.

2.2 PROPERTIES OF AMBIENT PM

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 characterized by size, formation mechanism, origin, chemical composition, and atmospheric behavior. This section generally focuses on size since classes of particles have historically been characterized largely in that manner. Fine particles and coarse particles, which are defined in Section 2.2.1.1, are relatively distinct entities with fundamentally different sources and formation processes, chemical composition, atmospheric residence times and behaviors, transport distances, and

optical and radiative properties. The CD concludes that these differences justify consideration of fine and coarse particles as separate subclasses of PM pollution (CD, pp. 2-111 and 9-21).

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 and spatial distribution of particles in ambient air, key considerations 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 influenced by 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 specific PM sampling devices. The terminology used in this Staff Paper for describing these classifications is summarized in Table 2-1 and discussed 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	The distribution of particles that are mostly larger than the intermodal minimum in volume or mass distributions; also referred to as coarse-mode particles. This intermodal minimum generally occurs between 1 and 3 μm.
Thoracic Coarse Particles	A subset of coarse particles that includes particles that can be inhaled and penetrate to the thoracic region (i.e., the tracheobronchial and the gas-exchange regions) of the lung. This subset includes the smaller coarse particles, ranging in size up to those with a nominal aerodynamic diameter less than or equal to 10 microns.
Fine Particles	The distribution of particles that are mostly 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.
Ultrafine Particles	A subset of fine particles with diameters below about 0.1 μm, encompassing the Aitkin and nucleation modes.
Aitkin-Mode Particles	A subset of ultrafine particles with diameters between about 0.01 and 0.1 μm.
Nucleation-Mode Particles	Freshly formed particles with diameters below about 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 a subset of coarse particles, and is an indicator for particles that can be inhaled and penetrate to the thoracic region of the lung; also referred to as thoracic particles.
$PM_{2.5}$	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. This measurement, which generally includes all fine particles, is an indiator for fine particles; also referred to as fine-fraction particles. A small portion of coarse particles may be included depending on the sharpness of the sampler efficiency curve.
$PM_{10-2.5}$	Particles measured directly using a dichotomous sampler or by 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 coarse fraction of thoracic particles; also referred to as thoracic coarse particles or coarse-fraction 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. 2-25). The distribution of particles that are mostly larger than this minimum make up the coarse mode and 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] Ultrafine particles are apparent as the largest peak in the number distribution in panel (a), and are 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).

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 50 percent of particles of

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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 times density.

[4] Whitby (1978) did not identify multiple ultrafine particle modes between 0.01 and 0.1 μm, and therefore separate nucleation and Aitkin modes are not illustrated in Figure 2-1. See CD Figure 2-6 for a depiction of all particle modes.

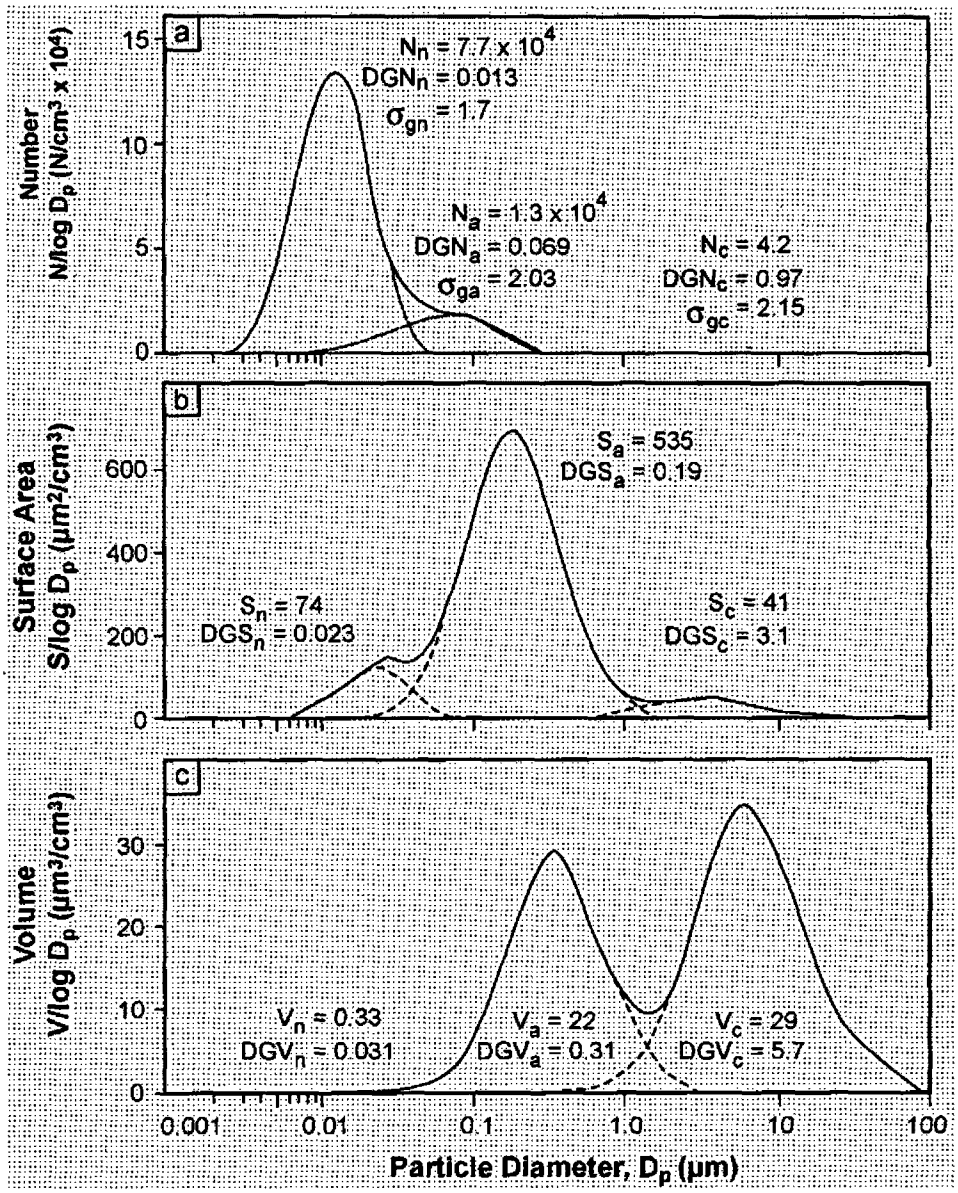

Figure 2-1. Distribution of coarse (c), accumulation (a), and nuclei (n) mode particles by three characteristics: (a) number, N; (b) surface area, S; and (c) volume, V for the grand average continental size distribution. DGV = geometric mean diameter by volume; DGS = geometric mean diameter by surface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

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

a specified diameter are captured by the inlet, and their penetration efficiency as a function of particle size. The usual notation for these classifications is "PM_x", where x refers to measurements with a 50 percent cut point of x μm aerodynamic diameter. Because of the overlap in the size distributions of fine and coarse-mode ambient particles, and the fact that inlets do not have perfectly sharp cut points, no single sampler can completely separate them. Given a specific size cut, the smaller the particles the greater the percentage of particles that are captured. The objective of size-selective sampling is usually to measure particle size fractions that provide a 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 attention on particles small enough to be inhaled and to penetrate into the thoracic region of the human respiratory tract. In 1997, EPA established standards for fine particles measured as $PM_{2.5}$ (i.e., the fine fraction of PM_{10}). 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 particles, and the distribution captured by the $PM_{2.5}$ FRM sampler[7], including generally all fine particles and potentially capturing a small subset of coarse particles.

The EPA is now considering establishing standards for another PM indicator identified in Table 2-1 as $PM_{10-2.5}$, which represents the subset of coarse particles small enough to be inhaled and to penetrate into the thoracic region of the respiratory tract (i.e., the coarse fraction of PM_{10}, or thoracic coarse particles). Section 2.3 discusses measurement methods for this indicator.

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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.

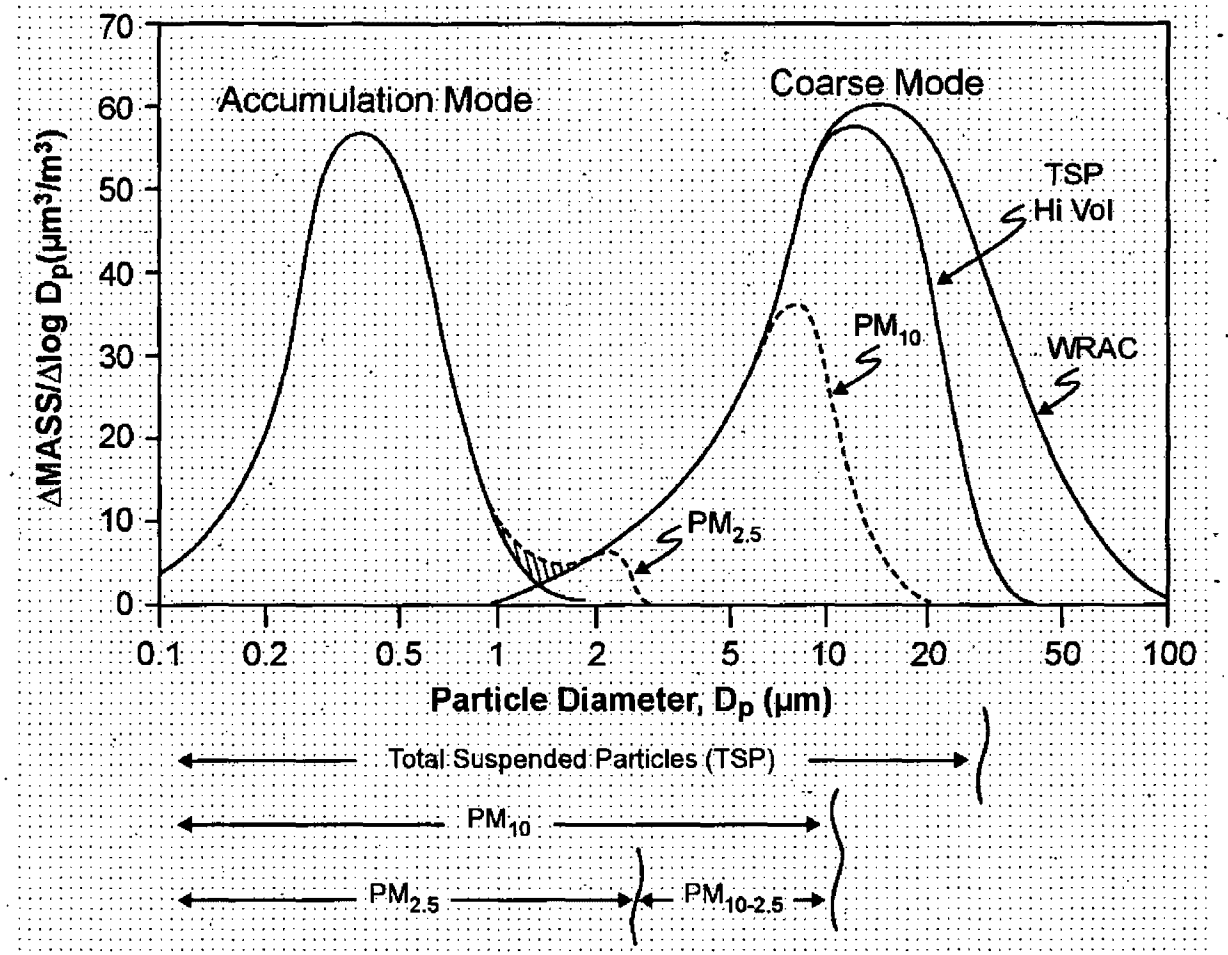

Figure 2-2. An idealized distribution of ambient PM showing fine and coarse particles and the fractions collected by size-selective samplers. (WRAC is the Wide Range Aerosol Classifier which collects the entire coarse mode).

Source: Adapted from Wilson and Suh (1997) and Whitby (1978); CD page 2-18

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 as coarse particles.

By contrast, a significant amount of fine particles are produced through combustion processes and atmospheric chemistry reactions. 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 and off-highway equipment, power generation facilities, industrial facilities, residential wood burning, agricultural burning, and forest fires.

The formation and growth of fine particles are influenced by several processes including: (1) nucleation (i.e., gas molecules coming together to form a new particle); (2) condensation of gases onto existing particles; (3) coagulation of particles, the weak bonding of two or more particles into one larger particle; (4) hygroscopic uptake of water; and (5) gas phase reactions which form secondary PM. 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 coarse particles (CD, p. 2-29).

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) vapor that reacts further with ammonia to form ammonium nitrate (NH_4NO_3) particles; and (3) reactions involving volatile organic compounds (VOC) yielding organic compounds with low ambient temperature (saturation) vapor pressures that nucleate or condense on existing particles to form secondary organic aerosol particles (CD, p. 3-65 to 3-71). In most of the ambient monitoring data displays shown later in this chapter, the first two types of secondary PM are generally labeled plurally as 'sulfates' and 'nitrates' (respectively), which implies that the ammonium content is encompassed. The third type of secondary PM may be lumped with the directly emitted elemental carbon particles and labeled 'total carbonaceous mass,' or the two types of carbonaceous PM may be reported separately as elemental carbon (EC) and organic carbon (OC).

2.2.3 Chemical Composition

Based on studies conducted in most parts of the U.S., the CD reports that a number of chemical components of ambient PM are found predominately in fine particles including: sulfate, ammonium, and hydrogen ions; elemental carbon[8], secondary organic compounds, and primary organic species from cooking and combustion; and certain metals, primarily from combustion processes. Chemical components found predominately in coarse particles include: crustal-related materials such as calcium, aluminum, silicon, magnesium, and iron; and primary organic materials such as pollen, spores, and plant and animal debris (CD, p. 2-38).

Some components, such as nitrate and potassium, may be found in both fine and coarse particles. 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. 2-38). Potassium in coarse particles comes primarily from soil, with additional contributions from sea salt in coastal areas. Potassium in fine particles, generally not a significant contributor to overall mass, comes mainly from emissions of burning wood, with infrequent but large contributions from fireworks, as well as significant proportions from the tail of the distribution of coarse soil particles (i.e., < 2.5 μm in diameter) in areas with high soil concentrations.

[8] Also called light absorbing carbon and black carbon.

Many ambient particles also contain water (i.e., particle-bound water) as a result of an equilibrium between water vapor and hygroscopic PM (CD, p. 2-40). Particle-bound water influences the size of particles and in turn their aerodynamic and light scattering properties (discussed in section 2.2.5). Particle-bound water can also act as a carrier to convey dissolved gases or reactive species into the lungs which, in turn, may cause heath consequences. (CD, p. 2-112). The amount of particle-bound water in ambient particulate matter will vary with the particle composition and the ambient relative humidity. Sulfates, nitrates, and some secondary organic compounds are much more hygroscopic than elemental carbon (BC), primary organic carbon (OC), and crustal material.

2.2.4 Fate and Transport

Fine and coarse particles typically exhibit different behaviors 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 have a very short life, on the order of minutes to hours, since they grow rapidly into the accumulation mode. However, their chemical content persists in the accumulation mode. 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 in falling rain drops. Accumulation-mode particles that are not involved in cloud processes are eventually removed from the atmosphere by gravitational settling and impaction on surfaces.

By contrast, coarse particles can settle rapidly from the atmosphere with lifetimes ranging from minutes to days depending on their size, atmospheric conditions, and their altitude. Larger coarse 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 coarse particles extending into the tail of the distribution can have longer lifetimes and travel longer distances, especially in extreme

circumstances, such as intercontinental dust storms (CD, p. 2-49). Coarse particles also are readily removed by falling rain drops (CD, p. 2-50).

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

2.2.5 Optical Properties of Particles

Particles and gases in the atmosphere scatter and absorb light and, thus, affect visibility. As discussed in section 4.3 of the CD, the efficiency of particles in causing visibility impairment depends on particle size, shape, and composition. Accumulation-mode particles are more efficient per unit mass than coarse particles in causing visibility impairment. The accumulation-mode particle components principally responsible for visibility impairment are sulfates, nitrates, organic matter, and elemental carbon. Soil dust in the fine tail of the coarse particle distribution can also impair visibility. All of these particles scatter light to some degree, but, of these, only elemental carbon (also called light absorbing carbon) plays a significant role in light absorption. Since elemental carbon, which is a product of incomplete combustion from activities such as the burning of wood or diesel fuel, is a relatively small component of PM in most areas, visibility impairment is generally dominated by light scattering rather than by light absorption.

Because humidity causes hygroscopic particles to grow in size, humidity plays a significant role in particle-related visibility 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, dry particle size distribution, and composition would likely cause greater visibility impairment in an eastern versus a western location. The relationship between ambient PM and visibility impairment is discussed below in Section 2.8.

2.2.6 Radiative Properties of Particles

Ambient particles scatter and absorb electromagnetic radiation across the full spectrum, including ultraviolet, visible, and thermal infrared wavelengths, affecting climate processes and the amount of ultraviolet radiation that reaches the earth. As discussed in section 4.5 of the CD, 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

TABLE 2-2. COMPARISON OF AMBIENT PARTICLES, FINE PARTICLES (Ultrafine plus Accumulation-Mode) AND COARSE PARTICLES

	Fine		
	Ultrafine	**Accumulation**	**Coarse**
Formation Processes:	Combustion, high-temperature processes, and atmospheric reactions		Break-up of large solids/droplets
Formed by:	Nucleation Condensation Coagulation	Condensation Coagulation Reactions 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 Elemental carbon Metal compounds Organic compounds with very low saturation vapor pressure at ambient temperature	Sulfate, nitrate, ammonium, and hydrogen ions Elemental carbon Large variety of organic compounds Metals: compounds of Pb, Cd, V, Ni, Cu, Zn, Mn, Fe, etc. Particle-bound water	Suspended soil or street dust Fly ash from uncontrolled combustion of coal, oil, and wood Nitrates/chlorides/sulfates from $HNO_3/HCl/SO_2$ reactions with coarse particles Oxides of crustal elements (Si, Al, Ti, Fe) $CaCO_3$, $CaSO_4$, 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 nonhygroscopic
Sources:	Combustion Atmospheric transformation of SO_2 and some organic compounds High temperature processes	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 processes, smelters, steel mills, etc.	Resuspension of industrial dust and soil tracked onto roads and streets Suspension from disturbed soil (e.g., farming, mining, unpaved roads) Construction and demolition Uncontrolled coal and oil combustion Ocean spray Biological sources
Atmospheric half-life:	Minutes to hours	Days to weeks	Minutes to hours
Removal Processes:	Grows into accumulation mode Diffuses to raindrops	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 (small size tail, 100s to 1000s in dust storms)

Source: Adapted from Wilson and Suh (1997); CD, p. 2-52.

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.

The effects of PM on the transfer of radiation in the visible and infrared spectral regions play a role in global and regional climate. Direct effects of particles on climatic processes 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 elemental 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 elemental 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.

The transmission of solar radiation in the ultraviolet (UV) range through the earth's atmosphere is affected by ozone and clouds as well as by particles. The effect of particles on radiation in the ultraviolet-B (UV-B) range, which has been associated with various biological effects, is of particular interest. Relative to ozone, the effects of ambient particles on the transmission of UV-B radiation are more complex. The 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.

2.3 AMBIENT PM MEASUREMENT METHODS

The methods used to measure ambient PM are important to understanding population exposure to PM, evaluating health and welfare 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.[9] 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).

2.3.1 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. Methods can also be segregated as either discrete or continuous according to whether samples require laboratory analysis or the data are available in real-time. Discrete methods provide time integrated data points (typically over a 24-hour period) that allow for post-sampling gravimetric analyses in the laboratory. These methods are typically directly linked to the historical data sets that have been used in health studies that provide the underlying basis for having a NAAQS. Continuous methods can provide time resolution on the order of minutes and automated operation up to several weeks, facilitating the cost-effective collection of greater amounts of data compared with discrete methods.

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 before and after collection to determine mass by difference. Examples include the FRM monitors for $PM_{2.5}$ and PM_{10}. Dichotomous samplers contain a separator that splits the air stream

[9] Refer to 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).

from a PM_{10} inlet into two streams so that both fine- and coarse-fraction particles can be collected on separate filters. These gravimetric methods require weighing the filters after they are subjected to specific equilibrium conditions (e.g., 22° C, 35 percent RH).

Discrete, gravimetric methodologies have been refined over the past 20 years as PM monitoring networks have evolved from sampling based on the high volume TSP and PM_{10} method to the $PM_{2.5}$ FRM. The inclusion of such measures as size-selective inlets and separators, highly specific filter media performance criteria, active flow control to account for ambient changes in temperature and pressure, and highly prescriptive filter weighing criteria have reduced levels of measurement uncertainty, compared with earlier methods.

National quality assurance data analyzed by EPA between 1999-2001 indicate that the $PM_{2.5}$ FRM has been a robust indicator of ambient levels by meeting the data quality objectives (DQO) established at the beginning of the monitoring program. Three-year average estimates from reporting organizations aggregated on a national basis for collocated sampler precision (7.2 percent), flow rate accuracy (0.18 percent), and method bias (-2.06 percent, from the Performance Evaluation Program)[10] are well within their respective goals of $\pm$10 percent, $\pm$4 percent, and $\pm$10 percent.

There are a number of continuous PM measurement techniques. A commonly 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).

The next generation of the TEOM® is the Filter Dynamics Measurement System (FDMS® monitor). This method is based upon the differential TEOM that is described in the CD (CD, p. 2-78). The FDMS method employs an equilibration system integrated with a TEOM® having alternating measurements of ambient air and filtered air. This self-referencing approach allows the method to determine the amount of volatile PM that is evaporating from the TEOM sensor for 6 of every 12 minutes of operation. An hourly measurement of the total aerosol mass

[10] The Performance Evaluation Program (PEP) is designed to determine total bias for the $PM_{2.5}$ sample collection and laboratory analysis processes. Federally referenced audit samplers are collocated adjacent to a monitoring site's routine sampler and run for a 24-hour period. The concentrations are then determined independently by EPA laboratories and compared in order to assess bias. The performance evaluations are conducted four times per year (once per quarter) at one-fourth (25 percent) of the sampling sites in a reporting organization.

concentration, including non-volatile and volatile PM, is calculated and reported every 6 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.

2.3.2 Particle Indirect Optical Methods

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.[11] 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 collecting samples from a 4.5 μm inlet onto white filter paper where blackness of the stain is measured by light absorption. Smoke particles composed primarily of elemental carbon (EC), including black carbon (BC), typically make the largest contribution to stain darkness. CoH is determined using a light transmittance method. This involves collecting 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 widely by location and time of year, the correlation between BS, COH, and nephelometer measurements and PM mass is highly site- and time-specific. The optical methods described here, as well as the particle counters described below, are based on the measurement of properties

[11] See Section 2.2.5 of this chapter for a discussion of the optical properties of PM.

such as light scattering and electric mobility, which are inherently different than previous methods described based on aerodynamic diameter.

2.3.3 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.

2.3.4 Chemical Composition Measurement Methods

There are a variety of methods used to identify and describe the characteristic components of ambient PM.[12] 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 elemental carbon (EC) or black carbon (BC) in ambient filter samples. Thermal optical reflectance (TOR), thermal manganese oxidation (TMO), and thermal optical transmittance (TOT) have been commonly applied in aerosol studies in the United States. The thermal optical transmission (TOT) method, used in the EPA speciation program, uses a different temperature profile than TOR, which is used in the Interagency Monitoring of Protected Visual Environments (IMPROVE) visibility

[12] The reader is referred to Chapter 2, section 2.2, of the 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).

monitoring program. The two methods yield comparable estimates of total carbon, but give a different split between OC and EC.

Commercial instruments are now available to measure carbon (OC, EC, TC); nitrate; and sulfate on a near-continuous basis. These instruments provide time-resolved measurements from a few minutes to a few hours. The semi-continuous methods involved a variety of techniques that include thermal reduction; wet impaction and flash vaporization; and thermal oxidation with non-dispersive infrared (NDIR) detection. They have been field tested and compared through the EPA's Environmental Technology Verification (ETV) program and the Supersites program and proven to be good candidates for additional testing (EPA, 2004a). Data are now becoming available from regional planning and multi-state organizations and the EPA to understand the comparison with filter-based methods and the inherent limitations of these technologies.

The U.S. EPA is coordinating a pilot study of semi-continuous speciation monitors at five Speciation Trends Network (STN) sites. The pilot study began in 2002. The goals of the pilot study are to assess the operational characteristics and performance of continuous carbon, nitrate, and sulfate monitors for routine application at STN sites; work with the pilot participants and the vendors to improve the measurement technologies used; and evaluate the use of an automated data collection and processing system for real time display and reporting. After the pilot monitoring and data evaluation phase, proven semi-continuous monitors will become the framework for a long-term network of up to 12 STN sites equipped with semi-continuous sulfate, nitrate, and carbon monitors.

2.3.5 Measurement Issues

There is no perfect PM 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 semivolatile 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 percent of total $PM_{2.5}$ mass, as shown in southern California studies (CD, p. 2-68). 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 and conversion of PM components to gas-phase chemicals 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 ambient PM mass under conditions where relative humidity is more than 60 percent (CD; p. 2-63, p. 2-109). It can also represent a substantial fraction of gravimetric mass at normal equilibrium conditions (i.e., 22° C, 35 percent RH) when the aerosol has high sulfate content. 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, although the newer generation TEOM's incorporates less reliance on heat for water management (CD, p. 2-100, Table 2-7).

Particle bounce from the impaction plate 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, thus overstating the mass of fine-fraction particles. The addition of a $PM_{1.0}$ measurement in these circumstances can provide greater insights into the magnitude of this problem (CD, p. 9-12).

2.4 PM CONCENTRATIONS, TRENDS, AND SPATIAL PATTERNS

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 2003, based on data publically available from EPA's Air Quality System (AQS) as of August 2004, are presented here. $PM_{2.5}$ data from the IMPROVE network are also presented. Many data summaries are presented by region, as shown in Figure 2-3. These regions are the same as those defined in the CD and have proven useful for understanding potential differences in the characteristics of PM in different parts of the U.S. As is the case with all surface-based ambient monitoring data, these data can be considered representative of exposures in typical breathing zones in the lowest 15 meters of the atmosphere.

2.4.1 $PM_{2.5}$

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. Over 90 percent of the monitors are located in urban areas. These monitors use the $PM_{2.5}$ FRM which, when its procedures are followed, assures that PM data are collected using standard equipment, operating procedures, and filter handling techniques.[13] Most of these FRM monitors began operation in 1999. The EPA has analyzed the available data collected by this network from 2001-2003. Data from the monitors were screened for completeness with the purpose of avoiding seasonal bias. To be included in these analyses, a monitoring site needed all 12 quarters (2001-2003), each with 11 or more observations. A total of 827 FRM sites in the U.S. met these criteria.[14]

The 3-year average annual $PM_{2.5}$ mean concentrations range from about 4 to 28 $\mu g/m^3$, with a median of about 13 $\mu g/m^3$. The 3-year average annual 98th percentiles of the 24-hour average concentrations range from about 9 to 76 $\mu g/m^3$, with a median of about 32 $\mu g/m^3$. Figures 2-4 and 2-5 depict the regional distribution of site-specific 3-year average annual mean and 3-year average 98th percentile 24-hour average $PM_{2.5}$ (and $PM_{10-2.5}$, discussed in section 2.4.3)

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

[14] 810 of the 827 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.

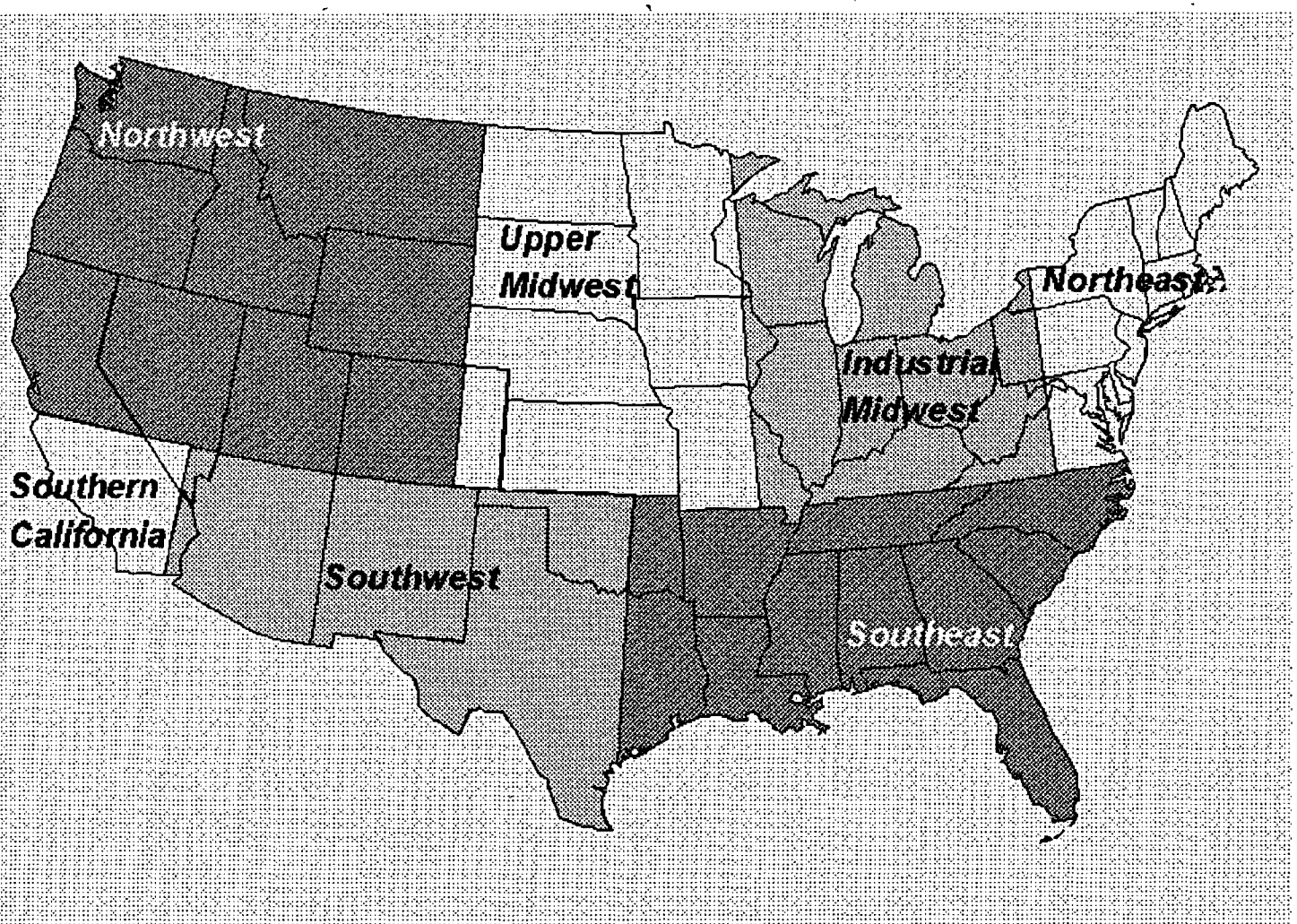

Figure 2-3. Regions used in PM Staff Paper data analyses summaries.

concentrations, respectively, by geographic region (excluding Alaska, Hawaii, Puerto Rico, and the Virgin Islands). In general, with the exception of southern California, $PM_{2.5}$ annual average mass is greater in the eastern regions than in the western regions, whereas $PM_{10-2.5}$ annual average mass is greater in the western regions. Figures 2-6 and 2-7 are national maps that depict county-level 3-year average annual mean and 3-year average annual 98th percentile 24-hour average $PM_{2.5}$ concentrations, respectively, from the FRM network.[15] The site 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 $\mu g/m^3$. Mean $PM_{2.5}$ concentrations were above 18 $\mu g/m^3$ in several urban areas throughout the eastern U.S., including Chicago, Cleveland, Detroit, Indianapolis, 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 3-year average annual mean $PM_{2.5}$ concentrations, most below 12 $\mu g/m^3$. Three-year average annual 98th percentile 24-hour average $PM_{2.5}$ concentrations above 65 $\mu g/m^3$ appear only in California. 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 3-year average 98th percentile $PM_{2.5}$ concentrations were more typically below 40 $\mu g/m^3$, with many below 25 $\mu g/m^3$.

The PM maps shown in this chapter encompass all valid data, including days that were flagged for episodic events, either natural or anthropogenic. Examples of such events include biomass burning, meteorological inversions, dust storms, and volcanic and seismic activity. PM concentrations can increase dramatically with these 'natural' or 'exceptional' events. Although these events are rare (e.g., affecting less than 1 percent of reported $PM_{2.5}$ concentrations between 2001 and 2003), they can affect people's short-term PM exposure, briefly pushing daily PM levels into the unhealthy ranges of the Air Quality Index (AQI). An analyses of 2001-2003 $PM_{2.5}$ data found that over 9 percent of the days above (site-based) 98th percentile 24-hour

[15] Readers are 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. Although 11 samples per quarter, as required in these analyses, is sufficient to show nonattainment, additional data capture (at least 75 percent per quarter) is required to show attainment of the standards. Not all of the PM federal reference method (FRM) sites that contributed data to the summaries presented here recorded 75 percent data capture for all four calendar quarters for each of the 3 years.

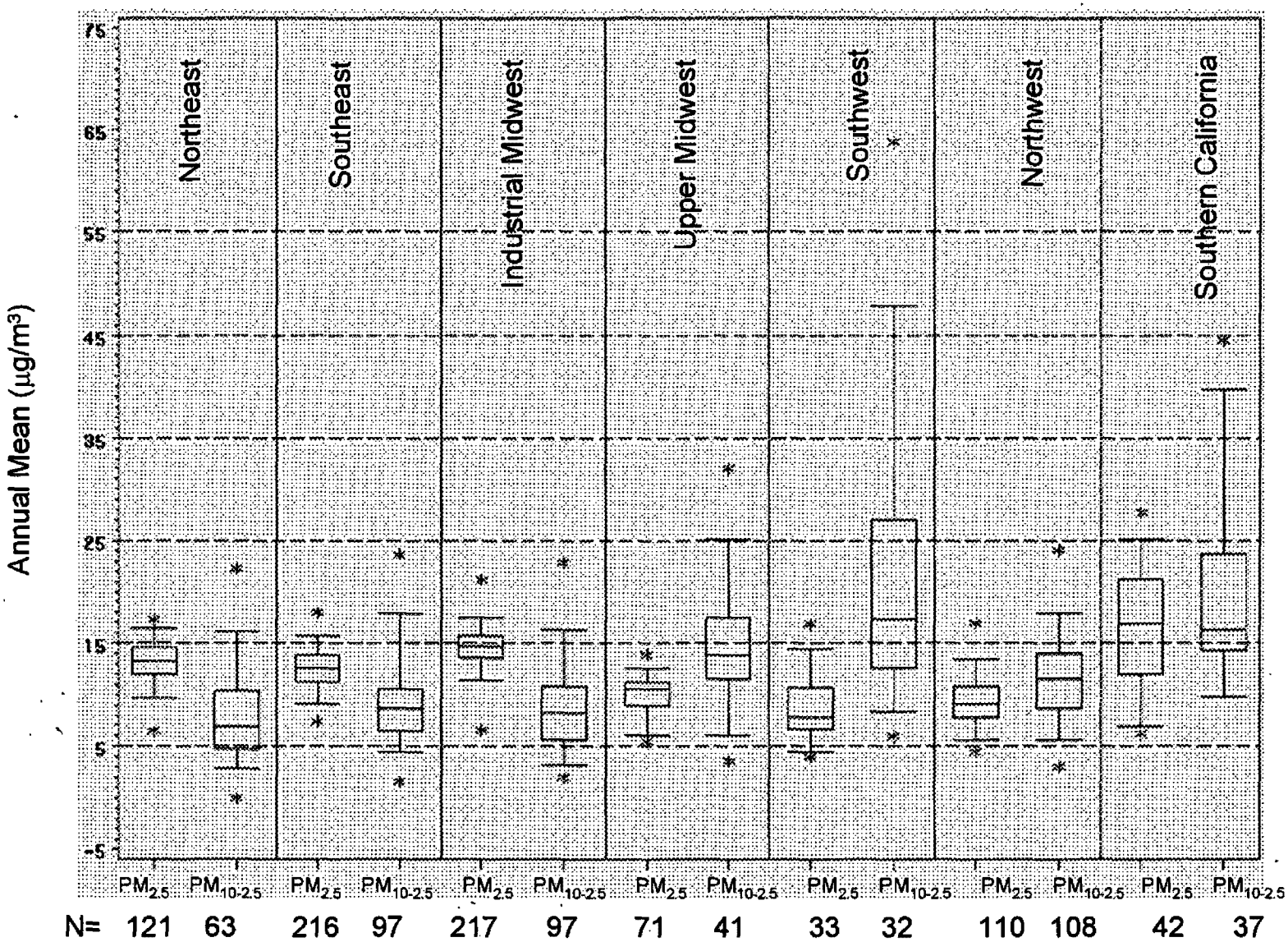

Figure 2-4. Distribution of annual mean $PM_{2.5}$ and estimated annual mean $PM_{10-2.5}$ concentrations by region, 2001-2003. Box depicts interquartile range and median; whiskers depict 5th and 95th percentiles; asterisks depict minima and maxima. N = number of sites.

Source: Schmidt et al. (2005)

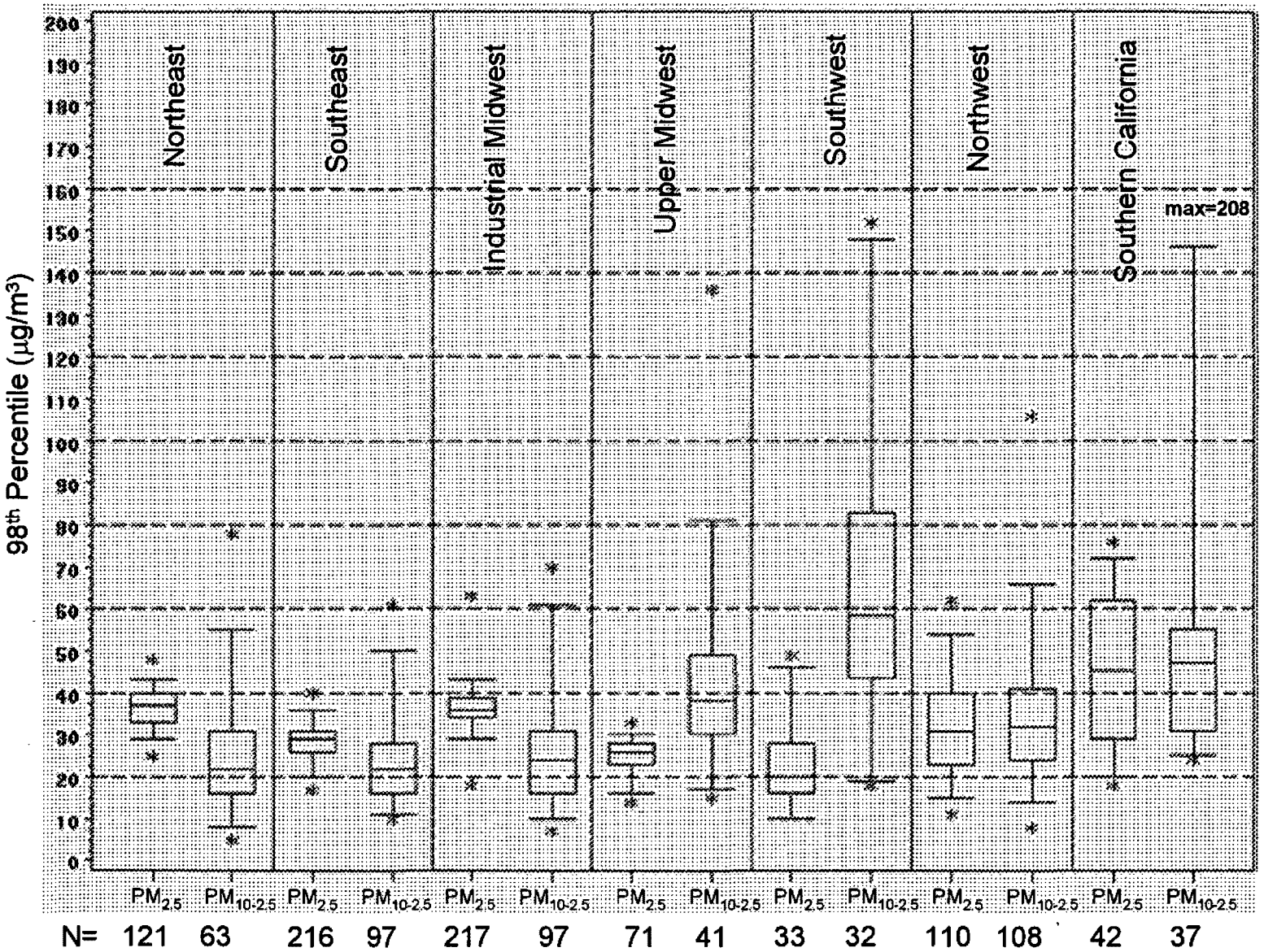

Figure 2-5. Distribution of 98th percentile 24-hour average $PM_{2.5}$ and estimated $PM_{10-2.5}$ concentrations by region, 2001-2003. Box depicts interquartile range and median; whiskers depict 5th and 95th percentiles; asterisks depict minima and maxima. N = number of sites.

Source: Schmidt et al. (2005)

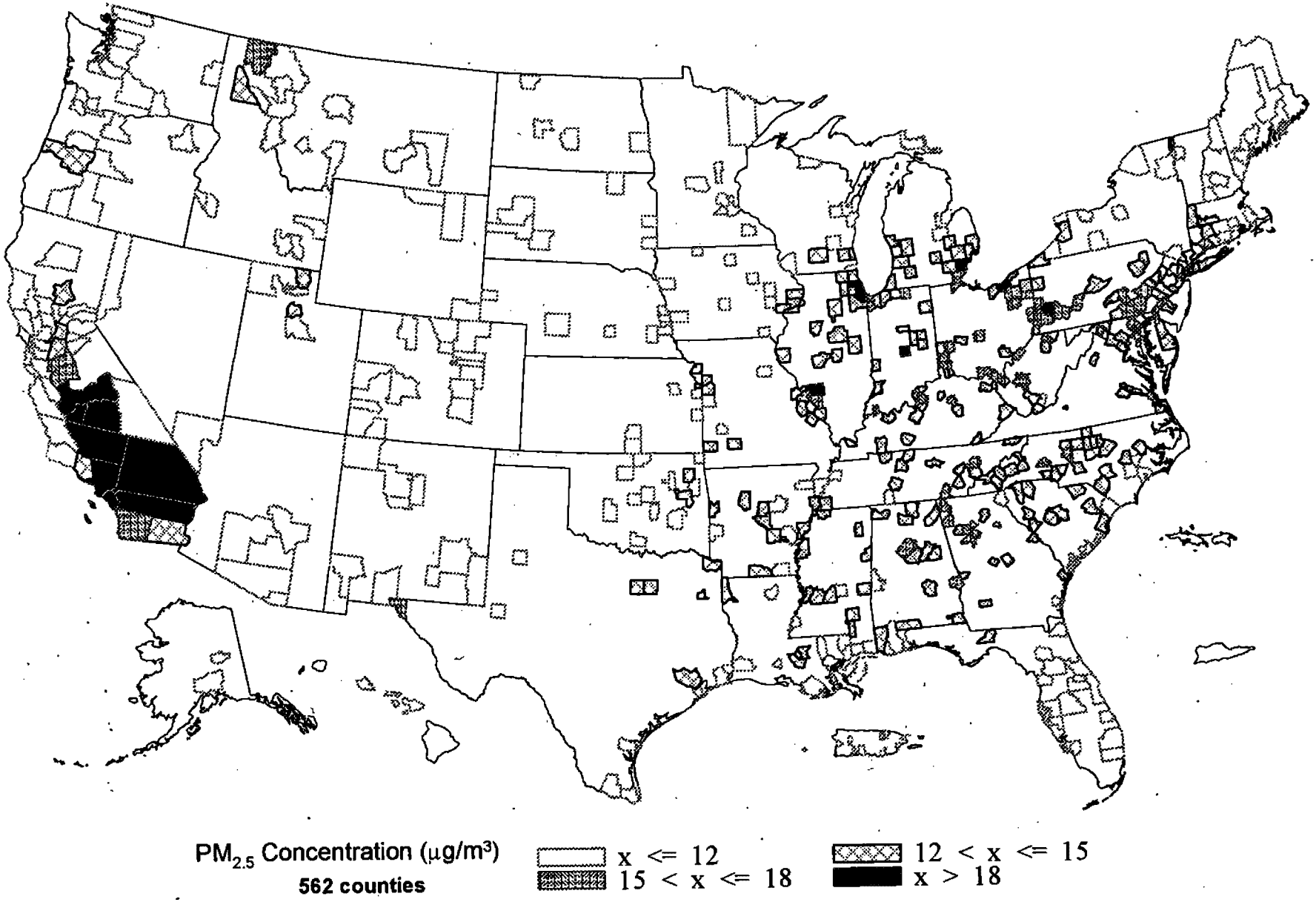

Figure 2-6. County-level maximum annual mean $PM_{2.5}$ concentrations, 2001-2003.

Source: Schmidt et al. (2005)

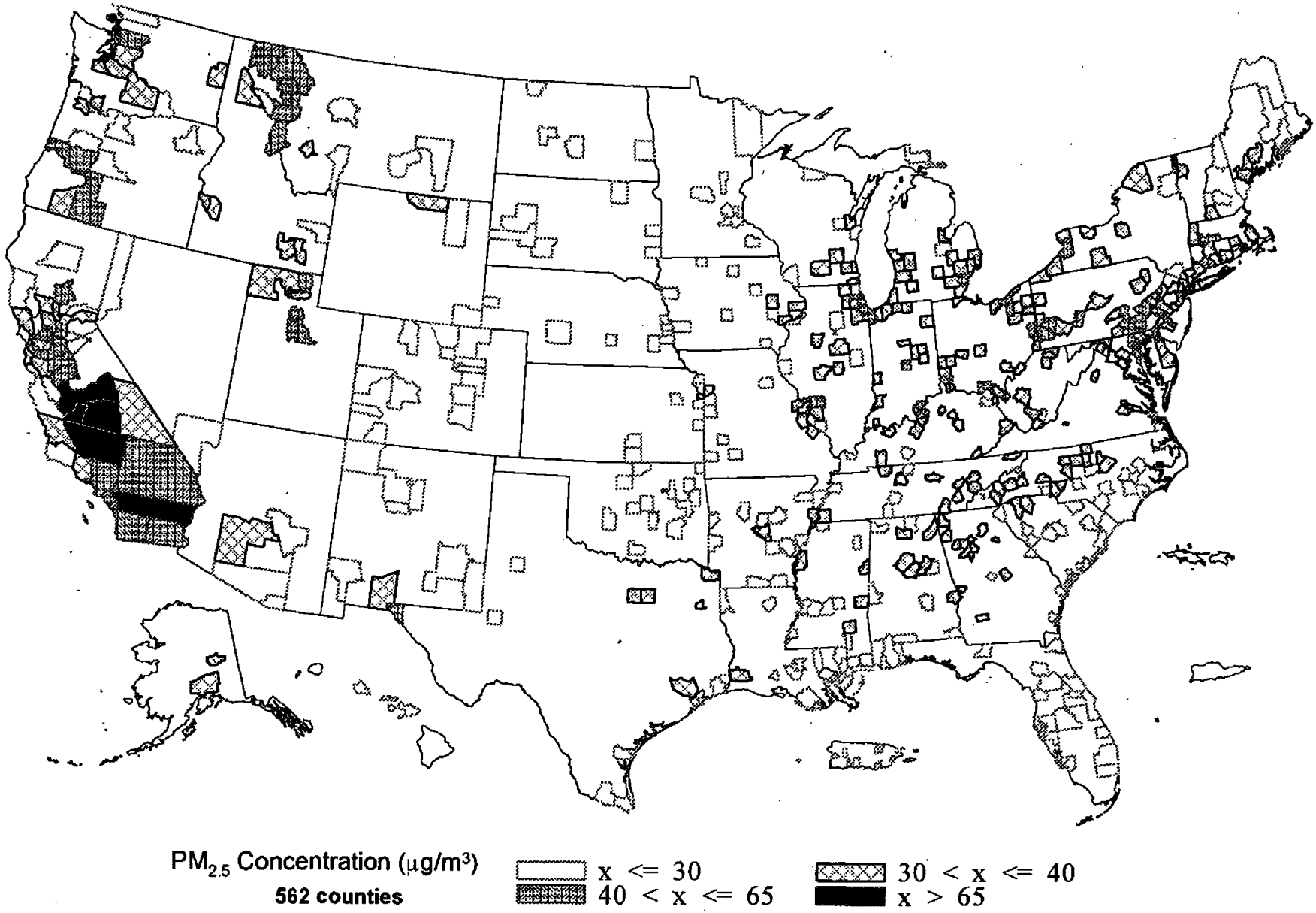

Figure 2-7. County-level maximum 98th percentile 24-hour average $PM_{2.5}$ concentrations, 2001-2003.

Source: Schmidt et al. (2005)

concentrations were flagged for events. The events, in fact, were found to cause the 98th percentiles to inflate by up to 18 $\mu g/m^3$, with an average increase of 0.8 $\mu g/m^3$. Natural and exceptional events, however, rarely have a significant effect on annual or longer averages of PM. In the afore-mentioned analyses of 2001-2003 $PM_{2.5}$ data, the average effect of natural and exceptional events on 3-year annual means was less than 0.1 $\mu g/m^3$ (Schmidt, et al., 2005). Episodic event-flagged data are often excluded from trends-type analyses and are addressed for the purpose of determining compliance with the NAAQS by EPA's national and exceptional events policies, as described below in section 2.6.

$PM_{2.5}$ short-term trends were recently evaluated by EPA in The Particle Pollution Report (EPA, 2004, p. 14). In the EPA FRM network, $PM_{2.5}$ annual average concentrations decreased 10 percent nationally from 1999 to 2003. The Northeast, where moderate concentrations are found, was the only region that did not show a decline between these years; annual concentrations in that region rose about 1 percent over the 5-year period. Except in the Northeast, $PM_{2.5}$ generally decreased the most in the regions with the highest concentrations - the Southeast (20 percent), southern California (16 percent), and the Industrial Midwest (9 percent) from 1999 to 2003. The remaining regions with lower concentrations (the Upper Midwest, the Southwest, and the Northwest) posted modest declines in $PM_{2.5}$; see Figure 2-8 (EPA, 2004, p. 15).

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.[16] Figure 2-9 shows the composite long-term trend at 8 eastern sites, 17 western sites, and one urban site in Washington, D.C. The 4 westmost U.S. subregions (Northwest, southern California, Upper Midwest, and Southwest) are considered the 'west' and the 3 eastern ones (Northeast, Southeast, and Industrial Midwest) are termed the 'east.' At the rural eastern sites, measured $PM_{2.5}$ mass decreased about 23 percent from 1993 to 2003. At the rural western sites $PM_{2.5}$ mass decreased about 21 percent from 1993 to 2003. At the Washington, D.C., site the annual average $PM_{2.5}$ concentration in 2003 was about 31 percent lower than the value in 1993.

The relative spatial homogeneity of the ambient air across a specified area can be assessed by examining the values at multiple sites using several indicators, including: (1) site pair

[16]IMPROVE monitoring instruments and protocols (defined at http://vista.cira.colostate.edu/improve/) are not identical to FRM monitors.

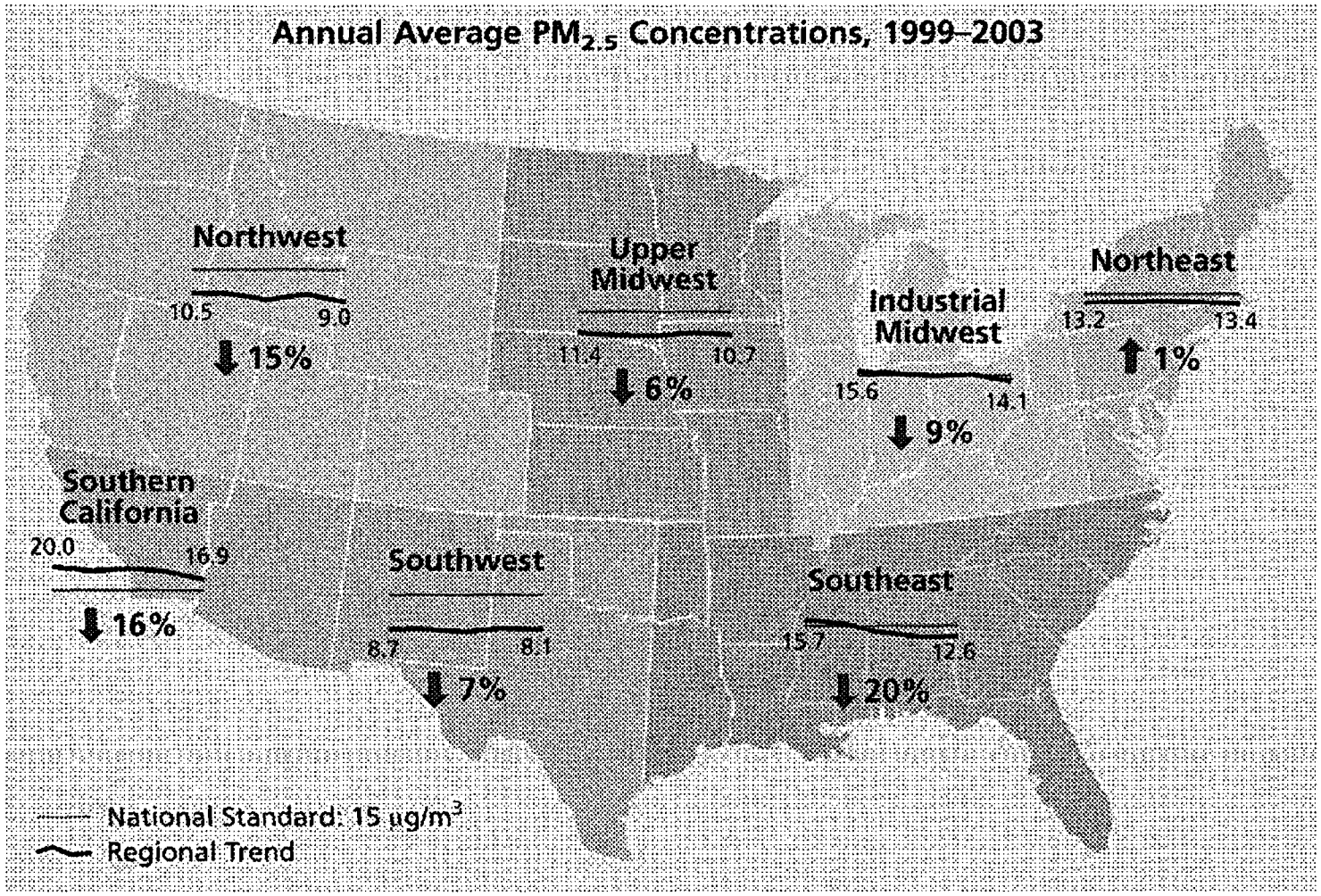

Figure 2-8. Regional trends in annual average PM2.5 concentrations in the EPA network, 1999-2003.

Source: EPA (2004b)

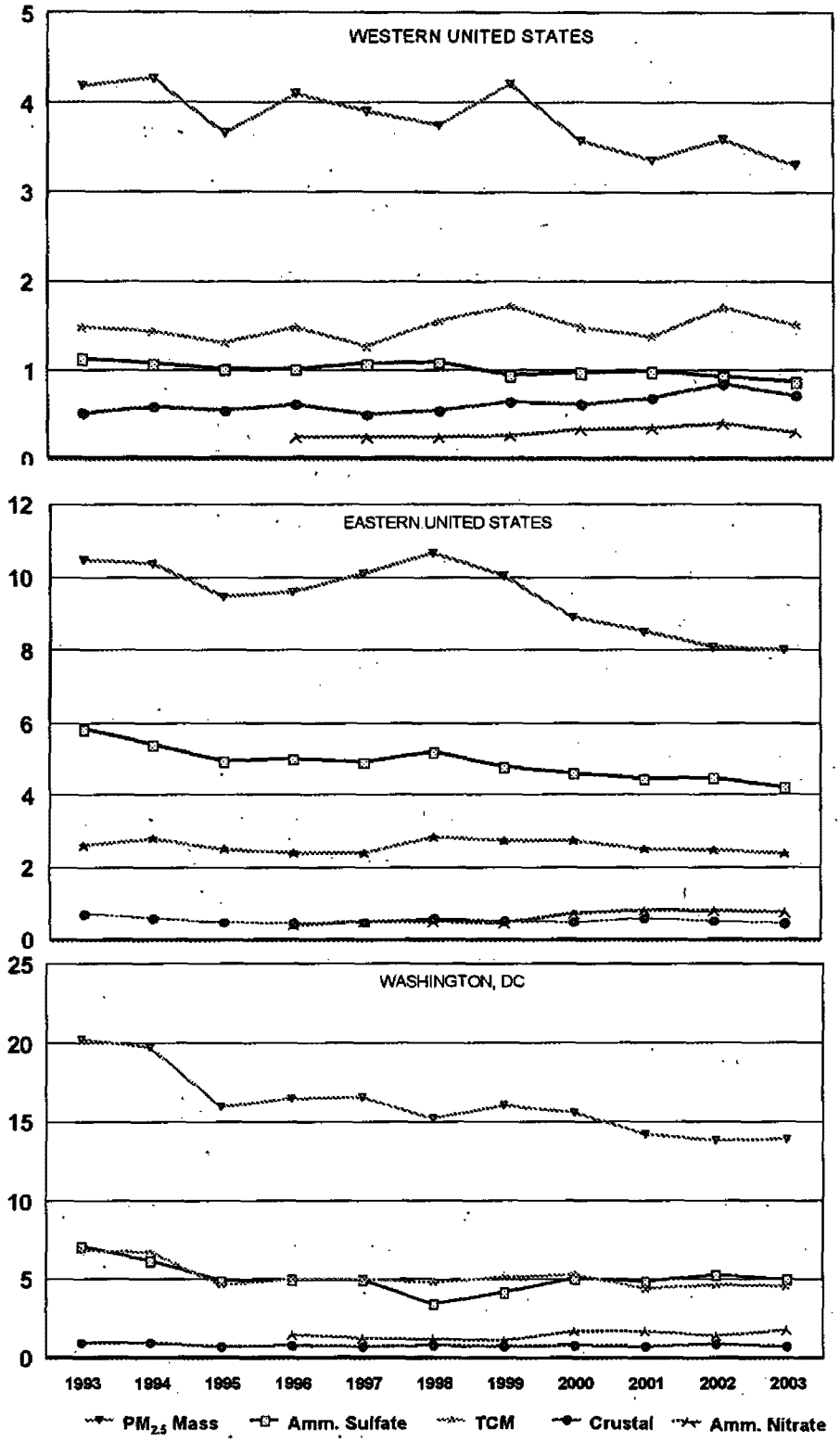

Figure 2-9. Average annual average trend in $PM_{2.5}$ mass, ammonium sulfate, ammonium nitrate, total carbonaceous mass, and crustal material at IMPROVE sites, 1993-2003.

Source: Schmidt et al. (2005)

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 Metropolitan Statistical Areas (MSAs) using $PM_{2.5}$ FRM monitoring data from 1999-2001 is included in the CD (CD, Appendix 3A).

An analysis of site 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 larger, more recent (2001-2003) $PM_{2.5}$ FRM database was similarly analyzed; the median between-site correlation for more than 2,000 site pairs across the nation was about 0.9 (Schmidt, et al., 2005).

A summary of the analyses of long-term and short-term concentration differences for the 27 urban areas is shown in Table 2-3. 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, meteorology, 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 CD also examined differences in 24-hour average concentrations between the urban site pairs. Small differences throughout the distribution would indicate relatively homogeneous concentration levels between the sites. Table 2-3 presents a summary of the 90th percentile of the distribution (P_{90}) of daily site pair differences in each urban area. The site pairs 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 was generally highly correlated ($r_{max} \geq 0.70$), and in some cases was more highly correlated than the sites with the largest annual mean differences.

Table 2-3. 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

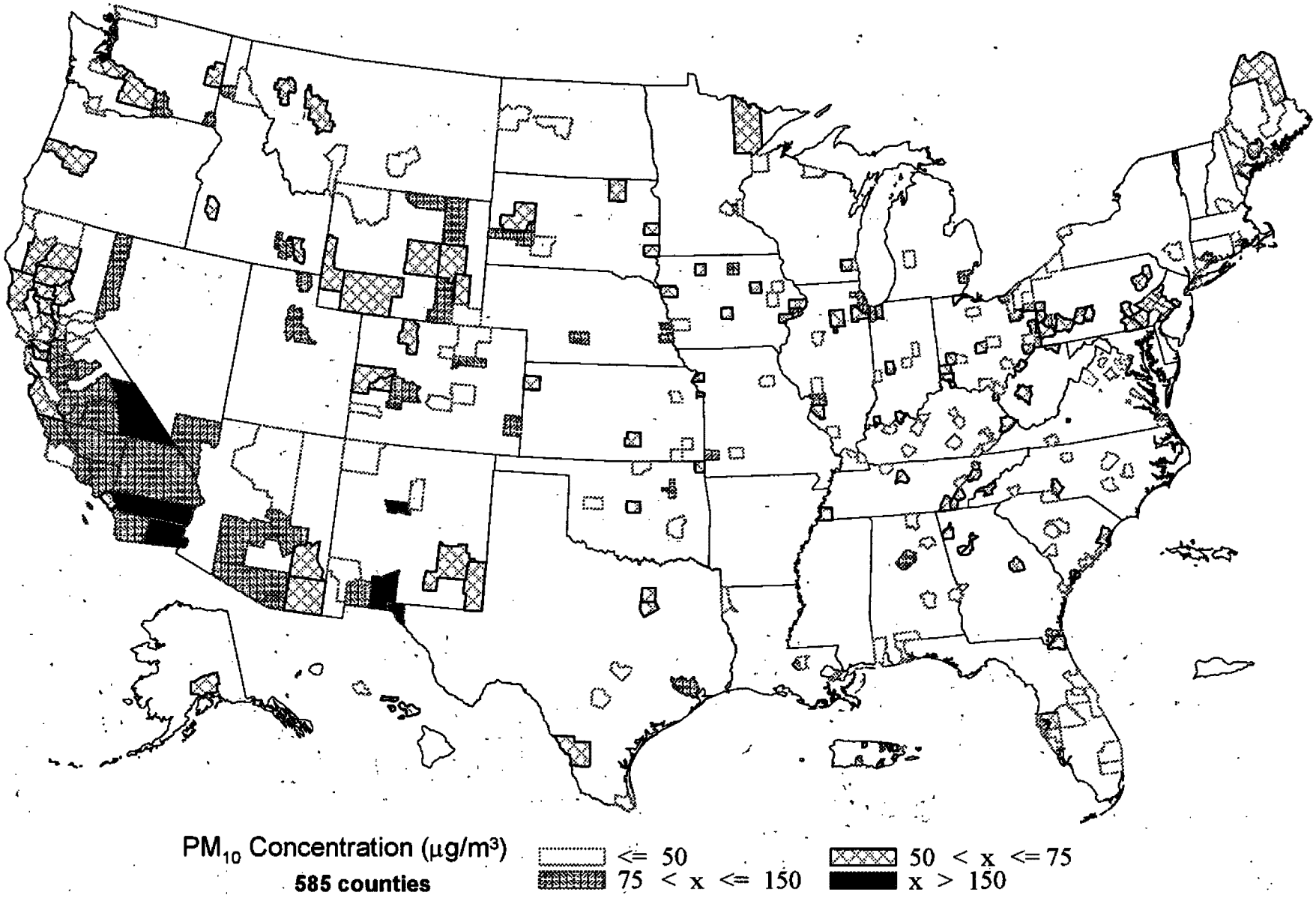

Figure 2-11. County-level maximum 98th percentile 24-hour average PM_{10} concentrations, 2001-2003.

Source: Schmidt et al. (2005)

Since the protocol for each monitor is not usually identical, the consistency of these $PM_{10-2.5}$ measurements is relatively uncertain, and they are referred to as "estimates" in this Staff Paper.[19]

The 98th percentile 24-hour average $PM_{10-2.5}$ concentrations range from about 5 to 208 $\mu g/m^3$, with a median of about 28 $\mu g/m^3$. The box plots in Figures 2-4 and 2-5 (introduced in section 2.4.1) 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 2-12 and 2-13 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 40 $\mu g/m^3$, with one maximum value as high as 64 $\mu g/m^3$ (see Figure 2-4), and with a median of about 10-11 $\mu g/m^3$. 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). As noted before, these maps include days that were flagged for natural or exceptional episodic events. Episodic events can affect $PM_{10-2.5}$ 98th percentiles even more than for $PM_{2.5}$. An evaluation of 2001-2003 $PM_{10-2.5}$ data found that events caused 98th percentiles to be elevated by an average of 2.5 $\mu g/m^3$ (Schmidt, et al., 2005).

The IMPROVE monitoring network provides long-term $PM_{10-2.5}$ trends for generally rural areas. Figure 2-14 presents the composite long-term trend at 7 eastern sites, 17 western sites, and

[19]Note that the urban $PM_{10-2.5}$ estimates derived in this review, labeled '2001-2003', actually represent either the entire 12-quarter period or the most recent consecutive 4- or 8-quarter period (from that 3-year period) with 11 or more samples each. This technique was used to maximize the number of usable sites (and not introduce seasonal bias). Of the 489 total sites, 230 had 12 complete quarters, 122 sites had 8 quarters, and 137 had 4. Similar to $PM_{2.5}$ and PM_{10} processing, 'annual' means and 'annual' 98th percentiles were first constructed from 4-quarter periods, albeit for $PM_{10-2.5}$, not all necessarily from the same calender year. The 4-quarter statistics were then averaged together for the 8- and 12-quarter sites. Hence there is some temporal variability intrinsic in 2001-2003 estimates. The 1-, 2-, or 3-year averages of the 'annual' statistics are subsequently referred to simply as 'annual means' or '98th percentiles'.

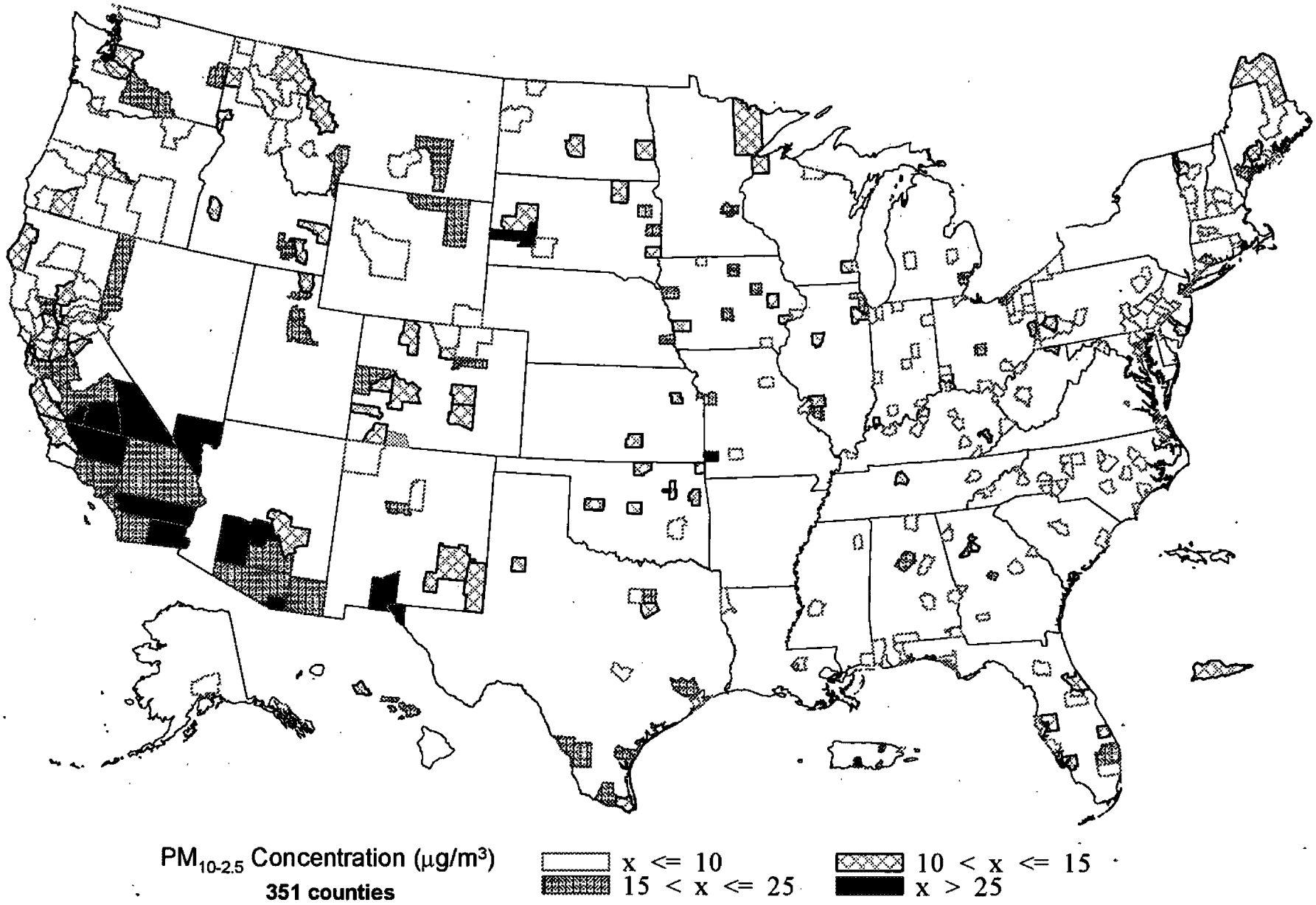

Figure 2-12. Estimated county-level maximum annual mean $PM_{10-2.5}$ concentrations, 2001-2003.

Source: Schmidt et al. (2005)

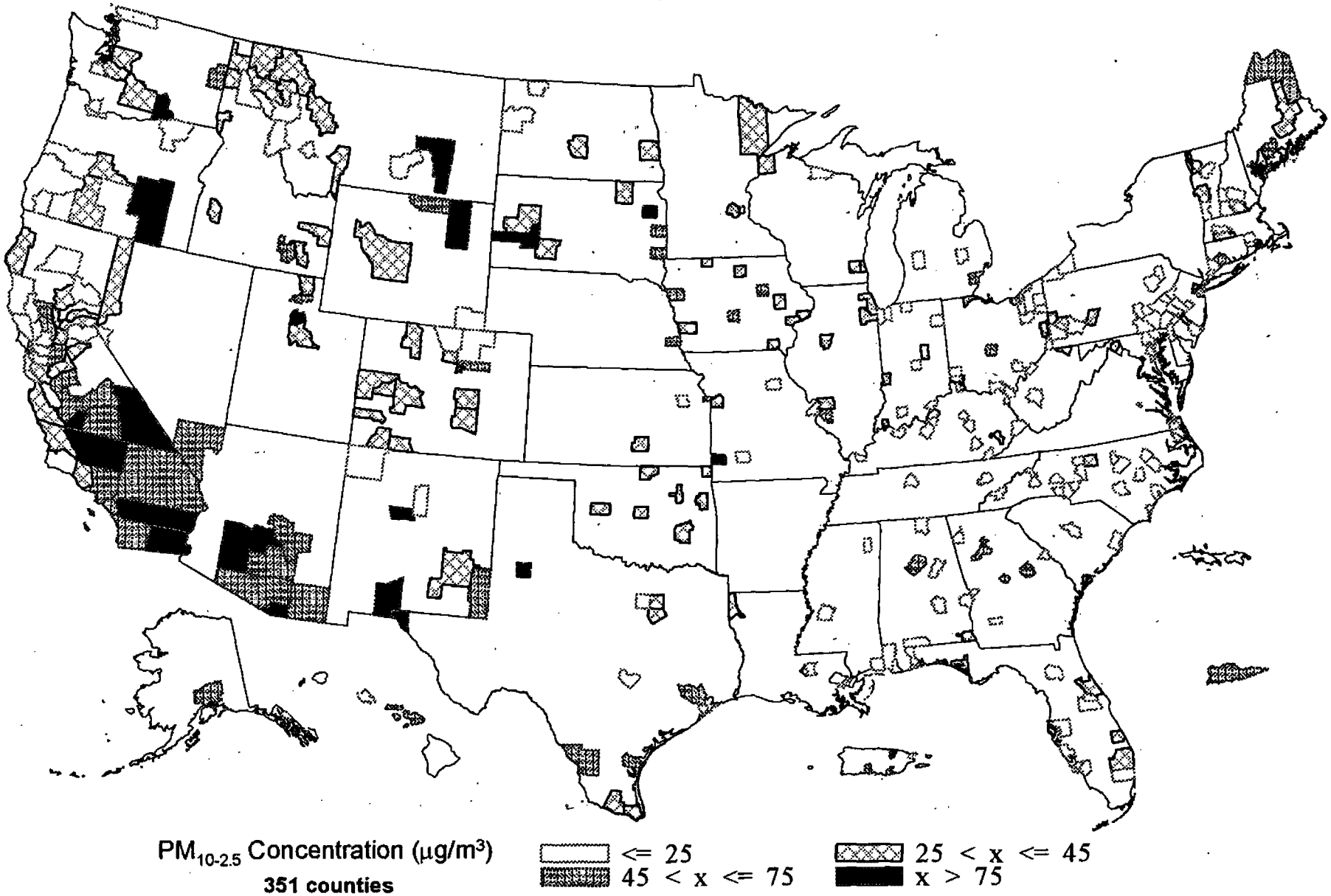

Figure 2-13. Estimated county-level maximum 98th percentile 24-hour average $PM_{10-2.5}$ concentrations, 2001-2003.

Source: Schmidt et al. (2005)

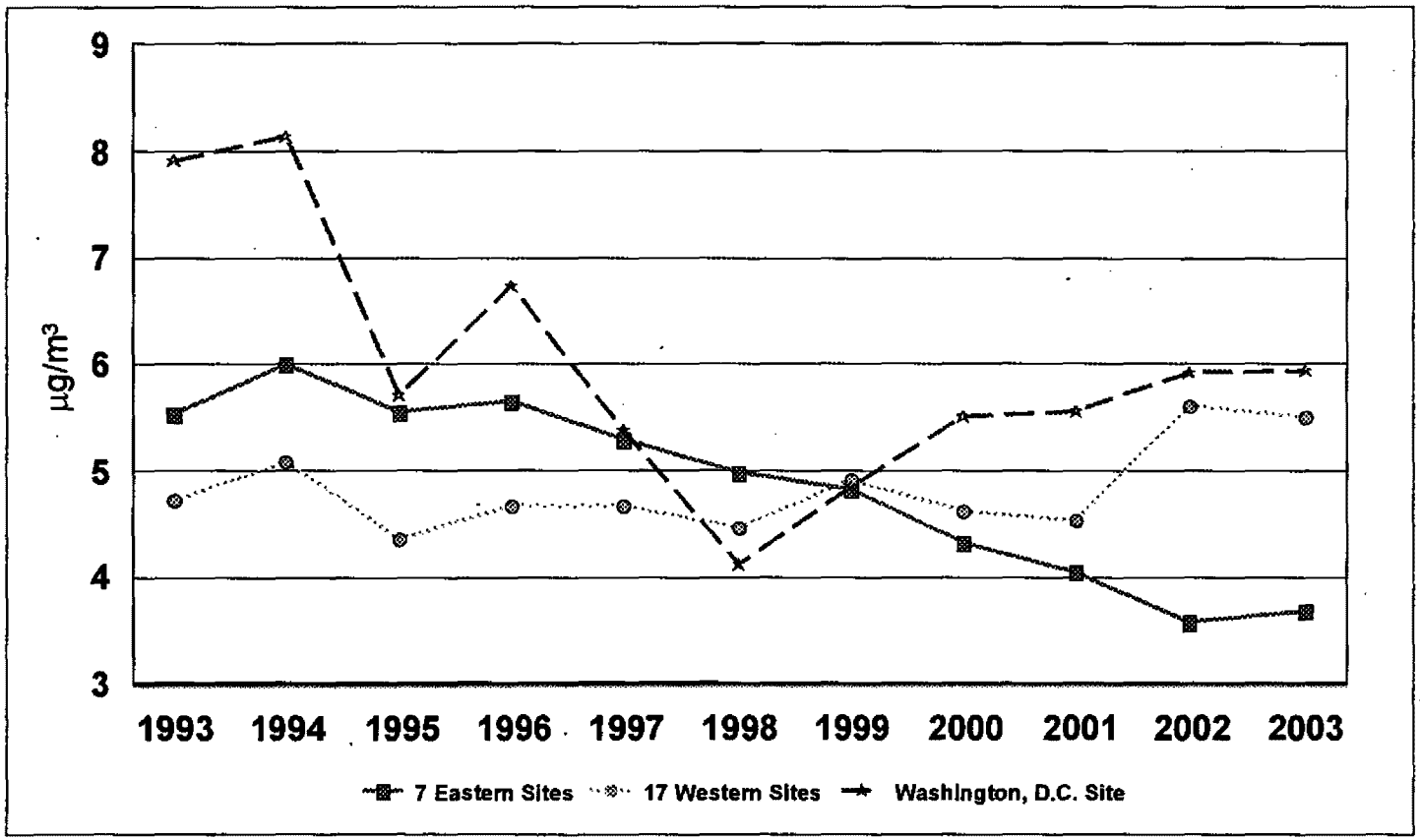

Figure 2-14. Average measured annual average $PM_{10-2.5}$ concentration trend at IMPROVE sites, 1993-2003.

Source: Schmidt et al. (2005)

one urban site in Washington, D.C. At the rural eastern sites, measured $PM_{10-2.5}$ in 2003 was about 33 percent lower then the corresponding value in 1993. At the rural western sites, measured $PM_{10-2.5}$ was about 17 percent higher in 2003 than the corresponding value in 1993. At the Washington, D.C., site, the annual average $PM_{10-2.5}$ concentration in 2003 was about 25 percent lower than the 10-year peak in 1994, but nearly 2 $\mu g/m^3$ higher than the 1998 low point.

The CD contains an analysis of 1999-2001 $PM_{10-2.5}$ estimates in 17 MSAs 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 2.4.1 and summarized earlier in Table 2-4. However, since there were fewer site pairings, fewer urban areas covered, and because of higher uncertainty in daily concentration estimates, 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 2-4. 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 percent (426 of 491) of the pairs for $PM_{2.5}$.

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 2.4.1) 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 analyses indicate that $PM_{10-2.5}$ is more heterogeneous than $PM_{2.5}$ in many 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 $PM_{2.5}$.

2.4.4 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 (CD, p. 2B-21). 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

Table 2-4. Summary of Estimated $PM_{10-2.5}$ Analysis in 17 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}
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

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. Similarly poor correlations between $PM_{2.5}$ mass and number of ultrafine particles were confirmed for sites in Los Angeles and nearby Riverside, CA (Kim et al, 2002). 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 (CD, p. 2-35 to 2-36). Ultrafine PM concentrations were found to decrease exponentially with distance from the roadway source, and were equal to the upwind "background" location at 300 m downwind.

2.4.5 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 2.2, fine and coarse particles have fundamentally different sources and composition. Recent data from the rural IMPROVE network and from the EPA urban speciation network provide indications of regional composition differences for fine particles. Although both programs provide detailed estimates of specific PM chemical components (individual metals, ions, etc.), only gross-level speciation breakouts are shown here. Figure 2-15 shows urban and rural 2003 annual average $PM_{2.5}$ mass apportionment among chemical components averaged over several sites within each of the U.S. regions. In general:

- $PM_{2.5}$ mass is higher in urban areas than in rural areas.
- $PM_{2.5}$ in the eastern U.S. regions is dominated by ammonium sulfate and carbon.
- $PM_{2.5}$ in the western U.S. regions has a greater proportion of carbon.
- Ammonium nitrate is more prevalent in urban aerosols than in rural aerosols, especially in the midwest regions and in southern California.

Though most of the speciation data available are from $PM_{2.5}$, there is a limited amount of data available on speciation profiles for other size fractions as well. One such data source is the

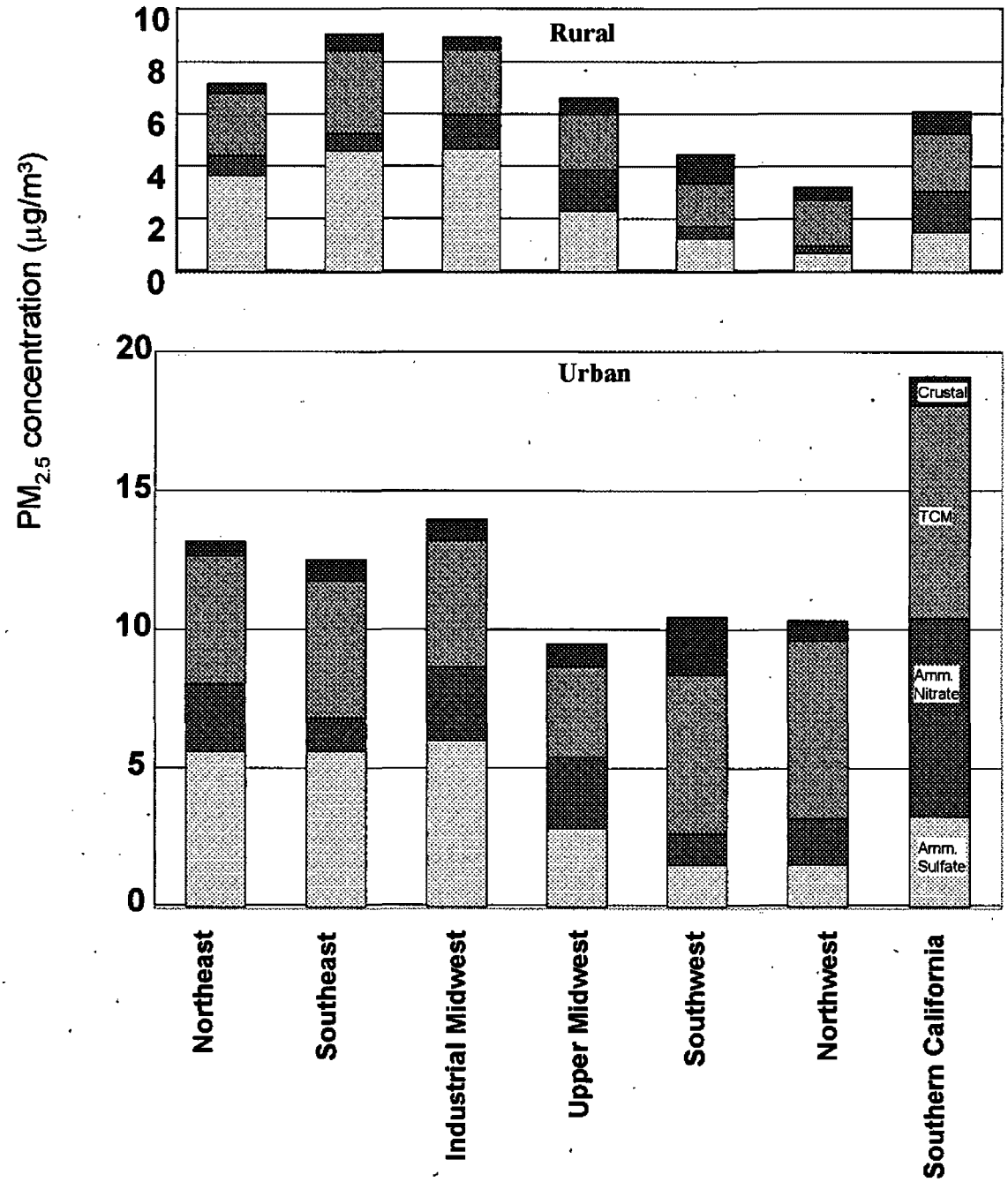

Figure 2-15. Annual average composition of $PM_{2.5}$ by region, 2003. Rural data (top panel) from IMPROVE network, urban data (bottom panel) from EPA Speciation Network. Components (from top to bottom) are crustal material, total carbonaceous mass (TCM), ammonium nitrate, and ammonium sulfate.

Source: Schmidt et al. (2005)

PM Supersites program, established by EPA. This monitoring program addresses a number of scientific issues associated with PM.[20] At a Supersite location in the Los Angeles metropolitan area, speciation data have been collected for fine, coarse, and ultrafine. Speciated data from this source-influenced site are shown in Figure 2-16. These data show that fine, coarse, and ultrafine PM have different compositions (in the Los Angeles area). For these PM size fractions, there are differences in the relative amounts of nitrates, sulfates, crustal (metals and trace elements), and carbon. Carbon, shown here as organic (OC) and elemental carbon (EC), makes up a large fraction of ultrafine and fine PM; crustal material dominates the coarse fraction.

Trends in rural area and urban Washington, D.C., concentrations of fine particle components based on data from the IMPROVE network from 1993 to 2003 are shown in Figure 2-9 (introduced above in section 2.4.1 on $PM_{2.5}$). The top two panels of this figure aggregate rural IMPROVE sites in the eastern and western U.S. The bottom panel shows the urban IMPROVE data for Washington, D.C., for the same time period. Levels of rural annual average $PM_{2.5}$ mass are significantly higher in the east than in the west. Annual levels of ammonium sulfates have decreased the most (and contributed the most to the reductions in $PM_{2.5}$ mass) both in eastern and western rural areas. At the Washington, D.C., IMPROVE site, mass has decreased 31 percent from 1993-2003. Total carbon (34 percent reduction) and ammonium sulfates (down 29 percent) are the biggest contributors to the mass reduction over the past 10 years. In addition, at the Washington, D.C., site, both total carbon and sulfates 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.

2.4.6 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.[21] Figure 2-17 shows the distribution of ratios of annual mean $PM_{2.5}$ to PM_{10} at sites in different geographic regions for 2001-2003. The ratios are highest in the eastern U.S. regions with median ratios of

[20] More information can be found at http://www.epa.gov/ttn/amtic/supersites.html.

[21] In this section's analyses, information was gleaned from the 489 site (4-, 8-, 12-quarter) $PM_{10-2.5}$ database for all 3 sizes in order to get seasonally unbiased estimates of their statistical relationships (i.e., to ensure a minimum number of data pairs each quarter for 4-, 8-, or 12 quarters).

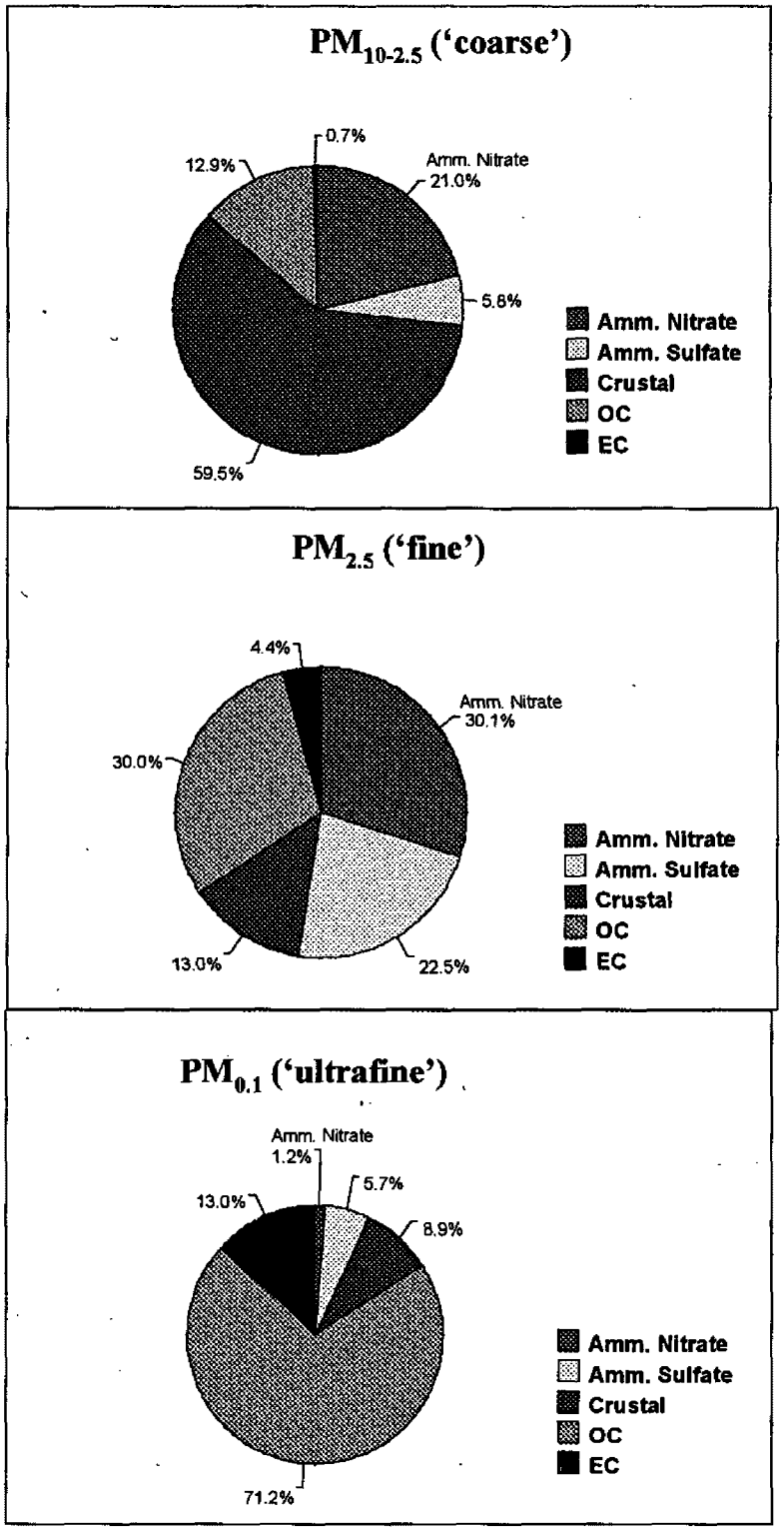

Figure 2-16. Average $PM_{10-2.5}$ $PM_{2.5}$, and $PM_{0.1}$ (ultrafine) chemical composition at an EPA 'supersite' monitor in Los Angeles, CA, 10/2001 to 9/2002. Components shown in clockwise order (starting with ammonium nitrate) as listed in legend from top to bottom.

Source: EPA (2004b)

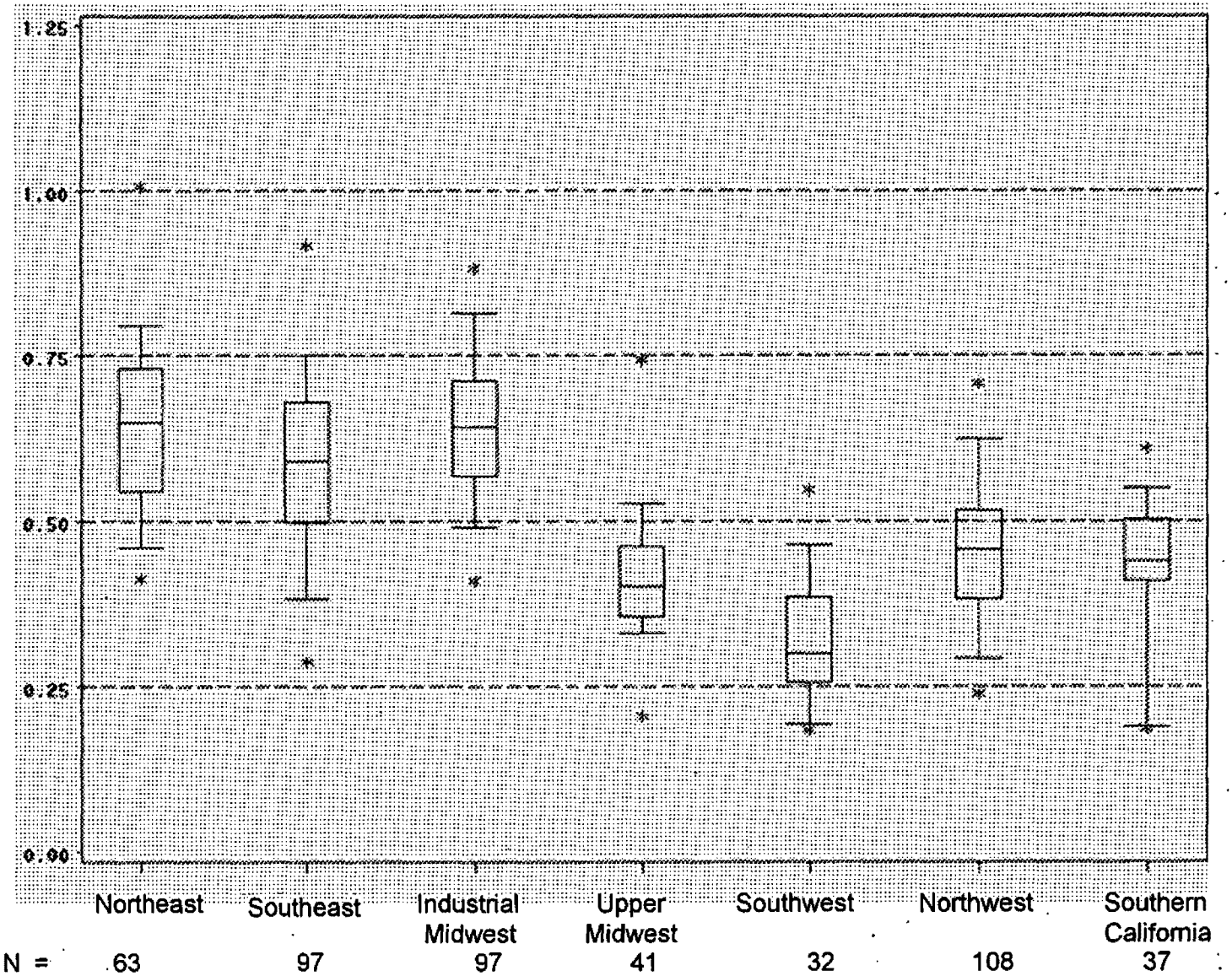

Figure 2-17. Distribution of ratios of $PM_{2.5}$ to PM_{10} by region, 2001-2003. Box depicts interquartile range and median; whiskers depict 5th and 95th percentiles; asterisks depict minima and maxima. N = number of sites.

Source: Schmidt et al. (2005)

about 0.6 to 0.65, and lowest in the Southwest region, with a median ratio near 0.3. 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 uncertainty 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. Figure 2-18 shows the results of a nationwide analysis of correlations among PM size fractions using 24-hour average data from the FRM monitoring networks for 2001-2003. $PM_{2.5}$ 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 or coarse particles, depending upon location-specific 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.

2.5 PM TEMPORAL PATTERNS

2.5.1 $PM_{2.5}$ and $PM_{10-2.5}$ Patterns

Data from the PM FRM network from 2001-2003 generally show distinct seasonal variations in $PM_{2.5}$ and estimated $PM_{10-2.5}$ concentrations. Although distinct, the seasonal fluctuations are generally not as sharp as those seen for ozone concentrations. Figure 2-19 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, 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.

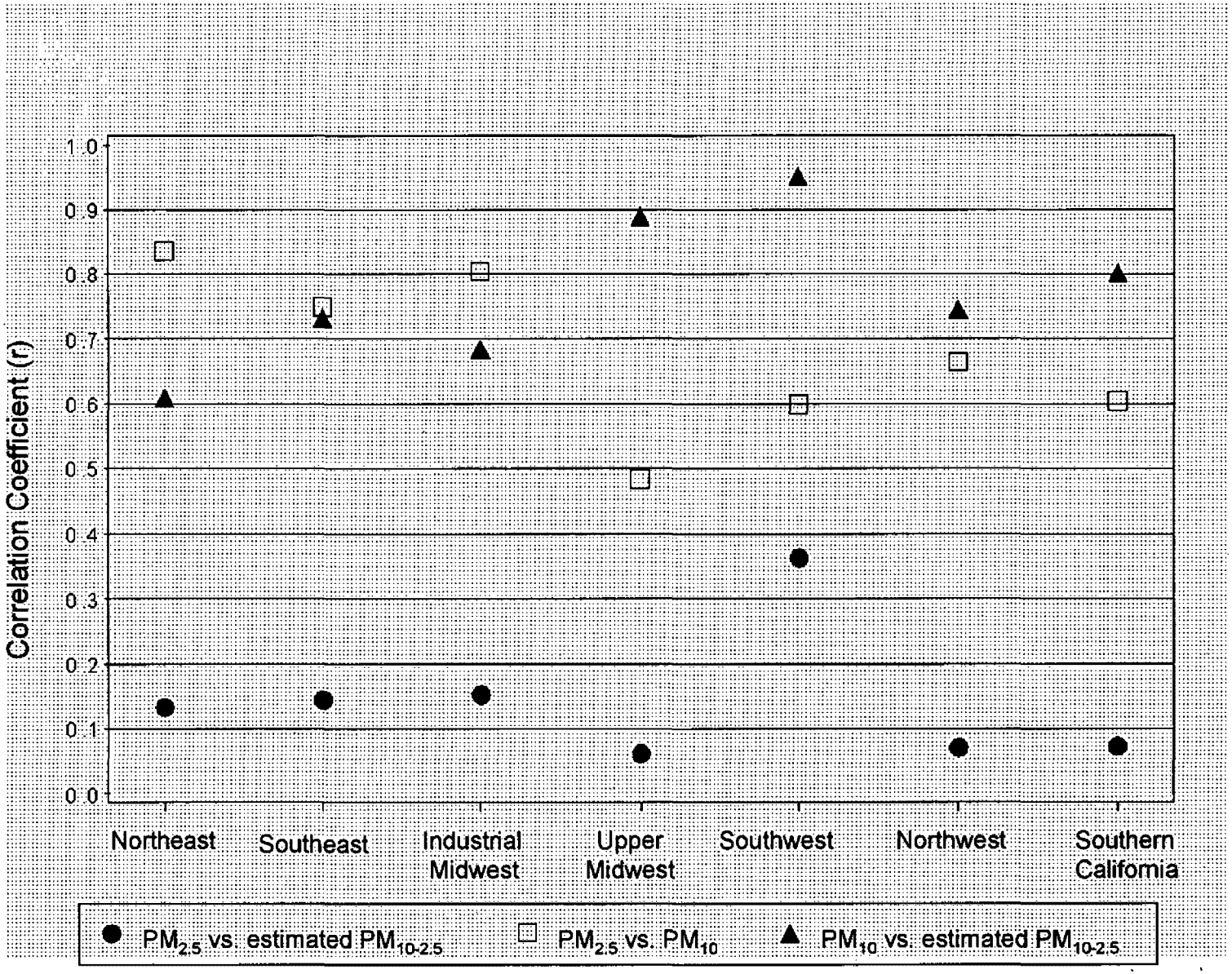

Figure 2-18. Regional average correlations of 24-hour average PM by size fraction.

Source: Schmidt et al. (2005)

Figure 2-20 shows the distributions of estimated 24-hour average urban $PM_{10-2.5}$ concentrations by U.S. geographic region. The lowest concentrations generally occur in the winter months. Elevated levels are apparent in the easternmost regions in April. In the upper midwest, northwest, and southern California regions, the highest levels occur in the mid- to late-summer to mid-fall. The southwest region exhibits the greatest range of variability throughout the year. Elevated levels are apparent in the spring, consistent with winds that contribute to windblown dust. In the southwest and southern California, highly elevated levels in the fall, especially October, were caused by forest fires in the vicinity of the monitoring sites.

The chemical components of fine particles also exhibit seasonal patterns. Figures 2-21 and 2-22 show seasonal 2003 urban and rural patterns for each of the U.S. regions. Seasonal patterns are shown by calendar quarter. In general:

- $PM_{2.5}$ values in the east are typically higher in the third calendar quarter (July-September) when sulfates are more readily formed from SO_2 emissions from power plants predominantly located there and sulfate formation is supported by increased photochemical activity.

- Urban $PM_{2.5}$ values tend to be higher in the first (January-February) and fourth (October-December) calendar quarters in many areas of the western U.S., in part because more carbon is produced when woodstoves and fireplaces are used and particulate nitrates are more readily formed in cooler weather. In addition, the effective surface layer mixing depth often is restricted due to inversion events, as well as limited by reduced radiative heating.

- Urban concentrations of $PM_{2.5}$ are seen to be generally higher than rural concentrations in all four quarters, though in the west the difference seems to be greatest in the cooler months.

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 2-23 and 2-24 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. Although there is a clear monotonic relationship between 98th percentiles and annual means, 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 of 98th percentiles spans about 5 to 6 $\mu g/m^3$ for each 1 $\mu g/m^3$ interval.. The range between the 5th and 95th

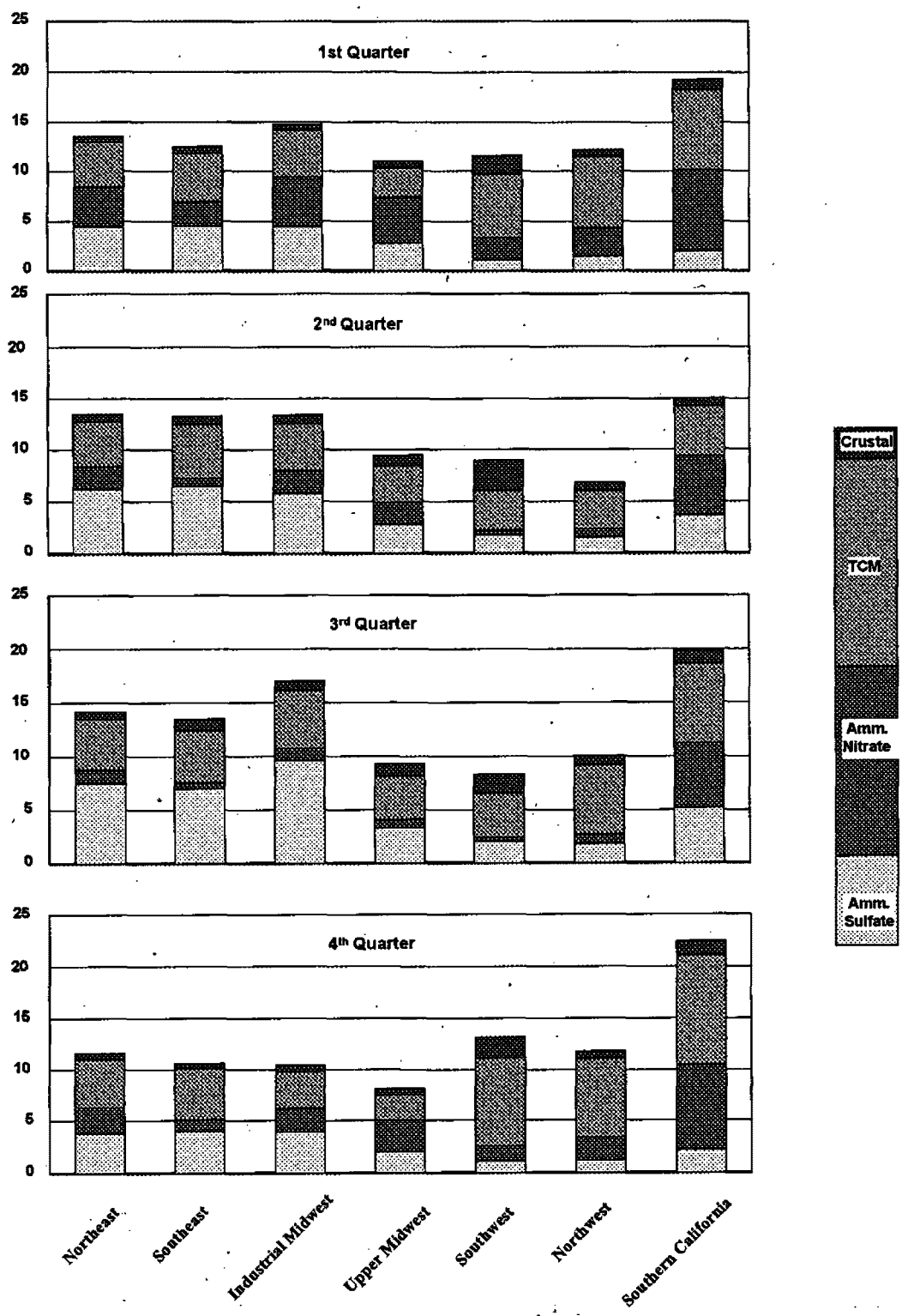

Figure 2-21. Seasonal average composition of urban $PM_{2.5}$ by region, 2003. Data from EPA Speciation Network. Components (from top to bottom) are crustal material, total carbonaceous mass (TCM), ammonium nitrate, and ammonium sulfate.

Source: Schmidt et al. (2005)

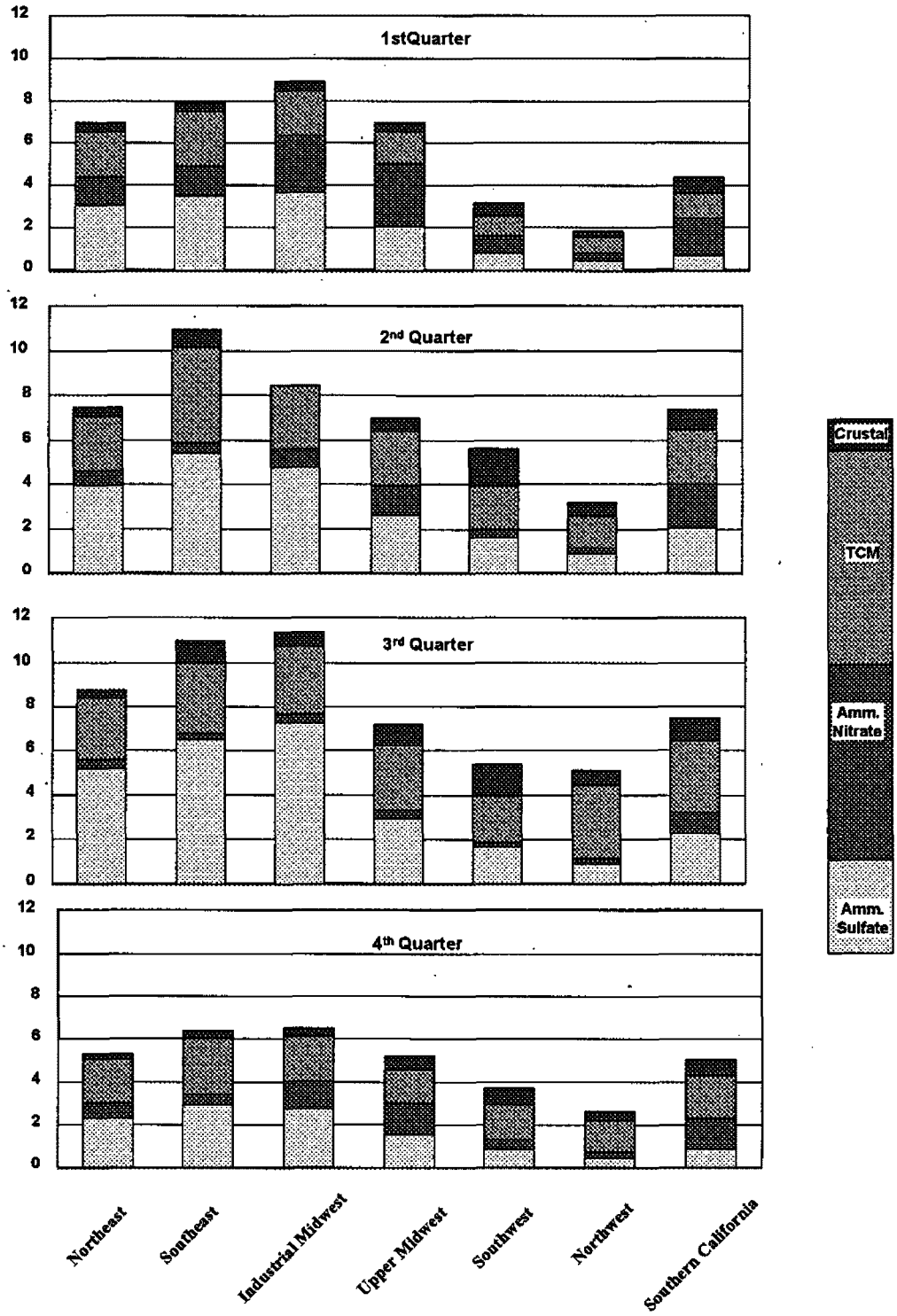

Figure 2-22. Seasonal average composition of rural $PM_{2.5}$ by region, 2003. Data from IMPROVE Network. Components (from top to bottom) are crustal material, total carbonaceous mass (TCM), ammonium nitrate, and ammonium sulfate.

Source: Schmidt et al. (2005)

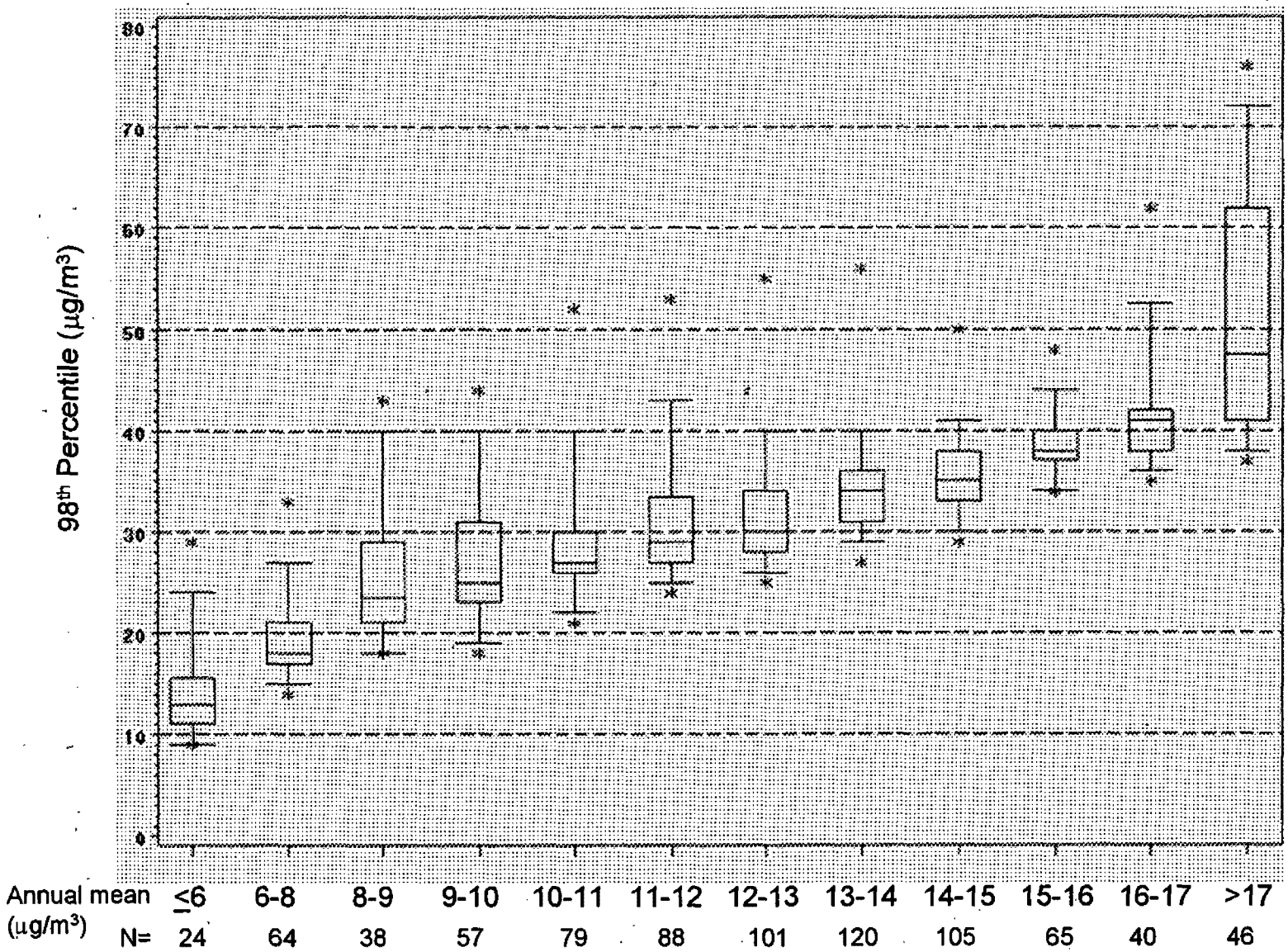

Figure 2-23. Distribution of annual mean vs. 98th percentile 24-hour average $PM_{2.5}$ concentrations, 2001-2003. Box depicts interquartile range and median; whiskers depict 5th and 95th percentiles; asterisks depict minima and maxima. N= number of sites.

Source: Schmidt et al. (2005)

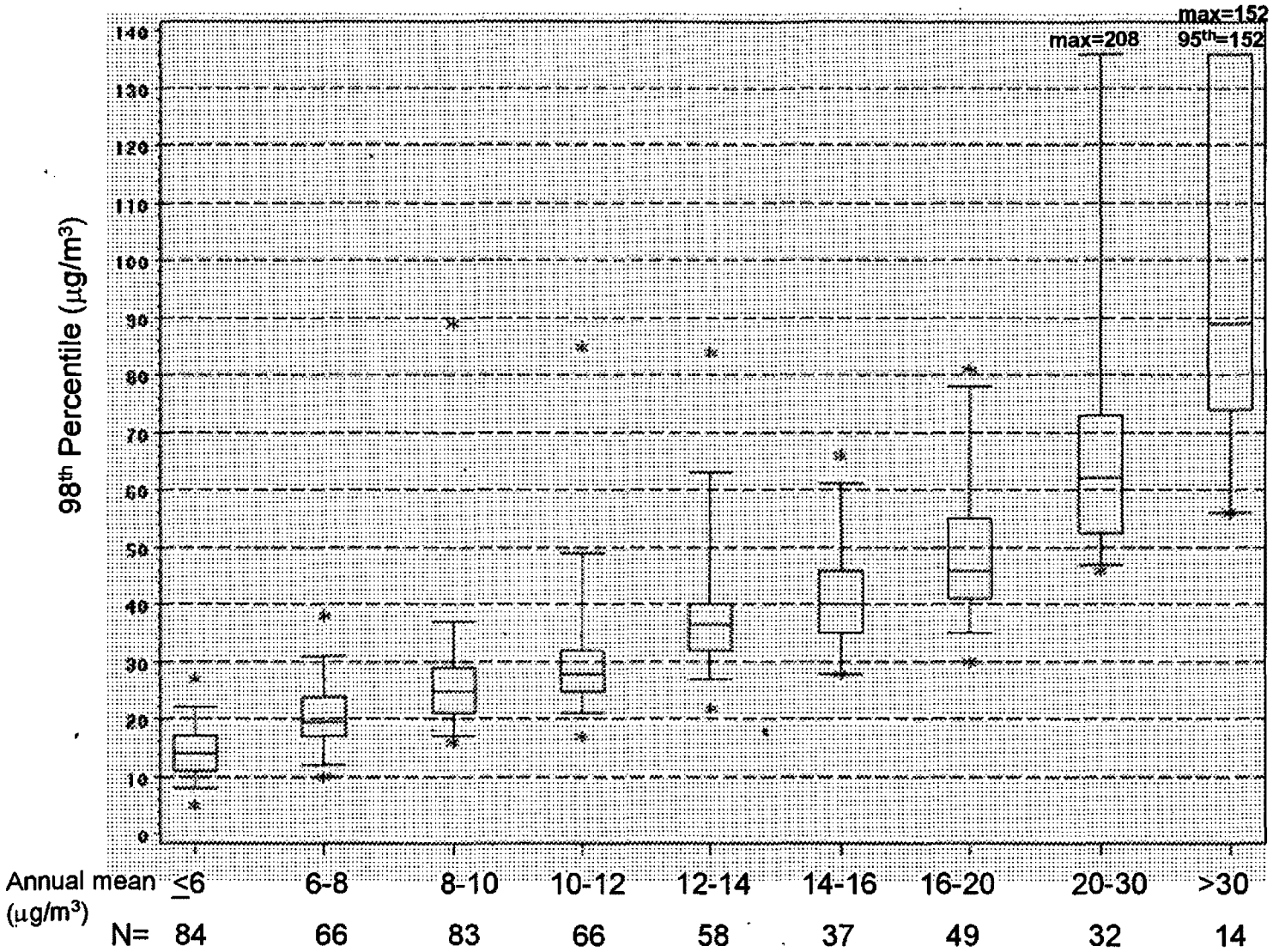

Figure 2-24. Distribution of estimated annual mean vs. 98th percentile 24-hour average $PM_{10-2.5}$ concentrations, 2001-2003. Box depicts interquartile range and median; whiskers depict 5th and 95th percentiles; asterisks depict minima and maxima. N= number of sites.

Source: Schmidt et al. (2005)

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 for annual mean intervals above 20 $\mu g/m^3$.

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 and welfare effects. Examples of average hourly profiles for $PM_{2.5}$ and $PM_{10-2.5}$ from 2001-2003 are shown in Figures 2-25 and 2-26 for a monitoring site in the Greensboro, NC, metropolitan area. As with most eastern urban sites, the $PM_{2.5}$ concentrations are significantly higher than those for $PM_{10-2.5}$. Profiles, for both $PM_{2.5}$ and $PM_{10-2.5}$, in Figure 2-25 indicate that 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. An evening peak starting about 5:00 pm is also evident for both size indicators. The 95th percentile concentrations during peak hours can be as high as three to four times the median level for the same hour. As indicated in Figure 2-26, the lowest seasonal levels for both size fractions occur in the winter. For $PM_{2.5}$, the summer concentrations are considerably higher than the other season. These profiles of hourly average $PM_{2.5}$ and $PM_{10-2.5}$ levels are typical of many, but not all, eastern U.S. urban areas.

Figure 2-27 shows hourly average $PM_{2.5}$ and $PM_{10-2.5}$ concentrations for a monitoring site in the El Paso, TX metropolitan area from 2001-2003. Like many western U.S. sites for all hours of the day, the $PM_{10-2.5}$ concentrations are higher than the $PM_{2.5}$ levels. However, this particular site is atypical of most urban ones, even in the west. Note the increased variability in the hourly concentrations compared to the Greensboro site; the 95th percentile concentrations for some hours are more than ten times the median levels. Note also that hourly means are significantly higher than the medians, and in some cases, the 75th percentiles. Episodic events are causing these significant excursions from the typical day. Figure 2-28 highlights one of several such episodic events that affected this site. On April 26, 2002, there was a dust storm that caused the $PM_{2.5}$ and $PM_{10-2.5}$ concentrations to be extremely elevated. The dust particles from the storm had a greater impact on the $PM_{10-2.5}$ concentrations than the $PM_{2.5}$. (Note that the $PM_{10-2.5}$ scale is about 3 times as large as the $PM_{2.5}$ scale.) Hourly $PM_{10-2.5}$ levels approaching 3000 $\mu g/m^3$ were recorded this day.

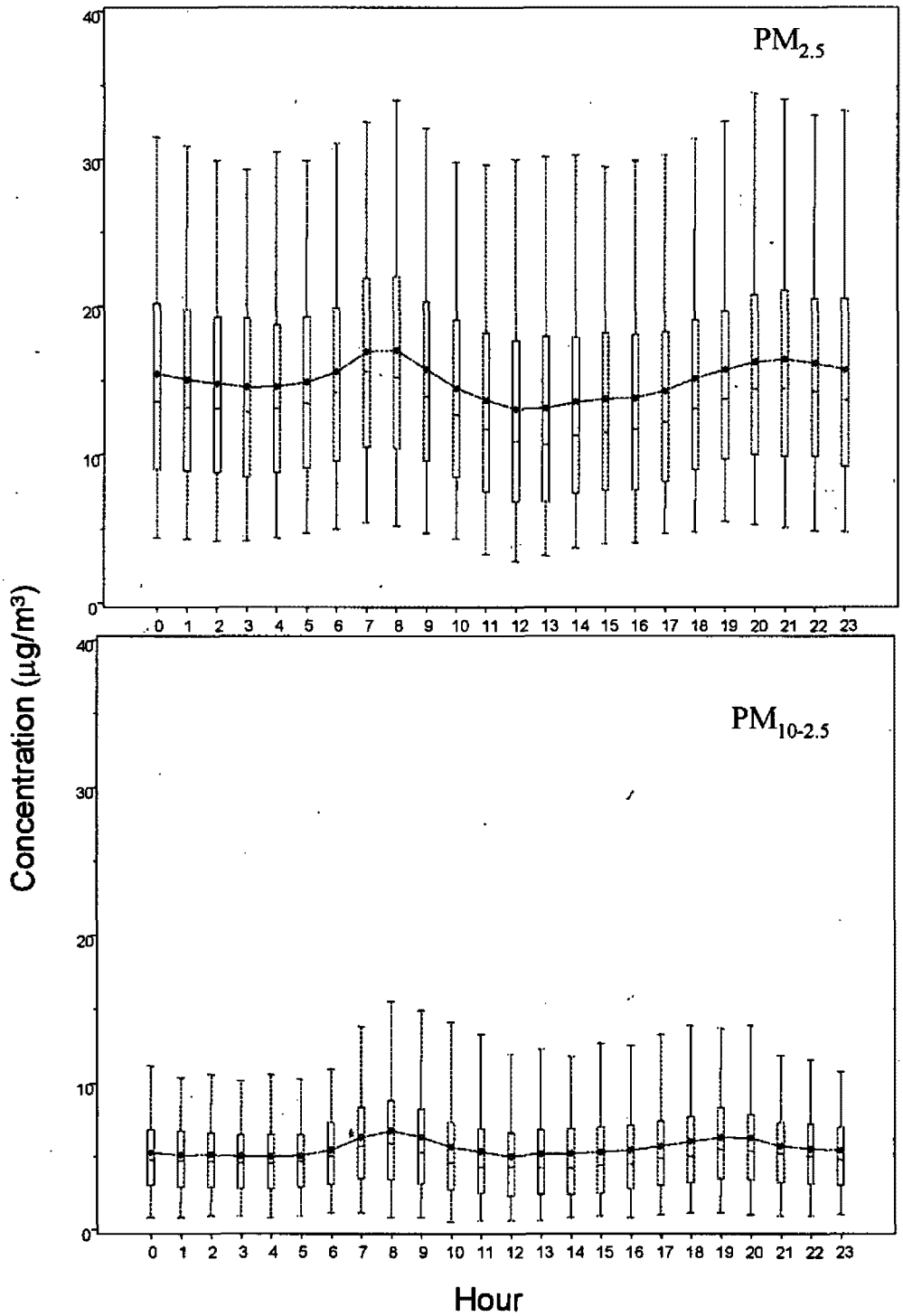

Figure 2-25. Hourly average $PM_{2.5}$ and $PM_{10\text{-}2.5}$ concentrations at a Greensboro, NC monitoring site, 2001-2003. Upper panel shows the distribution of $PM_{2.5}$ concentrations and the lower panel shows the distribution of $PM_{10\text{-}2.5}$ concentrations. (Box plots of interquartile ranges, means, medians, 5th and 95th percentiles.)

Source: Schmidt et al. (2005)

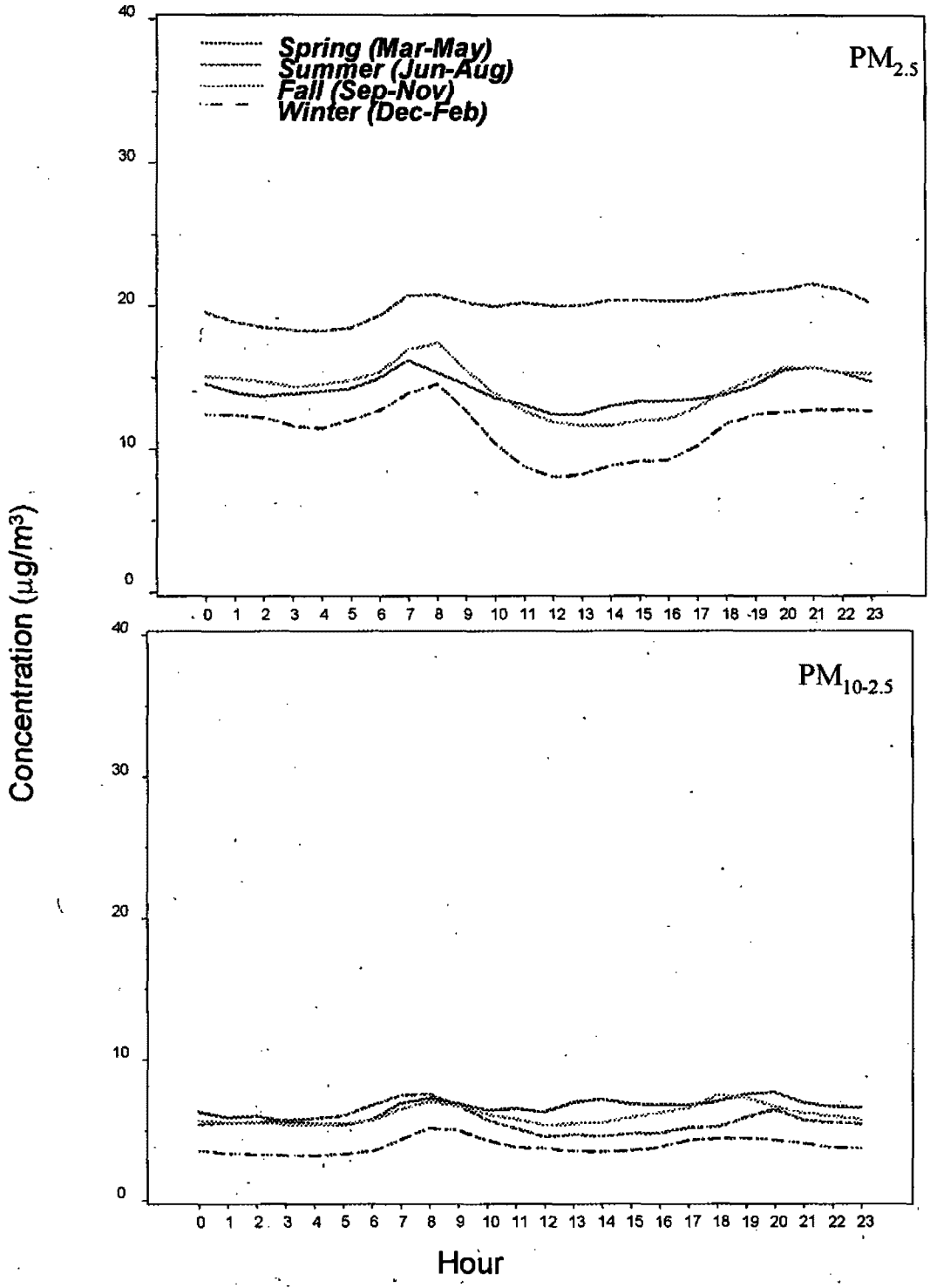

Figure 2-26. Seasonal hourly average $PM_{2.5}$ and $PM_{10-2.5}$ concentrations at a Greensboro, NC monitoring site, 2001-2003. Upper panel shows the $PM_{2.5}$ concentrations and the lower panel shows the $PM_{10-2.5}$ concentrations.

Source: Schmidt et al. (2005)

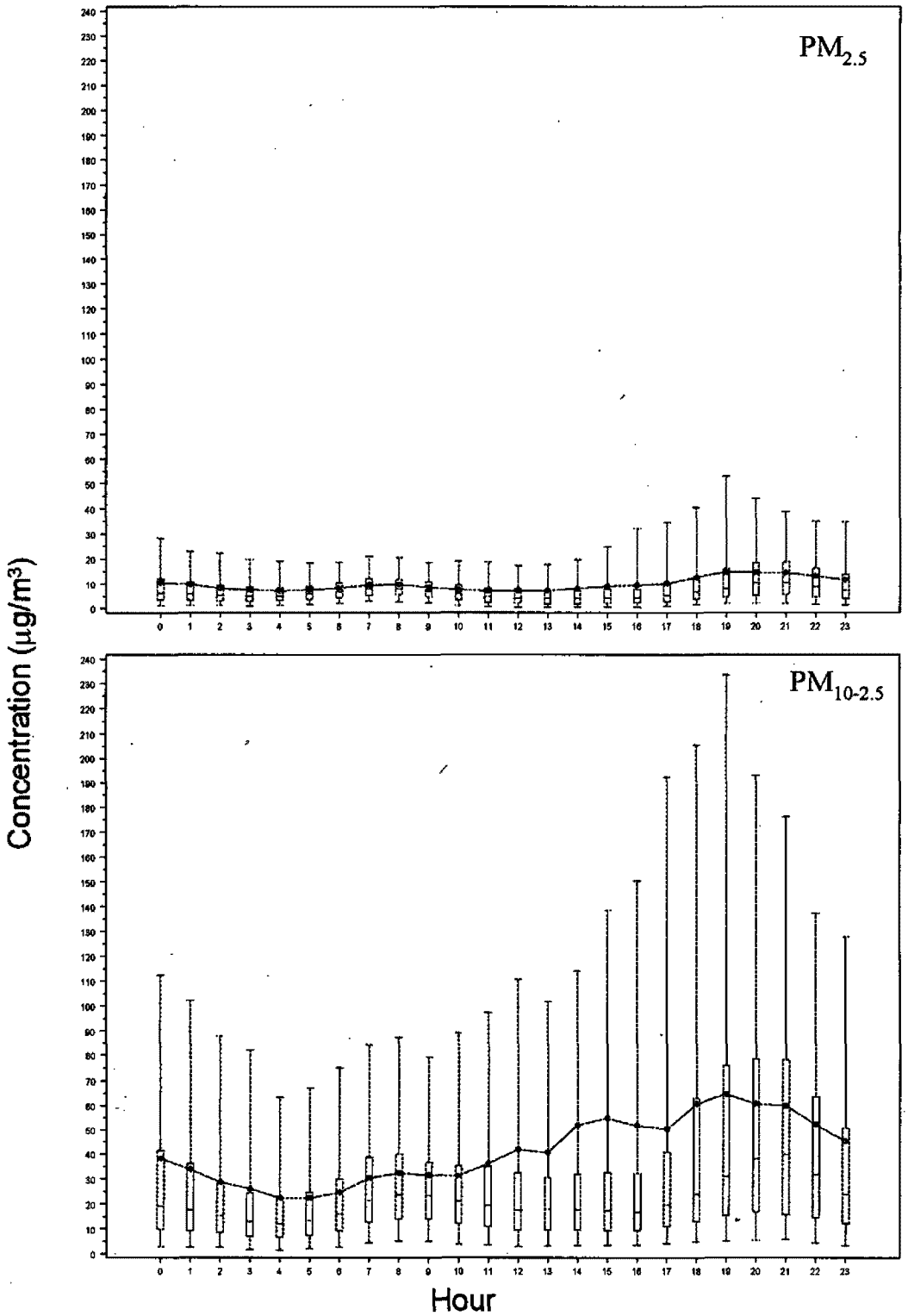

Figure 2-27. Hourly average $PM_{2.5}$ and $PM_{10-2.5}$ concentrations at an El Paso. TX monitoring site, 2001-2003. Upper panel shows the distribution of $PM_{2.5}$ concentrations and the lower panel shows the distribution of $PM_{10-2.5}$ concentrations. (Box plots of interquartile ranges, means, medians, 5th and 95th percentiles.)

Source: Schmidt et al. (2005)

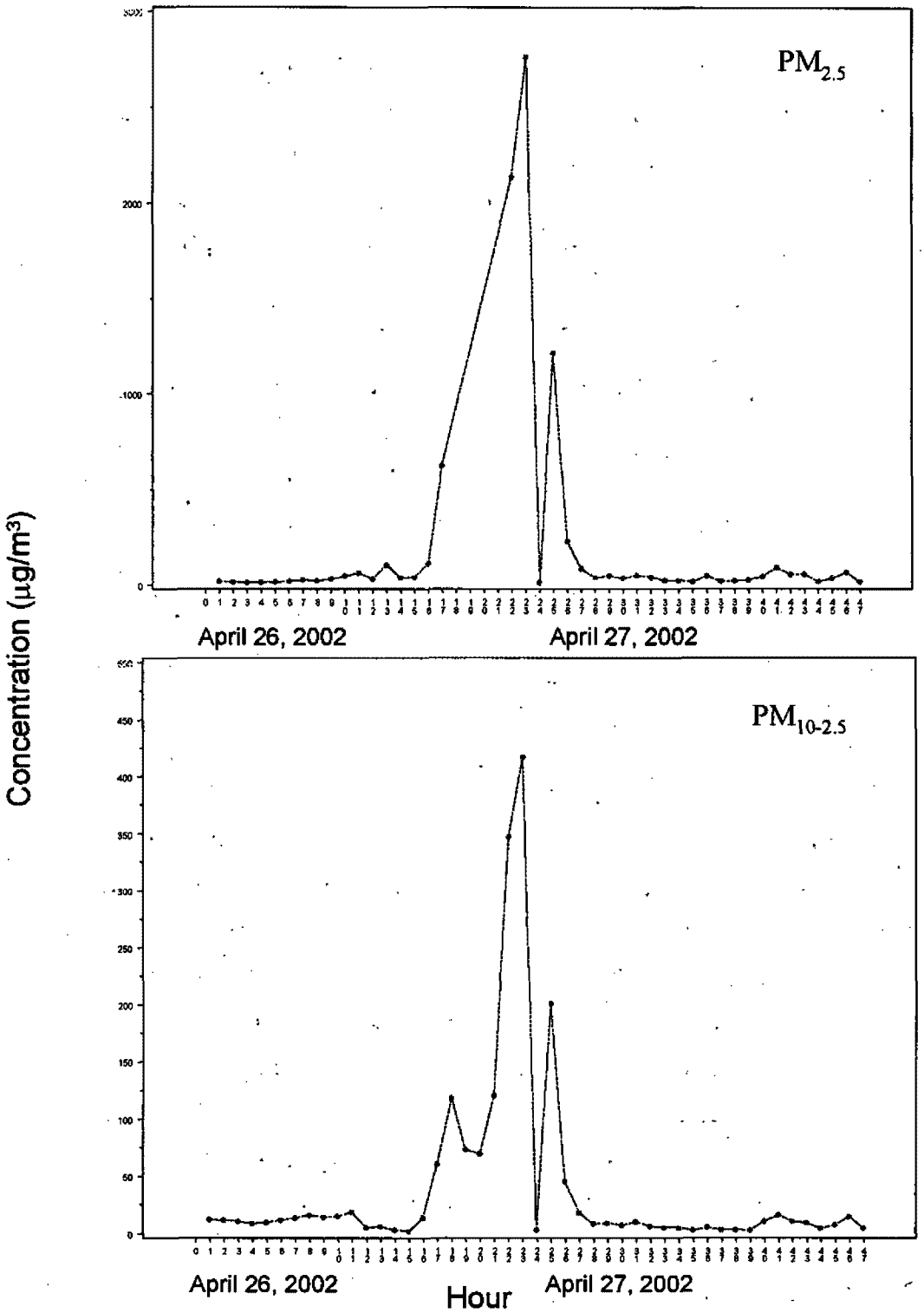

Figure 2-28. Hourly $PM_{2.5}$ and $PM_{10\text{-}2.5}$ concentrations at a El Paso, TX monitoring site, April 26, 2002-April 27, 2002. Upper panel shows the hourly $PM_{2.5}$ concentrations and the lower panel shows the hourly $PM_{10\text{-}2.5}$ concentrations. Note the different scales.

Source: Schmidt et al. (2005)

The hourly ranges shown in Figures 2-25 and 2-27 suggest that hour-to-hour changes in $PM_{2.5}$ concentrations encompass several $\mu g/m^3$; however, extreme values for hour-to-hour variations can be much larger. An analysis of the distribution of increases in hour-to-hour concentrations at multiple sites across the U.S. for 2001-2003 found site-level median hourly increases ranging up to 6 $\mu g/m^3$ (maximum), with an average median increase of about 1.8 $\mu g/m^3$.

2.5.2 Ultrafine Patterns

Diurnal or seasonal patterns for ultrafine particles have been studied in relatively few areas of the U.S. A study done at the most extensively studied urban location in the U.S., Atlanta, GA, is discussed in the CD (p.3-32). In this study, (CD, p. 3-32 to 3-33) ultrafine particle number concentrations were found to be higher in the winter than in the summer. 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 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. .

2.6 PM BACKGROUND LEVELS

For the purposes of this document, policy-relevant background (PRB) (referred to as "background" in the rest of this section) PM is defined as the distribution of PM concentrations that would be observed in the U.S. in the absence of anthropogenic (man-made) emissions of primary PM and precursor emissions (e.g., VOC, NO_x, SO_2, and NH_3) in the U.S., Canada, and Mexico. The reason for defining background in this manner is that for purposes of determining the adequacy of current standards and the need, if any, to revise the standards, EPA is focused on the effects and risks associated with pollutant levels that can be controlled by U.S. regulations or through international agreements with border countries. Thus, as defined here, background includes PM from natural sources in the U.S. and transport of PM from both natural and man-made sources outside of the U.S. and its neighboring countries.

Section 3.3.3 of the CD discusses annual average background PM levels, and states that "[e]stimates of annually averaged PRB concentrations or their range have not changed from the 1996 PM AQCD" (CD, p. 3-105). These ranges for $PM_{2.5}$ and PM_{10} are reproduced in Table 2-5. The lower bounds of these ranges are based on estimates of "natural" background midrange

concentrations. The upper bounds are derived from the multi-year annual averages of the remote monitoring sites in the IMPROVE network (EPA, 1996a, p. 6-44). The ranges for $PM_{10-2.5}$ are derived from the $PM_{2.5}$ ranges and the PM_{10} ranges by subtraction (CD, p. 3-83). Since the IMPROVE data unavoidably reflect some contributions from the effects of anthropogenic emissions from within the U.S., Canada, and Mexico, as well as background, they likely overestimate the U.S. background concentrations as defined here.

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 particles and more water associated with hygroscopic fine particles than the western U.S. due to generally higher humidity levels.

Table 2-5. Estimated Ranges 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
$PM_{10-2.5}$	0 - 7	0 - 9

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 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 background 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. 3-82). 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 the U.S., Canada, and Mexico can be significant on an episodic, but probably not on an annual basis (CD, p. 3-91). Several studies

have focused on identifying the origin, sources, and impacts of recent transnational transport events from Canada, Mexico, 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. 3-86).

- 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. 3-87).

- 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. 3-84).

- 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. (2001) 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. 3-84).

Background concentrations of $PM_{2.5}$, $PM_{10-2.5}$, and PM_{10} may be conceptually viewed as comprised of baseline and episodic components. The baseline component is the contribution from natural sources within the U.S., Canada, and Mexico and from transport of natural and anthropogenic sources outside of the U.S., Canada, and Mexico that is reasonably well characterized by a consistent pattern of daily values each year, although they may vary by region and season.

In addition to this baseline contribution to background concentrations, a second component consists of more rare episodic high-concentration events over shorter periods of time (e.g., days or weeks) both within the U.S., Canada, and Mexico (e.g., volcanic eruptions, large forest fires) and from outside of the U.S., Canada, and Mexico (e.g., transport related to dust storms from deserts in North Africa and Asia). Over shorter periods of time (e.g., days or weeks), the range of background concentrations is much broader than the annual averages. Specific natural events such as wildfires, volcanic eruptions, and dust storms, both of U.S. 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 can be essentially uncontrollable, EPA has in place policies that can remove consideration of them, where appropriate, from attainment decisions.[22]

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. EPA staff have conducted an analysis of daily $PM_{2.5}$ measurements from 1990 to 2002 at IMPROVE sites across the U.S., focused on the non-sulfate components of $PM_{2.5}$ (Langstaff, 2005). Ambient sulfate concentrations are almost entirely due to anthropogenic sources (with the exception of sulfates from volcanic eruptions), so while non-sulfate $PM_{2.5}$ is partly of anthropogenic origin, it captures almost all of the background.

Based on regional differences in geography and land use, the U.S. is divided into a number of regions for estimating regional background levels. The "Eastern U.S." region extends west to include Minnesota, Iowa, Missouri, Arkansas, and Louisiana. The "Central West" region comprises states west of the Eastern U.S. region and east of Washington, Oregon, and California. Washington, Oregon, and northern California make up the "North West Coast" and southern California (south of about 40 degrees latitude) makes up the "South West Coast" regions.[23]

To arrive at estimates of background we use the averaged measured non-sulfate $PM_{2.5}$ values at IMPROVE sites in these regions. The Eastern U.S. region is heavily impacted by anthropogenic emissions and we selected sites in northern states, which we judge to be affected to a lesser extent by anthropogenic pollution, to derive estimates of background concentrations, using all IMPROVE sites in the selected states. In all of the other regions we include all of the

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[22] There are two policies which allow PM data to be flagged for special consideration due to natural events: the Exceptional Events Guideline (EPA, 1986) and the PM10 Natural Events Policy (Nichols, 1996). Under these policies, 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_{10} events are specified in the natural events policy: volcanic or seismic activity, wildland fires, and high wind dust events. The exceptional events policy covers natural and other events not expected to recur at a given location and applies to all criteria pollutants. Categories of events covered in the exceptional events guidance include, but are not limited to, high winds, volcanic eruptions, forest fires, and high pollen counts. EPA is drafting further guidance concerning how to handle data affected by natural events related to the PM standards.

[23] The 'Eastern' region roughly equates to the combined Southeast, Northeast, Industrial Midwest, and eastern portion (MN, IA, & MO) of the Upper Midwest regions as defined previously in this chapter (Figure 2-4). The 'Central West' region roughly corresponds to the western portion of the Upper Midwest region and the eastern two thirds (ID, MT, CO, UT, NV) of the Northwest region. The 'North West Coast' approximates the remaining one third (northern CA, OR, and WA) of the Northwest region. The 'South West Coast' area is similar to the southern California region.

IMPROVE sites. Table 2-6 describes the IMPROVE sites selected to represent these different regions of the U.S. We recognize that these estimates will likely be biased high, as they include an anthropogenic component, some sites more than others.

The 99th percentile concentrations at each of these sites were calculated to assess high values measured at these sites, while avoiding excursions that potentially reflect exceptional natural events. Standard deviations were also calculated for characterization of the daily variation of background concentrations. Table 2-7 presents the results of this analysis as means and ranges of individual site statistics within each of the background regions.

Table 2-6. IMPROVE sites selected for estimates of regional background

Region	IMPROVE Sites
Eastern U.S.	All sites in Maine, New Hampshire, Vermont, Minnesota, and Michigan
Central West	All sites in this region (sites in ID, MT, WY, ND, SD, CO, UT, NV, AZ)
North West Coast	All sites in this region (all Washington and Oregon sites, and the northern California sites REDW and LAVO)
South West Coast	All sites in this region (all California sites except the northern sites REDW and LAVO)
Alaska	All sites in Alaska
Hawaii	All sites in Hawaii

Table 2-7. Estimates of long-term means, daily standard deviations and 99th percentiles of $PM_{2.5}$ background concentrations ($\mu g/m^3$)

Region	# Sites	Means	St Devs	99th %iles
Eastern U.S.	7	3.0 (2.5-3.6)	2.5 (2.1-2.8)	13 (11-15)
Central West	37	2.5 (1.6-4.6)	1.9 (1.3-3.7)	10 (6-17)
North West Coast	8	3.4 (2.2-6.6)	2.8 (2.1-4.2)	14 (10-21)
South West Coast	8	5.2 (2.6-8.6)	3.7 (1.8-6.8)	20 (9-33)
Alaska	1	1.2	1.5	9
Hawaii	3	1.1 (0.7-1.8)	0.9 (0.8-1.0)	4 (4-5)

Notes:
1) Some of these estimates likely contain a significant North American anthropogenic component.
2) The "Means" column has the mean of the long-term averages of the sites representing the region followed by the minimum and maximum of the long-term averages of these sites in parentheses. Similarly for the "St Devs" column, which presents standard deviations of the daily concentrations about the annual means, and the "99th %iles" column, which presents the 99th percentiles of the daily concentrations over the 23-year period.

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 an estimate of the highest daily background concentrations. Staff notes that these recent findings are generally consistent with those from the last review, which suggested 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. (EPA, 1996b).

2.7 RELATIONSHIP BETWEEN AMBIENT PM MEASUREMENTS AND HUMAN EXPOSURE

The statutory focus of the primary NAAQS for PM is protection of 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 outdoor atmosphere that are either emitted directly by sources or formed in the atmosphere from precursor emissions. 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).

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, source origin, and 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.[24] The focus here is on particle size distinctions; the CD (CD, Section 5.4) also discusses exposure relationships related to compositional differences.

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

2.7.1 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 and particles formed in the atmosphere from emissions of gaseous precursors. This includes emissions not only from outdoor sources such as smokestacks, industrial sources, and automobiles, but also from sources located indoors with emissions vented outdoors, such as fireplaces, wood stoves, and some cooking appliances. 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 only due to indoor sources of particles that are not vented outdoors such as smoking, cooking, other non-vented sources of combustion, cleaning, mechanical processes, and chemical interactions producing particles. In characterizing human exposure to PM concentrations relevant to setting standards for ambient air quality, the 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 PM exposure*).

Outdoor concentrations of PM are affected by emissions, meteorology, topography, 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, resuspension of particles, and removal mechanisms such as particle deposition, ventilation, and air-conditioning and air cleaning devices (CD, p. 5-122). 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, referred to as the personal cloud.

Epidemiologic studies generally use measurements from central monitors to represent the ambient concentrations in an urban or rural 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 many 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.

2.7.2 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 1996 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 coarse fraction particles. As discussed in the 1996 Staff Paper, 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 nearby 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 current CD, and provide additional support for these findings. The CD discusses two classes of fine particles: ultrafine and accumulation-mode particles (see Chapter 2). Ultrafine, accumulation-mode, and coarse particles have different chemical and physical properties which affect personal exposures in different ways (CD, Table 9-2, p. 9-17).

$PM_{10-2.5}$ and ultrafine particles penetrate indoors less readily than $PM_{2.5}$ and deposit to surfaces more rapidly than $PM_{2.5}$, a greater proportion of $PM_{2.5}$ of ambient origin is found indoors than $PM_{10-2.5}$ and ultrafine particles, relative to their outdoor concentrations. Thus, the particle size distribution influences the amounts of PM of ambient origin found indoors.

Since people typically spend a large part of their time indoors at home, the air exchange rate of the home has a large impact on exposures to ambient pollution. Homes with low air exchange rates are more protected from outdoor sources, and vice-versa. Homes in regions with moderate climate tend to be better ventilated and have higher air exchange rates than areas which have very cold or very hot climates. Thus, climate plays an important role in regional population exposure to ambient pollution.

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. 5-82). This factor, however, does not influence exposure to PM of ambient origin.

These three factors related to total personal exposure can give rise to measurement error in estimating exposures to fine and coarse PM (CD, Section 5.5.3), thus making the quantification of relationships between concentrations measured at central site monitors and health effects more difficult due to reduction in statistical power. Moreover, exposure measurement errors can also affect the magnitude and the precision of the health effects estimates. However, as discussed in the CD and below in Chapter 3, exposure measurement errors under most ordinary circumstances are not expected to influence the overall interpretation of findings from either the long-term exposure or time-series epidemiologic studies that have used ambient concentration data (CD, p. 5-121).

The 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. The explanation of this finding is that 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 concentrations of ambient origin is correlated with ambient concentrations. Thus, it is not surprising that health effects might correlate with central site PM concentrations, because exposure to PM of ambient origin correlates with these concentrations, and the lack of correlation of total exposure with central site PM concentrations does not statistically alter that relationship. By their statistical design, time-series epidemiologic studies of this type only address the ambient component of exposure, since the impact of day-to-day fluctuations in ambient PM on acute health effects is examined.

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[25] provides important findings, as discussed in the CD (p. 5-63 to 5-66 and 5-125 to 5-126). The PTEAM study demonstrated that central site ambient PM_{10} concentrations are well correlated with personal exposure to PM_{10} of ambient origin, while such concentrations are only weakly correlated with total personal exposure. This study also found that estimated exposure to nonambient PM_{10} is effectively independent of PM_{10} concentrations at central site monitors, and that nonambient exposures are highly variable due to differences in indoor sources across the study homes.

When indoor sources only have minor contributions to personal exposures, total exposure is mostly from PM of ambient origin. In these cases high correlations are generally found between total personal exposure and ambient PM measured at a central site (CD, p. 5-54). For example, measurements of ambient sulfate, which is mostly in the fine fraction, have been found to be highly correlated with total personal exposure to sulfate (CD, p. 5-124). 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 possible presence of small indoor-generated sulfates in some environments.

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[25] 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_{10}, 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.

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 more difficult to detect a true underlying association between the exposure metric and the health outcome of interest. However, the use of ambient PM concentrations as a surrogate for personal ambient exposures is not expected to change the principal conclusions from PM epidemiological studies that use community average health and pollution data (CD, p. 5-121). Based on these considerations and on the review of the available exposure-related studies, the CD concludes that for epidemiologic studies, ambient $PM_{2.5}$ concentration as measured at central site monitors is a useful surrogate for exposure to $PM_{2.5}$ of ambient origin. However, for coarse and ultrafine PM, such ambient concentrations are not likely to be as good a surrogate for personal ambient exposure. While nonambient PM may also be responsible for health effects, since the ambient and nonambient components of personal exposure are independent, the health effects due to nonambient PM exposures generally will not bias the risk estimated for ambient PM exposures (CD, p. 9-17).

2.8 RELATIONSHIP BETWEEN AMBIENT PM AND VISIBILITY

The effect of ambient particles on visibility is dependent upon particle size and composition, atmospheric illumination, the optical properties of the atmosphere, and the optical properties of the target being viewed. The optical properties of particles, discussed in section 2.2.5, can be well characterized in terms of a light extinction coefficient. For a given distribution of particle sizes and compositions, the light extinction coefficient is strictly proportional to the particle mass concentration. Light extinction is a measure of visibility impairment, and, as such, provides a linkage between ambient PM and visibility, as discussed below in section 2.8.1. Other measures directly related to the light extinction coefficient are also used to characterize visibility impairment, including visual range and deciviews, as discussed below in section 2.8.2. Light extinction associated with background levels of PM is also discussed below in section 2.8.3.

2.8.1 Particle Mass and Light Extinction

Fine particle 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, sulfates and

nitrates contribute substantially more to light scattering per unit mass than other constituents, especially as relative humidity levels exceed 70 percent. 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, regional estimates, or actual day-specific measurements of the component-specific percentage of total mass, however, one can develop reasonable estimates of light extinction from PM mass concentrations (see section 6.2.2 for further discussion).

The light extinction coefficient has been widely used in the U.S. for many years as a metric to describe the effect of concentrations of particles and gases 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 (b_{ext}) is represented by the following equation (CD, 4-155):

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

where b_{ap} = light absorption by particles

b_{ag} = light absorption by gases

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

b_{sp} = light scattering 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-veraged light extinction coefficient. Transmissometers operate continuously, and data are 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 for the coarse fraction mass, plus 10 Mm^{-1} for light extinction due to Rayleigh scattering, discussed below. This algorithm is represented by the following equation (CD, 4-169):

$$
\begin{aligned}
b_{ext} = \; & (3)f(RH)\,[SULFATE] \\
& + (3)f(RH)\,[NITRATE] \\
& + (1.4)\,[ORGANIC\ CARBON] \\
& + (10)\,[LIGHT\ ABSORBING\ CARBON] \\
& + (1)\,[SOIL] \\
& + (0.6)\,[COARSE\ PM] \\
& + 10\ (for\ Rayleigh\ scattering\ by\ gases)
\end{aligned}
\qquad (5\text{-}2)
$$

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, *f(RH)*, to account for their hygroscopic behavior (CD, p. 4-169). The relative humidity adjustment factor increases significantly with higher humidity, ranging from about 2 at 70 percent, to 4 at 90 percent, and over 7 at 95 percent relative humidity (CD, p. 4-170, Figure 4-38).

Rayleigh scattering represents the degree of natural light scattering found in a particle-free atmosphere, caused by the gas molecules that make up "blue sky" (e.g., N_2, O_2). 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 nm (CD, p. 4-156 to 4-157). 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. 4-157). 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.

The light 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. By apportioning the light extinction coefficient to different aerosol constituents, one can estimate changes in visibility due to changes in constituent concentrations (Pitchford and Malm, 1994).

2.8.2 Other Measures of Visibility

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):

$$\text{Visual Range (km)} = 3912/b_{ext}(\text{Mm}^{-1}) \qquad (5\text{-}3)$$

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 percent change in the extinction coefficient (Pitchford and Malm, 1994). Deciview can be calculated from the light extinction coefficient (b_{ext}) by the equation:

$$\text{Haziness (dv)} = 10 \ln(b_{ext}/10\ \text{Mm}^{-1})$$

Figure 2-29 graphically illustrates the relationships among light extinction, visual range, and deciview.

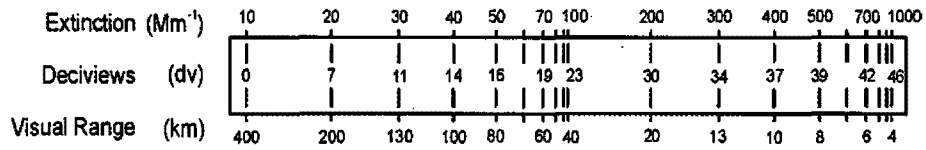

Figure 2-29. Relationship between light extinction, deciviews, and visual range.

Source: Malm (1999)

2.8.3 Visibility at PM Background Conditions

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. Background PM is defined and discussed in detail in section 2.6, and Table 2-5 provides the annual average regional background $PM_{2.5}$ mass ranges for the eastern and western U.S.

The National Acid Precipitation Assessment Program report (NAPAP, 1991) provides estimates of extinction contributions from background levels of fine and coarse particles, plus Rayleigh scattering. 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 scattering, annual average background levels of fine and coarse particles are estimated to account for approximately 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 background conditions, discussed above in section 2.6, 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. This issue is discussed further in Chapter 6, section 6.2.

Fine particles originate from both natural and anthropogenic, or man-made, sources. Background concentrations of fine particles are those originating from natural sources. On an annual average basis, 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. Anthropogenic contributions account for about one-third of the average extinction coefficient in the rural West and more than 80 percent in the rural East (NAPAP, 1991).

It is important to note that, even in 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. As discussed in the CD, 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. 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.

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3. POLICY-RELEVANT ASSESSMENT OF HEALTH EFFECTS EVIDENCE

3.1 INTRODUCTION

This chapter assesses key policy-relevant information on 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. More specifically, this assessment focuses on health effects associated with exposures to ambient fine particles and to thoracic coarse particles, consistent with EPA's decision in the last review to establish new standards for fine particles separate from those intended to address effects related to thoracic coarse particles. The presentation here first summarizes the qualitative assessment of health evidence contained in the CD, as a basis for development of staff conclusions and recommendations related to primary standards for PM, as discussed in Chapter 5. Secondly, this assessment addresses key issues relevant to quantitative assessment of the epidemiologic health evidence available in this review so as to provide a foundation for quantitative health risk assessment, as discussed in Chapter 4.

In the last review of the PM NAAQS, 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. The epidemiologic evidence for PM-related effects was found to be strong, suggesting a "likely causal role" of ambient PM in contributing to a range of health effects (62 FR 38657). Of special importance in the last review were the conclusions that (1) ambient particles smaller than 10 μm that penetrate into the thoracic region of the respiratory tract remained 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 added 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.

EPA's conclusion in the last review that fine and thoracic coarse particles should be considered as separate pollutants was based on differences in physical and chemical properties, sources, atmospheric formation and transport, relationships with human exposure, and evidence

of health effects (62 FR 38667). In this review, the CD has evaluated the newly available evidence related to the physics and chemistry of particulate matter, exposure relationships, and particle dosimetry. The CD notes that the chemical and physical distinctions between fine and coarse particles recognized in the last review remain generally unchanged; recent studies continue to show that fine and coarse particles generally have different sources and composition and different formation processes (see Table 2-2 herein). Recent exposure research finds that accumulation-mode fine particles can infiltrate into buildings more readily than can thoracic coarse particles, and that ambient concentrations of $PM_{10-2.5}$ are less well correlated and less uniform across a community than ambient concentrations of $PM_{2.5}$ (CD, p. 9-21). The CD also concludes that the new evidence from dosimetry studies continues to reinforce some distinctions between fine and coarse particles, and submodes within fine particles, with regard to deposition patterns in the respiratory tract (CD, p. 9-21). While there is significant overlap between particle size classes, thoracic coarse particles have somewhat greater deposition fractions in the upper regions of the respiratory tract, while fine particles generally (though not the larger accumulation-mode particles) are more likely to be deposited in the alveolar region than are thoracic coarse particles (CD, p. 9-21). Based on these considerations, the CD concludes that it remains appropriate to consider fine and thoracic coarse particles as separate subclasses of PM (CD, p. 9-22).

The assessment of health evidence in this chapter therefore focuses on health effects associated with fine and thoracic coarse particles. This assessment is based on the CD's evaluation and conclusions on the body of evidence from health studies, summarized in Chapters 6 through 9 of the CD, with particular emphasis on the integrative synthesis presented in Chapter 9. That integrative synthesis focuses on integrating newly available scientific information with that available from the last review, as well as integrating information from various disciplines, so as to address a set of issues central to EPA's assessment of scientific information upon which this review of the PM NAAQS is to be based. It is intended to provide a coherent framework for assessment of human health effects posed by ambient PM in the U.S., and to facilitate consideration of the key, policy-related issues to be addressed in this Staff Paper, including recommendations as to appropriate indicators, averaging times, levels, and forms for PM NAAQS. As described in section 9.1 of the CD, the integrative synthesis focuses not only on

what has been learned since the last review, but also highlights important uncertainties that remain and the value of continuing PM research efforts in a number of areas.

As summarized in Chapters 6 through 9 of the CD, 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, toxicologic, controlled human exposure, and dosimetry studies. As was true in the last review, evidence from epidemiologic studies plays a key role in the CD's evaluation of the scientific evidence. As discussed further in section 3.3, some highlights of the new evidence 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 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 thoracic coarse 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 or physiological changes.

- 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 lead to a better understanding of the relationship between ambient PM levels and personal exposures to PM of ambient origin.

- 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).

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

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

In addition, the body of evidence on PM-related effects has greatly expanded with findings from studies that help inform mechanism of action, including important new dosimetry, toxicologic and controlled human exposure studies.

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

- Dosimetry studies using new modeling methods 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.

In presenting that evidence and conclusions based on it, this chapter first summarizes information from the CD's evaluation of health evidence from the different disciplines. Sections 3.2 and 3.3 provide overviews of the CD's findings on the evidence of potential mechanisms for PM-related effects and on the nature of effects associated with PM exposures, respectively. Drawing from the integration of evidence in Chapter 9 of the CD, the chapter summarizes the CD's integrative findings and conclusions regarding causality in section 3.4, with a particular focus on results for fine and thoracic coarse particles. Section 3.5 also draws from the CD's integrative synthesis to characterize potential at-risk subpopulations and potential public health impacts of exposure to ambient PM. Finally, section 3.6 addresses several key issues relevant to the staff's interpretation and quantitative assessment of the health evidence, including: (1) considerations related to air quality measurements and data used in the health studies; (2) exposure error in fine and thoracic coarse particle studies; (3) specification of models used in epidemiologic studies; (4) approaches to evaluating the role of co-pollutants and potential confounding in PM-effects associations; (5) questions of temporality in associations between air quality and health effects, including lag periods used in short-term exposure studies and the selection of time periods used to represent exposures in long-term exposures studies; and (6) questions related to the form of concentration-response relationships and potential threshold

levels. In this final section, staff builds upon the CD's detailed evaluation and integration of the scientific evidence on these issues to reach conclusions regarding the use of the health study results in quantitative evaluation and risk assessments that inform staff recommendations on potential revisions to the primary PM NAAQS presented in Chapter 5.

3.2 MECHANISMS

This section provides an overview of evidence presented in the CD on potential mechanisms by which exposure to PM may result in effects, drawing from Chapters 6 and 7 of the CD. Evidence from dosimetry studies has played a key rôle in previous PM NAAQS reviews, especially in the decision to revise the indicator from TSP to PM_{10} to focus on thoracic particles (52 FR 24634, July 1, 1987). In contrast, in previous reviews of the PM NAAQS there has been little available evidence on potential biological mechanisms by which deposited particles could affect the lungs or heart.

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. Particles must be deposited and retained in the respiratory tract for biological effects to occur (CD, p. 6-1). Briefly, 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.

Fine particles, including accumulation mode and ultrafine prticles, and thoracic coarse particles can all penetrate into and be deposited in the alveolar and tracheobronchial regions of the respiratory tract, though there are differences among these size fractions. The CD finds that deposition patterns are generally similar for ultrafine and coarse particles, with a large fraction of particles being deposited in the extrathoracic region. Removal of particles by the extrathoracic region is less efficient for accumulation-mode fine particles, and thus penetration is increased to the tracheobronchial and alveolar regions (CD, 6-105). The CD concludes that fractional deposition into the alveolar region of the respiratory system for healthy individuals is greatest for particles in the size ranges of approximately 2.5 to 5 μm and 0.02 to 0.03 μm, and fractional

deposition to the tracheobronchial region is greatest for particles in the size range of approximately 4 to 6 μm (CD, p. 6-109). 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 and thoracic coarse particles show enhanced deposition of particles at airway bifurcations (CD, p. 6-20).

Breathing patterns and respiratory disease status can affect regional particle deposition patterns. 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. 6-34). In such cases, 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 (see section 3.5).

The potential effects of deposited particles are influenced by the speed and nature of removal. The predominant clearance and translocation mechanisms vary across the three regions of the respiratory system. For example, dissolution or absorption of particles or particle constituents and endocytosis by cells such as macrophages are two primary mechanisms operating in the alveolar region. These mechanisms also apply in the tracheobronchial region, where two key additional mechanisms for particle clearance or translocation are mucociliary transport and coughing (CD, 6-44, Table 6-2). Soluble components of particles may also move into the circulatory system and thus throughout the body. Recent studies have also suggested that ultrafine particles may be able to move directly from the lungs into the systemic circulation, providing a pathway by which ambient PM exposure could rapidly affect extrapulmonary organs (CD, p. 6-55).

In summary, new evidence from dosimetry studies has advanced our understanding of the complex and different patterns of particle deposition and clearance in the respiratory tract exhibited by fine particles in the accumulation mode, ultrafine particles, and thoracic coarse particles. The evidence shows that all size fractions of thoracic particles can enter the tracheobronchial or alveolar regions of the respiratory system and potentially cause effects.

A major research need identified in the last review was the need to understand the potential biological mechanisms by which deposited particles could result in the varying effects

observed in epidemiological studies with PM exposure. New evidence from toxicologic and controlled human exposure studies has helped to identify and provide support for a number of potential pathways by which particles could have biological effects, as discussed in Chapter 7 of the CD. 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 evidence summarized in the CD has provided important insights that contribute to the plausibility of effects observed in community health studies, 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 ambient air would act alone through any single pathway of response. Accordingly, it is plausible that several health 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 CD developed a summary of current thinking on pathophysiological mechanisms for the effects related to PM exposure. Section 7.10.1 of the CD discusses a series of potential mechanisms or general pathways for effects on the heart and lung, and the CD's conclusions on the evidence supporting different types of effects is briefly 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 CD highlights the variability

of results that exist among different approaches, investigators, animal models, and even day-to-day within studies. Nonetheless, the CD states that "Findings since 1996 have provided evidence supporting many hypotheses regarding induction of PM effects; and this body of evidence has grown substantially." (CD, p. 7-205). For the most part, the evidence from toxicologic and controlled human exposure studies discussed below reflects the effects of fine particles or fine particle constituents.

Direct Pulmonary Effects. Potential pathways for direct pulmonary effects include: lung injury and inflammation; increased airway reactivity and asthma exacerbation; and increased susceptibility to respiratory infections. The CD finds "particularly compelling" evidence that PM exposure causes lung injury and inflammation. Evidence that supports hypotheses on direct pulmonary effects includes toxicologic 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 that ambient particles can cause lung inflammation and injury (CD, p. 7-48). 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 7-46, 7-47). Administration of residual oil fly ash (ROFA, an example of a combustion source particle type) has been shown to produce acute lung injury and severe inflammation, with effects including recruitment of neutrophils, eosinophils and monocytes into the airway (CD, p. 7-60). New toxicologic or controlled human exposure studies using exposure to CAPs have reported some evidence of inflammatory responses in animals, as well as increased susceptibility to infections, though the results of this group of studies are more equivocal (CD, p. 7-85). *In vitro* studies, summarized in section 7.4.2 of the CD, also report evidence of lung injury, inflammation, or altered host defenses with exposure to ambient particles or particle constituents. 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, section 7.3.5). Finally, some new evidence suggests that particles can initiate neurogenic responses in

the respiratory system. For example, several studies have indicated that some particles can activate sensory nerve receptors in the airways, leading to inflammatory responses such as cytokine release (CD, section 7.4.4.4)

Systemic Effects Secondary to Lung Injury. Adding to the list of direct pulmonary effects, these pathways include: impairment of lung function leading to heart injury; pulmonary inflammation and cytokine production leading to systemic hemodynamic effects; lung inflammation leading to increased blood coagulability; and lung inflammation leading to hematopoiesis effects. 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. 7-209 to 7-212).

Effects on the Heart. In addition, potential pathways for effects on the heart include: effects on the heart from uptake of particles or particle constituents in the blood; and effects on the autonomic control of the heart and circulatory system. In the last review, little or no evidence was available on potential cardiovascular effects from toxicologic studies. More recent studies have provided some initial evidence that particles can have direct cardiovascular effects. As shown in Figure 7-1 of the CD, 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. 7-8). 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. 6-55). The CD concludes that the data remain limited but provide some new insights into mechanisms by which particles, primarily fine particles, could affect the cardiovascular system (CD, 7-35, 7-212).

The above list of potential mechanisms 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. Some

mechanistic evidence is available, however, for potential carcinogenic or genotoxic effects of particles. Section 7.10.1 of the CD also includes a discussion of the evidence for mutagenic or genotoxic effects of particles or particle constituents, concluding that "both ambient PM and combustion products of coal, wood, diesel, and gasoline are mutagenic/genotoxic." (CD, p.7-215).

While new evidence is available from studies exposing animals or humans to ambient fine particles, many toxicologic and controlled human exposure studies have used exposures to fine particle constituents or emission-related particles, such as fly ash or diesel exhaust particles. The evidence related to particle types or components is summarized in section 7.10.2 of the CD. Overall, the findings indicate that different health responses are linked with different particle characteristics, and that both individual components and the complex particle mixtures appear to be responsible for many biological responses (CD, p. 7-206).

Particles may also help carry other airborne substances into the respiratory tract, as summarized in section 7.9 of the CD. Particles can take up moisture and grow in the humid atmosphere of the respiratory tract, thus potentially altering the deposition and clearance patterns of the particles. Water-soluble gases can be carried into the lung on particles, and delivery of reactive gases such as SO_2 and formaldehyde to the lower respiratory regions can be increased when carried on particles since these gases would otherwise be more likely trapped in the upper airways. Particles can also carry reactive oxygen species, such as hydrogen peroxide, and other toxic compounds such as polynuclear aromatic hydrocarbons or allergens, into the lower respiratory regions (CD, pp, 7-203, 7-204).

Beyond the dosimetric evidence summarized above, few studies have assessed potential biological mechanisms for effects seen with $PM_{10-2.5}$, for either acute or chronic exposures (CD, p. 9-55). However, the CD includes results from a few new toxicologic studies that assess the effects of thoracic coarse particles. Section 7.4.2 of the CD includes discussion of two studies that report inflammatory responses in cells exposed to ambient thoracic coarse particles collected in Chapel Hill, NC, that appeared to be linked to the endotoxin content of the particles (CD, pp. 7-83, 7-102). A study in Japan also reported effects on immune cells with exposure to resuspended coarse particles (CD, p. 7-135). Another research group exposed blood cells to

ambient fine and thoracic coarse particles, and reported greater effects with fine particles (CD, p. 7-102).

Many of the newer studies use high doses (in mg or hundreds of μg), though some have used doses that are close to ambient concentrations. A key consideration for evaluating the results of animal toxicologic studies is the relation between effects reported with high dose exposures to animals to effects that would be expected in human populations with ambient exposures. The CD presents an illustrative set of analyses evaluating the doses and responses reported in human and animal studies in Appendix 7A of the CD. In the analyses, dosimetry models were used to predict doses of deposited and retained particles in various regions of the respiratory system for humans and rats. In this series of analyses, the dose ratios for humans to rats were quite variable across dose metrics and respiratory system regions. For example, using data from combustion particle (residual oil fly ash) exposures, the equivalent exposure ratios for rats to humans in Table 7A-8a of the CD range from about 0.1 to 16 (CD, p. 7A-34). Using particle number and surface area-based dose metrics resulted in a broader range of equivalent exposure ratios, for example, ranging from 0.008 to 1,300 for particle surface area (CD, p. 7A-36). The CD also evaluated relative dose levels using data from two sets of studies in which toxicologic and controlled human exposure studies used the same type of ambient particles (Utah Valley dust and concentrated ambient particles). Tables 7A-11a through 7A-11c in the CD show estimations for both deposited or retained doses in the alveolar and tracheobronchial regions for three scenarios. In each case the differences between humans and rats is not overly large; for example, deposited doses were roughly two- to four-fold higher for rats than for humans in analyses from inhalation exposure studies using concentrated ambient particles (CD, pp. 7A-52, 7A-53). Recognizing the limitations of this small set of illustrative analyses, the CD concludes that larger doses in rats may be dosimetrically equivalent to lower doses in humans, given the faster particle clearance rates in rats (CD, p. 7A-62). However, the CD also observed that the prediction of dose levels depends on a number of factors, and estimated equivalent exposure ratios for rats and humans vary substantially (CD, 7-163).

In summary, while investigation of potential mechanisms for the effects of particles remains an important research question, new mechanistic studies provide evidence to support a number of hypothesized mechanisms of action. In evaluating this new body of evidence, the CD

more than 80 new time-series studies of the relationship between short-term exposure to PM and mortality have been published, including several multi-city studies that are responsive to the recommendations from the last review (CD, p. 8-23).

In the last review, much consideration was given to assessing the relative roles of PM and co-pollutants, acting alone and in combination, in producing the associations with adverse health effects in epidemiologic studies. Much attention was focused on a series of analyses and reanalyses using data from one U.S. city, Philadelphia, which reported associations between mortality and TSP and gaseous co-pollutants. However, 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 (Samet et al., 1997; EPA, 1996a, p. 13-56). Indeed, the limitations of even the most comprehensive single-city analyses precluded definitive conclusions concerning the role of PM. The results of reanalyses of these data were reviewed by an expert panel, the Health Effects Institute review panel, who 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). The summary report from this panel recommended that future research into the role of co-pollutants should improve upon the examination of multiple single-city studies by different investigators and by conducting multi-city studies, using consistent analytical approaches across cities. Consistent with these views, the 1996 CD and Staff Paper examined the consistency and coherence of reported effects across studies of individual cities having different pollutant mixtures, climate, and other factors.

In this review, the CD has emphasized the results of the multi-city studies as being 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).

The National Morbidity, Mortality and Air Pollution Study (NMMAPS) is the largest available multi-city analysis, and included analyses of PM_{10} effects on mortality in 90 U.S. cities (Samet et al., 2000a,b; Dominici et al., 2003a). Additional, more detailed, analyses were conducted in a subset of the 20 largest U.S. cities (Samet et al., 2000b). The NMMAPS study was, in fact, designed to use a multi-city approach such as that recommended above (Samet et al., 2000c, p. 1). 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. The authors reported associations between total and cardiorespiratory mortality and PM_{10} that were robust to different modeling approaches and to adjustment for gaseous co-pollutants. For total mortality, the overall risk estimate for all cities is a statistically significant increase of 1.4% (using more stringent GAM) or 1.1% (using GLM) per 50 $\mu g/m^3$ PM_{10}, lagged one day (Dominici et al., 2003a; CD, p. 8-33).. Key components to the NMMAPS analyses include assessment of the potential heterogeneity in effects and effects of co-pollutants, as discussed below in sections 3.4.3 and 3.6.4, respectively.

Another major multi-city study used data from 10 U.S. cities where every-day PM_{10} monitoring data were available (in many areas, monitoring is done on a 1-in-3 or 1-in-6 day basis) (Schwartz, 2003b). The authors reported a statistically significant association between PM_{10} and total mortality, with an effect estimate of an increase of 3.4% per 50 $\mu g/m^3$ PM_{10} (in reanalyzed GAM results) or 2.8% per 50 $\mu g/m^3$ PM_{10} (using GLM) (Schwartz, 2003b; CD, p. 8-38). The CD observes that the effect estimates from this study are larger than those reported in NMMAPS, and suggests that the availability of more frequent monitoring data may partly account for the differences (CD, p. 8-39).

In the previous review, results for one key multi-city study were available, in which associations were assessed between daily mortality and PM, using fine and thoracic coarse particle measurements from six U.S. cities (the "Six Cities" study) (Schwartz, et al., 1996). The authors reported significant associations for total mortality with $PM_{2.5}$ and PM_{10}, but not with $PM_{10-2.5}$. Reanalyses of Six Cities data have reported results consistent with the findings of the original study, with statistically significant increases in total mortality ranging from 2% to over

3% reported for results from more stringent GAM or GLM analyses using either $PM_{2.5}$ (per 25 $\mu g/m^3$ increment) or PM_{10} (per 50 $\mu g/m^3$ increment), whereas $PM_{10-2.5}$ was not significantly associated with mortality (Schwartz, 2003a; Klemm and Mason, 2003; CD, p. 8-40).

Using data for the eight largest Canadian cities, mortality was associated with $PM_{2.5}$, PM_{10}, and $PM_{10-2.5}$ and the effect estimates were of similar magnitude for each PM indicator (Burnett et al., 2000; Burnett and Goldberg, 2003). Using either more stringent GAM or GLM, the authors reported increases ranging from 2% to 3% in total mortality for each PM indicator. The association between mortality and $PM_{2.5}$ generally remained statistically 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. Associations with PM_{10}, and $PM_{10-2.5}$ did not reach statistical significance, though the effect estimates were similar in magnitude to those for $PM_{2.5}$. While the associations reported with $PM_{10-2.5}$ were somewhat increased in magnitude in reanalyses, they did not reach statistical significance. The CD concludes that it is difficult to compare the relative significance of associations with $PM_{2.5}$ and $PM_{10-2.5}$, but for this study, "overall, they do not appear to be markedly different" (Burnett and Goldberg, 2003; CD, p. 8-42).

The CD also highlights results of analyses from a major European multi-city study, the Air Pollution and Health: A European Approach (APHEA) study, that evaluated associations between mortality and various PM measures (CD, section 8.2.2.3.3). In the analyses that included data from 29 European cities, overall effect estimates of 2 to 3% increased risk of mortality per 50 $\mu g/m^3$ PM_{10} were reported; reanalysis produced essentially identical results to those of the initial studies (Katsouyanni et al., 2003; CD, p. 8-47).

Numerous studies have been conducted in single cities or locations in the U.S. or Canada, as well as locations in Europe, Mexico City, South America, Asia and Australia (Table 8A in the CD). As was observed based on the more limited studies available in the last review, the associations reported in the recent studies on short-term exposure to PM_{10} and mortality are largely positive, and frequently statistically significant. Overall, the CD concludes that multi-city studies in the U.S., Canada, and Europe reported statistically significant associations with effect estimates ranging from ~1.0 to 3.5% increased risk of total mortality per 50 $\mu g/m^3$ PM_{10}, and from 2 to over 3% increased risk in mortality per 25 $\mu g/m^3$ $PM_{2.5}$ (CD, p. 8-50). Combining

total mortality effect estimates from many individual-city studies with those from the multi-city studies, the CD finds that they generally fall in the range of ~1.0 to 8.0% per 50 $\mu g/m^3$ PM_{10} (CD, p. 8-337).

Effect estimates from U.S. and Canadian multi-city and single-city studies are presented in Figure 3-1 for associations between PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ and mortality.[5] Figure 3-1 shows that, for $PM_{2.5}$, almost all effect estimates are positive and a number are statistically significant, particularly when focusing on the results of studies with greater precision. As summarized in the CD, effect estimates for total mortality from the multi-city studies range from ~1 to 3.5% per 25 $\mu g/m^3$ $PM_{2.5}$, and from approximately 2 to 6% per 25 $\mu g/m^3$ $PM_{2.5}$ from the relatively more precise single-city studies (CD, p. 9-28). Figure 3-1 also shows effect estimates for $PM_{10-2.5}$ that are generally positive and similar in magnitude to those for $PM_{2.5}$ and PM_{10}, but for total mortality, none reach statistical significance. Staff notes that on a unit mass basis, the effect estimates for both $PM_{2.5}$ and $PM_{10-2.5}$ are generally larger than those for PM_{10}, which is consistent with $PM_{2.5}$ and $PM_{10-2.5}$ having independent effects (CD, p. 9-25).

In general, effect estimates are somewhat larger for respiratory and cardiovascular mortality than for total mortality. In the NMMAPS analyses using data from the 20 largest U.S. cities, the effect estimates for deaths from cardiorespiratory causes were somewhat larger than those for deaths from all causes (1.6% versus 1.1% increased risk per 50 $\mu g/m^3$ PM_{10}, using GLM) (Domenici, 2003; CD, p. 8-78). In Figure 3-1, for all three PM indicators, it can be seen that not only is the effect estimate size generally larger for cardiovascular mortality, but the effect estimates are also more likely to reach statistical significance. This is particularly true for $PM_{10-2.5}$, where two of the five effect estimates for cardiovascular mortality shown are positive and statistically significant (Mar et al., 2003; Ostro et al., 2003). For respiratory mortality, effect estimates are often larger than those for either total or cardiovascular mortality, but they are often less precise, which would be expected since respiratory deaths comprise a small

[5] The effect estimates in Figure 3-1 (for mortality effects) and in Figure 3-2 (for morbidity effects; discussed below in section 3.3.2) have been plotted in order of decreasing study power, using as an indicator the natural log of the product of the number of study days and number of health events per day.

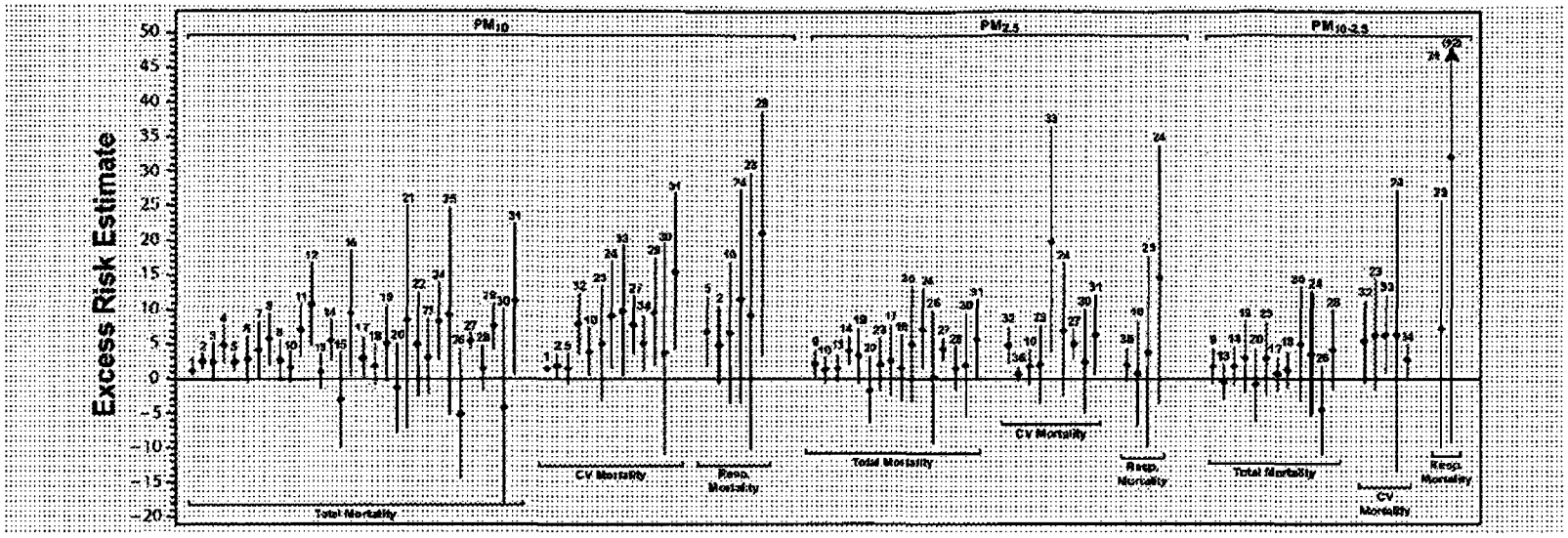

Figure 3-1. Excess risk estimates for total nonaccidental, cardiovascular, and respiratory mortality in single-pollutant models for U.S. and Canadian studies, including aggregate results from two multicity studies (denoted in bold print below). PM increments: 50 μg/m³ for PM_{10} and 25 μg/m³ for $PM_{2.5}$ and $PM_{10-2.5}$. Results presented from time-series studies that did not use GAM or were reanalyzed using GLM. (Source: CD Figure 9-4)

1. **Dominici et al. (2003a), 90 U.S. cities**
2. Moolgavkar (2003), Cook County
3. Kinney et al. (1995), Los Angeles
4. Schwartz (2003b), Chicago
5. Ito and Thurston (1996), Cook County
6. Schwartz (2003b), Pittsburgh
7. Styer et al. (1995), Cook County
8. Schwartz (2003b), Detroit
9. **Burnett and Goldberg (2003), 8 Canadian cities**
10. Moolgavkar (2003), Los Angeles
11. Schwartz (2003b), Seattle
12. Schwartz (2003b), Minneapolis
13. Klemm and Mason (2003), St. Louis
14. Klemm and Mason (2003), Boston
15. Schwartz (2003b), Birmingham
16. Schwartz (2003b), New Haven
17. Chock et al. (2000), Pittsburgh (< 75 y.o.)
18. Chock et al. (2000), Pittsburgh (75+ y.o.)
19. Klemm and Mason (2003), Kingston-Harriman
20. Klemm and Mason (2003), Portage
21. Schwartz (2003b), Canton
22. Schwartz (2003b), Spokane
23. Ito (2003), Detroit
24. Fairley (2003), Santa Clara County
25. Schwartz (2003b), Colorado Springs
26. Klemm and Mason (2003), Topeka
27. Tsai et al. (2000), Newark
28. Klemm and Mason (2003), Steubenville
29. Pope et al. (1992), Utah Valley
30. Tsai et al. (2000), Elizabeth
31. Tsai et al (2000), Camden
32. Lipfert et al. (2000), Philadelphia
33. Mar et al. (2003), Phoenix
34. Ostro et al. (2003), Coachella Valley
35. Klemm and Mason (2000), Atlanta
36. Ostro et al. (1995), Southern California

proportion of total deaths. The CD concludes that effect estimates fall in the range of 3 to 7% per 25 μg/m^3 $PM_{2.5}$ for cardiovascular or cardiorespiratory mortality, and 2 to 7% per 25 μg/m^3 $PM_{2.5}$ for respiratory mortality in U.S. and Canadian cities. The magnitude of the effect estimates for $PM_{10-2.5}$ are similar to those for $PM_{2.5}$, generally falling in the range of 3 to 8% for cardiovascular mortality and 3 to 16% for respiratory mortality per 25 μg/m^3 $PM_{10-2.5}$ (CD, p. 8-306).

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. While effect estimates for different PM indicators may not be quantitatively comparable, the CD observes that "many of the newly reported analyses continue to show statistically significant associations between short-term (24-hr) 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. 8-24). These effect estimates are generally within (but toward the lower end of) the range of PM_{10} estimates previously reported in the 1996 PM AQCD.

As discussed in section 8.2.2.5 of the CD, associations have been reported between mortality and short-term exposure to a number of PM components, especially fine particle components. Recent studies have evaluated 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 (for example, combustion and crustal sources). These studies have suggested that fine particles of some source types, especially combustion sources, may contribute more to associations with mortality than other particles, such as those from crustal material in fine particles (CD, p. 8-85).

The evidence from time-series studies is also buttressed by findings of several "intervention studies" that have assessed improvement in health in areas where policy, economic or regulatory changes resulted in reduced air pollutant concentrations (section 8.2.3.4 in the CD). Studies conducted in Dublin and Hong Kong reported reduced mortality risk following regulations that banned the use of bituminous coal and reduced sulfur in fuel oil, respectively, though it was difficult to distinguish effects of reductions in the individual pollutants.

Overall, the CD finds that the expanded body of evidence provides "especially strong" evidence for associations between short-term exposure to thoracic particles and mortality (CD, p. 8-335). From the full body of multi-city and single-city studies, the CD observes that "many of the newly reported analyses 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. 8-24).

3.3.1.2 Mortality and Long-term PM Exposure

In the 1996 PM CD, 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 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). New studies discussed in the CD (section 8.2.3) include a comprehensive reanalysis of data from the Six Cities and ACS studies, new analyses using updated data from the AHSMOG and ACS studies, and a new analysis using data from a cohort of veterans. Effect estimates from all four of these studies are provided in Appendix 3B.

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. The reanalysis investigators replicated the original results, confirming the original investigators' findings of associations with both total and cardiorespiratory mortality (Krewski et al., 2000; CD, p. 8-95). In single-pollutant models, none of the gaseous co-pollutants was significantly associated with mortality except SO_2. The reanalyses included multi-pollutant models with the gaseous pollutants, and the associations between mortality and both fine particles and sulfates were unchanged in these models, except for those including SO_2. SO_2 is a precursor for fine particle sulfates, making it difficult to distinguish effects of SO_2 and sulfates or fine particles (CD, p. 9-37). 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 (Krewski et al., 2000; CD, p. 8-95).

The extended analyses for the ACS cohort study included follow-up health data and air quality data from the new fine particle monitoring network for 1999-2000, and reported significant associations between long-term exposure to fine particles (using various averaging periods for air quality concentrations) and premature mortality from all causes, cardiopulmonary diseases, and lung cancer (Pope et al., 2002; CD p. 8-102). 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 for the PM-mortality relationship (e.g., new methods for spatial smoothing and random effects models in the Cox proportional hazards model) (CD, p. 8-97). 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); however, as was found in the reanalysis of the original ACS study, education level was found to be an effect modifier, in that larger and more statistically significant effect estimates were reported in the group with the lowest education level (Pope et al., 2002; CD, p. 8-104). In both the reanalyses and extended analyses of the ACS cohort study, long-term exposure to $PM_{10-2.5}$ was not significantly associated with mortality (Krewski et al., 2000; Pope et al., 2002).

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 to the original study in which no statistically significant results were reported with TSP, a significant association was reported between total mortality and PM_{10} for males, but not for females (CD, pp. 3-41, 3-42). 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 the estimates were generally not statistically significant (McDonnell et al., 2000; CD, p. 8-117). In the VA cohort study, analyses were done using subsets of PM exposure and mortality time periods, and 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 (Lipfert et al., 2000b).

Based on an evaluation of all the available long-term exposure studies, the CD places greatest weight on the results of the Six Cities and ACS studies. In so doing, the CD notes that the Six Cities and ACS studies (including reanalyses and extended 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, pp. 8-116; 9-33).

One new effect reported in the extended analysis of the ACS study was a statistically significant association between fine particle and sulfate concentrations and lung cancer mortality, with a 13% increased risk of lung cancer mortality per 10 $\mu g/m^3$ $PM_{2.5}$, using air quality data averaged across all available years (CD, p. 8-99). This effect estimate is little changed and remains significant with adjustment for covariates, random effects modeling and spatial smoothing methods (CD, Figure 8-8). Also, 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 (CD, p. 8-317).

The epidemiologic findings of associations between fine particles and lung cancer mortality are supported by the results of recent toxicologic studies that have examined the mutagenic potential of ambient particles. These toxicologic studies have provided evidence of mutagenicity or genotoxicity with exposure to combustion-related particles or to ambient particles collected in Los Angeles, Germany and the Netherlands (CD, p. 9-76). 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 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. 8-318). A number of toxicologic studies, summarized in section 7.10.1 of the CD, report evidence of genotoxicity or mutagenicity with particles. The CD also finds that the evidence indicates that fine particles may be more mutagenic than thoracic coarse particles (CD, p. 7-214), which is consistent with the evidence from epidemiologic studies. Considered with the results of toxicologic studies, the CD finds that

this new evidence supports the plausibility of a relationship between fine particles and lung cancer mortality (CD, p. 9-78).

Thus, emphasizing the results from the Six Cities and ACS cohorts, the CD finds that there are significant associations for mortality with long-term exposure to $PM_{2.5}$. The effect estimates for deaths from all causes fall in a range of 6 to 13% increased risk per 10 $\mu g/m^3$ $PM_{2.5}$, while effect estimates for deaths from cardiopulmonary causes fall in a range of 6 to 19% per 10 $\mu g/m^3$ $PM_{2.5}$. For lung cancer mortality, the effect estimate was a 13% increase per 10 $\mu g/m^3$ $PM_{2.5}$ in the results of the extended analysis from the ACS cohort (Pope etl al., 2002; CD, Table 8-12). In addition, based on evidence from reanalyses and extended analyses using ACS cohort data, the CD concludes that the long-term exposure studies provide evidence that long-term exposure to thoracic coarse particles is not associated with mortality (CD, p. 8-307).

3.3.2 Morbidity

The epidemiologic evidence also includes associations between various indicators of PM and a wide range of endpoints reflecting both respiratory- and cardiovascular-related morbidity effects. The following sections summarize the CD's findings on PM-related morbidity effects, beginning with hospital admissions and medical visits for respiratory and cardiovascular diseases. Subsequent sections provide overviews of the CD's evaluation of evidence for effects on the respiratory and cardiovascular systems. Effect estimates for associations between short-term exposure to $PM_{2.5}$ or $PM_{10-2.5}$ with hospitalization and medical visits from U.S. and Canadian studies are presented below in Figure 3-2. Appendix 3A includes effect estimates for associations with hospitalization and medical visits, as well as those for respiratory symptoms and lung function and physiological cardiovascular effects, with short-term exposures to PM_{10}, $PM_{2.5}$ or $PM_{10-2.5}$ from U.S. and Canadian studies. 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 CD in section 8.3, and a more complete discussion of all studies is available in Appendix 8B of the CD.

3.3.2.1 Hospitalization and Medical Visits

Numerous recent studies have continued to report significant associations between short-term exposures to PM and hospital admissions or emergency department visits for respiratory or

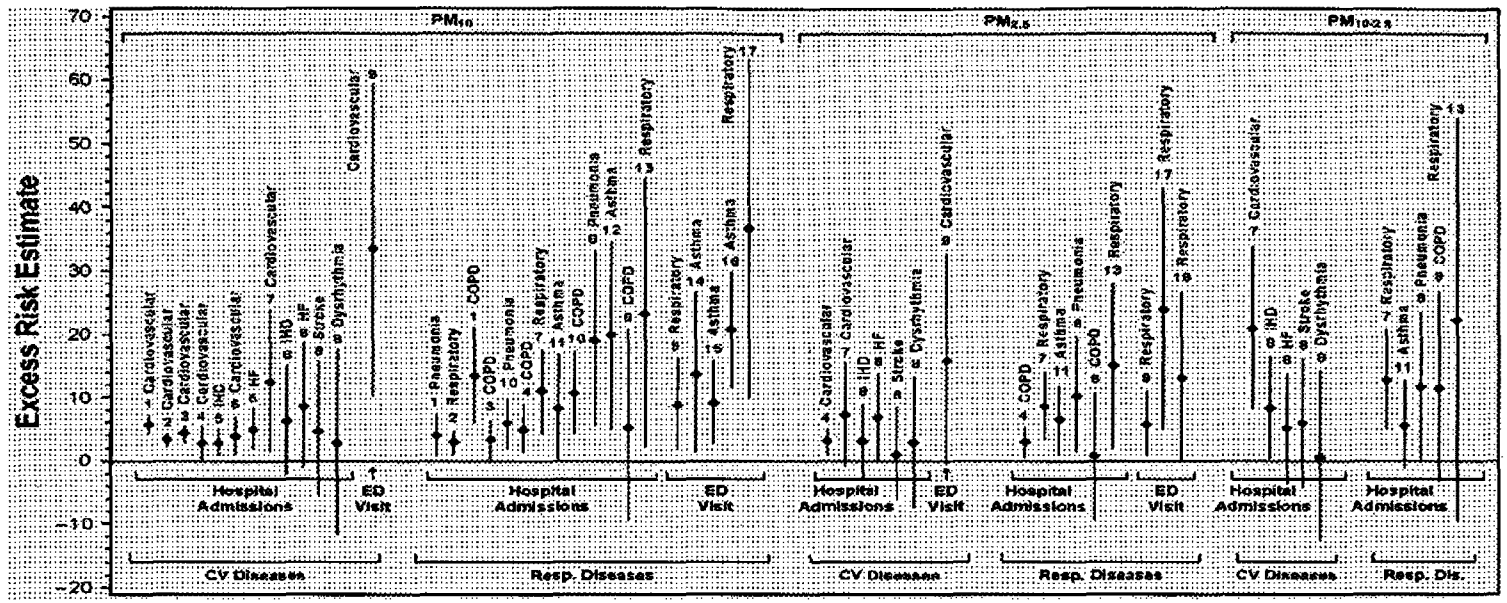

Figure 3-2. Excess risk estimates for hospital admissions and emergency department visits for cardiovascular and respiratory diseases in single-pollutant models from U.S. and Canadian studies, including aggregate results from one multicity study (as denoted in bold below). PM increments: 50 μg/m³ for PM_{10} and 25 μg/m³ for $PM_{2.5}$ and $PM_{10-2.5}$. Results presented from time-series studies that did not use GAM or were reanalyzed using GLM. PM effect size estimate (± 95% confidence intervals) are depicted for the studies listed below. (Source: CD Figure 9-5)

1. **Zanobetti and Schwartz (2003) U.S. 14 cities**
2. Linn et al. (2000), Los Angeles
3. Moolgavkar (2003), Cook County
4. Moolgavkar (2003), Los Angeles
5. Schwartz and Morris (1995), Detroit
6. Morris and Naumova (1998), Chicago
7. Burnett et al. (1997), Toronto
8. Ito (2003), Detroit
9. Stieb et al. (2000), St. John
10. Schwartz (1994), Detroit
11. Sheppard (2003), Seattle
12. Nauenberg and Basu (1999), Los Angeles
13. Thurston et al. (1994), Toronto
14. Tolbert et al. (2000), Atlanta
15. Lipsett et al. (1997), Santa Clara County
16. Choudhury et al. (1997), Montreal
17. Delfino et al. (1997), Montreal
18. Delfino et al. (1998), Montreal

cardiovascular diseases. The new studies have included multi-city analyses, numerous assessments using cardiovascular admissions/visits, and evaluation of the effects of fine and thoracic coarse particles.

One new multi-city study, the NMMAPS, included analyses of associations with hospital admissions among the elderly, and reported statistically significant associations between PM_{10} and hospital admissions in the elderly for cardiovascular diseases, pneumonia and chronic obstructive pulmonary disease (COPD) in 14 cities (Samet et al., 2000; Schwartz et al., 2003). Increases of 5% in hospital admissions for cardiovascular disease and 8% and 6% in hospital admissions for COPD or pneumonia, respectively, per 50 $\mu g/m^3$ PM_{10} were reported. In the NMMAPS multi-city analyses on hospitalization for respiratory and cardiovascular diseases, effect estimates with PM_{10} were not correlated with city-specific correlations between PM_{10} and co-pollutant levels, which the authors conclude indicates a lack of confounding by co-pollutants (CD, p. 8-146, 8-175).

Numerous single-city studies have also been published that report associations between short-term PM exposure and hospitalization or medical visits for respiratory diseases. The effect estimates from these studies generally fall in a range of 5 to 20% increased risk per 50 $\mu g/m^3$ PM_{10}, with somewhat higher estimates for asthma visits (CD, p. 8-193). The findings from studies of medical visits for respiratory diseases offer new evidence of acute respiratory effects with exposure to ambient PM (the studies generally used PM_{10}) that provides new insight into the scope of respiratory morbidity (CD, p. 9-180).

Figure 3-2 shows associations between $PM_{2.5}$ and hospitalization or emergency room visits for the general category of respiratory diseases that 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. Similarly, associations between hospital admissions for respiratory and cardiovascular diseases and $PM_{10-2.5}$ are generally positive and, as evident in Figure 3-2, the more precise estimates are statistically significant. Overall, the CD finds that excess risks for

The CD finds that the recent epidemiologic findings are consistent with those of the previous review in showing associations with both respiratory symptom incidence and decreased lung function (CD, p. 9-70). PM_{10} and $PM_{2.5}$ were associated with small decreases in lung function and increases in respiratory symptoms, though the associations were not always statistically significant, and a few new studies reported associations between $PM_{10-2.5}$ and respiratory morbidity. 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 CD concludes that "[t]he fact that instillation of ambient PM collected from different geographical areas has been shown to cause pulmonary inflammation and injury tends to support epidemiologic studies that report increased PM-associated respiratory effects living in some of the same geographical areas" (CD, p. 7-48).

3.3.2.3 Effects on the Respiratory System from Long-term Exposures

In the last review, several studies had reported that long-term PM exposure was linked with increased respiratory disease and decreased lung function. One study, using data from 24 U.S. and Canadian cities ("24 Cities" study), reported associations with these effects and long-term exposure to fine particles or acidic particles, but not with PM_{10} exposure (Dockery et al., 1996; Raizenne et al., 1996). The 1996 Staff Paper included further evaluation of the evidence that indicated no relationship between lung function decrements and long-term exposure to thoracic coarse particles (EPA, 1996b, p. V-67a).

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. In the U.S., studies have been based on data from two cohorts, a cohort of schoolchildren in 12 Southern California Communities and an adult cohort of Seventh Day Adventists (AHSMOG). Results for the new studies, together with the findings available in the last review, are presented in Appendix 3B.

In general, these studies have indicated that long-term exposure to PM, for both PM_{10} or $PM_{2.5}$, is associated with reduced lung function growth and increased risk of developing chronic respiratory illness (CD, p. 8-215). In section 8.3.3.2.2, the CD describes results from the

Southern California cohort, where significant decreases in lung function growth were associated with increasing exposure to PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ in one analysis (Gauderman et al., 2000), while in a second group of children recruited in this cohort there were decreases in lung function growth with long-term exposure to PM_{10} and $PM_{2.5}$ ($PM_{10-2.5}$ data were not included in this study) but the results were generally not statistically significant (Gauderman et al., 2002). In an analysis of cohort participants who moved during the course of the study, those who moved to areas with lower PM concentrations (using PM_{10} as the indicator) showed increased lung function growth, whereas lung function growth decreased in the group of children who moved to areas with high pollution levels (Avol et al., 2001; CD, p. 8-213). A number of long-term studies of respiratory effects also have been conducted in non-North American countries, and many report significant associations between indicators of long-term PM exposure and either decreases in lung function or increased respiratory disease prevalence (Table 8-B8 of the CD).

Considered together, the CD finds that the long-term exposure studies on respiratory morbidity reported positive and statistically associations between fine particles or fine particle components and lung function decrements or chronic respiratory diseases, such as chronic bronchitis (CD pp. 8-313, 8-314). The CD observes that little evidence is available on potential effects of long-term to exposure to $PM_{10-2.5}$ (CD pp. 8-313, 8-314); one analysis from a Southern California cohort suggests a link between decreased lung function and long-term $PM_{10-2.5}$ exposure, but an earlier report from the 24 Cities study finds no such associations.

3.3.2.4 Effects on the Cardiovascular System

In contrast with the limited information available in the previous review, the CD observes that new toxicologic and epidemiologic studies provide much more evidence of effects on the cardiovascular system with short-term exposures to PM (CD, p. 9-67). These new findings help to shed light on biological mechanisms that underlie associations between short-term PM exposure and cardiovascular mortality and hospitalization that have been reported previously. The CD also observes that, while epidemiologic studies have shown associations between long-term exposure to particles, especially fine particles, and cardiovascular mortality, only limited evidence is available on potential cardiopulmonary morbidity responses to long-term PM exposure, or mechanisms underlying such responses (CD, p. 9-69).

Epidemiologic studies have reported associations between short-term exposures to ambient PM (often using PM_{10}) and measures of changes in cardiac function such as arrhythmia, alterations in electrocardiogram (ECG) patterns, heart rate or heart rate variability changes, and incidence of myocardial infarction (CD, p. 8-166). Recent studies have also reported increases in blood components or characteristics such as increased levels of C-reactive protein and fibrinogen (CD, p. 8-169). Several of these studies report significant associations between short-term $PM_{2.5}$ exposures and cardiovascular health indicators. Only one of the new set of studies included $PM_{10-2.5}$, in which significant associations were reported between onset of myocardial infarction and short-term $PM_{2.5}$ exposures but not with $PM_{10-2.5}$ exposures (Peters et al., 2001).

As noted in section 3.2, a number of toxicologic and controlled human exposure studies have reported some similar cardiovascular responses with exposure to different types of particles. In section 9.2.3.2.1, the CD summarizes evidence from both epidemiologic and toxicologic studies on subtle changes in cardiovascular health. These changes include increased blood fibrinogen and fibrin formation, certain ECG parameters (e.g., heart rate variability or HRV), and vascular inflammation. The CD notes that vascular inflammation induces release of C-reactive proteins and cytokines that may cause further inflammatory responses which, on a chronic basis, could lead to atherosclerosis.

Where a series of studies have been conducted in the same location, these studies can provide evidence for coherence of effects, linking results from different study types for exposure to PM in the same airshed. As discussed in the CD, in Boston, epidemiologic associations were reported between $PM_{2.5}$ and incidence of myocardial infarction, increases in recorded discharges from implanted cardiovertex defibrillators, and decreases in HRV measures. Toxicologic studies in Boston, using $PM_{2.5}$ CAPs exposures in dogs, also suggested changes in cardiac rhythm with $PM_{2.5}$ mass and changes in blood parameters with certain $PM_{2.5}$ components (CD, p. 9-68, 9-69).

3.3.3 Developmental effects

Some new evidence is available that is suggestive of adverse effects of exposure to PM and gaseous co-pollutants on prenatal development, including both mortality and morbidity effects. Several recent studies have shown significant associations between PM_{10} concentration averaged over a month or a trimester of gestation and risk of intrauterine growth reduction

(IUGR) and low birth weight. In addition, several new studies have suggested that infant mortality may be associated with exposure to PM and gaseous co-pollutants during gestation. The CD concludes that these effects are emerging as potentially more important than was appreciated in the 1996 CD, but the evidence is still preliminary regarding these effects (CD, pp.8-347).

3.3.4 Summary

In summary, the CD finds that the many new available studies build upon what was previously known, reporting associations between PM exposure, using various PM indicators, with a broad range of cardiovascular and respiratory health endpoints (CD, p. 9-23). The new studies support findings from the last review on associations between PM and cardiorespiratory mortality, hospitalization and emergency department visits for respiratory diseases, respiratory symptoms and decreased lung function. Recent studies also broaden the range of health effects associated with exposure to PM. Evidence for respiratory effects is expanded with studies showing associations with visits to physicians or clinics for respiratory illnesses. New evidence is available to link PM exposure, especially fine particles, with effects on the cardiovascular system, including changes in physiological indicators or biomarkers for cardiovascular health.

3.4 INTEGRATIVE ASSESSMENT OF HEALTH EVIDENCE

In Chapter 9, the CD assesses the new health evidence, integrating findings from epidemiologic studies with experimental (e.g., dosimetric and toxicologic) studies, to make judgments about the extent to which causal inferences can be made about observed associations between health endpoints and various indicators or constituents of ambient PM, acting alone and/or in combination with other pollutants. In evaluating the evidence from epidemiologic studies in section 9.2.2, the CD focuses on well-recognized criteria, including (1) the *strength* of reported associations; (2) the *robustness* of reported associations to the use of alternative model specifications, potential confounding by co-pollutants, and exposure misclassification related to measurement error; (3) the *consistency* of findings in multiple studies of adequate power, and in different persons, places, circumstances and times; (4) *temporality* between exposure and observed effects; (5) the nature of *concentration-response* relationships; and (6) information

from so-called *natural experiments* or intervention studies (CD, p. 9-23). Integrating more broadly across epidemiologic and experimental evidence in section 9.2.3, the CD focuses on the coherence and plausibility of observed PM-related health effects to reach judgments about causality.

The following discussion summarizes the conclusions and judgments from the CD's integrative assessment, focusing first on the strength, robustness, and consistency of the epidemiologic evidence, and ending with a focus on the CD's assessment of coherence and biological plausibility of PM-related health effects. Other related issues, including temporality of effects and the form of PM concentration-response relationships, are discussed below in section 3.6, with a focus on how these issues affect the use of epidemiologic results in the quantitative risk assessments discussed in Chapter 4.

3.4.1 Strength of Associations

Considering the magnitude, statistical significance, and the degree of precision of the effect estimates derived from epidemiologic analyses, the CD finds that the results from recent studies expand and support epidemiologic evidence that was found to be "fairly strong" in the last review (EPA, 1996a, p. 13-92). From the short-term exposure studies, the CD concludes that the "epidemiological evidence is strong" for associations between $PM_{2.5}$ and PM_{10} and total or cardiovascular mortality (CD, p. 9-32). Associations between $PM_{10-2.5}$ and mortality are similar in magnitude, but less precise, than those for $PM_{2.5}$ or PM_{10}; the CD finds this evidence "not as strong" but suggestive of associations with mortality (CD, p. 9-32). For both $PM_{2.5}$ and $PM_{10-2.5}$ there is a series of positive associations with hospitalization and emergency department visits for cardiovascular or respiratory diseases; many are statistically significant, but the associations with $PM_{10-2.5}$ are somewhat less precise than those for $PM_{2.5}$ (CD, p. 9-29). Studies of respiratory symptoms or lung function changes show associations with both fine and thoracic coarse particles (CD, p. 8-343), while the studies of more subtle cardiovascular health outcomes have shown associations with fine, but not thoracic coarse particles. Taken together, the CD concludes that there is strong epidemiological evidence linking short-term exposures to fine particles with a range of cardiorespiratory morbidity and mortality effects. The more limited

evidence on effects of $PM_{10-2.5}$ is suggestive of both mortality and morbidity effects, with greater strength in the evidence for morbidity, especially respiratory morbidity.

For long-term exposures, the evidence supports associations between $PM_{2.5}$ and mortality for cardiovascular and respiratory diseases and lung cancer, as well as the development of chronic respiratory illness and decreased lung function (CD, p. 9-34). For $PM_{10-2.5}$, available studies provide evidence of the absence of associations with mortality. Since long-term exposure morbidity studies have generally not included $PM_{10-2.5}$ data, no conclusions can be drawn regarding long-term exposure to $PM_{10-2.5}$ and morbidity effects (CD, p. 9-34).

3.4.2 Robustness of Associations

In section 9.2.2.2, the CD evaluates the robustness of epidemiologic associations in part by considering the effect of differences in statistical model specification, potential confounding by co-pollutants and exposure error on PM-health associations. The 1996 CD included an assessment of evidence then available on these issues, and concluded that the effects observed in epidemiologic studies "cannot be wholly attributed to" issues such as confounding by co-pollutants, differing model specifications, or measurement error (EPA, 1996a, p. 13-92). These issues have been further evaluated in many new studies available in this review.

As discussed below in section 3.6.3, the CD assesses the findings of studies that evaluated alternative modeling strategies, with a particular focus on the recent set of analyses to address issues related to the use of GAM in time-series epidemiologic studies. The reanalyses included the use of alternative statistical models and methods of control for time-varying effects, such as weather or season. In the results of these reanalyses, some studies showed little change in effect estimates, while others reported reduced effect estimate size, though the CD observes that the reductions were often not substantial (CD, p. 9-35). Overall, the CD concludes that associations between short-term exposure to PM and various health outcomes are generally robust to the use of alternative modeling strategies, though further evaluation of alternative modeling strategies is warranted (CD, p. 9-35). In addition, the reanalysis and extended analyses of data from prospective cohort studies have shown that reported associations between mortality and long-term exposure to fine particles are robust to alternative modeling strategies, as discussed below in section 3.6.3.

The CD also included extensive evaluation of the sensitivity of PM-health responses to confounding by gaseous co-pollutants, as discussed in detail in section 8.4.3 of the CD, and more briefly below in section 3.5.6. In the new multi-city studies, as well as many of the single-city studies, health outcome associations with short-term exposures to PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ are little changed in multi-pollutant models including one or more of the gaseous co-pollutants (CD, p. 8-253). However, in some single-city analyses, PM-health outcome associations were attenuated in multi-pollutant models; the CD observes that collinearity between co-pollutants can make interpretation of multi-pollutant models difficult (CD, p. 8-253). Overall, the CD concludes that these studies indicate that effect estimates for associations between mortality and morbidity and various PM indices are robust to confounding by co-pollutants (CD, p. 9-37).

Finally, as discussed in section 3.6.2, a number of recent studies have evaluated the influence of exposure error on PM-health associations. Exposure error includes both consideration of measurement error, and the degree to which measurements from an individual monitor reflect exposures to the surrounding community. Several studies have shown that fairly extreme conditions (e.g., very high correlation between pollutants and no measurement error in the "false" pollutant) are needed for complete "transfer of causality" of effects from one pollutant to another (CD, p. 9-38). In comparing fine and thoracic coarse particles, the CD observes that exposure error is likely to be more important for associations with $PM_{10-2.5}$ than with $PM_{2.5}$, since there is generally greater error in $PM_{10-2.5}$ measurements, $PM_{10-2.5}$ concentrations are less evenly distributed across a community, and less likely to penetrate into buildings (CD, p. 9-38). Therefore, while the CD concludes that associations reported with PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ are generally robust, the CD recognizes that factors related to exposure error may result in reduced precision for epidemiologic associations with $PM_{10-2.5}$ (CD, p. 9-46).

3.4.3 Consistency

The 1996 CD reported associations between short-term PM exposure and mortality or morbidity from studies conducted in locations across the U.S. as well as in other countries, and concluded that the epidemiologic data base had "general internal consistency" (EPA, 1996a, p. 13-30). This epidemiologic data base has been greatly expanded with numerous studies conducted in single locations, as well as several key multi-city studies. As described above, the

CD finds that the epidemiologic studies generally report positive and often statistically significant associations with various cardiorespiratory health outcomes. The larger body of evidence also has shown more variability in effect estimate size for a given health outcome than was apparent in the last review.

New multi-city studies have allowed evaluation of consistency in effect estimates across geographic locations, using uniform statistical modeling approaches. In the NMMAPS results, effect estimates for many individual cities exhibited wide confidence ranges, with varied effect estimate sizes, that suggested potentially more heterogeneity in effect estimates across cities than had been seen with single-city studies in the last review. However, the authors observed that there was no statistically significant heterogeneity across the effect estimates in the NMMAPS analyses (Samet et al., 2000; Dominici et al., 2003a). The Canadian multi-city study also reported some limited evidence suggesting heterogeneity in responses for $PM_{2.5}$ and $PM_{10-2.5}$ in the reanalysis to address GAM questions, whereas there been no evidence of heterogeneity in initial study findings (Burnett and Goldberg, 2003; CD, p. 9-39). Finally, in the European multi-city, there were differences seen between effect estimates from eastern and western European cities in initial analyses, but these differences were less clear with reanalysis to address GAM issues (Katsouyanni et al., 2003). Overall, the new multi-city study results suggest that effect estimates differ from one location to another, but the extent of heterogeneity is not clear.

The CD discusses a number of factors that would be likely to cause variation in PM-health outcomes in different populations and geographic areas in section 9.2.2.3. The CD recognizes that differences might well be expected in effects across locations, and discusses investigation of a number of factors that appeared to be associated with variation in effect estimates, including indicators of exposure to traffic-related pollution and climate-related increases in exposure to ambient pollution (CD, p. 9-39). Other factors might also be expected to cause variation in observed effects between locations, including population characteristics that affect susceptibility or exposure differences, distribution of PM sources, or geographic features that would affect the spatial distribution of PM (CD, p. 9-41). In addition, the CD observes that 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 (CD, p. 9-40). The Canadian

8-city study, as well, used PM data from a monitoring network that operated primarily on a 1-in-6 day collection schedule, although the data were available for a long time period. In general, use of data collected on every sixth day results in reduced statistical power, resulting in less precision for estimated effect estimates for the individual cities and increased potential variability in results (CD, p. 9-40).

Overall, the CD finds that "[f]ocusing on the studies with the most precision, it can be concluded that there is much consistency in epidemiologic evidence regarding associations between short-term and long-term exposures to fine particles and cardiopulmonary mortality and morbidity." (CD, p. 9-47). The CD also concludes that for short-term exposure to thoracic coarse particles, there is some consistency in effect estimates for hospitalization for cardiovascular and respiratory causes, though fewer studies are available on which to make such an assessment (CD, p. 9-47).

3.4.4 Coherence and Plausibility

Section 9.2.3 of the CD integrates and evaluates evidence from the different health disciplines to draw conclusions regarding the coherence of effects observed in the cardiovascular and respiratory systems, as well as evidence for biological plausibility of these effects. The CD finds that progress has been made in substantiating and expanding epidemiologic findings on cardiovascular- and respiratory-related effects of PM, and in obtaining evidence bearing on the biological plausibility of observed effects and potential mechanisms of action for particles (CD, p. 9-49).

As was concluded in the previous review, in considering evidence from epidemiologic studies using PM_{10} and other PM indicators the CD finds coherence for effects on the cardiovascular and respiratory systems. Figures 8-24 through 8-28 of the CD show effect estimates for associations between short-term exposures to PM_{10} and a range of cardiovascular and respiratory health endpoints from within the same geographic location. This evidence from epidemiologic studies in one location provides some broad support for coherence of effects related to PM. In addition, the new series of toxicologic and controlled human exposure studies using ambient particles (primarily PM_{10}) collected in Utah Valley show inflammatory effects that are consistent with evidence of respiratory effects from the epidemiologic studies (CD, p. 9-71).

Considering epidemiologic evidence for $PM_{2.5}$, the CD finds that epidemiologic studies report associations with a broad range of effects on the cardiovascular and respiratory systems, primarily from short-term exposure studies, but also supported by associations reported for long-term fine particle exposure with cardiovascular mortality (CD, pp. 9-67). As described briefly in section 3.2 above, and in more depth in Chapter 7 of the CD, the findings of new toxicologic and controlled human exposure studies, while still limited, support a number of potential biological mechanisms or pathways for PM-related effects, and this evidence is largely from studies of fine particles or fine particle components. The experimental and epidemiologic evidence together support the biological plausibility of observed effects on the cardiovascular system (CD, p. 9-70). In addition, the CD highlights evidence from a series of epidemiologic and toxicologic studies using ambient $PM_{2.5}$ exposures in Boston that provide evidence of coherence in effects on the cardiovascular system (CD, pp. 9-68, 9-69). The CD observes: "While many research questions remain, the convergence of evidence related to cardiac health from epidemiologic and toxicologic studies indicates both coherence and plausibility in this body of evidence." (CD, p. 9-78). In the last review, evidence was available suggesting coherence of effects on the respiratory system, and the CD finds that new epidemiologic and toxicologic studies expand upon that knowledge, particularly for $PM_{2.5}$ (CD, p. 9-74). In locations where epidemiologic studies have been conducted, toxicologic or controlled human exposure studies using exposures to concentrated ambient particles have shown effects related to lung inflammation, though minimal effects on lung function have been reported (CD, p. 9-72).

As was true in the last review, there is some coherence in epidemiologic evidence linking long-term exposure to fine particles with mortality and effects on the respiratory system. However, toxicologic studies that are currently available have generally not studied effects of long-term or chronic exposures to air pollution, so for the most part, no conclusions can be drawn regarding biological plausibility of observed effects with long-term $PM_{2.5}$ exposures (CD, p. 9-69). However, for lung cancer, the CD summarizes evidence that supports coherence and plausibility in the associations reported between long-term exposures to fine particles and lung cancer mortality. Toxicologic evidence of mutagenicity or genotoxicity of particles lends coherence and plausibility to evidence from epidemiologic studies linking long-term exposure to fine particles with lung cancer mortality (CD, p. 9-76).

Less information is available to allow conclusions to be drawn about coherence or plausibility for associations with $PM_{10-2.5}$. Based on the epidemiologic evidence discussed previously, the CD concludes that the results are suggestive of associations between short-term exposure to $PM_{10-2.5}$ and morbidity effects, including data on hospitalization for respiratory diseases as well as increased respiratory symptoms (CD, p. 9-90). Only limited evidence is available from toxicologic studies of $PM_{10-2.5}$, as noted in section 3.2, though the available evidence does provide some coherence for effects on the respiratory system As discussed above, fractional deposition to the tracheobronchial region is greatest for thoracic coarse particles in the size range of 4 to 6 μm (CD, p. 6-109). This would be consistent with epidemiological evidence linking $PM_{10-2.5}$ with respiratory morbidity, such as increased respiratory symptoms or risk of hospitalization for asthma. In addition, as observed in the CD, reduced precision in $PM_{10-2.5}$ effect estimates may be heavily influenced by the increased error in $PM_{10-2.5}$ measurements and exposure error related to greater spatial variability and reduced penetration indoors, thus larger standard errors would be expected for associations with $PM_{10-2.5}$ than for either PM_{10} or $PM_{2.5}$ (CD, p. 9-91).

3.4.5 Summary

The new evidence from epidemiologic studies builds upon the conclusions of the last review regarding the strength, robustness and consistency of the evidence. While uncertainties remain and the new studies raise some new questions, the CD concludes:

> In conclusion, the epidemiological evidence continues to support likely causal associations between $PM_{2.5}$ and PM_{10} and both mortality and morbidity from cardiovascular and respiratory diseases, based on an assessment of strength, robustness, and consistency in results. For $PM_{10-2.5}$, less evidence is available, but the studies using short-term exposures have reported results that are of the same magnitude as those for PM_{10} and $PM_{2.5}$, though less often statistically significant and thus having less strength, and the associations are generally robust to alternative modeling strategies or consideration of potential confounding by co-pollutants. (CD, p. 9-48).

Much more evidence is now available related to the coherence and plausibility of effects than in the last review. For short-term exposures, the CD finds that the integration of evidence from epidemiologic and toxicologic studies indicates both coherence and plausibility of effects on the cardiovascular and respiratory systems, particularly for fine particles (CD, p. 9-78). Also,

there is evidence supporting coherence and plausibility for the observed associations between long-term exposures to fine particles and lung cancer mortality (CD, p. 9-79). The smaller body of evidence on thoracic coarse particles, especially the limited evidence from toxicologic studies, provides only limited evidence of coherence for effects of thoracic coarse particles. Epidemiologic and dosimetric evidence, along with limited support from toxicologic studies, support associations between $PM_{10-2.5}$ and the respiratory system, with less evidence available on cardiovascular effects.

Finally, the evaluation of these criteria leads the CD to draw conclusions regarding causality of effects seen with fine or with thoracic coarse particles. Overall, the CD concludes that the available evidence supports the general conclusion that $PM_{2.5}$ or fine particle components are "likely causally related to cardiovascular and respiratory mortality and morbidity" (CD, p. 9-79). For $PM_{10-2.5}$, the more limited body of evidence is suggestive of causality between short-term (but not long-term) exposures and various mortality and morbidity effects, with stronger evidence for associations with morbidity (CD, p. 9-79, 9-80).

3.5 PM-RELATED IMPACTS ON PUBLIC HEALTH

The following discussion draws from sections 9.2.4 and 9.2.5 of the CD to characterize subpopulations potentially at risk for PM-related effects and potential public health impacts associated with exposure to ambient PM. In particular, the potential magnitude of at-risk population groups is discussed, along with other key considerations related to impacts on public health, such as the concept of "mortality displacement" or "harvesting."

3.5.1 Potentially Susceptible and Vulnerable Subpopulations

The CD summarizes information on potentially susceptible or vulnerable groups in section 9.2.4. As described there, the term *susceptibility* refers to innate (e.g., genetic or developmental) or acquired (e.g., personal risk factors, age) factors that make individuals more likely to experience effects with exposure to pollutants. A number of population subgroups have been identified as potentially susceptible to health effects as a result of PM exposure, including people with existing heart and lung diseases, including possibly diabetes, older adults and children. In addition, new attention has been paid to the concept of some population groups

having increased *vulnerability* to pollution-related effects due to factors including socioeconomic status (e.g., reduced access to health care or low socioeconomic status) or particularly elevated exposure levels, such as residence near sources such as roadways (CD, p. 9-81). Most available studies have used PM_{10} or other measures of thoracic particles, with little specific evidence on potential susceptibility to effects of $PM_{2.5}$ or $PM_{10-2.5}$.

A good deal of evidence indicates that people with existing heart or lung diseases are more susceptible to PM-related effects. In addition, new studies have suggested that people with diabetes, who are at risk for cardiovascular disease, may have increased susceptibility to PM exposures. This body of evidence includes findings from epidemiologic studies that associations with mortality or morbidity are greater in those with preexisting conditions, as well as evidence from toxicologic studies using animal models of cardiopulmonary disease (CD, section 9.2.4.1).

Two age groups, older adults and the very young, are also potentially at greater risk for PM-related effects. Epidemiologic studies have generally not shown striking differences between adult age groups. However, some epidemiologic studies have suggested that serious health effects, such as premature mortality, are greater among older populations (CD, p. 8-328). In addition, preexisting respiratory or cardiovascular conditions are more prevalent in older adults than younger age groups; thus there is some overlap between potentially susceptible groups of older adults and people with heart or lung diseases.

Epidemiologic evidence has reported associations with emergency hospital admissions for respiratory illness and asthma-related symptoms in children (CD, p. 8-328). The CD also observes that several factors may make children more susceptible to PM-related effects, including the greater ventilation per kilogram body weight in children and the fact that children are more likely to be active outdoors and thus have greater exposures (CD, p. 9-84). In addition, the CD describes a limited body of new evidence from epidemiologic studies for potential PM-related health effects in infants, but concludes that the available new results are too mixed to allow any clear conclusions to be drawn (CD, p. 8-335).

The CD also discusses other potentially susceptible groups for which less evidence is available. Gender is a potential factor, and there are suggested differences in epidemiologic study results, but the findings are not always consistent (CD, section 9.2.4.4). There is some new

suggestive evidence on genetic susceptibility to air pollution, but no conclusions can be drawn at this time (CD section 9.2.4.3).

Finally, there is some new evidence from epidemiologic studies that people from lower socioeconomic strata, or who have greater exposure to sources such as roadways, may be more vulnerable to PM exposure. Such population groups would be considered to be more vulnerable to potential effects on the basis of socioeonomic status or exposure conditions, as distinguished from susceptibility due to biological or individual health characteristics (CD, section 9.2.4.5).

In summary, there are several population groups that may be more susceptible or vulnerable to PM-related effects. These groups include those with preexisting heart and lung diseases, older adults and children. The available evidence does not generally allow distinctions to be drawn between $PM_{2.5}$ and $PM_{10-2.5}$.

3.5.2 Potential Public Health Impact

As summarized above, there are several populations groups that may be susceptible or vulnerable to effects from exposure to PM. The CD provides estimates of the size of population subgroups, such as young children or older adults, and people with prevalent heart or lung diseases (CD, section 9.2.5.1) that are the subpopulations considered to be likely susceptible to the effects of PM exposure. As shown in Table 9-4 of the CD, approximately 22 million people, or 11% of the U.S. population, have received a diagnosis of heart disease, about 20% of the population have hypertension and about 9% of adults and 11% of children in the U.S. have been diagnosed with asthma. In addition, about 26% of the U.S. population are under 18 years of age, and about 12% are 65 years of age or older (CD, p. 9-89). The CD concludes that combining fairly small risk estimates and small changes in PM concentration with large groups of the U.S. population would result in large public health impacts (CD, p. 9-93).

These health statistics also generally illustrate increasing frequency of less serious health outcomes that would be expected in a "pyramid of effects." In general, many PM-health studies have used the more severe outcome measures for which data are readily available, such as mortality or hospitalization. Incidence or frequency would be expected to increase in the population for less severe effects along the spectrum of severity, for example, from

cardiovascular mortality to the subtle measures of cardiovascular health, such as changes in heart rhythm or increased levels of C-reactive protein.

One issue that is important for interpreting the public health implications of the associations reported between mortality and short-term exposure to PM is whether mortality is occurring only in very frail individuals (sometimes referred to as "harvesting"), resulting in loss of just a few days of life expectancy. A number of new analyses are discussed in the CD (section 8.4.10.1) that assess the likelihood of such "harvesting" occurring in the short-term exposure studies. Overall, the CD concludes from the time-series studies that there appears to be no strong evidence to suggest that short-term exposure to PM is only shortening life by a few days (CD, p. 8-329).

In addition to evidence from short-term exposure studies discussed above, one new report used the mortality risk estimates from the ACS prospective cohort study to estimate potential loss of life expectancy from PM-related mortality in a population. The authors estimated that the loss of population life expectancy associated with long-term exposure to $PM_{2.5}$ was substantial, on the order of a year or so (CD, p. 9-94). Taken together, these results suggest that exposure to ambient PM can have substantial public health impacts (CD, p. 9-93).

3.6 ISSUES RELATED TO QUANTITATIVE ASSESSMENT OF EPIDEMIOLOGIC EVIDENCE

The 1996 CD included extensive discussions of methodological issues for epidemiologic studies, including questions about model specification or selection, co-pollutant confounding, measurement error in pollutant measurements, and exposure misclassification. Based on information available in the last review, the 1996 PM CD concluded that 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 1996a, p. 13-92). Little information was available at that time to allow for evaluation of these and other related methodological issues.

A large number of studies now available in this review have provided new insights on these and other issues as evaluated in Chapters 8 and 9 of the CD. The following discussion builds upon the CD's evaluation of key methodological issues related to epidemiologic studies as

a basis for staff judgments specifically regarding the use of epidemiologic evidence in quantitative assessments, as discussed in Chapters 4 and 5.

This section addresses a number of key methodological issues. Section 3.6.1 discusses air quality data reported in epidemiologic studies, which is one key component of quantitative risk assessment. Section 3.6.2 discusses the issue of exposure error associated with the use of ambient air concentrations as indicators of population exposures in epidemiologic studies. Section 3.6.3 addresses statistical modeling and model specifications used in epidemiologic studies. Section 3.6.4 addresses potential confounding by co-pollutants, to draw staff conclusions about the use of specific study results in quantitative assessments. Finally, two of the criteria discussed in the CD's integrative assessment of the health evidence – temporality and the nature of concentration-response functions – are discussed. Section 3.6.5 includes discussion of several topics in temporal relations between PM exposure and health outcomes. In section 3.6.6, the form of concentration-response relationships in both short-term and long-term exposure studies is discussed, as is evidence related to the potential existence of population threshold levels for effects.

3.6.1 Air Quality Data in Epidemiologic Studies

In general, epidemiologic studies use ambient measurements to represent population exposures to PM of ambient origin. This section discusses some considerations with regard to the ambient PM measurements: (1) whether the type of monitoring method influences the epidemiologic study findings; (2) how measurement error might affect estimates of effects for $PM_{2.5}$ and $PM_{10-2.5}$ and (3) how the frequency of PM measurement collection can influence the power and certainty of study results. Questions related to the influence of exposure error on epidemiologic study results are discussed in the following section.

Many studies have used $PM_{2.5}$ and $PM_{10-2.5}$ measurements from dichotomous samplers or Harvard impactors, but $PM_{2.5}$ and PM_{10} measurements from co-located TEOMs or BAMs also have been used, along with other methods (see Chapter 2 for more detailed descriptions of monitors). In reviewing results from studies using various monitoring methods for $PM_{2.5}$ and $PM_{10-2.5}$, staff finds that there appear to be no systematic differences in the effect estimates related to the use of differing monitoring methods.

For these various monitoring methods, however, another factor to consider is the degree to which uncertainty in the air quality measurements may affect epidemiologic associations with $PM_{10-2.5}$ or $PM_{2.5}$. The CD summarizes the findings of several new analyses that show the potential influence of differential measurement error on epidemiologic analysis results, for either PM with gaseous pollutants, or $PM_{10-2.5}$ and $PM_{2.5}$ as separate pollutants (section 8.4.5). Several studies used simulation analyses of a "causal" pollutant and a "confounder" with differing degrees of measurement error and collinearity between the pollutants. These studies 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. 8-282, 8-283). The conditions required for the error to substantially influence the epidemiologic findings are severe and unlikely to exist in current studies. 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 (CD, p. 9-38).

One analysis applied measurement error models to data from the Harvard Six Cities study, specifically testing relationships between mortality and either fine or thoracic coarse particles (Carrothers and Evans, 2000). The authors identified several variables that could result in biased 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; CD, p. 8-286). These analyses, using data from a study in which significant associations were reported for mortality with $PM_{2.5}$, but not with $PM_{10-2.5}$, indicate that

it is unlikely that measurement error in one PM measurement will result in "false negative" results for coarse particles or "false positive" results for fine particles (CD, p. 8-286). Thus, for either $PM_{2.5}$ or $PM_{10-2.5}$ measurement error is not likely to be falsely attributing effects from one pollutant to another pollutant in the existing epidemiologic studies.

However, it must be recognized that measurement error is a larger issue for $PM_{10-2.5}$ than for fine particles, especially when $PM_{10-2.5}$ concentrations are determined by subtraction of PM_{10} and $PM_{2.5}$ measurements (see section 2.4.3). It is likely that measurement error would increase the uncertainty of an epidemiologic association. With increased error in $PM_{10-2.5}$ monitoring methods, any reported epidemiologic associations would be less likely to reach statistical significance (CD, p. 5-126). Thus, a set of positive but generally not statistically significant associations between $PM_{10-2.5}$ and a health outcome could be reflecting a true association that is measured with error. Decreases in study precision would also occur even if gravimetric $PM_{10-2.5}$ were perfectly measured, but the sources and relative composition of the coarse particles were highly variable. In evaluating the implications of the epidemiologic studies showing effects of $PM_{10-2.5}$, therefore, staff places more emphasis on the pattern of results in a series of studies than on the statistical significance of any single effect estimate.

Finally, frequency of data collection can also affect the results reported from epidemiologic analyses. The CD discusses the use of less-than-everyday monitoring data as a source of uncertainty for time-series analyses (CD, p. 8-296). Many such studies were conducted in areas where PM was monitored on a daily basis; in fact, the availability of every-day monitoring is cited as a basis for study location in a number of reports. This is particularly true for panel studies on respiratory or cardiovascular symptoms, all of which use daily PM monitoring data, though generally for shorter time periods.

However, staff observes that a small number of the recent studies have been based on less frequently collected data. Data collection frequency is one component of statistical power for time-series studies, and missing data would result in increased uncertainty in study results. In addition, for either $PM_{2.5}$ or $PM_{10-2.5}$, one would expect that a substantial proportion of missing data may complicate time-series analyses (CD, p. 9-41). As illustrated in the CD, effect estimates for PM_{10} and mortality varied in size and statistical significance in a series of analyses of data collected on a 1-in-6 day schedule (CD, p. 8-297). The CD presents results from a study

had not been used in the original analyses (e.g., physical activity, lung function, marital status), did not alter the original findings. Data were also obtained for 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 reanalysis investigators 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 (CD, p. 8-92).

In summary, the sensitivity of epidemiologic study results to model specification has been investigated for both short-term and long-term exposure studies. In both cases, the reanalyses generally support the findings of the original studies, while raising questions for further research. For short-term exposure studies, staff concludes that it is appropriate to use the results of the reanalyzed time-series epidemiologic studies or the results of studies that did not use GAM in the original analyses. In addition, staff observes that the use of more appropriate convergence criteria in GAM has generally addressed questions about the magnitude of the effect estimate. To obtain correct standard errors for the estimates, additional reanalyses used GLM and parametric smoothing approaches that generally produced larger standard errors. For quantitative risk assessment, staff concludes that models using more stringent GAM criteria likely provide the most representative effect estimate sizes, while in illustrating the significance of associations (e.g., as presented in Figures 3-1 and 3-2) staff has chosen to use results from GLM-based analyses when available. For long-term exposure studies, staff concludes that results from the reanalyses or extended analyses, in particular the extended analysis of the ACS study, are most appropriate for use in quantitative assessment.

3.6.4 Co-pollutant Confounding and Effect Modification

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; epidemiologic 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.

it is unlikely that measurement error in one PM measurement will result in "false negative" results for coarse particles or "false positive" results for fine particles (CD, p. 8-286). Thus, for either $PM_{2.5}$ or $PM_{10-2.5}$ measurement error is not likely to be falsely attributing effects from one pollutant to another pollutant in the existing epidemiologic studies.

However, it must be recognized that measurement error is a larger issue for $PM_{10-2.5}$ than for fine particles, especially when $PM_{10-2.5}$ concentrations are determined by subtraction of PM_{10} and $PM_{2.5}$ measurements (see section 2.4.3). It is likely that measurement error would increase the uncertainty of an epidemiologic association. With increased error in $PM_{10-2.5}$ monitoring methods, any reported epidemiologic associations would be less likely to reach statistical significance (CD, p. 5-126). Thus, a set of positive but generally not statistically significant associations between $PM_{10-2.5}$ and a health outcome could be reflecting a true association that is measured with error. Decreases in study precision would also occur even if gravimetric $PM_{10-2.5}$ were perfectly measured, but the sources and relative composition of the coarse particles were highly variable. In evaluating the implications of the epidemiologic studies showing effects of $PM_{10-2.5}$, therefore, staff places more emphasis on the pattern of results in a series of studies than on the statistical significance of any single effect estimate.

Finally, frequency of data collection can also affect the results reported from epidemiologic analyses. The CD discusses the use of less-than-everyday monitoring data as a source of uncertainty for time-series analyses (CD, p. 8-296). Many such studies were conducted in areas where PM was monitored on a daily basis; in fact, the availability of every-day monitoring is cited as a basis for study location in a number of reports. This is particularly true for panel studies on respiratory or cardiovascular symptoms, all of which use daily PM monitoring data, though generally for shorter time periods.

However, staff observes that a small number of the recent studies have been based on less frequently collected data. Data collection frequency is one component of statistical power for time-series studies, and missing data would result in increased uncertainty in study results. In addition, for either $PM_{2.5}$ or $PM_{10-2.5}$, one would expect that a substantial proportion of missing data may complicate time-series analyses (CD, p. 9-41). As illustrated in the CD, effect estimates for PM_{10} and mortality varied in size and statistical significance in a series of analyses of data collected on a 1-in-6 day schedule (CD, p. 8-297). The CD presents results from a study

correlations. In such situations, while the epidemiologic associations may be illustrating true time-series relationships between PM and a health outcome, it is more difficult to draw inferences about the population exposure levels at which those effects are seen. From studies in which significant associations are reported with $PM_{10-2.5}$, the distribution of ambient monitoring data available for the study may reflect levels that are higher or lower than those experienced by neighborhoods in other parts of the community.

3.6.3 Alternative Model Specifications

As observed earlier, statistical modeling issues for epidemiologic studies were discussed in great detail in the 1996 PM CD (EPA, 1996a, sections 12.6.2 and 12.6.3). This evaluation lead to the conclusion that PM-related effects observed in epidemiologic studies were unlikely to be seriously biased by inadequate statistical modeling or confounded by weather (CD, p. 8-22). Statistical modeling issues have re-emerged in this review, however, and much attention has been given to further investigations of approaches to model specification for epidemiological analyses. The following discussions draw from the CD's evaluation of model specification issues for both short-term and long-term exposure studies.

Time-series epidemiologic studies

In 2002, questions were raised about the default convergence criteria and standard error calculations made using GAM, which have been commonly used in recent time-series epidemiologic studies. As discussed more completely in the CD (section 8.4.2), a number of time-series studies were reanalyzed using alternative methods, typically GAM with more stringent convergence criteria and alternative models such as GLM with natural smoothing splines. The results of the reanalyses have been compiled and reviewed in an HEI publication (HEI, 2003a). Reanalyzed PM_{10} mortality study results are presented in Figure 8-15 in the CD, where it can be seen that the reanalyses generally did not substantially change the findings of the original analyses, and the changes in effect estimates with alternative analysis methods were much smaller than the variation in effects across studies. Taking into account the conclusions of the HEI review, the CD finds that mortality effect estimates were often, but not always, reduced with the use of GAM with more stringent convergence criteria; however, the extent of these changes was not substantial in most cases (CD, p. 8-232). Further, for morbidity studies, the

CD finds that the impact of the reanalyses was relatively small and the basic conclusions regarding the significance of PM-related hospital admissions remained unchanged when more stringent GAM criteria were used (CD; p. 8-235).

These reanalyses also investigated alternative model specifications to control for potential weather effects and temporal trends. As shown in Figures 8-20 and 8-21 in the CD, the magnitude of the effect estimate for PM can decrease with increasing control for weather and temporal trend, though it generally stabilizes at some point. The CD observes that there is no clear consensus at this time as to what constitutes appropriate control for such variables, while recognizing that no single approach is likely to be most appropriate in all cases (CD, p. 8-340). 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 model 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 (CD, p. 8-236). 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. The CD concludes that the available studies appear to demonstrate that there are PM-related effects independent of weather influences, but that further evaluation is needed on how to best characterize possible combined effects of air pollution and weather (CD, p. 8-340).

Prospective cohort epidemiologic studies

Data from the ACS and Six Cities prospective cohort studies were used in a major reanalysis study that evaluated a number of issues that had been raised for the long-term exposure studies. These issues included whether the results were sensitive to alternative modeling strategies. The reanalysis included two major components, a replication and validation study, and a sensitivity analysis, where alternative risk models and analytic approaches were used to test the robustness of the original analyses. In the first phase, the data from the two studies were found to be of generally high quality, and the original results were replicated, confirming the original investigators' findings of associations with both total and cardiorespiratory mortality (Krewski et al., 2000; CD, p. 8-91). 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 alter the original findings. Data were also obtained for 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 reanalysis investigators 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 (CD, p. 8-92).

In summary, the sensitivity of epidemiologic study results to model specification has been investigated for both short-term and long-term exposure studies. In both cases, the reanalyses generally support the findings of the original studies, while raising questions for further research. For short-term exposure studies, staff concludes that it is appropriate to use the results of the reanalyzed time-series epidemiologic studies or the results of studies that did not use GAM in the original analyses. In addition, staff observes that the use of more appropriate convergence criteria in GAM has generally addressed questions about the magnitude of the effect estimate. To obtain correct standard errors for the estimates, additional reanalyses used GLM and parametric smoothing approaches that generally produced larger standard errors. For quantitative risk assessment, staff concludes that models using more stringent GAM criteria likely provide the most representative effect estimate sizes, while in illustrating the significance of associations (e.g., as presented in Figures 3-1 and 3-2) staff has chosen to use results from GLM-based analyses when available. For long-term exposure studies, staff concludes that results from the reanalyses or extended analyses, in particular the extended analysis of the ACS study, are most appropriate for use in quantitative assessment.

3.6.4 Co-pollutant Confounding and Effect Modification

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; epidemiologic 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.

8-10). Effect modifiers include variables that may influence the health response to the pollutant exposure (e.g., co-pollutants, individual susceptibility, smoking or age). Both are important considerations for evaluating effects in a mixture of pollutants, but for confounding, the emphasis is on controlling or adjusting for potential confounders in estimating the effects of one pollutant, while the emphasis for effect modification is on identifing and assessing the level of effect modification (CD, p. 8-12).

Co-pollutant Confounding

Potential confounding by gaseous copollutants has been most commonly assessed by using multi-pollutant models. As discussed in the CD (section 8.4.3.2), 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. 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. 8-12). This situation certainly occurs. For example, sources of fine particle 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.

In the NMMAPS multi-city analyses, one key objective was to characterize the effects of PM_{10} and the gaseous co-pollutants, alone and in combination. Multi-pollutant modeling was used in the NMMAPS mortality analyses for 20 and 90 U.S. cities, in which the authors added first O_3, then O_3 and another co-pollutant (e.g., CO, NO_2 or SO_2) to the models (CD, p. 8-35). The relationship between PM_{10} and mortality was little changed in models including control for O_3 and other gaseous pollutants (CD, Figure 8-4, p. 8-35). 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; Domenici, 2003).

Single- and multi-pollutant model results for a range of health outcomes with PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ from multi- and single-city studies are presented in Figures 8-16 through 8-19 of the CD. For the most part, the addition of gaseous co-pollutants had little influence on PM associations, although substantial reduction in associations with PM could be seen in some cases when gaseous pollutants are added to the model.

In the long-term exposure studies, multi-pollutant models have been tested in some analyses. The reanalysis of data from the ACS cohort indicated that associations between mortality and $PM_{2.5}$ or sulfates were reduced in size in co-pollutant models including SO_2 but not with the other gaseous pollutants. Since SO_2 is a precursor for fine particle sulfates, it is naturally difficult to distinguish effects from the precursor SO_2 and fine particles, as discussed above (CD, p. 9-37).

In addition to statistical approaches for assessing potential confounding, the CD also discusses information available on the biological plausibility of effects of the potentially confounding pollutants and consideration of exposure relationships. Information about the biological plausibility of effects can inform conclusions about which pollutant from a mixture of correlated pollutants is more likely responsible for the observed associations. For example, in evaluating results of the ACS study analyses described above, the authors concluded that an association between SO_2 and mortality was less plausible than the association between $PM_{2.5}$ and mortality (CD, p. 8-15). Further research is needed on biological mechanisms underlying air pollution-related effects to support future assessments.

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. These findings suggest that associations reported with ambient $PM_{2.5}$ are truly reflecting associations with fine particles and that fine particles are unlikely to be simply acting as surrogates for other gaseous pollutants (Sarnat et al., 2000, 2001; CD, p. 5-90).

Effect Modification

Some new studies have also assessed the potential for effect modification by the gaseous pollutants. In the NMMAPS morbidity analyses for14 U.S. cities, the authors tested 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 for cardiovascular or respiratory hospitalization and PM_{10}-co-pollutant correlations (CD, pp. 8-146, 8-175). These results indicate that associations reported in this study for PM_{10} are not dependent on the correlation between PM_{10} and the gaseous copollutants.

An alternative way to evaluate the effect of co-pollutants on associations reported with $PM_{2.5}$ is illustrated in Figure 3-3. 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 3-3 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 $PM_{2.5}$ that is not appreciably modified by differing levels of the gaseous pollutants.

In summary, the available evidence does not indicate that exposure to the gaseous pollutants is an effect modifier for PM-related health outcomes. With regard to confounding effects between pollutants, 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, as illustrated in Figures 8-16 through 8-19 of the CD. 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.

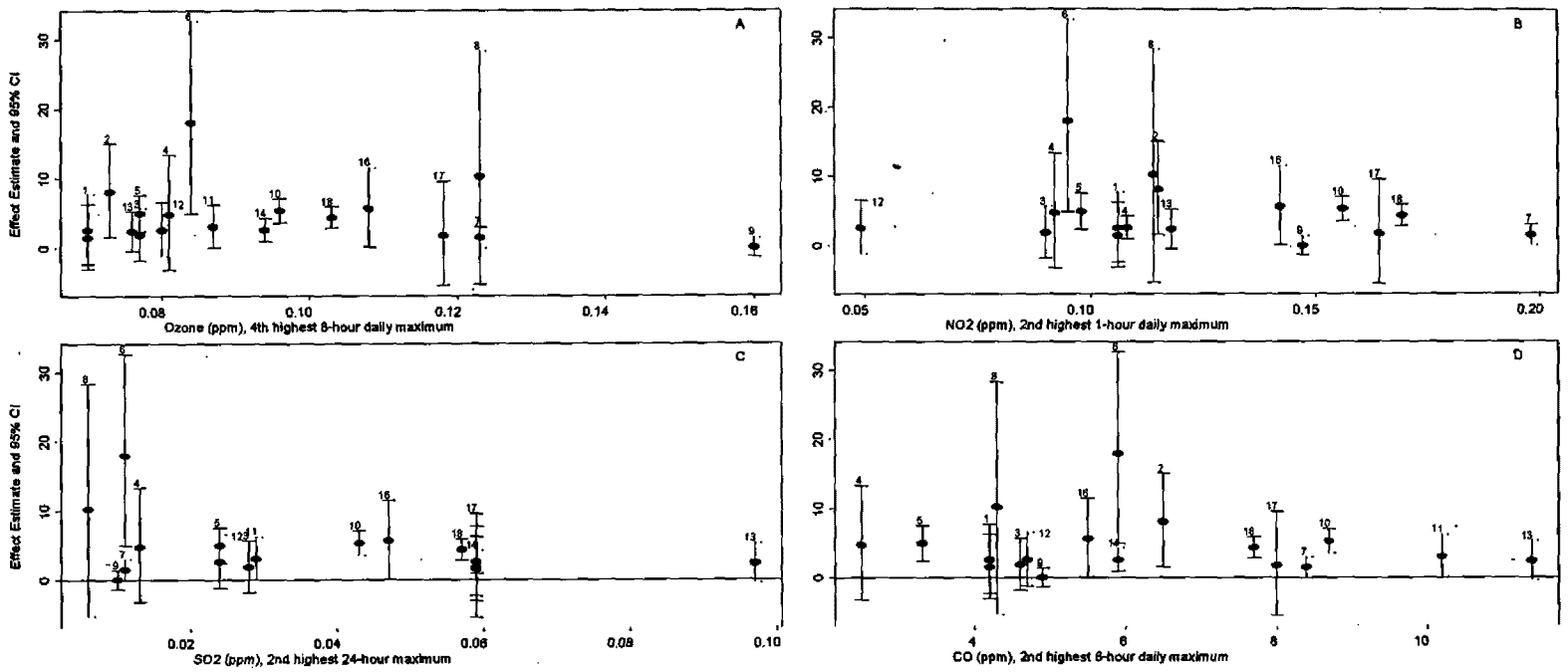

Figure 3-3. Associations between $PM_{2.5}$ and total mortality from U.S. studies, plotted against gaseous pollutant concentrations from the same locations. Air quality data obtained from the Air Quality System (AQS) for each study time period: (A) mean of 4th highest 8-hour ozone concentration; (B) 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. Study locations are identified below:

1. Chock et al., 2000, Pittsburgh, PA
2. Fairley, 2003, Santa Clara County, CA
3. Ito, 2003, Detroit, MI
4. Klemm and Mason, 2000, Atlanta, GA
5. Lipfert et al., 2000a, Philadelphia, PA
6. Mar et al., 2003, Phoenix, AZ
7. Moolgavkar, 2003, Los Angeles, CA
8. Ostro et al., 2003, Coachella Valley, CA
9. Ostro et al., 1995, Southern California
10. Schwartz, 2003a, Boston, MA
11. Schwartz, 2003a, Knoxville, TN
12. Schwartz, 2003a, Portage, WI
13. Schwartz, 2003a, St. Louis, MO
14. Schwartz, 2003a, Steubenville, OH
15. Schwartz, 2003a, Topeka, KS
16. Tsai et al., 2000, Camden NJ
17. Tsai et al., 2000, Elizabeth NJ
18. Tsai et al., 2000, Newark NJ

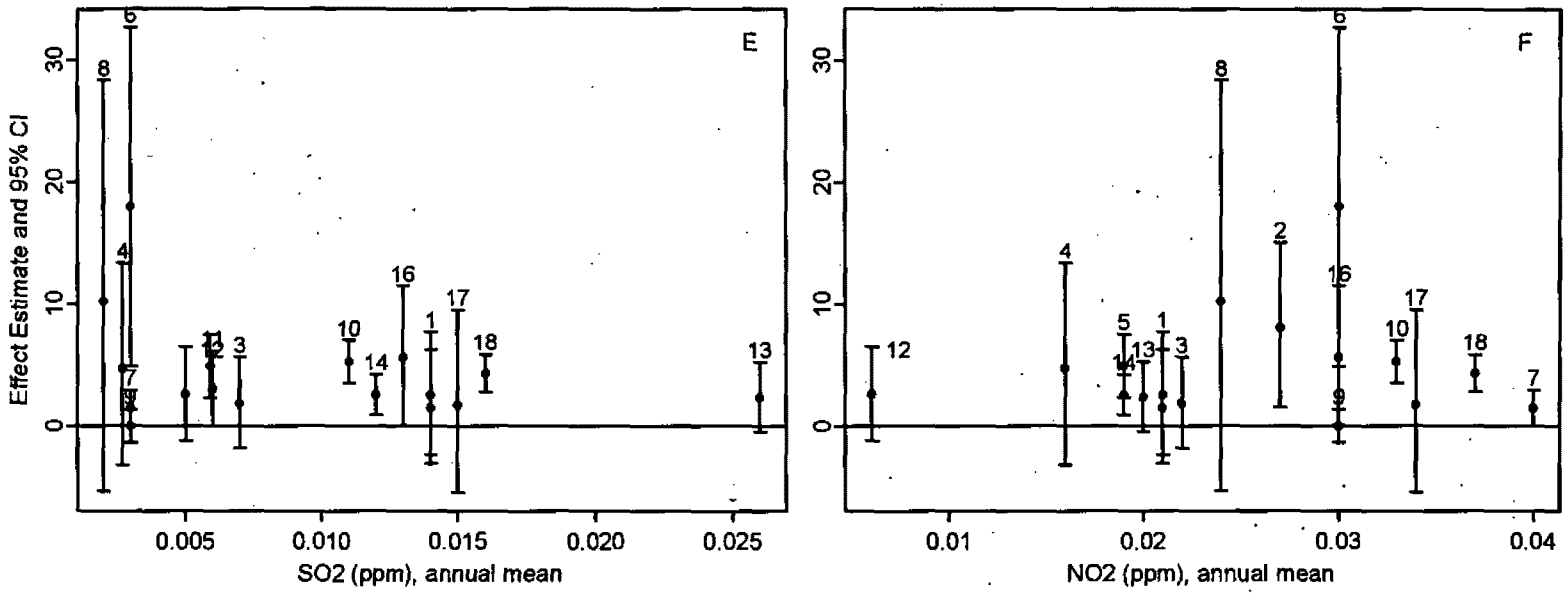

Figure 3-3 (continued). Associations between $PM_{2.5}$ and total mortality from U.S. studies, plotted against gaseous pollutant concentrations from the same locations. Air quality data obtained from the Air Quality System (AQS) for each study time period: (E) annual mean SO_2 concentration; (F) annual mean NO_2 concentration. Study locations are identified below (data in Appendix A)

1. Chock et al., 2000, Pittsburgh, PA
2. Fairley, 2003, Santa Clara County, CA
3. Ito, 2003, Detroit, MI
4. Klemm and Mason, 2000, Atlanta, GA
5. Lipfert et al., 2000a, Philadelphia, PA
6. Mar et al., 2003, Phoenix, AZ
7. Moolgavkar, 2003, Los Angeles, CA
8. Ostro et al., 2003, Coachella Valley, CA
9. Ostro et al., 1995, Southern California
10. Schwartz, 2003a, Boston, MA
11. Schwartz, 2003a, Knoxville, TN
12. Schwartz, 2003a, Portage, WI
13. Schwartz, 2003a, St. Louis, MO
14. Schwartz, 2003a, Steubenville, OH
15. Schwartz, 2003a, Topeka, KS
16. Tsai et al., 2000, Camden NJ
17. Tsai et al., 2000, Elizabeth NJ
18. Tsai et al., 2000, Newark NJ

Finally, 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. For example, recent animal toxicologic studies have tested effects of exposure to PM (e.g., urban PM, carbon particles, acid aerosols) in combination with O_3 and suggeted that 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 7-13). 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 CD concludes that while research questions remain, in general, "associations for various PM indices with mortality or morbidity are robust to confounding by co-pollutants." (CD, p. 9-37). This indicates that effect estimates from single-pollutant models can be used to represent the magnitude of a concentration-response relationship, though there will remain uncertainty with regard to potential contributions from other pollutants. For quantitative assessment, staff concludes that single-pollutant model results provide reasonable indicators of the magnitude of PM-related effects for the purpose of comparing risk estimates with different alternative standard scenarios, with additional sensitivity analyses to include multi-pollutant model results.

3.6.5 Temporality in Concentration-Response Relationships

3.6.5.1 PM short-term exposure time periods

While most time-series epidemiologic studies use 24-hour average PM measurements, several new studies have used ambient PM concentrations averaged over shorter time intervals, such as 1- or 4-hour averages. Many such studies have evaluated associations with cardiovascular health biomarkers or physiological changes. Section 8.3.1.3.4 of the CD describes several epidemiologic studies that report statistically significant associations between 2- to 4-hour PM_{10} or $PM_{2.5}$ concentrations and cardiovascular health endpoints, including myocardial infarction incidence and heart rate variability (CD, pp. 8-162 to 8-165). One study reported effect estimates for myocardial infarction incidence with $PM_{2.5}$ averaged over 2- and 24 hours that are quite similar in magnitude, and both are statistically significant (Peters et al., 2001; CD, p. 8-165).

For respiratory health outcomes, two panel studies of symptoms in asthmatic subjects are summarized in the CD (section 8.3.3.1.1). One study in a small Southern California community,

reported larger effect estimates for 1- or 8-hour concentrations than for 24-hour PM_{10} concentrations (Delfino et al., 1998), while the other, in Los Angeles, reported larger effect estimates for 24-hour PM_{10} concentrations (Ostro et al., 2001; CD, p. 8-206). However, several studies of hospital admissions or medical visits for respiratory diseases reported the strongest associations with several-day average PM concentrations (CD, p. 8-279).

Evidence of health effects associations with different exposure time periods can inform staff conclusions and recommendations regarding potential NAAQS averaging times. Staff observes that the very limited information available in the CD suggests that cardiovascular effects may be associated with acute exposure time periods on the order of an hour or so.

3.6.5.2 Lag Structure in Short-term Exposure Studies

In the short-term exposure epidemiologic studies, many investigators have tested associations for a range of lag periods between the health outcome and PM concentration (see CD, sections 8.4.4 and 9.2.2.4). As discussed in the CD, it is important to consider the pattern of results that is seen across the series of lag periods. If there is an apparent pattern of results across the different lags, then selecting the single-day lag with the largest effect from a series of positive associations is likely to underestimate the overall effect size, since single-day lag effect estimates do not fully capture the risk that may be distributed over adjacent or other days (CD, p. 8-270). Where effects are found for a series of lag periods, a distributed lag model will more accurately characterize the effect estimate size. However, if there is no apparent pattern or reported effects vary across lag days, the use of any single result may be inappropriate for quantitative assessment (CD, p. 9-42).

For selecting effect estimates from studies for use in quantitative risk assessment, or for evaluation of potential revisions to the standards, staff considered patterns of results for $PM_{2.5}$ or $PM_{10-2.5}$ across lag periods from U.S. and Canadian studies. Numerous investigators have reported quantitative results only for the strongest associations, after testing associations over a range of lags and finding a reasonably consistent pattern across lags. An example of such an evaluation is provided in an analysis using hospitalization for asthma (Sheppard et al.,1999; 2003). This study tested lags to 3-days and beyond, and reported consistent patterns across lags for associations between asthma hospitalization and PM_{10}, $PM_{2.5}$ or $PM_{10-2.5}$. Results for the strongest associations are presented, with the authors observing "When considering single (vs.

distributed) lag estimates, it is important to put the estimate in the context of the pattern of lags nearby and to recognize that effect estimates contain information from adjacent days owing to serial correlation of the pollutant series. The pollutant effects given for asthma are larger than and consistent with estimates obtained for adjacent lags. In contrast, adjacent lags to the same-day PM and SO_2 effects on appendicitis change much more abruptly, and the overall pattern is unstable." (Sheppard et al., 1999, p. 27)

Most of the studies included in Appendix 3A either selected lag periods *a priori*, or evaluated results for a range of lag periods, reporting effect estimates for one lag period based on this evaluation. An example of results that do not follow a consistent pattern across lags can be found in a study in Coachella Valley (Ostro et al., 2000; 2003). In this study, results for a series of lags show fairly consistent patterns for associations between PM_{10} and $PM_{10\text{-}2.5}$ and cardiovascular mortality, but not with total or respiratory mortality, nor for associations between $PM_{2.5}$ and total and cardiovascular mortality. Based on the greater uncertainty on the effect estimate size for the $PM_{2.5}$-mortality association from this study, staff concludes that it would not be appropriate to use the results for quantitative assessments.[6] In addition, a series of studies in Cook County, IL and Los Angeles County, CA, include effect estimates for 0- to 5-day lag periods and, in general, the results follow a pattern. However, the pattern of results for COPD mortality with $PM_{2.5}$ was quite inconsistent (Moolgavkar, 2000a,b,c; Moolgavkar, 2003, p. 191).[7] Based on the considerations described above, the results for COPD mortality from this study were not used in the risk assessment discussed in Chapter 4.

The CD concludes that it is likely that the most appropriate lag period for a study will vary, depending on the health outcome and the specific pollutant under study. Some general observations can be made about lag periods for different health outcomes. For total and cardiovascular mortality, it appears that the greatest effect size is generally reported for the 0-day

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[6]The air quality measurements available for $PM_{2.5}$ and $PM_{10\text{-}2.5}$ may also contribute to the more uncertain findings for $PM_{2.5}$ in this study. For $PM_{10\text{-}2.5}$, a 10-year series of concentrations was modeled from a 2 ½ year series of ambient measurements at co-located beta attenuation monitors, while predictive models for $PM_{2.5}$ concentrations were not reported to be adequate, so only the 2 ½ year series of measurements were used in $PM_{2.5}$ analyses.

[7] That only 1-in-6 day PM measurements were available in Los Angeles County is likely to be an important factor contributing to less consistent findings there.

lag and 1-day lag, generally tapering off for longer lag periods (CD, p. 8-279). This is true also for hospitalization for cardiovascular diseases. For cardiovascular effects such as myocardial infarction or HRV change, there appears to be a pattern of larger effects with shorter lag periods, such as 1- to 4-hours. For respiratory symptoms, many studies report effects over a series of lags, with larger effect estimates for moving average or distributed lag models. Similarly, for asthma hospitalization, there appear to be larger effects over longer average time periods, out to 5- to 7-day average lags.

A number of recent studies that have investigated associations with distributed lags provide effect estimates for health responses that persist over a period of time (days to weeks) after the exposure period. The available studies have generally used PM_{10} or other PM indicators, but not $PM_{2.5}$ or $PM_{10-2.5}$. Effect estimates from distributed lag models are often, but not always, larger in size that those for single-day lag periods (CD, p. 8-281). For example, in multi-city analyses of data from 10 U.S. cities, the effect estimates for total mortality from distributed lag models are about twice those from 0-1 day average lag models (Schwartz, 2003b). In the 14-city NMMAPS analysis of hospitalization in the elderly, the combined city effect estimate for COPD hospitalization is larger (about doubled) in results of distributed lag models than in 0-1 day average lag models, while the CVD hospitalization effect estimate is only increased by a small amount, and the effect estimate for pneumonia hospitalization is somewhat smaller in distributed lag models, compared with a 0-1 day average lag (Schwartz, et al., 2003).

In summary, the CD concludes that distributed lag results would likely provide more accurate effect estimates for quantitative assessment than an effect estimate for a single lag period (CD, p. 9-42). However, at this time, studies using $PM_{2.5}$ and $PM_{10-2.5}$ have not included distributed lag models Most U.S. and Canadian studies have reported consistent patterns in results for different lags; for these studies, an effect estimate for a single-day lag period is likely to underestimate the effect. In quantitative assessments for $PM_{2.5}$ and $PM_{10-2.5}$, since results are not available for distributed lag models, staff conclude that it is appropriate to use single-day lag period results, recognizing that this is likely to underestimate the effect. For quantitative assessment, staff concludes that it is appropriate to use results from lag period analyses consistent with those reported in the CD, focusing on shorter lag periods for cardiovascular effects and lag periods of several days for respiratory effects, depending on availability of results. For the few

studies that show inconsistent patterns, the use of single-day lag results are not appropriate for quantitative assessment.

3.6.5.3 Seasonal Differences in Time-Series Epidemiologic Results

As discussed in section 3.5.3, time-series epidemiologic studies generally use some temporal or seasonal terms in the models to control for seasonal changes in health outcomes. In addition, a few epidemiologic studies have also evaluated PM-health associations across seasons by doing analyses on data subdivided into different seasons, thus evaluating differences in effects across the season, rather than trying to control for seasonal influences. The CD observes that there can be seasonal differences in correlations between PM and other pollutants, or in PM levels across seasons (CD, p. 8-57).

The CD presents results for seasonal analyses for individual studies in Chapter 8 and the Appendices to Chapter 8. In 10 U.S. cities, the relationship between PM_{10} and mortality was the same in analyses for data divided into summer and winter seasons (Schwartz, et al., 2000). In Pittsburgh, relationships between $PM_{10-2.5}$ and $PM_{2.5}$ and mortality were "unstable" when statified by season, and there was evidence of differing multi-collinearity between seasons (Chock et al., 2000). In Coachella Valley, associations between mortality and several PM indicators were stronger in the winter season (October-May) than in the summer season (Ostro et al., 2000). However, an earlier analysis in two Southern California counties reported significant associations between estimated $PM_{2.5}$ and mortality in the summer (April-September) quarter only (Ostro et al., 1995). Seasonal analyses were done for the mortality-$PM_{2.5}$ relationship in San Jose, and there were no significant differences between the four seasons (Fairley, 2003). In Phoenix, the association between $PM_{10-2.5}$ and mortality was reported to be highest in spring and summer, when $PM_{10-2.5}$ concentrations were lowest (Mar et al., 2003). Associations between PM_{10} and hospitalization for cardiovascular diseases in Los Angeles were greater in the winter and fall seasons than in spring or summer (Linn et al., 2000). Asthma hospitalization was significantly associated with PM_{10} for both "wet" and "dry" seasons in Los Angeles, but the association was larger in magnitude during the wet season (January-March) (Nauenberg and Basu, 1999). In Seattle, associations between PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ and asthma hospitalization were positive in all seasons, but higher in spring and fall (Sheppard et al., 2003).

Staff observes that these few studies show no apparent pattern in results across seasons. The largest of these studies showed no seasonal differences in the results combining data from 10 U.S. cities (Schwartz et al., 2000). Most of the studies listed above show generally positive results across all seasons tested, with some reporting larger effect estimates in one or more season(s), but the differences were not statistically significant. Staff concludes that the available evidence does not support quantitative assessment of seasonal differences in relationships between PM and health outcomes.

3.6.5.4 Exposure Time Periods in Long-term Exposure Studies

Studies of effects related to long-term PM exposures have generally used air quality levels averaged over months or years as exposure indicators. It is important to recognize that these studies do not test specifically for latency in an exposure-effect relationship. Instead, the average PM levels are used to represent long-term exposure to ambient PM, and the exposure comparisons are basically cross-sectional in nature (CD, p. 9-42). As discussed in the CD, it is not easy to differentiate the role of historic exposures from more recent exposures, leading to potential exposure measurement error (CD, p. 5-118). This potential misclassification of exposure is increased if average PM concentrations change over time differentially between areas.

Several new studies have used different air quality periods for estimating long-term exposure and tested associations with mortality for the different exposure periods. In the extended analysis of the ACS study, Pope et al. (2002) reported associations between mortality and $PM_{2.5}$ using the original air quality data (1979-1983), data from the new fine particle monitoring network (1999-2000), and the average $PM_{2.5}$ concentrations from both time periods. The authors reported that the two data sets were well correlated, indicating that the ordering of the cities from low to high pollution levels had changed little. When using average $PM_{2.5}$ levels from all years, the associations for total, cardiopulmonary and lung cancer were slightly larger in size, though not significantly so, than for either individual air quality data set.

A 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 (1979 to 1986) to represent long-term PM exposure estimates, while the new analysis includes $PM_{2.5}$ data from more recent years and evaluates associations with $PM_{2.5}$ averaged 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. 8-97). This might be expected, if the most polluted cities had the greatest decline in pollutant levels as controls were applied (CD, p. 8-93). 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 (Villeneuve et al., 2002, p. 574).

Using essentially the same air quality data set as that used in the original ACS analyses, Lipfert et al. (2000b) investigated associations between mortality and PM (using several PM indicators) over numerous averaging periods. When using methods similar to those of the other prospective cohort studies, the authors report finding similar associations between fine particles and mortality (CD, p. 8-115). However, in analyses using mortality and PM data in different time segments, the results were varied, with some statistically significant negative associations reported. The authors report that the strongest positive associations were found with air quality data from the earliest time periods, as well as the average across all data.

All three analyses indicate that averaging PM concentrations over a longer time period results in stronger associations; as the Six Cities study authors observe, the longer series of data is likely a better indicator of cumulative exposure. In these studies, spatial variation in the PM concentrations is the key exposure indicator, and one key question is the extent to which concentrations change over time, particularly whether there are differential changes across cities. As observed above, the order of cities from high to low pollution levels changed little across time periods in the cities used in the ACS analyses. Where lower effect estimates are reported with data collected in more recent years, the CD observes: "This is likely indicative of the effectiveness of control measures in reducing source emissions importantly contributing to the toxicity of ambient particles in cities where PM levels were substantially decreased over time" (CD, p. 9-43). The CD concludes that further study is warranted on the importance of different time windows for exposure indicators in studies of effects of chronic PM exposure.

For use in quantitative assessments, staff concludes that it appropriate to use results from analyses that are based on averaging PM levels over longer time periods, since the recent studies indicate that this provides a better indicator of long-term PM exposure. Thus, as described in Chapter 4, the results from the extended ACS analyses using average $PM_{2.5}$ concentrations from both the original and more recent time periods are used in the PM risk assessment. Staff notes that this is consistent with the advice to EPA from the Health Effects Subcommittee (HES) of the SAB's Clean Air Act Compliance Council (SAB, 2004), in their review of methods used for EPA's health benefits assessments. The HES recommended using the results of ACS cohort analyses that used air quality data averaged over the full study time period, indicating that this represented the best period to use in order to reduce measurement error.

3.6.6 Concentration-Response Relationships and Potential Population Thresholds

In assessing or interpreting public health risk associated with exposure to PM, the form of the concentration-response function is a critical component. The CD recognized that it is reasonable to expect that, for individuals or groups of individuals with similar innate characteristics and health status, there may be biological thresholds for different effects. Individual thresholds would presumably vary substantially from person to person due to individual differences in genetic-level susceptibility and pre-existing disease conditions (and could even vary from one time to another for a given person). Thus, it would be difficult to detect a distinct threshold at the population level, especially if the most sensitive members of a population are unusually sensitive even down to very low concentrations. The person-to-person difference in the relationship between personal exposure to PM of ambient origin and the concentration observed at a monitor may also add to the variability in observed exposure-response relationships, further obscuring potential population thresholds (CD, p. 9-43, 9-44).

The 1996 CD evaluated evidence from epidemiologic studies regarding both functional form and whether a threshold for effects could be identified. Based on the few available studies, the 1996 CD concluded that linear model results "appear adequate for assessments of PM_{10} and $PM_{2.5}$ effects" (EPA, 1996a, p. 13-91). Among the new epidemiologic studies of short-term PM exposure are several that use different modeling methods to investigate potential threshold levels and concentration-response forms.

Several time-series studies have evaluated potential threshold levels for associations between mortality and short-term PM exposures. In plots of concentration-response curves from multi-city analyses, using the NMMAPS data, it is difficult to discern any evident threshold for relationships between PM_{10} and total or cardiorespiratory mortality. The authors also present posterior probabilities for the existence of thresholds at different levels of PM_{10} showing that if there is a threshold in the relationships between PM_{10} and total or cardiorespiratory mortality, the likelihood of the threshold being above about 25 $\mu g/m^3$ is essentially zero (Dominici et al., 2003b; CD, pp. 8-320, 8-321). One single-city analysis used various statistical methods to test for thresholds in simulated data sets that were created with assumed threshold levels ranging from 12.8 to 34.4 $\mu g/m^3$ for the relationship between PM_{10} and mortality. The authors of this analysis concluded that it was highly likely that standard statistical methods could detect a threshold level, if one existed (Cakmak et al., 1999; CD, p. 8-319).

One single-city study used $PM_{2.5}$ and $PM_{10-2.5}$ measurements in Phoenix and reported that there was no indication of a threshold in the association between $PM_{10-2.5}$ and mortality, but that there was suggestive evidence of a threshold for the mortality association with short-term exposure to $PM_{2.5}$ up to levels of about 20-25 $\mu g/m^3$ (Smith et al., 2000; CD, 8-322). In addition, single-city analyses in Birmingham and Chicago suggested that the concentration-response functions for PM_{10} and mortality changed to show increasing effects at levels of 80 to 100 $\mu g/m^3$ PM_{10}, but "not to an extent that statistically significant distinctions were demonstrated" (CD, p. 8-322).

For long-term exposure to PM and mortality, the shape of the concentration-response function was evaluated using data from the ACS cohort. The concentration-response relationships for associations between $PM_{2.5}$ and all-cause, cardiopulmonary and lung cancer mortality are shown in Figure 3-4. The authors reported that the associations for all-cause, cardiovascular and lung cancer mortality "were not significantly different from linear associations" (Pope, et al., 2002). It is apparent in this figure that the confidence intervals around each of the estimated concentration-response functions expand significantly as one looks below around 12-13 $\mu g/m^3$, indicating greater uncertainty in the shape of the concentration-

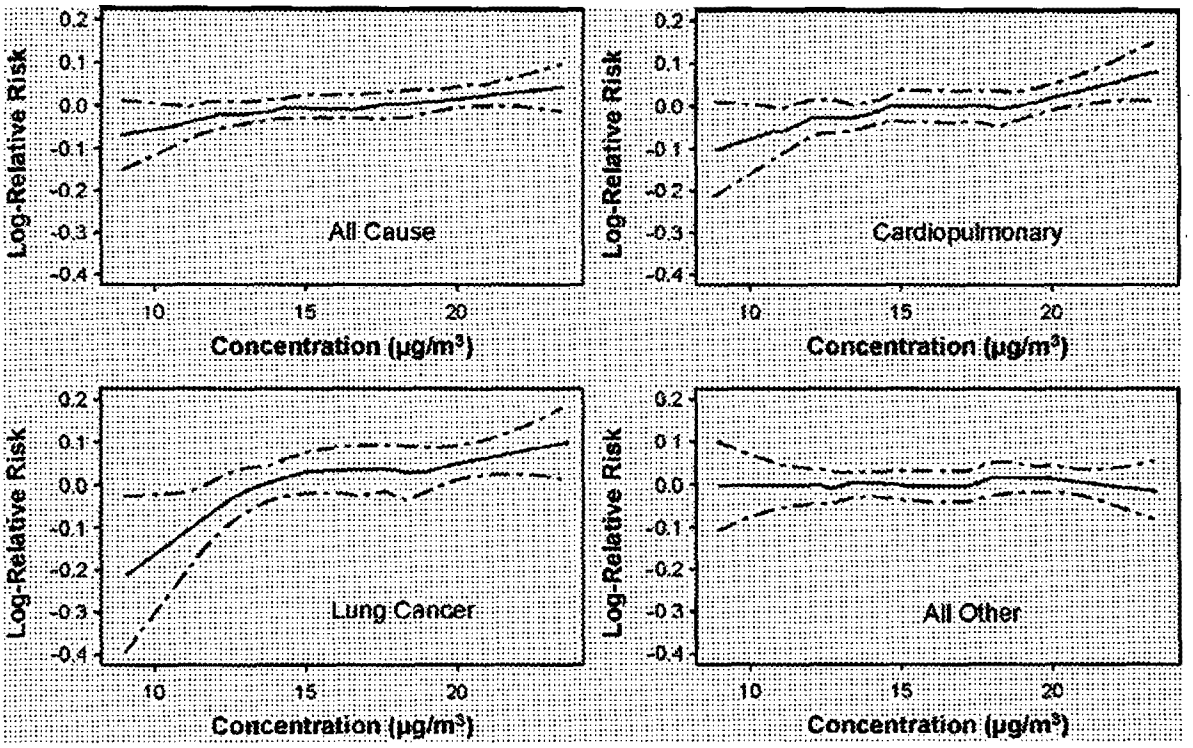

Figure 3-4. Natural logarithm of relative risk for total and cause-specific mortality per 10 µg/m³ $PM_{2.5}$ (approximately the excess relative risk as a fraction), with smoothed concentration-response functions. Based on Pope et al. (2002) mean curve (solid line) with pointwise 95% confidence intervals (dashed lines). (Source: CD, Figure 8-7).

response relationship at concentration ranges below this level. In addition, 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.8-98).

In summary, while staff recognizes that individual thresholds may likely exist for specific health responses, existing studies do not support or refute the existence of population thresholds for PM-mortality relationships, for either long-term or short-term PM exposures within the range of air quality observed in the studies (CD, p. 9-44). While epidemiologic analyses have not identified population thresholds in the range of air quality concentrations in the studies, it is possible that such thresholds exist within or below these ranges but cannot be detected due to variability in susceptibility across a population. Even in those few studies with suggestive evidence of population thresholds, the potential thresholds are at fairly low concentrations (CD, p. 9-45). Based on the above considerations, staff concludes that it is appropriate to focus on linear or log-linear concentration-response models reported in the studies for quantitative risk assessment. Recognizing that population thresholds may well exist below the lowest air quality levels observed in the studies, staff concludes it is not appropriate to extrapolate below these levels. Further, to address the possibility that population thresholds may exist at fairly low levels within the range of air quality observed in the studies, staff concludes that it is appropriate to consider alternative hypothetical threshold levels in the context of sensitivity analyses within the PM risk assessment.

3.7 SUMMARY AND CONCLUSIONS

Based on the available evidence and the evaluation of that evidence in the CD, summarized briefly above, staff concludes that the body of evidence supports an inference of causality for associations between $PM_{2.5}$ and a broad range of health effects. Short-term exposure to $PM_{2.5}$ is likely causally associated with mortality from cardiopulmonary diseases, hospitalization and emergency department visits for cardiopulmonary diseases, increased respiratory symptoms, decreased lung function, and physiological changes or biomarkers for cardiac changes. Long-term exposure to $PM_{2.5}$ is likely causally associated with mortality from cardiopulmonary diseases and lung cancer, and effects on the respiratory system such as decreased lung function or the development of chronic respiratory disease. Staff concludes that

there is less strength, but suggestive evidence of causality for short-term exposure to $PM_{10-2.5}$ and indicators of morbidity, including hospitalization for cardiopulmonary diseases, increased respiratory symptoms and decreased lung function. Staff concludes that it is appropriate to consider including the health outcomes listed above in quantitative assessments for $PM_{2.5}$ and $PM_{10-2.5}$. Further, staff notes that more equivocal evidence is available for other PM-health responses, such as associations between short-term exposure to $PM_{10-2.5}$ and mortality, and between PM and effects on infants. Staff believes that less certain evidence, while not appropriate for quantitative assessment, can inform more general assessments of the evidence.

Several issues that are relevant to the interpretation of health evidence for quantitative assessment of PM-related effects are discussed above. Measurement error and exposure error are issues that are distinctly more important for interpretation of results for $PM_{10-2.5}$ than $PM_{2.5}$. For $PM_{10-2.5}$, there is greater uncertainty in the relationship between ambient PM measured at central monitors and individuals' exposure to ambient PM, based on both variability in $PM_{10-2.5}$ concentrations across an area and decreased ability for coarse particles to penetrate into buildings. This uncertainty is likely to increase the confidence intervals around effect estimates. In interpreting results of associations with $PM_{10-2.5}$, staff places greater emphasis on evaluating results from the pattern of findings in multiple studies than on statistical significance of any individual result.

In the evaluation of different epidemiologic model specifications, as described above, some effect estimates differ upon reanalysis to address issues associated with the use of the default GAM procedures, but many are little affected. Recognizing that there is no single "correct" analytical approach, staff concludes that it is appropriate for quantitative assessment to use results from short-term exposure studies that were reanalyzed with more stringent GAM criteria or with other approaches such as GLM, or that did not use GAM in the original analysis.

Regarding potential confounding by co-pollutants, the CD concludes that the evidence supports the existence of independent effects of PM, while recognizing the difficulties in distinguishing effects from mixtures of correlated pollutants. Staff concludes that single-pollutant model effect estimates can be used as reasonable indicators of the magnitudes of effect sizes, with sensitivity analyses to evaluate the influence of adjustment for co-pollutants.

The CD concludes that distributed lag periods may provide the most representative quantitative estimates of effect for some health outcomes, such as mortality. Recognizing that distributed lags have not been used in the available studies of $PM_{2.5}$ and $PM_{10-2.5}$, staff concludes that a reasonable approach to selection of effect estimates for use in quantitative assessment is to evaluate the pattern of lag results available from studies. If the data show a reasonable pattern of results, then selecting a single lag period is appropriate, recognizing that this result is likely to underestimate effects. Conversely, if the pattern of results across lag periods is unstable, staff concludes that it is inappropriate to use such results for quantitative assessment.

For the long-term exposure studies, recent studies indicate that long-term PM exposure is likely to be better estimated from air quality data averaged over longer time periods (e.g., multiple years of data). Staff concludes that effect estimates based on PM data averaged over longer times periods are more representative of population health responses for use in risk assessment. Specifically, for the results from the extended analysis of the ACS study, staff concludes that it is most appropriate to use the concentration-response functions from the models using averaged air quality data over the full study time period for quantitative assessment.

Finally, evaluation of the health effects data summarized in the CD provides no evidence to support selecting any particular population threshold for $PM_{2.5}$ or $PM_{10-2.5}$, recognizing that it is reasonable to expect that, for individuals, there may be thresholds for specific health responses. Staff observes that uncertainty in the concentration-response function increases at the low end of the range of concentrations. Even in those studies where the existence of population thresholds is suggested, they are at fairly low concentrations. For the PM risk assessment, staff concludes that it is appropriate to focus on linear or log-linear concentration-response models reported in the studies, while considering alternative hypothetical threshold levels in the context of sensitivity analyses. Staff also concludes it is not appropriate to extrapolate below the lowest PM concentrations reported in the studies.

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4. CHARACTERIZATION OF HEALTH RISKS

4.1 INTRODUCTION

This chapter describes and presents the 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 1997 PM NAAQS review. This updated assessment includes estimates of (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 and discussed in Chapter 5 of this draft Staff Paper. The risk assessment discussed in this Chapter is more fully described and presented in the draft technical support document, *Particulate Matter Health Risk Assessment for Selected Urban Areas: Draft Report* (Abt Associates, 2005; henceforth referred to as the Technical Support Document (TSD) and cited as TSD).

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 both the actual values of model input variables (parameter uncertainty) and the physical systems or relationships (model uncertainty – e.g., the shapes of concentration-response (C-R) functions). In any risk assessment uncertainty is, ideally, reduced to the maximum extent possible, but significant uncertainty often remains. It can be reduced by improved measurement and improved model formulation. In addition, the degree of uncertainty can be characterized, sometimes quantitatively. For example, the statistical uncertainty surrounding the estimated $PM_{2.5}$ and $PM_{10-2.5}$ coefficients in the reported C-R functions is reflected in the confidence intervals provided for the risk estimates in this chapter and in the TSD. Additional uncertainties are addressed quantitatively through sensitivity analyses and/or qualitatively and are discussed in more detail in section 4.2.7.

As noted above, the updated risk assessment presents qualitative and quantitative considerations of uncertainty, including sensitivity analyses of key individual uncertainties. Given the existing data gaps in the scientific evidence and associated uncertainties, a more comprehensive integrated assessment of uncertainties, while desirable, would require use of techniques involving elicitation of probabilistic judgments from health scientists. While the Agency is currently developing these approaches, such comprehensive assessments of uncertainty are not available for the current risk assessment for this PM NAAQS review.

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 and composition 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 2003 air quality data for most locations and the most recent available mortality baseline incidence rates (from 2001)). However, we have not tried to predict future changes in inputs (e.g., future population levels or possible changes in baseline incidence rates).

The goals of the updated PM risk assessment are: (1) to provide estimates of the potential magnitude of PM-associated mortality and morbidity associated with current $PM_{2.5}$, and $PM_{10-2.5}$ levels and with attaining the current suite of $PM_{2.5}$ NAAQS (as well as the additional estimated

reductions in effects associated with alternative $PM_{2.5}$ and $PM_{10-2.5}$ standards identified as part of this review) in specific urban areas,[1] (2) to develop a better understanding of the influence of various inputs and assumptions on the risk estimates (e.g., choice of policy-relevant background (PRB) levels, consideration of potential hypothetical thresholds), and (3) to gain insights into the nature of the risks associated with exposures to ambient PM (e.g., patterns of risk reduction associated with meeting alternative annual and daily standards). 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 must take into account the significant uncertainties associated with this assessment, discussed in section 4.2.7 below.

4.1.1 Summary of Risk Assessment Conducted During Prior PM NAAQS Review

For the last review cycle, 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 included sensitivity analyses and integrated uncertainty analyses to better understand the

[1]Risk estimates associated with current PM_{10} levels also have been included in an appendix to the TSD for those urban areas where $PM_{2.5}$ risks have been estimated to provide additional context.

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

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 1997 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 ambient PM 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).

4.1.2 Development of Updated Assessment

The scope and methodology for the updated PM risk assessment have been developed over the last three years. 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, 2002) 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} (Hopke, 2002).

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. In August 2003 OAQPS released a draft technical report describing its draft PM risk assessment (Abt Associates, 2003b) in conjunction with the 1st draft Staff Paper.[3] The CASAC provided its comments on the draft PM risk assessment in its letter to the Administrator (Hopke, 2004). The revised draft risk assessment discussed in this Chapter and in the TSD (Abt Associates, 2005) has taken into consideration the CASAC and public comments received on the 2003 drafts and the evaluation of the health effects literature contained in the final CD.

4.2 GENERAL SCOPE OF PM RISK ASSESSMENT

As discussed in Chapter 3 above, the CD concludes (p.9-79) that "a growing body of evidence both from epidemiological and toxicological studies also supports the general conclusion that $PM_{2.5}$ (or one or more $PM_{2.5}$ components), acting alone and/or in combination with gaseous co-pollutants are likely causally related to cardiovascular and respiratory mortality and morbidity." With respect to $PM_{10-2.5}$, the CD (p.9-80) finds that there is "a much more limited body of evidence ... suggestive of associations between short-term (but not long-term) exposures to ambient coarse-fraction thoracic particles... and various mortality and morbidity effects observed at times in some locations." The CD further concludes that there is somewhat stronger evidence for coarse-fraction particle associations with morbidity (especially respiratory) endpoints than for mortality. As discussed in greater detail in Chapter 3, the evidence relating $PM_{10-2.5}$ concentrations and premature mortality is equivocal and, therefore, the quantitative risk assessment presented here and included in the TSD (Abt Associates, 2005) only includes morbidity health endpoints for $PM_{10-2.5}$. The $PM_{10-2.5}$ risk assessment is more limited than the $PM_{2.5}$ assessment because of the more limited air quality data as well as the smaller number of studies for which there is sufficient evidence to use in this assessment.

The updated risk assessment being conducted for the current NAAQS review is premised on the assumption that elevated ambient $PM_{2.5}$ concentrations are causally related to the mortality, morbidity, and symptomatic effects (alone and/or in combination with other

[3]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 primarily focuses on two PM indicators – $PM_{2.5}$ and $PM_{10-2.5}$.

pollutants) observed in the epidemiological studies. Similarly, the risk assessment for $PM_{10-2.5}$ is premised on the assumption that elevated ambient $PM_{10-2.5}$ concentrations are causally related to morbidity and symptomatic effects observed in the epidemiological studies. Staff concludes that these assumptions are well supported by the evaluation contained in the 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 PM indicators and the specific health endpoints (e.g., cardiovascular hospital admissions, COPD hospital admissions) and that the degree of uncertainty is directly related to differences in the relative weight of evidence.

This $PM_{2.5}$ risk assessment focuses on selected health endpoints such as increased excess daily mortality and mortality associated with long-term exposure, and increased hospital admissions for respiratory and cardiopulmonary causes and increased respiratory symptoms for children associated with short-term exposure. The $PM_{10-2.5}$ risk assessment includes increased hospital admissions for respiratory and cardiopulmonary causes and increased respiratory symptoms for children associated with short-term exposure. A consequence of limiting the assessment to these 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, the broad range of 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, community-oriented, ambient monitors. As discussed earlier in Chapter 2 (section 2.7) and Chapter 3 (section 3.6.2), measurements of daily variations of ambient PM 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 to ambient $PM_{2.5}$ for the populations represented by ambient monitoring stations. The CD concludes that "at this time, 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" (CD, p. 5-121). 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 3.6.2).

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 5 of the 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 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 scenarios in which air quality just meets the current set of standards;

- analyze additional reductions in 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; and

- 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, community-oriented monitors, the appropriate application of these functions in a PM risk assessment similarly requires the use of air quality data from fixed-site, community-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 $PM_{2.5}$ and 3 urban areas for $PM_{10-2.5}$. In addition, to provide some additional context, PM_{10} risk estimates are provided in Appendix I of Abt Associates (2005), for the same urban areas and short-term exposure health endpoints for which $PM_{2.5}$ and $PM_{10-2.5}$ risk estimates are available. As discussed in section 4.2.2. these areas have been chosen based on availability of PM C-R relationships, adequate PM air quality data, and baseline incidence 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).

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 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 available, are provided in Appendix 4A.

For long-term exposure mortality associated with $PM_{2.5}$, the LMLs for the relevant $PM_{2.5}$ epidemiology studies are 7.5, 10, and 11 $\mu g/m^3$, for the ACS-extended, ACS, and Six Cities studies, respectively. These LMLs are higher than the range of estimated $PM_{2.5}$ background levels in either the Eastern or Western U.S.. 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 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 would be underestimated to the extent that annual average $PM_{2.5}$ concentrations below the LMLs contribute to long-term exposure mortality. Where the LML for the epidemiology study that served as the basis for the C-R relationship was either below the estimated background PM concentration for an area or was not available, risks were only estimated above background PM concentrations. The rationale for this choice is that risks associated with concentrations above background are judged to be more relevant to policy decisions about the NAAQS than estimates that include risks potentially attributable to uncontrollable background PM concentrations.

The following sections 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 present key results from the assessment. A separate TSD (Abt Associates, 2005) is available which provides a more detailed discussion of the risk assessment methodology and additional 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 $PM_{10-2.5}$) 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 $PM_{2.5}$ and $PM_{10-2.5}$ from ambient monitors for the selected location, (2) estimates of background $PM_{2.5}$ and $PM_{10-2.5}$ concentrations appropriate for that location, and (3) a method for adjusting the "as is" data to reflect patterns of air quality estimated to occur when the 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; and

- 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 in an area before any changes in PM air quality.

Figure 4-1 provides a broad schematic depicting the role of these components in the risk assessment. Those points where EPA has conducted analyses of alternative assumptions, procedures, or data are indicated by a diamond 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 4-8). 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. While a significant number of new epidemiological studies have been published since the last review, and are evaluated in the CD, the PM risk assessment relies only on U.S. and Canadian studies to limit introducing uncertainty associated with the possible differences in population and characteristics of PM and co-pollutants between the U.S. and Canada 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 PM indicators are described below.

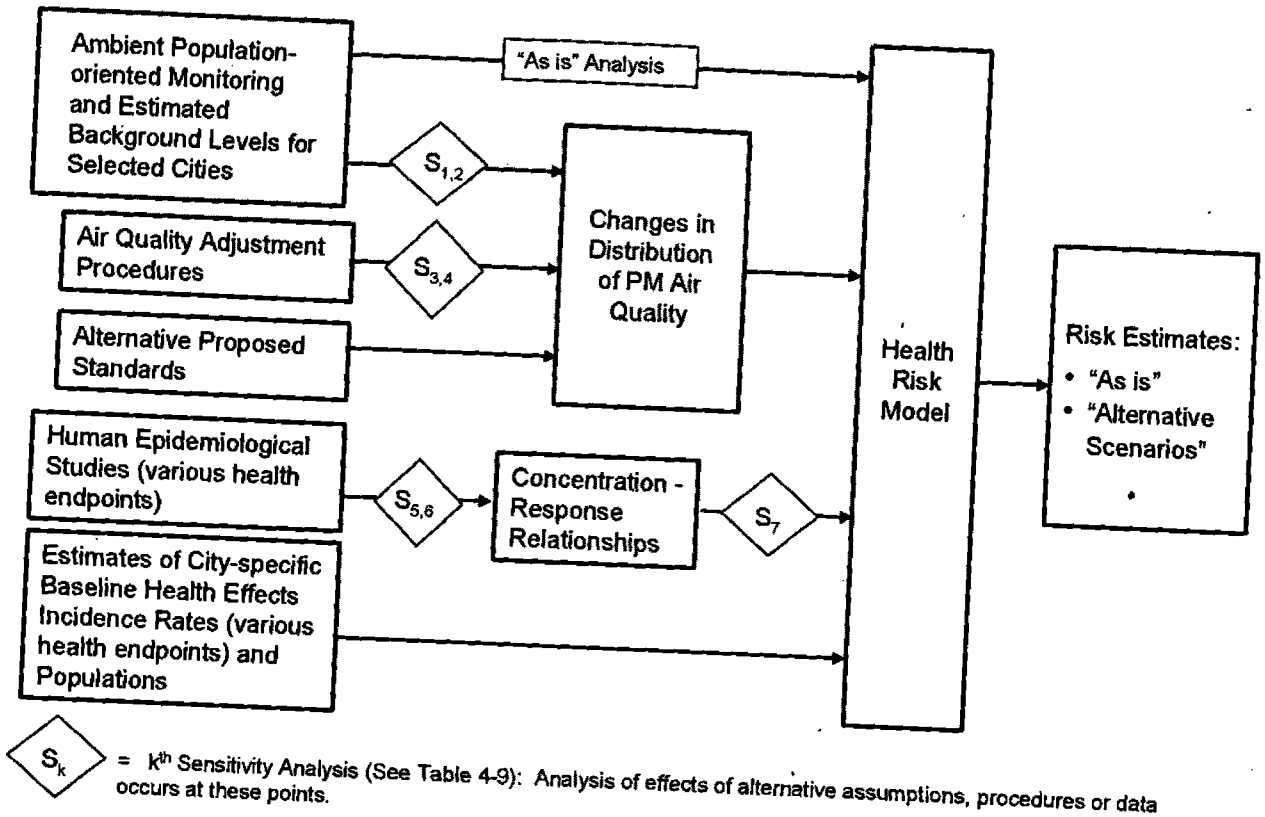

Figure 4-1. Major Components of Particulate Matter Health Risk Assessment.

4.2.2.1 Selection of Health Endpoint Categories

As discussed in Chapter 3, OAQPS staff carefully reviewed the health effects evidence evaluated in the 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 overall weight of the evidence from the collective body of studies supports the conclusion that there is likely to be a causal relationship between PM and the health effects category and for which baseline incidence data were available. In addition, 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 fine particles using $PM_{2.5}$ or $PM_{2.1}$, (2) estimating the concentration of fine particles using nepholometry data, and (3) estimating $PM_{10-2.5}$ concentrations based on co-located PM_{10} and $PM_{2.5}$ monitors or based on measurements using dichotomous samplers.

Based on a review of the evidence evaluated in the CD and discussed in Chapter 3, as well as the criteria discussed above, staff included the following broad categories of health endpoints in the risk assessment for $PM_{2.5}$:

related to short-term exposure:

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

related to long-term exposure:

- total, cardiopulmonary, and lung cancer mortality.

Other effects reported to be associated with $PM_{2.5}$, 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.

Based on a review of the evidence evaluated in the CD and discussed in Chapter 3, as well as the criteria discussed above, staff included the following categories of health endpoints associated with short-term exposures in the risk assessment for $PM_{10-2.5}$:

- hospital admissions for cardiovascular and respiratory causes;
- respiratory symptoms.

As discussed in Chapter 3 (section 3.4), more equivocal evidence is available for other health responses, such as associations between short-term exposure to $PM_{10-2.5}$ and mortality. Staff believe that these health endpoints, which are based on less certain evidence, are not appropriate for inclusion in the quantitative risk assessment. Staff have considered these endpoints in more qualitative assessments of the evidence presented in Chapter 3.

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 evaluation contained in the CD and staff assessment in Chapter 3 that suggests there may be 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 $PM_{2.5}$ 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 11 daily values per quarter based on data from co-located $PM_{2.5}$ and PM_{10} monitors. The criterion of at least 11 observations per quarter is based on EPA guidance on measuring attainment of the daily and annual PM standards and is contained in Appendix N of the July 18, 1997 Federal Register notice.

- 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).

[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.

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

- Where an area was considered based on PM-related hospital admission effects, an area had relatively recent area-specific baseline incidence data.

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 (i.e., hospital admissions). Based on a review of studies listed in Tables 8A and 8B of the CD (see also Appendices 3-A and 3-B 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.[5]

Staff next considered an indicator of study precision for the urban areas associated with the short-term exposure mortality studies identified in the first step. As discussed above in Chapter 3 (section 3.3.1.1) and in Chapter 8 of the CD (pp.8-324 - 8-325), 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 a rough indicator of the degree of precision of effect estimates; studies with larger values for this indicator should be accorded relatively greater study weight. While there is no bright line for selecting any particular cutoff, it was the staff's judgment to consider 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.[6] 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).

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[5]The Tolbert et al. (2000) study in Atlanta was excluded from consideration because the CD urged caution in interpreting these preliminary results given the incomplete and variable nature of the databases analyzed.

[6]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.

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 nine urban areas (i.e., Boston, Detroit, Los Angeles, Philadelphia, Phoenix, Pittsburgh, San Jose, Seattle, and St. Louis) in which epidemiological studies reported C-R relationships for $PM_{2.5}$ and mortality or hospital admissions and which had sufficient air quality data in a recent year.

The $PM_{2.5}$ risk assessment for long-term exposure mortality was conducted for nine urban areas. Eight of the nine urban areas, excluding Seattle, were already included in the $PM_{2.5}$ risk assessment based on short-term exposure mortality and are listed above. Since the C-R functions for $PM_{2.5}$-related mortality associated with long-term exposure used in the risk assessment are based on differences in long-term PM averages across multiple cities in the U.S., the issue of matching risk assessment locations with city-specific studies did not arise. Therefore, long-term exposure mortality risk estimates also were developed for Seattle.

Most of the short-term 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 $PM_{2.5}$ ambient air quality data). In addition, for the hospital admissions effect category, assessment was limited 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 Appendices 3A, 3B, and 4A of this draft Staff Paper and Appendix C of the TSD (Abt Associates, 2005).

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 $PM_{10-2.5}$ with the

morbidity endpoints (hospital admissions and respiratory symptoms) in any of the urban areas already selected for the $PM_{2.5}$ risk assessment. As summarized in Table 4-3 and noted earlier, 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 health endpoints and studies. Based on the available data, EPA has included in the $PM_{10-2.5}$ risk assessment the following health endpoints and locations: increased hospital admissions in Detroit and Seattle, and increased respiratory symptoms in St. Louis. Additional details about the epidemiological studies and the C-R functions used in the risk assessment based on these studies are provided in Appendices 3A, 3B, and 4A of this draft Staff Paper and Appendix C of the TSD (Abt Associates, 2005).

With respect to the $PM_{10-2.5}$ risk assessment, staff notes that the locations used in this part of the risk assessment are not representative of urban locations in the U.S. that experience the most significant elevated 24-hour $PM_{10-2.5}$ ambient concentrations. Thus, observations about risk reductions associated with alternative standards in the three urban areas (i.e., Detroit, Seattle, and St. Louis) may not be very relevant to the areas expected to have the greatest health risks associated with peak daily ambient $PM_{10-2.5}$ concentrations.

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 $PM_{2.5}$ and $PM_{10-2.5}$ from suitable monitors for each selected location, (2) estimates of background $PM_{2.5}$ and $PM_{10-2.5}$ 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 2003 from EPA's Air Quality System (AQS). As noted earlier, consistent with EPA guidance, 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. Generally, the most recent year of PM data was used for each study area and PM indicator subject to meeting this requirement.

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]			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, cardiopulmonary, 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

*Includes multi-city C-R function
[A] Reanalyzed in Ito (2003)
[B] Reanalyzed in Moolgavkar (2003)
[C] Reanalyzed in Sheppard (2003)

Table 4-3. 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 C-R function
[A] Reanalyzed in Ito (2003)
[B] Reanalyzed in Sheppard (2003)

Consistent with the approach used in the last PM risk assessment, a composite monitor data set was created for each assessment location based on averaging the 24-hour values from all monitors eligible for comparison with the standards for each day with any monitoring data. The resulting composite monitor data set provides a single series of daily concentrations for the urban area which serves as the surrogate index of exposure for the urban area. Table 4-4 provides a summary of the $PM_{2.5}$ and $PM_{10-2.5}$ ambient air quality data for the urban study areas based on the composite monitor values used 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 TSD (Abt Associates, 2005).

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 the U.S., Canada, and Mexico. For the base case risk estimates, the midpoint of the appropriate ranges of annual average estimates for $PM_{2.5}$ background presented in section 2.6 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 approximate mid-point of the annual average estimates for $PM_{10-2.5}$ background presented in section 2.6 were used. In sensitivity analyses, we examine the impact of assuming 1) a constant background set at the lower and upper ends of the range of estimated background levels for the eastern and western United States, depending on the assessment location and 2) a variable daily $PM_{2.5}$ background, using distributions whose means are equal to the values used in the base case analysis and whose distributions are based on an analysis of $PM_{2.5}$ data from relatively remote sites with the sulfate component removed (see Langstaff (2005)).

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). Since compliance with the standards is based on a 3-year average, air quality data from 2001 to 2003 have been used to determine the amount of reduction in $PM_{2.5}$ concentrations required to meet

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

Area	Population (millions)	$PM_{2.5}$**		$PM_{10-2.5}$**	
		Annual Avg.	24-hr Avg, 98th%	Annual Avg.	24-hr , 98th%
Boston, MA[a]	2.8	12.1	34.1		
Detroit, MI[b]	2.1	15.7	41.5	21.7	101.5
Los Angeles County, CA[c]	9.5	19.1	55.0		
Philadelphia County, PA[d]	1.5	14.3	38.4		
Phoenix, AZ[e]	3.1	10.4	28.9		
Pittsburgh, PA[f]	1.3	16.9	43.9		
San Jose, CA[g]	1.7	11.1	37.6		
Seattle, WA[h]	1.7	8.3	21.7	11.4	26.2
St. Louis[i]	2.5	14.0	30.6	12.0	24.1

*Based on air quality data for the year 2003, 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.
[b]Includes Wayne County.
[c]Includes Los Angeles County.
[f]Includes Hennepin and Ramsey Counties.
[d]Includes Philadelphia County.
[e]Includes Maricopa County; $PM_{2.5}$ air quality data are for 2001.
[f]Includes Allegheny County
[g]Includes Santa Clara County
[h]Includes King County
[i]Includes St. Louis, Franklin, Jefferson, St. Charles Counties in MO, Clinton, Madison, Monroe, and St. Claire Counties in IL and St. Louis City.

the current or alternative suites of standards. The amount of control has then been applied to a single year of data (i.e., 2003, unless otherwise specified) to estimate risks for a single year. Estimated design values (see Table 4-13 later in this Chapter) based on the highest community-oriented monitor within each study area are used to determine the percent adjustment necessary to just meet annual, 98th percentile daily, and 99th percentile daily standards.

Under the current annual $PM_{2.5}$ standard urban areas may, under certain circumstances, use the average of the annual averages of several monitors within an urban area to determine compliance, commonly referred to as the "spatial averaging approach." Therefore, a sensitivity analysis has been conducted for 3 urban areas to allow comparison of the estimated incidence and percent reduction in incidence associated with using either the highest monitor or the spatial average for determining the percent adjustment necessary to just meet the current and alternative annual standards.

The percent adjustment to simulate just meeting alternative standards is applied to the composite monitor for the urban area. The composite monitor is used because it is the best surrogate indicator of exposure that matches the type of exposure measure used in the original epidemiological studies.

When assessing the risks associated with long-term exposures, which use C-R functions from epidemiological studies that are specified in terms of long-term average concentrations, the annual mean is simply set equal to the standard level. In contrast, when assessing the risks associated with short-term exposures, which use C-R functions from epidemiological studies that consider the sequence of daily average concentrations, the distribution of 24-hour values that would occur upon just attaining a given 24-hour and/or annual PM standard has to be simulated.

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 more recent analysis of historical air quality data (see Abt Associates, 2005, Appendix B) have found that $PM_{2.5}$ levels in excess of estimated background concentrations in general have historically decreased in a roughly 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 $PM_{2.5}$ reductions that would result from just meeting a set of standards is to use a proportional adjustment (i.e., to decrease non-background PM levels on all days by the same percentage) for all concentrations exceeding the background level.[7] A sensitivity analysis also has been conducted to examine the impact on the $PM_{2.5}$ 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%).

Because the $PM_{10-2.5}$ historical air quality data are substantially more sparse, there was insufficient data to carry out the type of evaluation of historical data that was done for $PM_{2.5}$ to see whether the shape of the distribution of daily values has changed over time. In the absence of a clearly preferable alternative, the same proportional rollback approach used for $PM_{2.5}$ has been used for the $PM_{10-2.5}$ assessment. This increases the uncertainty about the $PM_{10-2.5}$ risk estimates associated with meeting alternative $PM_{10-2.5}$ standards.

In assessing health risks associated with $PM_{2.5}$ and $PM_{10-2.5}$, air quality just meeting the current or alternative $PM_{2.5}$ standards and alternative $PM_{10-2.5}$ standards is simulated by reducing the $PM_{2.5}$ or $PM_{10-2.5}$ 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 or ninety-ninth percentile daily values, depending on the form of the standard) with the level of the annual (or daily)

standard.[8] Because pollution abatement methods are applied largely to anthropogenic sources of $PM_{2.5}$ or $PM_{10-2.5}$, rollbacks were applied only to $PM_{2.5}$ or $PM_{10-2.5}$ concentrations above estimated background levels. Where sets of standards are considered, as is the case for $PM_{2.5}$ where both an annual and a daily standard are specified, the percent reduction is determined by the "controlling standard." The "controlling standard" is defined as the standard which would require the greatest reduction in PM levels to just meet the standard. For the current suite of

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[7] The portion of the distribution below the estimated background concentration is not rolled back, since air quality strategies adopted to meet the standards will not reduce the background contribution to PM concentrations.

[8] 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 community-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.

$PM_{2.5}$ standards, the existing annual standard of 15 $\mu g/m^3$ is the controlling standard for the five urban study areas (i.e., Detroit, Los Angeles, Philadelphia, Pittsburgh, and St. Louis) that do not meet the current standards. In four of these five urban areas suites of annual standards within the range of 12 to 15 $\mu g/m^3$ combined with the current daily standard of 65 $\mu g/m^3$, using a 98th percentile form, requires the same reduction as when these annual standards are combined with a daily standard of 40 $\mu g/m^3$, using the same daily form. Therefore, the risk assessment only included the 14 $\mu g/m^3$ annual standard combined with the current daily standard for the one location (i.e., Philadelphia) and annual standard scenario where there was a difference in the reduction required between daily standards of 40 and 65 $\mu g/m^3$.

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.[9] In this model,

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

where y is the incidence of the health endpoint of interest associated with ambient PM level x, β is the coefficient of ambient PM concentration, and B is the incidence of the health endpoint at x = 0, i.e., when there is no ambient $PM_{2.5}$ (or $PM_{10-2.5}$). 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 $PM_{2.5}$ (or $PM_{10-2.5}$) levels, $\Delta x = x_0 - x$, is then

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

[9]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 $PM_{2.5}$ (or $PM_{10-2.5}$) levels, Δx. Equations 4-2 and 4-3 are simply alternative ways of expressing the relation between a given difference in ambient $PM_{2.5}$ (or $PM_{10-2.5}$) 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 $PM_{2.5}$ and $PM_{10-2.5}$ health risk.

For the first part of the risk assessment, characterizing risks associated with "as is" ambient PM concentrations, Δx is the difference between the as is ambient PM concentration (on each day for the short-term exposure (i.e, daily or 24-hour) endpoints or the annual average for the long-term exposure (i.e., annual average or longer) endpoints and either the estimated PRB concentration or the LML in the epidemiology study providing the β, whichever is greater. For the second part of the risk assessment, characterizing the reduction in health effects incidence associated with alternative PM standards, Δx is the difference between the ambient PM concentration when the current PM standards are just met (on each day for the short-term exposure endpoints or the annual average for the long-term exposure endpoints) and the ambient PM concentration associated with just meeting the specified alternative standards.[10]

For short-term exposure health endpoints, the risk assessment first calculated the daily changes in incidence. Since most areas had at least some days for which no ambient PM concentration data were available, the estimated annual incidence was summed up for each quarter of the year and adjusted by using the ratio of the total number of days in each quarter to the number of days in the quarter for which air quality data was available.[11] This simple adjustment assumes that missing air quality data occur randomly within a quarter and that the

[10]For those areas already meeting the current $PM_{2.5}$ standards, Δx is the difference between the as is ambient PM concentration and the ambient PM concentration associated with just meeting the specified standards.

[11]Adjustment was done on a quarterly basis to reduce possible bias that would be introduced where missing data are not uniformly distributed throughout the year.

distribution of PM concentrations on the days with missing data is essentially the same as the distribution on days for which there are PM data. The quarterly incidence estimates were then summed to derive an annual estimate.

The daily time-series epidemiological studies used models estimating C-R functions in which the PM-related incidence on a given day depends only on some specified lagged PM concentration measure (e.g., 0-day lag, 1-day lag, 2-day lag, average of 0- and 1-day lag). As discussed in Chapter 3 (section 3.6.5.2), 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). As indicated in section 3.6.5.2, our use of single day lag models which ignore longer-term influences may result in the risk being underestimated. Currently, there is insufficient information to adjust for the impact of longer-term exposure (on the order of weeks or months) on mortality associated with short-term $PM_{2.5}$ 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.

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 just discussed, 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 be added together.

The statistical uncertainty surrounding the estimated $PM_{2.5}$ and $PM_{10-2.5}$ coefficients in the reported C-R functions is reflected in the confidence intervals provided for the risk estimates in sections 4.3 to 4.5. In addition, sensitivity analyses examine how the short- and long-term $PM_{2.5}$

exposure mortality risk estimates would vary if, instead of the reported C-R relationships, 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 were applied instead of the single lag models reported in the literature for short-term exposure mortality. 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. Finally, $PM_{2.5}$ risk estimates based on alternative model specifications, including the impact of different lags, statistical models (i.e., GAM vs. GLM), and degrees of freedom allowed (i.e., 30 vs. 100) are shown for short-term exposure mortality and morbidity endpoints in Los Angeles are included in the TSD (Abt Associates, 2005). The results of these sensitivity analyses are discussed 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. 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[12] and are summarized in Table 4-5. Where possible, county-specific incidence or incidence rates have been used. County-specific mortality incidences were available for the year 2001 from CDC Wonder (CDC, 2001), an interface for public health data dissemination provided by the Centers for Disease Control (CDC). The baseline mortality rates for each risk assessment location are provided in Table 4-6.

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 risk

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

assessment locations.[13] Baseline hospitalization rates used in each $PM_{2.5}$ and $PM_{10-2.5}$ risk assessment location are summarized in Table 4-7. 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 $PM_{2.5}$ risk assessment for short-term exposure include mortality, hospital admissions, and respiratory symptoms not requiring hospitalization and long-term exposure mortality is also estimated. The health endpoints that have been included in the $PM_{10-2.5}$ risk assessment for short-term exposure include hospital admissions and respiratory symptoms not requiring hospitalization. These health endpoints were included in the risk assessment because 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 these specific health endpoints. Once it had been determined that a health endpoint was to be included in the assessment, inclusion of a study on that health endpoint to estimate the magnitude of the response 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 8A and 8B of the CD (see also Appendices 3A and 3B of this SP).

[13]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.

Table 4-5. Relevant Population Sizes for PM Risk Assessment Locations

City	Population[a]							
	Total	Ages 7-14	Ages ≥25	Ages ≥30	Ages <65	Ages ≥ 65	Ages <75	Ages ≥75
Boston[1]	2,806,000	283,000 (10%)	1,903,000 (68%)	1,673,000 (60%)	---		---	---
Detroit[2]	2,061,000		---	1,153,000 (56%)	---	249,000 (12%)	---	---
Los Angeles[3]	9,519,000		---	5,092,000 (53%)	---	927,000 (10%)	---	---
Philadelphia[4]	1,518,000		---	852,000 (56%)	---	---	---	---
Phoenix[5]	3,072,000		---	1,684,000 (55%)	---	359,000 (12%)	---	---
Pittsburg[6]	1,282,000		---	814,000 (64%)	---	---	1,166,000 (91%)	116,000 (9%)
San Jose[7]	1,683,000		---	965,000 (57%)	---	---	---	---
Seattle[8]	1,737,000		---	1,044,000 (60%)	1,555,000 (90%)	---	---	---
St. Louis[9]	2,518,000	308,000 (12%)	1,637,000 (65%)	1,475,000 (59%)	---	---	---	---

[a] Total population and age-specific population estimates taken from the CDC Wonder website are based on 2000 U.S. Census data. See http://factfinder.census.gov/. Populations are rounded to the nearest thousand. The urban areas given in this exhibit are those considered in the studies used in the $PM_{2.5}$ risk assessment. The percentages in parentheses indicate the percentage of the total population in the specific age category.

[1] Middlesex, Norfolk, and Suffolk Counties. [2] Wayne County. [3] Los Angeles County. [4] Philadelphia County.
[5] Maricopa County. [6] Allegheny County. [7] Santa Clara County. [8] King County.
[9] St. Louis, Franklin, Jefferson, St. Charles, Clinton (IL), Madison (IL), Monroe (IL), and St. Clair (IL) Counties and St. Louis City.

Table 4-7. Baseline Hospitalization Rates for PM Risk Assessment Locations*

Health Effect	Detroit[1]	Los Angeles[2]	Seattle[3]
Hospital Admissions (per 100,000 general population/year)			
Pneumonia admissions (65 and over): ICD codes 480-486	250	—	—
COPD and asthma admissions (all ages): ICD codes 490-496	—	318	—
COPD and asthma admissions (65 and over): ICD codes 490-496	192	—	—
Asthma (<65): ICD code 493	—	—	92
Cardiovascular admissions (65 and over): ICD codes: 390-429	—	728	—
Ischemic heart disease (65 and over): ICD codes 410-414	487	—	—
Dysrhythmias (65 and over): ICD code 427	161	—	—
Congestive heart failure (65 and over): ICD code 428	341	—	—

[a]Hospitalization rates are presented only for the locations in which the C-R functions were estimated. For each location, the number of discharges was divided by the location's population from the 2000 U.S. Census estimates to obtain rates. All incidence rates are rounded to the nearest unit.
1. Wayne County. Year 2000 hospitalization data were obtained from the Michigan Health and Hospital Association.
2. Los Angeles County: Year 1999 hospitalization data were obtained from California's Office of Statewide Health Planning and Development – Health Care Information Resource Center.
3. King County. Year 2000 hospitalization data were obtained from the State of Washington Department of Health, Center for Health Statistics, Office of Hospital and Patient Data Systems.

As discussed in the CD (section 8.4.2) and Chapter 3 (section 3.6.3), 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 time-series studies employing generalized additive models (GAMs) in a commonly used statistical software package. 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, and based on the earlier staff conclusion in Chapter 3 (section 3.6.3) that models using more stringent GAM criteria provide the most representative effect estimate sizes, 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 Los Angeles.[14] It should be noted that the GAM stringent C-R functions 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 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.9-35).

More detailed information about the C-R relationships used in the PM risk assessment is provided in Appendix 4A of this draft Staff Paper. 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 5th and 95th confidence intervals.

[14] $PM_{2.5}$ risk estimates for various combinations of statistical estimation approaches (GAM and GLM with varying degrees of freedom) have been included for Los Angeles as a sensitivity analysis to illustrate the impact of alternative model specification choices.

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 health effects evidence examining whether or not a population threshold might exist for short- and long-term exposure health outcomes for $PM_{2.5}$ and short-term exposure health outcomes for $PM_{10-2.5}$ is discussed in section 3.6.6 of this SP and section 8.4.7 of the CD.

The $PM_{2.5}$ base case risk assessments presented in sections 4.3 and 4.4 below do not include a threshold, based on the conclusions in the CD that "there is no strong evidence of a clear threshold for PM mortality effects" and that the use of linear PM effect models appears to be appropriate (CD, p.8-345) . The base case 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 discussed in section 3.6.6, while the CD concludes that there is no strong evidence of a clear threshold for PM mortality effects, it also notes "nor is there clear evidence against possible thresholds for PM-related effects" (p.8-322). The CD also states that "some single-city studies do provide some suggestive hints for possible thresholds, but not in a statistically clear manner" (p.8-322). Therefore, as noted earlier, sensitivity analyses have been conducted that do include hypothetical alternative thresholds, where risks only are estimated due to $PM_{2.5}$ or $PM_{10-2.5}$ concentrations exceeding the assumed threshold concentrations. Based on the staff evaluation contained in section 3.6.6, three hypothetical thresholds (10, 15, and 20 $\mu g/m^3$) were included in sensitivity analyses for short-term exposure mortality for $PM_{2.5}$ and short-term exposure morbidity for $PM_{10-2.5}$ and two hypothetical thresholds (10 and 12 $\mu g/m^3$) were included in sensitivity analyses for long-term exposure mortality associated with $PM_{2.5}$. 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, 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 discussed in section 3.6.4 the 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 four gaseous co-pollutants examined. While not definitive, these consistent patterns indicate that it is more likely that there is an independent effect of $PM_{2.5}$ that is not appreciably modified by the gaseous co-pollutants.

For some of the gaseous co-pollutants, such as CO, NO_2, and SO_2, which tend to be highly correlated with ambient $PM_{2.5}$ concentrations in some cities (and, in the case of NO_x and SO_x, are PM precursors as well), it is difficult to sort out whether these pollutants are exerting any independent effect from that attributed to $PM_{2.5}$. As discussed in section 3.6.4, inclusion of pollutants that are highly correlated with one another can lead to misleading conclusions in identifying a specific causal pollutant. When such 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.8-241). Given that single and multi-pollutant models each have both potential advantages and disadvantages, with neither type clearly preferable over the other in all cases, risk estimates based on both single and multi-pollutant models have been developed.

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 CD (section 8.4.4) and in section 3.6.5 of this SP. As noted in those discussions, it is important to consider the pattern of results that is seen across the series of lag periods. When there is an observed pattern showing effects across different lags, use of the single-day lag with the largest effect, while reasonable, is likely to underestimate the overall effect size (since the largest single-lag day results do not fully capture the risk also distributed over adjacent or other days)(CD, p.8-270).

As discussed in the CD, a number of the $PM_{2.5}$ short-term exposure mortality studies reported stronger associations with shorter lags, with a pattern of results showing larger

associations at the 0- and 1-day lag period that taper off with successive lag days for the varying PM indicators. Several studies included in the $PM_{2.5}$ risk assessment only included 0- and 1-day lags in presenting results. Therefore, when a study reports several single day lag models, unless the study authors identify a "best lag", both the 0- and 1-day lag models for mortality (both total and cause-specific) were chosen for inclusion in the $PM_{2.5}$ risk assessment. In one study conducted in Los Angeles (Moolgavkar, 2003), there was no consistent pattern observed across the various lags examined for COPD mortality. Therefore, EPA did not include this particular endpoint in the $PM_{2.5}$ risk assessment for Los Angeles.

For hospital admissions, unless the study authors specified an optimal lag, both 0- and 1-day lag models were included for cardiovascular admissions since the CD indicates that recent evidence from time series studies strongly suggests maximal effects at 0-day lag with some carryover to 1-day lag and little evidence for effects beyond 1-day for this health endpoint (CD, p.8-279). Since many of the studies addressing COPD hospital admissions report effects at somewhat longer lags, 0-, 1-, and 2-day lag models (if all three were available) were included in the risk assessment for this health endpoint, unless the authors selected a different "best lag."

As discussed in section 3.6.5.2, there is recent evidence (Schwartz, 2000b, 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). As noted above, 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. Because of this, a distributed lag model may be preferable to a single lag model. However, distributed lag models have been used in only a few cases and only for PM_{10}. 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 CD and in section 3.6.5.2:

- 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.

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). This sensitivity analysis was included to provide a very rough sense of the possible underestimation of risk due to use of single-day lags models.

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

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. As discussed in section 3.3.1.2, based on the evaluation contained in the CD and the staff's assessment of the complete data base addressing mortality associated with long-term exposure to $PM_{2.5}$, staff have concluded that two cohorts that have been studied are particularly relevant for the $PM_{2.5}$ risk assessment. These include the Six Cities study cohort (referred to here as Krewski et al. (2000) - Six Cities) and the American Cancer Society (ACS) cohort (referred to as Krewski et al. (2000) – ACS) containing a larger sample of individuals from many more cities. 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). EPA's use of these particular cohort studies to estimate health risks associated with long-term exposure to $PM_{2.5}$ is consistent with the views expressed in the NAS (2002) report, "Estimating the Public Health Benefits of Proposed Air Pollution Regulations," and the SAB Clean Air Act Compliance Council review of the proposed methodology to estimate the health benefits associated with the Clean Air Act (SAB, 2004). Risk estimates have been developed using C-R functions from the Six Cities, ACS, and ACS-extended studies. As explained in section 3.6.5.4, three different indicators of long-term $PM_{2.5}$ exposure were considered in this extended ACS study and staff have selected the C-R function associated with an average of the 1979-1983 and 1999-2000 $PM_{2.5}$ ambient concentrations to use in the current risk assessment.

4.2.7 Characterizing Uncertainty and Variability

This section discusses the approaches used in the current PM risk assessment to address, and characterize, where feasible, uncertainties and variability. Although the weight of the

evidence is sufficient to support the conclusions in the CD that a variety of health endpoints are likely causally related to short- and long-term ambient exposures to $PM_{2.5}$ and short-term ambient exposures to $PM_{10-2.5}$, significant uncertainties remain affecting the quantitative assessment of health risks associated with varying exposure levels. The following briefly summarizes the major sources of these uncertainties and variability and how they are dealt with in the risk assessment :

- Causality. There is uncertainty about whether each of the estimated associations between the two PM indicators ($PM_{2.5}$ and $PM_{10-2.5}$) and the various health endpoints included in this risk assessment 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. For example, there is much greater uncertainty associated with the morbidity effects associated with $PM_{10-2.5}$ exposures compared to $PM_{2.5}$ due to the much smaller health effects data base. Chapter 3 presents a more detailed discussion of the staff's qualitative assessment of the varying weight of evidence associated with the effects included in the risk assessment.

- Empirically estimated C-R relationships. In estimating the C-R relationships, there are uncertainties: (1) surrounding estimates of PM coefficients in C-R functions used in the assessment, (2) concerning the specification of the C-R model (including the shape of the C-R relationship) and whether or not a population threshold exists within the range of concentrations examined in the studies, and (3) 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 and daily PM concentrations. For the few instances where multi-city PM C-R functions are included in the risk assessment (e.g., use of the Six-Cities study function for respiratory symptoms associated with short-term exposures to $PM_{2.5}$ applied in Boston and St. Louis), there also is uncertainty related to the transferability of PM C-R functions from multiple locations to the specific location selected for the risk assessment.[15] Statistical uncertainty, based on the standard errors reported in the epidemiology studies is incorporated in the risk assessment and is discussed below. Sensitivity analyses of potential alternative hypothetical thresholds also have been included in the risk assessment.

[15] A C-R function derived from a multi-cities study may not provide an accurate representation of the C-R relationship in a specific assessment location 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 exposure 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.

- Lag structure. There is some evidence from a few PM_{10} studies that the impact of any single day of exposure may be to cause effects across a number of subsequent days (i.e., a distributed lag), however most epidemiology studies have only analyzed single day lags. The use of single day lag C-R functions could result in a downward bias in the estimated incidence associated with a given reduction in PM concentrations. However, there are no available $PM_{2.5}$ or $PM_{10-2.5}$ studies that included distributed lag models. As discussed below, a limited sensitivity analysis has been conducted to illustrate the potential impact on $PM_{2.5}$ mortality risk estimates associated with short-term exposures.

- Extrapolation of C-R relationship beyond the range of observed PM data. There is significant uncertainty about the shape of the C-R relationship beyond the range of the PM data observed in the epidemiology studies. Risk estimates have not been calculated for PM levels below the lowest measured level (LML) in a study, if it was available. Where the LML was not available, risk was estimated only down to an estimated background level. This approach minimizes the uncertainty for risk estimates associated with concentrations within the range of the studies.

- Adequacy of ambient PM monitors as surrogate for population exposure. The extent to which there are differences in the relationship between spatial variation in ambient $PM_{2.5}$ or $PM_{10-2.5}$ concentrations and ambient exposures in the original epidemiology studies compared to more recent ambient $PM_{2.5}$ or $PM_{10-2.5}$ data introduces additional uncertainty in the risk estimates. This is expected to be more of a concern for $PM_{10-2.5}$ where greater spatial variability in ambient monitoring data within urban areas and over time has been observed.

- Adjustment of air quality distributions to simulate just meeting alternative standards. The shape of the daily distribution of $PM_{2.5}$ and $PM_{10-2.5}$ ambient concentrations that would result upon meeting alternative PM standards is unknown. Based on an analysis of historical data, staff believes it is a reasonable assumption that $PM_{2.5}$ concentrations would be reduced by roughly the same percentage. However, there is much greater uncertainty associated with the use of this same approach for meeting $PM_{10-2.5}$ standards given the lack of sufficient data to evaluate the reasonableness of this assumption.

- Background concentrations. Since risks have only been estimated in excess of background, where the LML is either not available or is lower than the estimated background, uncertainty about background concentrations contributes to uncertainty about the risk estimates. As discussed below, sensitivity analyses examining the impact of alternative constant and varying daily background levels on the risk estimates have been conducted.

- Baseline incidence rates and population data. There are uncertainties related to: (1) 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 simulate just meeting the current or

alternative standards; (2) the use of annual incidence rate data to develop daily health effects incidence data; and (3) related to the use of an overall combined incidence rate for six cities for the respiratory symptoms endpoint which is applied to individual cities (i.e., Boston and St. Louis). Variability in baseline incidence and population data is taken into account by use of city-specific data in most cases.

The uncertainties from some of these sources -- in particular, the statistical uncertainty surrounding estimates of the PM coefficients in C-R functions -- are characterized quantitatively in the PM risk assessment. It is 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.[16] In situations where the point estimate for a C-R function is positive, but the lower confidence limit estimate is less than 1.0, the lower confidence limit of the risk estimate is a negative value. Based on the overall body of evidence on the relationships between PM and health effects, the staff believes that these negative estimates should not be interpreted as implying that increasing PM levels will result in reduced risks, but rather that the negative risk estimates are simply a result of statistical uncertainty in the reported C-R relationships in the epidemiological studies.

Steps also have been taken to minimize some of the uncertainties noted above. For example, the current PM risk assessment includes only health endpoints for which the CD evaluation and staff assessment (see Chapter 3) find that the overall weight of the evidence supports the conclusion that $PM_{2.5}$ is likely causally related, or for $PM_{10-2.5}$ is suggestive of a causal relationship. Also, for most of the health endpoints and locations included in the risk assessment, this assessment uses the C-R functions derived from epidemiological studies carried out in those same locations. This serves to minimize the uncertainties, such as differences in composition and differences in factors affecting human exposure associated with applying C-R functions developed in one location to a different location.

As noted above, a variety of sensitivity analyses, summarized in Table 4-8, have been included in the risk assessment to address some of the major uncertainties. The results of these

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

sensitivity analyses are summarized in sections 4.3.2 (for as is risk estimates), 4.4.2 (for just meeting the current $PM_{2.5}$ standards), and 4.5.3 (for meeting alternative $PM_{2.5}$ and $PM_{10-2.5}$ standards).

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

4.3.1 Base Case Risk Estimates

The base case risk estimates associated with "as is" $PM_{2.5}$ and $PM_{10-2.5}$ concentrations in excess of background levels are presented 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. For each series of estimates, a point estimate is provided along with 95% confidence intervals. As noted above, in some cases, where the lower confidence limit of the C-R function is less than 1.0, the resulting lower confidence limit of the risk estimate is a negative value. The staff's interpretation of these negative values is that while they indicate statistical uncertainty about the C-R relationships, they do not at all suggest that risk reductions would be associated with an increase in PM levels. 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 TSD (Abt Associates, 2005). 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 4-2 through 4-6 present the $PM_{2.5}$ risk estimates across the various assessment locations associated with "as is" concentrations in excess of either background or the LML in the study providing the C-R function, whichever is greater. Figure 4-2 compares risk estimates for total non-accidental mortality incidence associated with short-term (i.e., 24-hour) exposure to $PM_{2.5}$ using single-pollutant, single-city models. The point estimates are in the range from about 0.5 to 2.5% of total non-accidental mortality incidence. Figure 4-3 compares the estimated percent of total incidence for non-accidental and cause-specific mortality associated with short-term exposure to $PM_{2.5}$ based on single city versus multi-city models. Generally, the estimated incidence for the single- and multi-city models are roughly comparable, with somewhat lower risk estimates seen in Boston for the multi-city models compared to the single-city models and

Table 4-8. Sensitivity Analyses

Analysis Number (Figure 4-1)	PM Indicator	Component of the Risk assessment	Sensitivity Analysis or Comparison
1	$PM_{2.5}$, $PM_{10-2.5}$	Air Quality	A sensitivity analysis of the effect of assuming different (constant) background PM levels
2	$PM_{2.5}$	Air Quality	A sensitivity analysis of the effect of assuming a constant background PM level versus a distribution of daily background levels
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 sensitivity analysis of the effect of just meeting the current and alternative annual $PM_{2.5}$ standards using the maximum versus the average of monitor-specific averages
5	$PM_{2.5}$	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	$PM_{2.5}$, $PM_{10-2.5}$	Concentration-Response	Sensitivity analyses assuming alternative hypothetical threshold concentration levels for the occurrence of $PM_{2.5}$- and $PM_{10-2.5}$-related response at concentrations above those for background or the LML for as is air quality, and for just meeting the current and alternative $PM_{2.5}$ standards.

Source: Abt Associates (2005).

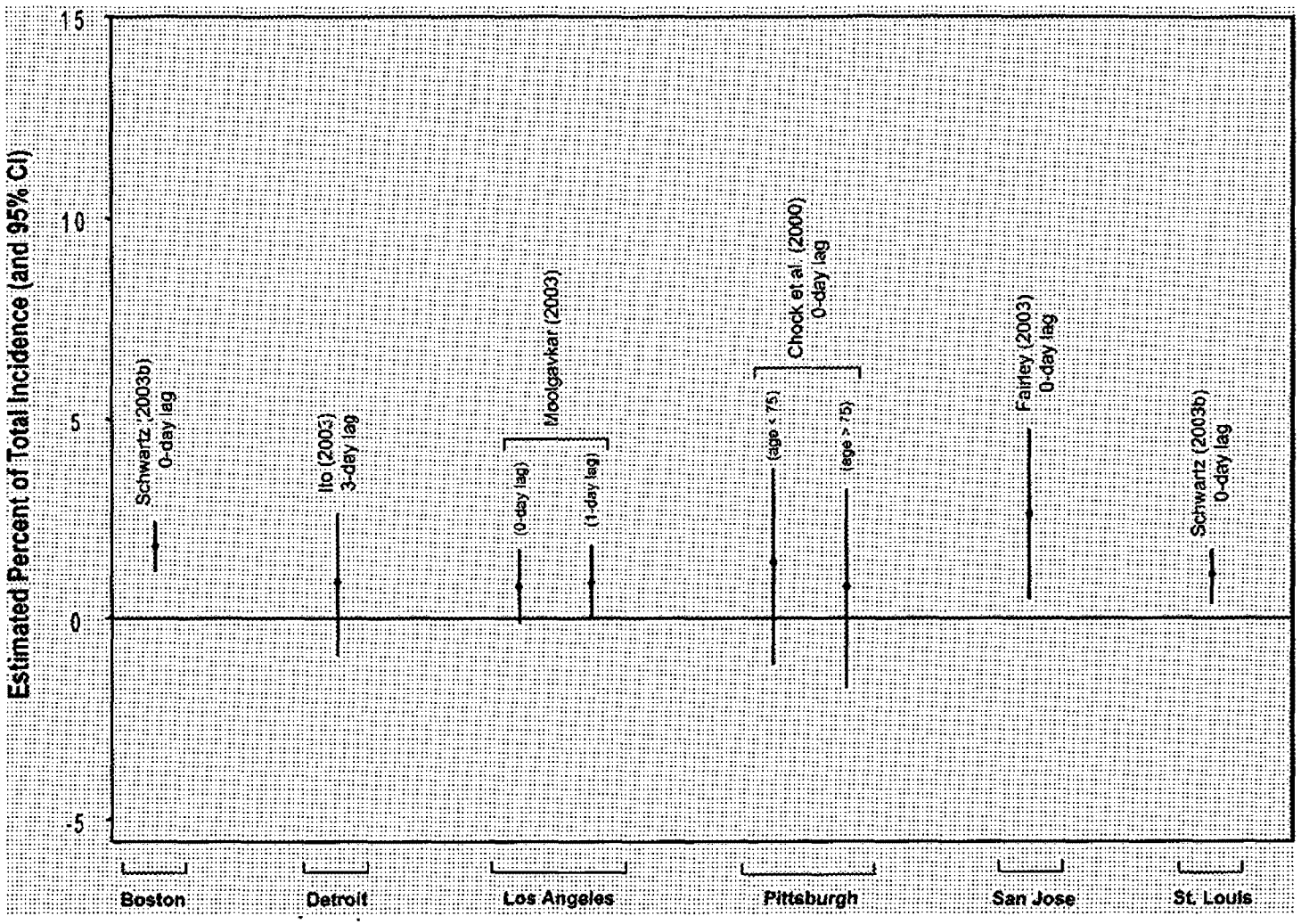

Figure 4-2. Estimated Annual Percent of Total (Non-Accidental) Mortality Associated with Short-Term Exposure to $PM_{2.5}$ (and 95 Percent Confidence Interval): Single-Pollutant, Single-City Models. Source: Abt Associates (2005)

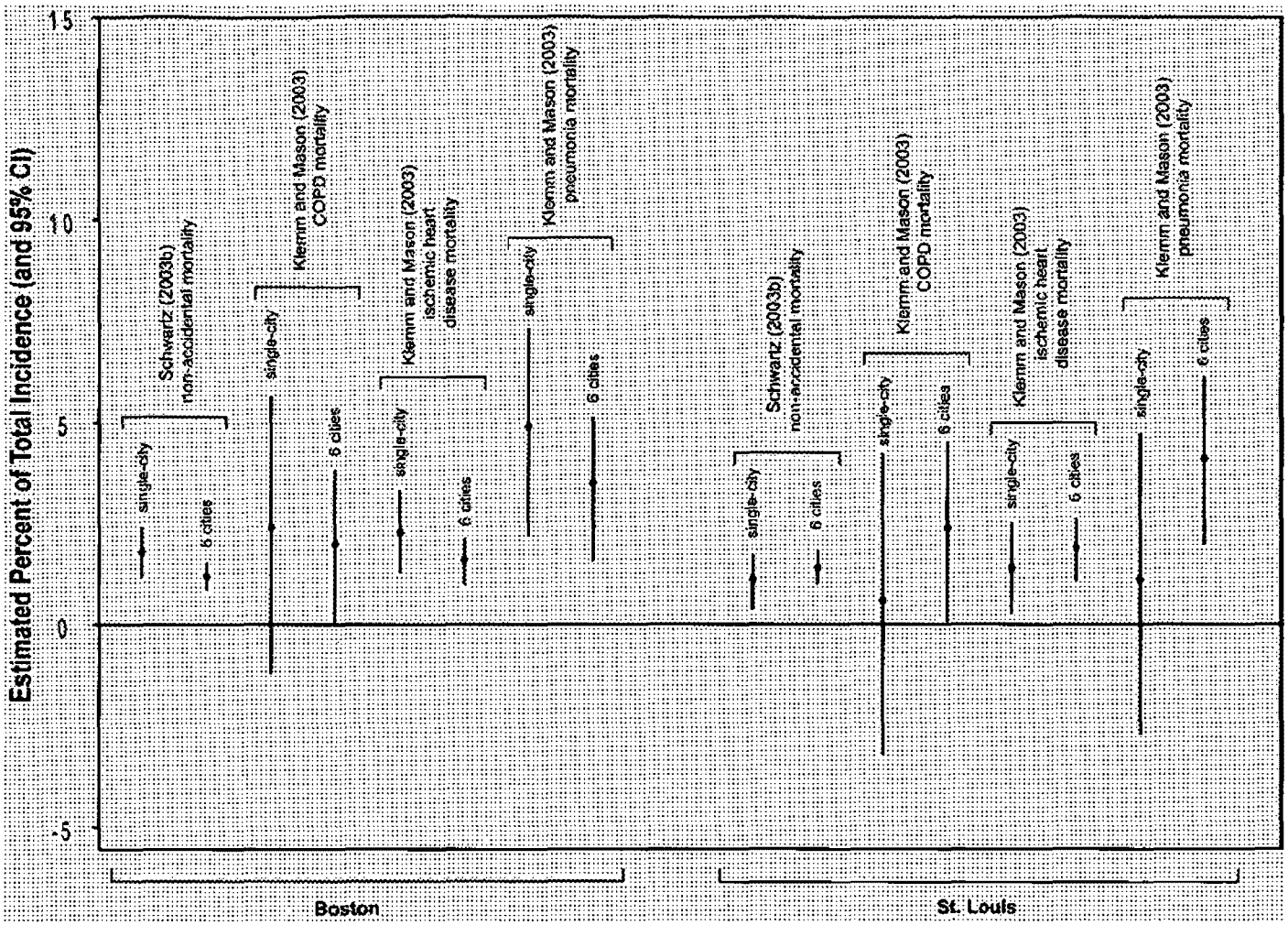

Figure 4-3. Estimated Annual Percent of Health Effects Associated with Short-Term Exposure to $PM_{2.5}$ (and 95 Percent Confidence Interval): Results Based on Single-City versus Multi-City Models. Source: Abt Associates (2005)

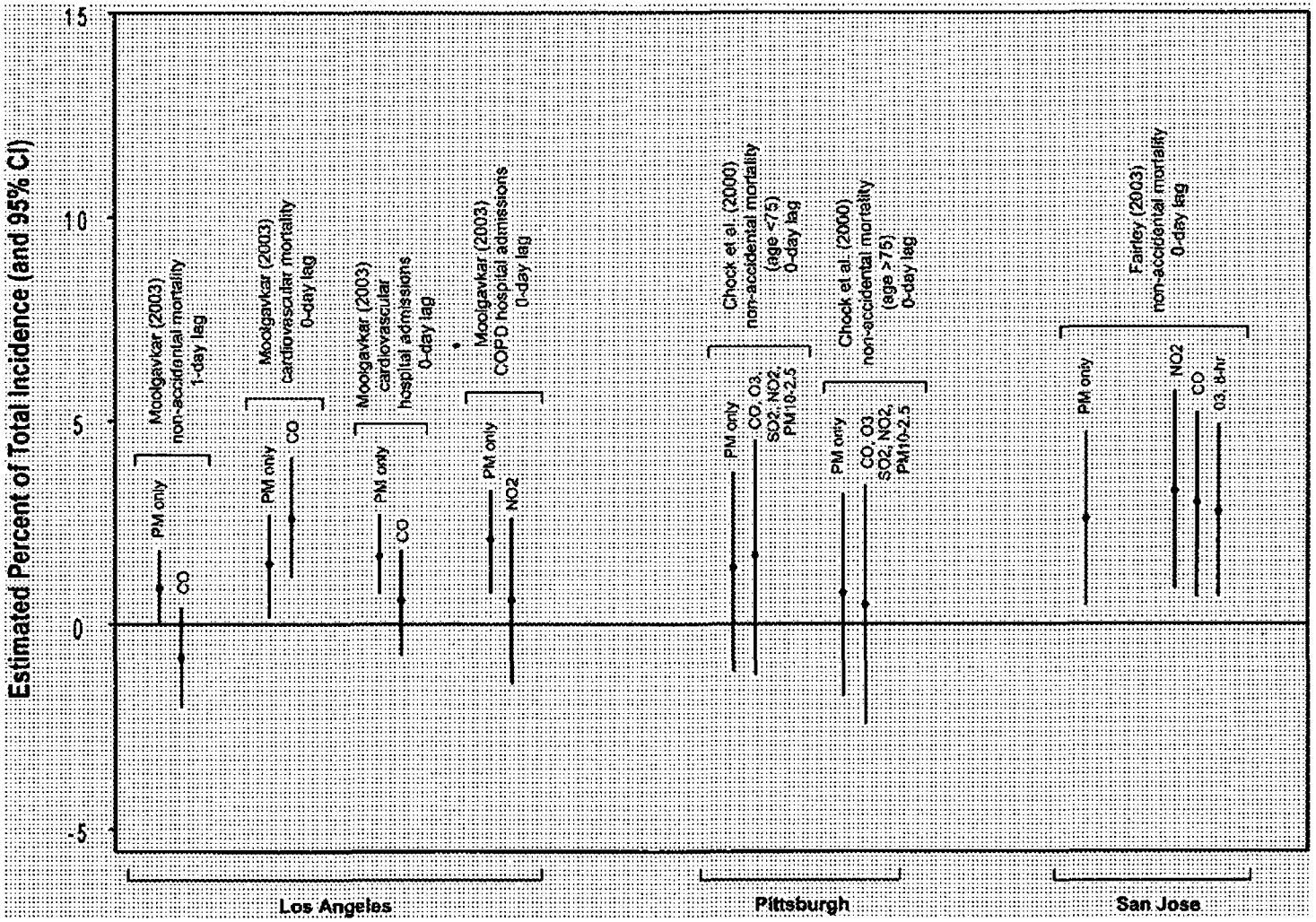

Figure 4-4. Estimated Annual Percent of Health Effects Associated with Short-Term Exposure to $PM_{2.5}$ (and 95 Percent Confidence Interval): Results Based on Single-Pollutant versus Multi-Pollutant Models. Source: Abt Associates (2005)

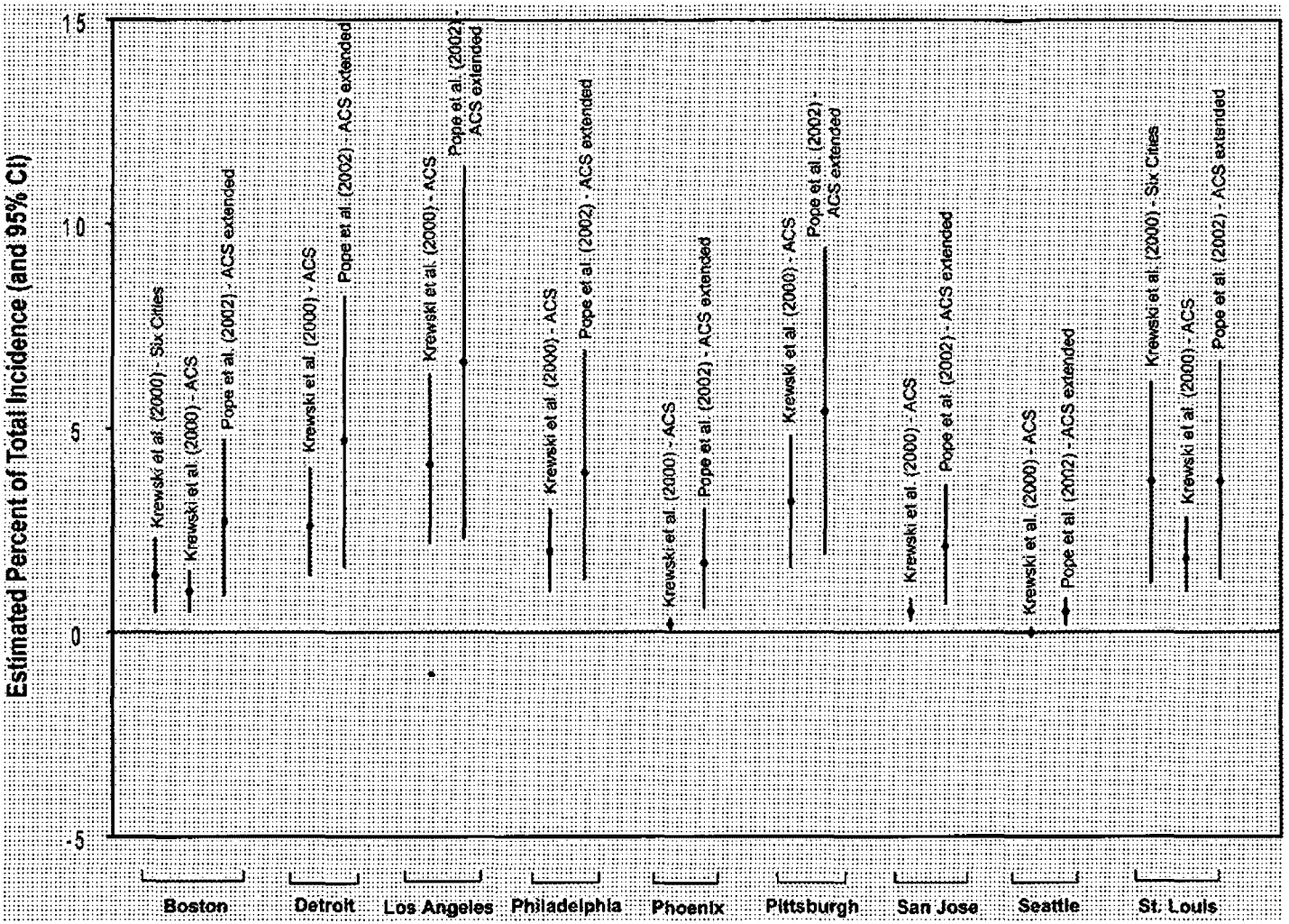

Figure 4-5. Estimated Annual Percent of Mortality Associated with Long-Term Exposure to $PM_{2.5}$ (and 95 Percent Confidence Interval): Single-Pollutant Models. Source: Abt Associates (2005)

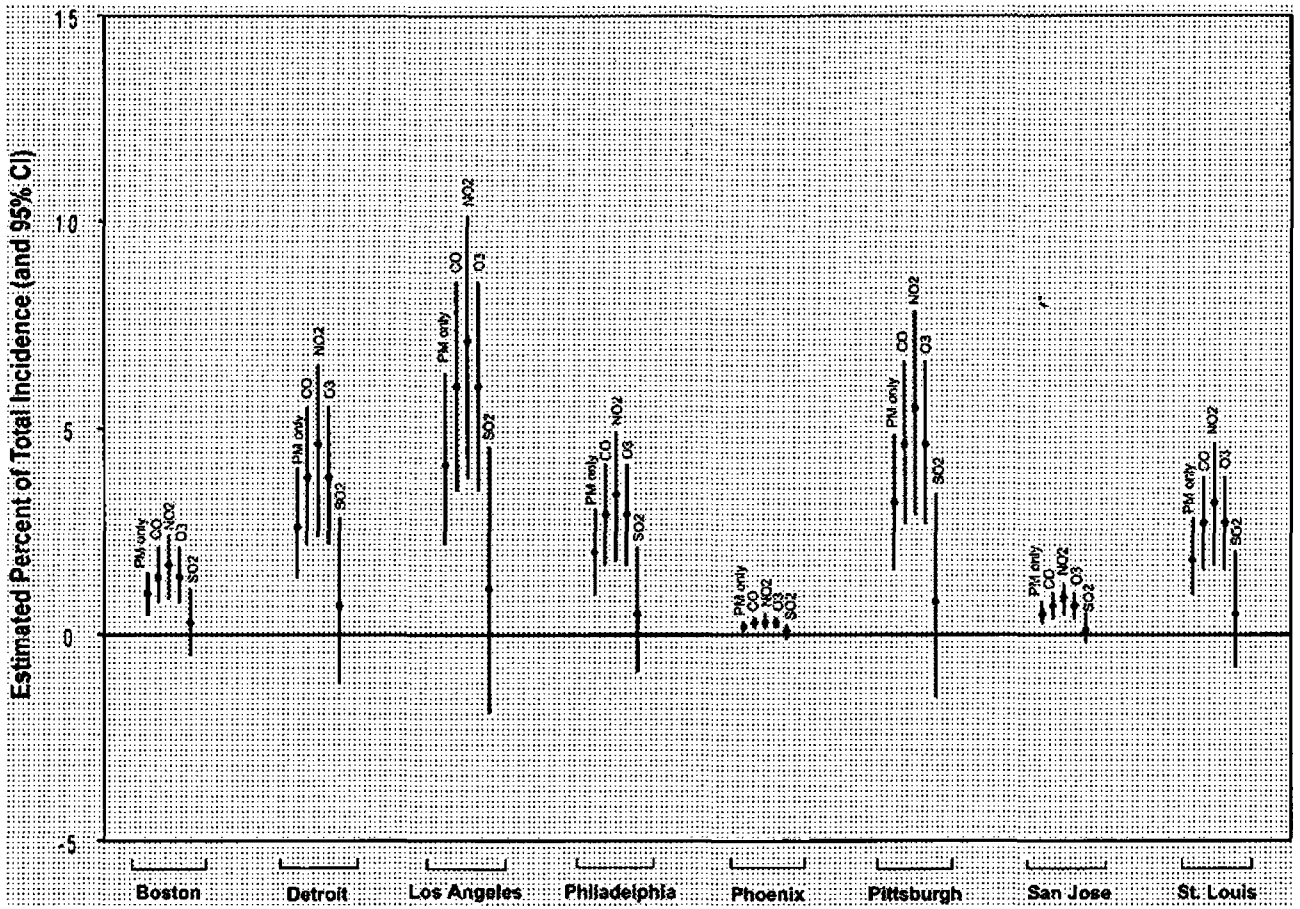

Figure 4-6. Estimated Annual Percent of Mortality Associated with Long-Term Exposure to $PM_{2.5}$ (and 95 Percent Confidence Interval): Single-Pollutant and Multi-Pollutant Models (Based on Krewski et al. (2000) - ACS Study).

Source: Abt Associates (2005)

the reverse being observed in St. Louis. As expected, the 95% confidence intervals are somewhat smaller for the multi-city models compared to the respective single-city models which is due to the greater sample size in the multi-city models.

Figure 4-4 compares risk estimates based on single-pollutant versus multi-pollutant C-R models provided in the epidemiological studies for $PM_{2.5}$ short-term exposure health endpoints. In two cases there is relatively little difference in the risk estimates between the single-pollutant and multi-pollutant models (i.e., Pittsburgh and San Jose), while in the third case (Los Angeles) there are larger differences when either CO or NO_2 are added to the model along with PM. Figures 4-5 and 4-6 show risk estimates for mortality related to long-term (i.e., annual average) exposure to $PM_{2.5}$ based on single- and multi-pollutant models, respectively. The point estimates for the single-pollutant models, based on the ACS-extended study (Pope et al., 2002), range from 0.5% in Seattle to as high as 6.6% of total mortality in Los Angeles, with most point estimates falling in the 2 to 5% range. The point estimates based on the original ACS study (Krewski et al., 2000) are lower in Phoenix, Seattle, and San Jose (ranging from 0 to 0.5%) because the "as is" annual averages at the composite monitors in these locations were not much higher than the LML in the ACS study (i.e., 10 $\mu g/m^3$) and risk estimates only were calculated down to the LML. As shown in Figure 4-6, the risk estimates based on multi-pollutant models, involving addition of different single co-pollutants 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.[17]

Figure 4-7 shows risk estimates for hospital admissions and respiratory symptoms associated with short-term exposure to $PM_{10-2.5}$ for three urban areas (Detroit, Seattle, and St. Louis). For Detroit risk estimates are provided for several categories of cardiovascular and respiratory-related hospital admissions and show point estimates ranging from about 2 to 7% of cause-specific admissions being associated with as is short-term exposures to $PM_{10-2.5}$. The point estimate for asthma hospital admissions associated with $PM_{10-2.5}$ exposures for Seattle, an area

[17] 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.

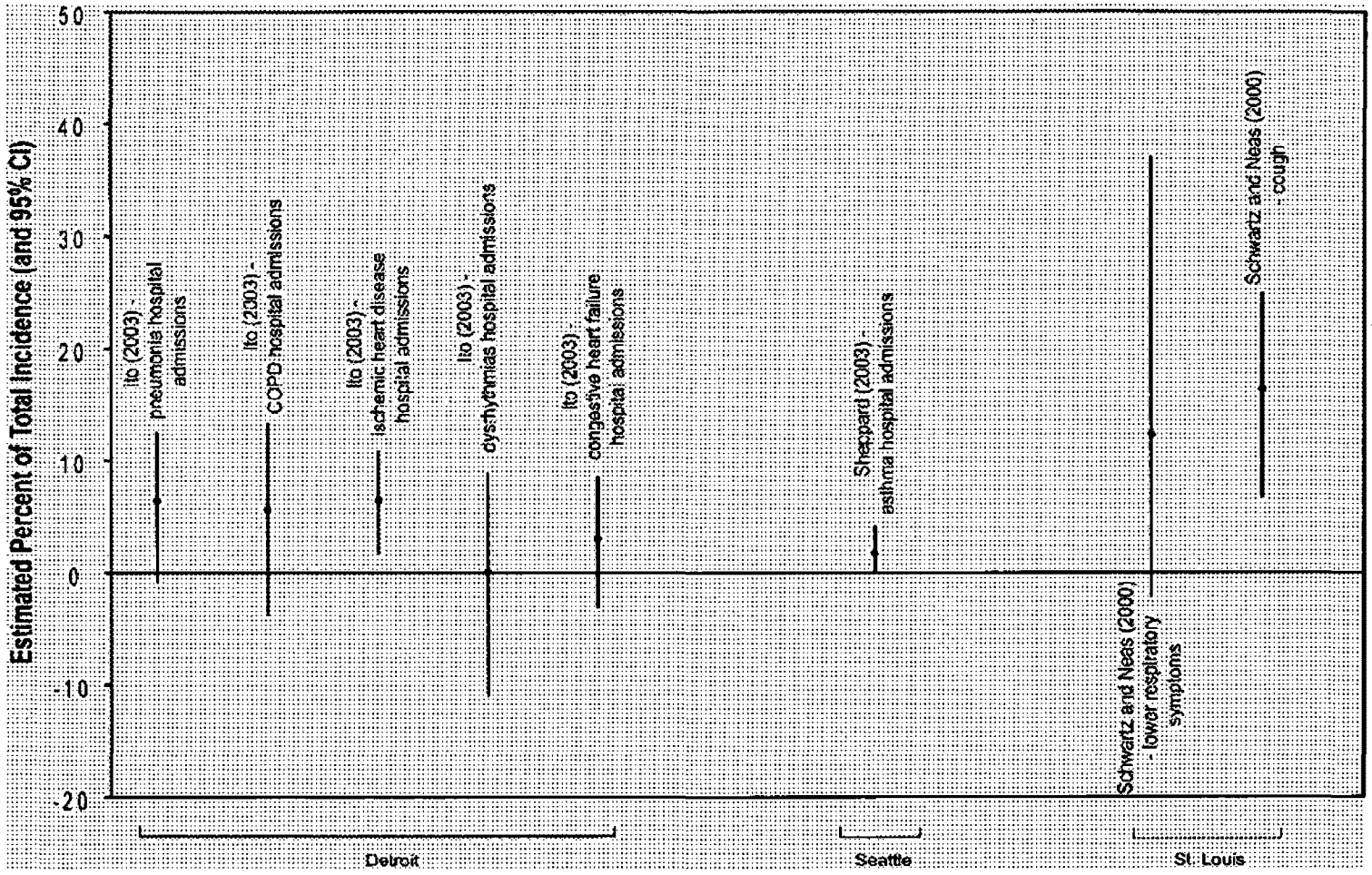

Figure 4-7. Estimated Annual Percent of Health Effects Associated with Short-Term Exposure to $PM_{10-2.5}$ (and 95 Percent Confidence Interval)
Source: Abt Associates (2005)

with lower $PM_{10-2.5}$ ambient concentrations, is about 1%. Point estimates for lower respiratory symptoms and cough in St. Louis are about 12 and 15%, respectively.

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 4-8 above for a summary of different types of sensitivity analyses). Most of these sensitivity analyses were conducted in each of the study areas and the complete results are in the TSD (Abt Associates, 2005). 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. In some cases, sensitivity analyses were conducted only in one location due to data constraints (e.g., only Los Angeles for alternative C-R model specifications since it was the only study that presented results for a wide range of alternative model specifications).

4.3.2.1 Alternative Background Levels

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 the U.S., Canada, and Mexico (discussed in section 2.6). One set of sensitivity analyses examined the impact of using the lower and upper end of the range of estimated background concentrations provided in section 2.6. For Detroit, the use of alternative estimated $PM_{2.5}$ background levels had only a relatively small 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 4 $\mu g/m^3$, which is lower than the upper range of background levels considered in the sensitivity analysis (i.e., 2 to 5 $\mu g/m^3$). There was no difference in the base case where background was assumed to be 3.5 $\mu g/m^3$ versus setting background at the lower end of the range (2.0 $\mu g/m^3$). With the background set at 5 $\mu g/m^3$, the short-term exposure risk estimates were about 10% smaller than the base case.

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 roughly +/- 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 LMLs in the long-term studies were 7.5, 10 or 11 $\mu g/m^3$, which all are larger than the range of estimated $PM_{2.5}$ background levels.

A sensitivity analysis also was conducted that focused 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. Staff developed a Monte Carlo simulation approach to generate a year long series of daily $PM_{2.5}$ background concentrations for specific urban areas based on using available distributional information for the observed and background concentrations to estimate their joint distribution, which yields the distribution of the background concentrations conditioned on the level of the observed concentrations (see Langstaff, 2004 for additional details describing the approach). This approach involved assigning a background value to an observed concentration by randomly selecting a value from the conditional distribution corresponding to the observed value. The analysis was done both without any correlation assumed and with a 0.4 correlation between background and observed concentrations. To implement this approach, the mean of the background distribution was assumed to be the mid-point estimate of $PM_{2.5}$ background discussed in section 2.6. Estimates of the variation in background concentrations for different regions of the United States were obtained by an analysis of daily data from IMPROVE sites with the sulfate component removed (Langstaff, 2005). It is important to recognize that all IMPROVE sites measure some $PM_{2.5}$ from anthropogenic sources, and that removing sulfate from the $PM_{2.5}$ component considered does not completely remove all anthropogenic contributions to the observed concentrations.

The sensitivity analysis examining varying daily background was carried out in Detroit and St. Louis using as is air quality levels for short-term exposure non-accidental mortality associated with $PM_{2.5}$. As shown in exhibit 7.8 (Abt Associates, 2005), the difference between the risk estimates based on a constant versus a varying daily background were extremely small in

Detroit (i.e., 0.8 percent of total incidence with varying daily background vs. 0.9 percent with assumed constant background). The difference was even smaller in St. Louis in both the no correlation and 0.4 correlation cases, with essentially no difference in risk estimates between the constant and varying daily background cases (Abt Associates, 2005).

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 background concentrations within areas affected by wildfires and dust storms and long range transport from outside the United States, Canada, and Mexico (see section 2.6). While the current $PM_{2.5}$ base case risk estimates, therefore, do 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 2.6). The $PM_{2.5}$ risk assessment also included a sensitivity analysis which used 2002 air quality data for Boston to examine the impact of an extreme example (i.e., the Quebec fire episode in July 2002) of this type of natural episodic event on short- and long-term exposure mortality (see Exhibits 7.9 and 7.10 in Abt Associates, 2005). This sensitivity analysis showed that there was hardly any difference (i.e., differences ranged from 0 to 0.1% of total incidence) in estimated short-term exposure mortality associated with $PM_{2.5}$ when one included or excluded this fairly extreme, but

short-term episode.[18] This same sensitivity analysis showed a difference of about 0.2% in total long-term exposure mortality incidence associated with $PM_{2.5}$ with and without inclusion of the Quebec fire episode days.

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 about a 16% increase in the risk estimates for various respiratory and cardiovascular-related short-term exposure hospital admissions in Detroit between the base case (which used a value of 4.5 $\mu g/m^3$ for background) and the lower end where background was estimated to be 1 $\mu g/m^3$. At the upper end, where background was estimated to be 9 $\mu g/m^3$, the short-term exposure hospital admission risk

[18] This extreme episode included 2 days with $PM_{2.5}$ levels above 30 $\mu g/m^3$ and 1 day above 50 $\mu g/m^3$.

estimates were reduced by about 19% (see Exhibit 7.12 in the TSD (Abt Associates (2005)). The effect of different background concentrations for the other two $PM_{10-2.5}$ locations can be found in Exhibits D.84 and D.86 through D.89 in the TSD.

4.3.2.2 Hypothetical Thresholds

One of the most significant uncertainties continues to be the issue of hypothetical thresholds below which there may be no $PM_{2.5}$ or $PM_{10-2.5}$ health effects. As discussed above in sections 3.6.6 and 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 population thresholds exist within the range of concentrations examined. 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 two different hypothetical thresholds or cutpoints (10 and 12 $\mu g/m^3$) 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 hockey stick would be approximately a weighted average of the two slopes of a hockey stick - namely, zero and the slope of the upward-sloping portion of the hockey stick (see the TSD (Abt Associates, 2005) 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 $\mu g/m^3$ for long-term exposure mortality, given that the LMLs in the long-term exposure mortality studies were 7.5, 10, and 11 $\mu g/m^3$. Staff believes that the slope adjustment method used in this risk assessment is a reasonable approach to illustrate the potential impact of using a non-linear approach. A more definitive evaluation of the effect of hypothetical thresholds and use of alternative non-linear approaches would require re-analysis of the original health and air quality data, which is beyond the scope of this risk assessment.

The results of these sensitivity analyses examining the impact of hypothetical thresholds for short-term exposure mortality risk estimates for the "as is" $PM_{2.5}$ levels in Detroit show that

the short- and long-term exposure risk estimates are particularly sensitive to the application of hypothetical thresholds. A hypothetical threshold of 10 $\mu g/m^3$ reduces the percent of total non-accidental mortality incidence associated with short-term exposure to $PM_{2.5}$ from 0.9% to 0.5%, a 44% decrease in the risk estimate and the highest hypothetical threshold of 20 $\mu g/m^3$ reduces it to 0.2%, nearly an 80% reduction from the base case. To illustrate the impact on long-term exposure mortality, using the risk estimates based on the ACS-extended all cause mortality results, a hypothetical threshold of 10 $\mu g/m^3$ reduces the risk estimate from 4.7% of total incidence to 3.7%, a reduction of about 20% from the base case estimate and a hypothetical threshold of 12 $\mu g/m^3$ reduces the risk estimate to 2.7%, a reduction of over 40% from the base case estimate.

4.3.2.3 Alternative Concentration-Response Models

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 Associates, 2005; 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 4-9. 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.

- Various respiratory and cardiovascular cause-specific hospital admission point estimates associated with short-term exposure to $PM_{10-2.5}$ range from 1 to 7%, depending on location and type of admission. Point estimates for lower respiratory symptoms and cough were about 12 and 15% of total incidence for as is levels in a single urban area (St. Louis).

The wide variability in risk estimates associated with a recent year of air quality for the two 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 original 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.

Based on the results from the sensitivity analyses, the following key observations are made:

- The single most important factor influencing the risk estimates is whether or not a hypothetical threshold exceeding the estimated background level or LML in the studies exists.

- The following uncertainties have a moderate impact on the risk estimates in some or all of the cities: choice of an alternative estimated constant 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). Figures 4-8 (a,b) provide an example of the annual distribution of 24-hour $PM_{2.5}$ concentrations in Detroit and the corresponding

Health Effect and Model**	Urban Study Area	"As Is" Incidence	Incidence Remaining Upon Attaining Current $PM_{2.5}$ NAAQS	Percent of $PM_{2.5}$-Related Incidence Reduced	Reduction in Incidence Expressed as Percent of Total Incidence
total mortality (age ≥ 30)*** long-term exposure	Pittsburgh	816 (282 - 1430)	403 (141 - 699)	50.6 (50.1 - 51.1)	2.7 (0.9 - 4.8)
non-accidental mortality, short-term exposure, mean of lag 0 & 1-day, all ages (Schwartz, 2003)**	St. Louis	233 (86 - 379)	191 (70 - 311)	18.0 (17.9 - 18.0)	0.2 (0.1 - 0.3)
total mortality (age ≥ 30)*** long-term exposure	St. Louis	842 (290 - 1486)	596 (206 - 1047)	29.2 (29.0 - 29.8)	1.1 (0.4 - 1.9)

*Risk reductions are relative to the "as is" (year 2003) air quality base case risk estimates. **These risk reductions are based on single pollutant model from the study cited and include all ages unless otherwise noted. ***These risk reductions are based on the Pope et al. (2000) ACS-extended study.

- Various respiratory and cardiovascular cause-specific hospital admission point estimates associated with short-term exposure to $PM_{10-2.5}$ range from 1 to 7%, depending on location and type of admission. Point estimates for lower respiratory symptoms and cough were about 12 and 15% of total incidence for as is levels in a single urban area (St. Louis).

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Based on the results from the sensitivity analyses, the following key observations are made:

- The single most important factor influencing the risk estimates is whether or not a hypothetical threshold exceeding the estimated background level or LML in the studies exists.

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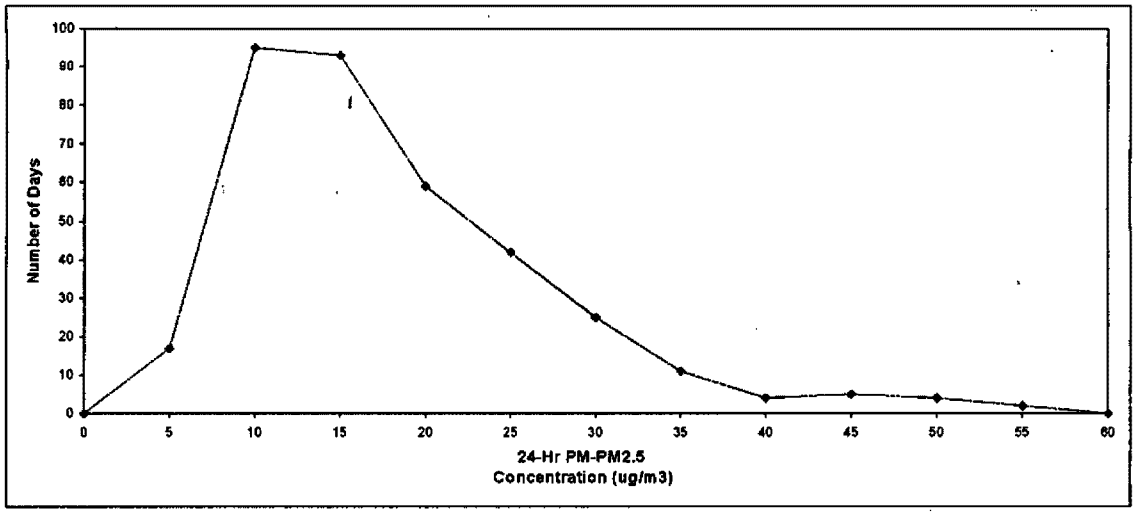

Figure 4-8a. Distribution of 24-Hour $PM_{2.5}$ Concentrations in Detroit (2003 Air Quality Data).

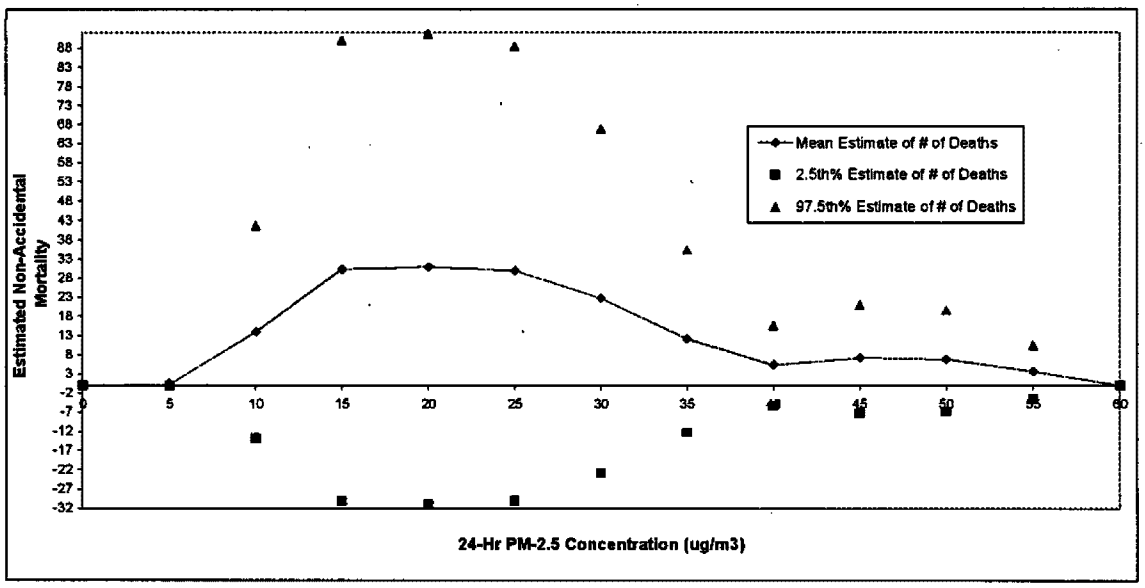

Figure 4-8b. Estimated Non-Accidental Mortality in Detroit Associated with $PM_{2.5}$ Concentrations (2003 Air Quality Data) (Based on Ito, 2003).

distribution of estimated mortality incidence (for $PM_{2.5}$) based on the short-term exposure epidemiology study included in the current PM risk assessment.[19] Consistent with the observation made in the previous PM NAAQS review, the highest 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 based on typical distributions observed in urban areas.

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

4.4.1 Base Case 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 2001-2003 air quality data (i.e., Detroit, Philadelphia, Pittsburgh, Los Angeles, and St. Louis). 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 risk assessment described in the TSD and discussed here, the approach used to simulate just meeting the current annual average standard for the base case risk estimates used the maximum of the monitor-specific annual averages as shown in Table 4-10. Since an area could potentially use the spatial average of the community-oriented monitors to determine whether or not it met the annual average standard, Table 4-10 also presents the percent rollbacks and annual average design values that would have resulted from using this alternative approach in each urban study area which does not meet the current annual standard and which meets the minimum criteria for

[19]The Detroit $PM_{2.5}$ example uses the C-R function for non-accidental mortality from Lippmann et al. (2000), reanalyzed in Ito (2003).

Table 4-10. 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 Current Annual $PM_{2.5}$ Standard		Design Value Based on 2001-2003 Data	
	Using Maximum of Monitor-Specific Annual Averages	Using Average of Monitor-Specific Annual Averages	Annual Based on Maximum Monitor	Annual Based on Average of Monitor-Specific Annual Averages
Detroit	28.1%	11.5%	19.5	16.5
Los Angeles*	59.2%	--	23.6	--
Philadelphia	10.9%	-0.9%	16.4	14.9
Pittsburgh	35.0%	22.8%	21.2	18.4
St. Louis	17.9%	13.5%	17.5	16.8

*Los Angeles does not meet the minimum requirements for use of spatial averaging.
Source: Abt Associates (2005)

use of spatial averaging. A sensitivity analysis examining the impact of using design values based on spatial averaging is discussed in section 4.5.3.2.

Drawing on the detailed risk estimates contained in Exhibit 8.1 and Appendix E of the TSD (Abt Associates, 2005), Figure 4-9 displays the estimated percent reductions in total incidence for non-accidental mortality associated with short-term exposure to $PM_{2.5}$ concentrations when air quality goes from as is concentrations to just meeting the current annual and daily $PM_{2.5}$ suite of standards in four of the risk assessment study area that do not meet the current standards.[20] The point estimates generally are in the range of 0.3 - 0.5 percent reduction

[20]Short-term exposure non-accidental mortality estimates were not included for Philadelphia because the C-R function did not include confidence limits for this endpoint.

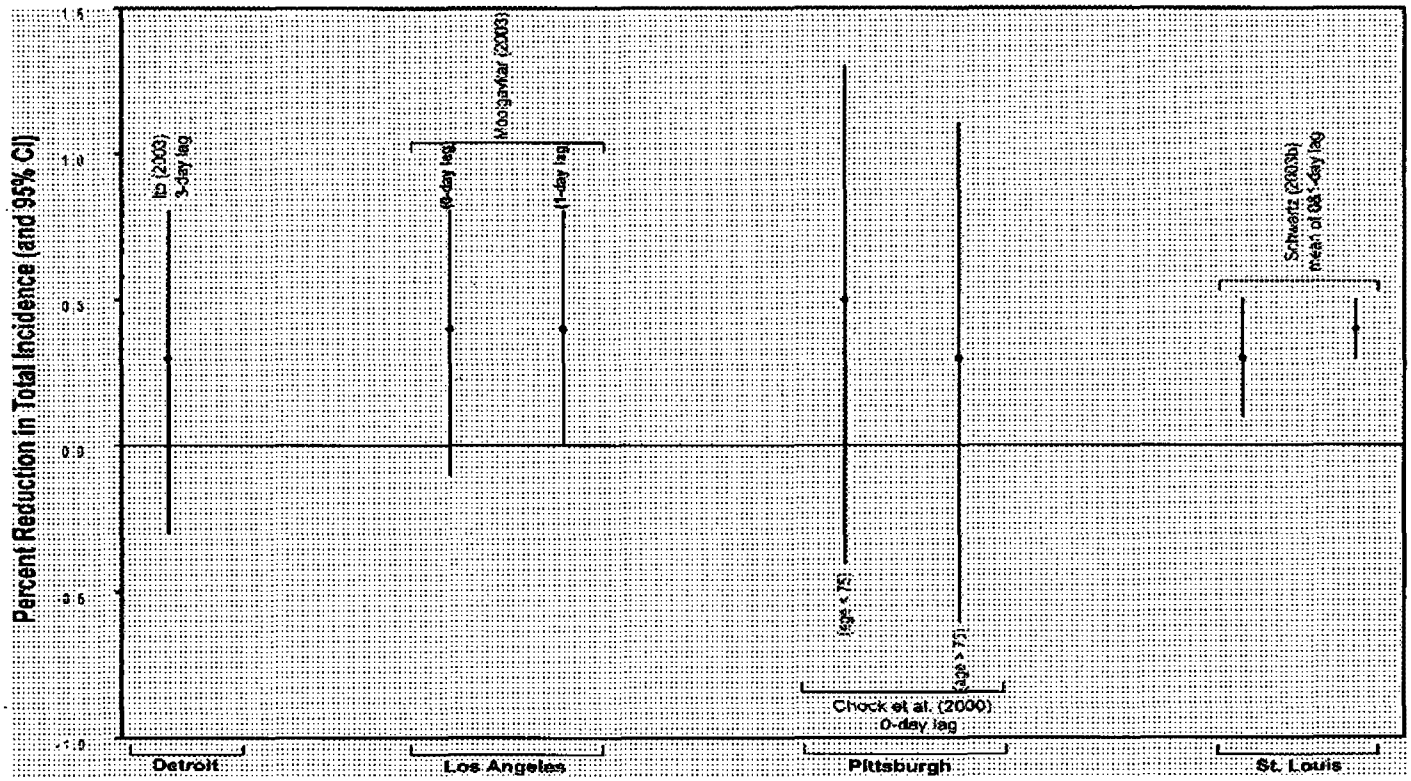

Figure 4-9. Estimated Annual Percentage Reduction of Health Risks Associated with Rolling Back $PM_{2.5}$ Concentrations to Just Meet the Current Standards (and 95 Percent Confidence Intervals): Non-Accidental Mortality Associated with Short-Term Exposure to $PM_{2.5}$.

Source: Abt Associates, 2005

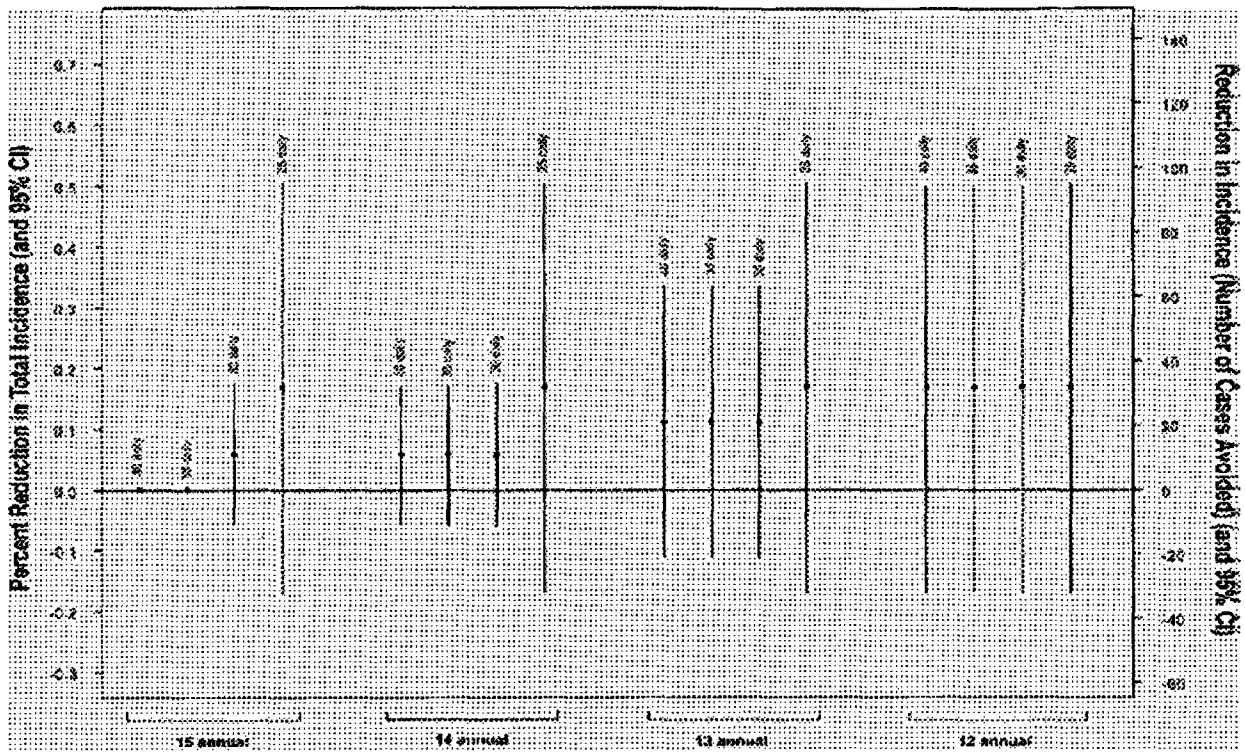

Figure 4-10. Estimated Annual Percentage Reduction of Health Risks Associated with Rolling Back $PM_{2.5}$ Concentrations to Just Meet the Current Annual Standards (and 95 Percent Confidence Interval): Mortality Associated with Long-Term Exposure to $PM_{2.5}$. Source: Abt Associates, 2005

1 **Table 4-11. 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	Incidence Remaining Upon Attaining Current $PM_{2.5}$ NAAQS	Percent of $PM_{2.5}$-Related Incidence Reduced	Reduction in Incidence Expressed as Percent of Total Incidence
non-accidental mortality, short-term exposure, 3-day lag, (Ito, 2003)**	Detroit	163 (-163 - 481)	115 (-116 - 338)	29.4 (29.2 - 29.7)	0.3 (-0.3 - 0.8)
total mortality (age ≥ 30)*** long-term exposure	Detroit	906 (33 - 1592)	522 (181 - 910)	42.4 (42.0 - 42.8)	2.0 (0.7 - 3.5)
non-accidental mortality, short-term exposure, 1-day lag, (Moolgavkar, 2003)**	Los Angeles	491 (1 - 971)	270 (1 - 533)	44.9 (44.8 - 45.1)	0.4 (0.0 - 0.8)
total mortality (age ≥ 30)*** long-term exposure	Los Angeles	3684 (1280 - 6426)	1507 (531 - 2587)	59.1 (58.6 - 59.8)	3.9 (1.3 - 6.8)
cardiovascular, short-term exposure, 1-day lag, Lipfert et al., 2000)**	Philadelphia	412 (197 - 628)	367 (175 - 560)	10.9 (10.9 - 10.9)	0.3 (0.1 - 0.4)
total mortality (age ≥ 30)*** long-term exposure	Philadelphia	650 (224 - 1146)	536 (185 - 943)	17.5 (17.3 - 17.7)	0.7 (0.2 - 1.2)
non-accidental mortality (age >75) (Chock et al., 2000)**	Pittsburgh	77 (-166 - 311)	50 (-108 - 200)	35.2 (34.7 - 35.6)	0.3 (-0.6 - 1.1)

Health Effect and Model**	Urban Study Area	"As Is" Incidence	Incidence Remaining Upon Attaining Current $PM_{2.5}$ NAAQS	Percent of $PM_{2.5}$-Related Incidence Reduced	Reduction in Incidence Expressed as Percent of Total Incidence
total mortality (age ≥ 30)* long-term exposure**	Pittsburgh	816 (282 - 1430)	403 (141 - 699)	50.6 (50.1 - 51.1)	2.7 (0.9 - 4.8)
non-accidental mortality, short-term exposure, mean of lag 0 & 1-day, all ages (Schwartz, 2003)**	St. Louis	233 (86 - 379)	191 (70 - 311)	18.0 (17.9 - 18.0)	0.2 (0.1 - 0.3)
total mortality (age ≥ 30)* long-term exposure**	St. Louis	842 (290 - 1486)	596 (206 - 1047)	29.2 (29.0 - 29.8)	1.1 (0.4 - 1.9)

*Risk reductions are relative to the "as is" (year 2003) air quality base case risk estimates. **These risk reductions are based on single pollutant model from the study cited and include all ages unless otherwise noted. ***These risk reductions are based on the Pope et al. (2000) ACS-extended study.

in total incidence, which represent from about 11 to 45% reductions in the PM-related incidence. Figure 4-10 shows the estimated percent reductions in total incidence for mortality associated with long-term exposure to $PM_{2.5}$ concentrations for this same air quality change in all five of the risk assessment study areas that do not meet the current standards. The point estimates are in the range 0.5 to nearly 4.0 percent reduction in total incidence, which represents from about 18 to 59% reductions in the PM-related incidence. Table 4-11 shows the estimated short- and long-exposure mortality incidence to facilitate a comparison both within and across the five study areas. For short-term exposure mortality, single-pollutant, non-accidental mortality estimates are selected since they are available for four of the study areas, and cardiovascular mortality is shown for the fifth area, Philadelphia. For long-term exposure mortality, the ACS-extended estimates for total (all cause) mortality are selected for comparison. In Table 4-11 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 reductions in both short- and long-term exposure mortality associated with $PM_{2.5}$ are ranked in the same order as the percent rollback required to bring as is concentrations down to just attaining the current standards, with Los Angeles having the biggest percentage reduction in risk and Philadelphia the least. Also, both the risk remaining upon just meeting the current $PM_{2.5}$ standards and the size of the reduction in risk in moving from as is concentrations to just meeting the current standards are larger associated with long-term exposure mortality estimates.

4.4.2 Sensitivity Analyses

The base case risk assessment used 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 TSD (Abt Associates, 2005). 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 was used which reduced 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 small difference (less than 1%) in the percent change in PM-associated incidence (see Exhibit 8.2 and Appendix E, exhibits E5-E8 in Abt Associates, 2005).

4.4.3 Key Observations

Sections 4.4.1 and 4.4.2 have presented the PM health risk estimates and sensitivity analyses associated with just meeting the current $PM_{2.5}$ standards. Presented 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 18% in St. Louis. Similarly, using the ACS-extended study the percent of $PM_{2.5}$-related incidence reduced for long-term exposure mortality ranges from roughly 60% in Los Angeles to about 18% in Philadelphia.

- 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

4.5.1 Base Case Risk Estimates for Alternative $PM_{2.5}$ Standards

The third part of the $PM_{2.5}$ risk assessment estimates the risk reductions associated with just meeting alternative suites of annual and daily $PM_{2.5}$ standards. For the five urban areas that exceeded the current $PM_{2.5}$ suite of standards (i.e., Detroit, Los Angeles, Philadelphia, Pittsburgh, and St. Louis), the estimated risk reductions were those associated with a further reduction in $PM_{2.5}$ concentrations from just meeting the current standards to just meeting various suites of alternative $PM_{2.5}$ standards. For the four urban areas that met the current $PM_{2.5}$ standards based on 2001-2003 levels (i.e., Boston, Phoenix, San Jose, and Seattle), the estimated risk reductions were those associated with a reduction from as is air quality levels to just meeting various suites of alternative $PM_{2.5}$ standards.

The selection of the suites of alternative annual and daily standards included in the risk assessment was based on the preliminary staff recommendations described in Chapter 6 of the draft 2003 Staff Paper (EPA, 2003) and consideration of public and CASAC comments. Annual standards of 15, 14, 13, and 12 $\mu g/m^3$ were each combined with 98th percentile daily standards of 40, 35, 30, and 25 $\mu g/m^3$, and 99th percentile daily standards at the same levels.[21] In addition, an annual standard of 15 $\mu g/m^3$ was combined with a ninety-ninth percentile daily standard of 65 $\mu g/m^3$. The combinations of annual and daily alternative standards used in the $PM_{2.5}$ risk assessment are summarized in Table 4-12. The same proportional adjustment approach used to simulate air quality just meeting the current standards, described previously in section 4.2.3.2 and in section 2.3 of Abt Associates (2005), was used to simulate air quality just meeting the various alternative suites of standards. Table 4-13 provides the design values for the annual and

[21]In four of the five urban areas that do not meet the current suite of $PM_{2.5}$ standards, annual standards within the range of 12 to 15 $\mu g/m^3$ combined with the current daily standard of 65 $\mu g/m^3$, using a 98th percentile form, require the same reduction as when these annual standards are combined with a daily standard of 40 $\mu g/m^3$, using the same daily form. Therefore, the risk assessment only included the 14 $\mu g/m^3$ annual standard combined with the current daily standard for the one location (i.e., Philadelphia) and annual standard scenario where there was a difference in the reduction required between daily standards of 40 and 65 $\mu g/m^3$.

98th and 99th percentile daily standards for all of the $PM_{2.5}$ risk assessment study areas based on air quality data from 2001-2003 for the base case risk estimates.

The estimated risk reduction in total non-accidental mortality, presented both in terms of percent reduction in total incidence and in number of cases avoided, associated with short-term $PM_{2.5}$ exposures for alternative annual standards combined with ninety-eighth and ninety-ninth percentile daily standards, respectively, are given in Figures 4-11 and 4-12 for Detroit. Similarly, the estimated risk reduction in total mortality associated with long-term $PM_{2.5}$ exposures for these same alternative standards are given in Figures 4-13 and 4-14 for Detroit. Similar figures for the other risk assessment locations and additional risk estimates for cause-specific mortality, hospital admissions, and respiratory symptoms (depending on location) associated with alternative standards are presented in Chapter 8 and Appendix F of Abt Associates (2005). As with the estimated risk reductions presented earlier for just meeting the current $PM_{2.5}$ standards, when the percent reduction is expressed in terms of the estimated reduction in PM-related incidence rather than total incidence, the changes are much larger. The complete set of risk estimates is presented in Exhibits 8.5a through 8.5h for Detroit and the exhibits in Appendix F for the other 4 locations in the TSD (Abt Associates, 2005).

Some interesting patterns can be observed in the estimated risk reductions displayed in Figures 4-11 through 4-14. For example, in Figures 4-11 and 4-13 one observes there are no estimated reductions in risk in going from just meeting the current 15 $\mu g/m^3$ annual standard/65 $\mu g/m^3$ 98th percentile daily standard to either a 40 or 35 $\mu g/m^3$ 98th percentile daily standard with the same 15 $\mu g/m^3$ annual standard. The reason for this is that the 28.1% reduction, required based on the 3-year estimated design value, when applied to the 2003 $PM_{2.5}$ distribution for the composite monitor to meet the current 15 $\mu g/m^3$ annual standard, brings down the 98th percentile daily value to below 35 $\mu g/m^3$. Thus, there is no additional reduction in air quality or risk when either a 40 or 35 $\mu g/m^3$ 98th percentile daily standard is considered in combination with a 15 $\mu g/m^3$ annual standard. Meeting lower daily 98th percentile standards of 30 or 25 $\mu g/m^3$ when combined with the current annual standard do require additional air quality reductions and, thus, result in additional estimated risk reductions compared to just meeting the current suite of

Table 4-12. Alternative Sets of $PM_{2.5}$ Standards Considered in the $PM_{2.5}$ Risk Assessment*

Annual Standard	98th Percentile Daily Standard					99th Percentile Daily Standard				
	65	40	35	30	25	65	40	35	30	25
15		x	x	x	x	x	x	x	x	x
14	x**	x	x	x	x		x	x	x	x
13		x	x	x	x		x	x	x	x
12		x	x	x	x		x	x	x	x

*All standards are in $\mu g/m^3$.

**Only in Philadelphia.

Table 4-13. Estimated Design Values for Annual and 98th and 99th Percentile Daily $PM_{2.5}$ Standards Based on 2001-2003 Air Quality Data*

Location	Annual	98th Percentile Daily	99th Percentile Daily
Boston	14.4	44	60
Detroit	19.5	44	48
Los Angeles	23.6	62	96
Philadelphia	16.4	51	89
Phoenix	11.5	35	41
Pittsburgh	21.2	63	70
St. Louis	17.5	42	46
San Jose	14.6	47	53
Seattle	11.1	41	48

*The calculation of design values is explained in Schmidt (2005). All design values are in $\mu g/m^3$.

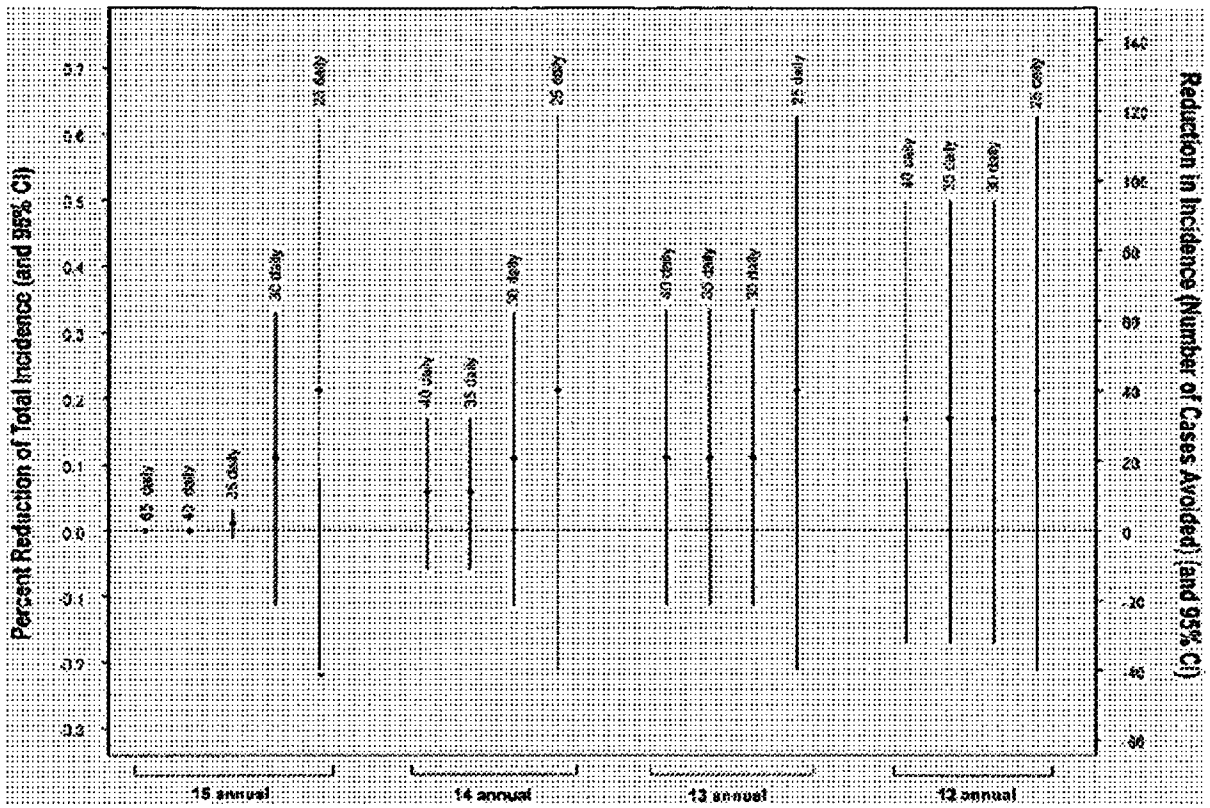

Figure 4-11. Estimated Annual Reduction in Short-Term Exposure Mortality Associated with Rolling Back $PM_{2.5}$ Concentrations that Just Meet the Current Annual Standard of 15 ug/m3 and the Current Daily Standard of 65 ug/m3 to $PM_{2.5}$ Concentrations that Just Meet Alternative Suites of $PM_{2.5}$ Annual and Daily 98th Percentile Standards: Detroit, MI, 2003.*

*Based on Ito (2003)

Source: Abt Associates (2005)

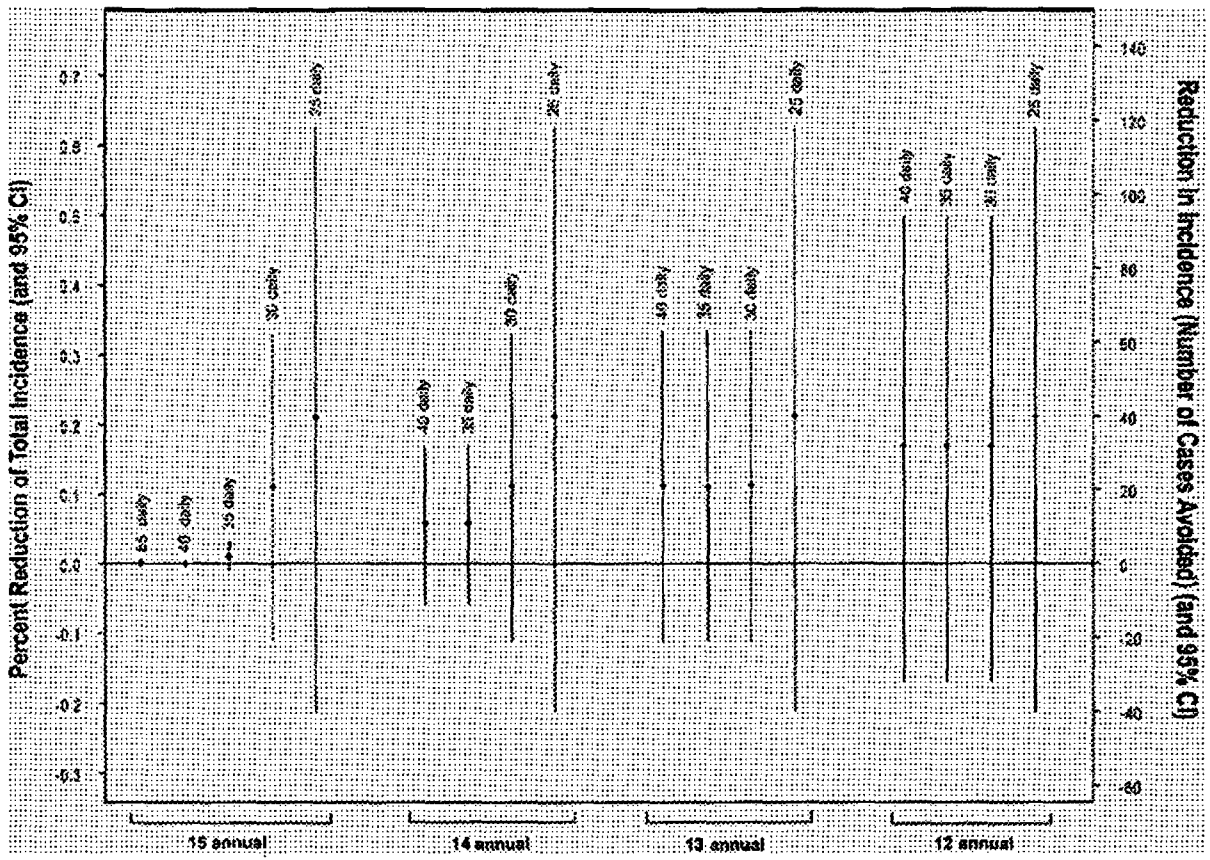

Figure 4-12. Estimated Annual Reduction in Short-Term Exposure Mortality Associated with Rolling Back $PM_{2.5}$ Concentrations that Just Meet the Current Annual Standard of 15 ug/m3 and the Current Daily Standard of 65 ug/m3 to $PM_{2.5}$ Concentrations that Just Meet Alternative Suites of $PM_{2.5}$ Annual and Daily 99th Percentile Standards: Detroit, MI, 2003.*

*Based on Ito (2003)

Source: Abt Associates (2005)

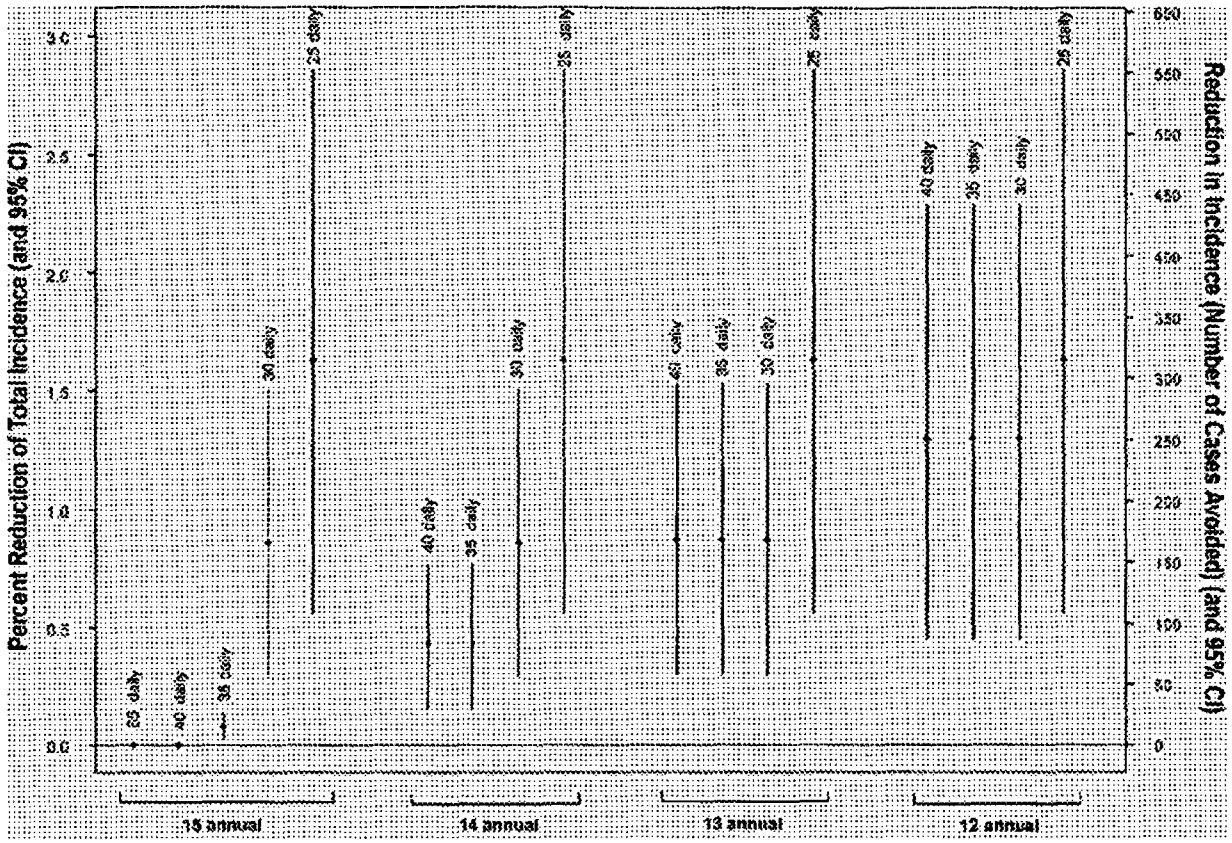

Figure 4-13. Estimated Annual Reduction in Long-Term Exposure Mortality Associated with Rolling Back $PM_{2.5}$ Concentrations that Just Meet the Current Annual Standard of 15 ug/m3 and the Current Daily Standard of 65 ug/m3 to $PM_{2.5}$ Concentrations that Just Meet Alternative Suites of $PM_{2.5}$ Annual and Daily 98th Percentile Standards: Detroit, MI, 2003.*

*Based on Pope et al. (2002) – ACS extended

Source: Abt Associates (2005)

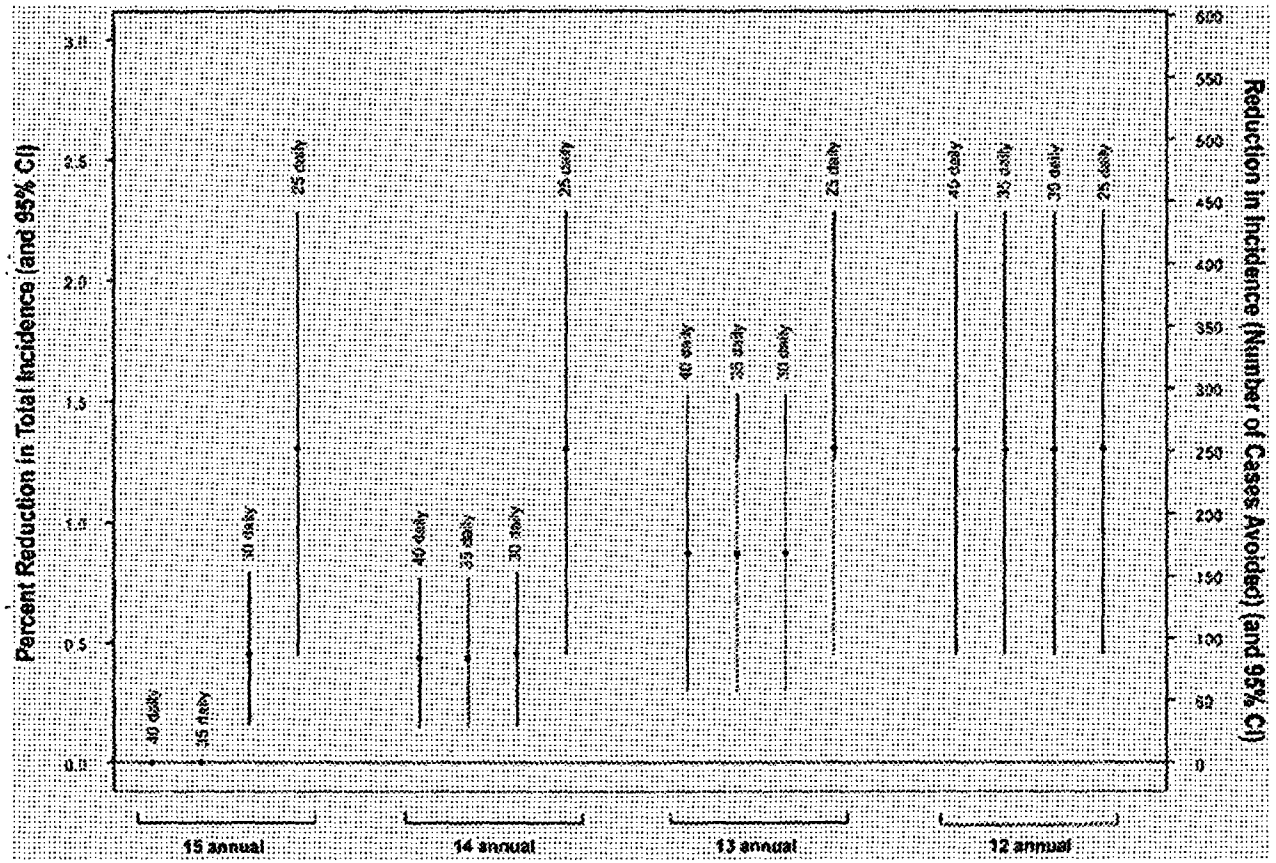

Figure 4-14. Estimated Annual Reduction in Long-Term Exposure Mortality Associated with Rolling Back $PM_{2.5}$ Concentrations that Just Meet the Current Annual Standard of 15 ug/m3 and the Current Daily Standard of 65 ug/m3 to $PM_{2.5}$ Concentrations that Just Meet Alternative Suites of $PM_{2.5}$ Annual and Daily 99th Percentile Standards: Detroit, MI, 2003.*

*Based on Pope et al. (2002) – ACS extended

Source: Abt Associates (2005)

standards. The maximum incremental risk reduction from the current standards, with respect to both short- and long-term exposure $PM_{2.5}$-associated mortality, is estimated to occur for meeting the daily 98th and 99th percentile daily standards set at 25 μg/m³. For daily standards set at this level the estimated risk reduction does not depend on the level of the annual standard within the range of standards considered. Within four of the five study areas, just meeting 98th or 99th percentile daily standards set at 30 μg/m³ results in the same short- or long-term exposure mortality risk reductions no matter which annual standards (from 12 to 15 μg/m³) they are paired with. Similar, although not identical, patterns are observed in the other four risk assessment locations that do not meet the current $PM_{2.5}$ standards (see Figures F1 through F14 in the TSD (Abt Associates (2005)).

4.5.2 Base Case Estimates for Alternative $PM_{10-2.5}$ Standards

The second part of the $PM_{10-2.5}$ risk assessment estimates the risk reductions associated with just meeting alternative daily $PM_{10-2.5}$ standards for the three locations examined earlier (Detroit, St. Louis, and Seattle). Estimated reductions in risk were developed for going from as is levels (based on 2003 air quality) to just meeting alternative $PM_{10-2.5}$ standards. Staff selected the alternative daily standards to be included in the risk assessment based on the preliminary staff recommendations described in Chapter 6 of the draft 2003 Staff Paper (EPA, 2003) and consideration of public and CASAC comments. Table 4-14 summarizes the sets of 98th and 99th percentile daily standards that were included in the $PM_{10-2.5}$ risk assessment. The estimated design values which were used to determine the air quality adjustment to be used in simulating just meeting alternative $PM_{10-2.5}$ standards are shown in Table 4-15.

The estimated annual reduction in hospital admissions for ischemic heart disease, presented both in terms of percent reduction in total incidence and in number of cases avoided, associated with short-term $PM_{10-2.5}$ exposures for alternative 98th and 99th percentile daily standards, respectively, are given in Figure 4-15 for Detroit. Daily $PM_{10-2.5}$ standards set at 80 (for 98th percentile form) and 100 or 80 (for 99th percentile form) result in no reduction in risk in Detroit. The reason why no estimated risk reductions are observed with these alternative standards is that the percent reduction of $PM_{10-2.5}$ concentrations at the composite monitor to just meet a standard is determined by comparing the alternative standard level with the design value

for that location based on 2001-2003 air quality data. In Detroit, the design value for the 98th percentile daily $PM_{10-2.5}$ standards is 70 μg/m³ whereas the 98th percentile daily value in 2003 is 105.9 μg/m³. Because the design value is lower than 80 μg/m³, the highest 98th percentile daily $PM_{10-2.5}$ standard, zero risk reductions were estimated to result from this standard, even though the 98th percentile daily value at the composite monitor in 2003, 105.9 μg/m³, is well above the standard level. Similarly, the design value for the 99th percentile daily $PM_{10-2.5}$ standards is 77 μg/m³ for Detroit, whereas the 99th percentile daily value at the composite monitor in Detroit in 2003 is substantially greater than 100 μg/m³, the highest 99th percentile daily $PM_{10-2.5}$ standard. So zero risk reductions were similarly estimated to result from both a 100 and 80 μg/m³ standards. In general, estimated risk reductions increase and the confidence intervals around the estimates widen as lower daily standards are considered.

As expected, the maximum reduction in risk is achieved with the 98th percentile 25 μg/m³ standard and 99th percentile 30 μg/m³ standard. The point estimate is that about a 4% reduction in hospital admissions for ischemic heart disease, equating to roughly 450 fewer cases, would result from meeting either of these daily standards. Similar patterns in risk reduction are observed for the other hospital admission endpoints in Detroit which are included in Chapter 9 of Abt Associates (2005). Additional risk estimates for hospital admissions for asthma in Seattle and cough and lower respiratory symptoms in St. Louis can be found in Appendix G of Abt Associates (2005). Based on the point estimates, there are no risk reductions associated with just meeting daily 98th percentile $PM_{10-2.5}$ standards of 80 μg/m³ in Detroit, and 80, 65, and 50 μg/m³ in St. Louis or Seattle. Similarly, there are no risk reductions associated with just meeting daily 99th percentile $PM_{10-2.5}$ standards of 100 or 80 μg/m³ in Detroit, and 100, 80, or 60 μg/m³ in St. Louis or Seattle.

4.5.3 Sensitivity Analyses for Alternative $PM_{2.5}$ and $PM_{10-2.5}$ Standards

4.5.3.1 Hypothetical Thresholds

An important observation from the sensitivity analyses on estimated health risks associated with "as is" $PM_{2.5}$ concentrations was that the impact of hypothetical thresholds was the greatest on the estimated risks. In order to gain insight into the impact of this important

Table 4-14. Alternative $PM_{10-2.5}$ Standards Considered in the $PM_{10-2.5}$ Risk Assessment*

Daily Standards Based on the 98th Percentile Value	Daily Standards Based on the 99th Percentile Value
80	100
65	80
50	60
30	35
25	30

*All standards are in $\mu g/m^3$.

Table 4-15. Estimated Design Values for 98th and 99th Percentile Daily $PM_{10-2.5}$ Standards Based on 2001-2003 Air Quality Data*

Location	98th Percentile Daily	99th Percentile Daily
Detroit	70	77
St. Louis	33	47
Seattle	31	39

*The calculation of design values is explained in Schmidt (2005). All design values are in $\mu g/m^3$.

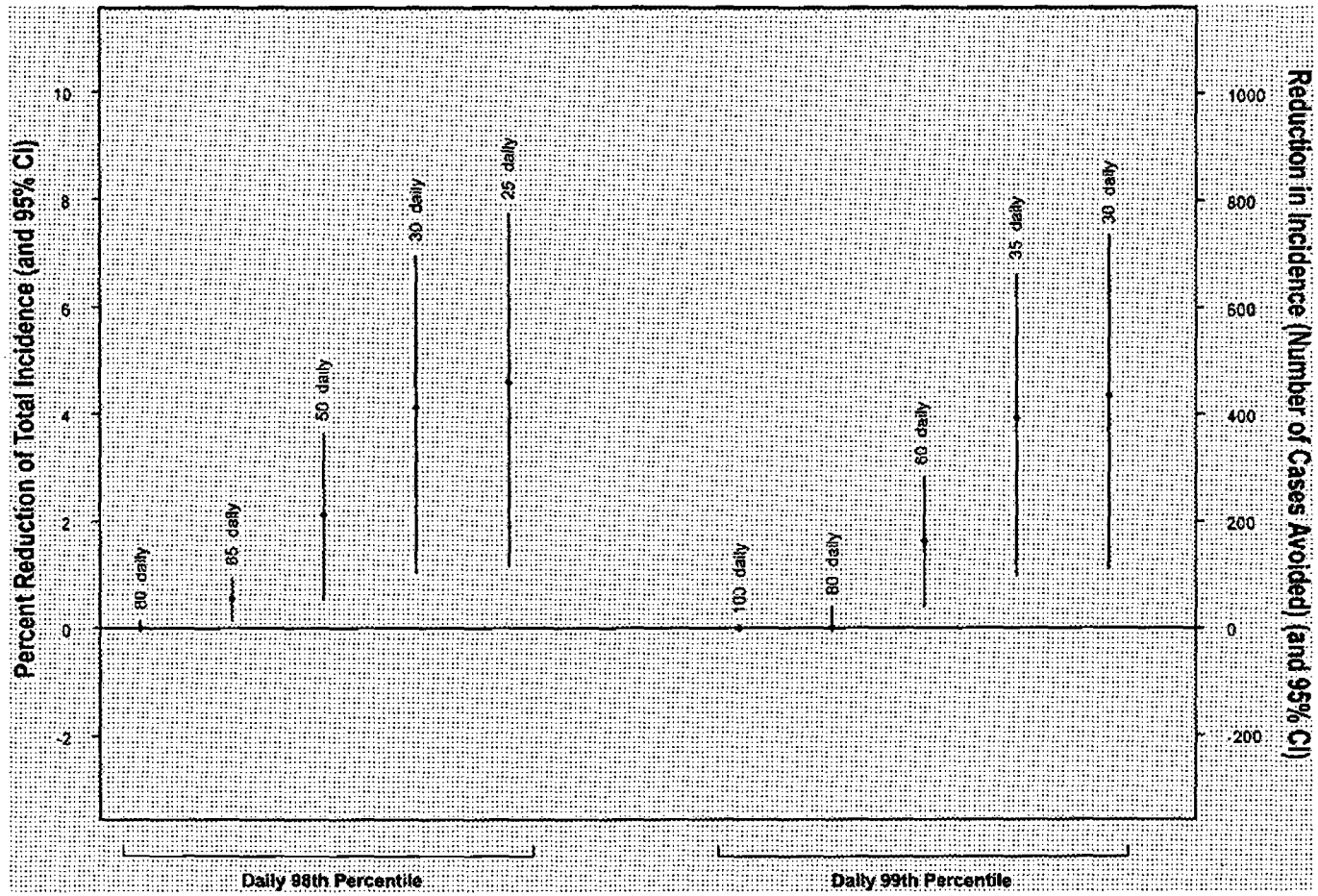

Figure 4-15. Estimated Annual Reduction of Hospital Admissions for Ischemic Heart Disease Associated with Rolling Back "As Is" $PM_{10-2.5}$ Concentrations to $PM_{10-2.5}$ Concentrations that Just Meet Alternative $PM_{10-2.5}$ Daily Standards: Detroit, MI, 2003.*

*Based on Ito (2003) Source: Abt Associates (2005)

uncertainty on the risk estimates, an additional set of sensitivity analyses was developed to examine the impact of different hypothetical threshold assumptions on estimated risks associated with just meeting the current and alternative $PM_{2.5}$ standards and alternative $PM_{10-2.5}$ standards. For those locations and cases where either the current $PM_{2.5}$ standards or any of the alternative suites of standards were already met under as is air quality, the estimated risks associated with "as is" $PM_{2.5}$ (or $PM_{10-2.5}$) concentrations in excess of either background or the LML for the health endpoint, whichever is greater, were calculated.

For $PM_{2.5}$ this sensitivity analysis included estimates of risk for all cause mortality, cardiopulmonary mortality, and lung cancer mortality associated with long-term exposure to $PM_{2.5}$ based on Pope et al. (2002) – ACS extended. Since the patterns observed were identical, only the all cause mortality results are presented in Appendix 4B (See Abt Associates, 2005 for the cause-specific mortality estimates). In addition, this sensitivity analysis also included non-accidental mortality (or cause-specific if there was no suitable function for non-accidental mortality available) associated with short-term exposure to $PM_{2.5}$. As in the earlier sensitivity analysis for as is air quality, hypothetical thresholds of 10, 15, and 20 $\mu g/m^3$ were considered for health endpoints associated with short-term exposures, and hypothetical thresholds of 10 and 12 $\mu g/m^3$ were considered for the mortality endpoints associated with long-term exposure.

The sensitivity analysis results for all-cause mortality associated with long-term exposure and mortality associated with short-term exposure for Detroit, Los Angeles, Philadelphia, Pittsburgh, and St. Louis are shown in Appendix 4B to this Chapter (Tables 4B-1 through 4B-10) The results for cardiopulmonary and lung cancer mortality associated with long-term exposure to $PM_{2.5}$ based on Pope et al. (2002) – ACS extended are shown in Appendix H of Abt Associates (2005). Not surprisingly, estimated PM-related incidences varied substantially with both hypothetical threshold assumptions and alternative standards. In Detroit, for example, the estimated number of cases of non-accidental mortality associated with short-term exposure to $PM_{2.5}$ when the current standards are just met decreases from 115, under the assumption of no threshold, to 54, 26, and 12 under hypothetical threshold assumptions of 10, 15, and 20 $\mu g/m^3$, respectively. Because meeting increasingly lower level standards removes estimated cases at the higher concentrations and considering higher hypothetical thresholds increasingly removes estimated cases at concentrations between background (or the LML) and the threshold, one would expect to see an increase in the percent reduction associated with just meeting alternative

standards for higher hypothetical thresholds. This is exactly what is found. For example, as seen in Table 4B-1, going from just meeting the current standards (15 μg/m³ annual and 65 μg/m³ daily 98th percentile value) to just meeting the lowest set of standards considered (12 μg/m³ annual and 25 μg/m³ daily 99th percentile value) results in a reduction in short-term exposure mortality incidence of (115 - 75)/115 = 34.8 percent under the assumption of no threshold, but under the assumption of a threshold of 10 μg/m³ it results in a reduction of (54 - 22)/54 = 59 percent. Under hypothetical short-term exposure thresholds of 15 and 20 μg/m³, the reductions are 73 percent and 83 percent, respectively. As shown in Table 4B-2 for all-cause mortality associated with long-term exposure in Detroit, the reduction in mortality incidence is even more dramatic when alternative hypothetical thresholds are considered. Going from just meeting the current standards to just meeting the lowest set of standards considered (12 μg/m³ annual and 25 μg/m³ daily 99th percentile value) results in a reduction in long-term exposure mortality incidence of (522-207)/522= 60% under the assumption of no threshold, but under the assumptions of a long-term exposure threshold of 10 μg/m³ it results in a reduction of (282 - 0)/282 =100 percent. With a hypothetical long-term exposure threshold of 12 μg/m³ estimated incidence is reduced to 41 upon just meeting the current suite of standards and a 100% reduction is achieved upon meeting either a 15 μg/m³ annual standard with a 30 μg/m³ daily 98th percentile standard or a 14 μg/m³ annual with a 40 μg/m³ daily 98th percentile value. The same general patterns can be seen in all locations and for all health endpoints considered.

The sensitivity analysis results examining alternative $PM_{10-2.5}$ standards with hypothetical thresholds associated with short-term exposure morbidity endpoints for Detroit, Seattle, and St. Louis also are shown in Appendix B to this Chapter (Tables 4B-11 through 4B-13). The health endpoints included hospital admissions for ischemic heart disease in Detroit; hospital admissions for asthma (age < 65) in Seattle; and days of cough among children in St. Louis, all associated with short-term exposures to $PM_{10-2.5}$ exposures. Hypothetical short-term exposure thresholds of 10, 15, and 20 μg/m³ were considered.

4.5.3.2 Spatial Averaging Versus Maximum Community Monitor

As discussed previously in section 4.2.3.2, under the current annual $PM_{2.5}$ standard urban areas may, under certain circumstances, use the average of the annual averages of several monitors within an urban area to determine compliance with the annual standard, commonly referred to as the "spatial averaging approach." Four of the five urban areas included in the $PM_{2.5}$

- In four of the five risk assessment locations that do not meet the current $PM_{2.5}$ standards, daily standards of 40 $\mu g/m^3$, 98th percentile or 65 $\mu g/m^3$, 99th percentile when combined with the current 15 $\mu g/m^3$ annual standard provide no additional risk reduction in terms of short-term exposure mortality.

- In all five of the risk assessment locations that do not meet the current $PM_{2.5}$ standards, the maximum risk reduction with respect to both short- and long-term $PM_{2.5}$-associated mortality is estimated to occur upon meeting the 98th and 99th percentile daily standards set at 25 $\mu g/m^3$. For these standards the estimated risk reduction does not depend on the level of the annual standard within the range of standards examined.

- For four of the five risk assessment locations the estimated risk reduction within each area associated with meeting either a 98th or 99th percentile daily $PM_{2.5}$ standard set at 30 $\mu g/m^3$ is the same no matter which annual standard is included within the range of standards examined.

- For the $PM_{10-2.5}$ risk estimates, the maximum reduction in risk is achieved with the 98th percentile 25 $\mu g/m^3$ standard or 99th percentile 30 $\mu g/m^3$ standard. The point estimate is that about a 4% reduction in hospital admissions for ischemic heart disease, equating to roughly 450 fewer cases, would result from meeting either of these daily standards. The confidence intervals get significantly larger as lower $PM_{10-2.5}$ standards are considered. Similar patterns in risk reduction are observed for the other hospital admission endpoints in Detroit.

- Based on the point estimates, there are no risk reductions associated with just meeting daily 98th percentile $PM_{10-2.5}$ standards of 80 $\mu g/m^3$ in Detroit, and 80, 65, and 50 $\mu g/m^3$ in St. Louis or Seattle. Similarly, there are no risk reductions associated with just meeting daily 99th percentile $PM_{10-2.5}$ standards of 100 or 80 $\mu g/m^3$ in Detroit, and 100, 80, or 60 $\mu g/m^3$ in St. Louis or Seattle.

Section 4.5.3 presented the results of the following two sets of sensitivity analyses: (1) considering the impact on risk estimates associated with just meeting the current and alternative $PM_{2.5}$ standards and alternative $PM_{10-2.5}$ standards when hypothetical threshold models are included and (2) considering the impact on risk estimates associated with just meeting the current and alternative $PM_{2.5}$ standards when the average of the annual averages of several monitors within an urban area are used to determine compliance with the annual standard, commonly referred to as the "spatial averaging approach." Presented below are key observations resulting from this part of the risk assessment:

- For short-term exposure mortality associated with $PM_{2.5}$, there is a significant decrease in the incidence avoided as one considers higher hypothetical thresholds. There also is a

significant increase observed in the percent reduction in PM-associated incidence upon just meeting alternative standards with higher hypothetical thresholds. The reduction in incidence and increase in percent reduction in PM-associated incidence is even more dramatic for long-term exposure mortality as higher alternative hypothetical thresholds are considered.

- For short-term exposure morbidity associated with $PM_{10-2.5}$, there is a significant decrease in the incidence avoided as one considers higher hypothetical thresholds.

- There is an increase in estimated short-term exposure mortality incidence associated with $PM_{2.5}$ when a spatial averaging approach is used to determine compliance with the current annual standard or alternative suites of standards where the daily standard is not the controlling standard.

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5. STAFF CONCLUSIONS AND RECOMMENDATIONS ON PRIMARY PM NAAQS

5.1 INTRODUCTION

This chapter presents staff conclusions and recommendations for the Administrator to consider in deciding whether the existing primary PM standards should be revised and, if so, what revised standards are appropriate.[1] The existing suite of primary PM standards includes annual and 24-hour $PM_{2.5}$ standards, to protect public health from exposure to fine particles, and annual and 24-hour PM_{10} standards, to protect public health from exposure to thoracic coarse particles. Each of these standards is defined in terms of four basic elements: indicator, averaging time, level and form. Staff conclusions and recommendations on these standards are based on the assessment and integrative synthesis of information presented in the CD and on staff analyses and evaluations presented in Chapters 2 through 4 herein.

In recommending a range of primary standard options for the Administrator to consider, 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, discussed more fully below, is based on a recognition that the available health effects evidence generally reflects a continuum consisting of ambient levels at which scientists generally agree that health effects are likely to occur 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 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 to public health.

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[1] As noted in Chapter 1, staff conclusions and recommendations presented herein are provisional; final staff conclusions and recommendations, to be included in the final version of this document, will be informed by comments received from CASAC and the public in their reviews of this draft document.

5.2 APPROACH

As an initial matter, $PM_{2.5}$ standards for fine particles and PM_{10} standards for thoracic coarse particles are addressed separately, consistent with the decision made by EPA in the last review and with the conclusion in the CD that fine and thoracic coarse particles should continue to be considered as separate subclasses of PM pollution. As discussed in Chapter 3, section 3.2.3, this conclusion is based in part on long-established information on the differences in sources, properties, and atmospheric behavior between fine and coarse particles, and is reinforced by new information that advances our understanding of differences in human exposure relationships and dosimetric patterns, and the apparent independence of health effects that have been associated with these two subclasses of PM pollution in epidemiologic studies.

In general, in evaluating whether the current primary standards are adequate or whether revisions are appropriate, and in developing recommendations on the elements of possible alternative standards for consideration, staff's approach in this review builds upon and broadens the general approach used by EPA in the last review. In setting $PM_{2.5}$ standards in 1997, the Agency mainly used an evidence-based approach that placed primary emphasis on epidemiologic evidence from short-term exposure studies of fine particles, judged to be the strongest evidence at that time, in reaching decisions to set a generally controlling annual $PM_{2.5}$ standard and a 24-hour $PM_{2.5}$ standard to provide supplemental protection. The risk assessment conducted in the last review provided qualitative insights, but was judged to be too limited to serve as a quantitative basis for decisions on the standards. In this review, the more extensive and stronger body of evidence now available on health effects related to both short- and long-term exposure to $PM_{2.5}$, together with the availability of much more extensive $PM_{2.5}$ air quality data, have facilitated a more comprehensive risk assessment for $PM_{2.5}$. As a result, staff has used a broader approach in this review of the $PM_{2.5}$ standards that takes into account both evidence-based and quantitative risk-based considerations, placing greater emphasis on evidence from long-term exposure studies and quantitative risk assessment results for fine particles than was done in the last review. Staff has applied this approach to a more limited degree in reviewing the PM_{10} standards, reflecting the far more limited nature of the health effects evidence and air quality data available for thoracic coarse particles.

Staff has taken into account evidence-based considerations primarily by assessing the epidemiologic evidence of associations with health endpoints that the CD has judged to be likely causal based on an integrative synthesis of the entire body of evidence. Less weight is given to evidence of associations that are judged to be only suggestive of possible causal relationships, taking this information into account as part of margin of safety considerations. In so doing, staff has placed greater weight on U.S. and Canadian studies reporting statistically significant associations, providing relatively more precise effects estimates, using relatively more reliable air quality data, and reporting associations that are generally robust to alternative model specifications and the inclusion of potentially confounding co-pollutants. By considering the ambient particle levels present during specific studies, staff has reached conclusions as to the degree to which alternative standards could be expected to protect against the observed health effects, while being mindful of the inherent limitations and uncertainties in such evidence.

Staff has also taken into account quantitative risk-based considerations, drawn from the results of the risk assessment conducted in several example urban areas (discussed in Chapter 4). More specifically, staff has considered estimates of the magnitude of PM-related risks associated with current air quality levels, as well as the risk reductions likely to be associated with attaining the current or alternative standards. In so doing, staff recognizes the considerable uncertainties inherent in such risk estimates, and has taken such uncertainties into account by considering the sensitivity of the risk estimates to alternative assumptions likely to have substantial impact on the estimates.

More specifically, in this review a series of questions frames staff's approach to reaching conclusions and recommendations, based on the available evidence and information, as to whether consideration should be given to retaining or revising the current primary PM standards. Staff's review of the adequacy of the current standards begins by considering whether the currently available body of evidence assessed in the CD suggests that revision of any of the basic elements of the standards would be appropriate. This evaluation of the adequacy of the current standards involves addressing questions such as the following:

- To what extent does newly available information reinforce or call into question evidence of associations with effects identified in the last review?

- To what extent does newly available information reinforce or call into question any of the basic elements of the current standards?

- To what extent have important uncertainties identified in the last review been reduced and have new uncertainties emerged?

To the extent that the evidence suggests that revision of the current standards would be appropriate, staff then considers whether the currently available body of evidence supports consideration of standards that are either more or less protective by addressing the following questions:

- Is there evidence that associations, especially likely causal associations, extend to air quality levels that are as low as or lower than had previously been observed, and what are the important uncertainties associated with that evidence?

- Are health risks estimated to occur in areas that meet the current standards; are they important from a public health perspective; and what are the important uncertainties associated with the estimated risks?

To the extent that there is support for consideration of revised standards, staff then identifies ranges of standards (in terms of indicators, averaging times, levels and forms) that would reflect a range of alternative public health policy judgments, based on the currently available evidence, as to the degree of protection that is requisite to protect public health with an adequate margin of safety. In so doing, staff addresses the following questions:

- Does the evidence provide support for considering different PM indicators?

- Does the evidence provide support for considering different averaging times?

- What range of levels and forms of alternative standards is supported by the evidence, and what are the uncertainties and limitations in that evidence?

- To what extent do specific levels and forms of alternative standards reduce the estimated risks attributable to PM, and what are the uncertainties in the estimated risk reductions?

Based on the evidence, estimated risk reductions, and related uncertainties, staff makes recommendations as to ranges of alternative standards for the Administrator's consideration in reaching decisions as to whether to retain or revise the primary PM NAAQS.

Standards for fine particles are addressed in section 5.3 below, beginning with staff's consideration of the adequacy of the current primary $PM_{2.5}$ standards. Subsequent subsections address each of the major elements that define specific PM standards: pollutant indicator, averaging time, level and form. Staff has evaluated separately the protection that a suite of $PM_{2.5}$ standards would likely provide against effects associated with long-term exposures (section 5.3.4) and those associated with short-term exposures (section 5.3.5). These separate evaluations provide the basis for integrated recommendations on alternative suites of standards that protect against effects associated with both long- and short-term exposures, based on considering how a suite of standards operate together to protect public health. In a similar manner, standards for thoracic coarse particles are addressed in section 5.4 below. This chapter concludes with a summary of key uncertainties associated with establishing primary PM standards and related staff research recommendations in section 5.5.

5.3 FINE PARTICLE STANDARDS

5.3.1 Adequacy of Current $PM_{2.5}$ Standards

In considering the adequacy of the current $PM_{2.5}$ standards, staff has first considered the extent to which newly available information reinforces or calls into question evidence of associations with effects identified in the last review, as well as considering the extent to which important uncertainties have been reduced or have resurfaced as being more important than previously understood. In looking across the extensive epidemiologic evidence available in this review, the CD addresses these questions by concluding that "the available findings demonstrate well that human health outcomes are associated with ambient PM" (CD, p. 9-24) and, more specifically, that there is now "strong epidemiological evidence" for $PM_{2.5}$ linking short-term exposures with cardiovascular and respiratory mortality and morbidity, and long-term exposures with cardiovascular and lung cancer mortality and respiratory morbidity (CD, p. 9-46). This latter conclusion reflects greater strength in the epidemiologic evidence specifically linking $PM_{2.5}$ and various health endpoints than was observed in the last review, when the CD concluded that the epidemiologic evidence for PM-related effects was "fairly strong," noting that the

studies "nonetheless provide ample reason to be concerned" about health effects attributable to PM at levels below the then-current PM NAAQS (EPA, 1996, p. 13-92).

As discussed in Chapter 3 (section 3.5) and the CD (section 9.2.2), the CD concludes that the extensive body of epidemiologic evidence now available continues to support likely causal associations between $PM_{2.5}$ and the above health outcomes based on an assessment of strength, robustness, and consistency in results. The CD finds "substantial strength" in the evidence of $PM_{2.5}$ associations, especially for total and cardiovascular mortality (CD, p. 9-28). The CD recognizes that while the relative risk estimates are generally small in magnitude, a number of new studies provide more precise estimates that are generally positive and often statistically significant. Overall, the CD finds the new evidence substantiates that the associations are generally robust to confounding by co-pollutants, noting that much progress has been made in sorting out contributions to observed health effects of PM and its components relative to other co-pollutants. On the other hand, the CD notes that effect estimates are generally more sensitive than previously recognized to different modeling strategies to adjust for temporal trends and weather variables. While some studies showed little sensitivity, different modeling strategies altered conclusions in other studies.

Although greater variability in effects estimates across study locations is seen in the much larger set of studies now available, in particular in the new multi-city studies, the CD finds much consistency in the epidemiologic evidence particularly in studies with the most precision. There also are persuasive reasons why variation in associations in different locations could be expected. Further, the CD concludes that new source apportionment studies and "found experiments," showing improvements in community health resulting from reductions in PM and other air pollutants, lend additional support to the results of other studies that focused specifically on $PM_{2.5}$.

Looking more broadly to integrate epidemiologic evidence with that from exposure-related, dosimetric and toxicologic studies, the CD (section 9.2.3) considered the coherence of the evidence and the extent to which the new evidence provides insights into mechanisms by which PM, especially fine particles, may be affecting human health. Progress made in gaining insights into mechanisms lends support to the biological plausibility of results observed in

epidemiologic studies. For cardiovascular effects, the CD finds that the convergence of important new epidemiologic and toxicologic evidence builds support for the plausibility of associations especially between fine particles and physiological endpoints indicative of increased risk of cardiovascular disease and changes in cardiac rhythm. This finding is supported by new cardiovascular effects research focused on fine particles that has notably advanced our understanding of potential mechanisms by which PM exposure, especially in susceptible individuals, could result in changes in cardiac function or blood characteristics that are risk factors for cardiovascular disease. For respiratory effects, the CD finds that toxicologic studies have provided evidence that supports plausible biological pathways for fine particles, including inflammatory responses, increased airway responsiveness, or altered responses to infectious agents. Further, the CD finds coherence across a broad range of cardiovascular and respiratory health outcomes from epidemiologic and toxicologic studies done in the same location, particularly noting, for example, the series of studies conducted in or evaluating ambient PM from Boston and the Utah Valley. The CD also finds that toxicologic evidence examining combustion-related particles supports the plausibility of the observed relationship between fine particles and lung cancer mortality. With regard to PM-related infant mortality and developmental effects, the CD finds this to be an emerging area of concern, but notes that current information is still very limited in support of the plausibility of potential ambient PM relationships.

Based on the above considerations and findings from the CD, staff concludes that the newly available information generally reinforces the associations between $PM_{2.5}$ and mortality and morbidity effects observed in the last review. Staff recognizes that important uncertainties and research questions remain, notably including questions regarding modeling strategies to adjust for temporal trends and weather variables in time-series epidemiologic studies. Nonetheless, staff notes that progress has been made in reducing some key uncertainties since the last review, including important progress in advancing our understanding of potential mechanisms by which ambient $PM_{2.5}$, alone and in combination with other pollutants, is causally linked with cardiovascular, respiratory, and lung cancer associations observed in epidemiologic

studies. Thus, staff finds clear support in the available evidence, as assessed in the CD, for fine particle standards that are at least as protective as the current $PM_{2.5}$ standards.

Having reached this initial conclusion, staff also has addressed the question of whether the available evidence supports consideration of standards that are more protective than the current $PM_{2.5}$ standards. In so doing, staff has considered first whether there is evidence that health effects associations with short- and long-term exposures to fine particles extend to lower air quality levels than had previously been observed, or to levels below the current standards. In addressing this question, staff first notes that the available evidence does not either support or refute the existence of thresholds for the effects of PM on mortality across the range of concentrations in the studies, as discussed in Chapter 3 (section 3.4.6) and the CD (section 9.2.2.5). More specifically, while there are likely threshold levels for individuals and specific health responses, existing studies show little evidence for thresholds for PM-mortality relationships in populations, for either long-term or short-term PM exposures (CD, p. 9-44). Further, the CD notes that in the multi-city and most single-city studies, statistical tests comparing linear and various nonlinear or threshold models have not shown statistically significant distinctions between them (CD, p. 9-44). Even in those few studies with suggestive evidence for thresholds, the potential thresholds are at fairly low concentrations (CD, p. 9-45). While acknowledging that for some health endpoints, such as total nonaccidental mortality, it is likely to be extremely difficult to detect thresholds, the CD concludes that "epidemiologic studies suggest no evidence for clear thresholds in PM-mortality relationships within the range of ambient PM concentrations observed in these studies." (CD, p. 9-48).

In considering the available epidemiologic evidence (summarized in Chapter 3, section 3.3 and Appendices 3A and 3B), staff has focused on specific epidemiologic studies that show statistically significant associations between $PM_{2.5}$ and health effects for which the CD judges associations with $PM_{2.5}$ to be likely causal. Many more U.S. and Canadian studies are 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 (15 $\mu g/m^3$), which was set to provide protection against health effects related to both short- and long-term exposures to fine particles. Notably, a few of the newly available short-term exposure

mortality studies provide evidence of statistically significant associations with $PM_{2.5}$ in areas with long-term average air quality below that ambient level (summarized in Appendix 3A). In considering this group of studies, staff has focused on those studies that include adequate gravimetric $PM_{2.5}$ mass measurements, and where the associations are generally robust to alternative model specification and to the inclusion of potentially confounding co-pollutants. Three such studies conducted in Phoenix (Mar et al., 1999, 2003), Santa Clara County, CA (Fairley, 1999, 2003) and eight Canadian cities (Burnett et al., 2000 and Burnett and Goldberg, 2003) report statistically significant 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 $\mu g/m^3$. These studies were reanalyzed to address questions about the use of GAM with default convergence criteria, 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, other studies provide evidence of statistically significant associations with morbidity. Three studies of emergency department visits were conducted in areas where the mean $PM_{2.5}$ concentrations were approximately 12 $\mu g/m^3$ or below, although these studies either had not been reanalyzed to address the default convergence criteria problem with GAM, did not assess the potential for confounding by co-pollutants or were not robust to the inclusion of co-pollutants, or were done only during a single season. Another new study reported statistically significant associations with incidence of myocardial infarction where the mean $PM_{2.5}$ concentration was just above 12 $\mu g/m^3$; however, the CD urges caution in interpreting the results of the new body of evidence related to such cardiovascular effects (CD, p. 8-166). Thus, these studies provide no clear evidence of statistically significant associations with $PM_{2.5}$ at such low concentrations.

New evidence is also available from U.S. and Canadian studies of long-term exposure to fine particles (summarized in Appendix 3B). In evaluating this evidence (CD, section 9.2.3), the CD notes that new studies have built upon studies available in the last review and these studies have confirmed and strengthened the evidence of associations for both mortality and respiratory

morbidity. For mortality, the CD places greatest weight on the reanalyses and extensions of the Six Cities and the ACS studies, finding that these studies provide "strong evidence" for associations with fine particles (CD, p. 9-34), notwithstanding the lack of consistent results in other long-term exposure studies. For morbidity, the CD finds that new studies of a cohort of children in Southern California have built upon earlier limited evidence to provide "fairly strong" evidence that long-term exposure to fine particles is associated with development of chronic respiratory disease and reduced lung function growth (CD, p. 9-34).

As discussed in the CD and in Chapter 3 above, mortality studies of the Six Cities and ACS cohorts available in the last review had aggregate long-term 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. The extended analyses using the ACS cohort also continued to report statistically significant associations with $PM_{2.5}$ with the inclusion of more recent $PM_{2.5}$ air quality data, with an average range across the old and new time periods from about 7.5 to 30 $\mu g/m^3$ (from figure 1, Pope et al., 2002) with a long-term mean of approximately 17.7 $\mu g/m^3$ (Pope et al., 2002). As with the earlier cohort studies, no evidence of a threshold was observed in the relationships with total, cardiovascular, and lung cancer mortality reported in this extended study. In the morbidity studies of the Southern California children's cohort, the means of 2-week average $PM_{2.5}$ concentrations 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). Staff notes that in figures depicting relationships between lung function growth and average PM concentration, there is no evidence of a threshold in this study (Gauderman et al., 2000, 2002).

Beyond the epidemiologic studies using $PM_{2.5}$ as an indicator of fine particles, a large body of newly available evidence from studies that used PM_{10}, as well as other indicators or components of fine particles (e.g., sulfates, combustion-related components), 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 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 particles). Further, the CD concludes that new epidemiologic studies of ambient PM associations with potential PM-related infant mortality and/or developmental effects are very limited, although if further substantiated by future research, would significantly increase estimates of the extent of life shortening due to PM-related premature mortality (CD, p. 9-94). The CD also notes that new epidemiologic studies of asthma-related increased physicians visits and symptoms, as well as new studies of cardiac-related risk factors, suggest likely much larger public health impacts due to ambient fine particles than just those indexed by the mortality/morbidity effects considered in the last review (CD, p. 9-94).

Staff recognizes, however, that important limitations and uncertainties associated with this expanded body of evidence for $PM_{2.5}$ and other indicators or components of fine particles, as discussed in Chapter 3 herein and section 9.2.2 of the CD, need to be carefully considered in determining the weight to be placed on the studies available in this review. For example, the CD notes that 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 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 associations for various PM indicators with mortality and morbidity are robust to confounding by co-pollutants (CD, p. 9-37).

Another issue of particular importance 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. As discussed in section 3.5.3 herein and in section 9.2.2 of the CD, 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. These reanalyses suggest that weather continues to be a potential confounder of concern and highlight that no one model is likely to be most appropriate

in all cases. The HEI Review Panel, in reviewing these reanalyses, concluded that this awareness introduces a degree of uncertainty in evaluating the findings from time-series epidemiological studies that had heretofore not been widely appreciated.

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 CD 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.

While the limitations and uncertainties in the available evidence suggest caution in interpreting the epidemiologic studies at the lower levels of air quality observed in the studies, staff concludes that the evidence now available provides strong support for considering fine particle standards that would provide increased protection from that afforded by the current $PM_{2.5}$ standards. More protective standards would reflect the generally stronger and broader body of evidence of associations with mortality and morbidity now available in this review, at lower levels of air quality and at levels below the current standards, and with more understanding of possible underlying mechanisms.

In addition to this evidence-based evaluation, staff has also considered the extent to which health risks estimated to occur upon attainment of the current $PM_{2.5}$ standards may be judged to be important from a public health perspective, taking into account key uncertainties associated with the estimated risks. Based on the risk assessment presented in Chapter 4, staff considered as a base case the estimated risks attributable to $PM_{2.5}$ concentrations above background levels, or above the lowest measured levels in a given study if that was higher than background, so as to avoid extrapolating risk estimates beyond the range of air quality upon which the concentration-response functions were based. In the case of estimated risk associated with long-term exposure, based on the extended ACS study, risk was estimated down to an annual level of 7.5 $\mu g/m^3$, the lowest measured level in that study; for estimated risk associated

with short-term exposure, risk was estimated down to daily levels ranging from 2.5 to 4 $\mu g/m^3$, based on estimated background or the lowest measured level in a particular study.

In the absence of evidence for clear thresholds in any of the studies used in this risk assessment, the base case estimates in this analysis reflect the linear or near-linear concentration-response functions reported in the studies. To reflect the uncertainty as to whether thresholds may exist within the range of air quality observed in the studies, but may not be discernable with currently applied statistical methods, staff also has considered estimates of risk based on concentration-response functions modified to incorporate various assumed hypothetical threshold levels, as discussed in Chapter 4. Based on the sensitivity analyses conducted as part of the risk assessment, the uncertainty associated with alternative hypothetical thresholds had by far the greatest impact on estimated risks. Other uncertainties have a more moderate and often variable impact on the risk estimates in some or all of the cities, including the use of single- versus multi-pollutant models, single- versus multi-city models, use of a distributed lag model, alternative assumptions about the relevant air quality for long-term exposure mortality, and alternative constant or varying background levels.

Table 5-1 summarizes the estimated $PM_{2.5}$-related annual incidence of total mortality associated with long- and short-term exposure for the base case and for alternative hypothetical thresholds in the nine example urban areas included in the risk assessment. In looking particularly at the annual incidence of $PM_{2.5}$-related mortality estimated to occur upon attainment of the current $PM_{2.5}$ standards in the five study areas that do not meet the current standards based on 2001-2003 air quality data, staff notes that there is a fairly wide range of estimated incidence across the areas. Such variation would be expected considering, for example, differences in total population, demographics, exposure considerations (e.g., degree of air conditioning use), presence of co-pollutants and other environmental stressors, and exposure measurement error across urban areas; as well as differences in concentration-response relationships across studies that might be due in part to variation in these factors across locations. Staff also recognizes that there are uncertainties associated with the procedure used to simulate air quality that would just attain the current standards and in the degree to which various components of the fine particle mix would likely be reduced in similar proportion to the simulated reduction in $PM_{2.5}$ as a whole.

Table 5-1 Estimated $PM_{2.5}$-related Annual Incidence of Total Mortality when Current $PM_{2.5}$ Standards are Met (Base Case and Assumed Alternative Hypothetical Thresholds)*

	Short-term Exposure: Annual Non-Accidental Mortality (except as noted)				Long-term Exposure: Annual All-Cause Mortality		
	Base case Estimate, 95% CI	Assumed Hypothetical Short-term Exposure Thresholds			Base case Estimate, 95% CI	Assumed Hypothetical Long-term Exposure Thresholds	
		10 μg/m³	15 μg/m³	20 μg/m³		10 μg/m³	12 μg/m³
Risks associated with just meeting current $PM_{2.5}$ standards							
Detroit	115 -116 to 338	54 -55 to 159	26 -27 to 77	12 -12 to 35	522 181 to 910	282 98 to 494	41 14 to 72
Los Angeles	248 -31 to 519	115 -14 to 240	58 -7 to 121	29 -4 to 61	1,507 531 to 2,587	823 290 to 1415	138 48 to 237
Philadelphia (short-term: cardiovascular mortality)	367 175 to 560	189 90 to 288	106 51 to 162	57 27 to 87	536 185 to 943	338 116 to 597	137 47 to 244
Pittsburgh (short-term: over age 74)	50 -108 to 200	22 -48 to 87	10 -23 to 41	5 -11 to 18	403 141 to 699	215 75 to 373	25 9 to 43
St. Louis	191 70 to 311	75 28 to 122	29 11 to 46	9 3 to 14	596 206 to 1,047	311 107 to 548	23 8 to 40
Risks associated with "as is" air quality (in areas that meet current $PM_{2.5}$ standards)							
Boston	390 265 to 514	173 118 to 228	82 56 to 109	41 28 to 53	594 204 to 1053	309 106 to 551	20 7 to 36
Phoenix (short-term: cardiovascular mortality over age 64)	323 97 to 536	115 35 to 190	67 21 to 109	43 13 to 69	349 119 to 620	76 26 to 136	0 0 to 0
San Jose	218 45 to 387	80 17 to 141	44 9 to 77	28 6 to 50	172 59 to 306	58 20 to 104	0 0 to 0
Seattle**	--				50 17 to 89	0 0 to 0	0 0 to 0

* These estimates of annual incidence of $PM_{2.5}$-related mortality are based on using the maximum monitor in an area to calculate the percent rollback needed to just attain the current $PM_{2.5}$ annual standard, and applying that percent rollback to the composite monitor in the area, as described in Chapter 4, section 4.2.3. Estimates of annual mortality incidence based on using a spatially averaged concentration to calculate the percent rollback needed to just attain the current standard, where this is allowed, would be higher than the estimates shown here.

** No short-term exposure concentration-response function is available for mortality in Seattle.

Staff observes that base case point estimates of annual incidence of total $PM_{2.5}$-related mortality associated with just meeting the current $PM_{2.5}$ standards in the five areas shown range

from approximately 400 to 600 in four areas (or from roughly 25 to 35 deaths per 100,000 population in these areas) to over 1500 annual deaths in Los Angeles (i.e., roughly 16 deaths per 100,000 population) associated with long-term exposure. These estimated incidences associated with long-term exposure represent 2.6 to 3.2 percent of total mortality incidence due to all causes. Expressing the risk estimates in terms of percentage of total incidence takes into account city-to-city differences in population size and baseline mortality incidence rate. In some areas, the 95% confidence ranges associated with the estimates of total annual mortality incidence related to short-term exposure (but not long-term exposure) extend to below zero, reflecting appreciably more uncertainty in estimates based on positive but not statistically significant associations. In the other four areas that meet the current standards based on recent air quality data, base case point estimates of annual incidence of total $PM_{2.5}$-related mortality associated with long-term exposure range from a lower end of about 50 deaths in Seattle (which represents a rate of about 3 per 100,000 population) to an upper end of almost 600 deaths in Boston (a rate of 21 per 100,000 population). It is much more difficult to make comparisons among the urban areas with regard to short-term exposure mortality incidence or incidence rates because of the different population groups and mortality types examined in the epidemiology studies for the different locations. There also is greater variability in the estimates for mortality associated with short-term exposure due to the use of different city-specific concentration-response relationships.

In looking beyond the base case estimates, staff also considered the extent to which the assumption of the presence of hypothetical thresholds in the concentration-response relationships would influence the risk estimates. As expected, risk estimates are substantially smaller when hypothetical threshold concentration-response functions are considered. Point estimates of annual incidence of total $PM_{2.5}$-related mortality associated with long-term exposure are roughly 50% of base case estimates when a hypothetical threshold of 10 $\mu g/m^3$ is assumed, whereas when a hypothetical threshold of 12 $\mu g/m^3$ is assumed, point estimates are roughly 5 to 20% of base case estimates in nonattainment areas (and even smaller in attainment areas). A similar pattern is seen when considering the impact of alternative hypothetical thresholds in the range of 10 to 20 $\mu g/m^3$ on risks associated with short-term exposure.

In considering these estimates of $PM_{2.5}$-related incidence of annual total mortality upon meeting the current standards in a number of example urban areas, together with the uncertainties in these estimates, staff concludes that they are indicative of risks that can reasonably be judged to be important from a public health perspective and provide support for consideration of standards that would provide increased protection from that afforded by the current $PM_{2.5}$ standards. In the absence of evidence of clear thresholds, staff believes that it is appropriate to give most weight to the base case risk estimates. These estimates indicate the likelihood of thousands of premature deaths per year in urban areas across the U.S. Beyond the estimated incidences of mortality discussed above, staff also recognizes that similarly substantial numbers of incidences of hospital admissions, emergency room visits, aggravation of asthma and other respiratory symptoms, and increased cardiac-related risk are also likely in many urban areas, based on risk assessment results presented in Chapter 4 and on the discussion related to the pyramid of effects drawn from section 9.2.5 of the CD. Staff also believes that it is important to recognize how highly dependent these risk estimates are on the shape of the underlying concentration-response functions. In so doing, staff nonetheless notes that in considering even the largest assumed hypothetical thresholds, estimated mortality risks are not completely eliminated when current $PM_{2.5}$ standards are met in a number of example urban areas, including all such areas that do not meet the standards based on recent air quality.

Staff well recognizes that as the body of available evidence has expanded, it has added greatly both to our knowledge of PM-related effects, as well as to the complexity inherent in interpreting the evidence in a policy-relevant context as a basis for setting appropriate standards. In considering available evidence, risk estimates, and related limitations and uncertainties, staff concludes that the available information clearly calls into question the adequacy of the current suite of $PM_{2.5}$ standards, and provides strong support for giving consideration to revising the current $PM_{2.5}$ standards to provide increased public health protection. Staff conclusions and recommendations for indicators, averaging times, and levels and forms of alternative, more protective primary standards for fine particles are discussed in the following sections.

5.3.2 Indicators

In 1997, EPA established $PM_{2.5}$ as the indicator for fine particles. In reaching this decision, the Agency first considered whether the indicator should be based on the mass of a size-differentiated sample of fine particles or on one or more components within the mix of fine particles. Secondly, in establishing a size-based indicator, a size cut point needed to be selected that would appropriately distinguish fine particles from particles in the coarse mode.

In addressing the first question in the last review, EPA determined that it was more appropriate to control fine particles as a group, as opposed to singling out any particular component or class of fine particles based on the following considerations. Community health studies had found significant associations between various indicators of fine particles (including $PM_{2.5}$ or PM_{10} in areas dominated by fine particles) and health effects in areas with significant mass contributions of differing components or sources of fine particles, including sulfates, wood smoke, nitrates, secondary organic compounds and acid sulfate aerosols. In addition, 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. It also was not possible to rule out any component within the mix of fine particles as not contributing to the fine particle effects found in epidemiologic studies. Thus, it was determined that total mass of fine particles was the most appropriate indicator for fine particle standards rather than an indicator based on PM composition (62 FR 38667, July 18, 1997).

Having selected a size-based indicator for fine particles, the Agency then based its selection of a specific cut point on a number of considerations. In focusing on a cut point within the size range of 1 to 3 μm (i.e., the intermodal range between fine and coarse mode particles), EPA recognized that the choice of any specific sampling cut point within this range was largely a policy judgment. In making this judgment, the Agency noted that the available epidemiologic studies of fine particles were based largely on $PM_{2.5}$; only very limited use of PM_1 monitors had been made. While it was recognized that using PM_1 as an indicator of fine particles would exclude the tail of the coarse mode in some locations, in other locations it would miss a portion of the fine PM, especially under high humidity conditions, which would result in falsely low fine

PM measurements on days with some of the highest fine PM concentrations. The selection of a 2.5 μm cut point reflected the regulatory importance that was placed on defining an indicator for fine particle standards that would more completely capture fine particles under all conditions likely to be encountered across the U.S., especially when fine particle concentrations are likely to be high, while recognizing that some small coarse particles would also be captured by $PM_{2.5}$ monitoring.[2] Thus, EPA's selection of 2.5 μm as the cut point for the fine particle indicator was based on considerations of consistency with the epidemiologic studies, the regulatory importance of more completely capturing fine particles under all conditions, and the limited potential for intrusion of coarse particles in some areas; it also took into account the general availability of monitoring technology (62 FR 38668).

In this current review, staff observes that the same considerations apply for selection of an appropriate indicator for fine particles. As an initial matter, staff notes that the available epidemiologic studies linking mortality and morbidity effects with short- and long-term exposures to fine particles continue to be largely indexed by $PM_{2.5}$. Some epidemiologic studies also have continued to implicate various PM components (e.g., sulfates, nitrates, carbon, organic compounds, and metals) as being associated with adverse effects; effects have been reported with a broad range of PM components, as summarized in Table 9-13 of the CD (p. 9-31). Animal toxicologic and controlled human exposure studies, evaluated in Chapter 7 of the CD, have continued to link a variety of PM components or particle types (e.g., sulfates or acid aerosols, metals, organic constituents, bioaerosols, diesel particles) with health effects, though often at high concentrations (CD section 7.10.2). In addition, some recent studies have suggested that the ultrafine subset of fine particles may also be associated with adverse effects (CD, pp. 8-66, 8-199).

Staff recognizes that, for a given health response, some PM components are likely to be more closely linked with that response than others (CD, p. 9-30). That different PM constituents may have differing biological responses is an important source of uncertainty in interpreting epidemiologic evidence. For specific effects there may be stronger correlation with individual

[2] In reaching this decision, EPA indicated that it might be appropriate to address undue intrusion of coarse mode particles resulting in violations of $PM_{2.5}$ standards in the context of policies established to implement such standards (62 FR 38668).

PM components than with particle mass. For example, in some toxicologic studies of cardiovascular effects, such as changes in heart rate, electrocardiogram measures, or increases in arrhythmia, PM exposures of equal mass did not produce the same effects, indicating that PM composition was important (CD, p. 7-30). In addition, section 9.2.3.1.3 of the CD indicates that particles, or particle-bound water, can act as carriers to deliver other toxic agents into the respiratory tract, highlighting the fact that exposure to particles may elicit effects that are linked with a mixture of components more than with any individual PM component.

Thus, epidemiologic and toxicologic studies summarized above and discussed in the CD have provided evidence for effects associated with various fine particle components or size-differentiated subsets of fine particles. The CD concludes: "These studies suggest that many different chemical components of fine particles and a variety of different types of source categories are all associated with, and probably contribute to, mortality, either independently or in combinations" (CD, p. 9-31). Conversely, the CD provides no basis to conclude that any individual fine particle component *cannot* be associated with adverse health effects. There is no evidence that would lead toward the selection of one or more PM components as being primarily responsible for effects associated with fine particles, nor is there any component that can be eliminated from consideration. Staff continues to recognize the importance of an indicator that not only captures all of the most harmful components of fine PM (i.e., an effective indicator), but also places greater emphasis for control on those constituents or fractions, including most sulfates, acids, transition metals, organics, and ultrafine particles, that are most likely to result in the largest risk reduction (i.e., an efficient indicator). Taking into account the above considerations, staff concludes that it remains appropriate to control fine particles as a group; i.e., that total mass of fine particles is the most appropriate indicator for fine particle standards.

With regard to an appropriate cut point for a size-based indicator of total fine particle mass, the CD most generally concludes that advances in our understanding of the characteristics of fine particles continue to support the use of particle size as an appropriate basis for distinguishing between these subclasses, and that a nominal cut point of 2.5 μm remains appropriate (CD, p. 9-22). This conclusion follows from a recognition that within the intermodal range of 1 to 3 μm there is no unambiguous definition of an appropriate cut point for the

separation of the overlapping fine and coarse particle modes (CD, p. 9-8). Within this range, staff considered cut points of both 1 μm and 2.5 μm. Consideration of these two cut points took into account that there is generally very little mass in this intermodal range, although in some circumstances (e.g., windy, dusty areas) the coarse mode can extend down to and below 1 μm, whereas in other circumstances (e.g., high humidity conditions, usually associated with very high fine particle concentrations) the fine mode can extend up to and above 2.5 μm. The same considerations that led to the selection of a 2.5 μm cut point in the last review – that the epidemiologic evidence was largely based on $PM_{2.5}$ and that it was more important from a regulatory perspective to more completely capture fine particles under all conditions likely to be encountered across the U.S. (especially when fine particle concentrations are likely to be high) than to avoid some coarse-mode intrusion into the fine fraction in some areas – also lead to the same conclusion in this review. In addition, section 9.2.1.2.3. of the CD discusses the potential health significance of particles as carriers of water, oxidative compounds, and other components into the respiratory system. This consideration adds to the importance of ensuring that larger accumulation-mode particles are included in the fine particle size cut. Therefore, as observed previously in section 3.1.2, the scientific evidence leads the CD to conclude that 2.5 μm remains an appropriate upper cut point for a fine particle mass indicator.

Thus, consistent with the CD's conclusion that 2.5 μm remains an appropriate cut point for including the larger accumulation-mode fine particles while limiting intrusion of coarse particles, staff recommends that $PM_{2.5}$ be retained as the indicator for fine particles. Staff further concludes that currently available studies do not provide a sufficient basis for supplementing mass-based fine particle standards with standards for any specific fine particle component or subset of fine particles, or for eliminating any individual component or subset of components from fine particle mass standards.

Further, staff notes that since the last review an extensive $PM_{2.5}$ monitoring network has been deployed and operated in cooperative efforts with State, local and Tribal agencies and with instrument manufacturers. At the same time, EPA has been working on the development of strategies and programs to implement the 1997 $PM_{2.5}$ standards, based on the Federal Reference Monitor (FRM) for $PM_{2.5}$. The new monitoring network has provided substantial new air quality

potentially be used to provide information to the public based on episodic very short-term peak fine particle levels that may be of public health concern.

In the last review, having decided to set both annual and 24-hour $PM_{2.5}$ standards, EPA also made judgments as to the most effective and efficient approach to establishing a suite of standards that, taken together, would appropriately protect against effects associated with both long- and short-term exposures. At that time, EPA selected an approach that was based on treating the annual standard as the generally controlling standard for lowering the entire distribution of $PM_{2.5}$ concentrations, with the 24-hour standard providing additional protection against the occurrence of peak 24-hour concentrations. The 24-hour standard was intended to address in particular those peaks that result in 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 approach was supported by results of the PM risk assessment from the last review which indicated that peak 24-hour $PM_{2.5}$ concentrations contribute 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 was seen to be 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.

In this review, staff recognizes that some key considerations that led to establishing a generally controlling annual standard in the last review are still valid. In particular, staff observes that:

- EPA's updated risk assessment supports 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, as discussed in Chapter 4 (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 clear population thresholds, but rather reflect relationships between health effects and ambient PM across a wide distribution of PM concentrations. Thus, as in the last review, staff recognizes that these studies do not provide a basis for identifying a lowest-observed-effect level that would clearly translate into a 24-hour standard that would protect against all effects related to short-term exposures.

Nonetheless, staff believes that the greatly expanded body of epidemiologic evidence and air quality data provide the basis for considering alternative approaches to establishing a suite of $PM_{2.5}$ standards. Thus, staff has not focused *a priori* on an annual standard as the generally controlling standard for protection against effects associated with both long- and short-term exposures. Rather, staff has broadened its view to consider both evidence-based and risk-based approaches to evaluating the protection that a suite of $PM_{2.5}$ standards can provide against effects associated with long-term exposures and against short-term exposures. These evaluations, discussed in the next two sections, provide the basis for integrated recommendations on ranges of alternative suites of standards that, when considered together, protect against effects associated with both long- and short-term exposures.

5.3.4 Alternative $PM_{2.5}$ Standards to Address Health Effects Related to Long-term Exposure

In considering alternative $PM_{2.5}$ standards that would provide protection against health effects related to long-term exposures, staff has taken into account both evidence-based and risk-based considerations. As discussed below in this section, staff has first evaluated the available evidence from long-term exposure studies, as well as the uncertainties and limitations in that evidence, to assess the degree to which alternative annual $PM_{2.5}$ standards can be expected to provide protection against effects related to long-term exposures. Secondly, staff has considered the quantitative risk estimates for long-term exposure effects, discussed in Chapter 4, to assess the extent to which alternative annual and/or 24-hour standards can be expected to reduce the

estimated risks attributable to long-term exposure to $PM_{2.5}$. Staff conclusions as to ranges of alternative annual and/or 24-hour standards that would provide protection against health effects related to long-term exposures are summarized at the end of this section. The integrated staff recommendations presented in section 5.3.7 are based in part on the conclusions from this section and in part on staff conclusions from the next section, in which alternative $PM_{2.5}$ standards to address health effects related to short-term exposures are assessed.

5.3.4.1 Evidence-based Considerations

In taking into account evidence-based considerations, staff has focused on long-term exposure studies of fine particles in the U.S. As discussed above, staff notes that the reanalyses and extensions of earlier studies have confirmed and strengthened the evidence of long-term associations for both mortality and morbidity effects. The assessment in the CD of these mortality studies, taking into account study design, the strength of the study (in terms of statistical significance and precision of result), and the consistency and robustness of results, concluded that it was appropriate to give the greatest weight to the reanalyses of the Six Cities study and the ACS study, and in particular to the results of the extended ACS study (CD, p. 9-33). The assessment in the CD of the relevant morbidity studies noted in particular the results of the new studies of the children's cohort in Southern California as providing evidence of respiratory morbidity with long-term PM exposures.

Staff believes it is appropriate to consider a level for an annual $PM_{2.5}$ standard that is somewhat below the averages of the long-term concentrations across the cities in each of these studies, recognizing that the evidence of an association in any such study is strongest at and around the long-term average where the data in the study are most concentrated. For example, the interquartile range of long-term average concentrations within a study, or a range within one standard deviation around the study mean, might be used to characterize the range over which the evidence of association is strongest. Staff also believes it is appropriate to consider the long-term average concentration at the point where the confidence interval becomes notably wider, suggestive of a concentration below which the association becomes appreciably more uncertain and the possibility that an effects threshold may exist becomes more likely. Staff further notes that in considering a level for a standard that is to provide protection with an adequate margin of

safety, it is appropriate to take into account evidence of effects for which the reported associations provide only suggestive evidence of a potentially causal association.

In looking first at the long-term exposure mortality studies, staff notes that the long-term mean $PM_{2.5}$ concentration in the Six Cities study was 18 μg/m³, within an overall range of 11 to 30 μg/m³. In the studies using the ACS cohort, the long-term mean $PM_{2.5}$ concentration across the cities was 21 μg/m³ in the initial study and in the reanalysis of that study, within an overall range of 9 to 34 μg/m³. In the extended ACS study, the mean for the more recent time period used in the analysis (from 1999 to 2000) was 14 μg/m³; in looking at the association based on the air quality averaged over both time periods (which was the basis for the concentration-response functions from this study used in the risk assessment), the long-term mean $PM_{2.5}$ concentration was 17.7 μg/m³, with a standard deviation of ± 4, ranging down to 7.5 μg/m³. The CD notes that the confidence intervals around the relative risk functions in this extended study, as in the initial ACS study, start to become appreciably wider below approximately 12 to 13 μg/m³. In considering the Southern California children's cohort study showing evidence of decreased lung function growth, staff notes that the long-term mean $PM_{2.5}$ concentration was 15 μg/m³, ranging from 7 to 32 μg/m³ across the cities. This is approximately equal to the long-term mean $PM_{2.1}$ concentration in the earlier 24 City study, showing effects on children's lung function, in which the long-term mean concentration was 14.5 μg/m³, ranging from 9 to 17 μg/m³ across the cities.

In considering this evidence, staff concludes that these studies provide a basis for considering an annual $PM_{2.5}$ standard somewhat below 15 μg/m³, down to about 12 μg/m³. A standard of 14 μg/m³ would reflect some consideration of the more recent long-term exposure studies that show associations over a somewhat lower range of air quality than had been observed in the studies available in the last review. A standard of 13 μg/m³ would be consistent with a judgment that appreciable weight should be accorded these long-term exposure studies, particularly taking into account the most recent extended ACS mortality study and the Southern California children's cohort morbidity study. A standard level of 13 μg/m³ would be well below the long-term mean in the Six Cities mortality study and approximately one standard deviation below the extended ACS mortality study mean, while being somewhat closer to the long-term means in the morbidity studies discussed above. A standard of 12 μg/m³ would be consistent

City Assumed threshold (μg/m³) base = 7.5 μg/m³ (LML in ACS Extended Study)	Detroit			Los Angeles			Philadelphia			Pittsburgh			St. Louis		
	base	10	12	base	10	12	base	10	12	base	10	12	base	10	12
Incidence Associated with Meeting Current Standards	*520*	*280*	*40*	*1510*	*820*	*140*	*540*	*340*	*140*	*400*	*220*	*30*	*600*	*310*	*20*
13 μg/m³ annual and 65 μg/m³ daily; 98th percentile	32	67		33	67		30	54		32	68		33	70	
40	32	67		33	67		30	54		32	68		33	70	
35	32	67		33	67		44	79		34	73		33	70	
30				45	92		64	100		58	100				
25	48	100		74	100		84			82			60	100	
40 μg/m³ daily; 99th percentile	32	67		66	100		89	100		32	68		33	70	
35				84			100			50	100				
30				100						72			45	97	
25	60	100								93			72	100	
12 μg/m³ annual and 65 μg/m³ daily; 98th percentile	48	100		49	100		45	80		48	100		49	100	
40	48			49			45	80		48			49		
35															
30							64	100		58					
25				74			84			82			60		
40 μg/m³ daily; 99th percentile	48			66			89	100		48			49		
35				84			100			50					
30				100						72					
25	60									93			72		

The alternative annual $PM_{2.5}$ standards considered here include a range of levels from 15 to 12 μg/m^3, and simulating attainment of the standards is based on a percent rollback calculated using the highest monitor in an area, as noted in Table 5-1 and discussed in Chapter 4, section 4.2.3. The alternative 24-hour $PM_{2.5}$ standards considered here include a range of levels from 65 to 25 μg/m^3 in conjunction with two different forms, including the 98th percentile form of the current 24-hour $PM_{2.5}$ standard and an alternative 99th percentile form. Further discussion of alternative forms of the annual and 24-hour standards is presented below in section 5.3.6.

In looking at the base case estimates, staff has first considered the estimated reductions associated with lower levels of the annual $PM_{2.5}$ standard, without changing the 24-hour standard. From Table 5-2, staff observes that alternative annual standard levels of 14, 13, and 12 μg/m^3 result in generally consistent estimated risk reductions from long-term exposure to $PM_{2.5}$ of roughly 20, 30, and 50 percent, respectively, across all five example cities. Thus, for the base case assessment in which mortality risks are estimated down to the lowest measured level in the extended ACS study, estimated reductions in mortality associated with long-term exposure to $PM_{2.5}$ are no greater than 50 percent in any of the five example cities with changes in the annual standard down to a level of 12 μg/m^3.

Staff also examined the effect on mortality reduction if the 24-hour standard were to change. Staff first notes that the estimated reductions in long-term mortality risk associated with changes to the 24-hour standard are much more variable across cities than with changes in just the annual standard. Further, no combination of standards within the ranges that staff has considered result in the elimination of all estimated long-term mortality risk in all example cities. This assessment indicates that estimated reductions in long-term mortality risk of approximately 50 percent or greater in the five example cities generally result from 24-hour standards set at 30 to 25 μg/m^3, based on either the 98th or 99th percentile form of such a standard, depending on the city.

Staff further considered the effects of various combinations of the annual and 24-hour standard. Staff notes in particular that the base case estimates of long-term mortality risk reduction associated with a 24-hour standard set at 25 μg/m^3 provides the same degree of risk reduction regardless of the level of the annual standard within the range of 15 to 12 μg/m^3; a 24-

hour standard set at 30 μg/m^3 provides the same degree of risk reduction in most but not all cases. That is, in the range of 30 to 25 μg/m^3, the 24-hour standard would be the generally controlling standard in most cases relative to an annual standard in the range of 15 to 12 μg/m^3; and, in those cases, lowering the annual standard to as low as 12 μg/m^3 would result in no additional estimated reductions in long-term mortality risks.

Beyond this base case assessment, staff also has considered the extent to which the assumption of the presence of hypothetical thresholds in the concentration-response relationships would influence the estimated risk reductions. As noted above (section 5.3.1), the estimated incidence of $PM_{2.5}$-related mortality associated with long-term exposure when the current standards are met are appreciably smaller, although still present, under these assumed hypothetical thresholds. In considering an assumed threshold of 10 μg/m^3, staff observes that lowering the annual standard to alternative levels of 14, 13, and 12 μg/m^3 (without changing the 24-hour standard) results in estimated risk reductions of roughly 30 to 40 percent, 50 to 70 percent, and 80 to 100 percent, respectively, across the five example cities. In considering changes to the annual and/or 24-hour $PM_{2.5}$ standards in this case, staff first notes that mortality risk associated with long-term exposure is estimated to be reduced by 100 percent in all five cities with a 24-hour standard set at 25 μg/m^3, in combination with the current annual standard. For a 24-hour standard set at 35 μg/m^3, with a 99th percentile form, estimated risk reductions remained at 100 percent in three of the cities, but were only 40 and 6 percent in the other two cities. Under this assumed threshold of 10 μg/m^3, similar to the base case, there is little if any additional reduction obtained in lowering the annual standard below 15 μg/m^3 in conjunction with 24-hour standards in this range. Thus, in this case, as in the base case, changes in the 24-hour standard, while retaining the current annual standard, can result in larger but much more variable estimated reductions in risks associated with long-term exposures across the five cities.

Further, in considering an assumed hypothetical threshold of 12 μg/m^3, staff observes that lowering the annual standard to a level of 14 μg/m^3 (without changing the 24-hour standard) results in estimated risk reductions of 100 percent in all five cities. In considering changes to the 24-hour $PM_{2.5}$ standard alone in this case, staff notes that long-term mortality risk is estimated to

be reduced by 100 percent in all five cities with a 24-hour standard set at 30 $\mu g/m^3$, 98th percentile form.

5.3.4.3 Summary

In summary, in considering the epidemiologic evidence, estimates of risk reductions associated with alternative annual and/or 24-hour standards, and the related limitations and uncertainties, staff concludes that there is clear support for considering revisions to the suite of current $PM_{2.5}$ standards to provide additional protection against health effects associated with long-term exposures. In looking specifically at the evidence of associations between long-term exposure to $PM_{2.5}$ and serious health effects, including total, cardiovascular, and lung cancer mortality, as well as respiratory-related effects on children, staff concludes that it is appropriate to consider an annual $PM_{2.5}$ standard in the range of 15 down to 12 $\mu g/m^3$. In considering the results of the quantitative risk assessment, in the absence of evidence of clear thresholds, staff believes that it is appropriate to give significant weight to base case risk estimates, while also considering the implications of potential thresholds within the range of the air quality data from the relevant studies. In so doing, staff finds further support for considering an annual $PM_{2.5}$ standard in the range of 14 to 12 $\mu g/m^3$. Alternatively, staff also finds support for a revised 24-hour standard, in conjunction with retaining the current annual standard, in the range of 35 to 25 $\mu g/m^3$, with an emphasis on a 99th percentile form especially with a standard level in the middle or upper end of this range. Staff notes that a 24-hour standard at a level of 40 $\mu g/m^3$ is estimated to provide no additional protection against the serious health effects associated with long-term $PM_{2.5}$ exposures in two or three of the five example cities (for a 99th or 98th percentile form, respectively) relative to that afforded by the current annual $PM_{2.5}$ standard, regardless of the weight that is given to the potential for a threshold within the range considered by staff. Staff believes that a suite of $PM_{2.5}$ standards selected from the alternatives identified above could provide an appropriate degree of protection against the mortality and morbidity effects associated with long-term exposure to $PM_{2.5}$ in studies in urban areas across the U.S..

5.3.5 Alternative $PM_{2.5}$ Standards to Address Health Effects Related to Short-term Exposure

In considering alternative $PM_{2.5}$ standards that would provide protection against health effects related to short-term exposures, staff has similarly taken into account both evidence-based and risk-based considerations. As discussed below in this section, staff has first evaluated the available evidence from short-term exposure studies, as well as the uncertainties and limitations in that evidence, to assess the degree to which alternative 24-hour and/or annual $PM_{2.5}$ standards can be expected to provide protection against effects related to short-term exposures. Secondly, staff has considered the quantitative risk estimates for short-term exposure effects, discussed in Chapter 4, to assess the extent to which alternative annual and/or 24-hour standards can be expected to reduce the estimated risks attributable to short-term exposure to $PM_{2.5}$. Staff conclusions as to ranges of alternative annual and/or 24-hour standards that would provide protection against health effects related to short-term exposures are summarized at the end of this section. As noted above, the integrated staff recommendations presented in section 5.3.7 are based in part on the conclusions from this section and in part on staff conclusions from the previous section, in which alternative $PM_{2.5}$ standards to address health effects related to long-term exposures are assessed.

5.3.5.1 Evidence-based Considerations

In taking into account evidence-based considerations, staff has evaluated the available evidence from short-term exposure studies, as well as the uncertainties and limitations in that evidence. In so doing, staff has focused on U.S. and Canadian short-term exposure studies of fine particles (Appendix 3A). We took into account reanalyses that addressed GAM-related statistical issues and considered the extent to which the studies report statistically significant and relatively precise relative risk estimates; the reported associations are robust to co-pollutant confounding and alternative modeling approaches; and the studies used relatively reliable air quality data. In particular, staff has focused on those specific studies, identified above in section 5.3.1, that provide evidence of associations in areas that would have met the current annual and 24-hour $PM_{2.5}$ standards during the time of the study. Staff believes that this body of evidence

can serve as a basis for 24-hour and/or annual $PM_{2.5}$ standards that would provide increased protection against effects related to short-term exposures.

As an initial matter, staff recognizes, as discussed above, that these short-term exposure studies provide no evidence of clear thresholds, or lowest-observed-effects levels, in terms of 24-hour average concentrations. Staff notes that of the two $PM_{2.5}$ studies that explored potential thresholds, one study in Phoenix provided some suggestive evidence of a threshold possibly as high as 20 to 25 $\mu g/m^3$, whereas the other study provided evidence suggesting that if a threshold existed, it would likely be appreciably below 25 $\mu g/m^3$. While there is no evidence for clear thresholds within the range of air quality observed in the epidemiologic studies, for some health endpoints (such as total nonaccidental mortality) it is likely to be extremely difficult to detect threshold levels (CD, p.9-45). As a consequence, this body of evidence is difficult to translate directly into a specific 24-hour standard that would independently protect against all effects associated with short-term exposures. Staff notes that the distributions of daily $PM_{2.5}$ concentrations in these studies often extend down to or below background levels, such that consideration of the likely range of background concentrations across the U.S., as discussed in Chapter 2, section 2.6, becomes important in identifying a lower bound of a range of 24-hour standards appropriate for consideration.

Being mindful of the difficulties posed by issues relating to threshold and background levels, staff has first considered this short-term exposure epidemiologic evidence as a basis for alternative 24-hour $PM_{2.5}$ standards. In so doing, staff has focused on the upper end of the distributions of daily $PM_{2.5}$ concentrations, particularly in terms of the 98th and 99th percentile values, reflecting the form of the current 24-hour standard and an alternative form considered in the risk assessment, respectively. In looking at the specific studies identified in section 5.3.1 that report statistically significant association in areas that would have met the current $PM_{2.5}$ standards, including studies in Phoenix (Mar et al., 1999, 2003), Santa Clara County, CA (Fairley, 1999, 2003) and eight Canadian cities (Burnett et al., 2000 and Burnett and Goldberg, 2003), staff notes that the 98th percentile values range from approximately 32 to 39 $\mu g/m^3$ in Phoenix and the eight Canadian cities, up to 59 $\mu g/m^3$ in Santa Clara Country; 99th percentile values range from 34 to 45 $\mu g/m^3$ in Phoenix and the eight Canadian cities, up to 69 $\mu g/m^3$ in

evidence discussed above, giving little weight to the remaining uncertainties in the broader body of short-term exposure evidence, including the possibility of a threshold within the range of air quality in the studies and the recognition that results may be sensitive to selection of models beyond the range of models examined in these particular studies.

Consistent with the conclusions reached in the last review (62 FR 38674-7), however, staff continues to believe that an annual standard cannot be expected to offer an adequate margin of safety against the effects of all short-term exposures, especially in areas with unusually high peak-to-mean ratios of $PM_{2.5}$ levels, possibly associated with strong local or seasonal sources, or for potential $PM_{2.5}$-related effects that may be associated with shorter-than-daily exposure periods (noted above in section 5.3.3). As a result, if an alternative annual standard were adopted to provide primary protection against effects associated with short-term exposures, staff believes it is appropriate also to consider an alternative 24-hour $PM_{2.5}$ standard to provide such supplemental protection. Such a supplemental 24-hour standard could reasonably be based on air quality information (from 2001 to 2003) in Chapter 2, Figure 2-23, that shows the distribution of 98th percentile values as a function of annual means values in urban areas across the U.S. Based on this information, staff concludes that a supplemental standard in the range of approximately 40 to 35 $\mu g/m^3$ would limit peak concentrations in areas with relatively high peak-to-mean ratios (i.e., generally in the upper quartile to the upper 5th percentile, respectively) and with annual mean concentrations in the range of 12 to 15 $\mu g/m^3$.

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, and the population in those 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 562 counties, are shown in Tables 5-3(a) and (b).

Table 5-3(a). Predicted percent of counties with monitors (and percent of population in counties with monitors) not likely to meet alternative annual and 24-hour (98th percentile form) $PM_{2.5}$ standards

Alternative Standards and Levels (µg/m³)	Percent of counties, total and by region, (and total percent population) not likely to meet stated standard and level*								
	Total counties (population)	Northeast	Southeast	Industrial Midwest	Upper Midwest	Southwest	Northwest	Southern CA	Outside Regions**
No. of counties with monitors (Population, in thousands)	562 (185,780)	83	168	130	49	21	81	15	15
Annual standard only:									
15	14 (30)	19	7	29	0	0	4	60	0
14	25 (41)	28	21	51	0	5	5	67	0
13	40 (55)	47	40	76	4	5	7	67	0
12	54 (66)	70	61	89	12	5	12	67	0
Combined annual /24-hour:									
15 / 65	14 (30)	19	7	29	0	0	4	60	0
15 / 50	15 (31)	19	7	29	0	0	9	60	0
15 / 45	15 (33)	19	7	29	0	10	12	60	0
15 / 40	17 (35)	20	7	30	0	10	19	60	0
15 / 35	27 (48)	45	8	47	0	10	36	60	7
15 / 30	51 (72)	78	29	87	6	19	51	80	13
15 / 25	78 (86)	98	77	99	51	43	65	80	13
14 / 65	25 (41)	28	21	51	0	5	5	67	0
14 / 50	26 (43)	28	21	51	0	5	10	67	0
14 / 45	26 (44)	28	21	51	0	10	12	67	0
14 / 40	27 (46)	28	21	52	0	10	19	67	0
14 / 35	34 (55)	45	22	58	0	10	36	67	7
14 / 30	53 (72)	78	33	88	6	19	51	80	13
14 / 25	78 (86)	98	77	99	51	43	65	80	13

	Alternative Standards and Levels (µg/m3)	Percent of counties, total and by region, (and total percent population) not likely to meet stated standard and level*								
		Total counties (population)	Northeast	Southeast	Industrial Midwest	Upper Midwest	Southwest	Northwest	Southern CA	Outside Regions**
	No. of counties with monitors (Population, in thousands)	562 (185,780)	83	168	130	49	21	81	15	15
1	13 / 65	40 (55)	47	40	76	4	5	7	67	0
2	13 / 50	40 (56)	47	40	76	4	5	10	67	0
3	13 / 45	41 (57)	47	40	76	4	10	12	67	0
4	13 / 40	42 (58)	47	40	76	4	10	19	67	0
5	13 / 35	45 (62)	53	40	77	4	10	36	67	7
6	13 / 30	57 (74)	78	43	90	8	19	51	80	13
7	13 / 25	78 (86)	98	77	99	51	43	65	80	13
8	12 / 65	54 (66)	70	61	89	12	5	12	67	0
9	12 / 50	54 (66)	70	61	89	12	5	12	67	0
10	12 / 45	54 (67)	70	61	89	12	10	14	67	0
11	12 / 40	55 (68)	70	61	89	12	10	20	67	0
12	12 / 35	58 (71)	70	61	89	12	10	36	67	7
13	12 / 30	64 (78)	84	62	94	14	19	51	80	13
14	12 / 25	79 (86)	98	78	99	51	43	65	80	13

15 * Based on 2001-2003 data for sites with at least 11 samples per quarter for all 12 quarters. As such, these estimates are not based on the same air quality data
16 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
17 counties that are likely not to attain the given standards and should be interpreted with caution.

18 ** "Outside Regions" includes Alaska, Hawaii, Puerto Rico, and the Virgin Islands.

Table 5-3(b). Predicted percent of counties with monitors (and percent of population in counties with monitors) not likely to meet alternative annual and 24-hour (99th percentile form) $PM_{2.5}$ standards

Alternative Standards and Levels (mg/m3)	Percent of counties, total and by region, (and total percent population) not likely to meet stated standards and levels*								
	Total counties (population)	Northeast	Southeast	Industrial Midwest	Upper Midwest	Southwest	Northwest	Southern CA	Outside Regions**
No. of counties with monitors (Population, in thousands)	562 (185,780)	83	168	130	49	21	81	15	15
Annual only:									
15	14 (30)	19	7	29	0	0	4	60	0
14	25 (41)	28	21	51	0	5	5	67	0
13	40 (55)	47	40	76	4	5	7	67	0
12	54 (66)	70	61	89	12	5	12	67	0
Combined annual / 24-hour:									
15 / 65	14 (30)	19	7	29	0	0	5	60	0
15 / 50	16 (33)	19	7	29	0	10	15	60	0
15 / 45	18 (35)	24	7	32	0	10	21	60	0
15 / 40	27 (46)	47	9	42	0	10	36	67	7
15 / 35	44 (68)	72	17	77	0	19	51	80	13
15 / 30	68 (82)	96	54	97	35	38	59	80	13
15 / 25	85 (89)	100	86	99	69	48	73	87	13
14 / 65	25 (41)	28	21	51	0	5	6	67	0
14 / 50	27 (44)	28	21	51	0	10	15	67	0
14 / 45	28 (45)	30	21	52	0	10	21	67	0
14 / 40	35 (53)	48	23	57	0	10	36	73	7
14 / 35	47 (70)	72	27	78	0	19	51	80	13
14 / 30	68 (82)	96	54	97	35	38	59	80	13
14 / 25	85 (89)	100	86	99	69	48	73	87	13

Alternative Standards and Levels (mg/m3)	Percent of counties, total and by region, (and total percent population) not likely to meet stated standards and levels*								
	Total counties (population)	Northeast	Southeast	Industrial Midwest	Upper Midwest	Southwest	Northwest	Southern CA	Outside Regions**
No. of counties with monitors (Population, in thousands)	562 (185,780)	83	168	130	49	21	81	15	15
13 / 65	40 (55)	47	40	76	4	5	9	67	0
13 / 50	41 (57)	47	40	76	4	10	15	67	0
13 / 45	42 (58)	49	40	76	4	10	21	67	0
13 / 40	47 (62)	59	40	77	4	10	36	73	7
13 / 35	54 (73)	75	40	85	4	19	51	80	13
13 / 30	70 (82)	96	58	97	35	38	59	80	13
13 / 25	85 (89)	100	86	99	69	48	73	87	13
12 / 65	54 (66)	70	61	89	12	5	12	67	0
12 / 50	55 (67)	70	61	89	12	10	16	67	0
12 / 45	56 (68)	71	61	89	12	10	22	67	0
12 / 40	59 (71)	75	62	89	12	10	36	73	7
12 / 35	63 (77)	80	62	92	12	19	51	80	13
12 / 30	73 (83)	96	68	98	35	38	59	80	13
12 / 25	85 (89)	100	86	99	69	48	73	87	13

* Based on 2001-2003 data for sites with at least 11 samples per quarter for all 12 quarters. As such, these estimates are not based on the same 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.

For example, from Table 5-3(a) it can be seen that for an annual standard set at 15 $\mu g/m^3$, 24-hour standard levels ranging from 40 to 35 $\mu g/m^3$, with a 98th percentile form, would add approximately 3 to 13 percent 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; with a 99th percentile form, as seen in Table 5-3(b), these percentages increase to 13 to 30 percent.. For an annual standard set at 12 $\mu g/m^3$, 24-hour standard levels in this range would add approximately 1 to 4 percent, or 5 to 9 percent, to the percentage of counties for standards with a 98th or 99th percentile form, respectively. As seen in Tables 5-3(a) and (b), the percentage of the population that would be afforded greater public health protection from these alternative standards would increase somewhat more than would the percentage of counties not likely to attain the standards.

5.3.5.2 Risk-based Considerations

Beyond looking directly at the relevant epidemiologic evidence, staff has also considered the extent to which specific levels and forms of alternative 24-hour and annual $PM_{2.5}$ standards are likely to reduce the estimated risks attributable to short-term exposure to $PM_{2.5}$, and the uncertainties in the estimated risk reductions. As discussed above (section 5.3.1), staff has based this evaluation on the risk assessment results presented in Chapter 4, in which short-term exposure risks were estimated down to background or the lowest measured level (LML) in a particular study, whichever is higher. Staff also has considered the sensitivity of these results to the uncertainty related to potential thresholds by using concentration-response functions modified to incorporate assumed hypothetical threshold levels.

Table 5-4 summarizes estimated percentage reductions in mortality attributable to short-term exposure to $PM_{2.5}$ in going from meeting the current $PM_{2.5}$ standards to meeting alternative annual and 24-hour $PM_{2.5}$ standards in the five example cities that do not meet the current standards based on 2001-2003 air quality data. Base case estimated percentage risk reductions are given in the table, along with reductions associated with assumed alternative hypothetical thresholds. The percentage reductions presented in Table 5-4 represent approximate reductions relative to the total estimated short-term mortality incidence presented above in Table 5-1.

Table 5-4 Estimated Percent Reduction in $PM_{2.5}$-attributable Short-term Risk (mortality/morbidity) for Alternative Standards Relative to Meeting Current Standards (Base Case and Assumed Alternative Hypothetical Thresholds

City	Detroit				Los Angeles				Philadelphia				Pittsburgh				St. Louis			
Assumed threshold (µg/m³) (base = background or LML)	base	10	15	20	base	10	15	20	base	10	15	20	base	10	15	20	base	10	15	20
Incidence Associated with Meeting Current Standards	*120*	*50*	*30*	*10*	*250*	*120*	*60*	*30*	*370*	*190*	*110*	*60*	*50*	*20*	*10*	*5*	*190*	*80*	*30*	*10*
15 µg/m³ annual and 65 µg/m³ daily; 98th percentile	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
40	0	0	0	0	0	0	0	0	14	24	33	40	6	14	10	20	0	0	0	0
35	0	0	0	0	9	17	22	24	26	44	58	68	18	36	50	60	1	0	3	11
30	10	17	23	33	26	43	55	59	37	62	76	88	32	59	70	80	16	35	52	67
25	28	48	62	75	43	66	78	83	49	78	90	96	44	77	90	100	32	63	83	89
65 µg/m³ daily; 99th percentile	0	0	0	0	0	0	0	0	19	33	45	54	0	0	0	0	0	0	0	0
40	0	0	0	0	38	61	72	76	52	81	92	98	16	32	40	40	0	0	0	0
35	2	2	4	8	49	74	83	90	59	88	97	100	28	50	60	80	10	21	34	44
30	18	31	42	50	59	84	91	97	65	94	99	100	38	68	80	80	24	49	69	78
25	35	59	73	83	69	92	97	100	72	97	100	100	50	82	90	100	38	73	90	100
14 µg/m³ annual and 65 µg/m³ daily; 98th percentile	10	17	23	33	10	17	22	24	14	24	33	40	8	18	20	40	8	19	31	44
40	10	17	23	33	10	17	22	24	14	24	33	40	8	18	20	40	8	19	31	44
35	10	17	23	33	10	17	22	24	26	44	58	68	18	36	50	60	8	19	31	44
30	10	17	23	33	26	43	55	59	37	62	76	88	32	59	70	80	16	35	52	67
25	28	48	62	75	43	66	78	83	49	78	90	96	44	77	90	100	32	63	83	89
40 µg/m³ daily; 99th percentile	10	17	23	33	38	61	72	76	52	81	92	98	16	32	40	40	8	19	31	44
35	10	17	23	33	49	74	83	90	59	88	97	100	28	50	60	80	10	21	34	44
30	18	31	42	50	59	84	91	97	65	94	99	100	38	68	80	80	24	49	69	78
25	35	59	73	83	69	92	97	100	72	97	100	100	50	82	90	100	38	73	90	100

City Assumed threshold (µg/m³) (base = background or LML)	Detroit				Los Angeles				Philadelphia				Pittsburgh				St. Louis			
	base	10	15	20	base	10	15	20	base	10	15	20	base	10	15	20	base	10	15	20
Incidence Associated with Meeting Current Standards	*120*	*50*	*30*	*10*	*250*	*120*	*60*	*30*	*370*	*190*	*110*	*60*	*50*	*20*	*10*	*5*	*190*	*80*	*30*	*10*
13 µg/m³ annual and 65 µg/m³ daily; 98th percentile	18	33	46	50	19	33	41	45	17	30	42	49	18	32	40	60	17	37	55	67
40	18	33	46	50	19	33	41	45	17	30	42	49	18	32	40	60	17	37	55	67
35	18	33	46	50	19	33	41	45	26	44	58	68	18	38	50	60	17	37	55	67
30	18	33	46	50	26	43	55	59	37	62	76	88	32	59	70	80	17	37	55	67
25	28	48	62	75	43	66	78	83	49	78	90	96	44	77	90	100	32	63	83	89
40 µg/m³ daily; 99th percentile	18	33	46	50	38	61	72	76	52	81	92	98	18	32	40	60	17	37	55	67
35	18	33	46	50	49	74	83	90	59	88	97	100	28	50	60	80	17	37	55	67
30	18	33	46	50	59	84	91	97	65	94	99	100	38	68	80	80	24	49	69	78
25	35	59	73	83	69	92	97	100	72	97	100	100	50	82	90	100	38	73	90	100
12 µg/m³ annual and 65 µg/m³ daily; 98th percentile	28	48	62	75	28	47	59	62	26	45	58	68	26	50	60	80	26	53	72	89
40	28	48	62	75	28	47	59	62	26	45	58	68	26	50	60	80	26	53	72	89
35	28	48	62	75	28	47	59	62	26	45	58	68	26	50	60	80	26	53	72	89
30	28	48	62	75	28	47	59	62	37	62	76	88	32	59	70	80	26	53	72	89
25	28	48	62	75	43	66	78	83	49	78	90	96	44	77	90	100	32	63	83	89
40 µg/m³ daily; 99th percentile	28	48	62	75	38	61	72	76	52	81	92	98	26	50	60	80	26	53	72	89
35	28	48	62	75	49	74	83	90	59	88	97	100	28	50	60	80	26	53	72	89
30	28	48	62	75	59	84	91	97	65	94	99	100	38	68	80	80	26	53	72	89
25	35	59	73	83	69	92	97	100	72	97	100	100	50	82	90	100	38	73	90	100

The same alternative standards are considered here as were considered above in section 5.2.4. That is, the alternative annual $PM_{2.5}$ standards considered here include a range of levels from 15 to 12 $\mu g/m^3$, and simulating meeting these standards is based on a percent rollback calculated using the highest monitor in an area, as noted in Table 5-1 and discussed in Chapter 4, section 4.2.3. The alternative 24-hour $PM_{2.5}$ standards considered here again include a range of levels from 65 to 25 $\mu g/m^3$ in conjunction with two different forms, including the 98th percentile form of the current 24-hour $PM_{2.5}$ standard and an alternative 99th percentile form. Further discussion of these alternative forms for annual and 24-hour standards is presented below in section 5.3.6.

In looking at the base case estimates, staff first considered the estimated reductions associated with lower levels of the annual $PM_{2.5}$ standard, without changing the 24-hour standard. From Table 5-4, staff observes that lowering the annual standard to alternative levels of 14, 13, and 12 $\mu g/m^3$ results in small but generally consistent estimated risk reductions of roughly 10 to 15 percent, 15 to 20 percent, and 25 to 30 percent, respectively, across all five example cities. Thus, for the base case assessment in which mortality risks are estimated down to background or the lowest measured level in the relevant study, estimated reductions in mortality associated with short-term exposure to $PM_{2.5}$ are no greater than 30 percent in any of the five example cities with changes in the annual $PM_{2.5}$ down to a level of 12 $\mu g/m^3$.

In considering changes to the 24-hour and/or annual $PM_{2.5}$ standards for base case estimates, staff first notes that the estimated reductions in short-term mortality risk associated with changes to the 24-hour standard are generally larger and much more variable across cities than with changes in just the annual standard. Further, no combination of standards within the ranges that staff has considered results in the elimination of all estimated mortality risk associated with short-term exposure in all example cities. More specifically, a 24-hour standard of 25 $\mu g/m^3$ results in base case estimates of reductions in short-term mortality ranging from approximately 30 to 50 percent (98th percentile form) and 35 to 70 percent (99th percentile form) across the five cities in conjunction with any annual standard in the range of 15 to 12 $\mu g/m^3$. A 24-hour standard of 30 $\mu g/m^3$ results in base case estimates of reductions in short-term mortality ranging from approximately 25 to 35 percent (98th percentile form) and 25 to 65 percent (99th

percentile form) across the five cities in conjunction with an annual standard of 12 μg/m^3; the lower end, but not the upper end, of these ranges decreases somewhat in conjunction with annual standards from 13 to 15 μg/m^3. As in the assessment of risk related to long-term exposures discussed in section 5.3.4.2, this assessment indicates that 24-hour standards of 30 to 25 μg/m^3 become generally controlling standards in most cases within this range of annual standards.

Beyond this base case assessment, staff also has considered the extent to which the assumption of the presence of hypothetical thresholds in the concentration-response relationships would influence the estimated risk reductions. As noted above (section 5.3.1), the estimated incidence of $PM_{2.5}$-related mortality associated with short-term exposure when the current standards are met are appreciably smaller under these assumed hypothetical thresholds. In considering an assumed threshold of 10 μg/m^3, staff observes that lowering the annual standard to alternative levels of 14, 13, and 12 μg/m^3 (without changing the 24-hour standard) results in estimated risk reductions of roughly 15 to 25 percent, 30 to 35 percent, and 45 to 55 percent, respectively, across all five example cities. In considering changes to the 24-hour and/or annual $PM_{2.5}$ standards in this case, staff notes that a 24-hour standard of 25 μg/m^3 results in estimates of reductions in short-term mortality ranging from approximately 45 to 80 percent (98th percentile form) and 60 to 95 percent (99th percentile form) across the five cities in conjunction with any annual standard in the range of 15 to 12 μg/m^3. A 24-hour standard of 30 μg/m^3 results in estimates of reductions in short-term mortality ranging from approximately 45 to 60 percent (98th percentile form) and 50 to 95 percent (99th percentile form) across the five cities in conjunction with an annual standard of 12 μg/m^3; as with the base case, the lower end, but not the upper end, of these ranges decreases appreciably in conjunction with annual standards from 13 to 15 μg/m^3. Thus, in this case, as in the base case, changes in the 24-hour standard, while retaining the current annual standard, can result in generally larger but much more variable estimated reductions in risks associated with short-term exposures across the five cities than with changes in just the annual standard.

Further, in considering assumed hypothetical thresholds of 15 or 20 μg/m^3, staff observes that lowering the annual standard to alternative levels of 14, 13, and 12 μg/m^3 (without changing the 24-hour standard) results in estimated risk reductions of roughly 20 to 45 percent, 40 to 65

percent, and 60 to 90 percent, respectively, across all five example cities. In considering changes to the 24-hour and/or annual $PM_{2.5}$ standards in this case, staff notes that a 24-hour standard of 25 μg/m^3 results in estimates of reductions in short-term mortality ranging from approximately 60 to 100 percent (98th percentile form) and 70 to 100 percent (99th percentile form) across the five cities in conjunction with any annual standard in the range of 15 to 12 μg/m^3. A 24-hour standard of 30 μg/m^3 results in estimates of reductions in short-term mortality ranging from approximately 60 to 90 percent (98th percentile form) and 60 to 100 percent (99th percentile form) across the five cities in conjunction with an annual standard of 12 μg/m^3; similarly, the lower end, but not the upper end, of these ranges decreases appreciably in conjunction with annual standards from 13 to 15 μg/m^3. Thus, in this case as well, changes in the 24-hour standard, while retaining the current annual standard, can result in generally larger but much more variable estimated reductions in risks associated with short-term exposures across the five cities than with changes in just the annual standard.

5.3.5.3 Summary

In summary, in considering the relevant epidemiologic evidence, estimates of risk reductions associated with alternative annual and/or 24-hour standards, and the related limitations and uncertainties, staff concludes that there is clear support for considering revisions to the suite of current $PM_{2.5}$ standards to provide additional protection against health effects associated with short-term exposures. In looking specifically at the evidence of associations between short-term exposure to $PM_{2.5}$ and serious health effects, with a particular focus on mortality associations, staff concludes that it is appropriate to consider a revised 24-hour standard in the range of 30 to 25 μg/m^3 in conjunction with retaining the current annual standard level of 15 μg/m^3. Alternatively, staff also believes the evidence supports consideration of a revised annual standard, in the range of 13 to 12 μg/m^3, in conjunction with a revised 24-hour standard, to provide supplemental protection, in the range of 40 to 35 μg/m^3. In considering the results of the quantitative risk assessment, in the absence of evidence of clear thresholds, staff believes that it is appropriate to give significant weight to base case risk estimates, while also considering the implications of potential thresholds within the range of the air quality data from the relevant studies. In so doing, staff also finds support for considering a revised 24-hour

standard, in conjunction with retaining an annual standard level of 15 μg/m^3, in the range of 30 to 25 μg/m^3. Staff notes that a 24-hour standard at a level of 35 μg/m^3 is estimated to provide less than 30 percent reduction in mortality incidence in two or three of the five example cities (for a 99th or 98th percentile form, respectively), in either the base case or under an assumed hypothetical threshold of 10 μg/m^3, relative to that afforded by the current annual $PM_{2.5}$ standard alone. Further, staff finds little support based on the risk assessment for addressing short-term exposure effects solely with a revised annual standard in a range down to 12 μg/m^3. Staff believes that a suite of $PM_{2.5}$ standards selected from the alternatives identified above could provide an appropriate degree of protection against the mortality and morbidity effects associated with short-term exposure to $PM_{2.5}$ in studies in urban areas across the U.S..

5.3.6 Alternative Forms for Annual and 24-Hour $PM_{2.5}$ Standards

5.3.6.1 Form of Annual 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 the community-oriented monitoring site recording the highest level, or, if specific constraints are met, measurements from multiple community-oriented monitoring sites may be averaged (62 FR at 38,672). The constraints on allowing the use of spatially averaged measurements were intended to limit averaging across poorly correlated or widely disparate air quality values. 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 across multiple monitors in an area or were 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 annual standard levels, staff is also reconsidering the appropriateness of continuing to

allow spatial averaging across monitors as part of the form of an annual standard. There now exist much more $PM_{2.5}$ air quality data than were available in the last review. Consideration of the spatial variability across urban areas that is revealed by this new database (see Chapter 2, section 2.4 above, and the CD Chapter 3, section 3.2.5) raises questions as to whether an annual standard that allows for spatial averaging, within currently specified or alternative constraints, would provide appropriate public health protection. In conducting analyses to assess these questions, as discussed below, staff has taken into account both aggregate population risk across an entire urban area and the potential for disproportionate impacts on potentially vulnerable subpopulations within an area.

The effect of allowing the use of spatial averaging on aggregate population risk was considered as part of the sensitivity analyses included in the health risk assessment discussed in Chapter 4. In particular, a sensitivity analysis was done in several example urban areas (Detroit, Pittsburgh, and St. Louis) that compared estimated mortality risks (associated with both long- and short-term exposures) based on air quality values from the highest community-oriented monitor in an area with estimated risks based on air quality values averaged across all such monitors within the constraints allowed by the current standard. As discussed in Chapter 4, section 4.2.3, the monitored air quality values were used to determine the design value for the annual standard in each area, as applied to a "composite" monitor to reflect area-wide exposures. Changing the basis of the annual standard design value from the concentration at the highest monitor to the average concentration across all monitors reduces the air quality adjustment needed to just meet the current or alternative annual standards. As expected, the estimated risks remaining upon attainment of the current annual standard are greater when spatial averaging is used than when the highest monitor is used. Based on the results of this analysis in the three example cities, estimated mortality incidence associated with long-term exposure based on the use of spatial averaging is about 10 to over 40% higher than estimated incidence based on the use of the highest monitor. For estimated mortality incidence associated with short-term exposure, the use of spatial averaging results in risk estimates that range from about 5 to 25% higher. In considering estimated risks remaining upon attainment of alternative suites of annual and 24-hour $PM_{2.5}$ standards, spatial averaging only has an impact in those cases when the

annual standard is the "controlling" standard. For such cases in the three example cities, the estimated mortality incidence associated with long-term exposure in most cases ranges from about 10 to 60% higher when spatial averaging is used, and estimated mortality incidence associated with short-term exposure in most cases ranges from about 5 to 25% higher.

In considering the potential for disproportionate impacts on potentially vulnerable subpopulations, staff has assessed whether any such groups are more likely to live in census tracts in which the monitors recording the highest air quality values in an area are located. Data were obtained for demographic parameters measured at the census tract level, including education level, income level, and percent minority. These data from the census tract in which the highest air quality values were monitored were compared to area-wide average values (Schmidt et al., 2005). Recognizing the limitations of such cross-sectional analyses, staff observes that the results suggest that the highest concentrations in an area tend to be measured at monitors located in areas where the surrounding population is more likely to have lower education and income levels, and higher percentage minority levels. Staff notes that some epidemiologic study results, most notably the associations between mortality and long-term $PM_{2.5}$ exposure in the ACS cohort, have shown larger effect estimates in the cohort subgroup with lower education levels (CD, p. 8-103). As discussed in Chapter 3, section 3.4, people with lower socioeconomic status (e.g., lower education and income levels), or who have greater exposure to sources such as roadways, may have increased vulnerability to the effects of PM exposure. Combining evidence from health studies suggesting that people with lower socioeconomic status may be considered a population more vulnerable to PM-related effects with indications from air quality analyses showing that higher $PM_{2.5}$ concentrations are measured in local communities with lower socioeconomic status, staff finds that this is additional evidence which supports a change from spatial averaging across $PM_{2.5}$ monitors to provide appropriate protection from public health risks associated with exposure to ambient $PM_{2.5}$.

In considering whether alternative constraints on the use of spatial averaging may be appropriate to consider, staff has analyzed existing data on the correlations and differences between monitor pairs in metropolitan areas (Schmidt et al., 2005). For all pairs of $PM_{2.5}$ monitors, the median correlation coefficient based on annual air quality data is approximately

0.9; i.e., substantially higher than the current criterion for correlation of at least 0.6, which was met by nearly all monitor pairs. Similarly, the current criterion that differences in mean air quality values between monitors not exceed 20% was met for most monitor pairs, while the annual median and mean differences for all monitor pairs are 5% and 8%, respectively. This analysis also showed that in some areas with highly seasonal air quality patterns (e.g., due to seasonal woodsmoke emissions), substantially lower seasonal correlations and larger seasonal differences can occur relative to those observed on an annual basis. The spatial averaging requirements established in 1997 were intended to represent a relatively stable measure of air quality and to characterize area-wide $PM_{2.5}$ concentrations, while also precluding averaging across monitors that would leave a portion of a metropolitan area with substantially greater exposures than other areas (62 FR 38672). Based on the $PM_{2.5}$ air quality data now available, staff believes that the existing constraints on spatial averaging may not be adequate to achieve this result.

In considering the results of the analyses discussed above, staff concludes that it is appropriate to consider eliminating the provision that allows for spatial averaging from the form of an annual $PM_{2.5}$ standard. Further, staff concludes that if consideration is given to retaining an allowance for spatial averaging, more restrictive criteria should be considered. Staff believes that it would be appropriate to consider alternative criteria such as a correlation coefficient of at least 0.9, determined on a seasonal basis, with differences between monitor values not to exceed about 10%.

5.3.6.2 Form of 24-Hour Standard

In 1997 EPA established the form of the 24-hour $PM_{2.5}$ standard as the 98th 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.[4] 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

[4] 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.

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.

In this review, in conjunction with recommending that consideration be given to alternative 24-hour standard levels, staff is also considering the appropriateness of recommending that the current 98th percentile form, averaged over 3 years, be retained or revised. As an initial matter, staff believes that it is appropriate to retain a concentration-based form that is defined in terms of a specific percentile of the distribution of 24-hour $PM_{2.5}$ concentrations at each population-oriented monitor within an area, averaged over 3 years. Staff bases this recommendation on the same reasons that were the basis for EPA's selection of this type of form in the last review. As to the specific percentile value to be considered, staff has narrowed the focus of this review to the 98th and 99th percentile forms. This focus is based on the observation that the current 98th percentile form already allows the level of the standard to be exceeded seven days per year, on average (with every-day monitoring), while potentially allowing many more exceedance days in the worst year within the 3-year averaging period (Schmidt et al., 2005). As a result, in areas that just attain the standards, EPA's communication to the public through the Air Quality Index will on one hand indicate that the general level of air quality is satisfactory (since the standards are being met), but on the other hand it may identify many days throughout the year as being unhealthy, particularly for sensitive groups. Thus, staff does not believe it would be appropriate to consider specifying the form in terms of an even lower percentile value.

In considering differences between 98th and 99th percentile forms, staff believes it is appropriate to take into consideration the relative risk reduction afforded by these alternative forms at the same standard level. Based on the risk assessment results discussed in Chapter 4, and the risk reductions associated with alternative levels and forms discussed above in sections 5.3.4 and 5.3.5, staff notes that the 99th percentile can, in some instances, result in appreciably greater risk reductions in particular areas than that associated with a standard at the same level but with a 98th percentile form. More specifically, staff considered the differences in risk reductions associated with attaining alternative standards with 98th and 99th percentile forms in five example urban areas (Detroit, Los Angeles, Philadelphia, Pittsburgh, and St. Louis). In looking at estimated risk reductions associated with meeting a 24-hour standard of 30 $\mu g/m^3$, for example, estimated risk reductions for mortality associated with long-term exposures were higher with the use of a 99th percentile form in some areas by approximately 15%, ranging up to over 50% higher in Los Angeles. For estimated risk reductions for mortality associated with short-term exposures, the use of a 99th percentile form resulted in estimated reductions that were higher by less than 10% to over 30% across the five urban areas.

Staff also analyzed the available air quality data from 2001 to 2003 to compare the 98th and 99th percentile forms in terms of the numbers of days that would be expected to exceed the level of the standard (on average over 3 years and in the worst year within a 3-year averaging period) and by how much the standard would typically be exceeded on such days (Schmidt et al., 2005). In so doing, as noted above, staff observes that the current 98th percentile form allows the level of the standard to be exceeded seven days per year, on average (with every-day monitoring), and finds that this form allows up to about 20 days in the worst year within the 3-year averaging period. A 99th percentile form would allow the level of the standard to be exceeded three days per year, on average (with every-day monitoring), while allowing up to about 13 days in the worst year within the 3-year averaging period. Further, staff observes that for either form, daily peak concentrations in the upper 1 to 2% of the annual air quality distributions are within 5 $\mu g/m^3$ of the 98th or 99th percentile value somewhat more than half the

time and are almost always within 10 to 15 μg/m^3 above the 98th or 99th percentile values, with very few excursions above this range.[5]

Based on these considerations, staff recommends either retaining the 98th percentile form or revising it to be based on the 99th percentile air quality value. In selecting between these alternative forms, staff believes primary consideration should be given to the estimated level of risk reduction that is associated with standards based on either form. Staff also believes it is appropriate to take into account whether the 24-hour standard is set to supplement protection afforded by an annual standard, or is intended to be the primary basis for providing protection against effects associated with short-term exposures. In choosing between forms of alternative standards that provide generally equivalent levels of public health protection, staff believes it is appropriate to consider the implications from a public health communication perspective of the extent to which alternative forms allow different numbers of days in a year to be above the level of the standard in areas that attain the standard. In particular, staff notes that the use of a 99th percentile form would result in a more consistent public health message to the general public in the context of the wide-spread use of the Air Quality Index.

5.3.7 Summary of Staff Recommendations on Primary $PM_{2.5}$ NAAQS

Staff recommendations for the Administrator's consideration in making decisions on the primary $PM_{2.5}$ standards, together with supporting conclusions from sections 5.3.1 through 5.3.6, are briefly summarized below. Staff recognizes that selecting from among alternative standards will necessarily reflect consideration of the qualitative and quantitative uncertainties inherent in the relevant evidence and in the assumptions that underlie the quantitative risk assessment. In recommending these alternative suites of primary standards and ranges of levels for consideration, staff is mindful that the Act requires standards to be set that are requisite to

[5] This analysis also looked at the number of days in which the reported air quality values were "flagged" as being heavily influenced by natural events (including forest fires, dust storms) or exceptional events, for which the Agency's natural and exceptional events policies would likely apply. While flagged days generally account for less than 1% of all reported 24-hour average $PM_{2.5}$ concentrations, they account for about 40% of the highest 100 days across the country. In looking at the reported values that are above the 99th or 98th percentiles of the distribution of values, approximately 3 to 6% of the highest 2% of days (above the 98th percentile) were flagged, and approximately 5 to 10% of the highest 1% of days (above the 99th percentile) were flagged.

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.

(1) Consideration should be given to revising the current $PM_{2.5}$ primary standards to provide increased public health protection from the effects of both long- and short-term exposures to fine particles in the ambient air. This recommendation is based in general on the evaluation in the CD of the newly available epidemiologic, toxicologic, dosimetric, and exposure-related evidence, and more specifically on the evidence of mortality and morbidity effects in areas where the current standards were met, together with judgments as to the public health significance of the estimated incidence of effects upon just attaining the current standards.

(2) The indicator for fine particle standards should continue to be $PM_{2.5}$. This recommendation is based on the conclusion that the available evidence does not provide a sufficient basis for replacing or supplementing a mass-based fine particle indicator with an indicator for any specific fine particle component or subset of fine particles, nor does it provide a basis for excluding any components; on the evaluation in the CD of air quality within the intermodal particle size range of 1 to 3 μm; and on the policy judgment made in the last review to place regulatory importance on defining an indicator that would more completely capture fine particles under all conditions likely to be encountered across the U.S., while recognizing that some limited intrusion of small coarse particles will occur in some circumstances. Consideration should be given to modifying the FRM for $PM_{2.5}$ based on instrumentation and operational improvements that have been made since the $PM_{2.5}$ monitoring network was deployed in 1999, and to the adoption of FEMs for appropriate continuous measurement methods.

(3) Averaging times for $PM_{2.5}$ standards should continue to include annual and 24-hour averages to protect against health effects associated with short-term (hours to days) and

long-term (seasons to years) exposure periods; consideration of other averaging times, especially on the order of one or more hours, was limited by a lack of adequate information at this time.

(a) Consideration should be given to revising the form of the annual standard to one based on the highest community-oriented monitor in an area or, alternatively, to one with more constrained requirements for the use of spatial averaging across community-oriented monitors.

(b) Consideration should be given to revising the form of the 24-hour standard to a 99th percentile form or, alternatively, to retaining the 98th percentile form, based in part on considering the level of risk reduction likely to result from a standard using either form.

(4) Consideration should be given to alternative suites of $PM_{2.5}$ standards to provide protection against effects associated with both long- and short-term exposures, taking into account both evidence-based and risk-based considerations. Integrated recommendations on ranges of alternative suites of standards that, when considered together, protect against effects associated with both long- and short-term exposures include:

(a) Staff recommends consideration of an annual $PM_{2.5}$ standard at the current level of 15 $\mu g/m^3$ together with a revised 24-hour $PM_{2.5}$ standard in the range of 35 to 25 $\mu g/m^3$. Staff judges that such a suite of standards, particularly in conjunction with a 99th percentile form for a 24-hour standard set at the middle to upper end of this range, could provide an appropriate degree of protection against serious mortality and morbidity effects associated with long- and short-term exposures to fine particles.

(b) Alternatively, staff also recommends consideration of a revised annual $PM_{2.5}$ standard, within the range of 14 to 12 $\mu g/m^3$, together with a revised 24-hour $PM_{2.5}$ standard to provide supplemental protection against episodic localized or seasonal peaks, in the range of 40 to 35 $\mu g/m^3$. Staff judges that such a suite of

standards, particularly with an annual standard set at the middle or low end of this range, could provide an appropriate degree of protection against serious mortality and morbidity effects associated with long- and short-term exposures to fine particles.

5.4 THORACIC COARSE PARTICLE STANDARDS

5.4.1 Adequacy of Current PM_{10} Standards

In considering the adequacy of the current PM_{10} standards to control thoracic coarse particles, in conjunction with separate standards for $PM_{2.5}$, staff has first considered the appropriateness of using PM_{10} as an indicator for thoracic coarse particles. In 1997, in conjunction with establishing new $PM_{2.5}$ standards, EPA determined that the new function of PM_{10} standards was to protect against potential effects associated with thoracic coarse particles in the size range of 2.5 to 10 μm (62 FR 38,677). Although staff had given some consideration to a more narrowly defined indicator that did not include fine particles (e.g., $PM_{10-2.5}$), EPA decided to continue to use PM_{10} as the indicator for standards to control thoracic coarse particles. This decision was based in part on the recognition that the only studies of clear quantitative relevance to health effects most likely associated with thoracic coarse particles used PM_{10} in areas where the coarse fraction was the predominant component of PM_{10}, namely two fugitive dust studies in areas that substantially exceeded the PM_{10} standards (62 FR 38,679). Also, the decision reflected the fact that there was only very limited ambient air quality data then available specifically on thoracic coarse particles, in contrast to the extensive monitoring network already in place for PM_{10}. In essence, EPA concluded at that time that it was appropriate to continue to control thoracic coarse particles, which, like fine particles, are capable of penetrating to the thoracic region of the respiratory tract, but that the only information available upon which to base such standards was indexed in terms of PM_{10}.[6]

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[6] As discussed in Chapter 1, however, in subsequent litigation regarding the 1997 PM NAAQS revisions, the court held in part that PM_{10} is a "poorly matched indicator" for thoracic coarse 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 thoracic coarse particles, the court nonetheless vacated the 1997 revised PM_{10} standards for the control of thoracic coarse particles.

taking into account key uncertainties associated with the estimated risks. Estimates of risks attributable to short-term exposure to $PM_{10-2.5}$ are presented in Chapter 4 for Detroit, Seattle and St. Louis, the urban areas in which the studies discussed above were conducted. These estimated risks are attributable to $PM_{10-2.5}$ concentrations above background levels, or above the lowest measured levels in a given study if higher than background, so as to avoid extrapolating risk estimates beyond the range of air quality upon which the concentration-response functions were based.

In the absence of evidence for clear thresholds in any of the studies used in the risk assessment, the base case estimates in the analysis reflect the linear or near-linear concentration-response functions reported in the studies. To reflect the uncertainty as to whether thresholds may exist within the range of air quality observed in the studies, but may not be discernable with currently applied statistical methods, staff has also considered estimates of risk based on concentration-response functions modified to incorporate various assumed hypothetical threshold levels. Based on the sensitivity analyses conducted as part of the risk assessment, the uncertainty associated with alternative hypothetical thresholds had by far the greatest impact on estimated risks.

Table 5-5 summarizes the estimated $PM_{10-2.5}$-related annual incidence of hospital admissions and respiratory symptoms (cough) in children associated with short-term exposure for the base case and for alternative hypothetical thresholds in the three example urban areas included in the risk assessment. Staff observes that the base case estimates of cardiac-related hospital admissions in Detroit are an order of magnitude greater than asthma-related admissions in Seattle. Such large differences are in part attributable to the large differences in $PM_{10-2.5}$ air quality levels in these two areas, in which the 2003 annual average $PM_{10-2.5}$ concentration in Detroit (21.7 $\mu g/m^3$) is much higher than in Seattle (11.4 $\mu g/m^3$). Further, staff notes that the 2003 annual average $PM_{10-2.5}$ concentration in St. Louis (12.0 $\mu g/m^3$) is similarly far below that in Detroit. In looking beyond the base case estimates, staff observes that, as expected, the risk estimates are substantially smaller when concentration-response functions adjusted to reflect hypothetical thresholds are considered. At the largest assumed hypothetical threshold, estimates

effects of the crustal contribution in thoracic coarse particles. The CD notes that particles of crustal origin may be linked with morbidity effects, or may serve as carriers for other more toxic components, such as metals or organic compounds (CD, p. 9-63). The CD discusses some coarse particle components (e.g., metals, biogenic constituents) or sources contributing to coarse particles (e.g., wood burning) that may be linked with health effects, but little evidence is available on any of the components or sources within the coarse fraction at present (CD, p.9-32). 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 be eliminated from consideration.

Taking into account the above considerations, staff concludes that a mass-based indicator continues to be the most appropriate indicator for any thoracic coarse particle standards. Staff recommends that such an indicator retain 10 μm as the upper cut point, and that the lower cut point of 2.5 μm be used so as to most clearly differentiate between thoracic coarse ($PM_{10-2.5}$) and fine ($PM_{2.5}$) particles. In considering the evidence that suggests that high PM concentrations linked with dust storm events may be of less concern, staff notes that EPA has historically used natural events policies to address such issues in the implementation of PM standards.

In conjunction with considering $PM_{10-2.5}$ as an indicator for standards to address thoracic coarse particles, EPA is evaluating various ambient monitoring methods. This evaluation is being performed through field studies of commercially ready and prototype methods to characterize the measurement of thoracic coarse particles.[8] The $PM_{10-2.5}$ methods evaluation has resulted in characterizing the performance of multiple $PM_{10-2.5}$ measurement technologies under a variety of aerosol and meteorological conditions. This characterization has demonstrated that the majority of commercially available methods for the measurement of $PM_{10-2.5}$ have good precision and are well correlated with filter-based gravimetric methods such as the difference method that has primarily been used to date (i.e., operation of collocated PM_{10} and $PM_{2.5}$ low volume FRMs and calculating $PM_{10-2.5}$ by difference). EPA is working with the instrument manufacturers to address design issues that should reduce biases that have been observed among methods, in preparation for another field study examining the performance of the methods.

[8] This work is being done in consultation with the CASAC AAMM Subcommittee.

taking into account key uncertainties associated with the estimated risks. Estimates of risks attributable to short-term exposure to $PM_{10-2.5}$ are presented in Chapter 4 for Detroit, Seattle and St. Louis, the urban areas in which the studies discussed above were conducted. These estimated risks are attributable to $PM_{10-2.5}$ concentrations above background levels, or above the lowest measured levels in a given study if higher than background, so as to avoid extrapolating risk estimates beyond the range of air quality upon which the concentration-response functions were based.

In the absence of evidence for clear thresholds in any of the studies used in the risk assessment, the base case estimates in the analysis reflect the linear or near-linear concentration-response functions reported in the studies. To reflect the uncertainty as to whether thresholds may exist within the range of air quality observed in the studies, but may not be discernable with currently applied statistical methods, staff has also considered estimates of risk based on concentration-response functions modified to incorporate various assumed hypothetical threshold levels. Based on the sensitivity analyses conducted as part of the risk assessment, the uncertainty associated with alternative hypothetical thresholds had by far the greatest impact on estimated risks.

Table 5-5 summarizes the estimated $PM_{10-2.5}$-related annual incidence of hospital admissions and respiratory symptoms (cough) in children associated with short-term exposure for the base case and for alternative hypothetical thresholds in the three example urban areas included in the risk assessment. Staff observes that the base case estimates of cardiac-related hospital admissions in Detroit are an order of magnitude greater than asthma-related admissions in Seattle. Such large differences are in part attributable to the large differences in $PM_{10-2.5}$ air quality levels in these two areas, in which the 2003 annual average $PM_{10-2.5}$ concentration in Detroit (21.7 $\mu g/m^3$) is much higher than in Seattle (11.4 $\mu g/m^3$). Further, staff notes that the 2003 annual average $PM_{10-2.5}$ concentration in St. Louis (12.0 $\mu g/m^3$) is similarly far below that in Detroit. In looking beyond the base case estimates, staff observes that, as expected, the risk estimates are substantially smaller when concentration-response functions adjusted to reflect hypothetical thresholds are considered. At the largest assumed hypothetical threshold, estimates

in Detroit are 50 percent smaller than base case estimates, whereas in St. Louis estimates are 90 percent smaller.

Table 5-5 Estimated $PM_{10-2.5}$-related Annual Incidence of Hospital Admissions and Cough in Children with 2003 Air Quality in Areas that Meet the Current PM_{10} Standards (Base Case and Assumed Alternative Hypothetical Thresholds)

	Short-term Exposure			
	Base case Estimate, 95% CI	Assumed Hypothetical Thresholds		
		10 μg/m³	15 μg/m³	20 μg/m³
Detroit: hospital admissions for ischemic heart disease	654 169 to 1083	505 131 to 836	386 100 to 636	294 77 to 483
Seattle: hospital admissions for asthma (age <65)	27 0 to 65	11 0 to 26	4 0 to 10	1 0 to 3
St. Louis: days of cough in children	27,000 11,000 to 40,900	11,500 4,700 to 17,400	5,400 2,200 to 8,000	2,600 1,100 to 3,700

Beyond the specific health endpoints presented in Table 5-5, for which sensitivity analyses have been done, staff notes that hundreds of additional hospital admissions for other cardiac- and respiratory-related diseases are also estimated in Detroit, based on risk assessment results presented in Chapter 4, as are thousands of additional days in which children are likely to experience other symptoms of the lower respiratory tract in St. Louis. In considering these limited estimates, even when hypothetical thresholds are assumed, staff concludes that they are indicative of risks that can reasonably be judged to be important from a public health perspective, especially in areas in which $PM_{10-2.5}$ concentrations approach those observed in Detroit.

In considering the evidence and risk estimates for thoracic coarse particles discussed above, and the related limitations and uncertainties, staff concludes that this information is sufficient to support consideration of revised standards for thoracic coarse particles to afford

protection from effects related to short-term exposure to current ambient levels of $PM_{10-2.5}$ in some urban areas. Staff conclusions and recommendations on an indicator and associated monitoring methods, averaging times, and alternative levels and forms for thoracic coarse particle standards that would afford an appropriate degree of protection from such effects are discussed in the following sections.

5.4.2 Indicators

Section 5.4.1 above discusses EPA's decision in 1997 to continue to use PM_{10} as the indicator for standards intended to protect against the effects most likely associated with thoracic coarse particles. In considering the adequacy of such standards, staff has taken into account information now available on health effects and air quality in which thoracic coarse particles are indexed by $PM_{10-2.5}$, concluding that such information should form the basis for consideration of standards for thoracic coarse particles using an indicator that does not include the fine fraction of PM_{10}.

The CD concludes that the recent scientific information supports EPA's previous decision to use an indicator based on PM mass, as discussed above in section 5.3.2 for fine particles. In addition, currently available information from dosimetric studies supports retaining 10 μm as the appropriate cut point for particles capable of penetrating to the thoracic regions of the lung. In conjunction with $PM_{2.5}$ standards, an appropriate mass-based indicator for thoracic coarse particles thus would be $PM_{10-2.5}$. As noted above, this is the indicator that has been used to index thoracic coarse particles in newly available epidemiologic studies and in characterizations of air quality.

There is limited evidence to support consideration of other indicators for thoracic coarse particles, such as individual components within this PM fraction. In general, less is known about the composition of thoracic 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. While several studies have suggested that the crustral or geological component of fine particles is not significantly associated with mortality (CD, p. 8-66), no studies have focused on potential

effects of the crustal contribution in thoracic coarse particles. The CD notes that particles of crustal origin may be linked with morbidity effects, or may serve as carriers for other more toxic components, such as metals or organic compounds (CD, p. 9-63). The CD discusses some coarse particle components (e.g., metals, biogenic constituents) or sources contributing to coarse particles (e.g., wood burning) that may be linked with health effects, but little evidence is available on any of the components or sources within the coarse fraction at present (CD, p.9-32). 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 be eliminated from consideration.

Taking into account the above considerations, staff concludes that a mass-based indicator continues to be the most appropriate indicator for any thoracic coarse particle standards. Staff recommends that such an indicator retain 10 μm as the upper cut point, and that the lower cut point of 2.5 μm be used so as to most clearly differentiate between thoracic coarse ($PM_{10-2.5}$) and fine ($PM_{2.5}$) particles. In considering the evidence that suggests that high PM concentrations linked with dust storm events may be of less concern, staff notes that EPA has historically used natural events policies to address such issues in the implementation of PM standards.

In conjunction with considering $PM_{10-2.5}$ as an indicator for standards to address thoracic coarse particles, EPA is evaluating various ambient monitoring methods. This evaluation is being performed through field studies of commercially ready and prototype methods to characterize the measurement of thoracic coarse particles.[8] The $PM_{10-2.5}$ methods evaluation has resulted in characterizing the performance of multiple $PM_{10-2.5}$ measurement technologies under a variety of aerosol and meteorological conditions. This characterization has demonstrated that the majority of commercially available methods for the measurement of $PM_{10-2.5}$ have good precision and are well correlated with filter-based gravimetric methods such as the difference method that has primarily been used to date (i.e., operation of collocated PM_{10} and $PM_{2.5}$ low volume FRMs and calculating $PM_{10-2.5}$ by difference). EPA is working with the instrument manufacturers to address design issues that should reduce biases that have been observed among methods, in preparation for another field study examining the performance of the methods.

[8] This work is being done in consultation with the CASAC AAMM Subcommittee.

EPA has begun the process of examining data quality objectives for potential $PM_{10-2.5}$ standards. On the basis of preliminary analyses, it is apparent that greater sampling frequency will be important due to the high variability of $PM_{10-2.5}$ in the atmosphere; this would be particularly important for a short-term $PM_{10-2.5}$ standard. Due to the resource intensive nature of filter sampling on a daily basis, staff believes that it will be critical to include continuous monitoring in any network deployment strategy for a possible $PM_{10-2.5}$ standard. In addition to providing high temporal resolution to $PM_{10-2.5}$ data, continuous monitors would also support public reporting of $PM_{10-2.5}$ episodes and inclusion of $PM_{10-2.5}$ in an air quality forecasting program. As noted above and elsewhere in this document, $PM_{10-2.5}$ is more highly variable in the atmosphere than $PM_{2.5}$, such that spatial robustness will be a particularly important consideration in monitoring network design.

5.4.3 Averaging Times

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 thoracic coarse particles (62 FR at 38,677-79). This decision was based in part on qualitative considerations related to the expectation that deposition of thoracic coarse 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 thoracic coarse particles including fugitive dust. Further, potential build-up of insoluble thoracic coarse particles in the lung after long-term exposures to high levels was also considered plausible.

Information available in this review on thoracic coarse particles, while still limited, represents a significant expansion of the evidence available in the last review. As discussed above in section 5.4.1, a number of epidemiologic studies are now available that report statistically significant associations between short-term (24-hour) exposure to $PM_{10-2.5}$ and morbidity effects, which the CD concludes are suggestive of causal associations, and mortality, which the CD concludes provide less support for possible causal associations. With regard to

long-term exposure studies, while one recent study reported a link between reduced lung function growth and long-term exposure to $PM_{10-2.5}$ and $PM_{2.5}$, the CD concludes that the evidence is not sufficient to be suggestive of a causal association. Staff notes that no evidence is available to suggest associations between $PM_{10-2.5}$ and very short exposure periods of one or more hours.

Based on these considerations, staff concludes that the newly available evidence provides support for considering a 24-hour standard for control of thoracic coarse particles, based primarily on evidence suggestive of associations between short-term exposure and morbidity effects, reflecting as well the potential for associations with mortality. Noting the absence of evidence judged to be even suggestive of an association with long-term exposures, staff concludes that there is little support for an annual standard, although staff recognizes that it may be appropriate to consider an annual standard to provide a margin of safety against possible effects related to long-term exposure to thoracic coarse particles that future research may reveal. Staff observes, however, that a 24-hour standard that would reduce 24-hour exposures would also likely reduce long-term average exposures, thus providing some margin of safety against the possibility of health effects associations with long-term exposures.

5.4.4 Alternative $PM_{10-2.5}$ Standards to Address Health Effects Related to Short-term Exposure

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 of 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 thoracic coarse particles. Because of the very limited nature of this evidence, staff concluded that while it supported retention of a standard to control thoracic coarse 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 5.3.6. In making this change, EPA selected a 99th percentile form,[9] 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.

Since the last review, as discussed above in section 5.4.1, new evidence specific to $PM_{10-2.5}$ has become available that suggests associations between short-term $PM_{10-2.5}$ concentrations and morbidity effects and, to a lesser degree, mortality. In considering this evidence as a basis for setting a 24-hour $PM_{10-2.5}$ standard, staff has focused on U.S. and Canadian short-term exposure studies of thoracic coarse particles (Appendix 3A). In so doing, staff has taken into account reanalyses that addressed GAM-related statistical issues and has considered the extent to which the studies report statistically significant and relatively precise relative risk estimates; the reported associations are robust to co-pollutant confounding and alternative modeling approaches; and the studies used relatively reliable air quality data. In particular, staff has focused first on those specific morbidity studies that provide evidence of associations in areas that would have met the current PM_{10} standards during the time of the study.

As an initial matter, staff recognizes, as discussed in Chapter 3 (section 3.6.6), that these short-term exposure studies provide no evidence of clear thresholds, or lowest-observed-effects levels, in terms of 24-hour average concentrations. Staff notes that in the one study that explored a potential $PM_{10-2.5}$ threshold, conducted in Phoenix, no evidence of a threshold was observed for $PM_{10-2.5}$, even though that study provided some suggestion of a potential threshold for $PM_{2.5}$. The CD concludes that while there is no evidence of a clear threshold within the range of air quality observed in the studies, for some health endpoints (such as total nonaccidental mortality) it is likely to be extremely difficult to detect threshold levels (CD, p.9-45). As a consequence, this body of evidence is difficult to translate directly into a specific 24-hour standard that would protect against all effects associated with short-term exposures. Staff notes that the distributions

[9] As noted above, the court vacated the 1997 24-hour PM_{10} standard that had been revised to incorporate a 99th percentile form.

of daily $PM_{10-2.5}$ concentrations in these studies often extend down to or below background levels, such that the likely range of background concentrations across the U.S., as discussed in Chapter 2, section 2.6, could be a relevant consideration in this policy evaluation. Staff recognizes, however, that there is insufficient data to estimate daily distributions of background $PM_{10-2.5}$ levels (as was done for background $PM_{2.5}$ levels, as discussed in Chapter 2, section 2.6).

Being mindful of the difficulties posed by uncertainties related to potential thresholds and insufficient data to characterize daily distributions of $PM_{10-2.5}$ background concentrations, as well as the limited nature of the available evidence, staff has considered the short-term exposure epidemiologic evidence as a basis for alternative 24-hour $PM_{10-2.5}$ standards. In so doing, staff has focused on the upper end of the distributions of daily $PM_{10-2.5}$ concentrations, particularly in terms of the 98th and 99th percentile values, consistent with the forms considered in section 5.3.6 above for $PM_{2.5}$. In looking at the specific morbidity studies identified in section 5.4.1 that report statistically significant associations with respiratory- and cardiac-related hospital admissions in areas that had ambient air quality levels that would have met the current PM_{10} standards at the time of the study, including studies in Toronto (Burnett et al., 1997), Seattle (Sheppard et al.,1999, 2003), and Detroit (Lippmann et al., 2000; Ito, 2003), staff notes that the reported 98th percentile values range from approximately 30 to 36 $\mu g/m^3$ in all three areas, and the 99th percentile values range from 36 to 40 $\mu g/m^3$ (Ross and Langstaff, 2005).

In looking more closely at these studies, staff recognizes that the uncertainty related to exposure measurement error is potentially quite large in epidemiologic studies linking effects to $PM_{10-2.5}$ air quality measures. For example, in looking specifically at the Detroit study, staff notes that the $PM_{10-2.5}$ air quality values were based on air quality monitors located in Windsor, Canada. The study authors determined that the air quality values from these monitors were generally well correlated with air quality values monitored in Detroit, where the hospital admissions data were gathered, and, thus concluded that these monitors were appropriate for use in exploring the association between air quality and hospital admissions in Detroit. Staff has observed, however, that the $PM_{10-2.5}$ levels reported in this study are significantly lower than the $PM_{10-2.5}$ levels measured at some of the Detroit monitors in 2003 -- an annual mean level of 13.3 $\mu g/m^3$ is reported in the study, based on 1992 to 1994 data, as compared to an average annual

mean level of 21.7 μg/m^3 measured at two urban-center monitors in 2003 (which is used as the basis for the risk assessment presented in Chapter 4). This observation prompted staff to further explore the comparison between $PM_{10-2.5}$ levels monitored at Detroit and Windsor sites. This exploration has shown that in recent years, based on available Windsor and Detroit data from 1999 to 2003, the Windsor monitors used in this study typically have recorded $PM_{10-2.5}$ levels that are generally less than half the levels recorded at urban-center Detroit monitors, though the concentrations measured in Windsor are more similar to concentrations reported for suburban areas well outside the city (Ross and Langstaff, 2005). These observations lead staff to conclude that the statistically significant, generally robust association with hospital admissions in Detroit reflects population exposures that may be appreciably higher than what would be estimated using data from the Windsor monitors. Taking these observations into account, staff nonetheless believes that these studies in general, and the Detroit study in particular, do provide evidence of associations between short-term exposures to $PM_{10-2.5}$ and hospital admissions. Staff does conclude, however, that the association observed in the Detroit study, which staff judges to be the strongest of these studies, likely reflects exposure levels potentially much higher in the central city area than those reported in that study. Based on this information, staff believes that alternative 24-hour $PM_{10-2.5}$ standards appropriate for consideration in this review need not necessarily extend to levels down to or below the ranges reported in these studies in order to provide protection from the morbidity effects related to short-term exposures to $PM_{10-2.5}$.

Staff has also looked at the evidence from U.S. and Canadian studies that report statistically significant and generally robust associations with mortality and short-term exposures to $PM_{10-2.5}$. As discussed in section 9.2.3 of the CD, the evidence associating mortality with short-term exposures to $PM_{10-2.5}$ is too uncertain to infer a likely causal relationship, although it is suggestive of a possible causal relationship. Staff identified two such studies, conducted in Phoenix (Mar et al., 2000, 2003) and Coachella Valley, CA (Ostro et al., 2000, 2003), that report 98th percentile $PM_{10-2.5}$ values of approximately 70 and 107 μg/m^3, and 99th percentile values of 75 and 134 μg/m^3, respectively. Staff notes that these studies were conducted in areas with air quality levels that would not have met the current PM_{10} standards. A staff analysis of PM_{10} and estimated $PM_{10-2.5}$ concentrations from the AQS database for 2001 to 2003 suggests that 98th

Table 5-6(b). Predicted percent of counties with monitors (and percentage of population in counties with monitors) not likely to meet alternative 24-hour (99th percentile form) $PM_{10-2.5}$ standards or current PM_{10} standards

Alternative Standards and Levels (µg/m³)	Percent of counties (and percent population) not likely to meet stated standards and levels*								
	Total counties (population)	Northeast	Southeast	Industrial Midwest	Upper Midwest	Southwest	Northwest	Southern CA	Outside Regions**
No. of counties with $PM_{10-2.5}$ monitors (Population, in thousands)	382 (150,595)	57	82	73	33	20	88	15	14
24-hour $PM_{10-2.5}$ standards:									
100	6 (13)	4	2	0	3	40	1	40	14
95	7 (13)	4	2	3	3	45	3	40	14
90	9 (14)	5	2	4	6	50	6	40	14
85	12 (20)	5	4	7	12	55	11	40	14
80	13 (22)	5	4	8	15	60	13	40	14
75	14 (24)	5	6	10	15	60	13	53	21
70	16 (26)	9	9	10	21	60	14	60	21
65	21 (32)	11	10	14	33	65	17	60	50
60	24 (38)	12	11	16	33	70	23	67	64
55	29 (44)	12	12	18	48	70	33	73	71
50	34 (47)	18	17	23	52	70	40	73	79
45	41 (53)	18	24	27	58	80	51	87	86
40	45 (56)	21	24	34	70	80	55	87	93
35	53 (67)	32	34	45	79	80	64	93	93
30	62 (72)	42	45	56	85	85	73	93	93
25	75 (82)	56	66	68	94	90	85	100	93
No. of counties with PM_{10} monitors (Population, in thousands)	*585 (170,157)*								
PM_{10} annual and 24-hour standards:									
50/150	8 (11)								

* Based on 2001-2003 data for sites with 4, 8, or 12 consecutive quarters with at least 11 samples per quarter. As such, these estimates are not based on the same 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.

Beyond looking directly at the relevant epidemiologic evidence, staff has also considered the extent to which the $PM_{10-2.5}$ risk assessment results discussed in Chapter 4 can help inform consideration of alternative 24-hour $PM_{10-2.5}$ standards. While one of the goals of the $PM_{10-2.5}$ risk assessment was to provide estimates of the risk reductions associated with just meeting alternative $PM_{10-2.5}$ standards, staff has concluded that the nature and magnitude of the uncertainties and concerns associated with this portion of the risk assessment weigh against use of these risk estimates as a basis for recommending specific standard levels. These uncertainties and concerns include, but are not limited to the following:

- as discussed above, concerns that the current $PM_{10-2.5}$ levels measured at ambient monitoring sites in recent years may be quite different from the levels used to characterize exposure in the original epidemiologic studies based on monitoring sites in different location, thus possibly over- or underestimating population risk levels;

- greater uncertainty about the reasonableness of the use of proportional rollback to simulate attainment of alternative $PM_{10-2.5}$ daily standards in any urban area due to the limited availability of $PM_{10-2.5}$ air quality data over time;

- concerns that the locations used in the risk assessment are not representative of urban areas in the U.S. that experience the most significant 24-hour peak $PM_{10-2.5}$ concentrations, and thus, observations about relative risk reductions associated with alternative standards may not be relevant to the areas expected to have the greatest health risks associated with elevated ambient $PM_{10-2.5}$ levels; and

- concerns about the much smaller health effects database that supplies the C-R relationships used in the risk assessment, compared to that available for $PM_{2.5}$, which limits our ability to evaluate the robustness of the risk estimates for the same health endpoints across different locations.

In summary, in considering the relevant epidemiologic evidence and the related limitations and uncertainties, staff concludes that there is support for considering a 24-hour $PM_{10-2.5}$ standard to replace the current PM_{10} standards to provide protection against health effects associated with short-term exposures to thoracic coarse particles. In looking primarily at the evidence of associations between short-term exposure to $PM_{10-2.5}$ and mortality, staff concludes that it is appropriate to consider a 24-hour standard in the range of 65 to 75 $\mu g/m^3$, with a 98th percentile form, or in the range of 75 to 85 $\mu g/m^3$, with a 99th percentile form. A

standard set within either of these ranges could be expected to provide a margin of safety to protect against the potential, but uncertain, mortality effects of $PM_{10-2.5}$, while continuing to provide protection against the effects of $PM_{10-2.5}$ associated with high levels of PM_{10} that were the basis for the decision made by EPA in 1997 to retain the levels of the PM_{10} standards. In addition, staff observes that several epidemiologic studies have reported associations with morbidity effects in areas with lower $PM_{10-2.5}$ that could support consideration of standard levels as low as approximately 30 $\mu g/m^3$, 98th percentile, or 35 $\mu g/m^3$, 99th percentile.

Staff recognizes, however, that the epidemiologic evidence on morbidity and mortality effects related to $PM_{10-2.5}$ exposure is very limited at this time. A key area of uncertainty in this evidence is the potentially quite large uncertainty related to exposure measurement error for $PM_{10-2.5}$, as compared with fine particles. $PM_{10-2.5}$ concentrations can vary substantially across a metropolitan area and thoracic coarse particles are less able to penetrate into buildings than fine particles; thus, the ambient concentrations reported in epidemiologic studies may not well represent area-wide population exposure levels. Other key uncertainties include the lack of information on the composition of thoracic coarse particles and the effects of thoracic coarse particles from various sources, and the lack of evidence on potential mechanisms for effects of thoracic coarse particles. Staff believes that taking these uncertainties into account leads to consideration of standard levels toward the upper end of the ranges identified above.

5.4.5 Summary of Staff Recommendations on Primary $PM_{10-2.5}$ NAAQS

Staff recommendations for the Administrator's consideration in making decisions on standards for thoracic coarse particles, together with supporting conclusions from sections 5.4.1 through 5.4.4, are briefly summarized below. In making these recommendations, 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.

(1) The current primary PM_{10} standards should be revised in part by replacing the PM_{10} indicator with an indicator of thoracic coarse particles that does not include fine particles. Any such revised standards should be based on available health effects evidence and air quality data generally indexed by $PM_{10-2.5}$, to provide public health protection more specifically directed toward effects related to exposure to thoracic coarse particles in the ambient air.

(2) The indicator for a thoracic coarse particle standard should be $PM_{10-2.5}$, consistent with the recommendation made in section 5.3.7 to retain $PM_{2.5}$ as the indicator for fine particle standards.

(a) As noted above, this recommendation is based primarily on the evaluation in the CD of air quality within the intermodal particle size range of 1 to 3 μm and the policy judgment made in the last review to place regulatory importance on defining an indicator that would more completely capture fine particles under all conditions likely to be encountered across the U.S., while recognizing that some limited intrusion of small coarse particles will occur in some circumstances.

(b) In support of this recommendation, work should continue on the development of an FRM for $PM_{10-2.5}$ based on the ongoing field program to evaluate various types of monitors, and consideration should be given to the adoption of FEMs for appropriate continuous measurement methods.

(3) A 24-hour averaging time should be retained for a $PM_{10-2.5}$ standard to protect against health effects associated with short-term exposure periods, with consideration given to the use of either a 98th or 99th percentile form. Consideration could also be given to retaining an annual averaging time, in considering the appropriate margin of safety against possible health effects that might be associated with long-term exposure periods.

(4) Consideration should be given to setting a 24-hour $PM_{10-2.5}$ standard about as protective as the current daily PM_{10} standard, with a level in the range of approximately 65 to 75

$\mu g/m^3$, 98th percentile, or approximately 75 to 85 $\mu g/m^3$, 99th percentile. Staff also believes there is some support for consideration of a $PM_{10-2.5}$ standard level down to approximately 30 $\mu g/m^3$, 98th percentile, or 35 $\mu g/m^3$, 99th percentile, while recognizing that a standard set at such a relatively low level would place a great deal of weight on very limited and uncertain epidemiologic associations. Consideration of $PM_{10-2.5}$ standards within the ranges recommended above, and design considerations for an associated $PM_{10-2.5}$ monitoring network, should take into account the especially large variability seen in currently available information on ambient concentrations and composition of $PM_{10-2.5}$.

5.5 SUMMARY OF KEY UNCERTAINTIES AND RESEARCH RECOMMENDATIONS RELATED TO SETTING PRIMARY PM STANDARDS

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 health-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 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 CD. Still, the 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 thoracic coarse particles has been expanded, but the uncertainties regarding thoracic coarse particles are still much greater than those for fine particles. As discussed in Chapter 2, the spatial variability of thoracic coarse particles is generally greater than that for fine particles, which will increase uncertainty in the associations between health effects and thoracic coarse particles measured at central site monitors. Additional exposure research is needed to understand the influence of measurement error and exposure error on thoracic coarse 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 thoracic coarse particles, but it is possible that there are components of thoracic coarse 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 $PM_{2.5}$-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 evidence for a clear threshold level in relationships between various PM indicators and mortality, within the range of concentrations observed in the studies, though some studies have suggested potential levels.

(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 co-pollutants, 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 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.

(6) Selection of appropriate averaging times for PM air quality standards is important for public health protection, and available information suggests that some effects, including cardiac-related risk factors, 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 thoracic 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.

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6. POLICY-RELEVANT ASSESSMENT OF PM-RELATED WELFARE EFFECTS

6.1 INTRODUCTION

This chapter assesses key policy-relevant information on the known and potential effects on public welfare 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 CD and other significant reports referenced therein. The welfare effects to be considered in this review of the secondary PM NAAQS include effects on visibility (section 6.2), vegetation and ecosystems (section 6.3), materials (section 6.4), and climate change processes[1] (section 6.5). For each category of effects, this chapter presents a 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. Staff conclusions and recommendations related to secondary standards for PM are presented in Chapter 7.

It is important to note that discussion of PM-related effects on visibility, vegetation and ecosystems, and climate change processes in Chapters 4 and 9 of the CD builds upon and includes by reference extensive information from several other significant scientific reviews of these topics. 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).

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[1] In assessing information on PM-related effects on climate change processes, consideration is given to potential indirect impacts on human health and the environment that may be a consequence of changes in climate and solar radiation attributable to changes in ambient PM.

6.2 EFFECTS ON VISIBILITY

Visibility can be defined as the degree to which the atmosphere is transparent to visible light (NRC, 1993; CD, 4-153). Visibility impairment is the most noticeable effect of fine particles present in the atmosphere. Particle 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.

This section discusses the role of ambient PM in the impairment of visibility, drawing upon the most relevant information contained in the CD (Chapters 4 and 9), as well as significant reports on the science of visibility referenced therein, and building upon information presented in section 2.8 of this document. In particular, this section includes new information on the following topics:

- Summary findings of analyses of hourly $PM_{2.5}$ measurements and reconstructed light extinction coefficients for urban areas, for 2003, that demonstrate a significant correlation between $PM_{2.5}$ and light extinction across the U.S. during daylight hours.
- An overview of 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 visual air quality, as demonstrated by efforts to improve visibility in national parks and wilderness areas, as well as in urban areas.

This section summarizes available information as follows: (1) information on the general types of visibility impairment; (2) trends and conditions in Class I and non-urban areas; (3) visibility conditions in urban areas; (4) studies of the economic value of improving visual air quality; (5) current policy approaches to addressing visibility impairment; and (6) approaches to evaluating public perceptions of visibility impairment and judgments about the acceptability of varying degrees of visibility impairment.

6.2.1 Overview of Visibility Impairment

Visibility effects are manifested in two principal ways: as local impairment (e.g., localized hazes and plumes) and as regional haze. This distinction is significant because this difference impacts both how visibility goals may be set and how air quality management strategies may be devised.

Local-scale visibility degradation commonly occurs either in the form of 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." Visibility impairment caused by a specific source or small group of sources has been generally termed "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 Act) as being "reasonably attributable" to a particular source.[2]

The second type of impairment, regional haze, results from pollutant emissions from a multitude of sources located across a broad geographic region. Regional haze impairs visibility in every direction over a large area, in some cases over multi-state regions. It also 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, regional haze, that is principally responsible for impairment in national parks and wilderness areas across the country (NRC, 1993).

While visibility impairment in urban areas at times may be dominated by local sources, it 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.

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[2]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.

6.2.2 Visibility Trends and Current Conditions in Class I and Non-Urban Areas

In conjunction with the National Park Service, other Federal land managers, and State organizations, EPA has supported visibility monitoring in national parks and wilderness areas since 1988. The monitoring 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).

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, with adjustment for the relative humidity. 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 light extinction levels are reasonable in establishing current visibility conditions. Aerosol-derived light extinction is used to document spatial and temporal trends and to determine how proposed changes in atmospheric constituents would affect future visibility conditions.

Annual average visibility conditions (reflecting light extinction due to both 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, for 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 (CD, p 4-179).

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. 4-182; 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 light extinction in the summer exceeds the annual average by 40% (Sisler et al., 1996).

Regional trends in Class I area visibility are updated and presented 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 a 16% improvement. However, visibility in the East remains significantly impaired, with an average visual range of approximately 20 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 approximately 100 km.

6.2.3 Visibility Conditions in Urban Areas

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. 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.

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 $PM_{2.5}$ and, thus, higher visibility impairment than monitored Class I areas. As discussed in Chapter 2, sections 2.4 and 2.5, annual mean levels of 24-hour average $PM_{2.5}$ levels are generally higher in urban areas 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.

6.2.3.1 ASOS Airport Visibility Monitoring Network

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., 2001).

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. 4-174). 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 these truncated data are not ideal 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 for a subset of the ASOS sites.[3]

6.2.3.2 Correlation between Urban Visibility and $PM_{2.5}$ Mass

In an effort to better characterize urban visibility, staff has analyzed the extensive new data now available on $PM_{2.5}$ primarily in urban areas. This rapidly expanding national database, including FRM measurements of $PM_{2.5}$ mass, continuous measurements of hourly $PM_{2.5}$ mass, and $PM_{2.5}$ chemical speciation measurements, has now provided the opportunity to conduct such an analysis. In this analysis, described below and documented in detail in Schmidt et al. (2005), staff has sought to explore the factors that have historically complicated efforts to address visibility impairment nationally, including regional differences related to levels of primarily fine particles and relative humidity. Taking these factors into account, staff has compared correlations between visibility, in terms of reconstructed light extinction (using the IMPROVE methodology discussed in Chapter 2, section 2.8), with hourly $PM_{2.5}$ concentrations in urban areas across the U.S. and in eastern and western regions.

As an initial matter, staff has explored the factors contributing to the substantial East/West differences that have been characterized primarily for Class I areas across the country, as discussed above in section 6.2.2. In considering fine particle levels, staff notes that East/West differences are substantially smaller in urban areas than in rural areas. As shown in Figure 6-1, 24-hour average $PM_{2.5}$ concentrations in urban areas in the East and West are much more similar than in rural areas. A significantly lower East/West ratio is observed in urban areas, based on data from either the FRM or the EPA Speciation Network, than in rural areas, based on data from the IMPROVE network.

In considering relative humidity levels, staff notes that, while the average daily relative humidity levels are generally higher in eastern than western areas, in both regions relative humidity levels are appreciably lower during daylight as compared to night time hours. These

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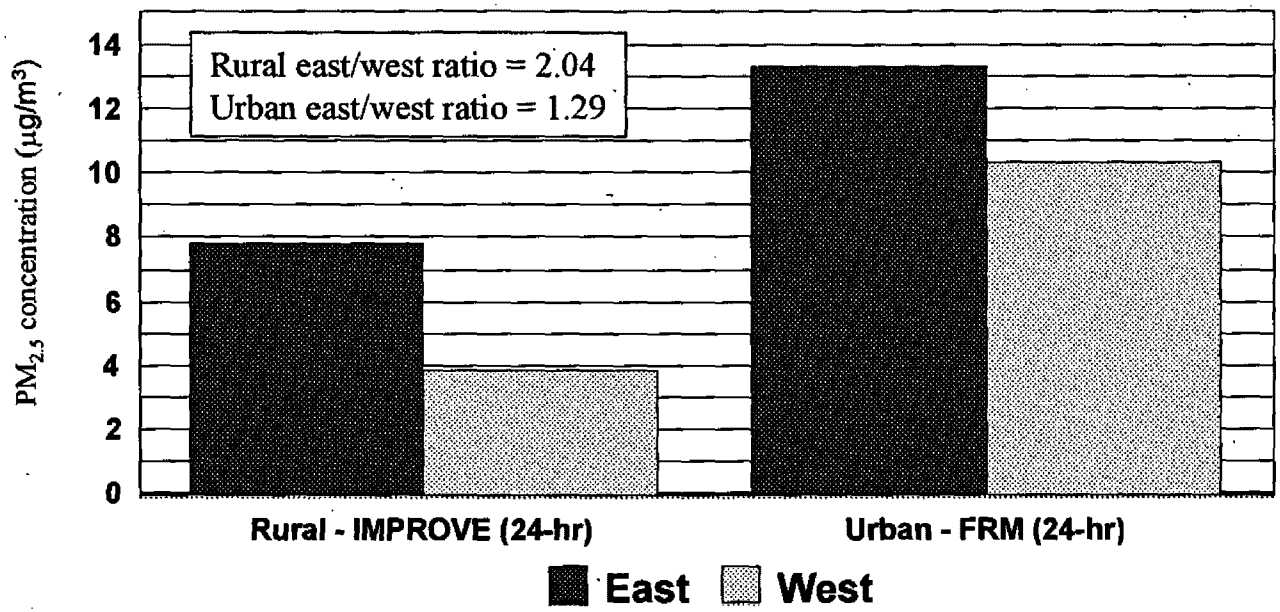
[3] A preliminary analysis of the archived data for 63 cities across the U.S. was presented in the first draft Staff Paper (August 2003), but further analysis has not been conducted. While the preliminary analysis demonstrated relatively well-characterized correlations between predicted $PM_{2.5}$ concentrations (based on ASOS extinction values) and measured $PM_{2.5}$ concentrations in some urban areas, such correlations were not consistently observed in urban areas across the country.



Note: Urban IMPROVE sites and rural FRM sites excluded.

Figure 6-1. $PM_{2.5}$ concentration differences between eastern and western areas and between rural and urban areas for 2003.

Source: Schmidt et al. (2005)

differences can be seen in Figure 6-2, based on data from National Weather Service (NWS) sites. As discussed in Chapter 2, section 2.8, the reconstructed light extinction coefficient, for a given mass and concentration, increases sharply as relative humidity rises. Thus, visibility impacts related to East/West differences in average relative humidity are minimized during daylight hours, when relative humidity is generally lower.

Taking these factors into account, staff has considered both 24-hour and shorter-term daylight hour averaging periods in evaluating correlations between $PM_{2.5}$ concentrations in urban areas and visibility, in terms of reconstructed light extinction (RE), in eastern and western areas, as well as nationwide. Figure 6-3 shows clear and similarly strong correlations between RE and 24-hour average $PM_{2.5}$ in eastern, western, and all urban areas. Figure 6-3 is based on data from 161 urban continuous $PM_{2.5}$ mass monitoring sites across the country with co-located or nearby 24-hour $PM_{2.5}$ speciation data. RE values were calculated based on a constructed hourly speciated $PM_{2.5}$ data set, hourly relative humidity data (either co-located or from nearby NWS sites), and a coarse PM data set (estimated either by difference method from the continuous $PM_{2.5}$ and co-located continuous PM_{10} instruments, or based on regional ratios of PM fractions) (Schmidt et al., 2005). In calculating RE, the relative humidity was capped at 95%, reflecting the lack of accuracy in higher relative humidity values and their highly disproportionate impact on RE.

For these analyses, staff has considered both 10 years of relative humidity data, converted to 10-year average hourly *f(RH)*[4] values (Figure 6-3, panel a), as well as actual hourly relative humidity data for 2003, converted to *f(RH)* values (Figure 6-3, panel b). Staff recognizes that 10-year average hourly *f(RH)* data are more reflective of long-term humidity patterns, and may provide a more appropriate basis for relating ambient $PM_{2.5}$ levels to visibility impairment in the context of consideration of a potential secondary standard to protect against PM-related visibility impairment. On the other hand, since there can be significant day-to-day variance in relative humidity that is not reflected in long-term average *f(RH)* data, actual hourly

[4] *f(RH)* is the relative humidity adjustment factor; it increases significantly with higher humidity. See section 2.8.1 and Chapter 4 of the CD (CD, pp. 4-149 to 4-170) for further information.

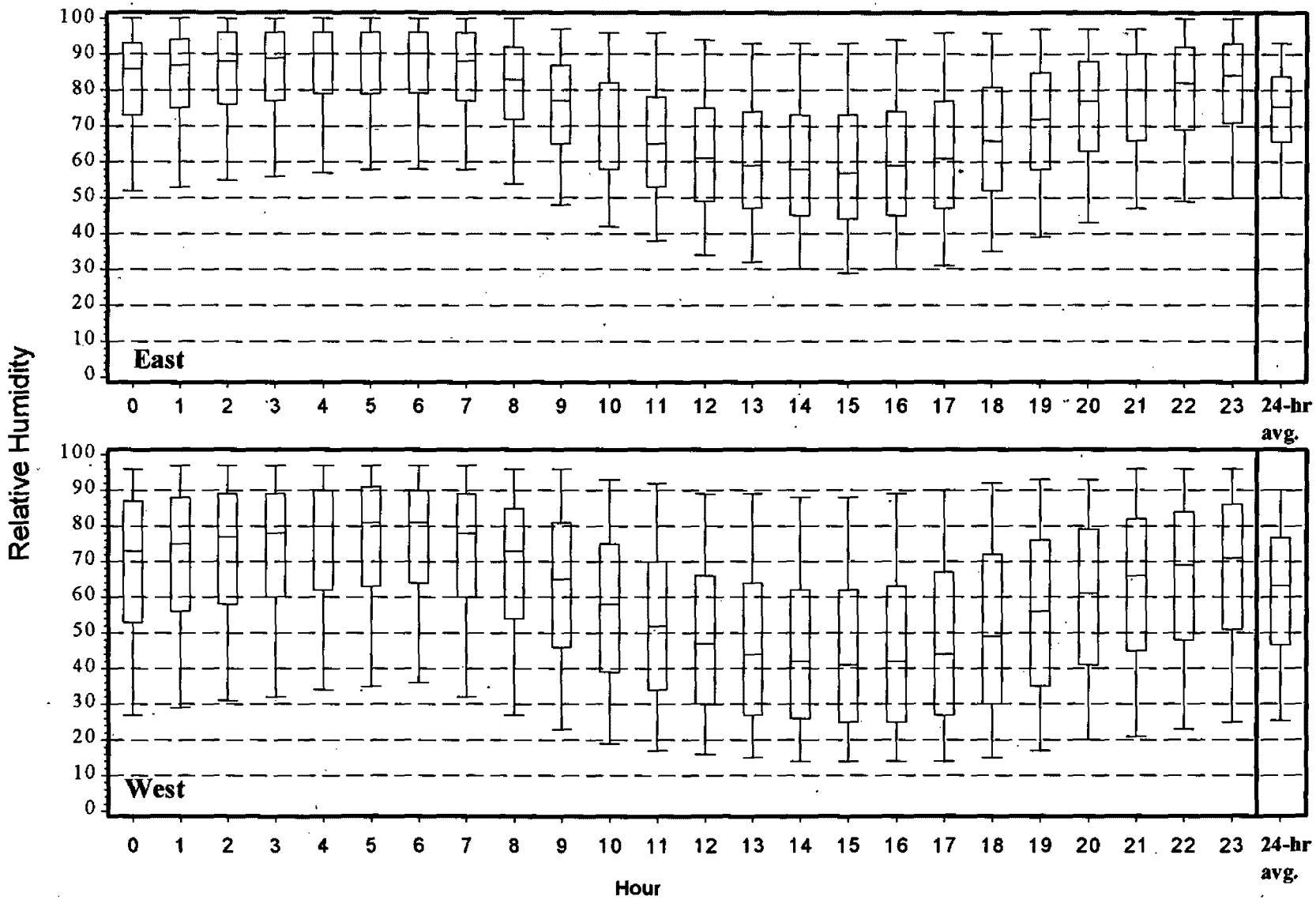


Figure 6-2. Distribution of hourly and 24-hour average relative humidity at eastern and western U.S. National Weather Service Sites, 2003. Box depicts interquartile range and median; whiskers depict 5th and 95th percentiles.

Source: Schmidt et al. (2005)

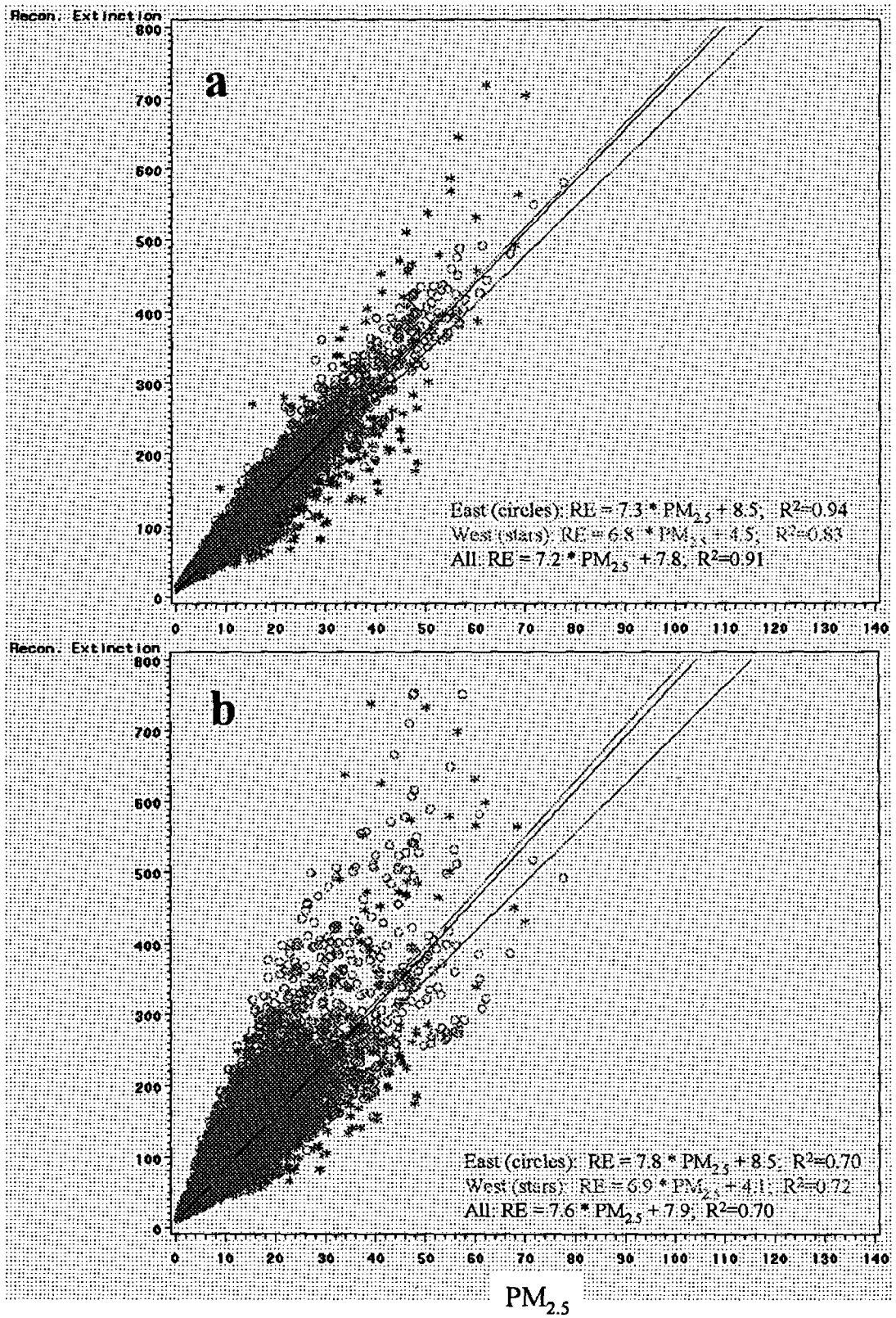


Figure 6-3. Relationship between reconstructed light extinction (RE) and 24- hour average $PM_{2.5}$, 2003. RE in top panel (a) computed with 10-year average *f(RH)*; RE in bottom panel (b) computed using actual *f(RH)*.

Source: Schmidt et al. (2005)

f(RH) data were also included in the analyses, to reflect the potential ranges of high and low relative humidity levels likely to occur over the course of a year.

In considering shorter-term daylight hour averaging periods, staff also evaluated the slope and strength of the correlations between RE and $PM_{2.5}$ concentrations on an hourly basis (Schmidt et al., 2005). Figure 6-4 shows plots of the average slope of the correlation between hourly RE and corresponding $PM_{2.5}$ concentrations (i.e., the increase in RE due to the incremental increase in $PM_{2.5}$) by region, in eastern and western areas, and nationwide. The slopes are all lower during daytime hours when the disproportionate effects of relative humidity on the light extinction coefficients for fine particle sulfates and nitrates are diminished. Thus, during daylight hours, the slope more closely represents the influence of $PM_{2.5}$ mass on visibility than the influence of relative humidity. In addition, Figure 6-4 shows that the slopes (and hence, the relationships between RE and $PM_{2.5}$) are more comparable across regions during daylight hours. In considering the strength of these correlations, staff notes that the correlations between RE and $PM_{2.5}$, as indicated by the model R^2 values, are strong for individual daylight hours, similar to that for the 24-hour average (Schmidt et al., 2005). On a national basis, daytime (9 a.m. to 6 p.m.) hourly model R^2 values are all above 0.6 for the RE's calculated with actual *f(RH)* values and above 0.8 for the RE's calculated with 10-year average *f(RH)* values (Schmidt et al., 2005).

On the basis of lower slopes and more inter-region comparability, staff selected a number of daylight time periods to consider in evaluating additional correlations between $PM_{2.5}$ concentrations and RE in eastern and western regions, as well as nationwide. Evaluated time periods included 7 a.m. to 7 p.m.; 9 a.m. to 5 p.m.; 10 a.m. to 6 p.m.; 10 a.m. to 4 p.m.; 12 p.m. to 4 p.m.; and 8 a.m. to 4 p.m. With a focus on minimizing slope, minimizing regional and East/West slope differences, maximizing R^2 values, and considering other related factors, staff selected the 12 p.m. to 4 p.m. time period for further analyses (Schmidt et al., 2005).

Using the same data as were used for Figure 6-3, Figure 6-5 shows examples of the correlations between RE and $PM_{2.5}$ concentrations averaged over a 4-hour time period, for 10-year average hourly *f(RH)* data (panel a) and for actual hourly *f(RH)* data in 2003 (panel b). As seen in this figure, the correlations between RE and $PM_{2.5}$ concentrations during daylight hours

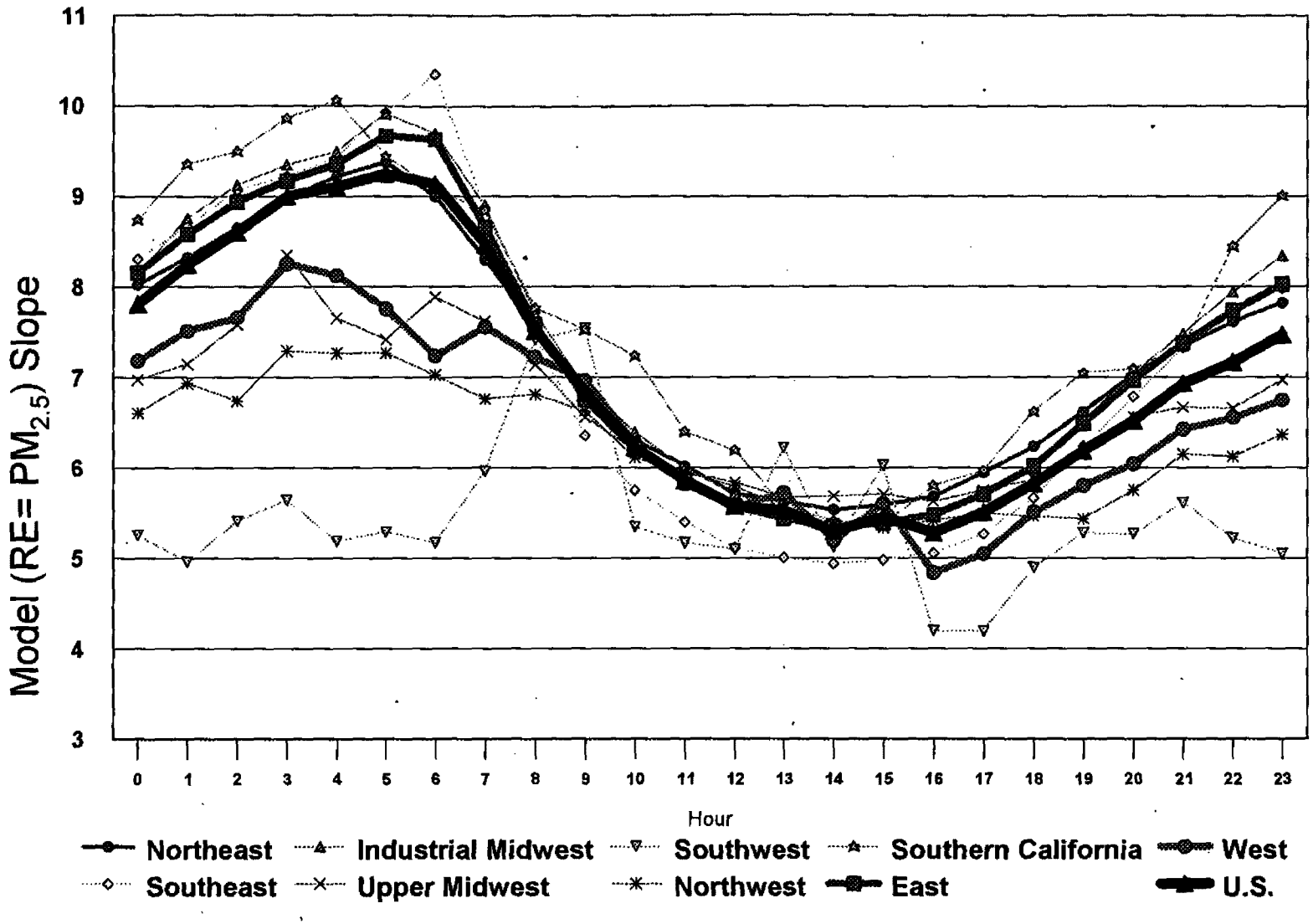


Figure 6-4. Model slope for relationship between reconstructed light extinction (RE) and hourly $PM_{2.5}$ (increase in RE due to incremental increase in $PM_{2.5}$), 2003. RE computed using 10-year average *f(RH)*.

Source: Schmidt et al. (2005)

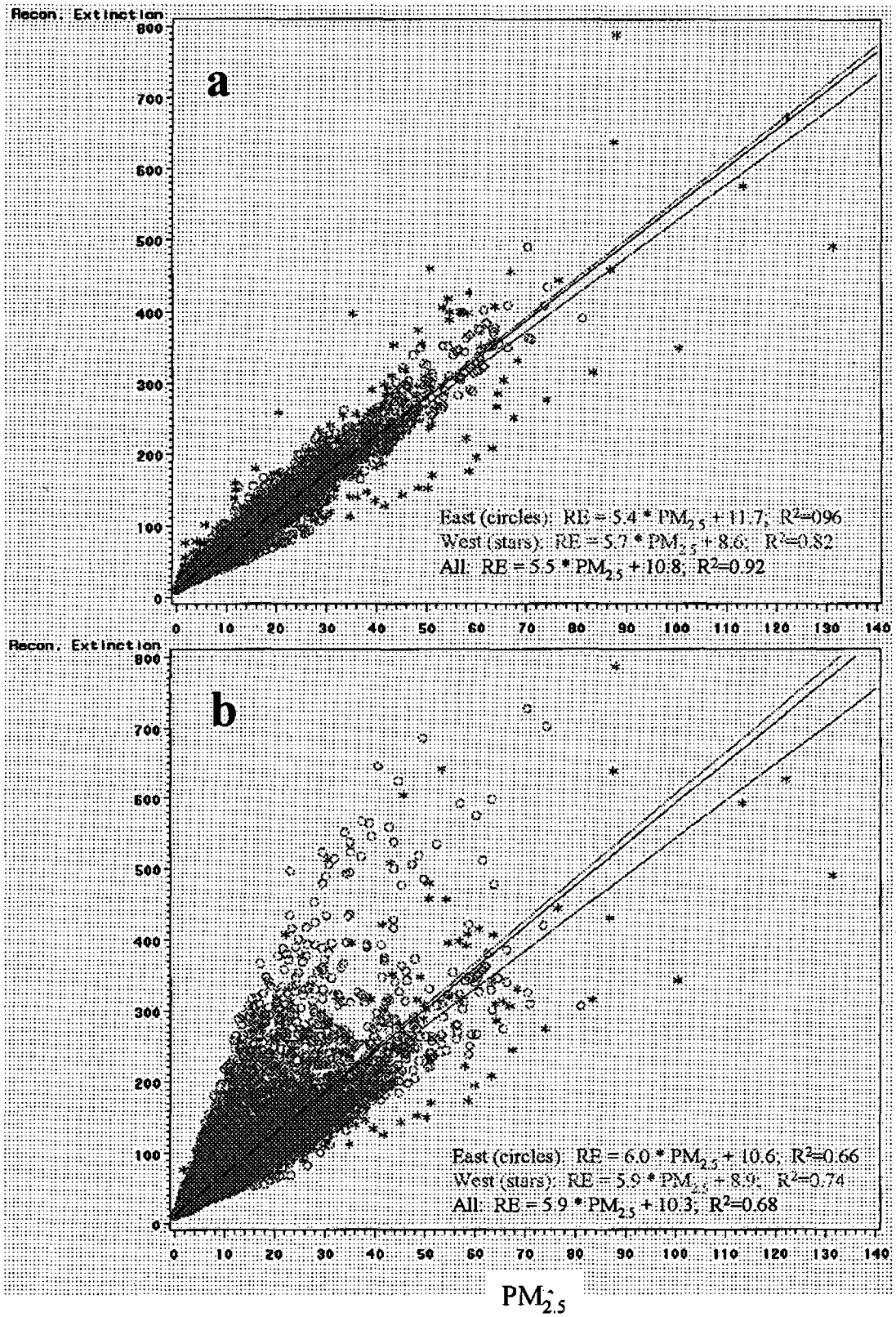


Figure 6-5. Relationship between reconstructed light extinction (RE) and 12 p.m. – 4 p.m. average $PM_{2.5}$, 2003. RE in top panel (a) computed with 10-year average *f(RH)*; RE in bottom panel (b) computed using actual *f(RH)*.

Source: Schmidt et al. (2005)

in urban areas are comparably strong (similar R^2 values), yet more reflective of $PM_{2.5}$ mass rather than relative humidity effects (i.e., lower slopes), in comparison to the correlations based on a 24-hour averaging time. Further, these correlations in urban areas are generally similar in the East and West, in sharp contrast to the East/West differences observed in rural areas.

6.2.4 Economic and Societal Value of Improving Visual Air Quality

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 (Chestnut and Rowe, 1991).

Individuals value good visibility for the sense of 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. These economic benefits are often described by economists as either use values or non-use values. Use values are those aspects of environmental quality that directly affect an individual's welfare. These include improved aesthetics during daily activities (e.g., driving or walking, looking out windows, daily recreations), for special activities (e.g., visiting parks and

scenic vistas, hiking, hunting), and for viewing scenic photography. Aesthetic benefits of better visibility also include improved road and air safety.

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, and for wilderness areas.

In addition, staff notes that the concept of option value is a key component of the measured values. The option value represents the value that is tied to preserving improved visibility in the event of a visit, even though a visit is not certain. This component is considered by some as a use value and by others as a non-use value.

Tourism in the U.S. is a significant contributor to the economy. 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 et al., 1998). A similar estimate of economic benefits resulting from visitation to national forests and other public lands could increase this estimate significantly.

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. 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 CD (CD, pp. 4-187 to 4-190), the 1996 Criteria Document (EPA, 1996a, p. 8-83, Table 8-5; p. 8-85, Table 8-6) and in Chestnut and Rowe (1991) and Chestnut et al. (1994). Past studies by Schulze et al. (1983) and Chestnut and Rowe (1990) 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 improvements in visibility can be difficult because visibility is not directly or indirectly valued in markets. Many of 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).

Some of the studies cited above used an alternative valuation method known as hedonic pricing. Hedonic pricing is a technique used to measure components of property value (e.g., proximity to schools). It relies on the measurement of differentials in property values under various environmental quality conditions, including air pollution and environmental amenities, such as aesthetic views. This method works by analyzing the way that market prices change

with changes in environmental quality or amenity. EPA believes that well-designed and well-executed hedonic valuation studies, in combination with public perception surveys, are useful for estimating the benefits of environmental effects such as improved visibility.

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, 1982). 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 National Transportation Safety Board (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.

6.2.5 Programs and Goals for Improving Visual Air Quality

Specific discussion is provided below on regional visibility programs in the U.S., as well as local visibility programs established by States, localities, and other countries in an effort to protect visual air quality.

6.2.5.1 Regional Protection

Due to differences in visibility impairment levels (due to differences in chemical composition of haze and in relative humidity levels) between the East and West, EPA, land managers, and States have taken a regional approach, rather than a national approach, to protecting visibility in non-urban areas in the U.S.. Protection against visibility impairment in special areas is provided for in sections 169A, 169B, and 165 of the Act, in addition to that provided by the secondary NAAQS. 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 (EPA, 1999; 65 FR 35713). 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.

6.2.5.2 Local, State, and International 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 because they are illustrative of the significant value that the public places on improving visibility, and because they have made use of

developed methods for evaluating public perceptions and judgments about the acceptability of varying degrees of visibility impairment.

Several state and local governments have developed programs to improve visual air quality in specific urban areas, including Denver, CO; Phoenix, AZ; and, Lake Tahoe, CA. At least two States have established statewide standards to protect visibility. In addition, visibility protection efforts have been undertaken in other countries, including Australia, New Zealand, and Canada. Examples of these efforts are highlighted below.

In 1990, the State of Colorado adopted a visibility standard for the city of Denver. The Denver standard is a short-term standard that establishes a limit of a four-hour average light extinction level of 76 Mm^{-1} (equivalent to a visual range of approximately 50 km) during the hours between 8 a.m. and 4 p.m. (Ely et al., 1991). In 2003, the Arizona Department of Environmental Quality created the Phoenix Region Visibility Index, which focuses on an averaging time of 4 hours during actual daylight hours. This visiblity index establishes visual air quality categories (i.e., excellent to very poor) and establishes the goals of moving days in the poor/very poor categories up to the fair category, and moving days in the fair category up to the good/excellent categories (Arizona Department of Environmental Quality, 2003). This approach results in a focus on improving visibility to a visual range of approximately 48-36 km. In 1989, the state of California revised the visibility standard for the Lake Tahoe Air Basin and established an 8-hour visibility standard equal to a visual range of 30 miles (approximately 48 km) (California Code of Regulations).

California and Vermont each have standards to protect visibility, though they are based on different measures. Since 1959, the state of California has had an air quality standard for particle pollution where the "adverse" level was defined as the "level at which there will be . . . reduction in visibility or similar effects." California's general statewide visibility standard is a visual range of 10 miles (approximately 16 km) (California Code of Regulations). In 1985, Vermont established a state visibility standard that is expressed as a summer seasonal sulfate concentration of 2 $\mu g/m^3$, that equates to a visual range of approximately 50 km. This standard was established to represent "reasonable progress toward attaining the congressional visibility

goal for the Class 1 Lye Brook National Wilderness Area, and applies to this Class 1 area and to all other areas of the state with elevations greater than 2500 ft.

Outside of the U.S., efforts have also been made to protect visibility. 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). A survey was undertaken for the Lower Fraser Valley in British Columbia, with responses from this pilot study being supportive of a standard in terms of a visual range of approximately 40 km for the suburban township of Chilliwack and 60 km for the suburban township of Abbotsford, although no visibility standard has been adopted for the Lower Fraser Valley at this time.

6.2.6 Approaches to Evaluating Public Perceptions and Attitudes

New methods and tools have been developed 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. New survey methods have been applied and evaluated in various studies, such as those for Denver, Phoenix, and the Lower Fraser Valley in British Columbia, and these studies are described below in more detail. 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. For the Denver and British Columbia studies, actual slides taken in the areas of interest, and matched with transmissometer and nephelometer readings, respectively, were used to assess public perceptions about visual air quality. For the Phoenix study, WinHaze, a newly available image modeling program, discussed below, was used for simulating images. Staff finds that, even with variations in each study's approaches, the survey methods used for the Denver, Phoenix, and British Columbia studies 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.

6.2.6.1 Photographic Representations of Visual Air Quality

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 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 et al. (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).

The simulation technique has the advantage of being readily applicable to any location as long as a very clear base photo is available for that location. 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. An alternative

technique to using simulated images is 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.

As part of a pilot study[5] in Washington, D.C., both survey and photographic techniques were applied (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 Attachment 6-A: Images of Visual Air Quality in Selected Urban Areas in the U.S.). Included as part of Attachment 6-A, this website also contains actual photographs of Chicago illustrating visibility conditions associated with a range of $PM_{2.5}$ concentrations, as well as simulated images for Denver and Phoenix, as discussed below.

6.2.6.2 Survey Methods

Denver, Colorado: Visibility Standard

The process by which the Denver visibility standard was developed 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-

[5] A small pilot study for Washington, D.C. was conducted by EPA and was briefly discussed in the preliminary draft staff paper (2001).

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.

Though images used in the Denver study were actual photographs, more recently, WinHaze has been used to generate images that illustrate visual air quality in Denver under a range of visibility conditions (generally corresponding to 10th, 20th, 30th, 40th, 50th, 60th 80th, and 90th percentile values), and these images are available in Attachment 6-A at http://www.epa.gov/ttn/naaqs/standards/pm/s_pm_cr_sp.html.

Data analyses using extensive new monitoring data now available on $PM_{2.5}$ primarily in urban areas show a consistently high correlation between hourly $PM_{2.5}$ data and RE coefficients for urban areas across regions of the U.S. during daylight hours. These correlations in urban areas are generally similar in the East and West, in sharp contrast to the East/West differences observed in rural areas.

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. 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.

In some urban areas, poor visibility has led to more localized efforts to better characterize, as well as improve, urban visibility conditions. The public perception 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. In the cases of Denver and Phoenix, these studies provided the basis for the establishment of their visibility standards and goals.

Staff believes that the findings of the new data analyses, in combination with recognized benefits to public welfare of improved visual air quality and an established approach for determining acceptable visual range, provide a basis for considering revisions to the secondary $PM_{2.5}$ standards to protect against PM-related visibility effects in urban areas.

6.3 EFFECTS ON VEGETATION AND ECOSYSTEMS

Information and conclusions regarding what is currently known about the impacts of ambient PM on ecosystems and individual components of ecosystems such as vegetation, soils, water, and wildlife are discussed in Chapters 4 and 9 of the CD. This section seeks to build upon and focus this body of science using EPA's ecological risk paradigm in a manner that highlights the usefulness and policy relevance of the scientific information. In so doing, staff has drawn

from EPA's *Guidelines for Ecological Risk Assessment* (*Guidelines*) (EPA, 1998), which expanded upon the earlier document, *Framework for Ecological Risk Assessment* (EPA, 1992), with the goal of improving the quality of ecological risk assessments and increasing the consistency of assessments across the Agency.

According to the *Guidelines* document, the three main phases of ecological risk assessment are problem formulation, analysis, and risk characterization. In problem formulation, the purpose for the assessment is articulated, the problem is defined, assessment endpoints are selected, a conceptual model is prepared and an analysis plan is developed. Initial work in problem formulation includes the integration of available information on sources, stressors, effects, and ecosystem and receptor characteristics.

In the analysis phase data are evaluated to determine how exposure to stressors is likely to occur (exposure profile) and the relationship between stressor levels and ecological effects (stressor-response profile). These products provide the basis for the risk characterization phase.

During the third phase, risk characterization, the exposure and stressor-response profiles are integrated through the risk estimation process. Risk characterization includes a summary of assumptions, scientific uncertainties, and strengths and limitations of the analyses. The final product is a risk description in which the results of the integration are presented, including an interpretation of ecological adversity and description of uncertainty and lines of evidence.

Keeping these goals and guidelines in mind, this section organizes information into the following seven subsections: major ecosystem stressors in PM (6.3.1); direct vegetation effects of PM stressor deposition (6.3.2); ecosystem effects of PM stressor deposition (6.3.3); characteristics and location of sensitive ecosystems within the U.S. (6.3.4); ecosystem exposures to PM deposition (6.3.5); consideration of critical loads as an approach for effects characterization and/or as a management tool (6.3.6); and summary and conclusions (6.3.7).

This review will also consider and reference where applicable the extent to which PM affects the essential ecological attributes (EEAs) outlined in 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), ad described in subsections 4.2.1 and 4.2.3 of the CD.

6.3.1 Major Ecosystem Stressors in PM

As previously discussed, PM is not a single pollutant, but a heterogeneous mixture of particles differing in size, origin, and chemical composition. 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. 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. 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. Because PM size classes do not necessarily have specific differential relevance for vegetation or ecosystem effects (Whitby, 1978; EPA, 1996a), it is the opinion of the staff that an ecologically relevant indicator for PM would be based on one or multiple chemical stressors found in ambient PM. At this time it remains to be studied as to what extent NAAQS standards focused on a given size fraction would result in reductions of the ecologically relevant constituents of PM for any given area.

A number of different chemical species found within ambient PM and their effects on vegetation and ecosystems were discussed in chapter 4 of the PM CD. In particular, the CD focused on nitrates and sulfates, concluding that these PM constituents are considered to be the stressors of greatest environmental significance (CD, p. 9-114). Other components of PM, such as dust, trace metals, and organics, which can also be toxic to plants and other organisms at high levels, were also discussed. However, because such high levels occur only near a few limited point sources and/or on a very local scale, they do not appear significant at the national level. Therefore, the remainder of this section will narrow its focus to consideration of the impacts of particulate nitrates and sulfates, both separately and in combination with acidifying compounds, on sensitive ecosystem components and essential ecological attributes, which in turn, impact overall ecosystem structure and function.

6.3.2 Direct Vegetation Effects of PM Stressor Deposition

Nitrogen is a critical limiting nutrient for plant growth. The process of photosynthesis uses approximately 75% of the nitrogen in a plant leaf, and, thus, to a large extent, governs the utilization of other nutrients such as phosphorus, potassium (CD, p. 4-95). Plants usually absorb nitrogen (as NH_4^+ or NO_3^-) through their roots. However, particle deposition of nitrate, together with other nitrogen-containing gaseous and precipitation-derived sources, can represent a substantial fraction of total nitrogen reaching vegetation. In nitrogen-limited ecosystems, this influx of N can act as a fertilizer. Though it is known that foliar uptake of nitrate can occur, the mechanism of foliar uptake is not well established, 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. 4-69, 4-70).

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 also 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 (CD, pp. 4-71, 4-72).

Staff therefore conclude that at current ambient levels, risks to vegetation from short term exposures to dry deposited particulate nitrate or sulfate are low. Additional studies are needed, however, on the effects of sulfate particles on physiological characteristics of plants following chronic exposures (CD, p. 4-72).

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).

Acid precipitation with levels of acidity associated with the foliar effects described above are currently found in some locations in the U.S.. For example, in the eastern U.S., the mean precipitation pH ranges from 4.3 (Pennsylvania and New York) to 4.8 (Maine)(EPA, 2003). It can be assumed that occult (mist or fog) deposition impacting high elevations more frequently, would contain even higher concentrations of acidity. Thus, staff conclude that the risks of foliar injury occurring from acid deposition is high. The contribution of particulate sulfates and nitrates to the total acidity found in the acid deposition impacting eastern vegetation is not clear.

6.3.3 Ecosystem Effects of PM Stressor Deposition

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 nutrient cycling and/or shifts in biodiversity. The most

significant PM-related ecosystem-level effects result from 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, usually by 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 availability of this type of long-term data is limited. The following discussion is organized according to the speciated effects of PM on ecosystems.

6.3.3.1 Environmental Effects of Reactive Nitrogen (Nr) Deposition

In the environment, nitrogen may be divided into two types: nonreactive, molecular nitrogen (N_2) and reactive nitrogen (Nr). Molecular nitrogen is the most abundant element in the atmosphere. However, it only becomes available to support the growth of plants and microorganisms after it is converted into a reactive form. In nature, Nr creation is accomplished by certain organisms that have developed the capability of converting N_2 to biologically active reduced forms (Galloway and Cowling, 2002; Hornung and Langan, 1999; EPA, 1993). By the mid-1960's, however, Nr creation through natural terrestrial processes had been overtaken by Nr creation as a result of human processes (CD, p. 4-95). The deposition of nitrogen in the U.S. from human activity doubled between 1961 and 1997, with the largest increase occurring in the 1960s and 1970s (CD, p. 4-98). Reactive nitrogen is now accumulating in the environment on all spatial scales – local, regional and global. The three main sources of anthropogenic Nr are: (1) the Haber-Bosch process, which converts N_2 to Nr to sustain food production and some industrial activities; (2) widespread cultivation of legumes, rice and other crops that promote the conversion of N_2 to organic nitrogen through biological nitrogen fixation; and (3) combustion of fossil fuels, which converts both atmospheric N_2 and fossil nitrogen to reactive NO_x (CD, pp. 4-95, 4-96; Galloway and Cowling, 2002; Galloway et al., 2003). Currently available forms of reactive nitrogen include 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, p. 4-95).

Emissions of nitrogen oxides from fuel burning increased exponentially from1940 until the 1970s, leveled off after the passage of the 1970 amendments to the Clean Air Act, and stabilized at approximately 7 Tg NO_x /yr in the late 1990s. Contemporary emissions of NO_x in the U.S. from fossil fuel burning are nearly two-thirds the rate of Nr released from the use of inorganic fertilizers and comprise 30% of the global emissions of NO_x from fossil fuel combustion. Despite decreases in emissions from fossil fuel burning industries, emissions from automobiles have increased approximately 10% since 1970 due to greater total miles driven (Howarth et al., 2002). Some NO_x emissions are transformed into a portion of ambient air PM (particulate nitrate) and deposited onto sensitive ecosystems.

The term "nitrogen cascade" refers to the sequential transfers and transformations of Nr molecules as they move from one environmental system or reservoir (atmosphere, biosphere, hydrosphere) to another, and the multiple linkages that develop among the different ecological components, as shown in Figure 6-6. Because of these linkages, adding anthropogenic Nr alters a wide range of biogeochemical processes and exchanges as the Nr moves among the different environmental reservoirs, with the consequences accumulating through time (Galloway and Cowling, 2002; Galloway et al., 2003). These changes in the nitrogen cycle are contributing to both beneficial and detrimental effects to the health and welfare of humans and ecosystems (Rabalais, 2002; van Egmond et al., 2002; Galloway, 1998).

Large uncertainties, still exist, however, concerning the rates of Nr accumulation in the various environmental reservoirs which limit our ability to determine the temporal and spatial distribution of environmental effects for a given input of Nr. These uncertainties are of great significance because of the sequential nature of Nr effects on environmental processes. Reactive nitrogen does not cascade at the same rate through all environmental systems. The only way to eliminate Nr accumulation and stop the cascade is to convert Nr back to nonreactive N_2 (Galloway et al., 2003).

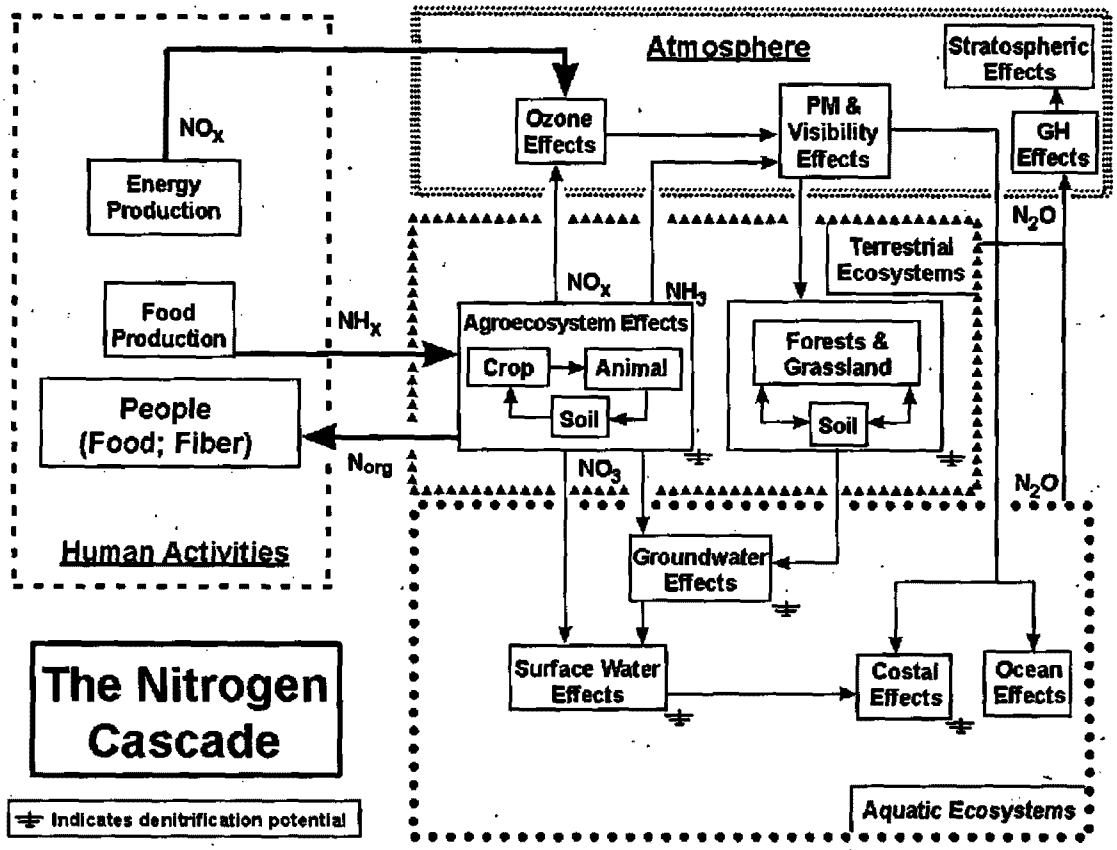


Figure 6-6 Illustration of the nitrogen cascade showing the movement of human-produced reactive nitrogen (Nr) as it cycles through the various environmental reservoirs in the atmosphere and in terrestrial and aquatic ecosystems (Galloway et al., 2003; Figure 4-15, CD p. 4-97).

Some of the more significant detrimental effects resulting from chronic increased inputs of atmospheric Nr (e.g., particulate nitrates) include: (1) decreased productivity, increased mortality, and/or shifts in terrestrial plant community composition, often leading to decreased biodiversity in many natural habitats wherever atmospheric Nr deposition increases significantly and critical thresholds are exceeded (Aber et al., 1995); (2) leaching of excess nitrate and associated base cations from terrestrial soils into streams, lakes and rivers and mobilization of soil aluminum; (3) eutrophication, hypoxia, loss of biodiversity, and habitat degradation in coastal ecosystems, now considered a major pollution problem in coastal waters (Rabalais,

2002); (4) acidification and loss of aquatic flora and fauna biodiversity in lakes and streams in many regions of the world when associated with sulfur deposition (Vitousek et al., 1997); and (5) alteration of ecosystem processes such as nutrient and energy cycles through changes in the functioning and species composition of beneficial soil organisms (Galloway and Cowling 2002).

Additional, indirect detrimental effects of excess Nr on societal values include: (1) increases in fine PM resulting in regional hazes that decrease visibility at scenic rural and urban vistas and airports (discussed above in section 6.2); (2) depletion of stratospheric ozone by N_2O emissions which can in turn affect ecosystems and human health; (3) global climate change induced by emissions of N_2O (Galloway et al., 2003); (4) formation of O_3 and ozone-induced injury to crops, forests, and natural ecosystems and the resulting predisposition to attack by pathogens and insects, as well as human health related impacts (EPA, 1996); (5) decrease in quantity or quality of available critical habitat for threatened and endangered species (Fenn et al., 2003); and (6) alteration of fire cycles in a variety of ecoystem types (Fenn et al., 2003).

A number of the more significant effects of chronic, long-term deposition of Nr on terrestrial and aquatic ecosystems will be discussed below, specifically those effects which seem to pose the greatest long-term risks to species or ecosystem health and sustainability or that threaten ecosystem flows of goods and services important to human welfare.

Nitrogen Saturation of Terrestrial Ecosystems

Long-term, chronic additions of Nr (including nitrate deposition from ambient PM) to terrestrial ecosystems is resulting in numerous ecosystems shifting to a detrimental ecological condition known as "nitrogen saturation." Nitrogen saturation does not occur at a specific point in time, but is a set of gradually developing critical changes in ecosystem processes which represent the integrated response of a system to increased nitrogen availability over time (Aber, 1992). It occurs when nitrogen inputs exceed the capacity of plants and soil microorganisms to utilize and retain the nitrogen (Aber et al., 1989, 1998; Garner, 1994; EPA, 1993). Under conditions of nitrogen saturation, some other resource generally replaces nitrogen in limiting biotic functions. The appearance of nitrate in soil solution (leaching) is an early symptom of excess Nr accumulation.

Not all vegetation, organisms, or ecosystems react in the same manner to increased Nr availability from atmospheric deposition. This is due in part to the variation both within and

across species in their inherent capacity to utilize additional Nr and the suite of other factors that influence the range of community or ecosystem types possible at any given location. Such factors can include the mineral composition of the underlying bedrock, the existing soil nutrient pools, the local climatic conditions including weather extremes such as drought, high/low temperatures, topography, elevations, natural/land use history, and fire regimes.

In U.S. ecosystems, the nutrient whose supply most often sets the limit of possible primary productivity at a given site is biologically available nitrogen. However, in any given ecosystem, not all plants are equally capable of utilizing extra nitrogen. Those plants that are predisposed to capitalize on any increases in Nr availability gain an advantage over those that are not as responsive to added nutrients. Over time, this shift in the competitive advantage may lead to shifts in overall plant community composition. Whether or not this shift is considered adverse would depend on the management context within which that ecosystem falls and the ripple effects of this shift on other ecosystem components, essential ecological attributes (EEAs), and ecosystems.

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 the U.S. and Europe. These studies suggest that some forests receiving chronic inputs of nitrogen may decline in productivity and experience greater mortality (Fenn et al. 1998). For example, fertilization and nitrogen gradient experiments at Mount Ascutney, VT suggest that nitrogen saturation may lead to the replacement of slow-growing, slow nitrogen-cycling spruce-fir forest stands by fast-growing deciduous forests that cycle nitrogen rapidly (Fenn et al. 1998). Similarly, experimental studies of the effects of Nr deposition over a 12-year period on Minnesota grasslands dominated by native warm-season grasses observed the shift to low-diversity mixtures dominated by cool-season grasses at all but the lowest rates of Nr addition (Wedin and Tilman, 1996). The shift to low-diversity mixtures was associated with the decrease in biomass carbon to N (C:N) ratios, increased Nr mineralization, increased soil nitrate, high nitrogen losses, and low carbon storage. Grasslands with high nitrogen retention and carbon storage rates were the most vulnerable to loss of species and major shifts in nitrogen cycling. (Wedin and Tilman, 1996).

The carbon-to-nitrogen (C:N) ratio of the forest floor can be changed by nitrogen deposition over time. In Europe, low C:N ratios coincide with high deposition regions. A strong decrease in forest floor root biomass has also been observed with increased nitrogen availability, and appears to occur when the ecosystem becomes nitrogen saturated. If root growth and mycorrhizal formation are impaired by excessive nitrogen deposition, the stability of the forest floor vegetation may be affected. The forest floor C:N ratio has been used as a rough indicator of ecosystem nitrogen status in mature coniferous forests and the risk of nitrate leaching. Nitrate leaching has been significantly correlated with forest floor nitrate status, but not with nitrate deposition. 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. Understanding the variability in forest ecosystem response to nitrogen input is essential in assessing pollution risks (Gundersen et al., 1998; CD, pp. 4-106, 4-107).

In the U.S., forests that are now showing severe symptoms of nitrogen saturation include: 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 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).

The composition and structure of the plant community within an ecosystem in large part determines the food supply and habitat types available for use by other organisms. In terrestrial systems, plants serve as the integrators between above-ground and below-ground environments and are influenced by and influence conditions in each. It is because of these linkages that chronic excess Nr additions can lead to complex, dramatic, and severe ecosystem level/wide changes/responses. Changes in soil Nr influence below ground communities as well. A mutualistic relationship exists in the rhizosphere (plant root zone) between plant roots, fungi, and microbes. Because the rhizosphere is an important region of nutrient dynamics, its function is critical for the growth of the organisms involved. The plant roots provide shelter and carbon for the symbionts, whereas the symbionts provide access to limiting nutrients such as nitrogen and phosphorus for the plant. Bacteria make N, S, Ca, P, Mg, and K available for plant use while fungi in association with plant roots form mycorrhizae that are essential in the uptake by plants of mineral nutrients, such as N and P (Section 4.3.3; Wall and Moore, 1999; Rovira and Davy, 1974). Mycorrhizal fungal diversity is associated with above-ground plant biodiversity, ecosystem variability, and productivity (Wall and Moore, 1999). Studies suggest that during nitrogen saturation, soil microbial communities change from being predominately fungal, and dominated by mycorrhizae, to being dominated by bacteria (Aber et al., 1998; CD, pp. 4-107, 4-108), dramatically affecting both above- and below-ground ecosytems. These types of effects have been observed in the field. For example, the coastal sage scrub (CSS) community in California has been declining in land area and in drought deciduous shrub density over the past 60 years, and is being replaced in many areas by Mediterranean annual grasses. At the same time, larger-spored below-ground 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 the coastal sage scrub species can be directly linked to

the decline of the arbuscular mycorrhizal community (Edgerton-Warburton and Allen, 2000; Allen et al., 1998; Padgett et al., 1999)(CD, pp. 4-108, 4-109).

Impacts on threatened and endangered species. In some rare and unique U.S. ecosystems, the chronic additions of atmospherically-derived nitrogen have already had some dire and perhaps irreversible consequences. For example, California has many species that occur in shrub, forb, and grasslands affected by N deposition, with up to 200 sensitive plant species in southern California CSS alone (Skinner and Pavlik, 1994). Some 25 plant species are already extinct in California, most of them annual and perennial forbs that occurred in sites now experiencing conversion to annual grassland. As CSS converts more extensively to annual grassland dominated by invasive species, loss of additional rare species may be inevitable. Though invasive species are often identified as the main threat to rare species, it is more likely that invasive species combine with other factors, such as excess N deposition, to promote increased productivity of invasive species and resulting species shifts.

Not surprisingly, as sensitive vegetation is lost, wildlife that depend on these plants are adversely affected. Included among these wildlife species are several threatened or endangered species listed by the U.S. Fish and Wildlife Service, such as the desert tortoise and checkerspot butterfly. A native to San Francisco Bay area, the bay checkerspot butterfly (*Euphydryas editha bayensis*), has been declining steadily over the past decade, with local extirpations in some reserves. This decline has been associated with the invasion of exotic grasses replacing the native forbs on which the butterfly depends. In particular, the larval stage is dependent on primarily one host plant, *Plantago erecta*, which is increasingly being out-competed by exotic grasses.

Similarly, the desert tortoise has declined due to a number of co-occurring stresses, including grazing, habitat destruction, drought, disease, and a declining food base. In the desert shrub inter-spaces, sites where native forbs once flourished, invasive grasses now dominate, reducing the nutritional quality of foods available to the tortoise (Fenn et al., 2003; Nagy et al., 1998). Nitrogen deposition contributes to the productivity and density of N-fertilized grasses at the expense of native forbs (Brooks, 2003). "Thus, protection of endangered species will require increased exotic grass control, but local land management strategies to protect these

endangered species may not succeed unless they are accompanied by policy changes at the regional or national level that reduce air pollution" (Fenn et al., 2003).

Community composition of epiphytic lichens is readily altered by small increases in nitrogen deposition, an effect that seems to be widespread in the West (Fenn et al., 2003). Most epiphytic lichens meet their nutritional requirements from atmospheric deposition and can store N in excess of their nutritional needs (van Herk, 1999). In the San Bernardino Mountains, up to 50% of the lichen species that occurred in the region in the early 1900s have disappeared, with a disproportionate number of the locally extinct species being (epiphytic) cyanolichens (Fenn et al., 2003; Nash and Sigal, 1999). The Pacific Northwest, in contrast, still has widespread populations of pollution-sensitive lichens (Fenn et al., 2003). However, in urban areas, intensive agricultural zones and downwind of major urban and industrial centers, there is a sparsity of sensitive lichen species and high levels of N concentrations have been measured in lichen tissue (Fenn et al., 2003). Replacement of sensitive lichens by nitrophilous species has undesirable ecological consequences. In late-successional, naturally N-limited forests of the Coast Range and western Cascades, epiphytic cyanolichens make important contributions to mineral cycling and soil fertility (Pike 1978, Sollins et al., 1980, Antoine, 2001), and together with other large, pollution-sensitive macrolichens, are an integral part of the food web for large and small mammals, insects and birds (McCune and Geiser, 1997).

Alteration of native fire cycles. Several lines of evidence suggest that N deposition may be contributing to greater fuel loads and thus altering the fire cycle in a variety of ecosystem types, although further study is needed (Fenn et al., 2003). Invasive grasses promote a rapid fire cycle in many locations (D'Antonio and Vitousek, 1992). The increased productivity of flammable understory grasses increases the spread of fire and has been hypothesized as one mechanism for the recent conversion of CSS to grassland (Minnich and Dezzani, 1998).

Thus, through its effect on habitat suitability, genetic diversity, community dynamics and composition, nutrient status, energy and nutrient cycling, and frequency and intensity of natural disturbance regimes (fire), excess Nr deposition is having profound and adverse impact on the essential ecological attributes associated with terrestrial ecosystems. Strong correlation between the stressor and adverse environmental response exists in many locations, and N-addition studies

have confirmed this relationship between stressor and response. Loss of species and genetic diversity are clearly adverse ecological effects and adverse to the public welfare. Research efforts should be made to elucidate what role particulate deposition is playing in contributing to these effects so as to facilitate the mitigation of such effects.

Effects of Nitrogen Addition on Aquatic Habitats

Aquatic ecosystems (streams, rivers, lakes, estuaries or oceans) receive increased nitrogen inputs either from direct atmospheric deposition (including nitrogen-containing particles), surface runoff, or leaching from nitrogen saturated soils into ground or surface waters. The primary pathways of Nr loss from forest ecosystems are hydrological transport beyond the rooting zone into groundwater or stream water, or surface flows of organic nitrogen as nitrate and Nr loss associated with soil erosion (Fenn et al., 1998). In the east, high nitrate concentrations have been observed in streams draining nitrogen saturated watersheds in the southern Appalachian Mountains (Fenn et al., 1998). The Great Smoky Mountains National Park in Tennessee and North Carolina receives elevated levels of 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 (CD, pp. 4-110, 4-111; see also section 6.3.3.2 on acidification from PM deposition).

In the west, the Los Angeles Air Basin exhibited the highest stream water NO_3^- concentrations in wilderness areas of North America (Bytnerowicz and Fenn, 1996; Fenn et al., 1998). Chronic N deposition in southern California, in the southwestern Sierra Nevada, and in the Colorado Front Range leads to increased net N mineralization and nitrification rates in soil and to elevated NO_3^- concentrations in lakes and streams. These symptoms occur in low- and mid-elevation, high-deposition areas (>15 kg N/ha/yr) and in high elevation sites with relatively low N deposition (4 to 8 kg N/ha/yr) but little capacity to assimilate and retain added N.

Estuaries are among the most intensely fertilized systems on Earth (Fenn et al., 1998). They receive far greater nutrient inputs than other systems. In the Northeast, for example, nitrogen is the element most responsible for eutrophication in coastal waters of the region. Since the early 1900s, there has been a 3- to 8-fold increase in nitrogen flux from10 watersheds in the

northeast. 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, averaging approximately 64%, was derived directly or indirectly from nitrogen oxide emissions (CD, pp. 4-109, 4-110).

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, has also been the subject of recent research (Paerl et al., 2001) to characterize the effects of nitrogen deposition on the estuary. 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. Especially under extreme rainfall events (e.g., hurricanes), massive influxes of nitrogen (in combination with excess loadings of metals or other nutrients) into watersheds and sounds can lead to dramatic decreases of oxygen in water and the creation of widespread "dead zones" and/or increases in algae blooms that can cause extensive fish kills and damage to commercial fish and sea food harvesting (Paerl et al., 2001; CD, pp. 4-109, 4-110).

6.3.3.2 Environmental Effects of PM-Related Acidic Deposition

Acidic deposition has emerged over the past quarter century as a critical environmental stress that affects diverse terrestrial and aquatic ecosystems in North America, Europe, and Asia (Driscoll et al., 2001). In the eastern U.S. for example, the current acidity in precipitation is at least twice as high as in pre-industrial times, with mean precipitation pH ranges from 4.3 (Pennsylvania and New York) to 4.8 (Maine) (EPA, 2003). Acidic deposition is highly variable across space and time, can originate from transboundary air pollution, can travel hundreds of miles before being deposited, thereby affecting large geographic areas. It is composed of ions, gases, and particles derived from the precursor gaseous emissions of SO_2, NO_x, NH_3 and

particulate emissions of other acidifying compounds. Acid deposition disturbs forest and aquatic ecosystems by giving rise to harmful chemical conditions (Dricoll et al., 2001).

Terrestrial Effects

Acidic deposition has changed the chemical composition of soils by depleting the content of available plant nutrient cations (e.g., Ca^{2+}, Mg^{2+}, K^{+}) by increasing the mobility of Al, and by increasing the S and N content (Driscoll et al., 2001). Soil leaching is often of major importance in cation cycles, and many forest ecosystems show a net loss of base cations (CD, pp. 4-118). In acid sensitive soils, mineral weathering (the primary source of base cations in most watersheds) is insufficient to keep pace with leaching rates accelerated by acid deposition (Driscoll et al., 2001).

In the absence of acid deposition, cation leaching in northeastern forest soils is driven largely by naturally occurring organic acids derived from the decomposition of organic matter. Organic acids tend to mobilize Al through formation of organic-Al complexes, most of which are deposited lower in the soil profile through adsorption to mineral surfaces. This process, termed podzolization, results in surface waters with low concentrations of Al. Such concentrations are primarily in a nontoxic, organic form (Driscoll et al., 1998). Acid deposition, however, has altered podzolization by solubilizing Al with mobile inorganic anions, facilitating the transport of inorganic Al into surface waters. In forest soils with base saturation values less than 20%, acidic deposition leads to increased Al mobilization and a shift in chemical speciation of Al from organic to inorganic forms that are toxic to terrestrial and aquatic biota.

The toxic effect of Al on forest vegetation is attributed to its interference with plant uptake of essential nutrients, such as Ca and Mg. 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 (Smith, 1990a). There are large variations in Al sensitivity among ecotypes, between and within species, due to differences in nutritional demands and physiological status, that are related to age and climate, which change over time (CD, pp. 4-126).

Acidic deposition has been firmly implicated as a causal factor in the northeastern high-elevation decline of red spruce (DeHayes et al., 1999). Red spruce is common in Maine, where it is an important commercial species. It is also common at high elevations in mountainous regions throughout the Northeast, where it is valued for recreation and aesthetics, as well as for providing a habitat for unique and endangered species. Dieback has been most severe at high elevations in the Adirondack and Green Mountains, where more than 50% of the canopy trees died during the 1970s and 1980s. In the White Mountains, about 25% of the canopy spruce died during that same period (Craig and Friedland 1991). Dieback of red spruce trees has also been observed in mixed hardwood-conifer stands at relatively low elevations in the western Adirondack Mountains, areas that receive high inputs of acidic deposition (Shortle et al., 1997). Results of controlled exposure studies show that acidic mist or cloud water reduces the cold tolerance of current-year red spruce needles by 3-10 degrees C (DeHayes et al., 1999). This increased susceptibility to freezing occurs due to the loss of membrane-associated Ca^{2+} from needles through leaching caused by the hydrogen ion. The increased frequency of winter injury in the Adirondack and Green Mountains since 1955 coincides with increased exposure of red spruce canopies to highly acidic cloud water (Johnson et al., 1984). Recent episodes of winter injury have been observed throughout much of the range of red spruce in the Northeast. (DeHayes et al., 1999). 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, p. 4-120).

Although less well established, there is also a strong possibility that low Ca to Al ratios in soils may also be impacting northeastern red spruce. Cronan and Grigal (1995) concluded that a Ca:Al ratio of less than 1.0 in soil water indicated a greater than 50% probability of impaired growth in red spruce. They cite examples of studies from the northeast where soil solutions in the field were found to exhibit Ca:Al ionic ratios less than 1.0.

Acidic deposition may also be contributing to episodic dieback of sugar maple in the Northeast through depletion of nutrient cations from marginal soils. Horsley et al. (1999) found that dieback at 19 sites in northwestern and north-central Pennsylvania and south-western New York was correlated with combined stress from defoliation and deficiencies of Mg and Ca. Dieback occurred predominately on ridgetops and on upper slopes, where soil base availability was much lower than at mid and low slopes of the landscape (Bailey et al., 1999). Because multiple factors such as soil mineralogy and landscape position affect soil base status, the extent to which sugar maple dieback can be attributed to acidic deposition is not clear.

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 S and N deposition for many years.

In contrast to contributing to the adverse impacts of acid deposition, particles can also provide a beneficial supply of base cations to sites with very low rates of supply from mineral sources. In these areas, atmospheric inputs of bass cations can help ameliorate the acidifying effects of acid particles. 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. This study showed that in the IFS ecosystems, 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. 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 (CD, p. 4-132). 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 (CD, pp. 4-127, 4-128).

In a follow up study, Johnson et al. (1999) used the nutrient cycling moded, NuCM, 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 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 only a minor effect on soils and soil solutions; whereas S and N deposition had delayed but major effects on C_B leaching (CD, pp. 4-136, 4-137).

Aquatic Effects

Inputs of acidic deposition to regions with base-poor soils have resulted in the acidification of soil waters, shallow ground waters, streams, and lakes in a number of locations within the U.S. In addition, perched seepage lakes, which derive water largely from direct precipitation inputs, are highly sensitive to acidic deposition (Charles, 1991). These processes usually result in lower pH and, for drainage lakes, higher concentrations of inorganic monomeric Al. Such changes in chemical conditions are toxic to fish and other aquatic animals. (Driscoll et al., 2001).

A recent report, *Response of Surface Water Chemistry to the Clean Air Act of 1990* (EPA, 2003), analyzes data from 1990 through 2000 obtained from EPA's Long Term Monitoring (LTM) and Temporally Integrated Monitoring of Ecosystems (TIME) projects, part of EMAP (Environmental Monitoring and Assessment Program). The report assesses recent changes in surface water chemistry in response to changes in deposition, in the northern and eastern U.S., specifically in the acid sensitive regions defined as New England (Maine, New Hampshire, Vermont and Massachusetts), the Adirondack Mountains of New York, the Northern Appalachian Plateau (New York, Pennsylvania and West Virginia), the Ridge and Blue Ridge Provinces of Virginia, and the Upper Midwest (Wisconsin and Michigan). Acidic waters are defined as having acid neutralizing capacity (ANC) less than zero (i.e., no acid buffering capacity in the water), corresponding to a pH of about 5.2. Increases in surface water ANC values and/or pH would indicate improved buffering capacity and signal the beginning of recovery (EPA, 2003).

Using National Atmospheric Deposition Program (NADP) data, trends in sulfate and N (nitrate + ammonium) deposition were analyzed, along with C_B deposition, sulfate and nitrate

concentrations in surface waters, ANC and pH levels. Over this timeframe, sulfate deposition declined significantly across all regions, while N declined slightly in the Northeast and increased slightly in the Upper Midwest. Base cation deposition showed no significant changes in the East and increased slightly in the Upper Midwest. Concurrently, all regions except the Ridge/Blue Ridge province in the mid-Atlantic showed significant declines in sulfate concentrations in surface waters, while nitrate concentrations decreased in two regions with the highest ambient nitrate concentrations (Adirondacks, Northern Appalachian Plateau) but were relatively unchanged in regions with low concentrations.

Given the declines in S and N deposition measured for these areas, one would expect to find increasing values of ANC, pH or both in response. ANC values did increase in the Adirondacks, Northern Appalachian Plateau and Upper Midwest, despite a decline in base cations (Ca and Mg) in each region. The loss of base cations limited the extent of ANC and pH increase. Toxic Al concentrations also declined slightly in the Adirondacks. In New England and Ridge/Blue Ridge, however, regional surface water ANC did not change significantly (EPA, 2003).

Modest increases in ANC have reduced the number of acidic lakes and stream segments in some regions. There are an estimated 150 Adirondack lakes with ANC less than 0, or 8.1% of the population, compared to 13% (240 lakes) in the early 1990s. In the Upper Midwest, an estimated 80 of 250 lakes that were acidic in mid-1980s are no longer acidic. TIME surveys of streams in the Northern Appalachian Plateau region estimated that 8.5% (3,600 kilometers) of streams remain acidic at the present time, compared to 12% (5,014 kilometers) of streams that were acidic in 1993-94. In these three regions taken together, approximately one-fourth to one-third of formerly acidic surface waters are no longer acidic, although still with very low ANC. The report finds little evidence of regional change in the acidity status of New England or the Ridge/Blue Ridge regions and infers that the numbers of acidic waters remain relatively unchanged. Despite a general decline in base cations and a possible increase in natural organic acidity, there is no evidence that the number of acidic waters have increased in any region (EPA, 2003).

Acidification has marked effects on the trophic structure of surface waters. Decreases in pH and increases in Al concentrations contribute to declines in species richness and in the abundance of zooplankton, macroinvertebrates, and fish (Schindler et al.,1985; Keller and Gunn 1995). Numerous studies have shown that fish species richness (the number of fish species in a water body) is positively correlated with pH and ANC values (Rago and Wiener, 1986 Kretser et al., 1989). Decreases in pH result in decreases in species richness by eliminating acid-sensitive species (Schindler et al. 1985). Of the 53 fish species recorded by the Adirondack Lakes Survey Corporation, about half (26 species) are absent from lakes with pH below 6.0. Those 26 species include important recreational fishes, such as Atlantic salmon, tiger trout, redbreast sunfish, bluegill, tiger musky, walleye, alewife, and kokanee (Kretser et al. 1989), plus ecologically important minnows that serve as forage for sport fishes.

A clear link exists between acidic water, which results from atmospheric deposition of strong acids, and fish mortality. The Episodic Response Project (ERP) study showed that streams with moderate to severe acid episodes had significantly higher fish mortality during bioassays than nonacidic streams (Van Sickle et al., 1996). The concentration of inorganic monomeric Al was the chemical variable most strongly related to mortality in the four test species (brook trout, mottled sculpin, slimy sculpin, and blacknose dace). The latter three species are acid sensitive. In general, trout abundance was lower in ERP streams with median episode pH less than 5.0 and inorganic monomeric Al concentrations greater than 3.7 - 7.4 mmol L^{-1}. Acid sensitive species were absent from streams with median episode pH less than 5.2 and with a concentration of inorganic monomeric Al greater than 3.7 mmol L^{-1}..

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).

Driscoll et al. (2001) envision a recovery process that will involve two phases. Initially, a decrease in acidic deposition following emissions controls will facilitate a phase of chemical recovery in forest and aquatic ecosystems. Recovery time for this phase will vary widely across ecosystems and will be a function of the following:

- the magnitude of decreases in atmospheric deposition
- the local depletion of exchangeable soil pools of base cations
- the local rate of mineral weathering and atmospheric inputs of base cations
- the extent to which soil pools of S and N are released as SO_4^{2-} or as NO_3^- to drainage waters and the rate of such releases (Galloway et al. 1983).

In most cases, it seems likely that chemical recovery will require decades, even with additional controls on emissions. The addition of base cations, e.g., through liming, could enhance chemical recovery at some sites.

The second phase in ecosystem recovery is biological recovery, which can occur only if chemical recovery is sufficient to allow survival and reproduction of plants and animals. The time required for biological recovery is uncertain. For terrestrial ecosystems, it is likely to be at least decades after soil chemistry is restored because of the long life of tree species and the complex interactions of soil, roots, microbes, and soil biota. For aquatic systems, research suggests that stream macroinvertebrate populations may recover relatively rapidly (approximately 3 years), whereas lake populations of zooplankton are likely to recover more slowly (approximately 10 years) (Gunn and Mills 1998). Some fish populations may recover in 5 to 10 years after the recovery of zooplankton populations. Stocking could accelerate fish population recovery (Driscoll et al., 2001)

Projections made using an acidification model (PnET-BGC) indicate that full implementation of the 1990 CAAA will not afford substantial chemical recovery at Hubbard Brook EF and at many similar acid-sensitive locations (Driscoll et al., 2001) . Model calculations indicate that the magnitude and rate of recovery from acidic deposition in the northeastern U.S. are directly proportional to the magnitude of emissions reductions. Model evaluations of policy proposals calling for additional reductions in utility SO_2 and NO_X

emissions, year round emissions controls, and early implementation indicate greater success in facilitating the recovery of sensitive ecosystems (Driscoll et al., 2001).

Indirect Vegetation and Ecosystem Effects from Atmospheric PM

In addition to the direct and indirect effects of deposited PM, ambient atmospheric PM can effect radiation and climate conditions that influence overall plant/ecosystem productivity. The degree to which these effects occur in any given location will depend on the chemical and physical composition and concentration of the ambient PM. Because plants are adapted to the overall light and temperature environments in which they grow, any PM-related changes to these conditions potentially alter the overall competititive success these plants will have in that ecosystem.

With respect to radiation, the characteristics and net receipts of solar and terrestrial radiation determine rates of both photosynthesis and the heat-driven process of water cycling. Atmospheric turbidity (the degree of scattering occurring in the atmosphere due to particulate loading) influences the light environment of vegetative canopy in two ways: through conversion of direct to diffuse radiation and by scattering or reflecting incoming radiation back out into space. Diffuse radiation increases canopy photosynthetic productivity by distributing radiation more uniformly throughout the canopy so that it also reaches the lower leaves and improves the canopy radiation use efficiency (RUE). Acting in the opposite direction, non-absorbing, scattering aerosols present in PM reduce the overall amount of radiation reaching vegetative surfaces, by scattering or reflecting it back into space. It appears that global albedo has been increasing due to an increasing abundance of atmospheric particles. Using World Meteorological Organization (WMO) data, Stanhill and Cohen (2001) have estimated that average solar radiation receipts have declined globally by an average of 20 W m-2 since 1958. The net effect of atmospheric particles on plant productivity is not clear, however, as the enrichment in photosynthetically active radiation (PAR) present in diffuse radiation may offset a portion of the effect of decreased solar radiation receipts in some instances (CD, pp. 4-92, 4-93).

Plant processes also are sensitive to temperature. Some atmospheric particles (most notably black carbon) absorb short-wavelength solar radiation, leading to atmospheric heating and reducing total radiation received at the surface. Canopy temperature and transpirational water use by vegetation are particularly sensitive to long-wave, infrared radiation. Atmospheric

heating by particles can potentially reduce photosynthetic water uptake efficiency and vertical temperature gradients, potentially reducing the intensity of atmospheric turbulent mixing. Stanhill and Cohen (2001) suggested that plant productivity is more affected by changes in evapotranspiration induced by changes in the amount of solar radiation plants receive than by changes in the amount of PAR plants receive (CD, p. 4-93).

6.3.4 Characteristics and Location of Sensitive Ecosystems in the U.S.

Ecosystems sensitive to anthropogenically derived nitrogen and/or acid deposition tend to have similar characteristics. Some of these ecosystems and characteristics have already been mentioned in earlier sections but are repeated here to provide a more comprehensive list that can help ecological risk assessors/managers identify areas of known or potential concern. For example, lower nitrogen and/or resource environments, such as those with infertile soils, shaded understories, deserts, or tundras, are populated with organisms specifically adapted to survive under those conditions. Plants adapted to these conditions have been observed to have similar characteristics, including inherently slower growth rates, lower photosynthetic rates, and lower capacity for nutrient uptake, and grow in soils with lower soil microbial activity. When N becomes more readily available, such plants will be replaced by nitrophilic plants which are better able to use increased amounts of Nr (Fenn et al., 1998).

Additionally, in some instances, there seem to be important regional distinctions in exposure patterns, environmental stressors, and ecosystem characteristics between the eastern and western U.S.. A seminal report describing these distinctive characteristics for the western U.S. (11 contiguous states located entirely west of the 100th meridian) is Fenn et al., 2003.

In the western U.S., vast areas receive low levels of atmospheric deposition, interspersed with hotspots of elevated N deposition downwind of large, expanding metropolitan centers or large agricultural operations. In other words, spatial patterns of urbanization largely define the areas where air pollution impacts are most severe. The range of air pollution levels for western wildlands is extreme, spanning from near-background to the highest exposures in all of North America, with the possible exception of forests downwind of Mexico City. Over the same geographic expanse, climatic conditions and ecosystem types vary widely. Some regions receive

more than 1000 millimeters of precipitation, namely the Pacific coastal areas, the Sierra Nevada, the Colorado Rockies, and northern Idaho, while other regions are arid or semiarid, with more than 300 clear days per year (Riebsame et al., 1997). In these latter regions, the contribution of atmospheric dry deposition is likely to be most important. These characteristics which are unique to the West require special consideration, and often make application of models and ecological effects thresholds developed for other regions inappropriate.

In summary, sensitive or potentially sensitive ecosystems in the west include those that:

- are located downwind of large urban source areas; regions with a mix of emissions sources that may include urban, mobile, agricultural, and industrial sources; and/or sites near large point sources of N.

- contain inherently N sensitive ecosystem components, such as lichens, diatoms, or poorly buffered watersheds which produce high streamwater NO_3- levels. These sensitive components can be affected by N deposition rates as low as 3-8 kg/ha/yr.

- occur on top of siliclastic/crystalline bedrock with little potential for buffering acidity.

- are naturally nitrogen limited. For example, the approximately 16,000 high elevation western mountain lakes are generally oligotrophic and especially sensitive to the effects of atmospheric deposition.

A seminal report describing key characteristics of sensitive ecosystems for the eastern and in particular the northeastern U.S. is Driscoll et al. (2001). In the northeastern United States, atmospheric deposition is largely a regional problem. Because S and N most often occur together in the eastern atmosphere and deposit to the environment as acidic deposition, acidic deposition is seen as a critical environmental stress.

Several critical chemical thresholds appear to coincide with the onset of deleterious effects to biotic resources resulting from acid deposition. Thus, ecosystems sensitive to additional acid inputs include those with the following characteristics:

- a molar Ca:Al ratio of soil water that is less than 1.0;
- soil percentage base cation saturation less than 20%;
- surface water pH less than 6.0;
- ANC less than 50 meq L-1; and
- concentrations of inorganic monomeric Al greater than 2 mmol L-1.

Knowledge of such indicators is necessary for restoring ecosystem structure and function.

6.3.5 Ecosystem Exposures to PM Stressor Deposition

In order for any specific chemical stressor present in ambient PM to impact ecosystems, it must first be removed from the atmosphere through deposition. Deposition can occur in three modes: wet (rain/frozen precipitation), dry, or occult (fog, mist or cloud). At the national scale, 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. (CD, p. 4-8 to 4-10). For example, in large parts of the western U.S. which are arid or semiarid, dry deposition may be the source of most deposited PM (Fenn, et al., 2003). However, in coastal areas or high elevation forests, wet or occult deposition may predominate. Where the latter is the case, deposition levels may greatly exceed PM levels measured in the ambient 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 can be especially at risk from depositional impacts because they 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. 4-29, 4-44).

In order to establish exposure-response profiles useful in ecological risk assessments, two types of monitoring networks need to be in place. First, a deposition network is needed that can track changes in deposition rates of PM stressors (nitrates/sulfates) occurring in sensitive or symptomatic areas/ecosystems. Secondly, a network or system of networks that measure the

response of key ecological indicators sensitive to changes in atmospheric deposition of PM stressors is also needed.

Currently in the U.S., national deposition monitoring networks routinely measure total wet or dry deposition of certain compounds. 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 CAAA, 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; (http://www.epa.gov/castnet/).

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 (http://nadp.sws.uiuc.edu/).

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 section 2.5, 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.

Data from these deposition networks demonstrate that N and S compounds are being deposited onto soils and aquatic ecosystems in sufficient amounts to impact ecosystems at local, regional and national scales. Though the percentages of N and S containing compounds in PM vary spatially and temporally, nitrates and sulfates make up a substantial portion of the chemical composition of PM. In the future, speciated data from these networks may allow better

understanding of the specific components of total deposition that are most strongly influencing PM-related ecological effects.

Unfortunately, at this time there is only limited long-term ecosystem response monitoring taking place at the national level. Two exceptions are the Hubbard Brook Experimental Forest research site, that provides the longest continuous record of precipitation and stream chemistry in the U.S. (Likens and Bormann, 1995) and EPA's LTM and TIME projects which monitor changes in surface water chemistry in the acid sensitive regions of the northern and eastern U.S.. Because the complexities of ecosystem response make predictions of the magnitude and timing of chemical and biotic recovery uncertain, it is strongly recommended that this type of long-term surface water chemistry monitoring network be continued, and that a biological monitoring program be added. Data from these long-term monitoring sites will be invaluable for the evaluation of the response of forested watersheds and surface waters to a host of research and regulatory issues related to acidic deposition, including soil and surface water recovery, controls on N retention, mechanisms of base cation depletion, forest health, sinks for S in watersheds, changes in dissolved organic carbon and speciation of Al, and various factors related to climate change (EPA, 2003).

6.3.6 Critical Loads

The critical load (CL) 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 load 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 pollutant stress.

Key to the establishment of a critical load is the selection of appropriate ecological endpoints or indicators that are measurable characteristics related to the structure, composition, or functioning of ecological systems (i.e., indicators of condition). 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). A number of different indicators for monitoring ecosystem status have been proposed. Indicators of ecosystems at risk of N saturation could include: 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 increased and prolonged NO_3^- loss below the main rooting zone and in stream water or in soil solution; and flux rates of nitrogenous trace gases from soil (Fenn et al., 1998). Seasonal patterns of stream water nitrate concentrations are especially good indicators of watershed N status. Biological indicators that have been suggested for use in the critical load calculation in forest ecosystems 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). Lichen community composition in terrestrial ecosystems or lichen N tissue levels are also fairly responsive to changes in N deposition over time (Fenn et al., 2003). In aquatic systems, diatom species composition can be a good indicator of changes in water chemistry (Fenn et al., 2003). 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).

Within North America, a number of different groups have recently begun to use or develop critical loads. As discussed below, these groups include the U.S. Federal Land Managers (FLMs), such as the National Park Service and the Forest Service, a binational group known as New England Governors/Eastern Canadian Premiers (NEG/ECP), and several Canadian Provinces.

Federal Land Managers have hosted a number of meetings over the last few years to discuss how the CL concept might be used in helping them fulfill their mandate of providing

protection for the lands they manage. In trying to develop a consistent approach to using CL, a number of issues and considerations have been identified. First, the distinction between critical loads (which are based on modeled or measured dose-response data) and target loads (which can be based on political, economic, spatial or temporal considerations in addition to scientific information) needs to be recognized. When using the critical or target load (TL) approach, one must indicate the spatial (or geographic) scope, the temporal scope (timeframe to ecological or ecosystem recovery), and a description of the sensitive receptors (or resource) to be protected, the sensitive receptor indicators (physical, chemical biological, or social characteristics of the receptor that can be measured), and the harmful effect on the receptor that is of concern. Additionally, one would need to specify what is the "desired condition" that the critical or target load is meant to achieve. For any given location, there may be a range or suite of possible critical or target loads based on different sensitive receptors and/or receptor indicators found at that site. Alternatively, one could focus on the most sensitive receptor and select a single CL or TL for that receptor. Several aspects of the CL approach make it attractive for use by the FLMs. Specifically, it can provide a quantitative, objective and consistent approach for evaluating resource impacts. In an effort to progress the CL approach, the Forest Service is testing the applicability of the European protocol to several U.S. case study sites.

Under the auspices of the NEG/ECP, and other binational efforts, Canadian and U.S. scientists are involved in joint forest mapping projects. A Forest Mapping Work Group has been tasked with conducting a regional assessment of the sensitivity of northeastern North American forests to current and projected sulfur and nitrogen emissions levels, identifying specific forested areas most sensitive to continued deposition and estimating deposition rates required to maintain forest health and productivity. They have completed the development of methods, models and mapping techniques, and identification of data requirements. Some of these data requirements include: pollution loading to forest landscapes; the interaction of pollutants with forest canopies; plant nutrient requirements; and the ability of soils to buffer acid inputs and replenish nutrients lost due to acidification.

In addition to the CL measure, they have also defined a "deposition index" as the difference between the CL and current deposition levels. Positive values of the index reflect the

capacity of a forest ecosystem to tolerate additional acidic deposition. Negative index values correspond to the reduction in S and N deposition required to eliminate or deter the development of future nutrient limitations. This allows an assessor to identify areas where the deposition problems are most severe, and which sites might be under the CL level currently but not far from reaching or exceeding that level should deposition levels increase. Currently maps exist for Vermont and Newfoundland, though the goal is to develop maps that will cover Quebec and the Atlantic provinces of Canada, along with the remaining New England states. These maps show that 31% of Vermont forests and 23% of Newfoundland forests are sensitive (e.g., current levels of S and N deposition are causing cation depletion).

Though these current activities hold promise for using the CLs approach in environmental assessments and in informing management decisions, widespread use of CLs in the U.S. is not yet possible. Critical loads is a very data-intensive approach, and, at the present time, there is a paucity of ecosystem- level data for most sites. However, for a limited number of areas which already have a long-term record of ecosystem monitoring, (e.g., Rocky Mountain National Park in Colorado and the Lye Brook Wilderness in Vermont), FLMs may be able to develop site-specific CLs. Further, in areas already exceeding the CL, it may be difficult to determine what the management goals are/should be for each mapped area (e.g., what is the "desired condition" or level of protection) without historic baseline data. More specifically, with respect to PM deposition, there are insufficient data for the vast majority of U.S. ecosystems that differentiate the PM contribution to total N or S depostion 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. Though atmospheric sources of Nr and acidifying compounds, including ambient PM, are clearly contributing to the overall excess pollutant load or burden entering ecosystems annually, insufficient data are available at this time to quantify the contribution of ambient PM to total Nr or acidic deposition as its role varies both temporally and spatially along with a number of other factors. Thus, it is not clear whether a CL could be developed just for the portion of the total N or S input that is contributed by PM.

6.3.7 Summary and Conclusions

The above discussions identify a group of ecosystems known to be sensitive to excess N and S inputs and a list of characteristics that can be used to predict or locate other potentially sensitive ecosystems within the U.S. Further, exposures of these sensitive ecosystems to atmospherically derived pollutants (e.g., N and S) have been measured and documented, in some cases for decades. Clear linkages between reduced atmospheric concentrations of these pollutants and reduced deposition rates have been made. The mechanisms of environmental and ecosystem responses to these inputs are increasingly understood, though very complex. Fertilization and acidification studies have verified observed ecosystem responses to these pollutants in the field. Ecosystem-level effects associated with excess N and S inputs are profound, but in most cases potentially reversible. New assessment and management tools, such as critical and target loads, are being developed to better characterize the relationship between deposition loads and ecosystem response. The success of these tools will depend on the availability of sufficient ecosystem response data, which is currently limited to a few long-term monitoring networks/sites (e.g., TIME/LTM). The current risk to sensitive ecosystems and especially sensitive species like the checkerspot butterfly, desert tortoise, epiphytic lichens, native shrub and forb species, and aquatic diatom communities is high. The loss of species and whole ecosystem types is adverse and should receive increased protection.

A number of ecosystem-level conditions (e.g., nitrogen saturation, terrestrial and aquatic acidification, coastal eutrophication) have been associated with chronic, long-term exposure of ecosystems to elevated inputs of compounds containing Nr, sulfur and/or associated hydrogen ions. These ecosystem level changes profoundly impact almost all of the EEAs identified in the EPEC Framework (SAB, 2002) and described in sections 4.2.1 and 4.2.3 of the CD. These impacted EEAs include Landscape Condition, Biotic Condition, Chemical and Physical Characteristics, Ecological Processes, and Natural Disturbance Regimes. Given that humans, as well as other organisms, are dependent on the services ecosystems provide, ecosystem changes of this magnitude are of concern and can lead to adverse impacts on human health and welfare.

Based on the information included in the above discussions and Chapters 4 and 9 of the CD, staff has reached the following conclusions:

- An ecologically-relevant indicator for PM would be based on one or multiple chemical stressors found in ambient PM (e.g. N or S containing compounds).

- Ecosystem effects can be associated with long-term high or even low levels of excess inputs. Thus, there is no bright line or threshold for effects, but rather a "syndrome" of complex changes over time. Additionally, ecosystem recovery can occur but may take decades, and may require controls beyond those already established.

- Excess N or acid deposition acts in conjunction with other co-occurring stresses (e.g., invasive species, reduced grazing pressure) that jointly determine ecological outcomes. Therefore, these pollution-related stresses should not be considered in isolation. Additionally, all forms of airborne nitrogen and acidic compounds need to be considered and managed in harmony.

- Monitoring networks may be sufficient to measure air concentrations or deposition but are not generally sufficient to monitor ecosystem response. For example, in the West, more environmental monitoring is needed downwind of large urban areas.

Unfortunately, our ability to relate ambient concentrations of PM to ecosystem response is hampered by a number of significant data gaps and uncertainties. First, U.S. monitoring networks have only recently begun to measure speciated PM. Historically, measurements were focused only on a particular size fraction such as PM_{10} and, more recently, $PM_{2.5}$. An exception to this is the IMPROVE network, which collects speciated measurements. Additionally, except for the IMPROVE and some CASTNet sites, much of the PM monitoring effort has focused on urban or near urban exposures, rather than on those in sensitive ecosystems. Thus, the lack of a long-term, historic database of annual speciated PM deposition rates precludes establishing relationships between PM deposition (exposure) and ecosystem response at this time. As a result, 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.

A second source of uncertainty lies in predicting deposition velocities based on ambient concentrations of PM. There are a multitude of factors that influence the amounts of PM that get deposited from the air onto sensitive receptors, including the mode of deposition (wet, dry, and occult), wind speed, surface roughness or stickiness, elevation, particle characteristics (e.g., size,

shape, chemical composition), and relative humidity. Therefore, modeled deposition rates, used in the absence of monitored data, can be highly uncertain.

Third, each ecosystem has developed within a context framed by the topography, underlying bedrock, soils, climate, meteorology, hydrologic regime, natural and land use history, species associations that co-occur at that location (e.g., soil organisms, plants), and successional stage, making it unique from all others. Because of this variety, and insufficient baseline data on each of these features for most ecosystems, it is currently not possible to extrapolate with much confidence any effect from one ecosystem to another, or to predict an appropriate "critical load" for the vast majority of U.S. ecosystems.

As additional PM speciated air quality and deposition monitoring data become available, there is much room for fruitful research into the areas of uncertainty identified above. At this time, however, staff concludes that there is insufficient information available to recommend for consideration an ecologically defined secondary standard that is specifically targeted for protection of vegetation and ecosystems against the adverse effects potentially associated with the levels of PM-related stressors of nitrate and sulfate found in the ambient air.

6.4 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 (section 4.4) of the CD, the following sections summarize the physical damage and aesthetic soiling effects of PM on materials including metals, paint finishes, and stone and concrete.

6.4.1 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 4-193).

The CD (p. 4-194, 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 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. 4-196).

With respect to damage to building stone, numerous studies discussed in the CD (pp. 4-196 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. 4-200).

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. 4-201 , 4-202).

6.4.2 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. 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).

6.4.3 Summary and Conclusions

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. Thus, staff concludes that PM effects on materials can play no quantitative role in considering whether any revisions of the secondary PM NAAQS are appropriate at this time.

6.5 EFFECTS ON CLIMATE CHANGE AND SOLAR RADIATION

Atmospheric particles alter the amount of electromagnetic radiation transmitted through the earth's atmosphere by both scattering and absorbing radiation. As discussed above in Chapter 2 (section 2.2.6), 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) absorb incoming solar radiation or outgoing terrestrial 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 and the effects on human health and the environment associated with these atmospheric processes is briefly summarized below, based on the information in section 4.5 of the 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.

6.5.1 Climate Change and Potential Human Health and Environmental Impacts

As discussed in section 4.5.1 of the 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.

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. As discussed in Section 4.5.1.2 of the 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 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. 216). The 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. 4-240).

More specifically, the CD 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 (CD, p. 4-207). 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, calculations of indirect physical effects of particles on climate (e.g., related to alteration of cloud properties and disruption of hydrological cycles) are subject to much larger uncertainties than those related to the direct radiative effects of particles (CD, p. 4-219). The CD concludes that at present 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 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.

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

As discussed in section 4.5.2 of the CD, 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. Several studies cited in the CD 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. 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.

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 (CD, p. 4-221). 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 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 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, 4-226).

At present, models are not available to take such complex factors into account, nor is sufficient data available to characterize input variables that would be necessary for any such modeling. The CD concludes, however, that the outcome of such modeling efforts would likely vary from location to location, even as to the direction of changes in the levels of exposures to UV-B radiation, due to location-specific changes in ambient PM concentrations and/or composition (CD, p. 4-227). Beyond considering just average levels of exposures to UV-B radiation in general, the CD notes that ambient PM can affect the directional characteristics of UV-B radiation scattering at ground-level, and thus its biological effectiveness. Also, ambient PM can affect not only biologically damaging UV-B radiation, but can also reduce the ground-level ratio of photorepairing UV-A radiation to damaging UV-B radiation. Further, the CD notes

that ambient PM deposition is a major source of PAH in certain water bodies, which can enhance the adverse effects of solar UV-B radiation on aquatic organisms, such that the net effect of ambient PM in some locations may be to increase UV-B radiation-related biological damage to certain aquatic and terrestrial organisms. (CD, p. 4-227).

6.5.3 Summary and Conclusions

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, staff concludes that 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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7. STAFF CONCLUSIONS AND RECOMMENDATIONS ON SECONDARY PM NAAQS

7.1 INTRODUCTION

This chapter presents staff conclusions and recommendations for the Administrator to consider in deciding whether the existing secondary PM standards should be revised and, if so, what revised standards are appropriate.[1] The existing suite of secondary PM standards, which is identical to the suite of primary PM standards, includes annual and 24-hour $PM_{2.5}$ standards and annual and 24-hour PM_{10} standards to address visibility impairment associated with fine particles and materials damage and soiling related to both fine and coarse particles. Each of these standards is defined in terms of four basic elements: indicator, averaging time, level and form. Staff conclusions and recommendations on these standards are based on the assessment and integrative synthesis of information related to welfare effects presented in the CD and on staff analyses and evaluations presented in Chapters 2 and 6 herein.

In recommending a range of secondary standard options for the Administrator to consider, staff notes that the final decision is largely a public policy judgment. A final decision must draw upon scientific evidence 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 relevant information. The NAAQS provisions of the Act require the Administrator to establish secondary standards that are requisite to protect public welfare[2] from any known or anticipated adverse effects associated with the presence of the pollutant in the ambient air. 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 secondary standards be set to eliminate all

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[1] As noted in Chapter 1, staff conclusions and recommendations presented herein are provisional; final staff conclusions and recommendations, to be included in the final version of this document, will be informed by comments received from CASAC and the public in their reviews of this draft document.

[2] As noted in Chapter 1, 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."

welfare effects, but rather at a level requisite to protect public welfare from those effects that are judged to be adverse.

7.2 APPROACH

Similar to the approach discussed in Chapter 5, section 5.2, for the review of the primary NAAQS, staff's approach here can be framed by a series of questions that may be applicable for each category of PM-related welfare effects identified in the CD as being associated with the presence of the pollutant in the ambient air. Staff's review of the adequacy of the current PM standards for each effects category involves addressing questions such as:

- To what extent does the available information demonstrate or suggest that PM-related effects are occurring at current ambient conditions or at levels that would meet the current standards?
- To what extent does the available information inform judgments as to whether any observed or anticipated effects are adverse to public welfare?
- To what extent are the current secondary standards likely to be effective in achieving protection against any identified adverse effects?

To the extent that the available information suggests that revision of the current secondary standards would be appropriate for an effects category, staff then identifies ranges of standards (in terms of indicators, averaging times, levels, and forms) that would reflect a range of alternative policy judgments as to the degree of protection that is requisite to protect public welfare from known or anticipated adverse effects. In so doing, staff addresses questions such as:

- Does the available information provide support for considering different PM indicators?
- Does the available information provide support for considering different averaging times?
- What range of levels and forms of alternative standards is supported by the information, and what are the uncertainties and limitations in that information?
- To what extent would specific levels and forms of alternative standards reduce adverse impacts attributable to PM, and what are the uncertainties in the estimated reductions?

Based on the available information, estimated reductions in adverse impacts, and related uncertainties, staff makes recommendations as to ranges of alternative standards for the Administrator's consideration in reaching decisions as to whether to retain or revise the secondary PM NAAQS.

In presenting this approach, staff well recognizes that for some welfare effects the currently available information falls short of what is considered sufficient to serve as a basis for a distinct standard defined specifically in terms of the relationship between ambient PM and that effect. In the case of visibility impairment, however, the available information may well provide a basis for a distinctly defined standard. In either case, staff believes it is appropriate to consider the extent to which the current or recommended primary standards may afford protection against the identified welfare effects.

Staff first considers information related to the effects of ambient PM, especially fine particles, on visibility impairment in section 7.3, and makes recommendations that consideration be given to a revised $PM_{2.5}$ standard. Other PM-related welfare effects, including effects on vegetation and ecosystems, materials, and global climate change processes, are addressed in section 7.4. This chapter concludes with a summary of key uncertainties associated with establishing secondary PM standards and related staff research recommendations in section 7.5.

7.3 STANDARDS TO ADDRESS VISIBILITY IMPAIRMENT

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 future 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 fine particles, 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 in 1997, EPA recognized, based on observations from available monitoring data, primarily from rural sites in the IMPROVE monitoring network, that the selection of an appropriate level for a national secondary standard to address visibility protection was complicated by regional differences in visibility impairment. These differences were 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 to protect against visibility conditions generally considered to be adverse in all areas.

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 anticipated 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 regional planning organizations have been formed and are now developing strategies, to be adopted over the next few years, that will make reasonable progress in meeting these goals.

7.3.1 Adequacy of Current $PM_{2.5}$ Standards

In considering the information now available in this review, as discussed in Chapters 2 and 6 (section 6.2), 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, especially fine particles, in visibility impairment from the last review. However, extensive new information now available from visibility and fine particle monitoring networks has allowed for updated characterizations of visibility trends and current levels in urban areas, as well as Class I areas. These new data are a critical component of the analysis presented in section 6.2.3 that better characterizes visibility impairment in urban areas.

Based on this information, staff has first considered the extent to which available information shows PM-related impairment of visibility at current ambient conditions in areas across the U.S. Taking into account the most recent monitoring information and analyses, 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).

- In urban areas, visibility levels show far less difference between eastern and western regions. For example, based on reconstructed light extinction values calculated from 24-hour average $PM_{2.5}$ concentrations, the average visual ranges on the 20% haziest days in eastern and western urban areas are approximately 21 km and 28 km, respectively. Even

more similarity is seen in considering 4-hour (12:00 to 4:00 pm) average $PM_{2.5}$ concentrations, for which the average visual ranges on the 20% haziest days in eastern and western urban areas are approximately 26 km and 30 km, respectively. (Schmidt et al., 2005)

Based on this information, and on the recognition that efforts are now underway to address all human-caused visibility impairment in Class I areas through the regional haze program implemented under sections 169A and 169B of the Act, as discussed above, staff has focused in this review on visibility impairment primarily in urban areas. In so doing, staff has considered whether information now available can inform judgments as to the extent to which existing levels of visibility impairment in urban areas can be considered adverse to public welfare. In so doing, staff has looked at studies in the U.S. and abroad that have provided the basis for the establishment of standards and programs to address specific visibility concerns in local areas, as discussed in section 6.2.5. These studies 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 particle pollution levels and environmental conditions. As discussed in section 6.2.6, methods involving the use of surveys 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, which ranged from approximately 40 to 60 km in visual range.

Beyond the information available from such programs, staff believes it is appropriate to make use directly 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 6.2.6, photographic representations of varying levels of visual air quality have been developed for several urban areas 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, and Phoenix (for which $PM_{2.5}$ concentrations are reported), 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 $PM_{2.5}$ concentrations below 35 to 40 $\mu g/m^3$, or at visual ranges generally above 20 km for the urban areas considered.

While being mindful of the limitations in using visual representations from a small number of areas as a basis for considering national visibility-based secondary standards, staff nonetheless concludes that the observations discussed above support consideration of revising the current $PM_{2.5}$ secondary standards to enhance visual air quality, particularly with a focus on urban areas. Thus, in the sections that follow, staff evaluates information related to indicator, averaging time, level and form to identify a range of alternative PM standards for consideration that would protect visual air quality, primarily in urban areas.

7.3.2 Indicators

As discussed in Chapter 2, section 2.8, fine particles contribute to visibility impairment directly in proportion to their concentration in the ambient air. Hygroscopic components of fine particles, in particular sulfates and nitrates, contribute disproportionately to visibility impairment under high humidity conditions, when such components can reach particle diameters up to and even above 2.5 μm. Particles in the coarse mode generally contribute only marginally to visibility impairment in urban areas. Thus, fine particles, as indexed by $PM_{2.5}$, are an appropriate indicator of PM pollution to consider for the purpose of standards intended to address visibility impairment.

In analyzing how well $PM_{2.5}$ concentrations correlate with visibility in urban locations across the U.S., as discussed above in section 6.2.3 and in more detail in Schmidt et al. (2005), staff concludes that the observed correlations are strong enough to support the use of $PM_{2.5}$ as the indicator for such standards. More specifically, clear correlations exist between 24-hour average $PM_{2.5}$ concentrations and reconstructed light extinction (RE), which is directly related to visual range, and these correlations are similar in eastern and western regions. These correlations are less influenced by relative humidity and more consistent across regions when $PM_{2.5}$

concentrations are averaged over shorter, daylight time periods (e.g., 4 to 8 hours). Thus, staff concludes that it is appropriate to use $PM_{2.5}$ as an indicator for standards to address visibility impairment in urban areas, especially when the indicator is defined for a relatively short period of daylight hours.

7.3.3 Averaging Times

In considering appropriate averaging times for a standard to address visibility impairment, staff has considered averaging times that range from 24 to 4 hours, as discussed in section 6.2.3. Within this range, as noted above, correlations between $PM_{2.5}$ concentrations and RE are generally less influenced by relative humidity and more consistent across regions as the averaging time gets shorter. Based on the regional and national average statistics considered in this analysis, staff observes that in the 4-hour time period between 12:00 and 4:00 p.m., the slope of the correlation between $PM_{2.5}$ concentrations and hourly RE is lowest and most consistent across regions than for any other 4-hour or longer time period within a day (Chapter 6, Figure 6-4). Staff also recognizes that these advantages remain in looking at a somewhat wider time period, from approximately 10:00am to 6:00 pm. Staff concludes that an averaging time from 4 to 8 hours, generally within the time period from 10:00 am to 6:00 pm, should be considered for a standard to address visibility impairment.

In reaching this conclusion, staff recognizes that the national $PM_{2.5}$ FRM monitoring network provides 24-hour average concentrations, such that implementing a standard with a less-than-24-hour averaging time would necessitate the use of continuous monitors that can provide hourly time resolution. Given that the data used in the analysis discussed above are from commercially available $PM_{2.5}$ continuous monitors, such monitors clearly could provide the hourly data that would be needed for comparison with a potential visibility standard with a less-than-24-hour averaging time. Decisions as to which $PM_{2.5}$ continuous monitors are providing data of sufficient quality to be used in a visibility standard would follow protocols for approval of reference and equivalent methods that can provide data in at least hourly intervals. Development of the criteria for approval of these reference or equivalent methods for support of a visibility standard would be based upon a data quality objective process that considers

uncertainties associated with the measurement system and the level of the standard under consideration.

7.3.4 Alternative $PM_{2.5}$ Standards to Address Visibility Impairment

In considering alternative short-term (4- to 8-hour) $PM_{2.5}$ standards that would provide requisite protection against PM-related impairment of visibility primarily in urban areas, staff has taken into account the results of public perception and attitude surveys in the U.S. and Canada, State and local visibility standards within the U.S., and visual inspection of photographic representations of several urban areas across the U.S. Staff believes that these sources provide a basis for bounding a range of levels appropriate for consideration in setting a national visibility standard primarily for urban areas.

As discussed above in section 6.2, public perception and attitude surveys conducted in Denver, CO and Phoenix, AZ resulted in judgments reflecting the acceptability of a visual range of approximately 50 and 40 km, respectively. A similar survey approach in the Fraser Valley in British Columbia, Canada reflected the acceptability of a visual range of 40 to 60 km. Visibility standards established for the Lake Tahoe area in California and for areas within Vermont are both targeted at a visual range of approximately 50 km. Staff notes that, in contrast to this convergence of standards and goals around a visual range from 40 to 60 km, California's long-standing general state-wide visibility standard is a visual range of approximately 16 km. Staff believes that consideration should be given to national visibility standards for urban areas across the U.S. that are somewhat less stringent than local standards and goals set to protect scenic resources in and around certain urban areas that are particularly highly valued by people living in those areas, suggesting an upper end of the range of consideration below 40 km.

Staff has also inspected the photographic representations of varying levels of visual air quality that have been developed for Washington, D.C., Chicago, Phoenix, and Denver (available on EPA's website, http:/www.epa.gov/ttn/naaqs/standards/pm/s_pm_cr_sp.html, as an attachment to this document). Staff observes that scenic views (e.g., historic monuments, lake front and mountain vistas) depicted in these images (around and within the three urban areas for which $PM_{2.5}$ concentrations are reported) are significantly obscured from view at $PM_{2.5}$ concentrations of 35 to 40 $\mu g/m^3$ in Chicago, Washington, D.C., and Phoenix, corresponding to

reported visual ranges in Washington, D.C. and Phoenix of 12 to 20 km, respectively. Staff also observes that visual air quality appears to be good in these areas at $PM_{2.5}$ concentrations generally below 20 $\mu g/m^3$, corresponding to reported visual ranges in Washington, D.C. and Phoenix above approximately 25 to 35 km, respectively. In looking at the images in Denver, staff observes that visual air quality appears to be generally good, specifically in terms of the ability to view nearby mountain ranges, at a visual range above 52 km. These observations are interpreted by staff as suggesting consideration of a national visibility standard in the range of 30 to 20 $\mu g/m^3$. The upper end of this range is below the levels at which scenic views are significantly obscured, and the lower end is around the level at which visual air quality generally appeared to be good in these areas. Staff recognizes that the above observations about visual air quality in urban areas inherently take into account the nature and location of scenic views that are notable within and around a given urban area, which has implications for the appropriate design of an associated monitoring network.

Building upon the analysis discussed above in section 6.2.3, staff has characterized the distributions of $PM_{2.5}$ concentrations, 4-hour averages in the 12:00 to 4:00 pm time frame, by region, that correspond to various visual range target levels. The results are shown in Figure 7-1, panels (a) through (c), for visual range levels of 25, 30, and 35 km, respectively. This figure shows notable consistency across regions in the median concentrations that correspond to the target visual range level, with what more variation in regional mean values as well as notable variation within each region. In focusing on the median values, staff observes that 4-hour average $PM_{2.5}$ concentrations of approximately 30, 25, and 20 $\mu g/m^3$ correspond to the target visual range levels of 25, 30, and 35 km, respectively. Thus, a standard set within the range of 30 to 20 $\mu g/m^3$ can be expected to correspond generally to median visual range levels of approximately 25 to 35 km in urban areas across the U.S.. Staff notes, however, that a standard set at any specific $PM_{2.5}$ concentration will necessarily result in visual ranges that vary somewhat in urban areas across the country, reflecting in part the less-than-perfect correlation between $PM_{2.5}$ concentrations and reconstructed light extinction. Staff also notes that the range of $PM_{2.5}$ concentrations from 30 to 20 $\mu g/m^3$, suggested by staff's analysis and observations of photographic representations, is generally consistent with national target visual range levels

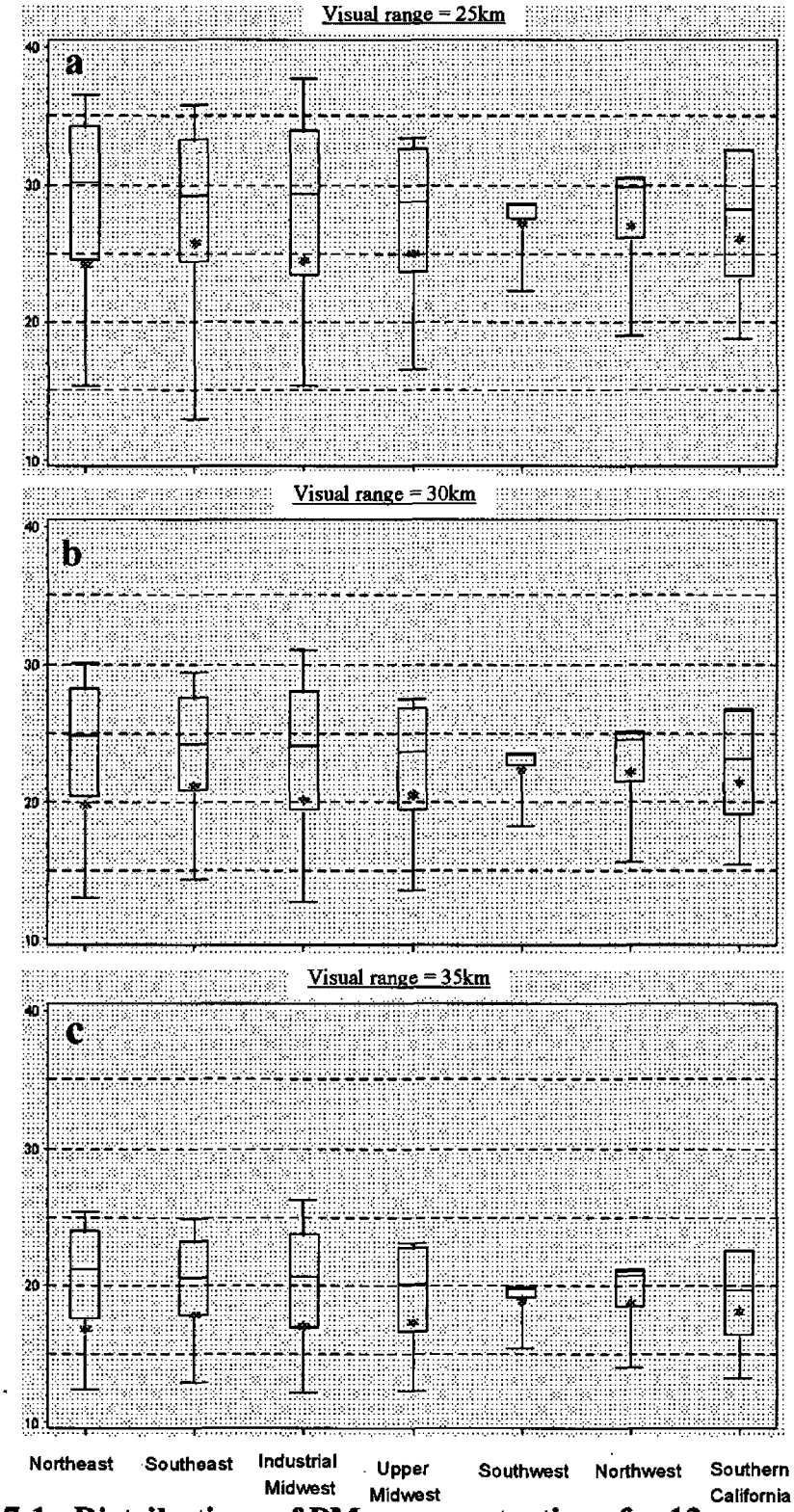

Figure 7-1. Distributions of $PM_{2.5}$ concentrations for 12 p.m. – 4 p.m. corresponding to visual ranges of 25km (panel a) , 30km (panel b), and 35km (panel c) – by region. Box depicts interquartile range and median; whiskers depict 5th and 95th percentiles; star denotes mean.

Source: Schmidt et al. (2005)

below 40 km, the level suggested by the public perception surveys and the local visibility standards and goals discussed above.

In considering a standard down to 20 μg/m^3, staff has again looked to information on $PM_{2.5}$ background concentrations, as was done in considering primary $PM_{2.5}$ standard levels in Chapter 5, section 5.3.5. In both instances, staff recognizes that an appropriate standard level intended to provide protection from man-made pollution should be clearly above background levels. In considering background levels in conjunction with a primary standard, staff focused on the 99th percentile of the distribution of estimated background levels; consistent with consideration of a 98th or 99th percentile form for a primary standard, concluding in that case that 25 μg/m^3 was an appropriate lower end to the range of 24-hour primary $PM_{2.5}$ standards for consideration. For reasons discussed below, staff believes that a lower percentile form would be appropriate to consider for a visibility standard, and thus has looked to a lower percentile in the distribution of estimated background levels as a basis for comparison with the lower end of the range of short-term secondary $PM_{2.5}$ standards for consideration. As discussed in Chapter 2, section 2.6, staff notes that, while long-term average daily $PM_{2.5}$ background levels are quite low (ranging from 1 to 5 μg/m^3 across the U.S.), the estimated 90th percentile values in distributions of daily background levels are appreciably higher, but generally well below 15 μg/m^3, with levels below 10 μg/m^3 in most areas, and these levels may include some undetermined contribution from anthropogenic emissions (Langstaff, 2005). In addition, staff again notes that even higher daily background levels result from episodic occurrences of extreme natural events (e.g., wildfires, global dust storms), but levels related to such events are generally excluded from consideration under EPA's natural events policy, as noted in section 2.6. Taking these considerations into account, staff believes that 20 μg/m^3 is an appropriate lower end to the range of short-term $PM_{2.5}$ standards for visibility protection for consideration in this review.

As in the last review, staff believes that a national visibility standard should be considered in conjunction with the regional haze program as a means of achieving appropriate levels of protection against PM-related visibility impairment in urban, non-urban, and Class I areas across the country. Staff recognizes that programs implemented to meet a national standard focused primarily on urban areas can be expected to improve visual air quality in surrounding non-urban areas as well, as would programs now being developed to address the

requirements of the regional haze rule established for protection of visual air quality in Class I areas. 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. Based on these considerations, and taking into account the observations and analysis discussed above, staff concludes that consideration should be given to a short-term (4- to 8-hour daylight average) secondary $PM_{2.5}$ standard in the range of 30 to 20 $\mu g/m^3$ for protection of visual air quality primarily in urban areas.

7.3.5 Alternative Forms of a Short-term $PM_{2.5}$ Standard

In considering an appropriate form for a short-term $PM_{2.5}$ standard for visibility, staff has taken into account the same general factors that were taken into account in considering an appropriate form for the primary $PM_{2.5}$ standard, as discussed above in Chapter 5, section 5.3.6. In that case, as in the last review, staff has concluded that a concentration-based form should be considered because of its advantages over the previously used expected-exceedance form[3]. One such advantage is that a concentration-based form is more reflective of the impacts 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. Staff notes that the same advantage would apply for a visibility standard as to a health-based standard, in that it would give proportionally greater weight to days when PM-related visibility impairment is substantially higher than to days just above the standard. Further, staff recognizes that 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. Taking these factors into account, staff concludes that consideration should be given to a percentile-based form for a visibility standard.

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[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.

To identify a range of concentration percentiles that would be appropriate for consideration, staff first concludes that constraints on the number of days in which a standard can be exceeded should be appreciably tighter for a standard intended to protect against serious health effects than would be appropriate for a standard intended to protect against visibility impairment, as noted above. Thus, staff believes that the upper end of the range of consideration should be below the 98th to 99th percentiles being considered for a 24-hour primary $PM_{2.5}$ standard. Staff has also considered that the regional haze program targets the 20% most impaired days for improvements in visual air quality in Class I areas. If a similar target of the 20% most impaired days were judged to be appropriate for protecting visual air quality in urban areas, a percentile well above the 80th percentile would be appropriate to increase the likelihood that days in this range would be improved by control strategies intended to attain the standard. A focus on improving the 20% most impaired days suggests to staff that the 90th percentile, which represents the middle of the distribution of the 20% worst days, would be an appropriate form.

To assist in understanding the implications of alternative percentile forms in combination with alternative levels of a standard, staff assessed the percentage of days estimated to exceed various $PM_{2.5}$ concentrations in counties across the U.S., as shown in Figure 7-2. This analysis is based on 2001 to 2003 air quality data, using the 4-hour average concentration from 12:00 to 4:00 pm at the maximum monitor in each county. This assessment is intended to provide some rough indication of the breadth of additional protection potentially afforded by alternative percentile forms for a given standard level: Staff notes that a 90th percentile form, averaged over 3 years, that allows 10% of the days to be above the level of the standard provides additional protection of visual air quality in far fewer areas at a standard level of 30 $\mu g/m^3$ than at a level of 20 $\mu g/m^3$.

Based on the factors discussed above, staff concludes that a percentile-based form should be considered, based on a percentile at or somewhat above the 90th percentile. Staff believes that a form selected from within this range could provide an appropriate balance between adequately limiting the occurrence of peak concentrations and providing for a relatively stable standard.

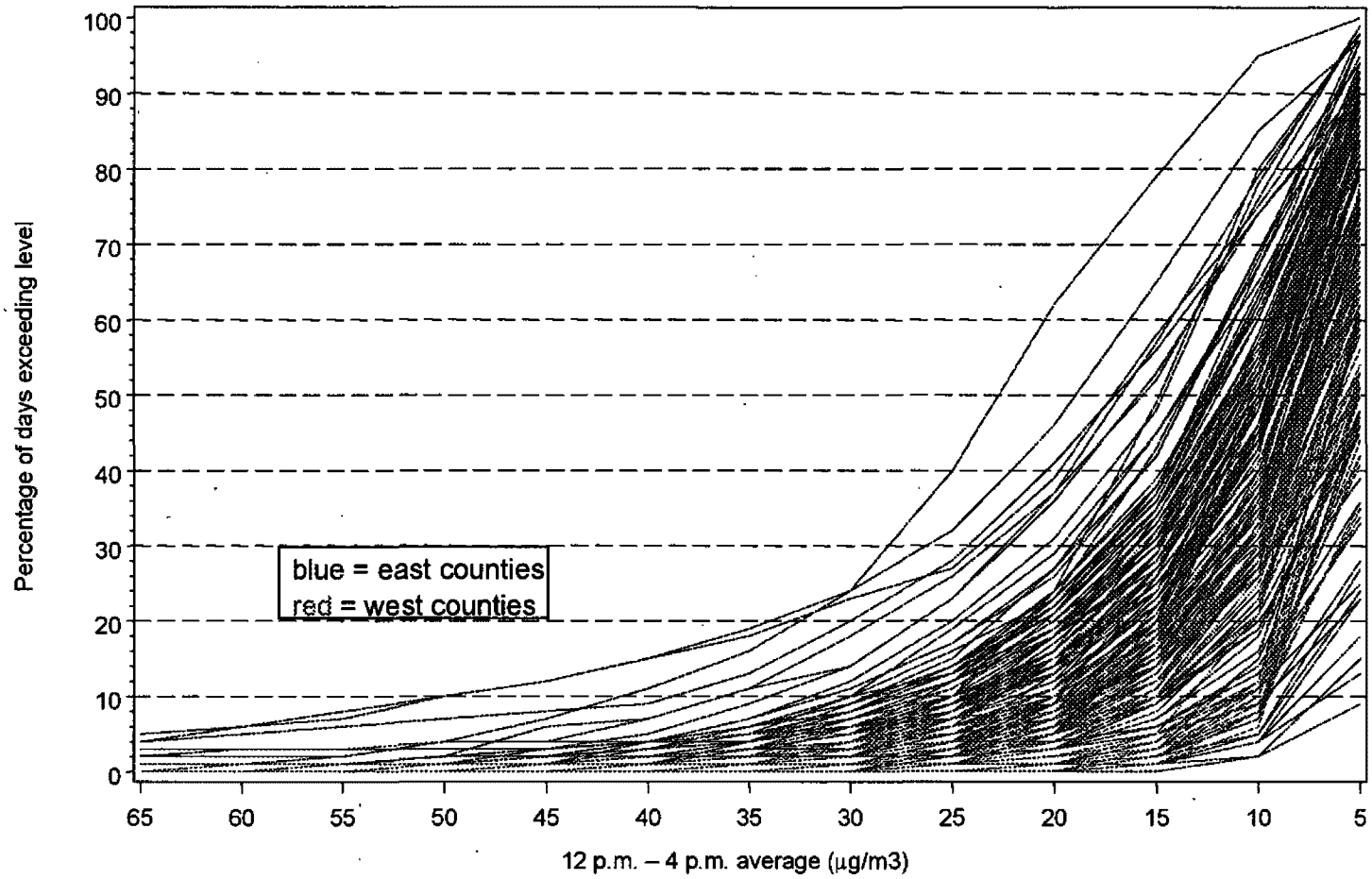

Figure 7-2. Estimated exceedances (%) of various $PM_{2.5}$ levels for 12 p.m. - 4p.m. based on daily county maximum, 2001-2003.

Source: Schmidt et al. (2005)

7.3.5 Summary of Staff Recommendations

Staff recommendations for the Administrator's consideration in making decisions on the secondary $PM_{2.5}$ standards to address PM-related visibility impairment, together with supporting conclusions from sections 7.3.1 through 7.3.4, are briefly summarized below. Staff recognizes that selecting from among alternative standards will necessarily reflect consideration of the qualitative and quantitative uncertainties inherent in the relevant information. In making the following recommendations, staff is mindful that the Act requires secondary standards to be set that are requisite to protect public welfare from those effects that are judged to be adverse, such that the standards are neither more nor less stringent than necessary. The provisions do not require that secondary standards be set to eliminate all welfare effects.

(1) Consideration should be given to revising the current suite of secondary $PM_{2.5}$ standards to provide increased and more targeted protection primarily in urban areas from visibility impairment related to fine particles.

(2) The indicator for a fine particle visibility standard should be $PM_{2.5}$, reflecting the strong correlation between short-term average $PM_{2.5}$ in urban areas across the U.S. and light extinction, which is a direct measure of visibility impairment.

(3) Consideration should be given to a short-term averaging time for a $PM_{2.5}$ standard, within the range of 4 to 8 hours, within a daylight time period between approximately 10:00 am to 6:00 pm. To facilitate implementation of such a standard, consideration should be given to the adoption of FEMs for appropriate continuous methods for the measurement of short-term average $PM_{2.5}$ concentrations.

(4) Consideration should be given to alternative $PM_{2.5}$ standards to provide protection against visibility impairment primarily in urban areas. This recommendation reflects the recognition that programs implemented to meet such a standard can be expected to improve visual air quality in non-urban areas as well, just as programs now being developed to address the requirements of the regional haze rule, for protection of visual

air quality in Class I areas, can also be expected to improve visual air quality in some urban areas. Recommendations on ranges of alternative levels and forms for such a standard include:

(a) Staff recommends consideration of a 4- to 8-hour $PM_{2.5}$ standard within the range of 30 to 20 $\mu g/m^3$. Staff judges that a standard within this range could provide an appropriate degree of protection against visibility impairment, generally resulting in a visual range of approximately 25 to 35 km, primarily in urban areas, as well as improved visual air quality in surrounding non-urban areas.

(b) Staff also recommends consideration of a percentile-based form for such a standard, focusing on a range at or somewhat above the 90th percentile of the annual distribution of daily short-term $PM_{2.5}$ concentrations, averaged over 3 years.

7.4 STANDARDS TO ADDRESS OTHER PM-RELATED WELFARE EFFECTS

EPA's decision in 1997 to revise the suite of secondary PM standards took into account not only visibility protection, but also materials damage and soiling, the other PM-related welfare effect considered in the last review. 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 7.3, 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 consider whether the reductions in fine and coarse particles likely to result from the suite of primary PM 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, in addition to addressing visibility impairment, the CD has broadened its scope to include effects on ecosystems and vegetation, discussed in Chapter 6, section 6.3, and also addresses PM-related effects on materials, discussed in section 6.4, and the role of ambient PM in atmospheric processes associated with climate change and the transmission of solar radiation, discussed in section 6.5. 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, on the basis of the evaluation of the information discussed in Chapter 6, which highlighted the substantial 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 in the following sections whether the reductions in fine and coarse particles likely to result from the current secondary standards, or the range of recommended revisions to the primary standards and the secondary $PM_{2.5}$ standard to address visibility impairment, would provide appropriate protection against these other PM-related welfare effects.

7.4.1 Vegetation and Ecosystems

With regard to PM-related effects on ecosystems and vegetation, staff notes that the 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 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.

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 to the prevention of 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 may 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 revisions to the 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 secondary PM 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.

7.4.2 Materials Damage and Soiling

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 secondary $PM_{2.5}$ standards or revising those standards to be consistent with any revisions made to the primary $PM_{2.5}$ standards or to the secondary $PM_{2.5}$ standards to address visibility impairment, 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.

7.4.3 Climate Change and Solar Radiation

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 6.5, 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 secondary PM standards are appropriate at this time based on ambient PM's role in atmospheric processes related to climate or the transmission of solar radiation.

7.4.4 Summary of Staff Recommendations

Taking into account the conclusions presented in sections 7.4.1 through 7.4.3 above, staff makes the following recommendations with regard to PM-related effects on vegetation and ecosystems and materials damage and soiling:

(1) Consideration should be given to retaining secondary standards for fine and coarse particles that at a minimum retain the level of protection afforded by the current $PM_{2.5}$ and PM_{10} standards so as to continue control of ambient particles, especially long-term deposition of particles, especially particulate nitrates and sulfates, that contribute to adverse impacts on vegetation and ecosystems and materials damage and soiling.

(2) For consistency with the primary standards, secondary standards for fine and coarse particles should be indexed by $PM_{2.5}$ and $PM_{10-2.5}$. While staff recognizes that PM-related impacts on vegetation and ecosystems in particular are associated with chemical components in either size fraction rather than with particle size per se, staff also recognizes that sufficient information is not available at this time to recommend consideration of an ecologically based indicator in terms of a specific chemical component of PM.

In making these recommendations, staff has taken into account both the available evidence linking fine and coarse particles with effects on vegetation and ecosystems and material damage and soiling, 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 primary PM standards.

7.5 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 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.

With regard to welfare-related effects, discussed in Chapter 4 of the CD and Chapter 6 herein, staff has identified the following key uncertainties and research questions that have been highlighted in this review of the welfare-based secondary standards:

(1) 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 reviews of a visibility-based secondary standard. 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.

(2) 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 nitrogen deposition in particular. 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.

(3) 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.[4]

[4] This recommendation is consistent with the views of the National Research Council (NRC) contained in its recent review of air quality management in the U.S. (NRC, 2004). This report recognizes that for some resources at risk from air pollutants, including soils, groundwaters, surface waters, and coastal ecosystems, a deposition-based standard could be appropriate, and identifies "critical loads"as one potential approach for establishing such a deposition-based standard.

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APPENDIX 2A. 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. Episodic emissions from dust storms and forest fires are difficult to quantify and to allocate accurately in space and time, and discerning between natural and anthropogenic "causality" for these source categories is especially challenging.

Table 2A-1 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.

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 (total of all sources) shown in Table 2A-1 are about 2.5 times higher than estimates of $PM_{2.5}$ emissions – 16.3 million short tons compared to 6.6 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.

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 2A-1). 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 2A-1, direct emissions from fuel combustion, industrial processes, fires, and motor vehicles contribute more to primary $PM_{2.5}$ than to primary $PM_{10-2.5}$. Recent improvements to methodologies for estimating emissions, reflected in the values in Table A-1, include:

- Wildfires and prescribed burning - use of state-specific fuel loading factors and improved emission factors
- Residential wood combustion (woodstoves & fireplaces) - recalculation of emissions using updated wood consumption data
- Condensible PM emissions - added these emissions; were not previously included
- Animal husbandry - updated NH3 emissions for this category based on recent work by EPA's Emission Standards Division/OAQPS
- Mobile source emissions - updated estimates using the latest MOBILE and NONROAD models

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 2A-1 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.

APPENDIX 3A. Mortality and Morbidity Effect Estimates and PM Concentrations from U.S. and Canadian Studies for Short-term Exposures to PM_{10}, $PM_{2.5}$, and $PM_{10-2.5}$

Original study* Study Location Reanalysis study	Analysis Comments	% increase (95% CI) per 50 μg/m³ PM_{10}	% increase (95% CI) per 25 μg/m³ $PM_{2.5}$	% increase (95% CI) per 25 μg/m³ $PM_{10-2.5}$	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*	---	*5.3 (3.5, 7.1)*	(	*PM_{10} 24.5 (SD 12.8)*
	GLM NS		*5.7 (3.7, 7.6)*		*$PM_{2.5}$ 15.7 (SD 9.2)*
	GLM BS		*5.0 (3.1, 7.0)*		*$PM_{10-2.5}$ 8.8 (SD 7.0)*
	GLM PS		*4.5 (2.5, 6.5)*	*0.7 (-1.9, 3.4)*	
Schwartz et al., 1996 Knoxville, TN Schwartz, 2003a	*GAM Strict*	---	*3.1 (0.0, 6.2)*		*PM_{10} 32.0 (SD 14.5)*
	GLM NS		*3.0 (-0.3, 6.6)*		*$PM_{2.5}$ 20.8 (SD 9.6)*
	GLM BS		*2.8 (-0.5, 6.3)*		*$PM_{10-2.5}$ 11.2 (SD 7.4)*
	GLM PS		*2.6 (-0.8, 6.1)*	*1.7 (-2.7, 6.3)*	
Schwartz et al., 1996 St. Louis, MO Schwartz, 2003a	*GAM Strict*	---	*2.6 (0.9, 4.3)*		*PM_{10} 30.6 (SD 16.2)*
	GLM NS		*2.4 (0.6, 4.1)*		*$PM_{2.5}$ 18.7 (SD 10.5)*
	GLM BS		*2.6 (0.9, 4.4)*		*$PM_{10-2.5}$ 11.9 (SD 8.5)*
	GLM PS		*2.3 (0.6, 4.1)*	*0.3 (-2.1, 2.7)*	

Original study* Study Location Reanalysis study	Analysis Comments	% increase (95% CI) per 50 μg/m³ PM_{10}	% increase (95% CI) per 25 μg/m³ $PM_{2.5}$	% increase (95% CI) per 25 μg/m³ $PM_{10-2.5}$	PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ Mean (Range) Levels Reported**
Schwartz et al., 1996	*GAM Strict*	---	*2.4 (-0.4, 5.3)*		*PM_{10} 45.6 (SD 32.3)*
Steubenville, OH	*GLM NS*		*1.7 (-1.3 4.8)*		*$PM_{2.5}$ 29.6 (SD 21.9)*
Schwartz, 2003a	*GLM BS*		*1.5 (-1.5, 4.6)*		*$PM_{10-2.5}$ 16.1 (SD 13.0)*
	GLM PS		*1.8 (-1.2, 4.9)*	*5.2 (0.0, 10.7)*	
Schwartz et al., 1996	*GAM Strict*	---	*2.6 (-1.2, 6.6)*		*PM_{10} 17.8 (SD 11.7)*
Portage, WI	*GLM NS*		*0.8 (-3.3, 5.1)*		*$PM_{2.5}$ 11.2 (SD 7.8)*
Schwartz, 2003a	*GLM BS*		*1.5 (-2.7, 5.8)*		*$PM_{10-2.5}$ 6.6 (SD 6.8)*
	GLM PS		*1.1 (-3.1, 5.4)*	*0.7 (-4.0, 5.6)*	
Schwartz et al., 1996	*GAM Strict*	---	*1.6 (-5.3, 9.0)*		*PM_{10} 26.7 (SD 16.1)*
Topeka, KS	*GLM NS*		*2.7 (-5.0, 10.9)*		*$PM_{2.5}$ 12.2 (SD 7.4)*
Schwartz, 2003a	*GLM BS*		*1.3 (-6.2, 9.3)*		*$PM_{10-2.5}$ 14.5 (SD 12.2)*
	GLM PS		*1.4 (-6.3, 9.6)*	*-3.0 (-8.1, 2.3)*	
Schwartz et al., 1996	*GAM Strict*	---	*3.5 (2.5, 4.5)*	---	*PM_{10} means 17.8-45.6*
6 Cities, Overall	*GLM NS*		*3.3 (2.2, 4.3)*		*$PM_{2.5}$ means 11.2-29.6*
Schwartz, 2003a	*GLM BS*		*3.0 (2.0, 4.0)*		*$PM_{10-2.5}$ means 6.6-16.1*
	GLM PS		*2.9 (1.8, 4.0)*		
Styer et al., 1995	*GAM not used*	*4.08 (0.08, 8.24)*	---	---	*PM_{10} 37 (4, 365)*
Chicago, IL					
Samet et al., 2000a,b	GAM strict	1.4 (0.9, 1.9)	---	---	PM_{10} mean range
90 Largest U.S. Cities	GLM NS	1.1 (0.5, 1.7)			15.3-52.0
Dominici et al. (2003)					
Schwartz, 2000a	GAM Strict	3.4 (2.6, 4.1)	---	---	PM_{10} mean range
10 U.S. cities	GLM NS	2.8 (2.0, 3.6)			27.1-40.6
Schwartz, 2003b					
Burnett et al., 2000	GAM Strict	3.2 (1.1, 5.5)	2.8 (1.2, 4.4)	1.9 (-0.1, 3.9)	PM_{10} 25.9 (max 121)
8 Canadian Cities	GLM NS (6	2.7 (-0.1, 5.5)	2.1 (0.1, 4.2)	1.8 (-0.6, 4.4)	$PM_{2.5}$ 13.3 (max 86)
Burnett and Goldberg, 2003	knots/yr)				$PM_{10-2.5}$ 12.9 (max 99)
Chock et al., 2000	GAM not used		<75 years 2.6 (-2.0, 7.7)	<75 years 0.7 (-1.7, 3.)	$PM_{2.5}$ 20.5 (3.0, 86.0)
Pittsburgh, PA			>75 years 1.5 (-3.0, 6.3)	>75 years 1.3 (-1.3, 3.8)	$PM_{10-2.5}$ 21.6 (0, 208.0)

Original study* Study Location Reanalysis study	Analysis Comments	% increase (95% CI) per 50 μg/m³ PM_{10}	% increase (95% CI) per 25 μg/m³ $PM_{2.5}$	% increase (95% CI) per 25 μg/m³ $PM_{10-2.5}$	PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ Mean (Range) Levels Reported**
Clyde et al., 2000 Phoenix, AZ	GAM not used	6 (>0, 11)	---	---	PM_{10} mean 45.4
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 and Mason, 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 and Mason, 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 and Mason, 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 and Mason, 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 and Mason, 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)

Original study* Study Location Reanalysis study	Analysis Comments	% increase (95% CI) per 50 μg/m³ PM_{10}	% increase (95% CI) per 25 μg/m³ $PM_{2.5}$	% increase (95% CI) per 25 μg/m³ $PM_{10-2.5}$	PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ Mean (Range) Levels Reported**
Klemm et al., 2000 Six City reanalysis - Madison Klemm and Mason, 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)
Klemm et al., 2000 Six City reanalysis - overall Klemm and Mason, 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)

Original study* Study Location Reanalysis study	Analysis Comments	% increase (95% CI) per 50 μg/m³ PM_{10}	% increase (95% CI) per 25 μg/m³ $PM_{2.5}$	% increase (95% CI) per 25 μg/m³ $PM_{10-2.5}$	PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ Mean (Range) Levels Reported**
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)
Schwartz, 2000 Chicago, IL Schwartz, 2003b	Strict GAM (dist. lag)	5.41 (2.36, 8.56)	---	---	PM_{10} mean 36.5
Schwartz, 2000 Pittsburgh, PA Schwartz, 2003b	Strict GAM (dist. lag)	3.14 (0.25, 6.11)	---	---	PM_{10} mean 36.4
Schwartz, 2000 Detroit, MN Schwartz, 2003b	Strict GAM (dist. lag)	6.83 (3.73, 10.02)	---	---	PM_{10} mean 36.9
Schwartz, 2000 Seattle, WA Schwartz, 2003b	Strict GAM (dist. lag)	7.46 (3.94, 11.10)	---	---	PM_{10} mean 32.5
Schwartz, 2000 Minneapolis, MN Schwartz, 2003b	Strict GAM (dist. lag)	10.25 (4.67, 16.12)	---	---	PM_{10} mean 27.5
Schwartz, 2000 Birmingham, AL Schwartz, 2003b	Strict GAM (dist. lag)	1.71 (-3.44, 7.13)	---	---	PM_{10} mean 34.8
Schwartz, 2000 New Haven, CT Schwartz, 2003b	Strict GAM (dist. lag)	9.17 (1.04, 17.96)	---	---	PM_{10} mean 28.6
Schwartz, 2000 Canton, OH Schwartz, 2003b	Strict GAM (dist. lag)	8.79 (-4.69, 24.18)	---	---	PM_{10} mean 29.31

Original study* Study Location Reanalysis study	Analysis Comments	% increase (95% CI) per 50 μg/m³ PM_{10}	% increase (95% CI) per 25 μg/m³ $PM_{2.5}$	% increase (95% CI) per 25 μg/m³ $PM_{10-2.5}$	PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ Mean (Range) Levels Reported**
Schwartz, 2000 Spokane, WA Schwartz, 2003b	Strict GAM (dist. lag)	5.62 (-0.31, 11.91)	---	---	PM_{10} mean 40.6
Schwartz, 2000 Colorado Springs, CO Schwartz, 2003b	Strict GAM (dist. lag)	8.58 (-3.94, 22.73)	---	---	PM_{10} mean 27.1
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)*

Original study* Study Location Reanalysis study	Analysis Comments	% increase (95% CI) per 50 μg/m³ PM_{10}	% increase (95% CI) per 25 μg/m³ $PM_{2.5}$	% increase (95% CI) per 25 μg/m³ $PM_{10-2.5}$	PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ Mean (Range) Levels Reported**
Pope et al., 1992 Utah Valley, UT	*GAM not used*	*9.36 (1.91, 17.36)*	---	---	*PM_{10} 47 (11, 297)*
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)

Original study* Study Location Reanalysis study	Analysis Comments	% increase (95% CI) per 50 μg/m³ PM_{10}	% increase (95% CI) per 25 μg/m³ $PM_{2.5}$	% increase (95% CI) per 25 μg/m³ $PM_{10-2.5}$	PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ Mean (Range) Levels Reported**
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)
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/m³ PM_{10}	% increase (95% CI) per 25 μg/m³ $PM_{2.5}$	% increase (95% CI) per 25 μg/m³ $PM_{10-2.5}$	PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ Mean (Range) Levels Reported**
CARDIOVASCULAR MORBIDITY					
Total Cardiovascular Hospital Admissions:					
Samet et al., 2000 14 U.S. Cities (>65 years) Zanobetti and Schwartz (2003b)	strict GAM strict GAM (dist lag) GLM NS GLM PS	4.95 (3.95 ,5.95)) 5.73 (4.27, 7.20) 4.8 (3.55, 6.0) 5.0 (4.0, 5.95)	---	---	PM_{10} means 24.4-45.3
Linn et al., 2000 Los Angeles, CA (>29 years)	GAM not used	3.25 (2.04, 4.47)	---	---	PM_{10} 45.5 (5, 132)
Moolgavkar, 2000b Cook Co., IL (all ages) Moolgavkar, 2003	strict GAM_{100df} GLM NS_{100df}	4.05 (2.9, 5.2) 4.25 (3.0, 5.5)	---	---	PM_{10} median 35 (3, 365)
Moolgavkar, 2000b Los Angeles, CA (all ages) Moolgavkar, 2003	GAM_{30df} GAM_{100df} GLM NS_{100df}	3.35 (1.2, 5.5) 2.7 (0.6, 4.8) 2.75 (0.1, 5.4)	3.95 (2.2, 5.7) 2.9 (1.2, 4.6) 3.15 (1.1, 5.2)	---	PM_{10} median 44 (7, 166) $PM_{2.5}$ median 22 (4, 86)
Stieb et al., 2000 St. John, CAN (all ages)	GAM not used	39.2 (5.0, 84.4)	15.11 (0.25, 32.8)	---	summer 93 PM_{10} 14.0 (max 70.3) $PM_{2.5}$ 8.5 (max 53.2)
Burnett et al., 1997 Toronto, CAN (all ages)	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)

Original study* Study Location Reanalysis study	Analysis Comments	% increase (95% CI) per 50 μg/m³ PM_{10}	% increase (95% CI) per 25 μg/m³ $PM_{2.5}$	% increase (95% CI) per 25 μg/m³ $PM_{10-2.5}$	PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ Mean (Range) Levels Reported**
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)
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)
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/m³ PM_{10}	% increase (95% CI) per 25 μg/m³ $PM_{2.5}$	% increase (95% CI) per 25 μg/m³ $PM_{10-2.5}$	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)
Other Cardiovascular Effects, Including Physiological Changes or Biomarkers					
Gold et al., Boston, MA	GAM stringent	---	(heart rate) -2.3 (-4.2, -0.3) (r-MSSD) -6.3 (10.2, -2.3)	---	$PM_{2.5}$ (4-hr) 15.3 (2.9, 48.6)
Peters et al., 2000 Boston, MA	GAM not used	(cardiac arrhythmia, 10+ events) 144.6 (-2.8, 515.8)	(cardiac arrhythmia, 10+ events) 75.4 (3.2, 198.2)	---	PM_{10} 19.3 (max = 62.5) $PM_{2.5}$ 12.7 (max = 53.2)
Peters et al., 2001 Boston, MA	GAM not used	(myocardial infarction) 132.7 (18.7, 356.3)	(myocardial infarction) 82.8 (16.0, 188.1)	(myocardial infarction) 73.1 (-17.0, 261.1)	PM_{10} 19.4 (SD=9.4) $PM_{2.5}$ 12.1 (SD=6.6) $PM_{10-2.5}$ 7.4 (SD=4.4)
Schwartz et al., 2001 U.S. population (NHANES)	GAM not used	(fibrinogen) 25,7 (8.8, 42.6)	---	---	PM_{10} 35.2 (SD=20.5)
Pope et al., 1999 Utah Valley, UT	GAM not used	(heart rate) 34.5 (3.1, 65.9)	---	---	PM_{10} NR (15,145 from figure)
Liao et al., 1999 Baltimore, MD	GAM not used	---	(heart rate variability) -0.1 (-0.18, -0.03)		$PM_{2.5}$ 16.1 (8.0, 32.2)
Levy et al., 2001 Seattle, WA	GAM not used	(cardiac arrest) -30.3 (-53.4, 4.3)	---	---	PM_{10} 31.9 (6.0, 178.0)

Original study* Study Location Reanalysis study	Analysis Comments	% increase (95% CI) per 50 μg/m³ PM_{10}	% increase (95% CI) per 25 μg/m³ $PM_{2.5}$	% increase (95% CI) per 25 μg/m³ $PM_{10-2.5}$	PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ Mean (Range) Levels Reported**
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
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)
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%)*

Original study* Study Location Reanalysis study	Analysis Comments	% increase (95% CI) per 50 μg/m³ PM_{10}	% increase (95% CI) per 25 μg/m³ $PM_{2.5}$	% increase (95% CI) per 25 μg/m³ $PM_{10-2.5}$	PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ Mean (Range) Levels Reported**
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)
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)

Original study* Study Location Reanalysis study	Analysis Comments	% increase (95% CI) per 50 μg/m³ PM_{10}	% increase (95% CI) per 25 μg/m³ $PM_{2.5}$	% increase (95% CI) per 25 μg/m³ $PM_{10-2.5}$	PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ Mean (Range) Levels Reported**
Moolgavkar, 2000c Los Angeles, CA (all ages)	Strict GAM: 30 df	7.78 (4.30, 11.38)	4.69 (2.06, 7.39)		PM_{10} median 44 (7, 166) $PM_{2.5}$ median 22 (4, 86)
Moolgavkar 2003	Strict GAM: 100 df	5.52 (2.53-8.59)	2.87 (0.53, 5.27)		
	GLM NS: 100df	5.00 (1.22, 8.91)	2.59 (-0.29, 5.56)		
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., 2000 Atlanta, GA (<17 years)	GAM not used	13.2 (1.2, 26.7)	---	---	PM_{10} 38.9 (9, 105)
Sheppard et al., 1999 Seattle, WA (<65 years) Sheppard et al., 2003	Strict GAM	10.9 (2.8, 19.6)	8.7 (3.2, 14.4)	5.5 (0, 14.0)	PM_{10} 31.5 (90 55)
	GLM NS	8.1 (0.1, 16.7)	6.5 (1.1,12.0)	5.5 (−2.7, 11.1)	$PM_{2.5}$ 16.7 (90 32) $PM_{10-2.5}$ 16.2 (90 29)

Original study* Study Location Reanalysis study	Analysis Comments	% increase (95% CI) per 50 μg/m³ PM_{10}	% increase (95% CI) per 25 μg/m³ $PM_{2.5}$	% increase (95% CI) per 25 μg/m³ $PM_{10-2.5}$	PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ Mean (Range) Levels Reported**
Respiratory Symptoms		Odds Ratio (95% CI) for 50 ug/m³ % increase in PM_{10}	Odds Ratio (95% CI) for 25 ug/m³ % increase in $PM_{2.5}$	Odds Ratio (95% CI) for 25 ug/m³ % 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/m³ PM_{10}	% increase (95% CI) per 25 μg/m³ $PM_{2.5}$	% increase (95% CI) per 25 μg/m³ $PM_{10-2.5}$	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/m³ PM_{10}	% increase (95% CI) per 25 μg/m³ $PM_{2.5}$	% increase (95% CI) per 25 μg/m³ $PM_{10-2.5}$	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)
Lung Function Changes		Lung Function change (L/min) (95% CI) for 50 ug/m³ % increase in PM_{10}	Lung Function change (L/min) (95% CI) for 25 ug/m³ % increase in $PM_{2.5}$	Lung Function change (L/min) (95% CI) for 25 ug/m³ % increase in $PM_{10-2.5}$	$PM_{10-2.5}$ Mean (Range) Levels Reported**

Original study* Study Location Reanalysis study	Analysis Comments	% increase (95% CI) per 50 μg/m³ PM_{10}	% increase (95% CI) per 25 μg/m³ $PM_{2.5}$	% increase (95% CI) per 25 μg/m³ $PM_{10-2.5}$	PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ 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/m³ $SO_4^=$)	---	$SO_4^=$ 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
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.

APPENDIX 3B. Mortality and Morbidity Effect Estimates and PM Concentrations from U.S. and Canadian Studies for Long-Term Exposures to PM_{10}, $PM_{2.5}$, and $PM_{10-2.5}$

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[U] (9, 34)
	$SO_4^=$ (15 $\mu g/m^3$)	1.11 (1.06, 1.16)	11[U] (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)	1.04 (1.01, 1.08)	21 (9, 34)
	$PM_{2.5}$ (10 $\mu g/m^3$) (1999-00)	1.06 (1.02, 1.10)	14 (5, 20)
	$PM_{2.5}$ (10 $\mu g/m^3$) (average)	1.06 (1.02, 1.11)	18 (7.5, 30)
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 $\mu g/m^3$)	—[V]	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[U] (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)
ACS Study Extended Analyses[E]	$PM_{2.5}$ (10 $\mu g/m^3$) (1979-83)	1.06 (1.02, 1.10)	21 (9, 34)
	$PM_{2.5}$ (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 (7.5, 30)

Study	Indicator (Increment)	Relative Risk (95% CI)	Study Concentrations ($\mu g/m^3$)
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[U] (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) $PM_{2.5}$ (10 $\mu g/m^3$) (1999-00) $PM_{2.5}$ (10 $\mu g/m^3$) (average)	1.08 (1.01, 1.16) 1.13 (1.04, 1.22) 1.14 (1.05, 1.24)	21 (9, 34) 14 (5, 20) 18 (7.5, 30)
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)
Increased Bronchitis in Children			
Six City[I]	$PM_{15/10}$ (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[J]	$SO_4^=$ (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[K]	$SO_4^=$ (15 $\mu g/m^3$)	1.39 (0.99, 1.92)	—
12 Southern California communities[L] (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[M] (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[L] (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[M] (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

Study	Indicator (Increment)	Relative Risk (95% CI)	Study Concentrations ($\mu g/m^3$)
Increased Airway Obstruction in Adults			
AHSMOG[K]	PM_{10} (20 $\mu g/m^3$)	1.19 (0.84, 1.68)	NR
Decreased Lung Function in Children			
Six City[I]	$PM_{15/10}$ (50 $\mu g/m^3$)	NS Changes	NR (20, 59)
24 City[J]	$SO_4^=$ (15 $\mu g/m^3$) $PM_{2.1}$ (10 $\mu g/m^3$) PM_{10} (20 $\mu g/m^3$)	-6.56% (-9.64, -3.43) FVC -2.15% (-3.34, -0.95) FVC -2.80% (-4.97, -0.59) FVC	4.7 (0.7, 7.4) 14.5 (5.8, 20.7) 23.8 (15.4, 32.7)
12 Southern California communities[P] (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[P] (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[Q] (4th grade cohort)	PM_{10} (20 $\mu g/m^3$) $PM_{2.5}$ (10 $\mu g/m^3$) $PM_{10-2.5}$ (10 $\mu g/m^3$)	-0.23 (-0.44, -0.01) FVC % growth -0.18 (-0.36, 0.0) FVC % growth -0.22 (-0.47, 0.02) FVC % growth	NR (15, 70)[X] NR (10, 35)[X] NR
12 Southern California communities[Q] (4th grade cohort)	PM_{10} (20 $\mu g/m^3$) $PM_{2.5}$ (10 $\mu g/m^3$) $PM_{10-2.5}$ (10 $\mu g/m^3$)	-0.51 (-0.94, -0.08) MMEF % growth -0.4 (-0.75, -0.04) MMEF % growth -0.54 (-1.0, -0.06) MMEF % growth	NR (15, 70)[X] NR (10, 35)[X] NR
12 Southern California communities[R] (second 4th grade cohort)	PM_{10} (20 $\mu g/m^3$) $PM_{2.5}$ (10 $\mu g/m^3$)	-0.12 (-0.26, 0.24) FVC % growth -0.06 (-0.30, 0.18) FVC % growth	NR (10, 80)[Y] NR (5, 30)[Y]
12 Southern California communities[R] (second 4th grade cohort)	PM_{10} (20 $\mu g/m^3$) $PM_{2.5}$ (10 $\mu g/m^3$)	-0.26 (-0.75, 0.23) MMEF % growth -0.42 (-0.84, 0.0) MMEF % growth	NR (10, 80)[Y] NR (5, 30)[Y]
12 Southern California communities[R] (second 4th grade cohort)	PM_{10} (20 $\mu g/m^3$) $PM_{2.5}$ (10 $\mu g/m^3$)	-0.16 (-0.62, 0.30) PEFR % growth -0.20 (-0.64, 0.25) PEFR % growth	NR (10, 80)[Y] NR (5, 30)[Y]

Study	Indicator (Increment)	Relative Risk (95% CI)	Study Concentrations ($\mu g/m^3$)
12 Southern California communities[S]	PM_{10} (20 $\mu g/m^3$)	-3.6 (-18, 11) FVC growth	NR (15.0, 66.2)
12 Southern California communities[S]	PM_{10} (20 $\mu g/m^3$)	-33 (-64, -2.2) MMEF growth	NR (15.0, 66.2)
12 Southern California communities[S]	PM_{10} (20 $\mu g/m^3$)	-70 (-120, -20) PEFR growth	NR (15.0, 66.2)
Lung Function Changes in Adults			
AHSMOG[T] (% 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[T] (% 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)
AHSMOG[T] (% 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[T] (% 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)

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)
[I] Dockery et al. (1989)
[J] Dockery et al. (1996)
[K] Abbey et al. (1995a,b,c)
[L] Peters et al. (1999a)
[M] McConnell et al. (1999)
[N] Berglund et al. (1999)
[O] Raizenne et al. (1996)
[P] Peters et al. (1999)
[Q] Gauderman et al. (2000)
[R] Gauderman et al. (2002)
[S] Avol et al. (2001)
[T] Abbey et al. (1998)

Note: Study concentrations are presented as mean (min, max), or mean (±SD); NS Changes = No significant changes (no quantitative results reported); NR=not reported.

[U] Median
[V] Results only for smoking category subgroups.
[X] Estimated from Figure 1, Gauderman et al. (2000)
[Y] Estimated from figures available in online data supplement to Gauderman et al. (2002)

APPENDIX 4A

Study-Specific Information on Short- and Long-term Exposure Studies in Cities included in $PM_{2.5}$ Assessment and on Short-term Exposure Studies in Cities included in $PM_{10-2.5}$ Assessment

Table 4A-1. Study-Specific Information for Short-term Exposure $PM_{2.5}$ Studies in Boston, MA

Study	Health Effect	ICD-9 Codes	Ages	Model	Other Pollutants in Model	Observed Concentrations min.	Observed Concentrations max.	Lag	Exposure Metric	PM2.5 Coeff.	Lower Bound	Upper Bound
Short-Term Exposure Total Mortality – Single Pollutant Models												
Schwartz (2003b) [reanalysis of Schwartz et al. (1996)]	Non-accidental	< 800	all	log-linear, GAM (stringent)	none	0	70.8	mean of lag 0 & 1	2-day avg	0.00206	0.00139	0.00273
Schwartz (2003b) [reanalysis of Schwartz et al. (1996)] – 6 cities	Non-accidental	< 800	all	log-linear, GAM (stringent)	none	0	174	mean of lag 0 & 1	2-day avg	0.00137	0.00098	0.00176
Short-Term Exposure Cause-Specific Mortality – Single Pollutant Models												
Klemm and Mason (2003) [reanalysis of Klemm et al. (2000)]	COPD	490-492, 494-496	all	log-linear, GAM (stringent)	none	0	70.8	0 day	2-day avg	0.00276	-0.00131	0.00658
Klemm and Mason (2003) [reanalysis of Klemm et al. (2000)]	Ischemic heart disease	410-414	all	log-linear, GAM (stringent)	none	0	70.8	0 day	2-day avg	0.00266	0.00149	0.00383
Klemm and Mason (2003) [reanalysis of Klemm et al. (2000)]	Pneumonia	480-487	all	log-linear, GAM (stringent)	none	0	70.8	0 day	2-day avg	0.00573	0.00257	0.00871
Klemm and Mason (2003) [reanalysis of Klemm et al. (2000)] – 6 cities	COPD	490-492, 494-496	all	log-linear, GAM (stringent)	none	0	174	0 day	2-day avg	0.00227	0.00010	0.00440
Klemm and Mason (2003) [reanalysis of Klemm et al. (2000)] – 6 cities	Ischemic heart disease	410-414	all	log-linear, GAM (stringent)	none	0	174	0 day	2-day avg	0.00178	0.00109	0.00247
Klemm and Mason (2003) [reanalysis of Klemm et al. (2000)] – 6 cities	Pneumonia	480-487	all	log-linear, GAM (stringent)	none	0	174	0 day	2-day avg	0.00402	0.00188	0.00602
Respiratory Symptoms and Illnesses – Single Pollutant Models**												
Schwartz and Neas (2000) - 6 cities	Lower respiratory symptoms*	n/a	7-14	logistic	none	N/A	N/A	1 day	1-day avg	0.01901	0.00696	0.03049
Schwartz and Neas (2000) - 6 cities	cough*	n/a	7-14	logistic	none	N/A	N/A	0 day	3-day avg	0.00989	-0.00067	0.02050
Respiratory Symptoms and Illnesses – Multi-Pollutant Models**												
Schwartz and Neas (2000) - 6 cities	Lower respiratory symptoms*	n/a	7-14	logistic	PM10-2.5	N/A	N/A	1 day	1-day avg	0.01698	0.00388	0.03007
Schwartz and Neas (2000) - 6 cities	cough*	n/a	7-14	logistic	PM10-2.5	N/A	N/A	0 day	3-day avg	0.00451	-0.00702	0.01541

*The C-R functions for lower respiratory symptoms and cough were calculated for the summer period April 1 through August 31

Table 4A-2. Study-Specific Information for Short-term Exposure $PM_{2.5}$ Studies in Detroit, MI

Study	Health Effect	ICD-9 Codes	Ages	Model	Other Pollutants in Model	Observed Concentrations min.	Observed Concentrations max.	Lag	Exposure Metric	PM2.5 Coeff.	Lower Bound	Upper Bound
Short-Term Exposure Total Mortality -- Single Pollutant Models												
Ito (2003) [reanalysis of Lippmann et al. (2000)]	Non-accidental	<800	all	log-linear, GAM (stringent)	none	4	86	3 day	1-day avg	0.00074	-0.00073	0.00221
Short-Term Exposure Cause-Specific Mortality -- Single Pollutant Models												
Ito (2003) [reanalysis of Lippmann et al. (2000)]	Circulatory	390-459	all	log-linear, GAM (stringent)	none	4	86	1 day	1-day avg	0.00087	-0.00131	0.00305
Ito (2003) [reanalysis of Lippmann et al. (2000)]	Respiratory	460-519	all	log-linear, GAM (stringent)	none	4	86	0 day	1-day avg	0.00090	-0.00438	0.00618
Hospital Admissions -- Single Pollutant Models												
Ito (2003) [reanalysis of Lippmann et al. (2000)]	Pneumonia	480-486	65+	log-linear, GAM (stringent)	none	4	86	1 day	1-day avg	0.00398	0.00074	0.00725
Ito (2003) [reanalysis of Lippmann et al. (2000)]	COPD	490-496	65+	log-linear, GAM (stringent)	none	4	86	3 day	1-day avg	0.00117	-0.00287	0.00523
Ito (2003) [reanalysis of Lippmann et al. (2000)]	Ischemic heart disease	410-414	65+	log-linear, GAM (stringent)	none	4	86	2 day	1-day avg	0.00143	-0.00082	0.00371
Ito (2003) [reanalysis of Lippmann et al. (2000)]	Congestive heart failure	428	65+	log-linear, GAM (stringent)	none	4	86	1 day	1-day avg	0.00307	0.00055	0.00561
Ito (2003) [reanalysis of Lippmann et al. (2000)]	Dysrhythmias	427	65+	log-linear, GAM (stringent)	none	4	86	1 day	1-day avg	0.00125	-0.00274	0.00523

Table 4A-3. Study-Specific Information for Short-term Exposure $PM_{2.5}$ Studies in Los Angeles, CA

Study	Health Effect	ICD-9 Codes	Ages	Model	Other Pollutants in Model	Observed Concentrations min.	Observed Concentrations max.	Lag	Exposure Metric	PM2.5 Coeff.	Lower Bound	Upper Bound
Short-Term Exposure Total Mortality -- Single Pollutant Models												
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	<800	all	log-linear, GAM (stringent), 100 df	none	4	86	0 day	1-day avg	0.00032	-0.00023	0.00086
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	<800	all	log-linear, GAM (stringent), 100 df	none	4	86	1 day	1-day avg	0.00010	-0.00046	0.00066
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	<800	all	log-linear, GAM (stringent), 30 df	none	4	86	0 day	1-day avg	0.00054	-0.00007	0.00114
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	<800	all	log-linear, GLM, 30 df	none	4	86	0 day	1-day avg	0.00040	-0.00034	0.00113
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	<800	all	log-linear, GAM (stringent), 100 df	none	4	86	0 day	1-day avg	0.00032	-0.00023	0.00086
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	<800	all	log-linear, GLM, 100 df	none	4	86	0 day	1-day avg	0.00030	-0.00043	0.00102
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	<800	all	log-linear, GAM (stringent), 30 df	none	4	86	1 day	1-day avg	0.00059	0.00000	0.00117
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	<800	all	log-linear, GLM, 30 df	none	4	86	1 day	1-day avg	0.00055	-0.00017	0.00126
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	<800	all	log-linear, GAM (stringent), 100 df	none	4	86	1 day	1-day avg	0.00010	-0.00046	0.00066
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	<800	all	log-linear, GLM, 100 df	none	4	86	1 day	1-day avg	-0.00001	-0.00099	0.00097

Study	Health Effect	ICD-9 Codes	Ages	Model	Other Pollutants in Model	Observed Concentrations min.	max.	Lag	Exposure Metric	PM2.5 Coeff.	Lower Bound	Upper Bound
Short-Term Exposure Cause-Specific Mortality -- Single Pollutant Models												
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardiovascular	390-429	all	log-linear, GAM (stringent), 30 df	none	4	86	0 day	1-day avg	0.00099	0.00010	0.00187
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardiovascular	390-429	all	log-linear, GAM (stringent), 100 df	none	4	86	0 day	1-day avg	0.00097	0.00014	0.00179
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardiovascular	390-429	all	log-linear, GLM, 100 df	none	4	86	0 day	1-day avg	0.00097	-0.00002	0.00195
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardiovascular	390-429	all	log-linear, GAM (stringent), 30 df	none	4	86	1 day	1-day avg	0.00103	0.00016	0.00189
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardiovascular	390-429	all	log-linear, GAM (stringent), 100 df	none	4	86	1 day	1-day avg	0.00080	-0.00003	0.00162
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardiovascular	390-429	all	log-linear, GLM, 100 df	none	4	86	1 day	1-day avg	0.00069	-0.00032	0.00169
Short-Term Exposure Total Mortality -- Multi-Pollutant Models												
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	<800	all	log-linear, GAM (stringent), 30 df	CO	4	86	1 day	1-day avg	-0.00053	-0.00132	0.00025
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	<800	all	log-linear, GAM (stringent), 100 df	CO	4	86	1 day	1-day avg	-0.00033	-0.00105	0.00039
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	<800	all	log-linear, GLM, 100 df	CO	4	86	1 day	1-day avg	-0.00033	-0.00118	0.00051
Short-Term Exposure Cause-Specific Mortality -- Multi-Pollutant Models												
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardiovascular	390-429	all	log-linear, GAM (stringent), 100 df	CO	4	86	0 day	1-day avg	0.00178	0.00076	0.00279
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardiovascular	390-429	all	log-linear, GLM, 100 df	CO	4	86	0 day	1-day avg	0.00188	0.00068	0.00306
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardiovascular	390-429	all	log-linear, GAM (stringent), 100 df	CO	4	86	1 day	1-day avg	0.00091	-0.00012	0.00193
Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Cardiovascular	390-429	all	log-linear, GLM, 100 df	CO	4	86	1 day	1-day avg	0.00091	-0.00034	0.00215

Study	Health Effect	ICD-9 Codes	Ages	Model	Other Pollutants in Model	Observed Concentrations min.	Observed Concentrations max.	Lag	Exposure Metric	PM2.5 Coeff.	Lower Bound	Upper Bound
Hospital Admissions – Single Pollutant Models												
Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardiovascular	390-429	65+	log-linear, GAM (stringent), 30 df	none	4	86	0 day	1-day avg	0.00158	0.00091	0.00224
Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardiovascular	390-429	65+	log-linear, GAM (stringent), 100 df	none	4	86	0 day	1-day avg	0.00116	0.00051	0.00181
Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardiovascular	390-429	65+	log-linear, GLM, 100 df	none	4	86	0 day	1-day avg	0.00126	0.00045	0.00206
Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardiovascular	390-429	65+	log-linear, GAM (stringent), 30 df	none	4	86	1 day	1-day avg	0.00139	0.00070	0.00208
Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardiovascular	390-429	65+	log-linear, GAM (stringent), 100 df	none	4	86	1 day	1-day avg	0.00113	0.00047	0.00179
Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardiovascular	390-429	65+	log-linear, GLM, 100 df	none	4	86	1 day	1-day avg	0.00120	0.00039	0.00200
Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	490-496	all	log-linear, GAM (stringent), 30 df	none	4	86	0 day	1-day avg	0.00167	0.00069	0.00264
Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	490-496	all	log-linear, GAM (stringent), 100 df	none	4	86	0 day	1-day avg	0.00138	0.00052	0.00223
Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	490-496	all	log-linear, GLM, 100 df	none	4	86	0 day	1-day avg	0.00149	0.00042	0.00255
Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	490-496	all	log-linear, GAM (stringent), 30 df	none	4	86	1 day	1-day avg	0.00119	0.00023	0.00214
Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	490-496	all	log-linear, GAM (stringent), 100 df	none	4	86	1 day	1-day avg	0.00075	-0.00011	0.00160
Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	490-496	all	log-linear, GLM, 100 df	none	4	86	1 day	1-day avg	0.00077	-0.00027	0.00180
Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	490-496	all	log-linear, GAM (stringent), 30 df	none	4	86	2 day	1-day avg	0.00185	0.00084	0.00285
Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	490-496	all	log-linear, GAM (stringent), 100 df	none	4	86	2 day	1-day avg	0.00114	0.00022	0.00205
Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	490-496	all	log-linear, GLM, 100 df	none	4	86	2 day	1-day avg	0.00103	-0.00011	0.00216

Study	Health Effect	ICD-9 Codes	Ages	Model	Other Pollutants in Model	Observed Concentrations min.	max.	Lag	Exposure Metric	PM2.5 Coeff.	Lower Bound	Upper Bound
Hospital Admissions – Single City, Multi-Pollutant Models												
Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardiovascular	390-429	65+	log-linear, GAM (stringent), 100 df	CO	4	86	0 day	1-day avg	0.00039	-0.00044	0.00121
Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardiovascular	390-429	65+	log-linear, GLM, 100 df	CO	4	86	0 day	1-day avg	0.00058	-0.00041	0.00156
Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardiovascular	390-429	65+	log-linear, GAM (stringent), 100 df	CO	4	86	1 day	1-day avg	0.00024	-0.00065	0.00112
Moolgavkar (2003) [reanalysis of Moolgavkar (2000b)]	Cardiovascular	390-429	65+	log-linear, GLM, 100 df	CO	4	86	1 day	1-day avg	0.00027	-0.00075	0.00128
Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	490-496	all	log-linear, GAM (stringent), 100 df	NO2	4	86	0 day	1-day avg	0.00042	-0.00091	0.00173
Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	490-496	all	log-linear, GAM (stringent), 100 df	NO2	4	86	1 day	1-day avg	-0.00004	-0.00162	0.00152
Moolgavkar (2003) [reanalysis of Moolgavkar (2000c)]	COPD+	490-496	all	log-linear, GAM (stringent), 100 df	NO2	4	86	2 day	1-day avg	0.00035	-0.00103	0.00171

Table 4A-4. Study-Specific Information for Short-term Exposure $PM_{2.5}$ Studies in Philadelphia, PA

Study*	Health Effect	ICD-9 Codes	Ages	Model	Other Pollutants in Model	Observed Concentrations min.	max.	Lag	Exposure Metric	PM2.5 Coeff.	Lower Bound	Upper Bound
Short-Term Exposure Cause-Specific Mortality -- Single Pollutant Models												
Lipfert et al. (2000) -- 7 counties	Cardiovascular	390-448	all	linear	none	-0.6	72.6	1 day	1-day avg	0.10440	0.04983	0.15897

*The Lipfert et al. (2000) study does not provide the statistical uncertainties surrounding the PM2.5 non-accidental mortality coefficients and the cardiovascular mortality multi-pollutant coefficient.

Table 4A-5. Study-Specific Information for Short-term Exposure $PM_{2.5}$ Studies in Phoenix, AZ

Study	Health Effect	ICD-9 Codes	Ages	Model	Other Pollutants in Model	Observed Concentrations min.	max.	Lag	Exposure Metric	PM2.5 Coeff.	Lower Bound	Upper Bound
Short-term Exposure Cause-Specific Mortality -- Single Pollutant Models												
Mar (2003) [reanalysis of Mar (2000)]	Cardiovascular	390-448.9	65+	log-linear, GAM (stringent)	none	0	42	0 day	1-day avg	0.00371	-0.0010136	0.0084336
Mar (2003) [reanalysis of Mar (2000)]	Cardiovascular	390-448.9	65+	log-linear, GAM (stringent)	none	0	42	1 day	1-day avg	0.00661	0.0019256	0.0112944

Table 4A-6. Study-Specific Information for Short-term Exposure $PM_{2.5}$ Studies in Pittsburgh, PA

Study	Health Effect	ICD-9 Codes	Ages	Model	Other Pollutants	Observed Concentrations		Lag	Exposure Metric	PM2.5 Coeff.	Lower Bound	Upper Bound
Short-term Exposure Total Mortality -- Single Pollutant Models												
Chock et al. (2000)	Non-accidental	<800	<75	log-linear	none	3	86	0 day	1-day avg	0.00101	-0.00078964	0.00280964
Chock et al. (2000)	Non-accidental	<800	75+	log-linear	none	3	86	0 day	1-day avg	0.00059	-0.00124556	0.00242556
Short-term Exposure Total Mortality -- Multi-Pollutant Models												
Chock et al. (2000)	Non-accidental	<800	<75	log-linear	CO, O3, SO2, NO2, PM10-2.5	3	86	0 day	1-day avg	0.0013	-0.00085932	0.00345932
Chock et al. (2000)	Non-accidental	<800	75+	log-linear	CO, O3, SO2, NO2, PM10-2.5	3	86	0 day	1-day avg	0.0004	-0.00177778	0.00257778

Table 4A-7. Study-Specific Information for Short-term Exposure $PM_{2.5}$ Studies in San Jose, CA

Study	Health Effect	ICD-9 Codes	Ages	Model	Other Pollutants in Model	Observed Concentrations min.	max.	Lag	Exposure Metric	PM2.5 Coeff.	Lower Bound	Upper Bound
Short-Term Exposure Total Mortality -- Single Pollutant Models												
Fairley (2003) [reanalysis of Fairley (1999)]	Non-accidental	<800	all	log-linear, GAM (stringent)	none	2	105	0 day	1-day avg	0.00314	0.00064	0.00567
Fairley (2003) [reanalysis of Fairley (1999)]	Non-accidental	<800	all	log-linear, GAM (stringent)	none	2	105	1 day	1-day avg	-0.00153	-0.00380	0.00071
Short-Term Exposure Cause-Specific Mortality -- Single Pollutant Models												
Fairley (2003) [reanalysis of Fairley (1999)]	Respiratory	11, 35, 472-519, 710.0, 710.2, 710.4	all	log-linear, GAM (stringent)	none	2	105	0 day	1-day avg	0.00446	-0.00416	0.01307
Fairley (2003) [reanalysis of Fairley (1999)]	Cardiovascular	390-459	all	log-linear, GAM (stringent)	none	2	105	0 day	1-day avg	0.00248	-0.00168	0.00666
Short-Term Exposure Total Mortality -- Multi-Pollutant Models												
Fairley (2003) [reanalysis of Fairley (1999)]	Non-accidental	<800	all	log-linear, GAM (stringent)	NO2	2	105	0 day	1-day avg	0.00402	0.00106	0.00698
Fairley (2003) [reanalysis of Fairley (1999)]	Non-accidental	<800	all	log-linear, GAM (stringent)	CO	2	105	0 day	1-day avg	0.00363	0.00085	0.00636
Fairley (2003) [reanalysis of Fairley (1999)]	Non-accidental	<800	all	log-linear, GAM (stringent)	O3 - 8hr	2	105	0 day	1-day avg	0.00340	0.00085	0.00594

Table 4A-8. Study-Specific Information for Short-term Exposure $PM_{2.5}$ Studies in Seattle, WA

Study	Health Effect	ICD-9 Codes	Ages	Model	Other Pollutants in Model	Observed Concentrations min.	max.	Lag	Exposure Metric	PM2.5 Coeff.	Lower Bound	Upper Bound
Hospital Admissions - Single Pollutant Models												
Sheppard (2003) [reanalysis of Sheppard et al. (1999)]**	Asthma	493	<65	log-linear, GAM (stringent)	none	2.5	96	1 day	1-day avg	0.0033238	0.00084325	0.004938

**Sheppard (2003) [reanalysis of Sheppard et al. (1999)] used daily PM2.5 values obtained from nephelometry measurements rather than from air quality monitors.

Table 4A-9. Study-Specific Information for Short-term Exposure $PM_{2.5}$ Studies in St. Louis, MO

Study	Health Effect	ICD-9 Codes	Ages	Model	Other Pollutants in Model	Observed Concentrations min.	Observed Concentrations max.	Lag	Exposure Metric	PM2.5 Coeff.	Lower Bound	Upper Bound
Short-Term Exposure Total Mortality -- Single Pollutant Models												
Schwartz (2003b) [reanalysis of Schwartz et al. (1996)]	Non-accidental	< 800	all	log-linear, GAM (stringent)	none	0.9	88.9	mean of lag 0 & 1	2-day avg	0.00102	0.00037	0.00167
Schwartz (2003b) [reanalysis of Schwartz et al. (1996)] -- 6 cities	Non-accidental	< 800	all	log-linear, GAM (stringent)	none	0	174	mean of lag 0 & 1	2-day avg	0.00137	0.00098	0.00176
Short-Term Exposure Cause-Specific Mortality -- Single Pollutant Models												
Klemm and Mason (2003) [reanalysis of Klemm et al. (2000)]	COPD	490-492, 494-496	all	Log-linear, GAM (stringent)	none	0.9	88.9	0 day	2-day avg	0.00060	-0.00294	0.00411
Klemm and Mason (2003) [reanalysis of Klemm et al. (2000)]	Ischemic heart disease	410-414	all	Log-linear, GAM (stringent)	none	0.9	88.9	0 day	2-day avg	0.00129	0.00030	0.00237
Klemm and Mason (2003) [reanalysis of Klemm et al. (2000)]	Pneumonia	480-487	all	Log-linear, GAM (stringent)	none	0.9	88.9	0 day	2-day avg	0.00109	-0.00253	0.00459
Klemm and Mason (2003) [reanalysis of Klemm et al. (2000)] -- 6 cities	COPD	490-492, 494-496	all	Log-linear, GAM (stringent)	none	0	174	0 day	2-day avg	0.00227	0.00010	0.00440
Klemm and Mason (2003) [reanalysis of Klemm et al. (2000)] -- 6 cities	Ischemic heart disease	410-414	all	Log-linear, GAM (stringent)	none	0	174	0 day	2-day avg	0.00178	0.00109	0.00247
Klemm and Mason (2003) [reanalysis of Klemm et al. (2000)] -- 6 cities	Pneumonia	480-487	all	Log-linear, GAM (stringent)	none	0	174	0 day	2-day avg	0.00402	0.00188	0.00602
Respiratory Symptoms and Illnesses -- Single Pollutant Models**												
Schwartz and Neas (2000) - - 6 cities	Lower respiratory symptoms*	n/a	7-14	logistic	none	N/A	N/A	1 day	1-day avg	0.01901	0.00696	0.03049
Schwartz and Neas (2000) - - 6 cities	Cough*	n/a	7-14	logistic	none	N/A	N/A	0 day	3-day avg	0.00989	-0.00067	0.02050
Respiratory Symptoms and Illnesses -- Multi-Pollutant Models**												
Schwartz and Neas (2000) - - 6 cities	Lower respiratory symptoms*	n/a	7-14	logistic	PM10-2.5	N/A	N/A	1 day	1-day avg	0.01698	0.00388	0.03007
Schwartz and Neas (2000) - - 6 cities	Cough*	n/a	7-14	logistic	PM10-2.5	N/A	N/A	0 day	3-day avg	0.00451	-0.00702	0.01541

*The C-R functions for lower respiratory symptoms and cough were calculated for the summer period April 1 through August 31.

Table 4A-10. Study-Specific Information for Long-term Exposure $PM_{2.5}$ Studies

Long-Term Exposure Mortality -- Single Pollutant Models											
Krewski et al. (2000) - ACS	All cause	all	30+	log-linear	none	10	38	n/a	annual mean	0.00463	0.00238	0.00710
Pope et al. (2002) - ACS extended	All cause	all	30+	log-linear	none	7.5	30	n/a	annual mean	0.00583	0.00198	0.01044
Krewski et al. (2000) - ACS	Cardiopulmonary	401-440, 460-519	30+	log-linear	none	10	38	n/a	annual mean	0.00943	0.00606	0.01315
Pope et al. (2002) - ACS extended	Cardiopulmonary	401-440, 460-519	30+	log-linear	none	7.5	30	n/a	annual mean	0.00862	0.00296	0.01484
Pope et al. (2002) - ACS extended	Lung cancer	162	30+	log-linear	none	7.5	30	n/a	annual mean	0.01310	0.00392	0.02070
Long-Term Exposure Mortality -- Multi-Pollutant Models											
Krewski et al. (2000) - ACS	All cause	all	30+	log-linear	CO	10	38	n/a	annual mean	0.00676	0.00389	0.00976
Krewski et al. (2000) - ACS	All cause	all	30+	log-linear	NO2	10	38	n/a	annual mean	0.00812	0.00426	0.01164
Krewski et al. (2000) - ACS	All cause	all	30+	log-linear	O3	10	38	n/a	annual mean	0.00676	0.00389	0.00976
Krewski et al. (2000) - ACS	All cause	all	30+	log-linear	SO2	10	38	n/a	annual mean	0.00121	-0.00209	0.00499

Table 4A-11. Study-Specific Information for $PM_{10-2.5}$ Studies in Detroit, MI

Study	Health Effect	ICD-9 Codes	Ages	Model	Other Pollutants in Model	Observed Concentrations min.		Lag	Exposure Metric	PM Coarse Coefficient	Lower Bound	Upper Bound
Hospital Admissions – Single Pollutant Models												
Ito (2003) [reanalysis of Lippmann et al. (2000)]	Pneumonia	480-486	65+	log-linear, GAM (stringent)	none	1	50	1 day	1-day avg	0.0037814	-0.0004188	0.0079769
Ito (2003) [reanalysis of Lippmann et al. (2000)]	COPD+	490-496	65+	log-linear, GAM (stringent)	none	1	50	3 day	1-day avg	0.0033223	-0.0019622	0.0085917
Ito (2003) [reanalysis of Lippmann et al. (2000)]	Ischemic heart disease	410-414	65+	log-linear, GAM (stringent)	none	1	50	2 day	1-day avg	0.0038954	0.0009475	0.0068258
Ito (2003) [reanalysis of Lippmann et al. (2000)]	Dysrhythmias	427	65+	log-linear, GAM (stringent)	none	1	50	0 day	1-day avg	0.0000416	-0.0052791	0.0053863
Ito (2003) [reanalysis of Lippmann et al. (2000)]	Congestive heart failure	428	65+	log-linear, GAM (stringent)	none	1	50	0 day	1-day avg	0.0017142	-0.0016142	0.0050924

Table 4A-12. Study-Specific Information for $PM_{10-2.5}$ Studies in Seattle, WA

Study	Health Effect	ICD-9 Codes	Ages	Model	Other Pollutants in Model	Observed Concentrations min.	Lag	Exposure Metric	PM Coarse Coefficient	Lower Bound	Upper Bound
Hospital Admissions – Single Pollutant Models											
Sheppard (2003) (reanalysis of Sheppard et al. (1999)*	Asthma	493	<65	log-linear, GAM (stringent)	none	N/A	88	1 day	1-day avg	0.0021293	0.0000000	0.0052463

*Sheppard (2003) [reanalysis of Sheppard et al. (1999)] used daily PM2.5 values obtained from nephelometry measurements rather than from the difference between PM2.5 and PM10 air quality monitors.

Table 4A-13. Study-Specific Information for Studies in St. Louis, MO

Study	Health Effect	ICD-9 Codes	Ages	Model	Other Pollutants in Model	Observed Concentrations min.	Lag	Exposure Metric	PM Coarse Coefficient	Lower Bound	Upper Bound
Respiratory Symptoms and Illnesses* -- Single Pollutant Models											
Schwartz and Neas, 2000 -- 6 cities	Lower respiratory symptoms*	N/A	7-14	logistic	none	0	121	0 day	3-day avg	0.0163785	-0.0025253	0.0633522
Schwartz and Neas, 2000 -- 6 cities	Cough*	N/A	7-14	logistic	none	0	121	0 day	3-day avg	0.0227902	0.0084573	0.0375131
Respiratory Symptoms and Illnesses* -- Multi-Pollutant Models											
Schwartz and Neas, 2000 -- 6 cities	Lower respiratory symptoms*	N/A	7-14	logistic	PM2.5	0	121	0 day	3-day avg	0.0060988	-0.0131701	0.0258768
Schwartz and Neas, 2000 -- 6 cities	Cough*	N/A	7-14	logistic	PM2.5	0	121	0 day	3-day avg	0.0206893	0.0049026	0.0365837

*The C-R functions for lower respiratory symptoms and cough were calculated for the summer period April 1 through August 31.

APPENDIX 4B

Sensitivity Analyses: Estimated PM-Related Incidence Associated with Short- and Long-term Exposure to $PM_{2.5}$ and Short-term Exposure to $PM_{10-2.5}$

Table 4B-1. Sensitivity Analysis: Estimated Annual Mortality Associated with Short-Term Exposure to PM2.5 When Alternative Standards Are Just Met, in the Base Case and Using Alternative Hypothetical Threshold Models*
Detroit, MI, 2003

Alternative Standards		PM-Related Incidence Associated with PM-2.5**			
Annual (ug/m3)	Daily (ug/m3)	BASE CASE: Lowest Measured Level in Study =4 µg/m3	Hypothetical Threshold =10 µg/m3	Hypothetical Threshold =15 µg/m3	Hypothetical Threshold =20 µg/m3
15	65, 98th percentile value***	115 (-116 - 338)	54 (-55 - 159)	26 (-27 - 77)	12 (-12 - 35)
15	40, 98th percentile value	115 (-116 - 338)	54 (-55 - 159)	26 (-27 - 77)	12 (-12 - 35)
15	35, 98th percentile value	115 (-116 - 338)	54 (-55 - 159)	26 (-27 - 77)	12 (-12 - 35)
15	30, 98th percentile value	104 (-105 - 306)	45 (-45 - 131)	20 (-20 - 58)	8 (-9 - 24)
15	25, 98th percentile value	83 (-84 - 243)	28 (-29 - 82)	10 (-10 - 28)	3 (-4 - 10)
15	65, 99th percentile value	115 (-116 - 338)	54 (-55 - 159)	26 (-27 - 77)	12 (-12 - 35)
15	40, 99th percentile value	115 (-116 - 338)	54 (-55 - 159)	26 (-27 - 77)	12 (-12 - 35)
15	35, 99th percentile value	113 (-114 - 333)	53 (-53 - 154)	25 (-26 - 74)	11 (-12 - 33)
15	30, 99th percentile value	94 (-95 - 276)	37 (-37 - 107)	15 (-15 - 42)	6 (-6 - 16)
15	25, 99th percentile value	75 (-76 - 220)	22 (-23 - 65)	7 (-7 - 19)	2 (-2 - 6)
14	40, 98th percentile value	104 (-105 - 307)	45 (-46 - 132)	20 (-20 - 58)	8 (-9 - 24)
14	35, 98th percentile value	104 (-105 - 307)	45 (-46 - 132)	20 (-20 - 58)	8 (-9 - 24)
14	30, 98th percentile value	104 (-105 - 306)	45 (-45 - 131)	20 (-20 - 58)	8 (-9 - 24)
14	25, 98th percentile value	83 (-84 - 243)	28 (-29 - 82)	10 (-10 - 28)	3 (-4 - 10)
14	40, 99th percentile value	104 (-105 - 307)	45 (-46 - 132)	20 (-20 - 58)	8 (-9 - 24)
14	35, 99th percentile value	104 (-105 - 307)	45 (-46 - 132)	20 (-20 - 58)	8 (-9 - 24)
14	30, 99th percentile value	94 (-95 - 276)	37 (-37 - 107)	15 (-15 - 42)	6 (-6 - 16)
14	25, 99th percentile value	75 (-76 - 220)	22 (-23 - 65)	7 (-7 - 19)	2 (-2 - 6)
13	40, 98th percentile value	94 (-95 - 275)	36 (-37 - 106)	14 (-15 - 42)	6 (-6 - 16)

Alternative Standards		PM-Related Incidence Associated with PM-2.5**			
Annual (ug/m3)	Daily (ug/m3)	BASE CASE: Lowest Measured Level in Study =4 µg/m3	Hypothetical Threshold =10 µg/m3	Hypothetical Threshold =15 µg/m3	Hypothetical Threshold =20 µg/m3
13	35, 98th percentile value	94 (-95 - 275)	36 (-37 - 106)	14 (-15 - 42)	6 (-6 - 16)
13	30, 98th percentile value	94 (-95 - 275)	36 (-37 - 106)	14 (-15 - 42)	6 (-6 - 16)
13	25, 98th percentile value	83 (-84 - 243)	28 (-29 - 82)	10 (-10 - 28)	3 (-4 - 10)
13	40, 99th percentile value	94 (-95 - 275)	36 (-37 - 106)	14 (-15 - 42)	6 (-6 - 16)
13	35, 99th percentile value	94 (-95 - 275)	36 (-37 - 106)	14 (-15 - 42)	6 (-6 - 16)
13	30, 99th percentile value	94 (-95 - 275)	36 (-37 - 106)	14 (-15 - 42)	6 (-6 - 16)
13	25, 99th percentile value	75 (-76 - 220)	22 (-23 - 65)	7 (-7 - 19)	2 (-2 - 6)
12	40, 98th percentile value	83 (-84 - 244)	28 (-29 - 82)	10 (-10 - 28)	3 (-4 - 10)
12	35, 98th percentile value	83 (-84 - 244)	28 (-29 - 82)	10 (-10 - 28)	3 (-4 - 10)
12	30, 98th percentile value	83 (-84 - 244)	28 (-29 - 82)	10 (-10 - 28)	3 (-4 - 10)
12	25, 98th percentile value	83 (-84 - 243)	28 (-29 - 82)	10 (-10 - 28)	3 (-4 - 10)
12	40, 99th percentile value	83 (-84 - 244)	28 (-29 - 82)	10 (-10 - 28)	3 (-4 - 10)
12	35, 99th percentile value	83 (-84 - 244)	28 (-29 - 82)	10 (-10 - 28)	3 (-4 - 10)
12	30, 99th percentile value	83 (-84 - 244)	28 (-29 - 82)	10 (-10 - 28)	3 (-4 - 10)
12	25, 99th percentile value	75 (-76 - 220)	22 (-23 - 65)	7 (-7 - 19)	2 (-2 - 6)

*This sensitivity analysis was performed using Ito (2003). See text for an explanation of the slope adjustment method.

**In the base case, incidence was quantified across the range of PM concentrations observed in the study, when possible, but not below policy relevant background level. Under alternative threshold models, incidence was quantified down to the specified threshold. Incidences are rounded to the nearest whole number.

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

Table 4B-2. Sensitivity Analysis: Estimated Annual Mortality Associated with Long-Term Exposure to PM2.5 When Alternative Standards Are Just Met, in the Base Case and Using Alternative Hypothetical Threshold Models*
Detroit, MI, 2003

Alternative Standards		PM-Related Incidence Associated with PM-2.5**		
Annual (ug/m3)	Daily (ug/m3)	BASE CASE: Lowest Measured Level in Study =7.5 µg/m3	Hypothetical Threshold =10 µg/m3	Hypothetical Threshold =12 µg/m3
15	65, 98th percentile value	522 (181 - 910)	282 (98 - 494)	41 (14 - 72)
15	40, 98th percentile value	522 (181 - 910)	282 (98 - 494)	41 (14 - 72)
15	35, 98th percentile value	522 (181 - 910)	282 (98 - 494)	41 (14 - 72)
15	30, 98th percentile value	435 (151 - 757)	185 (64 - 323)	0 (0 - 0)
15	25, 98th percentile value	270 (94 - 468)	0 (0 - 0)	0 (0 - 0)
15	65, 99th percentile value	522 (181 - 910)	282 (98 - 494)	41 (14 - 72)
15	40, 99th percentile value	522 (181 - 910)	282 (98 - 494)	41 (14 - 72)
15	35, 99th percentile value	507 (176 - 884)	266 (92 - 465)	23 (8 - 40)
15	30, 99th percentile value	356 (124 - 619)	97 (34 - 168)	0 (0 - 0)
15	25, 99th percentile value	207 (72 - 358)	0 (0 - 0)	0 (0 - 0)
14	40, 98th percentile value	438 (152 - 762)	188 (65 - 328)	0 (0 - 0)
14	35, 98th percentile value	438 (152 - 762)	188 (65 - 328)	0 (0 - 0)

Alternative Standards		PM-Related Incidence Associated with PM-2.5**		
Annual (ug/m3)	Daily (ug/m3)	BASE CASE: Lowest Measured Level in Study =7.5 µg/m3	Hypothetical Threshold =10 µg/m3	Hypothetical Threshold =12 µg/m3
14	30, 98th percentile value	435 (151 - 757)	185 (64 - 323)	0 (0 - 0)
14	25, 98th percentile value	270 (94 - 468)	0 (0 - 0)	0 (0 - 0)
14	40, 99th percentile value	438 (152 - 762)	188 (65 - 328)	0 (0 - 0)
14	35, 99th percentile value	438 (152 - 762)	188 (65 - 328)	0 (0 - 0)
14	30, 99th percentile value	356 (124 - 619)	97 (34 - 168)	0 (0 - 0)
14	25, 99th percentile value	207 (72 - 358)	0 (0 - 0)	0 (0 - 0)
13	40, 98th percentile value	354 (123 - 615)	94 (33 - 164)	0 (0 - 0)
13	35, 98th percentile value	354 (123 - 615)	94 (33 - 164)	0 (0 - 0)
13	30, 98th percentile value	354 (123 - 615)	94 (33 - 164)	0 (0 - 0)
13	25, 98th percentile value	270 (94 - 468)	0 (0 - 0)	0 (0 - 0)
13	40, 99th percentile value	354 (123 - 615)	94 (33 - 164)	0 (0 - 0)
13	35, 99th percentile value	354 (123 - 615)	94 (33 - 164)	0 (0 - 0)
13	30, 99th percentile value	354 (123 - 615)	94 (33 - 164)	0 (0 - 0)
13	25, 99th percentile value	207 (72 - 358)	0 (0 - 0)	0 (0 - 0)
12	40, 98th percentile value	271 (94 - 469)	0 (0 - 1)	0 (0 - 0)

Alternative Standards		PM-Related Incidence Associated with PM-2.5**		
Annual (ug/m3)	Daily (ug/m3)	BASE CASE: Lowest Measured Level in Study =7.5 µg/m3	Hypothetical Threshold =10 µg/m3	Hypothetical Threshold =12 µg/m3
12	35, 98th percentile value	271 (94 - 469)	0 (0 - 1)	0 (0 - 0)
12	30, 98th percentile value	271 (94 - 469)	0 (0 - 1)	0 (0 - 0)
12	25, 98th percentile value	270 (94 - 468)	0 (0 - 0)	0 (0 - 0)
12	40, 99th percentile value	271 (94 - 469)	0 (0 - 1)	0 (0 - 0)
12	35, 99th percentile value	271 (94 - 469)	0 (0 - 1)	0 (0 - 0)
12	30, 99th percentile value	271 (94 - 469)	0 (0 - 1)	0 (0 - 0)
12	25, 99th percentile value	207 (72 - 358)	0 (0 - 0)	0 (0 - 0)

*This sensitivity analysis was performed using Pope et al. (2002) – ACS extended. See text for an explanation of the slope adjustment method.

**In the base case, incidence was quantified across the range of PM concentrations observed in the study, when possible, but not below policy relevant background level. Under alternative threshold models, incidence was quantified down to the specified threshold. Incidences are rounded to the nearest whole number.

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

Table 4B-3. Sensitivity Analysis: Estimated Annual Mortality Associated with Short-Term Exposure to PM2.5 When Alternative Standards Are Just Met, in the Base Case and Using Alternative Hypothetical Threshold Models*
Los Angeles, CA, 2003

Alternative Standards		PM-Related Incidence Associated with PM-2.5**			
Annual (ug/m3)	Daily (ug/m3)	BASE CASE: Lowest Measured Level in Study =4 µg/m3	Hypothetical Threshold =10 µg/m3	Hypothetical Threshold =15 µg/m3	Hypothetical Threshold =20 µg/m3
15	65, 98th percentile value	248 (-31 - 519)	115 (-14 - 240)	58 (-7 - 121)	29 (-4 - 61)
15	40, 98th percentile value	248 (-31 - 519)	115 (-14 - 240)	58 (-7 - 121)	29 (-4 - 61)
15	35, 98th percentile value	225 (-28 - 471)	96 (-12 - 200)	45 (-6 - 94)	22 (-3 - 46)
15	30, 98th percentile value	184 (-23 - 384)	65 (-8 - 135)	26 (-3 - 54)	12 (-2 - 25)
15	25, 98th percentile value	142 (-18 - 297)	39 (-5 - 80)	13 (-2 - 27)	5 (-1 - 11)
15	65, 99th percentile value	248 (-31 - 519)	115 (-14 - 240)	58 (-7 - 121)	29 (-4 - 61)
15	40, 99th percentile value	154 (-19 - 321)	45 (-6 - 94)	16 (-2 - 33)	7 (-1 - 14)
15	35, 99th percentile value	127 (-16 - 266)	30 (-4 - 63)	10 (-1 - 20)	3 (0 - 7)
15	30, 99th percentile value	101 (-13 - 211)	18 (-2 - 37)	5 (-1 - 10)	1 (0 - 3)
15	25, 99th percentile value	76 (-10 - 158)	9 (-1 - 18)	2 (0 - 4)	0 (0 - 1)
14	40, 98th percentile value	224 (-28 - 470)	96 (-12 - 199)	45 (-6 - 93)	22 (-3 - 45)
14	35, 98th percentile value	224 (-28 - 470)	96 (-12 - 199)	45 (-6 - 93)	22 (-3 - 45)
14	30, 98th percentile value	184 (-23 - 384)	65 (-8 - 135)	26 (-3 - 54)	12 (-2 - 25)
14	25, 98th percentile value	142 (-18 - 297)	39 (-5 - 80)	13 (-2 - 27)	5 (-1 - 11)
14	40, 99th percentile value	154 (-19 - 321)	45 (-6 - 94)	16 (-2 - 33)	7 (-1 - 14)
14	35, 99th percentile value	127 (-16 - 266)	30 (-4 - 63)	10 (-1 - 20)	3 (0 - 7)
14	30, 99th percentile value	101 (-13 - 211)	18 (-2 - 37)	5 (-1 - 10)	1 (0 - 3)
14	25, 99th percentile value	76 (-10 - 158)	9 (-1 - 18)	2 (0 - 4)	0 (0 - 1)

Alternative Standards		PM-Related Incidence Associated with PM-2.5**			
Annual (ug/m3)	Daily (ug/m3)	BASE CASE: Lowest Measured Level in Study =4 µg/m3	Hypothetical Threshold =10 µg/m3	Hypothetical Threshold =15 µg/m3	Hypothetical Threshold =20 µg/m3
13	40, 98th percentile value	201 (-25 - 421)	77 (-10 - 161)	34 (-4 - 69)	16 (-2 - 33)
13	35, 98th percentile value	201 (-25 - 421)	77 (-10 - 161)	34 (-4 - 69)	16 (-2 - 33)
13	30, 98th percentile value	184 (-23 - 384)	65 (-8 - 135)	26 (-3 - 54)	12 (-2 - 25)
13	25, 98th percentile value	142 (-18 - 297)	39 (-5 - 80)	13 (-2 - 27)	5 (-1 - 11)
13	40, 99th percentile value	154 (-19 - 321)	45 (-6 - 94)	16 (-2 - 33)	7 (-1 - 14)
13	35, 99th percentile value	127 (-16 - 266)	30 (-4 - 63)	10 (-1 - 20)	3 (0 - 7)
13	30, 99th percentile value	101 (-13 - 211)	18 (-2 - 37)	5 (-1 - 10)	1 (0 - 3)
13	25, 99th percentile value	76 (-10 - 158)	9 (-1 - 18)	2 (0 - 4)	0 (0 - 1)
12	40, 98th percentile value	178 (-22 - 372)	61 (-8 - 126)	24 (-3 - 50)	11 (-1 - 23)
12	35, 98th percentile value	178 (-22 - 372)	61 (-8 - 126)	24 (-3 - 50)	11 (-1 - 23)
12	30, 98th percentile value	178 (-22 - 372)	61 (-8 - 126)	24 (-3 - 50)	11 (-1 - 23)
12	25, 98th percentile value	142 (-18 - 297)	39 (-5 - 80)	13 (-2 - 27)	5 (-1 - 11)
12	40, 99th percentile value	154 (-19 - 321)	45 (-6 - 94)	16 (-2 - 33)	7 (-1 - 14)
12	35, 99th percentile value	127 (-16 - 266)	30 (-4 - 63)	10 (-1 - 20)	3 (0 - 7)
12	30, 99th percentile value	101 (-13 - 211)	18 (-2 - 37)	5 (-1 - 10)	1 (0 - 3)
12	25, 99th percentile value	76 (-10 - 158)	9 (-1 - 18)	2 (0 - 4)	0 (0 - 1)

*This sensitivity analysis was performed using Moolgavkar (2003). See text for an explanation of the slope adjustment method.

**In the base case, incidence was quantified across the range of PM concentrations observed in the study, when possible, but not below policy relevant background level. Under alternative threshold models, incidence was quantified down to the specified threshold. Incidences are rounded to the nearest whole number.

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

Table 4B-4. Sensitivity Analysis: Estimated Annual Mortality Associated with Long-Term Exposure to PM2.5 When Alternative Standards Are Just Met, in the Base Case and Using Alternative Hypothetical Threshold Models*
Los Angeles, CA, 2003

Alternative Standards		PM-Related Incidence Associated with PM-2.5**		
Annual (ug/m3)	Daily (ug/m3)	BASE CASE: Lowest Measured Level in Study =7.5 µg/m3	Hypothetical Threshold =10 µg/m3	Hypothetical Threshold =12 µg/m3
15	65, 98th percentile value	1507 (531 - 2587)	823 (290 - 1415)	138 (48 - 237)
15	40, 98th percentile value	1507 (531 - 2587)	823 (290 - 1415)	138 (48 - 237)
15	35, 98th percentile value	1265 (446 - 2168)	553 (195 - 949)	0 (0 - 0)
15	30, 98th percentile value	829 (293 - 1416)	65 (23 - 111)	0 (0 - 0)
15	25, 98th percentile value	396 (140 - 675)	0 (0 - 0)	0 (0 - 0)
15	65, 99th percentile value	1507 (531 - 2587)	823 (290 - 1415)	138 (48 - 237)
15	40, 99th percentile value	514 (182 - 876)	0 (0 - 0)	0 (0 - 0)
15	35, 99th percentile value	240 (85 - 408)	0 (0 - 0)	0 (0 - 0)
15	30, 99th percentile value	0 (0 - 0)	0 (0 - 0)	0 (0 - 0)
15	25, 99th percentile value	0 (0 - 0)	0 (0 - 0)	0 (0 - 0)
14	40, 98th percentile value	1259 (444 - 2158)	546 (192 - 937)	0 (0 - 0)
14	35, 98th percentile value	1259 (444 - 2158)	546 (192 - 937)	0 (0 - 0)
14	30, 98th percentile value	829 (293 - 1416)	65 (23 - 111)	0 (0 - 0)
14	25, 98th percentile value	396 (140 - 675)	0 (0 - 0)	0 (0 - 0)
14	40, 99th percentile value	514 (182 - 876)	0 (0 - 0)	0 (0 - 0)
14	35, 99th percentile value	240 (85 - 408)	0 (0 - 0)	0 (0 - 0)

Alternative Standards		PM-Related Incidence Associated with PM-2.5**		
Annual (ug/m3)	Daily (ug/m3)	BASE CASE: Lowest Measured Level in Study =7.5 µg/m3	Hypothetical Threshold =10 µg/m3	Hypothetical Threshold =12 µg/m3
14	30, 99th percentile value	0 (0 - 0)	0 (0 - 0)	0 (0 - 0)
14	25, 99th percentile value	0 (0 - 0)	0 (0 - 0)	0 (0 - 0)
13	40, 98th percentile value	1013 (358 - 1732)	270 (95 - 463)	0 (0 - 0)
13	35, 98th percentile value	1013 (358 - 1732)	270 (95 - 463)	0 (0 - 0)
13	30, 98th percentile value	829 (293 - 1416)	65 (23 - 111)	0 (0 - 0)
13	25, 98th percentile value	396 (140 - 675)	0 (0 - 0)	0 (0 - 0)
13	40, 99th percentile value	514 (182 - 876)	0 (0 - 0)	0 (0 - 0)
13	35, 99th percentile value	240 (85 - 408)	0 (0 - 0)	0 (0 - 0)
13	30, 99th percentile value	0 (0 - 0)	0 (0 - 0)	0 (0 - 0)
13	25, 99th percentile value	0 (0 - 0)	0 (0 - 0)	0 (0 - 0)
12	40, 98th percentile value	767 (271 - 1310)	0 (0 - 0)	0 (0 - 0)
12	35, 98th percentile value	767 (271 - 1310)	0 (0 - 0)	0 (0 - 0)
12	30, 98th percentile value	767 (271 - 1310)	0 (0 - 0)	0 (0 - 0)
12	25, 98th percentile value	396 (140 - 675)	0 (0 - 0)	0 (0 - 0)
12	40, 99th percentile value	514 (182 - 876)	0 (0 - 0)	0 (0 - 0)
12	35, 99th percentile value	240 (85 - 408)	0 (0 - 0)	0 (0 - 0)
12	30, 99th percentile value	0 (0 - 0)	0 (0 - 0)	0 (0 - 0)
12	25, 99th percentile value	0 (0 - 0)	0 (0 - 0)	0 (0 - 0)

*This sensitivity analysis was performed using Pope et al. (2002) – ACS extended. See text for an explanation of the slope adjustment method.

**In the base case, incidence was quantified across the range of PM concentrations observed in the study, when possible, but not below policy relevant background level. Under alternative threshold models, incidence was quantified down to the specified threshold. Incidences are rounded to the nearest whole number.

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

Table 4B-5. Sensitivity Analysis: Estimated Annual Cardiovascular Mortality Associated with Short-Term Exposure to PM2.5 When Alternative Standards Are Just Met, in the Base Case and Using Alternative Hypothetical Threshold Models*
Philadelphia, PA, 2003

Alternative Standards		PM-Related Incidence Associated with PM-2.5**			
Annual (ug/m3)	Daily (ug/m3)	BASE CASE: Policy Relevant Background =3.5 µg/m3	Hypothetical Threshold =10 µg/m3	Hypothetical Threshold =15 µg/m3	Hypothetical Threshold =20 µg/m3
15	65, 98th percentile value	367 (175 - 560)	189 (90 - 288)	106 (51 - 162)	57 (27 - 87)
15	40, 98th percentile value	317 (151 - 482)	143 (68 - 218)	71 (34 - 107)	34 (16 - 51)
15	35, 98th percentile value	273 (130 - 416)	106 (50 - 161)	45 (22 - 69)	18 (9 - 28)
15	30, 98th percentile value	230 (110 - 350)	71 (34 - 108)	25 (12 - 38)	7 (3 - 11)
15	25, 98th percentile value	187 (89 - 284)	41 (20 - 63)	11 (5 - 16)	2 (1 - 3)
15	65, 99th percentile value	297 (142 - 451)	126 (60 - 191)	58 (28 - 89)	26 (12 - 40)
15	40, 99th percentile value	176 (84 - 268)	35 (17 - 53)	8 (4 - 12)	1 (1 - 2)
15	35, 99th percentile value	152 (72 - 231)	22 (11 - 34)	3 (2 - 5)	0 (0 - 1)
15	30, 99th percentile value	128 (61 - 195)	12 (6 - 19)	1 (1 - 2)	0 (0 - 0)
15	25, 99th percentile value	104 (49 - 158)	5 (2 - 8)	0 (0 - 0)	0 (0 - 0)
14	65, 98th percentile value	336 (160 - 511)	160 (76 - 243)	83 (40 - 127)	42 (20 - 63)
14	40, 98th percentile value	317 (151 - 482)	143 (68 - 218)	71 (34 - 107)	34 (16 - 51)
14	35, 98th percentile value	273 (130 - 416)	106 (50 - 161)	45 (22 - 69)	18 (9 - 28)
14	30, 98th percentile value	230 (110 - 350)	71 (34 - 108)	25 (12 - 38)	7 (3 - 11)
14	25, 98th percentile value	187 (89 - 284)	41 (20 - 63)	11 (5 - 16)	2 (1 - 3)
14	40, 99th percentile value	176 (84 - 268)	35 (17 - 53)	8 (4 - 12)	1 (1 - 2)
14	35, 99th percentile value	152 (72 - 231)	22 (11 - 34)	3 (2 - 5)	0 (0 - 1)
14	30, 99th percentile value	128 (61 - 195)	12 (6 - 19)	1 (1 - 2)	0 (0 - 0)
14	25, 99th percentile value	104 (49 - 158)	5 (2 - 8)	0 (0 - 0)	0 (0 - 0)

Alternative Standards		PM-Related Incidence Associated with PM-2.6**			
Annual (ug/m3)	Daily (ug/m3)	BASE CASE: Policy Relevant Background =3.5 µg/m3	Hypothetical Threshold =10 µg/m3	Hypothetical Threshold =15 µg/m3	Hypothetical Threshold =20 µg/m3
13	40, 98th percentile value	304 (145 - 462)	132 (63 - 200)	62 (30 - 95)	29 (14 - 44)
13	35, 98th percentile value	273 (130 - 416)	106 (50 - 161)	45 (22 - 69)	18 (9 - 28)
13	30, 98th percentile value	230 (110 - 350)	71 (34 - 108)	25 (12 - 38)	7 (3 - 11)
13	25, 98th percentile value	187 (89 - 284)	41 (20 - 63)	11 (5 - 16)	2 (1 - 3)
13	40, 99th percentile value	176 (84 - 268)	35 (17 - 53)	8 (4 - 12)	1 (1 - 2)
13	35, 99th percentile value	152 (72 - 231)	22 (11 - 34)	3 (2 - 5)	0 (0 - 1)
13	30, 99th percentile value	128 (61 - 195)	12 (6 - 19)	1 (1 - 2)	0 (0 - 0)
13	25, 99th percentile value	104 (49 - 158)	5 (2 - 8)	0 (0 - 0)	0 (0 - 0)
12	40, 98th percentile value	272 (130 - 414)	104 (50 - 159)	44 (21 - 68)	18 (9 - 27)
12	35, 98th percentile value	272 (130 - 414)	104 (50 - 159)	44 (21 - 68)	18 (9 - 27)
12	30, 98th percentile value	230 (110 - 350)	71 (34 - 108)	25 (12 - 38)	7 (3 - 11)
12	25, 98th percentile value	187 (89 - 284)	41 (20 - 63)	11 (5 - 16)	2 (1 - 3)
12	40, 99th percentile value	176 (84 - 268)	35 (17 - 53)	8 (4 - 12)	1 (1 - 2)
12	35, 99th percentile value	152 (72 - 231)	22 (11 - 34)	3 (2 - 5)	0 (0 - 1)
12	30, 99th percentile value	128 (61 - 195)	12 (6 - 19)	1 (1 - 2)	0 (0 - 0)
12	25, 99th percentile value	104 (49 - 158)	5 (2 - 8)	0 (0 - 0)	0 (0 - 0)

*This sensitivity analysis was performed using Lipfert et al. (2000). See text for an explanation of the slope adjustment method.

**In the base case, incidence was quantified across the range of PM concentrations observed in the study, when possible, but not below policy relevant background level. Under alternative threshold models, incidence was quantified down to the specified threshold. Incidences are rounded to the nearest whole number.

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

Table 4B-6. Sensitivity Analysis: Estimated Annual Mortality Associated with Long-Term Exposure to PM2.5 When Alternative Standards Are Just Met, in the Base Case and Using Alternative Hypothetical Threshold Models*
Philadelphia, PA, 2003

Alternative Standards		PM-Related Incidence Associated with PM-2.5**		
Annual (ug/m3)	Daily (ug/m3)	BASE CASE: Lowest Measured Level in Study =7.5 µg/m3	Hypothetical Threshold =10 µg/m3	Hypothetical Threshold =12 µg/m3
15	65, 98th percentile value	536 (185 - 943)	338 (116 - 597)	137 (47 - 244)
15	40, 98th percentile value	408 (141 - 716)	194 (67 - 341)	0 (0 - 0)
15	35, 98th percentile value	299 (104 - 524)	72 (25 - 126)	0 (0 - 0)
15	30, 98th percentile value	191 (67 - 334)	0 (0 - 0)	0 (0 - 0)
15	25, 98th percentile value	84 (29 - 146)	0 (0 - 0)	0 (0 - 0)
15	65, 99th percentile value	357 (124 - 626)	137 (47 - 241)	0 (0 - 0)
15	40, 99th percentile value	58 (20 - 101)	0 (0 - 0)	0 (0 - 0)
15	35, 99th percentile value	0 (0 - 0)	0 (0 - 0)	0 (0 - 0)
15	30, 99th percentile value	0 (0 - 0)	0 (0 - 0)	0 (0 - 0)
15	25, 99th percentile value	0 (0 - 0)	0 (0 - 0)	0 (0 - 0)
14	65, 98th percentile value	456 (157 - 799)	247 (85 - 435)	37 (13 - 65)
14	40, 98th percentile value	408 (141 - 716)	194 (67 - 341)	0 (0 - 0)
14	35, 98th percentile value	299 (104 - 524)	72 (25 - 126)	0 (0 - 0)
14	30, 98th percentile value	191 (67 - 334)	0 (0 - 0)	0 (0 - 0)
14	25, 98th percentile value	84 (29 - 146)	0 (0 - 0)	0 (0 - 0)
14	40, 99th percentile value	58 (20 - 101)	0 (0 - 0)	0 (0 - 0)

Alternative Standards		PM-Related Incidence Associated with PM-2.5**		
Annual (ug/m3)	Daily (ug/m3)	BASE CASE: Lowest Measured Level in Study =7.5 µg/m3	Hypothetical Threshold =10 µg/m3	Hypothetical Threshold =12 µg/m3
14	35, 99th percentile value	0 (0 - 0)	0 (0 - 0)	0 (0 - 0)
14	30, 99th percentile value	0 (0 - 0)	0 (0 - 0)	0 (0 - 0)
14	25, 99th percentile value	0 (0 - 0)	0 (0 - 0)	0 (0 - 0)
13	40, 98th percentile value	375 (130 - 657)	157 (54 - 276)	0 (0 - 0)
13	35, 98th percentile value	299 (104 - 524)	72 (25 - 126)	0 (0 - 0)
13	30, 98th percentile value	191 (67 - 334)	0 (0 - 0)	0 (0 - 0)
13	25, 98th percentile value	84 (29 - 146)	0 (0 - 0)	0 (0 - 0)
13	40, 99th percentile value	58 (20 - 101)	0 (0 - 0)	0 (0 - 0)
13	35, 99th percentile value	0 (0 - 0)	0 (0 - 0)	0 (0 - 0)
13	30, 99th percentile value	0 (0 - 0)	0 (0 - 0)	0 (0 - 0)
13	25, 99th percentile value	0 (0 - 0)	0 (0 - 0)	0 (0 - 0)
12	40, 98th percentile value	295 (102 - 516)	67 (23 - 118)	0 (0 - 0)
12	35, 98th percentile value	295 (102 - 516)	67 (23 - 118)	0 (0 - 0)
12	30, 98th percentile value	191 (67 - 334)	0 (0 - 0)	0 (0 - 0)
12	25, 98th percentile value	84 (29 - 146)	0 (0 - 0)	0 (0 - 0)
12	40, 99th percentile value	58 (20 - 101)	0 (0 - 0)	0 (0 - 0)
12	35, 99th percentile value	0 (0 - 0)	0 (0 - 0)	0 (0 - 0)
12	30, 99th percentile value	0 (0 - 0)	0 (0 - 0)	0 (0 - 0)
12	25, 99th percentile value	0 (0 - 0)	0 (0 - 0)	0 (0 - 0)

*This sensitivity analysis was performed using Pope et al. (2002) -- ACS extended. See text for an explanation of the slope adjustment method.

**In the base case, incidence was quantified across the range of PM concentrations observed in the study, when possible, but not below policy relevant background level. Under alternative threshold models, incidence was quantified down to the specified threshold. Incidences are rounded to the nearest whole number.

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

Table 4B-7. Sensitivity Analysis: Estimated Annual Mortality Associated with Short-Term Exposure to PM2.5 When Alternative Standards Are Just Met, in the Base Case and Using Alternative Hypothetical Threshold Models*
Pittsburgh, PA, 2003

Alternative Standards		PM-Related Incidence Associated with PM-2.5**			
Annual (ug/m3)	Daily (ug/m3)	BASE CASE: Policy Relevant Background =3.5 µg/m3	Hypothetical Threshold =10 µg/m3	Hypothetical Threshold =15 µg/m3	Hypothetical Threshold =20 µg/m3
15	65, 98th percentile value	50 (-108 - 200)	22 (-48 - 87)	10 (-23 - 41)	5 (-11 - 18)
15	40, 98th percentile value	47 (-102 - 189)	19 (-43 - 77)	9 (-19 - 34)	4 (-9 - 15)
15	35, 98th percentile value	41 (-88 - 162)	14 (-31 - 56)	5 (-12 - 21)	2 (-5 - 8)
15	30, 98th percentile value	34 (-74 - 136)	9 (-21 - 37)	3 (-6 - 11)	1 (-2 - 4)
15	25, 98th percentile value	28 (-60 - 110)	5 (-12 - 20)	1 (-3 - 5)	0 (-1 - 2)
15	65, 99th percentile value	50 (-108 - 200)	22 (-48 - 87)	10 (-23 - 41)	5 (-11 - 18)
15	40, 99th percentile value	42 (-92 - 168)	15 (-34 - 61)	6 (-13 - 24)	3 (-6 - 10)
15	35, 99th percentile value	36 (-79 - 145)	11 (-24 - 43)	4 (-8 - 14)	1 (-3 - 5)
15	30, 99th percentile value	31 (-67 - 122)	7 (-15 - 27)	2 (-4 - 7)	1 (-2 - 3)
15	25, 99th percentile value	25 (-54 - 99)	4 (-8 - 14)	1 (-2 - 3)	0 (-1 - 1)
14	40, 98th percentile value	46 (-99 - 182)	18 (-40 - 72)	8 (-17 - 31)	3 (-8 - 13)
14	35, 98th percentile value	41 (-88 - 162)	14 (-31 - 56)	5 (-12 - 21)	2 (-5 - 8)
14	30, 98th percentile value	34 (-74 - 136)	9 (-21 - 37)	3 (-6 - 11)	1 (-2 - 4)
14	25, 98th percentile value	28 (-60 - 110)	5 (-12 - 20)	1 (-3 - 5)	0 (-1 - 2)
14	40, 99th percentile value	42 (-92 - 168)	15 (-34 - 61)	6 (-13 - 24)	3 (-6 - 10)
14	35, 99th percentile value	36 (-79 - 145)	11 (-24 - 43)	4 (-8 - 14)	1 (-3 - 5)
14	30, 99th percentile value	31 (-67 - 122)	7 (-15 - 27)	2 (-4 - 7)	1 (-2 - 3)
14	25, 99th percentile value	25 (-54 - 99)	4 (-8 - 14)	1 (-2 - 3)	0 (-1 - 1)

Alternative Standards		PM-Related Incidence Associated with PM-2.5**			
Annual (ug/m3)	Daily (ug/m3)	BASE CASE: Policy Relevant Background =3.5 µg/m3	Hypothetical Threshold =10 µg/m3	Hypothetical Threshold =15 µg/m3	Hypothetical Threshold =20 µg/m3
13	40, 98th percentile value	41 (-90 - 165)	15 (-32 - 58)	6 (-13 - 22)	2 (-5 - 9)
13	35, 98th percentile value	41 (-88 - 162)	14 (-31 - 56)	5 (-12 - 21)	2 (-5 - 8)
13	30, 98th percentile value	34 (-74 - 136)	9 (-21 - 37)	3 (-6 - 11)	1 (-2 - 4)
13	25, 98th percentile value	28 (-60 - 110)	5 (-12 - 20)	1 (-3 - 5)	0 (-1 - 2)
13	40, 99th percentile value	41 (-90 - 165)	15 (-32 - 58)	6 (-13 - 22)	2 (-5 - 9)
13	35, 99th percentile value	36 (-79 - 145)	11 (-24 - 43)	4 (-8 - 14)	1 (-3 - 5)
13	30, 99th percentile value	31 (-67 - 122)	7 (-15 - 27)	2 (-4 - 7)	1 (-2 - 3)
13	25, 99th percentile value	25 (-54 - 99)	4 (-8 - 14)	1 (-2 - 3)	0 (-1 - 1)
12	40, 98th percentile value	37 (-80 - 147)	11 (-25 - 44)	4 (-8 - 15)	1 (-3 - 6)
12	35, 98th percentile value	37 (-80 - 147)	11 (-25 - 44)	4 (-8 - 15)	1 (-3 - 6)
12	30, 98th percentile value	34 (-74 - 136)	9 (-21 - 37)	3 (-6 - 11)	1 (-2 - 4)
12	25, 98th percentile value	28 (-60 - 110)	5 (-12 - 20)	1 (-3 - 5)	0 (-1 - 2)
12	40, 99th percentile value	37 (-80 - 147)	11 (-25 - 44)	4 (-8 - 15)	1 (-3 - 6)
12	35, 99th percentile value	36 (-79 - 145)	11 (-24 - 43)	4 (-8 - 14)	1 (-3 - 5)
12	30, 99th percentile value	31 (-67 - 122)	7 (-15 - 27)	2 (-4 - 7)	1 (-2 - 3)
12	25, 99th percentile value	25 (-54 - 99)	4 (-8 - 14)	1 (-2 - 3)	0 (-1 - 1)

*This sensitivity analysis was performed using Chock et al. (2000), age 75+ model. See text for an explanation of the slope adjustment method.

**In the base case, incidence was quantified across the range of PM concentrations observed in the study, when possible, but not below policy relevant background level. Under alternative threshold models, incidence was quantified down to the *specified threshold*. Incidences are rounded to the nearest whole number.

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

Table 4B-8. Sensitivity Analysis: Estimated Annual Mortality Associated with Long-Term Exposure to PM2.5 When Alternative Standards Are Just Met, in the Base Case and Using Alternative Hypothetical Threshold Models*
Pittsburgh, PA, 2003

Alternative Standards		PM-Related Incidence Associated with PM-2.5**		
Annual (ug/m3)	Daily (ug/m3)	BASE CASE: Lowest Measured Level in Study =7.5 µg/m3	Hypothetical Threshold =10 µg/m3	Hypothetical Threshold =12 µg/m3
15	65, 98th percentile value	403 (141 - 699)	215 (75 - 373)	25 (9 - 43)
15	40, 98th percentile value	361 (126 - 626)	168 (58 - 291)	0 (0 - 0)
15	35, 98th percentile value	264 (93 - 456)	59 (21 - 102)	0 (0 - 0)
15	30, 98th percentile value	168 (59 - 289)	0 (0 - 0)	0 (0 - 0)
15	25, 98th percentile value	72 (25 - 124)	0 (0 - 0)	0 (0 - 0)
15	65, 99th percentile value	403 (141 - 699)	215 (75 - 373)	25 (9 - 43)
15	40, 99th percentile value	287 (100 - 495)	84 (29 - 145)	0 (0 - 0)
15	35, 99th percentile value	200 (70 - 345)	0 (0 - 0)	0 (0 - 0)
15	30, 99th percentile value	114 (40 - 197)	0 (0 - 0)	0 (0 - 0)
15	25, 99th percentile value	29 (10 - 50)	0 (0 - 0)	0 (0 - 0)
14	40, 98th percentile value	338 (118 - 585)	141 (49 - 245)	0 (0 - 0)
14	35, 98th percentile value	264 (93 - 456)	59 (21 - 102)	0 (0 - 0)
14	30, 98th percentile value	168 (59 - 289)	0 (0 - 0)	0 (0 - 0)
14	25, 98th percentile value	72 (25 - 124)	0 (0 - 0)	0 (0 - 0)
14	40, 99th percentile value	287 (100 - 495)	84 (29 - 145)	0 (0 - 0)
14	35, 99th percentile value	200 (70 - 345)	0 (0 - 0)	0 (0 - 0)
14	30, 99th percentile value	114 (40 - 197)	0 (0 - 0)	0 (0 - 0)
14	25, 99th percentile value	29 (10 - 50)	0 (0 - 0)	0 (0 - 0)

Alternative Standards		PM-Related Incidence Associated with PM-2.5**		
Annual (ug/m3)	Daily (ug/m3)	BASE CASE: Lowest Measured Level in Study =7.5 µg/m3	Hypothetical Threshold =10 µg/m3	Hypothetical Threshold =12 µg/m3
13	40, 98th percentile value	273 (96 - 471)	68 (24 - 118)	0 (0 - 0)
13	35, 98th percentile value	264 (93 - 456)	59 (21 - 102)	0 (0 - 0)
13	30, 98th percentile value	168 (59 - 289)	0 (0 - 0)	0 (0 - 0)
13	25, 98th percentile value	72 (25 - 124)	0 (0 - 0)	0 (0 - 0)
13	40, 99th percentile value	273 (96 - 471)	68 (24 - 118)	0 (0 - 0)
13	35, 99th percentile value	200 (70 - 345)	0 (0 - 0)	0 (0 - 0)
13	30, 99th percentile value	114 (40 - 197)	0 (0 - 0)	0 (0 - 0)
13	25, 99th percentile value	29 (10 - 50)	0 (0 - 0)	0 (0 - 0)
12	40, 98th percentile value	208 (73 - 358)	0 (0 - 0)	0 (0 - 0)
12	35, 98th percentile value	208 (73 - 358)	0 (0 - 0)	0 (0 - 0)
12	30, 98th percentile value	168 (59 - 289)	0 (0 - 0)	0 (0 - 0)
12	25, 98th percentile value	72 (25 - 124)	0 (0 - 0)	0 (0 - 0)
12	40, 99th percentile value	208 (73 - 358)	0 (0 - 0)	0 (0 - 0)
12	35, 99th percentile value	200 (70 - 345)	0 (0 - 0)	0 (0 - 0)
12	30, 99th percentile value	114 (40 - 197)	0 (0 - 0)	0 (0 - 0)
12	25, 99th percentile value	29 (10 - 50)	0 (0 - 0)	0 (0 - 0)

*This sensitivity analysis was performed using Pope et al. (2002) -- ACS extended. See text for an explanation of the slope adjustment method.

**In the base case, incidence was quantified across the range of PM concentrations observed in the study, when possible, but not below policy relevant background level. Under alternative threshold models, incidence was quantified down to the specified threshold. Incidences are rounded to the nearest whole number.

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

Table 4B-9. Sensitivity Analysis: Estimated Annual Mortality Associated with Short-Term Exposure to PM2.5 When Alternative Standards Are Just Met, in the Base Case and Using Alternative Hypothetical Threshold Models*
St. Louis, MO, 2003

Alternative Standards		PM-Related Incidence Associated with PM-2.5**			
Annual (ug/m3)	Daily (ug/m3)	BASE CASE: Policy Relevant Background =3.5 µg/m3	Hypothetical Threshold =10 µg/m3	Hypothetical Threshold =15 µg/m3	Hypothetical Threshold =20 µg/m3
15	65, 98th percentile value	191 (70 - 311)	75 (28 - 122)	29 (11 - 46)	9 (3 - 14)
15	40, 98th percentile value	191 (70 - 311)	75 (28 - 122)	29 (11 - 46)	9 (3 - 14)
15	35, 98th percentile value	190 (70 - 310)	75 (27 - 121)	28 (10 - 46)	8 (3 - 14)
15	30, 98th percentile value	160 (59 - 260)	49 (18 - 80)	14 (5 - 23)	3 (1 - 4)
15	25, 98th percentile value	130 (48 - 211)	28 (10 - 45)	5 (2 - 8)	1 (0 - 1)
15	65, 99th percentile value	191 (70 - 311)	75 (28 - 122)	29 (11 - 46)	9 (3 - 14)
15	40, 99th percentile value	191 (70 - 311)	75 (28 - 122)	29 (11 - 46)	9 (3 - 14)
15	35, 99th percentile value	172 (63 - 280)	59 (22 - 96)	19 (7 - 31)	5 (2 - 7)
15	30, 99th percentile value	145 (53 - 235)	38 (14 - 62)	9 (3 - 14)	2 (1 - 3)
15	25, 99th percentile value	118 (43 - 191)	20 (7 - 33)	3 (1 - 4)	0 (0 - 1)
14	40, 98th percentile value	175 (64 - 284)	61 (22 - 99)	20 (7 - 33)	5 (2 - 8)
14	35, 98th percentile value	175 (64 - 284)	61 (22 - 99)	20 (7 - 33)	5 (2 - 8)
14	30, 98th percentile value	160 (59 - 260)	49 (18 - 80)	14 (5 - 23)	3 (1 - 4)
14	25, 98th percentile value	130 (48 - 211)	28 (10 - 45)	5 (2 - 8)	1 (0 - 1)
14	40, 99th percentile value	175 (64 - 284)	61 (22 - 99)	20 (7 - 33)	5 (2 - 8)
14	35, 99th percentile value	172 (63 - 280)	59 (22 - 96)	19 (7 - 31)	5 (2 - 7)

Alternative Standards		PM-Related Incidence Associated with PM-2.5**			
Annual (ug/m3)	Daily (ug/m3)	BASE CASE: Policy Relevant Background =3.5 µg/m3	Hypothetical Threshold =10 µg/m3	Hypothetical Threshold =15 µg/m3	Hypothetical Threshold =20 µg/m3
14	30, 99th percentile value	145 (53 - 235)	38 (14 - 62)	9 (3 - 14)	2 (1 - 3)
14	25, 99th percentile value	118 (43 - 191)	20 (7 - 33)	3 (1 - 4)	0 (0 - 1)
13	40, 98th percentile value	158 (58 - 256)	47 (17 - 77)	13 (5 - 21)	3 (1 - 4)
13	35, 98th percentile value	158 (58 - 256)	47 (17 - 77)	13 (5 - 21)	3 (1 - 4)
13	30, 98th percentile value	158 (58 - 256)	47 (17 - 77)	13 (5 - 21)	3 (1 - 4)
13	25, 98th percentile value	130 (48 - 211)	28 (10 - 45)	5 (2 - 8)	1 (0 - 1)
13	40, 99th percentile value	158 (58 - 256)	47 (17 - 77)	13 (5 - 21)	3 (1 - 4)
13	35, 99th percentile value	158 (58 - 256)	47 (17 - 77)	13 (5 - 21)	3 (1 - 4)
13	30, 99th percentile value	145 (53 - 235)	38 (14 - 62)	9 (3 - 14)	2 (1 - 3)
13	25, 99th percentile value	118 (43 - 191)	20 (7 - 33)	3 (1 - 4)	0 (0 - 1)
12	40, 98th percentile value	141 (52 - 229)	35 (13 - 57)	8 (3 - 12)	1 (1 - 2)
12	35, 98th percentile value	141 (52 - 229)	35 (13 - 57)	8 (3 - 12)	1 (1 - 2)
12	30, 98th percentile value	141 (52 - 229)	35 (13 - 57)	8 (3 - 12)	1 (1 - 2)
12	25, 98th percentile value	130 (48 - 211)	28 (10 - 45)	5 (2 - 8)	1 (0 - 1)
12	40, 99th percentile value	141 (52 - 229)	35 (13 - 57)	8 (3 - 12)	1 (1 - 2)
12	35, 99th percentile value	141 (52 - 229)	35 (13 - 57)	8 (3 - 12)	1 (1 - 2)
12	30, 99th percentile value	141 (52 - 229)	35 (13 - 57)	8 (3 - 12)	1 (1 - 2)
12	25, 99th percentile value	118 (43 - 191)	20 (7 - 33)	3 (1 - 4)	0 (0 - 1)

*This sensitivity analysis was performed using Schwartz (2003b). See text for an explanation of the slope adjustment method.

Table 4B-10. Sensitivity Analysis: Estimated Annual Mortality Associated with Long-Term Exposure to PM2.5 When Alternative Standards Are Just Met, in the Base Case and Using Alternative Hypothetical Threshold Models*
St. Louis, MO, 2003

Alternative Standards		PM-Related Incidence Associated with PM-2.5**		
Annual (ug/m3)	Daily (ug/m3)	BASE CASE: Lowest Measured Level in Study =7.5 µg/m3	Hypothetical Threshold =10 µg/m3	Hypothetical Threshold =12 µg/m3
15	65, 98th percentile value	596 (206 - 1047)	311 (107 - 548)	23 (8 - 40)
15	40, 98th percentile value	596 (206 - 1047)	311 (107 - 548)	23 (8 - 40)
15	35, 98th percentile value	592 (204 - 1039)	306 (105 - 539)	17 (6 - 30)
15	30, 98th percentile value	414 (144 - 726)	107 (37 - 188)	0 (0 - 0)
15	25, 98th percentile value	239 (83 - 417)	0 (0 - 0)	0 (0 - 0)
15	65, 99th percentile value	596 (206 - 1047)	311 (107 - 548)	23 (8 - 40)
15	40, 99th percentile value	596 (206 - 1047)	311 (107 - 548)	23 (8 - 40)
15	35, 99th percentile value	486 (168 - 853)	188 (65 - 330)	0 (0 - 0)
15	30, 99th percentile value	327 (113 - 571)	8 (3 - 15)	0 (0 - 0)
15	25, 99th percentile value	168 (58 - 293)	0 (0 - 0)	0 (0 - 0)
14	40, 98th percentile value	498 (172 - 874)	201 (69 - 354)	0 (0 - 0)
14	35, 98th percentile value	498 (172 - 874)	201 (69 - 354)	0 (0 - 0)
14	30, 98th percentile value	414 (144 - 726)	107 (37 - 188)	0 (0 - 0)
14	25, 98th percentile value	239 (83 - 417)	0 (0 - 0)	0 (0 - 0)
14	40, 99th percentile value	498 (172 - 874)	201 (69 - 354)	0 (0 - 0)
14	35, 99th percentile value	486 (168 - 853)	188 (65 - 330)	0 (0 - 0)
14	30, 99th percentile value	327 (113 - 571)	8 (3 - 15)	0 (0 - 0)
14	25, 99th percentile value	168 (58 - 293)	0 (0 - 0)	0 (0 - 0)

Alternative Standards		PM-Related Incidence Associated with PM-2.5**		
Annual (ug/m3)	Daily (ug/m3)	BASE CASE: Lowest Measured Level in Study =7.5 µg/m3	Hypothetical Threshold =10 µg/m3	Hypothetical Threshold =12 µg/m3
13	40, 98th percentile value	401 (139 - 702)	92 (32 - 162)	0 (0 - 0)
13	35, 98th percentile value	401 (139 - 702)	92 (32 - 162)	0 (0 - 0)
13	30, 98th percentile value	401 (139 - 702)	92 (32 - 162)	0 (0 - 0)
13	25, 98th percentile value	239 (83 - 417)	0 (0 - 0)	0 (0 - 0)
13	40, 99th percentile value	401 (139 - 702)	92 (32 - 162)	0 (0 - 0)
13	35, 99th percentile value	401 (139 - 702)	92 (32 - 162)	0 (0 - 0)
13	30, 99th percentile value	327 (113 - 571)	8 (3 - 15)	0 (0 - 0)
13	25, 99th percentile value	168 (58 - 293)	0 (0 - 0)	0 (0 - 0)
12	40, 98th percentile value	304 (106 - 532)	0 (0 - 0)	0 (0 - 0)
12	35, 98th percentile value	304 (106 - 532)	0 (0 - 0)	0 (0 - 0)
12	30, 98th percentile value	304 (106 - 532)	0 (0 - 0)	0 (0 - 0)
12	25, 98th percentile value	239 (83 - 417)	0 (0 - 0)	0 (0 - 0)
12	40, 99th percentile value	304 (106 - 532)	0 (0 - 0)	0 (0 - 0)
12	35, 99th percentile value	304 (106 - 532)	0 (0 - 0)	0 (0 - 0)
12	30, 99th percentile value	304 (106 - 532)	0 (0 - 0)	0 (0 - 0)
12	25, 99th percentile value	168 (58 - 293)	0 (0 - 0)	0 (0 - 0)

*This sensitivity analysis was performed using Pope et al. (2002) – ACS extended. See text for an explanation of the slope adjustment method.

**In the base case, incidence was quantified across the range of PM concentrations observed in the study, when possible, but not below policy relevant background level. Under alternative threshold models, incidence was quantified down to the specified threshold. Incidences are rounded to the nearest whole number.

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

Table 4B-11. Sensitivity Analysis: Estimated Annual Hospital Admissions for Ischemic Heart Disease Associated with Short-Term Exposure to PM10-2.5 When Alternative Standards Are Just Met, in the Base Case and Using Alternative Hypothetical Threshold Models*

Detroit, MI, 2003

(As Is = 21.7 ug/m3 Annual Average; 105.9 ug/m3 98th Percentile Daily Value)

"As Is" PM10-2.5 Concentrations and Alternative Standards	PM-Related Incidence Associated with PM10-2.5**			
	BASE CASE: Policy Relevant Background =4.5 µg/m3	Hypothetical Threshold =10 µg/m3	Hypothetical Threshold =15 µg/m3	Hypothetical Threshold =20 µg/m3
"As is" PM10-2.5 concentrations	654 (169 - 1083)	569 (149 - 934)	489 (129 - 794)	426 (115 - 682)
80 ug/m3 daily 98th percentile value	654 (169 - 1083)	569 (149 - 934)	489 (129 - 794)	426 (115 - 682)
65 ug/m3 daily 98th percentile value	600 (156 - 989)	508 (134 - 829)	425 (114 - 683)	360 (99 - 567)
50 ug/m3 daily 98th percentile value	443 (117 - 719)	334 (90 - 532)	248 (69 - 384)	183 (54 - 271)
30 ug/m3 daily 98th percentile value	242 (65 - 386)	125 (36 - 190)	65 (20 - 91)	44 (15 - 57)
25 ug/m3 daily 98th percentile value	193 (52 - 307)	81 (24 - 120)	39 (13 - 52)	25 (9 - 30)
100 ug/m3 daily 99th percentile value	654 (169 - 1083)	569 (149 - 934)	489 (129 - 794)	426 (115 - 682)
80 ug/m3 daily 99th percentile value	654 (169 - 1083)	569 (149 - 934)	489 (129 - 794)	426 (115 - 682)
60 ug/m3 daily 99th percentile value	491 (129 - 801)	387 (104 - 621)	301 (83 - 472)	233 (67 - 353)
35 ug/m3 daily 99th percentile value	262 (70 - 419)	144 (41 - 221)	79 (24 - 113)	53 (18 - 68)
30 ug/m3 daily 99th percentile value	218 (59 - 347)	103 (30 - 154)	51 (16 - 70)	34 (12 - 43)

*This sensitivity analysis was performed using Ito (2003). See text for an explanation of the slope adjustment method.

**In the base case, incidence was quantified across the range of PM10-2.5 concentrations observed in the study, when possible, but not below policy relevant background level. Under alternative threshold models, incidence was quantified down to the specified threshold. Incidences are rounded to the nearest whole number.

Note: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM10-2.5 coefficient.

Table 4B-12. Sensitivity Analysis: Estimated Annual Hospital Admissions for Asthma (Age < 65) Associated with Short-Term Exposure to PM10-2.5 When Alternative Standards Are Just Met, in the Base Case and Using Alternative Hypothetical Threshold Models*

Seattle, WA, 2003

(As Is = 11.4 ug/m3 Annual Average; 26.2 ug/m3 98th Percentile Daily Value)

"As Is" PM10-2.5 Concentrations and Alternative Standards	PM-Related Incidence Associated with PM10-2.5**			
	BASE CASE: Policy Relevant Background =3.5 µg/m3	Hypothetical Threshold =10 µg/m3	Hypothetical Threshold =15 µg/m3	Hypothetical Threshold =20 µg/m3
"As is" PM10-2.5 concentrations	27 (0 - 65)	12 (0 - 28)	5 (0 - 11)	2 (0 - 4)
80 ug/m3 daily 98th percentile value	27 (0 - 65)	12 (0 - 28)	5 (0 - 11)	2 (0 - 4)
65 ug/m3 daily 98th percentile value	27 (0 - 65)	12 (0 - 28)	5 (0 - 11)	2 (0 - 4)
50 ug/m3 daily 98th percentile value	27 (0 - 65)	12 (0 - 28)	5 (0 - 11)	2 (0 - 4)
30 ug/m3 daily 98th percentile value	26 (0 - 63)	11 (0 - 26)	4 (0 - 10)	1 (0 - 3)
25 ug/m3 daily 98th percentile value	21 (0 - 51)	7 (0 - 16)	2 (0 - 5)	0 (0 - 1)
100 ug/m3 daily 99th percentile value	27 (0 - 65)	12 (0 - 28)	5 (0 - 11)	2 (0 - 4)
80 ug/m3 daily 99th percentile value	27 (0 - 65)	12 (0 - 28)	5 (0 - 11)	2 (0 - 4)
60 ug/m3 daily 99th percentile value	27 (0 - 65)	12 (0 - 28)	5 (0 - 11)	2 (0 - 4)
35 ug/m3 daily 99th percentile value	24 (0 - 58)	9 (0 - 22)	3 (0 - 8)	1 (0 - 2)
30 ug/m3 daily 99th percentile value	20 (0 - 48)	6 (0 - 14)	2 (0 - 4)	0 (0 - 1)

*This sensitivity analysis was performed using Sheppard (2003). See text for an explanation of the slope adjustment method.

**In the base case, incidence was quantified across the range of PM10-2.5 concentrations observed in the study, when possible, but not below policy relevant background level. Under alternative threshold models, incidence was quantified down to the specified threshold. Incidences are rounded to the nearest whole number.

Note: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM10-2.5 coefficient.

Table 4B-13. Sensitivity Analysis: Estimated Annual Days of Cough Among Children Associated with Short-Term Exposure to PM10-2.5 When Alternative Standards Are Just Met, in the Base Case and Using Alternative Hypothetical Threshold Models*

St. Louis, MO, 2003

(As Is = 12.0 ug/m3 Annual Average; 24.1 ug/m3 98th Percentile Daily Value)

"As Is" PM10-2.5 Concentrations and Alternative Standards	PM-Related Incidence Associated with PM10-2.5**			
	BASE CASE: Policy Relevant Background =4.5 µg/m3	Hypothetical Threshold =10 µg/m3	Hypothetical Threshold =15 µg/m3	Hypothetical Threshold =20 µg/m3
"As is" PM10-2.5 concentrations	27000 (11000 - 40900)	12100 (4900 - 18100)	5800 (2500 - 8600)	2900 (1300 - 4000)
80 ug/m3 daily 98th percentile value	27000 (11000 - 40900)	12100 (4900 - 18100)	5800 (2500 - 8600)	2900 (1300 - 4000)
65 ug/m3 daily 98th percentile value	27000 (11000 - 40900)	12100 (4900 - 18100)	5800 (2500 - 8600)	2900 (1300 - 4000)
50 ug/m3 daily 98th percentile value	27000 (11000 - 40900)	12100 (4900 - 18100)	5800 (2500 - 8600)	2900 (1300 - 4000)
30 ug/m3 daily 98th percentile value	23800 (9800 - 35600)	9100 (3800 - 13300)	4200 (1800 - 6000)	2200 (1000 - 2900)
25 ug/m3 daily 98th percentile value	18600 (7800 - 27400)	5300 (2300 - 7300)	2000 (900 - 2500)	1300 (600 - 1600)
100 ug/m3 daily 99th percentile value	27000 (11000 - 40900)	12100 (4900 - 18100)	5800 (2500 - 8600)	2900 (1300 - 4000)
80 ug/m3 daily 99th percentile value	27000 (11000 - 40900)	12100 (4900 - 18100)	5800 (2500 - 8600)	2900 (1300 - 4000)
60 ug/m3 daily 99th percentile value	27000 (11000 - 40900)	12100 (4900 - 18100)	5800 (2500 - 8600)	2900 (1300 - 4000)
35 ug/m3 daily 99th percentile value	18600 (7700 - 27300)	5200 (2300 - 7300)	1900 (900 - 2500)	1200 (600 - 1600)
30 ug/m3 daily 99th percentile value	15200 (6400 - 22200)	3300 (1500 - 4400)	1100 (600 - 1400)	700 (400 - 900)

*This sensitivity analysis was performed using Schwartz and Neas (2000) -- 6 cities. See text for an explanation of the slope adjustment method.

**In the base case, incidence was quantified across the range of PM concentrations observed in the study, when possible, but not below policy relevant background level. Under alternative threshold models, incidence was quantified down to the specified threshold. Incidences are rounded to the nearest 100.

Note: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the PM10-2.5 coefficient.

Table 4B-14. Sensitivity Analysis: Estimated Annual Mortality Associated with Short-Term Exposure to PM2.5 When Alternative Standards Are Just Met, in the Base Case and Using Alternative Hypothetical Threshold Models – Rollbacks to Meet Annual Standards Using Design Values Based on Maximum vs. Average of Monitor-Specific Averages*
Detroit, MI, 2003

Alternative Standards		PM-Related Incidence Associated with PM-2.5 Using Annual Standard Design Values Based on the Maximum of Monitor-Specific Averages**.				PM-Related Incidence Associated with PM-2.5 Using Annual Standard Design Values Based on the Average of Monitor-Specific Averages**			
		Percent Reduction in Incidence from Current Standards				Percent Reduction in Incidence from Current Standards			
Annual (ug/m3)	Daily (ug/m3)	BASE CASE: Lowest Measured Level in Study =4 µg/m3	Hypothetical Threshold =10 µg/m3	Hypothetical Threshold =15 µg/m3	Hypothetical Threshold =20 µg/m3	BASE CASE: Lowest Measured Level in Study =4 µg/m3	Hypothetical Threshold =10 µg/m3	Hypothetical Threshold =15 µg/m3	Hypothetical Threshold =20 µg/m3
15	65, 98th percentile value***	115 (-116 - 338) 0.0%	54 (-55 - 159) 0.0%	26 (-27 - 77) 0.0%	12 (-12 - 35) 0.0%	143 (-144 - 422) 0.0%	80 (-81 - 236) 0.0%	46 (-47 - 137) 0.0%	25 (-26 - 75) 0.0%
15	40, 98th percentile value	115 (-116 - 338) 0.0%	54 (-55 - 159) 0.0%	26 (-27 - 77) 0.0%	12 (-12 - 35) 0.0%	143 (-144 - 422) 0.0%	80 (-81 - 236) 0.0%	46 (-47 - 137) 0.0%	25 (-26 - 75) 0.0%
15	35, 98th percentile value	115 (-116 - 338) 0.0%	54 (-55 - 159) 0.0%	26 (-27 - 77) 0.0%	12 (-12 - 35) 0.0%	125 (-126 - 368) 12.6%	63 (-64 - 186) 21.3%	33 (-33 - 97) 28.3%	16 (-17 - 47) 36.0%
15	65, 99th percentile value	115 (-116 - 338) 0.0%	54 (-55 - 159) 0.0%	26 (-27 - 77) 0.0%	12 (-12 - 35) 0.0%	143 (-144 - 422) 0.0%	80 (-81 - 236) 0.0%	46 (-47 - 137) 0.0%	25 (-26 - 75) 0.0%
15	40, 99th percentile value	115 (-116 - 338) 0.0%	54 (-55 - 159) 0.0%	26 (-27 - 77) 0.0%	12 (-12 - 35) 0.0%	132 (-133 - 389) 7.7%	70 (-70 - 206) 12.5%	38 (-39 - 112) 17.4%	20 (-20 - 58) 20.0%
14	40, 98th percentile value	104 (-105 - 307) 9.6%	45 (-46 - 132) 16.7%	20 (-20 - 58) 23.1%	8 (-9 - 24) 33.3%	130 (-131 - 383) 9.1%	68 (-68 - 200) 15.0%	37 (-37 - 108) 19.6%	19 (-19 - 55) 24.0%
14	35, 98th percentile value	104 (-105 - 307) 9.6%	45 (-46 - 132) 16.7%	20 (-20 - 58) 23.1%	8 (-9 - 24) 33.3%	125 (-126 - 368) 12.6%	63 (-64 - 186) 21.3%	33 (-33 - 97) 28.3%	16 (-17 - 47) 36.0%
14	40, 99th percentile value	104 (-105 - 307) 9.6%	45 (-46 - 132) 16.7%	20 (-20 - 58) 23.1%	8 (-9 - 24) 33.3%	130 (-131 - 383) 9.1%	68 (-68 - 200) 15.0%	37 (-37 - 108) 19.6%	19 (-19 - 55) 24.0%
14	35, 99th percentile value	104 (-105 - 307) 9.6%	45 (-46 - 132) 16.7%	20 (-20 - 58) 23.1%	8 (-9 - 24) 33.3%	113 (-114 - 333) 21.0%	53 (-53 - 154) 33.8%	25 (-26 - 74) 45.7%	11 (-12 - 33) 56.0%

Alternative Standards		PM-Related Incidence Associated with PM-2.5 Using Annual Standard Design Values Based on the Maximum of Monitor-Specific Averages**				PM-Related Incidence Associated with PM-2.5 Using Annual Standard Design Values Based on the Average of Monitor-Specific Averages**			
		Percent Reduction in Incidence from Current Standards				Percent Reduction in Incidence from Current Standards			
Annual (ug/m3)	Daily (ug/m3)	BASE CASE: Lowest Measured Level in Study =4 µg/m3	Hypothetical Threshold =10 µg/m3	Hypothetical Threshold =15 µg/m3	Hypothetical Threshold =20 µg/m3	BASE CASE: Lowest Measured Level in Study =4 µg/m3	Hypothetical Threshold =10 µg/m3	Hypothetical Threshold =15 µg/m3	Hypothetical Threshold =20 µg/m3
13	40, 98th percentile value	94 (-95 - 275) 18.3%	36 (-37 - 106) 33.3%	14 (-15 - 42) 46.2%	6 (-6 - 16) 50.0%	117 (-118 - 344) 18.2%	56 (-57 - 165) 30.0%	28 (-28 - 81) 39.1%	13 (-13 - 38) 46.0%
13	35, 98th percentile value	94 (-95 - 275) 18.3%	36 (-37 - 106) 33.3%	14 (-15 - 42) 46.2%	6 (-6 - 16) 50.0%	117 (-118 - 344) 18.2%	56 (-57 - 165) 30.0%	28 (-28 - 81) 39.1%	13 (-13 - 38) 46.0%
13	30, 98th percentile value	94 (-95 - 275) 18.3%	36 (-37 - 106) 33.3%	14 (-15 - 42) 46.2%	6 (-6 - 16) 50.0%	104 (-105 - 306) 27.3%	45 (-45 - 131) 43.8%	20 (-20 - 58) 56.5%	8 (-9 - 24) 68.0%
13	40, 99th percentile value	94 (-95 - 275) 18.3%	36 (-37 - 106) 33.3%	14 (-15 - 42) 46.2%	6 (-6 - 16) 50.0%	117 (-118 - 344) 18.2%	56 (-57 - 165) 30.0%	28 (-28 - 81) 39.1%	13 (-13 - 38) 46.0%
13	35, 99th percentile value	94 (-95 - 275) 18.3%	36 (-37 - 106) 33.3%	14 (-15 - 42) 46.2%	6 (-6 - 16) 50.0%	113 (-114 - 333) 21.0%	53 (-53 - 154) 33.8%	25 (-26 - 74) 45.7%	11 (-12 - 33) 56.0%
13	30, 99th percentile value	94 (-95 - 275) 18.3%	36 (-37 - 106) 33.3%	14 (-15 - 42) 46.2%	6 (-6 - 16) 50.0%	94 (-95 - 276) 34.3%	37 (-37 - 107) 53.8%	15 (-15 - 42) 67.4%	6 (-6 - 16) 76.0%
12	40, 98th percentile value	83 (-84 - 244) 27.8%	28 (-29 - 82) 48.1%	10 (-10 - 28) 61.5%	3 (-4 - 10) 75.0%	104 (-105 - 305) 27.3%	45 (-45 - 131) 43.8%	20 (-20 - 58) 56.5%	8 (-9 - 24) 68.0%
12	35, 98th percentile value	83 (-84 - 244) 27.8%	28 (-29 - 82) 48.1%	10 (-10 - 28) 61.5%	3 (-4 - 10) 75.0%	104 (-105 - 305) 27.3%	45 (-45 - 131) 43.8%	20 (-20 - 58) 56.5%	8 (-9 - 24) 68.0%
12	30, 98th percentile value	83 (-84 - 244) 27.8%	28 (-29 - 82) 48.1%	10 (-10 - 28) 61.5%	3 (-4 - 10) 75.0%	104 (-105 - 305) 27.3%	45 (-45 - 131) 43.8%	20 (-20 - 58) 56.5%	8 (-9 - 24) 68.0%
12	40, 99th percentile value	83 (-84 - 244) 27.8%	28 (-29 - 82) 48.1%	10 (-10 - 28) 61.5%	3 (-4 - 10) 75.0%	104 (-105 - 305) 27.3%	45 (-45 - 131) 43.8%	20 (-20 - 58) 56.5%	8 (-9 - 24) 68.0%
12	35, 99th percentile value	83 (-84 - 244) 27.8%	28 (-29 - 82) 48.1%	10 (-10 - 28) 61.5%	3 (-4 - 10) 75.0%	104 (-105 - 305) 27.3%	45 (-45 - 131) 43.8%	20 (-20 - 58) 56.5%	8 (-9 - 24) 68.0%
12	30, 99th percentile value	83 (-84 - 244) 27.8%	28 (-29 - 82) 48.1%	10 (-10 - 28) 61.5%	3 (-4 - 10) 75.0%	94 (-95 - 276) 34.3%	37 (-37 - 107) 53.8%	15 (-15 - 42) 67.4%	6 (-6 - 16) 76.0%

*This sensitivity analysis was performed using Ito (2003). See text for an explanation of the slope adjustment method. Only those standard combinations for which the change in design value used for the annual standard makes a difference in estimated mortality are shown.

**In the base case, incidence was quantified across the range of PM concentrations observed in the study, when possible, but not below policy relevant background level. Under alternative threshold models, incidence was quantified down to the specified threshold. Incidences are rounded to the nearest whole number. Percents are rounded to the nearest tenth.

***Current standards.

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

Table 4B-15. Sensitivity Analysis: Estimated Annual Mortality Associated with Long-Term Exposure to PM2.5 When Alternative Standards Are Just Met, in the Base Case and Using Alternative Hypothetical Threshold Models -- Rollbacks to Meet Annual Standards Using Design Values Based on Maximum vs. Average of Monitor-Specific Averages*
Detroit, MI, 2003

Alternative Standards		PM-Related Incidence Associated with PM-2.5 Using Annual Standard Design Values Based on the Maximum of Monitor-Specific Averages**			PM-Related Incidence Associated with PM-2.5 Using Annual Standard Design Values Based on the Average of Monitor-Specific Averages**		
		Percent Reduction in Incidence from Current Standards			Percent Reduction in Incidence from Current Standards		
Annual (ug/m3)	Daily (ug/m3)	BASE CASE: Lowest Measured Level in Study =7.5 µg/m3	Hypothetical Threshold =10 µg/m3	Hypothetical Threshold =12 µg/m3	BASE CASE: Lowest Measured Level in Study =7.5 µg/m3	Hypothetical Threshold =10 µg/m3	Hypothetical Threshold =12 µg/m3
15	65, 98th percentile value***	522 (181 - 910) 0.0%	282 (98 - 494) 0.0%	41 (14 - 72) 0.0%	747 (259 - 1309) 0.0%	535 (185 - 941) 0.0%	322 (111 - 568) 0.0%
15	40, 98th percentile value	522 (181 - 910) 0.0%	282 (98 - 494) 0.0%	41 (14 - 72) 0.0%	747 (259 - 1309) 0.0%	535 (185 - 941) 0.0%	322 (111 - 568) 0.0%
15	35, 98th percentile value	522 (181 - 910) 0.0%	282 (98 - 494) 0.0%	41 (14 - 72) 0.0%	602 (209 - 1051) 19.4%	372 (129 - 652) 30.5%	140 (48 - 247) 56.5%
15	65, 99th percentile value	522 (181 - 910) 0.0%	282 (98 - 494) 0.0%	41 (14 - 72) 0.0%	747 (259 - 1309) 0.0%	535 (185 - 941) 0.0%	322 (111 - 568) 0.0%
15	40, 99th percentile value	522 (181 - 910) 0.0%	282 (98 - 494) 0.0%	41 (14 - 72) 0.0%	659 (229 - 1153) 11.8%	437 (151 - 766) 18.3%	212 (73 - 374) 34.2%
14	40, 98th percentile value	438 (152 - 762) 16.1%	188 (65 - 328) 33.3%	0 (0 - 0) 100.0%	642 (223 - 1123) 14.1%	418 (144 - 733) 21.9%	191 (66 - 336) 40.7%
14	35, 98th percentile value	438 (152 - 762) 16.1%	188 (65 - 328) 33.3%	0 (0 - 0) 100.0%	602 (209 - 1051) 19.4%	372 (129 - 652) 30.5%	140 (48 - 247) 56.5%
14	40, 99th percentile value	438 (152 - 762) 16.1%	188 (65 - 328) 33.3%	0 (0 - 0) 100.0%	642 (223 - 1123) 14.1%	418 (144 - 733) 21.9%	191 (66 - 336) 40.7%
14	35, 99th percentile value	438 (152 - 762) 16.1%	188 (65 - 328) 33.3%	0 (0 - 0) 100.0%	507 (176 - 884) 32.1%	266 (92 - 465) 50.3%	23 (8 - 40) 92.9%

Alternative Standards		PM-Related Incidence Associated with PM-2.5 Using Annual Standard Design Values Based on the Maximum of Monitor-Specific Averages**			PM-Related Incidence Associated with PM-2.5 Using Annual Standard Design Values Based on the Average of Monitor-Specific Averages**		
		Percent Reduction in Incidence from Current Standards			Percent Reduction in Incidence from Current Standards		
Annual (ug/m3)	Daily (ug/m3)	BASE CASE: Lowest Measured Level in Study =7.5 µg/m3	Hypothetical Threshold =10 µg/m3	Hypothetical Threshold =12 µg/m3	BASE CASE: Lowest Measured Level in Study =7.5 µg/m3	Hypothetical Threshold =10 µg/m3	Hypothetical Threshold =12 µg/m3
13	40, 98th percentile value	354 (123 - 615) 32.2%	94 (33 - 164) 66.7%	0 (0 - 0) 100.0%	538 (187 - 939) 28.0%	301 (104 - 526) 43.7%	61 (21 - 107) 81.1%
13	35, 98th percentile value	354 (123 - 615) 32.2%	94 (33 - 164) 66.7%	0 (0 - 0) 100.0%	538 (187 - 939) 28.0%	301 (104 - 526) 43.7%	61 (21 - 107) 81.1%
13	30, 98th percentile value	354 (123 - 615) 32.2%	94 (33 - 164) 66.7%	0 (0 - 0) 100.0%	435 (151 - 757) 41.8%	185 (64 - 323) 65.4%	0 (0 - 0) 100.0%
13	40, 99th percentile value	354 (123 - 615) 32.2%	94 (33 - 164) 66.7%	0 (0 - 0) 100.0%	538 (187 - 939) 28.0%	301 (104 - 526) 43.7%	61 (21 - 107) 81.1%
13	35, 99th percentile value	354 (123 - 615) 32.2%	94 (33 - 164) 66.7%	0 (0 - 0) 100.0%	507 (176 - 884) 32.1%	266 (92 - 465) 50.3%	23 (8 - 40) 92.9%
13	30, 99th percentile value	354 (123 - 615) 32.2%	94 (33 - 164) 66.7%	0 (0 - 0) 100.0%	356 (124 - 619) 52.3%	97 (34 - 168) 81.9%	0 (0 - 0) 100.0%
12	40, 98th percentile value	271 (94 - 469) 48.1%	0 (0 - 1) 100.0%	0 (0 - 0) 100.0%	435 (151 - 756) 41.8%	184 (64 - 322) 65.6%	0 (0 - 0) 100.0%
12	35, 98th percentile value	271 (94 - 469) 48.1%	0 (0 - 1) 100.0%	0 (0 - 0) 100.0%	435 (151 - 756) 41.8%	184 (64 - 322) 65.6%	0 (0 - 0) 100.0%
12	30, 98th percentile value	271 (94 - 469) 48.1%	0 (0 - 1) 100.0%	0 (0 - 0) 100.0%	435 (151 - 756) 41.8%	184 (64 - 322) 65.6%	0 (0 - 0) 100.0%
12	40, 99th percentile value	271 (94 - 469) 48.1%	0 (0 - 1) 100.0%	0 (0 - 0) 100.0%	435 (151 - 756) 41.8%	184 (64 - 322) 65.6%	0 (0 - 0) 100.0%
12	35, 99th percentile value	271 (94 - 469) 48.1%	0 (0 - 1) 100.0%	0 (0 - 0) 100.0%	435 (151 - 756) 41.8%	184 (64 - 322) 65.6%	0 (0 - 0) 100.0%
12	30, 99th percentile value	271 (94 - 469) 48.1%	0 (0 - 1) 100.0%	0 (0 - 0) 100.0%	356 (124 - 619) 52.3%	97 (34 - 168) 81.9%	0 (0 - 0) 100.0%

*This sensitivity analysis was performed using Pope et al. (2002) -- ACS extended. See text for an explanation of the slope adjustment method. Only those standard combinations for which the change in design value used for the annual standard makes a difference in estimated mortality are shown.

**In the base case, incidence was quantified across the range of PM concentrations observed in the study, when possible, but not below policy relevant background level. Under alternative threshold models, incidence was quantified down to the specified threshold. Incidences are rounded to the nearest whole number. Percents are rounded to the nearest tenth.

***Current standards.

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

United States
Environmental Protection
Agency

Office of Air Quality Planning and Standards
Air Quality Strategies and Standards Division
Research Triangle Park, NC

Publication No. EPA-452/D-05-001
January 2005