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

Policy Assessment of Scientific and Technical Information

OAQPS Staff Paper

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

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PREFACE TO DECEMBER 2005 EDITION

The purpose of this December 2005 edition is to include the September 15, 2005 letter from the Clean Air Scientific Advisory Committee (CASAC) to the Administrator providing its final comments and advice on the June 2005 edition of the OAQPS Staff Paper. The CASAC letter has been included as an Attachment to this edition (Attachment B).

In addition, this edition of the Staff Paper includes technical corrections which are listed in the errata.

ACKNOWLEDGMENTS

This Staff Paper is the product of the Office of Air Quality Planning and Standards (OAQPS). For the section on PM-related health effects and primary standards, the principal authors include Dr. Mary Ross, who also led the PM NAAQS review team, Mr. Harvey Richmond, and Dr. Karen Martin, who also managed the project. For the section on PM-related welfare effects and secondary standards, the principal authors include Mr. Rich Damberg, Ms. Amy Vasu, Mr. Mark Schmidt, Ms. Vicki Sandiford, and Dr. Karen Martin. The principal authors of the chapter on the characterization of ambient PM include Mr. Mark Schmidt, Mr. John Langstaff, and Mr. Scott Mathias. The authors would also like to acknowledge Mr. Venkatesh Rao, Mr. David Mintz, Mr. Lance McCluney, Mr. Neil Frank, Mr. Tim Hanley, and Mr. Lewis Weinstock for providing input and analyses on specific topics. Staff from other EPA offices, including the Office of Research and Development, the Office of General Counsel, and the Office of Transportation and Air Quality, also provided valuable comments. Special acknowledgment is given to Mr. John Bachmann for his leadership in past PM NAAQS reviews and for his expert advice and valuable input in this review.

Earlier drafts of this document were formally reviewed by the Clean Air Scientific Advisory Committee (CASAC) and made available for public comment. This document has been informed by the expert advice and comments received from CASAC, as well as by public comments submitted by a number of independent scientists, officials from State and local air pollution organizations, environmental groups, and industrial groups and companies.

Table of Contents

PM-RELATED HEALTH EFFECTS AND PRIMARY PM NAAQS

PM-RELATED WELFARE EFFECTS AND SECONDARY PM NAAQS

CHAPTER APPENDICES:

ATTACHMENT A: Clean Air Scientific Advisory Committee Letter, June 6, 2005

ATTACHMENT B: Clean Air Scientific Advisory Committee Letter, September 15, 2005

List of Tables

List of Figures

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
CBSA	Core Based Statistical Area
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
CSA	Combined Statistical Area
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
$UPM_{10-2.5}$	Thoracic coarse urban particulate matter
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

ERRATA

Corrections made to figures and appendices:
The legends for Figures 5-2(a) through 5-2(d) have been revised, deleting the phrase "based on ACS extended study," and correcting the numbers on one of the axes on Figure 5-2(a)

Appendix 5A, figures have been replaced with the intended figures depicting risk estimates for fine particle alternative daily standards with the 99th percentile form.

Appendix 4B, Tables 4B-2 and 4B-6 have been replaced with the intended tables. One set of alternative standards was inadvertently left out in the June 2005 Staff Paper.

Minor corrections made in the text of the staff paper:
Page 3-23, in the last line of last full paragraph, "to" has been deleted.

Page 4-78, in the reference for Abt Associates Inc. (2005b), "Draft Report" has been revised to "Final Report"

Page 5-19, "section 3.5.5.1" has been revised to "section 3.6.5.3"

Page 5-19, "section 3.5.5" has been revised to "section 3.6.5.1"

Page 5-20, "section 3.5.5.3" has been revised to "section 3.6.5.2"

Page 5-21, first line of first full paragraph, "section 4.3.6.6" has been revised to "section 3.4.5"

Page 5-32, "Chapter 2, Figure 2-23" has been revised to "Chapter 2, Figure 2-25"

Page 5-33, "section 4.2.2" has been revised to "section 4.3.1"

Page 5-41, "section 4.2.2" has been revised to "section 4.3.1"

Page 5-42, "section 3.4" has been revised to "section 3.5"

Page 5-47, "section 2.5.3" has been revised to "section 2.3.5"

Page 5-48, "section 3.4.2" has been revised to "section 3.2"

Page 6-32, first line of bottom paragraph, "Impacts on threatened and endangered species" is now in bold text.

Page 6-37, in the top paragraph, "spruce (Picea glacus)" is revised to "spruce (Picea glauca), and "Pinus strobus" is now italicized.

Back page, publication number has been changed to "EPA-452/R-05-005a"

1. INTRODUCTION

1.1 PURPOSE

This 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 Staff Paper also presents and interprets results from 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). Finally, this Staff Paper presents 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 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 Staff Paper should be of use to all parties interested in the PM NAAQS review, it is written for those decision makers, scientists, and staff who have some familiarity with the technical discussions contained in the CD.

1.2 BACKGROUND

1.2.1 Legislative Requirements

Two sections of the Clean Air Act (Act) govern the establishment and revision of the NAAQS. Section 108 (42 U.S.C. 7408) directs the Administrator to identify and list "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 to issue air quality criteria for those that are listed. Air quality criteria are intended to "accurately reflect the latest scientific knowledge useful in indicating the kind

[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.

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 listed 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 the standards. See generally *Whitman v. American Trucking Associations*, 531 U.S. 457, 465-472, 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, 449 U.S. 1042 (1980); *American Petroleum Institute v. Costle*, 665 F.2d 1176, 1186 (D.C. Cir. 1981), cert. denied, 455 U.S. 1034 (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

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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."

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) 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 24634, 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

[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.

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 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, 498 U.S. 1082 (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 removed the vacated 1997 PM_{10} standards from the Code of Federal Regulations (CFR) (69 FR 45592, July 30, 2004) and deleted the regulatory provision [at 40 CFR section 50.6(d)] that controlled 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 a more narrowly defined indicator.

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

1.2.4 Current PM NAAQS Review

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

Shortly after EPA released the third draft CD, the Health Effects Institute (HEI)[5] announced that researchers at Johns Hopkins University had discovered problems with applications of statistical software used in a number of important epidemiological studies that had been discussed in that draft CD. In response to this significant issue, EPA took steps in consultation with CASAC to encourage researchers to reanalyze affected studies and to submit them expeditiously for peer review by a special expert panel convened at EPA's request by HEI. EPA subsequently incorporated the results of this reanalysis and peer-review process into a fourth draft CD, which was released in June 2003 and reviewed by CASAC and the public at a meeting held in August 2003.

The first draft Staff Paper, based on the fourth draft CD, was released at the end of August 2003, and was reviewed by CASAC and the public at a meeting held in November 2003. During that meeting, EPA also consulted with CASAC on a new framework for the final chapter (integrative synthesis) of the CD and on ongoing revisions to other CD chapters to address previous CASAC comments. EPA held additional consultations with CASAC at public meetings held in February, July, and September 2004, leading to publication of the final CD in October 2004. The second draft Staff Paper, based on the final CD, was released at the end of January 2005, and was reviewed by CASAC and the public at a meeting held in April 2005. The CASAC's advice and recommendations to the Administrator, based on their review of the second draft Staff Paper, were further discussed during a public teleconference held in May 2005 and are provided in a letter to the Administrator (Henderson, 2005), which is reproduced in Attachment 2. This final Staff Paper takes into account the advice and recommendations from CASAC and public comments received on the earlier drafts of this document. Any subsequent

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

advice and recommendations received from CASAC related to this final Staff Paper will also be considered by the Administrator.

The schedule for completion of this review is now governed by a consent decree resolving a lawsuit filed in March 2003 by a group of plaintiffs representing national environmental organizations. The lawsuit alleged that EPA had failed to perform its mandatory duty, under section 109(d)(1), of completing the current review within the period provided by statute. *American Lung Association v. Whitman* (No. 1:03CV00778, D.D.C. 2003). An initial consent decree was entered by the court in July 2003 after an opportunity for public comment. The consent decree, as modified by the court, provides that EPA will sign for publication notices of proposed and final rulemaking concerning its review of the PM NAAQS no later than December 20, 2005 and September 27, 2006, respectively.

1.3 GENERAL APPROACH AND ORGANIZATION OF DOCUMENT

This policy assessment is based on staff evaluation of the policy implications of the scientific evidence contained in the CD and the results of quantitative analyses based on that evidence, which taken together help inform staff conclusions and recommendations on the 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 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 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 staff recommendations on ranges of alternative primary standards for consideration by the Administrator.

Chapters 6 and 7 comprise the third main part of this 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 protection of visual air quality. 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 staff recommendations on ranges of alternative secondary standards for consideration by the Administrator.

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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.

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 particle 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 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.

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[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).

[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 per logarithmic increment in particle diameter 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.

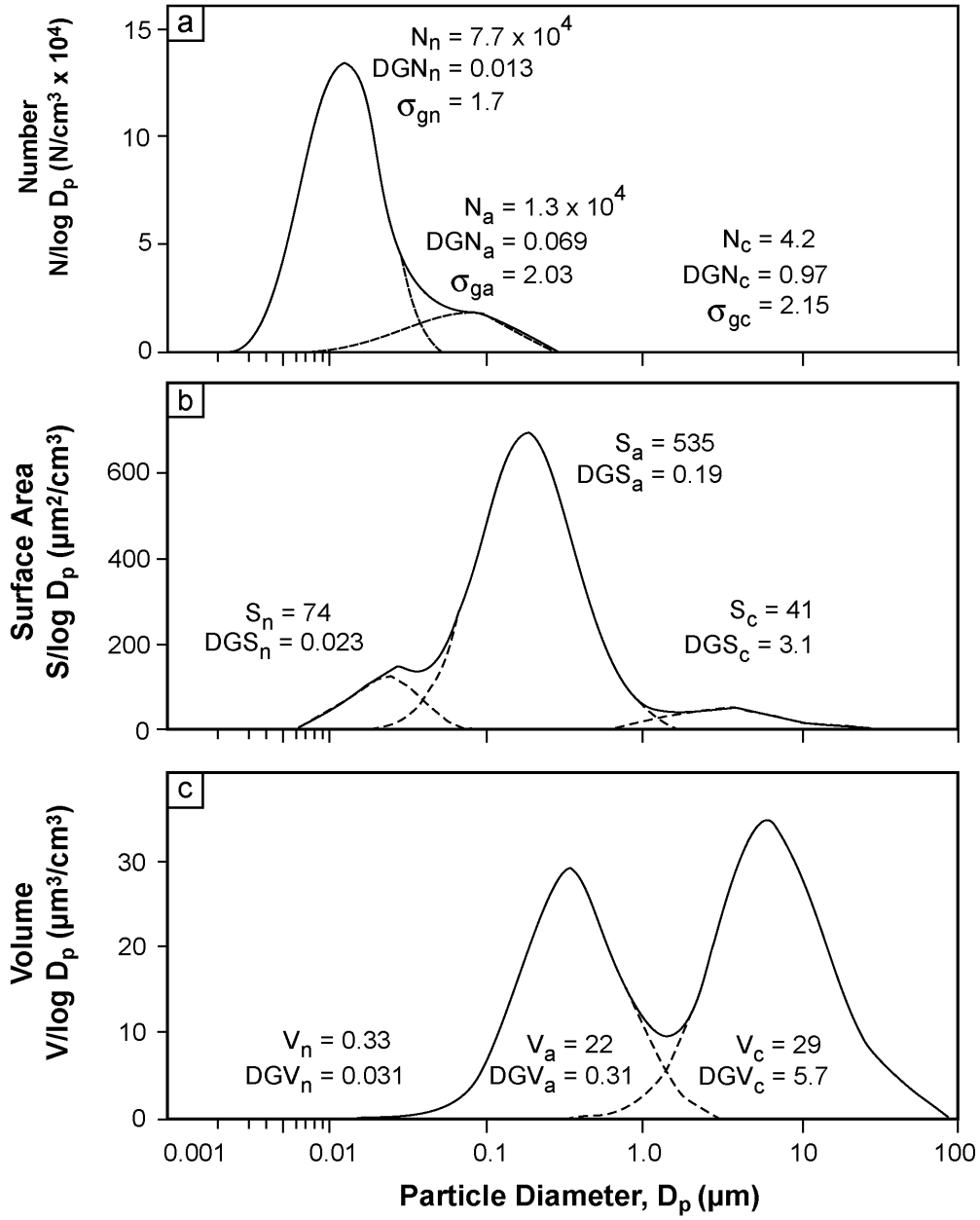

Figure 2-1. Distribution of coarse (c), accumulation (a), and nuclei (n) mode particles by three characteristics: (a) number, N; (b) surface area, S; and (c) volume, V for the grand average continental size distribution. DGV = geometric mean diameter by volume; DGS = geometric mean diameter by surface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

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

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," "Aitken mode," and "accumulation mode." Together, nucleation-mode and Aitken-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 Aitken-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 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.

Since 1987, the EPA has 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 the upper size cut varies substantially with placement and wind velocity . When EPA established new PM standards in 1987, the selection of PM_{10} as an indicator was

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

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

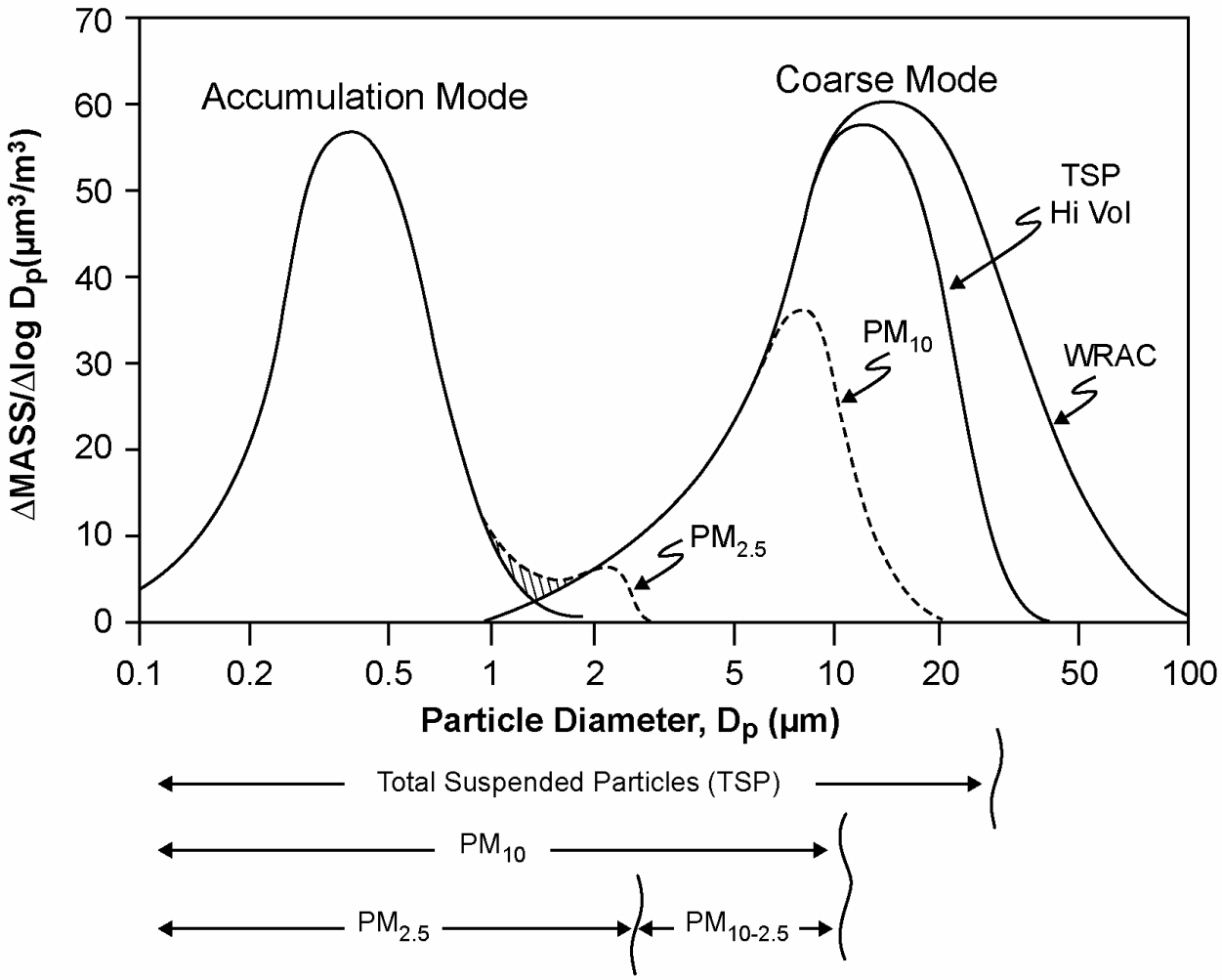

Figure 2-2. An idealized distribution of ambient PM showing fine and coarse particles and the fractions collected by size-selective samplers. (WRAC is the Wide Range Aerosol Classifier which collects the entire coarse mode).

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

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.

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 industrial process emissions, fugitive emissions from storage piles, traffic related emissions including tire and paving materials and grinding and resuspension of crustal, biological, industrial, and combustion materials that have settled on or near roadways, construction and demolition activities, agriculture, mining and mineral processing, sea spray, and wind-blown dust and biological materials. 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 transformation processes of precursor gases. 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.

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[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.

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, Aitken, 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 Aitken and nucleation modes.
Aitken-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 referrred 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.

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) uptake of water by hygroscopic components; 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 efficiency of coagulation for 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 gaseous 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 gaseous 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 or organic 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

[8] Also called light absorbing carbon and black carbon. The terms elemental carbon and black carbon are often used interchangeably, but may be defined differently by different users. Black carbon is most often used in discussions of optical properties and elemental carbon is most often used when referring to chemical composition. In many cases, there is little difference between the two, but care must be taken when comparing data from studies with different purposes. In addition, the term soot is also used in many instances to refer to either EC or BC. The differences between soot and either EC or BC can be significant, as soot refers to elemental carbon formed from gas phase hydrocarbons in the combustion process, and tends to be in the submicron fraction and often in the fraction of particles that are smaller than 0.10 microns in aerodynamic diameter. EC and BC both include carbonaceous particles formed from incomplete burnout of solid carbonaceous fuels; these particles have distinctly different physical characteristics compared to char. As an additive to automotive tires, commercially produced 'carbon black' and associated contaminants can also be found in resuspended urban road dust.

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.

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 (EC), 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 are more likely to reach 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 (i.e. accumulate) in the atmosphere because they are too large to diffuse rapidly to surfaces or to other particles and too small to settle out or impact on stationary objects. 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 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. For example, dust storms in desert areas of Africa and Asia lift coarse particles to high elevations and these 'dust clouds' are readily observed to undergo intercontinental transport to North America (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, particularly 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, elemental carbon plays the most 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.

Table 2-2. Comparison of Ambient 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.

2.2.6 Other Radiative Properties of Particles

In addition to the optical properties related to visibility summarized above, ambient particles scatter and absorb radiation across the full electromagnetic spectrum, including ultraviolet, visible, and 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 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 relative proportions of scattering and absorption by ambient particles are 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 refractive index, mineral aerosol can reflect or absorb radiation. Dark minerals absorb across the solar and infrared spectral regions 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 a complex mixture of substances with differing physical and chemical properties , 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 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

[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).

are subjected to specific equilibrium conditions (i.e., 20 - 23° C and 30 - 40 percent RH in most cases).

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 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

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[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.

(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.

The number of continuous $PM_{2.5}$ monitors across the U.S. has increased from 300 to over 500 between 2003 and 2005. Although a subset of these monitors were required by regulation to be placed in metropolitan areas of greater than 1 million population, a higher percentage were installed to provide improved temporal resolution for daily air quality index reporting and $PM_{2.5}$ forecast verifications through EPA's AIRNOW program. Some of the continuous $PM_{2.5}$ data reported through the AIRNOW program are adjusted to better match FRM results.[11] The continuous data used in the analyses in this chapter were obtained from EPA's Air Quality System (AQS); some of these AQS data are adjusted and some are not. There is currently an effort underway to better characterize this facet of the continuous data in AQS. Still, the AQS continuous data utilized in analyses here do show excellent correlation with collocated FRM measurements; over 95 percent of the continuous/FRM site pairs had a correlation coefficient of over 0.72, and almost 75 percent had a correlation of 0.9 or higher (Schmidt et al., 2005).

Work also continues on the development of national approval criteria for determining regional and national equivalency for continuous $PM_{2.5}$ monitors. Once promulgated, these criteria would provide the regulatory basis for approving appropriate continuous methods as equivalent to FRMs, and permit the assessment of NAAQS attainment status with continuous $PM_{2.5}$ data, reducing the number of manually-operated FRM monitors that need to continue operating.

2.3.2 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

[11] When data are sent to the AIRNOW website, they are assumed to be "FRM like" which means that their values are highly correlated ($R^2 > 0.8$) with actual FRM concentrations so that values can be compared not only to the FRM measurements but also across State boundaries. Statistical adjustments to the raw continuous data are necessary because some of the sampling methodologies, such as the TEOM monitors, have inlets heated from 30ºC to 50ºC which causes semi-volatile fine particulate matter including nitrates to be vaporized and never measured. The result of this vaporization is a lower measured TEOM concentration when compared to the FRM. Adjustments have been accomplished on a seasonal basis as well as using meteorologic variables (e.g., ambient temperature) with linear and non-linear regression techniques. The need to adjust the continuous data can depend on several factors including the type of method, the location of the site in the country and the composition of the ambient particulate matter being measured.

on a filter.[12] 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 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.[13] 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)

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

[13] 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 $PM_{2.5}$ Speciation Trends Network and other special monitoring programs are summarized in Solomon et al. (2001).

are used to measure ions such as nitrate (NO_3^-), sulfate ($SO_4^=$), chloride (Cl^-), ammonium (NH_4^+), 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 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 potential 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 that epidemiologic researchers find between ambient particles and health effects.

The semi-volatile components of PM can create both positive and negative measurement artifacts. Negative artifacts arise from evaporation of the semi-volatile 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 use diffusion dryers rather than heating to reduce the relative humidity (CD, p. 2-100, Table 2-7).

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 publicly 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..

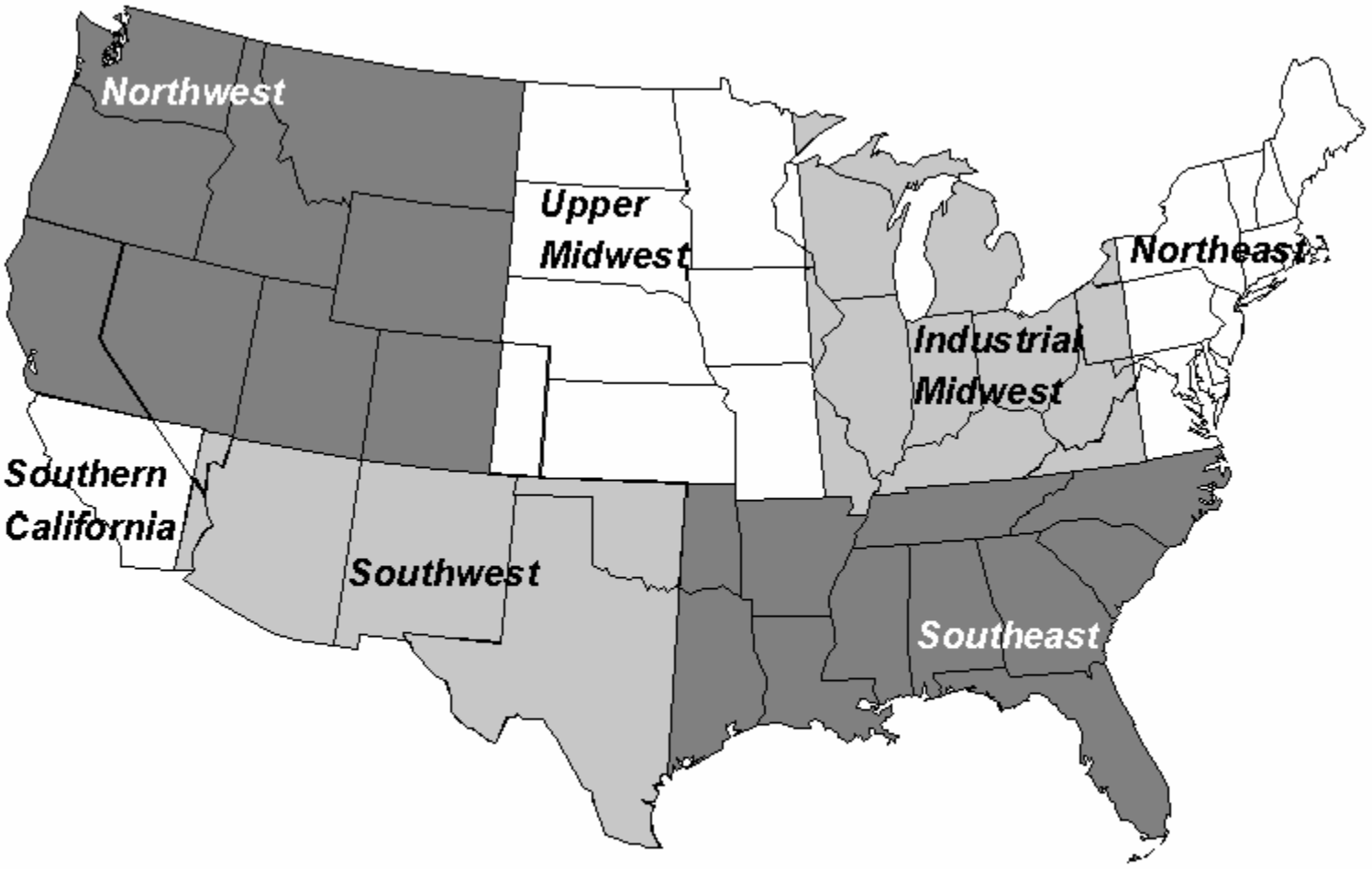

Figure 2-3. Regions used in PM Staff Paper in data analysis summaries.

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.[14] 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.[15]

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) 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.[16] 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,

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

[15] 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.

[16] No conclusions should be drawn from these data summaries regarding the potential attainment status of any area. 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.

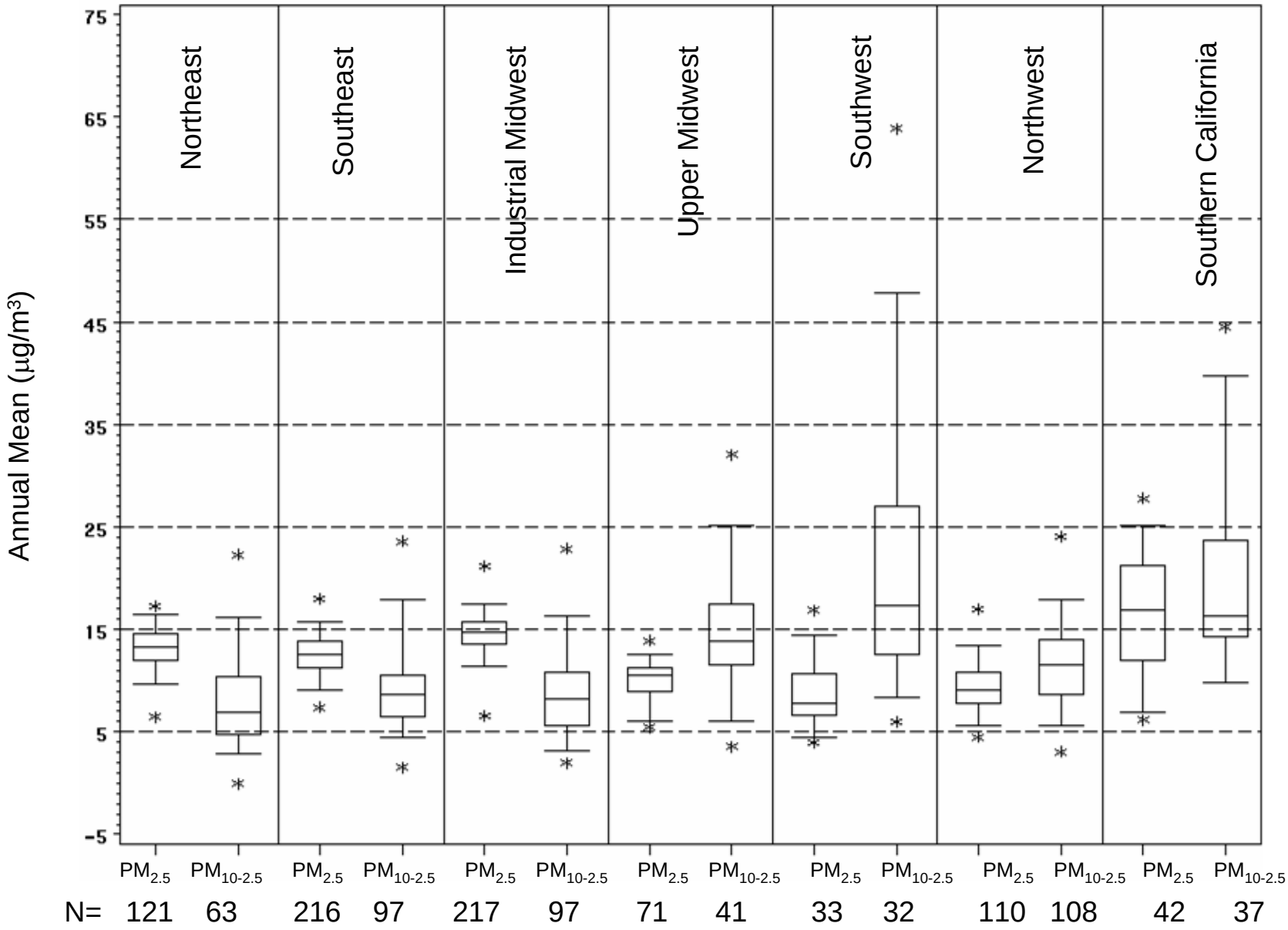

Figure 2-4. Distribution of annual mean $PM_{2.5}$ and estimated annual mean $PM_{10-2.5}$ concentrations by region, 2001-2003. Box depicts interquartile range and median; whiskers depict 5th and 95th percentiles; asterisks depict minima and maxima. N = number of sites.

Source: Schmidt et al. (2005)

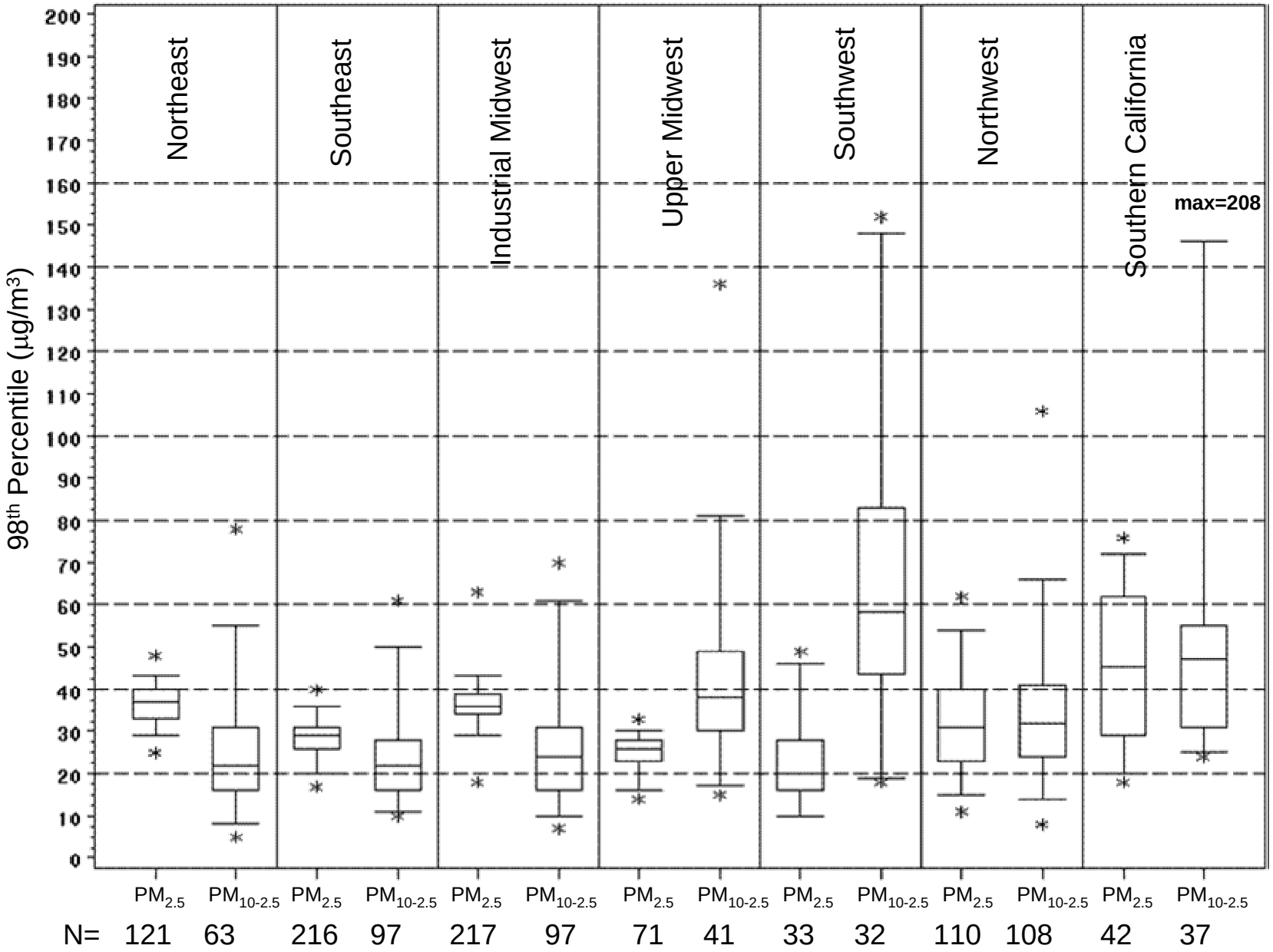

Figure 2-5. Distribution of 98th percentile 24-hour average $PM_{2.5}$ and estimated $PM_{10-2.5}$ concentrations by region, 2001-2003. Box depicts interquartile range and median; whiskers depict 5th and 95th percentiles; asterisks depict minima and maxima. N = number of sites.

Source: Schmidt et al. (2005)

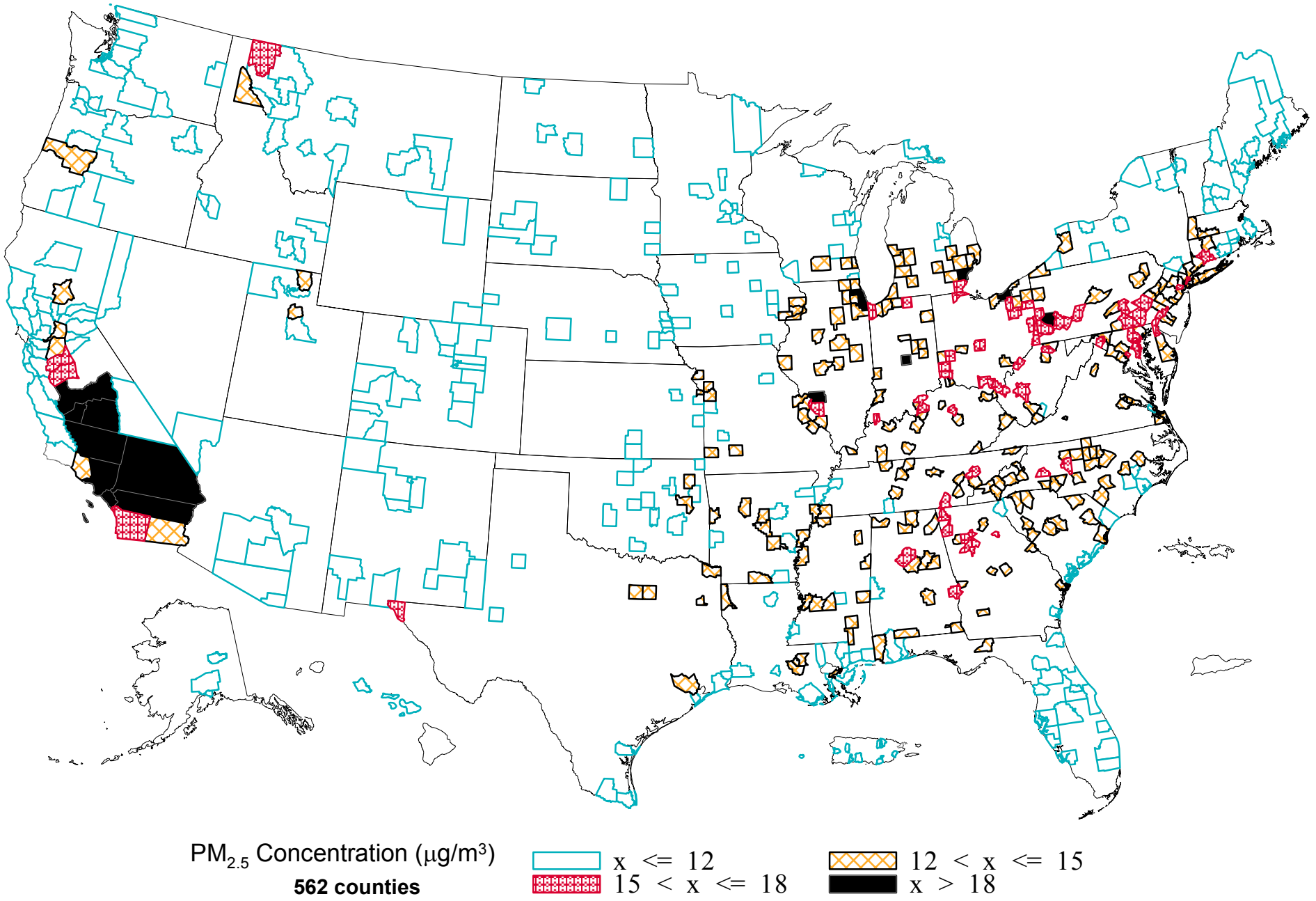

Figure 2-6. County-level maximum annual mean $PM_{2.5}$ concentrations, 2001-2003.

Source: Schmidt et al. (2005)

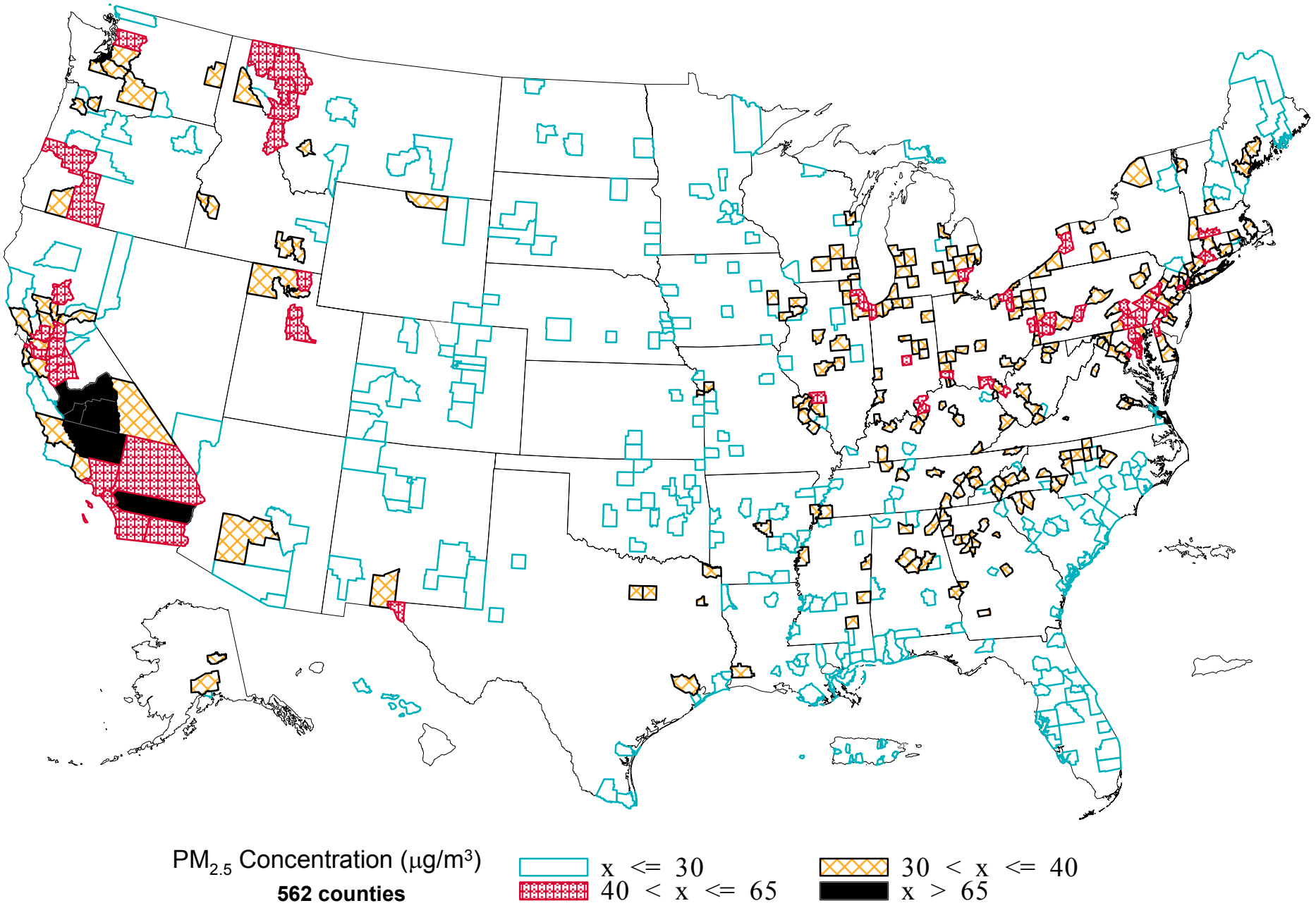

Figure 2-7. County-level maximum 98th percentile 24-hour average $PM_{2.5}$ concentrations, 2001-2003.

Source: Schmidt et al. (2005)

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 lower 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, construction/demolition activities, 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). Analyses of 2001-2003 $PM_{2.5}$ data found that over 9 percent of the days above (site-based) 98th percentile 24-hour 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, 2004b, 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 were somewhat flat or rose slightly (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, 2004b, p. 15).

The IMPROVE monitoring network, which consists of sites located primarily in national parks and wilderness areas throughout the U.S., generally provides data for long-term $PM_{2.5}$

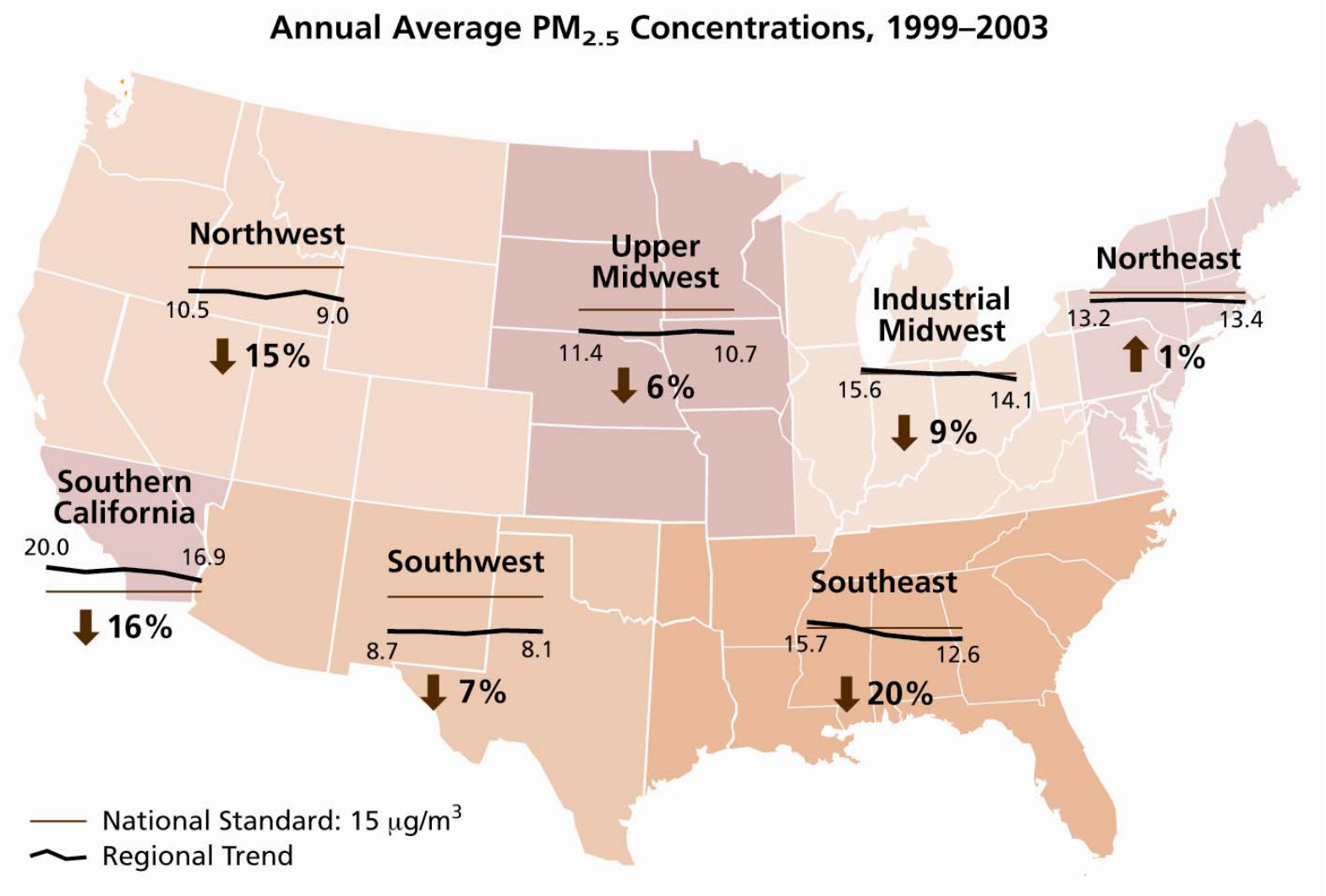

Figure 2-8. Regional trends in annual average PM2.5 concentrations in the EPA network, 1999-2003.

Source: EPA (2004b)

trends in rural areas.[17] 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 westernmost U.S. subregions (northwest, southern California, upper midwest, and southwest) are considered 'western sites' and the 3 eastern subregions (northeast, southeast, and industrial midwest) are considered 'eastern sites.' At the eastern rural sites, measured $PM_{2.5}$ mass decreased about 23 percent from 1993 to 2003. At the western rural 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 correlations, (2) differences in long-term (e.g., annual and multi-year) 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). A similar analysis, for 49 urban areas defined as either Core Based Statistical Areas (CBSAs) or Combined Statistical Areas (CSAs), was conducted on $PM_{2.5}$ FRM monitoring data from 2001-2003 (Schmidt et al., 2005).[18]

An analysis of site pairs from each of the 49 urban areas indicates that multiple sites in these areas were highly correlated throughout the period. About 83 percent (1901 out of 2290) of the between-site correlation coefficients in all 49 areas were greater than or equal to 0.80, and more than 48 percent (1113 out of 2290) 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 0.82.

A summary of the analyses of long-term and short-term concentration differences for the 49 urban areas is shown in Table 2-3. The difference in 3-year average annual mean $PM_{2.5}$ concentrations between monitor pairs in the 49 cities ranged from less than 1 $\mu g/m^3$ in four areas to about 18 $\mu g/m^3$ in Los Angeles. Large differences in 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). Most sites in the 49 areas had annual means within 15 percent of the area spatial average; the largest percent

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

[18] Metropolitan areas for use in federal statistical activities, as defined by the Office of Management and Budget, include core-based statistical areas (CBSA) that are comprised of "metropolitan" and "micropolitan" areas, and combined statistical areas (CSA) that are comprised of two or more core-based statistical areas. Counties are the geographic building blocks for defining CBSA's. The analysis described here, and other analyses throughout this and subsequent chapters, utilize the latest area definitions which are available at: http://www.whitehouse.gov/omb/bulletins/fy05/b05-02.html.

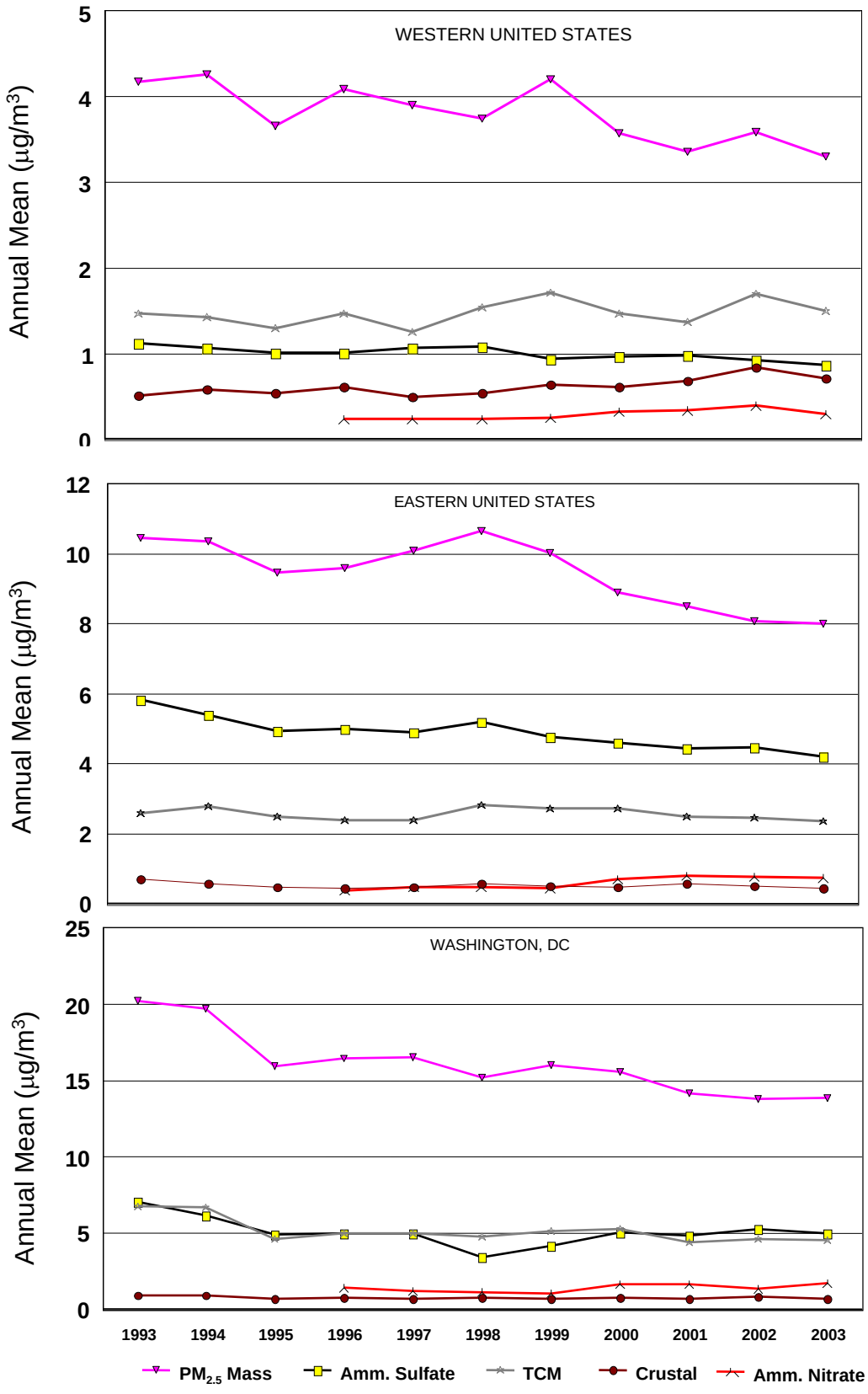

Figure 2-9. Average annual mean trend in $PM_{2.5}$ mass, ammonium sulfate, ammonium nitrate, total carbonaceous mass, and crustal material at IMPROVE sites, 1993-2003.

Source: Schmidt et al. (2005)

Table 2-3. Summary of $PM_{2.5}$ FRM Data Analyses in 49 Metropolitan Areas, 2001-2003

Area *	N Sites	3-year Average Annual Mean						24-Hour P_{90} ($\mu g/m^3$) **		
		Levels ($\mu g/m^3$)			Percent Difference		r (Max site versus Min site)	Max Pair	Min Pair	r (Max Pair)
		Area Avg	Max Site	Min Site	Largest diff., any site versus Area Avg	Max site versus Min site				
Albuquerque, NM	4	7.0	10.2	5.0	31%	51%	0.42	10.9	2.6	0.42
Atlanta-Sandy Springs-Gainesville, GA	8	15.9	18.0	14.1	12%	22%	0.71	9.4	3.5	0.71
Bakersfield, CA	5	15.3	21.8	6.7	56%	69%	0.00	44.8	6.0	0.16
Baton Rouge-Pierre Part, LA	5	12.3	13.1	10.8	12%	18%	0.85	7.7	2.4	0.62
Birmingham-Hoover-Cullman, AL	8	14.8	18.0	12.6	18%	30%	0.78	12.7	3.5	0.78
Charlotte-Gastonia-Salisbury, NC-SC	5	14.3	14.9	14.0	4%	6%	0.94	4.1	1.7	0.92
Chicago-Naperville-Michigan City, IL-IN-WI	28	14.7	17.7	11.7	20%	34%	0.77	13.6	2.2	0.73
Cincinnati-Middletown-Wilmington, OH-KY-IN	12	16.0	17.8	14.5	10%	19%	0.95	7.0	2.4	0.95
Cleveland-Akron-Elyria, OH	13	15.5	18.3	13.4	15%	27%	0.87	11.4	3.2	0.87
Dallas-Fort Worth, TX	7	12.8	13.9	11.7	9%	16%	0.92	5.2	2.3	0.92
Denver-Aurora-Boulder, CO	6	8.7	10.8	4.5	48%	58%	0.40	11.4	4.0	0.42
Detroit-Warren-Flint, MI	14	15.2	19.5	12.6	22%	35%	0.85	14.1	3.2	0.85
Eugene-Springfield, OR	4	9.4	13.4	6.6	30%	51%	0.57	19.3	4.8	0.57
Grand Rapids-Muskegon-Holland, MI	4	13.0	13.8	12.3	6%	11%	0.91	5.8	3.2	0.90
Greensboro--Winston-Salem--High Point, NC	4	14.6	15.8	14.0	8%	11%	0.94	5.5	2.5	0.93
Houston-Baytown-Huntsville, TX	6	11.7	14.2	9.6	18%	32%	0.78	8.9	6.2	0.64
Indianapolis-Anderson-Columbus, IN	6	15.3	16.7	13.6	11%	19%	0.93	6.8	2.0	0.93
Kansas City-Overland Park-Kansas City, MO-KS	10	12.0	13.9	10.8	14%	22%	0.76	9.1	1.4	0.76
Knoxville-Sevierville-La Follette, TN	5	15.3	16.7	14.2	8%	15%	0.86	6.2	2.7	0.86
Las Vegas-Paradise-Pahrump, NV	5	7.1	11.0	4.0	44%	64%	0.03	17.6	2.5	-0.03
Lexington-Fayette--Frankfort--Richmond, KY	4	14.4	15.7	13.5	8%	14%	0.86	5.9	3.3	0.86
Little Rock-North Little Rock-Pine Bluff, AR	5	13.0	14.1	11.9	8%	16%	0.79	7.6	5.1	0.78
Los Angeles-Long Beach-Riverside, CA	22	19.0	27.8	9.9	48%	64%	0.50	39.6	5.3	0.50
Louisville-Elizabethtown-Scottsburg, KY-IN	6	15.6	16.9	14.1	10%	17%	0.85	8.2	3.9	0.85
Memphis, TN-MS-AR	6	13.1	14.0	11.7	11%	16%	0.86	6.3	2.2	0.82
Miami-Fort Lauderdale-Miami Beach, FL	6	8.2	9.5	7.4	14%	22%	0.73	5.5	1.7	0.73
Milwaukee-Racine-Waukesha, WI	6	13.1	13.2	12.5	5%	5%	0.96	4.1	2.2	0.93
Minneapolis-St. Paul-St. Cloud, MN-WI	12	10.5	12.0	9.7	13%	19%	0.79	8.0	2.6	0.79
New Orleans-Metairie-Bogalusa, LA	4	11.5	12.2	10.4	10%	15%	0.91	4.0	2.8	0.90
New York-Newark-Bridgeport, NY-NJ-CT-PA	29	13.5	16.4	11.2	18%	32%	0.85	12.5	2.0	0.84
Omaha-Council Bluffs-Fremont, NE-IA	7	10.4	10.7	9.8	6%	8%	0.86	5.2	2.1	0.78
Philadelphia-Camden-Vineland, PA-NJ-DE-MD	14	14.9	16.4	13.8	9%	16%	0.94	7.6	3.1	0.94
Phoenix-Mesa-Scottsdale, AZ	5	9.3	11.4	6.3	32%	45%	0.22	14.0	4.2	0.22
Pittsburgh-New Castle, PA	13	15.8	21.2	13.2	25%	38%	0.75	21.8	3.2	0.69
Portland-Vancouver-Beaverton, OR-WA	6	8.2	9.5	6.1	26%	36%	0.84	9.5	3.0	0.76
Provo-Orem, UT	4	9.8	10.9	8.8	10%	19%	0.88	6.5	3.0	0.92
Raleigh-Durham-Cary, NC	5	13.3	13.9	12.2	8%	12%	0.93	5.7	2.4	0.88
Richmond, VA	5	13.4	14.0	12.8	4%	9%	0.88	5.8	3.2	0.88
Sacramento--Arden-Arcade--Truckee, CA-NV	5	9.9	12.5	7.6	23%	39%	0.37	16.0	6.0	0.21
Salt Lake City-Ogden-Clearfield, UT	7	11.4	14.0	9.0	21%	36%	0.92	11.0	3.8	0.92
San Diego-Carlsbad-San Marcos, CA	5	15.0	15.9	12.8	15%	19%	0.89	10.6	4.6	0.69
San Jose-San Francisco-Oakland, CA	9	10.8	11.8	8.4	22%	29%	0.67	13.5	4.7	0.67
San Juan-Caguas-Fajardo, PR	5	7.2	9.3	5.1	29%	45%	0.71	6.8	1.7	0.71
Seattle-Tacoma-Olympia, WA	10	9.4	11.1	5.3	44%	52%	0.30	19.1	2.9	0.30
St. Louis-St. Charles-Farmington, MO-IL	12	15.0	17.5	14.0	14%	20%	0.82	10.3	2.2	0.76
Virginia Beach-Norfolk-Newport News, VA-NC	5	12.5	13.0	11.9	5%	8%	0.93	4.6	2.7	0.90
Washington-Baltimore-Northern Virginia, DC-MD-VA-WV	20	14.5	16.7	12.2	16%	27%	0.82	9.7	2.6	0.82
Weirton-Steubenville, WV-OH	4	17.1	17.8	16.2	5%	9%	0.87	8.3	6.1	0.86
Wichita-Winfield, KS	4	10.9	11.1	10.2	6%	8%	0.96	2.9	1.3	0.91

* 'Area' is the larger of a Combined Statistical Area (CSA) or a Core Based Statistical Area (CBSA). See http://www.whitehouse.gov/omb/bulletins/fy05/b05-02.html.

** 'P_{90}' is the 90th percentile of the distribution of differences in 24-hour averages between two sites in the same urban area.

difference between any site in an area and the area's spatial average ranged from 4 to 56 percent with a median of 14 percent. In most urban areas (39 of the 49), the site pair with the maximum and minimum annual mean concentration was highly correlated ($r_{(max,min)} \geq 0.70$); there are, however, some notable exceptions (i.e., 8 areas had $r_{(max,min)} \leq 0.50$).

The spatial analysis 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 2290 monitor pairs in the 49 urban areas ranged from about 1 to 45 $\mu g/m^3$. 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.

2.4.2 PM_{10}

For the purpose of comparison to $PM_{2.5}$ and $PM_{10-2.5}$ concentrations, PM_{10} data from 2001-2003 are presented in Figures 2-10 and 2-11. Figure 2-10 shows the PM_{10} annual mean concentrations and Figure 2-11 shows the concentration-based 24-hour average 'design value' type metric.[19,20] As in the earlier $PM_{2.5}$ maps, the monitor with the highest value in each monitored county is used to represent the value in each county. Most areas of the country had concentrations below the level of the annual PM_{10} standard of 50 $\mu g/m^3$. Exceptions include six counties in central and southern California. Most areas of the country also had concentrations below the level of the 24-hour standard of 150 $\mu g/m^3$, with exceptions concentrated in the southwestern U.S. and isolated counties scattered across the east.

EPA recently examined national and regional PM_{10} trends from 1988 to 2003 (EPA, 2004b, p. 13). The EPA found a national average decline in annual average concentrations of approximately 31 percent over the 16-year period, with regional average declines ranging from 16 to 39 percent.

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[19] These figures do not depict officially designated PM_{10} nonattainment areas. As of January 1, 2005, there were a total of 58 areas classified as moderate or serious nonattainment areas, mostly in the western U.S. See designated nonattainment areas at www.epa.gov/oar/oaqps/greenbk/pnc.html. Further, note that these maps (like the other PM ones in this Chapter) do not exclude event-flagged data (natural or exceptional). Data flagged for events are sometimes excluded from regulatory design value calculations.

[20] The form of the 1987 PM_{10} 24-hour standard is based on the number of exceedances; the metric used for this map, "concentration-based 24-hour 'design value' type metric" is almost always calculated to be 150 $\mu g/m^3$ or higher when the monitoring site violates the explicit exceedance-based NAAQS. Utilization of the concentration-based metric permits delineation of gradients and facilitates comparisons with $PM_{2.5}$ and $PM_{10-2.5}$.

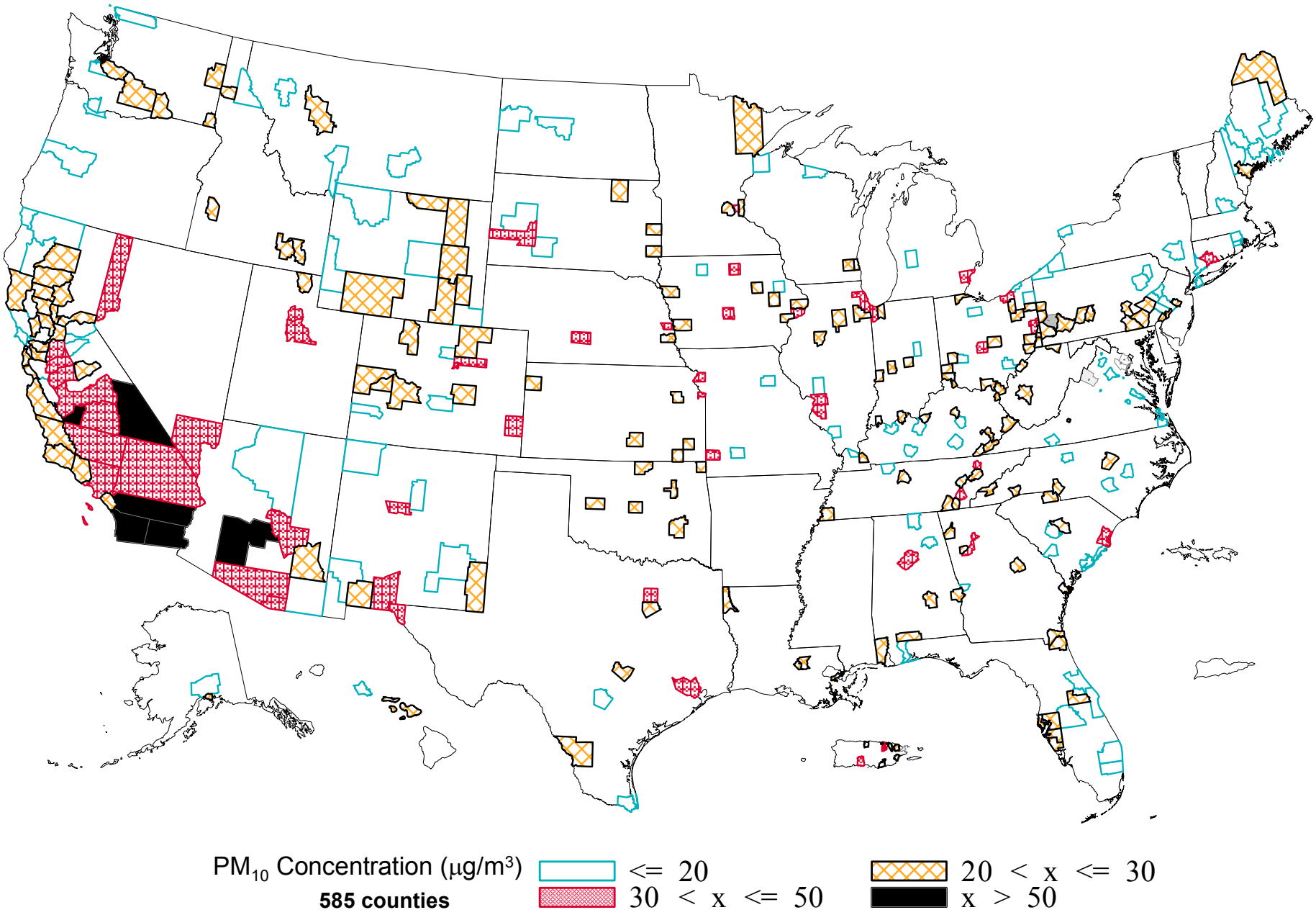


Figure 2-10. County-level maximum PM_{10} annual mean concentrations, 2001-2003.

Source: Schmidt et al. (2005)

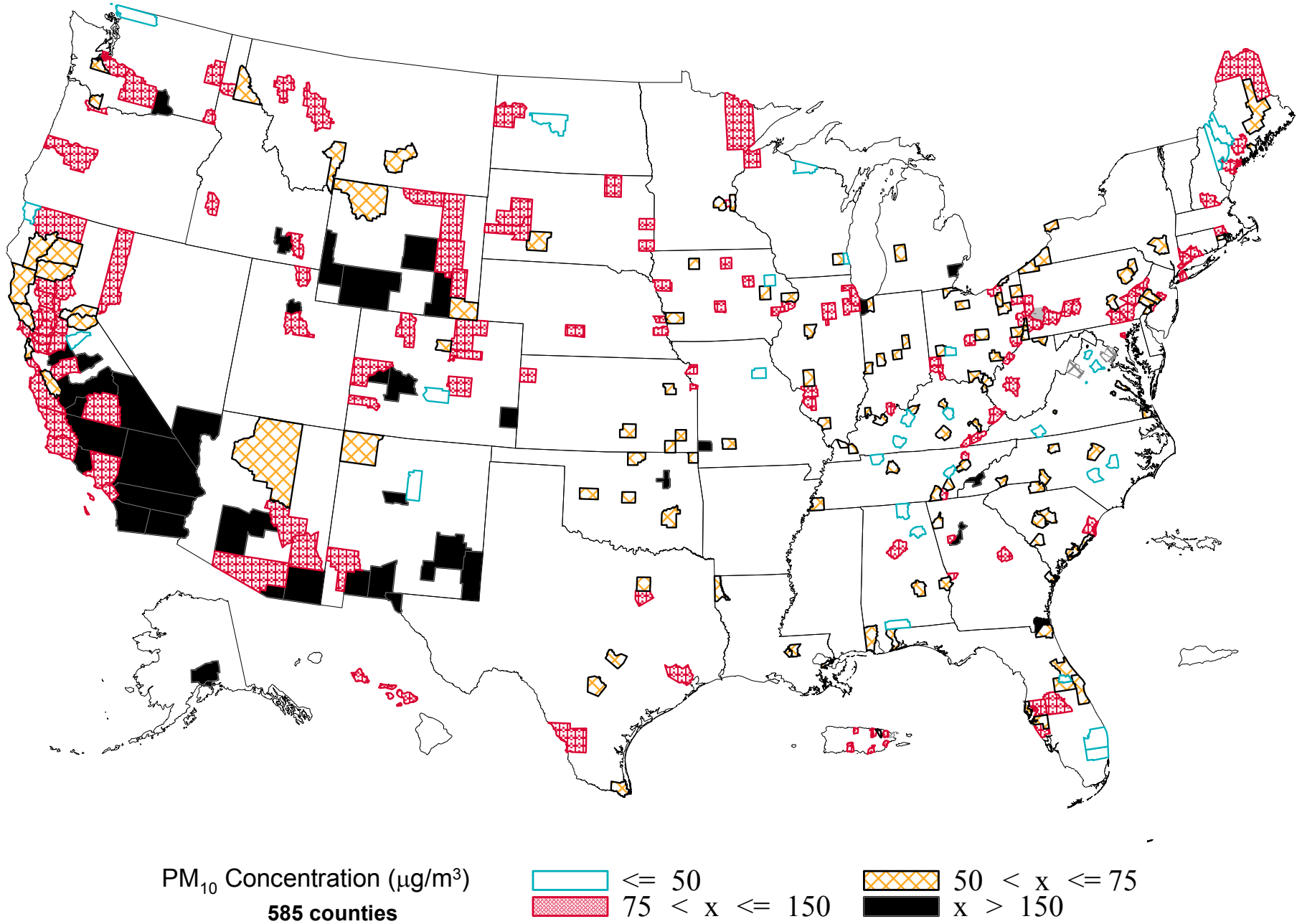


Figure 2-11. County-level maximum 24-hour PM_{10} 'design value' concentrations, 2001-2003.

Source: Schmidt et al. (2005)

2.4.3 $PM_{10-2.5}$

$PM_{10-2.5}$ is a measure of the coarse-mode fraction of PM_{10} being considered in this review. It can be directly measured by a dichotomous sampler, or by using a difference method with collocated PM_{10} and $PM_{2.5}$ monitors. For the latter, collocated PM_{10} and $PM_{2.5}$ monitors using identical inlets, sampling flow rates, and analysis protocols produce the most precise results. A nationwide network of samplers with the specific intent to consistently and accurately measure $PM_{10-2.5}$ does not currently exist. The EPA is currently evaluating a variety of monitoring platforms, including alternative continuous methods, to permit establishment of reference and equivalent methods for $PM_{10-2.5}$. These could be used in the future to design a national network of monitors to measure coarse-fraction particles. Until such a network is established, estimates of $PM_{10-2.5}$ can be generated for a limited number of locations using a difference method on same-day data. For this review, PM measurements collected from collocated PM_{10} and $PM_{2.5}$ FRM monitors are utilized. 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.[21]

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

[21] 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'.

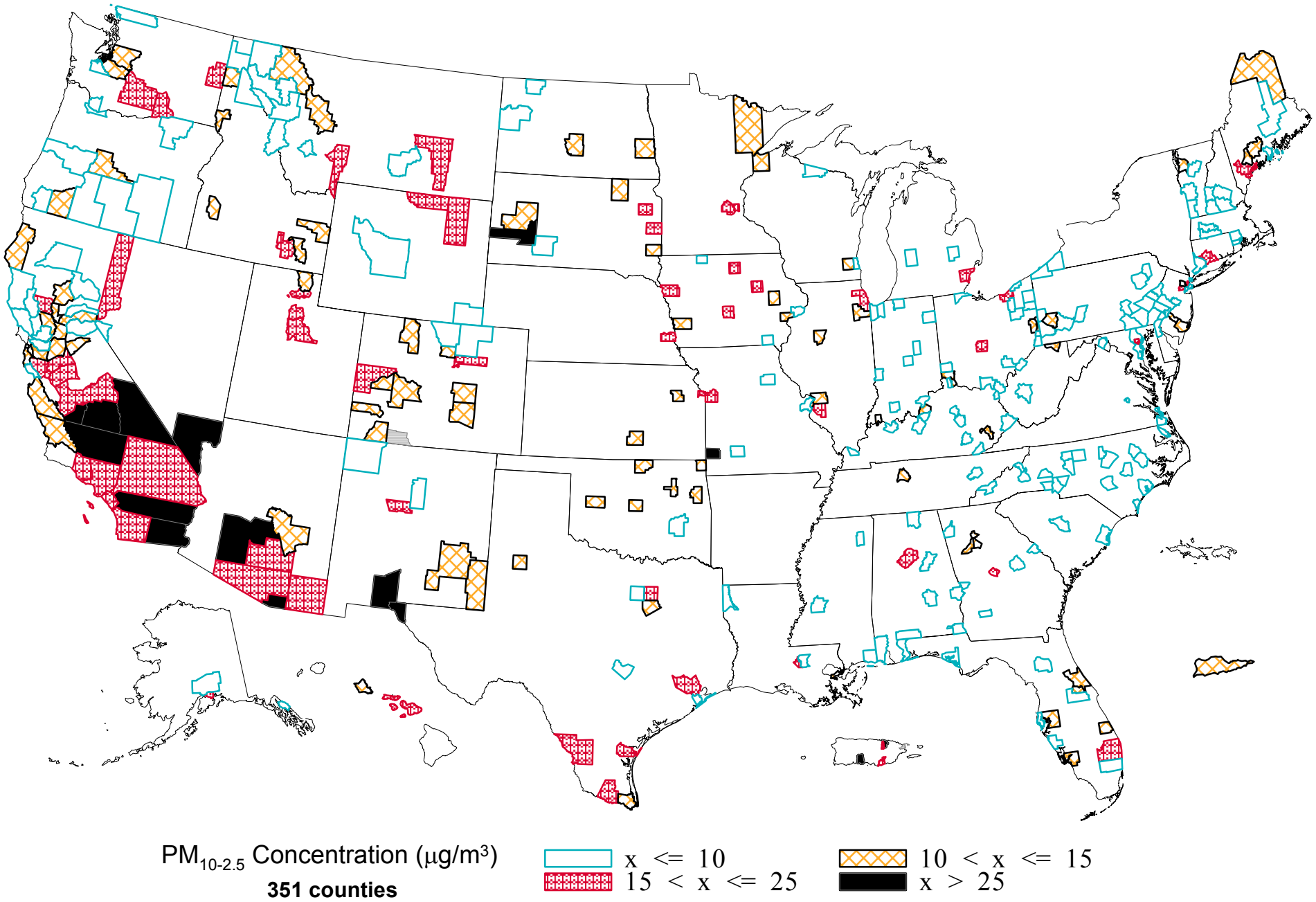


Figure 2-12. Estimated county-level maximum annual mean $PM_{10-2.5}$ concentrations, 2001-2003.

Source: Schmidt et al. (2005)

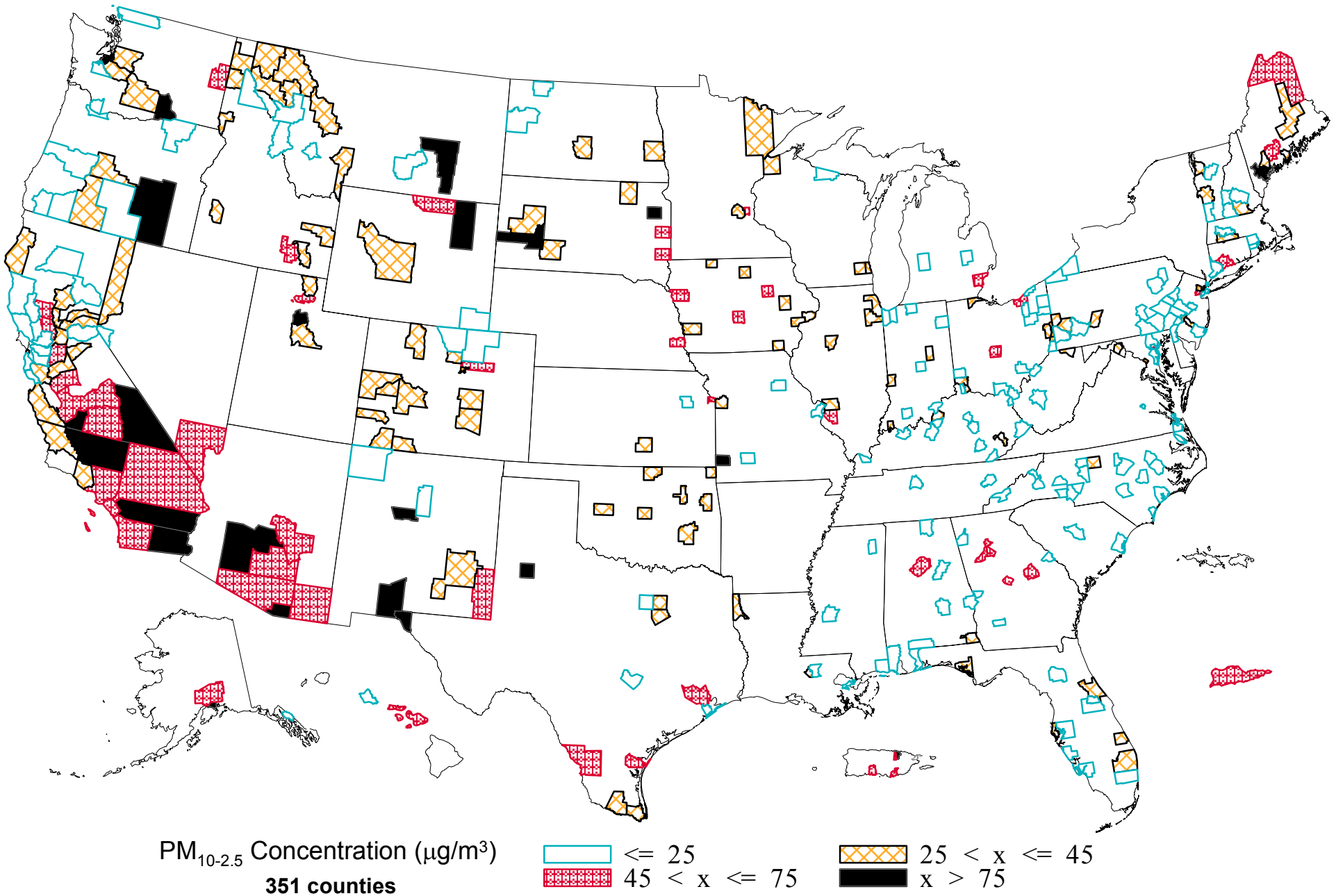


Figure 2-13. Estimated county-level maximum 98th percentile 24-hour average $PM_{10-2.5}$ concentrations, 2001-2003.

Source: Schmidt et al. (2005)

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 $PM_{10-2.5}$ 98^{th} percentiles even more than for $PM_{2.5}$. An evaluation of 2001-2003 $PM_{10-2.5}$ data found that such events caused 98^{th} percentile values to be elevated by an average of 2.5 $\mu g/m^3$ (Schmidt et al., 2005).

The IMPROVE monitoring network generally provides long-term $PM_{10-2.5}$ trends for rural areas. Figure 2-14 presents the composite long-term trend at 7 eastern sites, 17 western sites, and one urban site in Washington, D.C. At the eastern rural sites, measured $PM_{10-2.5}$ in 2003 was about 33 percent lower then the corresponding value in 1993. At the western rural 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 $\mu g/m^3$ (over 40%) higher than the 1998 low point.

The long-term $PM_{10-2.5}$ levels in the relatively remote non-urban IMPROVE sites shown in Figure 2-14 are notably lower than those found in most urban areas. While $PM_{10-2.5}$ concentrations in rural areas affected by sources such as windblown dry lake beds, unpaved roads, or agricultural activities can be quite high, comparison of paired urban and nearby rural sites suggest that $PM_{10-2.5}$ levels are generally higher in urban areas. Figure 2-15 shows urban and corresponding rural $PM_{10-2.5}$ concentrations for several large metropolitan areas in the eastern and western U.S.. The urban data represent inter-city or suburban monitoring sites located in densely populated regions of the metro areas, and the rural data typically represent one or more sites situated on the outskirts of the areas where population density is low. In all the metro areas shown, the urban $PM_{10-2.5}$ concentrations exceed those in the nearby rural locations.

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). A similar analysis, for 21 urban areas defined as either Core Based Statistical Areas (CBSAs) or Combined Statistical Areas (CSAs), was conducted on $PM_{10-2.5}$ estimates from 2001-2003 (Schmidt et al., 2005). These analyses are similar to the 49-city analysis for $PM_{2.5}$ discussed in section 2.4.1 and summarized earlier in Table 2-3. 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 200 monitor pairs analyzed, only 17 (9%) had correlation coefficients greater than or equal to 0.80, in contrast to around 83 percent (1901 of 2290) of the pairs for $PM_{2.5}$.

The difference in estimated annual mean $PM_{10-2.5}$ between site pairs in the 21 areas also covered a greater range than was seen for $PM_{2.5}$, with differences up to almost 31 $\mu g/m^3$ in Los

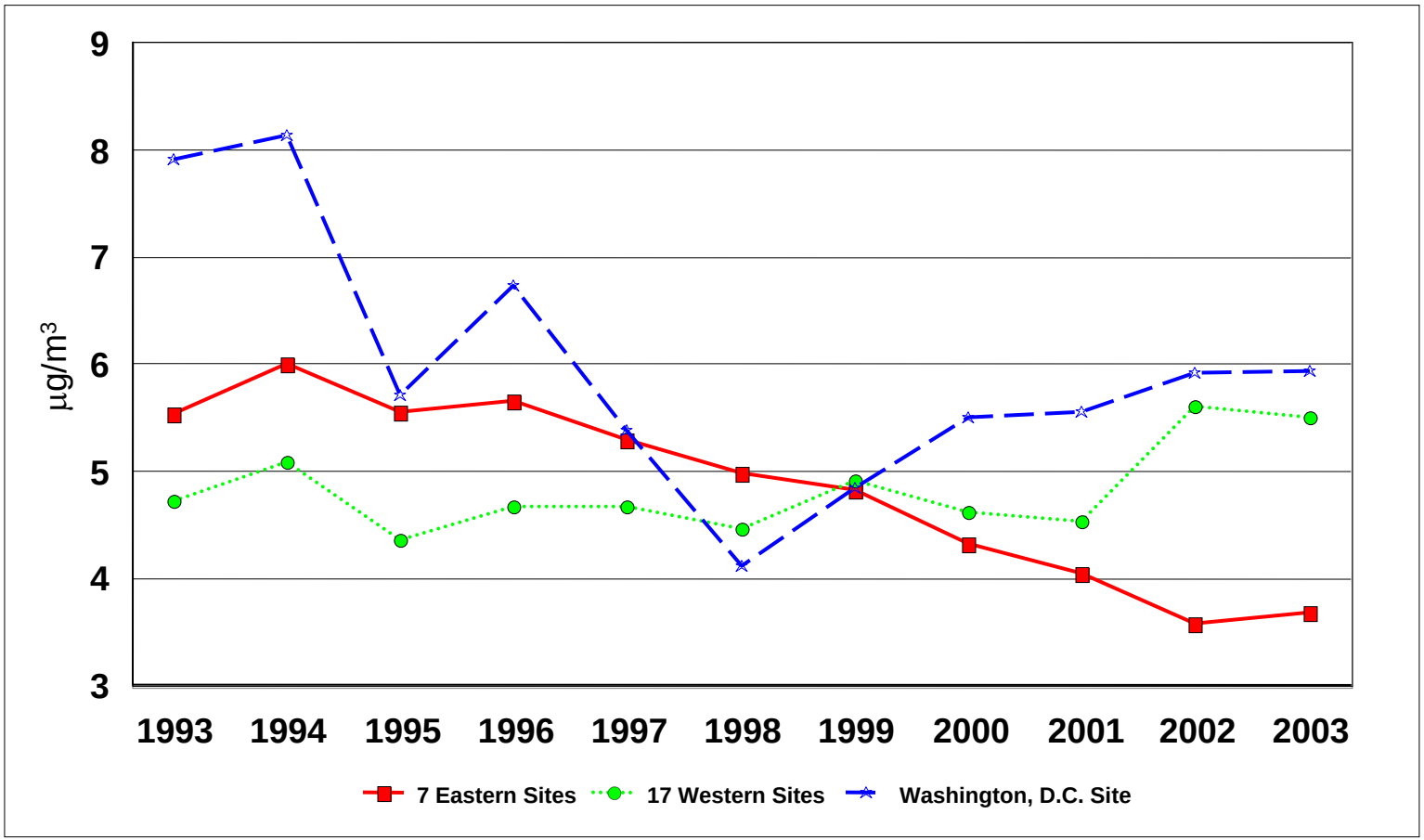


Figure 2-14. Average measured annual average $PM_{10-2.5}$ concentration trend at IMPROVE sites, 1993-2003.

Source: Schmidt et al. (2005)

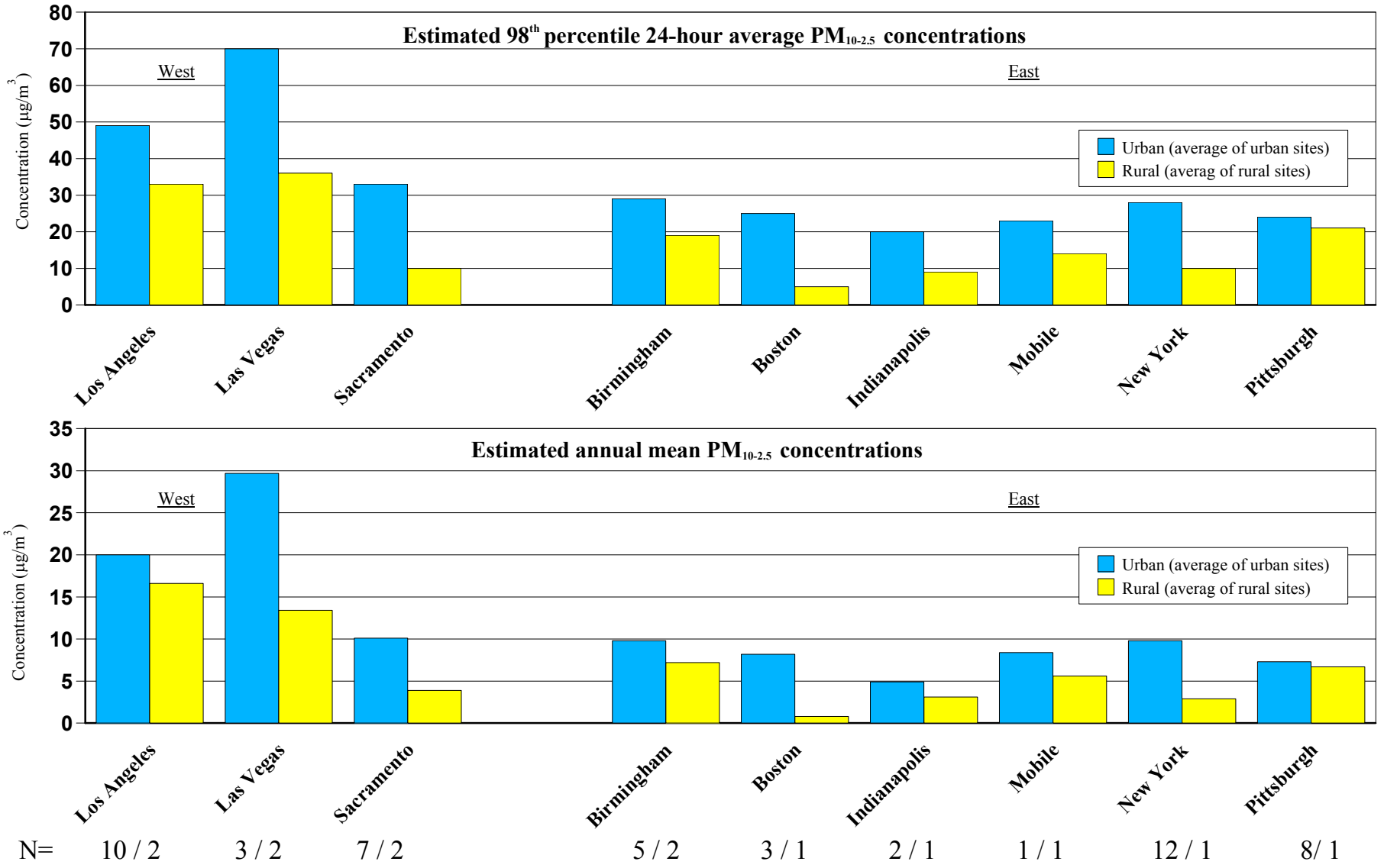


Figure 2-15. Urban versus rural estimated $PM_{10-2.5}$ concentrations in select areas. Estimated 98th percentile 24-hour average $PM_{10-2.5}$ concentrations shown in top panel and estimated annual mean $PM_{10-2.5}$ concentrations shown in bottom panel. Urban bar (left) is average of urban sites in area, rural bar (right) is average of nearby rural sites. Urban / rural designation from AQS. N= number of sites (urban / rural).

Schmidt et al. (2005)

Table 2-4. Summary of Estimated $PM_{10-2.5}$ Analyses in 21 Metropolitan Areas, 2001-2003

Area *	N Sites	3-year Average Annual Mean						24-Hour P_{90} ($\mu g/m^3$) **		
		Levels ($\mu g/m^3$)			Percent Difference		r (Max site versus Min site)	Max Pair	Min Pair	r (Max Pair)
		Area Avg	Max Site	Min Site	Largest diff., any site versus Area Avg	Max site versus Min site				
Anchorage, AK	3	14.8	23.7	9.6	38%	59%	0.13	52.3	22.5	0.13
Birmingham-Hoover-Cullman, AL	5	7.0	9.0	5.6	22%	38%	0.76	10.0	3.0	0.55
Cleveland-Akron-Elyria, OH	8	11.6	16.3	5.6	52%	66%	0.55	26.0	8.0	0.64
Denver-Aurora-Boulder, CO	3	15.5	22.1	7.7	50%	65%	0.54	29.3	14.5	0.54
Detroit-Warren-Flint, MI	3	15.3	18.7	8.8	42%	53%	0.60	30.5	25.0	0.32
El Paso, TX	4	23.2	28.3	13.9	40%	51%	0.89	31.0	15.0	0.92
Las Vegas-Paradise-Pahrump, NV	5	23.2	33.3	9.0	61%	73%	0.65	40.0	17.0	0.65
Los Angeles-Long Beach-Riverside, CA	11	21.6	44.5	13.7	51%	69%	0.38	57.5	8.5	0.03
Miami-Fort Lauderdale-Miami Beach, FL	4	10.2	15.3	8.4	33%	45%	0.63	14.0	3.0	0.63
Minneapolis-St. Paul-St. Cloud, MN-WI	3	19.1	23.6	15.5	19%	34%	0.62	23.0	19.5	0.38
New York-Newark-Bridgeport, NY-NJ-CT-PA	5	8.7	22.3	2.9	67%	87%	0.21	35.3	6.5	0.21
Orlando-The Villages, FL	3	9.5	10.2	8.5	11%	17%	0.71	6.0	4.0	0.71
Philadelphia-Camden-Vineland, PA-NJ-DE-MD	3	5.5	6.4	4.3	22%	33%	0.48	10.0	6.0	0.48
Pittsburgh-New Castle, PA	6	6.4	8.5	3.5	45%	59%	0.67	13.0	5.0	0.46
Sacramento--Arden-Arcade--Truckee, CA-NV	3	10.4	12.0	8.2	21%	32%	0.38	17.5	6.5	0.25
Salt Lake City-Ogden-Clearfield, UT	3	17.9	24.1	14.4	26%	40%	0.72	24.0	9.0	0.72
San Jose-San Francisco-Oakland, CA	7	10.8	13.4	7.8	28%	42%	0.69	13.5	4.5	0.53
San Juan-Caguas-Fajardo, PR	3	24.4	30.2	18.0	26%	40%	0.64	22.0	17.0	0.64
Virginia Beach-Norfolk-Newport News, VA-NC	3	4.2	4.5	4.0	7%	11%	0.54	5.0	3.0	0.54
Weirton-Steubenville, WV-OH	4	12.4	13.8	10.7	14%	22%	0.53	15.0	11.5	0.43
Wichita-Winfield, KS	3	11.9	13.7	10.3	13%	25%	0.81	11.0	5.0	0.69

* 'Area' is the larger of a Combined Statistical Area (CSA) or a Core Based Statistical Area (CBSA). See http://www.whitehouse.gov/omb/bulletins/fy05/b05-02.html.

** 'P_{90}' is the 90th percentile of the distribution of differences in 24-hour averages between two sites in the same urban area.

Angeles, CA. The largest percent difference between any site's annual mean and it's corresponding area spatial average ranged from 7 to 67 percent with a median of 28 percent, which is about double the median from the corresponding $PM_{2.5}$ analyses. Of the 18 common metropolitan areas analyzed for both $PM_{2.5}$ and $PM_{10-2.5}$, only 2 areas (Sacramento, CA and San Juan, PR) had higher values for this indicator (largest percent difference for any site in area verus area spatial average) for $PM_{2.5}$ compared to $PM_{10-2.5}$. The P_{90} values (described in section 2.4.1) for the 200 $PM_{10-2.5}$ site pairs ranged from about 3 $\mu g/m^3$ to about 58 $\mu g/m^3$, which is wider than the range of about 1 to 45 $\mu g/m^3$ observed for $PM_{2.5}$.

These analyses indicate that spatial distribution of $PM_{10-2.5}$ is more heterogeneous than $PM_{2.5}$ in many locations but may be similar in other areas. 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 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 2-16 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:

- While $PM_{2.5}$ mass and all component concentrations are higher in urban areas than in IMPROVE sites, in general, nitrates and carbonaceous components appear to have a greater urban/rural enhancement as compared to sulfates.
- $PM_{2.5}$ in the eastern U.S. regions is dominated by sulfates and carbonaceous mass.
- $PM_{2.5}$ in the western U.S. urban sites has a greater proportion of carbonaceous mass.

Trends concentrations of fine particle components from the IMPROVE network from 1993 to 2003 are shown in Figure 2-9 for rural areas and for urban Washington, D.C. (section 2.4.1 above). The top two panels of this figure aggregate rural IMPROVE sites in the eastern and western U.S. The bottom panel shows a comparable period for the Washington, D.C. urban IMPROVE site. Consistent with more recent data in Figure 2-15, levels of rural annual average $PM_{2.5}$ mass are significantly higher in the east than in the west, but are trending downward in both regions. Annual levels of 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 carbonaceous mass (34 percent reduction) and sulfates (down 29 percent) are the biggest contributors to the mass reduction over the past 10 years. Both total carbonaceous mass and sulfates dropped significantly at this site 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.

Though most of the speciation data currently available are for $PM_{2.5}$, there are a limited amount of recent data available on speciation profiles for the coarse fraction, and still less for ultrafine particles. The EPA "Supersite" program addresses a number of scientific issues associated with PM.[22] A Supersite location in the Los Angeles metropolitan area (USC site) provides a unique comparison of the composition of ultrafine, fine, and coarse particles (Sardar et al., 2005). Based on the reported measurement data, ultrafine, fine, and coarse PM have distinctly different compositions at this site (Figure 2-17). Increasing in size from ultrafine to fine to coarse, the relative fraction of organic carbon (dominant for ultrafine) drops, and the crustal element portion goes from a minor component (ultrafine, fine) to the dominant fraction

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

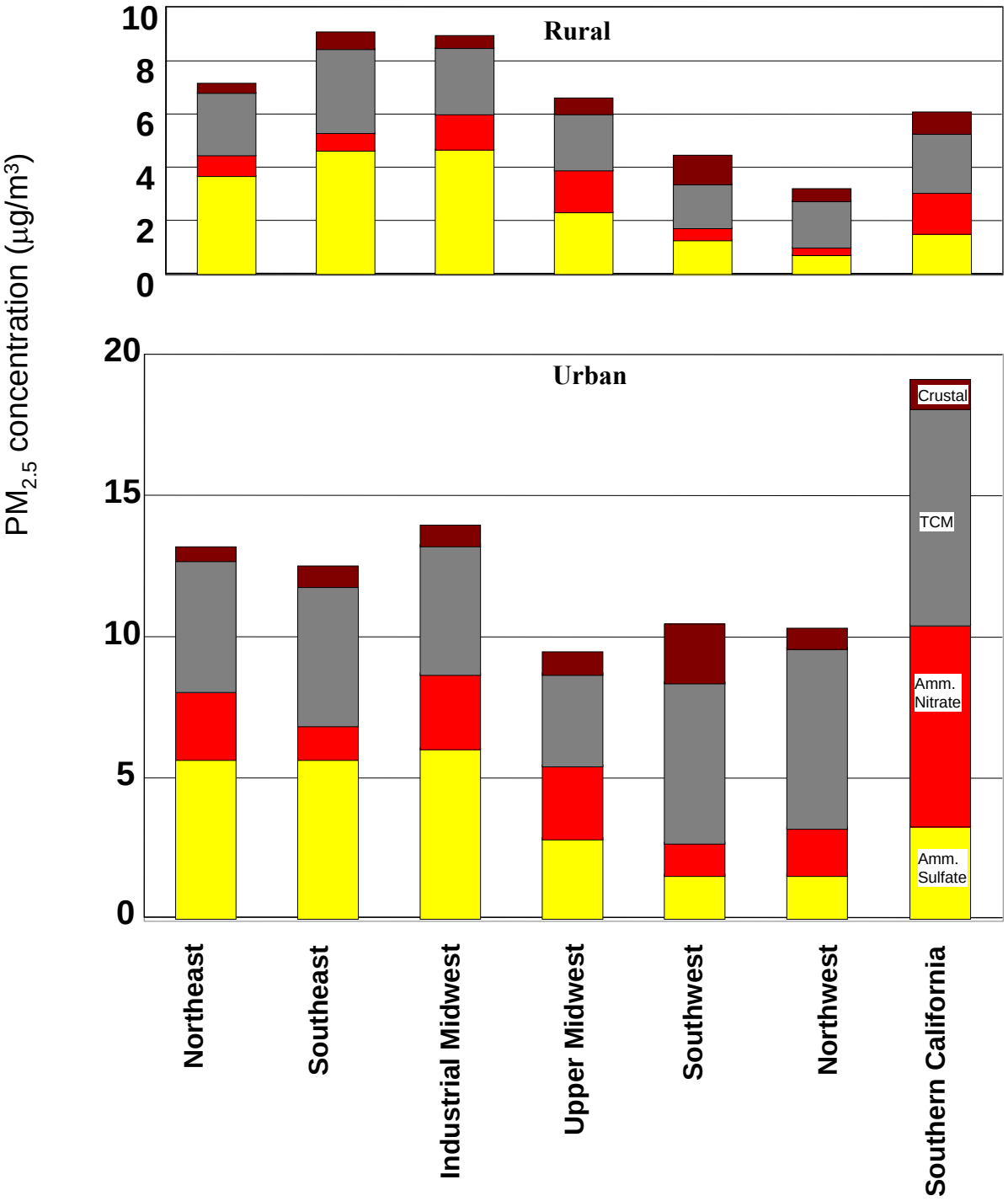


Figure 2-16. Annual average composition of $PM_{2.5}$ by region, 2003. Rural data (top panel) from IMPROVE network, urban data (bottom panel) from EPA Speciation Network. Components (from top to bottom) are crustal material, total carbonaceous mass (TCM), ammonium nitrate, and ammonium sulfate.

Source: Schmidt et al. (2005)

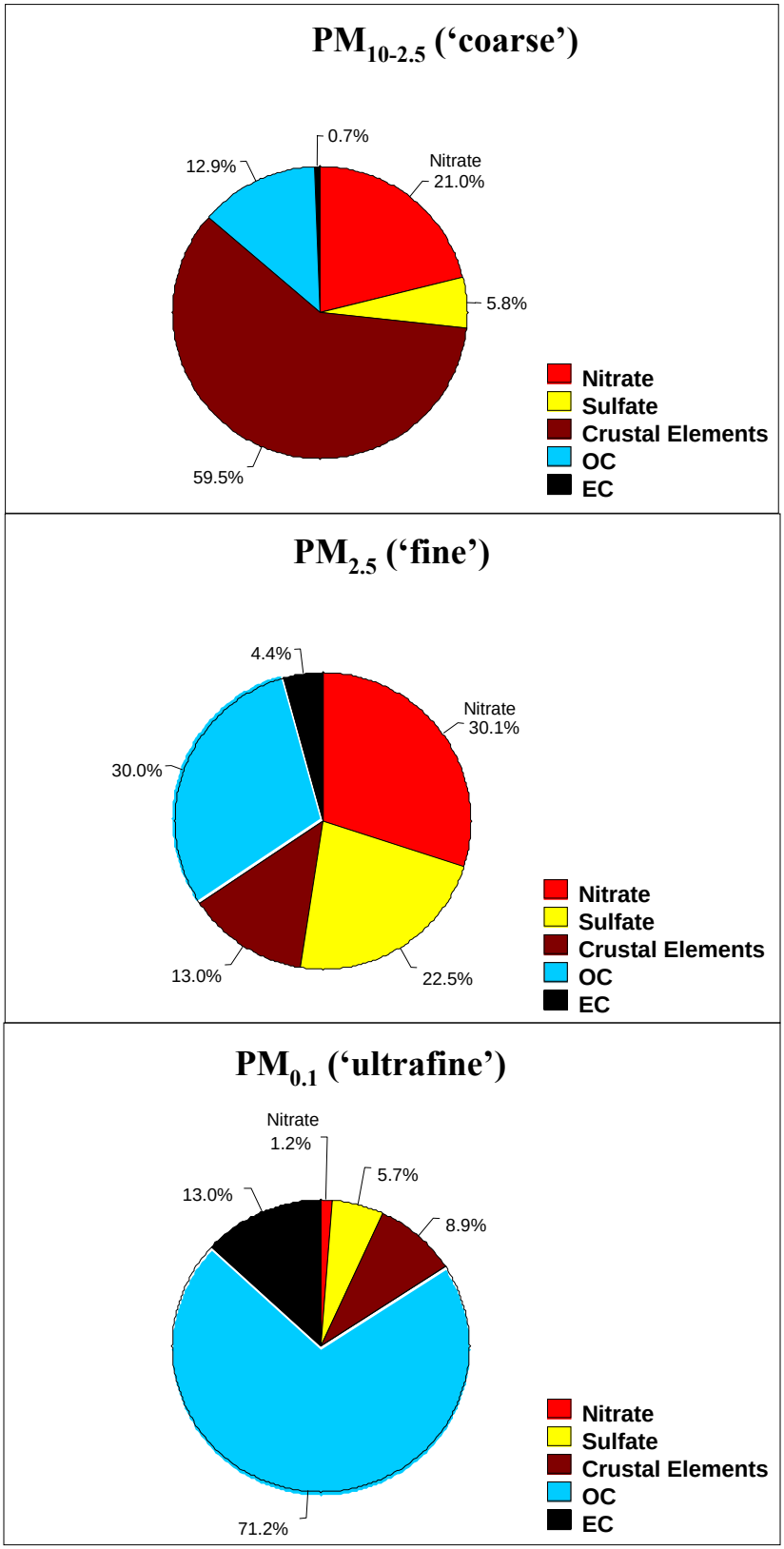


Figure 2-17. Average $PM_{10-2.5}$, $PM_{2.5}$, and $PM_{0.1}$ (ultrafine) chemical composition at the USC EPA 'supersite' monitor in Los Angeles, CA, 10/2001 to 9/2002. components represent measured ions, carbon and crustal elements including trace metals and are shown in clockwise order (starting with nitrate) as listed in legend from top to bottom.

Source: Sardar et al. (2005)

(coarse). The ultrafine results are consistent with other work in Southern California (CD, p. 3-39). The large crustal fraction in the coarse mode is typical of earlier work on western sites reported in the 1996 Criteria Document (EPA, 1996a, p. 6-165 to 6-167, Figure 6-85a-c), as well as for more recent work in Phoenix (CD, p. 3-36).

Other recent work on ambient $PM_{10-2.5}$ particle composition comes from the SouthEastern Aerosol Research and Characterization (SEARCH) Study.[23] This study examined two urban sites (Birmingham, AL and Atlanta, GA) and nearby rural sites in the southeast. Figure 2-18 presents the results of this work together with the Los Angeles results. In this graphic, the measured chemical components are presented in terms of their estimated coarse particle mass, as derived from the reported measurement data.[24]

Although the scope of these results are limited, staff notes the following:

- Consistent with the mass-based comparisons in Section 2.4.3, the western site has more coarse mass than any of the 4 eastern locations, and the urban concentrations are clearly higher than nearby non-urban sites.

- The larger absolute and relative crustal and nitrate contributions in LA appear to be the main source of the higher mass. In contrast, the carbonaceous fraction is more significant at the eastern sites. While this may be due in part to a greater contribution of biological materials in the southeast, such materials would not explain the larger elemental carbon contribution, particularly in Birmingham.

- The higher urban concentrations of $PM_{10-2.5}$ in the southeast appear to be due to higher crustal and carbonaceous components than are found in nearby rural areas, suggesting urban sources make a substantial contribution to both components.

These recent studies have focused more on the indicators of the major categories of coarse particles - crustal, carbonaceous, and inorganic anions, and less so on trace elements and specific organic constituents. The CD notes that the concentrations of a number of trace elements in the coarse fraction can be comparable or higher than that for fine particles (e.g. Cr, Ni Zn, Pb, Cu), while the crustal elements (Al, Si, K, Ca, Fe) are, of course, much higher (CD, p3-37-38). While urban sources apparently increase total crustal materials, the relative proportions of some crustal elements may be enriched by urban sources relative to the proportion

[23] See http://www.atmospheric-research.com/ for information on SEARCH.

[24] Inorganic nitrate and sulfate concentrations were assumed to be solely associated with their ammonium salts, the crustal component reflect the measured elements plus their common oxides and organic carbon mass was estimated by multiplying measured organic carbon by a factor of 2.5 to account for the mass of H, O, and other elements in the coarse particle organic compounds. For the SEARCH sites, the total carbonaceous mass is estimated as the difference between measured coarse particle mass and its inorganic constituents. The OC-EC split is derived from a special carbon measurement study during 2000, 2001, 2003 and 2004.

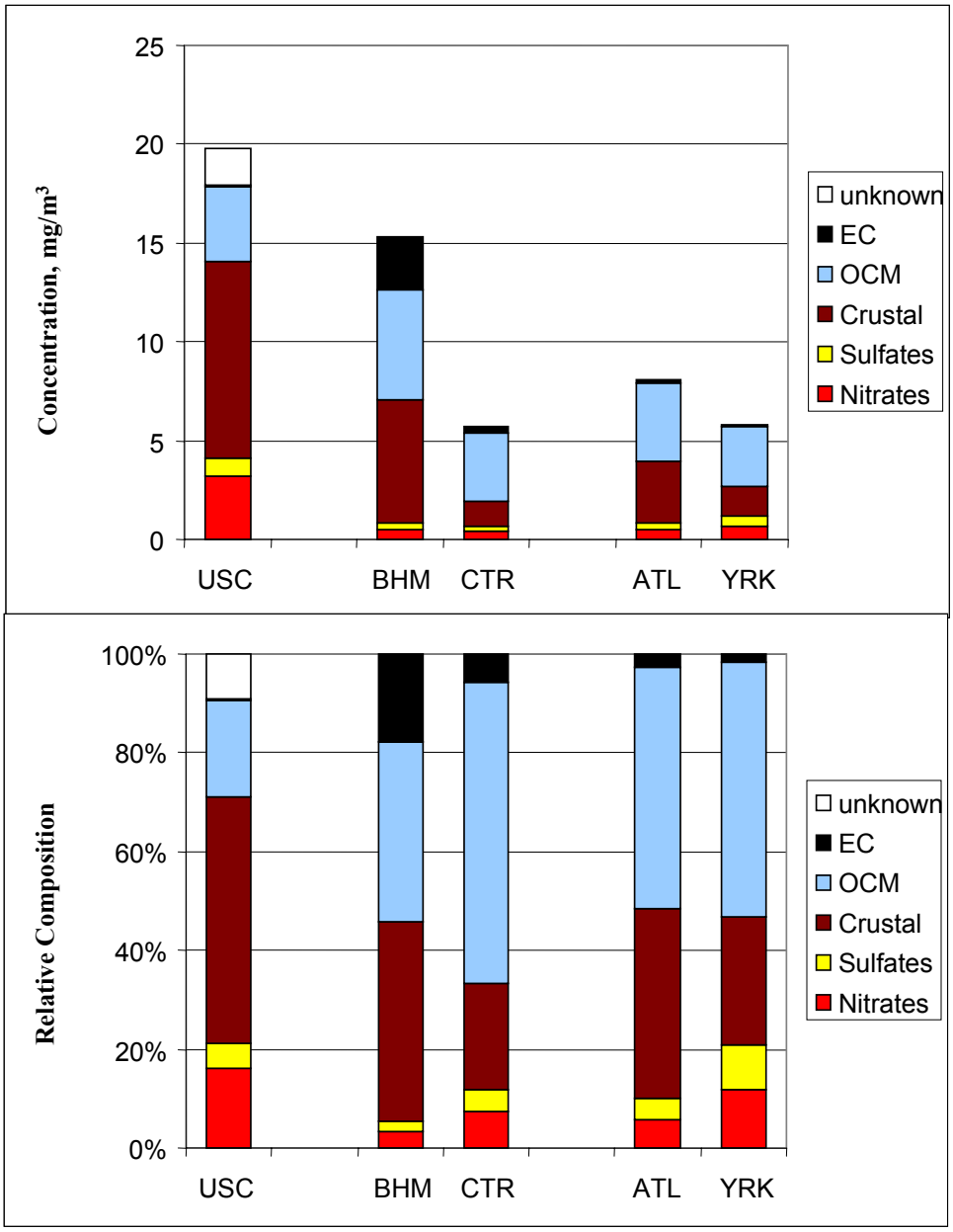


Figure 2-18. Average $PM_{10-2.5}$ composition for Los Angeles and two eastern urban-rural pairs. Based on USC Supersite data (10/2002 to 9/2003), and Birmingham, AL (BHM, urban), Centerville, AL (CTR, rural), Atlanta, GA (ATL, urban) and Yorkville, GA (YRK, rural) monitoring sites in the Southeastern Aerosol Research and Characterization (SEARCH) Study, 4/2003-12/2003. The top panel shows mass concentration in $\mu g/m^3$ and the bottom panel shows composition as percent of measured mass.

Source: USC site data (Sardar et al., 2005); eastern data (SEARCH website) adjusted as described in Schmidt et al. (2005)

found in soils. For example, urban industrial (e.g. steel) and automobiles (rust) can increase the relative amount of iron in urban coarse particles.

The CD review (Appendix 3C) lists no recent studies that speciated any substantial portion of organic components of $PM_{10-2.5}$, but some inferences can be drawn from analyses of the composition of road dusts. The CD reports on two California studies that found organic substances consistent with particles of biologic origin, tire and brake wear, asphalt, and combustion in fine fraction samples of resuspended road dust particles (CD, p. 3D-3 to 5). One of these (Rogge et al., 1993) suggests that the action of automobile traffic on leaves and other vegetative debris on roads may serve to elevate their atmospheric concentrations and decrease particle size as compared what might be found in more natural settings. The findings regarding road dust as well as trace elements are buttressed by a very recent report of similar work comparing urban and rural road dust in and near Pittsburgh (Robinson et al., 2005). These authors found that most of the over 100 organic species examined were "significantly enriched" in urban as compared to rural resuspended road dust samples. Marker substances suggested both vegetative debris and non-biological sources. Comparing trace elements, the authors found that urban road dust in Pittsburgh was enriched in metals associated with anthropogenic sources, notably Fe, Zn, Cu, Pb, Cr, Ni, Mo, and Sb. Ca and Mn were more prevalent in the rural road dust sample.

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.[25] Figure 2-19 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 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 2-20 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 in the western regions, particularly in the upper midwest. PM_{10} is fairly well correlated with

[25] 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).

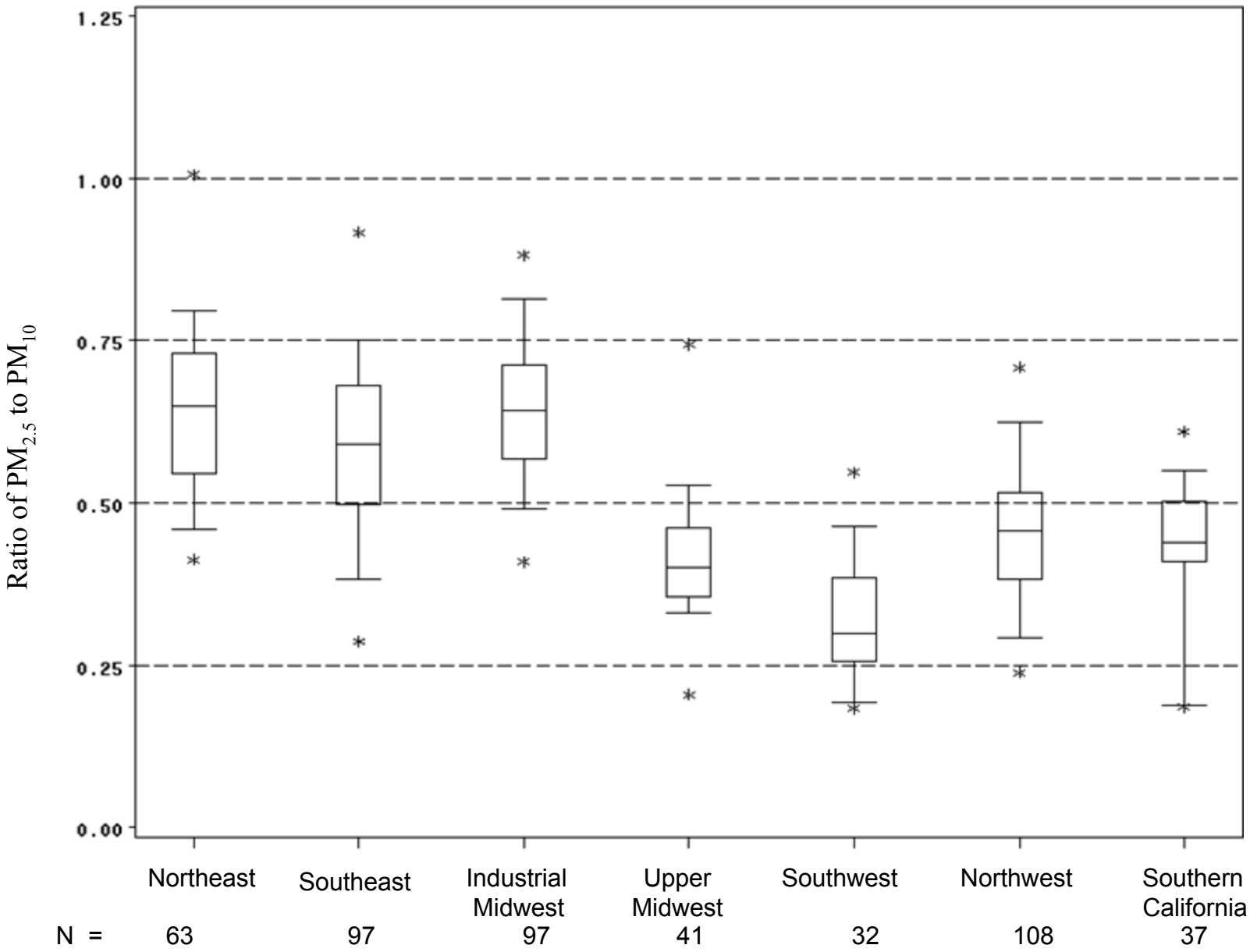


Figure 2-19. Distribution of ratios of $PM_{2.5}$ to PM_{10} by region, 2001-2003. Box depicts interquartile range and median; whiskers depict 5th and 95th percentiles; asterisks depict minima and maxima. N = number of sites.

Source: Schmidt et al. (2005)

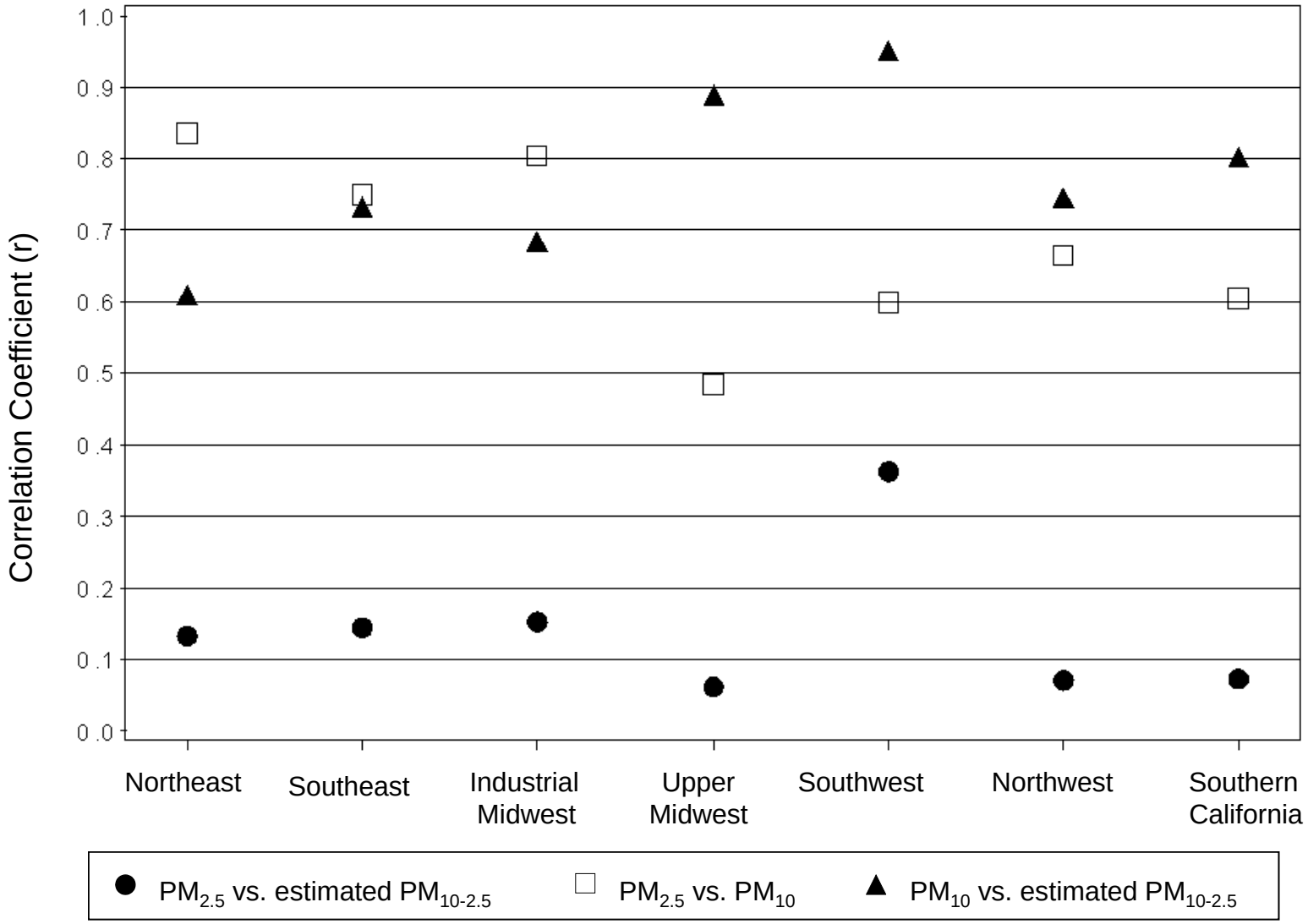


Figure 2-20. Regional average correlations of 24-hour average PM by size fraction.

Source: Schmidt et al. (2005)

estimated $PM_{10-2.5}$ in most regions, with the highest average correlations in the southwest, upper midwest, 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 networks 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 2-21 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 2-22 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 2-23 and 2-24 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-March) 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

Figure 2-21. Urban 24-hour average $PM_{2.5}$ concentration distributions by region and month, 2001-2003. Box depicts interquartile range and median; line connects monthly means. Counts above boxes indicate number of 24-hour observations

Source: Schmidt et al. (2005)

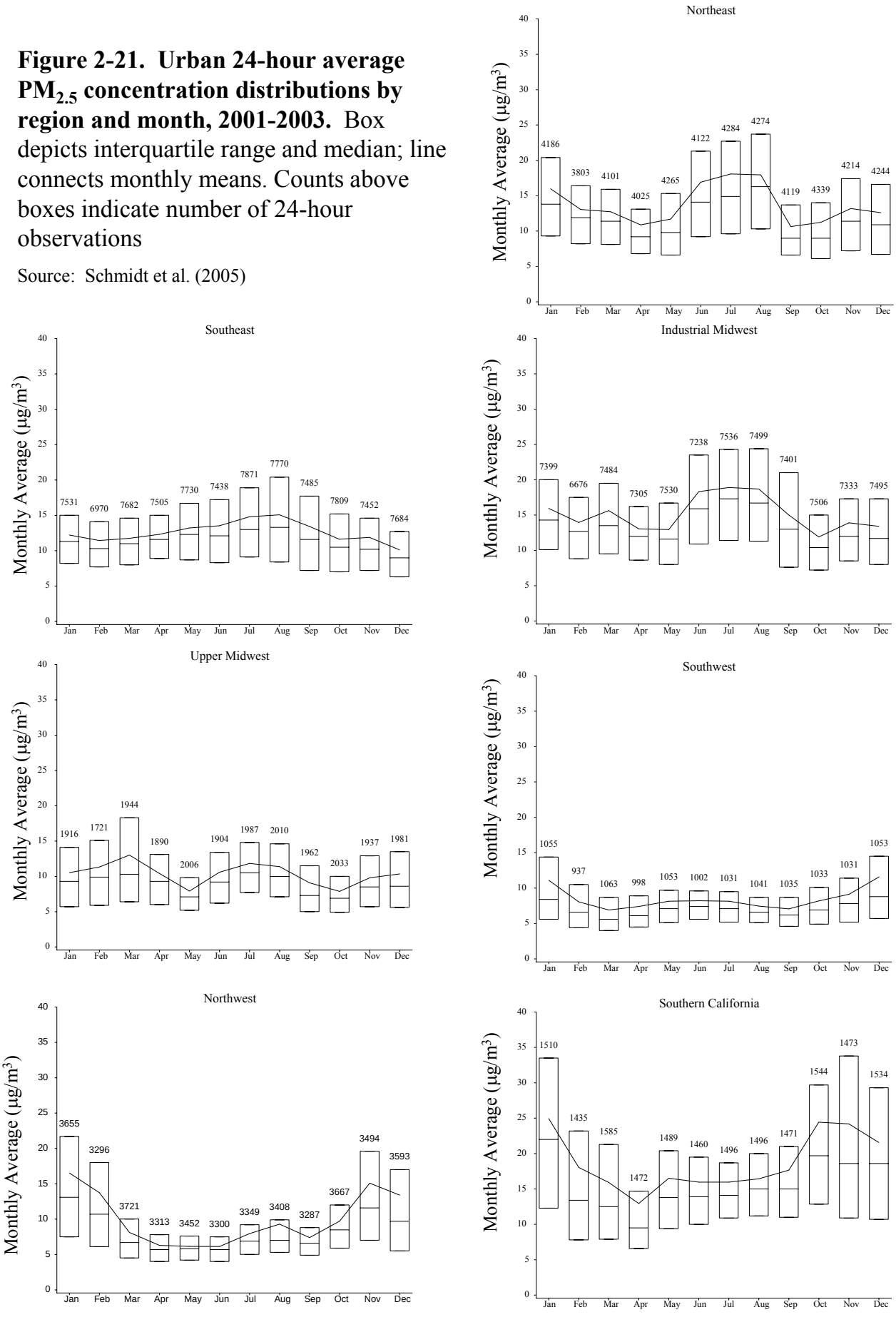
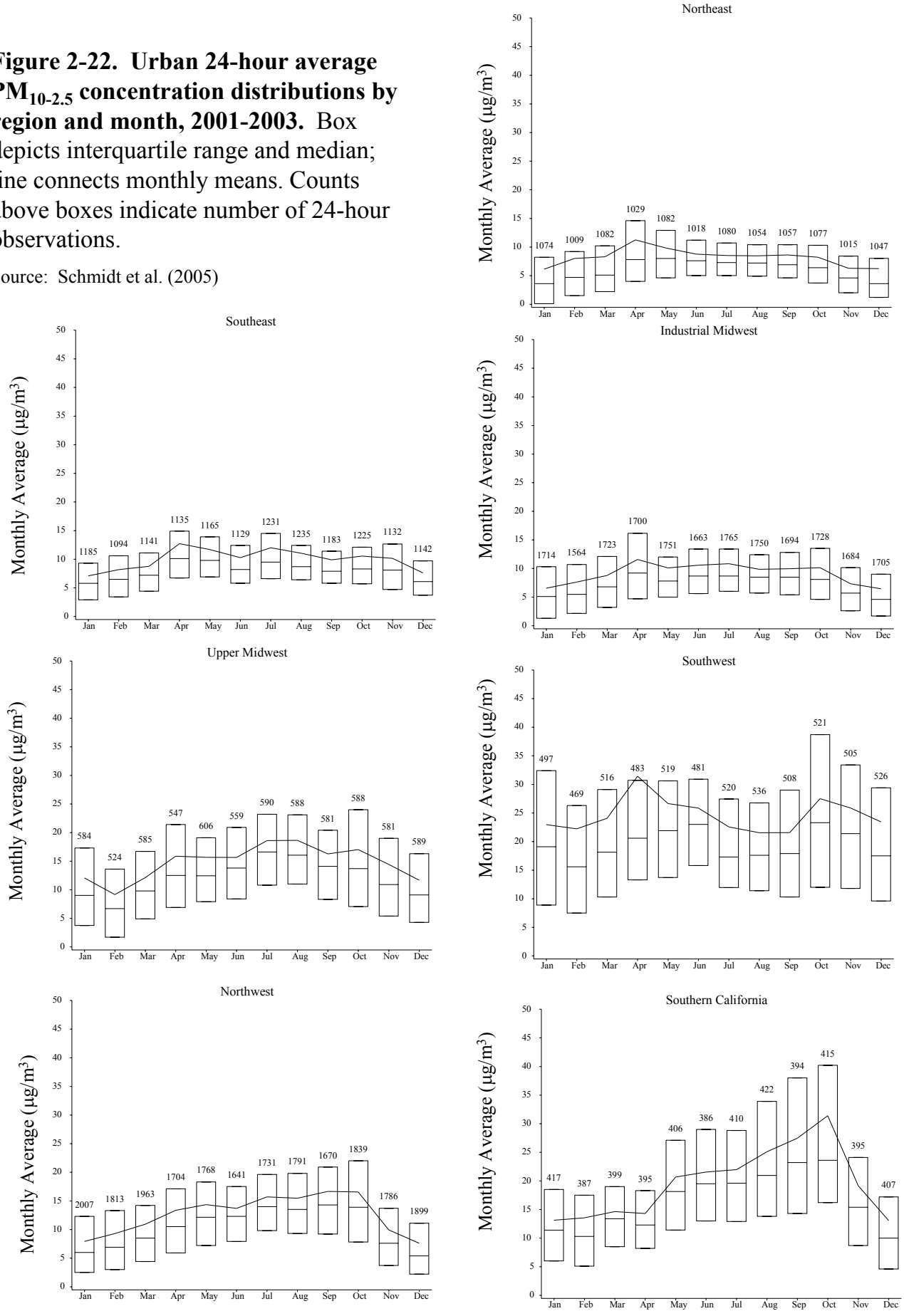


Figure 2-22. Urban 24-hour average $PM_{10-2.5}$ concentration distributions by region and month, 2001-2003. Box depicts interquartile range and median; line connects monthly means. Counts above boxes indicate number of 24-hour observations.

Source: Schmidt et al. (2005)



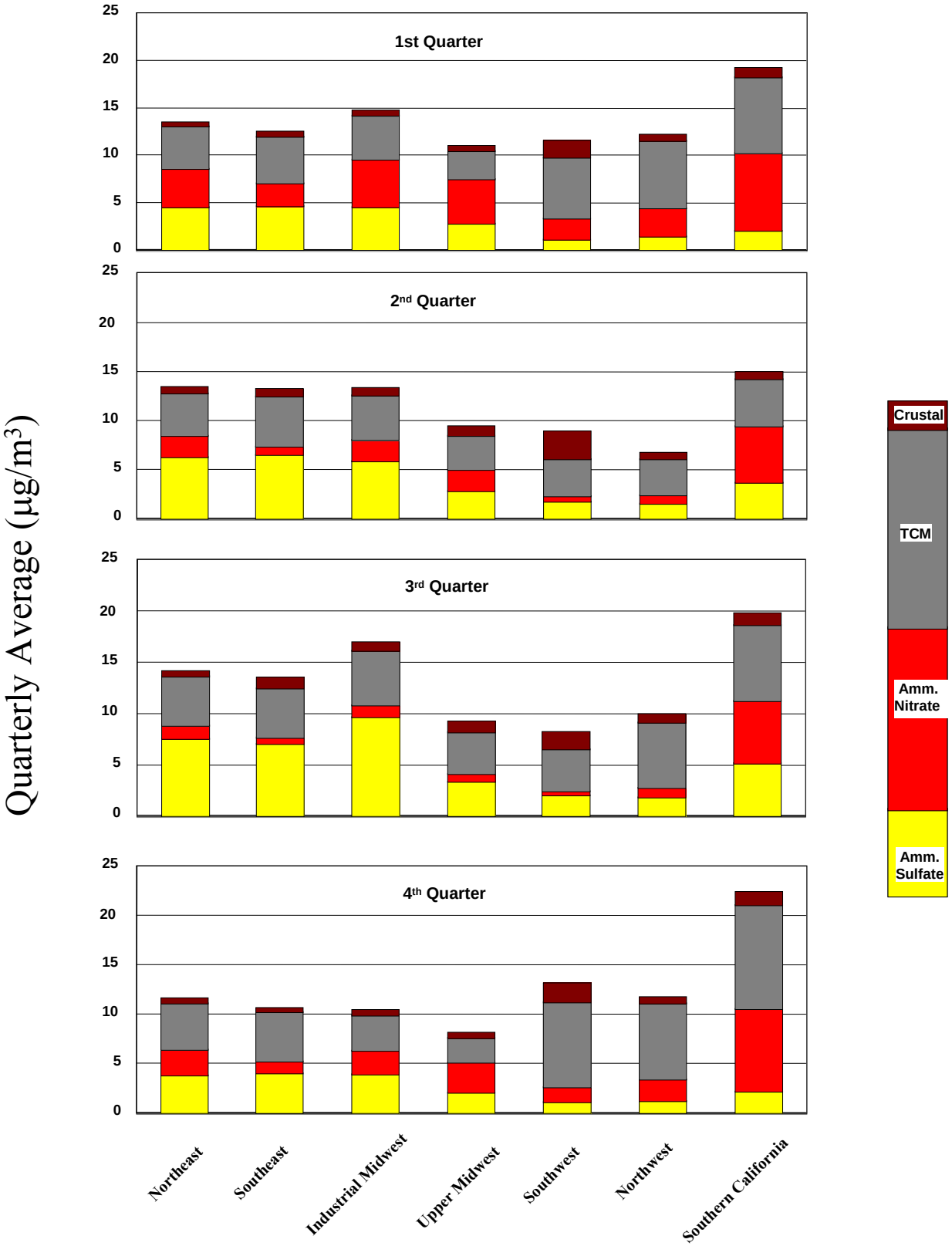


Figure 2-23. Seasonal (calendar quarter) average composition of urban $PM_{2.5}$ by region, 2003. Data from EPA Speciation Network. Components (from top to bottom) are crustal material, total carbonaceous mass (TCM), ammonium nitrate, and ammonium sulfate.

Source: Schmidt et al. (2005)

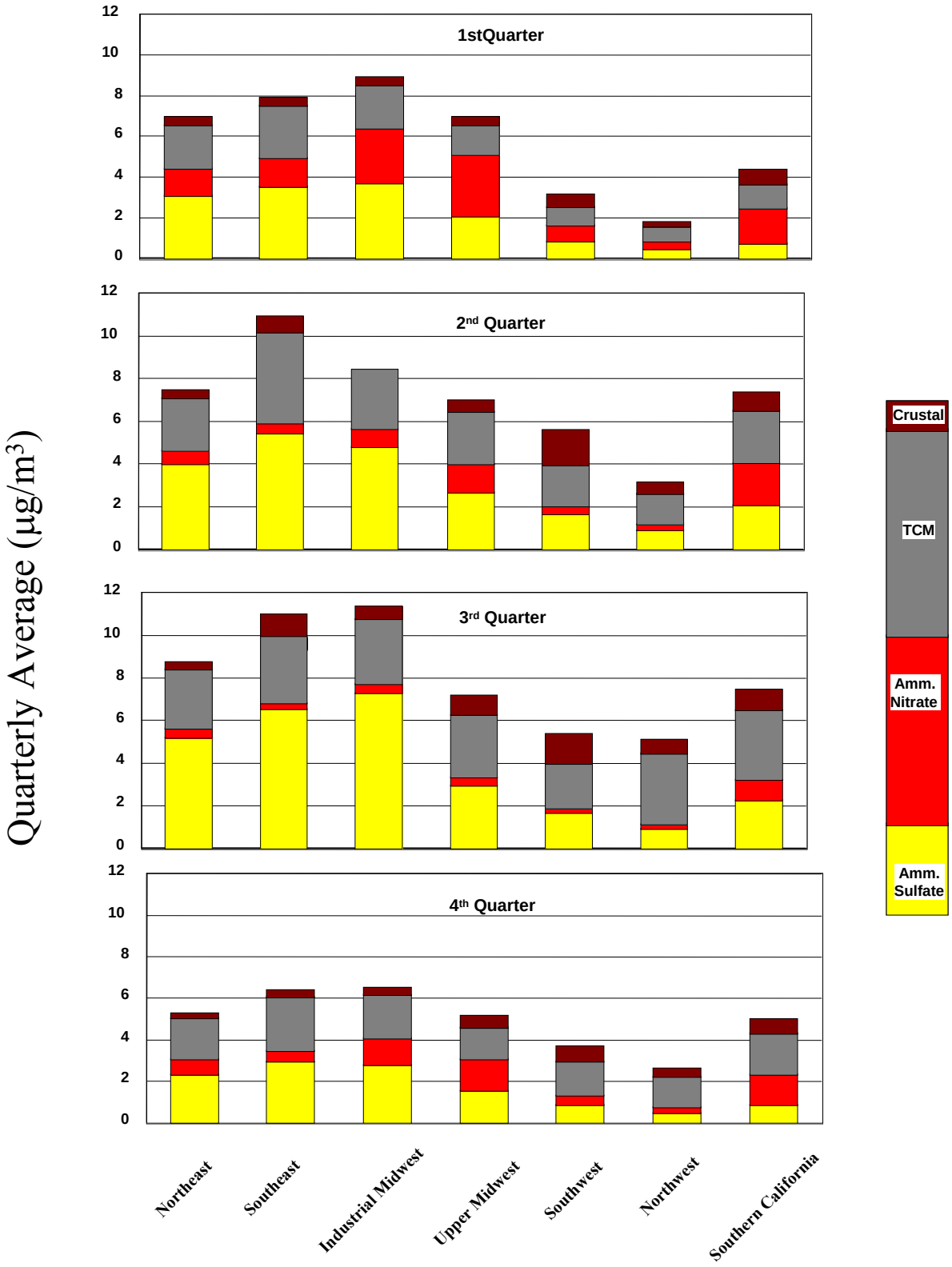


Figure 2-24. Seasonal (calendar quarter) average composition of rural $PM_{2.5}$ by region, 2003. Data from IMPROVE Network. Components (from top to bottom) are crustal material, total carbonaceous mass (TCM), ammonium nitrate, and ammonium sulfate.

Source: Schmidt et al. (2005)

particulate nitrates are more readily formed in cooler weather. In addition, the effective mixing depth is restricted due to enhanced thermal stability in the planetary boundary layer during the cooler seasons.

- 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 2-25 and 2-26 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 percentile values for each interval varies substantially. For all sites with an annual mean less than or equal 15 $\mu g/m^3$, the corresponding 98th percentile value is less than 65 $\mu g/m^3$. 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 $\mu g/m^3$.

Staff evaluated speciated $PM_{2.5}$ data for 2003 from the urban EPA network in order to compare the component profiles on high $PM_{2.5}$ mass days to annual average profiles (Schmidt et al., 2005). Table 2-5 shows the analysis results for 8 different sites in large metropolitan areas (in the east: Birmingham, AL; Atlanta, GA; New York City, NY; Cleveland, OH; Chicago, IL; and St. Louis, MO; in the west: Salt Lake City, UT; and Fresno, CA). Mass is proportioned into four categories: sulfates, nitrates, crustal, and total carbonaceous mass (TCM, the sum of EC and OCM). For each site, the table shows the 2003 annual average speciation pattern, the profile for the five highest PM2.5 mass days in that year -- both individually and averaged together -- and corresponding FRM mass values (annual average, five highest days, and average of five highest). The table shows some notable differences in the percentage contribution of each of the species to total mass when looking at the high end of the distribution versus the annual average. In all of the eastern city sites, the percentage of sulfates is somewhat higher on the five high days as compared to the annual averages. In the two western cities, the percentage of nitrates is higher on the five high days as compared to the annual averages. TCM appears somewhat lower percentage on the five high days compared to the annual averages in most cities. It is of note

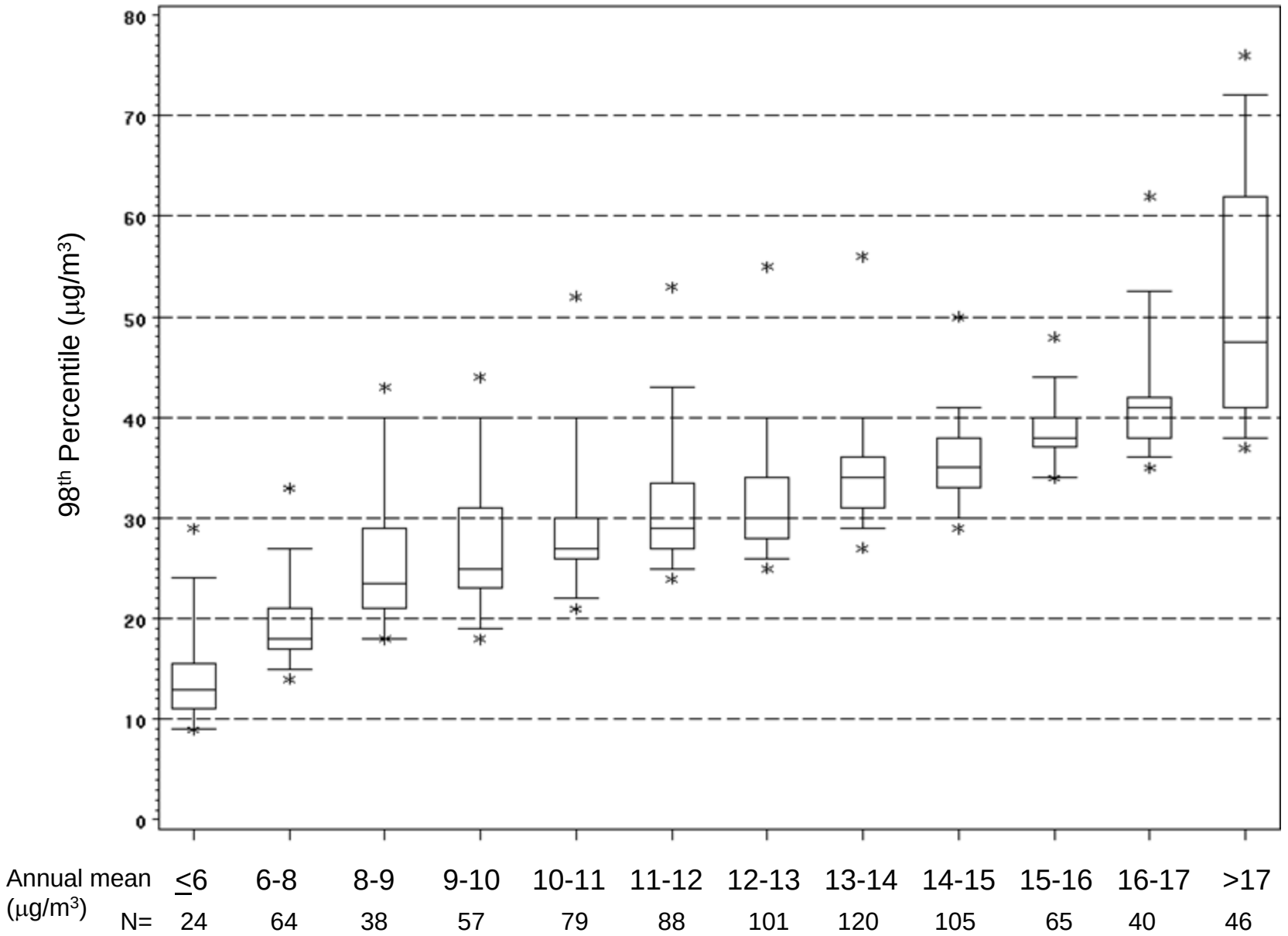


Figure 2-25. Distribution of annual mean vs. 98th percentile 24-hour average $PM_{2.5}$ concentrations, 2001-2003. Box depicts interquartile range and median; whiskers depict 5th and 95th percentiles; asterisks depict minima and maxima. N= number of sites.

Source: Schmidt et al. (2005)

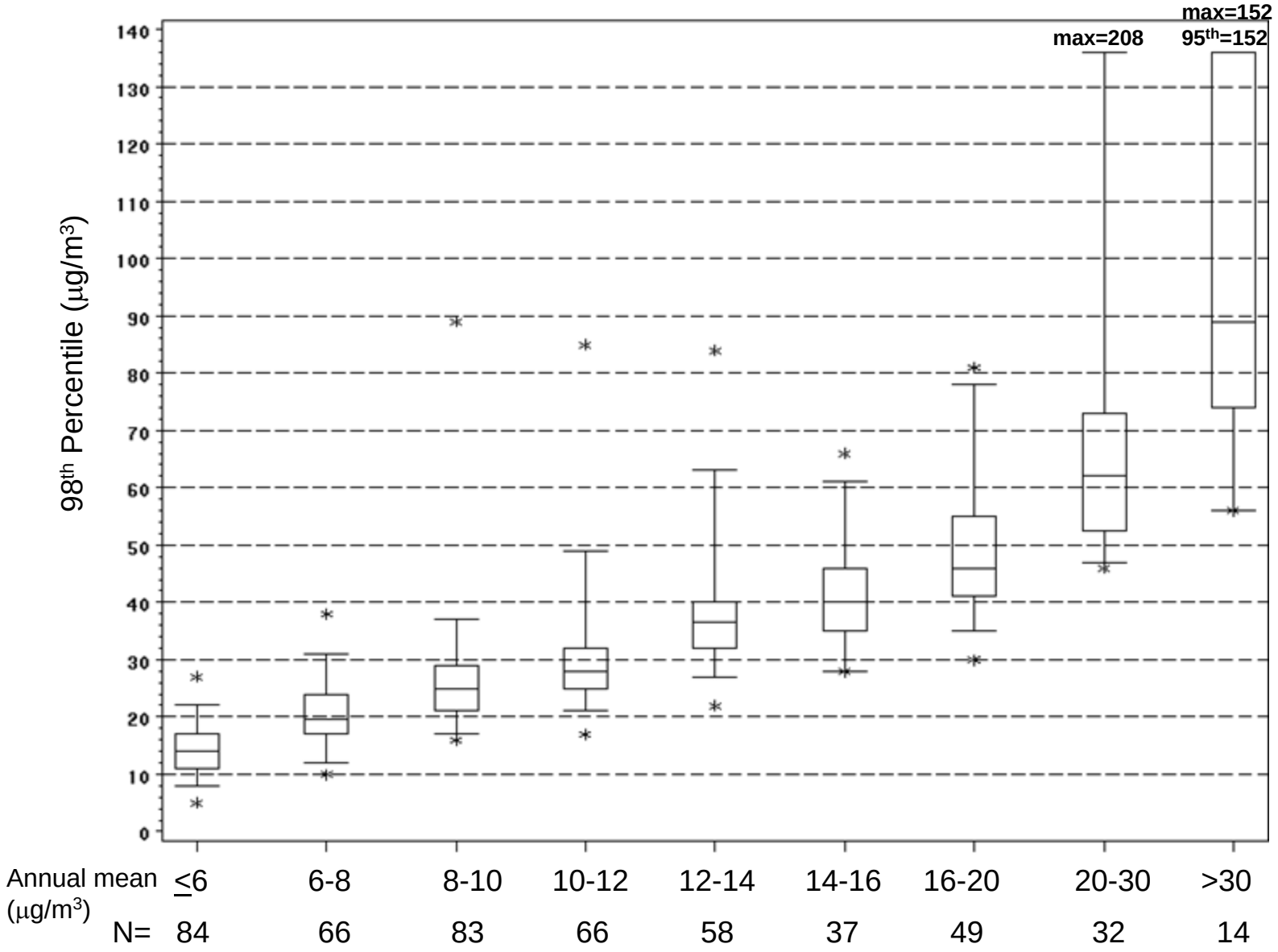
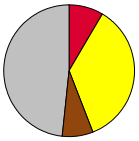
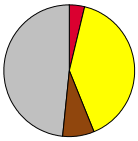
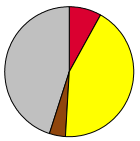
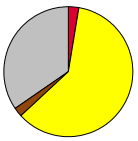
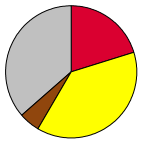
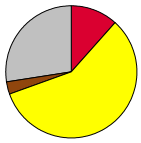
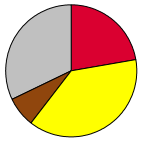
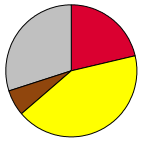
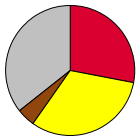
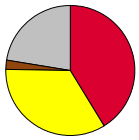
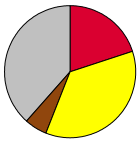
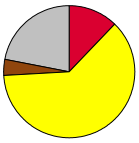
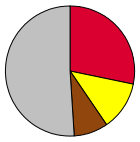
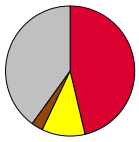
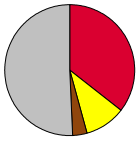
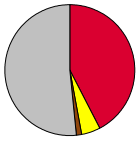


Figure 2-26. Distribution of estimated annual mean vs. 98th percentile 24-hour average $PM_{10-2.5}$ concentrations, 2001-2003. Box depicts interquartile range and median; whiskers depict 5th and 95th percentiles; asterisks depict minima and maxima. N= number of sites.

Source: Schmidt et al. (2005)

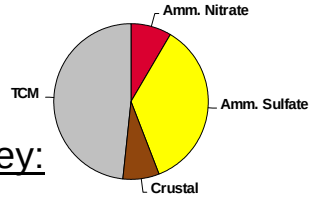
Table 2-5. $PM_{2.5}$ composition on high mass days in select urban areas, 2003

Urban Area	Statistic*	Composition Percents (%)				$PM_{2.5}$ mass** (µg/m3)	Annual average	Average of 5 highest days
		Amm. Nitrate	Amm. Sulfate	Crustal	TCM			
Birmingham, AL	• Annual average	8.5	35.6	7.6	48.3	**17.9**		
	• Average of 5 highest $PM_{2.5}$ mass days	3.8	40.0	7.8	48.3	**40.7**		
	• Highest $PM_{2.5}$ mass day	1.9	55.1	5.5	37.4	**46.6**		
	• 2nd highest $PM_{2.5}$ mass day	4.2	26.9	11.0	57.9	**40.4**		
	• 3rd highest $PM_{2.5}$ mass day	15.3	15.7	10.7	58.4	**39.2**		
	• 4th Highest $PM_{2.5}$ mass day	2.7	51.1	7.4	38.7	**39.1**		
	• 5th Highest $PM_{2.5}$ mass day	2.6	34.6	6.4	56.3	**38.3**		
Atlanta, GA	• Annual average	8.1	42.8	4.0	45.0	**15.2**		
	• Average of 5 highest $PM_{2.5}$ mass days	2.6	60.1	2.3	34.3	**35.2**		
	• Highest $PM_{2.5}$ mass day	2.0	70.5	1.9	25.6	**37.8**		
	• 2nd highest $PM_{2.5}$ mass day	2.0	47.8	2.5	47.8	**37.1**		
	• 3rd highest $PM_{2.5}$ mass day	2.4	67.6	2.1	27.9	**36.8**		
	• 4th Highest $PM_{2.5}$ mass day	3.2	50.8	2.9	43.1	**35.0**		
	• 5th Highest $PM_{2.5}$ mass day	3.6	67.5	1.9	27.0	**29.3**		
New York City, NY	• Annual average	20.2	38.3	5.1	36.4	**13.1**		
	• Average of 5 highest $PM_{2.5}$ mass days	11.6	57.9	3.0	27.4	**40.5**		
	• Highest $PM_{2.5}$ mass day	3.6	58.3	5.5	32.6	**45.9**		
	• 2nd highest $PM_{2.5}$ mass day	5.0	69.0	1.4	24.6	**45.8**		
	• 3rd highest $PM_{2.5}$ mass day	27.8	42.1	3.1	27.0	**38.2**		
	• 4th Highest $PM_{2.5}$ mass day	5.1	59.4	4.6	30.9	**36.4**		
	• 5th Highest $PM_{2.5}$ mass day	9.7	62.2	2.0	26.1	**36.0**		
Cleveland, OH	• Annual average	22.3	38.3	7.4	32.1	**17.6**		
	• Average of 5 highest $PM_{2.5}$ mass days	21.4	42.5	6.3	30.0	**44.1**		
	• Highest $PM_{2.5}$ mass day	32.7	43.2	2.3	21.7	**57.9**		
	• 2nd highest $PM_{2.5}$ mass day	25.1	41.5	4.0	29.3	**46.4**		
	• 3rd highest $PM_{2.5}$ mass day	4.8	64.4	8.7	22.1	**45.5**		
	• 4th Highest $PM_{2.5}$ mass day	8.8	37.5	14.7	39.0	**35.7**		
	• 5th Highest $PM_{2.5}$ mass day	31.4	20.5	4.0	44.0	**35.0**		
Chicago, IL	• Annual average	28.0	31.8	4.6	35.6	**15.2**		
	• Average of 5 highest $PM_{2.5}$ mass days	41.2	34.0	2.3	22.4	**34.4**		
	• Highest $PM_{2.5}$ mass day	46.0	30.7	1.2	22.1	**38.3**		
	• 2nd highest $PM_{2.5}$ mass day	49.2	36.4	0.8	13.6	**35.3**		
	• 3rd highest $PM_{2.5}$ mass day	51.8	27.7	1.2	19.3	**35.1**		
	• 4th Highest $PM_{2.5}$ mass day	5.6	61.7	3.8	28.9	**32.5**		
	• 5th Highest $PM_{2.5}$ mass day	47.8	16.1	5.3	30.8	**30.7**		
St. Louis, MO	• Annual average	20.0	36.0	5.6	38.4	**14.5**		
	• Average of 5 highest $PM_{2.5}$ mass days	12.2	61.9	3.9	22.0	**35.9**		
	• Highest $PM_{2.5}$ mass day	6.2	69.1	3.6	21.0	**50.6**		
	• 2nd highest $PM_{2.5}$ mass day	5.0	67.0	2.0	26.0	**36.0**		
	• 3rd highest $PM_{2.5}$ mass day	6.4	69.2	3.2	21.3	**33.1**		
	• 4th Highest $PM_{2.5}$ mass day	5.0	58.9	8.2	28.1	**30.8**		
	• 5th Highest $PM_{2.5}$ mass day	40.2	42.3	2.7	14.7	**28.9**		
Salt Lake City, UT	• Annual average	28.3	12.2	8.5	51.1	**10.0**		
	• Average of 5 highest $PM_{2.5}$ mass days	46.3	10.8	2.9	40.0	**40.6**		
	• Highest $PM_{2.5}$ mass day	50.6	6.3	2.5	40.5	**59.5**		
	• 2nd highest $PM_{2.5}$ mass day	43.5	11.9	2.6	42.0	**52.1**		
	• 3rd highest $PM_{2.5}$ mass day	42.4	13.5	3.7	40.4	**34.2**		
	• 4th Highest $PM_{2.5}$ mass day	48.2	5.9	4.7	41.3	**28.7**		
	• 5th Highest $PM_{2.5}$ mass day	45.4	20.2	1.5	32.8	**28.4**		
Fresno, CA	• Annual average	35.5	10.2	3.6	50.7	**18.0**		
	• Average of 5 highest $PM_{2.5}$ mass days	42.4	4.7	1.3	51.6	**54.2**		
	• Highest $PM_{2.5}$ mass day	55.2	4.6	2.1	38.2	**59.0**		
	• 2nd highest $PM_{2.5}$ mass day	58.4	8.5	0.9	32.2	**56.3**		
	• 3rd highest $PM_{2.5}$ mass day	17.5	1.5	1.3	79.7	**54.4**		
	• 4th Highest $PM_{2.5}$ mass day	35.1	5.3	1.0	58.6	**52.6**		
	• 5th Highest $PM_{2.5}$ mass day	44.6	3.7	1.3	50.3	**50.0**		

* The 5 highest days shown (and aggregated) for each site actually represent the 5 highest days (based on collocated FRM mass; see next bullet) that the speciation monitor sampled. FRM monitors at different locations in the metropolitan area and/or collocated FRM measurements on days that the speciation sampler did not record valid data may have had higher values than some or all of the 5 high values shown. Event-flagged data were omitted from this analyses.

** '$PM_{2.5}$ mass' concentration represents the collocated (w/ speciation monitor) same-day FRM measurement unless not available, in which case the speciation monitor gravimetric mass was substituted.

Key:



that event-flagged data were excluded from this analyses; the carbonaceous fraction of mass would be significantly higher on sites where peak days are affected by smoke from wildfires.

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 2-27 and 2-28 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 2-27 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 95^{th} 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 2-28 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 2-29 shows hourly average $PM_{2.5}$ and $PM_{10-2.5}$ concentrations for a monitoring site in the Denver metropolitan area from 2001-2003. Like many western U.S. sites, the $PM_{10-2.5}$ concentrations are higher than the $PM_{2.5}$ levels for all hours of the day. Similar to the eastern example site, this western one also shows a morning and afternoon rush hour traffic signal. Some western monitoring sites, located in areas subject to routine episodes of windblown dust, can have unusual diurnal concentration distributions (e.g., 95^{th} percentile concentrations for some hours more than ten times the median levels; and hourly means significantly higher than the medians and even 75^{th} percentiles) (Schmidt et al., 2005). Figure 2-30 highlights how continuous data can be used to pinpoint an unusual or episodic source, in this case a short but significant dust storm in El Paso, Texas. On April 26, 2002, this dust storm caused large increases in both $PM_{2.5}$ and $PM_{10-2.5}$ concentrations. As might be expected, the dust 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 6 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.

The hourly ranges shown in Figures 2-27 and 2-29 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 $\mu g/m^3$.

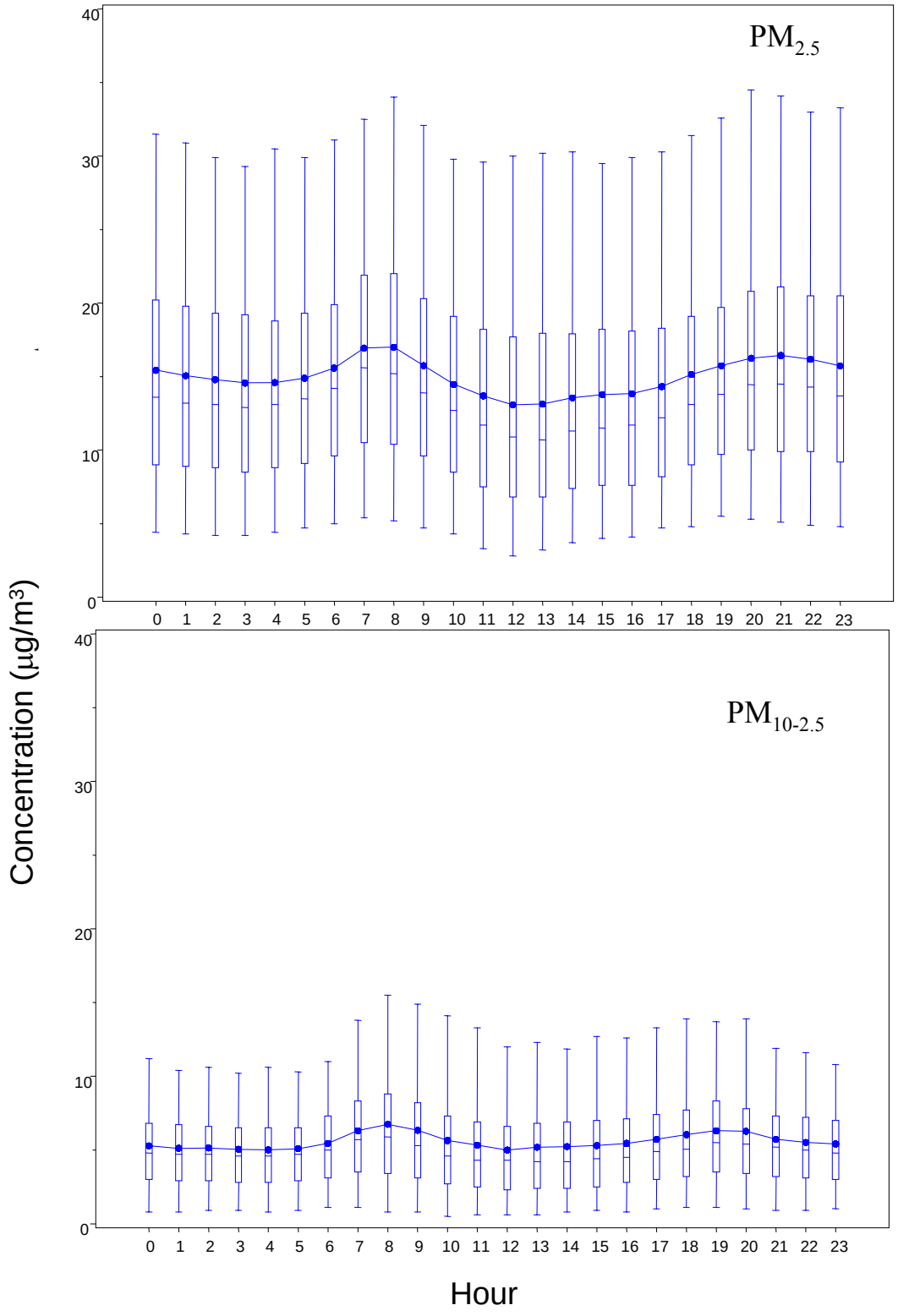


Figure 2-27. Hourly average $PM_{2.5}$ and $PM_{10-2.5}$ concentrations at a Greensboro, NC monitoring site, 2001-2003. Upper panel shows the distribution of $PM_{2.5}$ concentrations and the lower panel shows the distribution of $PM_{10-2.5}$ concentrations. (Box plots of interquartile ranges, means,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

Source: Schmidt et al. (2005)

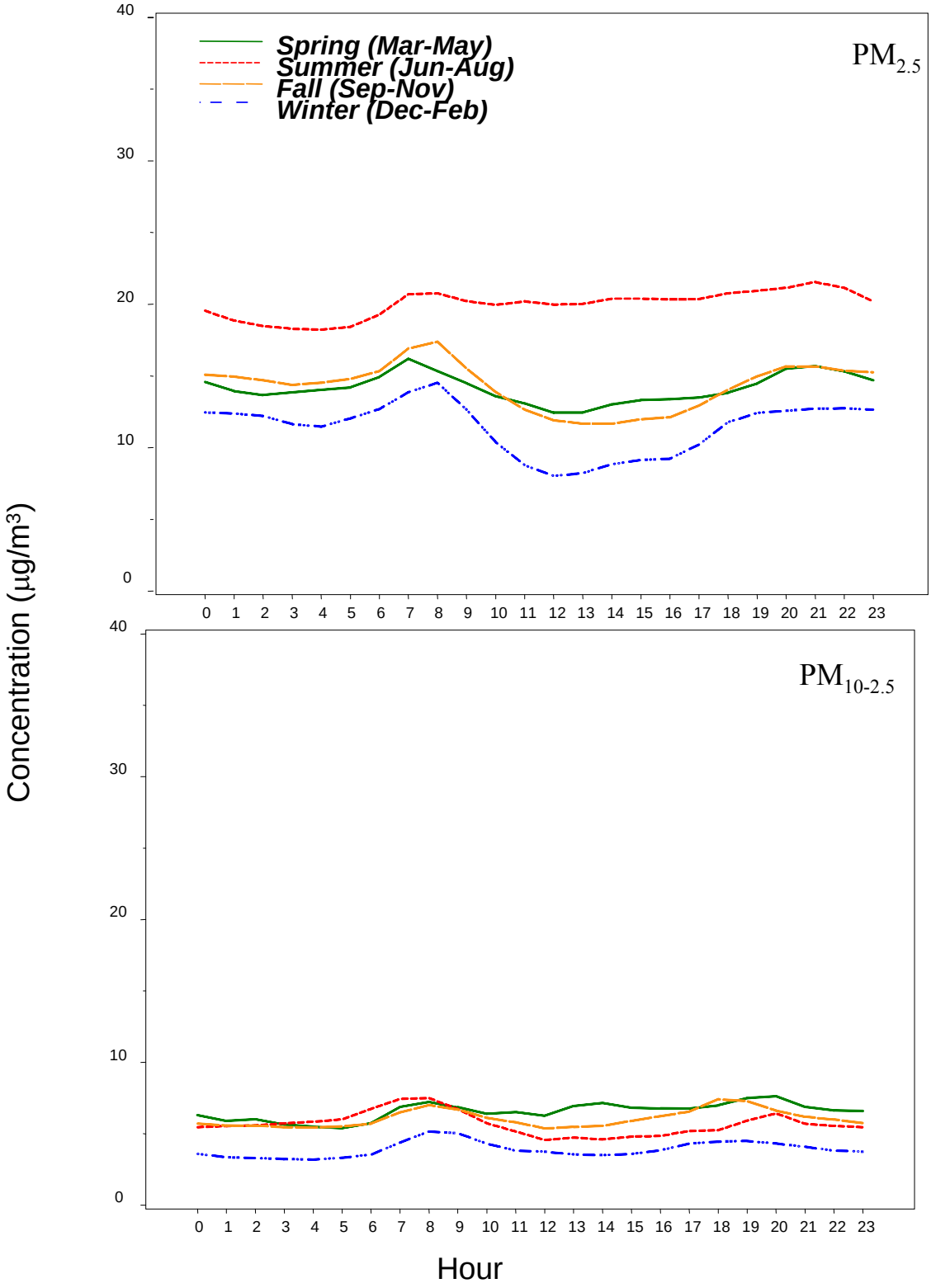


Figure 2-28. Seasonal hourly average $PM_{2.5}$ and $PM_{10-2.5}$ concentrations at a Greensboro, NC monitoring site, 2001-2003. Upper panel shows the $PM_{2.5}$ concentrations and the lower panel shows the $PM_{10-2.5}$ concentrations.

Source: Schmidt et al. (2005)

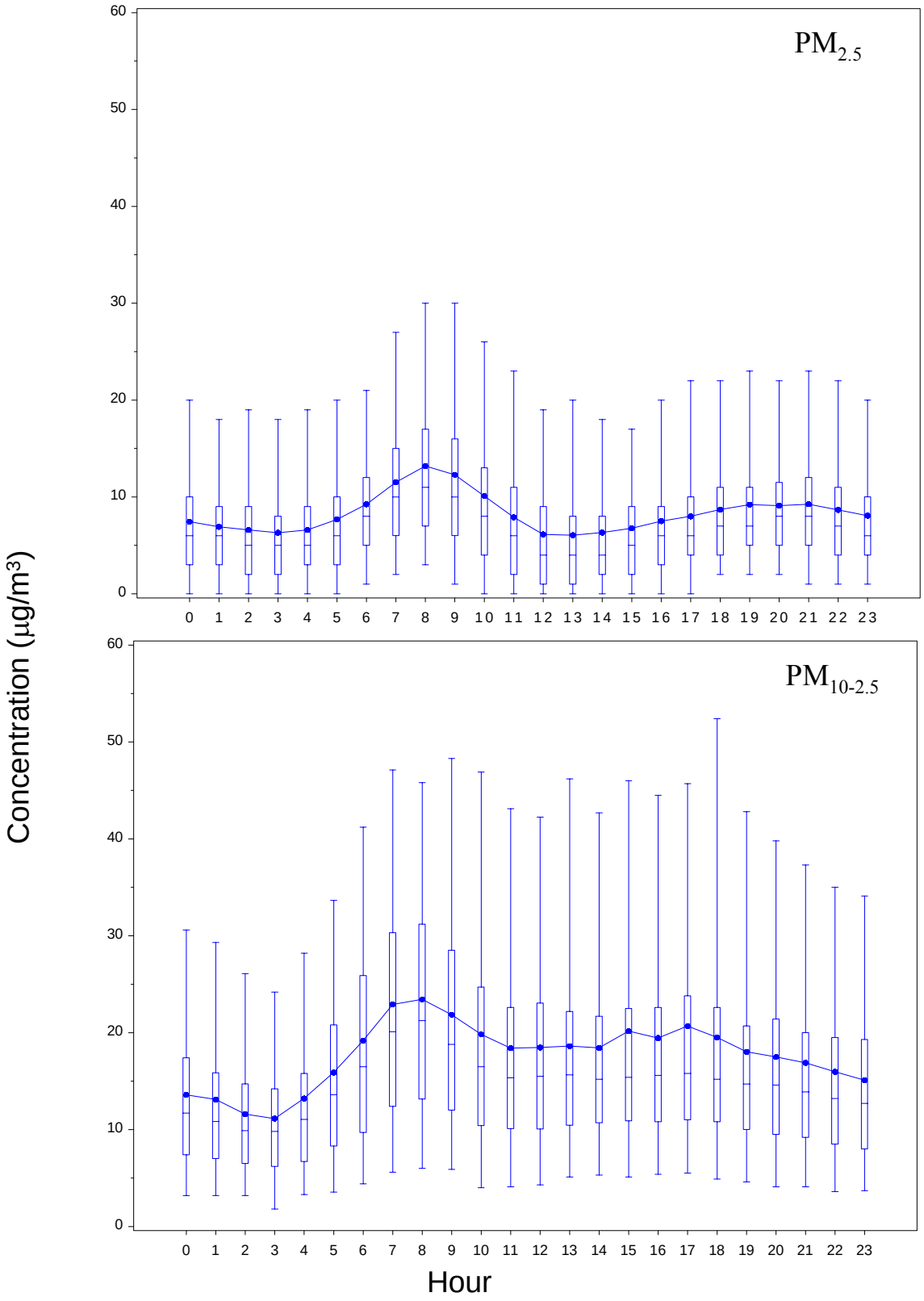


Figure 2-29. Hourly average $PM_{2.5}$ and $PM_{10-2.5}$ concentrations at a Denver, CO monitoring site, 2001-2003. Upper panel shows the distribution of $PM_{2.5}$ concentrations and the lower panel shows the distribution of $PM_{10-2.5}$ concentrations. (Box plots of interquartile ranges, means, medians, 5th and 95th percentiles.)

Source: Schmidt et al. (2005)

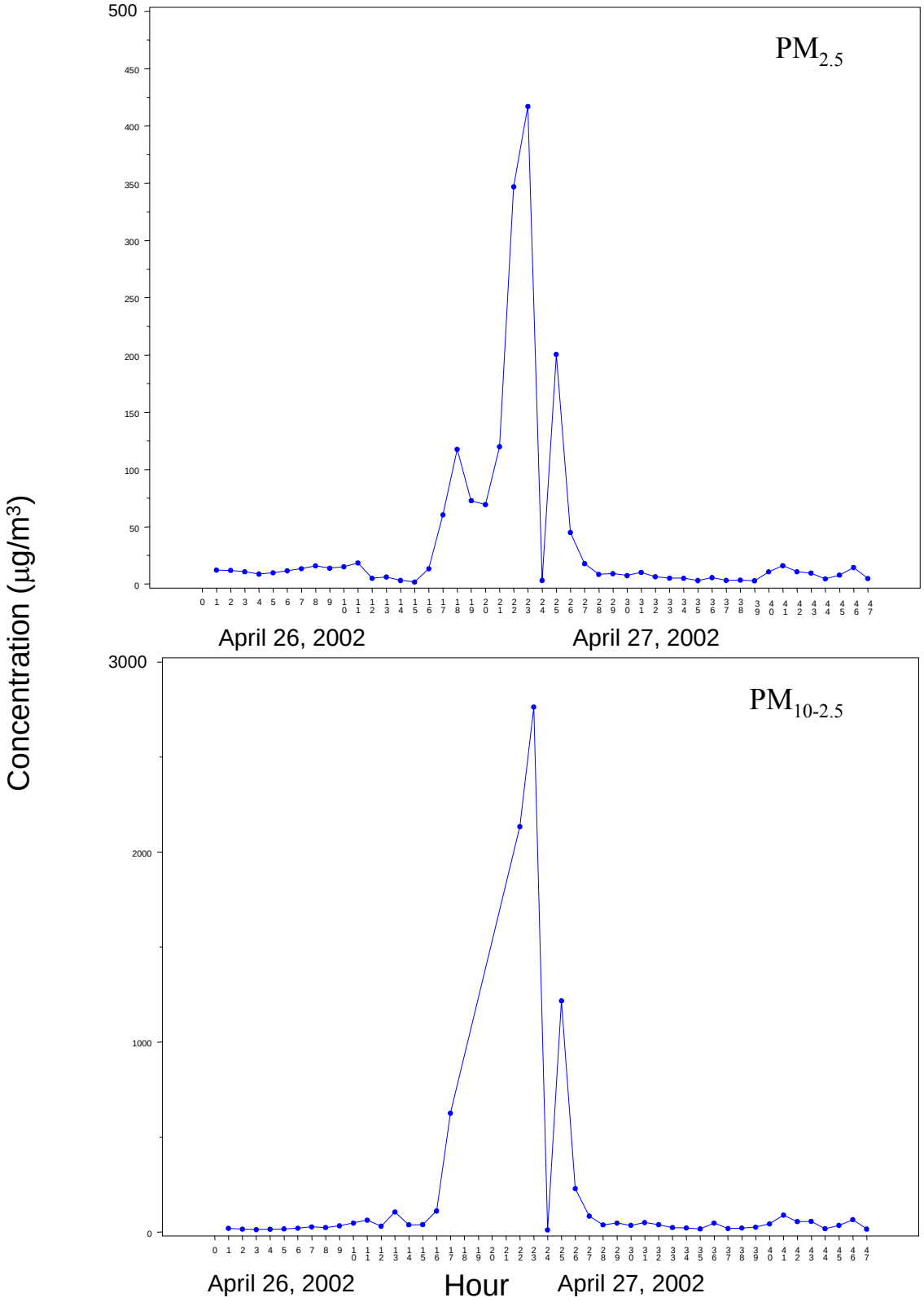


Figure 2-30. Hourly $PM_{2.5}$ and $PM_{10-2.5}$ concentrations at a El Paso, TX monitoring site, April 26, 2002-April 27, 2002. Upper panel shows the hourly $PM_{2.5}$ concentrations and the lower panel shows the hourly $PM_{10-2.5}$ concentrations. Note the different scales.

Source: Schmidt et al. (2005)

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, background 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. Background levels so defined are referred to policy-relevant background, since this definition of background facilitates separating pollution levels that can be controlled by U.S. regulations (or through international agreements with neighboring countries) from levels that are generally uncontrollable by the U.S.. 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). Annual average background estimates for PM_{10} range from 4 to 8 $\mu g/m^3$ in the western U.S. and 5 to 11 $\mu g/m^3$ in the eastern U.S.; for $PM_{2.5}$, estimates range from 1 to 4 $\mu g/m^3$ in the west and 2 to 5 $\mu g/m^3$ in the east. 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). Ranges presented in the CD for background $PM_{10-2.5}$ levels were derived from the PM_{10} and $PM_{2.5}$ ranges by subtraction, resulting in relatively wide ranges with mid-point estimates of 3.5 $\mu g/m^3$ in the west and 4.5 $\mu g/m^3$ in the east (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.

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 trans-national 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.[26]

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. Estimates of background concentrations for time scales shorter than daily averages are not feasible at this time, since almost all of the rural measurements of speciated PM are 24-hour averages. 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 is comprised of 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

[26] 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 PM_{10} 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.

southern California (south of about 40 degrees latitude) makes up the "south west coast" regions.[27]

To arrive at estimates of background we use the averaged non-sulfate $PM_{2.5}$ values[28] 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 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	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

[27] 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.

[28] Non-sulfate $PM_{2.5}$ is defined as measured $PM_{2.5}$ minus reported ammonuim sulfate.

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 exposure to ambient PM – 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 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.[29] The focus here is on particle size distinctions; the CD (CD, Section 5.4) also discusses exposure relationships related to compositional differences.

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

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

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).

An individual's total personal exposure to PM may differ from the ambient concentration measured at the central site monitor because: (1) spatial differences in ambient PM concentrations exist across a city or region; (2) generally only a fraction of the ambient PM is present in indoor or in-vehicle environments, whereas individuals generally spend a large percentage of time indoors; and (3) a variety of indoor sources of PM contribute to total personal exposure. Thus, the amount of time spent outdoors, indoors, and in vehicles and the types of activities engaged in (e.g., smoking, cooking, vacuuming) also will heavily influence personal

exposure to PM. The first two factors are important for determining the strength of the relationship between ambient PM and ambient personal exposure.

With regard to the first factor that influences the relationship between total personal exposure and concentrations measured at central sites, the spatial variability of PM plays a large role. As discussed in Section 2.4, for many areas $PM_{2.5}$ concentrations are fairly uniform spatially, with higher concentrations near roadways and other direct sources of $PM_{2.5}$. Analyses of $PM_{2.5}$ data for 27 urban areas indicate that differences in annual mean concentrations between monitoring sites in an urban area range from less than 1 $\mu g/m^3$ to as much as 8 $\mu g/m^3$. However, the correlations of daily $PM_{2.5}$ between sites are typically greater than 0.80. Daily mean $PM_{2.5}$ concentrations exhibit much higher spatial variability than annual means, even when the daily concentrations at sites are highly correlated. Although the spatial variability of $PM_{2.5}$ varies for different urban areas, overall, some degree of uniformity results from the widespread formation and long lifetime of the high regional background of secondary $PM_{2.5}$. In summarizing the key findings related to spatial variability in $PM_{2.5}$ concentrations, the CD states (p. 3-101):

> Differences in annual mean $PM_{2.5}$ concentrations between monitoring sites in urban areas examined are typically less than 6 or 7 $\mu g/m^3$. However, on individual days, differences in 24-h average $PM_{2.5}$ concentrations can be much larger. Some sites in metropolitan areas are highly correlated with each other but not with others, due to the presence of local sources, topographic barriers, etc. Although $PM_{2.5}$ concentrations at sites within a MSA can be highly correlated, significant differences in their concentrations can occur on any given day. Consequently, additional measures should be used to characterize the spatial variability of $PM_{2.5}$ concentrations. The degree of spatial uniformity in $PM_{2.5}$ concentrations in urban areas varies across the country. These factors should be considered in using data obtained by the $PM_{2.5}$ FRM network to estimate community-scale human exposure, and caution should be exercised in extrapolating conclusions obtained in one urban area to another. $PM_{2.5}$ to PM_{10} ratios were generally higher in the east than in the west, and values for this ratio are consistent with those found in numerous earlier studies presented in the 1996 PM AQCD.

Relative to fine particles, coarse and ultrafine particles are likely to be more variable across urban scales. Daily mean $PM_{10-2.5}$ concentrations tend to be more variable and have lower inter-site correlations than $PM_{2.5}$, possibly due to their shorter atmospheric lifetime (travel distances < 1 to 10s of km) and the more sporadic nature of $PM_{10-2.5}$ sources (CD, Section 3.2.5). Ultrafine particles also have shorter atmospheric lifetimes (travel distances < 1 to 10s of km, compared with 100s to 1000s of km for $PM_{2.5}$) and spatially variable sources. High concentrations of ultrafine particles have been measured near roadways, but with concentrations falling off rapidly with increasing distance from the roadway. Both coarse and ultrafine particles also have reduced concentrations indoors compared to $PM_{2.5}$, due to lower infiltration rates,

greater deposition rates, and coagulation of ultrafine particles into larger particles. These differences make it more difficult to find a relationship between ambient concentrations and personal exposures to these size fractions than for $PM_{2.5}$.

The second factor influencing the relationship between ambient PM concentrations measured at central sites and total personal exposure to PM is the extent to which ambient PM penetrates indoors and remains suspended in the air. If the flow of ambient PM into the home from the outdoors is very restricted, the relationship between ambient PM concentrations measured at a central site and total exposure to PM will tend to be weaker than in a situation where ambient PM flows more readily into the home and is a greater part of the overall indoor PM concentrations. This is heavily dependent on the building air exchange rate, and also on penetration efficiency and deposition or removal rate, both of which vary with particle aerodynamic size. Air exchange rates (the rates at which the indoor air in a building is replaced by outdoor air) are influenced by building structure, the use of air conditioning and heating, opening and closing of doors and windows, and meteorological factors (e.g., difference in temperature between indoors and outdoors). Based on physical mass-balance considerations, usually the higher the air exchange rate the greater the fraction of PM of ambient origin found in the indoor and in-vehicle environments. Higher air exchange rates also dilute the concentration of indoor- generated PM. Rates of infiltration of outdoor PM into homes through cracks and crevices are higher for $PM_{2.5}$ than for PM_{10}, $PM_{10-2.5}$, or ultrafine particles (CD, p. 5-123). Since $PM_{10-2.5}$ 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). They will more likely affect the magnitude of the effects found from these studies and result in higher effects estimates, since exposure measurement errors tend to bias towards the null hypothesis.

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[30] 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.

[30] 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.

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.

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):

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

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 estimated from ambient pollutant concentrations. Transmissometers measure the light transmitted through the atmosphere over a distance of 1 to 15 kilometers. The light transmitted between the light source (transmitter) and the light-monitoring component (receiver) is converted to the path-averaged light extinction coefficient. Transmissometers operate continuously, and data 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.

EPA guidance for tracking progress under the regional haze rule specifies 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):

$$
\begin{aligned}
b_{ext} = \; & (3)f(RH)\,[SULFATE] \\
& + (3)f(RH)\,[NITRATE] \\
& + (4)\,[ORGANIC\ CARBON] \\
& + (10)\,[LIGHT\ ABSORBING\ CARBON] \\
& + (1)\,[SOIL] \\
& + (0.6)\,[COARSE\ PM] \\
& + 10\ (for\ Rayleigh\ scattering\ by\ gases)
\end{aligned}
\qquad (5\text{-}2)
$$

The estimated 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 ignoring the Earth's curvature. 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):

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

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:

$$\text{Haziness (dv)} = 10 \ln(b_{ext}/10\ \text{Mm}^{-1}) \quad (5\text{-}4)$$

Figure 2-31 graphically illustrates the relationships among light extinction, visual range, and deciview.

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 2-65 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 $\pm$ 7 Mm^{-1} in the east, and 17 $\pm$ 2.5 Mm^{-1} in the west. These equate to a naturally-occurring visual range in the east of 150 $\pm$ 45 km, and 230 $\pm$ 35 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

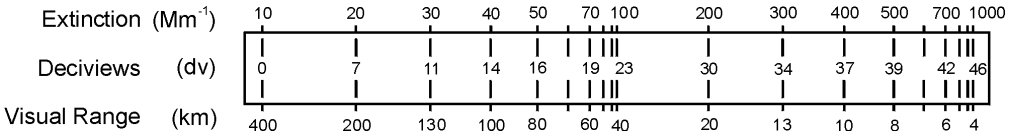


Figure 2-31. Relationship between light extinction, deciviews, and visual range.

Source: Malm (1999)

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 the evidence-based assessment of primary standards for PM presented 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 the quantitative health risk assessment discussed in Chapter 4 and used in the risk-based assessment of primary standards for PM presented in Chapter 5.

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 biologic 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 distinctions between fine and coarse particles, and submodes within fine particles, with regard to deposition patterns in the respiratory tract, though there is significant overlap between particle size classes (CD, p. 9-21 to 9-22). 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, drawing from the CD's evaluation and conclusions on the full 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 and integrated 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 elicited 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 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 dosimetric 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 various indicators 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 associations. These studies provide more precise estimates of the magnitude of an effect of exposure to PM 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 ambient PM.

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

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

- Several "intervention studies" have reported improvements in health in areas where policy, economic or regulatory changes resulted in reduced air pollutant concentrations (section 8.2.3.4 in the CD).

In addition, the body of evidence on PM-related effects has greatly expanded with findings from studies that help inform mechanisms of action, including important new dosimetric, 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 and cytokine levels, heart rate variability), and animal models simulating sensitive subpopulations, that are relevant to demonstrating plausibility of the epidemiologic evidence and provide insights into potential mechanisms for PM-related effects.

- Dosimetric 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

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

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) measurement error and 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 related to exposure time periods used 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 the PM risk assessment discussed in Chapter 4.

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 dosimetric studies has played a key role in previous PM NAAQS reviews, especially in the decision to revise the indicator from total suspended particulates (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 biologic 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 (CD, p. 6-1). Briefly, the human respiratory tract can be divided into three main regions: (1) extrathoracic, (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 health concerns related to ambient particles are greater for the two lower regions.

Fine particles, including accumulation mode and ultrafine particles, and thoracic coarse particles can all penetrate into and be deposited in the tracheobronchial and alveolar regions of the respiratory tract, though (as noted above) there are differences among these size fractions. Penetration into the tracheobronchial and alveolar regions is greater for accumulation mode particles than coarse or ultrafine particles, since coarse and ultrafine particles are more

efficiently removed from the air in the extrathoracic region than are accumulation-mode fine particles (CD, 6-105).

Once past the extrathoracic region, deposition fraction in the tracheobronchial and alveolar regions varies with different exertion levels or breathing patterns, and whether breathing is through the mouth or nose. As shown in Figures 6-16 and 6-17 of the CD, deposition fractions in these regions are largest for particles in the coarse fraction and ultrafine modes. More specifically, the CD concludes that fractional deposition in 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).

Respiratory disease status can also affect regional particle deposition patterns. Studies have indicated that, in general, enhanced deposition of particles occurs at airway bifurcations (CD, p. 6-20). New evidence confirms that people with chronic obstructive lung diseases 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 overall as well as increased doses to localized 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 rate 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 occur in the tracheobronchial region, but the primary mechanisms for particle clearance or translocation from the tracheobronchial region 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 also suggest that ultrafine particles or their soluble constituents may move directly from the lungs into the systemic circulation, providing a pathway by which ambient PM exposure could 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 three size fractions 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 biologic mechanisms by which deposited particles could result in the varying effects observed in epidemiologic 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 biologic 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 attained. Some of the more important findings presented in chapter 7 of the CD, 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 physiological 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 potential general pathways for effects on the heart and lung. 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 "[f]indings 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 inhalation or intratracheal instillation of ambient particles from various locations have shown that ambient particles can cause lung inflammation and injury (CD, Tables 7-4 and 7-5). 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 lactate dehydrogenase (CD, p 7-46, 7-47). Administration of residual oil fly ash 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 cardiac effects, 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 related to 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 or their soluble constituents 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 and/or general pathways for effects 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 some new studies have exposed 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 fine 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 complex particle mixtures appear to be responsible for many biologic responses relevant to fine particle exposures (CD, p. 7-206).

In addition to the evidence discussed above that related primarily to fine particles, there is some limited evidence from toxicologic studies on $PM_{10-2.5}$, for either acute or chronic exposures (CD, p. 9-55). The CD includes results from several *in vitro* toxicologic studies that provide some insight into potential effects of thoracic coarse particles, particularly related to inflammatory or allergic effects. Two recent studies report inflammatory responses in cells exposed to extracts of water-soluble and water-insoluble materials from thoracic coarse particles and fine particles collected in Chapel Hill, NC (CD, p. 7-83, Monn and Becker, 1999; and CD, p. 7-101 and 7-102, Soukup and Becker, 2001). One study focused on water-soluble materials, and reported significant cytotoxicity and cytokine production with water-soluble extracts of ambient $PM_{10-2.5}$, in contrast to the lack of effects observed with extracts from ambient $PM_{2.5}$ as well as indoor-collected $PM_{10-2.5}$ and $PM_{2.5}$. The authors report that endotoxin appeared to have a role in inflammatory effects, while metals appeared to have a role in the cytoxocity of thoracic coarse particle materials (CD, p. 7-83, Monn and Becker, 1999). Soukup and Becker (2001) used both soluble and insoluble components of thoracic coarse particles and fine particles, and report that the insoluble materials from thoracic coarse particles resulted in cytokine production, decreased phagocytic ability and oxidant generation (CD, p. 7-101 and 7-102). In this extract of thoracic

coarse particles, endotoxin appeared to be the most pro-inflammatory component, but "other moieties" (not endotoxin or metals) appeared to contribute to oxidant generation (CD, p. 7-102). Using particles collected in two urban areas in the Netherlands, Becker et al. (2003) reported that thoracic coarse particles, but not fine or ultrafine particles, resulted in effects related to inflammation and decreased pulmonary defenses (CD, p. 7-106). This small group of studies thus suggests that exposure to thoracic coarse particles may cause pro-inflammatory effects, as well as cytotoxicity and oxidant generation.

In addition, Diociaiuti et al. (2001) reported greater hemolytic effects with fine particles than with thoracic coarse particles when exposing blood cell cultures to extracts of particles collected in an urban area of Rome; increased hemolysis was seen with only the highest $PM_{10-2.5}$ dose (CD, p. 7-102). Hornberg et al. (1998) reported evidence of genotoxic activity in human bronchoepithelial cells exposed to both $PM_{2.5}$ and $PM_{10-2.5}$, with stronger evidence for genotoxicity in fine particles (CD, p. 7-171). These two studies suggest only limited hemolytic or carcinogenic effects of thoracic coarse particle exposures.

Road dust is a common source of thoracic coarse particles and can be considered as a PM sample that is more representative of thoracic coarse particles than fine particles. In the 1996 Staff Paper, results from one key toxicologic study were highlighted in which immunological and cellular toxicity was observed in rats with exposure to road dust. Higher concentrations of road dust were needed to cause effects, compared with exposures to fine particle components (e.g., sulfates, nitrates), but it was observed that some of the apparent differential toxicity was due to differential penetration efficiencies of particles in the rat (EPA, 1996b, p. V-70). A recent study reported that road tunnel dust particles had greater adjuvant activity in two animal models of allergy than several other particle samples, including residual oil fly ash and diesel exhaust particles (Steerenberg et al., 2003; CD, p. 7-136 to 7-137). In contrast, a number of studies have reported that Mt. St. Helens volcanic ash, which is generally in the size range of thoracic coarse particles, has very little toxicity in animal or *in vitro* toxicologic studies (CD, p. 7-216).

Many of the newer studies use relatively 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 in animals with high dose exposures 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, dosimetric 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, even when humans and rats are similarly exposed (i.e., exposed at rest to the same aerosol for 6 hours) the equivalent exposure ratios can range from 0.09 to 33 (CD, p. 7A-30, Table 7A-7a). 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). Based on these data, deposited and retained doses in the alveolar and tracheobronchial regions were estimated for three studies using concentrated ambient particles, and doses were 40- to 67-fold higher in rats than in humans from these inhalation exposure studies (CD, pp. 7A-61). However, the CD observed that similar and/or lesser inflammatory responses were reported for rats than for humans, suggesting that rats may be less susceptible to effects of concentrated ambient particles than healthy humans (CD, p. 7A-61). 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).

The CD also observes that particles may help carry other airborne substances into the respiratory tract (CD, section 7.9). For example, hygroscopic 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).

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 for ambient PM, primarily for fine PM. In evaluating this new body of evidence, the CD states: "Thus, there appear to be multiple biologic mechanisms that may be responsible for observed morbidity/mortality due to exposure to ambient PM. It also appears that many biologic responses are produced by PM whether it is composed of a single component or a complex mixture" (CD, p. 7-206).

3.3 NATURE OF EFFECTS

An extensive body of new epidemiologic studies has been published since completion of the 1996 PM CD. In the last review, epidemiologic evidence indicated that exposure to PM (using various indicators) was associated with increased risk for various cardiopulmonary effects, including mortality and a range of indices of morbidity associated with respiratory and cardiovascular disease such as hospital admissions and emergency room visits, school absences, work loss days, restricted activity days, effects on lung function and symptoms, morphological changes, and altered host defense mechanisms. The CD finds that recent epidemiologic studies have continued to report associations between various indicators of ambient PM and effects such

as premature mortality, hospital admissions or emergency department visits for respiratory and cardiovascular disease, and effects on lung function and symptoms (CD, p. 9-23). In addition, recent studies now identify several new types of health outcomes reported to be associated with exposure to various indicators of PM, including physicians' office or clinic visits, cardiovascular health indicators such as heart rate variability or increased C-reactive protein levels, and developmental effects such as low birth weight, and infant mortality (CD, p. 9-23, 9-24).

The discussions that follow draw primarily from epidemiologic evidence evaluated in Chapter 8 of the CD as well as the CD's integration of evidence across disciplines (section 9.2). The CD evaluates evidence from the full body of epidemiologic studies conducted world-wide and summarizes results of all such mortality and morbidity studies in Appendices 8A and 8B, respectively, in the CD. For the purposes of this Staff Paper, staff draws from the CD's qualitative evaluation of all studies, but focuses on those conducted in the U.S. and Canada for quantitative assessments.[2] Effect estimates for mortality and morbidity effects associated with increments of PM_{10}, $PM_{2.5}$, and $PM_{10-2.5}$ from multi-city and single-city U.S. and Canadian studies are summarized in Appendices 3A and 3B to this chapter for short-term and long-term exposure studies, respectively, as a consolidated reference for the following discussions.[3]

A number of the new time-series epidemiologic studies have used generalized additive models (GAM) in their analyses, and issues have been found with the convergence criteria and the method for determining standard errors when using GAM, as discussed in section 3.6.3 more fully and in section 8.4.2 of the CD. In Appendix 3A, results are presented from those short-term exposure studies that have been reanalyzed to address issues related to GAM, or that did not use GAM in their analyses. In presenting study results in figures in this section, for studies in which multiple reanalysis results were presented, staff has selected effect estimates based on the authors' stated judgments, where offered, or selected results from models using generalized linear models (GLM).[4]

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[2] Findings of U.S. and Canadian studies are more directly applicable for quantitative considerations in this review, since studies conducted in other countries may well reflect quite different population and air pollution characteristics.

[3] For consistency across studies, the effect estimates summarized in Appendices 3A and 3B, and the results presented in figures in this section, are from single-pollutant models. Results of multi-pollutant models are discussed in the text. As presented in the CD, effect estimates are presented using standardized PM increments to allow for comparison across studies. For short-term exposures studies, increments of 50 $\mu g/m^3$ for PM_{10} and 25 $\mu g/m^3$ for $PM_{2.5}$ and $PM_{10-2.5}$ were used; for long-term exposures studies, increments of 20 $\mu g/m^3$ for PM_{10} and 10 $\mu g/m^3$ for $PM_{2.5}$ and $PM_{10-2.5}$ were used (CD, p. 8-4).

[4] For studies that include results for GLM analyses using several methods to adjust for temporal or weather variables, if no judgment is offered by the authors on model selection, staff has presented results from the models using adjustment methods most closely matching those of the initial study.

3.3.1 Premature Mortality

This section includes an overview of the CD's findings on (1) mortality associations with short-term PM exposure, with emphasis on results from newly available multi-city analyses; and (2) mortality associations with long-term PM exposure.

3.3.1.1 Mortality and Short-term PM Exposure

Historical reports of dramatic pollution episodes have provided clear evidence of mortality associated with high levels of PM and other pollutants, as summarized in the 1996 CD (EPA, 1996a, pp. 12-28 to 12-31). More recently, associations between increased daily mortality and various indicators of PM have been reported at much lower concentrations in a large number of areas with differing climates, PM composition, and levels of gaseous co-pollutants. Since the last review, a large number of new time-series studies of the relationship between short-term exposure to various indicators of PM and mortality have been published, including several multi-city studies that are responsive to the recommendations from the last review (CD, p. 8-24). Included in the PM CD are results from numerous studies that 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. Staff have focused on the results of studies conducted in the U.S. and Canada in this assessment; 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]

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

[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.

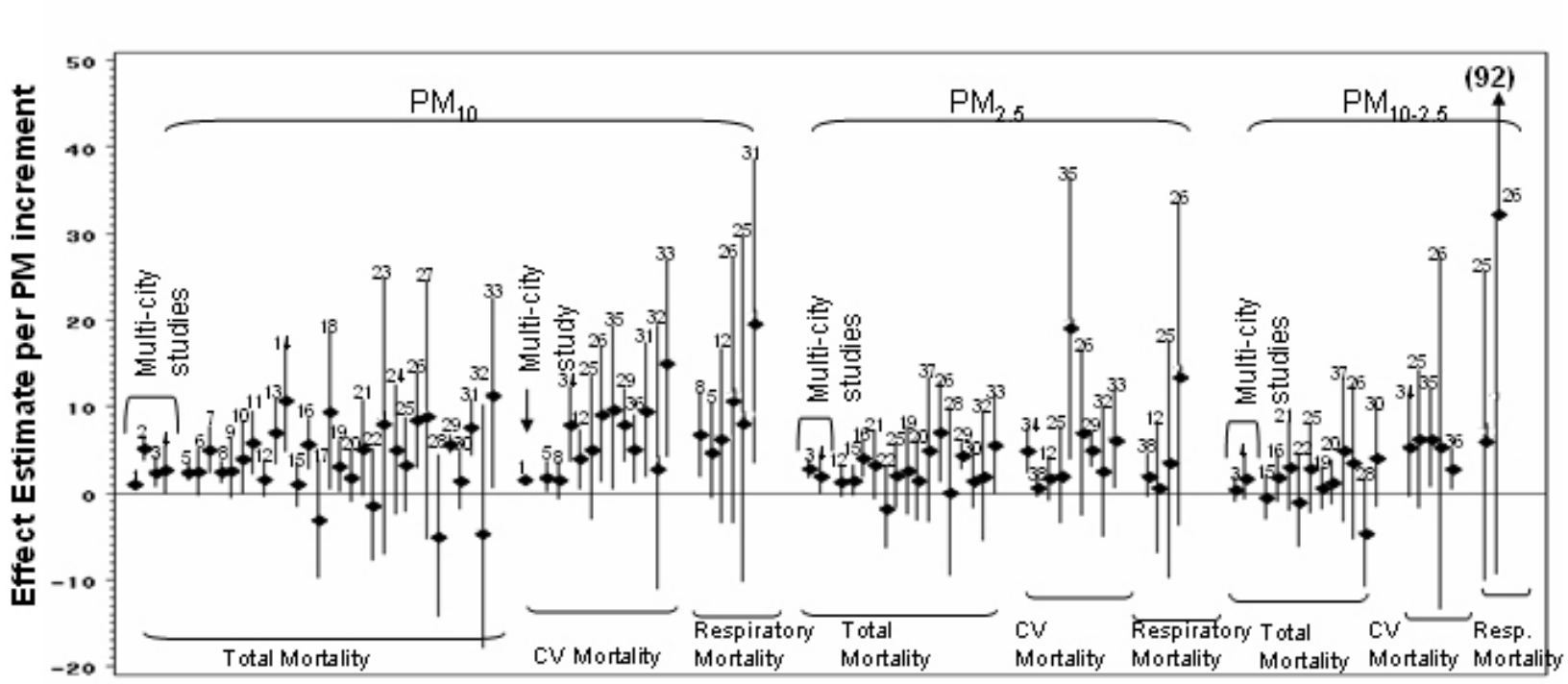

Figure 3-1. Excess risk estimates for total nonaccidental, cardiovascular, and respiratory mortality in multi-pollutant (in bold font below) and single-pollutant models for U.S. and Canadian studies. PM increments: 50 µg/m^3 for PM_{10} and 25 µg/m^3 for $PM_{2.5}$ and $PM_{10-2.5}$. Results presented from time-series studies that did not use GAM or were reanalyzed using GLM.

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

was designed to use a multi-city approach such as that recommended following an earlier report of time-series study reanalyses that recommended investigating the role of co-pollutants in PM-health outcome relationships by conducting multi-city studies, using consistent analytical approaches across cities (HEI, 1997, p. 38; Samet et al., 2000c, p. 1). The NMMAPS used a uniform methodology 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} (Dominici et al., 2003a; CD, p. 8-33). Key components of 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 that were selected from NMMAPS cities where daily 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_{10}, $PM_{2.5}$, and $PM_{10-2.5}$ 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 only significantly associated with mortality in one of the six cities (Steubenville) (Schwartz, 2003a; Klemm and Mason, 2003; CD, p. 8-40 to 8-41).

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 resulted in reduced effect estimate size, though the authors conclude that their findings are robust to the application of alternative modeling strategies (Katsouyanni et al., 2003; CD, p. 8-47). Taken together, 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} (CD, p. 8-50).

In considering the results from single-city analyses, Figure 3-1 shows that almost all effect estimates for $PM_{2.5}$ 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 $\mu g/m^3$ $PM_{2.5}$. For the relatively more precise single-city studies, effect estimates range from approximately 2 to 6% per 25 $\mu g/m^3$ $PM_{2.5}$ (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) (Dominici, et al., 2003a; 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 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, black smoke (BS) or coefficient of haze (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. Three recent studies have used $PM_{2.5}$ speciation data to evaluate the effects of air pollutant combinations or mixtures using factor analysis or source apportionment methods to link effects with different $PM_{2.5}$ source types. These studies reported that fine particles from combustion sources, including motor vehicle emissions, coal combustion, oil burning and vegetative burning, were associated with increased mortality. No significant increase in mortality was reported with a source factor representing crustal material in fine particles (CD, p. 8-85). These studies indicate that exposure fine particles from combustion sources, but not crustal material, is associated with mortality.

The findings of these studies, while providing some insight into what sources of fine particles might be associated with mortality, are not directly relevant to evaluating effects of thoracic coarse particles from different sources. Combustion sources are a major contributor to $PM_{2.5}$ emissions, but not $PM_{10-2.5}$, while crustal material is an important component of $PM_{10-2.5}$ but only a small portion of $PM_{2.5}$. Staff observes that no epidemiologic evidence is available to evaluate effects of different components or sources of thoracic coarse particles. One study that does have some relevance to considering the effects of $PM_{10-2.5}$ from different sources assessed the contribution of dust storms to PM_{10}-related mortality. The authors focused on days when dust storms or high wind events occurred, during which thoracic coarse particles are the dominant fraction of PM_{10}, in Spokane. No evidence was reported of increased mortality on days with high PM_{10} levels related to dust storms (average PM_{10} level was 221 μg/m^3 higher on

dust storm days than on other study days) (Schwartz, et al., 1999), suggesting that $PM_{10-2.5}$ from wind-blown dust is also not likely associated with mortality.[6]

3.3.1.2 Mortality and Long-term PM Exposure

In the 1996 PM CD, results were presented for three 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. Further reanalyses of the ACS study 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. While recognizing that increased mortality may be attributable to more than one component of ambient air pollution, the authors report that 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

[6]In addition, studies conducted in several areas in the western U.S. have reported that associations between PM_{10} and mortality or morbidity remained unchanged or became larger and more precise when days indicative of wind-blown dust or high PM_{10} concentration days were excluded from the analyses (Pope et al., 1999; Schwartz, 1997; Chen et al., 2000; Hefflin et al., 1994). This group of studies does not provide conclusive evidence, however, of any effects or lack of effects associated with wind-blown dust or high concentration days, but does indicate that associations between PM_{10} and health outcomes in these western areas are not overly influenced or "driven by" such days.

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 associations were stronger for those with lower education levels (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 (CD, p. 8-105; Krewski et al., 2000; Pope et al., 2002).

There are also new analyses using updated data from the AHSMOG cohort. These include 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 (CD, pp. 8-105, 8-110; 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, p. 8-106). Additional analyses were conducted using data from males only 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, pp. 8-110 and 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 (CD, pp. 8-110 to 8-111; 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 and 8-118; 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, positive associations were reported between long-term PM_{10} exposure lung cancer mortality that were statistically significant for males, but not females (CD, p. 8-108 and 8-109).

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 $PM_{2.5}$. Based on these studies, 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 et 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 find no associations between long-term exposure to thoracic coarse particles and 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_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ 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 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.

The NMMAPS multi-city analysis 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

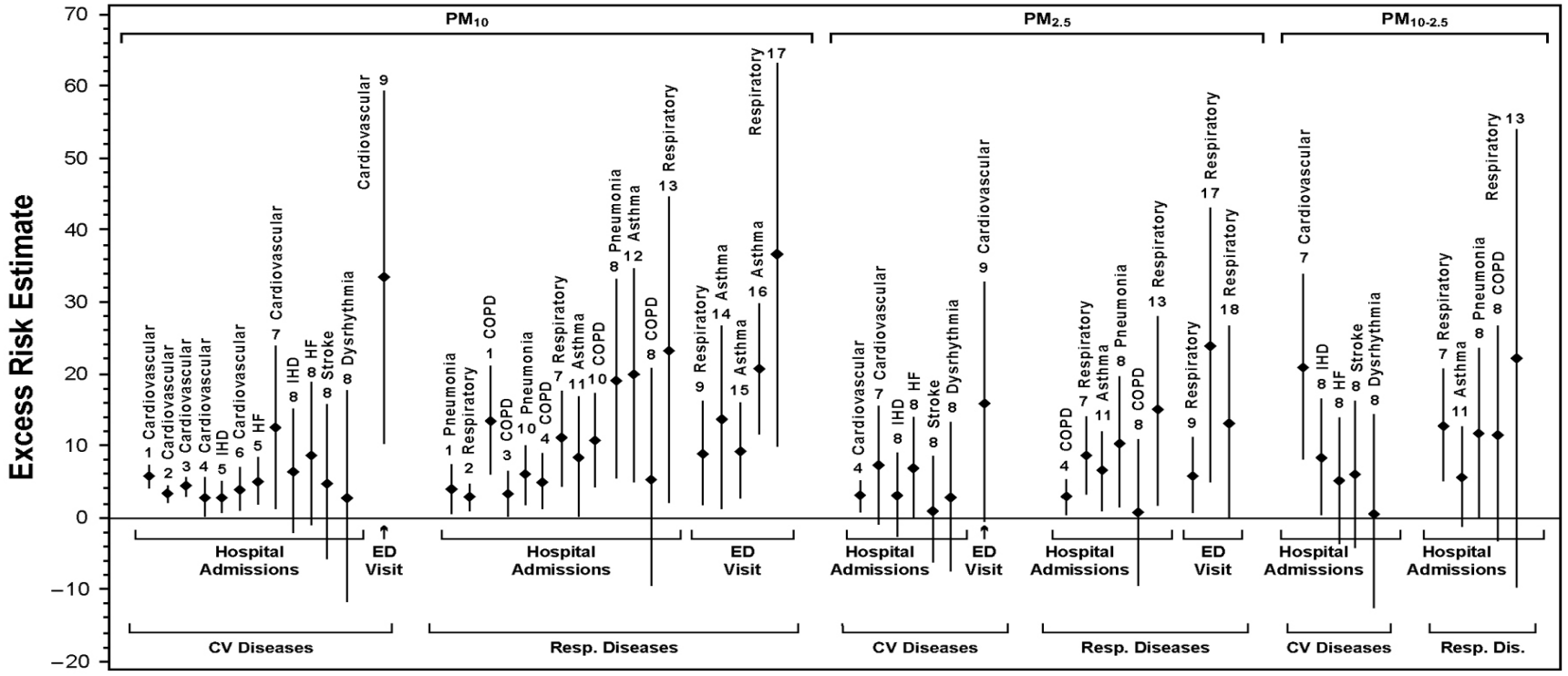

Figure 3-2. Excess risk estimates for hospital admissions and emergency department visits for cardiovascular and respiratory diseases in single-pollutant models from U.S. and Canadian studies, including aggregate results from one multicity study (as denoted in bold below). PM increments: 50 µg/m^3 for PM_{10} and 25 µg/m^3 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

NMMAPS. 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, although the CD recognizes that further evaluation is needed on this method to assess potential confounding (CD, p. 8-146, 8-175).

Numerous single-city studies have been published that report associations between short-term PM_{10} exposure and hospitalization or medical visits for cardiovascular or respiratory diseases. Overall, the CD reports that the more precise effect estimates for hospitalization range from 2 to 6% per 50 $\mu g/m^3$ PM_{10} for cardiovascular diseases and 2 to 12% per 50 $\mu g/m^3$ PM_{10} for respiratory diseases. The CD also observes that new studies reporting associations between PM_{10} and medical (e.g., physicians' office) visits for respiratory diseases offer a link between the more severe endpoints, such as increased mortality and hospital admissions or emergency room visits for respiratory diseases, and less serious effects such as respiratory symptoms and decreased lung function. These new studies also indicate the potentially more widespread public health impact of exposure to PM (CD, p. 8-194).

As shown in Figure 3-2, 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 the more precise estimates are statistically significant. Overall, the CD finds that excess risks for cardiovascular admissions range from about 1 to 10% per 25 $\mu g/m^3$ $PM_{2.5}$ or $PM_{10-2.5}$ (CD, p. 8-310). For total respiratory or COPD admissions, risk estimates tend to fall in the range of 5 to 15% per 25 $\mu g/m^3$ $PM_{2.5}$ or $PM_{10-2.5}$ (CD, p. 8-193).

Many studies using PM_{10} or other PM indicators have been conducted in areas where fine particles are the dominant fraction of PM_{10}; results of these studies would likely be reflective of associations with fine particles. In the last review, staff recognized that information about the effects of thoracic coarse particles can also come from studies linking health effects with PM_{10} in areas where thoracic coarse particles are the dominant fraction of PM_{10}. Evidence available at that time suggested that aggravation of asthma and respiratory infections and symptoms were associated with PM_{10} in areas where thoracic coarse particles were dominant, such as Anchorage, AK, and southeast Washington (62 FR 38679). Staff observes that several recent studies have also been conducted in urban areas where thoracic coarse particles are the dominant fraction of $PM_{10,}$ such as Reno, NV; Tucson, AZ; and Anchorage, AK, and these findings support the evidence from the limited group of studies that have found associations between measured

$PM_{10-2.5}$ and morbidity. In these areas, most of which have levels exceeding the PM_{10} standards, statistically significant associations have been reported between PM_{10} and increased hospitalization for cardiovascular diseases (Schwartz, 1997), hospitalization for respiratory diseases (Chen et al., 2000) and medical visits for asthma (Choudhury et al., 1997).

3.3.2.2 Effects on the Respiratory System from Short-term Exposures

As was found in the last review, some significant associations have been reported between increased respiratory symptoms and decreased lung function and short-term exposures to PM (section 8.3.3 in the CD). For asthmatic subjects, associations were reported between PM_{10} and $PM_{2.5}$ and decreases in lung function measures (e.g., decreased peak expiratory flow rate); some but not all of the associations reached statistical significance. In addition, positive associations were reported between PM_{10} and $PM_{2.5}$ and one or more of a range of respiratory symptoms (e.g., cough, wheeze, shortness of breath), but the findings were less consistent than those for lung function (CD, p. 8-199). In studies of nonasthmatic subjects, while inconsistent results were reported for changes in lung function, there were generally positive associations for respiratory symptoms that often were not statistically significant. Generally similar results were found for both PM_{10} and $PM_{2.5}$ (CD, p. 8-206).

Few studies of respiratory symptoms and lung function have included both $PM_{2.5}$ and $PM_{10-2.5}$ data. The CD summarizes findings from a Six Cities study analysis (Schwartz and Neas, 2000), a study in Philadelphia (Neas et al., 1999) and a study in Kupio, Finland (Tiittanen et al., 1999). The findings of these studies suggest roles for both fine and thoracic coarse PM in reduced lung function and increased respiratory symptoms (CD, p. 8-313). For example, in the Six Cities study, lower respiratory symptoms were found to be significantly increased for children with $PM_{2.5}$ but not with $PM_{10-2.5}$, while the reverse was true for cough. When both $PM_{2.5}$ and $PM_{10-2.5}$ were included in models, the effect estimates were reduced for each, but $PM_{2.5}$ retained significance in the association with lower respiratory symptoms and $PM_{10-2.5}$ retained significance in the association with cough (Schwartz and Neas, 2000). The new epidemiologic studies continue to show effects of short-term exposure to PM_{10} and $PM_{2.5}$ and offer additional evidence for associations between $PM_{10-2.5}$ and respiratory morbidity (CD, p. 8-312).

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.

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 staff evaluation of the data from this study that suggested that lung function decrements were not associated with 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 new European, Asian, and Australian studies have also been published. In the U.S., studies have been based on data from two cohort studies, cohorts 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 $PM_{2.5}$ is associated with reduced lung function growth and increased risk of developing chronic respiratory illness (CD, p. 8-313). In section 8.3.3.2.2, the CD describes results from Southern California cohorts, 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 cohort of children recruited in this study 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 exposure to $PM_{10-2.5}$ (CD pp. 8-313, 8-314).

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 epidemiologic studies provide much more evidence of effects on the cardiovascular system with short-term exposures to PM, particularly PM_{10} and $PM_{2.5}$ (CD, p. 9-67).

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, though the CD urges caution in drawing conclusions regarding the effects of PM on heart rhythm (CD, p. 8-166). Recent studies have also reported increases in blood components or biomarkers such as increased levels of C-reactive protein and fibrinogen (CD, p. 8-169). In addition, one new study reported an association between $PM_{2.5}$ and onset of myocardial infarction, though another study reported no significant associations between PM_{10} and sudden cardiac death (CD, pp. 8-165 to 8-166). Several of these studies report significant associations between various cardiovascular endpoints and short-term $PM_{2.5}$ exposures; 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 (CD, p. 8-165; Peters et al., 2001). These new epidemiologic findings can provide some insight into potential biologic mechanisms that underlie associations between short-term PM exposure and cardiovascular mortality and hospitalization that have been reported previously (see Section 3.2).

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 (AEGIR) 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, exposure to various PM indicators is associated with a broad range of cardiovascular and respiratory health endpoints. Newer studies report associations between short-term exposure to various indicators of PM and cardiopulmonary mortality, hospitalization or emergency department visits, and respiratory symptoms. In addition, there is now evidence for associations with cardiovascular health outcomes, such as myocardial infarction or physiological changes such as C-reactive protein increases. There are also a broader range of respiratory health effects associated with exposure to various indicators of PM than those previously documented. These effects include visits to physicians or clinics for treatment of respiratory illnesses (CD, p. 9-23).

More specifically, the epidemiologic evidence includes associations between short-term exposure to $PM_{2.5}$ and cardiorespiratory mortality, hospitalization and emergency department visits for respiratory diseases, respiratory symptoms and decreased lung function, as well as effects on the cardiovascular system, including changes in physiological indicators or biomarkers for cardiovascular health (CD, pp. 8-338, 8-342). New studies also build upon previous evidence for associations between long-term exposure to fine particles and cardiopulmonary mortality or respiratory morbidity, with new evidence suggesting that long-term exposure to fine particles is associated with lung cancer mortality (CD, p. 8-345).

Epidemiologic studies have linked short-term exposure to $PM_{10-2.5}$ with respiratory morbidity, such as hospitalization or respiratory symptoms, with suggested associations with mortality in some areas. Available studies have not supported a link between long-term exposure to $PM_{10-2.5}$ and mortality or morbidity.

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 also 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.

3.4.1 Strength of Associations

The strength of associations most directly refers to the magnitude of the reported relative risk estimates. Taking a broader view, the CD draws upon the criteria summarized in a recent report from the U.S. Surgeon General, which define strength of an association as "the magnitude of the association and its statistical strength" which includes assessment of both effect estimate size and precision, which is related to the statistical power of the study (CD, p. 9-6; CDC, 2004). In general, when associations are strong in terms of yielding large relative risk estimates, it is

less likely that the association could be completely accounted for by a potential confounder or some other source of bias (CDC, 2004). With associations that yield small relative risk estimates it is especially important to consider potential confounding and other factors in assessing causality.

As observed in the previous PM NAAQS review, in historical air pollution episodes with very high concentrations, reported relative risks were quite large.[7] In more recent studies with much lower ambient concentrations, the CD observes that the associations reported between health effects and PM yield much smaller relative risk estimates (CD, p. 9-24). Focusing on the results from more precise mortality studies done in the U.S. and Canada, the CD reports that associations with short-term exposure, expressed as relative risks, are in the range of about 1.02 to 1.06 per 25 $\mu g/m^3$ $PM_{2.5}$ or $PM_{10-2.5}$ (CD, p. 9-28), while relative risks associated with long-term exposure range upward to about 1.2 per 25 $\mu g/m^3$ $PM_{2.5}$ for cardiovascular mortality (CD, p. 8-117). Regarding the size of relative risk estimates, the CD states: "In contrast with the marked increase in health effects observed during historic episodes of very high air pollution levels, relatively small effect estimates would generally be expected with current ambient PM concentrations in the United States. The etiology of most air pollution-related health outcomes is multifactorial, and the impact of ambient air pollution exposure on these outcomes may be small in comparison to that of other risk factors." (CD, p. 9-24). Thus, while the associations reported in the more recent body of epidemiologic studies are appropriately characterized as being weak in terms of the magnitude of the relative risk estimates, such weak associations are generally coherent with outcomes that may reasonably be expected.

In considering both the magnitude and statistical strength of the associations, the CD observes a pattern of positive and often statistically significant associations for cardiovascular and respiratory health outcomes with short-term exposure to PM_{10} and $PM_{2.5}$ with estimates of similar magnitude but less precision with $PM_{10-2.5}$ (CD, p. 9-32). Of particular note are several multi-city studies that have yielded relative risk estimates for associations between short-term exposure to various indices of PM and mortality or morbidity that, while small in size, have great precision due to the statistical power of the studies. Such associations are strong relative to the precision of the studies; that is, the associations were strong enough to have been reliably measured by the studies such that many of the associations can be distinguished from the null hypothesis with statistical confidence.

In considering the strength of the associations between long-term exposure to fine particles and mortality or morbidity, the CD concludes that the magnitude and precision of associations with long-term exposure to $PM_{2.5}$ constitute "strong evidence" for associations with

[7] For example, in the week of the well-documented episode that occurred in London in 1952, when PM concentrations exceeded 500 $\mu g/m^3$, the relative risk of all-cause mortality was 2.6, and the relative risk for bronchitis mortality was 9.3 (62 FR 38659).

mortality and "fairly strong evidence" for associations with respiratory morbidity. However, the little evidence available for $PM_{10-2.5}$ provide no evidence for associations with mortality and allow no conclusions to be drawn regarding associations with morbidity (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 (id.). The CD also notes that the results of reanalyses indicated that effect estimates were more sensitive to the modeling approach used to account for temporal effects and weather variables than to the GAM specifications, and recommended further exploration of alternative modeling approaches for time-series analyses (CD, pp. 8-236 to 8-237).

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 stated in the reanalysis report, "The risk estimates reported by the Original Investigators were remarkably robust to alternative specifications of the underlying risk models, thereby strengthening confidence in the original findings" (Krewski et al., 2000, p. 232).

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 3.6.4. In the new multi-city studies, as well as in 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 (id.). Similarly, in the prospective cohort studies, associations between long-term exposure to $PM_{2.5}$ and mortality were generally not sensitive to inclusion of co-pollutants, except for SO_2, which was also associated with mortality (CD, p. 8-136). 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, while recognizing that disentangling the effects attributable to various pollutants within an air pollution mixture is challenging (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. This includes both consideration of error in measurements of PM, and the degree to which measurements from an individual monitor reflect exposures to the surrounding community. As further discussed in section 3.6.2, 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

Consistency refers to the persistent finding of an association between exposure and outcome in multiple studies of adequate power in different persons, places, circumstances and times (CDC, 2004). 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 had been no evidence of heterogeneity in initial study findings (Burnett and Goldberg, 2003; CD, p. 9-39). Finally, in the European multi-city study, 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 (CD, pp. 8-46 to 8-47; 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 than that used in most single-city studies (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, the CD observes that the 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). For short-term exposure to thoracic coarse particles, the CD concludes that 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 Temporality

Temporality refers to the occurrence of a cause before its purported effect, and is most relevant to studies of diseases that develop over time. This factor is difficult to investigate in situations where the pollutant concentrations are correlated over time as is the case to some degree in PM time series studies and to a greater degree in the cohort studies. The short-term exposure studies evaluate associations between acute health outcomes and PM measured on a daily or hourly basis. In many studies, associations have been reported between health events and pollutants measured contemporaneously. For example, in studies of total and cardiovascular mortality, the CD observes that effects have been most clearly linked with PM measured on the same day or the preceding day (CD, p. 8-273). This would be expected for acute health effects, however, it is difficult to characterize these associations in terms of temporality. Issues related to the evaluation and selection of lag periods among studies are further discussed in section 3.6.5.

The 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). Thus, the long-term exposure studies do not allow an assessment of the temporal relationship between exposure and health outcome. Taken together, it is difficult to assess temporality in the available studies of both short-term and especially long-term exposures to PM, given that PM concentrations are generally correlated over time in any given area.

3.4.5 Nature of concentration-response relationships

This is an assessment of whether increases in the potential causal factor result in increased effects, also referred to as a biologic gradient. In epidemiologic time-series analyses, the results have consistently shown positive associations, indicating that increases in various PM indicators are associated with increases in health outcomes (CD, pages 9-28 to 9-29). The prospective cohort studies have also generally reported positive associations between long-term exposure to PM, primarily $PM_{2.5}$, and increases in mortality or morbidity (CD, pp. 8-344 to 8-345). The available toxicologic studies have generally not been designed to quantify dose-response relationships (CD, p. 7-2). Among the studies reviewed in chapter 7 are some that report no evidence of a dose-response gradient (CD, p. 7-152), while some do (CD, p. 7-155), and the CD draws no overall conclusions regarding dose-response relationships from toxicologic studies. Therefore, while epidemiologic studies provide clear indication of increasing response with increasing concentration, no conclusions can be drawn from toxicologic evidence.

3.4.6 Natural Experiment Studies

Few studies are available that assess the extent to which reductions in ambient PM actually lead to reductions in health effects attributable to PM. As discussed in section 9.2.2.6 of the CD, and in somewhat more detail in section 3.4.7 below, one set of studies in the Utah Valley were conducted over a time period when a major source of PM was closed, resulting in markedly decreased PM_{10} concentrations. An epidemiologic study reported that respiratory hospital admissions decreased during the plant closure time period (CD, p. 8-131; Pope et al., 1989). Newly available controlled human exposure and animal toxicology studies, using particles extracted from stored PM_{10} sampling filters from the Utah Valley, have shown inflammatory responses that are greater with extracts of particles collected during the time period of source operation than when the source was closed (CD, p. 9-73). Epidemiologic studies in Dublin, Ireland and Hong Kong also provides evidence for reduced relative risks for mortality when PM (measured as BS or PM_{10}) and/or SO_2 were reduced as the result of interventions aimed at reducing air pollution (CD, pp. 8-131 to 8-135). From this small group of new studies, the CD concludes:

> By providing evidence for improvement in community health following reduction in air pollutant emissions, these studies add further support to the results of the hundreds of other epidemiologic studies linking ambient PM exposure to an array of health effects. Such studies showing improvements in health with reductions in emissions of ambient PM and/or gaseous co-pollutants provide strong evidence that reducing emissions of PM and gaseous pollutants has beneficial public health impacts. (CD, p. 9-45 to 9-46).

3.4.7 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 biologic 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 biologic 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. In addition, the CD finds that epidemiologic studies report associations for $PM_{2.5}$ 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 biologic mechanisms or pathways for PM-related effects, and this evidence is largely from studies of fine particles or fine particle components.

Focusing first on effects related to the cardiovascular system, 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). 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 (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 discussed in section 3.2, toxicologic and controlled human exposure studies have provided substantial evidence that particles can cause lung injury and inflammatory responses.

Interesting new evidence that links toxicologic and epidemiologic findings is available from some "intervention studies" in the Utah Valley area. Epidemiologic studies in the Utah Valley area observed that respiratory hospital admissions decreased during a period when a major source of PM_{10} (a steel mill) was closed. More recent toxicologic and controlled human exposure studies have used particles collected from this locale during the same time period, and reported increased inflammatory responses with particles collected while the PM source was operating than when it was closed. Several *in vitro* studies have also reported evidence of

increased oxidative stress in lung cell cultures exposed to particles collected in Utah Valley. In some toxicologic studies, the transition metal content of the particles appeared to be more closely linked to reported effects than the quantity of particles (CD pp. 7-46 to 7-48). 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). Staff observes that, in contrast with most evidence discussed here, this group of studies may well implicate thoracic coarse particles, since such particles generally dominate PM_{10} concentrations in the Utah Valley area.

As was true in the last review, there is some coherence in the epidemiologic evidence linking long-term exposure to fine particles with mortality and effects on the respiratory system. Available toxicologic studies have generally not studied cardiopulmonary effects of long-term or chronic exposures to ambient air pollution mixtures, so for the most part, no conclusions can be drawn regarding biologic plausibility of observed effects with long-term $PM_{2.5}$ exposures and mortality from heart and lung diseases (CD, p. 9-69). However, for lung cancer, the CD summarizes evidence that supports coherence and plausibility in the epidemiologic associations reported between long-term exposures to fine particles and lung cancer mortality. The CD discusses toxicologic evidence on mutagenic or genotoxic potential of ambient PM, particles from wood and coal combustion, and particles from diesel and gasoline engine emissions (CD Section 7.8). 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).

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, especially effects on the respiratory system (CD, p.

9-80). From the limited number of toxicologic studies using $PM_{10-2.5}$, as noted in section 3.2, there is some evidence supporting effects such as inflammation or oxidative stress. In addition, allergic adjuvant effects were linked with road dust exposures, but coarse particle sample of geologic origin, Mt. St. Helens ash, has not been linked with effects in toxicologic studies. 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 epidemiologic evidence linking $PM_{10-2.5}$ with respiratory morbidity, such as increased respiratory symptoms or risk of hospitalization for asthma.

3.4.8 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 "much more limited body of evidence is suggestive of associations

between short-term (but not long-term) exposures . . . and various mortality and morbidity effects observed at times in some locations." (CD, p. 9-79).

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 diabetes, and 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). In addition, as described previously in section 3.2, dosimetric evidence indicates that deposition of particles is increased, and can be focused in "hot spots" in the respiratory tract, in people with chronic respiratory diseases.

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 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, using various PM indicators. Results from this body of evidence, though mixed, are suggestive of possible effects; more research is needed to further elucidate the potential risks of PM exposure for these health outcomes (CD, p. 8-222).

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).

In considering populations groups that might be more vulnerable to PM-related effects, 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 biologic or individual health characteristics (CD, section 9.2.4.5).

In summary, there are several population groups that may be susceptible or vulnerable to PM-related effects. These groups include those with preexisting heart and lung diseases, older adults and children. Emerging evidence indicates that people from lower socioeconomic strata or who have particularly elevated exposures may be more vulnerable to PM-related effects. The available evidence does not generally allow distinctions to be drawn between the PM indicators, in terms of which groups might have greater susceptibility or vulnerability to $PM_{2.5}$ and/or $PM_{10-2.5}$

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 pre-existing 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." Along the spectrum of severity, it is expected that incidence or frequency of health endpoints would be larger for the less severe effects, such as respiratory symptoms or the more subtle measures of cardiovascular health such as levels of C-reactive protein. In contrast, with more severe health outcomes, such as hospitalization or mortality, lower incidence would be expected.

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. 8-334).

In addition to the 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, especially $PM_{2.5}$, can have substantial public health impacts (CD, p. 9-93). Furthermore, in the ACS cohort, the strongest associations between $PM_{2.5}$ and mortality were among the less educated participants who form a relatively small portion of the total study cohort. If the education distribution were adjusted to reflect the education distribution in the general U.S. population, the summary effect estimate would increase.

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 issues related to air quality data used in epidemiologic studies, and section 3.6.2 discusses the potential impact of measurement error and exposure error, related to the use of ambient air concentrations as indicators of population exposures, on epidemiologic studies. Section 3.6.3 addresses statistical modeling and model specifications used in epidemiologic studies. Section 3.6.4 addresses the issue of potential confounding by co-pollutants, as it relates to staff conclusions about the use of specific study results in quantitative assessments. Section 3.6.5 includes discussion of several topics related to the exposure periods used in epidemiologic studies, with an emphasis on the question of lag periods. 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. First, staff observes that PM measurements from several different monitoring methods were used in epidemiologic studies. Many studies have used $PM_{2.5}$ and $PM_{10-2.5}$ measurements from dichotomous samplers or Harvard impactors, as well as $PM_{2.5}$ and PM_{10} measurements from co-located TEOMs or BAMs, and 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.

In considering the frequency of PM data collection, staff observes that it can have a systematic effect on 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 and reduced precision 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 in Chicago, IL, where a significant association was reported between PM_{10} and mortality using data collected on a daily basis (Ito et al., 1996). However, when the data set was divided into 6 subsets representing 1-in-6 day monitoring frequency, the effect estimates for the PM_{10}-mortality association were quite variable in size and more uncertain. Consistent with the CD's observation that uncertainty is increased in studies using infrequently collected PM data, staff judges that greater weight should be placed on those studies with daily or near-daily PM data collection in drawing quantitative conclusions (CD, p. 9-41).

3.6.2 Measurement Error and Exposure Error

Measurement error, or uncertainty in the air quality measurements can be an important source of uncertainty in 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 calculated as the difference between 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.

Exposure error is an issue that is closely linked with the preceding discussion of PM air quality monitoring. Concentrations measured at ambient monitoring stations are generally used to represent a community's exposure to ambient PM. For time-series studies, the emphasis is on the temporal (usually daily) changes in ambient PM. In cohort or cross-sectional studies, air quality data averaged over a period of months to years are used as indicators of a community's long-term exposure to ambient PM and other pollutants.

As discussed in section 2.7, one component of exposure error is how evenly distributed PM is across a community, as indicated by levels at different monitoring sites; another component is how well particles penetrate from ambient air into indoor environments. Several factors affect how readily particles can move into buildings and remain suspended in indoor air. In general, fine particles move indoors and remain suspended more easily than do thoracic coarse particles. In time-series analyses, measurements of $PM_{2.5}$ made at a central site are found to be better correlated with indoor measurements than are measurements of $PM_{10-2.5}$ (see section 2.7.2). A number of recent studies have evaluated the effect of this type of exposure error on epidemiologic study results. The results of these studies, primarily focused on fine particles,

indicate that exposure error related to the use of PM data from central monitoring sites is likely to result in underestimation of the effect of PM exposure on health (CD, p. 8-288).

Analyses of site-to-site variability for $PM_{2.5}$ measurements, including time-series correlations of measurements across monitors and differences in mean concentrations between monitors, are presented in Table 2-3. The temporal correlation coefficients between monitors are high, often exceeding 0.80, indicating good correlation between time-series $PM_{2.5}$ measurements. However, a few areas, such as Los Angeles and Seattle, had lower temporal correlation coefficients, in the range of 0.60. As observed in the CD, western areas are less influenced by regional sources of fine particles (CD, p. 8-293), and geographic or topographic features may make $PM_{2.5}$ levels less homogeneous. Even where there is good temporal correlation between monitors, there may be a spatial gradient in $PM_{2.5}$ across the area. As discussed in the CD (Table 8-40), some areas had strong correlation coefficients (on the order of 0.90) but substantial differences in annual means were found between some monitor pairs. For example, correlation coefficients averaged about 0.90 between $PM_{2.5}$ monitor pairs in Detroit, but annual mean differences of up to 6 $\mu g/m^3$ were found between monitor pairs.

This same type of analysis was done using available data for $PM_{10\text{-}2.5}$, as discussed in section 2.4.3. Table 2-4 shows that there are greater differences in concentrations between paired $PM_{10\text{-}2.5}$ monitors than were seen in data from paired $PM_{2.5}$ monitors. Differences in annual mean values of over 20 $\mu g/m^3$ are shown between some paired $PM_{10\text{-}2.5}$ monitors, representing differences of 60-70% in some cases. Correlations between the monitoring sites were also somewhat lower than those for $PM_{2.5}$, ranging from about 0.3 to 0.8. In some cities, for example Cleveland, OH and Detroit, MI, the $PM_{10\text{-}2.5}$ measurements at paired monitors show both a large difference in magnitude as well as poor correlation in day-to-day changes; for both cities, the values are 60-70% different between the monitor pairs, and the correlation coefficient is about 0.4. However, for a number of the cities shown in Table 2-4, the correlation coefficients between data from paired monitors are in the range of 0.7 to 0.8, indicating that the data are fairly well correlated temporally, but there remain substantial differences in annual mean concentrations between the monitors. In interpreting the results of epidemiologic associations with $PM_{10\text{-}2.5}$, the data from the central monitoring sites may be characterizing day-to-day changes in $PM_{10\text{-}2.5}$ concentrations adequately, but staff observes that it is difficult to determine how well such concentrations characterize the magnitude of population exposures to $PM_{10\text{-}2.5}$.

In summary, there are some key exposure-related distinctions between $PM_{2.5}$ and $PM_{10\text{-}2.5}$. In section 9.2.1, the CD concludes that $PM_{2.5}$ concentrations are frequently evenly distributed across cities, and frequently have high site-to-site correlations; as summarized above, there can be differences in some locations. In contrast, the CD concludes that $PM_{10\text{-}2.5}$ is "seldom" evenly distributed across cities and that there are "frequently low" site-to-site 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 led 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 epidemiologic analyses. The following discussions draw from the CD's evaluation of model specification issues for both short-term and long-term exposure studies.

3.6.3.1 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. In the HEI reanalyses, 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).

In summary, the reanalyses generally support the findings of the original studies, while raising questions for further research. For quantitative assessment, staff considers it appropriate to use results from short-term exposure studies that did not use GAM initially, or that used either more stringent GAM or GLM analyses. As recognized in the CD, there is no one correct approach for model specification or covariate adjustment (CD, p. 9-35). An advantage to the use of GAM is that the model is "data-driven" and selects the degree of smoothing or adjustment for covariates that best fits the data. The GLM approach is advantageous in allowing more accurate calculation of standard errors.

3.6.3.2 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).

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 identifying and assessing the level of effect modification (CD, p. 8-12).

In addition to acting as confounders or effect modifiers, the CD recognizes that pollutants may act together in an ambient pollution mixture, potentially having additive or synergistic effects. 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 suggested 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).

3.6.4.1 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, if PM constituents are derived from gaseous pollutants (e.g., sulfates from SO_2), or if meteorological conditions contribute to the formation of both PM and gaseous pollutants (CD, p. 8-12). These situations certainly occur. 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. In addition, SO_2 and $PM_{2.5}$ are often derived from the same sources in an area (e.g., coal-fired power plants) and thus simultaneous inclusion in models may result in diminished effects for one or both pollutants, which can be misleading (CD, p. 8-14).

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; Dominici, 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.

Using an alternative approach, investigators in the NMMAPS morbidity analyses for 14 U.S. cities 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). The authors concluded that associations with PM_{10} were not dependent on the correlation between PM_{10} and the gaseous copollutants, though as mentioned previously, the CD highlights the need for additional evaluation of this type of analysis (CD, pp. 8-146).

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 inherently difficult to distinguish effects from the precursor SO_2 and fine particles (CD, p. 9-37).

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).

In summary, where various indicators of 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 various indicators 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 unlikely that a relationship found between a health endpoint and ambient $PM_{2.5}$ concentrations is actually representing relationships with other pollutants.

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). As shown in figures 8-16 through 8-19 of the CD, effect estimates for PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ were little changed in multi-pollutant models, as compared with single-pollutant models. 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, supported by analyses including multi-pollutant model results as available.

3.6.4.2 Effect Modification

One approach 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 the effect of $PM_{2.5}$ is not appreciably modified by differing levels of the gaseous pollutants.

An alternative approach would be assessment within a single study of whether the effect of time-varying PM is modified by time-varying concentrations of the gases in the time-series models, through addition of interaction terms, for example. However, such studies have not been conducted. While potential effect modification between various indicators of PM and the gaseous pollutants has been little studied, the limited available evidence indicates that the gases do not have a major role as effect modifiers for PM-related health outcomes.

3.6.5 Issues Related to Alternative Exposure Periods in Epidemiologic Studies

3.6.5.1 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, such as that shown in Figure 8-22 of the CD, 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, any result for a single day may well be biased (CD, p. 9-42). Staff also observes that the high degree of autocorrelation in PM measurements complicates the assessment of various lag periods.

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. As discussed below, most of the studies included in Appendix 3A evaluated results for a range of lag periods, with many authors reporting effect estimates for one lag period based on this evaluation. However, a few researchers selected lag periods *a priori*. Examples of studies that used *a priori* selection of lag periods includes Liao et al. (1999), in which the 24-hour $PM_{2.5}$ average preceding measurement of cardiac function was used, and Schwartz et al. (1996), in which an average of 0-day and 1-day lagged PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ measurements was used in analyses of associations with mortality.

Most authors report testing associations across a range of lag periods, and in many cases the authors reported a pattern of positive associations across several lag periods. Figure 8-22 in the CD presents associations for $PM_{2.5}$ levels over both a series of days and a series of hours preceding myocardial infarction incidence, and positive associations can be seen over several adjacent lag periods (CD, p. 8-270; Peters et al., 2001). In an analysis using hospitalization for asthma, researchers report testing associations for lags to 3-days and beyond, and reported consistent patterns across lags for associations between asthma hospitalization and PM_{10}, $PM_{2.5}$ or $PM_{10-2.5}$ (CD, p. 8-270; Sheppard et al.,1999; 2003). Results for the strongest associations are presented in this study, 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

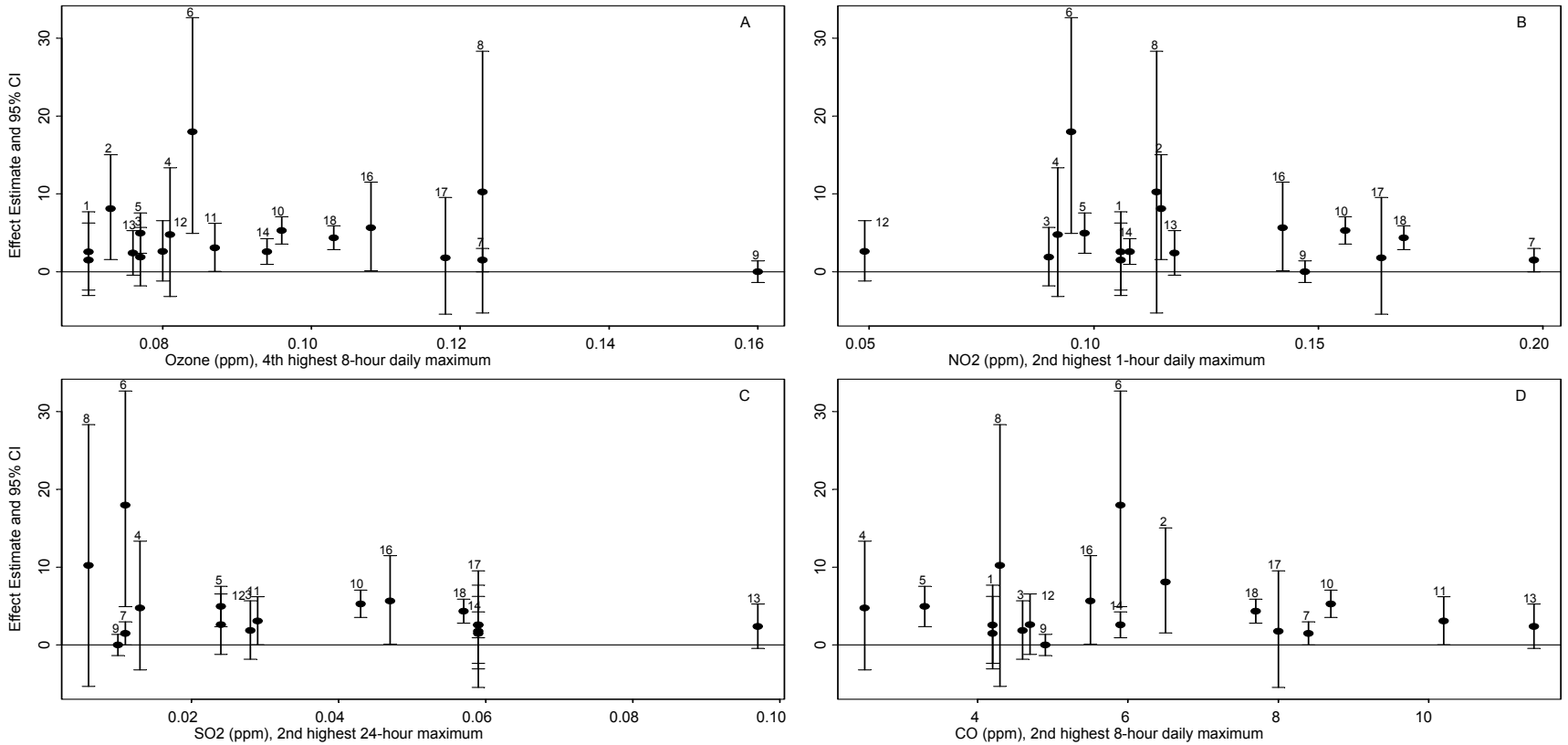

Figure 3-3. Associations between $PM_{2.5}$ and total mortality from U.S. studies, plotted against gaseous pollutant concentrations from the same locations. Air quality data obtained from the Air Quality System (AQS) for each study time period: (A) mean of 4th highest 8-hour ozone concentration; (B)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

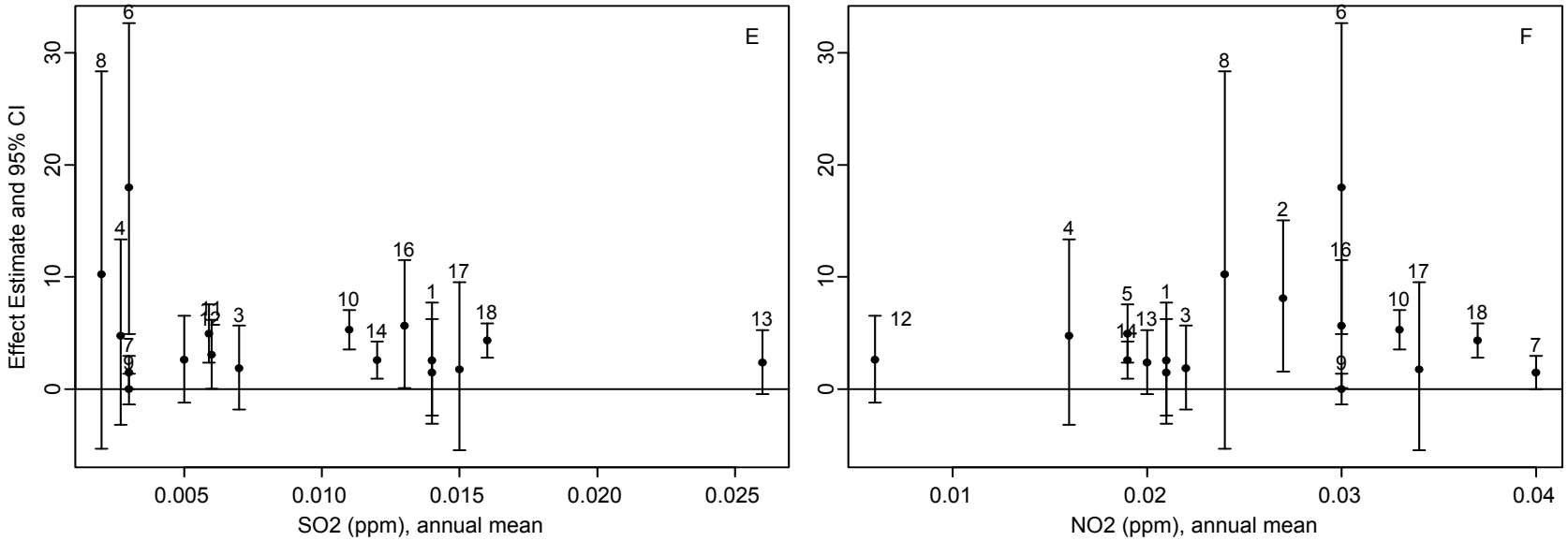

Figure 3-3 (continued). **Associations between $PM_{2.5}$ and total mortality from U.S. studies, plotted against gaseous pollutant concentrations from the same locations. Air quality data obtained from the Air Quality System (AQS) for each study time period: (E) annual mean SO_2 concentration; (F) annual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

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

appendicitis change much more abruptly, and the overall pattern is unstable. (Sheppard et al., 1999, p. 27)

In a study of mortality in Phoenix, positive associations are reported with PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ out to a lag period of 4 days, but effect estimates were larger and more often statistically significant for the 0- or 1-day lag periods (Mar et al., 1999; 2003). In an analysis of mortality and hospitalization in Detroit, results for models including individual lag days and moving average lags periods for PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ are presented in appendices and the results of the most significant lag results are presented in the body of the report, with the observation that significant associations often occurred at multiple lags (Lippmann et al., 2000, p. 24). Among U.S. and Canadian studies, the CD observes that many authors report finding a pattern of PM-related effects across adjacent lags (CD, p. 8-279).

An example of results that *do not* follow a consistent pattern across lags can be found in results for the association between $PM_{2.5}$ and mortality in Coachella Valley (Ostro et al., 2000; 2003). In this study, the pattern of results across a series of lag periods was not consistent in associations between $PM_{2.5}$ and total or respiratory mortality[8]. 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 these $PM_{2.5}$ results for quantitative assessments.[9] 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, for most health endpoints, the results follow a pattern. However, the pattern of results specifically for COPD mortality with $PM_{2.5}$ was quite inconsistent (Moolgavkar, 2000a,b,c; Moolgavkar, 2003, p. 191).[10] Based on the considerations described above, staff concludes that it would not be appropriate to use the results for COPD mortality from this study for quantitative assessment.

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 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

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[8]Staff observes that the results for a series of lags show fairly consistent patterns for associations between PM_{10} and $PM_{10-2.5}$ and cardiovascular mortality in this analysis.

[9]The air quality measurements available for $PM_{2.5}$ and $PM_{10-2.5}$ may also contribute to the more uncertain findings for $PM_{2.5}$ in this study. For $PM_{10-2.5}$, 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.

[10] 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.

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 (CD, p. 8-279).

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 generally not available for distributed lag models, staff concludes 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.

3.6.5.2 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 stratified 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 yielded larger effect estimates 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 effect estimates were larger 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 effect estimates were larger in spring and fall (Sheppard et al., 2003).

Staff observes that these few studies show no general 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 at this time.

3.6.5.3 Health effects related to different 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.

3.6.5.4 Exposure periods used in prospective cohort studies

The prospective cohort studies have used air quality measurements averaged over long periods of time, such as several years, to characterize the long-term ambient levels in the community. The exposure comparisons are basically cross-sectional in nature, and do not provide evidence concerning any temporal relationship between exposure and effect (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 $PM_{2.5}$ concentrations for the different time periods 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 $PM_{2.5}$ and PM_{10} 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 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 there likely are biologic thresholds for different health effects in individuals or groups of individuals with similar innate characteristics and health status. 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, below which no individual would experience a given effect, especially if some 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 concentration-response relationships, further obscuring potential population thresholds within the range of observed concentrations (CD, p. 9-43, 9-44).

The 1996 CD evaluated evidence from epidemiologic studies regarding both linear and nonlinear forms of concentration-response relationships and whether any effect thresholds 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 alternative forms of concentration-response functions and potential threshold levels.

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). In one single-city analysis, various statistical methods were used 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, in the data for this city, it was highly likely that standard statistical methods could detect a threshold level, if one existed (Cakmak et al., 1999; CD, p. 8-319). Thus, a number of studies have thus been unable to detect threshold levels in the PM-mortality relationship, and in fact one single-city analysis suggests that statistical methods would allow detection of a threshold in the epidemiologic data if a clear threshold existed.

However, a few analyses in individual cities have provided suggestions of some potential threshold levels. 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-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 there likely are individual biologic thresholds for specific health responses, existing studies do not support or refute the existence of thresholds in PM-mortality relationships at the population level, 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 thresholds in observed associations in the range of air quality concentrations in the studies, it is possible that such thresholds exist toward the lower end of these ranges (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 such thresholds, the potential thresholds are at fairly low concentrations (CD, p. 9-45).

Based on the above considerations, staff concludes in part that it is appropriate to use the linear or log-linear concentration-response models reported in epidemiologic studies in the quantitative risk assessment. Staff also recognizes, however, the possibility that thresholds may exist in reported associations at fairly low levels within the range of air quality observed in the studies, though no specific threshold levels have been clearly identified. While the biologic plausibility of the existence of individual thresholds supports the potential that concentration-response relationships may be non-linear at the lower end of the range of observed concentrations, statistical evaluations comparing linear and non-linear concentration-models have been unable to resolve this question. Therefore, the staff also concludes that the implications of assuming a non-linear concentration-response relationship also should be

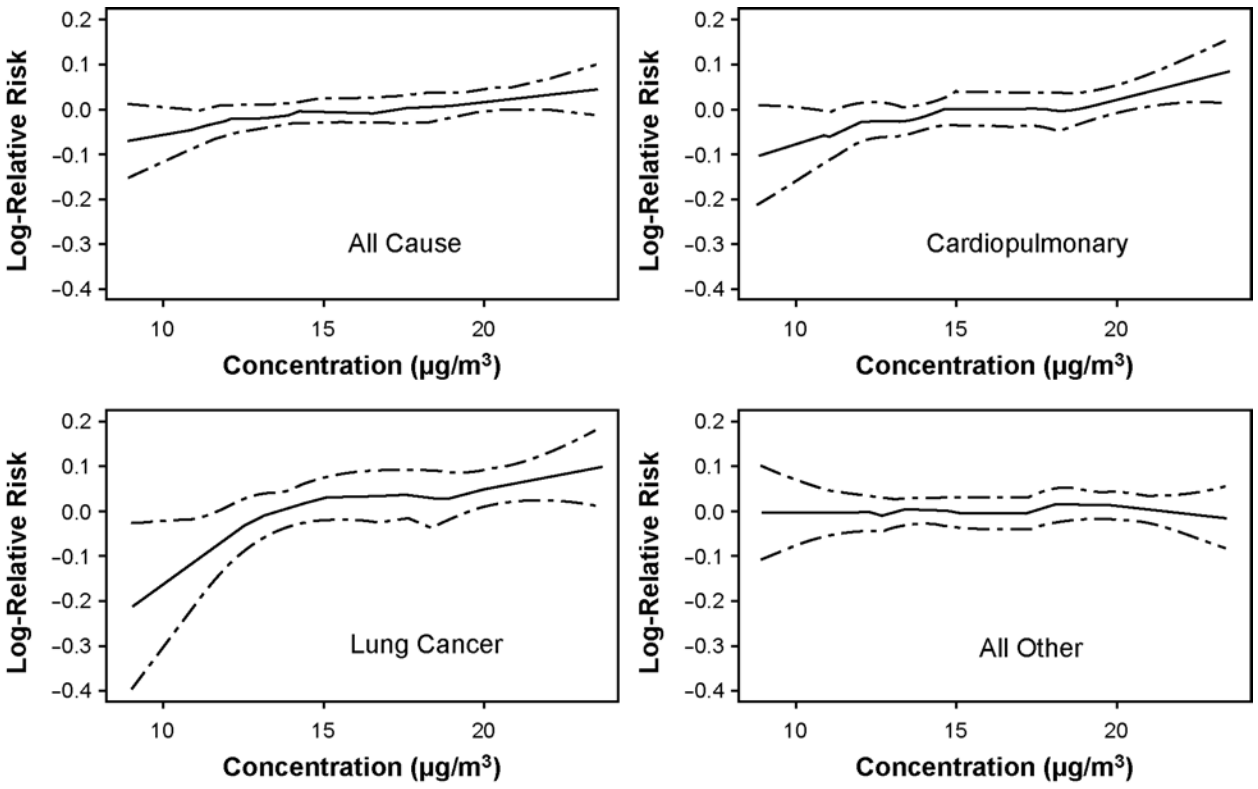

Figure 3-4. Natural logarithm of relative risk for total and cause-specific mortality per 10 µg/m^3 $PM_{2.5}$ (approximately the excess relative risk as a fraction), with smoothed concentration-response functions. Based on Pope et al. (2002) mean curve (solid line) with pointwise 95% confidence intervals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

included in the quantitative risk assessment. In the absence of published concentration-response models reflecting typical sigmoidal or "hockey-stick" shaped relationships, staff has included in the quantitative risk assessment (described in greater detail in Chapter 4) analyses incorporating a modified linear slope with an imposed cut point. This approach is used as a surrogate for a non-linear, sigmoidal-shaped function, in which the cut point is intended to reflect an inflection point at the lower end of the relationship, below which it is assumed that there is little or no population response.

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 broaden 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. For quantitative assessment, staff concludes that single-pollutant model effect estimates can be used as reasonable indicators of the magnitudes of effect sizes, especially for comparing results across studies. Additional analyses using multi-pollutant model results, where available, can allow assessment of risks related to PM exposure with 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 since the "best" lag day result may be biased upward.

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. Based on the above considerations, staff concludes in part that it is appropriate to use the linear or log-linear concentration-response models reported in epidemiologic studies in the quantitative risk assessment. Staff also recognizes, however, the possibility that thresholds may exist in reported associations at fairly low levels within the range of air quality observed in the studies, though no specific threshold levels have been clearly identified. Therefore, the staff also concludes that the implications of assuming a non-linear concentration-response relationship also should be included in the quantitative risk assessment. To do so, alternative cutpoints can be used as surrogate for a non-linear, sigmoidal-shaped function, to reflect an inflection point at the lower end of the relationship, below which it is assumed that there is little or no population response.

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4. CHARACTERIZATION OF HEALTH RISKS

4.1 INTRODUCTION

This chapter presents information regarding the results from an updated PM health risk assessment that builds upon the methodology used in the more limited assessment conducted as part of the last PM NAAQS review. This updated assessment includes estimates of (1) risks of mortality, morbidity, and symptoms associated with recent ambient $PM_{2.5}$ and $PM_{10-2.5}$ levels; (2) risk reductions associated with just meeting the current suite of $PM_{2.5}$ NAAQS; and (3) risk reductions associated with just meeting various alternative $PM_{2.5}$ and $PM_{10-2.5}$ standards. This risk assessment is more fully described and presented in a technical support document, *Particulate Matter Health Risk Assessment for Selected Urban Areas* (Abt Associates, 2005b; henceforth referred to as the Technical Support Document and cited as TSD).

The goals of this PM risk assessment are: (1) to provide estimates of the potential magnitude of mortality and morbidity effects associated with current $PM_{2.5}$ and $PM_{10-2.5}$ levels, and with meeting the current suite of $PM_{2.5}$ NAAQS and alternative $PM_{2.5}$ and $PM_{10-2.5}$ standards, in specific urban areas;[1] (2) to develop a better understanding of the influence of various inputs and assumptions on the risk estimates; and (3) to gain insights into the distribution of risks and patterns of risk reductions associated with meeting alternative suites of PM standards. Staff recognizes that there are many sources of uncertainty and variability inherent in the inputs to this assessment and that there is a high degree of uncertainty in the resulting PM risk estimates. While some of these uncertainties have been addressed quantitatively in the form of estimated confidence ranges around central risk estimates, other uncertainties and the variability in key inputs are not reflected in these confidence ranges, but rather are addressed through separate sensitivity analyses or characterized qualitatively.

Following this introductory section, this chapter discusses the scope of the risk assessment, including selection of urban areas and health endpoints; components of the risk model; characterization of uncertainty and variability associated with the risk estimates; and key results from the assessment for both $PM_{2.5}$ and $PM_{10-2.5}$. The TSD provides a more detailed discussion of the risk assessment methodology and includes additional risk estimates beyond those summarized herein.

4.1.1 Overview of Risk Assessment From Last Review

In the last review, PM-associated risks were estimated for two urban areas: Philadelphia and Los Angeles counties (Abt Associates, 1996). The PM health risk model used in the last

[1]To provide a broader perspective on health risks associated with ambient PM, 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.

assessment combined information about daily PM air quality for these two areas with estimated concentration-response functions derived from epidemiologic studies and baseline health incidence data for specific health endpoints to derive estimates of the annual incidence of specific health effects associated with recent air quality levels (termed "as is" air quality in both the previous and current TSD). 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 type of meta-analysis (referred to as a "pooled analysis") was conducted that combined the results of those studies that met specified criteria. The assessment also examined the reduction in estimated incidence that would result from just meeting the existing PM_{10} standards and various alternative $PM_{2.5}$ standards. In addition, the assessment included sensitivity analyses and integrated uncertainty analyses to better understand the influence of various inputs and assumptions on the risk estimates. The methodological approach followed in conducting the last risk assessment and risk estimates are 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).

In the 1997 review of the PM NAAQS, 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 concentration-response functions and quantitative risk estimates derived from them. Nevertheless, EPA judged that the assessment provided reasonable estimates as to the possible extent of risk for those effects given the available information (62 FR at 38656).

4.1.2 Development of Approach for Current Risk Assessment

The scope and methodology for this updated PM risk assessment have been developed over the last few years. In June 2001, OAQPS released a draft document, *PM NAAQS Risk Analysis Scoping Plan* (EPA, 2001c), for CASAC consultation and public comment, which described staff's general plan for this assessment. In January 2002, OAQPS released a more detailed draft document, *Proposed Methodology for Particulate Matter Risk Analyses for Selected Urban Areas* (Abt Associates, 2002), for CASAC review and public comment, which described staff's plans to assess (a) $PM_{2.5}$-related risks for several health endpoints, including mortality, hospital admissions, and respiratory symptoms and (b) $PM_{10-2.5}$-related risks for hospital admissions and respiratory symptoms. During a February 2002 teleconference, CASAC discussed this draft document and public comments were made; CASAC sent an advisory letter to the Administrator documenting its advice in May 2002 (Hopke, 2002). In its advisory letter, CASAC "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).

In response to a request from CASAC to provide additional details about the planned scope of the $PM_{10-2.5}$ and PM_{10} components of the assessment, in April 2003 OAQPS released a draft memorandum (Abt, 2003a) for CASAC consultation and public comment, addressing these topics. In August 2003, OAQPS released a draft PM risk assessment report (Abt Associates, 2003b) in conjunction with the first draft PM Staff Paper. The CASAC provided its comments on the draft PM risk assessment in a letter to the Administrator (Hopke, 2004). A second draft PM risk assessment report (Abt Associates, 2005a) was released in January 2005 in conjunction with the second draft PM Staff Paper, and was reviewed by CASAC and the public at a meeting held in April 2005. The final PM risk assessment report (Abt Associates, 2005b) takes into account the advice from CASAC and public comments received on the earlier drafts of that document.

4.2 SCOPE OF PM RISK ASSESSMENT

This risk assessment estimates risks of various health effects associated with exposure to ambient $PM_{2.5}$ and $PM_{10-2.5}$ in a number of urban areas selected to illustrate the public health impacts of these pollutants. The health endpoints selected for the $PM_{2.5}$ assessment, discussed in section 4.2.1, include those related to short- and long-term exposure for which the CD concludes that the association with $PM_{2.5}$ (or one or more $PM_{2.5}$ components), acting alone and/or in combination with gaseous co-pollutants, is likely causal (CD, p. 9-79). The health endpoints selected for the $PM_{10-2.5}$ assessment, also discussed in section 4.2.1, include those related to short-term exposure for which the CD concludes that the scientific evidence is suggestive of an association that the staff judges to be likely causal. This assessment includes risk estimates for nine urban areas for $PM_{2.5}$ and three urban areas for $PM_{10-2.5.}$ The basis for selection of these areas is discussed below (section 4.2.2). This assessment is intended to estimate risks attributable to anthropogenic sources and activities (i.e., risk associated with concentrations above policy-relevant background or above various higher cutpoints that reflect possible population thresholds).

This assessment uses concentration-response functions from epidemiologic studies of short- and long-term exposures to ambient PM based on PM concentrations measured at fixed-site, community-oriented, ambient monitors. As discussed 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, have a plausible linkage to the daily variations of exposure to ambient $PM_{2.5}$ and $PM_{10-2.5}$ 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 epidemiologic studies that use community average health and pollution data" (CD, p. 5-121)**.** The possible impact of

exposure misclassification on the estimated concentration-response relationships derived from the community epidemiologic studies is discussed above in Chapter 3 (section 3.6.2). Since the currently available epidemiologic health effects evidence relates ambient PM concentrations, not exposures, to health effects, this assessment does not include a quantitative exposure analysis. While quantitative estimates of personal or population exposure do not enter into this risk assessment, an understanding of the nature of the relationships between ambient PM concentrations and its various components and human exposure underlies the conceptual basis for this assessment.[2]

While the NAAQS are intended to provide protection from health effects associated with 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.

4.2.1 Selection of Health Endpoint Categories

As discussed in Chapter 3, OAQPS staff carefully reviewed the health effects evidence evaluated in the CD to identify potential health effect categories for inclusion in this assessment. Given the large number of endpoints and studies addressing PM-related effects, staff included only the more severe and better understood (in terms of health consequences) health endpoint categories. In addition, the staff included only those health endpoints for which the overall weight of the evidence from the collective body of studies supports the CD conclusion that there is likely to be a causal relationship or that the scientific evidence is sufficiently suggestive of a causal relationship that staff judges the effects to be likely causal between PM and the health effects category. Finally, for the three PM indicators ($PM_{2.5}$, PM_{10}, $PM_{10-2.5}$), staff considered only those endpoint categories which provided concentration-response 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.

[2]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 and to collect new information in PM exposure measurement field studies that will improve the scientific bases for exposure analyses that may be considered in future reviews.

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 $PM_{2.5}$ and $PM_{10-2.5}$:

Related to short-term $PM_{2.5}$ exposure:

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

Related to long-term $PM_{2.5}$ exposure:

- total, cardiopulmonary, and lung cancer mortality.

Related to short-term $PM_{10-2.5}$ exposure:

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

As discussed in Chapter 3 (sections 3.4 and 3.7), the available evidence for other health responses, such as associations between short-term exposure to $PM_{10-2.5}$ and mortality, is more equivocal. Staff believe that these health endpoints, which are based on less certain evidence, are not appropriate for inclusion in the quantitative risk assessment.

4.2.2 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 epidemiologic studies are available that estimate concentration-response 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 concentration-response 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 is the same as or close to the location where at least one concentration-response function, for one of the selected health endpoints, has been estimated by an epidemiologic study that satisfies the study selection criteria (see below).

- An area had relatively recent area-specific baseline incidence data available for those locations with epidemiologic studies reporting PM-related hospital admissions.

- An area is one in which epidemiologic studies exist that had relatively greater precision, as discussed below.

- An area had sufficient air quality data for a recent year (1999 or later). Sufficient $PM_{2.5}$ data are defined as having at least one PM monitor at which there are at least 11 observations per quarter for a one year period.[3] Sufficient air quality data for $PM_{10-2.5}$ are 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.[4]

For the $PM_{2.5}$ risk assessment, staff focused on selecting urban areas in which studies reported total and/or cardiovascular mortality associated with short-term exposure to $PM_{2.5}$ concentrations, 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 3A and 3B of this Staff Paper), 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.

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 to 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 was no bright line for selecting any particular cutoff, staff chose 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 (i.e., approximately 8,000 deaths) for total non-accidental mortality.[5] As a result of applying this criterion, six urban areas were excluded as potential study areas (Camden, NJ; Coachella Valley, CA; Elizabeth, NJ; Newark, NJ; Steubenville, OH; and Topeka, KS).

Finally, staff considered which of the remaining potential study locations identified from steps one and two above also had sufficient $PM_{2.5}$ ambient monitoring data consistent with the above criterion. This final criterion excluded two of the remaining potential study areas (Knoxville, TN and Portage, WI), leaving nine urban areas (i.e., Boston, MA; Detroit, MI; Los

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[3]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.

[4]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.

[5]Most of the epidemiologic 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 concentration-response functions from such studies, as long as the natural log of total mortality-days was greater than or equal to 9.0.

Angeles, CA; Philadelphia, PA; Phoenix, AZ; Pittsburgh, PA; San Jose, CA; Seattle, WA; and St. Louis, MO) in which epidemiologic studies reported concentration-response 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 also was conducted for these same nine urban areas.

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 the same criteria, focusing on locations which had studies with relatively greater precision, had adequate $PM_{2.5}$ ambient air quality data, and, for the hospital admissions effect category, had the necessary baseline incidence data.

The selection of urban areas to include for the $PM_{10-2.5}$ risk assessment was based on examining the pool of epidemiologic 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 noted earlier, the $PM_{10-2.5}$ risk assessment is more limited because of the more limited air quality 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.

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; endpoints and locations for the $PM_{10-2.5}$ risk assessment are summarized in Table 4-3. These tables also list the specific studies that provided the estimated concentration-response functions used in the $PM_{2.5}$ and $PM_{10-2.5}$ risk assessment. More detailed information on the studies selected can be found in Appendices 3A, 3B, and 4A of this Staff Paper and Appendix C of the TSD.

4.3 COMPONENTS OF THE RISK MODEL

In order to estimate the incidence of a particular health effect associated with recent 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 or various cutpoints, as well as 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) recent 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 recent 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;

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
Seattle, WA					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 concentration-response 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 provide total and cardiopulmonary mortality and Pope et al. (2002)-ACS extended provide total, cardiopulmonary, and lung cancer mortality coefficients

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 concentration-response function

[A] Reanalyzed in Ito (2003); COPD+ is indicated here because the authors included asthma in their definition of COPD.

[B] Reanalyzed in Moolgavkar (2003); COPD+ is indicated here because the authors included asthma in their definition of COPD.

[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 concentration-response function

[A] Reanalyzed in Ito (2003); COPD+ is indicated here because the authors included asthma in their definition of COPD.

[B] Reanalyzed in Sheppard (2003)

- relative risk-based concentration-response 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. EPA evaluated several base case scenarios, using various alternative cutpoints (see section 4.3.4.2). Those points where EPA has conducted analyses of alternative assumptions, procedures, or data across the various locations are indicated by a diamond with S_x in it. A summary description of the sensitivity analyses performed is included in Table 4-4.[6] Each of the key components (i.e., air quality information, estimated concentration-response functions, and baseline incidence and population data) is discussed below, highlighting those points at which judgments have been made.

The concentration-response relationships used in the PM risk assessment are empirically estimated relationships between average ambient PM concentrations and the health endpoints of interest reported by epidemiologic studies for specific urban areas. Most epidemiologic studies estimating relationships between PM and health effects used a method referred to as "Poisson regression" to estimate exponential (or log-linear) concentration-response functions.[7] 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 when there is no ambient $PM_{2.5}$ or $PM_{10\text{-}2.5}$. 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 $PM_{10\text{-}2.5}$) levels, $Sx = x_0 - x$, is then

[6]Two additional sensitivity analyses were carried out in single locations: one addressing the impact of an exceptional event episode in Boston and one examining the effect of different model specifications on annual health risks associated with recent air quality levels in Los Angeles.

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

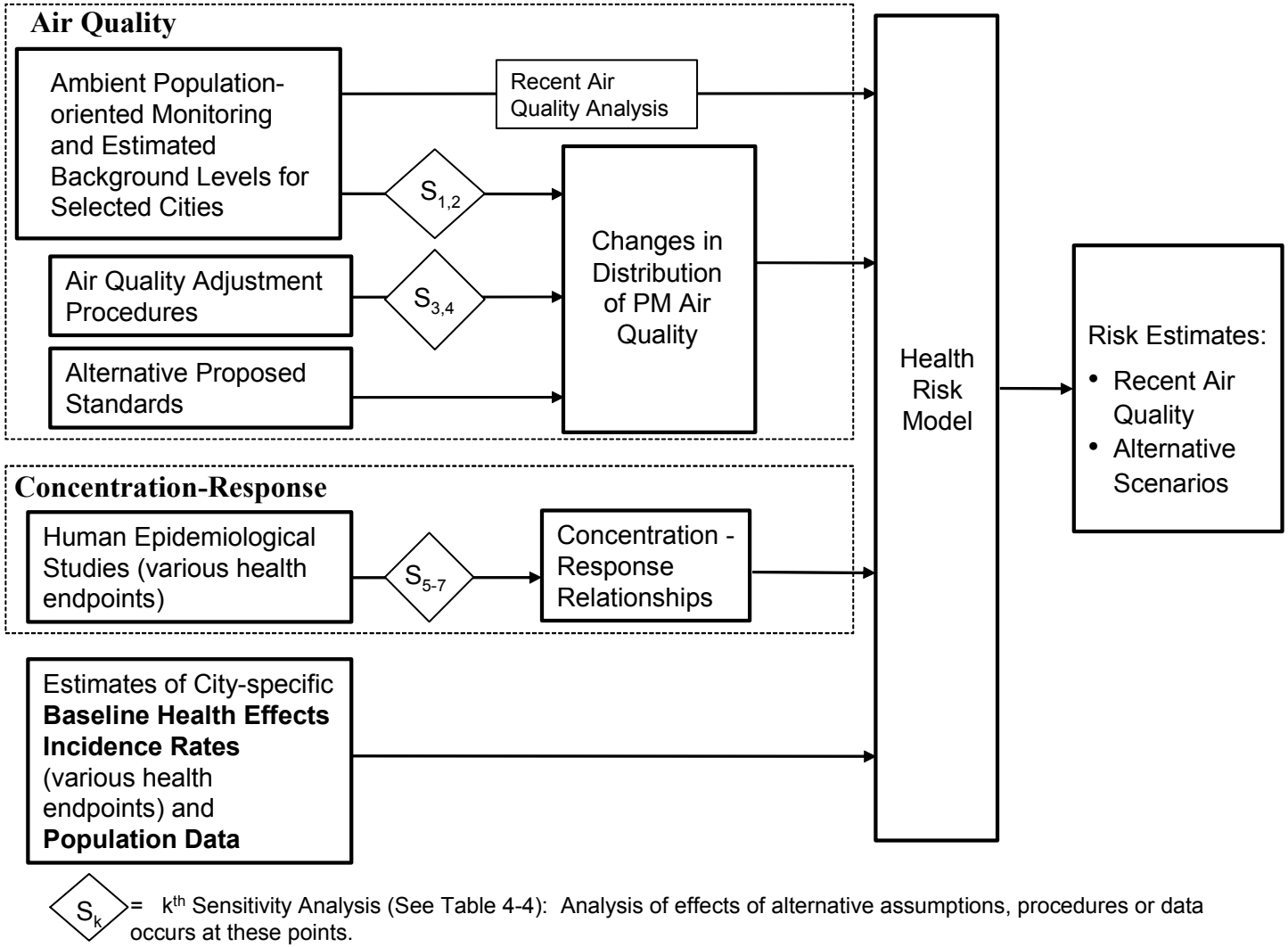

Figure 4-1. Major components of particulate matter health risk assessment.

Table 4-4. Sensitivity Analyses

Analysis Number (Figure 4-1)	PM Indicator	Component of the Risk assessment	Sensitivity Analyses or Comparisons
1	$PM_{2.5}$, $PM_{10-2.5}$	Air Quality	Sensitivity analyses of the effect of assuming different (constant) background PM levels
2	$PM_{2.5}$	Air Quality	Sensitivity analyses of the effect of assuming a constant background PM level versus a distribution of daily background levels
3	$PM_{2.5}$	Air Quality	Sensitivity analyses 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
4	$PM_{2.5}$	Air Quality	Sensitivity analyses 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
5	$PM_{2.5}$	Concentration-Response	Sensitivity analyses using an approach to estimate the possible impact of using a distributed lag concentration-response function
6	$PM_{2.5}$	Concentration-Response	Sensitivity analyses 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	$PM_{2.5}$	Concentration-Response	Sensitivity analysis of the impact on mortality associated with short-term exposure of using a multi-city concentration-response function compared to location-specific concentration-response functions from single-city studies

Source: Abt Associates (2005b)

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

or, alternatively,

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

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 relationship between a given difference in ambient $PM_{2.5}$ (or $PM_{10-2.5}$) levels and the corresponding difference in health effects. These are the key equations that combine air quality information, concentration-response information, and baseline health effects incidence and population information to estimate ambient $PM_{2.5}$ and $PM_{10-2.5}$ health risks.

For the first part of the risk assessment that characterizes risks associated with recent ambient PM concentrations, Δx is the difference between the recent ambient PM concentrations (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 policy-relevant background concentration or alternative cutpoints for short-term exposure endpoints or 7.5 $\mu g/m^3$ or the alternative cutpoints for long-term exposure mortality.[8] 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 ambient PM concentrations 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 ambient PM concentrations associated with just meeting the specified alternative standards.[9]

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

[8]As indicated previously, staff judges that the most relevant risk estimates are for those PM levels in excess of an estimated policy-relevant background and various cutpoints well above this background. As discussed more fully in Section 4.3.2.6, risk estimates for long-term exposure mortality are calculated in excess of the minimum of the lowest measured levels for the long-term exposure studies included in the risk assessment and in excess of two alternative cutpoint levels.

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

the number of days in the quarter for which air quality data was available.[10] This simple adjustment assumes that missing air quality data occur randomly with respect to level within a quarter and that the 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 epidemiologic studies used models estimating concentration-response 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 3.6.5.1), 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 $PM_{10\text{-}2.5}$ coefficients in the reported concentration-response functions is reflected in the confidence intervals provided for the risk estimates in sections 4.4 and 4.5. As discussed in greater detail in section 4.3.2.1, due to the significant uncertainty associated with whether or not the concentration-response relationships are approximately linear down to policy-relevant background, additional base case risk estimates are presented using alternative cutpoints for both short- and long-term exposure

[10]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.

mortality associated with $PM_{2.5}$ concentrations. As summarized in Table 4-4, a number of sensitivity analyses (S_1 through S_7 in Figure 4-1) were also conducted. The results of these sensitivity analyses are discussed in sections 4.4 and 4.5.

4.3.1 Air Quality Considerations

As illustrated in Figure 4-1, and noted earlier, air quality information required to conduct the PM risk assessment includes: (1) recent 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) air quality adjustment procedures to modify the recent data to reflect changes in the distribution of PM 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). Staff calculated $PM_{10-2.5}$ concentrations from co-located $PM_{2.5}$ and PM_{10} monitors that met the minimum observation cutoff criterion. Generally, the most recent year of PM data were used for each study area and PM indicator subject to meeting this requirement.

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. 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. The use of a composite monitor value to represent ambient PM air quality most closely matches the approach taken in the epidemiology studies that serve as the source of the concentration-response relationships used in the risk assessment. Table 4-5 provides a summary of the $PM_{2.5}$ and $PM_{10-2.5}$ ambient air quality data for the urban study areas, including the range of annual and 24-hr average statistics across monitors in each study area and 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.

4.3.1.1 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 initial 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 $PM_{10-2.5}$ analyses, the approximate mid-points of the annual average estimates for $PM_{10-2.5}$ background presented in section 2.6 were used for the initial base risk estimates.

Table 4-5. Summary of PM Ambient Air Quality Data for Risk Assessment Study Areas*

Area	Population (millions)	$PM_{2.5}$ ** ($\mu g/m^3$)				$PM_{10-2.5}$ ** ($\mu g/m^3$)			
		Annual Average		24-hr Average, 98thO%ile		Annual Average		24-hr , 98thO%ile	
		Range Across Monitors	Composite Monitor	Range Across Monitors	Composite Monitor	Range Across Monitors	Composite	Range Across Monitors	Composite Monitor
Boston, MA[a]	2.8	11.4-13.6	12.1	30.6-41.3	34.1				
Detroit, MI[b]	2.1	14.1-19.1	15.7	33.6-46.2	41.5	10.9-25.0	21.7	40.2-105.9	105.9
Los Angeles County, CA[c]	9.5	9.4-22.1	19.1	17.0-61.3	55.0				
Philadelphia County, PA[d]	1.5	13.2-16.1	14.3	35.6-42.3	38.4				
Phoenix, AZ[e]	3.1	9.2-10.9	10.4	22.7-35.3	28.9				
Pittsburgh, PA[f]	1.3	12.0-20.2	16.9	30.7-66.6	43.9				
San Jose, CA[g]	1.7	10.1-11.7	11.1	36.9-40.1	37.6				
Seattle, WA[h]	1.7	7.8-10.8	8.3	10.9-28.4	21.7	10.0-12.6	11.4	25.4-30.3	26.2
St. Louis[i]	2.5	13.0-17.5	14.0	30.5-40.8	30.6	10.1-14.9	12.0	24.2-33.3	24.1

*Based on air quality data for the year 2003, unless otherwise noted in footnotes below.
**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.
[c]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
[k]Includes King County
[i]Includes St. Louis, Franklin, Jefferson, St. Charles Counties in MO, Clinton, Madison, Monroe, and St. Clair Counties in IL and St. Louis City.

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 (S_1 in Figure 4-1), 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 (S_2 in Figure 4-1) (see Langstaff, 2005).

4.3.1.2 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 the current or alternative suites of standards. Estimated design values[11] (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. The amount of control has then been applied to a single year of data (2003, unless otherwise specified) to estimate risks for a single year.

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 (S_3 in Figure 4-1) has been conducted for three urban areas which satisfy the criteria for use of spatial averaging 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 epidemiologic studies. When assessing the risks associated with long-term exposures, which use concentration-response functions from epidemiologic 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 concentration-response functions from epidemiologic studies that consider the sequence of daily average concentrations, the distribution of 24-hour values that would occur upon just meeting a given 24-hour and/or annual PM standard has to be simulated.

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[11] A design value is a statistic that describes the air quality status of a given area relative to the level of the NAAQS. Design values are often based on multiple years of data, consistent with the specification of the NAAQS in Part 50 of the CFR. For example, for the base case analyses for the current $PM_{2.5}$ NAAQS, the 3-year averages (of annual means or 98th percentiles) based on the maximum monitor within an urban area are the design values.

There are many possible ways to create an alternative distribution of daily concentrations that just meets a specified set of PM standards. Both the 1996 assessment (see Abt Associates, 1996, section 8.2) and a more recent analysis of historical air quality data (see Appendix B in the TSD) 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.[12] We are using that approach in the base case here. The assessment also includes a sensitivity analysis (S_4 in Figure 4-1) 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 were 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.

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 example, for the current suite of $PM_{2.5}$ 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 based on design values.[13] 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 98^{th} 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 includes the 14 $\mu g/m^3$ annual standard combined with the current daily standard for one

[12] 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.

[13] See www.epa.gov/airtrends/pdfs/ for a discussion of how design values are calculated, noting in particular that concentrations flagged as natural events (e.g, high winds, wildfires, volcanic eruptions) or exceptional events (e.g., construction, prescribed burning) are not included in these calculations and that no regulatory decisions on attainment status have been made at this time based on these data.

location (i.e., Philadelphia) where there was a difference in the reduction required between daily standards of 40 and 65 $\mu g/m^3$.

4.3.2 Concentration-Response Functions

As indicated in Figure 4-1, another key component in the risk model is the set of concentration-response functions which provide estimates of the relationship between each health endpoint of interest and ambient PM concentrations. 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; 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. Once it had been determined that a health endpoint was to be included in the assessment, the assessment includes all estimates of response magnitude from studies judged suitable for inclusion in this assessment, including those which are not statistically significant. As discussed in section 4.2.2 above, one of the criteria for inclusion of studies in the risk assessment is that studies have enough sample size to provide a sufficient degree of precision. Effect estimates that are not statistically significant are used from studies judged suitable for inclusion in this assessment to avoid introducing bias into the estimate of the magnitude of the effect. Both single-pollutant and, where available, multi-pollutant, concentration-response functions are used from the studies listed in Tables 8A and 8B of the CD (see also Appendices 3A and 3B of this Staff Paper).

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, the PM risk assessment included concentration-response 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) concentration-response 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) concentration-response functions are somewhat

[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.

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 concentration-response relationships used in the PM risk assessment is provided in Appendix 4A of this 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 5^{th} and 95^{th} confidence intervals.

4.3.2.1 Linear and Nonlinear Models

In assessing or interpreting public health risk associated with exposure to PM, the form of the concentration-response relationships is a critical component. As discussed in Chapter 3 (section 3.6.6), staff recognizes that while there are likely biological thresholds in individuals for specific health responses, the available epidemiologic studies do not support or refute the existence of thresholds at the population level for either long-term or short-term PM exposures within the range of air quality observed in the studies. Thus, staff has concluded that it is appropriate to consider health risks estimated not only with the reported linear or log-linear concentration-response functions, but also with modified functions that incorporate alternative assumed cutpoints as surrogates for potential population thresholds.

For short-term exposure mortality and morbidity outcomes associated with $PM_{2.5}$ and $PM_{10-2.5}$, the initial base case uses linear or log-linear concentration-response models reported in the epidemiology studies. These concentration-response relationships are applied down to the estimated policy-relevant background concentration level. Generally, the lowest measured concentrations in the short-term exposure studies were relatively near or below the estimated policy-relevant background levels such that little or no extrapolation was required beyond the range of data in the studies. In the case of the long-term exposure mortality studies for $PM_{2.5}$ that have been included in the risk assessment, the lowest measured long-term levels were in the range 7.5 to 11 $\mu g/m^3$. Staff concludes that the initial base case scenario for this endpoint should include the reported linear models applied down to 7.5 $\mu g/m^3$, which is the lowest of the lowest measured levels in these long-term studies. Going down to an estimated policy-relevant background level for short-term exposure studies and to 7.5 $\mu g/m^3$ for long-term studies provides a consistent framework which facilitates comparison of risk estimates across urban locations within each group of studies and avoids significant extrapolation beyond the range of concentrations included in these studies.

Additional base case scenarios involved the use of alternative concentration-response functions. The approach used to develop the alternative functions incorporates a modified linear slope with an imposed cutpoint (i.e., an assumed threshold) that is intended to reflect an

inflection point in a typical non-linear, "hockeystick" shaped function, below which there is little or no population response. This approach also is a surrogate for a non-linear, sigmoidal-shaped function, in which the cut point is intended to reflect the inflection point at the lower end of the relationship, below which there is assumed to be little or no population response.

The staff recognizes that the alternative cutpoint analyses assume such a hockeystick shaped relationship, and it is appropriate to adjust the slope of the upper part of the hockeystick to be consistent with this assumption. If the data in the original study actually supported a hockeystick model better than a log-linear model, then the slope of the log-linear fitted relationship reported by the study would have understated the degree to which PM is associated with mortality or morbidity above the cutpoint, as shown in Figure 4-2. This rationale applies equally in the case of long- and short-term exposure mortality and morbidity. Therefore, the slope of the upward-sloping portion of the hockeystick should not use the slope reported for the concentration-response relationship but should be adjusted upward.

For the base case scenarios involving alternative cutpoints, the slope of the concentration-response relationship has been adjusted assuming that the upward-sloping portion of the hockeystick would be the slope estimated in the original epidemiologic study adjusted by the inverse of the proportion of the range of PM levels observed in the study that was above the cutpoint. Staff believes that this simple slope adjustment approach represents a reasonable approach to illustrate the potential impact of possible non-linear concentration-response relationships. A more definitive evaluation of the effect of alternative cutpoints and non-linear models is a subject that should be explored in much needed further research.

Based on the staff evaluation contained in section 3.6.6, a cutpoint of 20 $\mu g/m^3$ was selected as the highest value for inclusion in base case scenarios for short-term exposure mortality for $PM_{2.5}$ and short-term exposure morbidity for $PM_{10-2.5}$. Two additional alternative cutpoints, 10 and 15 $\mu g/m^3$, also were selected to be included in base case scenarios for these short-term exposure health outcomes, so as to span the range between the initial cutpoint (i.e., estimated policy-relevant background) and the upper cutpoint value at roughly 5 $\mu g/m^3$ intervals. With regard to long-term exposure mortality associated with $PM_{2.5}$ exposures, staff selected 12 $\mu g/m^3$ as the highest value for an alternative cutpoint based on the following two considerations: 1) the confidence intervals in the ACS extended study (Pope et al., 2002) begin to expand significantly starting around 12 to 13 $\mu g/m^3$ (see Figure 3-4) indicating greater uncertainty about the shape of the reported concentration-response relationship at and below this level and 2) it is unlikely that the relationship is non-linear near the reported mean concentration levels in the long-term exposure studies (e.g., 14 $\mu g/m^3$ in the ACS extended study). An additional alternative cutpoint of 10 $\mu g/m^3$ has been included as a base case scenarios for long-term exposure mortality, representing an approximate midpoint value between the cutpoints already selected. Results of these analyses are discussed below in section 4.4 and 4.5.

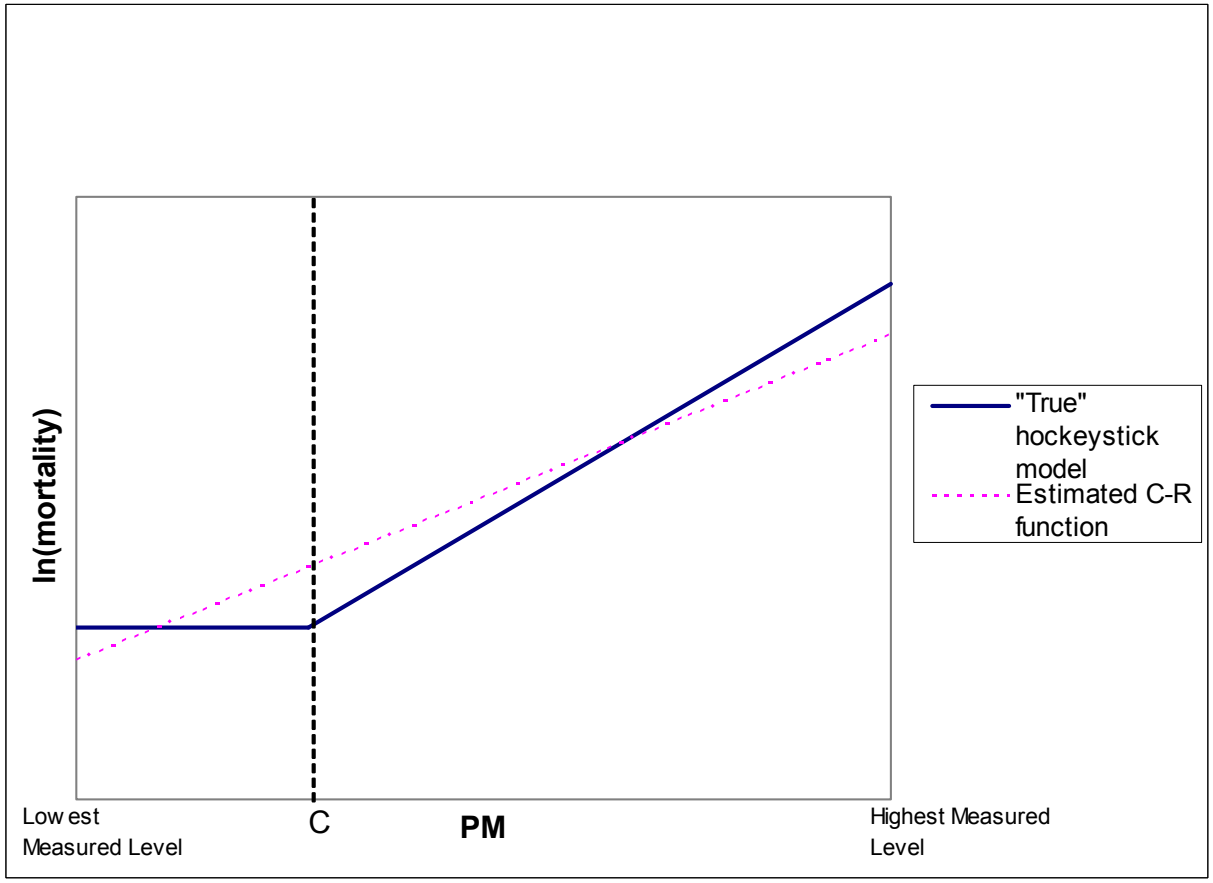

Figure 4-2. Relationship between estimated log-linear concentration-response relationship and hockeystick model with cutpoint C.
Source: Abt Associates (2005b)

4.3.2.2 Single and Multi-City Models

As described in section 4.2, staff have selected urban areas based on where epidemiologic studies have estimated concentration-response relationships. This approach avoids uncertainties associated with estimating health risks for an area based on a relationship developed for a different location. Staff has included both single-city and multi-city concentration-response functions in the current assessment. As discussed in section 3.3.1.1 and in the CD, there are a number of advantages of using concentration-response relationships obtained from multi-city studies which combine data from a number of locations that may vary in climate, sources and concentrations, and other potential risk factors. These advantages include, but are not limited to: (1) more precise effect estimates due to larger data sets, (2) greater consistency in data handling and model specification that can eliminate city-to-city variation due to study design, and (3) less likelihood of publication bias or exclusion of reporting of negative or nonsignificant findings. However, at this time very few multi-city studies have been carried out in the U.S. that report concentration-response relationships for $PM_{2.5}$ and/or $PM_{10-2.5}$. In the one instance where both single- and multi-city concentration-response relationships are available for the locations included in the risk assessment (e.g., the Six Cities study), risk estimates have been developed using both the single- and multi-city concentration-response relationships. In addition, a sensitivity analysis (S_7 in Figure 4-1) has been conducted to examine the potential impact on short-term exposure mortality of using a single multi-city concentration-response function from the Six Cities study (Schwartz, 2003b) across five of the $PM_{2.5}$ locations included in the risk assessment compared to use of location-specific concentration-response functions from single-city studies. The results of this sensitivity analysis are presented in section 4.4.3.2.

4.3.2.3 Single and Multi-Pollutant Models

For several of the epidemiologic studies from which concentration-response relationships for the PM risk assessment were obtained, concentration-response 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 concentration-response functions are based on single-pollutant models. However, the CD finds that associations for various PM indices with mortality or morbidity are robust to confounding by co-pollutants (CD, p.9-37). 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 for the assessment.

4.3.2.4 Single, Multiple, and Distributed Lag Functions

The question of lags and the problem 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 Staff Paper. As noted in those discussions, it is important to consider the pattern of results that is seen across the series of lag periods. In staff's judgment, observation of a consistent pattern of results across adjacent lags in a study supports use of a study in the risk assessment. In contrast, where an inconsistent pattern of results has been observed, staff judges that it would be inappropriate to include results from such studies in the risk assessment.

As noted in section 3.6.5.1, staff concludes that it is appropriate to use single-day lag period results for the risk assessment. When a study reports several single lag models, unless the study authors identify a "best lag," the following lag models were included in the risk assessment based on the assessment in the CD and in section 3.6.5.1:

- 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.

When there is an observed pattern showing effects across different lags, use of any 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 section 3.6.5.1, there is recent evidence (Schwartz, 2000b, reanalyzed in Schwartz, 2003b), that the relationship 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 –1, day –2 and so on). If this is the case, a model that includes only a single lag (e.g., a 0-day lag or a 1-day lag) is likely to understate the total impact of PM. 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}.

The risk assessment includes a sensitivity analysis (S_5 in Figure 4-1) which examines 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 has been included to provide a very rough sense of the possible underestimation of risk due to use of single-day lags models.

4.3.2.5 Alternative Short-Term Exposure Model Specifications

As discussed in section 3.6.3, time series studies investigating health effects associated with PM have used a range of alternative model specifications. For the risk assessment base case analyses, only the concentration-response functions using the more stringent convergence criterion, denoted "GAM (stringent), have been included to provide a consistent basis for comparison across studies and locations. In fact, most studies use a limited number of modeling approaches, in part to avoid producing an unwieldy and confusing number of different estimates. One study that used a wide variety of modeling approaches is the Moolgavkar (2003) study which is the basis for the short-term $PM_{2.5}$ exposure mortality and morbidity estimates for Los Angeles. This study included two different versions of the "GAM stringent" approach, one with 30 degrees of freedom and the other with 100 degrees of freedom, as well as models using GLM. The risk assessment includes a sensitivity analysis to examine the potential impact of alternative model specifications on estimates of short-term exposure morbidity and mortality in Los Angeles based on the results from Moolgavkar (2003).

4.3.2.6 Long-term Exposure Models

The available long-term exposure mortality concentration-response 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 (a) the Six Cities study cohort, referred to here as Krewski et al. (2000) - Six Cities, and (b) 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).

As explained in section 3.6.5.4, three different indicators of long-term $PM_{2.5}$ exposure were considered in the extended ACS study; and staff have selected the concentration-response function associated with an average of the 1979-1983 and 1999-2000 $PM_{2.5}$ ambient concentrations to use in the current risk assessment. The assessment includes a sensitivity analysis (S_6 in Figure 4-1) which examines the potential 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.

4.3.3 Baseline Health Effects Incidence Rates and Population Estimates

As illustrated in Figure 4-1, the most common health risk model expresses the reduction in health risk (Δy) associated with a given reduction in PM concentrations (Sx) 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 recent 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[15] and are summarized in Table 4-6. 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 4-7 and are expressed as a rate per 100,000 general population.[16]

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 assessment locations.[17] Baseline hospitalization rates used in each $PM_{2.5}$ and $PM_{10\text{-}2.5}$ risk assessment location are summarized in Table 4-8 and are expressed a rate per 100,000 general population. For respiratory symptoms in children, the only available estimates of baseline incidence rates were from the studies that estimated the concentration-response 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.

4.3.4 Characterizing Uncertainty and Variability

An important issue associated with any population health risk assessment is the characterization of uncertainty and variability. *Uncertainty* refers to the lack of knowledge regarding 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

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

[16]Since the baseline incidence rates are expressed in terms of cases per 100,000 general population, the general population estimates have been used in combination with these rates to generate the baseline incidence in each location for various effects in calculating the risk estimates.

[17]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. Use of the annual discharge rate is based on the assumption 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 4-6. 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]	2806000	283,000 (10%)	1,903,000 (68%)	1,673,000 (60%)	---		---	---
Detroit[2]	2061000		---	1,153,000 (56%)	---	249,000 (12%)	---	---
Los Angeles[3]	9519000		---	5,092,000 (53%)	---	927,000 (10%)	---	---
Philadelphia[4]	1518000		---	852,000 (56%)	---	---	---	---
Phoenix[5]	3072000		---	1,684,000 (55%)	---	359,000 (12%)	---	---
Pittsburg[6]	1282000		---	814,000 (64%)	---	---	1,166,000 (91%)	116,000 (9%)
San Jose[7]	1683000		---	965,000 (57%)	---	---	---	---
Seattle[8]	1737000		---	1,044,000 (60%)	1,555,000 (90%)	---	---	---
St. Louis[9]	2518000	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 table 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 Mortality Rates for 2001 for $PM_{2.5}$ Risk Assessment Locations

Health Effect	Boston[1]	Detroit[2]	Los Angeles[3]	Philadelphia[4]	Phoenix[5]	Pittsburgh[6]	San Jose[7]	St. Louis[8]	Seattle[9]	National Average
A. Mortality Rates Used in Risk Analysis for Short-Term Exposure Studies[a,b] (deaths per 100,000 general population/year)										
Non-accidental (all ages): ICD-9 codes < 800	776	916	581	1070	---	---	494	869	---	791
Non-accidental (75+): ICD-9 codes < 800	---	---	---	---	---	761	---	---	---	469
Non-accidental (<75): ICD-9 codes < 800	---	---	---	---	---	399	---	---	---	322
Cardiovascular (all ages): ICD-9 codes: 390-459	---	416	---	---	---	---	206	---	---	328
Cardiovascular (all ages): ICD-9 codes: 390-448	---	---	---	418	---	---	---	---	---	324
Cardiovascular (65+): ICD-9 codes: 390-448	---	---	---	---	211	---	---	---	---	273
Cardiovascular (all ages): ICD-9 codes: 390-429	---	---	207	---	---	---	---	---	---	252
Ischemic Heart Disease (all ages): ICD-9 codes: 410-414	122	---	---	---	---	---	---	206	---	152
Respiratory (all ages): ICD-9 codes: 11, 35, 472-519, 710.0, 710.2, 710.4	---	---	---	---	---	---	51	---	---	80
Respiratory (all ages): ICD-9 codes: 460-519	---	72	---	---	---	---	---	---	---	79

Health Effect	Boston[1]	Detroit[2]	Los Angeles[3]	Philadelphia[4]	Phoenix[5]	Pittsburgh[6]	San Jose[7]	St. Louis[8]	Seattle[9]	National Average
COPD without Asthma (all ages): ICD-9 codes: 490-492, 494-496	**36**	**---**	**---**	**---**	**---**	**---**	**---**	**39**	**---**	**42**
Pneumonia (all ages): ICD-9 codes: 480-487	**26**	**---**	**---**	**---**	**---**	**---**	**---**	**27**	**---**	**22**
B. Mortality Rates Used in Risk Analysis for Long-term Exposure Studies[a,b] (deaths per 100,000 general population/year)										
Total mortality (25+): ICD-9 codes: all	**803**	**---**	**---**	**---**	**---**	**---**	**---**	**905**	**---**	**822**
Total mortality (30+): ICD-9 codes: all	**797**	**937**	**591**	**1100**	**676**	**1189**	**499**	**897**	**637**	**814**
Cardiopulmonary Mortality (25+): ICD-9 codes: 400-440, 485-495	**297**	**---**	**---**	**---**	**---**	**---**	**---**	**391**	**---**	**341**
Cardiopulmonary Mortality (30+): ICD-9 codes: 401-440, 460-519	**347**	**468**	**313**	**489**	**313**	**573**	**247**	**439**	**287**	**391**
Lung Cancer Mortality (30+): ICD-9 code: 162	**55**	**64**	**33**	**72**	**42**	**78**	**30**	**61**	**44**	**55**

*The epidemiologic studies used in the risk assessment reported causes of mortality using the 9th revision of the International Classification of Diseases (ICD-9) codes. However, the 10th revision has since come out, and baseline mortality incidence rates for 2001 shown in this table use ICD-10 codes. The groupings of ICD-9 codes used in the epidemiologic studies and the corresponding ICD-10 codes used to calculate year 2001 baseline incidence rates is given in Exhibit 5.4 of the TSD (Abt Associates, 2005b).

[a] Mortality figures were obtained from CDC Wonder for 2001. See http://wonder.cdc.gov/.

[b] Mortality rates are presented only for the locations in which the concentration-response functions were estimated. All incidence rates are rounded to the nearest unit. Mortality rates for St. Louis may be slightly underestimated because some of the mortality counts in the smaller counties were reported as missing in CDC Wonder.

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

Table 4-8. 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**	**---**	**---**

* Hospitalization rates are presented only for the locations in which the concentration-response 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.

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 concentration-response 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 have been discussed throughout section 4.3.

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, would be desirable, but in the staff's judgment 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 variable of interest that is inherent and cannot be reduced through further research. For example, there may be variability among concentration-response 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 concentration-response 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).

Key Uncertainties and Assumptions

The following briefly summarizes the major sources of 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 than for $PM_{2.5}$ due to the much smaller health effects data base and greater concerns about exposure measurement error. 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 concentration-response relationships. In estimating the concentration-response relationships, there are uncertainties: (1) surrounding estimates of PM coefficients in concentration-response functions used in the assessment, (2) concerning the specification of the concentration-response model (including the shape of the concentration-response relationship) and whether or not a population threshold or non-linear relationship exists within the range of concentrations examined in the studies, and (3) related to the extent to which concentration-response functions derived from studies in a given location and time when PM concentrations were higher provide accurate representations of the concentration-response relationships for the same location with lower annual and daily PM concentrations. For the few instances where multi-city PM concentration-response functions are included in the base case analyses (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 concentration-response functions from multiple locations to the specific location selected for the risk assessment.[18] Statistical uncertainty, based on the standard errors reported in the epidemiologic studies, is incorporated in the risk assessment and is discussed below. Base case risk estimates incorporating various cutpoints have been included in the risk assessment to reflect the uncertainty about whether or not population thresholds

[18]A concentration-response function derived from a multi-cities study may not provide an accurate representation of the concentration-response 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.

or non-linear concentration-response relationships might exist at the lower range of ambient $PM_{2.5}$ and $PM_{10-2.5}$ concentrations. As discussed previously (see section 4.3) several sensitivity analyses have been presented related to uncertainties in the concentration-response relationships.

- 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 has been observed.

- Adjustment of air quality distributions to simulate just meeting specified 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 one of the base case scenarios includes estimating risks in excess of estimated policy-relevant background, uncertainty about background concentrations contributes to uncertainty about the risk estimates. As discussed previously, the assessment includes sensitivity analyses examining the impact of alternative constant and varying daily background levels on the risk estimates.

- Baseline incidence rates and population data. There are uncertainties related to: (1) the extent to which baseline incidence rates, age distribution, and other relevant 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). Spatial 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 concentration-response 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.[19] In situations where the point estimate for a concentration-response 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 concentration-response relationships in the epidemiologic 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 or staff assessment (see Chapter 3) find that the overall weight of the evidence supports the conclusion that $PM_{2.5}$ is likely causally related. Also, for most of the health endpoints and locations included in the risk assessment, this assessment uses the concentration-response functions derived from epidemiologic 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 concentration-response functions developed in one location to a different location. However, the use of functions from single-city studies does suffer the disadvantage of introducing possible publication bias and single-city studies generally have lower precision than larger multi-city studies.

In summary, the key assumptions on which the current PM risk assessment is based include the following:

- The relationship between $PM_{2.5}$ and PM_{10-25} and the health endpoints included in the assessment are causal;
- Concentration-response models are appropriately specified (including the functional form and lag structure);
- Baseline incidence rates and population size and age distributions have not changed appreciably from those used in the assessment;
- For short-term endpoints, that obtaining average daily incidence rates from annual baseline incidence rates and using them to estimate daily incidences associated with exposure to PM does not bias the estimates;

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

- The distribution of PM concentrations on missing days is essentially the same as the distribution on days for which we have PM data;

- The estimated policy-relevant background concentrations for $PM_{2.5}$ and $PM_{10-2.5}$ are appropriate for each urban area included in the analysis;

- A single year of air quality data is appropriate to characterize risks associated with recent air quality levels and just meeting specified standards;

- Proportional rollback of concentrations over an estimated policy-relevant background appropriately reflects the air quality distribution when specified standards would just be met.

4.4 $PM_{2.5}$ RISK ESTIMATES

Several "base case" analyses for $PM_{2.5}$ are presented in this section and include risk estimates associated with a recent year of air quality (generally, 2003), air quality adjusted to just meet the current $PM_{2.5}$ standards, and air quality adjusted to simulate just meeting alternative $PM_{2.5}$ standards. The initial base case analyses for the recent air quality and just meeting the current $PM_{2.5}$ standards scenarios include concentration-response models that extend down to estimated policy-relevant background for short-term exposure health outcomes (i.e., equivalent to setting a cutpoint at estimated policy-relevant background) and extend down to 7.5 $\mu g/m^3$ for long-term exposure mortality (equivalent to setting a cutpoint at 7.5 $\mu g/m^3$). For this initial set of base case analyses, the slope of the concentration-response function is based on that obtained directly from the published studies. A variety of models (single city with different lags, single city vs. multi-city, single pollutant vs. multi-pollutant) and health outcomes (mortality, hospital admissions, respiratory symptoms) are included using this set of initial base case analyses.

Following this initial set of base case analyses, risk estimates, additional base case estimates are developed only for non-accidental short-term exposure mortality (or if not available, cardiovascular mortality) and for all-cause mortality with long-term exposure for each study area employing the same cutpoints indicated above, as well as several additional alternative cutpoints. For the additional alternative cutpoints, the slope of the concentration-response function has been modified based on a simple hockeystick model (see discussion in section 4.3.2.1). Staff considers the initial set of base case analyses, as well as the analyses estimating health risks associated with alternative cutpoints, as being part of the complete set of base case analyses.

4.4.1 Recent Air Quality

4.4.1.1 Base Case Risk Estimates Above Initial Cutpoint

The base case risk estimates associated with recent $PM_{2.5}$ concentrations in excess of policy-relevant background levels for short-term exposure outcomes and in excess of 7.5 $\mu g/m^3$

for long-term exposure mortality are presented in a series of figures in this section. The risk estimates are expressed both in terms of percent of total incidence (the top panel in each figure) and cases per hundred thousand general population (the bottom panel in each figure). The percent of total incidence provides information about what portion of total incidence for a given health outcome is estimated to be due to exposure to ambient $PM_{2.5}$ levels. Expressing risk in terms of cases per hundred thousand general population provides a metric that takes into account the variation in population size for each of the urban areas. For each series of estimates, a point estimate is provided along with 95% confidence intervals.[20] Additional detailed tables which present the estimated incidence (both as the number of effects and as a percentage of total incidence) for each risk assessment location are included in the TSD. Risk estimates in a given assessment location are presented only for those health endpoints for which there is at least one acceptable concentration-response function reported for that location. Therefore, the set of health effects shown in the figures varies for the different locations.

Figures 4-3 through 4-7 present the $PM_{2.5}$ risk estimates across the various assessment locations associated with recent concentrations in excess of an initial cutpoint. For short-term exposure outcomes, this initial cutpoint is the estimated policy-relevant background. For long-term exposure mortality the initial cutpoint is 7.5 $\mu g/m^3$, the lowest of the lowest measured levels used in the long-term exposure studies included in the risk assessment. Figure 4-3 compares risk estimates for mortality associated with short-term (i.e., 24-hour) exposure to $PM_{2.5}$ above policy-relevant background using single-pollutant, single-city models. The point estimates are in the range from about 0.8 to 2.5% of total non-accidental mortality incidence. In terms of cases per hundred thousand general population, the point estimates range from about 4 to 13. The differences in estimates across locations is due to several factors including differences in recent air quality levels, use of different concentration-response functions from various single-city studies, and variation in baseline incidence rates. Differences in concentration-response functions across the various single-city studies may reflect methodological differences between studies and/or real differences due to differences in the population and extent of population exposure to ambient $PM_{2.5}$ concentrations. In addition, there are significant differences in baseline incidence rates among the cities which also contribute to city-to-city variation. For example, as shown in Table 4-7, the baseline mortality rate for non-accidental mortality (all ages) is nearly twice as large in Philadelphia as in Los Angeles.

Figure 4-4 compares risk estimates for non-accidental and cause-specific mortality associated with short-term exposure to $PM_{2.5}$ above policy-relevant background based on single

[20] As noted above, in some cases, where the lower confidence limit of the concentration-response 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 concentration-response relationships, they do not at all suggest that risk reductions would be associated with an increase in PM levels.

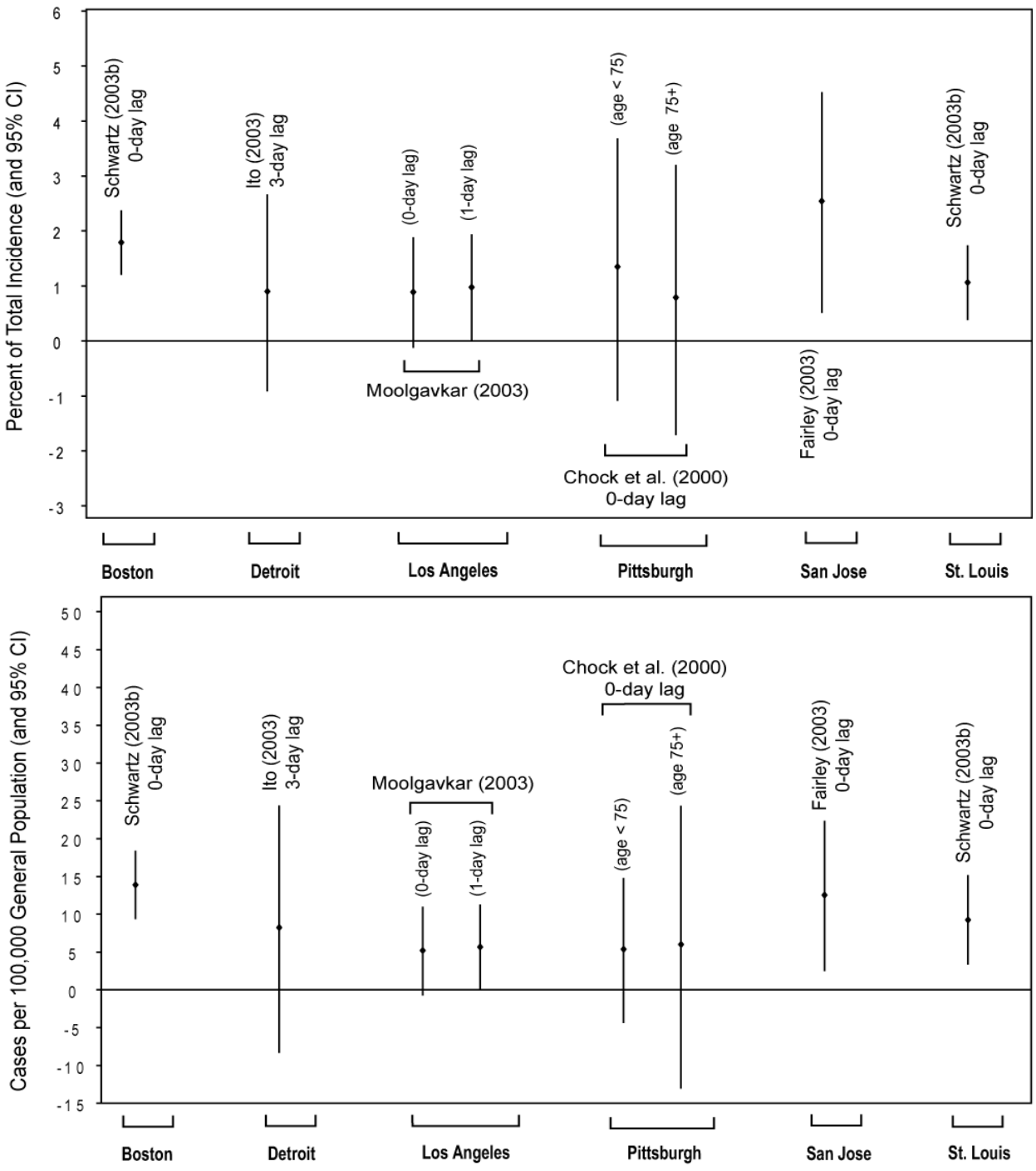


Figure 4-3. Estimated annual percent (top panel) and cases per 100,000 general population (bottom panel) of total (non-accidental) mortality associated with short-term exposure to $PM_{2.5}$ above background (and 95 percent confidence intervals): single-pollutant, single-city models.
Source: Abt Associates (2005b)

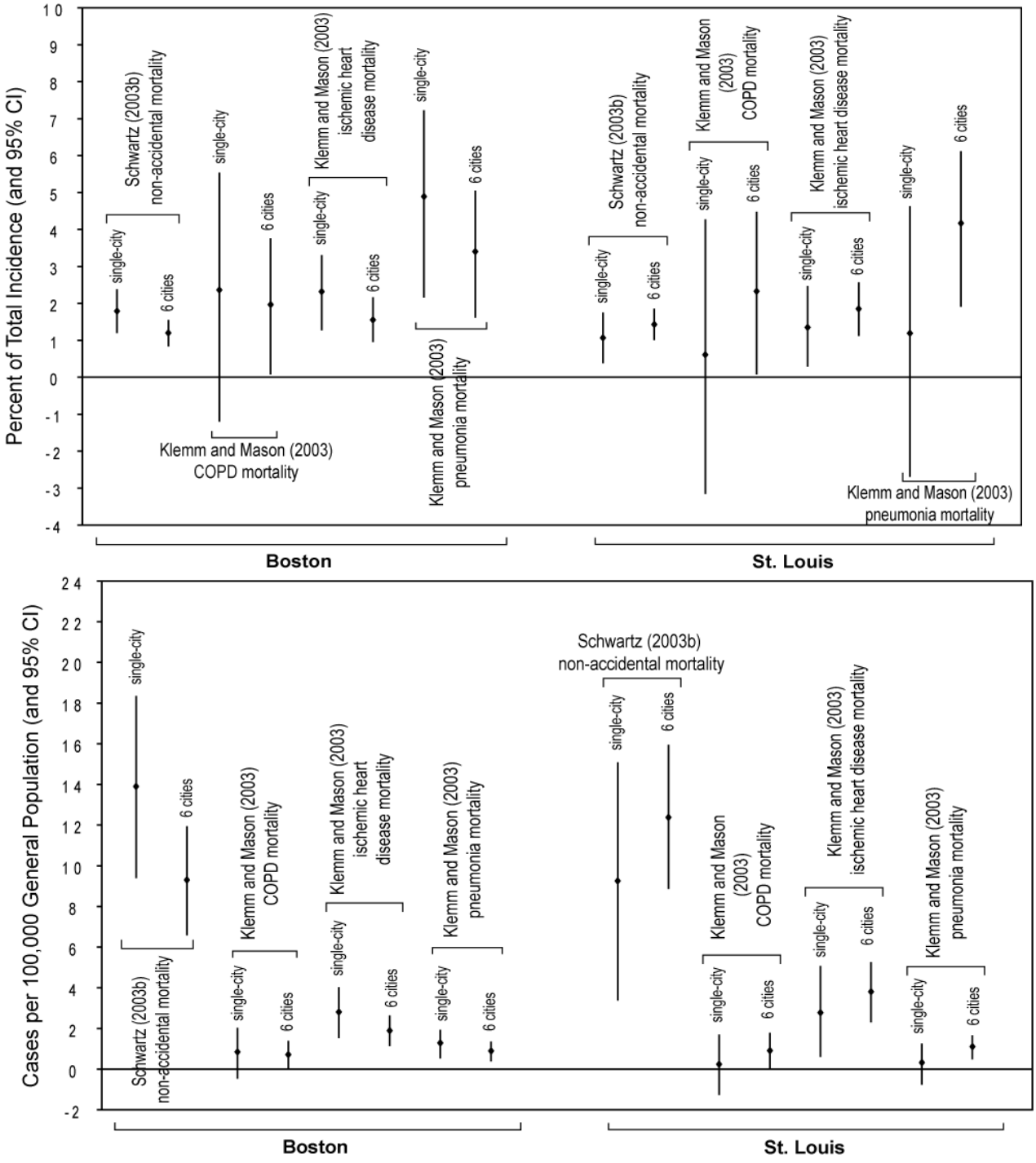


Figure 4-4. Estimated annual percent (top panel) and cases per 100,000 general population (bottom panel) of total (non-accidental) mortality associated with short-term exposure to $PM_{2.5}$ above background (and 95 percent confidence intervals): single-city versus multi-city models.
Source: Abt Associates (2005b)

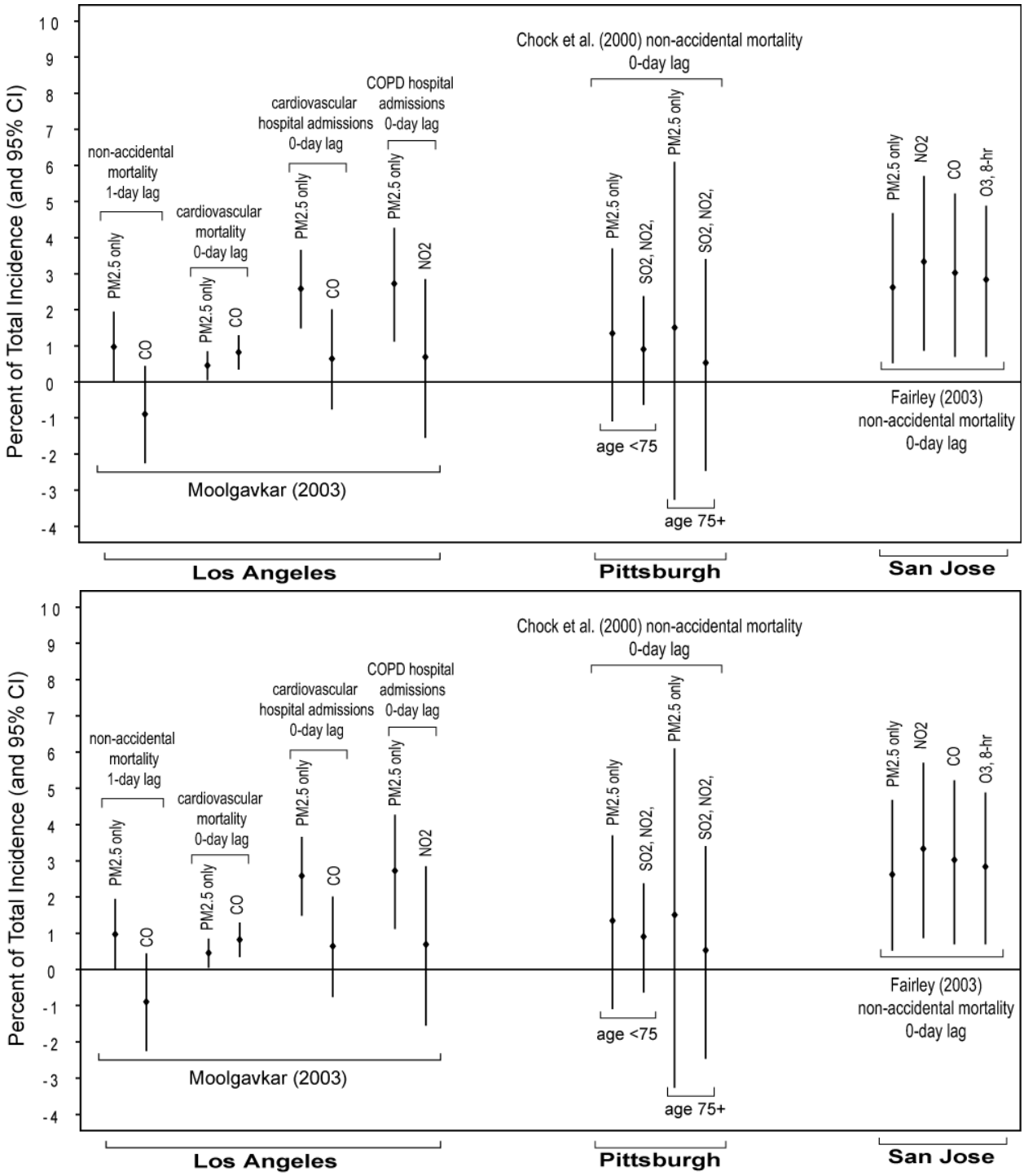


Figure 4-5. Estimated annual percent (top panel) and cases per 100,000 general population (bottom panel) of health effects associated with short-term exposure to $PM_{2.5}$ above background (and 95 percent confidence intervals): single-pollutant versus multi-pollutant models.

Source: Abt Associates (2005b)

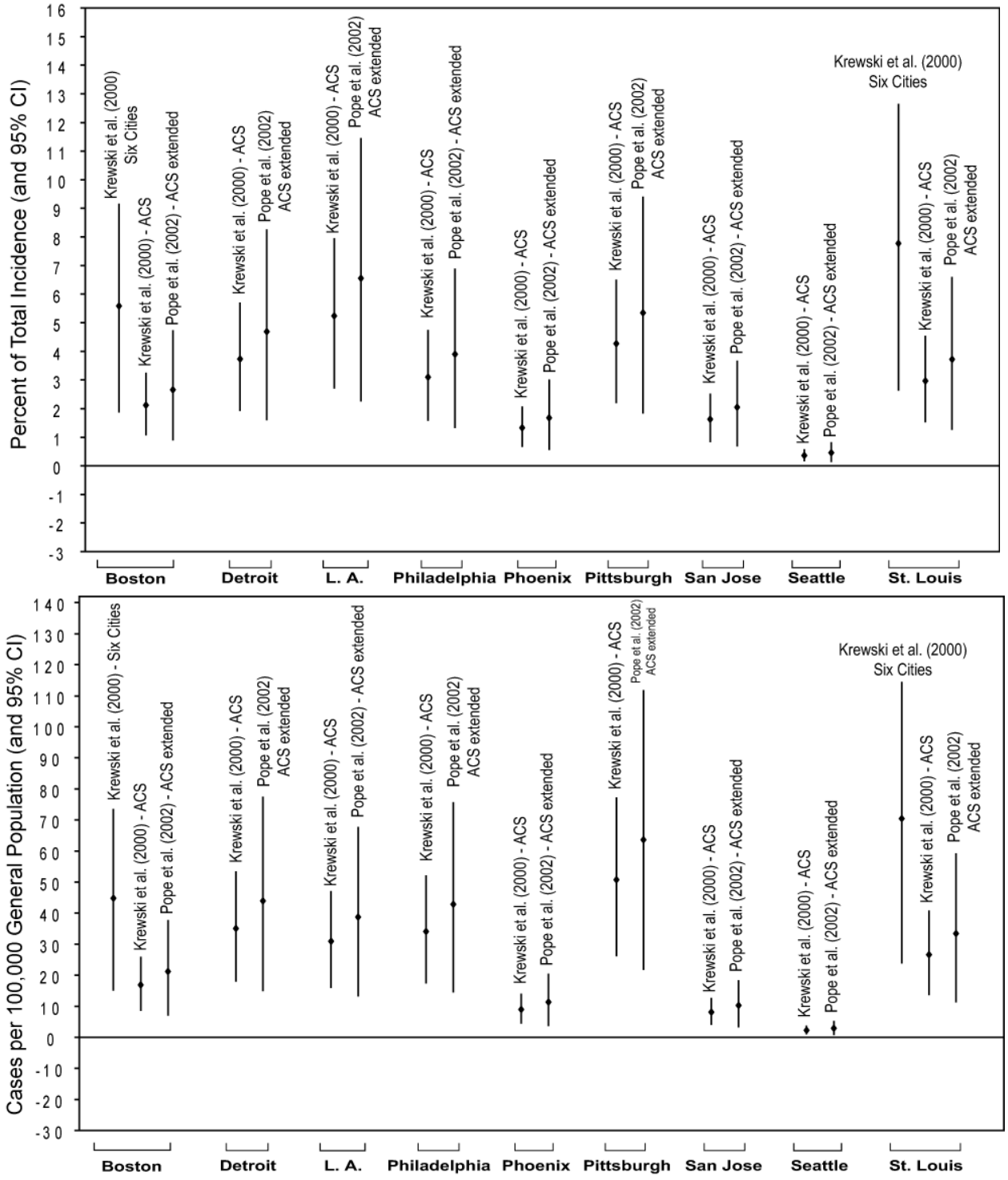


Figure 4-6. Estimated annual percent (top panel) and cases per 100,000 general population (bottom panel) of total (non-accidental) mortality associated with long-term exposure to $PM_{2.5}$ above 7.5 μg/m^3 (and 95 percent confidence intervals): single-pollutant models. Source: Abt Associates (2005b)

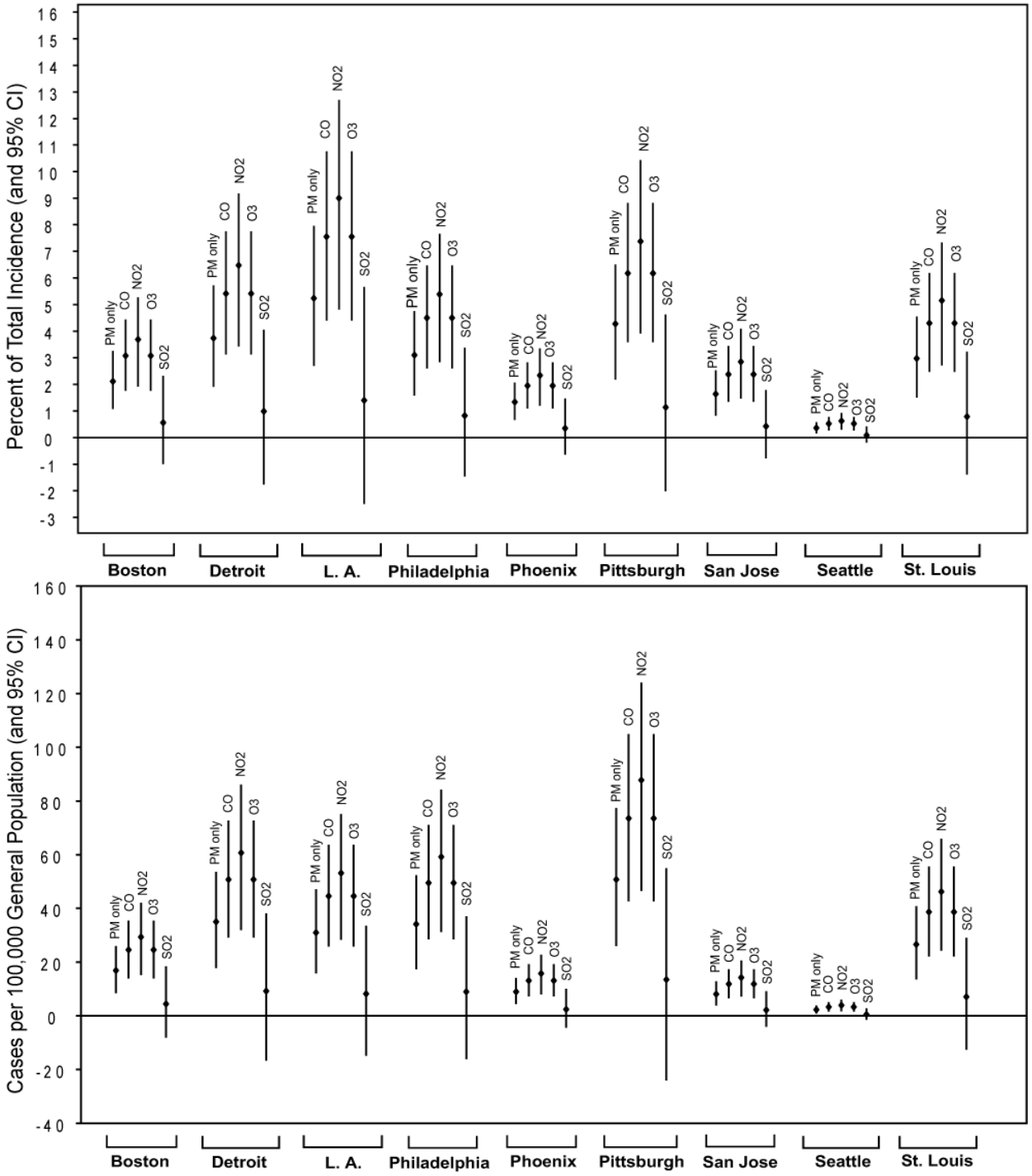


Figure 4-7. Estimated annual percent (top panel) and cases per 100,000 population (bottom panel) of total (non-accidental) mortality associated with long-term exposure to $PM_{2.5}$ above 7.5 μg/m^3 (and 95 percent confidence intervals): single-pollutant and multi-pollutant models (based on Krewski et al. (2000) - ACS study). Source: Abt Associates (2005b)

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 the reverse being observed in St. Louis.

Figure 4-5 compares risk estimates based on single-pollutant versus multi-pollutant concentration-response models provided in the epidemiologic studies for $PM_{2.5}$ short-term exposure health endpoints above policy relevant background. As noted earlier, the multi-pollutant models reflect where $PM_{2.5}$ and one or more other measured gaseous co-pollutants (i.e., O_3, NO_2, SO_2, CO) were entered into the health effects model. 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 $PM_{2.5}$.

Figures 4-6 and 4-7 show risk estimates for mortality related to long-term (i.e., annual average) exposure to $PM_{2.5}$ levels above 7.5 $\mu g/m^3$ 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 about 0.5% in Seattle to as high as about 6.5% 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 somewhat lower in all of the study areas (ranging from about 0.2 to 5% in terms of percent of total incidence). For Boston and St. Louis, the point estimates based on the Six Cities study (Krewski et al., 2000) are more than twice as large as the estimates based on the ACS extended study. As noted in Chapter 3 (section 3.5.2), the strongest associations between $PM_{2.5}$ and mortality in the ACS study were among the less educated participants who form a relatively small portion of the total study cohort. If the education distribution were adjusted to reflect the education distribution in the general U.S. population, the summary effect estimate would increase and this would narrow the difference observed in the risk estimates between the ACS and Six Cities studies. As shown in Figure 4-7, 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.[21]

4.4.1.2 Base Case Risk Estimates Above Various Cutpoints

As discussed above, the assessment includes additional base case annual short- and long-term mortality risk estimates associated with recent air quality levels assuming not only the

[21] The addition of a second pollutant reduced the number of cities available for estimating the concentration-response 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.

initial cutpoint included in the previous section but the alternative cutpoint levels as well. For short-term exposure mortality, a single non-accidental mortality function has been included, except for Philadelphia and Phoenix where cardiovascular mortality has been used since a suitable non-accidental mortality concentration-response function is not available. For long-term exposure mortality, a single all cause mortality concentration-response function has been included based on the ACS-extended study (Pope et al., 2002). Tables 4-9 and 4-10 present the annual health risks for short- and long-term exposure mortality, respectively. Both tables present the risk estimates expressed in terms of incidence (i.e., cases), cases per 100,000 general population, and percent of total incidence, along with 95 percent confidence intervals for each of these risk metrics.

4.4.1.3 Risk Estimates from Sensitivity Analyses

As discussed previously, several sensitivity analyses have been carried out to provide some perspective on the impact of various assumptions and uncertainties on the health risk estimates (see Table 4-4 above for a summary of different types of sensitivity analyses). Most of these sensitivity analyses have been conducted in each of the study areas and use the initial set of cutpoints (i.e., policy relevant background for short-term exposure outcomes and 7.5 $\mu g/m^3$ for long-term exposure mortality). The complete results of the sensitivity analyses are included in the TSD. In some cases, sensitivity analyses have been conducted only in one location due to data constraints (for example Los Angeles is the only city where the sensitivity analysis uses alternative concentration-response model specifications since, as explained in section 4.3.2.5 above, it is the only study that presents results for a wide range of alternative model specifications).

Alternative Background Levels

As explained earlier, for purposes of informing decisions about the PM NAAQS, we are interested in PM-related risks due to concentrations over policy-relevant background levels, where background excludes anthropogenic emissions of PM and its precursors in the U.S., Canada, and Mexico (discussed in section 2.6). One set of sensitivity analyses has examined the impact of using the lower and upper end of the range of estimated background concentrations provided in section 2.6. In the nine locations, using the upper- and lower-end of the range of estimated background generally has 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. Alternative estimated $PM_{2.5}$ background levels have no impact on long-term exposure mortality in any of the $PM_{2.5}$ locations, because the range of alternative policy-relevant background levels is lower than the lowest cutpoint of 7.5 $\mu g/m^3$ used for these analyses.

A sensitivity analysis also has been conducted that focuses on the impact of using a varying estimated $PM_{2.5}$ background concentration instead of the fixed level used in each study area in the base case assessment. Staff developed a Monte Carlo simulation approach to

Table 4-9. Estimated Annual Health Risks of Short-Term Exposure Mortality Associated with Recent $PM_{2.5}$ Concentrations Assuming Various Cutpoint Levels*

Urban Area	Study	Type	Ages	Lag	Incidence Associated with $PM_{2.5}$ Assuming Various Cutpoint Levels (95% Confidence Interval) Incidence per 100,000 General Population (95% Confidence Interval) Percent of Total Incidence (95% Confidence Interval)			
					Policy Relevant Background** =2.5 or 3.5 µg/m^3	Cutpoint*** =10 µg/m^3	Cutpoint*** =15 µg/m^3	Cutpoint*** =20 µg/m^3
Boston	Schwartz (2003b) [reanalysis of Schwartz et al. (1996)]	Non-accidental	all	mean of lag 0 & 1 day	390 (265 - 514)	173 (118 - 228)	82 (56 - 109)	41 (28 - 53)
					14 (9 - 18)	6 (4 - 8)	3 (2 - 4)	1 (1 - 2)
					1.8% (1.2% - 2.4%)	0.8% (0.5% - 1.1%)	0.4% (0.3% - 0.5%)	0.2% (0.1% - 0.2%)
Detroit	Ito (2003) [reanalysis of Lippmann et al. (2000)]	Non-accidental	all	3 day	170 (-170 - 501)	99 (-99 - 293)	62 (-62 - 184)	37 (-38 - 110)
					8 (-8 - 24)	5 (-5 - 14)	3 (-3 - 9)	2 (-2 - 5)
					0.9% (-0.9% - 2.7%)	0.5% (-0.5% - 1.6%)	0.3% (-0.3% - 1.0%)	0.2% (-0.2% - 0.6%)
Los Angeles	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	0 day	494 (-62 - 1038)	308 (-38 - 647)	212 (-26 - 445)	146 (-18 - 306)
					5 (-1 - 11)	3 (0 - 7)	2 (0 - 5)	2 (0 - 3)
					0.9% (-0.1% - 1.9%)	0.6% (-0.1% - 1.2%)	0.4% (-0.1% - 0.8%)	0.3% (0.0% - 0.6%)
Philadelphia	Lipfert et al. (2000) -- 7 counties	Cardiovascular	all	1 day	412 (197 - 628)	231 (110 - 352)	141 (67 - 215)	83 (40 - 127)
					27 (13 - 41)	15 (7 - 23)	9 (4 - 14)	5 (3 - 8)
					2.5% (1.2% - 3.9%)	1.4% (0.7% - 2.2%)	0.9% (0.4% - 1.3%)	0.5% (0.2% - 0.8%)
Phoenix	Mar (2003) [reanalysis of Mar (2000)]	Cardiovascular	65+	1 day	323 (97 - 536)	86 (26 - 143)	56 (17 - 93)	39 (12 - 63)
					11 (3 - 17)	3 (1 - 5)	2 (1 - 3)	1 (0 - 2)
					5.0% (1.5% - 8.3%)	1.3% (0.4% - 2.2%)	0.9% (0.3% - 1.4%)	0.6% (0.2% - 1.0%)

Urban Area	Study	Type	Ages	Lag	Incidence Associated with $PM_{2.5}$ Assuming Various Cutpoint Levels (95% Confidence Interval) Incidence per 100,000 General Population (95% Confidence Interval) Percent of Total Incidence (95% Confidence Interval)			
					Policy Relevant Background =2.5 or 3.5 µg/m^3**	**Cutpoint*** =10 µg/m^3**	**Cutpoint*** =15 µg/m^3**	**Cutpoint*** =20 µg/m^3**
Pittsburgh	Chock et al. (2000)	Non-accidental	75+	0 day	77 (-166 - 311)	48 (-103 - 193)	31 (-67 - 125)	20 (-43 - 80)
					6 (-13 - 24)	4 (-8 - 15)	2 (-5 - 10)	2 (-3 - 6)
					0.8% (-1.7% - 3.2%)	0.5% (-1.1% - 2.0%)	0.3% (-0.7% - 1.3%)	0.2% (-0.4% - 0.8%)
San Jose	Fairley (2003) [reanalysis of Fairley (1999)]	Non-accidental	all	0 day	218 (45 - 387)	80 (17 - 141)	44 (9 - 77)	28 (6 - 50)
					13 (3 - 23)	5 (1 - 8)	3 (1 - 5)	2 (0 - 3)
					2.6% (0.5% - 4.7%)	1.0% (0.2% - 1.7%)	0.5% (0.1% - 0.9%)	0.3% (0.1% - 0.6%)
St. Louis	Schwartz (2003b) [reanalysis of Schwartz et al. (1996)]	Non-accidental	all	mean of lag 0 & 1 day	233 (86 - 379)	114 (42 - 185)	55 (20 - 89)	23 (8 - 38)
					9 (3 - 15)	5 (2 - 7)	2 (1 - 4)	1 (0 - 1)
					1.1% (0.4% - 1.7%)	0.5% (0.2% - 0.8%)	0.3% (0.1% - 0.4%)	0.1% (0.0% - 0.2%)

*All results are for single pollutant, non-accidental mortality models, unless otherwise specified.

**Policy relevant background is 2.5 µg/m^3 in the West (Los Angeles, Phoenix, and San Jose) and 3.5 µg/m^3 in the East (Boston, Detroit, Philadelphia, Pittsburgh, and St. Louis).

***For these alternative cutpoints the slope of the concentration-response relationship has been modified based on a simple hockeystick model (see discussion in section 4.3.2.1).

Table 4-10. Estimated Annual Health Risks of Long-Term Exposure Mortality Associated with Recent $PM_{2.5}$ Concentrations Assuming Various Cutpoint Levels*

Urban Areas	Incidence Associated with $PM_{2.5}$ Assuming Various Cutpoint Levels (95% Confidence Interval) Incidence per 100,000 General Population (95% Confidence Interval) Percent of Total Incidence (95% Confidence Interval)		
	Cutpoint = 7.5 µg/m³	**Cutpoint** =10 µg/m³**	**Cutpoint** =12 µg/m³**
Boston	594 (204 - 1053)	309 (106 - 551)	20 (7 - 36)
	21 (7 - 38)	11 (4 - 20)	1 (0 - 1)
	2.7% (0.9% - 4.7%)	1.4% (0.5% - 2.5%)	0.1% (0.0% - 0.2%)
Detroit	906 (313 - 1592)	713 (245 - 1259)	519 (178 - 920)
	44 (15 - 77)	35 (12 - 61)	25 (9 - 45)
	4.7% (1.6% - 8.2%)	3.7% (1.3% - 6.5%)	2.7% (0.9% - 4.8%)
Los Angeles	3684 (1280 - 6426)	3267 (1132 - 5715)	2846 (984 - 4994)
	39 (13 - 68)	34 (12 - 60)	30 (10 - 52)
	6.6% (2.3% - 11.4%)	5.8% (2.0% - 10.2%)	5.1% (1.8% - 8.9%)
Philadelphia	650 (224 - 1146)	466 (160 - 825)	280 (96 - 497)
	43 (15 - 76)	31 (11 - 54)	18 (6 - 33)
	3.9% (1.3% - 6.9%)	2.8% (1.0% - 4.9%)	1.7% (0.6% - 3.0%)
Phoenix	349 (119 - 620)	55 (19 - 98)	0 (0 - 0)
	11 (4 - 20)	2 (1 - 3)	0 (0 - 0)
	1.7% (0.6% - 3.0%)	0.3% (0.1% - 0.5%)	0.0% (0.0% - 0.0%)
Pittsburgh	816 (282 - 1430)	678 (234 - 1193)	539 (185 - 951)
	64 (22 - 112)	53 (18 - 93)	42 (14 - 74)
	5.4% (1.9% - 9.4%)	4.5% (1.5% - 7.8%)	3.5% (1.2% - 6.2%)
San Jose	172 (59 - 306)	58 (20 - 104)	0 (0 - 0)
	10 (4 - 18)	3 (1 - 6)	0 (0 - 0)
	2.1% (0.7% - 3.6%)	0.7% (0.2% - 1.2%)	0.0% (0.0% - 0.0%)
Seattle	50 (17 - 89)	0 (0 - 0)	0 (0 - 0)
	3 (1 - 5)	0 (0 - 0)	0 (0 - 0)
	0.5% (0.2% - 0.8%)	0.0% (0.0% - 0.0%)	0.0% (0.0% - 0.0%)
St. Louis	842 (290 - 1486)	587 (201 - 1041)	330 (113 - 587)
	33 (12 - 59)	23 (8 - 41)	13 (4 - 23)
	3.7% (1.3% - 6.6%)	2.6% (0.9% - 4.6%)	1.5% (0.5% - 2.6%)

*Based on Pope et al. (2002) -- ACS extended, all cause mortality among ages 30 and older.
**For these alternative cutpoints the slope of the concentration-response relationship has been modified based on a simple hockeystick model (see discussion in section 4.3.2.1).

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 involves assigning a background value to an observed concentration by randomly selecting a value from the conditional distribution corresponding to the observed value. The analysis has been 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 is 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 have been 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 has been carried out in Detroit and St. Louis using recent air quality levels for short-term exposure non-accidental mortality associated with $PM_{2.5}$. As shown in Exhibit 7.9 in the TSD, the difference between the risk estimates based on a constant versus a varying daily background are very 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 is 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 (see section 7.2 in the TSD).

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 do not capture these unusual events, it should be noted that there are 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 includes 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 Exhibit 7.11 in the TSD). This sensitivity analysis shows that there is hardly any difference (i.e., differences ranged from 0 to 0.1% of total incidence) in estimated short-term exposure mortality

associated with $PM_{2.5}$ including or excluding this fairly extreme, but short-term episode.[22] This same sensitivity analysis shows 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.

Alternative Concentration-Response Models

Another sensitivity analysis illustrates the impact on the risk estimates if the concentration-response functions used for short-term exposure mortality had used distributed lag models instead of single lag models. Schwartz (2000b) 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 in this sensitivity analysis. As would be expected, the risk estimates are almost doubled using the distributed lag approximation (see Appendix D in TSD).

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 are examined in the assessment: 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 epidemiologic study. These levels have been picked by staff to give a very rough indication of the possible impact of previous higher ambient $PM_{2.5}$ levels. Assuming that the relevant $PM_{2.5}$ concentrations were 50% higher than and twice as high as the levels reported in the original studies reduces long-term exposure mortality risk estimates by about one-third and one-half, respectively.

As noted earlier, while few studies have reported $PM_{2.5}$ concentration-response functions using a wide variety of alternative model specifications (e.g., GAM vs. GLM, different degrees of freedom, alternative lags), Moolgavkar (2003) did for his study in Los Angeles. Exhibits 7.12.a and 7.12.b in the TSD show the results as a sensitivity analysis for different models that employed either the more stringent GAM approach or GLM, with either 30 or 100 degrees of freedom, and included both single and multi-pollutant models. For this particular study, use of GLM instead of GAM in single-pollutant models tended to either have no impact or to lower by a small amount the estimated percent incidence of mortality in single pollutant models (e.g., changing the estimate from 0.9 to 0.7% of total incidence for 0-day lag with 30 degrees of freedom). For multi-pollutant models, use of GLM instead of GAM tended to either increase by 0.2 to 0.3% total incidence for cause-specific mortality and hospital admission estimates for 0-day lag with 100 degrees of freedom. Generally, but not always, the confidence intervals were

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

a little wider when GLM functions were used compared to GAM functions. Also, the use of a greater number of degrees of freedom tended to reduce the estimated incidence for both mortality and hospital admissions.

4.4.2 Just Meeting Current $PM_{2.5}$ Standards

The second part of the $PM_{2.5}$ risk assessment estimates the risk reductions that would result if the current suite of $PM_{2.5}$ standards (15 $\mu g/m^3$ annual average and 65 $\mu g/m^3$ daily average) 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 24-hour standard (65 $\mu g/m^3$).

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, 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 4-11. 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 4-11 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 use of spatial averaging. A sensitivity analysis examining the impact of using design values based on spatial averaging is discussed in section 4.4.3.2 for both the current and alternative $PM_{2.5}$ annual average standards.

4.4.2.1 Base Case Risk Estimates Above Initial Cutpoint

Similar to the presentation of risk estimates in section 4.4.1 associated with recent air quality levels, this section presents risk estimates for $PM_{2.5}$ exposures after $PM_{2.5}$ levels are reduced to levels associated with just meeting the current set of standards, using the initial cutpoint. For short-term exposure outcomes, the initial cutpoint is the estimated policy-relevant background level. For long-term exposure mortality the initial cutpoint is 7.5 $\mu g/m^3$. Risks are expressed both in terms of percent of total incidence and cases per hundred thousand general population. Figure 4-8 shows the estimates for four of the risk assessment study areas that do not meet the current $PM_{2.5}$ standards when their air quality is adjusted to simulate meeting the current standards.[23] The point estimates are in the range of about 0.5 to 1 percent of total incidence or 5 to 10 cases per hundred thousand general population across the four study areas.

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

Table 4-11. Air Quality Adjustments Required to Just Meet the Current Annual $PM_{2.5}$ Standard of 15 µg/m^3 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 (2005b)

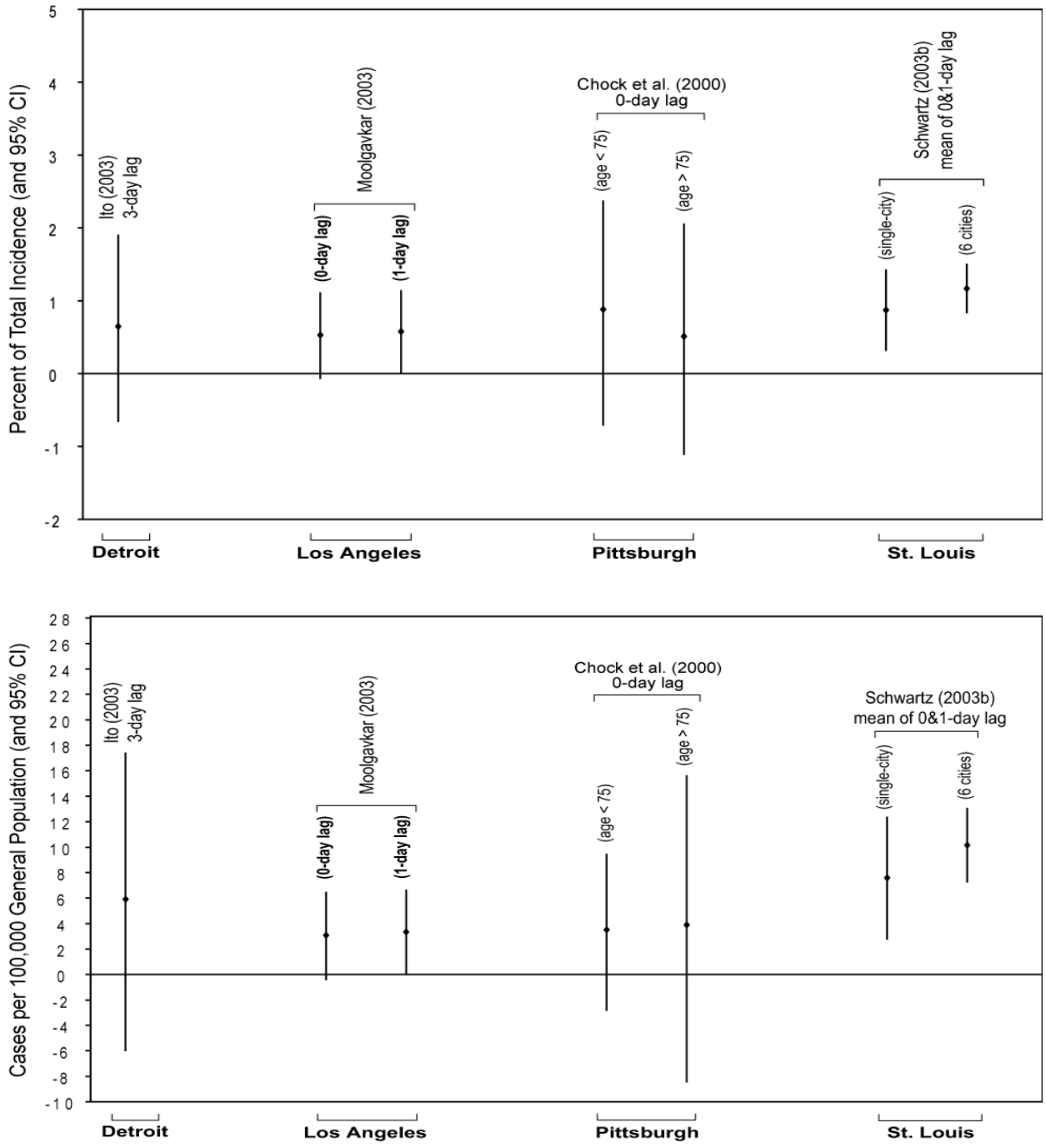


Figure 4-8. Estimated annual percent (top panel) and cases per 100,000 general population (bottom panel) of total (non-accidental) mortality associated with short-term exposure to $PM_{2.5}$ above background (and 95 percent confidence intervals) for air quality just meeting the current $PM_{2.5}$ standards.
Source: Abt Associates (2005b)

Similarly, Figure 4-9 displays the annual risk estimates in terms of percent of total incidence and cases per hundred thousand general population for all cause mortality associated with long-term exposure to $PM_{2.5}$ concentrations above a cutpoint of 7.5 $\mu g/m^3$ after air quality is adjusted to simulate just meeting the current standards in the five areas that do not meet the current $PM_{2.5}$ standards. The point estimates generally are in the range of about 2 to 5 percent of total incidence or 12 to 45 cases per hundred thousand general population across the five study areas.

4.4.2.2 Base Case Risk Estimates Above Various Cutpoints

In the same manner as the risk estimates for recent air quality levels, additional base case short- and long-term exposure annual mortality risk estimates have been developed associated with air quality levels just meeting the current standards including both the initial cutpoint used in the previous section and the same alternative cutpoints discussed previously. For short-term exposure mortality, a single non-accidental mortality function has been included, except for Philadelphia and Phoenix where cardiovascular mortality has been used since a suitable non-accidental mortality concentration-response function is not available. For long-term exposure mortality, a single all cause mortality concentration-response function has been included based on the ACS-extended study (Pope et al., 2002). Tables 4-12 and 4-13 present the annual health risks for short- and long-term exposure mortality, respectively, in terms of incidence (i.e., cases), cases per 100,000 general population, and percent of total incidence, along with 95 percent confidence intervals for each of these risk metrics.

4.4.2.3 Risk Estimates from Sensitivity Analyses

Three sensitivity analyses have been conducted associated with the air quality scenario of just meeting the current $PM_{2.5}$ standards. Two of these sensitivity analyses are discussed in this section. The third one examines the impact of using a spatial average of annual average monitor values versus the use of the maximum of annual average monitor values to determine the design value. The design value then determines the amount of adjustment required to just meet a specified set of standards. This third sensitivity analysis is presented in section 4.4.3.2 for both the current and alternative standards.

The first sensitivity analysis examines the impact of alternative approaches to simulating air quality levels that just meet the current standards. The base case risk analyses use 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 4.3.1.2 and in more detail in Appendix B of the TSD. 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 has been used which reduces the top 10 percent of the distribution of $PM_{2.5}$ concentrations by 1.6 times as much as the lower 90 percent of concentrations. The

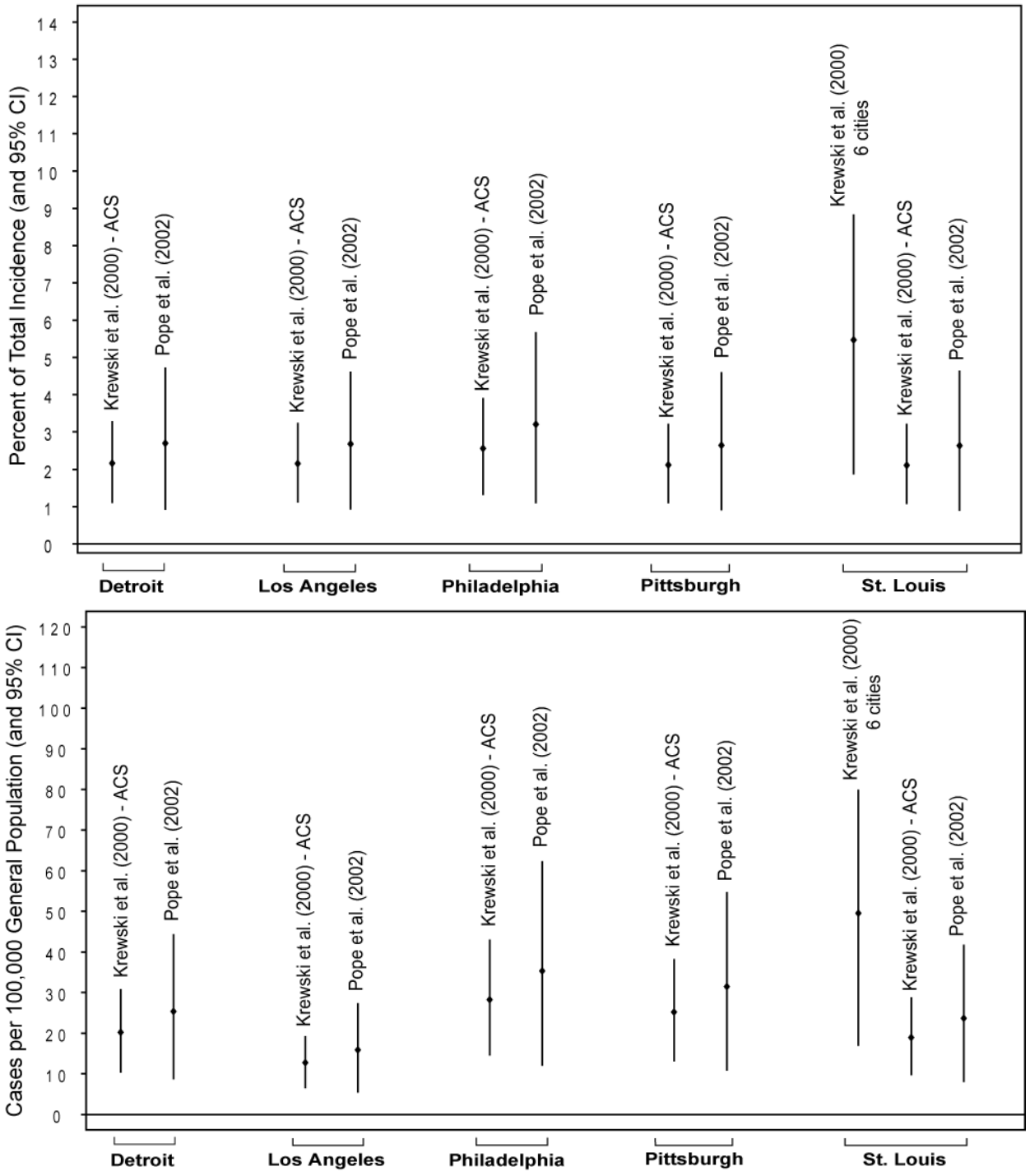


Figure 4-9. Estimated annual percent (top panel) and cases per 100,000 general population (bottom panel) of total mortality associated with long-term exposure to $PM_{2.5}$ above 7.5 $\mu g/m^3$ (and 95 percent confidence intervals) for air quality just meeting the current $PM_{2.5}$ standards.

Source: Abt Associates (2005b)

Table 4-12. Estimated Annual Mortality Associated with Short-Term Exposure to $PM_{2.5}$ When the Current Annual Standard of 15 µg/m3 and the Current Daily Standard of 65 µg/m3 Are Just Met, Assuming Various Cutpoint Levels*

Urban Area	Study	Type	Ages	Lag	Incidence Associated with $PM_{2.5}$ Assuming Various Cutpoint Levels (95% Confidence Interval) Incidence per 100,000 Population (95% Confidence Interval) Percent of Total Incidence (95% Confidence Interval)			
					Policy Relevant Background =2.5 or 3.5 µg/m^3**	**Cutpoint*** =10 µg/m^3**	**Cutpoint*** =15 µg/m^3**	**Cutpoint*** =20 µg/m^3**
Detroit	Ito (2003) [reanalysis of Lippmann et al. (2000)]	Non-accidental	all	3 day	122 (-123 - 358)	54 (-55 - 159)	26 (-27 - 77)	12 (-12 - 35)
					6 (-6 - 17)	3 (-3 - 8)	1 (-1 - 4)	1 (-1 - 2)
					0.7% (-0.7% - 1.9%)	0.3% (-0.3% - 0.8%)	0.1% (-0.1% - 0.4%)	0.1% (-0.1% - 0.2%)
Los Angeles	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental	all	0 day	292 (-37 - 612)	115 (-14 - 240)	58 (-7 - 121)	29 (-4 - 61)
					3 (0 - 6)	1 (0 - 3)	1 (0 - 1)	0 (0 - 1)
					0.5% (-0.1% - 1.1%)	0.2% (0.0% - 0.4%)	0.1% (0.0% - 0.2%)	0.1% (0.0% - 0.1%)
Philadelphia	Lipfert et al. (2000) -- 7 counties	Cardiovascular	all	1 day	367 (175 - 560)	189 (90 - 288)	106 (51 - 162)	57 (27 - 87)
					24 (12 - 37)	12 (6 - 19)	7 (3 - 11)	4 (2 - 6)
					5.8% (2.8% - 8.8%)	3.0% (1.4% - 4.5%)	1.7% (0.8% - 2.6%)	0.9% (0.4% - 1.4%)
Pittsburgh	Chock et al. (2000)	Non-accidental	75+	0 day	50 (-108 - 200)	22 (-48 - 87)	10 (-23 - 41)	5 (-11 - 18)
					4 (-8 - 16)	2 (-4 - 7)	1 (-2 - 3)	0 (-1 - 1)
					0.5% (-1.1% - 2.1%)	0.2% (-0.5% - 0.9%)	0.1% (-0.2% - 0.4%)	0.1% (-0.1% - 0.2%)
St. Louis	Schwartz (2003b) [reanalysis of Schwartz et al. (1996)]	Non-accidental	all	mean of lag 0 & 1	191 (70 - 311)	75 (28 - 122)	29 (11 - 46)	9 (3 - 14)
					8 (3 - 12)	3 (1 - 5)	1 (0 - 2)	0 (0 - 1)
					0.9% (0.3% - 1.4%)	0.3% (0.1% - 0.6%)	0.1% (0.1% - 0.2%)	0.0% (0.0% - 0.1%)

*All results are for single pollutant, non-accidental mortality models, unless otherwise specified.

**Policy relevant background is 2.5 µg/m^3 in the West (Los Angeles) and 3.5 µg/m^3 in the East (Detroit, Philadelphia, Pittsburgh, and St. Louis).

***For these alternative cutpoints the slope of the concentration-response relationship has been modified based on a simple hockeystick model (see discussion in section 4.3.2.1).

Table 4-13. Estimated Annual Mortality Associated with Long-Term Exposure to $PM_{2.5}$ When the Current Annual Standard of 15 µg/m^3 and the Current Daily Standard of 65 µg/m^3 Are Just Met, Assuming Various Cutpoint Levels*

Urban Areas	Incidence Associated with $PM_{2.5}$ Assuming Various Cutpoint Levels (95% Confidence Interval) Incidence per 100,000 Population (95% Confidence Interval) Percent of Total Incidence (95% Confidence Interval)		
	Cutpoint = 7.5 µg/m^3	**Cutpoint** =10 µg/m^3**	**Cutpoint** =12 µg/m^3**
Detroit	522 (181 - 910)	282 (98 - 494)	41 (14 - 72)
	25 (9 - 44)	14 (5 - 24)	2 (1 - 3)
	2.7% (0.9% - 4.7%)	1.5% (0.5% - 2.6%)	0.2% (0.1% - 0.4%)
Los Angeles	1507 (531 - 2587)	823 (290 - 1415)	138 (48 - 237)
	16 (6 - 27)	9 (3 - 15)	1 (1 - 2)
	2.7% (0.9% - 4.6%)	1.5% (0.5% - 2.5%)	0.2% (0.1% - 0.4%)
Philadelphia	536 (185 - 943)	338 (116 - 597)	137 (47 - 244)
	35 (12 - 62)	22 (8 - 39)	9 (3 - 16)
	3.2% (1.1% - 5.7%)	2.0% (0.7% - 3.6%)	0.8% (0.3% - 1.5%)
Pittsburgh	403 (141 - 699)	215 (75 - 373)	25 (9 - 43)
	31 (11 - 55)	17 (6 - 29)	2 (1 - 3)
	2.7% (0.9% - 4.6%)	1.4% (0.5% - 2.5%)	0.2% (0.1% - 0.3%)
St. Louis	596 (206 - 1047)	311 (107 - 548)	23 (8 - 40)
	24 (8 - 42)	12 (4 - 22)	1 (0 - 2)
	2.6% (0.9% - 4.6%)	1.4% (0.5% - 2.4%)	0.1% (0.0% - 0.2%)

*Based on Pope et al. (2002) -- ACS extended, all cause mortality among ages 30 and older.

**For these alternative cutpoints the slope of the concentration-response relatonship has been modified based on a simple hockeystick model (see discussion in section 4.3.2.1).

result of this alternative hypothetical adjustment which reduces the highest days more than the rest of the distribution shows only a small difference (less than 1%) in the percent change in PM-associated incidence (see Exhibit 8.9 and Appendix E, Exhibits E.33 to E.36, in the TSD).

The second sensitivity analysis explores the potential impact on the short-term exposure non-accidental mortality risk estimates if the same multi-city concentration-response relationship is used in five risk assessment locations compared to the single-city concentration-response relationships used in the base case analysis. As noted earlier, the multi-city concentration-response relationship used in this sensitivity analysis is from the Six-Cities study (Schwartz, 2003b), the only U.S. multi-city study on $PM_{2.5}$ short-term exposure mortality that is currently available. Table 4-14 shows the results of this sensitivity analysis, including the results from the base case analysis which used the single-city concentration-response relationships. As expected, given the generally larger relative risk reported in the Six Cities study, the estimated incidence and deaths per 100,000 general population are somewhat larger in four of the five locations using the Six Cities study function. The range of risk estimates across the five locations also considerably narrows when the same concentration-response relationship is used in all five locations. For example, using the risk metric that normalizes across locations with different population sizes, the range goes from 4 to 24 deaths per 100,000 general population using the single-city functions to 8 to 14 deaths per 100,000 general population using the Six Cities study function. As noted previously, there are a number of possible sources for the differences observed in risk estimates based on single-city studies. These include known differences in baseline mortality incidence rates, possible differences in study methodology, increased statistical uncertainty due to smaller sample sizes in some single-city studies, differences in degree and patterns of exposure to ambient $PM_{2.5}$, differences in sources or components that might impact the toxicity, and differences in co-pollutants or other unidentified factors that may play a role in modifying the concentration-response relationship.

4.4.3 Just Meeting Alternative $PM_{2.5}$ Standards

4.4.3.1 Base Case Risk Estimates

The third part of the $PM_{2.5}$ risk assessment estimates the risk associated with just meeting alternative suites of annual and daily $PM_{2.5}$ standards, along with the risk reduction associated with going to these levels from the current standards. For the five urban areas that exceed the current $PM_{2.5}$ suite of standards (i.e., Detroit, Los Angeles, Philadelphia, Pittsburgh, and St. Louis), the estimated risk reductions are 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 meet the current $PM_{2.5}$ standards based on our analysis of 2001-2003 levels (i.e., Boston, Phoenix, San Jose, and Seattle), the estimated risk reductions are those associated with a reduction from recent air quality levels to just meeting various suites of alternative $PM_{2.5}$ standards.

Table 4-14. Sensitivity Analysis Comparing the Use of Multi-City vs. Single-City Concentration-Response Relationships On Estimates of Short-Term Exposure Mortality Associated with Just Meeting the Current PM2.5 Standards

Urban Area	Study	Type of Mortality and Single vs. Multi-City Concentration-Response Relationship*	Ages	Lag	Incidence Associated with $PM_{2.5}$ Assuming Various Cutpoint Levels (95% Confidence Interval) Incidence per 100,000 General Population (95% Confidence Interval) Percent of Total Incidence (95% Confidence Interval)			
					Policy Relevant Background** =2.5 or 3.5 µg/m^3	Cutpoint*** =10 µg/m^3	Cutpoint*** =15 µg/m^3	Cutpoint*** =20 µg/m^3
Detroit	Ito (2003) [reanalysis of Lippmann et al. (2000)]	Non-accidental Single City	all	3 day	122 (-123 - 358)	54 (-55 - 159)	26 (-27 - 77)	12 (-12 - 35)
					6 (-6 - 17)	3 (-3 - 8)	1 (-1 - 4)	1 (-1 - 2)
					0.7% (-0.7% - 1.9%)	0.3% (-0.3% - 0.8%)	0.1% (-0.1% - 0.4%)	0.1% (-0.1% - 0.2%)
	Schwartz (2003b) [reanalysis of Schwartz et al. (1996)]	Non-accidental Multi-City	all	mean of lag 0 & 1 day	224 (160 - 286)	87 (62 - 111)	34 (25 - 44)	15 (11 - 19)
					11 (8 - 14)	4 (3 - 5)	2 (1 - 2)	1 (1 - 1)
					1.2% (0.9% - 1.5%)	0.5% (0.3% - 0.6%)	0.2% (0.1% - 0.2%)	0.1% (0.1% - 0.1%)
Los Angeles	Moolgavkar (2003) [reanalysis of Moolgavkar (2000a)]	Non-accidental Single City	all	0 day	292 (-37 - 612)	115 (-14 - 240)	58 (-7 - 121)	29 (-4 - 61)
					3 (0 - 6)	1 (0 - 3)	1 (0 - 1)	0 (0 - 1)
					0.5% (-0.1% - 1.1%)	0.2% (0.0% - 0.4%)	0.1% (0.0% - 0.2%)	0.1% (0.0% - 0.1%)
	Schwartz (2003b) [reanalysis of Schwartz et al. (1996)]	Non-accidental Multi-City	all	mean of lag 0 & 1 day	731 (526 - 935)	270 (194 - 344)	123 (89 - 157)	55 (40 - 70)
					8 (6 - 10)	3 (2 - 4)	1 (1 - 2)	1 (0 - 1)
					1.3% (1.0% - 1.7%)	0.5% (0.4% - 0.6%)	0.2% (0.2% - 0.3%)	0.1% (0.1% - 0.1%)
Philadelphia	Lipfert et al. (2000) -- 7 counties	Cardiovascular Single City	all	1 day	367 (175 - 560)	189 (90 - 288)	106 (51 - 162)	57 (27 - 87)
					24 (12 - 37)	12 (6 - 19)	7 (3 - 11)	4 (2 - 6)
					5.8% (2.8% - 8.8%)	3.0% (1.4% - 4.5%)	1.7% (0.8% - 2.6%)	0.9% (0.4% - 1.4%)
	Schwartz (2003b) [reanalysis of Schwartz et al. (1996)]	Non-accidental Multi-City	all	mean of lag 0 & 1 day	213 (153 - 273)	103 (74 - 132)	50 (36 - 65)	24 (17 - 31)
					14 (10 - 18)	7 (5 - 9)	3 (2 - 4)	2 (1 - 2)
					1.3% (0.9% - 1.7%)	0.6% (0.5% - 0.8%)	0.3% (0.2% - 0.4%)	0.2% (0.1% - 0.2%)
Pittsburgh	Chock et al. (2000)	Non-accidental Single City	75+	0 day	50 (-108 - 200)	22 (-48 - 87)	10 (-23 - 41)	5 (-11 - 18)
					4 (-8 - 16)	2 (-4 - 7)	1 (-2 - 3)	0 (-1 - 1)
					0.5% (-1.1% - 2.1%)	0.2% (-0.5% - 0.9%)	0.1% (-0.2% - 0.4%)	0.1% (-0.1% - 0.2%)
	Schwartz (2003b) [reanalysis of Schwartz et al. (1996)]	Non-accidental Multi-City	all	mean of lag 0 & 1 day	174 (125 - 223)	68 (49 - 87)	27 (19 - 34)	11 (8 - 14)
					14 (10 - 17)	5 (4 - 7)	2 (1 - 3)	1 (1 - 1)
					1.2% (0.8% - 1.5%)	0.5% (0.3% - 0.6%)	0.2% (0.1% - 0.2%)	0.1% (0.1% - 0.1%)
St. Louis	Schwartz (2003b) [reanalysis of Schwartz et al. (1996)]	Non-accidental Single City	all	mean of lag 0 & 1 day	191 (70 - 311)	75 (28 - 122)	29 (11 - 46)	9 (3 - 14)
					8 (3 - 12)	3 (1 - 5)	1 (0 - 2)	0 (0 - 1)
					0.9% (0.3% - 1.4%)	0.3% (0.1% - 0.6%)	0.1% (0.1% - 0.2%)	0.0% (0.0% - 0.1%)
	Schwartz (2003b) [reanalysis of Schwartz et al. (1996)]	Non-accidental Multi-City	all	mean of lag 0 & 1 day	256 (183 - 328)	97 (69 - 124)	36 (25 - 46)	10 (7 - 13)
					10 (7 - 13)	4 (3 - 5)	1 (1 - 2)	0 (0 - 1)
					1.2% (0.8% - 1.5%)	0.4% (0.3% - 0.6%)	0.2% (0.1% - 0.2%)	0.1% (0.0% - 0.1%)

*All results are for single pollutant models.

**Policy relevant background is 2.5 µg/m^3 in the West (Los Angeles) and 3.5 µg/m3 in the East (Detroit, Philadelphia, Pittsburgh, and St. Louis).

***For these alternative cutpoints the slope of the concentration-response function has been modified based on a simple hockeystick model (see discussion in section 4.3.2.1).

The selection of the suites of alternative annual and daily standards included in the risk assessment has been based, in part, on consideration of CASAC and public comments, and is consistent with the staff recommendations described in Chapter 5. Annual standards of 15, 14, 13, and 12 $\mu g/m^3$ are 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.[24] In addition, an annual standard of 15 $\mu g/m^3$ has been combined with a 99th 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 4-15. The same proportional adjustment approach used to simulate air quality just meeting the current standards, described previously in section 4.3.1.2 and in section 2.3 of the TSD, has been used to simulate air quality just meeting the various alternative suites of standards. Table 4-16 provides the design values for the annual and 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 base case analyses examining alternative $PM_{2.5}$ standards include non-accidental mortality (or cause-specific if there was no suitable function for non-accidental mortality available) associated with short-term exposure to $PM_{2.5}$ above policy-relevant background and several alternative cutpoint levels. In addition, the base case analyses include estimates of risk for all cause mortality, cardiopulmonary mortality, and lung cancer mortality associated with long-term exposure to $PM_{2.5}$ above 7.5 $\mu g/m^3$ and two alternative cutpoint levels based on Pope et al. (2002) – ACS extended. Since the patterns observed were identical, only the all cause long-term exposure mortality results are presented in this Staff Paper (see the TSD for the cause-specific mortality estimates). As in the earlier base case analyses, in addition to having a cutpoint set equal to policy-relevant background, cutpoints of 10, 15, and 20 $\mu g/m^3$ have been included for health endpoints associated with short-term exposures. For long-term exposure mortality, cutpoints set equal to 7.5 $\mu g/m^3$, the lowest measured level in the ACS-extended study, and alternative cutpoints of 10 and 12 $\mu g/m^3$ have been included in the base case analysis.

The base case analysis results for alternative annual standards combined with 98th and 99th percentile daily standards, respectively, are given in Tables 4-17 for Detroit for mortality associated with short-term exposure. Short-term exposure mortality risk estimates for the other four urban locations (Los Angeles, Philadelphia, Pittsburgh, and St. Louis) are provided in Tables 4B-1 through 4B-4 in Appendix 4B. Similarly, the estimated risk reduction in total all cause mortality associated with long-term $PM_{2.5}$ exposures for these same alternative standards

[24]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 $\mu g/m^3$.

Table 4-15. 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 4-16. 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 $\mu g/m^3$. The design values summarized here for the alternative standards are based on use of the maximum monitor in each urban area.

Table 4-17. Estimated Annual Mortality Associated with Short-Term Exposure to $PM_{2.5}$ When Alternative Standards Are Just Met, Assuming Various Cutpoint Levels for Detroit, MI*

Alternative Standards		Incidence Associated with $PM_{2.5}$ Assuming Various Cutpoint Levels (95% Confidence Interval)			
		Percent Reduction in Incidence from Current Standards			
Annual (µg/m3)	Daily (µg/m3)	Policy Relevant Background =3.5 µg/m^3	Cutpoint** =10 µg/m^3	Cutpoint** =15 µg/m^3	Cutpoint** =20 µg/m^3
15	65, 98th percentile value***	122 (-123 - 358) 0.0%	54 (-55 - 159) 0.0%	26 (-27 - 77) 0.0%	12 (-12 - 35) 0.0%
	40, 98th percentile value	122 (-123 - 358) 0.0%	54 (-55 - 159) 0.0%	26 (-27 - 77) 0.0%	12 (-12 - 35) 0.0%
	35, 98th percentile value	122 (-123 - 358) 0.0%	54 (-55 - 159) 0.0%	26 (-27 - 77) 0.0%	12 (-12 - 35) 0.0%
	30, 98th percentile value	111 (-112 - 325) 9.0%	45 (-45 - 131) 16.7%	20 (-20 - 58) 23.1%	8 (-9 - 24) 33.3%
	25, 98th percentile value	90 (-91 - 263) 26.2%	28 (-29 - 82) 48.1%	10 (-10 - 28) 61.5%	3 (-4 - 10) 75.0%
	65, 99th percentile value	122 (-123 - 358) 0.0%	54 (-55 - 159) 0.0%	26 (-27 - 77) 0.0%	12 (-12 - 35) 0.0%
	40, 99th percentile value	122 (-123 - 358) 0.0%	54 (-55 - 159) 0.0%	26 (-27 - 77) 0.0%	12 (-12 - 35) 0.0%
	35, 99th percentile value	120 (-121 - 352) 1.6%	53 (-53 - 154) 1.9%	25 (-26 - 74) 3.8%	11 (-12 - 33) 8.3%
	30, 99th percentile value	101 (-102 - 296) 17.2%	37 (-37 - 107) 31.5%	15 (-15 - 42) 42.3%	6 (-6 - 16) 50.0%
	25, 99th percentile value	82 (-83 - 239) 32.8%	22 (-23 - 65) 59.3%	7 (-7 - 19) 73.1%	2 (-2 - 6) 83.3%
14	40, 98th percentile value	111 (-112 - 326) 9.0%	45 (-46 - 132) 16.7%	20 (-20 - 58) 23.1%	8 (-9 - 24) 33.3%
	35, 98th percentile value	111 (-112 - 326) 9.0%	45 (-46 - 132) 16.7%	20 (-20 - 58) 23.1%	8 (-9 - 24) 33.3%
	30, 98th percentile value	111 (-112 - 325) 9.0%	45 (-45 - 131) 16.7%	20 (-20 - 58) 23.1%	8 (-9 - 24) 33.3%
	25, 98th percentile value	90 (-91 - 263) 26.2%	28 (-29 - 82) 48.1%	10 (-10 - 28) 61.5%	3 (-4 - 10) 75.0%
	40, 99th percentile value	111 (-112 - 326) 9.0%	45 (-46 - 132) 16.7%	20 (-20 - 58) 23.1%	8 (-9 - 24) 33.3%
	35, 99th percentile value	111 (-112 - 326) 9.0%	45 (-46 - 132) 16.7%	20 (-20 - 58) 23.1%	8 (-9 - 24) 33.3%
	30, 99th percentile value	101 (-102 - 296) 17.2%	37 (-37 - 107) 31.5%	15 (-15 - 42) 42.3%	6 (-6 - 16) 50.0%
	25, 99th percentile value	82 (-83 - 239) 32.8%	22 (-23 - 65) 59.3%	7 (-7 - 19) 73.1%	2 (-2 - 6) 83.3%

Alternative Standards		Incidence Associated with $PM_{2.5}$ Assuming Various Cutpoint Levels (95% Confidence Interval) Percent Reduction in Incidence from Current Standards			
Annual (µg/m3)	**Daily (µg/m3)**	**Policy Relevant Background =3.5 µg/m^3**	**Cutpoint** =10 µg/m^3**	**Cutpoint** =15 µg/m^3**	**Cutpoint** =20 µg/m^3**
13	40, 98th percentile value	101 (-101 - 295) 17.2%	36 (-37 - 106) 33.3%	14 (-15 - 42) 46.2%	6 (-6 - 16) 50.0%
	35, 98th percentile value	101 (-101 - 295) 17.2%	36 (-37 - 106) 33.3%	14 (-15 - 42) 46.2%	6 (-6 - 16) 50.0%
	30, 98th percentile value	101 (-101 - 295) 17.2%	36 (-37 - 106) 33.3%	14 (-15 - 42) 46.2%	6 (-6 - 16) 50.0%
	25, 98th percentile value	90 (-91 - 263) 26.2%	28 (-29 - 82) 48.1%	10 (-10 - 28) 61.5%	3 (-4 - 10) 75.0%
	40, 99th percentile value	101 (-101 - 295) 17.2%	36 (-37 - 106) 33.3%	14 (-15 - 42) 46.2%	6 (-6 - 16) 50.0%
	35, 99th percentile value	101 (-101 - 295) 17.2%	36 (-37 - 106) 33.3%	14 (-15 - 42) 46.2%	6 (-6 - 16) 50.0%
	30, 99th percentile value	101 (-101 - 295) 17.2%	36 (-37 - 106) 33.3%	14 (-15 - 42) 46.2%	6 (-6 - 16) 50.0%
	25, 99th percentile value	82 (-83 - 239) 32.8%	22 (-23 - 65) 59.3%	7 (-7 - 19) 73.1%	2 (-2 - 6) 83.3%
12	40, 98th percentile value	90 (-91 - 264) 26.2%	28 (-29 - 82) 48.1%	10 (-10 - 28) 61.5%	3 (-4 - 10) 75.0%
	35, 98th percentile value	90 (-91 - 264) 26.2%	28 (-29 - 82) 48.1%	10 (-10 - 28) 61.5%	3 (-4 - 10) 75.0%
	30, 98th percentile value	90 (-91 - 264) 26.2%	28 (-29 - 82) 48.1%	10 (-10 - 28) 61.5%	3 (-4 - 10) 75.0%
	25, 98th percentile value	90 (-91 - 263) 26.2%	28 (-29 - 82) 48.1%	10 (-10 - 28) 61.5%	3 (-4 - 10) 75.0%
	40, 99th percentile value	90 (-91 - 264) 26.2%	28 (-29 - 82) 48.1%	10 (-10 - 28) 61.5%	3 (-4 - 10) 75.0%
	35, 99th percentile value	90 (-91 - 264) 26.2%	28 (-29 - 82) 48.1%	10 (-10 - 28) 61.5%	3 (-4 - 10) 75.0%
	30, 99th percentile value	90 (-91 - 264) 26.2%	28 (-29 - 82) 48.1%	10 (-10 - 28) 61.5%	3 (-4 - 10) 75.0%
	25, 99th percentile value	82 (-83 - 239) 32.8%	22 (-23 - 65) 59.3%	7 (-7 - 19) 73.1%	2 (-2 - 6) 83.3%

*This analysis was performed using Ito (2003).
**For these alternative cutpoints the slope of the concentration-response function has been modified based on a simple hockeystick model (see discussion in section 4.3.2.1).
***Current standards.
Note: Incidences are rounded to the nearest whole number; percents are rounded to the nearest tenth.

are given in Table 4-18 for Detroit and in Appendix 4B (see Tables 4B-5 through 4B-8) for the other four urban areas.

Not surprisingly, estimated PM-related incidences varied substantially with both alternative cutpoint levels and with 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 (when the cutpoint is set equal to policy-relevant background) or to 54, 26, and 12 under alternative cutpoints 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 alternative cutpoint increasingly removes estimated cases at concentrations between background and the cutpoint, one would expect to see an increase in the percent reduction associated with just meeting alternative standards for higher cutpoints. This is exactly what is found. For example, as seen in Table 4-17, going from just meeting the current standards (15 $\mu g/m^3$ annual and 65 $\mu g/m^3$ daily 98^{th} percentile value) to just meeting the lowest set of standards considered (12 $\mu g/m^3$ annual and 25 $\mu g/m^3$ daily 99^{th} percentile value) results in a reduction in short-term exposure mortality incidence of (115 - 75)/115 = 34.8 percent when the cutpoint equals policy-relevant background; but, with a cutpoint equal to 10 $\mu g/m^3$, it results in a reduction of (54 - 22)/54 = 59 percent.

As shown in Table 4-18 for all-cause mortality associated with long-term exposure in Detroit, the reduction in mortality incidence is even more dramatic when alternative cutpoint levels are considered. Going from just meeting the current standards to just meeting the lowest set of standards considered (12 $\mu g/m^3$ annual and 25 $\mu g/m^3$ daily 99^{th} percentile value) results in a reduction in long-term exposure mortality incidence of (522-207)/522= 60% with a cutpoint equal to 7.5 $\mu g/m^3$; but, with the cutpoint set equal to 10 $\mu g/m^3$, it results in a reduction of (282 - 0)/282 =100 percent. The same general patterns can be seen in all locations and for all health endpoints considered.

4.4.3.2 Risk Estimates from Sensitivity Analyses

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 "spatial averaging approach" to determine compliance with the annual standard. This involves using the average of the annual averages of several monitors within the urban area. Four of the five urban areas included in the $PM_{2.5}$ risk assessment that do not meet the current annual standard based on the maximum community-oriented monitor meet the minimum requirements to allow use of spatial averaging. The design values and percent rollback required to meet the current annual standard for these four areas are shown in Table 4-11. Tables 4B-9 and 4B-10 in Appendix 4B present the PM-related mortality risk estimates associated with short- and long-term exposure, respectively, in Detroit using the maximum versus the average of monitor-specific averages to determine the design value for the annual standards. Risk estimates for alternative suites of standards are expressed in terms of

Table 4-18. Estimated Annual Mortality Associated with Long-Term Exposure to $PM_{2.5}$ When Alternative Standards Are Just Met, Assuming Various Cutpoint Levels for Detroit, MI*

Alternative Standards		Incidence Associated with $PM_{2.5}$ Assuming Various Cutpoint Levels (95% Confidence Interval) Percent Reduction in Incidence from Current Standards		
Annual ($\mu g/m^3$)	Daily ($\mu g/m^3$)	Cutpoint** =7.5 $\mu g/m^3$	Cutpoint** =10 $\mu g/m^3$	Cutpoint** =12 $\mu g/m^3$
15	65, 98th percentile value***	522 (181 - 910) 0.0%	282 (98 - 494) 0.0%	41 (14 - 72) 0.0%
	40, 98th percentile value	522 (181 - 910) 0.0%	282 (98 - 494) 0.0%	41 (14 - 72) 0.0%
	35, 98th percentile value	522 (181 - 910) 0.0%	282 (98 - 494) 0.0%	41 (14 - 72) 0.0%
	30, 98th percentile value	435 (151 - 757) 16.7%	185 (64 - 323) 34.4%	0 (0 - 0) 100.0%
	25, 98th percentile value	270 (94 - 468) 48.3%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
	65, 99th percentile value	522 (181 - 910) 0.0%	282 (98 - 494) 0.0%	41 (14 - 72) 0.0%
	40, 99th percentile value	522 (181 - 910) 0.0%	282 (98 - 494) 0.0%	41 (14 - 72) 0.0%
	35, 99th percentile value	507 (176 - 884) 2.9%	266 (92 - 465) 5.7%	23 (8 - 40) 43.9%
	30, 99th percentile value	356 (124 - 619) 31.8%	97 (34 - 168) 65.6%	0 (0 - 0) 100.0%
	25, 99th percentile value	207 (72 - 358) 60.3%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
14	40, 98th percentile value	438 (152 - 762) 16.1%	188 (65 - 328) 33.3%	0 (0 - 0) 100.0%
	35, 98th percentile value	438 (152 - 762) 16.1%	188 (65 - 328) 33.3%	0 (0 - 0) 100.0%
	30, 98th percentile value	435 (151 - 757) 16.7%	185 (64 - 323) 34.4%	0 (0 - 0) 100.0%
	25, 98th percentile value	270 (94 - 468) 48.3%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
	40, 99th percentile value	438 (152 - 762) 16.1%	188 (65 - 328) 33.3%	0 (0 - 0) 100.0%
	35, 99th percentile value	438 (152 - 762) 16.1%	188 (65 - 328) 33.3%	0 (0 - 0) 100.0%
	30, 99th percentile value	356 (124 - 619) 31.8%	97 (34 - 168) 65.6%	0 (0 - 0) 100.0%
	25, 99th percentile value	207 (72 - 358) 60.3%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%

Alternative Standards		Incidence Associated with $PM_{2.5}$ Assuming Various Cutpoint Levels (95% Confidence Interval) Percent Reduction in Incidence from Current Standards		
Annual ($\mu g/m^3$)	Daily ($\mu g/m^3$)	Cutpoint** =7.5 $\mu g/m^3$	Cutpoint** =10 $\mu g/m^3$	Cutpoint** =12 $\mu g/m^3$
13	40, 98th percentile value	354 (123 - 615) 32.2%	94 (33 - 164) 66.7%	0 (0 - 0) 100.0%
	35, 98th percentile value	354 (123 - 615) 32.2%	94 (33 - 164) 66.7%	0 (0 - 0) 100.0%
	30, 98th percentile value	354 (123 - 615) 32.2%	94 (33 - 164) 66.7%	0 (0 - 0) 100.0%
	25, 98th percentile value	270 (94 - 468) 48.3%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
	40, 99th percentile value	354 (123 - 615) 32.2%	94 (33 - 164) 66.7%	0 (0 - 0) 100.0%
	35, 99th percentile value	354 (123 - 615) 32.2%	94 (33 - 164) 66.7%	0 (0 - 0) 100.0%
	30, 99th percentile value	354 (123 - 615) 32.2%	94 (33 - 164) 66.7%	0 (0 - 0) 100.0%
	25, 99th percentile value	207 (72 - 358) 60.3%	0 (0 - 0) 100.0%	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%
	35, 98th percentile value	271 (94 - 469) 48.1%	0 (0 - 1) 100.0%	0 (0 - 0) 100.0%
	30, 98th percentile value	271 (94 - 469) 48.1%	0 (0 - 1) 100.0%	0 (0 - 0) 100.0%
	25, 98th percentile value	270 (94 - 468) 48.3%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
	40, 99th percentile value	271 (94 - 469) 48.1%	0 (0 - 1) 100.0%	0 (0 - 0) 100.0%
	35, 99th percentile value	271 (94 - 469) 48.1%	0 (0 - 1) 100.0%	0 (0 - 0) 100.0%
	30, 99th percentile value	271 (94 - 469) 48.1%	0 (0 - 1) 100.0%	0 (0 - 0) 100.0%
	25, 99th percentile value	207 (72 - 358) 60.3%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%

*This analysis was performed using Pope et al. (2002) -- ACS extended.
**For these alternative cutpoints the slope of the concentration-response function has been modified based on a simple hockeystick model (see discussion in section 4.3.2.1).
***Current standards.
Note: Incidences are rounded to the nearest whole number; percents are rounded to the nearest tenth.

estimated mortality incidence and percent reduction in incidence from just meeting the current standards using the initial cutpoint and assuming alternative cutpoints. Similar tables for Pittsburgh and St. Louis (the other two locations that do not meet the current standards and for which both approaches result in positive percent rollbacks) are given in Exhibits E.37 to E.40 in the TSD. Alternative suites of annual and daily $PM_{2.5}$ standards, where the daily standard is the controlling standard under both design value approaches, have not been included in this sensitivity analysis, since there is no change in the risk estimates.

For those cases where the annual standard is the controlling standard under both design value approaches, use of spatial averaging requires less reduction in $PM_{2.5}$, thus higher mortality incidence and less reduction in risk are associated with the current and alternative annual standards compared to use of the maximum monitor based approach. There are also cases where the annual standard is the controlling standard under the maximum monitor based approach, but the daily standard becomes controlling when the same annual standard is considered using the spatial averaging approach. When this occurs, the estimated incidence reduction associated with the spatially averaged annual standard combined with the daily standard is determined by the daily standard. In this case, the incidence reduction will be less than that associated with meeting the annual standard using the maximum-monitor based approach but greater than the incidence reduction associated with meeting the annual standard using the spatial averaging approach.

Based on the risk estimates for the three example urban areas (Detroit, Pittsburgh, and St. Louis) using the initial cutpoint, the estimated mortality incidence associated with long-term exposure is about 10 to over 40% higher for the current suite of standards where compliance with the annual standard is based on spatial averaging than the estimated incidence where compliance is based on the highest population-oriented monitor. The estimated mortality incidence associated with short-term exposure using the initial cutpoint ranges from about 5 to 25% higher when the spatial averaging approach is used for the current standards in these three example urban areas.

As noted above, the use of spatial averaging for alternative suites of standards only has an impact on risk estimates compared to the maximum-monitor based approach where the annual standard is controlling for at least one of these approaches. For such cases in the three example urban areas, the estimated mortality incidence associated with long-term exposure using the initial cutpoint in most cases ranges from about 10 to 60% higher when spatial averaging is used to determine compliance with the annual average. In these three example urban areas, the estimated mortality incidence associated with short-term exposure using the intial cutpoint in most cases ranges from about 5 to 25% higher when spatial averaging is used.

Changing from a maximum-monitor based approach to the spatial average approach impacts the estimated risks associated with just meeting both the current and lower alternative standards. Comparing the estimated percent reductions in mortality incidence associated with

going from just meeting the current standard to alternative lower standards between the two design value approaches for the three example urban areas (Detroit, Pittsburgh, and St. Louis), there does not seem to be any clear pattern.

4.4.4 Key Observations

Recent PM Air Quality Levels

Sections 4.4.1.1, 4.4.1.2, and 4.4.1.3 have presented the $PM_{2.5}$ health risk estimates and sensitivity analyses associated with recent PM air quality levels. Presented below are key observations resulting from this part of the risk assessment:

- A fairly wide range of risk estimates are observed for $PM_{2.5}$-related morbidity and mortality incidence across the urban areas analyzed associated with recent air quality.

- Most of the point estimates for $PM_{2.5}$ for the base case analysis are in the range 0.8 to 3% for short-term exposure total non-accidental mortality when the cutpoint equals estimated policy-relevant background. Generally, the point estimates for the single- and multi-city models are roughly similar in most of the urban areas analyzed. The impact of adding additional co-pollutants to the models was variable; sometimes there was relatively little difference, while in other cases there were larger differences.

- The point estimates for long-term exposure mortality associated with $PM_{2.5}$ range from about 0.5% to as high as 6.6% with most estimates falling in the 2 to 5% range for single-pollutant models (based on the ACS-extended study) when the cutpoint equals 7.5 $\mu g/m^3$. Addition of a single co-pollutant resulted in higher risk estimates when CO, NO_2, or O_3 were added to the models for the ACS study and lower risk estimates when SO_2 was added.

- The single most important factor influencing the risk estimates is the consideration of which of the alternative concentration-response functions included in this assessment best represents the unknown "true" concentration-response relationships.

The wide variability in risk estimates associated with a recent year of air quality is to be expected given the wide range of $PM_{2.5}$ levels across the urban areas analyzed and the variation observed in the concentration-response relationships obtained from the original epidemiologic 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 of the sensitivity analyses, the following key observation is made:

- 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 estimating exposure levels for long-term exposure mortality. Use of a distribution of daily background concentrations had very little impact on the risk estimates.

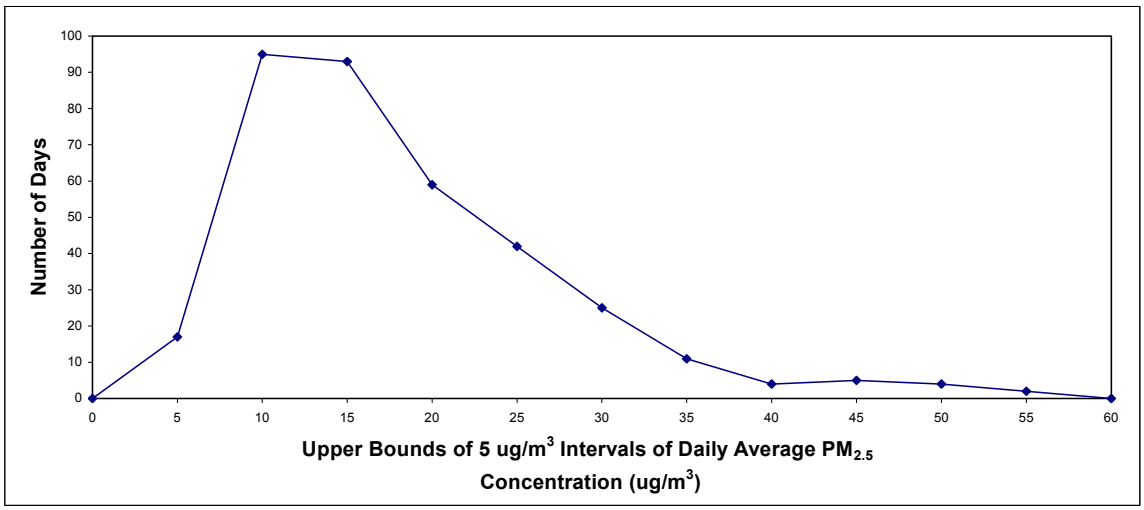
Staff was interested in obtaining insight into the overall pattern of risk associated with short-term $PM_{2.5}$ exposures across the distribution of $PM_{2.5}$ air quality, as typically observed in urban areas. Figure 4-10 illustrates the relative contribution of different portions of a typical urban ambient $PM_{2.5}$ concentration distribution to mortality risk associated with short-term $PM_{2.5}$ exposures. The top panel in Figure 4-10 shows the annual distribution of 24-hour $PM_{2.5}$ concentrations in Detroit. The middle panel shows the estimated incidence expressed in terms of deaths per day for the upper bound of each 5 $\mu g/m^3$ increment based on the short-term exposure epidemiology study included in the current $PM_{2.5}$ risk assessment.[25] The bottom panel shows the corresponding distribution of estimated mortality incidence (for $PM_{2.5}$) for each 5 $\mu g/m^3$ increment taking into account the number of days in each interval and the concentration-response relationship. Not surprisingly, the middle panel shows that higher 24-hour $PM_{2.5}$ concentrations pose greater risk in terms of deaths per day. However, as shown in the bottom panel, on an annual basis, the very highest days contribute less to the total annual health risk associated with short-term exposures than the middle of the distribution (i.e., in the range of about 10 to 35 $\mu g/m^3$ in this example), due to the much greater number of days that occur in this part of the air quality distribution. As shown in the prior review (61 FR at 65652, December 13, 1996), a similar, if somewhat scaled-back pattern, was observed when concentration-response relationships were used that assumed a cutpoint (or hypothetical threshold).

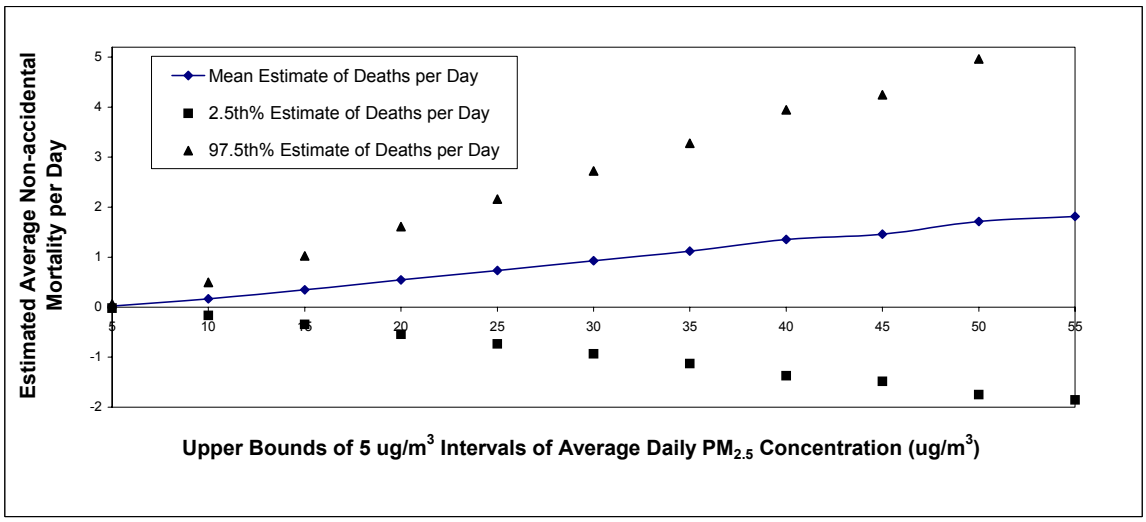
Meeting the Current $PM_{2.5}$ Standards

Sections 4.4.2.1, 4.4.2.2, and 4.4.2.3 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 $PM_{2.5}$-related incidence of short-term exposure mortality and morbidity remaining across the five urban areas analyzed. This is likely due, in large part, to differences in concentration-response relationships among

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





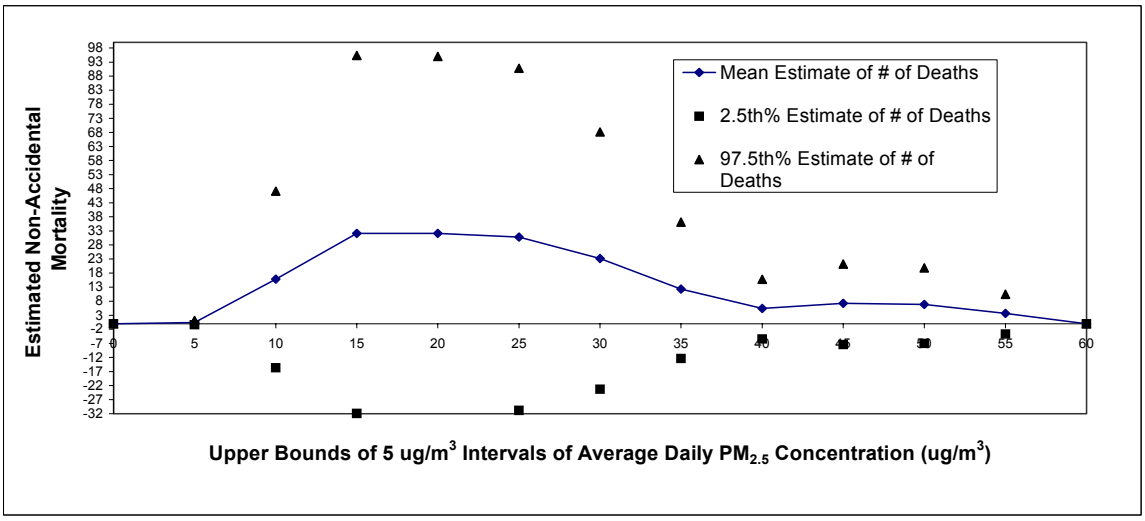


Figure 4-10. Distribution of average daily $PM_{2.5}$ concentrations in Detroit (2003 air quality data) (top panel), estimated non-accidental mortality per day in Detroit associated with exposure to daily $PM_{2.5}$ concentrations (middle panel), and estimated non-accidental mortality in Detroit associated with exposure to daily $PM_{2.5}$ concentrations over the course of a year (bottom panel). Source: Abt Associates (2005b)

single-location short-term exposure studies, differences in baseline incidence rates, and varying population sizes.

- Results of a sensitivity analysis which applied one multi-city concentration-response function to all five urban areas analyzed narrowed considerably the range of risk estimates when a risk metric was used that normalized for different population sizes. However, it is still unknown whether the wider range of estimates observed using single-city concentration-response functions reflect methodological differences between studies and/or real city-to-city differences related to exposure, population, composition of the particles, or other factors.

- The single most important factor influencing the risk estimates is the consideration of which of the alternative concentration-response functions included in this assessment best represents the unknown "true" concentration-response relationships.

- 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 meeting 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.

Meeting Alternative $PM_{2.5}$ Standards

Section 4.4.3.1 presented the base case $PM_{2.5}$-related incidence associated with meeting alternative $PM_{2.5}$ standards and the percent reduction in incidence from the current $PM_{2.5}$ standards. Presented below are key observations resulting from this part of the risk assessment:

- The most important factor influencing the base case risk estimates for both short- and long-term exposure mortality associated with $PM_{2.5}$ concentrations just meeting alternative standards is the consideration of which of the alternative concentration-response functions included in this assessment best represents the unknown "true" concentration-response relationships.

- For short-term exposure mortality, there is a significant decrease in the incidence remaining as one considers alternative higher cutpoints. There also is a

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

Section 4.4.3.2 presented the results of a sensitivity analysis considering the impact on risk estimates associated with just meeting the current and alternative standards when the spatial averaging approach is used to determine compliance with the annual standard. A key observation resulting from this part of the risk assessment follows:

- There is an increase in estimated short-term and long-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.

4.5 $PM_{10-2.5}$ RISK ESTIMATES

A similar approach has been taken for $PM_{10-2.5}$ risk estimates, with initial base case risk estimates for recent air quality using estimated policy-relevant background as the initial cutpoint and, then, additional base case estimates for recent air quality and alternative $PM_{10-2.5}$ standards including the initial and alternative cutpoints. For the alternative cutpoints, the slope of the concentration-response relationship has been modified based on the same simple hockeystick model approach used for $PM_{2.5}$.

4.5.1. Recent Air Quality

4.5.1.1 Base Case Risk Estimates

Figure 4-11 shows risk estimates for hospital admissions associated with short-term exposure to $PM_{10-2.5}$ for Detroit and Seattle, and Figure 4-12 shows risk estimates associated with respiratory symptoms for St. Louis associated with recent $PM_{10-2.5}$ air quality levels. 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 with lower $PM_{10-2.5}$ ambient concentrations than either Detroit or St. Louis, is about 1%. Point estimates for lower respiratory symptoms and cough in St. Louis are about 12 and 15%, respectively. These estimates use estimated policy-relevant background as the cutpoint. Table 4-21, discussed below, provides risk estimates associated with recent $PM_{10-2.5}$ air quality levels using policy-relevant background and higher alternative cutpoints.

4.5.1.2 Risk Estimates from Sensitivity Analyses

For $PM_{10-2.5}$, the sensitivity analysis examining the effects of using the lower- and upper-end of the range of estimated policy-relevant background levels shows 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 estimates are reduced by about 19% (see Exhibit 9.5 in the TSD). The effect of different background concentrations for the other two $PM_{10-2.5}$ locations is about $\pm$ 30% for asthma hospital admissions in Seattle (see Exhibit F.7 in the TSD) and about $\pm$ 50% for respiratory symptoms in St. Louis (see Exhibit F.8 in the TSD).

4.5.2 Just Meeting Alternative $PM_{10-2.5}$ Standards

The second part of the $PM_{10-2.5}$ risk assessment estimates the risk associated with just meeting alternative daily $PM_{10-2.5}$ standards for the three locations examined earlier (Detroit, St. Louis, and Seattle), as well as the risk reductions associated with going to these levels from the current air quality levels. 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 regarding risk reductions associated with alternative standards in these three urban areas may not be fully relevant to the areas expected to have the greatest health risks associated with peak daily ambient $PM_{10-2.5}$ concentrations.

Estimated reductions in risk were developed for going from recent air quality levels (based on 2003 air quality) to just meeting alternative $PM_{10-2.5}$ standards. Staff selected the daily standards to be included in the risk assessment based on the preliminary staff recommendations described in Chapter 5 of the draft 2005 Staff Paper (EPA, 2005) and consideration of public and CASAC comments. Table 4-19 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 4-20.

The estimated number of hospital admissions for ischemic heart disease associated with short-term $PM_{10-.2.5}$ exposures for alternative 98th and 99th percentile daily standards, respectively, are given in Table 4-21 for Detroit. This table includes risk estimates which are based on the cutpoint being policy-relevant background as well as three higher alternative cutpoints. 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

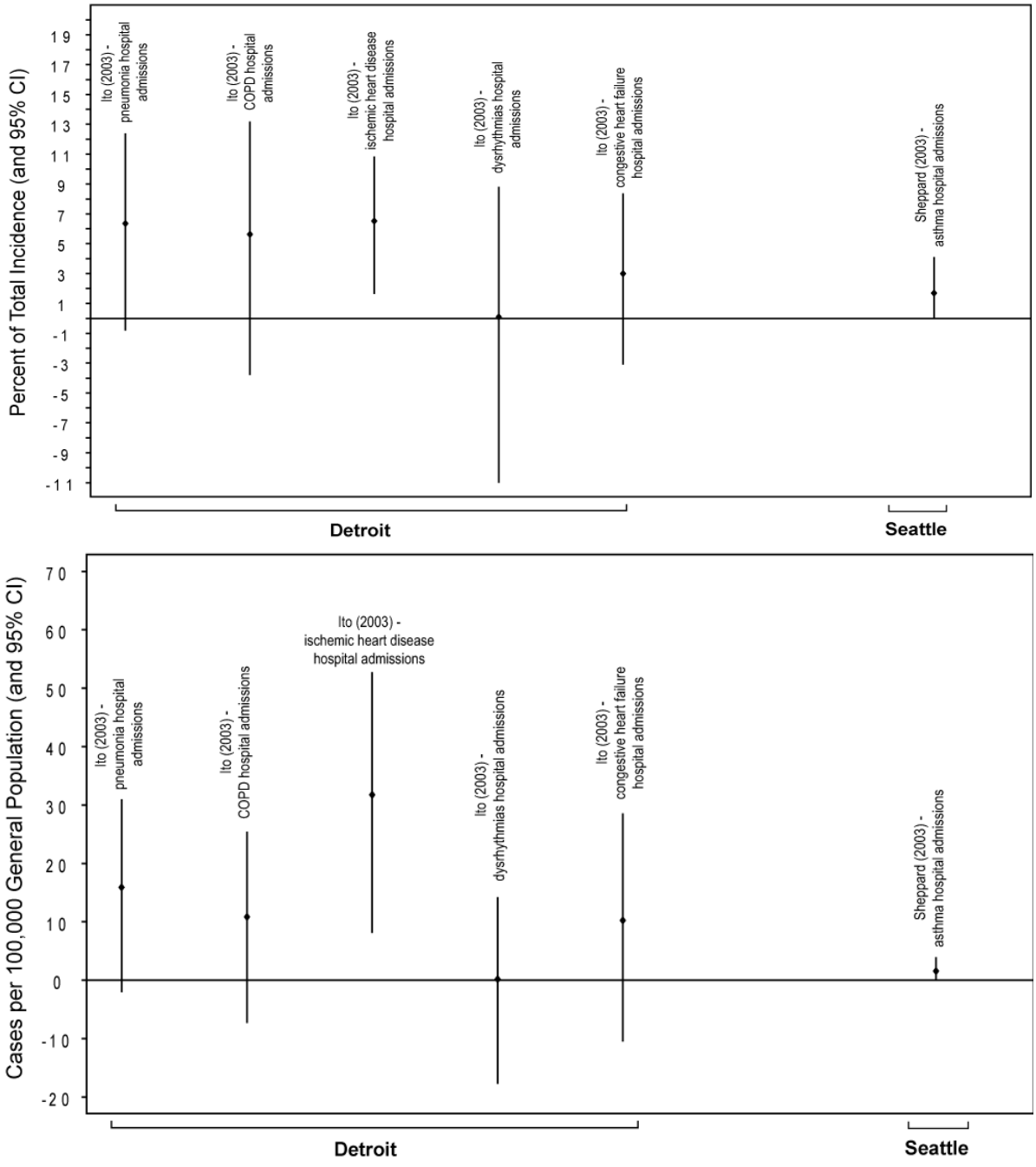


Figure 4-11. Estimated annual percent (top panel) and cases per 100,000 general population (bottom panel) of hospital admissions associated with short-term exposure to $PM_{10-2.5}$ above background for recent air quality (and 95 percent confidence intervals). Source: Abt Associates (2005b)

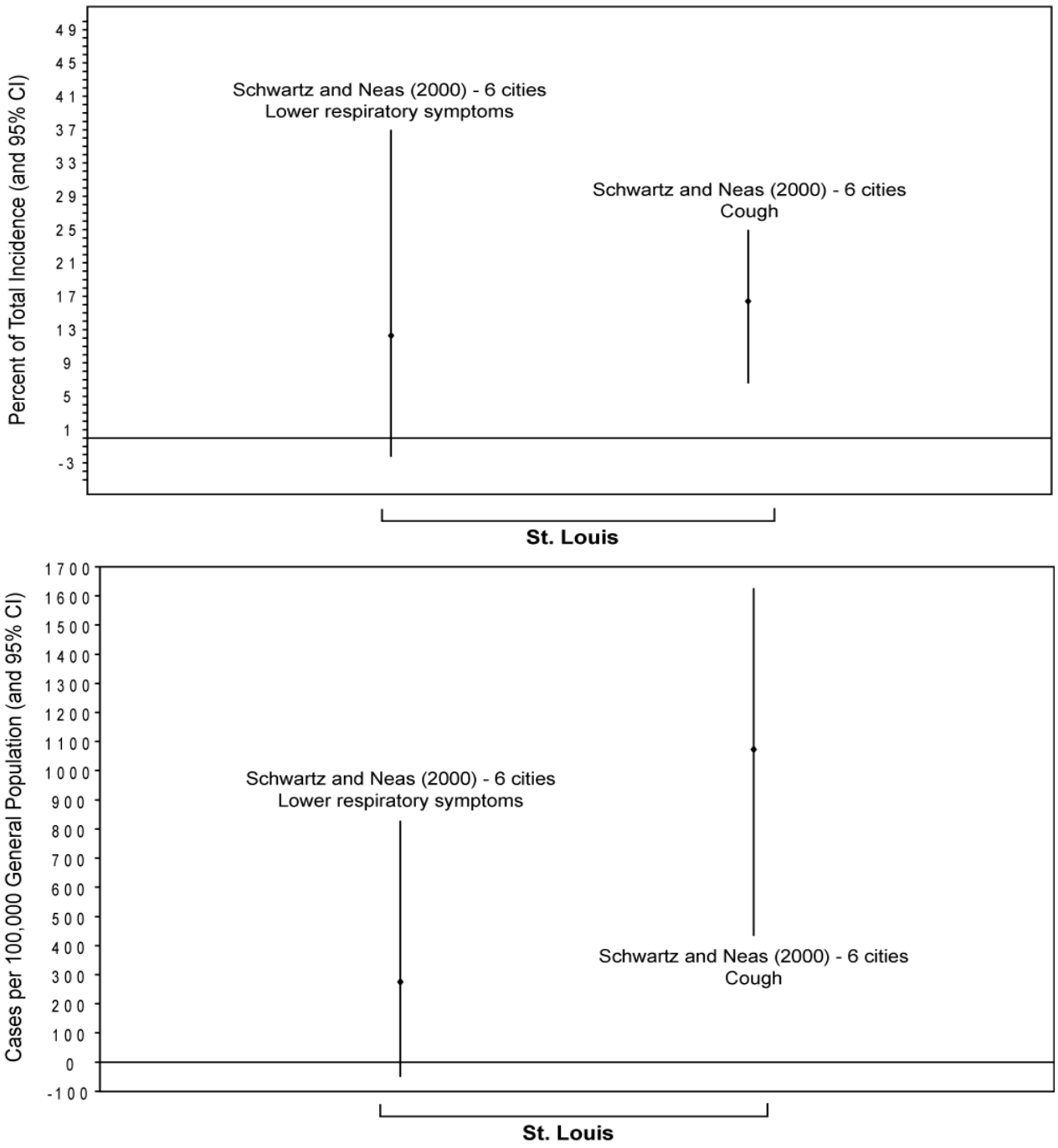


Figure 4-12. Estimated annual percent (top panel) and cases per 100,000 general population (bottom panel) of respiratory symptoms associated with short-term exposure to $PM_{10-2.5}$ above background for recent air quality (and 95 percent confidence intervals). Source: Abt Associates (2005b)

Table 4-19. 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 4-20. 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 $\mu g/m^3$.

Table 4-21. Estimated Annual Hospital Admissions for Ischemic Heart Disease Associated with Short-Term Exposure to $PM_{10-2.5}$ When Alternative Standards Are Just Met, Assuming Various Cutpoint Levels* Detroit, MI, 2003

(Recent Air Quality Levels = 21.7 µg/m^3 Annual Average; 105.9 µg/m^3, 98th Percentile Daily Value)

Recent $PM_{10-2.5}$ Air Quality Levels and Alternative Daily Standards (µg/m^3)	Incidence Associated with $PM_{10-2.5}$ Assuming Various Cutpoint Levels (95% Confidence Interval) Percent Reduction in Incidence from Recent $PM_{10-2.5}$ Air Quality Levels			
	Policy Relevant Background =4.5 µg/m^3	Cutpoint** =10 µg/m^3	Cutpoint** =15 µg/m^3	Cutpoint** =20 µg/m^3
Recent $PM_{10-2.5}$ air quality levels	654 (169 - 1083) 0.0%	569 (149 - 934) 0.0%	489 (129 - 794) 0.0%	426 (115 - 682) 0.0%
80 µg/m^3 daily 98th percentile value	654 (169 - 1083) 0.0%	569 (149 - 934) 0.0%	489 (129 - 794) 0.0%	426 (115 - 682) 0.0%
65 µg/m^3 daily 98th percentile value	600 (156 - 989) 8.3%	508 (134 - 829) 10.7%	425 (114 - 683) 13.1%	360 (99 - 567) 15.5%
50 µg/m^3 daily 98th percentile value	443 (117 - 719) 32.3%	334 (90 - 532) 41.3%	248 (69 - 384) 49.3%	183 (54 - 271) 57.0%
30 µg/m^3 daily 98th percentile value	242 (65 - 386) 63.0%	125 (36 - 190) 78.0%	65 (20 - 91) 86.7%	44 (15 - 57) 89.7%
25 µg/m^3 daily 98th percentile value	193 (52 - 307) 70.5%	81 (24 - 120) 85.8%	39 (13 - 52) 92.0%	25 (9 - 30) 94.1%
100 µg/m^3 daily 99th percentile value	654 (169 - 1083) 0.0%	569 (149 - 934) 0.0%	489 (129 - 794) 0.0%	426 (115 - 682) 0.0%
80 µg/m^3 daily 99th percentile value	654 (169 - 1083) 0.0%	569 (149 - 934) 0.0%	489 (129 - 794) 0.0%	426 (115 - 682) 0.0%
60 µg/m^3 daily 99th percentile value	491 (129 - 801) 24.9%	387 (104 - 621) 32.0%	301 (83 - 472) 38.4%	233 (67 - 353) 45.3%
35 µg/m^3 daily 99th percentile value	262 (70 - 419) 59.9%	144 (41 - 221) 74.7%	79 (24 - 113) 83.8%	53 (18 - 68) 87.6%
30 µg/m^3 daily 99th percentile value	218 (59 - 347) 66.7%	103 (30 - 154) 81.9%	51 (16 - 70) 89.6%	34 (12 - 43) 92.0%

*This analysis was performed using Ito (2003).

**For these alternative cutpoints the slope of the concentration-response function has been modified based on a simple hockeystick model (see discussion in section 4.3.2.1).

Note: Incidences are rounded to the nearest whole number; percents are rounded to the nearest tenth.

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 $\mu g/m^3$ whereas the 98th percentile daily value in 2003 is 105.9 $\mu g/m^3$. Because the design value is lower than 80 $\mu g/m^3$, the highest 98th percentile daily $PM_{10-2.5}$ standard considered in the assessment, 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 $\mu g/m^3$, is well above the standard level. Similarly, the design value for the 99th percentile daily $PM_{10-2.5}$ standards is 77 $\mu g/m^3$ for Detroit, whereas the 99th percentile daily value at the composite monitor in Detroit in 2003 is substantially greater than 100 $\mu g/m^3$, the highest 99th percentile daily $PM_{10-2.5}$ standard considered. Thus, zero risk reductions were estimated to result from both 100 and 80 $\mu g/m^3$ 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, for the set of alternative standards included in the analysis, is achieved with the 98th percentile 25 $\mu g/m^3$ standard and 99th percentile 30 $\mu g/m^3$ standard. The point estimate is that about a 4% reduction in hospital admissions for ischemic 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.

4.5.3 Key Observations

Sections 4.5.1.1 and 4.5.1.2 presented the $PM_{10-2.5}$ health risk estimates and sensitivity analyses associated with recent $PM_{10-2.5}$ air quality levels. Presented below are key observations resulting from this part of the risk assessment:

- 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 recent air quality levels in a single urban area (St. Louis).
- Results of a sensitivity analysis examining the impact of assuming different values for policy relevant background showed moderate changes in short-term morbidity risk estimates ranging from $\pm$ 16 to $\pm$50% depending on the health endpoint and location considered.

Section 4.5.2 presented the base case $PM_{10-2.5}$-related incidence associated with meeting alternative $PM_{2.5}$ standards and the percent reduction in incidence from recent air quality levels. Presented below are key observations resulting from this part of the risk assessment:

- For short-term exposure morbidity, there is a significant decrease in the remaining estimated incidence associated with $PM_{10-2.5}$ as one considers higher alternative cutpoints for all of the standards that require reductions in recent $PM_{10-2.5}$ air quality levels. There also is a significant increase observed in the percent reduction in PM-associated incidence upon just meeting these same alternative standards with higher alternative cutpoints compared to recent $PM_{10-2.5}$ air quality levels.

- Based on the point estimates, there are no risk reductions associated with just meeting daily 98^{th} 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 99^{th} 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.

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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. 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, in the Administrator's judgment, 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 Act does not require that primary standards be set at a zero-risk level, but rather at a level that avoids unacceptable risks to public health.

5.2 APPROACH

As an initial matter, PM standards for fine particles and 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 it is reinforced by new information that advances our understanding of differences in human exposure relationships and dosimetric patterns characteristic of these two subclasses of PM pollution, as well as the apparent independence of health effects that have been associated with them 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 an annual $PM_{2.5}$ standard that was generally controlling, and to set 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.

In reviewing the $PM_{2.5}$ standards, for example, 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 ranges of levels and forms of alternative standards are 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 would protect against effects associated with both long- and short-term exposures, based on considering how a suite of standards would 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 with 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 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 1996 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 the strength of the evidence, including the strength and robustness of reported associations and the consistency of the results. The CD recognizes that while the relative risk estimates are generally small in magnitude, a number of new studies provide relatively precise estimates that are generally positive and often statistically significant. Overall, the CD finds that 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, especially 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 biologic 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 $PM_{2.5}$ exposure, especially in susceptible individuals, could result in changes in cardiac function or blood parameters that are risk factors for cardiovascular disease. For respiratory effects, the CD finds that toxicologic studies have provided evidence that supports plausible biologic 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 recognizes that there are likely biologic threshold levels in individuals for specific health responses. Staff notes, however, that the available epidemiologic evidence neither supports nor refutes the existence of thresholds at the population level for the effects of $PM_{2.5}$ on mortality across the range of concentrations in the studies, for either long-term or short-term $PM_{2.5}$ exposures, as discussed in Chapter 3 (section 3.6.6) and the CD (section 9.2.2.5). Further, the CD notes that in the multi-city studies 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).

5.3.1.1 Evidence-based Considerations

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 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 the level of the current annual $PM_{2.5}$ standard (summarized in Appendix 3A). In considering these 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; 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 reported statistically significant associations between short-term $PM_{2.5}$ exposure and morbidity in such areas. 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, 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 that 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) and 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, no apparent threshold is evident 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. However, if these findings were further substantiated by future research, estimates of the extent of life shortening due to PM-related premature mortality would likely significantly increase (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 and 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 epidemiologic 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 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 other components, 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 beyond 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, both

at lower levels of air quality and at levels below the current standards, and with more understanding of possible underlying mechanisms.

5.3.1.2 Risk-based Considerations

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. In so doing, staff first notes that the risk assessment discussed in Chapter 4 addresses a number of key uncertainties through various base case analyses, as well as through several sensitivity analyses. Most importantly, a series of base case analyses were conducted to characterize the uncertainty associated with the form of the concentration-response functions drawn from the studies used in the assessment, which had by far the greatest impact on estimated risks. Other uncertainties, 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, have a more moderate and often variable impact on the risk estimates in some or all of the cities.

In considering the health risks estimated to occur upon attainment of the current $PM_{2.5}$ standards, staff focused in particular on base case risk estimates, while recognizing that the confidence ranges in the selected base case estimates do not reflect all the identified uncertainties. These risks were estimated using not only the linear or log-linear functions reported in the studies,[1] but also using a series of alternative modified linear functions as surrogates for assumed non-linear functions that would reflect the possibility that thresholds may exist in the reported associations within the range of air quality observed in the studies. The approach used to develop the alternative functions, discussed more fully in Chapter 4 (section 4.3.2.1), incorporates a modified linear slope with an imposed cutpoint (i.e., an assumed threshold) that is intended to reflect an inflection point in a typical non-linear, "hockey-stick" shaped function, below which there is little or no population response. As discussed in Chapter 3 (section 3.6.6), staff recognizes that while there are likely biological thresholds in individuals for specific health responses, the available epidemiologic studies do not support or refute the existence of thresholds at the population level for either long-term or short-term PM exposures within the range of air quality observed in the studies (CD, p. 9-44). Thus, staff has concluded that it is appropriate to consider health risks estimated not only with the reported linear or log-

[1] As discussed in Chapter 4, the reported linear or log-linear functions were applied down to 7.5 $\mu g/m^3$ in estimating risk associated with long-term exposure (i.e., the lowest measured level in the extended ACS study), and down to the estimated policy-relevant background level in estimating risk associated with short-term exposure (i.e., 3.5 $\mu g/m^3$ for eastern urban areas and 2.5 $\mu g/m^3$ for western urban areas).

linear concentration-response functions, but also with modified functions that incorporate alternative assumed cutpoints as surrogates for potential population thresholds.

Tables 5-1(a) and (b) summarize the estimated $PM_{2.5}$-related annual incidence and incidence rate (in terms of incidence per 100,000 general population) of total mortality associated with long-term and short-term exposures, respectively, assuming various cutpoint levels in the example urban areas included in the risk assessment.[2] In first looking 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 (Detroit, Los Angeles, Philadelphia, Pittsburgh, and St. Louis), staff notes that there is a fairly wide range of estimated incidence across the areas for both long- and short-term exposures. Such variation would be expected considering, for example, differences in total population, demographics, baseline mortality rates, exposure considerations (e.g., degree of air conditioning use), presence of co-pollutants and other environmental stressors, and exposure measurement error across urban areas. The somewhat greater variation in the estimated incidence associated with short-term exposure than with long-term exposure would also be expected, since the assessment uses the same long-term exposure concentration-response function in all areas, whereas the assessment used different short-term exposure functions (for different mortality endpoints in some cases) from studies conducted in each area. 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 (as discussed in Chapter 2, section 2.5.1).

In the five study areas that do not meet the current standards, staff observes for long-term exposure that point estimates of annual incidence of total $PM_{2.5}$-related mortality associated with just meeting the current $PM_{2.5}$ standards, based on the lowest cutpoint of 7.5 $\mu g/m^3$, range from approximately 400 to 600 in four areas (from roughly 25 to 35 deaths per 100,000 general population in these areas) to over 1500 annual deaths in Los Angeles (roughly 16 deaths per 100,000 general 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. In the same five areas, the annual incidence associated with short-term exposure, based on a cutpoint equal to policy-relevant background, ranges from less than 20 % to over 50% of the estimated incidence associated with long-term exposure. 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.

[2] These tables include risk estimates drawn from Tables 4-9, 4-10, 4-12, and 4-13 in Chapter 4.

Table 5-1(a) Estimated $PM_{2.5}$-related annual total mortality associated with long-term exposure when current $PM_{2.5}$ standards are met*

	Annual Incidence of All-Cause Mortality and 95% CI (deaths/yr)			Annual Incidence Rate of All-Cause Mortality and 95% CI (deaths/yr/100,000 general population)		
	Cutpoints			Cutpoints		
	7.5 μg/m^3	10 μg/m^3	12 μg/m^3	7.5 μg/m^3	10 μg/m^3	12 μg/m^3
Risks associated with just meeting current $PM_{2.5}$ standards						
Detroit	520 180- 910	280 100 - 490	40 10 - 70	25 9 - 44	14 5 - 24	2 1 - 3
Los Angeles	1,510 530 - 2,590	820 290 - 1420	140 50-o 240	16 6 - 27	9 3 - 15	1 1 - 2
Philadelphia	540 190 - 940	340 120-o 600	140 50 - 240	35 12 - 62	22 8 - 39	9 3 - 16
Pittsburgh	400 140-o 700	220 80-o 370	30 10 - 40	31 11 - 55	17 6 - 29	2 1 - 3
St. Louis	600 210 - 1,050	310 110 - 550	20 10 - 40	24 8 - 42	12 4 - 22	1 0 - 2
Risks associated with "as is" air quality (in areas that meet current $PM_{2.5}$ standards)						
Boston	590 200 - 1050	310 110 - 550	20 10 - 40	21 7 - 38	11 4 - 20	1 0 - 1
Phoenix	350 120 - 620	80 30 - 140	0	11 4 - 20	2 1 - 3	0
San Jose	170 60 - 310	60 20-o 100	0	10 4 - 18	3 1 - 6	0
Seattle	50 20 - 90	0	0	3 1 - 5	0	0

***** These estimates 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.

Table 5-1(b) Estimated $PM_{2.5}$-related annual mortality associated with short-term exposure when current $PM_{2.5}$ standards are met*

	Annual Incidence of Non-Accidental Mortality and 95% CI (except as noted) (deaths/yr)				Annual Incidence Rate of Non-Accidental Mortality and 95% CI (except as noted) (deaths/yr/100,000 general population)			
	Cutpoints				Cutpoints			
	Policy-relevant Background**	10 μg/m^3	15 μg/m^3	20 μg/m^3	Policy-relevant Background**	10 μg/m^3	15 μg/m^3	20 μg/m^3
Risks associated with just meeting current $PM_{2.5}$ standards								
Detroit	120 -120 - 360	50 -60 - 160	30 -30 - 80	10 -10 to 40	6 -6 - 17	3 -3 - 8	1 -1 - 4	1 -1 - 2
Los Angeles	290 -40 - 610	120 -10 - 240	60 -10 - 120	30 -4 to 60	3 0 - 6	1 0 - 3	1 0 - 1	0 0 - 1
Philadelphia cardiovascular mortality	370 180 - 560	190 90 - 290	110 50 - 160	60 30 to907	24 12 - 37	12 6 - 19	7 3 - 11	4 2 - 6
Pittsburgh over age 74	50 -110 - 200	20 -50 - 90	10 -20 - 40	5 -10 to 20	4 -8 - 16	2 -4 - 7	1 -2 - 3	0 -1 - 1
St. Louis	190 70 - 310	80 30 - 120	30 10 - 50	9 3 to 14	8 3 - 12	3 1 - 5	1 0 - 2	0 0 - 1
Risks associated with "as is" air quality (in areas that meet current $PM_{2.5}$ standards)								
Boston	390 270 - 510	170 120 - 230	80 60 - 110	40 30 - 50	14 9 - 18	6 4 - 8	3 2 - 4	1 1 - 2
Phoenix cardiovascular mortality over age 64	320 100 - 540	90 30 - 140	60 20 - 90	40 10 - 60	11 3 - 17	3 1 - 5	2 1 - 3	1 0 - 2
San Jose	220 50 - 390	80 20 - 140	40 10 - 80	30 10 - 50	13 3 - 23	5 1 - 8	3 1 - 5	2 0 - 3

***** These estimates 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.

** Estimated policy-relevant background levels are 3.5 μg/m^3 for eastern urban areas and 2.5 μg/m^3 for western urban areas.

In the other four areas that meet the current standards based on recent air quality data (Boston, Phoenix, San Jose, and Seattle), point estimates of annual incidence of total $PM_{2.5}$-related mortality associated with long-term exposure range from about 50 deaths in Seattle (roughly 3 deaths per 100,000 general population) to almost 600 deaths in Boston (roughly 21 deaths per 100,000 general population). Estimated incidence associated with short-term exposure in these four areas generally falls within the range of the estimates associated with long-term exposure.

In considering the estimated incidences associated with long-term exposure based on assumed cutpoint of 10 $\mu g/m^3$, staff observes that these estimates are roughly about half as large as the estimates based on a cutpoint of 7.5 $\mu g/m^3$. Under this assumption, point estimates of annual incidence of total $PM_{2.5}$-related mortality associated with just meeting the current $PM_{2.5}$ standards range from about 200 to over 300 in four of the areas that do not meet the current standards (from roughly 12 to 22 deaths per 100,000 general population in these areas) to over 800 annual deaths in Los Angeles (roughly 9 deaths per 100,000 general population) associated with long-term exposure. In considering an assumed cutpoint as high as 12 $\mu g/m^3$, point estimates associated with long-term exposure in these five areas are roughly 5 to 20% of the estimates based on the lowest cutpoint. A similar pattern is seen when considering the impact of alternative assumed cutpoints in the range of 10 to 20 $\mu g/m^3$ on risks associated with short-term exposure.

5.3.1.3 Summary

In considering these estimates of $PM_{2.5}$-related 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 that they provide support for consideration of standards that would provide increased protection beyond that afforded by the current $PM_{2.5}$ standards. In the absence of evidence of clear thresholds, staff believes it is appropriate to consider all the estimates associated with the range of assumed cutpoints used in the risk assessment. Staff believes that a relatively more precautionary approach to interpreting this evidence would give more weight to the estimates based on the lowest cutpoints considered. Staff also takes note of the view expressed by the CASAC PM Panel which "favored the primary use of an assumed threshold of 10 $\mu g/m^3$." (Henderson, 2005). Regardless of the relative weight placed on the estimates associated with either an assumed cutpoint of 10 $\mu g/m^3$ or the lowest cutpoints considered, the risk assessment indicates the likelihood that thousands of premature deaths per year would occur in urban areas across the U.S. even upon attainment of the current $PM_{2.5}$ standards. 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 it is important to recognize how highly dependent any specific risk estimates are on the shape of the underlying concentration-response functions. In so doing, staff nonetheless reiterates that based on even the highest assumed cutpoints used in the risk assessment, 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 data.

Staff also 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 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. 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. For these reasons, EPA concluded 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 size cut on a number of considerations. In focusing on a size cut 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 size cut 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 size cut 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.[3] Thus, EPA's selection of 2.5 μm as the size cut 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 potential for limited 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, the same considerations continue to apply for selection of an appropriate indicator for fine particles. As an initial matter, 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 9-3 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. 8-67 and 8-68, 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

[3] 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).

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 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, suggesting 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 not sufficient 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 sulfates, transition metals, and organics that have been associated with health effects in epidemiologic and/or toxicologic studies, 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 size cut 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 size cut 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 size cut for the separation of the overlapping fine and coarse particle modes (CD, p. 9-8). Within this range, staff considered size cuts of both 1 μm and 2.5 μm. Consideration of these two size cuts 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 size cut 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 – 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 size cut for a fine particle mass indicator.

Consistent with that conclusion, 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 method (FRM) sampler for $PM_{2.5}$. The new monitoring network has provided substantial new air quality information, in terms of $PM_{2.5}$, that has been and is being used in ongoing PM research and air quality analyses that inform this review. EPA also has conducted studies to evaluate options for improvements to the FRM. As a result of continuing evaluation of the monitoring network, staff is considering changes to the $PM_{2.5}$ FRM to improve performance and minimize the burden on agencies conducting the monitoring.[4] Staff is also considering the addition of federal equivalent method (FEM) designation criteria for continuous fine particle monitors.[5] Continuous monitoring is advantageous in providing additional data for many purposes, including compliance monitoring, health studies, and air quality forecasting, and it can also ease the burden of data collection for regulatory agencies.

[4] Changes to the $PM_{2.5}$ FRM being considered by staff are discussed in Hanley (2005).

[5] This work is being done in consultation with the CASAC Subcommittee on Ambient Air Monitoring and Methods (AAMM).

5.3.3 Averaging Times

In the last review, EPA established two $PM_{2.5}$ standards, based on annual and 24-hour averaging times (62 FR at 38,668-70). This decision was based in part on evidence of health effects related to both short-term (from less than 1 day to up to several days) and long-term (from a year to several years) measures of PM. EPA noted that the large majority of community epidemiologic studies reported associations based on 24-hour averaging times or on multiple-day averages. Further, EPA noted that a 24-hour standard could also effectively protect against episodes lasting several days, as well as providing some degree of protection from potential effects associated with shorter duration exposures. EPA also recognized that an annual standard would provide effective protection against both annual and multi-year, cumulative exposures that had been associated with an array of health effects, and that a much longer averaging time would complicate and unnecessarily delay control strategies and attainment decisions. The possibility of seasonal effects also was considered, although the very limited available evidence of such effects and the seasonal variability of sources of fine particle emissions across the country did not provide a satisfactory basis for establishing a seasonal averaging time.

In considering whether the information available in this review supports consideration of different averaging times for $PM_{2.5}$ standards, staff notes that the available information is generally consistent with and supportive of the conclusions reached in the last review to set $PM_{2.5}$ standards with both annual and 24-hour averaging times. In considering the new information, staff makes the following observations:

- There is a growing body of studies that provide additional evidence of effects associated with exposure periods shorter than 24-hours (e.g., one to several hours), as discussed in Chapter 3 (section 3.6.5.3). While staff concludes that this information remains too limited to serve as a basis for establishing a shorter-than-24-hour fine particle primary standard at this time, staff believes that it gives added weight to the importance of a standard with a 24-hour averaging time. Staff recognizes shorter-than-24-hour exposures as an important area of research that could provide a basis for the consideration of a shorter-term standard in the future.

- As discussed in Chapter 3 (section 3.6.5.1), some recent PM_{10} studies have used a distributed lag over several days to weeks preceding the health event, although this modeling approach has not been extended to studies of fine particles. While such studies continue to suggest consideration of a multiple day averaging time, staff notes that limiting 24-hour concentrations of fine particles will also protect against effects found to be associated with PM averaged over many days in health studies. Consistent with the conclusion reached in the last review, staff again concludes that a multiple-day averaging time would add complexity but would not provide more effective protection than a 24-hour average.

- While some newer studies have investigated seasonal effects, as noted in Chapter 3 (section 3.6.5.2), staff concludes that currently available evidence of such effects is still too limited to serve as a basis for considering seasonal standards.

Based on the above considerations, staff concludes that the currently available information supports keeping, and provides no adequate basis for changing, the averaging times of the current $PM_{2.5}$ standards. Staff notes that study of shorter-term averaging times, on the order of one or more hours, is an important research priority, with a particular focus on associations between exposure to fine particles and fine-particle constituents and indicators of cardiac-related risk factors. Thus, a shorter-term averaging time may be an important consideration in the next review of the PM NAAQS. Staff also notes that at present EPA has in place a significant harm level program (40 CFR Part 51) and a widely disseminated Air Quality Index that could potentially be adapted 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, some key considerations that led to establishing a generally controlling annual standard in the last review are still valid. In particular,

- EPA's updated risk assessment supports the previous 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 4.4.4). 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 3.4.5, 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 effects associated with 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 (CD, pp. 9-33 to 9-34).

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 long-term exposure 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, may reasonably 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 $\mu g/m^3$, within an overall range of 11 to 30 $\mu g/m^3$. In the studies using the ACS cohort, the long-term mean $PM_{2.5}$ concentration across the cities was 21 $\mu g/m^3$ in the initial study and in the reanalysis of that study, within an overall range of 9 to 34 $\mu g/m^3$. In the extended ACS study, the mean for the more recent time period used in the analysis (from 1999 to 2000) was 14 $\mu g/m^3$; 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, as explained in Chapter 4), the long-term mean $PM_{2.5}$ concentration was 17.7 $\mu g/m^3$, with a standard deviation of $\pm$ 4, ranging down to 7.5 $\mu g/m^3$. 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 $\mu g/m^3$. 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 $\mu g/m^3$, ranging from 7 to 32 $\mu g/m^3$ 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 $\mu g/m^3$, ranging from 9 to 17 $\mu g/m^3$ 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 $\mu g/m^3$, down to about 12 $\mu g/m^3$. A standard of 14 $\mu g/m^3$ 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 $\mu g/m^3$ 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 $\mu g/m^3$ 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 $\mu g/m^3$ would be consistent with a judgment that a more precautionary standard was warranted, potentially reflecting consideration of the seriousness of the mortality effects, for which there is strong evidence of likely causal relationships, and of the limited but suggestive evidence of possible links to effects on fetal and infant development and mortality. As discussed in Chapter 1, these factors are relevant to judgments about providing an adequate margin of safety to prevent pollution levels that may pose an unacceptable risk of harm, even if the risk is not precisely identified as to nature or degree. In staff's view, a standard set below this range would be highly precautionary, giving little weight to the remaining uncertainties in the broader body of evidence, which includes other long-term exposure studies that provide far more inconsistent results.

5.3.4.2 Risk-based Considerations

Beyond looking directly at the relevant epidemiologic evidence, staff also has considered the extent to which specific levels and forms of alternative $PM_{2.5}$ standards are likely to reduce the estimated risks attributable to long-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 long-term exposure mortality risks, based on the extended ACS study, were estimated using the reported concentration-response function down to a level of 7.5 $\mu g/m^3$, the lowest measured level (LML) in that study, as well as using modified concentration-response functions that incorporate alternative assumed cutpoints as surrogates for potential population thresholds.

Figures 5.1(a), (b), and (c) show the estimated percentage reductions in mortality attributable to long-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 (with a 98th percentile form) in the five example cities that do not meet the current standards (based on 2001-2003 air quality data), based on assumed cutpoints of 7.5, 10, and 12 $\mu g/m^3$, respectively. To put the estimated percentage reductions in perspective, these figures also include the estimated $PM_{2.5}$-related annual incidence rate (in terms of deaths/year/100,000 general population) and annual incidence (in terms of deaths/year) of total mortality associated with long-term exposure associated with just meeting the current $PM_{2.5}$ standards. A similar series of figures is shown in Appendix 5A for meeting alternative 24-hour standards with a 99th percentile form. The alternative annual $PM_{2.5}$ standards considered in these figures include a range of levels from 15 to 12 $\mu g/m^3$. Attainment of the standards is simulated based on a percent rollback calculated using the highest monitor in an area, as noted in Tables 5-1(a) and (b) and discussed in Chapter 4, section 4.2.2. The alternative 24-hour $PM_{2.5}$ standards considered in these figures include a range of levels from 65 to 25 $\mu g/m^3$. Further discussion of alternative forms of the annual and 24-hour standards is presented below in section 5.3.6.

In considering the estimates based on a cutpoint of 7.5 $\mu g/m^3$ [Figures 5-1(a) and 5A-1(a)], staff first examined the estimated reductions associated with lower levels of the annual $PM_{2.5}$ standard, without changing the 24-hour standard. Staff observes that alternative annual standard levels of 14, 13, and 12 $\mu g/m^3$ 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 this assumed cutpoint, 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 $\mu g/m^3$. Staff also examined the effect on mortality reduction associated with alternative 24-hour standards, without changing the annual standard. 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 $\mu g/m^3$, 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 estimates of long-term mortality risk reduction, based on a cutpoint of 7.5 $\mu g/m^3$, associated with a 24-hour standard set at 25 $\mu g/m^3$ provides the same degree of risk reduction regardless of the level of the annual standard within the range of 15 to 12 $\mu g/m^3$; a 24-hour standard set at 30 $\mu g/m^3$ provides the same degree of risk reduction in most

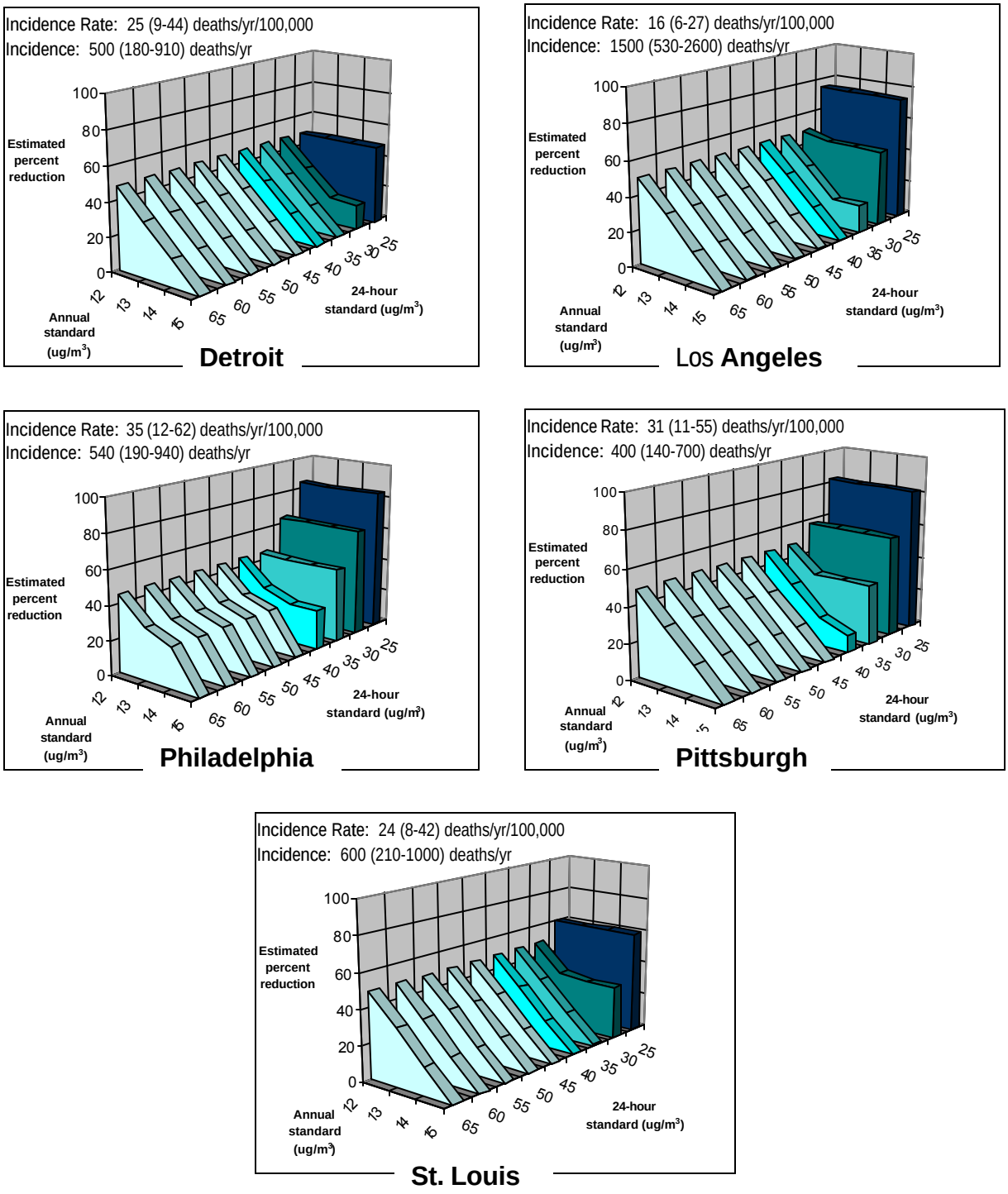

Figure 5-1(a) Estimated percent reduction in $PM_{2.5}$-related long-term mortality risk for alternative standards *(98th percentile form)* relative to risk associated with meeting current standards (based on assumed cutpoint of 7.5 $\mu g/m^3$). Risk associated with meeting current $PM_{2.5}$ standards, based on ACS extended study, is shown in figures in terms of estimated annual incidence rate and annual incidence (and 95% confidence ranges).

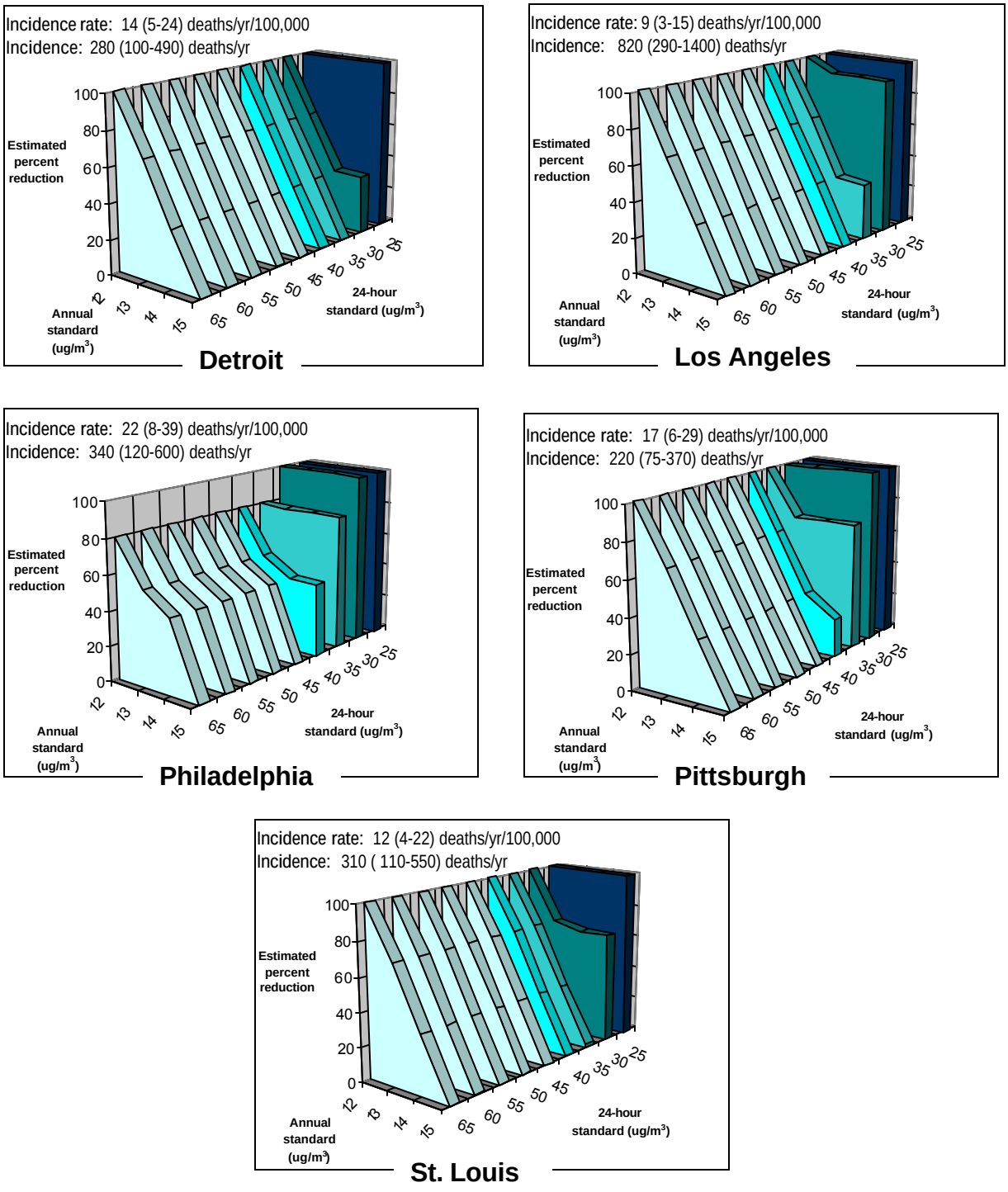

Figure 5-1(b) Estimated percent reduction in $PM_{2.5}$-related long-term mortality risk for alternative standards *(98th percentile form)* relative to risk associated with meeting current standards (based on assumed cutpoint of *10 µg/m³*). Risk associated with meeting current $PM_{2.5}$ standards, based on ACS extended study, is shown in figures in terms of estimated annual incidence rate and annual incidence (and 95% confidence ranges).

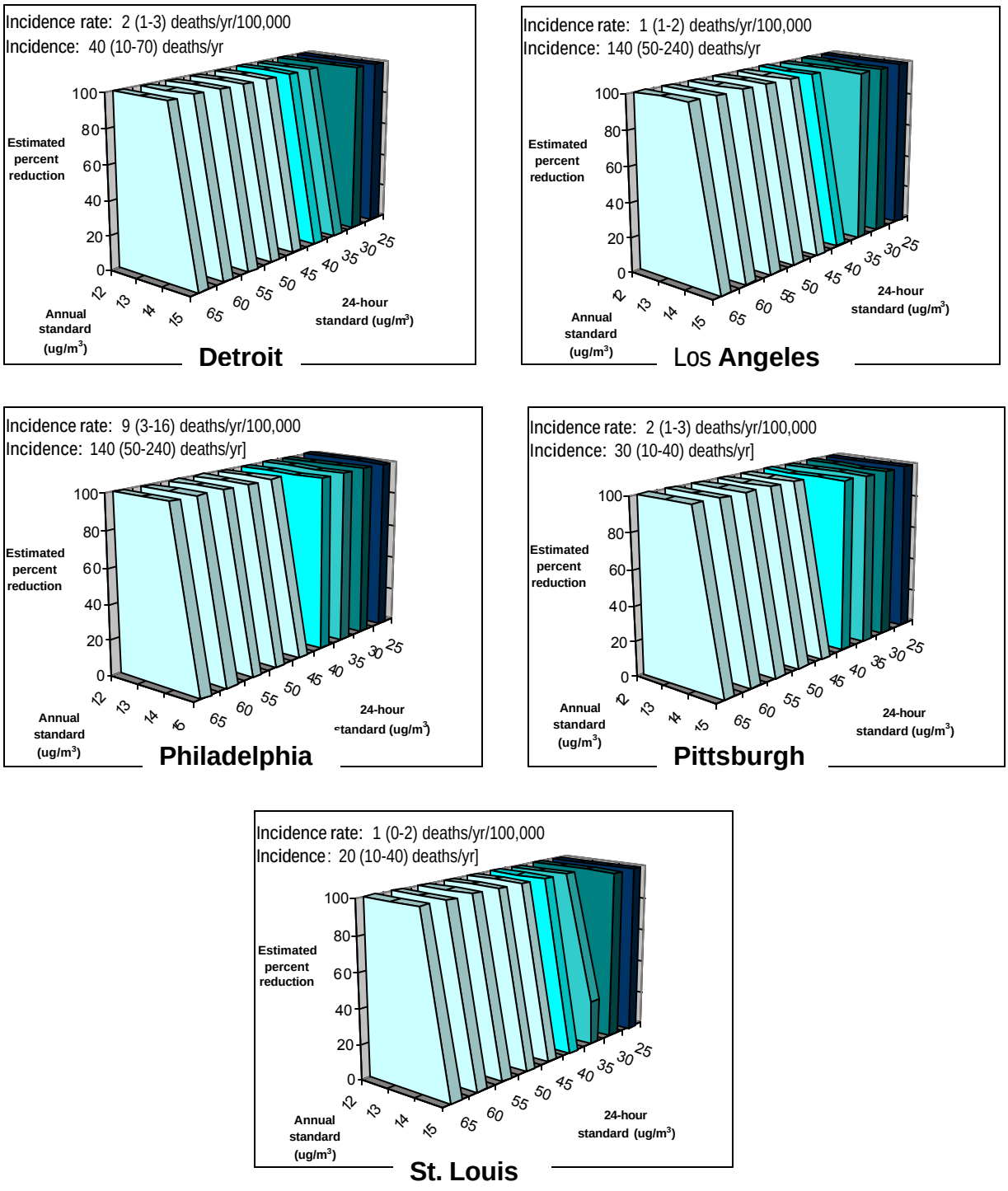

Figure 5-1(c) Estimated percent reduction in $PM_{2.5}$-related long-term mortality risk for alternative standards *(98th percentile form)* relative to risk associated with meeting current standards (based on assumed cutpoint of *12 µg/m³*). Risk associated with meeting current $PM_{2.5}$ standards, based on ACS extended study, is shown in figures in terms of estimated annual incidence rate and annual incidence (and 95% confidence ranges).

but not all cases. That is, in the range of 30 to 25 $\mu g/m^3$, 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 $\mu g/m^3$; and, in those cases, lowering the annual standard to as low as 12 $\mu g/m^3$ would result in no additional estimated reductions in long-term mortality risks.

In considering the estimates of risk reductions based on an assumed cutpoint of 10 $\mu g/m^3$ [Figures 5-1(b) and 5A-1(b)], staff again notes that the estimates of mortality incidence and incidence rate associated with meeting the current standards are roughly about half as large as the estimates based on a cutpoint of 7.5 $\mu g/m^3$, as discussed above in section 5.3.1. Staff observes that lowering the annual standard to alternative levels of 14, 13, and 12 $\mu g/m^3$ (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 $\mu g/m^3$ (with either a 98th or 99th percentile form), in combination with the current annual standard. For a 24-hour standard set at 30 $\mu g/m^3$ with a 98th percentile form, in combination with the current annual standard, estimated risk reductions remain at or close to 100 percent in three of the cities, but are appreciably lower in the other two cities. A 24-hour standard set at 35 $\mu g/m^3$ with a 98th percentile form results in appreciable risk reductions in only two of the cities in conjunction with the current annual standard, although appreciable risk reductions are observed with this 24-hour standard in conjunction with a lower annual standard.

Further, in considering an assumed cutpoint of 12 $\mu g/m^3$ [Figures 5-1(c) and 5A-1(c)], staff observes that lowering the annual standard to a level of 14 $\mu g/m^3$ (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 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, staff believes that it is appropriate to consider all the estimates associated with the range of assumed cutpoints used in the risk assessment. As discussed above

in section 5.3.1.3, staff believes that a relatively more precautionary approach to interpreting this evidence would give more weight to the estimates based on the lowest cutpoint considered, while giving more weight to the estimates based on an assumed cutpoint of 10 $\mu g/m^3$ is consistent with the view of the CASAC PM Panel. Taking into account the estimated risk reductions based on the use of either cutpoint, 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 $\mu g/m^3$, in conjunction with a 99^{th} percentile form especially with a standard level in the middle to upper end of this range or with a 98^{th} percentile form with a standard level in the middle to lower 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 99^{th} or 98^{th} percentile form, respectively) relative to that afforded by the current annual $PM_{2.5}$ standard, regardless of the weight that is given to the alternative assumed cutpoints in 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 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 extent to which the reported associations are robust to co-pollutant confounding and alternative modeling approaches; and the extent to which 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 typical background levels, such that consideration of the likely range of policy-relevant 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 98^{th} and 99^{th} 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 associations 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 98^{th} 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; 99^{th} 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 Santa Clara Country. These ranges also encompass the 98^{th} and 99^{th} percentile values from the

short-term exposure studies that reported positive PM-related effects and have long-term mean $PM_{2.5}$ concentrations at and somewhat above the current annual $PM_{2.5}$ standard [up to 18 $\mu g/m^3$, as summarized in Ross and Langstaff (2005)]. Based on this information, staff believes that the range of alternative 24-hour $PM_{2.5}$ standards appropriate for consideration should extend below the ranges of 98th and 99th percentile values reported in the studies identified above, so as to provide protection from the short-term exposure effects seen in these studies.

Since the available epidemiologic evidence provides no clear basis for identifying the lower end of the range of consideration for a 24-hour standard level, staff has looked to the information on background concentrations, recognizing that a standard intended to provide protection from man-made pollution should be set above background levels. As discussed in Chapter 2, section 2.6, staff notes that long-term average $PM_{2.5}$ daily background levels are quite low (ranging from 1 to 5 $\mu g/m^3$ across the U.S.), although the upper end (99th percentile values) of daily distributions of background levels are estimated to extend from approximately 10 to 20 $\mu g/m^3$ in regions across the U.S, although such levels may include some undetermined contribution from anthropogenic emissions (Langstaff, 2004). Even higher daily background levels result from episodic occurrences of extreme natural events (e.g., wildfires, 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. Based on consideration of these background levels, staff believes that 25 $\mu g/m^3$ is an appropriate lower end to the range of 24-hour $PM_{2.5}$ standards for consideration in this review. Thus, based on this evidence, staff concludes it is appropriate to consider alternative 24-hour $PM_{2.5}$ standards, with either a 98th or 99th percentile form, that range down to as low as 25 $\mu g/m^3$ to provide protection from effects associated with short-term exposures to $PM_{2.5}$.

As in the last review, staff believes it is also appropriate to consider the evidence discussed above as a basis for a alternative annual $PM_{2.5}$ standards that would address risks associated with short-term exposures. In the last review, annual standard levels were considered at or somewhat below the long-term mean concentrations in short-term exposure studies reporting statistically significant associations, recognizing that the evidence of an association in such studies is strongest at and around this long-term mean, where the data in the study are most concentrated. This approach follows from the observation that, when aggregated on an annual basis, much of the risk related to daily exposures 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 4.4.4) and in section 5.3.3 above. Thus, to reduce the aggregate risk, it is necessary to shift the bulk of the distribution to lower levels, not just to limit the concentrations on days when the $PM_{2.5}$ concentrations are relatively high. Shifting the distribution can be accomplished through control strategies aimed at attaining either an annual or 24-hour standard.

Using this approach, the same short-term exposure studies identified above can be considered as a basis for alternative levels of an annual standard that would provide additional

protection from effects associated with short-term exposures. In particular, the multi-city Canadian study (Burnett et al., 2000 and Burnett and Goldberg, 2003) reports statistically significant associations between short-term $PM_{2.5}$ exposure and total and cardiovascular mortality across areas with an aggregate long-term mean $PM_{2.5}$ concentration of 13.3 $\mu g/m^3$. The other two studies, conducted in Phoenix (Mar et al., 1999, 2003) and Santa Clara County, CA (Fairley, 1999, 2003), each had long-term mean $PM_{2.5}$ concentrations of approximately 13 $\mu g/m^3$. In considering this evidence, staff concludes that these studies provide a basis for considering an annual $PM_{2.5}$ standard within the range of 13 $\mu g/m^3$ to about 12 $\mu g/m^3$. An annual standard of 13 $\mu g/m^3$ would be consistent with a judgment that appreciable weight should be accorded these studies as a basis for an annual standard that would protect against $PM_{2.5}$-related mortality associated with short-term exposure. An annual standard of 12 $\mu g/m^3$, somewhat below the long-term means in these studies, would be consistent with a judgment that a more precautionary standard was warranted. Such a standard could potentially reflect consideration of the seriousness of the mortality effects, for which there is strong evidence of a likely causal relationship, as well as the much more uncertain evidence of respiratory-related emergency department visits, discussed above in section 5.3.1, in studies with long-term mean $PM_{2.5}$ concentrations of approximately 12 $\mu g/m^3$ and below. As discussed in Chapter 1 and above in section 5.3.4.1, these considerations are relevant to judgments about providing an adequate margin of safety to prevent pollution levels that may pose an unacceptable risk of harm, even if the risk is not precisely identified as to nature or degree. In staff's view, an annual standard set below this range would be highly precautionary based on the 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 statistical 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, in conjunction with an annual standard that may be adopted in part to provide protection against effects associated with short-term exposures, staff believes it is appropriate also to consider alternative 24-hour $PM_{2.5}$ standards as well. Such a 24-hour standard could reasonably be based on air quality information (from 2001 to 2003) in Chapter 2, Figure 2-25, that shows the distribution of 98^{th} percentile values as a function of annual means values in urban areas across the U.S. Based on this information, staff concludes that a 24-hour standard in the range of approximately 40 to 35 $\mu g/m^3$ could 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 13 μg/m^3.

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 using reported city-specific concentration-response functions down to policy-relevant background, as well as using modified concentration-response functions that incorporate alternative assumed cutpoints as surrogates for potential population thresholds.

Figures 5-2(a), (b), (c), and (d) show the 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 (with a 98th percentile form) in the five example cities that do not meet the current standards (based on 2001-2003 air quality data), based on assumed cutpoints equal to estimated policy-relevant background and 10, 15, and 20 μg/m^3, respectively. To put the estimated percentage reductions in perspective, these figures also include the estimated $PM_{2.5}$-related annual incidence rate (in terms of deaths/year/100,000 general population) and annual incidence (in terms of deaths/year) of total mortality associated with short-term exposure associated with just meeting the current $PM_{2.5}$ standards. A similar series of figures is shown in Appendix 5A for meeting alternative 24-hour standards with a 99th percentile form. As in the figures for long-term exposures discussed in section 5.3.4.2, the alternative annual $PM_{2.5}$ standards considered in these figures include a range of levels from 15 to 12 μg/m^3, and attainment of the standards is simulated based on a percent rollback calculated using the highest monitor in an area, as noted in Tables 5-1(a) and (b) and discussed in Chapter 4, section 4.3.1. The alternative 24-hour $PM_{2.5}$ standards considered in these figures include a range of levels from 65 to 25 μg/m^3. Further discussion of alternative forms of the annual and 24-hour standards is presented below in section 5.3.6.

In considering the estimates based on a cutpoint level equal to estimated policy-relevant background [Figures 5-2(a) and 5A-2(a)], staff first examined the estimated reductions associated with lower levels of the annual $PM_{2.5}$ standard, without changing the 24-hour standard. Staff observes that lowering the annual standard to alternative levels of 14, 13, and 12 μg/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 this assumed cutpoint, 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 μg/m^3. In examining the effect of changes to the 24-hour and/or annual $PM_{2.5}$ standards in this case, staff first notes that the estimated reductions in

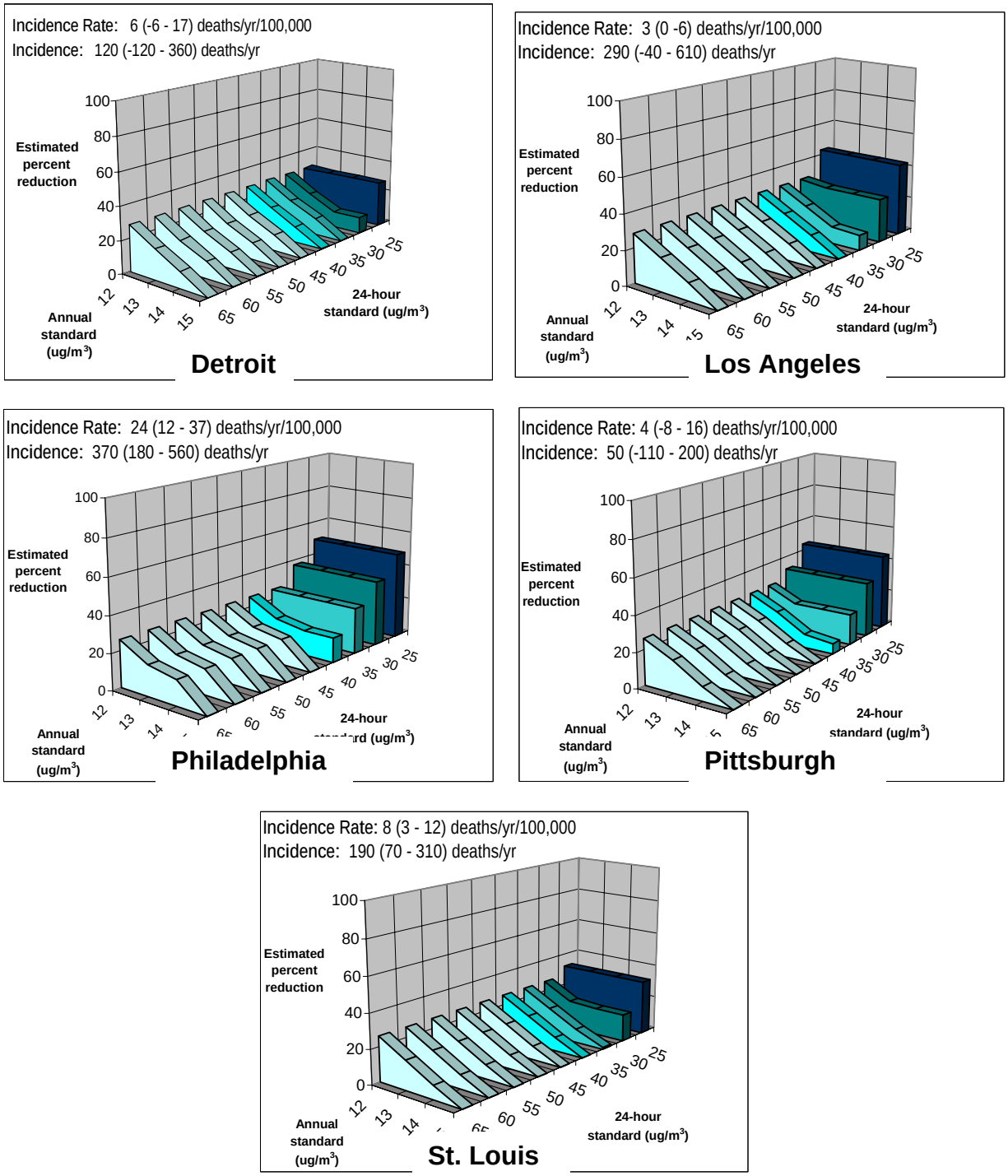

Figure 5-2(a) Estimated percent reduction in $PM_{2.5}$-related short-term mortality risk for alternative standards *(98th percentile form)* relative to risk associated with meeting current standards (based on assumed cutpoint equal to policy-relevant background). Risk associated with meeting current $PM_{2.5}$ standards is shown in figures in terms of estimated annual incidence rate and annual incidence (and 95% confidence ranges). Estimated policy-relevant background is 3.5 µg/m^3 in eastern cities and 2.5 µg/m^3 in western cities.

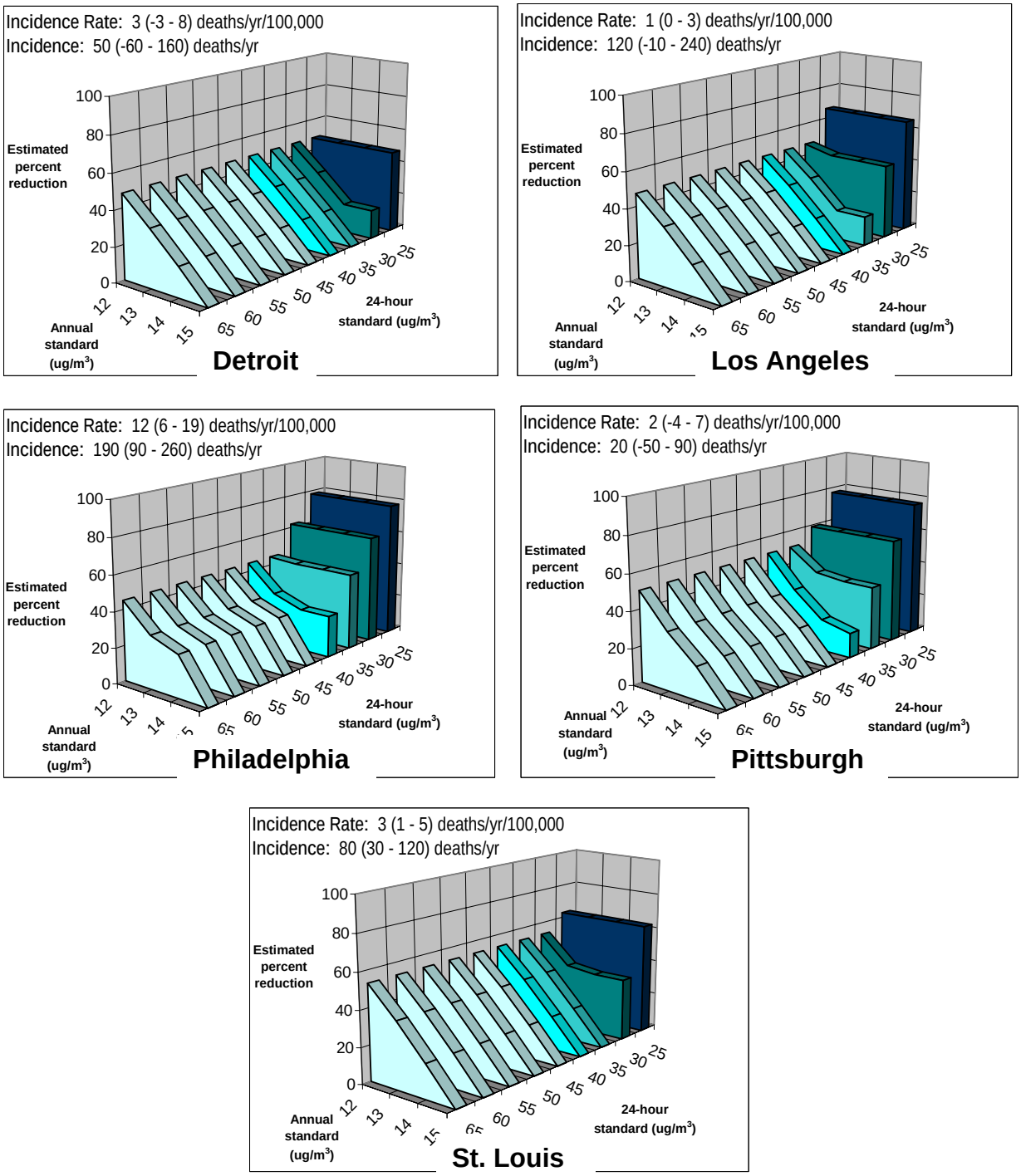

Figure 5-2(b) Estimated percent reduction in $PM_{2.5}$-related short-term mortality risk for alternative standards *(98th percentile form)* relative to risk associated with meeting current standards (based on assumed cutpoint of 10 µg/m^3). Risk associated with meeting current $PM_{2.5}$ standards is shown in figures in terms of estimated annual incidence rate and annual incidence (and 95% confidence ranges).

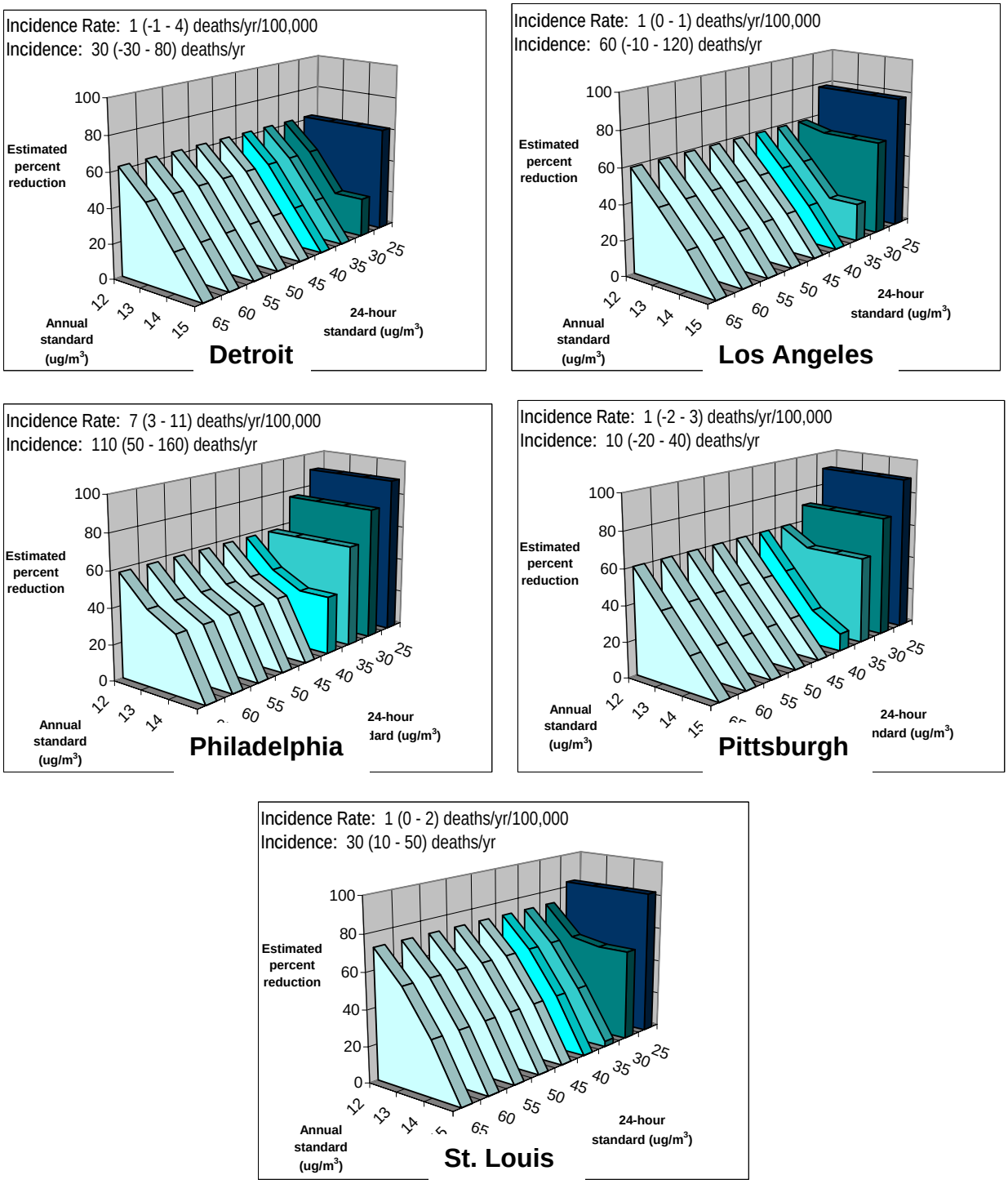

Figure 5-2(c) Estimated percent reduction in $PM_{2.5}$-related short-term mortality risk for alternative standards *(98th percentile form)* relative to risk associated with meeting current standards (based on assumed cutpoint of 15 µg/m^3). Risk associated with meeting current $PM_{2.5}$ standards is shown in figures in terms of estimated annual incidence rate and annual incidence (and 95% confidence ranges).

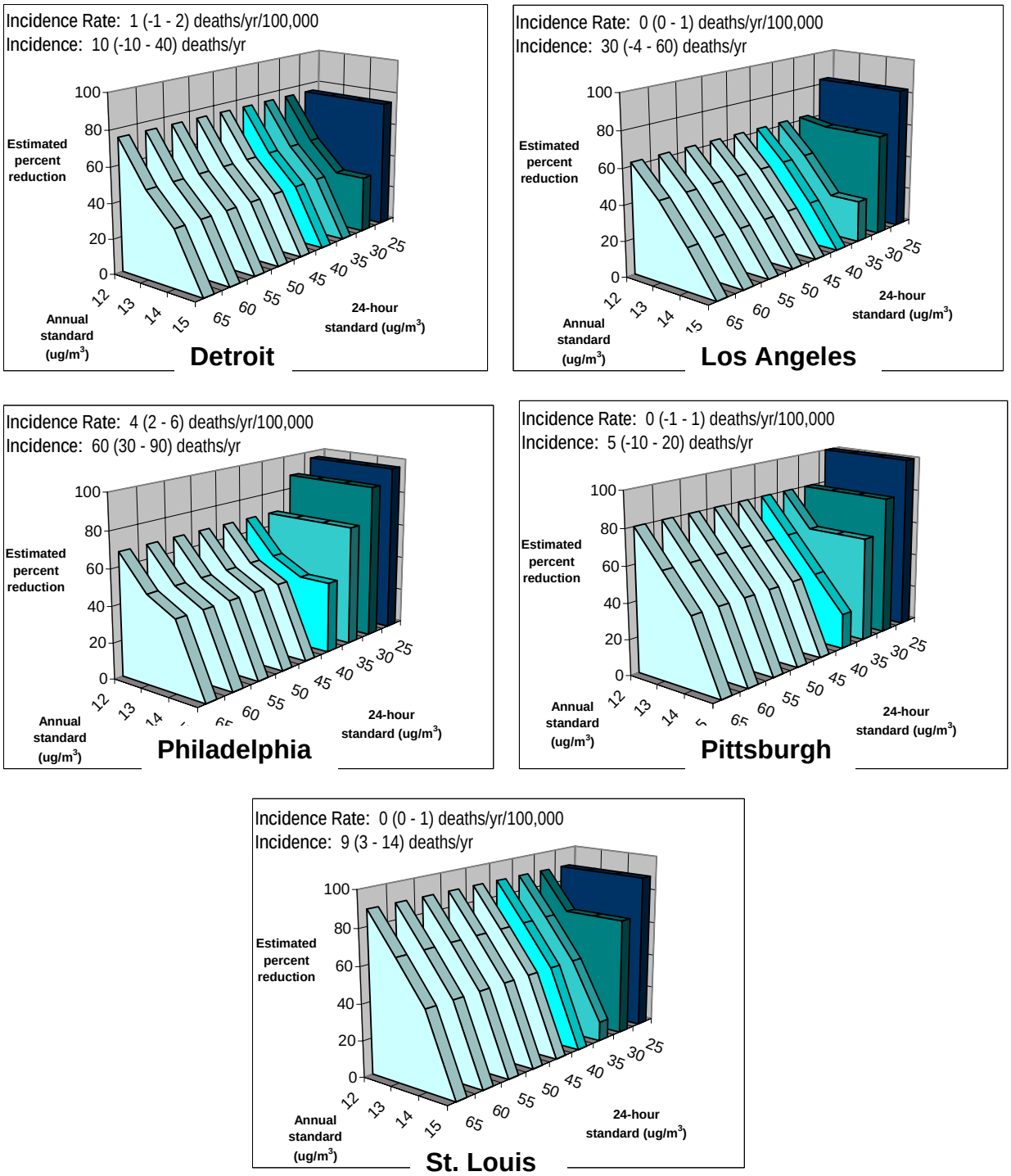

Figure 5-2(d) Estimated percent reduction in $PM_{2.5}$-related short-term mortality risk for alternative standards *(98th percentile form)* relative to risk associated with meeting current standards (based on assumed cutpoint of 20 µg/m³). Risk associated with meeting current $PM_{2.5}$ standards is shown in figures in terms of estimated annual incidence rate and annual incidence (and 95% confidence ranges).

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 estimated reductions in short-term mortality ranging from approximately 30 to 50 percent (98^{th} percentile form) and 35 to 70 percent (99^{th} 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 estimates of reductions in short-term mortality ranging from approximately 25 to 35 percent (98^{th} percentile form) and 25 to 65 percent (99^{th} percentile form) across the five cities in conjunction with an annual standard of 12 $\mu g/m^3$; the lower end, but not the upper end, of these ranges decreases somewhat in conjunction with annual standards from 13 to 15 $\mu g/m^3$. 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 $\mu g/m^3$ become generally controlling standards in most cases within this range of annual standards.

In considering the estimates of risk reductions based on an assumed cutpoint of 10 $\mu g/m^3$ [Figures 5-2(b) and 5A-2(b)], staff observes that lowering the annual standard to alternative levels of 14, 13, and 12 $\mu g/m^3$ (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 $\mu g/m^3$ results in estimates of reductions in short-term mortality ranging from approximately 45 to 80 percent (98^{th} percentile form) and 60 to 95 percent (99^{th} 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 estimates of reductions in short-term mortality ranging from approximately 45 to 60 percent (98^{th} percentile form) and 50 to 95 percent (99^{th} percentile form) across the five cities in conjunction with an annual standard of 12 $\mu g/m^3$; as with the previous case (based on a cutpoint equal to policy-relevant background), the lower end, but not the upper end, of these ranges decreases appreciably in conjunction with annual standards from 13 to 15 $\mu g/m^3$. Thus, in this case, as in the previous 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 cutpoints of 15 or 20 $\mu g/m^3$, staff observes that lowering the annual standard to alternative levels of 14, 13, and 12 $\mu g/m^3$ (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 these cases, staff notes that a 24-hour standard of 25 $\mu g/m^3$ 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 $\mu g/m^3$. A 24-hour standard of 30 $\mu g/m^3$ 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 $\mu g/m^3$; similarly, the lower end, but not the upper end, of these ranges decreases appreciably in conjunction with annual standards from 13 to 15 $\mu g/m^3$. Thus, in these cases 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 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 $\mu g/m^3$ in conjunction with retaining the current annual standard level of 15 $\mu g/m^3$. Alternatively, staff also believes the evidence supports consideration of a revised annual standard, in the range of 13 to 12 $\mu g/m^3$, in conjunction with a revised 24-hour standard in the range of 40 to 35 $\mu g/m^3$.

In considering the results of the quantitative risk assessment, staff believes that it is appropriate to consider all the estimates associated with the range of assumed cutpoints used in the risk assessment. As discussed above in sections 5.3.1.3 and 5.3.4.3, staff believes that a relatively more precautionary approach to interpreting this evidence would give more weight to the estimates based on the lowest cutpoint considered, while giving more weight to the estimates based on an assumed cutpoint of 10 $\mu g/m^3$ is consistent with the view of the CASAC PM Panel. Taking into account the estimated risk reductions based on the use of either cutpoint, staff finds additional support for considering a revised 24-hour standard in the range of 30 to 25 $\mu g/m^3$ in conjunction with retaining an annual standard level of 15 $\mu g/m^3$. In either case, a 24-hour standard at a level of 35 $\mu g/m^3$ 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) relative to that afforded by the current annual $PM_{2.5}$ standard alone. Further, in conjunction with a lower annual standard down to 12 $\mu g/m^3$, staff finds support for considering a revised 24-hour standard in the range of 35 to 30 $\mu g/m^3$. 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 $\mu g/m^3$. 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 areas across the U.S..

To provide some perspective on the implications of applying various combinations of alternative annual and 24-hour standards, staff assessed (based on 2001 to 2003 air quality) 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, shown in Appendix 5B (Tables 5B-1(a) and (b), for 98th and 99th percentile forms of the 24-hour standards, respectively), was not considered as a basis for the above staff conclusions.

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 current 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 calculating compliance using air quality values from the highest community-oriented monitor in an area, with estimated risks based on using air quality values averaged across all such monitors within the constraints allowed by the current standard. As discussed in Chapter 4, section 4.3.1, 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 (i.e., the estimated reductions in risk associated with just attaining the current or alternative annual standards are less when spatial averaging 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 where 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 3.5, 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 $PM_{2.5}$.

The allowance to use spatial averaging under certain constraints established in 1997 was intended to provide for 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 areas 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 avoid substantially greater exposures in some areas, potentially resulting in disproportionate impacts on potentially vulnerable subpopulations. Thus, in considering whether alternative constraints on the use of spatial averaging may be appropriate, 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, which is 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.

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 98^{th} percentile of 24-hour concentrations at each population-oriented monitor within an area, averaged over three years (62 FR at 38671-74). EPA selected such a concentration-based form because of its

advantages over the previously used expected-exceedance form.[6] A concentration-based form is more reflective of the health risk posed by elevated $PM_{2.5}$ concentrations because it gives proportionally greater weight to days when concentrations are well above the level of the standard than to days when the concentrations are just above the standard. Further, a concentration-based form better compensates for missing data and less-than-every-day monitoring; and, when averaged over 3 years, it has greater stability and, thus, facilitates the development of more stable implementation programs. After considering a range of concentration percentiles from the 95th to the 99th, EPA selected the 98th percentile as an appropriate balance between adequately limiting the occurrence of peak concentrations and providing increased stability and robustness. Further, by basing the form of the standard on concentrations measured at population-oriented monitoring sites (as specified in 40 CFR part 58), EPA intended to provide protection for people residing in or near localized areas of elevated concentrations.

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,

[6] 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.

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 which do not meet the current annual standard (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 $\mu g/m^3$ above the 98th or 99th percentile values, with very few excursions above this range.[7]

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 degree of risk reduction likely to result from the combination of the form and the level of a standard. Staff also

[7] 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.

believes it is appropriate to take into account whether the 24-hour standard is set so as to add to the protection afforded by a revised 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 relatively stability of a standard with either form as well as the implications from a public health communication perspective of the extent to which either form allows 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 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 99^{th} percentile form or, alternatively, to retaining the 98^{th} percentile form, based in part on considering the degree of risk reduction likely to result from the combination of the form and the level of a standard.

(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 $\mu g/m^3$, based a 98^{th} percentile form for a standard set at the middle to lower end of this range, or a 99^{th} percentile form for a standard set at the middle to upper end of this range. Staff judges that such a suite of standards 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 in the range of 30 to 40 $\mu g/m^3$. Staff judges that a suite of

standards that includes either the annual or the 24-hour standard, or both, set at the middle to lower end of these ranges 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 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 dominant component of PM_{10}, namely two fugitive dust studies in areas that substantially exceeded the PM_{10} standards (62 FR 38,679). The decision also reflected the fact that there were 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, but that the only information available upon which to base such standards was indexed in terms of PM_{10}.[8]

In the present review, staff has taken into account the information now available from a growing, but still limited, body of evidence on health effects associated with thoracic coarse particles from studies that use $PM_{10-2.5}$ as a measure of thoracic coarse particles. In addition, staff notes that there is now much more information available to characterize air quality in terms of estimated $PM_{10-2.5}$ than was available in the last review.[9] In considering this information, staff now finds that the major considerations that formed the basis for EPA's 1997 decision to retain PM_{10} as the indicator for thoracic coarse particles, rather than a more narrowly defined indicator that does not include fine particles, no longer apply. In particular, staff concludes that the

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[8] 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 $PM_{2.5}$. American Trucking Associations v. EPA, 175 F. 3d. at 1054. Although the court found "ample support" (id.) for EPA's decision to regulate thoracic coarse particles, it vacated the 1997 revised PM_{10} standards for that reason.

[9] As noted in section 2.3.5, coarse particle concentrations in EPA's monitoring network are currently estimated, not measured directly, using a difference method in locations with same-day data from co-located PM_{10} and $PM_{2.5}$ FRM monitors, resulting in air quality characterizations that are more uncertain than those available for $PM_{2.5}$ or PM_{10}.

continued use of PM_{10} as an indicator for standards intended to protect against health effects associated with thoracic coarse particles is no longer appropriate since information is now available that supports the use of a more directly relevant indicator, $PM_{10-2.5}$. Further, staff concludes that continuing to rely principally on health effects evidence indexed by PM_{10} is no longer appropriate since more directly relevant studies, indexed by $PM_{10-2.5}$, are also now available. Thus, quite aside from any legal considerations, staff finds that it is appropriate to revise the current PM_{10} standards in part by revising the indicator for thoracic coarse particles, and by basing any such revised standard principally on the currently available evidence and air quality information indexed by $PM_{10-2.5}$, but also considering evidence from studies using PM_{10} in locations where $PM_{10-2.5}$ is the predominant fraction.

Staff has also considered whether the currently available evidence and information support consideration of standards for thoracic coarse particles that afford either a similar or notably different degree of public health protection compared to that afforded by the current PM_{10} standards. In so doing, staff first focused on dosimetric and toxicologic evidence, then on relevant findings from epidemiologic studies, followed by consideration of risk-based information, as discussed below.

Dosimetric evidence formed the primary basis for initial development of the PM_{10} indicator. While considerable advances have been made, the available evidence continues to support the basic conclusions reached in the 1987 and 1997 reviews of the standards regarding penetration and deposition of size specific particles; an aerodynamic size of 10 μm remains a reasonable separation point for particles that penetrate and potentially deposit in the thoracic regions of the lungs, particularly for the more sensitive case of mouth breathing. As discussed in Chapter 3, both fine and thoracic coarse particles penetrate to and deposit in the alveolar and tracheobronchial regions. For a range of typical ambient size distributions, the total deposition of thoracic coarse particles to the alveolar region can be comparable to or even larger than that for fine particles. For areas with appreciable coarse particle concentrations, coarse particles would tend to dominate particle deposition to the tracheobronchial region for mouth breathers (CD, p. 6-16).

As noted in past reviews (EPA, 1981b, 1996b), deposition of a variety of particle types in the tracheobronchial region, including resuspended urban dust and coarse-fraction organic materials, has the potential to affect lung function and aggravate symptoms, particularly in asthmatics. Of particular note are limited toxicologic studies that found urban road dust can produce cellular and immunological effects (e.g., Kleinman, et al., 1995; Steerenberg et al., 2003). In addition, the CD notes that some very limited *in vitro* toxicologic studies show some evidence that coarse particles may elicit pro-inflammatory effects (CD, section 7.4.4), as discussed further in section 3.2. The staff assessment of the physicochemical properties and occurrence of ambient coarse particles (Chapter 2) suggests that both the chemical makeup and

spatial distribution of coarse particles are likely to be more heterogeneous than fine particles. In general, however, urban coarse particles can contain all of the components found in more rural areas, but be contaminated by a number of additional materials, from motor vehicle-related emissions to metals and transition elements associated with industrial operations. Taken together, staff believes that the weight of the dosimetric, limited toxicologic, and atmospheric science evidence lends support to the plausibility of the effects reported in urban epidemiologic studies, as discussed in Chapter 3 (section 3.4), and provides support for retaining standards for thoracic coarse particles so as to continue programs to protect public health from such $PM_{10-2.5}$-related effects.

Staff has considered also the available epidemiologic evidence of associations between thoracic coarse particles, as indexed by $PM_{10-2.5}$, and health endpoints, as well as evidence from PM_{10} studies conducted in areas in which the coarse fraction is dominant. As summarized in Chapter 3 (section 3.4 and Appendix 3-A), several U.S. and Canadian studies now provide evidence of associations between short-term exposure to $PM_{10-2.5}$ and various morbidity endpoints. Three such studies conducted in Toronto (Burnett et al., 1997), Seattle (Sheppard et al.,1999, 2003), and Detroit (Lippmann et al., 2000; Ito, 2003) report statistically significant associations between short-term $PM_{10-2.5}$ exposure and respiratory- and cardiac-related hospital admissions, and a fourth study (Schwartz and Neas, 2000) conducted in six U.S. cities including Boston, St. Louis, Knoxville, Topeka, Portage, and Steubenville reports statistically significant associations with respiratory symptoms in children. The extent to which the results from these studies are robust to the inclusion of co-pollutants varies depending on the various models used and the number of co-pollutants included in the models. Staff observes that the morbidity studies were done in areas in which $PM_{2.5}$, rather than $PM_{10-2.5}$, is the predominant fraction of ambient PM_{10}, such that they are not representative of areas with relatively high levels of thoracic coarse particles.

The CD found that evidence from health studies on associations between short-term exposure to $PM_{10-2.5}$ and mortality was "not as strong" as evidence for associations with $PM_{2.5}$ or PM_{10} but nonetheless was suggestive of associations with mortality (CD, p. 9-32). As described in Section 3.4, associations between $PM_{10-2.5}$ and mortality are similar in magnitude, but less precise, than those for $PM_{2.5}$ or PM_{10}. Statistically significant mortality associations were reported in studies conducted in areas with relatively high $PM_{10-2.5}$ concentrations, including Phoenix (Mar et al., 2000, 2003), Coachella Valley, CA (Ostro et al., 2000, 2003), and Steubenville (as part of the Harvard Six Cities study, Schwartz et al., 1996; Klemm et al., 2003). In areas with lower $PM_{10-2.5}$ concentrations, no statistically significant associations were reported with mortality, though many were positive but not statistically significant.

In addition, some epidemiologic studies that used PM_{10} and were conducted in areas where PM_{10} is typically dominated by coarse fraction particles can provide information relevant to the evaluation of coarse fraction particles. Such studies include findings of associations with

hospitalization for cardiovascular diseases in Tucson, AZ (Schwartz, 1997), hospitalization for COPD in Reno/Sparks, NV (Chen et al., 2000), and medical visits for asthma or respiratory diseases in Anchorage, AK (Gordian et al., 1996; Choudhury et al., 1997). In addition, a number of epidemiologic studies have reported significant associations with mortality, respiratory hospital admissions and respiratory symptoms in the Utah Valley area (e.g., Pope et al., 1989; 1991; 1992). This group of studies provides additional supportive evidence for associations between coarse fraction particles and health effects, particularly morbidity effects, in areas with concentrations generally not meeting the PM_{10} standard levels (all areas except Tucson).

Taken together, staff concludes that the health evidence, including dosimetric, toxicologic and epidemiologic study findings, supports retaining standards to protect against effects associated with short-term exposure to thoracic coarse particles. Staff believes that the substantial uncertainties associated with this limited body of epidemiologic evidence on health effects related to exposure to $PM_{10-2.5}$, however, suggests a high degree of caution in interpreting this evidence, especially at lower levels of ambient particle concentrations as observed in the morbidity studies discussed above.

Beyond this evidence-based evaluation, staff has also considered the extent to which $PM_{10-2.5}$-related health risks estimated to occur at current levels of ambient air quality may be judged to be important from a public health perspective, taking into account key uncertainties associated with the estimated risks. Consistent with the approach used to address this issue for $PM_{2.5}$-related health risks, discussed above in section 5.3.1.2, staff has considered the results of a series of base case analyses that reflect in part the uncertainty associated with the form of the concentration-response functions drawn from the studies used in the assessment, as presented in Chapter 4, section 4.5.[10] Health risks were estimated in these analyses by using the reported linear or log-linear concentration-response functions as well as modified functions that incorporate alternative assumed cutpoints as surrogates for potential population thresholds. Such estimates of risks attributable to short-term exposure to $PM_{10-2.5}$ have been developed for Detroit, Seattle, and St. Louis.[11]

Table 5-2 summarizes the estimated $PM_{10-2.5}$-related annual incidence and incidence rates (in terms of incidence per 100,000 general population) of hospital admissions and respiratory symptoms (cough) in children associated with short-term exposure in these three example urban areas. As an initial matter, staff observes that the range of estimates of cardiac-related hospital admissions in Detroit is generally more than an order of magnitude greater than the range of

[10] Uncertainties related to estimated policy-relevant background levels of $PM_{10-2.5}$ were addressed in a sensitivity analysis, which showed negligible impact on the risk estimates.

[11] This table includes risk estimates for Detroit drawn from Table 4-20 in Chapter 4 and for Seattle and St. Louis drawn from Exhibits E.33 and E.34, respectively, in the Technical Support Document.

Table 5-2 Estimated $PM_{10-2.5}$-related annual incidence of hospital admissions and cough in children associated with short-term exposure with 2003 air quality

	Annual Incidence and 95% CI (events/yr)				Annual Incidence Rate and 95% CI (events/yr/100,000 general population)			
	Cutpoints				Cutpoints			
	Policy-relevant Background*	10 μg/m^3	15 μg/m^3	20 μg/m^3	Policy-relevant Background*	10 μg/m^3	15 μg/m^3	20 μg/m^3
Detroit: hospital admissions for ischemic heart disease	650 170 - 1,100	570 150 - 930	490 130 - 790	430 120 - 680	32 8 - 53	28 7 - 45	24 6 - 39	21 6 - 33
Seattle: hospital admissions for asthma (age <65)	30 0 - 70	10 0 - 20	5 0 - 10	2 0 - 4	2 0 - 4	1 0 - 2	0 0 - 1	0 0 - 0
St. Louis: days of cough in children	27,000 11,000 - 41,000	12,000 4 900 - 18,000	5,800 2,500 - 8,600	2,900 1,300 - 4,000	1,070 440 - 1,600	480 190 - 720	230 100 - 340	120 50 - 160

* Estimated policy-relevant background levels are 4.5 μg/m^3 for eastern urban areas and 3.5 μg/m^3 for western urban areas.

estimated asthma-related admissions in Seattle, which can be attributed in part to differences in baseline risks related to cardiovascular- and respiratory-related health endpoints as well as to differences in $PM_{10-2.5}$ air quality levels in these two areas. To provide some context for considering these risks estimates, staff notes that Detroit and St. Louis did not meet the current 24-hour PM_{10} standard of 150 $\mu g/m^3$ based on 2001 to 2003 air quality data (with 24-hour PM_{10} design values of 191 and 224 $\mu g/m^3$, respectively), whereas Seattle, with much lower daily concentrations (with a 24-hour PM_{10} design value of 73 $\mu g/m^3$), meets the current PM_{10} standards.[12] More specifically, in considering the risk estimates based on the lowest cutpoint considered, the point estimate of annual incidence of $PM_{10-2.5}$-related hospital admissions for ischemic heart disease in Detroit is approximately 650 events per year (roughly 32 events per year per 100,000 general population), and the estimate of days of cough in children is approximately 27,000 days per year (over 1,000 days per year per 100,000 general population, which would be roughly an order of magnitude higher in terms of days per year per 100,000 children). In considering the estimated incidences based on an assumed cutpoint of 10 $\mu g/m^3$, staff observes that these estimates are about 15 percent lower in Detroit and over 50 percent lower in St. Louis, whereas at the highest cutpoint considered, the estimates are about 35 percent lower in Detroit and close to 90 percent lower in St. Louis.

Beyond the specific health endpoints presented in Table 5-2, 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 (across the range of cutpoints considered), as are thousands of additional days in which children are likely to experience other lower respiratory tract symptoms in St. Louis. In considering these limited ranges of estimates, staff concludes that they are indicative of risks that can reasonably be judged to be important from a public health perspective, in contrast to the appreciably lower respiratory morbidity risks estimated in Seattle.

In summary, staff recognizes that the substantial uncertainties associated with the limited available epidemiologic evidence present inherent difficulties in interpreting the evidence for purposes of setting appropriate standards for thoracic coarse particles. Nonetheless, in considering the available evidence, the public health implications of estimated risks associated with current levels of air quality, and the related limitations and uncertainties, staff concludes that this information supports consideration of standards to provide public health protection from morbidity effects and possibly mortality associated with current levels of short-term exposure to thoracic coarse particles in some urban areas. Staff conclusions and recommendations for

[12] See www.epa.gov/airtrends/pdfs/pm10_design_values_2001-2003.pdf for a discussion of how these design values are calculated, noting in particular that concentrations flagged as natural events (e.g, high winds, wildfires, volcanic eruptions) or exceptional events (e.g., construction, prescribed burning) are not included in these calculations and that no regulatory decisions on attainment status have been made at this time based on these data.

indicators, averaging times, and levels and forms of alternative primary standards for thoracic coarse particles are discussed in the following sections.

5.4.2 Indicators

In considering an appropriate indicator for a standard intended to afford protection from health effects associated with exposure to thoracic coarse particles, staff makes the following observations:

- The most obvious choice for a thoracic coarse particle standard is the size-differentiated, mass-based indicator used in the epidemiologic studies that provide the most direct evidence of such health effects, $PM_{10-2.5}$.

- The upper size cut of a $PM_{10-2.5}$ indicator is consistent with the dosimetric evidence discussed above that continues to reinforce the finding from past reviews that an aerodynamic size of 10 μm is a reasonable separation point for particles that penetrate to and potentially deposit in the thoracic regions of the respiratory tract.

- The lower size cut of such an indicator is consistent with the choice of 2.5 μm as a reasonable separation point between fine and coarse fraction particles, based on consideration of evidence from atmospheric sciences; it is also consistent with the recommended continued use of $PM_{2.5}$ as the indicator for fine particles (as discussed above in section 5.3.2), while recognizing that it would exclude the tail of the coarse mode in some locations.

- Further, the limited available information is not sufficient to define an indicator for thoracic coarse particles solely in terms of metrics other than size-differentiated mass, such as specific chemical components.

- The available epidemiologic evidence for effects of $PM_{10-2.5}$ exposure is quite limited and is inherently characterized by large uncertainties, reflective in part of the more heterogeneous nature of the spatial distribution and chemical composition of thoracic coarse particles and the more limited and uncertain measurement methods that have generally been used to characterize their ambient concentrations.

In evaluating relevant information from atmospheric sciences, toxicology, and epidemiology related to thoracic coarse particles, staff notes that there appears to be a clear distinction between the character and nature of exposures and evidence concerning associated health effects of coarse particles as found in urban as compared to those found in nonurban and, more specifically, rural areas. As discussed more fully below, this evidence leads staff to consider a more narrowly defined indicator for thoracic coarse particles that focuses on thoracic coarse particles characteristic of urban areas. This is consistent with CASAC's recommendation

to "qualify the $PM_{10-2.5}$ standard . . . with a focus on urban areas" where thoracic coarse particles are influenced by industrial or traffic-associated sources (Henderson, 2005, p. 8). The following discussion briefly summarizes key observations from the available scientific and technical information that are most relevant in comparing the potential health effects associated with thoracic coarse particles in urban and rural settings.

5.4.2.1 Evidence Related to Urban and Rural Thoracic Coarse Particles

The atmospheric sciences and monitoring information in Chapter 2 indicates not only that exposures to coarse particles tend to be higher in urban areas than in nearby rural locations, but also that urban coarse particles are enriched by a number of contaminants not commonly found in natural crustal materials that are typical of rural coarse particles. The elevation of urban $PM_{10-2.5}$ levels as compared to those at nearby rural sites indicates that sources located within urban areas are generally the cause of elevated urban concentrations; conversely, $PM_{10-2.5}$ concentrations in such urban areas are not largely composed of particles blown in from more distant regions. Important sources of thoracic coarse particles in urban areas include dense traffic that suspends significant quantities of road dust, as well as industrial and combustion sources that contribute to ambient coarse particles both directly and through deposition to soils and roads (Chapter 2, Table 2-2). It follows that thoracic coarse particles in urban areas would differ in composition from those in rural areas, being enriched in components from urban mobile, stationary, and area source emissions.

While detailed composition data are more limited for $PM_{10-2.5}$ than for $PM_{2.5}$, available measurements from some areas as well as studies of road dust components do show a significant influence of urban sources on urban thoracic coarse particle composition and mass. Although crustal elements and natural biological materials represent a significant fraction of thoracic coarse particles in urban areas, both their relative quantity and character may be altered by urban sources. For example, in industrial cities, primary particle emissions from industrial sources and resuspended road dust can increase the relative amount of iron, one of the metals that has been noted as being of some interest in the studies of mechanisms of toxicity for PM, as well as other industrial process-related and potentially toxic materials such as nickel, cadmium, and chromium (CD, p. 9-63). Traffic-related activities can grind and resuspend vegetative materials into forms not as common in more natural areas (Rogge et al., 1993). Studies of urban road dusts find that levels of a variety of components are increased from traffic as well as from other anthropogenic urban sources, including products of incomplete combustion (e.g. polycyclic aromatic hydrocarbons) from motor vehicle emissions and other sources, brake and tire wear, rust, salt and biological materials (CD, p. 3D-3). As discussed in Chapter 2, limited ambient coarse fraction composition data from various comparisons find that metals and sometimes elemental carbon contribute a greater proportion of thoracic coarse particle mass in urban areas than in nearby rural areas. In addition, while large uncertainties exist in emissions inventory data, staff observes that major sources of $PM_{10-2.5}$ emissions in the urban counties in which

epidemiologic studies have been conducted are paved roads and "other" sources (largely construction), and that such areas also have larger contributions from industrial emissions, whereas unpaved roads and agriculture are the main sources of $PM_{10-2.5}$ emissions nationwide.

Toxicologic studies, although quite limited, support the view that sources of coarse particles common in urban areas are of greater concern than uncontaminated materials of geologic origin. As noted above, one major source of urban coarse particles is paved road dust, and the CD discusses results from a recent study in which road tunnel dust particles had greater allergic adjuvant activity than several other particle samples, including residual oil fly ash and diesel exhaust particles, in two animal models of allergy (Steerenberg et al., 2003; CD, p. 7-136-137). This supports evidence available in the previous PM NAAQS review regarding potential effects of road dust particles (EPA, 1996b, p. V-70). In contrast, a number of studies have reported that Mt. St. Helens volcanic ash, an example of natural crustal material of geologic origin, has very little toxicity in animal or *in vitro* toxicologic studies (CD, p. 7-216).

A few toxicologic studies have used ambient thoracic coarse particles from urban/suburban locations ($PM_{10-2.5}$), and the results suggest that effects can be linked with several components of $PM_{10-2.5}$. As described in more detail in sections 5.4.1 and 3.2, these *in vitro* toxicologic studies linked coarse fraction particles with effects including cytotoxicity, oxidant formation, and inflammatory effects. These studies suggest that several components (e.g., metals, endotoxin, other materials) may have roles in various health responses but do not suggest a focus on any individual component.

Although largely focused on undifferentiated PM_{10}, the series of epidemiologic observations and toxicologic experiments related to the Utah Valley suggest that directly emitted (fine and coarse) and resuspended (coarse) urban industrial emissions are of concern. Of particular interest are area studies spanning a 13-month period when a major source of PM_{10} in the area, a steel mill, was not operating. Observational studies found that respiratory hospital admissions for children were lower when the plant was shut down (Pope et al., 1989). More recently, a set of toxicologic and controlled human exposure studies have used particles extracted from filters from ambient PM_{10} monitors from periods when the plant did and did not operate. In both human volunteers and animals, greater lung inflammatory responses were reported with particles collected when the source was operating, as compared to the period when the plant was closed (CD, p. 9-73). In addition, in some studies it was suggested that the metal content of the particles was most closely related to the effects reported (CD, p. 9-74). Staff observes that while peak days in the Utah Valley occur in conditions that enhance fine particle concentrations, over the long run, over half of the PM_{10} was in the coarse fraction. The aggregation of particles collected on the filters during the study period reflect this long-term composition. At a minimum, the filter-derived particles represent the kinds of industrial components that would be incorporated in road dusts in the area.

Taken together, the epidemiologic studies that examine exposures to thoracic coarse particles generally found in urban environments and to natural crustal materials support the view that urban thoracic coarse particles are of concern to public health, in contrast to uncontaminated natural crustal dusts. With respect to the urban results, several recent studies have shown associations between $PM_{10-2.5}$ and health outcomes in a number of sites across the U.S. Associations have been consistently reported with morbidity in urban areas, some of which had relatively low $PM_{10-2.5}$ concentrations. For mortality, statistically significant associations have been reported only for urban areas that have notably higher ambient $PM_{10-2.5}$ concentrations. These associations are with short-term exposures to aggregated $PM_{10-2.5}$ mass, and no epidemiologic evidence is available on associations with different components or sources of $PM_{10-2.5}$. However, staff observes that the studies have all been conducted in urban areas of the U.S., and thus reflect effects of thoracic coarse particles generally present in urban environments from urban sources.

By contrast, recent evidence from epidemiologic studies has suggested that mortality and possibly other health effects are not associated with thoracic coarse particles from dust storms or other such wind-related events that result in suspension of natural crustal materials of geologic origin. The clearest example is provided by a study in Spokane, WA, which specifically assessed whether mortality was increased on dust-storm days using case-control analysis methods. The average PM_{10} level was more than 200 $\mu g/m^3$ higher on dust storm days than on control days, and the authors report no evidence of increased mortality on these specific days (Schwartz et al., 1999). One caveat of note is the possibility that people may reduce their exposure to ambient particles on the most dusty days (e.g., Gordian et al., 1996; Ostro et al., 2000). Nevertheless, the Spokane study provides no suggestion of significant health effects from uncontaminated natural crustal materials that would typically form a major fraction of coarse particles in non-urban or rural areas.

Beyond the urban and rural distinctions discussed above, staff has also considered the extent to which there is evidence of effects with exposure to ambient thoracic coarse particles in communities predominantly influenced by agricultural or mining sources. For example, in the last review, staff considered health evidence related to long-term silica exposures from mining activities, but found that there was a lack of evidence that such emissions contribute to effects linked with ambient PM exposures (EPA, 1996b, p. V-28). Similarly in this review, there is an absence of evidence related to such community exposures. While dust generated from agricultural activity can include biological material such as fungal or bacterial material, and some occupational studies discussed in the CD report effects at occupational exposure levels (Table7B-3, p. 7B-11), such studies do not provide relevant evidence for much lower levels of community exposures. Further, it is unlikely that such sources contribute to the effects that have been observed in the recent urban epidemiologic studies.

The CD concludes its integrated assessment of the effects of natural crustal materials as follows:

> Certain classes of ambient particles appear to be distinctly less toxic than others and are unlikely to exert human health effects at typical ambient exposure concentrations (or perhaps only under special circumstances). For example, particles of crustal origin, which are predominately in the coarse fraction, are relatively non-toxic under most circumstances, compared to combustion-related particles (such as from coal and oil combustion, wood burning, etc.) However, under some conditions, crustal particles may become sufficiently toxic to cause human health effects. For example, resuspended crustal particles may be contaminated with toxic trace elements and other components from previously deposited fine PM, e.g., metals from smelters (Phoenix) or steel mills (Steubenville, Utah Valley), PAHs from automobile exhaust, or pesticides from agricultural lands. (CD, p. 8-344)

This is consistent with CASAC's conclusion that the available evidence from health studies suggests that the focus of an indicator for thoracic coarse particles should be on such particles found in urban, not rural environments (Henderson, 2005).

The staff assessment of the available evidence relevant to the appropriate scope of an indicator for coarse particles can be summarized as follows. Thoracic coarse particle concentrations generally reflect contributions from local sources, and the limited information available from speciation of thoracic coarse particles and emissions inventory data indicate that the sources of urban thoracic coarse particles generally differ from those found in nonurban areas. As a result, the kinds of thoracic coarse particles people are exposed to in urban areas can be expected to differ significantly from the kinds found in non-urban or rural areas. Ambient $PM_{10-2.5}$ exposure is associated with health effects in studies conducted in urban areas, and the limited available health evidence more strongly implicates coarse particles from industrial and traffic-related sources than from uncontaminated soil or geologic sources. The limited evidence does not support either the existence or the lack of causative associations for community exposures to agricultural or mining industries. Given the apparent differences in composition and in the epidemiologic evidence, it is not appropriate to conclude that evidence of associations with health effects related to urban coarse particles would also apply to nonurban or rural coarse particles.

Collectively, the evidence suggests that a more narrowly defined indicator for thoracic coarse particles should be considered that would protect public health against effects linked with thoracic coarse particles present in urban areas. Such an indicator would be principally based on particle size, but also reflect a focus on those thoracic coarse particles that are generally present in urban environments. Staff recommends consideration of thoracic coarse urban particulate matter ($UPM_{10-2.5}$) as an indicator for a thoracic coarse particle standard, referring to airborne particles between 2.5 and 10 μm in diameter that are generally present in urban environments,

which, as discussed above, are principally comprised of resuspended road dust typical of high traffic-density areas and emissions from industrial sources. Staff considers that $UPM_{10-2.5}$ would more likely be an effective indicator for standards to protect against effects of thoracic coarse particles than a more broadly focused $PM_{10-2.5}$ indicator. Staff notes that this indicator would also be consistent with an appropriately cautious interpretation of the epidemiologic evidence that does not potentially over-generalize the results of the limited available studies.

5.4.2.2 Related Requirements for $PM_{10-2.5}$ Monitors and Monitoring Network Design

Along with staff's recommendations on the definition for an indicator for urban thoracic coarse particles, it is important to recognize that requirements for federal reference and equivalent methods and monitoring network design are essential components in fully defining and applying a PM indicator. While these efforts are not described in detail in this Staff Paper, the discussions below highlight key components of these activities for an urban thoracic coarse particle standard indicator.[13]

First, in conjunction with considering $UPM_{10-2.5}$ as an indicator for standards to address thoracic coarse particles, EPA is evaluating various ambient monitoring methods, including continuous methods. This evaluation is being performed through field studies of commercially ready and prototype methods to characterize the measurement of $PM_{10-2.5}$ in urban areas.[14] This $PM_{10-2.5}$ methods evaluation has resulted in characterizing the performance of multiple $PM_{10-2.5}$ measurement technologies under a variety of aerosol and meteorological conditions typical of urban areas in regions across the country. 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.

EPA has also begun the process of examining data quality objectives for a potential 24-hour $UPM_{10-2.5}$ standard. 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 in urban environments. 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 $UPM_{10-2.5}$ standard. In addition to providing high temporal resolution to

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[13] EPA plans to issue proposed and final revisions to related requirements for federal reference and equivalent methods and monitoring network design concurrently with proposed and final decisions regarding revisions to the PM NAAQS.

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

$PM_{10-2.5}$ data, continuous monitors would also support public reporting of $UPM_{10-2.5}$ episodes and inclusion of $UPM_{10-2.5}$ in an air quality forecasting program.

In addition, EPA has also commenced the examination of monitoring network design issues for a possible $UPM_{10-2.5}$ standard. One key focus in monitoring network design is consistency with information from the health studies and with staff conclusions regarding the appropriateness of an indicator based on $PM_{10-2.5}$ mass generally present in urban environments and reflecting local urban sources. The available epidemiologic studies have reported associations with $PM_{10-2.5}$ mass that has been measured at sites that are located in or near heavily populated areas, but not in close proximity to industrial or other specific sources, to reflect community exposure levels. In the context of applying such a standard, staff has examined various measures for designing an appropriate monitoring network for an urban $PM_{10-2.5}$ standard, including indicators of traffic density and locations of industrial sources, as well as total population and population density. Staff observes that these traffic- and population-related measures are very highly correlated with one another. On an initial basis, a focus on CBSA/CSAs with populations of at least 100,000 would result in a base monitoring network that would include approximately 350 areas. Further refinements in network size in terms of the number of monitors to be placed within these areas can be accomplished through additional criteria such as a graduated scale that increases monitoring requirements for more populous CBSA/CSAs, as well as a hybrid strategy that combines population/traffic-related measures with comparisons to historical PM_{10} or estimated $PM_{10-2.5}$ design values to focus additional monitors in areas with elevated concentrations associated with traffic-related and industrial sources, and potentially reduce monitoring in CBSA/CSAs with lower concentrations levels of thoracic coarse particles.

As noted elsewhere in this document, $PM_{10-2.5}$ is more highly variable in the atmosphere than $PM_{2.5}$, such that the specific criteria regarding monitor placement will be a particularly important consideration in the implementation of a $PM_{10-2.5}$ monitoring network. Staff has examined various options for targeting monitors within larger CBSA/CSAs to focus measurements in locations of expected high concentrations of urban coarse thoracic particles that also represent the population-oriented objective of monitors underlying key epidemiologic studies. One useful metric for guiding monitor placement appears to be population density, for example, the placement of monitors in U.S. Census Block Groups characterized by population densities greater than 500 people per square mile. This type of criterion effectively guides monitors toward the urban/center city and suburban location settings that likely represent areas of high population exposure to elevated concentrations, as compared to the rural parts of urbanized CBSA/CSAs. Staff notes, in addition, that the population density metric based on Census Block Group has the potential to identify and de-emphasize monitoring in "population holes" within the urbanized area, such as areas with only industrial development. For PM_{10}, some monitors have historically been located in such heavily industrialized areas away from

significant population exposure, including some existing source-oriented monitors placed on facility fence-lines to investigate specific emission-related complaints. Staff believes that it would be inconsistent with the basis of a $UPM_{10-2.5}$ indicator and, thus, inappropriate to include any such monitors as part of a $UPM_{10-2.5}$ monitoring network.

Consideration of specific measurement scale guidance for placement of $PM_{10-2.5}$ monitors is influenced by the rapid fallout of coarse fraction particles with increasing distance from sources. Analysis of existing $PM_{10-2.5}$ concentrations in cities such as Birmingham, Cleveland, and Las Vegas has revealed large differences in three-year mean, 98th percentile design values, as well as poor daily correlations, between monitors located within several kilometers of each other. In many cases, these large differences can be explained by the immediate proximity of the higher reading monitor to areas of industrial and high traffic activity, compared with lower reading monitors that may be farther from coarse-particle generating or resuspending activities. As a result, staff believes that to be consistent with the population-oriented objective of the monitors underlying key epidemiologic studies, population-oriented monitors that represent middle scale (i.e., 100 meters to 0.5 km) or neighborhood scale (i.e., 0.5 to 4.0 km) sized areas of relatively uniform land use would be most appropriate for a $UPM_{10-2.5}$ network, recognizing that a significant spatial gradient in coarse thoracic particle concentrations may still exist across middle scale-sized areas.[15] Such preferred locations for $PM_{10-2.5}$ monitors in a $UPM_{10-2.5}$ network would include densely populated communities located several hundred meters from significant sources, such as industrial sources or heavily traveled roadways.[16]

Finally, with regard to elevations in thoracic coarse particle levels that may occur in urban areas as a result of dust storms or other such events, staff observes that EPA has historically used implementation policies to address such issues in the implementation of PM standards. Examples of such policies include the "natural events" policy for implementation of the PM_{10} standards. This policy includes guidance regarding exclusion of PM_{10} measurements from natural events in making determinations on attainment or nonattainment of the standards. The natural events discussed in this policy include volcanic and seismic activity, wildland fires and high wind events. EPA is now in the process of revising policies for natural events and exceptional events to address issues related to the $PM_{2.5}$ NAAQS, as well as potential new $UPM_{10-2.5}$ standards.[17]

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[15] Staff notes that these discussions of measurement scale for monitors are consistent with definitions in 40 CFR Part 58, Appendix D, http://www.access.gpo.gov/nara/cfr/waisidx_04/40cfr58_04.html.

[16] Staff observes that $PM_{10-2.5}$ monitors may also be sited to provide data for other purposes such as research or trends assessment that would be outside the scope of a monitoring network for a $UPM_{10-2.5}$ standard.

[17] Current policy available at http://www.epa.gov/ttn/oarpg/t1/memoranda/nepol.pdf.). EPA plans to issue proposed and final revisions to the natural events policy concurrently with any proposed and final decisions regarding revisions to the PM NAAQS.

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.

New information available in this review on thoracic coarse particles, as discussed above in section 5.4.1, includes several epidemiologic studies that report statistically significant associations between short-term (24-hour) exposure to $PM_{10-2.5}$ and various morbidity effects and mortality. 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 $PM_{2.5}$, other such studies reported no associations. The CD concludes that the evidence does not suggest an association with long-term exposure to $PM_{10-2.5}$ (CD, p. 9-79). Staff also 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 continues to support a 24-hour averaging time for a standard intended to control thoracic coarse particles, based primarily on evidence suggestive of associations between short-term (24-hour) exposure and morbidity effects and, to a lessor degree, mortality. Noting the absence of evidence judged to be even suggestive of an association with long-term exposures, staff concludes that there is no evidence that directly supports an annual standard, while recognizing that it could 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 associated with long-term exposures.

5.4.4 Alternative Standards to Address Health Effects Related to Short-term Exposure

As noted earlier, 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) for protection against effects of exposure to coarse fraction particles was based on two community studies of exposure to fugitive dust that showed health effects in areas experiencing large exceedances of that standard (Gordian et al., 1996; Hefflin et al., 1994), 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 in section 5.3.6. In making this change, EPA selected a 99th percentile form,[18] 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 thoracic coarse particles has become available that reports associations between short-term $PM_{10-2.5}$ concentrations in some urban areas and various morbidity effects and, to a lesser degree, mortality. In considering alternative standards that would provide protection against such health effects, as discussed below, staff has taken into account evidence-based considerations and has examined the extent to which risk-based considerations should also be taken into account.

5.4.4.1 Evidence-based Considerations

In considering the available evidence on associations between short-term $PM_{10-2.5}$ concentrations and morbidity and mortality effects as a basis for setting a 24-hour standard for thoracic coarse particles, staff has focused on relevant U.S. and Canadian studies (Appendix 3-A). As discussed above in section 5.4.1 and in Chapter 3, 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 extent to which the reported associations are generally robust to co-pollutant confounding and alternative modeling approaches; and the extent to which the studies used relatively reliable air quality data.

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 population 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

[18] As noted above, the court vacated the 1997 24-hour PM_{10} standard that had been revised to incorporate a 99th percentile form.

threshold within the range of air quality observed in the studies,[19] for some health endpoints 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 the range of effects that have been associated with short-term exposures.

In considering the evidence, staff is mindful of these uncertainties as well as the limited nature of the available evidence. In examining the available evidence to identify a basis for a range of standard levels that would be appropriate for consideration, staff has focused on the upper end of the distributions of daily $PM_{10-2.5}$ concentrations in the relevant studies, particularly in terms of the 98th and 99th percentile values, consistent with the forms considered in section 5.3.6 above for a short-term $PM_{2.5}$ standard. Staff's examination of the evidence is discussed below, based on air quality information and analyses presented in Ross and Langstaff (2005) and Ross (2005).

In looking first at the morbidity studies identified in section 5.4.1 that report statistically significant associations with respiratory- and cardiac-related hospital admissions in Toronto (Burnett et al., 1997), Seattle (Sheppard et al.,1999, 2003), and Detroit (Lippmann et al., 2000; Ito, 2003), the reported 98th percentile values in the three areas range from approximately 30 to 36 $\mu g/m^3$, and the 99th percentile values range from 36 to 40 $\mu g/m^3$. To provide some perspective on these $PM_{10-2.5}$ levels, staff notes that the level of the 24-hour PM_{10} standard was exceeded only on a few occasions during the time periods of the studies in Detroit and Seattle.[20]

Staff has also looked at the studies identified in section 5.4.1 that report statistically significant and generally robust associations with mortality and short-term exposures to $PM_{10-2.5}$. Studies conducted in Phoenix (Mar et al., 2000, 2003) and Coachella Valley, CA (Ostro et al., 2000, 2003) report 98th percentile $PM_{10-2.5}$ values of approximately 70 and 107 $\mu g/m^3$, and 99th percentile values of 75 and 134 $\mu g/m^3$, respectively. These studies were conducted in areas with air quality levels that did not meet the current PM_{10} standards. In addition, a statistically significant association was reported between $PM_{10-2.5}$ and mortality in Steubenville as part of the Harvard Six Cities analysis (Schwartz et al., 1996; Klemm et al., 2003). $PM_{10-2.5}$ concentrations were fairly high in this eastern city, with reported 98th and 99th percentile $PM_{10-2.5}$ values of 53 and 61 $\mu g/m^3$, respectively. Staff notes that, in contrast with most areas, $PM_{10-2.5}$ and $PM_{2.5}$ concentrations were highly correlated (r=0.69) in Steubenville during the study period (Schwartz

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[19] Staff notes that the distributions of daily $PM_{10-2.5}$ concentrations in these studies often extend down to or below typical 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 are 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).

[20] As shown in air quality data trends reports: for Seattle, *1997 Air Quality Annual Report for Washington State*, p. 17, at http://www.ecy.wa.gov/pubs/97208.pdf; for Detroit, *Michigan's 2003 Annual Air Quality Report*, p. 46, at http://www.deq.state.mi.us/documents/deq-aqd-air-reports-03AQReport.pdf.

et al., 1996; Klemm et al., 2003). In contrast to the statistically significant mortality associations with $PM_{10-2.5}$ reported in these studies, staff notes that no such associations were reported in a number of other studies, including in the five other cities that were part of the Harvard Six Cities study (Boston, St. Louis, Knoxville, Topeka, and Portage), San Jose, Detroit, Philadelphia, and Pittsburgh. With the exception of Pittsburgh, these cities had much lower 98th percentile $PM_{10-2.5}$ values, ranging from 18 to 49 $\mu g/m^3$.

Based on the air quality information reported in these studies, staff makes the following observations:

- In the morbidity studies that reported statistically significant associations with respiratory or cardiovascular hospitalization in Detroit and Seattle, and with respiratory symptoms in six U.S. cities, the reported 98th percentile $PM_{10-2.5}$ values ranged from approximately 30 up to 40 $\mu g/m^3$.

- In the mortality studies that reported statistically significant associations in Steubenville, Coachella Valley, and Phoenix, the reported 98th percentile $PM_{10-2.5}$ values were all above 50 $\mu g/m^3$, ranging from 53 $\mu g/m^3$ up to 107 $\mu g/m^3$ in Coachella Valley.

- In the mortality studies that reported no statistically significant associations in five of the cities in the Harvard Six Cities study (Boston, St. Louis, Knoxville, Topeka, and Portage), and in San Jose, Detroit, Philadelphia, and Pittsburgh, the reported 98th percentile $PM_{10-2.5}$ values were below 50 $\mu g/m^3$ for all cities except Pittsburgh, ranging from 18 to 49 $\mu g/m^3$.

In looking more closely at air quality data used in the morbidity and mortality studies discussed above, however, staff recognizes that the uncertainty related to exposure measurement error can be potentially quite large. 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 $\mu g/m^3$ 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. These observations lead staff to conclude that the statistically significant, generally robust association with hospital admissions in Detroit likely reflects population exposures that may be appreciably higher in the central city area, but not necessarily across the broader study area, than would be estimated using data from the Windsor monitors.

Staff also looked more specifically at the Coachella Valley mortality study (Ostro et al., 2000; 2003), in which data were used from a single monitoring site in one city, Indio, within the study area where daily measurements were available. The mean $PM_{10-2.5}$ concentration during the study period (1989-1998) was 30.5 $\mu g/m^3$ at this monitoring site. Consistently lower concentrations were measured at another city in the Coachella Valley area, Palm Springs; during the study period the mean value at this site was 17 $\mu g/m^3$. The authors report that the data for the two sites were correlated, with correlation coefficients of about 0.6 for each of the three PM indicators. Using 2001-2003 data, mean $PM_{10-2.5}$ levels were reported to be 44 and 15 $\mu g/m^3$ at the Indio and Palm Springs sites, respectively. Thus, in Coachella Valley, mortality was significantly associated with $PM_{10-2.5}$ measurements made at the Indio site, but a portion of the study population would have been expected to experience appreciably lower ambient exposure levels. In contrast to the Detroit study, air quality data used in the mortality study conducted in Coachella Valley appear to represent concentrations on the high end of $PM_{10-2.5}$ levels for Coachella Valley communities.

A closer examination of the air quality data used in the other studies discussed above generally shows less disparity between air quality levels at the monitoring sites used in the studies and the broader pattern of air quality levels across the study areas than that described above in the Detroit and Coachella Valley studies. More specifically, less variation across monitoring sites was seen in air quality data in both Seattle and St. Louis. In Steubenville, the $PM_{10-2.5}$ concentrations measured at the centrally located monitor used in the study are somewhat higher than those reported at other area monitors. In Phoenix, data from a larger network of PM_{10} monitors across the area shows a large gradient in concentrations across the urban area, with lower concentrations measured at sites on the north side to appreciably higher concentrations on the south side; data from the one centrally located monitoring site used in the study appears to fall in the mid-range of concentrations along the north-south gradient.

This closer examination of air quality information generally reinforces the view that exposure measurement error is potentially quite large in these $PM_{10-2.5}$ studies. As a consequence, the air quality levels reported in these studies, as measured by ambient concentrations at monitoring sites within the study areas, are not necessarily good surrogates for population exposures that are likely associated with the observed effects in the study areas or that would likely be associated in other urban areas across the country. The Detroit example suggests that population exposures were probably appreciably underestimated in the Detroit

morbidity study, such that the observed effects are likely associated with higher $PM_{10-2.5}$ levels than reported. In contrast, the Coachella Valley mortality study provides an example in which population levels were probably appreciably overestimated, such that the observed effects may well be associated with lower $PM_{10-2.5}$ levels than reported. At relatively low levels of air quality, population exposures implied by these studies as being associated with the observed effects likely become more uncertain, suggesting an even higher degree of caution in interpreting the group of morbidity studies as a basis for identifying a standard level that would protect against the observed effects.

Taking into account this closer examination of the studies, staff concludes that this evidence suggests consideration of a standard for urban thoracic coarse particles at a $PM_{10-2.5}$ level at least down to 50 $\mu g/m^3$, in conjunction with a 98^{th} percentile form (which would be roughly equivalent to a level of 60 $\mu g/m^3$ in conjunction with a 99^{th} percentile form). While lower levels may be considered to provide a margin of safety against morbidity effects that may possibly occur at such lower levels, staff believes that consideration of a standard below these levels may not be warranted based on this evidence. Staff's view takes into account the conclusion that this evidence is particularly uncertain as to population exposures, especially from the morbidity studies reporting effects at relatively low concentrations, as well as the general lack of evidence of associations from the group of mortality studies with reported concentrations below these levels. A standard set at or somewhat above these levels could be expected to provide protection against the potential mortality effects observed in studies that reported ambient concentrations above this level, as well as morbidity effects that may occur above this level.

An even more cautious or restrained approach to interpreting the limited body of $PM_{10-2.5}$ epidemiologic evidence would be to judge that the uncertainties in this whole group of studies as to population exposures that are associated with the observed effects are too large to use the reported air quality levels directly as a basis for setting a specific standard level. Staff notes that such a judgment would not be inconsistent with the conclusion reached above in section 5.4.1 that these studies, together with other dosimetric and toxicologic evidence, provide support for retaining standards for thoracic coarse particles at some level to protect against the morbidity and mortality effects observed in the studies, regardless of whether an associated population exposure level can be clearly discerned from the studies.

Considering this more cautious interpretation, staff believes that it would be reasonable to interpret the available epidemiologic evidence more generally, by considering whether it provides a sufficient basis for a standard that would afford protection generally "equivalent" to that afforded by the current PM_{10} standards. Considering the available evidence in this way leads to the following observations:

- The statistically significant mortality associations with short-term exposure to $PM_{10-2.5}$ reported in the Phoenix and Coachella Valley studies were observed in areas that did not meet the current PM_{10} standards.

- The statistically significant morbidity associations with short-term exposure to $PM_{10-2.5}$ reported in the Detroit and Seattle studies were observed in areas that exceeded the level of the current 24-hour PM_{10} standard only on few occasions during the time periods of the studies.

- All but one of the statistically significant morbidity and mortality associations with short-term exposure to PM_{10} reported in areas dominated by coarse fraction particles (including Reno/Sparks, NV, Tucson, AZ, Anchorage, AK, and the Utah Valley area, as discussed above in section 5.4.1) were observed in areas that did not meet the current PM_{10} standards.

Based on these considerations, staff finds little basis for concluding that the degree of protection afforded by the current PM_{10} standards is greater than warranted, since potential mortality effects have been associated with air quality levels not allowed by the current standards, but have not been associated with air quality levels that would generally meet the current standards. Further, staff finds little basis for concluding that a greater degree of protection is warranted in light of the very high degree of uncertainty in the relevant population exposures implied by the morbidity studies. Staff judges, therefore, that it is reasonable to interpret the available evidence as supporting consideration of a short-term standard for urban thoracic coarse particles set so as to provide generally "equivalent" protection to that afforded by the current PM_{10} standards, recognizing of course that no one $PM_{10-2.5}$ level will be strictly equivalent to a specific PM_{10} level in all areas. Such a standard would likely provide protection against morbidity effects especially in urban areas where, unlike the study areas, PM_{10} is generally dominated by coarse-fraction rather than fine-fraction particles. Such a standard would also likely provide protection against the more serious, but more uncertain, $PM_{10-2.5}$-related mortality effects generally observed at somewhat higher air quality levels.

To identify a range of levels for consideration for a short-term standard for urban thoracic coarse particles set so as to afford generally "equivalent" protection as the current PM_{10} standards, staff has analyzed available data on $PM_{10-2.5}$ and PM_{10} 24-hour average concentrations from monitors that would be included in the monitoring network design provisions discussed in section 5.4.2.2.[21] Based on a regression analysis of the 205 monitoring sites so identified (Schmidt et al., 2005), staff finds that a $UPM_{10-2.5}$ level of approximately 60 $\mu g/m^3$ in terms of a

[21] Monitors included in this analysis are those in CBSAs with at least 100,000 population and in census block groups with a population density of at least 500, and that also had 3 years of complete data in each quarter for both PM_{10} and $PM_{10-2.5}$.

98th percentile form (or approximately 70 μg/m^3 in terms of a 99th percentile form) would be roughly equivalent on average across the U.S. to a PM_{10} level of 150 μg/m^3 in terms of a one-expected-exceedance form.[22] While noting appreciable variability in the estimated point of equivalence across individual sites, these levels of approximate average equivalence are quite consistent across each of the five regions in which all of the areas that do not meet the current PM_{10} standards are located (including the southern California, southwest, northwest, upper mid-west, and southeast regions). Notably different average equivalence levels were observed in the other two regions, i.e., approximately 40 μg/m^3 in the northeast and over 70 μg/m^3 in the industrial mid-west (in terms of 98th percentile forms).

Another approach to identifying a $UPM_{10-2.5}$ standard that is generally "equivalent" to the current PM_{10} standards is to compare the number of areas, and the population in those areas, that would likely not meet a specific $UPM_{10-2.5}$ standard, set at a given level and form, with the same measures in areas that do not meet the current PM_{10} standards. Such an analysis, based on 2001 to 2003 data from monitors that would be included in the monitoring network design provisions discussed in section 5.4.2.2 provides some rough indication of the breadth of protection potentially afforded by alternative standards. The results of this analysis (Appendix 5B, Tables 5B-2(a), (b) and (c), for the 98th and 99th percentile forms of a 24-hour $UPM_{10-2.5}$ standard and the current PM_{10} standards, respectively) indicate that a $UPM_{10-2.5}$ standard of about 70 or 65 μg/m^3, 98th percentile form, (or approximately 85 or 80 μg/m^3, 99th percentile form) would impact approximately the same number of counties or number of people, respectively, as would the current PM_{10} standards.[23]

Based on these alternative analyses of generally "equivalent" $UPM_{10-2.5}$ standards, staff concludes that it is reasonable to consider a 24-hour $UPM_{10-2.5}$ standard in the range of approximately 60 to 70 μg/m^3 with a 98th percentile form (approximately 70 to 85 μg/m^3 with a 99th percentile form). Considering standards within these ranges, somewhat above the levels identified above based on an examination of the air quality concentrations in the relevant epidemiologic studies (i.e., 50 μg/m^3, 98th percentile form, or 60 μg/m^3, 99th percentile form), would reflect an even higher degree of caution in interpreting the epidemiologic evidence. Consideration of a generally "equivalent" $UPM_{10-2.5}$ standard would reflect a judgment that while the epidemiologic evidence supports establishing a short-term standard for urban thoracic coarse

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[22] Across the U.S., the 95% confidence intervals around these point estimates are approximately $\pm$ 3 μg/m^3, while region-specific intervals are approximately $\pm$ 10 μg/m^3 in the five regions in which all of the areas that do not meet the current PM_{10} standards are located.

[23] As shown in Table 5B-2(c), staff notes that there are 585 counties with PM_{10} monitoring sites used in determining compliance with the PM_{10} standards, whereas only 309 of those counties have monitor sites that would be included in the monitoring network design provisions discussed in section 5.4.2.2. Of these 309 counties, 259 have PM_{10} and $PM_{10-2.5}$ air quality data that meet the data completeness criteria defined for this analysis, which are somewhat less restrictive that the criteria that were applied in the regression analysis described above.

particles at such a generally "equivalent" level, the evidence concerning air quality levels of thoracic coarse particles in the studies is not strong enough to provide a basis for revising the level of protection generally afforded by the current PM_{10} standards.

5.4.4.2 Risk-based Considerations

Beyond looking directly at the relevant epidemiologic evidence and related air quality information, 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 concentration-response 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.

5.4.4.3 Summary

In considering the relevant dosimetric, toxicologic, and epidemiologic evidence, associated air quality information, and related limitations and uncertainties, staff concludes that there is support for considering a 24-hour $UPM_{10-2.5}$ standard to replace the current PM_{10} standards to provide protection against health effects associated with short-term exposures to thoracic coarse particles that are generally present in urban environments. In looking at the evidence of associations between short-term exposure to $PM_{10-2.5}$ and morbidity and mortality,

both on its own and in the context of considering a standard set so as to be generally "equivalent" to the current PM_{10} standards, staff concludes that it is appropriate to consider a 24-hour standard in the range of 50 to 70 $\mu g/m^3$, with a 98^{th} percentile form, or in the range of 60 to 85 $\mu g/m^3$, with a 99^{th} percentile form. A standard set within either of these ranges could be expected to provide protection against the morbidity effects of $PM_{10-2.5}$, especially those likely to occur in areas in which PM_{10} is dominated by thoracic coarse particles, as well as to protect against the potential mortality effects of $PM_{10-2.5}$.

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 very limited information available 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 placing relatively more weight on these uncertainties would focus consideration of standard levels toward the upper end of the ranges identified above, whereas a more precautionary approach would focus consideration on the lower end of these ranges.

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, in the Administrator's judgment, 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 urban thoracic coarse particles that does not generally include fine particles. Any such revised standards should be based primarily 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, together with consideration of evidence from studies using PM_{10} in locations where $PM_{10-2.5}$ is the dominant fraction.

(2) The indicator for a thoracic coarse particle standard should be $UPM_{10-2.5}$, which is 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 recommended indicator is primarily based on particle size, but with a more narrow focus on those thoracic coarse particles that are generally present in urban environments. The available evidence from studies on atmospheric chemistry and sources and health effects of thoracic coarse particles indicates that a $UPM_{10-2.5}$ indicator is more likely an effective indicator for standards to protect against health effects of thoracic coarse particles than a more broadly-focused $PM_{10-2.5}$ indicator.

(b) In support of this recommendation, work should continue on the development of an FRM for a $UPM_{10-2.5}$ indicator based on the ongoing field program to evaluate various types of $PM_{10-2.5}$ 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 $UPM_{10-2.5}$ standard to protect against health effects associated with short-term exposure periods, with consideration given to the use of either a 98^{th} or 99^{th} percentile form. There is little basis for also retaining an annual averaging time for protection against such health effects.

(4) Consideration should be given to setting a 24-hour $UPM_{10-2.5}$ standard with a level in the range of approximately 50 to 70 $\mu g/m^3$, 98^{th} percentile form, or approximately 60 to 85 $\mu g/m^3$, 99^{th} percentile form. Staff believes that a more precautionary approach would focus consideration on the lower end of these ranges, while consideration of a standard set toward the upper end of these ranges would place relatively more weight on the uncertainties inherent in the very limited epidemiologic evidence. Consideration of $UPM_{10-2.5}$ standards within the ranges recommended above, and design considerations for an associated $UPM_{10-2.5}$ monitoring network, should take into account the especially large variability seen in currently available information on ambient concentrations and composition of $PM_{10-2.5}$ in urban areas.

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 epidemiologic 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 research is needed on such intra-city variability as well as on inter-city variability and on temporal (e.g., seasonal) variability. 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 in non-urban areas) 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, epidemiologic, and toxicologic 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 epidemiologic studies were discussed at length in the previous review, and it appeared at the time that the epidemiologic 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 epidemiologic 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. It draws upon the most relevant information contained in the CD, as well as 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). In addition, 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) have been reviewed.

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

[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.

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 impairment is manifested in two principal ways: as local visibility impairment (e.g., localized plumes or "brown clouds") and as regional haze. In some cases, local-scale visibility degradation is considered to be "reasonably attributable" to a single source or small group of sources. Such impairment may take the form of a localized plume, a band or layer of discoloration appearing well above the terrain that results from complex local meteorological conditions. Localized plumes are composed of smoke, dust, or colored gas that obscures 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. Historically, sources of visible plumes were thought to be relatively minor contributors to visibility impairment in Class I areas (i.e., 156 national parks, wilderness areas, and international parks identified for visibility protection in section 162(a) of the Act). However, 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 as being "reasonably attributable" to a particular source.[2]

In other cases, localized visibility impairment is manifested as an urban haze, sometimes referred to as a "brown cloud." This type of impairment is predominantly caused by emissions from multiple sources in the urban area and is not typically attributable to a single nearby source or to long-range transport from more distant sources. Brown cloud conditions have been well-documented in a number of western cities.

The second type of impairment, regional haze, generally 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 are functions 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, in some cases, be significant contributors to regional-scale impairment in Class I and other rural areas.

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 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

[2] Two of the most notable cases leading to emission 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.

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, p. 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 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, now provides 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 the East and West are much more similar in urban areas than they are 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 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,

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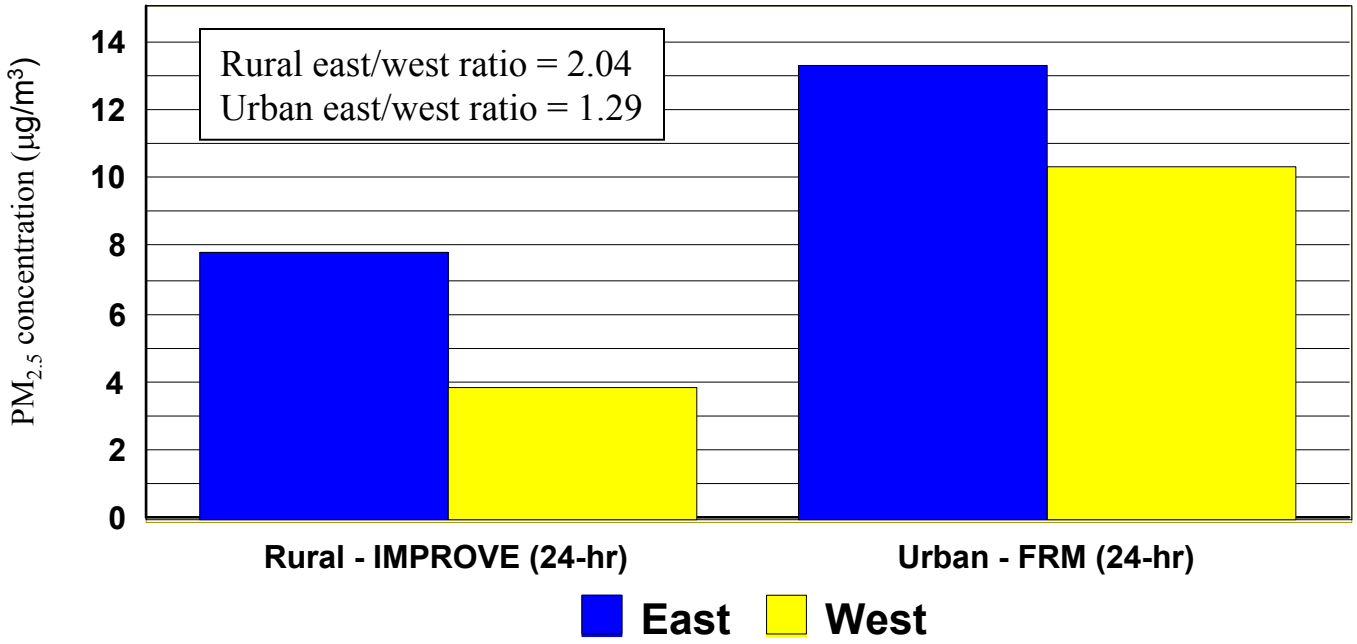
[3] A preliminary analysis of the archived data for 63 cities across the U.S. was presented in the first draft Staff Paper (August 2003), but further analysis has not been conducted. While the preliminary analysis demonstrated relatively well-characterized correlations between predicted $PM_{2.5}$ concentrations (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)

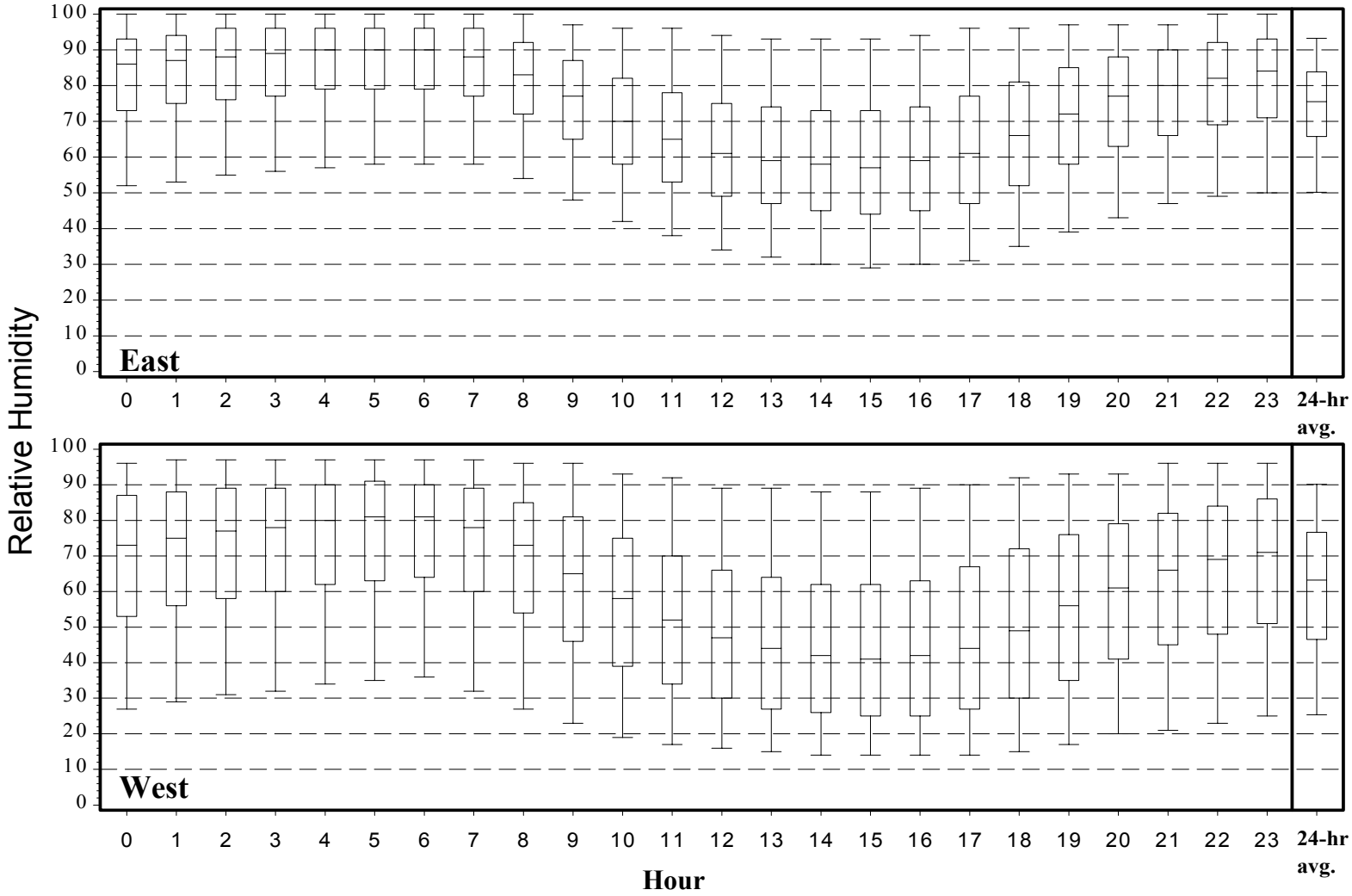


Figure 6-2. Distribution of hourly and 24-hour average relative humidity at eastern and western U.S. National Weather Service Sites, 2003. Box depicts interquartile range and median; whiskers depict 5th and 95th percentiles.

Source: Schmidt et al. (2005)

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). Of the two sets of data, 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 *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)*

[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.

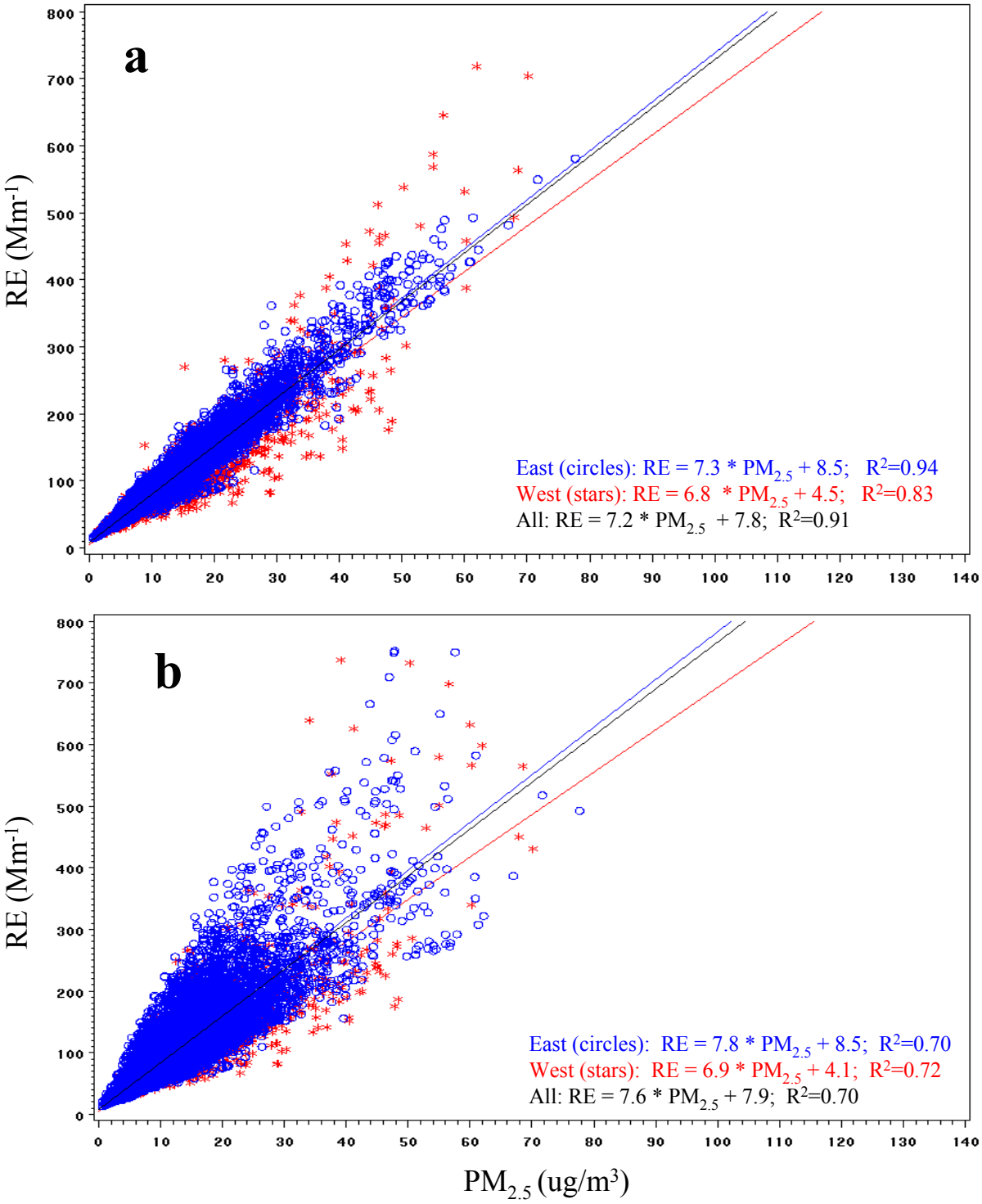


Figure 6-3. Relationship between reconstructed light extinction (RE) and 24-hour average $PM_{2.5}$, 2003. RE in top panel (a) computed with 10-year average *f(RH)*; RE in bottom panel (b) computed using actual *f(RH)*.

Source: Schmidt et al. (2005)

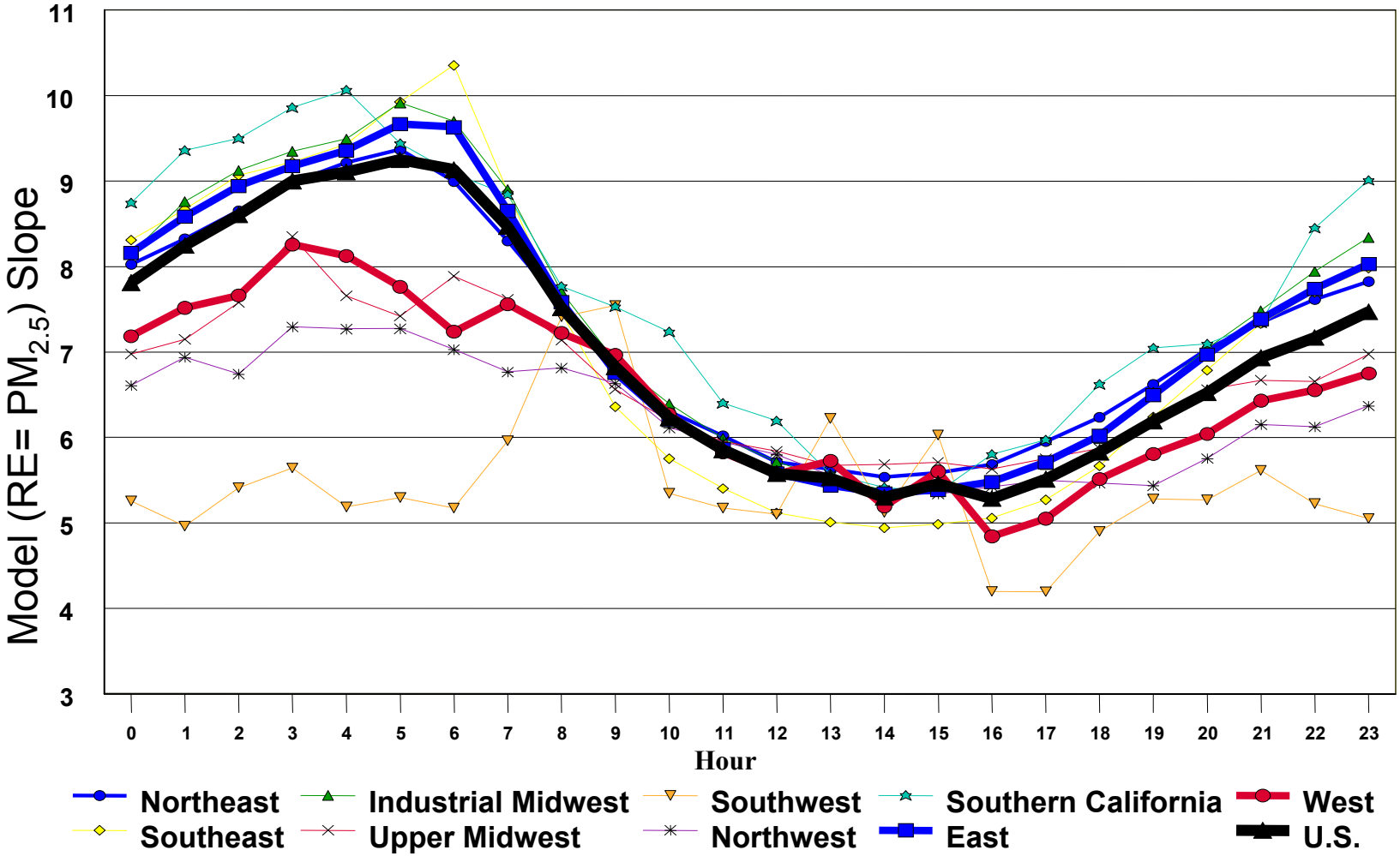


Figure 6-4. Model slope for relationship between reconstructed light extinction (RE) and hourly $PM_{2.5}$ (increase in RE due to incremental increase in $PM_{2.5}$), 2003. RE computed using 10-year average *f(RH)*.

Source: Schmidt et al. (2005)

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 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 Dennis, 1997; 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 (Chestnut and Dennis, 1997). These economic benefits are often described by economists

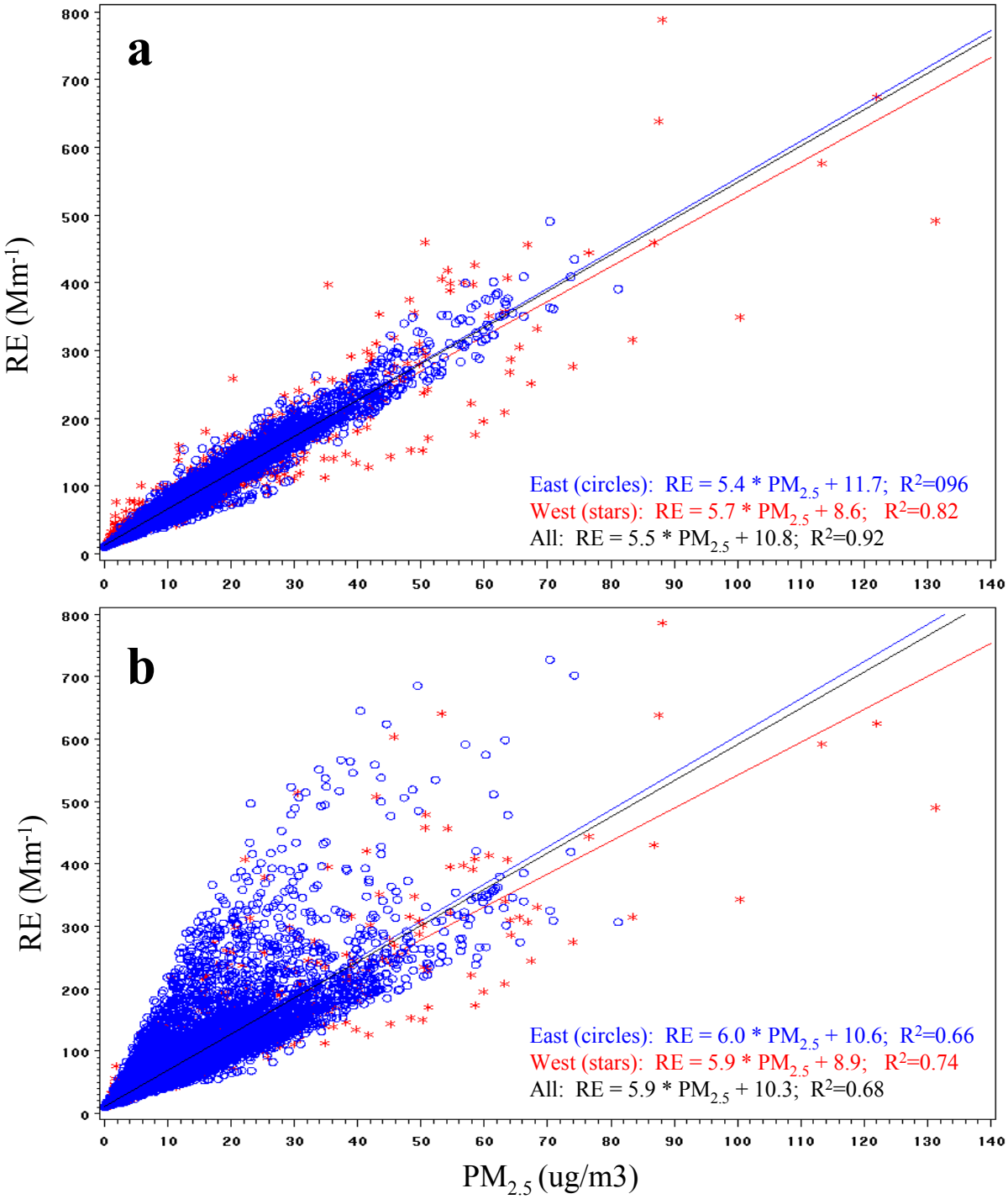


Figure 6-5. Relationship between reconstructed light extinction (RE) and 12 p.m.– 4 p.m. average $PM_{2.5}$, 2003. RE in top panel (a) computed with 10-year average *f(RH)*; RE in bottom panel (b) computed using actual *f(RH)*.

Source: Schmidt et al. (2005)

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. Use 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 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 local, State, and international efforts to protect visual air quality.

6.2.5.1 Regional Protection

Due to differences in visibility impairment levels (as a result of 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

A number of programs, goals, standards, and planning efforts 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, for the purpose of public perception surveys, 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 the 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 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 Appendix 6A: Images of Visual Air Quality in Selected Urban Areas in the U.S.). Included as part of Appendix 6A, 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

[5] A small pilot study for Washington, D.C. was conducted by EPA and was briefly discussed in the preliminary draft staff paper (2001).

three factors: 1) the standard was for an urban area, not a pristine national park area where the standards might be more strict; 2) standard violations should be at visual air quality levels considered to be unreasonable, objectionable, and unacceptable visually; and 3) judgments of standards violations should be based on visual air quality only, not on health effects.

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

The Denver visibility standard setting process produced the following findings:

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

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

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

The CDPHE researchers sorted the ratings for each slide by increasing order of light extinction and calculated the percent of participants that judged each slide to violate the standard. The Denver visibility standard was then established based on a 50% acceptability criterion. Under this approach, the standard was identified as the light extinction level that divides the slides into two groups: those found to be acceptable and those found to be unacceptable by a majority of study participants. The CDPHE researchers found this level to be reasonable because, for the slides at this level and above, a majority of the study participants judged the light extinction levels to be unacceptable. In fact, when researchers evaluated all citizen judgments made on all slides at this level and above as a single group, more than 85% of the participants found visibility impairment at and above the level of the selected standard to be unacceptable.

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 10^{th}, 20^{th}, 30^{th}, 40^{th}, 50^{th}, 60^{th} 80^{th}, and 90^{th} 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.

Phoenix, Arizona: Visibility Index

In 2002, the Arizona Department of Environmental Quality formed the Visibility Index Oversight Committee. The Committee's goal was to coordinate the involvement of Phoenix-area residents in the development of a visibility index. The Phoenix committee patterned its survey process after the process used by Denver to develop its visibility standard.

The survey included 385 participants in 27 separate sessions. Participants were carefully recruited to form a sample group that was demographically representative of the larger Phoenix population. Three sessions were held in Spanish.

Participants were shown a series of 25 images of the same vista of downtown Phoenix, with South Mountain in the background at a distance of about 40 km. Photographic slides of the images were developed using the WinHaze program. The visibility impairment levels ranged from 15 to 35 deciviews (87 to 12 km visual ranges). Participants first rated the randomly-shown slides on a scale of 1 (unacceptable) to 7 (excellent). Next, the participants rated slides, again shown in random order, as acceptable or unacceptable. This phase of the survey produced the following findings:

- At least 90 percent of all participants found visible air quality acceptable between 15 deciviews (87 km visual range) and 20 deciviews (53 km);

- At 24 deciviews (36 km), nearly half of all participants thought the visible air quality was unacceptable; and

- By 26 deciviews (29 km), almost three-quarters of participants said it was unacceptable, with nearly all participants considering levels of 31 deciviews (18 km) and higher to be unacceptable.

The information developed in this survey informed the development of recommendations by the Visibility Index Oversight Committee for a visibility index for the Phoenix Metropolitan Area (Arizona Department of Environmental Quality, 2003). A final report of the survey methods and results is available (BBC Research & Consulting, 2002). The Phoenix survey demonstrates that the rating methodology developed for gathering citizen input for establishing the Denver visibility standard can be reliably transferred to another city while relying on updated imaging technology to simulate a range of visibility impairment levels.

Images used in this study were generated using WinHaze. Similar images, also generated by WinHaze, which illustrate visual air quality in Phoenix under a range of visibility conditions, are available in Appendix 6A at http://www.epa.gov/ttn/naaqs/standards/pm/s_pm_cr_sp.html.

British Columbia, Canada: Public Perception Survey

In 1993, the REVEAL (Regional Visibility Experimental Assessment in the Lower Fraser Valley) field study was undertaken to characterize summertime visibility and ambient aerosol loadings in southwestern British Columbia. In 1994, researchers at the University of British

Columbia conducted a pilot study on the perception of acceptable visibility conditions in the area, using photographs and optical measurements taken during the summer of 1993 (Pryor, 1996). The study was based on the methodology used in setting the Denver visibility standard (Ely et al., 1991).

Participants in the study were shown slides of two suburban locations in British Columbia: Chilliwack and Abbotsford. After using the same general protocol, Pryor found that responses from this pilot study would indicate a standard in terms of visual range of approximately 40 km for Chilliwack and 60 km for Abbotsford. Pryor (1996) discusses some possible reasons for the variation in standard visibility judgments between the two locations. Factors discussed include the relative complexity of the scenes, different levels of development at each location, potential local source influence on site-specific nephelometer data, and potential bias of the sample population since only students participated. The author expressed the view that the pilot study reinforced the conclusion that the methodology originally developed for the Denver standard-setting process is a sound and effective one for obtaining public participation in a standard-setting process, and that it could be adapted for such use in another geographic location with only minor modifications (Pryor, 1996).

6.2.7 Summary and Conclusions

The CD and other reports referenced in section 6.2 provide a significant body of information documenting the effects of PM and its components on atmospheric visibility. Data on visibility conditions indicate that urban areas generally have higher loadings of $PM_{2.5}$ and, thus, higher visibility impairment than monitored Class I areas.

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 effects 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 doing so, 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. Problem formulation includes the integration of the available information on ecosystem stressors (which can include physical, chemical, and/or biological stressors), their sources, and the effects associated with exposure of sensitive ecosystem components to each stressor.

During analysis, data are evaluated to determine how exposure to stressors is likely to occur (exposure profile) and how stressor levels and ecological effects (stressor-response profile) are related. These products provide the basis for risk characterization.

During risk characterization, the exposure and stressor-response profiles are integrated through risk estimation. 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, the information is organized into the following seven subsections: major ecosystem stressors in PM (6.3.1); direct vegetation effects of particulate nitrate and sulfate deposition (6.3.2); ecosystem effects associated with chronic inputs of reactive nitrogen and acidifying compounds from PM and other sources (6.3.3); characteristics and location of nutrient and acid 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), as 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. The 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 should be based on one or more chemical species found in ambient PM. At this time it remains to be determined 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 of greatest and most widespread environmental significance (CD, p. 9-114). Other components of PM, such as dust, trace metals, and organics, which can at high levels affect plants and other organisms, were also discussed. However, some of these compounds, such as organics and some metals, are regulated under separate statutory authorities, e.g., section 112 of the Clean Air Act. Further, because the high levels necessary to cause injury occur only near a few limited point sources and/or on a very local scale, protection against these effects alone may not provide sufficient basis for recommending a separate secondary NAAQS. Therefore, the remainder of this section will focus on the effects of particulate nitrates and sulfates, either individually, in combination, and/or as contributors to total reactive nitrogen deposition and total deposition of acidifying compounds, on sensitive ecosystem components and essential ecological attributes, which in turn, affect overall ecosystem structure and function.

At the outset, it must be recognized that of particulate nitrogen and sulfur as ecosystem stressors with the recognition that nitrogen and sulfur in varying amounts are necessary and

beneficial nutrients for most organisms that make up ecosystems. Optimal amounts of these nutrients varies among organisms, populations, communities and ecosystems and across seasons and time scales. An individual of a given species found in different ecosystems might have different optimum nutrient requirements, and different species within a given ecosystem may be stimulated, inhibited or unaffected by a given amount of inputs of these nutrients. Therefore, it is impossible to generalize to all species in all circumstances as to the amount at which inputs of these nutrients or acidifying compounds become stressors. On the other hand, while some species are adapted to benefit from additions of nutrients in the short run, they may not continue to benefit into the future (or may not benefit at the same rate), due to supplies of other nutrients becoming short and limiting further growth. Further, species that benefit from nutrient additions often do so to the detriment of their competitors, shifting the delicate balance that has evolved under more nutrient-limited conditions.

The staff recognizes that the public welfare has benefitted from the use of nitrogen and sulfur nutrients in fertilizers in managed agricultural and commercial forest settings. The focus of this review is on identifying risks to sensitive species and ecosystems where unintentional additions of these atmospherically derived nutrient and acidifying compounds may be forcing unintended change on the nation's ecosystems and resulting in adverse impacts on essential ecological attributes including, species shifts, loss of species richness and diversity, impacts on threatened and endangered species, and alteration of native fire cycles. In these cases, deposited particulate nitrate and sulfate are appropriately termed ecosystem "stressors".

6.3.2 Direct Vegetation Effects of Particulate Nitrate and Sulfate 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).

Sulfur, similar to nitrogen, is an essential plant nutrient that can be deposited 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 concludes 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, to determine possible 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 acidifying deposition, such particles do have the potential to cause direct foliar injury. This is especially true when the acidifying deposition 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 acid 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 acid 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 concludes that the risks of foliar injury occurring from acid precipitation is high. The contribution of particulate sulfates and nitrates to the total acidity found in the acid precipitation impacting eastern vegetation is not clear.

6.3.3 Ecosystem Effects of Chronic Inputs of Reactive Nitrogen and Acidifying Compounds From PM Deposition and Other Sources

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 and acidifying 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). Though not currently regulated under the NAAQS program, reduced forms of Nr from food production are estimated to be approximately 2 - 4 times larger than emissions of oxidized forms of nitrogen produced during combustion of fossil fuels. Specifically, Galloway and Cowling (2002) estimate that per capita Nr creation for the world in the mid-1990s was 20 kg/N/person/year for food production and only 3.9 kg/N/person/year for energy production (see Table 3).

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. However, 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. These uncertainties limit our ability to determine the temporal and spatial distribution of environmental effects for a given input of Nr. These uncertainties are of particular 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).

Some of the more significant detrimental effects resulting from chronic increased inputs of atmospheric Nr (e.g., ammonium and nitrate compounds) 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

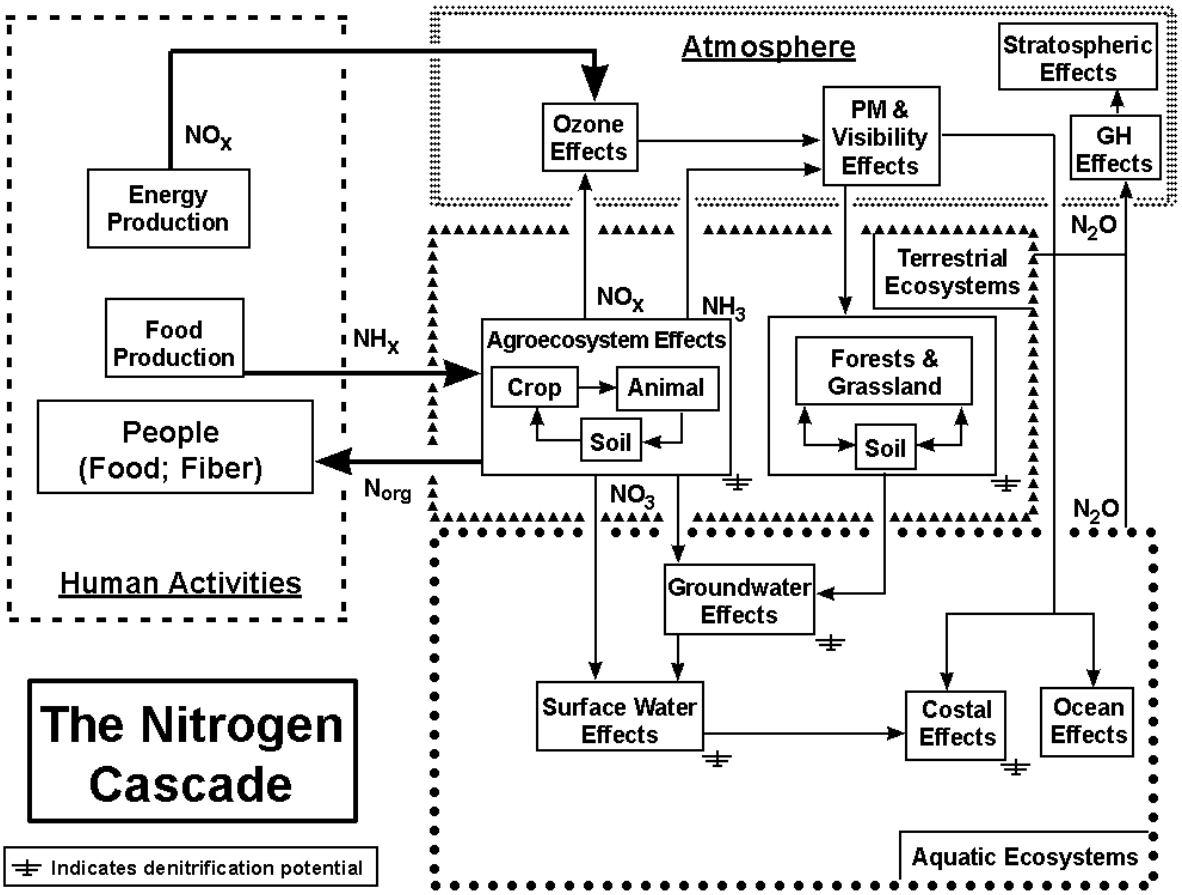


Figure 6-6 Illustration of the nitrogen cascade showing the movement of human-produced reactive nitrogen (Nr) as it cycles through the various environmental reservoirs in the atmosphere and in terrestrial and aquatic ecosystems (Galloway et al., 2003; Figure 4-15, CD p. 4-97).

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 ecosystem 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 ammonium and 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 Nr availability over time (Aber, 1992). It occurs when Nr 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 Nr 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 Nr 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 determine 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 ecosystems. 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, 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 species that depend on these plants are adversely affected. Included among these 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 Nr 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. 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 Nr 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 amounts of total atmospheric deposition of sulfur and nitrogen. A major portion of the atmospheric loading is from dry and occult 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 Nr 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 Nr (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 and Acidifying Deposition

Acid 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). Acid 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 (Driscoll et al., 2001).

Terrestrial Effects

Acid 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%, acid 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, 1990). 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).

Acid 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 acid 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 and acidifying 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 deposition altering 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 glauca*), 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.

Acid 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 acid 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 model, 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 acid 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 acid 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 decreases 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 decreases 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 acid 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 acid 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 Radiation and Climate Condition Effects from Atmospheric PM

In addition to the direct and indirect effects of deposited PM, ambient atmospheric PM can affect 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 (see section 6.5 below) potentially alter the overall competitive 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 amounts 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 stream water 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 acid deposition, acid 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 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, where wet or occult deposition may predominate, deposition amounts may greatly exceed PM amounts 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 should be established that measures the response of key sensitive ecological indicators over time to changes in atmospheric deposition of PM stressors.

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 amounts 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 in amounts known to be sufficient to affect sensitive terrestrial and aquatic ecosystems over time. 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 are only a few sites where long-term monitoring of sensitive indicators of ecosystem response to acidic and acidifying deposition is taking place within the U.S.. Two examples 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 acid 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 acid 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 pollutants in question 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. 4-125). The biological indicator is the organism used to indicate the status of the receptor ecosystem; the chemical criterion is the parameter that results in harm to the biological indicator; and the critical value is the value of the chemical criterion below which no significant harmful response occurs to the biological indicator (Lokke et al., 1996).

A number of different types of indicators and chemical criteria for monitoring ecosystem status have been proposed. Some examples for evaluating ecosystem nitrogen status include: foliar nitrogen content, 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 either the critical or the 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 a given ecosystem. 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 CL measures, they have also defined a corresponding "deposition index" for each CL value. The deposition index is the difference between the CL value for a given site and the current deposition rates at that site. Positive values of the index at a particular forested location reflect the capacity of that forest ecosystem to tolerate additional acid 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 applicable 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 CL approach in environmental assessments and in informing management decisions, widespread use of CLs in the U.S. is not yet possible. The CL approach is very data-intensive, 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 applicable 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 deposition 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 acid 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 have profound impacts on 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 should be based on one or multiple chemical stressors found in ambient PM (e.g. N or S containing compounds).

- PM, as a contributor to the chronic annual loads of total Nr and/or acidifying compounds entering sensitive ecosystems, has been associated with numerous effects on those ecosystems and their associated essential ecological attributes. There is no bright line or threshold for these effects, but rather a "syndrome" of complex changes over time. As levels of inputs of these pollutants are reduced, ecosystem recovery can occur but may take decades, and may require controls beyond those already established.

- Excess Nr or acidifying 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 acidifying compounds need to be considered and managed in harmony.

- Existing ambient air and deposition monitoring networks are not generally sufficient to characterize the associated ecosystem response. Additional, long-term, targeted ecosystem monitoring is needed (e.g., downwind of large urban areas in the West).

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 the amount of PM deposited to sensitive receptors from measured concentrations of PM in the ambient air. This makes it difficult to relate a given ambient air concentration to a receptor response, an important factor in being able to set a national ambient air quality standard. A multitude of factors (e.g., 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) exert varying degrees of influence on the deposition velocities for different PM components at any given point in time. Therefore, modeled deposition velocities, 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 may co-occur only at that location (e.g., soil organisms, plants), that make it unique from all others. Because of this variety, and lack of sufficient baseline data on each of these features for most ecosystems, it is currently not possible to extrapolate with confidence any effect from one ecosystem to another, or to predict appropriate "critical loads" 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 productive 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 impaired 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 (hydrated calcium sulfate) 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 tending to offset the "greenhouse effect" 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 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. A further 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 less 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. 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. This existing suite of secondary PM standards is intended 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, in the judgment of the Administrator, are requisite to protect public welfare[1] 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 Act does not require that secondary standards be set to eliminate all risk of adverse welfare effects, but rather at a level requisite to protect public welfare from those effects that are judged by the Administrator 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:

[1] 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."

- 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 effects, 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 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 those effects. 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 secondary $PM_{2.5}$ standard defined in terms of that effect. 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 38679-83). In reaching this decision, EPA first concluded that PM, especially fine particles, impairs 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, multi-state 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 20 km and 27 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 31 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 they have been 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 (Appendix 6A, available on EPA's website at http://www.epa.gov/ttn/naaqs/standards/pm/s_pm_cr_sp.html). 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 $\mu g/m^3$, which equates to 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 10:00 am 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 $PM_{2.5}$ Federal Reference Method (FRM) monitoring network provides 24-hour average concentrations, and, in some cases, on a third- or sixth-day sample schedule, 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 federal equivalent methods (FEMs) that can provide data in at least hourly intervals. Development of the criteria for approval of FEMs to support a visibility standard would be based upon a data quality objective process that considers uncertainties associated with the measurement system and the level and form 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 (Appendix 6A, available on EPA's website at http://www.epa.gov/ttn/naaqs/standards/pm/s_pm_cr_sp.html). 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 $PM_{2.5}$ concentration 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 $PM_{2.5}$

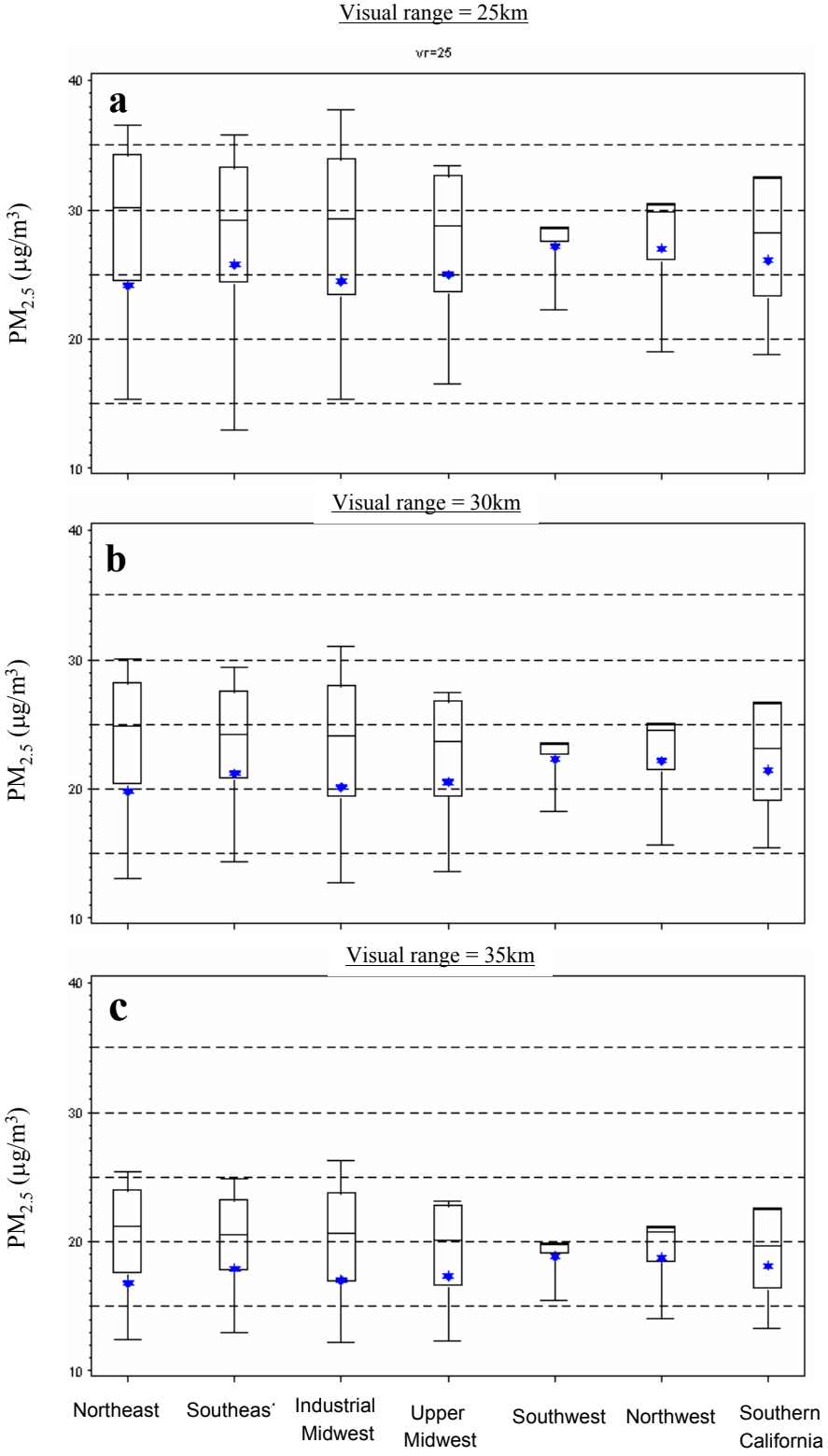

Figure 7-1. Distributions of $PM_{2.5}$ concentrations for 12 p.m. – 4 p.m. corresponding to visual ranges of 25 km (panel a), 30 km (panel b), and 35 km (panel c) – by region. Box depicts interquartile range and median; whiskers depict 5th and 95th percentiles; star denotes mean.

Source: Schmidt et al. (2005)

concentrations from 30 to 20 $\mu g/m^3$, suggested by staff's analysis and observations of photographic representations, is generally consistent with a national target visual range 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 $\mu g/m^3$, 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 a standard intended to provide protection from man-made pollution should be set 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 $\mu g/m^3$ 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 range of percentile forms extending below the 98th percentile would be appropriate to consider for a visibility standard, and thus staff has also looked to lower percentiles 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 $\mu g/m^3$ across the U.S.), the estimated 90th percentile values in distributions of daily background levels, for example, are appreciably higher, but generally well below 15 $\mu g/m^3$, with levels below 10 $\mu g/m^3$ 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 $\mu g/m^3$ is an appropriate lower end to the range of short-term $PM_{2.5}$ standards for visibility protection for consideration in this review if a percentile form ranging down to the 90th percentile were to be considered. Alternatively, if a percentile form at about the 98th percentile were considered, then, consistent with conclusions for the primary standard, staff believes that 25 $\mu g/m^3$ is an appropriate lower end to the range.

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 $\mu g/m^3$, depending in part on the form of the standard, as discussed below, 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[2]. 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 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.

To identify a range of concentration percentiles that would be appropriate for consideration, staff first has taken into account similar considerations as were discussed in Chapter 5, section 5.3.6.2, for the primary $PM_{2.5}$ standard as a basis for identifying an appropriate upper end to a range of percentile forms. More specifically, staff believes that the upper end of the range of consideration should be the 98^{th} to 99^{th} percentiles, consistent with the forms being considered for the 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 80^{th} 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%

[2] 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.

most impaired days suggests to staff that the 92nd percentile, which represents the mean of the distribution of the 20% worst days (Schmidt et al., 2005), would be an appropriate lower end of the range of forms for consideration.

Based on the factors discussed above, staff concludes that a percentile-based form should be considered, based on a percentile within a range of the 92nd to about the 98th 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.

To assist in understanding the implications of alternative percentile forms, in conjunction with alternative levels of a 4-hour secondary $PM_{2.5}$ standard, staff assessed the percentage of days estimated to exceed various $PM_{2.5}$ concentrations in counties across the U.S., as shown in Appendix 7A, Figure 7A-1. This analysis is based on 2001 to 2003 air quality data from continuous monitors, using the 4-hour average concentration from 12:00 to 4:00 pm at the maximum monitor in each county. Staff also assessed (based on the same air quality database) the percentage of counties, and the population in those counties, that would not likely attain various $PM_{2.5}$ secondary standards (Appendix 7A, Table 7A-1). These assessments are intended to provide some rough indication of the breadth of coverage potentially afforded by various combinations of alternative standards.

7.3.6 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, in the judgment of the Administrator, are requisite to protect public welfare from those known or anticipated effects that are judged by the Administrator to be adverse, such that the standards are neither more nor less stringent than necessary. The Act does not require that secondary standards be set to eliminate all risk of adverse 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. 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.

(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) Recommendations on ranges of alternative levels and forms for alternative $PM_{2.5}$ visibility standards include:

(a) Staff recommends consideration of a 4- to 8-hour $PM_{2.5}$ standard within the range of 30 to 20 $\mu g/m^3$, depending in part on the form of the standard.

(b) Staff also recommends consideration of a percentile-based form for such a standard, focusing on a range from the 92^{nd} up to the 98^{th} percentile of the annual distribution of daily short-term $PM_{2.5}$ concentrations, averaged over 3 years.

Staff judges that a standard within these ranges 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.

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 38683). 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 for PM based on any of these effects alone. These considerations lead staff to address in the following sections whether the reductions in PM likely to result from the current secondary PM standards, or from the range of recommended revisions to the primary PM standards and from the recommended 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). Currently, however, fundamental areas of uncertainty preclude establishing predictable relationships between ambient concentrations of particulate nitrogen and sulfur compounds and associated ecosystem effects. One source of uncertainty hampering the characterization of such relationships is the extreme complexity and variability that exist in estimating particle deposition rates. These rates are affected by numerous factors, including particle size and composition, associated atmospheric conditions, and the properties of the surfaces being impacted. A related source of uncertainty is establishing what portion of the total nitrogen and sulfur deposition occurring at a given site is attributable to ambient PM. 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. Further, it is not clear how well data from monitoring sites

may apply to non-monitored sites with different surface cover, meteorology, or other deposition-related factors.

In addition, ecosystems have different sensitivities and capacities to buffer or assimilate pollutants. Many of the documented ecosystem-level effects only became evident after long-term, chronic exposures to total annual loads of Nr or acidifying compounds that eventually exceeded the natural buffering or assimilative capacity of the system. In most cases, PM deposition is not the only contributor to the total load of Nr or acidifying compounds entering the affected system. Since it is difficult to predict the rate of PM deposition, and thus, the PM contribution to total deposition at a given site, it is difficult to predict the ambient concentration of PM that would likely lead to the observed adverse effects within any particular ecosystem. Equally difficult is the prediction of recovery rates for areas already affected, if PM deposition rates of various chemical species were to be reduced.

Despite these uncertainties, a number of significant 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 Nr 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 to address these effects. 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 of Nr and acidifying compounds that is likely attributable to ambient PM, national ambient air quality standards alone may not be an appropriate approach to protect against the adverse impacts of total Nr and acidifying compounds, partially contributed by ambient PM, on ecosystems and vegetation in all parts of the country. Nonetheless, staff believes that additional reductions in fine particles and related precursor emissions likely to result from the current suite of secondary PM standards, or the range of recommended revisions to the primary PM standards and to the secondary $PM_{2.5}$ standard to address visibility impairment, 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 believes that reductions in fine and coarse particles likely to result from the current suite of secondary PM standards, or the range of recommended revisions to the primary PM standards and to the secondary $PM_{2.5}$ standard to address visibility impairment, would contribute to protection against PM-related soiling and materials damage. Staff recommends that the potential for such protection be taken into account in considering whether to revise the current secondary PM 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 setting secondary PM standards that at a minimum retain the level of protection afforded by the current PM standards, so as to continue control of ambient fine and coarse-fraction particles, especially long-term deposition of particles such as particulate nitrates and sulfates, that contribute to adverse impacts on vegetation and ecosystems and/or to materials damage and soiling. Any such standards should be considered in conjunction with the 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.

(2) While staff recognizes that PM-related impacts on vegetation and ecosystems and PM-related soiling and materials damage are associated with chemical components in both fine and coarse-fraction PM, staff also recognizes that sufficient information is not available at this time to recommend consideration of either an ecologically based indicator or an indicator based distinctly on soiling and materials damage, in terms of specific chemical components of PM. Thus, for consistency with the primary standards, staff recommends that consideration be given to basing secondary standards on the same indicators that are used as the basis for the suite of primary PM standards.

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 SETTING SECONDARY 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 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, especially with respect to nitrogen and

sulfur deposition. Reduction in these uncertainties, particularly in key acid- and Nr-sensitive areas, will be important in developing the capability of quantitatively linking ambient PM concentrations with environmental exposures. In order to better understand the contribution of PM to cumulative long-term environmental impacts, more research needs to be conducted on the percentage of total Nr and acidifying deposition contributed by PM and where necessary, better monitoring methods and network designs should be developed.

(3) Atmospheric sources of Nr and acidifying compounds, including ambient PM, are clearly contributing to the total load of these pollutants entering U.S. ecosystems annually. However, the immense variability in ecosystem response to total Nr and acidifying deposition across the U.S., and the factors controlling ecosystem sensitivity and recovery, have not been adequately characterized. Data should be collected on a greater variety of ecosystems over longer time scales to determine how ecosystems respond to different loading rates over time. Such research, in conjunction with the development of improved predictive models, could help in future consideration within the U.S. of the "critical loads" concept, and in determining how much of any given critical load is contributed by different sources of pollutants.[3]

[3] 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 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/m^3 PM_{10}	% increase (95% CI) per 25 µg/m^3 $PM_{2.5}$	% increase (95% CI) per 25 µg/m^3 $PM_{10-2.5}$	PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ Mean (Range) Levels Reported**
Schwartz et al., 1996	*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/m^3 PM_{10}	% increase (95% CI) per 25 μg/m^3 $PM_{2.5}$	% increase (95% CI) per 25 μg/m^3 $PM_{10-2.5}$	PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ Mean (Range) Levels Reported**
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/m^3 PM_{10}	% increase (95% CI) per 25 µg/m^3 $PM_{2.5}$	% increase (95% CI) per 25 µg/m^3 $PM_{10-2.5}$	PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ Mean (Range) Levels Reported**
Klemm et al., 2000 Six City reanalysis - 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/m^3 PM_{10}	% increase (95% CI) per 25 μg/m^3 $PM_{2.5}$	% increase (95% CI) per 25 μg/m^3 $PM_{10-2.5}$	PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ Mean (Range) Levels Reported**
Schwartz, 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/m^3 PM_{10}	% increase (95% CI) per 25 µg/m^3 $PM_{2.5}$	% increase (95% CI) per 25 µg/m^3 $PM_{10-2.5}$	PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ Mean (Range) Levels Reported**
Schwartz, 2000 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)

Original study* Study Location Reanalysis study	Analysis Comments	% increase (95% CI) per 50 µg/m^3 PM_{10}	% increase (95% CI) per 25 µg/m^3 $PM_{2.5}$	% increase (95% CI) per 25 µg/m^3 $PM_{10-2.5}$	PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ Mean (Range) Levels Reported**
Total Cardiovascular Mortality					
Ito and Thurston, 1996 *Chicago, IL*	*GAM not used*	*1.49 (-0.72, 3.74)*	---	---	*PM_{10} 38 (max 128)*
Pope et al., 1992 *Utah Valley, UT*	*GAM not used*	*9.36 (1.91, 17.36)*	---	---	*PM_{10} 47 (11, 297)*
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)

Original study* Study Location Reanalysis study	Analysis Comments	% increase (95% CI) per 50 µg/m^3 PM_{10}	% increase (95% CI) per 25 µg/m^3 $PM_{2.5}$	% increase (95% CI) per 25 µg/m^3 $PM_{10-2.5}$	PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ Mean (Range) Levels Reported**
Ostro et al., 2000 Coachella Valley, CA Ostro et al., 2003	GAM Strict GLM NS	5.5 (1.6, 9.5) 5.1 (1.2, 9.1)	9.8 (-5.7, 27.9) 10.2 (-5.3, 28.3)	2.9 (0.7, 5.2) 2.7 (0.4, 5.1)	PM_{10} 47.4 (3, 417) $PM_{2.5}$ 16.8 (5, 48) $PM_{10-2.5}$ 17.9 (0, 149)
Ostro, 1995 San Bernadino and Riverside Counties, CA	GAM not used	---	0.69 (-0.34, 1.74)	---	$PM_{2.5}$ 32.5 (9.3, 190.1) (estimated from visibility)
Total Respiratory Mortality:					
Ito and Thurston, 1996 *Chicago, IL*	*GAM not used*	*6.77 (1.97, 11.79)*	---	---	*PM_{10} 38 (max 128)*
Pope et al., 1992 *Utah Valley, UT*	*GAM not used*	*19.78 (3.51, 38.61)*	---	---	*PM_{10} 47 (11, 297)*
Fairley, 1999 Santa Clara County, CA Fairley, 2003	GAM Strict GLM NS	10.7 (-3.7, 27.2) 10.8 (-3.4, 27.1)	11.7 (-9.8, 38.3) 13.5 (-3.6, 33.7)	32.1 (-9.1, 92.2)	PM_{10} 34 (6, 165) $PM_{2.5}$ 13 (2, 105) $PM_{10-2.5}$ 11 (0, 45)
Lippmann et al., 2000 Detroit, MI Ito, 2003	GAM Strict GLM NS	7.5 (-10.5, 29.2) 7.9 (-10.2, 29.7)	2.3 (-10.4, 16.7) 3.1 (-9.7, 17.7)	7.0 (-9.5, 26.5) 6.4 (-10.0, 25.7)	PM_{10} 31 (12, 105) $PM_{2.5}$ 18 (6, 86) $PM_{10-2.5}$ 13 (4, 50) mean (10%, 90%)
Ostro, 1995 San Bernadino and Riverside Counties, CA	GAM not used	---	2.08 (-0.35, 4.51)	---	$PM_{2.5}$ 32.5 (9.3, 190.1) (estimated from visibility)
COPD Mortality:					
Moolgavkar, 2000a Cook Co., IL Moolgavkar, 2003	GAM Strict GLM NS	5.5 (0.2, 11.0) 4.5 (-1.6, 11.0)	---	---	PM_{10} median 35 (3, 365)
Moolgavkar, 2000a Los Angeles, CA Moolgavkar, 2003	GAM Strict GLM NS	4.4 (-3.2, 12.6) 6.2 (-3.4, 16.7)	1.0 (-5.1, 7.4) 0.5 (-6.8, 8.4)	---	PM_{10} median 44 (7, 166) $PM_{2.5}$ 22 (4, 86)

Original study* Study Location Reanalysis study	Analysis Comments	% increase (95% CI) per 50 μg/m^3 PM_{10}	% increase (95% CI) per 25 μg/m^3 $PM_{2.5}$	% increase (95% CI) per 25 μg/m^3 $PM_{10-2.5}$	PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ Mean (Range) Levels Reported**
CARDIOVASCULAR MORBIDITY					
Total Cardiovascular Hospital Admissions:					
Samet et al., 2000 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/m^3 PM_{10}	% increase (95% CI) per 25 μg/m^3 $PM_{2.5}$	% increase (95% CI) per 25 μg/m^3 $PM_{10-2.5}$	PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ Mean (Range) Levels Reported**
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/m^3 PM_{10}	% increase (95% CI) per 25 µg/m^3 $PM_{2.5}$	% increase (95% CI) per 25 µg/m^3 $PM_{10-2.5}$	PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ Mean (Range) Levels Reported**
Lippmann et al., 2000 Detroit, MI (>65 years) Ito, 2003	Strict GAM GLM NS	5.00 (-5.27, 16.38) 4.41 (-5.81, 15.74)	1.94 (-5.16, 9.57) 0.97 (-6.06, 8.52)	5.00 (-4.59, 15.56) 5.63 (-4.02, 16.25)	PM_{10} 31 (max 105) $PM_{2.5}$ 18 (6, 86) $PM_{10-2.5}$ 13 (4, 50)
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/m^3 PM_{10}	% increase (95% CI) per 25 µg/m^3 $PM_{2.5}$	% increase (95% CI) per 25 µg/m^3 $PM_{10-2.5}$	PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ Mean (Range) Levels Reported**
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/m^3 PM_{10}	% increase (95% CI) per 25 μg/m^3 $PM_{2.5}$	% increase (95% CI) per 25 μg/m^3 $PM_{10-2.5}$	PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ Mean (Range) Levels Reported**
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/m^3 PM_{10}	% increase (95% CI) per 25 µg/m^3 $PM_{2.5}$	% increase (95% CI) per 25 µg/m^3 $PM_{10-2.5}$	PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ Mean (Range) Levels Reported**
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 GLM NS	10.9 (2.8, 19.6) 8.1 (0.1, 16.7)	8.7 (3.2, 14.4) 6.5 (1.1,12.0)	5.5 (0, 14.0) 5.5 (−2.7, 11.1)	PM_{10} 31.5 (90 55) $PM_{2.5}$ 16.7 (90 32) $PM_{10-2.5}$ 16.2 (90 29)

Original study* Study Location Reanalysis study	Analysis Comments	% increase (95% CI) per 50 µg/m^3 PM_{10}	% increase (95% CI) per 25 µg/m^3 $PM_{2.5}$	% increase (95% CI) per 25 µg/m^3 $PM_{10-2.5}$	PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ Mean (Range) Levels Reported**
Respiratory Symptoms		Odds Ratio (95% CI) for 50 ug/m^3 % increase in PM_{10}	Odds Ratio (95% CI) for 25 ug/m^3 % increase in $PM_{2.5}$	Odds Ratio (95% CI) for 25 ug/m^3 % increase in $PM_{10-2.5}$	$PM_{10-2.5}$ Mean (Range) Levels Reported**
Schwartz et al., 1994 6 U.S. cities (children, cough)	*GAM not used*	*1.39 (1.05, 1.85)*	*1.24 (1.00, 1.54)*	---	*PM_{10} median 30.0 (max 117) $PM_{2.5}$ median 18.0 (max 86)*
Schwartz et al., 1994 6 U.S. cities (children, lower respiratory symptoms)	*GAM not used*	*2.03 (1.36, 3.04)*	*1.58 (1.18, 2.10)*	---	*PM_{10} median 30.0 (max 117) $PM_{2.5}$ median 18.0 (max 86)*
Neas et al., 1995 Uniontown, PA (children, cough)	*GAM not used*	---	*2.45 (1.29, 4.64)*	---	*$PM_{2.5}$ 24.5 (max 88.1)*
Ostro et al., 1991 Denver, CO (adults, cough)	*GAM not used*	*1.09 (0.57, 2.10)*	---	---	*PM_{10} 22 (0.5, 73)*
Pope et al., 1991 Utah Valley, UT (lower respiratory symptoms, schoolchildren)	*GAM not used*	*1.28 (1.06, 1.56)*	---	---	*PM_{10} 44 (11, 195)*
Pope et al., 1991 Utah Valley, UT (lower respiratory symptoms, asthmatic patients)	*GAM not used*	*1.01 (0.81, 1.27)*	---	---	*PM_{10} 44 (11, 195)*

Original study* Study Location Reanalysis study	Analysis Comments	% increase (95% CI) per 50 μg/m^3 PM_{10}	% increase (95% CI) per 25 μg/m^3 $PM_{2.5}$	% increase (95% CI) per 25 μg/m^3 $PM_{10-2.5}$	PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ Mean (Range) Levels Reported**
Neas et al., 1996 State College, PA (children, cough)	GAM not used	NR	1.48 (1.17, 1.88) (1-d)	---	PM_{10} 31.9 (max 82.7) $PM_{2.1}$ 23.5 (max 85.8)
Neas et al., 1996 State College, PA (children, wheeze)	GAM not used	NR	1.59 (0.93, 2.70) (1-d)	---	PM_{10} 31.9 (max 82.7) $PM_{2.1}$ 23.5 (max 85.8)
Neas et al., 1996 State College, PA (children, cold)	GAM not used	NR	1.61 (1.21, 2.17) (0-d)	---	PM_{10} 31.9 (max 82.7) $PM_{2.1}$ 23.5 (max 85.8)
Ostro et al., 1995 Los Angeles, CA (children, asthma episode)	GAM not used	1.05 (0.64, 1.73)	---	---	PM_{10} 55.87 (19.63, 101.42)
Ostro et al., 1995 Los Angeles, CA (children, shortness of breath)	GAM not used	1.51 (1.04, 2.17)	---	---	PM_{10} 55.87 (19.63, 101.42)
Schwartz and Neas, 2000 Six Cities reanalysis (children, cough)	GAM not used	---	1.28 (0.98, 1.67)	1.77 (1.23, 2.54)	$PM_{2.5}$ (same as Six Cities) $PM_{10-2.5}$ NR
Schwartz and Neas, 2000 Six Cities reanalysis (children, lower respiratory symptoms)	GAM not used	---	1.61 (1.20, 2.16)	1.51 (0.66, 3.43)	$PM_{2.5}$ (same as Six Cities) $PM_{10-2.5}$ NR
Vedal et al., 1998 Port Alberni, CAN (children, cough)	GAM not used	1.40 (1.14, 1.73)	---	---	PM_{10} median 22.1 (0.2, 159.0) (north site)

Original study* Study Location Reanalysis study	Analysis Comments	% increase (95% CI) per 50 µg/m^3 PM_{10}	% increase (95% CI) per 25 µg/m^3 $PM_{2.5}$	% increase (95% CI) per 25 µg/m^3 $PM_{10-2.5}$	PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ Mean (Range) Levels Reported**
Vedal et al., 1998 Port Alberni, CAN (children, phlegm)	GAM not used	1.40 (1.03, 1.90)	---	---	PM_{10} median 22.1 (0.2, 159.0) (north site)
Vedal et al., 1998 Port Alberni, CAN (children, nose symptoms)	GAM not used	1.22 (1.00, 1.47)	---	---	PM_{10} median 22.1 (0.2, 159.0) (north site)
Vedal et al., 1998 Port Alberni, CAN (children, sore throat)	GAM not used	1.34 (1.06, 1.69)	---	---	PM_{10} median 22.1 (0.2, 159.0) (north site)
Vedal et al., 1998 Port Alberni, CAN (children, wheeze)	GAM not used	1.16 (0.82, 1.63)	---	---	PM_{10} median 22.1 (0.2, 159.0) (north site)
Vedal et al., 1998 Port Alberni, CAN (children, chest tightness)	GAM not used	1.34 (0.86, 2.09)	---	---	PM_{10} median 22.1 (0.2, 159.0) (north site)
Vedal et al., 1998 Port Alberni, CAN (children, dyspnea)	GAM not used	1.05 (0.74, 1.49)	---	---	PM_{10} median 22.1 (0.2, 159.0) (north site)
Vedal et al., 1998 Port Alberni, CAN (children, any symptom)	GAM not used	1.16 (1.00, 1.34)	---	---	PM_{10} median 22.1 (0.2, 159.0) (north site)

Original study* Study Location Reanalysis study	Analysis Comments	% increase (95% CI) per 50 µg/m^3 PM_{10}	% increase (95% CI) per 25 µg/m^3 $PM_{2.5}$	% increase (95% CI) per 25 µg/m^3 $PM_{10-2.5}$	PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ Mean (Range) Levels Reported**
Lung Function Changes		Lung Function change (L/min) (95% CI) for 50 ug/m^3 % increase in PM_{10}	Lung Function change (L/min) (95% CI) for 25 ug/m^3 % increase in $PM_{2.5}$	Lung Function change (L/min) (95% CI) for 25 ug/m^3 % increase in $PM_{10-2.5}$	$PM_{10-2.5}$ Mean (Range) Levels Reported**
Neas et al., 1995 *Uniontown, PA* *(children)*	*GAM not used*	---	*-2.58 (-5.33, +0.35)*	---	*$PM_{2.5}$ 24.5 (max 88.1)*
Thurston et al., (1997) Connecticut summer camp (children)	GAM not used	---	PEFR -5.4 (-12.3, 1.5) (15 µg/m^3 $SO_4^=$)	---	$SO_4^=$ 7.0 (1.1, 26.7)
Naeher et al., 1999 Southwest VA (adult women)	GAM not used	am PEFR -3.65 (-6.79, -0.51) pm PEFR -1.8 (-5.03, 1.43)	am PEFR -1.83 (-3.44, -0.21) pm PEFR -1.05 (-2.77, 0.67)	am PEFR -6.33 (-12.50, -0.15) pm PEFR -2.4 (-8.48, 3.68)	PM_{10} 27.07 (4.89, 69.07) $PM_{2.5}$ 21.62 (3.48, 59.65) $PM_{10-2.5}$ 5.72 (0.00, 19.78)
Neas et al., 1996 State College, PA (children)	GAM not used	---	pm PEFR -0.64 (-1.73, 0.44)	---	$PM_{2.5}$ 23.5 (max 85.8)
Neas et al., 1999 Philadelphia, PA (children)	GAM not used	am PEFR -8.17 (-14.81, -1.56) pm PEFR -1.44 (-7.33, 4.44)	am PEFR -3.29 (-6.64, 0.07) pm PEFR -0.91 (-4.04, 2.21)	am PEFR -4.31 (-11.44, 2.75) pm PEFR 1.88 (-4.75, 8.44)	$PM_{2.5}$ 22.2 (IQR 16.2) $PM_{10-2.5}$ 9.5 (IQR 5.1)
Schwartz and Neas, 2000 Uniontown, PA (reanalysis) (children)	GAM not used	---	pm PEFR -1.52, (-2.80, -0.24)	pm PEFR +1.73 (-2.2, 5.67)	$PM_{2.5}$ 24.5 (max 88.1) $PM_{10-2.5}$ NR
Schwartz and Neas, 2000 State College PA (reanalysis) (children)	GAM not used	---	pm PEFR -0.93 (-1.88, 0.01)	pm PEFR -0.28 (-3.45, 2.87)	$PM_{2.5}$ 23.5 (max 85.8) $PM_{10-2.5}$ NR

Original study* Study Location Reanalysis study	Analysis Comments	% increase (95% CI) per 50 µg/m^3 PM_{10}	% increase (95% CI) per 25 µg/m^3 $PM_{2.5}$	% increase (95% CI) per 25 µg/m^3 $PM_{10-2.5}$	PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ Mean (Range) Levels Reported**
Vedal et al., 1998 Port Alberni, CAN (children)	GAM not used	PEF -1.35 (-2.7, -0.05)	---	---	PM_{10} median 22.1 (0.2, 159.0) (north site)

* Studies in italics available in 1996 CD

** mean (minimum, maximum) 24-h PM level shown in parentheses unless otherwise noted.

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) $PM_{2.5}$ (10 $\mu g/m^3$) (1999-00) $PM_{2.5}$ (10 $\mu g/m^3$) (average)	1.04 (1.01, 1.08) 1.06 (1.02, 1.10) 1.06 (1.02, 1.11)	21 (9, 34) 14 (5, 20) 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) $PM_{2.5}$ (10 $\mu g/m^3$) (1999-00) $PM_{2.5}$ (10 $\mu g/m^3$) (average)	1.06 (1.02, 1.10) 1.08 (1.02, 1.14) 1.09 (1.03, 1.16)	21 (9, 34) 14 (5, 20) 18 (7.5, 30)
Southern California[F]	PM_{10} (20 $\mu g/m^3$)	1.01 (0.92, 1.10)	51 (0, 84)

Study	Indicator (Increment)	Relative Risk (95% CI)	Study Concentrations ($\mu g/m^3$)
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
Increased Airway Obstruction in Adults			
AHSMOG[K]	PM_{10} (20 $\mu g/m^3$)	1.19 (0.84, 1.68)	NR

Study	Indicator (Increment)	Relative Risk (95% CI)	Study Concentrations ($\mu g/m^3$)
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] (4^{th} 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] (4^{th} 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 4^{th} 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 4^{th} 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 4^{th} 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]
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)

Study	Indicator (Increment)	Relative Risk (95% CI)	Study Concentrations ($\mu g/m^3$)
12 Southern California communities[S]	PM_{10} (20 $\mu g/m^3$)	-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)

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.	Observed Concentrations 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.	Observed Concentrations 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.	Observed Concentrations 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.	Observed Concentrations 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.	Observed Concentrations 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.	Observed Concentrations 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.

Table 4B-1. Estimated Annual Mortality Associated with Short-Term Exposure to $PM_{2.5}$ When Alternative Standards Are Just Met, Assuming Various Cutpoint Levels*
Los Angeles, CA, 2003

Alternative Standards		Incidence Associated with $PM_{2.5}$ (95% Confidence Interval) Percent Reduction in Incidence from Current Standards			
Annual ($\mu g/m^3$)	Daily ($\mu g/m^3$)	Policy Relevant Background =2.5 $\mu g/m^3$	Cutpoint** =10 $\mu g/m^3$	Cutpoint** =15 $\mu g/m^3$	Cutpoint** =20 $\mu g/m^3$
15	65, 98th percentile value***	292 (-37 - 612) 0.0%	115 (-14 - 240) 0.0%	58 (-7 - 121) 0.0%	29 (-4 - 61) 0.0%
	40, 98th percentile value	292 (-37 - 612) 0.0%	115 (-14 - 240) 0.0%	58 (-7 - 121) 0.0%	29 (-4 - 61) 0.0%
	35, 98th percentile value	269 (-34 - 564) 7.9%	96 (-12 - 200) 16.5%	45 (-6 - 94) 22.4%	22 (-3 - 46) 24.1%
	30, 98th percentile value	228 (-28 - 476) 21.9%	65 (-8 - 135) 43.5%	26 (-3 - 54) 55.2%	12 (-2 - 25) 58.6%
	25, 98th percentile value	186 (-23 - 389) 36.3%	39 (-5 - 80) 66.1%	13 (-2 - 27) 77.6%	5 (-1 - 11) 82.8%
	65, 99th percentile value	292 (-37 - 612) 0.0%	115 (-14 - 240) 0.0%	58 (-7 - 121) 0.0%	29 (-4 - 61) 0.0%
	40, 99th percentile value	197 (-25 - 413) 32.5%	45 (-6 - 94) 60.9%	16 (-2 - 33) 72.4%	7 (-1 - 14) 75.9%
	35, 99th percentile value	171 (-21 - 358) 41.4%	30 (-4 - 63) 73.9%	10 (-1 - 20) 82.8%	3 (0 - 7) 89.7%
	30, 99th percentile value	145 (-18 - 302) 50.3%	18 (-2 - 37) 84.3%	5 (-1 - 10) 91.4%	1 (0 - 3) 96.6%
	25, 99th percentile value	118 (-15 - 247) 59.6%	9 (-1 - 18) 92.2%	2 (0 - 4) 96.6%	0 (0 - 1) 100.0%
14	40, 98th percentile value	269 (-34 - 562) 7.9%	96 (-12 - 199) 16.5%	45 (-6 - 93) 22.4%	22 (-3 - 45) 24.1%
	35, 98th percentile value	269 (-34 - 562) 7.9%	96 (-12 - 199) 16.5%	45 (-6 - 93) 22.4%	22 (-3 - 45) 24.1%
	30, 98th percentile value	228 (-28 - 476) 21.9%	65 (-8 - 135) 43.5%	26 (-3 - 54) 55.2%	12 (-2 - 25) 58.6%
	25, 98th percentile value	186 (-23 - 389) 36.3%	39 (-5 - 80) 66.1%	13 (-2 - 27) 77.6%	5 (-1 - 11) 82.8%
	40, 99th percentile value	197 (-25 - 413) 32.5%	45 (-6 - 94) 60.9%	16 (-2 - 33) 72.4%	7 (-1 - 14) 75.9%
	35, 99th percentile value	171 (-21 - 358) 41.4%	30 (-4 - 63) 73.9%	10 (-1 - 20) 82.8%	3 (0 - 7) 89.7%
	30, 99th percentile value	145 (-18 - 302) 50.3%	18 (-2 - 37) 84.3%	5 (-1 - 10) 91.4%	1 (0 - 3) 96.6%
	25, 99th percentile value	118 (-15 - 247) 59.6%	9 (-1 - 18) 92.2%	2 (0 - 4) 96.6%	0 (0 - 1) 100.0%

Alternative Standards		Incidence Associated with $PM_{2.5}$ (95% Confidence Interval) Percent Reduction in Incidence from Current Standards			
Annual ($\mu g/m^3$)	Daily ($\mu g/m^3$)	Policy Relevant Background =2.5 $\mu g/m^3$	Cutpoint** =10 $\mu g/m^3$	Cutpoint** =15 $\mu g/m^3$	Cutpoint** =20 $\mu g/m^3$
13	40, 98th percentile value	245 (-31 - 513) 16.1%	77 (-10 - 161) 33.0%	34 (-4 - 69) 41.4%	16 (-2 - 33) 44.8%
	35, 98th percentile value	245 (-31 - 513) 16.1%	77 (-10 - 161) 33.0%	34 (-4 - 69) 41.4%	16 (-2 - 33) 44.8%
	30, 98th percentile value	228 (-28 - 476) 21.9%	65 (-8 - 135) 43.5%	26 (-3 - 54) 55.2%	12 (-2 - 25) 58.6%
	25, 98th percentile value	186 (-23 - 389) 36.3%	39 (-5 - 80) 66.1%	13 (-2 - 27) 77.6%	5 (-1 - 11) 82.8%
	40, 99th percentile value	197 (-25 - 413) 32.5%	45 (-6 - 94) 60.9%	16 (-2 - 33) 72.4%	7 (-1 - 14) 75.9%
	35, 99th percentile value	171 (-21 - 358) 41.4%	30 (-4 - 63) 73.9%	10 (-1 - 20) 82.8%	3 (0 - 7) 89.7%
	30, 99th percentile value	145 (-18 - 302) 50.3%	18 (-2 - 37) 84.3%	5 (-1 - 10) 91.4%	1 (0 - 3) 96.6%
	25, 99th percentile value	118 (-15 - 247) 59.6%	9 (-1 - 18) 92.2%	2 (0 - 4) 96.6%	0 (0 - 1) 100.0%
12	40, 98th percentile value	222 (-28 - 464) 24.0%	61 (-8 - 126) 47.0%	24 (-3 - 50) 58.6%	11 (-1 - 23) 62.1%
	35, 98th percentile value	222 (-28 - 464) 24.0%	61 (-8 - 126) 47.0%	24 (-3 - 50) 58.6%	11 (-1 - 23) 62.1%
	30, 98th percentile value	222 (-28 - 464) 24.0%	61 (-8 - 126) 47.0%	24 (-3 - 50) 58.6%	11 (-1 - 23) 62.1%
	25, 98th percentile value	186 (-23 - 389) 36.3%	39 (-5 - 80) 66.1%	13 (-2 - 27) 77.6%	5 (-1 - 11) 82.8%
	40, 99th percentile value	197 (-25 - 413) 32.5%	45 (-6 - 94) 60.9%	16 (-2 - 33) 72.4%	7 (-1 - 14) 75.9%
	35, 99th percentile value	171 (-21 - 358) 41.4%	30 (-4 - 63) 73.9%	10 (-1 - 20) 82.8%	3 (0 - 7) 89.7%
	30, 99th percentile value	145 (-18 - 302) 50.3%	18 (-2 - 37) 84.3%	5 (-1 - 10) 91.4%	1 (0 - 3) 96.6%
	25, 99th percentile value	118 (-15 - 247) 59.6%	9 (-1 - 18) 92.2%	2 (0 - 4) 96.6%	0 (0 - 1) 100.0%

*This analysis was performed using Moolgavkar (2003).

**For the cutpoints above policy relevant background, the slope of the concentration-response function has been modified based on a simple hockeystick model (see discussion in section 4.3.2.1).

***Current standards.

Note: Incidences are rounded to the nearest whole number; percents are rounded to the nearest tenth.

Table 4B-2. Estimated Annual Cardiovascular Mortality Associated with Short-Term Exposure to PM2.5 When Alternative Standards Are Just Met, Assuming Various Cutpoint Levels*
Philadelphia, PA, 2003

Alternative Standards		Incidence Associated with PM2.5 (95% Confidence Interval) Percent Reduction in Incidence from Current Standards			
Annual (µg/m3)	Daily (µg/m3)	Policy Relevant Background =3.5 µg/m3	Cutpoint** =10 µg/m3	Cutpoint** =15 µg/m3	Cutpoint** =20 µg/m3
15	65, 98th percentile value***	367 (175 - 560) 0.0%	189 (90 - 288) 0.0%	106 (51 - 162) 0.0%	57 (27 - 87) 0.0%
15	40, 98th percentile value	317 (151 - 482) 13.6%	143 (68 - 218) 24.3%	71 (34 - 107) 33.0%	34 (16 - 51) 40.4%
15	35, 98th percentile value	273 (130 - 416) 25.6%	106 (50 - 161) 43.9%	45 (22 - 69) 57.5%	18 (9 - 28) 68.4%
15	30, 98th percentile value	230 (110 - 350) 37.3%	71 (34 - 108) 62.4%	25 (12 - 38) 76.4%	7 (3 - 11) 87.7%
15	25, 98th percentile value	187 (89 - 284) 49.0%	41 (20 - 63) 78.3%	11 (5 - 16) 89.6%	2 (1 - 3) 96.5%
15	65, 99th percentile value	297 (142 - 451) 19.1%	126 (60 - 191) 33.3%	58 (28 - 89) 45.3%	26 (12 - 40) 54.4%
15	40, 99th percentile value	176 (84 - 268) 52.0%	35 (17 - 53) 81.5%	8 (4 - 12) 92.5%	1 (1 - 2) 98.2%
15	35, 99th percentile value	152 (72 - 231) 58.6%	22 (11 - 34) 88.4%	3 (2 - 5) 97.2%	0 (0 - 1) 100.0%
15	30, 99th percentile value	128 (61 - 195) 65.1%	12 (6 - 19) 93.7%	1 (1 - 2) 99.1%	0 (0 - 0) 100.0%
15	25, 99th percentile value	104 (49 - 158) 71.7%	5 (2 - 8) 97.4%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
14	65, 98th percentile value	336 (160 - 511) 8.4%	160 (76 - 243) 15.3%	83 (40 - 127) 21.7%	42 (20 - 63) 26.3%
14	40, 98th percentile value	317 (151 - 482) 13.6%	143 (68 - 218) 24.3%	71 (34 - 107) 33.0%	34 (16 - 51) 40.4%
14	35, 98th percentile value	273 (130 - 416) 25.6%	106 (50 - 161) 43.9%	45 (22 - 69) 57.5%	18 (9 - 28) 68.4%
14	30, 98th percentile value	230 (110 - 350) 37.3%	71 (34 - 108) 62.4%	25 (12 - 38) 76.4%	7 (3 - 11) 87.7%
14	25, 98th percentile value	187 (89 - 284) 49.0%	41 (20 - 63) 78.3%	11 (5 - 16) 89.6%	2 (1 - 3) 96.5%
14	40, 99th percentile value	176 (84 - 268) 52.0%	35 (17 - 53) 81.5%	8 (4 - 12) 92.5%	1 (1 - 2) 98.2%
14	35, 99th percentile value	152 (72 - 231) 58.6%	22 (11 - 34) 88.4%	3 (2 - 5) 97.2%	0 (0 - 1) 100.0%
14	30, 99th percentile value	128 (61 - 195) 65.1%	12 (6 - 19) 93.7%	1 (1 - 2) 99.1%	0 (0 - 0) 100.0%
14	25, 99th percentile value	104 (49 - 158) 71.7%	5 (2 - 8) 97.4%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%

Alternative Standards		Incidence Associated with PM2.5 (95% Confidence Interval) Percent Reduction in Incidence from Current Standards			
Annual (µg/m3)	Daily (µg/m3)	Policy Relevant Background =3.5 µg/m3	Cutpoint** =10 µg/m3	Cutpoint** =15 µg/m3	Cutpoint** =20 µg/m3
13	40, 98th percentile value	304 (145 - 462) 17.2%	132 (63 - 200) 30.2%	62 (30 - 95) 41.5%	29 (14 - 44) 49.1%
13	35, 98th percentile value	273 (130 - 416) 25.6%	106 (50 - 161) 43.9%	45 (22 - 69) 57.5%	18 (9 - 28) 68.4%
13	30, 98th percentile value	230 (110 - 350) 37.3%	71 (34 - 108) 62.4%	25 (12 - 38) 76.4%	7 (3 - 11) 87.7%
13	25, 98th percentile value	187 (89 - 284) 49.0%	41 (20 - 63) 78.3%	11 (5 - 16) 89.6%	2 (1 - 3) 96.5%
13	40, 99th percentile value	176 (84 - 268) 52.0%	35 (17 - 53) 81.5%	8 (4 - 12) 92.5%	1 (1 - 2) 98.2%
13	35, 99th percentile value	152 (72 - 231) 58.6%	22 (11 - 34) 88.4%	3 (2 - 5) 97.2%	0 (0 - 1) 100.0%
13	30, 99th percentile value	128 (61 - 195) 65.1%	12 (6 - 19) 93.7%	1 (1 - 2) 99.1%	0 (0 - 0) 100.0%
13	25, 99th percentile value	104 (49 - 158) 71.7%	5 (2 - 8) 97.4%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
12	40, 98th percentile value	272 (130 - 414) 25.9%	104 (50 - 159) 45.0%	44 (21 - 68) 58.5%	18 (9 - 27) 68.4%
12	35, 98th percentile value	272 (130 - 414) 25.9%	104 (50 - 159) 45.0%	44 (21 - 68) 58.5%	18 (9 - 27) 68.4%
12	30, 98th percentile value	230 (110 - 350) 37.3%	71 (34 - 108) 62.4%	25 (12 - 38) 76.4%	7 (3 - 11) 87.7%
12	25, 98th percentile value	187 (89 - 284) 49.0%	41 (20 - 63) 78.3%	11 (5 - 16) 89.6%	2 (1 - 3) 96.5%
12	40, 99th percentile value	176 (84 - 268) 52.0%	35 (17 - 53) 81.5%	8 (4 - 12) 92.5%	1 (1 - 2) 98.2%
12	35, 99th percentile value	152 (72 - 231) 58.6%	22 (11 - 34) 88.4%	3 (2 - 5) 97.2%	0 (0 - 1) 100.0%
12	30, 99th percentile value	128 (61 - 195) 65.1%	12 (6 - 19) 93.7%	1 (1 - 2) 99.1%	0 (0 - 0) 100.0%
12	25, 99th percentile value	104 (49 - 158) 71.7%	5 (2 - 8) 97.4%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%

*This analysis was performed using Lipfert et al. (2000).

**For the cutpoints above policy relevant background, the slope of the concentration-response function has been modified based on a simple hockeystick model (see discussion in section 4.3.2.1).

***Current standards.

Note: Incidences are rounded to the nearest whole number; percents are rounded to the nearest tenth.

Table 4B-3. Estimated Annual Mortality Associated with Short-Term Exposure to $PM_{2.5}$ When Alternative Standards Are Just Met, Assuming Various Cutpoint Levels*
Pittsburgh, PA, 2003

Alternative Standards		Incidence Associated with $PM_{2.5}$ (95% Confidence Interval) Percent Reduction in Incidence from Current Standards			
Annual ($\mu g/m^3$)	Daily ($\mu g/m^3$)	Policy Relevant Background =3.5 $\mu g/m^3$	Cutpoint** =10 $\mu g/m^3$	Cutpoint** =15 $\mu g/m^3$	Cutpoint** =20 $\mu g/m^3$
15	65, 98th percentile value***	50 (-108 - 200) 0.0%	22 (-48 - 87) 0.0%	10 (-23 - 41) 0.0%	5 (-11 - 18) 0.0%
	40, 98th percentile value	47 (-102 - 189) 6.0%	19 (-43 - 77) 13.6%	9 (-19 - 34) 10.0%	4 (-9 - 15) 20.0%
	35, 98th percentile value	41 (-88 - 162) 18.0%	14 (-31 - 56) 36.4%	5 (-12 - 21) 50.0%	2 (-5 - 8) 60.0%
	30, 98th percentile value	34 (-74 - 136) 32.0%	9 (-21 - 37) 59.1%	3 (-6 - 11) 70.0%	1 (-2 - 4) 80.0%
	25, 98th percentile value	28 (-60 - 110) 44.0%	5 (-12 - 20) 77.3%	1 (-3 - 5) 90.0%	0 (-1 - 2) 100.0%
	65, 99th percentile value	50 (-108 - 200) 0.0%	22 (-48 - 87) 0.0%	10 (-23 - 41) 0.0%	5 (-11 - 18) 0.0%
	40, 99th percentile value	42 (-92 - 168) 16.0%	15 (-34 - 61) 31.8%	6 (-13 - 24) 40.0%	3 (-6 - 10) 40.0%
	35, 99th percentile value	36 (-79 - 145) 28.0%	11 (-24 - 43) 50.0%	4 (-8 - 14) 60.0%	1 (-3 - 5) 80.0%
	30, 99th percentile value	31 (-67 - 122) 38.0%	7 (-15 - 27) 68.2%	2 (-4 - 7) 80.0%	1 (-2 - 3) 80.0%
	25, 99th percentile value	25 (-54 - 99) 50.0%	4 (-8 - 14) 81.8%	1 (-2 - 3) 90.0%	0 (-1 - 1) 100.0%
14	40, 98th percentile value	46 (-99 - 182) 8.0%	18 (-40 - 72) 18.2%	8 (-17 - 31) 20.0%	3 (-8 - 13) 40.0%
	35, 98th percentile value	41 (-88 - 162) 18.0%	14 (-31 - 56) 36.4%	5 (-12 - 21) 50.0%	2 (-5 - 8) 60.0%
	30, 98th percentile value	34 (-74 - 136) 32.0%	9 (-21 - 37) 59.1%	3 (-6 - 11) 70.0%	1 (-2 - 4) 80.0%
	25, 98th percentile value	28 (-60 - 110) 44.0%	5 (-12 - 20) 77.3%	1 (-3 - 5) 90.0%	0 (-1 - 2) 100.0%
	40, 99th percentile value	42 (-92 - 168) 16.0%	15 (-34 - 61) 31.8%	6 (-13 - 24) 40.0%	3 (-6 - 10) 40.0%
	35, 99th percentile value	36 (-79 - 145) 28.0%	11 (-24 - 43) 50.0%	4 (-8 - 14) 60.0%	1 (-3 - 5) 80.0%
	30, 99th percentile value	31 (-67 - 122) 38.0%	7 (-15 - 27) 68.2%	2 (-4 - 7) 80.0%	1 (-2 - 3) 80.0%
	25, 99th percentile value	25 (-54 - 99) 50.0%	4 (-8 - 14) 81.8%	1 (-2 - 3) 90.0%	0 (-1 - 1) 100.0%

Alternative Standards		Incidence Associated with $PM_{2.5}$ (95% Confidence Interval) Percent Reduction in Incidence from Current Standards			
Annual ($\mu g/m^3$)	Daily ($\mu g/m^3$)	Policy Relevant Background =3.5 $\mu g/m^3$	Cutpoint** =10 $\mu g/m^3$	Cutpoint** =15 $\mu g/m^3$	Cutpoint** =20 $\mu g/m^3$
13	40, 98th percentile value	41 (-90 - 165) 18.0%	15 (-32 - 58) 31.8%	6 (-13 - 22) 40.0%	2 (-5 - 9) 60.0%
	35, 98th percentile value	41 (-88 - 162) 18.0%	14 (-31 - 56) 36.4%	5 (-12 - 21) 50.0%	2 (-5 - 8) 60.0%
	30, 98th percentile value	34 (-74 - 136) 32.0%	9 (-21 - 37) 59.1%	3 (-6 - 11) 70.0%	1 (-2 - 4) 80.0%
	25, 98th percentile value	28 (-60 - 110) 44.0%	5 (-12 - 20) 77.3%	1 (-3 - 5) 90.0%	0 (-1 - 2) 100.0%
	40, 99th percentile value	41 (-90 - 165) 18.0%	15 (-32 - 58) 31.8%	6 (-13 - 22) 40.0%	2 (-5 - 9) 60.0%
	35, 99th percentile value	36 (-79 - 145) 28.0%	11 (-24 - 43) 50.0%	4 (-8 - 14) 60.0%	1 (-3 - 5) 80.0%
	30, 99th percentile value	31 (-67 - 122) 38.0%	7 (-15 - 27) 68.2%	2 (-4 - 7) 80.0%	1 (-2 - 3) 80.0%
	25, 99th percentile value	25 (-54 - 99) 50.0%	4 (-8 - 14) 81.8%	1 (-2 - 3) 90.0%	0 (-1 - 1) 100.0%
12	40, 98th percentile value	37 (-80 - 147) 26.0%	11 (-25 - 44) 50.0%	4 (-8 - 15) 60.0%	1 (-3 - 6) 80.0%
	35, 98th percentile value	37 (-80 - 147) 26.0%	11 (-25 - 44) 50.0%	4 (-8 - 15) 60.0%	1 (-3 - 6) 80.0%
	30, 98th percentile value	34 (-74 - 136) 32.0%	9 (-21 - 37) 59.1%	3 (-6 - 11) 70.0%	1 (-2 - 4) 80.0%
	25, 98th percentile value	28 (-60 - 110) 44.0%	5 (-12 - 20) 77.3%	1 (-3 - 5) 90.0%	0 (-1 - 2) 100.0%
	40, 99th percentile value	37 (-80 - 147) 26.0%	11 (-25 - 44) 50.0%	4 (-8 - 15) 60.0%	1 (-3 - 6) 80.0%
	35, 99th percentile value	36 (-79 - 145) 28.0%	11 (-24 - 43) 50.0%	4 (-8 - 14) 60.0%	1 (-3 - 5) 80.0%
	30, 99th percentile value	31 (-67 - 122) 38.0%	7 (-15 - 27) 68.2%	2 (-4 - 7) 80.0%	1 (-2 - 3) 80.0%
	25, 99th percentile value	25 (-54 - 99) 50.0%	4 (-8 - 14) 81.8%	1 (-2 - 3) 90.0%	0 (-1 - 1) 100.0%

*This analysis was performed using Chock et al. (2000), age 75+ model.

**For the cutpoints above policy relevant background, the slope of the concentration-response function has been modified based on a simple hockeystick model (see discussion in section 4.3.2.1).

***Current standards.

Note: Incidences are rounded to the nearest whole number; percents are rounded to the nearest tenth.

Table 4B-4. Estimated Annual Mortality Associated with Short-Term Exposure to $PM_{2.5}$ When Alternative Standards Are Just Met, Assuming Various Cutpoint Levels*
St. Louis, MO, 2003

Alternative Standards		Incidence Associated with $PM_{2.5}$ (95% Confidence Interval) Percent Reduction in Incidence from Current Standards			
Annual ($\mu g/m^3$)	Daily ($\mu g/m^3$)	Policy Relevant Background =3.5 $\mu g/m^3$	Cutpoint** =10 $\mu g/m^3$	Cutpoint** =15 $\mu g/m^3$	Cutpoint** =20 $\mu g/m^3$
15	65, 98th percentile value***	191 (70 - 311) 0.0%	75 (28 - 122) 0.0%	29 (11 - 46) 0.0%	9 (3 - 14) 0.0%
	40, 98th percentile value	191 (70 - 311) 0.0%	75 (28 - 122) 0.0%	29 (11 - 46) 0.0%	9 (3 - 14) 0.0%
	35, 98th percentile value	190 (70 - 310) 0.5%	75 (27 - 121) 0.0%	28 (10 - 46) 3.4%	8 (3 - 14) 11.1%
	30, 98th percentile value	160 (59 - 260) 16.2%	49 (18 - 80) 34.7%	14 (5 - 23) 51.7%	3 (1 - 4) 66.7%
	25, 98th percentile value	130 (48 - 211) 31.9%	28 (10 - 45) 62.7%	5 (2 - 8) 82.8%	1 (0 - 1) 88.9%
	65, 99th percentile value	191 (70 - 311) 0.0%	75 (28 - 122) 0.0%	29 (11 - 46) 0.0%	9 (3 - 14) 0.0%
	40, 99th percentile value	191 (70 - 311) 0.0%	75 (28 - 122) 0.0%	29 (11 - 46) 0.0%	9 (3 - 14) 0.0%
	35, 99th percentile value	172 (63 - 280) 9.9%	59 (22 - 96) 21.3%	19 (7 - 31) 34.5%	5 (2 - 7) 44.4%
	30, 99th percentile value	145 (53 - 235) 24.1%	38 (14 - 62) 49.3%	9 (3 - 14) 69.0%	2 (1 - 3) 77.8%
	25, 99th percentile value	118 (43 - 191) 38.2%	20 (7 - 33) 73.3%	3 (1 - 4) 89.7%	0 (0 - 1) 100.0%
14	40, 98th percentile value	175 (64 - 284) 8.4%	61 (22 - 99) 18.7%	20 (7 - 33) 31.0%	5 (2 - 8) 44.4%
	35, 98th percentile value	175 (64 - 284) 8.4%	61 (22 - 99) 18.7%	20 (7 - 33) 31.0%	5 (2 - 8) 44.4%
	30, 98th percentile value	160 (59 - 260) 16.2%	49 (18 - 80) 34.7%	14 (5 - 23) 51.7%	3 (1 - 4) 66.7%
	25, 98th percentile value	130 (48 - 211) 31.9%	28 (10 - 45) 62.7%	5 (2 - 8) 82.8%	1 (0 - 1) 88.9%
	40, 99th percentile value	175 (64 - 284) 8.4%	61 (22 - 99) 18.7%	20 (7 - 33) 31.0%	5 (2 - 8) 44.4%
	35, 99th percentile value	172 (63 - 280) 9.9%	59 (22 - 96) 21.3%	19 (7 - 31) 34.5%	5 (2 - 7) 44.4%
	30, 99th percentile value	145 (53 - 235) 24.1%	38 (14 - 62) 49.3%	9 (3 - 14) 69.0%	2 (1 - 3) 77.8%
	25, 99th percentile value	118 (43 - 191) 38.2%	20 (7 - 33) 73.3%	3 (1 - 4) 89.7%	0 (0 - 1) 100.0%

Alternative Standards		Incidence Associated with $PM_{2.5}$ (95% Confidence Interval) Percent Reduction in Incidence from Current Standards			
Annual ($\mu g/m^3$)	Daily ($\mu g/m^3$)	Policy Relevant Background =3.5 $\mu g/m^3$	Cutpoint** =10 $\mu g/m^3$	Cutpoint** =15 $\mu g/m^3$	Cutpoint** =20 $\mu g/m^3$
13	40, 98th percentile value	158 (58 - 256) 17.3%	47 (17 - 77) 37.3%	13 (5 - 21) 55.2%	3 (1 - 4) 66.7%
	35, 98th percentile value	158 (58 - 256) 17.3%	47 (17 - 77) 37.3%	13 (5 - 21) 55.2%	3 (1 - 4) 66.7%
	30, 98th percentile value	158 (58 - 256) 17.3%	47 (17 - 77) 37.3%	13 (5 - 21) 55.2%	3 (1 - 4) 66.7%
	25, 98th percentile value	130 (48 - 211) 31.9%	28 (10 - 45) 62.7%	5 (2 - 8) 82.8%	1 (0 - 1) 88.9%
	40, 99th percentile value	158 (58 - 256) 17.3%	47 (17 - 77) 37.3%	13 (5 - 21) 55.2%	3 (1 - 4) 66.7%
	35, 99th percentile value	158 (58 - 256) 17.3%	47 (17 - 77) 37.3%	13 (5 - 21) 55.2%	3 (1 - 4) 66.7%
	30, 99th percentile value	145 (53 - 235) 24.1%	38 (14 - 62) 49.3%	9 (3 - 14) 69.0%	2 (1 - 3) 77.8%
	25, 99th percentile value	118 (43 - 191) 38.2%	20 (7 - 33) 73.3%	3 (1 - 4) 89.7%	0 (0 - 1) 100.0%
12	40, 98th percentile value	141 (52 - 229) 26.2%	35 (13 - 57) 53.3%	8 (3 - 12) 72.4%	1 (1 - 2) 88.9%
	35, 98th percentile value	141 (52 - 229) 26.2%	35 (13 - 57) 53.3%	8 (3 - 12) 72.4%	1 (1 - 2) 88.9%
	30, 98th percentile value	141 (52 - 229) 26.2%	35 (13 - 57) 53.3%	8 (3 - 12) 72.4%	1 (1 - 2) 88.9%
	25, 98th percentile value	130 (48 - 211) 31.9%	28 (10 - 45) 62.7%	5 (2 - 8) 82.8%	1 (0 - 1) 88.9%
	40, 99th percentile value	141 (52 - 229) 26.2%	35 (13 - 57) 53.3%	8 (3 - 12) 72.4%	1 (1 - 2) 88.9%
	35, 99th percentile value	141 (52 - 229) 26.2%	35 (13 - 57) 53.3%	8 (3 - 12) 72.4%	1 (1 - 2) 88.9%
	30, 99th percentile value	141 (52 - 229) 26.2%	35 (13 - 57) 53.3%	8 (3 - 12) 72.4%	1 (1 - 2) 88.9%
	25, 99th percentile value	118 (43 - 191) 38.2%	20 (7 - 33) 73.3%	3 (1 - 4) 89.7%	0 (0 - 1) 100.0%

*This analysis was performed using Schwartz (2003b).

**For the cutpoints above policy relevant background, the slope of the concentration-response function has been modified based on a simple hockeystick model (see discussion in section 4.3.2.1).

***Current standards.

Note: Incidences are rounded to the nearest whole number; percents are rounded to the nearest tenth.

Table 4B-5. Estimated Annual Mortality Associated with Long-Term Exposure to $PM_{2.5}$ When Alternative Standards Are Just Met, Assuming Various Cutpoint Levels*
Los Angeles, CA, 2003

Alternative Standards		Incidence Associated with $PM_{2.5}$ (95% Confidence Interval) Percent Reduction in Incidence from Current Standards		
Annual ($\mu g/m^3$)	Daily ($\mu g/m^3$)	Cutpoint** =7.5 $\mu g/m^3$	Cutpoint** =10 $\mu g/m^3$	Cutpoint** =12 $\mu g/m^3$
15	65, 98th percentile value***	1507 (531 - 2587) 0.0%	823 (290 - 1415) 0.0%	138 (48 - 237) 0.0%
	40, 98th percentile value	1507 (531 - 2587) 0.0%	823 (290 - 1415) 0.0%	138 (48 - 237) 0.0%
	35, 98th percentile value	1265 (446 - 2168) 16.1%	553 (195 - 949) 32.8%	0 (0 - 0) 100.0%
	30, 98th percentile value	829 (293 - 1416) 45.0%	65 (23 - 111) 92.1%	0 (0 - 0) 100.0%
	25, 98th percentile value	396 (140 - 675) 73.7%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
	65, 99th percentile value	1507 (531 - 2587) 0.0%	823 (290 - 1415) 0.0%	138 (48 - 237) 0.0%
	40, 99th percentile value	514 (182 - 876) 65.9%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
	35, 99th percentile value	240 (85 - 408) 84.1%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
	30, 99th percentile value	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
	25, 99th percentile value	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
14	40, 98th percentile value	1259 (444 - 2158) 16.5%	546 (192 - 937) 33.7%	0 (0 - 0) 100.0%
	35, 98th percentile value	1259 (444 - 2158) 16.5%	546 (192 - 937) 33.7%	0 (0 - 0) 100.0%
	30, 98th percentile value	829 (293 - 1416) 45.0%	65 (23 - 111) 92.1%	0 (0 - 0) 100.0%
	25, 98th percentile value	396 (140 - 675) 73.7%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
	40, 99th percentile value	514 (182 - 876) 65.9%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
	35, 99th percentile value	240 (85 - 408) 84.1%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
	30, 99th percentile value	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
	25, 99th percentile value	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%

Alternative Standards		Incidence Associated with $PM_{2.5}$ (95% Confidence Interval) Percent Reduction in Incidence from Current Standards		
Annual ($\mu g/m^3$)	Daily ($\mu g/m^3$)	Cutpoint** =7.5 $\mu g/m^3$	Cutpoint** =10 $\mu g/m^3$	Cutpoint** =12 $\mu g/m^3$
13	40, 98th percentile value	1013 (358 - 1732) 32.8%	270 (95 - 463) 67.2%	0 (0 - 0) 100.0%
	35, 98th percentile value	1013 (358 - 1732) 32.8%	270 (95 - 463) 67.2%	0 (0 - 0) 100.0%
	30, 98th percentile value	829 (293 - 1416) 45.0%	65 (23 - 111) 92.1%	0 (0 - 0) 100.0%
	25, 98th percentile value	396 (140 - 675) 73.7%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
	40, 99th percentile value	514 (182 - 876) 65.9%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
	35, 99th percentile value	240 (85 - 408) 84.1%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
	30, 99th percentile value	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
	25, 99th percentile value	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
12	40, 98th percentile value	767 (271 - 1310) 49.1%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
	35, 98th percentile value	767 (271 - 1310) 49.1%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
	30, 98th percentile value	767 (271 - 1310) 49.1%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
	25, 98th percentile value	396 (140 - 675) 73.7%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
	40, 99th percentile value	514 (182 - 876) 65.9%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
	35, 99th percentile value	240 (85 - 408) 84.1%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
	30, 99th percentile value	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
	25, 99th percentile value	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%

*This analysis was performed using Pope et al. (2002) -- ACS extended.

**For the cutpoints above policy relevant background, the slope of the C-R function has been modified based on a simple hockeystick model (see discussion in section 4.3.2.1).

***Current standards.

Note: Incidences are rounded to the nearest whole number; percents are rounded to the nearest tenth.

Table 4B-6. Estimated Annual Mortality Associated with Long-Term Exposure to $PM_{2.5}$ When Alternative Standards Are Just Met, Assuming Various Cutpoint Levels*
Philadelphia, PA, 2003

Alternative Standards		Incidence Associated with PM2.5 (95% Confidence Interval) Percent Reduction in Incidence from Current Standards		
Annual (µg/m3)	Daily (µg/m3)	Cutpoint** =7.5 µg/m3	Cutpoint** =10 µg/m3	Cutpoint** =12 µg/m3
15	65, 98th percentile value***	536 (185 - 943) 0.0%	338 (116 - 597) 0.0%	137 (47 - 244) 0.0%
15	40, 98th percentile value	408 (141 - 716) 23.9%	194 (67 - 341) 42.6%	0 (0 - 0) 100.0%
15	35, 98th percentile value	299 (104 - 524) 44.2%	72 (25 - 126) 78.7%	0 (0 - 0) 100.0%
15	30, 98th percentile value	191 (67 - 334) 64.4%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
15	25, 98th percentile value	84 (29 - 146) 84.3%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
15	65, 99th percentile value	357 (124 - 626) 33.4%	137 (47 - 241) 59.5%	0 (0 - 0) 100.0%
15	40, 99th percentile value	58 (20 - 101) 89.2%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
15	35, 99th percentile value	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
15	30, 99th percentile value	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
15	25, 99th percentile value	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
14	65, 98th percentile value	456 (157 - 799) 14.9%	247 (85 - 435) 26.9%	37 (13 - 65) 73.0%
14	40, 98th percentile value	408 (141 - 716) 23.9%	194 (67 - 341) 42.6%	0 (0 - 0) 100.0%
14	35, 98th percentile value	299 (104 - 524) 44.2%	72 (25 - 126) 78.7%	0 (0 - 0) 100.0%
14	30, 98th percentile value	191 (67 - 334) 64.4%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
14	25, 98th percentile value	84 (29 - 146) 84.3%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
14	40, 99th percentile value	58 (20 - 101) 89.2%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
14	35, 99th percentile value	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
14	30, 99th percentile value	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
14	25, 99th percentile value	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%

Alternative Standards		Incidence Associated with PM2.5 (95% Confidence Interval) Percent Reduction in Incidence from Current Standards		
Annual (µg/m3)	Daily (µg/m3)	Cutpoint** =7.5 µg/m3	Cutpoint** =10 µg/m3	Cutpoint** =12 µg/m3
13	40, 98th percentile value	375 (130 - 657) 30.0%	157 (54 - 276) 53.6%	0 (0 - 0) 100.0%
13	35, 98th percentile value	299 (104 - 524) 44.2%	72 (25 - 126) 78.7%	0 (0 - 0) 100.0%
13	30, 98th percentile value	191 (67 - 334) 64.4%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
13	25, 98th percentile value	84 (29 - 146) 84.3%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
13	40, 99th percentile value	58 (20 - 101) 89.2%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
13	35, 99th percentile value	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
13	30, 99th percentile value	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
13	25, 99th percentile value	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
12	40, 98th percentile value	295 (102 - 516) 45.0%	67 (23 - 118) 80.2%	0 (0 - 0) 100.0%
12	35, 98th percentile value	295 (102 - 516) 45.0%	67 (23 - 118) 80.2%	0 (0 - 0) 100.0%
12	30, 98th percentile value	191 (67 - 334) 64.4%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
12	25, 98th percentile value	84 (29 - 146) 84.3%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
12	40, 99th percentile value	58 (20 - 101) 89.2%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
12	35, 99th percentile value	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
12	30, 99th percentile value	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
12	25, 99th percentile value	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%

*This analysis was performed using Pope et al. (2002) -- ACS extended.

**For the cutpoints above 7.5 µg/m3, the slope of the concentration-response function has been modified based on a simple hockeystick model (see discussion in section 4.3.2.1).

***Current standards.

Note: Incidences are rounded to the nearest whole number; percents are rounded to the nearest tenth.

Table 4B-7. Estimated Annual Mortality Associated with Long-Term Exposure to $PM_{2.5}$ When Alternative Standards Are Just Met, Assuming Various Cutpoint Levels*
Pittsburgh, PA, 2003

Alternative Standards		Incidence Associated with $PM_{2.5}$ (95% Confidence Interval) Percent Reduction in Incidence from Current Standards		
Annual ($\mu g/m^3$)	Daily ($\mu g/m^3$)	Cutpoint** =7.5 $\mu g/m^3$	Cutpoint** =10 $\mu g/m^3$	Cutpoint** =12 $\mu g/m^3$
15	65, 98th percentile value***	403 (141 - 699) 0.0%	215 (75 - 373) 0.0%	25 (9 - 43) 0.0%
	40, 98th percentile value	361 (126 - 626) 10.4%	168 (58 - 291) 21.9%	0 (0 - 0) 100.0%
	35, 98th percentile value	264 (93 - 456) 34.5%	59 (21 - 102) 72.6%	0 (0 - 0) 100.0%
	30, 98th percentile value	168 (59 - 289) 58.3%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
	25, 98th percentile value	72 (25 - 124) 82.1%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
	65, 99th percentile value	403 (141 - 699) 0.0%	215 (75 - 373) 0.0%	25 (9 - 43) 0.0%
	40, 99th percentile value	287 (100 - 495) 28.8%	84 (29 - 145) 60.9%	0 (0 - 0) 100.0%
	35, 99th percentile value	200 (70 - 345) 50.4%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
	30, 99th percentile value	114 (40 - 197) 71.7%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
	25, 99th percentile value	29 (10 - 50) 92.8%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
14	40, 98th percentile value	338 (118 - 585) 16.1%	141 (49 - 245) 34.4%	0 (0 - 0) 100.0%
	35, 98th percentile value	264 (93 - 456) 34.5%	59 (21 - 102) 72.6%	0 (0 - 0) 100.0%
	30, 98th percentile value	168 (59 - 289) 58.3%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
	25, 98th percentile value	72 (25 - 124) 82.1%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
	40, 99th percentile value	287 (100 - 495) 28.8%	84 (29 - 145) 60.9%	0 (0 - 0) 100.0%
	35, 99th percentile value	200 (70 - 345) 50.4%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
	30, 99th percentile value	114 (40 - 197) 71.7%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
	25, 99th percentile value	29 (10 - 50) 92.8%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%

Alternative Standards		Incidence Associated with $PM_{2.5}$ (95% Confidence Interval) Percent Reduction in Incidence from Current Standards		
Annual ($\mu g/m^3$)	Daily ($\mu g/m^3$)	Cutpoint** =7.5 $\mu g/m^3$	Cutpoint** =10 $\mu g/m^3$	Cutpoint** =12 $\mu g/m^3$
13	40, 98th percentile value	273 (96 - 471) 32.3%	68 (24 - 118) 68.4%	0 (0 - 0) 100.0%
	35, 98th percentile value	264 (93 - 456) 34.5%	59 (21 - 102) 72.6%	0 (0 - 0) 100.0%
	30, 98th percentile value	168 (59 - 289) 58.3%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
	25, 98th percentile value	72 (25 - 124) 82.1%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
	40, 99th percentile value	273 (96 - 471) 32.3%	68 (24 - 118) 68.4%	0 (0 - 0) 100.0%
	35, 99th percentile value	200 (70 - 345) 50.4%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
	30, 99th percentile value	114 (40 - 197) 71.7%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
	25, 99th percentile value	29 (10 - 50) 92.8%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
12	40, 98th percentile value	208 (73 - 358) 48.4%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
	35, 98th percentile value	208 (73 - 358) 48.4%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
	30, 98th percentile value	168 (59 - 289) 58.3%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
	25, 98th percentile value	72 (25 - 124) 82.1%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
	40, 99th percentile value	208 (73 - 358) 48.4%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
	35, 99th percentile value	200 (70 - 345) 50.4%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
	30, 99th percentile value	114 (40 - 197) 71.7%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
	25, 99th percentile value	29 (10 - 50) 92.8%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%

*This analysis was performed using Pope et al. (2002) -- ACS extended.

**For the cutpoints above policy relevant background, the slope of the concentration-response function has been modified based on a simple hockeystick model (see discussion in section 4.3.2.1).

***Current standards.

Note: Incidences are rounded to the nearest whole number; percents are rounded to the nearest tenth.

Table 4B-8. Estimated Annual Mortality Associated with Long-Term Exposure to $PM_{2.5}$ When Alternative Standards Are Just Met, Assuming Various Cutpoint Levels*
St. Louis, MO, 2003

Alternative Standards		Incidence Associated with $PM_{2.5}$ (95% Confidence Interval) Percent Reduction in Incidence from Current Standards		
Annual ($\mu g/m^3$)	Daily ($\mu g/m^3$)	Cutpoint** =7.5 $\mu g/m^3$	Cutpoint** =10 $\mu g/m^3$	Cutpoint** =12 $\mu g/m^3$
15	65, 98th percentile value***	596 (206 - 1047) 0.0%	311 (107 - 548) 0.0%	23 (8 - 40) 0.0%
	40, 98th percentile value	596 (206 - 1047) 0.0%	311 (107 - 548) 0.0%	23 (8 - 40) 0.0%
	35, 98th percentile value	592 (204 - 1039) 0.7%	306 (105 - 539) 1.6%	17 (6 - 30) 26.1%
	30, 98th percentile value	414 (144 - 726) 30.5%	107 (37 - 188) 65.6%	0 (0 - 0) 100.0%
	25, 98th percentile value	239 (83 - 417) 59.9%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
	65, 99th percentile value	596 (206 - 1047) 0.0%	311 (107 - 548) 0.0%	23 (8 - 40) 0.0%
	40, 99th percentile value	596 (206 - 1047) 0.0%	311 (107 - 548) 0.0%	23 (8 - 40) 0.0%
	35, 99th percentile value	486 (168 - 853) 18.5%	188 (65 - 330) 39.5%	0 (0 - 0) 100.0%
	30, 99th percentile value	327 (113 - 571) 45.1%	8 (3 - 15) 97.4%	0 (0 - 0) 100.0%
	25, 99th percentile value	168 (58 - 293) 71.8%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
14	40, 98th percentile value	498 (172 - 874) 16.4%	201 (69 - 354) 35.4%	0 (0 - 0) 100.0%
	35, 98th percentile value	498 (172 - 874) 16.4%	201 (69 - 354) 35.4%	0 (0 - 0) 100.0%
	30, 98th percentile value	414 (144 - 726) 30.5%	107 (37 - 188) 65.6%	0 (0 - 0) 100.0%
	25, 98th percentile value	239 (83 - 417) 59.9%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
	40, 99th percentile value	498 (172 - 874) 16.4%	201 (69 - 354) 35.4%	0 (0 - 0) 100.0%
	35, 99th percentile value	486 (168 - 853) 18.5%	188 (65 - 330) 39.5%	0 (0 - 0) 100.0%
	30, 99th percentile value	327 (113 - 571) 45.1%	8 (3 - 15) 97.4%	0 (0 - 0) 100.0%
	25, 99th percentile value	168 (58 - 293) 71.8%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%

Alternative Standards		Incidence Associated with $PM_{2.5}$ (95% Confidence Interval) Percent Reduction in Incidence from Current Standards		
Annual ($\mu g/m^3$)	Daily ($\mu g/m^3$)	Cutpoint** =7.5 $\mu g/m^3$	Cutpoint** =10 $\mu g/m^3$	Cutpoint** =12 $\mu g/m^3$
13	40, 98th percentile value	401 (139 - 702) 32.7%	92 (32 - 162) 70.4%	0 (0 - 0) 100.0%
	35, 98th percentile value	401 (139 - 702) 32.7%	92 (32 - 162) 70.4%	0 (0 - 0) 100.0%
	30, 98th percentile value	401 (139 - 702) 32.7%	92 (32 - 162) 70.4%	0 (0 - 0) 100.0%
	25, 98th percentile value	239 (83 - 417) 59.9%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
	40, 99th percentile value	401 (139 - 702) 32.7%	92 (32 - 162) 70.4%	0 (0 - 0) 100.0%
	35, 99th percentile value	401 (139 - 702) 32.7%	92 (32 - 162) 70.4%	0 (0 - 0) 100.0%
	30, 99th percentile value	327 (113 - 571) 45.1%	8 (3 - 15) 97.4%	0 (0 - 0) 100.0%
	25, 99th percentile value	168 (58 - 293) 71.8%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
12	40, 98th percentile value	304 (106 - 532) 49.0%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
	35, 98th percentile value	304 (106 - 532) 49.0%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
	30, 98th percentile value	304 (106 - 532) 49.0%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
	25, 98th percentile value	239 (83 - 417) 59.9%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
	40, 99th percentile value	304 (106 - 532) 49.0%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
	35, 99th percentile value	304 (106 - 532) 49.0%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
	30, 99th percentile value	304 (106 - 532) 49.0%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
	25, 99th percentile value	168 (58 - 293) 71.8%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%

*This analysis was performed using Pope et al. (2002) -- ACS extended.

**For the cutpoints above policy relevant background, the slope of the concentration-response function has been modified based on a simple hockeystick model (see discussion in section 4.3.2.1).

***Current standards.

Note: Incidences are rounded to the nearest whole number; percents are rounded to the nearest tenth.

Table 4B-9. Sensitivity Analysis: Estimated Annual Mortality Associated with Short-Term Exposure to $PM_{2.5}$ When Alternative Standards Are Just Met, Assuming Various Cutpoint Levels -- Rollbacks to Meet Annual Standards Using Design Values Based on Maximum vs. Average of Monitor-Specific Averages*
Detroit, MI, 2003

Alternative Standards		Incidence Associated with $PM_{2.5}$ Using an Annual Design Value Based on the Maximum of Monitor-Specific Averages** (95% Confidence Interval)				Incidence Associated with $PM_{2.5}$ Using an Annual Design Value Based on the Average of Monitor-Specific Averages** (95% Confidence Interval)			
		Percent Reduction in Incidence from Current Standards				Percent Reduction in Incidence from Current Standards			
Annual ($\mu g/m^3$)	Daily ($\mu g/m^3$)	Policy Relevant Background =3.5 $\mu g/m^3$	Cutpoint** =10 $\mu g/m^3$	Cutpoint** =15 $\mu g/m^3$	Cutpoint** =20 $\mu g/m^3$	Policy Relevant Background =3.5 $\mu g/m^3$	Cutpoint** =10 $\mu g/m^3$	Cutpoint** =15 $\mu g/m^3$	Cutpoint** =20 $\mu g/m^3$
15	65, 98th percentile value***	122 (-123 - 358) 0.0%	54 (-55 - 159) 0.0%	26 (-27 - 77) 0.0%	12 (-12 - 35) 0.0%	150 (-151 - 442) 0.0%	80 (-81 - 236) 0.0%	46 (-47 - 137) 0.0%	25 (-26 - 75) 0.0%
	40, 98th percentile value	122 (-123 - 358) 0.0%	54 (-55 - 159) 0.0%	26 (-27 - 77) 0.0%	12 (-12 - 35) 0.0%	150 (-151 - 442) 0.0%	80 (-81 - 236) 0.0%	46 (-47 - 137) 0.0%	25 (-26 - 75) 0.0%
	35, 98th percentile value	122 (-123 - 358) 0.0%	54 (-55 - 159) 0.0%	26 (-27 - 77) 0.0%	12 (-12 - 35) 0.0%	132 (-133 - 388) 12.0%	63 (-64 - 186) 21.3%	33 (-33 - 97) 28.3%	16 (-17 - 47) 36.0%
	30, 98th percentile value	111 (-112 - 325) 9.0%	45 (-45 - 131) 16.7%	20 (-20 - 58) 23.1%	8 (-9 - 24) 33.3%	111 (-112 - 325) 26.0%	45 (-45 - 131) 43.8%	20 (-20 - 58) 56.5%	8 (-9 - 24) 68.0%
	25, 98th percentile value	90 (-91 - 263) 26.2%	28 (-29 - 82) 48.1%	10 (-10 - 28) 61.5%	3 (-4 - 10) 75.0%	90 (-91 - 263) 40.0%	28 (-29 - 82) 65.0%	10 (-10 - 28) 78.3%	3 (-4 - 10) 88.0%
	65, 99th percentile value	122 (-123 - 358) 0.0%	54 (-55 - 159) 0.0%	26 (-27 - 77) 0.0%	12 (-12 - 35) 0.0%	150 (-151 - 442) 0.0%	80 (-81 - 236) 0.0%	46 (-47 - 137) 0.0%	25 (-26 - 75) 0.0%
	40, 99th percentile value	122 (-123 - 358) 0.0%	54 (-55 - 159) 0.0%	26 (-27 - 77) 0.0%	12 (-12 - 35) 0.0%	139 (-140 - 409) 7.3%	70 (-70 - 206) 12.5%	38 (-39 - 112) 17.4%	20 (-20 - 58) 20.0%
	35, 99th percentile value	120 (-121 - 352) 1.6%	53 (-53 - 154) 1.9%	25 (-26 - 74) 3.8%	11 (-12 - 33) 8.3%	120 (-121 - 352) 20.0%	53 (-53 - 154) 33.8%	25 (-26 - 74) 45.7%	11 (-12 - 33) 56.0%
	30, 99th percentile value	101 (-102 - 296) 17.2%	37 (-37 - 107) 31.5%	15 (-15 - 42) 42.3%	6 (-6 - 16) 50.0%	101 (-102 - 296) 32.7%	37 (-37 - 107) 53.8%	15 (-15 - 42) 67.4%	6 (-6 - 16) 76.0%
	25, 99th percentile value	82 (-83 - 239) 32.8%	22 (-23 - 65) 59.3%	7 (-7 - 19) 73.1%	2 (-2 - 6) 83.3%	82 (-83 - 239) 45.3%	22 (-23 - 65) 72.5%	7 (-7 - 19) 84.8%	2 (-2 - 6) 92.0%
14	40, 98th percentile value	111 (-112 - 326) 9.0%	45 (-46 - 132) 16.7%	20 (-20 - 58) 23.1%	8 (-9 - 24) 33.3%	137 (-138 - 403) 8.7%	68 (-68 - 200) 15.0%	37 (-37 - 108) 19.6%	19 (-19 - 55) 24.0%
	35, 98th percentile value	111 (-112 - 326) 9.0%	45 (-46 - 132) 16.7%	20 (-20 - 58) 23.1%	8 (-9 - 24) 33.3%	132 (-133 - 388) 12.0%	63 (-64 - 186) 21.3%	33 (-33 - 97) 28.3%	16 (-17 - 47) 36.0%
	30, 98th percentile value	111 (-112 - 325) 9.0%	45 (-45 - 131) 16.7%	20 (-20 - 58) 23.1%	8 (-9 - 24) 33.3%	111 (-112 - 325) 26.0%	45 (-45 - 131) 43.8%	20 (-20 - 58) 56.5%	8 (-9 - 24) 68.0%
	25, 98th percentile value	90 (-91 - 263) 26.2%	28 (-29 - 82) 48.1%	10 (-10 - 28) 61.5%	3 (-4 - 10) 75.0%	90 (-91 - 263) 40.0%	28 (-29 - 82) 65.0%	10 (-10 - 28) 78.3%	3 (-4 - 10) 88.0%
	40, 99th percentile value	111 (-112 - 326) 9.0%	45 (-46 - 132) 16.7%	20 (-20 - 58) 23.1%	8 (-9 - 24) 33.3%	137 (-138 - 403) 8.7%	68 (-68 - 200) 15.0%	37 (-37 - 108) 19.6%	19 (-19 - 55) 24.0%
	35, 99th percentile value	111 (-112 - 326) 9.0%	45 (-46 - 132) 16.7%	20 (-20 - 58) 23.1%	8 (-9 - 24) 33.3%	120 (-121 - 352) 20.0%	53 (-53 - 154) 33.8%	25 (-26 - 74) 45.7%	11 (-12 - 33) 56.0%
	30, 99th percentile value	101 (-102 - 296) 17.2%	37 (-37 - 107) 31.5%	15 (-15 - 42) 42.3%	6 (-6 - 16) 50.0%	101 (-102 - 296) 32.7%	37 (-37 - 107) 53.8%	15 (-15 - 42) 67.4%	6 (-6 - 16) 76.0%
	25, 99th percentile value	82 (-83 - 239) 32.8%	22 (-23 - 65) 59.3%	7 (-7 - 19) 73.1%	2 (-2 - 6) 83.3%	82 (-83 - 239) 45.3%	22 (-23 - 65) 72.5%	7 (-7 - 19) 84.8%	2 (-2 - 6) 92.0%

Alternative Standards		Incidence Associated with $PM_{2.5}$ Using an Annual Design Value Based on the Maximum of Monitor-Specific Averages** (95% Confidence Interval)				Incidence Associated with $PM_{2.5}$ Using an Annual Design Value Based on the Average of Monitor-Specific Averages** (95% Confidence Interval)			
		Percent Reduction in Incidence from Current Standards				Percent Reduction in Incidence from Current Standards			
Annual ($\mu g/m^3$)	Daily ($\mu g/m^3$)	Policy Relevant Background =3.5 $\mu g/m^3$	Cutpoint** =10 $\mu g/m^3$	Cutpoint** =15 $\mu g/m^3$	Cutpoint** =20 $\mu g/m^3$	Policy Relevant Background =3.5 $\mu g/m^3$	Cutpoint** =10 $\mu g/m^3$	Cutpoint** =15 $\mu g/m^3$	Cutpoint** =20 $\mu g/m^3$
13	40, 98th percentile value	101 (-101 - 295) 17.2%	36 (-37 - 106) 33.3%	14 (-15 - 42) 46.2%	6 (-6 - 16) 50.0%	124 (-125 - 364) 17.3%	56 (-57 - 165) 30.0%	28 (-28 - 81) 39.1%	13 (-13 - 38) 48.0%
	35, 98th percentile value	101 (-101 - 295) 17.2%	36 (-37 - 106) 33.3%	14 (-15 - 42) 46.2%	6 (-6 - 16) 50.0%	124 (-125 - 364) 17.3%	56 (-57 - 165) 30.0%	28 (-28 - 81) 39.1%	13 (-13 - 38) 48.0%
	30, 98th percentile value	101 (-101 - 295) 17.2%	36 (-37 - 106) 33.3%	14 (-15 - 42) 46.2%	6 (-6 - 16) 50.0%	111 (-112 - 325) 26.0%	45 (-45 - 131) 43.8%	20 (-20 - 58) 56.5%	8 (-9 - 24) 68.0%
	25, 98th percentile value	90 (-91 - 263) 26.2%	28 (-29 - 82) 48.1%	10 (-10 - 28) 61.5%	3 (-4 - 10) 75.0%	90 (-91 - 263) 40.0%	28 (-29 - 82) 65.0%	10 (-10 - 28) 78.3%	3 (-4 - 10) 88.0%
	40, 99th percentile value	101 (-101 - 295) 17.2%	36 (-37 - 106) 33.3%	14 (-15 - 42) 46.2%	6 (-6 - 16) 50.0%	124 (-125 - 364) 17.3%	56 (-57 - 165) 30.0%	28 (-28 - 81) 39.1%	13 (-13 - 38) 48.0%
	35, 99th percentile value	101 (-101 - 295) 17.2%	36 (-37 - 106) 33.3%	14 (-15 - 42) 46.2%	6 (-6 - 16) 50.0%	120 (-121 - 352) 20.0%	53 (-53 - 154) 33.8%	25 (-26 - 74) 45.7%	11 (-12 - 33) 56.0%
	30, 99th percentile value	101 (-101 - 295) 17.2%	36 (-37 - 106) 33.3%	14 (-15 - 42) 46.2%	6 (-6 - 16) 50.0%	101 (-102 - 296) 32.7%	37 (-37 - 107) 53.8%	15 (-15 - 42) 67.4%	6 (-6 - 16) 76.0%
	25, 99th percentile value	82 (-83 - 239) 32.8%	22 (-23 - 65) 59.3%	7 (-7 - 19) 73.1%	2 (-2 - 6) 83.3%	82 (-83 - 239) 45.3%	22 (-23 - 65) 72.5%	7 (-7 - 19) 84.8%	2 (-2 - 6) 92.0%
12	40, 98th percentile value	90 (-91 - 264) 26.2%	28 (-29 - 82) 48.1%	10 (-10 - 28) 61.5%	3 (-4 - 10) 75.0%	111 (-112 - 325) 26.0%	45 (-45 - 131) 43.8%	20 (-20 - 58) 56.5%	8 (-9 - 24) 68.0%
	35, 98th percentile value	90 (-91 - 264) 26.2%	28 (-29 - 82) 48.1%	10 (-10 - 28) 61.5%	3 (-4 - 10) 75.0%	111 (-112 - 325) 26.0%	45 (-45 - 131) 43.8%	20 (-20 - 58) 56.5%	8 (-9 - 24) 68.0%
	30, 98th percentile value	90 (-91 - 264) 26.2%	28 (-29 - 82) 48.1%	10 (-10 - 28) 61.5%	3 (-4 - 10) 75.0%	111 (-112 - 325) 26.0%	45 (-45 - 131) 43.8%	20 (-20 - 58) 56.5%	8 (-9 - 24) 68.0%
	25, 98th percentile value	90 (-91 - 263) 26.2%	28 (-29 - 82) 48.1%	10 (-10 - 28) 61.5%	3 (-4 - 10) 75.0%	90 (-91 - 263) 40.0%	28 (-29 - 82) 65.0%	10 (-10 - 28) 78.3%	3 (-4 - 10) 88.0%
	40, 99th percentile value	90 (-91 - 264) 26.2%	28 (-29 - 82) 48.1%	10 (-10 - 28) 61.5%	3 (-4 - 10) 75.0%	111 (-112 - 325) 26.0%	45 (-45 - 131) 43.8%	20 (-20 - 58) 56.5%	8 (-9 - 24) 68.0%
	35, 99th percentile value	90 (-91 - 264) 26.2%	28 (-29 - 82) 48.1%	10 (-10 - 28) 61.5%	3 (-4 - 10) 75.0%	111 (-112 - 325) 26.0%	45 (-45 - 131) 43.8%	20 (-20 - 58) 56.5%	8 (-9 - 24) 68.0%
	30, 99th percentile value	90 (-91 - 264) 26.2%	28 (-29 - 82) 48.1%	10 (-10 - 28) 61.5%	3 (-4 - 10) 75.0%	101 (-102 - 296) 32.7%	37 (-37 - 107) 53.8%	15 (-15 - 42) 67.4%	6 (-6 - 16) 76.0%
	25, 99th percentile value	82 (-83 - 239) 32.8%	22 (-23 - 65) 59.3%	7 (-7 - 19) 73.1%	2 (-2 - 6) 83.3%	82 (-83 - 239) 45.3%	22 (-23 - 65) 72.5%	7 (-7 - 19) 84.8%	2 (-2 - 6) 92.0%

*This analysis was performed using Ito (2003).

**For the cutpoints above policy relevant background, the slope of the concentration-response function has been modified based on a simple hockeystick model (see discussion in section 4.3.2.1).

***Current standards.

Note: Incidences are rounded to the nearest whole number; percents are rounded to the nearest tenth.

Table 4B-10. Sensitivity Analysis: Estimated Annual Mortality Associated with Long-Term Exposure to $PM_{2.5}$ When Alternative Standards Are Just Met, Assuming Various Cutpoint Levels -- Rollbacks to Meet Annual Standards Using Design Values Based on Maximum vs. Average of Monitor-Specific Averages*
Detroit, MI, 2003

Alternative Standards		Incidence Associated with $PM_{2.5}$ Using an Annual Design Value Based on the Maximum of Monitor-Specific Averages** (95% Confidence Interval) Percent Reduction in Incidence from Current Standards			Incidence Associated with $PM_{2.5}$ Using an Annual Design Value Based on the Average of Monitor-Specific Averages** (95% Confidence Interval) Percent Reduction in Incidence from Current Standards		
Annual ($\mu g/m^3$)	Daily ($\mu g/m^3$)	Cutpoint** =7.5 $\mu g/m^3$	Cutpoint** =10 $\mu g/m^3$	Cutpoint** =12 $\mu g/m^3$	Cutpoint** =7.5 $\mu g/m^3$	Cutpoint** =10 $\mu g/m^3$	Cutpoint** =12 $\mu g/m^3$
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%
	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%
	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%
	30, 98th percentile value	435 (151 - 757) 16.7%	185 (64 - 323) 34.4%	0 (0 - 0) 100.0%	435 (151 - 757) 41.8%	185 (64 - 323) 65.4%	0 (0 - 0) 100.0%
	25, 98th percentile value	270 (94 - 468) 48.3%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%	270 (94 - 468) 63.9%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
	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%
	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%
	35, 99th percentile value	507 (176 - 884) 2.9%	266 (92 - 465) 5.7%	23 (8 - 40) 43.9%	507 (176 - 884) 32.1%	266 (92 - 465) 50.3%	23 (8 - 40) 92.9%
	30, 99th percentile value	356 (124 - 619) 31.8%	97 (34 - 168) 65.6%	0 (0 - 0) 100.0%	356 (124 - 619) 52.3%	97 (34 - 168) 81.9%	0 (0 - 0) 100.0%
	25, 99th percentile value	207 (72 - 358) 60.3%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%	207 (72 - 358) 72.3%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
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%
	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%
	30, 98th percentile value	435 (151 - 757) 16.7%	185 (64 - 323) 34.4%	0 (0 - 0) 100.0%	435 (151 - 757) 41.8%	185 (64 - 323) 65.4%	0 (0 - 0) 100.0%
	25, 98th percentile value	270 (94 - 468) 48.3%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%	270 (94 - 468) 63.9%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
	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%
	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%
	30, 99th percentile value	356 (124 - 619) 31.8%	97 (34 - 168) 65.6%	0 (0 - 0) 100.0%	356 (124 - 619) 52.3%	97 (34 - 168) 81.9%	0 (0 - 0) 100.0%
	25, 99th percentile value	207 (72 - 358) 60.3%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%	207 (72 - 358) 72.3%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%

Alternative Standards		Incidence Associated with $PM_{2.5}$ Using an Annual Design Value Based on the Maximum of Monitor-Specific Averages** (95% Confidence Interval) Percent Reduction in Incidence from Current Standards			Incidence Associated with $PM_{2.5}$ Using an Annual Design Value Based on the Average of Monitor-Specific Averages** (95% Confidence Interval) Percent Reduction in Incidence from Current Standards		
Annual ($\mu g/m^3$)	Daily ($\mu g/m^3$)	Cutpoint** =7.5 $\mu g/m^3$	Cutpoint** =10 $\mu g/m^3$	Cutpoint** =12 $\mu g/m^3$	Cutpoint** =7.5 $\mu g/m^3$	Cutpoint** =10 $\mu g/m^3$	Cutpoint** =12 $\mu g/m^3$
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%
	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%
	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%
	25, 98th percentile value	270 (94 - 468) 48.3%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%	270 (94 - 468) 63.9%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
	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%
	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%
	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%
	25, 99th percentile value	207 (72 - 358) 60.3%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%	207 (72 - 358) 72.3%	0 (0 - 0) 100.0%	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%
	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%
	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%
	25, 98th percentile value	270 (94 - 468) 48.3%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%	270 (94 - 468) 63.9%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%
	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%
	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%
	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%
	25, 99th percentile value	207 (72 - 358) 60.3%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%	207 (72 - 358) 72.3%	0 (0 - 0) 100.0%	0 (0 - 0) 100.0%

*This analysis was performed using Pope et al. (2002) -- ACS extended.

**For the cutpoints above policy relevant background, the slope of the concentration-response function has been modified based on a simple hockeystick model (see discussion in section 4.3.2.1).

***Current standards.

Note: Incidences are rounded to the nearest whole number; percents are rounded to the nearest tenth.

NOTE: Consistent with Figures 5-1(a), (b), and (c) and Figures 5-2(a), (b), (c), and (d) that appear in Chapter 5, the figures in this Appendix are based on risk reductions estimated for various combinations of alternative standards, including annual standards of 15, 14, 13, and 12 $\mu g/m^3$ and 24-hour standards of 65, 40, 35, 30, and 25 $\mu g/m^3$. Since in most cases the estimated risk reductions were the same or nearly so for alternative 24-hour standards of 65 and 40 $\mu g/m^3$, the interim points between these two levels were filled in so as to better depict the 3-dimensional surface of risk reductions. In those cases involving the 99^{th} percentile form of the 24-hour standard where there were appreciable differences in the estimated risk reductions between alternative 24-standards of 65 and 40 $\mu g/m^3$, the interim points were not filled in, since the shape of the surface between these points has not been calculated.

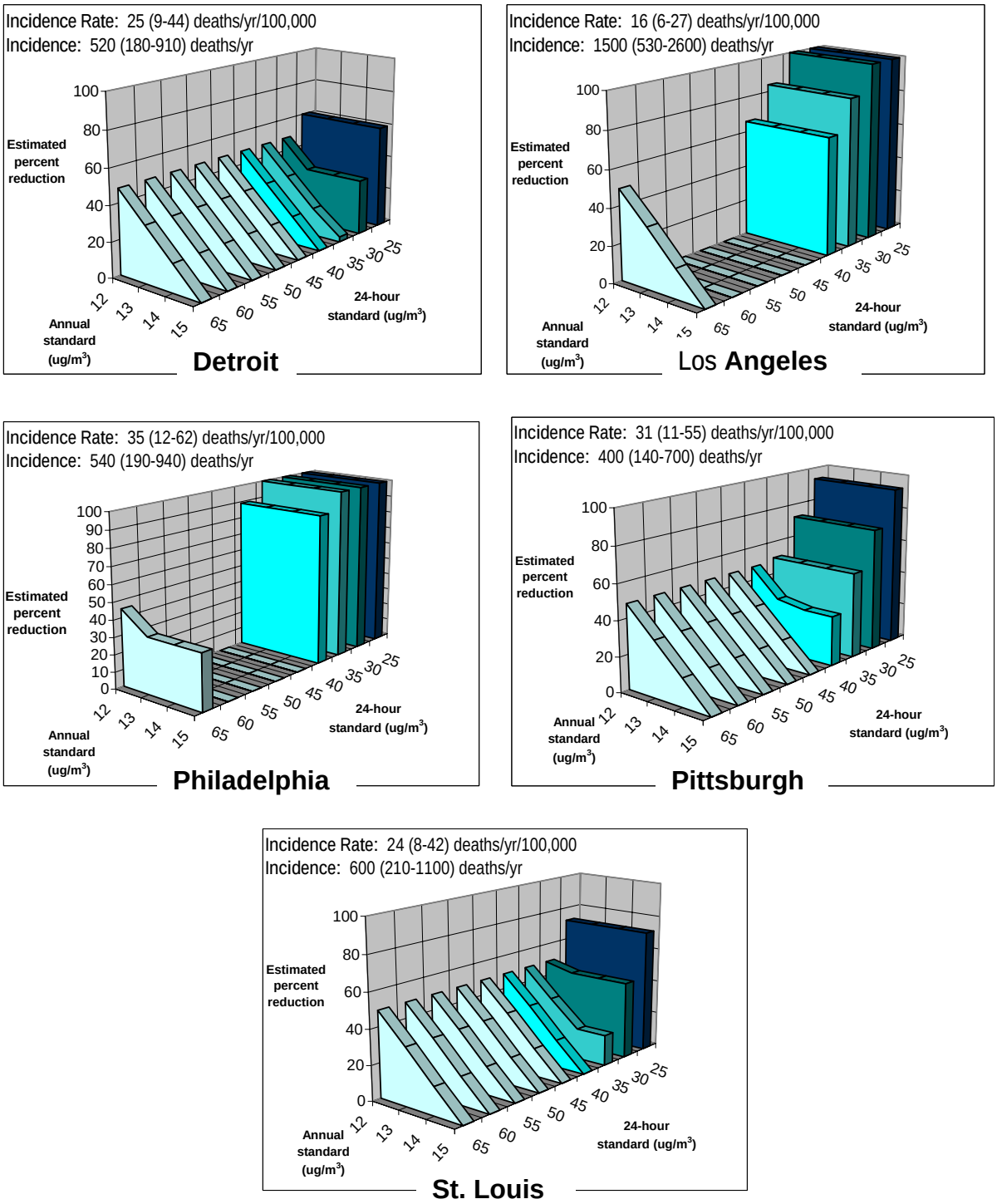


Figure 5A-1(a) Estimated percent reduction in $PM_{2.5}$-related long-term mortality risk for alternative standards *(99th percentile form)* relative to risk associated with meeting current standards (based on assumed cutpoint of 7.5 $\mu g/m^3$). Risk associated with meeting current $PM_{2.5}$ standards, based on ACS extended study, is shown in figures in terms of estimated annual incidence rate and annual incidence (and 95% confidence ranges).

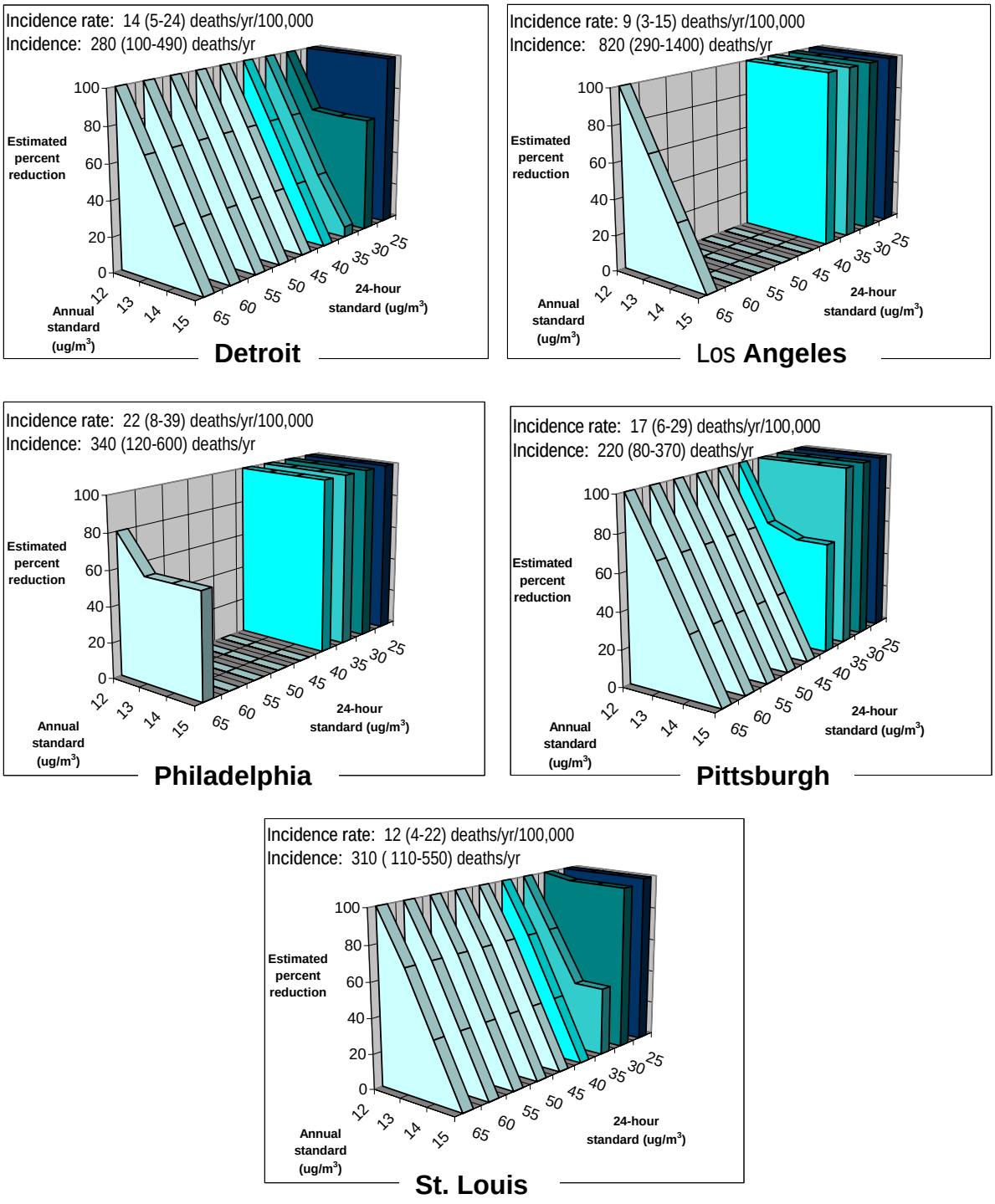


Figure 5A-1(b) Estimated percent reduction in $PM_{2.5}$-related long-term mortality risk for alternative standards *(99th percentile form)* relative to risk associated with meeting current standards (based on assumed cutpoint of *10 µg/m3*). Risk associated with meeting current $PM_{2.5}$ standards, based on ACS extended study, is shown in figures in terms of estimated annual incidence rate and annual incidence (and 95% confidence ranges).

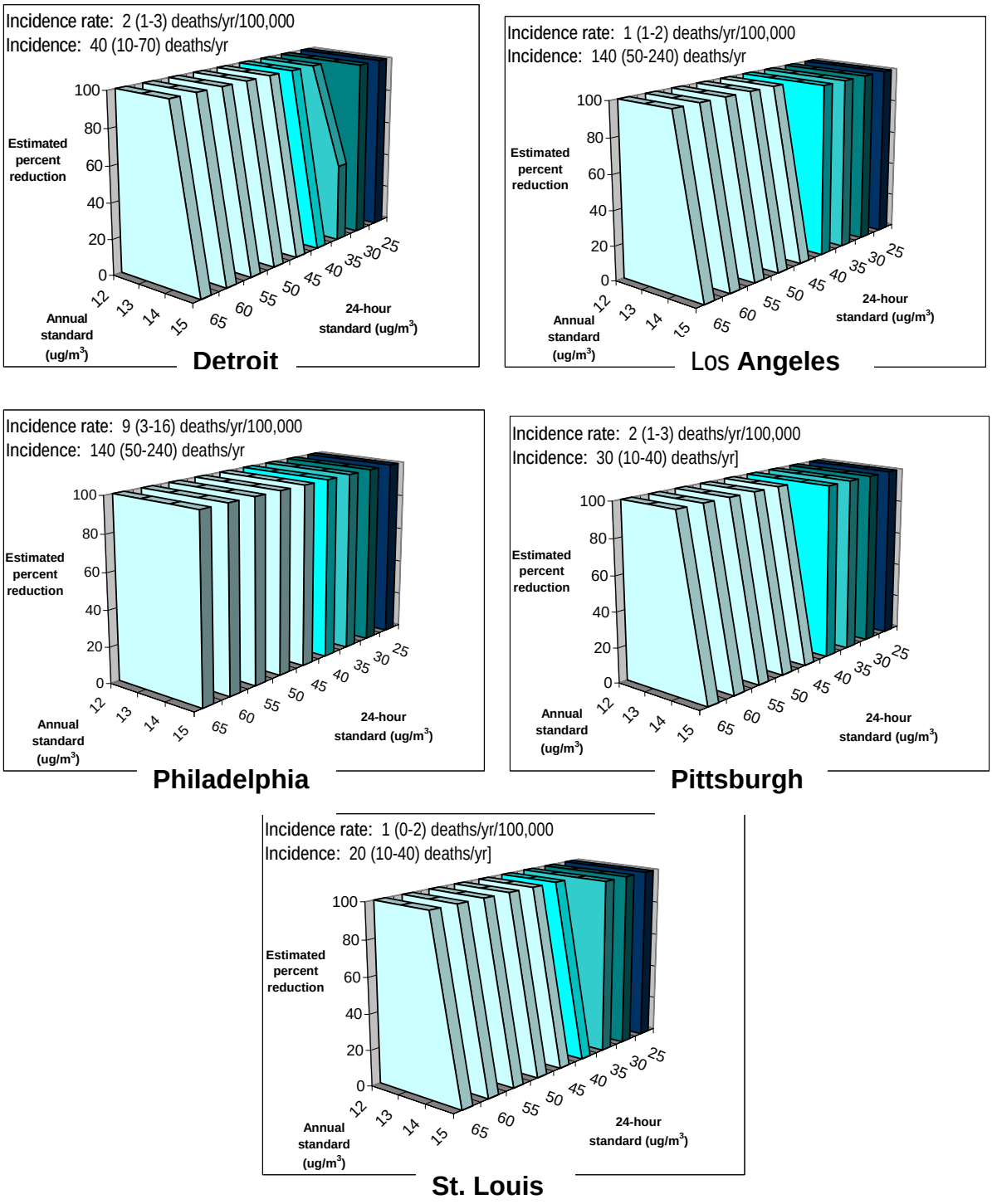


Figure 5A-1(c) Estimated percent reduction in $PM_{2.5}$-related long-term mortality risk for alternative standards *(99th percentile form)* relative to risk associated with meeting current standards (based on assumed cutpoint of *12 µg/m³*). Risk associated with meeting current $PM_{2.5}$ standards, based on ACS extended study, is shown in figures in terms of estimated annual incidence rate and annual incidence (and 95% confidence ranges).

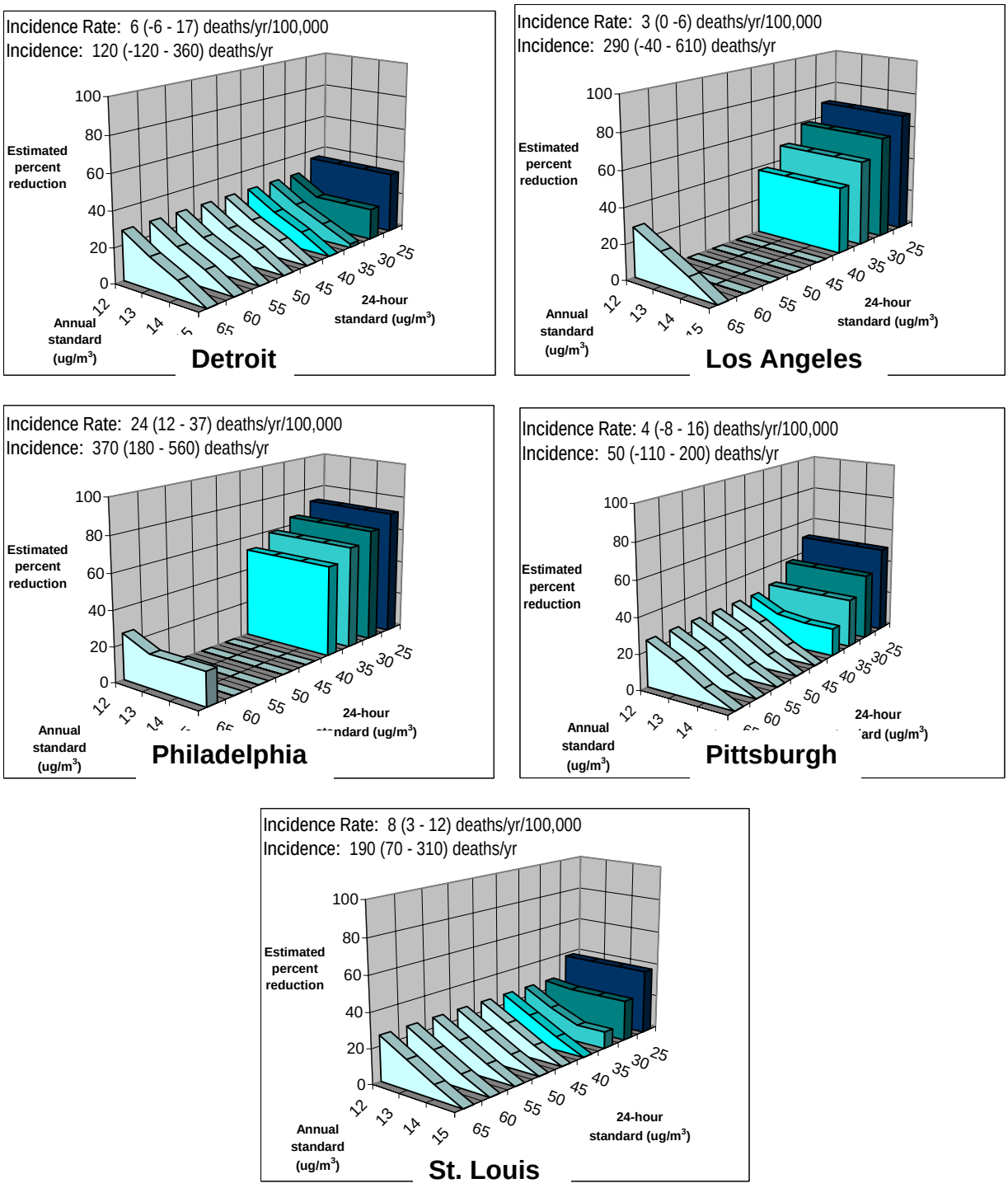


Figure 5A-2(a) Estimated percent reduction in $PM_{2.5}$-related short-term mortality risk for alternative standards (*99th percentile form*) relative to risk associated with meeting current standards (based on assumed cutpoint equal to policy-relevant background). Risk associated with meeting current $PM_{2.5}$ standards is shown in figures in terms of estimated annual incidence rate and annual incidence (and 95% confidence ranges). Estimated policy-relevant background is 3.5 µg/m^3 in eastern cities and 2.5 µg/m^3 in western cities.

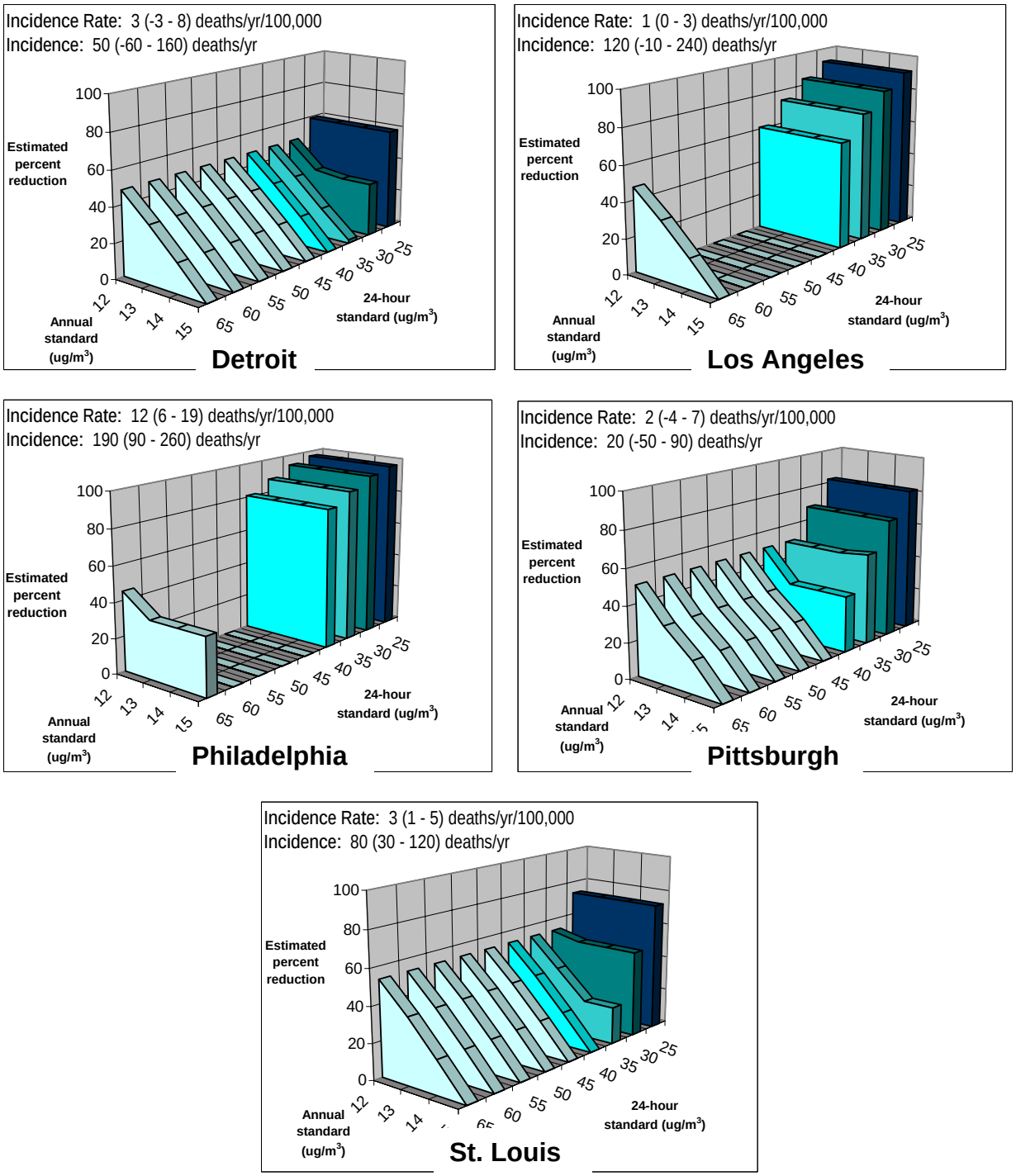


Figure 5A-2(b) Estimated percent reduction in $PM_{2.5}$-related short-term mortality risk for alternative standards *(99th percentile form)* relative to risk associated with meeting current standards (based on an assumed cutpoint of 10 µg/m^3). Risk associated with meeting current $PM_{2.5}$ standards is shown in figures in terms of estimated annual incidence rate and annual incidence (and 95% confidence ranges).

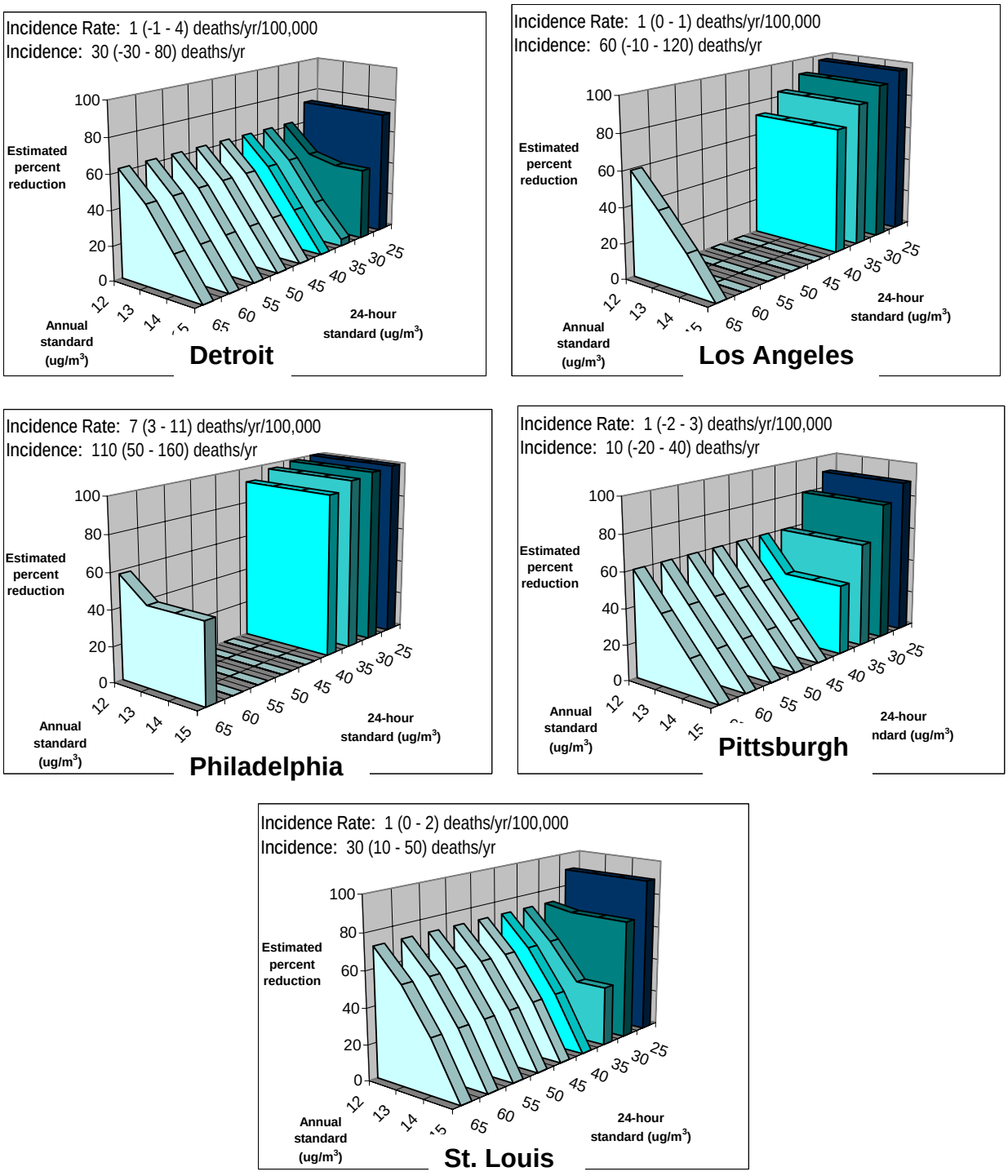


Figure 5A-2(c) Estimated percent reduction in $PM_{2.5}$-related short-term mortality risk for alternative standards *(99th percentile form)* relative to risk associated with meeting current standards (based on an assumed cutpoint of 15 µg/m^3). Risk associated with meeting current $PM_{2.5}$ standards is shown in figures in terms of estimated annual incidence rate and annual incidence (and 95% confidence ranges).

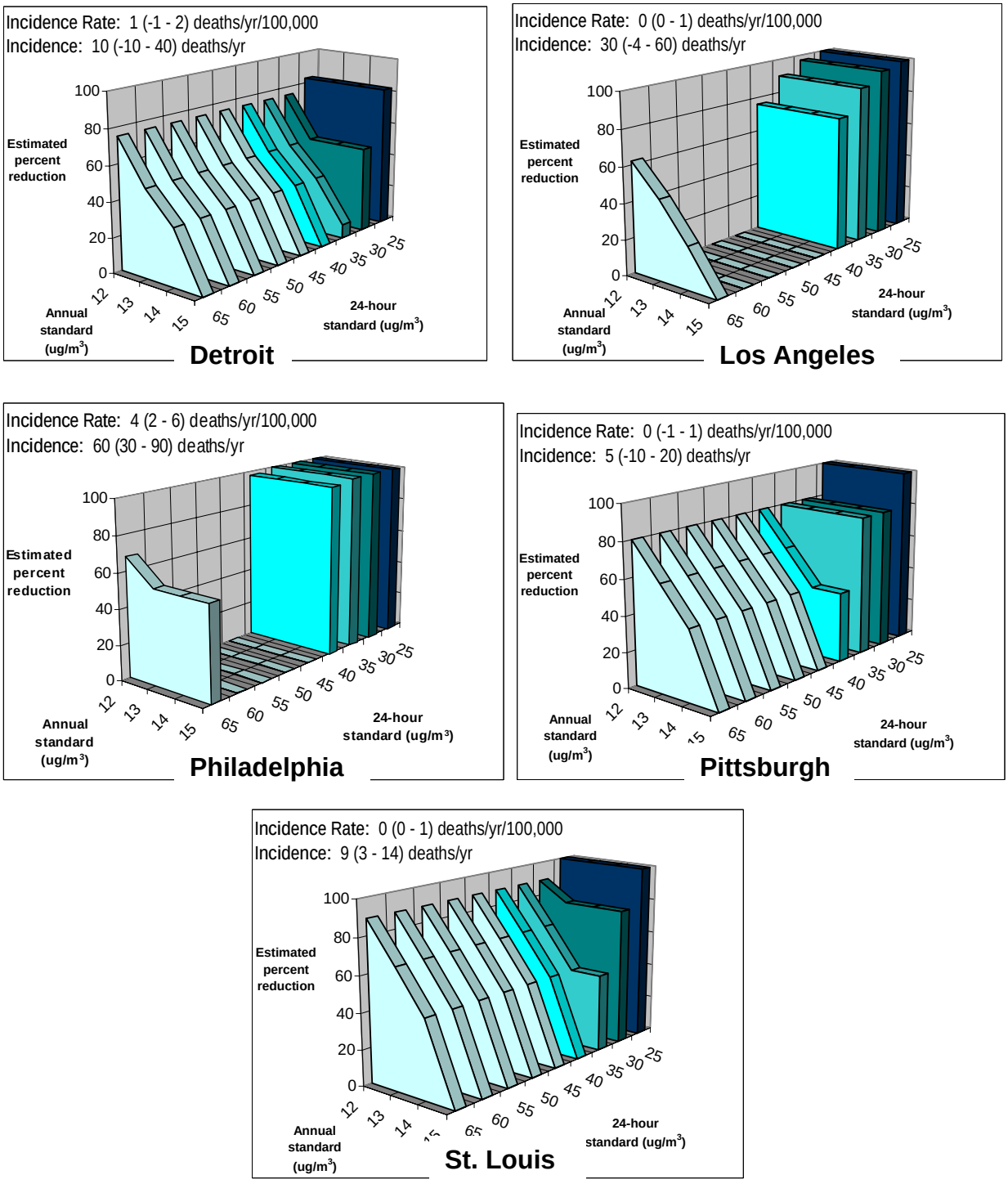


Figure 5A-2(d) Estimated percent reduction in $PM_{2.5}$-related short-term mortality risk for alternative standards *(99th percentile form)* relative to risk associated with meeting current standards (based on an assumed cutpoint of 20 µg/m^3). Risk associated with meeting current $PM_{2.5}$ standardsis shown in figures in terms of estimated annual incidence rate and annual incidence (and 95% confidence ranges).

Table 5B-1(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 ($\mu g/m_3$)	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
13 / 65	40 (55)	47	40	76	4	5	7	67	0
13 / 50	40 (56)	47	40	76	4	5	10	67	0
13 / 45	41 (57)	47	40	76	4	10	12	67	0
13 / 40	42 (58)	47	40	76	4	10	19	67	0
13 / 35	45 (62)	53	40	77	4	10	36	67	7
13 / 30	57 (74)	78	43	90	8	19	51	80	13
13 / 25	78 (86)	98	77	99	51	43	65	80	13
12 / 65	54 (66)	70	61	89	12	5	12	67	0
12 / 50	54 (66)	70	61	89	12	5	12	67	0
12 / 45	54 (67)	70	61	89	12	10	14	67	0
12 / 40	55 (68)	70	61	89	12	10	20	67	0
12 / 35	58 (71)	70	61	89	12	10	36	67	7
12 / 30	64 (78)	84	62	94	14	19	51	80	13
12 / 25	79 (86)	98	78	99	51	43	65	80	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.

Table 5B-1(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/m_3)	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/m_3)	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.

Table 5B-2(a) Percent of counties with monitors (and percent of population in counties with monitors) not likely to meet alternative 24-hour (98th percentile form) $UPM_{10-2.5}$ standards

Alternative Levels	Percent of counties, total and by region, (and total percent population) not likely to meet alternative 24-hour (98th percentile form) $PM_{10-2.5}$ standards or current PM_{10} standards*								
	Total Counties (population)	**Northeast**	**Southeast**	**Industrial Midwest**	**Upper Midwest**	**Southwest**	**Northwest**	**Southern CA**	**Outside Regions****
Number of counties with monitors (Population, in thousands)	***259 (141,859)***	***44***	***60***	***57***	***18***	***13***	***45***	***15***	***7***
70	7 (9)	2	3	4	0	46	4	33	29
65	9 (11)	2	3	5	6	46	9	40	29
60	12 (16)	2	5	7	6	62	13	40	43
55	13 (18)	5	5	7	17	62	13	40	43
50	16 (27)	5	7	9	22	62	16	53	57

* 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.

Table 5B-2(b) Percent of counties with monitors (and percent of population in counties with monitors) not likely to meet alternative 24-hour (99th percentile form) $UPM_{10-2.5}$ standards

Alternative Levels	Percent of counties, total and by region, (and total percent population) not likely to meet alternative 24-hour (99th percentile form) $PM_{10-2.5}$ standards or current PM_{10} standards*								
	Total Counties (population)	**Northeast**	**Southeast**	**Industrial Midwest**	**Upper Midwest**	**Southwest**	**Northwest**	**Southern CA**	**Outside Regions****
Number of counties with monitors (Population, in thousands)	***259 (141,859)***	***44***	***60***	***57***	***18***	***13***	***45***	***15***	***7***
85	8 (10)	7	2	4	6	46	11	20	14
80	10 (11)	7	3	4	6	54	11	20	29
75	12 (14)	7	5	5	6	54	11	40	29
70	13 (15)	7	7	5	17	54	13	40	29
65	16 (19)	9	8	7	33	54	16	40	43
60	19 (27)	11	10	7	39	62	16	60	43

* 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.

Table 5B-2(c) Percent of counties with monitors (and percentage of population in counties with monitors) not likely to meet current PM_{10} standards

Database	Percent of counties, total and by region, (and total percent population) not meeting the current PM_{10} standards								
	Total Counties (population)	**Northeast**	**Southeast**	**Industrial Midwest**	**Upper Midwest**	**Southwest**	**Northwest**	**Southern CA**	**Outside Regions****
***All PM_{10} sites** : [Number of counties with monitors (Population, in thousands)]**	***585 (170,118)***	***84***	***120***	***115***	***52***	***33***	***142***	***18***	***21***
Percent violating	8 (13)	0	3	3	6	27	10	61	10
***PM_{10} sites that meet 'urban' criteria:** [Number of counties with monitors (Population, in thousands)]	***309 (153,546)***	***59***	***70***	***67***	***21***	***17***	***50***	***15***	***10***
Percent violating	6 (12)	0	1	3	0	29	4	53	10
***Urban PM_{10} sites, also$PM_{10-2.5}$:** [Number of counties with monitor (Population, in thousands)]	***259 (141,859)***	***44***	***60***	***57***	***18***	***13***	***45***	***sp***	***7***
Percent violating	7 (11)	0	2	4	0	38	2	47	14

* Based on official EPA design values for 2001-2003; see http://epa.gov/airtrends/values.html.

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

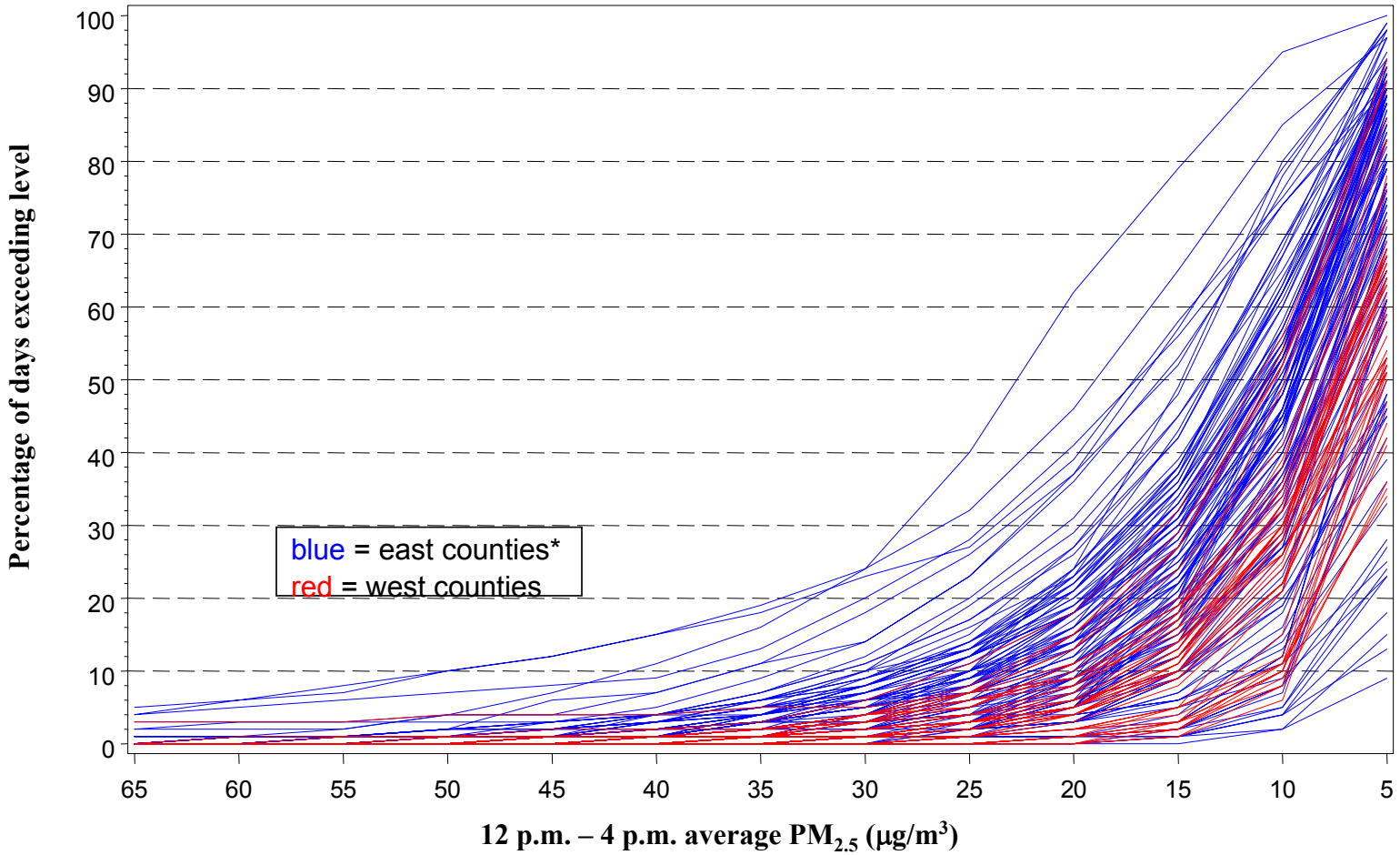


Figure 7A-1. Estimated exceedances (%) of various $PM_{2.5}$ levels for 12 p.m. – 4 p.m. based on daily county maximum, 2001-2003.

*** Figure 7A-1 can be seen in color at: http://www.epa.gov/ttn/naaqs/standards/pm/s_pm_cr_sp.html**

Source: Schmidt et al. (2005)

Table 7A-1. Predicted percent of counties with monitors (and percent of population in counties with monitors) not likely to meet alternative 4-hour (12 p.m. - 4 p.m.) $PM_{2.5}$ secondary standards.

Alternative Forms and Levels of Secondary $PM_{2.5}$ Standard	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**
Number of counties with monitors (Population, in thousands)	***168 (78,419)***	***33***	***45***	***30***	***16***	***14***	***25***	***3***	***2***
$PM_{2.5}$ standard levels and forms:									
20 µg/m^3, 92nd percentile	51 (67)	76	36	90	38	29	8	100	100
25 µg/m^3, 92nd percentile	27 (46)	52	16	53	0	7	4	100	0
30 µg/m^3, 92nd percentile	8 (17)	15	0	17	0	7	0	67	0
20 µg/m^3, 95th percentile	70 (83)	88	73	97	50	50	24	100	100
25 µg/m^3, 95th percentile	47 (67)	79	31	87	19	29	4	100	100
30 µg/m^3, 95th percentile	24 (43)	52	7	47	0	14	4	100	0
20 µg/m^3, 98th percentile	85 (96)	100	100	100	63	57	48	100	100
25 µg/m^3, 98th percentile	70 (81)	94	62	100	50	57	28	100	100
30 µg/m^3, 98th percentile	56 (73)	85	38	90	19	57	24	100	100

* Based on 2001-2003 data for sites with at least 1 year of complete data. Completeness criteria (per year) = Minimum of 3 hours per day (in 4-hour 12-4pm window), 275+ days per year. 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.

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON D.C. 20460

June 6, 2005

EPA-SAB-CASAC-05-007

OFFICE OF THE ADMINISTRATOR
SCIENCE ADVISORY BOARD

Honorable Stephen L. Johnson
Administrator
U.S. Environmental Protection Agency
1200 Pennsylvania Avenue, NW
Washington, DC 20460

Subject: Clean Air Scientific Advisory Committee (CASAC) Particulate Matter (PM) Review Panel's Peer Review of the Agency's *Review of the National Ambient Air Quality Standards for Particulate Matter: Policy Assessment of Scientific and Technical Information* (Second Draft PM Staff Paper, January 2005); and *Particulate Matter Health Risk Assessment for Selected Urban Areas: Second Draft Report* (Second Draft PM Risk Assessment, January 2005)

Dear Administrator Johnson:

EPA's Clean Air Scientific Advisory Committee (CASAC), supplemented by subject-matter-expert Panelists — collectively referred to as the CASAC Particulate Matter (PM) Review Panel ("Panel") — met in a public meeting held in Durham, NC, on April 5-6, 2005, to conduct a peer review of subject documents. The current Panel roster is found in Appendix A of this report.

This meeting was a continuation of the CASAC PM Review Panel's peer review of the *Review of the National Ambient Air Quality Standards for Particulate Matter: Policy Assessment of Scientific and Technical Information (*First Draft PM Staff Paper, August 2003*)* and a related draft technical report, Particulate Matter Health Risk Assessment for Selected Urban Areas (First Draft PM Risk Assessment, August 2003*).* The previous draft of the PM Staff Paper was a preliminary version since the Panel has not yet finished its review of the Air Quality Criteria Document (AQCD) for PM (which was completed in October 2004). In addition, further risk analyses and analyses of alternative forms of the PM standards were included in the Second Draft PM Staff Paper and Second Draft PM Risk Assessment. The charge questions provided to the Panel by EPA are found in Appendix B to this report. Panelists' individual review comments are provided in Appendix C of this report.

In its peer review of the Second Draft of the PM Staff Paper, most of the members of the CASAC PM Review Panel found the document was generally well-written and scientifically

well-reasoned for all but the short term primary $PM_{10-2.5}$ standard. A majority of the members of the Panel were in agreement with the following: the primary $PM_{2.5}$ 24-hour and annual PM national ambient air quality standards (NAAQS) should be modified to provide increased public health protection. Although the evidence for a standard for coarse-mode particles was weaker than for the $PM_{2.5}$, the Panel agreed that a 24-hour NAAQS for $PM_{10-2.5}$ was appropriate, especially in urban areas, with caveats to make exceptions for those types of rural dusts thought to have low toxicity. The Panel recommends that the Agency staff expand and strengthen the discussion of the exposure index (size-range plus composition and/or source) and the monitoring strategy to be used for the coarse-mode NAAQS, as well as the degree of public health protection against thoracic coarse PM expected relative to the protection afforded by the current PM_{10} short-term NAAQS. As discussed below, the CASAC PM Review Panel will need to review the final version of the PM Staff Paper before providing a final opinion to EPA on the adequacy of a short-term $PM_{10-2.5}$ NAAQS.

The approach used to set secondary NAAQS to protect the environment was considered appropriate, but it was strongly recommended that, in the future, Agency staff also give serious consideration to a shift to the European approach of critical loads to protect vegetation and ecosystems in the U.S. In addition, most of the Panel supported Agency staff recommendations regarding a standard to address the issue of urban visibility impairment.

1. Background

The CASAC, comprised of seven members appointed by the EPA Administrator, was established under section 109(d)(2) of the Clean Air Act (CAA or "Act") (42 U.S.C. § 7409) as an independent scientific advisory committee, in part to provide advice, information and recommendations on the scientific and technical aspects of issues related to air quality criteria and NAAQS under sections 108 and 109 of the Act. Section 109(d)(1) of the CAA requires that EPA carry out a periodic review and revision, where appropriate, of the air quality criteria and the NAAQS for "criteria" air pollutants such as PM. The CASAC, which is administratively located under EPA's Science Advisory Board (SAB) Staff Office, is a Federal advisory committee chartered under the Federal Advisory Committee Act (FACA), as amended, 5 U.S.C., App. The CASAC PM Review Panel is comprised of the seven members of the chartered (statutory) Clean Air Scientific Advisory Committee, supplemented by fifteen technical experts.

Under section 108 of the CAA, the Agency is required to establish NAAQS for each pollutant for which EPA has issued criteria, including PM. Section 109(d) of the Act subsequently requires periodic review and, if appropriate, revision of existing air quality criteria to reflect advances in scientific knowledge on the effects of the pollutant on public health and welfare. EPA is also to revise the NAAQS, if appropriate, based on the revised criteria. The purpose of the Second Draft PM Staff Paper is to evaluate the policy implications of the key scientific and technical information contained in a related document, EPA's revised PM AQCD (October 2004), and to identify critical elements that EPA believes should be considered in the review of the PM NAAQS. The Staff Paper for PM is intended to "bridge the gap" between the scientific review contained in the PM AQCD and the public health and welfare policy judgments required of the Administrator in reviewing the PM NAAQS.

This Second Draft PM Staff Paper is based on the information in the final PM AQCD, which had been the subject of review by the CASAC PM Review Panel since October 1999. (The report from the Panel's final meeting to review the PM AQCD, dated October 4, 2004, is posted on the SAB Web Site at: http://www.epa.gov/sab/pdf/casac05001.pdf. The Agency subsequently announced the availability of a final document, *Air Quality Criteria for Particulate Matter* (EPA/600/P-99/002aF, EPA/600/P-99/002bF) on October 29, 2004.) In addition, the Second Draft PM Staff Paper builds upon the First Draft PM Staff Paper, which was the subject of review by the CASAC PM Review Panel held on November 12-13, 2003. The report from the Panel's previous meeting to review these draft documents, dated February 18, 2004, is posted on the SAB Web Site at: http://www.epa.gov/sab/pdf/casac_04004.pdf. The Second Draft PM Staff Paper and the Second Draft PM Risk Assessment were made available for public review and comment on January 31, 2005 by EPA's Office of Air Quality Planning and Standards (OAQPS), within the Office of Air and Radiation (OAR). The Second Draft PM Risk Assessment, which builds upon the Agency's First Draft PM Risk Assessment, describes the methodology and presents the results from an updated PM health risk assessment for health risks associated with exposure to fine and thoracic coarse particles in a number of U.S. cities.

2. CASAC PM Review Panel's Peer Review of the Second Draft PM Staff Paper and Second Draft PM Risk Assessment

After reviewing the Second Draft PM Staff Paper and written comments from the public, and after hearing public comments at the meeting, a majority of the members of the CASAC PM Review Panel were in agreement with the following: the primary $PM_{2.5}$ 24-hour and annual NAAQS should be modified to provide increased public health protection. The evidence for a NAAQS for coarse mode particles is weaker than for $PM_{2.5}$. The Panel agreed, however, that a 24-hour NAAQS for $PM_{10-2.5}$ is appropriate, especially in urban areas and with caveats to make exceptions for those types of rural dusts thought to have low toxicity. Before the Panel renders its final recommendation concerning a daily $PM_{10-2.5}$ standard, the Panel recommends that the Agency staff expand and strengthen the discussion of the exposure index (size-range plus composition and/or source) and the monitoring strategy to be used for this standard, as well as the degree of public health protection expected relative to the protection against thoracic coarse PM afforded by the current PM_{10} short-term NAAQS. Accordingly, after the Panel has reviewed the Final Staff Paper and Risk Assessment for Particulate Matter following its issuance on June 30, 2005, the Panel will meet again this summer via a public teleconference to consider the final Staff Paper's recommendations concerning the setting of a coarse PM standard. Subsequent to the Panel's teleconference meeting, we will send you a separate letter providing the Panel's recommendations concerning $PM_{10-2.5}$ as an indicator together with our views on the averaging time, statistical form, and level of any potential daily $PM_{10-2.5}$ standard.

The approach used to set secondary standards to protect the environment was considered appropriate, but it was strongly recommended that, in the future, Agency staff give serious consideration to the European approach of critical loads to protect vegetation and ecosystems in the U.S. In addition, most of the Panel supported Agency staff recommendations regarding a standard to address the issue of urban visibility impairment.

In its peer review of the Second Draft of the PM Staff Paper, most of the members of the CASAC PM Review Panel found the document was generally well-written and scientifically

well-reasoned. The following represent summaries of advice and recommendations of the Panel in response to the charge questions provided by EPA, which are found in Appendix B to this report. More detailed responses are provided in the individual review comments of each member of the Panel included in Appendix C to this report.

The CASAC PM Review Panel has reached agreement on the following synopsis of advice and recommendations for the Agency:

AIR QUALITY

Chapter 2: **Characterization of Ambient PM**

Chapter 2 of the Second Draft PM Staff Paper was considered well-written, presenting an accurate and concise summary of Chapters 2, 3, and 5 of the PM Air Quality Criteria Document. The chapter was acceptable to the Panel reviewers as written, but some improvements were suggested in two areas. In the area of measurement methods, the Panel thought there should be more discussion of continuous PM monitoring methods in light of the recommended secondary fine particle standard based on 4- to 8-hour concentration averages and the likely availability of a continuous coarse particle monitor. A more quantitative characterization of PM mass measurement errors could be presented, especially for $PM_{10-2.5}$. Interest was expressed in a discussion of alternative PM indicators for future NAAQS considerations related to the source of the PM, especially for the potentially more toxic portion of coarse PM. In the area of health and visibility assessments, concern was expressed that spatial gradients near major arterials and other urban sources are not adequately addressed. It was suggested that spatial heterogeneity within a city might better be characterized in terms of departures of individual sites from the metropolitan average, in place of this draft's summary statistics of between-sites comparisons. Some members of the Panel expressed concerns about the policy-relevant background (PRB) estimates. The true background is not observable and is effectively unknowable. As indicated in the summary of Chapter 5 comments, alternative standards should be analyzed in ways that are insensitive to estimates of PRB.

HEALTH-BASED STANDARDS

Chapter 3: **Policy-Relevant Assessment of Health Effects Evidence**

Chapter 3 addresses each of the health effects issues relevant to PM NAAQS reconsideration. Agency staff have adequately reviewed advances in understanding effects from studies conducted subsequent to the 1997 NAAQS, as summarized in the latest PM AQCD. Overall, EPA staff have done a reasonably good job of summarizing the health effects basis for considering revised or new PM standards. However, there are instances where the summary of findings and their interpretation are overstated (see individual Panel member review comments, particularly on pages C-82 and C-83). Specifically, there was confusion over strength of association versus strength of evidence, between confounding and effect modification, and between temporality and lag structure. There are some areas where Agency staff have either over-interpreted or overstated the extent to which the health data support a particular PM indicator variable. These problems can be addressed if EPA staff give heed to the individual

comments of the CASAC PM Review Panel when revising the chapter. The discussion of the effect of co-pollutants in interpreting the results of PM health studies would benefit from a clearer discussion of EPA staff's approach to interpreting quantitative results from multi-pollutant studies.

Numerous epidemiological studies that are reviewed in this chapter have shown statistically significant associations between the concentrations of ambient air $PM_{2.5}$ and PM_{10} (including levels that are lower than the current PM NAAQS) and excess mortality and morbidity. Furthermore, the evidence presented indicates that the effects appear to be robust, in that inclusion of other environmental variables in regression analyses has not been found to materially affect the associations of the adverse health effects with ambient PM concentrations. On the other hand, the scientific evidence included in the PM Air Quality Criteria Document and draft Staff Paper provides substantially less data derived from controlled exposure studies in humans or experimental animals; or from studies of biological mechanisms in animals *in vivo* or cells and tissues *in vitro*, to support the biological plausibility of the effects of the relatively low concentrations found in the human population studies. In the case of controlled human studies, this appears to be due to the inherent limitations of such studies, which are largely confined to young, healthy subjects exposed for short time periods and the examination of mild, reversible effects. In the case of animal studies, it may be due to not having adequate animal models of human disease processes and exposures to individual chemical agents rather than realistic mixtures. Both types of studies may be inadequate to represent the real-world situation of susceptible subpopulations of humans undergoing long-term exposures and occasional peak levels of complex mixtures of PM, associated surface coatings of reactive chemicals, and gaseous co-pollutants. This apparent discontinuity needs to be addressed in future research.

The current health effects data base for coarse mode particles ($PM_{10-2.5}$) is relatively weak. Few epidemiology studies have been conducted where $PM_{10-2.5}$ was measured directly as opposed to obtaining an estimate of this indicator variable by subtracting data from collocated $PM_{2.5}$ and PM_{10} monitors. There is limited evidence that $PM_{10-2.5}$ may be related to cardiovascular mortality as well as to hospital admissions for respiratory diseases. The few controlled human studies that have been conducted with concentrated ambient particles have not shed any light on the morbidity findings from epidemiological studies. Moreover, animal toxicological studies using coarse mode particles are virtually nonexistent; they are difficult to perform because rodents are obligate nose breathers and thus few of these particles penetrate to the lungs. A further complication with current epidemiological studies of the health effects of $PM_{10-2.5}$ is that most have been conducted in urban areas, and because coarse mode particles from urban and rural areas may be markedly different, extrapolating these findings to rural settings may be difficult. Considerably more research with $PM_{10-2.5}$ is needed.

Chapter 4: **Characterization of Health Risks**

One major concern with the current version of the chapter is the clarity of presentation. Readers need to struggle through dense prose and jargon-ridden text to identify key aspects of the methods and findings. Key terms are sometimes used incorrectly or inconsistently across the chapter. The chapter could be substantially shortened, and redundancies need to be addressed. Figure 4-1 provides an overall framework for the risk assessment that could be used to shape the

chapter. We suggest that the chapter refer to it repetitively as the risk assessment methods and findings are described. Subheadings, such as "assumptions" and "sensitivity analyses," might be more effectively used to guide the reader through the individual sections of the chapter.

A second concern is with methodological issues. The issue of the selection of concentration-response (C-R) relationships based on locally-derived coefficients needs more discussion. The Panel did not agree with EPA staff in calculating the burden of associated incidence in their risk assessment using either the predicted background or the lowest measured level (LML) in the utilized epidemiological analysis. The available epidemiological database on daily mortality and morbidity does not establish either the presence or absence of threshold concentrations for adverse health effects. Thus, in order to avoid emphasizing an approach that assumes effects that extend to either predicted background concentrations or LML, and to standardize the approach across cities, for the purpose of estimating public health impacts, the Panel favored the primary use of an assumed threshold of 10 $\mu g/m^3$. The original approach of using background or LML, as well as the other postulated thresholds, could still be used in a sensitivity analysis of threshold assumptions.

The analyses in this chapter highlight the impact of assumptions regarding thresholds, or lack of threshold, on the estimates of risk. The uncertainty associated with threshold or nonlinear models needs more thorough discussion. A major research need is for more work to determine the existence and level of any thresholds that may exist or the shape of nonlinear concentration-response curves at low levels of exposure that may exist, and to reduce uncertainty in estimated risks at the lowest PM concentrations.

Chapter 5: **Staff Conclusions and Recommendations on Primary PM NAAQS**

The Panel had the following advice and recommendations for the PM 2.5 standard:

The tack taken by EPA staff in recommending a suite of standards for $PM_{2.5}$ by using both an evidence-based and a risk-based approach, while necessarily *ad hoc*, was felt to be reasonable. Most Panel members favored continued use of the 98th percentile form because it is more robust than the 99th percentile form and therefore would provide more stability to prevent areas from bouncing in and out of attainment from year to year. Some concern was expressed as to whether EPA staff would exclude days on which natural phenomena such as forest fires distort the distribution. The Panel felt that such days should be eliminated before standard compliance is assessed. The link between the percentile form and the exposure level chosen is well-illustrated in the type of three-dimensional figures created by Dr. Miller at the April meeting (based on the data in Figure 5-2 in the 2nd draft PM Staff Paper), which were endorsed by the Panel and later provided in expanded form by OAQPS staff. The Panel endorses the inclusion of these types of figures in the Staff Paper. It would be helpful if reductions in risk associated with different regulatory options were expressed in the form of absolute numbers normalized to a fixed population size, in addition to those already expressed as percentage reductions.

In recommending revisions to the $PM_{2.5}$ NAAQS, changes to either the annual or the 24-hour standard, or both, could be recommended. Three arguments were made that support placing more emphasis on lowering the 24-hour NAAQS. First, the vast majority of studies indicating

effects of short-term $PM_{2.5}$ exposures were carried out in settings in which $PM_{2.5}$ concentrations were largely below the current standard of 65 $\mu g/m^3$. Second, the amount of evidence on short-term effects, at least as reflected by the number of reported studies, is greater than for long-term effects. Third, toxicological findings largely relate to effects of short-term $PM_{2.5}$ exposures.

There was a consensus among the Panel members in agreement with the EPA staff recommendations that focused on decreasing $PM_{2.5}$ concentrations through lowering of the 24-hour PM standard, but the panel did not endorse the option of keeping the annual standard at its present level of 15 $\mu g/m^3$. It was appreciated that some cities have relatively high annual PM concentrations, but without much variation in concentrations from day to day. Such cities would only rarely exceed a 24-hour $PM_{2.5}$ standard, even if set at levels below the current standard. This observation indicates the desirability of lowering the level of the annual $PM_{2.5}$ standard as well.

Of the options presented by EPA staff for lowering the level of the PM standard, based on the above considerations and the predicted reductions in health impacts derived from the risk analyses, most Panel members favored the option of setting a 24-hour $PM_{2.5}$ NAAQS at concentrations in the range of 35 to 30 $\mu g/m^3$ with the 98th percentile form, in concert with an annual NAAQS in the range of 14 to 13 $\mu g/m^3$. The justification for not moving to the lowest staff-recommended levels within these ranges is that these were generally associated with only small additional predicted reductions in risk. In addition, the uncertainties associated with concentration-response relationships increase greatly below these ranges, as reflected in substantial widening of the confidence limits for point estimates.

The Panel had the following advice and recommendation for the $PM_{10-2.5}$ standard:

It was acknowledged that the scientific basis supporting a causal role of $PM_{10-2.5}$ in an array of adverse health effects is weaker than that of $PM_{2.5}$. Regardless, most of the Panel members felt that the evidence that exists supports a causal role for health effects for $PM_{10-2.5}$. Moreover, setting this NAAQS would allow continuation and expansion of the $PM_{10-2.5}$ monitoring network that would facilitate collection of data for future exposure assessment and epidemiology studies. Because the evidence for the toxicity of $PM_{10-2.5}$ comes from studies conducted primarily in urban areas and is related, in large part, to the re-entrainment of urban and suburban road dusts as well as primary combustion products, there is concern that the associations of adverse effects with $PM_{10-2.5}$ may not apply to rural areas where the $PM_{10-2.5}$ is largely composed of less-toxic components of windblown soil or products of agricultural operations for which there is either no or limited evidence of health issues.

Further, although there is some evidence that short-term changes in concentrations of $PM_{10-2.5}$ are associated with changes in mortality, particularly cardiovascular mortality, the evidence in support of effects on morbidity, especially respiratory morbidity, is stronger. Most Panel members therefore favored not including short-term mortality effects in the health impact predictions, in line with the approach taken by EPA staff. The Panel agreed with Agency staff's approach of not setting an annual NAAQS for $PM_{10-2.5}$ at this time.

One of the major reservations expressed by the Panel in recommending a 24-hour $PM_{10-2.5}$ NAAQS related to the non-specificity of the $PM_{10-2.5}$ mass metric. Given that most evidence indicates that the component of the coarse fraction in most rural areas has little or no toxicity at environmental concentrations, it was felt important to qualify the $PM_{10-2.5}$ standard by somehow allowing exceptions for regions where the coarse fraction was composed largely of material that was not contaminated by industrial- or motor vehicle traffic-associated sources. Options discussed by members of the Panel for attempting to achieve this approach included limiting the standard to cover "all" urban areas, the judicious siting of monitors with a focus on urban areas, or regulatory exceptions for regions where road dust is not an issue or where rural components dominate the source. No single option was favored.

The panel also agreed that there was a need for more research on the health effects of $PM_{10-2.5}$. Such research will require the continuation and expansion of the $PM_{10-2.5}$ monitoring network in both rural and urban areas. The Panel recommends that the Agency staff expand and strengthen the discussion of the exposure index (size-range plus composition and/or source) and the monitoring strategy to be used for this NAAQS as well as the degree of public health protection expected relative to the protection against thoracic coarse PM afforded by the current PM_{10} short-term NAAQS.

WELFARE-BASED STANDARDS

Chapters 6 & 7: **PM-Related Welfare Effects**

Overall, these chapters are well done. Comments are provided below regarding vegetation and ecosystem, materials soiling, and visibility.

Considering the effects of PM on vegetation and ecosystems, EPA staff are to be commended for a well-written and concise reflection of the key science as presented in the final PM AQCD. The ecological risk assessment is reasonable given the required "criteria pollutant" approach. That being said, the criteria pollutant approach in this case (*i.e.*, PM) has serious shortcomings when it comes to ensuring environmental protection of vegetation and ecosystems in the U.S. This is illustrated in the following discussion.

Scientific evidence presented in the PM Staff Paper and the PM AQCD indicates that forest ecosystems at a number of locations in the U.S. "are now showing severe symptoms of nitrogen saturation." The Staff Paper makes the point that this is the result of chronic long-term additions of reactive nitrogen (Nr) species that have been accumulating over time. The PM Staff Paper also makes the point that the issue of forest-ecosystem deterioration is broader and more complex than just Nr accumulation. The Staff Paper notes that, "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." A key point implied here and elaborated later in the PM Staff Paper text is that PM deposition is only partially-responsible for the observed ecosystem-level effects and that the extent of the role of PM deposition in these ecosystem-level effects needs to be determined. While this has scientific merit, the question must be asked as to whether knowing the role of PM alone will improve the

protection of vegetation and ecosystems in the U.S.? The answer to this question is critical because forest ecosystems are responding to the cumulative total load that has resulted from the chronic long-term deposition of both PM as well as gases and not to PM alone.

While EPA staff have done a commendable job within the context of the criteria pollutant approach, it is strongly recommended that in the future that Agency staff give serious consideration to a philosophical shift from the criteria pollutant approach to the European approach of "critical loads" when it comes to ensuring protection of vegetation and ecosystems in the U.S. The critical load is defined in the criteria document and is 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. The current criteria pollutant approach is a significant limitation in the efforts of the Agency staff to address the cumulative load of all the pollutant stressors to which ecosystems are responding.

Considering soiling and materials effects, several of the Panel members specifically asked EPA to add some discussion of the welfare effects caused by soiling from coarse particles. This may lead to consideration of a secondary $PM_{10-2.5}$ standard intended to protect against adverse welfare effects.

Considering visibility effects, most Panel members strongly supported the EPA staff recommendation to establish a new, secondary $PM_{2.5}$ standard to protect urban visibility. Overall, the Second Draft Staff Paper visibility sections (Chapters 6 and 7 and the detailed technical appendix by Schmidt *et al.*, 2005) are well-conceived and clearly-written. Agency staff can also be commended for responsiveness to comments previously submitted by this Panel on the PM AQCD and the First Draft PM Staff Paper. The recommended new standard was considered by most Panel members to be a reasonable complement to the Regional Haze Rules that protect Class I areas. The dissenting view is provided in one Panel member's individual review comments (see pages C-101 and C-102).

The recommended range of secondary standards includes an indicator ($PM_{2.5}$ mass), averaging time (4 to 8 daylight hours), level (20 to 30 $\mu g/m^3$) and form (90^{th} percentile "or slightly higher"). The sub-daily averaging time is an innovative approach that strengthens the quality of the $PM_{2.5}$ indicator by targeting the driest part of the day. An indirect but important benefit will come from the direct use of — and more intense scrutiny on the quality of —the hourly data from the widely deployed continuous $PM_{2.5}$ mass monitors. The net effect is a "responsive" standard that (for the first time) would directly link public perception of air pollution (predominantly due to visual effects of light scattering by fine particles in the ambient air) to a routinely measured pollutant indicator (*i.e.*, artificially-dried $PM_{2.5}$ mass).

The recommended level and form of the standard are more difficult to specify. The draft PM Staff Paper employs a "bounding" approach, suggesting a level that is below the "obviously adverse" level of the current secondary standard — under which extreme short-term concentrations exceeding 100 $\mu g/m^3$ have been observed on days when 24-hour concentrations do not exceed 65 $\mu g/m^3$. Some members of the Panel felt the recommended level (and form) of the standard were on the high side, but developing a more specific (and more protective) level in future standards would require updated and refined public visibility valuation studies. Agency

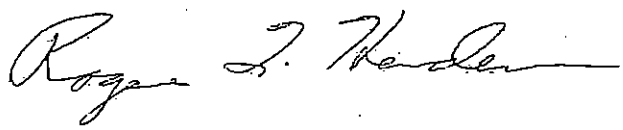
staff are strongly encouraged to support such studies prior to the next round of NAAQS review, even as it moves forward with the currently-recommended standard.

Some felt the recommended 90th percentile form of the standard was the weakest element of the EPA staff recommendation and the least well-justified. The visual effects of fine particle pollution provide the most direct public perception of air pollution of any regulated (or unregulated) pollutant, and the adversity of the effect is greatest on the haziest days that the 90th percentile would discard. Some Panel members recommend considering a higher percentile (92nd to 98th), accompanied by a level toward the upper-end of the recommended range, and/or otherwise softened by an "exceptional events" policy to assure that secondary non-attainment is not driven by natural source influences such as dust storms and wild fires.

To determine the degree of non-attainment that will result from a secondary standard, Agency staff should include — for different combinations of 4-hour and 24-hour levels and upper percentiles — estimates of concentrations and locations that would be expected to exceed a recommended secondary standard. EPA staff should also add some discussion of estimated "background" $PM_{2.5}$ conditions for the 4-hour daylight period.

In conclusion, the CASAC PM Review Panel encourages EPA in its efforts to protect the public health and our environment from the adverse effects of ambient air PM in the most effective manner possible. The Panel will continue to offer its advice and recommendations to help the Agency in meeting the mandates of the Clean Air Act and will review the final version of the staff paper with respect to EPA staff's approach to setting a $PM_{10-2.5}$ standard. As always, the CASAC PM Review Panel wishes the Agency well in this important endeavor.

Sincerely,



Dr. Rogene Henderson, Chair
Clean Air Scientific Advisory Committee

Appendix A – Roster of the CASAC Particulate Matter Review Panel

Appendix B – Charge to the CASAC Particulate Matter Review Panel

Appendix C – Review Comments from Individual CASAC Particulate Matter Review Panelists

Appendix A – Roster of the CASAC Particulate Matter Review Panel

U.S. Environmental Protection Agency
Science Advisory Board (SAB) Staff Office
Clean Air Scientific Advisory Committee (CASAC)
CASAC Particulate Matter Review Panel*

CHAIR

Dr. Rogene Henderson*, Scientist Emeritus, Lovelace Respiratory Research Institute, Albuquerque, NM

MEMBERS

Dr. Ellis Cowling*, University Distinguished Professor-at-Large, North Carolina State University, Colleges of Natural Resources and Agriculture and Life Sciences, North Carolina State University, Raleigh, NC

Dr. James D. Crapo*, Professor, Department of Medicine, Biomedical Research and Patient Care, National Jewish Medical and Research Center, Denver, CO

Dr. Philip Hopke**, Bayard D. Clarkson Distinguished Professor, Department of Chemical Engineering, Clarkson University, Potsdam, NY

Dr. Jane Q. Koenig, Professor, Department of Environmental Health, School of Public Health and Community Medicine, University of Washington, Seattle, WA

Dr. Petros Koutrakis, Professor of Environmental Science, Environmental Health , School of Public Health, Harvard University (HSPH), Boston, MA

Dr. Allan Legge, President, Biosphere Solutions, Calgary, Alberta

Dr. Paul J. Lioy, Associate Director and Professor, Environmental and Occupational Health Sciences Institute, UMDNJ - Robert Wood Johnson Medical School, NJ

Dr. Morton Lippmann, Professor, Nelson Institute of Environmental Medicine, New York University School of Medicine, Tuxedo, NY

Dr. Joe Mauderly, Vice President, Senior Scientist, and Director, National Environmental Respiratory Center, Lovelace Respiratory Research Institute, Albuquerque, NM

Dr. Roger O. McClellan, Consultant, Albuquerque, NM

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Dr. Gunter Oberdorster, Professor of Toxicology, Department of Environmental Medicine, School of Medicine and Dentistry, University of Rochester, Rochester, NY

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Dr. Warren H. White, Visiting Professor, Crocker Nuclear Laboratory, University of California - Davis, Davis, CA

Dr. George T. Wolff, Principal Scientist, General Motors Corporation, Detroit, MI

Dr. Barbara Zielinska*, Research Professor, Division of Atmospheric Science, Desert Research Institute, Reno, NV

SCIENCE ADVISORY BOARD STAFF

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* Members of the statutory Clean Air Scientific Advisory Committee (CASAC) appointed by the EPA Administrator

**Immediate past CASAC Chair

ATTACHMENT B

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON D.C. 20460

September 15, 2005

EPA-SAB-CASAC-05-012

OFFICE OF THE ADMINISTRATOR
SCIENCE ADVISORY BOARD

Honorable Stephen L. Johnson
Administrator
U.S. Environmental Protection Agency
1200 Pennsylvania Avenue, NW
Washington, DC 20460

Subject: Clean Air Scientific Advisory Committee (CASAC) Review of the EPA Staff Recommendations Concerning a Potential Thoracic Coarse PM Standard in the *Review of the National Ambient Air Quality Standards for Particulate Matter: Policy Assessment of Scientific and Technical Information* (Final PM OAQPS Staff Paper, EPA-452/R-05-005, June 2005)

Dear Administrator Johnson:

EPA's Clean Air Scientific Advisory Committee (CASAC or "Committee"), supplemented by subject-matter-expert Panelists — collectively referred to as the CASAC Particulate Matter (PM) Review Panel ("Panel") — held a public teleconference on August 11, 2005 to review the EPA staff recommendations concerning a potential thoracic coarse PM standard in the *Review of the National Ambient Air Quality Standards for Particulate Matter: Policy Assessment of Scientific and Technical Information* (Final PM OAQPS Staff Paper, EPA-452/R-05-005, June 2005).

The CASAC PM Review Panel agrees that this report adequately represents their views. The chartered CASAC — whose seven members are also members of the Panel — fully endorses the Panel's report and hereby forwards it to you as the Committee's consensus report on this subject. The current Clean Air Scientific Advisory Committee roster is found in Appendix A of this report, and the CASAC PM Review Panel roster is attached as Appendix B. Discussion questions for the Panel provided by the CASAC are contained in Appendix C to this report, and Panelists' individual review comments are provided in Appendix D.

This meeting was a continuation of the CASAC PM Review Panel's peer review of the Agency's *Review of the National Ambient Air Quality Standards for Particulate Matter: Policy Assessment of Scientific and Technical Information* (Second Draft PM Staff Paper, January 2005) and a related draft technical report, *Particulate Matter Health Risk Assessment for Selected Urban Areas: Second Draft Report* (Second Draft PM Risk Assessment, January 2005).

The Panel met in a public meeting held on April 6-7, 2005 in Durham, NC to conduct a peer review of these documents. This was followed by a public teleconference held on May 18, 2005 to review and approve the Panel's report from its April meeting. The Panel's letter/report to you from this April 2005 meeting, dated June 6, 2005, is found at the following URL: http://www.epa.gov/sab/pdf/casac-05-007.pdf.

In its review of the EPA staff recommendations concerning a potential thoracic coarse PM national ambient air quality standard (NAAQS), there was general concurrence among Panel members that there was a need for a specific primary standard to address particles in the size range of 2.5 to 10 microns, as EPA staff recommended in the Final PM Staff Paper. The Committee agrees with the summary of the scientific data regarding the potential adverse health effects from exposures to thoracic coarse particles in Section 5.4 of the Staff Paper. The Committee is also in general agreement with EPA staff observations that coarse particles in urban or industrial areas are likely to be enriched by anthropogenic pollutants that tend to be inherently more toxic than the windblown crustal material which typically dominates coarse particle mass in arid rural areas. Most but not all Panel members were supportive of the EPA staff recommendation to specify an urban coarse particle indicator of $UPM_{10-2.5}$. Some members recommended specifying a national $PM_{10-2.5}$ standard accompanied by monitoring and exceptional-events guidance that emphasized urban influences.

The CASAC notes that it is important to recognize that the urban coarse particle indicator for the standard represents a surrogate for the components of the urban coarse PM that differ in composition from coarse-mode particles of natural origin. Sufficient data are lacking at the present time to set standards based specifically on composition. Therefore, there is a need for substantial future research on the health effects of coarse-mode particles, and CASAC recommends that monitoring of both rural areas as well as urban areas be done for total $PM_{10-2.5}$ levels, distribution and composition. Moreover, the collection of coarse PM data in selected locations for compliance-monitoring purposes should be coordinated with epidemiological and toxicological research efforts as soon as this monitoring capability is in place. It is recognized that, as more information on the toxicity of rural dusts is acquired, the name and/or geographical focus of a coarse-particle indicator may need to be reconsidered.

The Committee also supports the use of a 24-hour averaging time and agrees that an annual averaging time for $PM_{10-2.5}$ is not currently warranted. Furthermore, the CASAC strongly recommends use of the 98th percentile, which is more statistically-robust than the 99^{th} percentile, together with the use of a three-year average of this statistic. In addition, there was general agreement among Panel members that Agency staff had presented a reasonable justification for the ranges proposed, with most members favoring levels at the upper end of the range for the 98^{th} percentile form. Overall, therefore, the Committee finds that the Agency has been responsive to the previous advice and recommendations of the CASAC. By use of the indicator $UPM_{10-2.5}$, the Agency is taking a next step toward including composition as well as size in its regulations of ambient air PM. The Committee sees this process as a progressive one and reiterates that, as our knowledge of the potential toxicity of rural $PM_{10-2.5}$ increases, the potential need for regulation of rural $PM_{10-2.5}$ particles may need to be reevaluated.

1. Background

The CASAC, comprised of seven members appointed by the EPA Administrator, was established under section 109(d)(2) of the Clean Air Act (CAA or "Act") (42 U.S.C. § 7409) as an independent scientific advisory committee, in part to provide advice, information and recommendations on the scientific and technical aspects of issues related to air quality criteria and NAAQS under sections 108 and 109 of the Act. Section 109(d)(1) of the CAA requires that EPA carry out a periodic review and revision, where appropriate, of the air quality criteria and the NAAQS for "criteria" air pollutants such as PM. The CASAC, which is administratively located under EPA's Science Advisory Board (SAB) Staff Office, is a Federal advisory committee chartered under the Federal Advisory Committee Act (FACA), as amended, 5 U.S.C., App. The CASAC PM Review Panel is comprised of the seven members of the chartered (statutory) Clean Air Scientific Advisory Committee, supplemented by fifteen technical experts.

Under section 108 of the CAA, the Agency is required to establish NAAQS for each pollutant for which EPA has issued criteria, including PM. Section 109(d) of the Act subsequently requires periodic review and, if appropriate, revision of existing air quality criteria to reflect advances in scientific knowledge on the effects of the pollutant on public health and welfare. EPA is also to revise the NAAQS, if appropriate, based on the revised criteria. The purpose of the PM Staff Paper is to evaluate the policy implications of the key scientific and technical information contained in a related document, EPA's revised Air Quality Criteria [Document] (AQCD) for Particulate Matter (EPA/600/P-99/002aF & -bF, October 2004) and to identify critical elements that EPA believes should be considered in the review of the PM NAAQS. The Staff Paper for PM is intended to "bridge the gap" between the scientific review contained in the PM AQCD and the public health and welfare policy judgments required of the Administrator in reviewing the PM NAAQS.

In the letter/report to the EPA Administrator from its April 6-7 meeting, the Panel informed you that:

> "… after the Panel has reviewed the Final Staff Paper and Risk Assessment for Particulate Matter following its issuance on June 30, 2005, the Panel will meet again this summer via a public teleconference to consider the final Staff Paper's recommendations concerning the setting of a coarse PM standard. Subsequent to the Panel's teleconference meeting, we will send you a separate letter providing the Panel's recommendations concerning $PM_{10-2.5}$ as an indicator together with our views on the averaging time, statistical form, and level of any potential daily $PM_{10-2.5}$ standard."

Section 5.4 of the Final PM staff Paper, which discusses the Thoracic Coarse Particle Standard, is found on pages 5-47 through 5-71 of the document. This section was the focal point of the Panel's teleconference meeting.

2. CASAC Review of the EPA Staff Recommendations Concerning a Potential Thoracic Coarse PM Standard in the Final PM OAQPS Staff Paper

a. Indicator for a Thoracic Coarse Particle Standard

There was general concurrence among the members of the Panel that there was a need for a specific primary standard to address particles in the size range of 2.5 to 10 microns, as the Agency staff recommended in the Final PM Staff Paper. The Committee agrees with the summary of the scientific data regarding the potential adverse health effects from exposures to thoracic coarse particles in Section 5.4 of the Staff Paper. The body of evidence on health effects associated with exposure to thoracic coarse particles that is relevant to $PM_{10-2.5}$ is limited. However, several U.S. and Canadian studies do provide convincing data that there is an association between short-term exposure to $PM_{10-2.5}$ and various morbidity endpoints. Associations with mortality endpoints were suggestive but not as convincing as were the morbidity endpoints. These data showing adverse health effects associated with exposure to $PM_{10-2.5}$ primarily come from studies of urban environments. Little is known about the potential toxicity of rural dusts, although the 2000 and 2003 Coachella Valley, CA studies from Ostro *et al.* showed significant adverse health effects, primarily involving exposures to coarse-mode particles arising from crustal sources. $PM_{10-2.5}$ concentrations in urban and industrial areas may be commonly enriched by a number of contaminants not commonly found in crustal material.

CASAC generally agrees with EPA staff conclusions that thoracic coarse particles in urban areas can be expected to differ in composition from those in rural areas and that evidence of associations with health effects related to urban coarse-mode particles would not necessarily apply to non-urban or rural coarse particles (although it is likely that there will be some overlap of the same contaminants in both areas.) Most Panel members concurred that the current scarcity of information on the toxicity of rural dusts makes it necessary for the Agency to base its regulations on the known toxicity of urban-derived coarse particles, and that an urban coarse particle indicator should be specified as $UPM_{10-2.5}$. Other Panel members recommended specifying a national $PM_{10-2.5}$ standard accompanied by monitoring and exceptional-events guidance that emphasized urban influences. Some members also expressed concerns whether EPA would be able to specify a clear definition of "urban" to effectively determine in advance the specific conditions in which the standard would (and would not) apply. It is recognized that, as more information on the toxicity of rural dusts is acquired, the name and/or geographical focus of a coarse-particle indicator may need to be reconsidered.

In establishing the parameters for the thoracic coarse particle monitoring network, the Agency should consider the estimated risk reduction in health outcomes (or, alternately stated, the potential national benefits of reducing the public health risks) associated with a reduction in thoracic coarse particles in areas with smaller populations as well as those with large populations. There is a paucity of data currently available on health outcomes related to thoracic coarse particles in rural areas and limited information on the composition and toxicity of rural area coarse particles. This underscores the need for monitoring thoracic coarse particle levels and for population-based health-effects studies in those rural areas where it is feasible to conduct such studies.

It is important to recognize that the use of an urban coarse particle standard with $UPM_{10-2.5}$ mass as an indicator is intended to provide protection against those components of $PM_{10-2.5}$ that arise from anthropogenic activities occurring in or near urban and industrial areas. Sufficient data are lacking at the present time to set standards based specifically on composition and, thus, there is a need for substantial future research on the health effects of coarse-mode particles. Data on both urban and rural exposures to coarse-mode particles as defined by $PM_{10-2.5}$ mass are needed, and there is a need for more data that relate the composition of the particulate matter to adverse health effects. We anticipate that future coarse- and fine-mode particulate standards will give greater weight to particulate composition as a critical element in defining the risk of adverse health effects. Data are needed on ambient concentrations in each size range in terms of mass concentrations and speciation. Continuous monitors for mass, as well as for key components or source-related tracers, will provide the best and most cost-effective means of collecting such data for both epidemiologic research and compliance monitoring. Moreover, the collection of coarse PM data in selected locations for compliance-monitoring purposes should be coordinated with epidemiological and toxicological research efforts as soon as this monitoring capability is in place.

CASAC recommends that monitoring of both rural and urban areas be done for total particulate levels, size distribution and composition. It is essential to have data collected on a wide range of both urban and rural areas in order to determine whether or not the proposed $UPM_{10-2.5}$ standard should be modified at the time of future reviews. Finally, some members of the Panel recommended that a secondary $PM_{10-2.5}$ standard be set at the same level as used for the (primary) $UPM_{10-2.5}$ standard. The geographical applicability of this secondary standard should not necessarily be constrained only to urban areas, as the irritant, nuisance soiling, materials damage and ecological effects of coarse particles are not uniquely related to urban contaminants.

b. Averaging Time and Statistical Form for a Thoracic Coarse Particle Standard

The limited results available from epidemiological studies suggest short-term morbidity effects (*e.g.*, respiratory- and cardiac-related hospital admissions, respiratory symptoms in children) are associated with $PM_{10-2.5}$ and that this indicator variable may indeed be causative, at least for those urban areas in which these studies were conducted. The evidence for short-term mortality effects from exposure to coarse particles is less convincing, and the epidemiological studies do not (except in a very few studies) suggest any clear associations with long-term, chronic exposures. The bases for EPA's proposed retention of a 24-hour averaging time and high-percentile (98^{th} or 99^{th}) statistical form, and the elimination of the annual averaging time for coarse particles, are well described in Chapter 5 of the Final PM Staff Paper. The CASAC finds this discussion both to be balanced and to reflect adequately the options that are reasonable for the EPA Administrator to consider for the averaging time and statistical form of a short-term $PM_{10-2.5}$ standard. Specifically, the Committee supports the use of a 24-hour averaging time and agrees that an annual averaging time for $PM_{10-2.5}$ is not currently warranted.

The CASAC also strongly recommends use of the 98th percentile, which is more statistically-robust than the 99^{th} percentile, together with the use of a three-year average of this statistic. This use of this statistic will tend to minimize measurement error and spatial variability, which are larger for coarse-mode particles than for fine PM. It would also tend to minimize the influence in arid areas of occasional but extreme excursion contributions from rural, coarse-

mode dust sources that are thought to be inherently less toxic than coarse-mode particles heavily enriched with urban source contaminants

To a certain degree, however, the conclusions reached by EPA staff regarding the lack of a basis for an annual averaging time reflect more an absence of evidence than they do any evidence of absence of effects from long-term exposures. In a similar way, the evidence suggesting that all types of "non-urban" coarse particles are relatively benign is also quite limited. The only way that these issues will be resolved is if additional epidemiological studies are conducted in both urban and rural areas that examine whether there are morbidity and/or mortality effects from short-term and long-term exposure to coarse-mode particulate matter. For these and other PM NAAQS studies, a rich database will be needed on ambient concentrations in each size range in terms of mass concentrations and composition.

c. Level for a Thoracic Coarse Particle Standard

There was general agreement among Panel members that Agency staff had presented a reasonable justification for the ranges of levels proposed. In contrast, there was one member who thought the lower bounds as proposed would leave a substantial portion of the population (particularly in the Northeast) at continued significant risk, and several members who supported the lower ends of the proposed ranges. However, because of the significant uncertainties resulting from the limited number of studies to date, in which $PM_{10-2.5}$ has been measured, and potentially large exposure measurement errors in the available coarse-particle databases, a more stringent lower bound was not generally supported.

Overall, the Panel found that the Agency has been responsive to the previous advice and recommendations of the CASAC. By use of the indicator $UPM_{10-2.5}$, the Agency is taking a next step toward including composition as well as size in its regulations of ambient air PM. The Committee sees this process as a progressive one and reiterates that, as our knowledge of the potential toxicity of rural $PM_{10-2.5}$ particles increases, the need for primary health standards for rural $PM_{10-2.5}$ particles may need to be reevaluated.

The Clean Air Scientific Advisory Committee and the CASAC Particulate Matter Review Panel have been pleased to advise the Agency in this extremely important and difficult task of setting appropriate standards for airborne coarse particles. As always, we wish EPA well as the Agency continues this process.

Sincerely,

Rogene L. Henderson

Dr. Rogene Henderson, Chair
Clean Air Scientific Advisory Committee

Appendix A – Roster of the Clean Air Scientific Advisory Committee

U.S. Environmental Protection Agency
Science Advisory Board (SAB) Staff Office
Clean Air Scientific Advisory Committee (CASAC)

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Dr. Rogene Henderson, Scientist Emeritus, Lovelace Respiratory Research Institute, Albuquerque, NM

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Dr. Frederick J. Miller, Consultant, Cary, NC

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* Immediate past CASAC Chair

Appendix B – Roster of the CASAC Particulate Matter Review Panel

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Clean Air Scientific Advisory Committee (CASAC)
CASAC Particulate Matter Review Panel*

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