Policy Assessment for the Review of the Particulate Matter National Ambient Air Quality Standards

Preliminary Draft

September 2009

DISCLAIMER

This preliminary draft document has been prepared by staff from the Ambient Standards Group, Office of Air Quality Planning and Standards, U.S. Environmental Protection Agency. Any opinions, findings, conclusions, or recommendations are those of the authors and do not necessarily reflect the views of the EPA. This document is being circulated for informational purposes and to facilitate discussion with the Clean Air Scientific Advisory Committee (CASAC) on the overall structure, areas of focus, and level of detail to be included in an external review draft Policy Assessment, which EPA plans to release for CASAC review and public comment later this year. Questions related to this preliminary draft document should be addressed to Beth Hassett-Sipple, U.S. Environmental Protection Agency, Office of Air Quality Planning and Standards, C504-06, Research Triangle Park, North Carolina 27711 (email: hassett-sipple.beth@epa.gov).

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LIST OF ACRONYMS/ABBREVIATIONS

A/C	Air conditioning
AAC	Abdominal Aortic Calcium
ACS	American Cancer Society
ABI	Ankle-brachial index
AERMOD	American Meteorological Society/EPA Regulatory Model Improvement Committee (AERMIC) air quality dispersion modeling system
AHR	Airway Hyperesponsiveness
AHRQ	Agency for Healthcare Research and Quality
AHS	American Housing Survey
AHSMOG	California Seventh Day Adventist Study
Al	Aluminum
ANS	Autonomic nervous system
APEX	EPA's Air Pollutants Exposure model, version 4
APHEA	Air Pollution and Health: a European Approach
AQCD	Air Quality Criteria Document
AQI	Air Quality Index
AQS	EPA's Air Quality System
AR4	Fourth Assessment Report of the Intergovernmental Panel on Climate Change
ARIES	Aerosol Research and Inhalation Epidemiological Study
As	Arsenic
ASOS	Automated Surface Observing System
BAD	Bronchial Artery Diameter
BAM	Beta Attenuation Mass Monitor
BASE	Building Assessment Survey and Evaluation
BC	Black carbon
BC	British Columbia
BenMAP	Benefits Mapping Analysis Program
BMI	Body Mass Index
BNP	B-type natriuretic peptide/Brain natriuretic peptide
BP	Blood pressure
BS	Black smoke

C	Carbon
Ca	Calcium
CAA	Clean Air Act
CAC	Coronary Artery Calcium
CAIR	Clean Air Interstate Rule
CAMx	Comprehensive Air quality Model
CAP	Concentrated Ambient Particles
CARB	California Air Resources Board
CASAC	Clean Air Scientific Advisory Committee
CBSA	Consolidated Business Statistical Area
CBVD	Cerebrovascular disease
CCN	Cloud Condensation Nuclei
CCSP	US Climate Change Science Program
Cd	Cadmium
CDPHE	Colorado Department of Public Health and Environment
CEM	Continuous Emission Monitoring
CHAD	EPA's Consolidated Human Activity Database
CHD	Coronary Heart disease
CHF	Congestive heart failure
CHS	Childrens Health Study
CIMT	Carotid intima-media thickness
CMAQ	Community Multiscale Air Quality
CNS	Central Nervous System
CO	Carbon Monoxide
CONUS	CMAQ simulations covering continental US
COPD	Chronic obstructive pulmonary disease
COV	Coefficient of Variation
CPL	Candidate Protection Level
Cr	Chromium
C-R	Concentration-response relationship
CRA	Charles River Associates
CRP	C-reactive protein
CSA	Consolidated Statistical Area
CSN	Chemical Speciation Network

CTM	Chemical transport models
CTS	CMAQ model run
Cu	Copper
CV	Cardiovascular
CVD	Cardiovascular disease
DBP	Diastolic Blood Pressure
DE	Diesel Exhaust
DNA	Deoxyribonucleic Acid
DRE	Direct Radiative Effects
dv	deciview
DVT	Deep Vein Thrombosis
EC	Elemental Carbon
ECG	Electrocardiogram
ED	Emergency department
EPA	Environmental Protection Agency
ETV	Environmental Technology Verification program
eVNA	enhanced Voronoi Neighbor Averaging
FEM	Federal Equivalent Method
FEV_1	Change in forced expiratory volume in one second
FIP	Federal Implementation Plan
FMD	Flow-mediated vasodilation
FRM	Federal Reference Method
GAMs	Generalized additive models
GEOS	Global Scale Air Circulation Model
GHG	Greenhouse Gas
GI	Group Interviews
GLMs	Generalized linear models
GSH	Glutathione
GST	Glutathione-S-transferase
HA	Hospital admissions
HCUP	Healthcare Cost and Utilization Project
HEAPSS	Health Effects of Air Pollution Among Susceptible Subpopulations study
HEI	Health Effects Institute

HF	High Frequency
Hg	Mercury
HR	Heart rate
HRV	Heart rate variability
ICAM-1	Intercellular adhesion molecule-1
ICD	International Classification of Disease
ICD	Implantable/Implanted Cardioverter defibrillator
ICR	Information Collection Request
IEC	Industrial Economics, Incorporated
IFG	Investigative Focus Groups
IHD	Ischemic heart disease
IMPROVE	Interagency Monitoring of Protected Visual Environment
IPCC	Intergovernmental Panel on Climate Change
IRP	Integrated Review Plan
ISA	Integrated Science Assessment
ISCST	EPA's Industrial Source Complex Short-Term model
IT	Intratracheal
Km	Kilometer
Lag	Time between one event and another
Lag 0	Same day as the death, test, hospital, ED, clinic, physician visit; that occurs on the same day as the exposure to the pollutant(s)
Lag 0-x	All the deaths test, hospital, ED, clinic, physician visit; that occurs on the same day as the exposure to the pollutant(s) and the x days following the day of exposure
LCD	Liquid Crystal Display
LF	Low Frequency
LOESS	Locally weighted Scatter Plot Smoothing
L/W	Ratio of lumen to wall
MAP	Mean Arterial Pressure
MCAPS	Medicare Air Pollution Study
ME	Microenvironment
MEA	Millennium Ecosystem Assessment
MENTOR	Modeling ENvironment for TOtal Risk
MI	Myocardial infarction
Mm	Megameter

MOA	Mode(s) or mechanism(s) of action
MSA	Metropolitan Statistical Area
N	Nitrogen
NAAQS	National Ambient Air Quality Standards
NAPS	National Air Pollution Surveillance
NARSTO	North American Research Strategy for Tropospheric Ozone
NCDC	National Climatic Data Center
NCEA	National Center for Environmental Assessment
NCore	National Core Monitoring Network
NEI	National Emissions Inventory
NEM	NAAQS Exposure Model
NERL	National Exposure Research Laboratory
NHANES	National Health and Nutrition Examination Survey
Ni	Nickel
NLCS	Netherlands Cohort Study
NMMAPS	National Morbidity, Mortality, and Air Pollution Study
NN intervals	Normal-to-normal (NN or RR, sinus) time interval between each QRS complex in the EKG
NOAA	National Oceanic and Atmospheric Administration
NO	Nitric Oxide
NO_2	Nitrogen dioxide
NO_3^-	Nitrate
NOx	Nitrogen oxides
NPS	National Park Service
NRC	National Research Council
NWS	National Weather Service
O_3	Ozone
OAQPS	Office of Air Quality Planning and Standards
OAR	Office of Air and Radiation
OC	Organic Carbon
OMB	Office of Management and Budget
OR	Odds Ratio
ORD	Office of Research and Development
OS	Observational Study

PA	Policy Assessment
PA	Pulmonary arterial
PAH	Polyaromatic Hydrocarbon
Pb	Lead
PBDE	Polybromiated diphenyl ether
PCA	Principal Component Analysis
PEF	Peak Expiratory Flow L/min
PM	Particulate matter
PM_{10}	Particles with a 50% upper cut-point of 10± 0.5 μm aerodynamic diameter and a penetration curve as specified in the Code of Federal Regulations.
$PM_{10-2.5}$	Particles with a 50% upper cut-point of 10 μm aerodynamic diameter and a lower 50% cut-point of 2.5 μm aerodynamic diameter.
$PM_{2.5}$	Particles with a 50% upper cut-point of 2.5 μm aerodynamic diameter and a penetration curve as specified in the Code of Federal Regulations.
PM_X	The legal definition for PM_X, as defined in the Code of Federal Regulations, includes both a 50% cut-point and a penetration curve. A 50% cut-point of X μm diameter means that 50% of particles with aerodynamic diameter of X are removed by the inlet and 50% pass through the inlet and are collected on the filter. Depending on the specific penetration curve specified, particles larger than X μm aerodynamic diameter are collected with an efficiently than decreases rapidly for particles larger than X while the collection efficiency for particles smaller than X increases rapidly with decreasing size until 100 % efficiency is reached.
PMF	Positive Matrix Factorization
POP	Persistant Organic Pollutant
PRB	Policy-Relevant Background
PSAS	The French National Program on Air Pollution Health Effects
PT	Prothrombin Time
PTT	Partial Thomboplastin Time
QA	Quality assurance
QT	Time for depolarization and repolarization of the ventricles
REA	Risk and Exposure Assessment
RF	Radiative forcing

RH	Relative humidity
RNS	Reactive Nitrogen Species
rMSSD	Square root of the mean squared successive NN interval differences in the EKG
ROFA	Residential Oil Fly Ash
ROS	Reactive Oxygen Species
RR	Relative risk
RV	Right Ventricular
SAB	Science Advisory Board
SANDWICH	Sulfate, Adjusted Nitrate, Derived Water, Inferred Carbonaceous mass approach
SAP	Synthesis and Assessment Product
SBP	Systolic Blood Pressure
SCAB	South Coast Air Basin
SD	Standard deviation
SDNN	standard deviation normal-to-normal (NN or RR) time interval between each QRS complex in the EKG
SEARCH	Southeastern Aerosol Research and Characterization Study
SEDD	State Emergency Department Databases
SES	Socioeconomic Status
SH	Spontaneously Hypertensive
SHEDS-PM	Stochastic Human Exposure and Dose Simulation model for PM
Si	Silicon
SID	State Inpatient Database
SMOKE	Sparse Matrix Operator Kernal Emissions
S	Sulfur
SO_2	Sulfur Dioxide
SO_4^{2-}	Sulfate
SO_x	Sulfur Oxides
SOPHIA	Study of Particulates and Health in Atlanta
STP	Standard Temperature and Pressure
TB	Tracheobronchial
TBARS	Thiobarbituric acid reactive substances
TEACH	Toxicity and Exposure Assessment for Children's Health
TEOM	Tapered Element Oscillating Microbalance
TNF-α	Tumor Necrosis Factor-Alpha

TSP	Total suspended particulate
UACR	Urinary Albumin/Creatine Ratio
UBC	University of British Columbia
UFPs	Ultrafine particles
UFVA	Urban-Focused Visibility Impact Assessment
V	Vanadium
VAQ	Visual Air Quality
VCAM-1	Vascular cell adhesion molecule-1
VOC	Volatile organic compounds
WACAP	Western Airborne Contaminants Assessment Project
WBC	White Blood Cell
WHI	Women's Health Initiative
Zn	Zinc

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1 INTRODUCTION

1.1 PURPOSE

The U.S. Environmental Protection Agency (EPA) is presently conducting a review of the particulate matter (PM) national ambient air quality standards (NAAQS). The EPA's overall plan and schedule for this PM NAAQS review were presented in the *Integrated Review Plan for the National Ambient Air Quality Standards for Particulate Matter* (US EPA, 2008a). The Integrated Review Plan (IRP) outlined the Clean Air Act (CAA) requirements related to the establishment and reviews of the NAAQS, the process and schedule for conducting the current PM NAAQS review, and the key components in the NAAQS review process: an Integrated Science Assessment (ISA), Risk and Exposure Assessments (REAs, referred to as assessment documents), and policy assessment/rulemaking. It also presented the key policy-relevant issues to be addressed in this review as a series of questions that frames our approach to determining whether the current primary (health-based) and secondary (welfare-based) NAAQS for PM should be retained or revised.

At this time, three draft documents prepared as part of this PM NAAQS review have recently been released for review by the public and EPA's Clean Air Scientific Advisory Committee (CASAC) PM NAAQS Review Panel at a public meeting to be held on October 5-6, 2009. These documents include the *Integrated Science Assessment for Particulate Matter: Second External Review Draft* (ISA, July 2009), prepared by EPA's National Center for Environmental Assessment, Research Triangle Park, NC (NCEA-RTP), and two draft assessment documents, prepared by EPA's Office of Air Quality Planning and Standards (OAQPS), *Risk Assessment to Support the Review of the PM Primary National Ambient Air Quality Standards -- External Review Draft* (September 2009) and *Particulate Matter Urban-Focused Visibility Assessment – External Review Draft* (September 2009).

In addition, OAQPS staff are preparing a Policy Assessment (PA) to help "bridge the gap" between the relevant scientific information and assessments and the judgments required of the EPA Administrator in determining whether it is appropriate to retain or revise the NAAQS for PM. Preparation of a PA by OAQPS staff reflects Administrator Jackson's decision to modify the NAAQS review process as presented in the IRP by reinstating the use of a policy assessment document (referred to as a Staff Paper in past reviews) in lieu of issuing a policy assessment in the form of an advance notice of proposed rulemaking (see http://www.epa.gov/ttn/naaqs/review.html).

When complete, the PA will present factors relevant to EPA's review of the primary (health-based) and secondary (welfare-based) PM NAAQS. It will consider both evidence- and risk-based approaches in evaluating the adequacy of the current PM standards and in identifying potential alternative standards for the Administrator's consideration. In preparing the PA, OAQPS staff considers the available scientific evidence presented in the ISA and the quantitative assessments presented in the REAs, together with related limitations and uncertainties, and focuses on information that is most pertinent to evaluating the basic elements of air quality standards: indicator[1], averaging time, form[2], and level. These elements, which together serve to define each standard, must be considered collectively in evaluating the health and welfare protection afforded by the PM standards.

We are releasing a preliminary draft PA at this time, prior to the upcoming CASAC meeting, for informational purposes and to facilitate discussion with the CASAC PM Panel on the overall structure, areas of focus, and level of detail to be included in an external review draft PA. This preliminary draft document, which is clearly a work in progress, draws from the information and conclusions presented in the second draft ISA (U.S. EPA, 2009a) and the two draft REAs (U.S. EPA, 2009b,c).

Following this introductory chapter, this preliminary draft PA includes early draft chapters addressing the following: a characterization of ambient PM (Chapter 2), an overview of the policy-relevant health effects evidence (Chapter 3), and an overview of the policy-relevant evidence of PM-related welfare effects and the assessment of PM-related visibility impairment (Chapter 6). Chapter 4, which will present an overview of the health risk assessment, is previewed by way of an annotated outline. Chapters 5 and 7, which will present staff conclusions on a range of policy options for the Administrator to consider concerning whether, and if so how, to revise the primary and secondary PM NAAQS, are previewed only by way of the organization shown in the draft table of contents. These two chapters will discuss both evidence- and risk-based approaches to informing consideration of the basic elements of each of the PM NAAQS: indicator, averaging time, form, and level.

As noted above, this preliminary draft PA is a work in progress. For example, we are still working to identify and potentially adapt figures from the ISA to sharpen our focus and discussion of the health effects evidence presented in Chapter 3. Additionally, we are continuing

[1] The "indicator" of a standard defines the chemical species or mixture that is to be measured in determining whether an area attains the standard.

[2] 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.

our efforts to obtain relevant air quality data from epidemiologic study authors to aid in our evidence-based approach to evaluating the current and potential alternative primary standards. We are also continuing to consider how to focus our presentation of the scientific evidence and quantitative analyses in a manner that will most directly help to address the key policy-relevant questions that frame this review. Staff will consider CASAC review and public comments on the second draft ISA and the two draft assessment documents, as well as CASAC discussion of this preliminary draft PA, in subsequently preparing a complete external review draft PA. We plan to release a complete external review draft PA later this year for review by the CASAC PM Panel and the public in early 2010.

While this preliminary draft PA should be of use to all parties interested in the PM NAAQS review, it is written with an expectation that the reader has some familiarity with the technical discussions contained in the second draft ISA (US EPA, 2009a) and two draft assessment documents (US EPA, 2009b,c).

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, cause or contribute to air pollution which 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 to "accurately reflect the latest scientific knowledge useful in indicating the kind and extent of all identifiable effects on public health or welfare which may be expected from the presence of [a] pollutant in ambient air . . ." 42 U.S.C. § 7408(b).

Section 109 (42 U.S.C. 7409) directs the Administrator to propose and promulgate "primary" and "secondary" NAAQS for pollutants for which air quality criteria are issued. Section 109(b)(1) defines a primary standard as one "the attainment and maintenance of which in the judgment of the Administrator, based on [air quality] criteria and allowing an adequate margin of safety, are requisite to protect the public health."[3] A secondary standard, as defined in

[3] 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).

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 [air quality] 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."[4]

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); *American Farm Bureau Federation v. EPA,* 559 F. 3d 512, 533 (D.C. Cir. 2009). 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, EPA considers such factors as the nature and severity of the health effects involved, the size of the sensitive population(s) at risk, and the kind and degree of the uncertainties that must be addressed. The selection of any particular approach to providing an adequate margin of safety is a policy choice left specifically to the Administrator's judgment. *Lead Industries Association v. EPA*, supra, 647 F.2d at 1161-62.

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, 471, 475-76 (2001).

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

[4] 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."

such revisions in such criteria and standards and promulgate such new standards as may be appropriate . ..." 42 U.S.C. § 7409(d)(1). 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 . . ." 42 U.S.C. § 7409(d)(2). Since the early 1980's, this independent review function has been performed by CASAC.

1.2.2 Overview of the NAAQS Review Process

Since completion of the last PM NAAQS review, the Agency has made a number of changes to the process for reviewing the NAAQS.[5] In making these changes, the Agency consulted with CASAC, which provides advice to the Administrator on key elements of NAAQS reviews, and the public. This revised process contains four major components: planning, science assessment, risk/exposure assessment, and policy assessment/rulemaking. Each of these components is described in this section.

The planning phase of the review process begins with a "kick-off" workshop early in the planning phase to get input from CASAC, internal and external experts, and the public regarding policy-relevant science issues that have emerged since the last review. The workshop discussions help inform the preparation of an IRP jointly by NCEA and OAQPS staff. A draft IRP is presented for consultation with CASAC and for public comment. A final IRP reflects CASAC and public comments together with early guidance from Agency management. The IRP includes the science-policy questions that will frame the review, an outline of the process and schedule that the entire review will follow, and more complete descriptions of the purpose, contents, and approach for developing each of the key documents in the review.

The science assessment phase involves the preparation of an ISA by NCEA-RTP staff. The ISA provides a concise evaluation and integration of the policy-relevant science, including key science judgments that are important to inform the design and scope of the risk and exposure assessments. The ISA and its supporting annexes provide a comprehensive assessment of the current scientific literature pertaining to known and anticipated effects on public health and welfare associated with the presence of the pollutant in the ambient air, emphasizing information that has become available since the last review. The process generally includes production of a first and second draft ISA, both of which undergo CASAC and public review prior to completion of the final ISA.

[5] See http://www.epa.gov/ttn/naaqs/ for more information.

In the risk/exposure assessment phase, OAQPS staff draws upon information and conclusions presented in the ISA to develop quantitative estimates of the risks/exposures for health and/or welfare effects associated with current ambient levels of PM, with levels that just meet the current standards, and with levels that just meet potential alternative standards. The REAs provide a concise presentation of methods, key results, observations, and related uncertainties. These assessments begin with the preparation of a planning document that discusses the scope and methods planned for use in conducting the quantitative assessments. Such Scope and Methods Plans are generally prepared in conjunction with the first draft ISA and presented for consultation with CASAC and for public comment. Comments received on the Scope and Methods Plan are considered in preparing a draft REA that undergoes CASAC and public review, generally in conjunction with review of the second draft ISA, prior to completion of a final REA.

The review process ends with a policy assessment/rulemaking phase. Under recent revisions to NAAQS review process, the EPA Administrator has reinstated the use of a Policy Assessment (PA), which is, like the previous Staff Paper, a document that provides a transparent staff analysis of the scientific basis for alternative policy options for consideration by the Administrator prior to the issuance of proposed and final rules (Jackson, 2009). The PA integrates and interprets the information from the ISA and REA to frame policy options for consideration by the Administrator. A draft PA is released for CASAC review and public comment prior to completion of the final PA. It is intended to facilitate CASAC's advice and recommendations to the Administrator on any new standards or revisions to existing standards as may be appropriate, as provided for in the CAA. Following issuance of the final PA, the Agency publishes a proposed rule, followed by a public comment period. Taking into account comments received on the proposed rule, the Agency issues a final rule to complete the rulemaking process.

1.2.3 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 from natural sources. Particles may be emitted directly or formed in the atmosphere by transformations of gaseous emissions such as sulfur oxides (SO_x), nitrogen oxides (NO_x), and volatile organic compounds (VOC). 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. Table 1-1 summarizes the NAAQS that have been promulgated for PM to date. These reviews are briefly described below.

The EPA first established NAAQS for PM in 1971, based on the original air quality 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 PM from TSP to PM_{10}, the latter including particles with a mean aerodynamic diameter[6] less than or equal to 10 μm, which delineates that subset of inhalable particles small enough to penetrate to the thoracic region (including the tracheobronchial and alveolar regions) of the respiratory tract (referred to as thoracic particles). The 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).

[6] The more precise term is 50 percent cutpoint 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.

Table 1-1. Summary of National Ambient Air Quality Standards Promulgated for Particulate Matter 1971-2006[7]

Final Rule	Indicator	Ave. Time	Level	Form
1971 (36 FR 8186)	**TSP - Total Suspended Particles** (≤ 25-45 μm)	**24-hour**	**260 μg/m^3** (primary) **150 μg/m^3** (secondary)	Not to be exceeded more than once per year
		Annual	**75 μg/m^3** (primary)	Annual average
1987 (52 FR 24634)	**PM_{10}**	**24-hour**	**150 μg/m^3**	Not to be exceeded more than once per year on average over a 3-year period
		Annual	**50 μg/m^3**	Annual arithmetic mean, averaged over 3 years
1997 (62 FR 38652)	**$PM_{2.5}$**	**24-hour**	**65 μg/m^3**	98th percentile, averaged over 3 years
		Annual	**15 μg/m^3**	Annual arithmetic mean, averaged over 3 years[8]
	PM_{10}	**24-hour**	**150 μg/m^3**	Initially promulgated 99th percentile, averaged over 3 years; when 1997 standards were vacated, the form of 1987 standards remained in place (not to be exceeded more than once per year on average over a 3-year period)
		Annual	**50 μg/m^3**	Annual arithmetic mean, averaged over 3 years
2006 (71 FR 61144)	**$PM_{2.5}$**	**24-hour**	**35 μg/m^3**	98th percentile, averaged over 3 years
		Annual	**15 μg/m^3**	Annual arithmetic mean, averaged over 3 years[9]
	PM_{10}	**24-hour**	**150 μg/m^3**	Not to be exceeded more than once per year on average over a 3-year period

[7] When not specified, primary and secondary standards are identical.

[8] The level of the 1997 annual $PM_{2.5}$ standard was to be compared to measurements made at the community-oriented monitoring site recording the highest level, or, if specific constraints were met, measurements from multiple community-oriented monitoring sites could be averaged ("spatial averaging"). These criteria and constraints were intended to ensure that spatial averaging would not result in inequities in the level of protection afforded by the $PM_{2.5}$ standards. Community-oriented monitoring sites were specified to be consistent with the intent that a spatially averaged annual standard provide protection for persons living in smaller communities, as well as those in larger population centers.

[9] In the revisions to the PM NAAQS finalized in 2006, EPA tighten the constraints on the spatial averaging criteria by further limiting the conditions under which some areas may average measurements from multiple community-oriented monitors to determine compliance (see 71 FR 61165-61167, October 17, 2006).

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. Most significantly, EPA determined that the fine and coarse fractions of PM_{10} should be considered separately. The Administrator's decision to modify the standards was based on evidence that serious health effects were associated with short- and long-term exposure to fine particles in areas that met the existing PM_{10} standards. The EPA accordingly added new standards, using $PM_{2.5}$, as the indicator for fine particles, and retained PM_{10} as the indicator for regulating coarse particles (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 $PM_{10-2.5}$). The 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. Also, EPA established a new reference method for the measurement of $PM_{2.5}$ in the ambient air and adopted protocols 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 99^{th} percentile of 24-hour PM_{10} concentrations at each monitor in an area. The EPA revised the secondary standards by making them identical in all respects to the primary standards.

Following promulgation of the revised PM NAAQS in 1997, 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. The pre-existing 1987 PM_{10} standards remained in place (65 FR 80776, December 22, 2000). The Court also upheld EPA's determination not to establish more stringent secondary standards for fine particles to address effects on visibility (175 F. 3d at 1027).

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 the ozone (O_3) 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 and D.C. Circuit precedent, the panel also reaffirmed its prior holdings that in setting NAAQS EPA is "not permitted to consider the cost of implementing those standards" Id. at 1040-41.

On EPA's petition for rehearing, the panel adhered to its position on these points. *American Trucking Associations v. EPA*, 195 F. 3d 4 (D.C. Cir. 1999). The full Court of Appeals denied EPA's request for rehearing en banc, with five judges dissenting. Id. at 13.

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).

In October 1997, EPA published its plans for the third periodic review of the air quality criteria and NAAQS for PM (62 FR 55201, October 23, 1997), including the 1997 $PM_{2.5}$ standards and the 1987 PM_{10} standards. After CASAC and public review of several drafts, EPA's NCEA finalized the *Air Quality Criteria Document for Particulate Matter* (henceforth, the

"Criteria Document") in October 2004 (U.S. EPA, 2004) and EPA's OAQPS finalized a technical support document, *Particulate Matter Health Risk Assessment for Selected Urban Areas* (Abt, 2005), and a "Staff Paper," *Review of the National Ambient Air Quality Standards for Particulate Matter: Policy Assessment of Scientific and Technical Information,* in December 2005 (U.S. EPA, 2005).

For the primary fine particle standards, most CASAC PM Panel members favored the option of revising the level of the 24-hour $PM_{2.5}$ standard in the range of 35 to 30 $\mu g/m^3$ with a 98^{th} percentile form, in concert with revising the level of the annual $PM_{2.5}$ standard in the range of 14 to 13 $\mu g/m^3$ (Henderson, 2005a). Most of the members of the CASAC PM Panel also strongly supported establishing a new, secondary $PM_{2.5}$ standard to protect urban visibility and recommended establishing a sub-daily (4- to 8-hour averaging time) $PM_{2.5}$ standard within the range of 20 to 30 $\mu g/m^3$ with a form within the range of the 92^{nd} to 98^{th} percentile (Henderson, 2005a). For thoracic coarse particles, there was general concurrence among CASAC PM Panel members to revise the PM_{10} standards by establishing a primary standard specifically targeted to address particles in the size range of 2.5 to 10 μm. The CASAC PM Panel was also in general agreement "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." Based on its review of the Staff Paper, there was general agreement among the CASAC PM Panel members that a 24-hour $PM_{10-2.5}$ standard with a level in the range of 50 to 70 $\mu g/m^3$, with a 98^{th} percentile form, was reasonably justified and that a $PM_{10-2.5}$ standard with an annual averaging time was not warranted (Henderson, 2005b).

On January 17, 2006, EPA proposed to revise the primary and secondary NAAQS for PM (71 FR 2620) and solicited comment on a broader range of options. Proposed revisions included: revising the level of the 24-hour $PM_{2.5}$ primary standard to 35 $\mu g/m^3$; revising the form, but not the level, of the annual $PM_{2.5}$ primary standard by tightening the constraints on the use of spatial averaging; replacing the 24-hour PM_{10} primary standard with a 24-hour standard defined in terms of a new indicator, $PM_{10-2.5}$ [10] set at a level of 70 $\mu g/m^3$; revoking the annual PM_{10} primary standard; and revising the secondary standards by making them identical in all respects to the proposed suite of primary standards for fine and coarse particles. Subsequent to the proposal, CASAC provided additional advice to EPA in a letter to the Administrator requesting

[10] This proposed indicator was qualified so as to include any ambient mix of $PM_{10-2.5}$ that is dominated by particles generated by high-density traffic on paved roads, industrial sources, and construction sources, and to exclude any ambient mix of particles dominated by rural windblown dust and soils and agricultural and mining sources.

reconsideration of CASAC's recommendations for both the primary and secondary $PM_{2.5}$ standards as well as the standards for thoracic coarse particles (Henderson, 2006a).

On September 21, 2006, EPA announced its final decisions to revise the primary and secondary NAAQS for PM to provide increased protection of public health and welfare, respectively (71 FR 61144, October 17, 2006). With regard to the primary and secondary standards for fine particles, EPA revised the level of the 24-hour $PM_{2.5}$ standard to 35 $\mu g/m^3$, retained the level of the annual $PM_{2.5}$ annual standard at 15 $\mu g/m^3$, and revised the form of the annual $PM_{2.5}$ standard by narrowing the constraints on the optional use of spatial averaging. The EPA revised the secondary standards for fine particles by making them identical in all respects to the primary standards. With regard to the primary and secondary standards for thoracic coarse particles, EPA retained PM_{10} as the indicator for coarse particles, retained the level and form of the 24-h PM_{10} standard (so the standard remained at a level of 150 $\mu g/m^3$ with a one expected exceedance form), and revoked the annual standard. The Agency concluded that dosimetric, toxicological, occupational and epidemiologic evidence supported retention of a primary standard that included all thoracic coarse particles (i.e. both urban and non-urban), consistent with the Act's requirement that primary NAAQS provide an adequate margin of safety. EPA further concluded that retention of PM_{10} as an indicator for thoracic coarse particles was reasonable, notwithstanding that PM_{10} measures both fine and coarse particles. The Agency reasoned that because fine particle levels are generally higher in urban areas, a PM_{10} standard set at a single unvarying level will generally result in less coarse particles being allowed in urban areas than in non-urban areas. This was considered to be an appropriate targeting of protection given that the strongest evidence of effects associated with thoracic coarse particles came from epidemiologic studies conducted in urban areas. The Agency concluded that epidemiologic evidence for both urban and non-urban coarse particles supported retention of the existing level of the 24-h standard, 150 $\mu g/m^3$.

In the same rulemaking revising the PM NAAQS, EPA also included a new Federal Reference Method (FRM) for the measurement of $PM_{10-2.5}$ in the ambient air (71 FR 61212 to 61213, October 17, 2006). Although the standards for thoracic coarse particles do not use a $PM_{10-2.5}$ indicator, the new FRM for $PM_{10-2.5}$ was established to provide a basis for approving Federal Equivalent Methods (FEMs) and to promote the gathering of scientific data to support future reviews of the PM NAAQS. With an FRM, researchers will likely include $PM_{10-2.5}$ measurements of thoracic coarse particles in health studies either by directly using the FRM or by utilizing approved FEMs.

In making final decisions for the $PM_{2.5}$ NAAQS in 2006, the Administrator relied primarily on evidence-based considerations to inform his conclusions on the levels for the 24-hour and annual standards. The Administrator believed, at that time, that the estimates of risks likely to remain upon attainment of the 1997 suite of $PM_{2.5}$ standards were indicative of risks that could be reasonably judged important from a public health perspective, and, thus, supported revision of the standards. The quantitative risk assessment provided information supporting the need to revise the $PM_{2.5}$ standards. However, the Administrator judged that this assessment had important limitations and did not provide an appropriate basis for selecting either the level of the 24-hour or annual $PM_{2.5}$ standard. The Administrator more heavily weighed the implications of the uncertainties associated with the quantitative risk assessment than CASAC apparently did in their comments on the proposed rulemaking. He also disagreed with CASAC and many public commenters that the risk assessment results could appropriately serve as a primary basis for a decision for the level of either the 24-hour or the annual $PM_{2.5}$ standards.[11]

Following issuance of the final rule, CASAC articulated its concern that "EPA's final rule on the NAAQS for PM does not reflect several important aspects of the CASAC's advice" (Henderson et al, 2006b). With respect to the 24-hour primary $PM_{2.5}$ standard, CASAC was pleased with the Agency's decision to revise the level to 35 $\mu g/m^3$, which was within the range recommended by most members of the CASAC PM Panel. With respect to the $PM_{2.5}$ annual standard, CASAC expressed serious concerns regarding the decision to retain the level of the standard at 15 $\mu g/m^3$. Specifically, CASAC stated:

> It is the CASAC's consensus scientific opinion that the decision to retain without change the annual $PM_{2.5}$ standard does not provide an "adequate margin of safety… requisite to protect the public health" (as required by the Clean Air Act), leaving parts of the population of this country at significant risk of adverse health effects from exposure to fine PM….to our knowledge there is no science, medical or public health group that disagrees with this very important aspect of the CASAC's recommendations (Henderson et. al, 2006b, p.2).

With regard to EPA's final decision to retain PM_{10} as the indicator for coarse particles and to retain the 24-hour standard at a level of 150 $\mu g/m^3$, CASAC acknowledged concerns associated with retaining this standard while recognizing the need to have a standard in place to protect against effects associated with short-term exposures to thoracic coarse particles. Specifically, CASAC stated:

[11] See discussion in Section II.F of the preamble to the final rule, 71 FR 61167-61177, October 17, 2006.

> Furthermore, the CASAC was completely surprised at the decision in the final PM NAAQS to revert to the use of PM_{10} as the indicator for coarse particles. In our September 15, 2005 letter, the CASAC recommended a new indicator of $PM_{10-2.5}$, which EPA put forward in its proposed rule for the PM NAAQS. The option of retaining the existing daily PM_{10} standard of 150 $\mu g/m^3$ was not discussed during the advisory process, and in fact the CASAC views this as highly-problematic since PM_{10} includes both fine and coarse particulate matter. The Committee acknowledges the need for the Agency to increase its understanding of the health risks of coarse particles and is concerned that ongoing dependence on PM_{10} sampling as an imprecise measure of coarse particulate matter will provide inadequate information on coarse PM concentrations, compositions and exposures in both urban and rural areas. However, the CASAC agrees that having a standard for PM_{10} is better than no standard at all for coarse particles, and was pleased with the Agency's decision against offering exemptions to specific industries (i.e., agricultural, mining) in its regulation of coarse particles (Henderson et. al, 2006b, p.2).

With regard to EPA's final decision to revise the secondary $PM_{2.5}$ standards to be identical in all respects to the revised primary $PM_{2.5}$ standards, CASAC expressed concerns that CASAC's advice to establish a distinct secondary standard for fine particles to address visibility impairment was not followed. Specifically, CASAC stated:

> In our June 6, 2005 letter, the CASAC affirmed the recommendation of Agency staff regarding a separate secondary fine particle standard to protect visibility. This sub-daily secondary $PM_{2.5}$ standard is a better indicator of visibility impairment than the 24-hour primary standard. The CASAC wishes to emphasize that continuing to rely on primary standards to protect against all PM-related adverse environmental and welfare effects assures neglect, and will allow substantial continued degradation, of visual air quality over large areas of the country (Henderson et. al, 2006b, p.2).

1.2.4 Litigation Related to the 2006 PM Standards

Several parties filed petitions for review following promulgation of the revised PM NAAQS in 2006. These petitions addressed the following issues: (1) selecting the level of the annual primary $PM_{2.5}$ standard, (2) setting the secondary $PM_{2.5}$ standards identical to the primary standards; (3) retaining PM_{10} as the indicator for coarse particles and retaining the level and form of the 24-hour PM_{10} standard, and (4) revoking the PM_{10} annual standard.

1.2.4.1 Fine Particles

On judicial review, the D.C. Circuit remanded the primary annual $PM_{2.5}$ NAAQS to EPA because the Agency failed to adequately explain why the standard provided the requisite protection from both short- and long-term exposures to fine particles including protection for at

risk populations. The court also remanded the secondary $PM_{2.5}$ NAAQS to EPA because the Agency failed to adequately explain why the standards provided the required protection from visibility impairment. *American Farm Bureau Federation v. EPA*, 559 F. 3d 512, (D.C. Cir. 2009).

With respect to human health protection from short-term $PM_{2.5}$ exposures, the court considered the different approaches used by EPA in the 1997 and 2006 PM NAAQS decisions. In the1997 decision, EPA determined that the primary annual $PM_{2.5}$ standard was the generally controlling standard for lowering both short- and long-term $PM_{2.5}$ ambient concentrations and the 24-hour standard was set to "provide an adequate margin of safety against infrequent or isolated peak concentrations that could occur in areas that attain the annual standard" (62 FR 38676-77, July 18, 1997). In the 2006 decision, the Administrator considered it appropriate to use a different evidence-based approach from that used in 1997 to set the level of the 24-hour and annual $PM_{2.5}$ standards. In the 2006 decision, the Administrator relied upon evidence from the short-term exposure studies as the principal basis for selecting the level of the 24-hour $PM_{2.5}$ standard and relied upon evidence from the long-term exposure studies as the principal basis for selecting the level of the annual $PM_{2.5}$ standard. The court found EPA failed to adequately explain this change in approach in light of CASAC and staff's recommendations to do otherwise. The court also found that EPA failed to adequately explain why a 24-hour $PM_{2.5}$ standard by itself would provide the protection needed from short-term exposures and remanded the annual $PM_{2.5}$ standard to EPA "for further consideration of whether it is set at a level requisite to protect the public health while providing an adequate margin of safety from the risk of short-term exposures to $PM_{2.5}$." *American Farm Bureau Federation*, 559 F. 3d at 520-24. .

With respect to protection from long-term exposure to fine particles, the court found that EPA failed to adequately explain how the current primary annual $PM_{2.5}$ standard provided an adequate margin of safety in children and other vulnerable subpopulations. Specifically, the court found that EPA did not provide a reasonable explanation of why certain studies, including a study of children in Southern California showing lung damage associated with long-term $PM_{2.5}$ exposure (Gauderman et.al, 2000) and a multi-city study (24-Cities Study) evaluating decreased lung function in children associated with long-term $PM_{2.5}$ exposures (Raizenne et al., 1996), did not call for a more stringent annual $PM_{2.5}$ standard. Id. at 522-23. Second, the court found that

> "EPA was unreasonably confident that, even though it relied solely upon long-term mortality studies, the revised standard would provide an adequate margin of safety with respect to morbidity among children. Notably absent from the final rule, moreover, is any indication of how the standard will adequately reduce risk to the elderly or to those

> with certain heart or lung diseases despite (a) the EPA's determination in its proposed rule that those subpopulations are at greater risk from exposure to fine particles and (b) the evidence in the record supporting that determination." *American Farm Bureau Federation*, 559 F. 3d at 525-26.

The court also remanded the secondary standards for fine particles, based on EPA's failure to adequately explain why setting the secondary PM NAAQS equivalent to the primary standards provided the required protection for public welfare including protection from visibility impairment. The court found that EPA failed to identify a target level of visibility impairment that would be requisite to protect public welfare. The court determined that this was contrary to the statute and resulted in a lack of a reasoned basis for the final decision. In addition, the court found that EPA's near exclusive reliance on a comparison of numbers of counties that would be in nonattainment under alternative standards scenarios was an inadequate basis for making a decision. The court concluded that EPA did not take into account the relative visibility protection of the alternative standards nor did the Agency consider the failure of a 24-hour $PM_{2.5}$ standard to address regional differences in humidity and its effect on visibility. Id. at 528-31.

1.2.4.2 Thoracic Coarse Particles

The court upheld EPA's decision to retain the primary 24-hour PM_{10} NAAQS to provide protection for coarse particle exposures and to revoke the annual PM_{10} standard. The court found that EPA reasonably included all coarse PM within the standard, both of urban and non-urban origin, to protect all of the country from exposure to coarse PM. The court rejected arguments that the evidence showed there are no risks from exposure to non-urban coarse PM. Id. at 531-33. The court further found that EPA had a reasonable basis to not set separate standards for urban and non-urban coarse PM, namely the inability to reasonably define what ambient mixes would be included under either 'urban' or 'non-urban' and that the evidence in the record supported EPA's cautious decision to provide "some protection from exposure to thoracic coarse particles… in all areas." Specifically, the court stated,

> Although the evidence of danger from coarse PM is, as EPA recognizes, "inconclusive," (71 FR 61193, October 17, 2006), the agency need not wait for conclusive findings before regulating a pollutant it reasonably believes may pose a significant risk to public health. The evidence in the record supports the EPA's cautious decision that "some protection from exposure to thoracic coarse particles is warranted in all areas." *Id.* As the court has consistently reaffirmed, the CAA permits the Administrator to "err on the side of caution" in setting NAAQS.

559 F. 3d at 533 (block citation of cases omitted)

The court also upheld EPA's decision to use PM_{10} as the indicator for coarse particles, and to retain the level of the standard at 150 $\mu g/m^3$. The EPA's final rule acknowledged that evidence of harm from urban-type coarse PM is stronger than for other types of coarse particles, and targeted protection at areas where urban-type coarse PM is most likely present (71 FR 61185-61203, October 17, 2006). The targeting is done by using the indicator PM_{10} for coarse particles. PM_{10} includes both coarse and fine particles. Urban and industrial areas tend to have higher levels of fine particles than rural areas, so that in those areas less coarse particles are allowed – the desired targeting. Conversely, fine particle levels tend to be lower in rural areas, so more coarse particles are allowed in those areas – again the desired targeting. Likewise, the court concluded that EPA's choice of the level for the PM_{10} standard was reasonable, for many of the same reasons. *American Farm Bureau Federation* at 533-36. The court also upheld EPA's decision to revoke the annual PM_{10} standard. Id. at 537-38.

1.2.5 Current PM NAAQS Review

The EPA initiated the current review of the air quality criteria for PM in June 2007 with a general call for information (72 FR 35462, June 28, 2007). EPA invited a wide range of external experts, as well as EPA staff, representing a variety of areas of expertise (e.g., epidemiology, human and animal toxicology, statistics, risk/exposure analysis, atmospheric science, ecology, biological sciences) to participate in two workshops: (1) Workshop to Discuss Policy-Relevant Science to Inform EPA's Integrated Plan for the Review of the Primary PM NAAQS (conducted July 11-13, 2007 in Research Triangle Park, NC) and (2) Workshop to Discuss Policy-Relevant Science to Inform EPA's Integrated Plan for the Review of the Secondary PM NAAQS (conducted July 16, 2007 in Chapel Hill, NC) (72 FR 34003 and 34005, June 20, 2007).[12] These workshops provided an opportunity for the participants to broadly discuss the key policy-relevant issues around which EPA would structure the PM NAAQS review and to discuss the most meaningful new science that would be available to inform our understanding of these issues.

Based in part on the workshop discussions, EPA developed a draft IRP outlining the schedule, the process, and the key policy-relevant science issues that would guide the evaluation of the air quality criteria for PM and the review of the primary and secondary PM NAAQS. On November 30, 2007, EPA held a consultation with CASAC on the draft IRP (72 FR 63177,

[12] See workshop materials http://www.regulations.gov/search/Regs/home.html#home Docket ID numbers EPA-HQ-OAR-2007-0492-008; EPA-HQ-OAR-2007-0492-009; EPA-HQ-OAR-2007-0492-010; and EPA-HQ-OAR-2007-0492-012.

November 8, 2007). Public comments were also requested on the draft plan and presented at that CASAC teleconference. The final IRP incorporated comments received from CASAC and the general public on the draft plan as well as input from senior Agency managers (US EPA, 2008a).

As part of the process of preparing the PM ISA, NCEA hosted a peer review workshop in June 2008 on preliminary drafts of key ISA chapters (73 FR 30391, May 27, 2008). The first external review draft ISA (US EPA, 2008b) was reviewed by CASAC at a meeting held in April 2009 (74 FR 2688, February 19, 2009). Based on CASAC and public comments, NCEA revised the draft ISA and released a second draft ISA (US EPA, 2009a) in July 2009 for CASAC review and public comment at an upcoming public meeting to be held on October 5-6, 2009.

In preparing the REA documents that build on the scientific evidence presented in the ISA, OAQPS released two planning documents, *Particulate Matter National Ambient Air Quality Standards: Scope and Methods Plan for Health Risk and Exposure Assessment* and *Particulate Matter National Ambient Air Quality Standards: Scope and Methods Plan for Urban Visibility Impact Assessment* (henceforth, Scope and Methods Plans)[13] outlining the scope, approaches, and key issues that staff planned to use in conducting the quantitative assessments (US EPA, 2009d,e). We considered CASAC comments on the Scope and Methods Plans made during an April 2009 consultation as well as public comments (74 FR 11580, March 18, 2009) in designing and conducting the assessments. OAQPS released two draft assessment documents, *Risk Assessment to Support the Review of the $PM_{2.5}$ Primary National Ambient Air Quality Standards: External Review Draft* - September 2009 (US EPA 2009b) and *Particulate Matter Urban-Focused Visibility Assessment - External Review Draft* - September 2009 (US EPA, 2009c) for CASAC review and public comment at the upcoming public meeting on October 5-6, 2009.

This document, a preliminary draft PA, builds upon the information presented in the second draft ISA and the two draft assessment documents. As noted above, this preliminary draft PA is being released at this time, prior to the upcoming CASAC meeting, for informational purposes and to facilitate discussion with the CASAC PM Panel at the upcoming meeting on the overall structure, areas of focus, and level of detail to be included in an external review draft PA. We will consider CASAC review and public comments on the second draft ISA and the two draft assessment documents, as well as CASAC discussion of this preliminary draft PA, in subsequently preparing a complete external review draft PA. We plan to release a complete

[13] EPA-452/P-09-001 and -002; February 2009 ; Available: http://www.epa.gov/ttn/naaqs/standards/pm/s_pm_2007_pd.html

external review draft PA later this year for review by the CASAC PM Panel and the public in early 2010.

1.3 GENERAL APPROACH AND ORGANIZATION OF THE DOCUMENT

The PA will include staff's evaluation of the policy implications of the scientific assessment of the evidence presented in the ISA and results of quantitative assessments based on that evidence, taking into consideration CASAC advice and public comments. Taken together, this information will inform various conclusions and the identification of policy options for the Administrator to consider in addressing public health and welfare effects associated with exposure to ambient PM.

Partly as a consequence of EPA's decision in the last review to continue 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, $PM_{10-2.5}$. Since the purpose of this review is to evaluate the adequacy of the current standards, which separately address fine and thoracic coarse particles, staff is focusing 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, we are considering PM_{10}-related evidence primarily to help inform our understanding of key issues and to help interpret and provide context for understanding the public health and welfare impacts of ambient fine and coarse particles.

Following this introductory chapter, this preliminary draft PA is organized into three main parts: the characterization of ambient PM (chapter 2), PM-related health effects and primary PM NAAQS (chapters 3, 4 and 5), and PM-related welfare effects and secondary PM NAAQS (chapters 6 and7). 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 subsequent chapters of this document.

Chapters 3 through 5 comprise the second main part of this policy assessment document 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 second draft ISA, and an examination of issues related to the quantitative assessment of health risks based on the epidemiologic health evidence. Chapter 4, to be developed for inclusion in the external review draft, will present key results and observations from a quantitative assessment of $PM_{2.5}$-related health risks, including risk estimates for current

air quality levels as well as those associated with just meeting the current $PM_{2.5}$ NAAQS and alternative standards under consideration. In this preliminary draft, chapter 4 is previewed by way of an annotated outline. Chapter 5, to be developed for inclusion in the external review draft, will present staff observations and conclusions related to the current primary standards for fine and thoracic coarse particles. As outlined in the table of contents of this preliminary draft, chapter 5 is planned to begin with a discussion of the broad approach used by staff in this review, generally reflecting consideration of both evidence-based and quantitative risk-based considerations. The discussion is then planned to focus on consideration of the adequacy of the current PM standards, staff conclusions as to alternative indicators, averaging times, levels and forms, and staff conclusions with regard to potential 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. The emphasis of this chapter is on visibility impairment, reflecting the availability of a significant amount of policy-relevant information and related assessments, which serve as the basis for staff consideration of a distinct secondary standard specifically for protection of visual air quality. Chapter 7, to be developed for inclusion in the external review draft, will present staff observations and conclusions related to the current secondary standards. As outlined in the table of contents of this preliminary draft, chapter 7 is planned to begin with a discussion of the approach used by staff in this review. The discussion is planned to focus on consideration of the adequacy of the current standards, staff conclusions as to alternative indicators, averaging times, levels and forms, and staff conclusions with regard to potential standards for consideration by the Administrator.

As noted above, we recognize that this preliminary draft PA is incomplete and that much work needs to be done to prepare an external review draft PA. Nonetheless, we decided to release this preliminary draft at this time both for informational purposes, to illustrate the most recent change made to the NAAQS review process with regard to the policy assessment, and to have the benefit of early discussion with the CASAC PM Panel on the overall structure, areas of focus, and level of detail to be included in an external review draft PA.

1.4 REFERENCES

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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 much more extensive treatment of these subjects in the second draft Integrated Science Assessment (ISA, US EPA, 2009a), the 2004 Air Quality Criteria Document (AQCD, US EPA, 2004) and US EPA, 2005 (US EPA, 2005) from the review completed in 2006, and other sources. (*To assist reviewers of this preliminary draft,* some *paragraphs that have been copied or condensed from one of these documents, but would not otherwise be easily identified as such, are denoted by a parenthetical comments in italics at the end of the paragraph. In some cases, these comments alert the reviewer that there has been augmentation of the original text for clarity or accuracy or to bring the material up to date.)*

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 describes the design and locations of ambient PM monitoring networks. Section 2.5 presents data on PM concentrations, trends, and spatial patterns in the U.S. Section 2.6 provides information on the temporal variability of PM. Much of the information in Sections 2.5 and 2.6 is derived from analyses of data collected by the nationwide networks of $PM_{2.5}$ and PM_{10} monitors in 2005 through 2007. Section 2.7 defines and discusses background levels of ambient PM. Section 2.8 addresses the relationships between ambient PM levels and human exposure to PM. Section 2.9 addresses the relationship between ambient $PM_{2.5}$ levels and visibility impairment.

2.2 PROPERTIES OF AMBIENT PM

Particulate matter 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. Classes of particles have historically been characterized largely in terms of particle size. Fine particles and coarse particles, which are defined below, 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. (*US EPA, 2005, section 2.2*)

2.2.1 Particle Size Distribution

2.2.1.1 Modes

The diameters of atmospheric particles span 5 orders of magnitude, ranging from 0.001 micrometers to 100 micrometers (μm).[14] 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. (*US EPA, 2005, section 2.2.1*)

Atmospheric chemical and microphysical processing of direct emissions of PM and its precursors together with mechanical generation of particles tend to produce distinct lognormal modes (Whitby, 1978 as shown in Figure 2-1). Discussions in this and subsequent sections will focus on particles in specific size ranges (i.e., $PM_{2.5}$, $PM_{10\text{-}2.5}$, and PM_{10}). The subscripts after PM refer to the aerodynamic diameter in micrometers (μm) of 50% cut points of sampling devices, see section 2.2.1.2. For example, EPA defines PM_{10} as particles collected by a sampler with an upper 50% cut point of 10 μm aerodynamic diameter and a specific, fairly sharp penetration curve, as defined in the Code of Federal Regulations (40 CFR Part 58). $PM_{2.5}$ is defined in an analogous way. Ultrafine particles, defined here as particles with a diameter less than or equal to 0.1 μm (typically based on physical size, thermal diffusivity, or electrical mobility), will also be discussed. (*US EPA, 2009a, section 3.2*)

In present usage, the term "fine particles" is most often associated with the $PM_{2.5}$ fraction, which includes the nucleation, Aitken, and accumulation modes and some particles

[14] In this preliminary draft Policy Assessment, 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 (US EPA 2009a, section 3.2).

from the lower-size tail of the coarse particle mode between about 1 and 2.5 μm aerodynamic diameter. "Thoracic coarse particles" are frequently used in reference to $PM_{10-2.5}$, which does not include the low-end tail of the coarse particle mode. With high relative humidity, larger hygroscopic particles in the accumulation mode could also extend into the 1 to 3 μm size range. These relationships can be seen in Figure 2-1, which shows the number distribution for ultrafine particles and the volume distribution (or mass distribution if particle density is constant across the size range) for fine and (thoracic) coarse particles. The figure is arranged this way because particle number is most highly concentrated in the ultrafine size range, but volume (or mass) is most concentrated in the larger size ranges. (*US EPA, 2009a, section 3.2*)

Figure 2-1. Particle size distributions by number and volume.

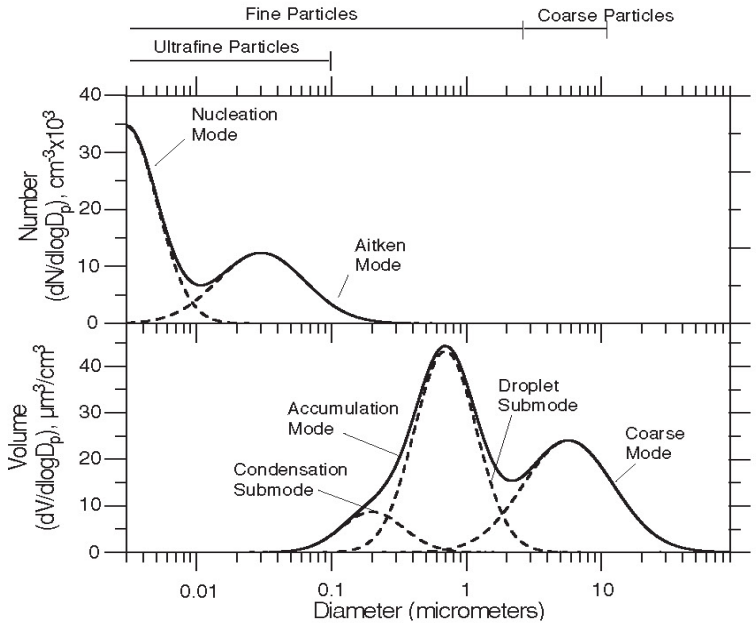


Figure 2-1 Notes: Dashed lines refer to values in individual modes and solid lines to their sum. Note that ultrafine particles are a subset of fine particles. (US EPA, 2009a, Figure 3-1). Source: Pandis (2004).

Characterizing particle size is important because different size particles penetrate to different regions of the human respiratory tract (see further discussion in section 3.2). Most commonly, $PM_{2.5}$ is used as an indicator of fine particles, $PM_{10-2.5}$ is used as an indicator of the thoracic component of coarse particles that is sometimes referred to as thoracic coarse (noting that it excludes some coarse particles below 2.5 μm and above 10 μm), and, PM_{10} is the sum of fine and thoracic coarse particles.. In the current NAAQS for PM, PM_{10} is used as the indicator for thoracic coarse particles. (*US EPA, 2009a, section 3.2*)

As can be seen from Table 2-1, particles in individual size modes are characterized by rather distinct sources, composition, chemical properties, lifetimes in the atmosphere (τ), and distances over which they can travel. Whereas particles in the smaller size modes are formed mainly by combustion processes and by nucleation and condensation of gases, coarse particles are generated mainly by mechanical activity, such as the action of wind on either the ground or the sea surface or construction or resuspension by traffic. Among the mechanical activities that generate coarse particles are various types of material handling and resuspension of dust at industrial facilities such as metallurgical operations, cement kilns, mines, quarries, etc. Particles in the ultrafine size range are either emitted directly to the atmosphere or are formed by nucleation of gaseous constituents in the atmosphere as shown in Table 2-2. (US EPA, 2005, *Table 2-1; US EPA 2009a, Table 3-1 and section 3.2; ISA text augmented*)

2.2.1.2 Sampler Cutpoints

Particle samplers typically use size-selective inlets that are defined by their 50 percent cutpoint, 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 "PMx", 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, although for particles on the order of 1-10 nm, many may diffuse to sampler surfaces before being captured on the filter. The objective of size-selective sampling is usually to measure particle size fractions that provide a relationship to human health impacts, visibility impairment, deposition, or emissions sources. (US EPA, 2005, section 2.2.1.2, modified regarding particles 1-10 nm).

Table 2-1. Particle Size Fraction Terminology

Term	Description
Size Distribution Modes	
Coarse Particles	The distribution or 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 μm.
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 referred to as thoracic particles. See 40 CFR Part 50, Appendix G.
$PM_{2.5}$	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 indicator 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. See 40 CFR Part 50, Appendix J.
$PM_{10-2.5}$	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 coarse particles; also referred to as thoracic coarse particles or coarse-fraction particles. See 40 CFR Part 50, Appendix O.

Source: modification of US EPA, 2005, Table 2-1

Table 2-2. Characteristics of ambient fine (ultrafine plus accumulation-mode) and coarse particles. (US EPA, 2009a, Table 3-1)

	Fine		Coarse
	Ultrafine	**Accumulation**	
Formation Processes	Combustion, high-temperature processes, and atmospheric reactions		Break-up of large solids/droplets
Formed by	Nucleation of atmospheric gases including H_2SO_4, NH_3 and some organic compounds Condensation of gases	Condensation of gases Coagulation of smaller particles 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 EC Metal compounds Organic compounds with very low saturation vapor pressure at ambient temperature	Sulfate, nitrate, ammonium, and hydrogen ions EC Large variety of organic compounds Metals: compounds of Pb, Cd, V, Ni, Cu, Zn, Mn, Fe, etc. Particle-bound water Bacteria, viruses	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 Bacteria, pollen, mold, fungal spores, plant and animal debris
Solubility	Not well characterized	Largely soluble, hygroscopic, and deliquescent	Largely insoluble and nonhygroscopic
Sources	High temperature combustion Atmospheric reactions of primary, gaseous compounds.	Combustion of fossil and biomass fuels, and high temperature industrial processes, smelters, refineries, steel mills etc. Atmospheric oxidation of NO_2, SO_2, and organic compounds, including biogenic organic species (e.g., terpenes)	Resuspension of particles deposited onto roads Tire, brake pad, and road wear debris Suspension from disturbed soil (e.g., farming, mining, unpaved roads) Construction and demolition Fly ash from uncontrolled combustion of coal, oil, and wood Ocean spray
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 (100s to 1,000s of km in dust storms for the small size tail)

Source: Wilson and Suh (1997) (adapted). US EPA, 2009a, Table 3-1.

Note: Coarse column may not yet adequately summarize industrial processes emitting coarse PM

Since 1987, EPA has defined indicators of PM for the PM NAAQS using cut points of interest. 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).[15] TSP typically includes particles with diameters less than about 40 μm, but the upper size cut varies substantially with placement, wind velocity, and wind direction relative to the non-cylindrically symmetrical shape of the sampler inlet. TSP samplers are still used in state regulatory monitoring networks, but only to monitor for lead (Pb) and other toxic metals. When EPA established new PM standards in 1987, the selection of PM_{10} as the new indicator was intended to focus regulatory attention on particles small enough to be inhaled and to penetrate into the thoracic region of the human respiratory tract. In 1997, EPA established standards for fine particles measured as $PM_{2.5}$ (i.e., the fine fraction of PM_{10}). Following the establishment of the 2.5 and 10 micron size cuts as indicators for the PM NAAQS, state monitoring networks have employed samplers with those cutpoints almost exclusively. Ongoing research networks and special monitoring studies for health effects research also are dominated by samplers with these cutpoints, with some use of samplers with other cutpoints. (*US EPA, 2005, section 2.2.1.2, augmented*)

In the PM NAAQS review completed in 2006, EPA defined but did not set a standard 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). The EPA also established a Federal Reference Method for measuring $PM_{10-2.5}$, to facilitate consistency in future research and air quality characterization relative to this indicator (71 FR 61212 to 61213, October 17, 2006).

2.2.2 Sources and Formation Processes

Particulate matter is composed of both primary (derived directly from emissions) and secondary (derived from atmospheric reactions involving gaseous precursors) components. Table 2-3 summarizes anthropogenic and natural sources for the major primary and secondary aerosol constituents of fine and coarse particles. Anthropogenic sources can be further divided into stationary and mobile sources. Stationary sources include fuel combustion for electrical utilities, residential space heating, and cooking; industrial processes; construction and demolition; metal, mineral, and petrochemical processing; wood products processing; mills and elevators used in agriculture; erosion from tilled lands; waste disposal and recycling; and

[15] 40 CFR Part 50, Appendix B, Reference Method for the Determination of Suspended Particulate Matter in the Atmosphere (High-Volume Method).

biomass combustion. Biomass combustion encompasses many emission activities including burning of wood for fuel, burning of vegetation to clear land for agriculture and construction, to dispose of agricultural and domestic waste, to control the growth of animal or plant pests, and to manage forest resources (prescribed burning). Wildlands also burn due to lightning strikes and arson. Mobile or transportation-related sources include direct emissions of primary PM and secondary PM precursors from highway vehicles and non-road sources as well as fugitive dust from paved and unpaved roads. Also shown in Table 2-3 are sources for several precursor gases, the oxidation of which can form secondary PM. Only major sources for each constituent within each broad category shown at the top of Table 2-3 are listed. Not all sources are equal in magnitude (*US EPA, 2009a, section 3.3*).

In general, the sources of fine particles are very different from those of coarse particles. Some of the mass in the fine size fraction forms during combustion from material that has volatilized in combustion chambers and then recondensed before after emission to the atmosphere. Some ambient $PM_{2.5}$ forms in the atmosphere from photochemical reactions involving precursor gases. Included in this category is the formation of new ultrafine particles by homogeneous nucleation of precursor gases in addition to the condensation of gases on pre-existing particles. Particulate matter formed by the first mechanism is referred to as primary, and PM formed by the second mechanism is referred to as secondary. Biological material also exists in the fine fraction including many types of microorganisms, especially viruses and bacteria and fragments of pollens and fungal spores. $PM_{10-2.5}$ is mainly primary in origin, as it is produced by surface abrasion or by suspension of biological material and fragments of living things (e.g., plant and insect debris). In addition, atmospheric reaction products condense on coarse particles. Some industrial facilities employ both combustion and abrasion/suspension processes, and thus can be significanat emitters of both fine and coarse particles. Because precursor gases undergo mixing during transport from their sources and reactions in the atmosphere can produce the same products, it is difficult to identify individual sources of secondary PM. Transport and transformation of precursors can occur over distances of hundreds of kilometers. $PM_{10-2.5}$ has a shorter lifetime in the atmosphere, so its effects tend to be more localized. However, intercontinental transport of dust from African and Asian deserts occurs, and some of this material is in the $PM_{10-2.5}$ size range. Major events are highly episodic but much smaller contributions can be made at other times. (*US EPA, 2009a, section 3.3, augmented regarding industrial sources with multiple processes*) *(Text borrowed from the second draft ISA, but may need amendment as it is not entirely clear (1) what "formed by the first mechanism" refers to*

and more generally (2) whether unreacted homogenous nucleation material and unreacted material condensed on pre-existing particles are being called primary or secondary.)

Precursors to secondary PM have natural and anthropogenic sources, just as primary PM has natural and anthropogenic sources. A substantial fraction of the fine particle mass, especially during the warmer months of the year, is secondary in nature, formed as the result of atmospheric reactions involving both inorganic and organic gaseous precursors. The major atmospheric chemical transformations leading to the formation of particulate nitrate (pNO_3) and sulfate (pSO_4) are relatively well understood, whereas those involving the formation of secondary organic aerosol (SOA) are less well understood. *(US EPA, 2009a, section 3.3.2*)

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. (*US EPA, 2005, section 2.2.2*)

Examples of secondary particle formation include:

- 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);

- 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

- 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.
  (US EPA, 2005 *section 2.2.2*)

Table 2-3. Constituents of atmospheric particles and their major sources

Primary (PM <2.5 µm)			Primary (PM >2.5 µm)		Secondary PM Precursors (PM <2.5 µm)	
Aerosol species	**Natural**	**Anthropogenic**	**Natural**	**Anthropogenic**	**Natural**	**Anthropogenic**
Sulfate (SO_4^{2-})	Sea spray	Fossil fuel combustion	Sea spray	—	Oxidation of reduced sulfur gases emitted by the oceans and wetlands and SO_2 and H_2S emitted by volcanism and forest fires	Oxidation of SO_2 emitted from fossil fuel combustion
Nitrate (NO_3^-)	—	Mobile source exhaust	—	—	Oxidation of NO_X produced by soils, forest fires, and lighting	Oxidation of NO_X emitted from fossil fuel combustion and in motor vehicle exhaust
Minerals	Erosion and re-entrainment	Fugitive dust from paved and unpaved roads, agriculture, forestry, construction, and demolition	Erosion and re-entrainment	Fugitive dust, paved and unpaved road dust, agriculture, forestry, construction, and demolition	—	—
Ammonium (NH_4^+)	—	Mobile source exhaust	—	—	Emissions of NH_3 from wild animals, and undisturbed soil	Emissions of NH_3 from motor vehicles, animal husbandry, sewage, and fertilized land
Organic carbon (OC)	Wildfires	Prescribed burning, wood burning, motor vehicle exhaust, cooking, tire wear and industrial processes	Soil humic matter	Tire and asphalt wear, paved and unpaved road dust	Oxidation of hydrocarbons emitted by vegetation (terpenes, waxes) and wild fires	Oxidation of hydrocarbons emitted by motor vehicles, prescribed burning, wood burning, solvent use and industrial processes
EC	Wildfires	Mobile source exhaust (mainly diesel), wood biomass burning, and cooking	—	Tire and asphalt wear, paved and unpaved road dust	—	—
Metals	Volcanic activity	Fossil fuel combustion, smelting and other metallurgical processes, and brake wear	Erosion, re-entrainment, and organic debris	—	. —	—
Bioaerosols	Viruses and bacteria	—	Plant and insect fragments, pollen, fungal spores, and bacterial agglomerates	—	—	—

Dash (—) indicates either very minor source or no known source of component.
Source: U.S. EPA,(2004).; US EPA, 2009a, Table 3-2)

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[16] (EC) and organic carbonaceous material (OCM), sometimes also referred to as organic aerosol. When only the mass of carbon atoms is being referred to, the term organic carbon (OC) is used. (*US EPA, 2005, section 2.2.2*)

2.2.3 Chemical Composition

As summarized in Table 2-3, a number of chemical components of ambient PM are found predominately in fine particles including: sulfate, ammonium, and hydrogen ions; elemental carbon, secondary organic compounds, and primary organic species from cooking and combustion; and certain metals, primarily from combustion processes. Chemical components found predominately in coarse particles include: crustal-related materials such as calcium, aluminum, silicon, magnesium, and iron; and primary organic materials such as pollen, spores, and plant and animal debris. (*US EPA, 2005, section 2.2.3*)

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. 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

[16] 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. Elemental carbon 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.

particles (i.e., less than 2.5 μm in diameter) in areas with high soil concentrations. (*US EPA, 2005, section 2.2.3*)

Many ambient particles also contain water (i.e., particle-bound water) as a result of an equilibrium between water vapor and hygroscopic PM. 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. 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 carbonaceous material (OCM), and crustal material. (*US EPA, 2005, section 2.2.3*)

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 and composition. Ultrafine particles have a very short life, on the order of minutes to hours, since they are more likely to reach the accumulation mode through agglomeration or condensation mechanisms or to be lost by diffusion to surfaces. 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 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. (*US EPA, 2005, section 2.2.4)*

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. Coarse particles also are readily removed by falling rain drops. (*US EPA, 2005, section 2.2.4)*

2.2.5 Optical Properties of Particles

As discussed in Chapter 9 of the second draft ISA, light extinction is the optical characteristic of the atmosphere that best determines the impact potential of PM on perceived visibility. Light extinction is the loss of light per unit of distance either by scattering out of the beam of light or by absorption, which converts the light to heat. Particulate matter and gases scatter and absorb light. Light scattering by gases (e.g., nitrogen, oxygen, etc.) that comprise the atmosphere (also known as Rayleigh or clear-air scattering) is related to the density of the air, which is sufficiently constant with elevation that it can be considered a known constant value for any location. NO_2 is the only atmospheric gas that absorbs visible light appreciably and its effects are generally small (i.e., less than 5%) compared to PM light extinction, so its contribution to ambient visibility impacts is often ignored. By this assumption, light extinction is approximated as the sum of PM light extinction (includes both scattering and absorption) plus Rayleigh light scattering, where the former characterizes the PM contribution to visibility impacts and the latter is taken to be a time-invariant constant depending only on elevation above sea level.

Light-absorbing carbon (e.g., diesel exhaust soot and smoke) and some crustal minerals are the only commonly occurring airborne particle components that absorb light. All particles scatter light, and generally particle light scattering is the largest of the four light extinction components. While a larger particle scatters more light than a similar shaped smaller particle of the same composition, the light scattered per unit of mass concentration is greatest for particles with diameters from about 0.3 to 1.0 μm. Particles composed of water soluble inorganic salts (i.e., ammoniated sulfate, ammonium nitrate, sodium chloride, etc.) are hygroscopic in that they absorb water as a function of relative humidity to form liquid solution droplets. The droplets become larger when relative humidity increases resulting in increased light scattering, hence the same particulate matter dry concentration produces greater haze levels. Because the extinction efficiency of PM depends on size, composition and humidity effects, there is no simple one-to-one correspondence between PM concentration and PM light extinction. The relationship between ambient PM and visibility impairment is further discussed below in section 2.8.

2.2.6 Other Radiative Properties of Particles

As discussed in section 9.3 of the second draft ISA, PM affects climate through direct and indirect effects. The direct effect is primarily realized as planet brightening when seen from space because most aerosols scatter most of the visible light that reaches them. The IPCC AR4 reported that the radiative forcing from this direct effect was -0.5 (±0.4) W/m^2 and identified the level of scientific understanding of this effect as "medium-low." The global mean direct radiative forcing from individual aerosol components varies from strongly negative for sulfate to positive for black carbon with weaker positive or negative effects for other components, all of which can vary strongly over space and time and with aerosol size. The indirect effects are primarily realized as an increase in cloud brightness (termed the "first indirect" or "Twomey" effect), changes in precipitation, and possible changes in cloud lifetime. The IPCC AR4 reported that the radiative forcing from the Twomey effect was -0.7 (range: -1.1 to +4) and identified the level of scientific understanding of this effect as "low." The other indirect effects from aerosols were not considered to be radiative-forcing.

Taken together, direct and indirect effects from aerosols increase Earth's shortwave albedo or reflectance, thereby reducing the radiative flux reaching Earth's surface from the Sun. This produces net climate cooling from aerosols. The current scientific consensus reported by IPCC AR4 is that the direct and indirect radiative forcing from anthropogenic aerosols computed at the top of the atmosphere, on a global average, is about -1.3 (range: -2.2 to -0.5) W/ m^2. Although the magnitude of this negative radiative forcing appears large in comparison to the analogous IPCC AR4 estimate of positive radiative forcing from anthropogenic GHG of about 2.9 (±0.3) W/ m^2, the spatial and temporal distributions of these two very different radiative forcing agents are dissimilar; therefore, they do not simply cancel, and regional differences can be large. These differences result from the much shorter atmospheric lifetime of aerosols than for the radiatively important trace gases, implying that the radiative effects of aerosols respond much more quickly to changes in emissions than do the effects from the gas-phase forcing agents.

2.3 AMBIENT PM MEASUREMENT METHODS

The measurement of PM can be accomplished with a number of sampling and analysis techniques. In the United States, much of the historical focus on PM methods has been associated with measuring gravimetric mass as Total Suspended Particulate (TSP), which was the PM indicator until 1987, and PM_{10}, which replaced TSP as the PM indicator in 1987. In 1997, a fine particle indicator was added, resulting in PM_{10} becoming the coarse particle indicator. PM_{10} Federal Reference Methods (FRMs) include the high-volume method, low-

volume method, and dichotomous method. The fine particle indicator is based on the $PM_{2.5}$ FRM, which also provides gravimetric mass, is a low volume method (i.e., it operates at 16.67 lpm or one cubic meter per hour), and has been operating throughout the country since 1999. Because PM is a complex mixture of substances with differing physical and chemical properties that vary in time and space, measuring and characterizing particles suspended in the atmosphere is a significant challenge.[17] Thus, to support characterizing the composition of $PM_{2.5}$, chemical speciation samplers have been implemented since 2001 as part of the Chemical Speciation Network (CSN). Additionally, most gravimetric samplers (i.e., the $PM_{2.5}$ FRM) require post-sampling laboratory analysis; therefore, $PM_{2.5}$ continuous mass monitors and associated data systems that provide for near real-time reporting of data have been deployed in most cities since 2004 to support timely reporting and forecasting air pollution to the public through the Air Quality Index (AQI). In addition to gravimetric mass and composition, other PM measurements include particle number, light scattering, and light absorption. Most samplers 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), black carbon (BC), 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

[17] Refer to second draft ISA Chapter 3 for more comprehensive assessments of particle measurement methods. A summary of PM measurement methods is also given in Fehsenfeld et al. (2003). Significant improvements and understanding of routine and advanced measurement methods is available through EPA's PM Supersites Program (see www.epa.gov/ttn/amtic/supersites.html).

weighed before and after collection to determine mass by difference. It should be noted that while filter-based samplers can be programmed to operate over just about any time interval, they are operated by state and local agencies on a schedule of midnight to midnight local standard time. Examples of the most commonly used filter-based samplers include the FRM monitors for $PM_{2.5}$ and PM_{10}. Dichotomous samplers contain a separator (i.e., a virtual impactor) 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. A low-volume dichotomous sampler (operated at 16.7 L/min), based on virtual impaction, was described in the 2004 AQCD (U.S. EPA, 2004, section 2.2.4.1.2). These gravimetric methods require weighing the filters after they 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 25 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 for the latest available three-year period (2005-2007) 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 $PM_{2.5}$ monitoring program. Three-year average estimates from reporting organizations aggregated on a national basis for collocated sampler precision (7.55 %), flow rate accuracy (0.007 %), and method bias (-2.97 %, from the Performance Evaluation Program)[18] are well within their respective goals of $\pm$10 %, $\pm$4 %, and $\pm$10 %. There are currently over 900 $PM_{2.5}$ FRM discrete monitors across the U.S. Of those, about 225 monitors are operating on a daily schedule.

The most widely deployed PM continuous technology 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).

[18] 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 an EPA laboratory and compared in order to assess bias.

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 ISA (ISA, Chapter 3.4.1.1). 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 average is calculated from the measurement of the total aerosol mass concentration, including non-volatile and volatile PM, every 6 minutes.

The other major PM monitoring device that provides for gravimetric mass is the beta attenuation monitor. A beta attenuation (or beta gauge) monitor 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.

In 2006, EPA finalized new performance criteria for approval of $PM_{2.5}$ and $PM_{10-2.5}$ monitors as Federal Equivalent Methods (FEMs). At the same time, EPA also finalized an approach to approving $PM_{2.5}$ continuous methods across an agencies network identified as an Approved Regional Method (ARM). Both the FEM and ARM are based on new performance criteria that utilized a data quality objective process that takes into account the existing data quality objectives of the network with the higher sampling frequency (i.e., daily sampling) provided for by PM continuous monitors. These new performance criteria have led to a number of approved FEMs for $PM_{2.5}$ including technologies that employ beta attenuation as well as use of the TEOM® technology in combination with an environmental conditioning system (i.e., the Thermo FDMS®). A few $PM_{10-2.5}$ technologies have also been approved as FEMs including the Met One BAM providing continuous measurement of coarse particles by difference and the Thermo 2025D, a dichotomous sampler that utilizes the same virtual impactor approved for use on its continuous dichotomous monitor, the FDMS® 1405D. No monitoring agency has applied for ARM approval, although a few were pursuing it until the methods they had implemented were approved as FEM's; thus making an ARM approval no longer needed. In addition to the recently approved $PM_{2.5}$ FEMs, many monitoring agencies continue to use conventional TEOMs and other $PM_{2.5}$ continuous technologies as part of their monitoring programs to report and support forecasting of the AQI. However, these methods are not used for comparison with the NAAQS.

The number of continuous $PM_{2.5}$ monitors across the U.S. has increased to over 700 stations. 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.[19] 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).

2.3.2 Indirect Optical Methods

Particulate matter has also been characterized in the U.S. and elsewhere by indirect optical methods that rely on the light scattering or absorbing properties of either suspended PM or PM collected on a filter.[20] These include BS, COH, Nephelometer, Aethalometer®, and light extinction 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. 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 in diameter. The Aethalometer® collects particles (typically without a size-selective inlet) onto a quartz filter tape where the attenuation of transmitted light or light absorption is measured. Since the mix of ambient particles varies widely by location and time of year, the correlation between optical

[19] 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 may be 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 meteorological 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.

[20] See section 2.2.5 for a discussion of the optical properties of PM.

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.[21] X-ray fluorescence (XRF) is a commonly used laboratory technique for analyzing the elemental composition of primary particles deposited on filters. Wet chemical analysis methods, such as ion chromatography (IC) and automated colorimetry (AC), are used to measure ions such as nitrate (NO_3^-), sulfate ($SO_4^=$), chloride (Cl^-), ammonium (NH_4^+), sodium (Na^+), organic cations (such as acetate), and phosphate (PO_4^{3-}). There are several thermal-optical methods for separating organic carbon (OC) and elemental carbon (EC) 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.

For EPA's CSN and IMPROVE monitoring networks, energy-dispersive X-ray fluorescence (EDXRF) is used for characterizing elements; IC is used for ions; and thermal-optical methods are used for OC and EC. The TOT method, which has been used primarily in the CSN, uses a different thermal temperature profile and optical correction for pyrolyis than TOR, which is used in the IMPROVE monitoring program. The two analysis methods yield

[21] See Chapter 3, section 3.4, of the second draft ISA for a more thorough discussion of sampling and analytical techniques for measuring PM. Methods used in EPA's National $PM_{2.5}$ Chemical Speciation Network and other special monitoring programs are summarized in Solomon et al. (2001).

comparable estimates of total carbon, but they give a different split between OC and EC when analyzing filters from the same sampling device. For consistency in OC and EC measurements across the U.S., CSN began transitioning the sampling and analysis methods to IMPROVE-like samplers and the IMPROVE_A analysis method. The transition began in May 2007 and will be completed in October 2009.

Commercial instruments are now available to measure elements, carbon (OC and EC), and sulfate on a semi-continuous basis. These instruments provide time-resolved measurements from a few minutes to a few hours. The semi-continuous methods involve a variety of techniques that include particle collection on a filter tape with on-board XRF analysis; thermal reduction; and thermal oxidation with non-dispersive infrared (NDIR) detection. Most of these techniques have been field tested and compared through the EPA's Environmental Technology Verification (ETV) program and the Supersites program (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. Proven semi-continuous monitors will become the framework for a long-term network of up to 12 CSN sites equipped with semi-continuous 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. 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 created as collected air moves across the filter. It has long been known that FRMs are subject to negative sampling artifacts or the loss of semi-volatile components of PM (e.g., ammonium nitrate and some organics). In comparison with

other sampling techniques that can measure both semi-volatile and nonvolatile PM, FRMs reported $PM_{2.5}$ or PM_{10} mass concentrations biased low by 10-30%. The bias of the FRMs depends on the components of ambient PM and the sampling conditions (e.g., ambient temperature and relative humidity), which vary from day to day and from season to season (US EPA, 2009a, section 3.x, p. 3-28). Positive artifacts arise when gas-phase organic 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. Despite these issues, the precision of the FRMs are quite high, and the method bias based on the performance audit program is well within the goal.

Particle-bound water is an important component of ambient PM mass (ISA; p. 3-30). It can also represent a substantial fraction of gravimetric mass at normal equilibrium conditions (i.e., 22° C, 35 % RH) when the aerosol has high sulfate content. The amount of particle-bound water will vary with the composition of particles, as discussed in the Provisional Assessment of Recent Studies on Health Effects of PM Exposure (US EPA, 2006). 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 TEOMs use diffusion dryers rather than heating to reduce the relative humidity (US EPA, 2009a, p. 2-100, Table 2-7).

2.4 AMBIENT PM MONITORING NETWORKS

The measurement of ambient air pollution in the United States is provided through a number of ambient air monitoring networks operated almost exclusively by state, local, and tribal air monitoring programs. This section briefly describes the network design criteria for each of the PM monitoring networks, what changes may be useful to improve the networks to best meet monitoring objectives, and what the existing network coverage is across the country.

The ambient air monitoring networks are designed to meet three basic monitoring objectives. Each objective is important and must be considered individually. The objectives are:

- to support compliance with ambient air quality standards and emissions strategy development, including comparison to the NAAQS, assess ambient exposures, development of attainment and maintenance plans, evaluation of regional air quality models used in developing emission strategies, and tracking trends in air pollution abatement control measures' impact on improving air quality;

- to provide air pollution data to the general public in a timely manner such as reporting the Air Quality Index (AQI) through AIRNow (www.airnow.gov), monitoring agency web sites, conventional media outlets such as newspapers, radio and television news, and emerging outlets such as social networking sites; and
- to support air pollution research studies, including atmospheric, health, and epidemiological studies that are used to inform future reviews of the NAAQS.

The sections below briefly summarize the monitoring networks for $PM_{2.5}$, including $PM_{2.5}$ speciation, PM_{10}, $PM_{10-2.5}$, and the forthcoming National Core (NCore) multi-pollutant network.

2.4.1 $PM_{2.5}$

The $PM_{2.5}$ monitoring requirements provide for monitors in MSAs based on a combination of population and design value (Table D-5, 40 CFR Part 58) with higher populated locations having more polluted air required to have the most monitors. Background and transport monitors are also required of each state with options for utilizing IMPROVE and other $PM_{2.5}$ data to provide for flexibility in meeting the requirement.

In urban areas, required $PM_{2.5}$ monitoring stations are sited to represent community-wide air quality. These monitoring stations will typically be at neighborhood or urban scale; however, where a population-oriented micro- or middle-scale $PM_{2.5}$ monitoring station represents many such locations throughout a metropolitan area, these smaller scales can be approved by the applicable EPA Regional Office to also represent community-wide air quality. The EPA's existing network design criteria for $PM_{2.5}$ states: "(1) at least one monitoring station is to be sited in a population-oriented area of expected maximum concentration and (2) for areas with more than one required SLAMS, a monitoring station is to be sited in an area of poor air quality" (40 CFR, PART 58, Appendix D, section 4.7). Since monitors sited for either of these network design criteria must represent community-wide air quality, they are also representative of population exposure. The most important spatial scale to effectively characterize the emissions of particulate matter from both mobile and stationary sources is the neighborhood scale for $PM_{2.5}$. For purposes of establishing monitoring sites to represent large homogenous areas other than the above scales of representativeness and to characterize regional transport, urban or regional scale sites would also be needed. Most $PM_{2.5}$ monitoring in urban areas should be representative of a neighborhood scale.

The $PM_{2.5}$ network design criteria were useful for establishing the network 10 years ago when the focus of the monitoring was on community-wide exposures, and the driver for attaining

the NAAQS was the annual average; however, two important things have changed since that time. First, with 10 years of data, we have a much better understanding of the spatial and temporal characterization of $PM_{2.5}$. Given the regional consistency of $PM_{2.5}$, especially in areas with relatively flat topography and uniform emission across the area, we can expect ambient exposures to be relatively consistent at neighborhood and urban scales for many, but not all areas. This validates devoting some portion of the required network to characterizing community-wide air quality in a neighborhood or urban scale of representation where we expect to find the expected maximum concentration. However, in some locations within urban areas, concentrations of $PM_{2.5}$ may differ from community-wide monitors due to nearby primary emission sources or terrain. The second major change occurred in 2006 when the 24-hour $PM_{2.5}$ NAAQS was finalized. This more protective daily NAAQS has led to some areas where the 24-hour standard is the controlling standard for an area. To better support protection with the daily $PM_{2.5}$ NAAQS, a portion of the required network could be targeted to monitor short-term $PM_{2.5}$ levels in areas that are not necessarily affirmatively designated as "community-wide" air quality. Requiring monitors in areas not designated as "community-wide" would ensure protection for those populations whose ambient exposures may be affected by unique local sources or terrain which are often located in areas with smaller spatial scales. These areas, such as population-oriented micro- and middle- scale "hot spots," are not required to be monitored in the current network design.

In rural areas, EPA's monitoring strategy relies on IMPROVE, rural NCore stations, a limited number of smaller cities, and partner monitoring agencies to provide for regional characterization of $PM_{2.5}$. Stations in these areas are typically sited to represent regional scale air quality and are therefore located away from any local sources, should they exist.

2.4.1.1 $PM_{2.5}$ FRM Network

The network of $PM_{2.5}$ FRMs has been operational since 1999. This network includes over 900 monitoring stations throughout the country. The number of $PM_{2.5}$ FRMs may decrease over the coming years as $PM_{2.5}$ continuous FEMs are now available and can replace FRMs without the loss of a data record. Figure 2-2 illustrates the locations of $PM_{2.5}$ FRMs reporting to the Air Quality System (AQS).

2.4.1.2 $PM_{2.5}$ Continuous Monitor Network

Continuous $PM_{2.5}$ monitors are required in metropolitan Statistical Areas (MSAs) at one half (rounded up) the number of monitoring stations that are required to have an FRM/FEM monitor. Since most deployed $PM_{2.5}$ continuous monitors are not approved as FEMs, many of

these monitors are collocated at monitoring locations with an FRM so that the availability of data from both instruments supports each of the major monitoring objectives described earlier in this section. Collocation with $PM_{2.5}$ FRMs and continuous monitors also ensures that reference data are available to validate the performance of the continuous monitor. While $PM_{2.5}$ continuous monitors primarily support forecasting and reporting the AQI, they are also used in interpreting the diurnal characterization of $PM_{2.5}$. The network of $PM_{2.5}$ continuous monitors has grown to over 700 locations throughout the country. Figure 2-3 illustrates the locations of $PM_{2.5}$ continuous monitors reporting to AQS, including those that are now approved as FEMs.

Figure 2-2. $PM_{2.5}$ FRMs Reporting to AQS

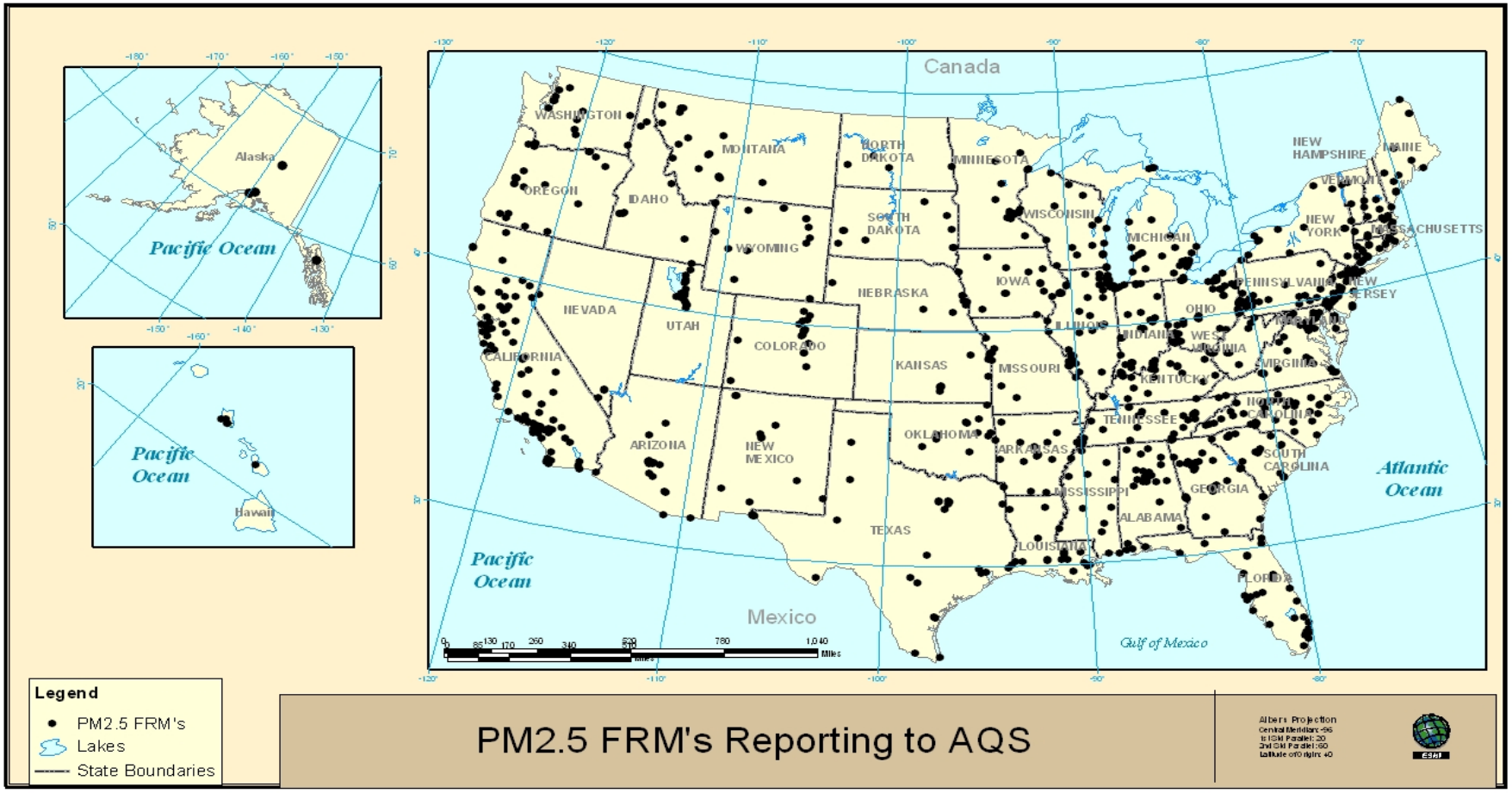
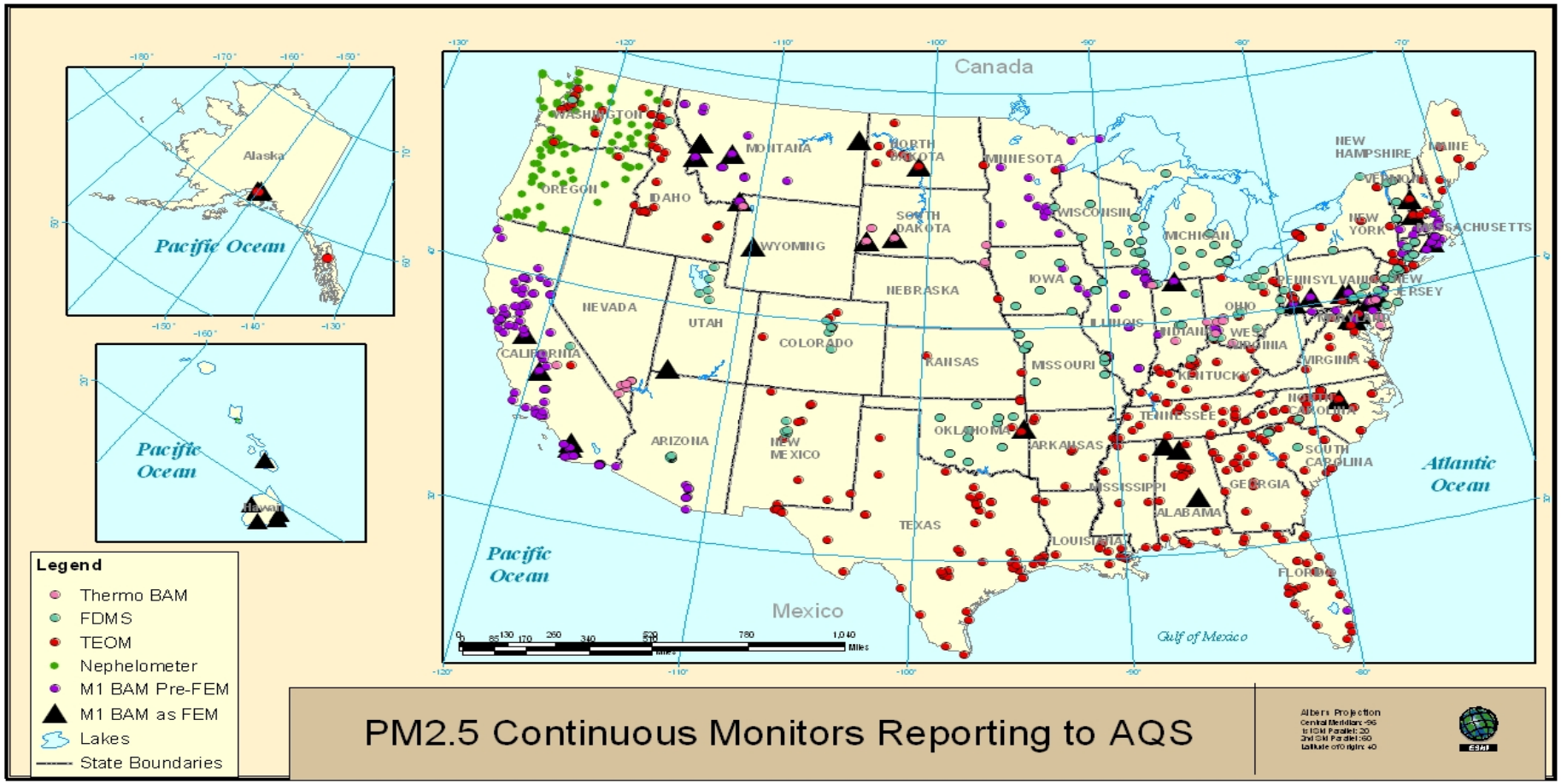


Figure 2-3. $PM_{2.5}$ Continuous Monitors Reporting to AQS



2.4.1.3 $PM_{2.5}$ Chemical Speciation Network (CSN)

As part of the $PM_{2.5}$ NAAQS review completed in 1997, EPA established a $PM_{2.5}$ Chemical Speciation Network (CSN) consisting of 54 Speciation Trends Network (STN) sites. The STN was established to conduct routine speciation monitoring in primarily urban areas to provide nationally consistent data for the assessment of trends and to provide a long-term record of the characterization of $PM_{2.5}$ in the United States. The initial STN monitoring began with a pilot of 13 sites in February 2000. In addition to the STN, EPA also implemented a network of about 200 supplemental speciation sites for multiple monitoring objectives, including support for development of modeling tools and the application of source apportionment modeling for control strategy development in support of the NAAQS; support for health effects and exposure research studies assessment of the effectiveness of emission reductions strategies through the characterization of air quality; support for programs aimed at improving environmental welfare; and state implementation plan (SIP) development. The STN and supplemental speciation monitoring sites together are referred to as the CSN. The CSN sampling apparatus do not include any FRM/FEMs; therefore, data produced from this network are not used for comparison to the NAAQS. However, FRM's are almost always collocated with the CSN since these are among the most important $PM_{2.5}$ sites in a network.

In 2005, EPA conducted an assessment specifically focused on the $PM_{2.5}$ speciation monitoring network. In consultation with state and local monitoring agencies, EPA evaluated CSN sites to determine which ones might be shut down so as to provide resources for future monitoring needs. EPA ranked the sites according to their overall information value. The ranking was based on several factors, including whether the site was in a non-attainment area and whether other sites were nearby. There was general agreement that some of the sites should be shut down when FY 2005 funding ran out. Other sites were identified as high value sites, particularly with regard to the $PM_{2.5}$ NAAQS program. EPA evaluated each of these sites when FY 2006 regional funding allocations for continued operation and maintenance were developed. In doing so, EPA balanced filter-based $PM_{2.5}$ speciation against other uses of $PM_{2.5}$ funding, such as FRM site operations, filter analysis, and startup of additional precursor gas sites and continuous speciation sites.

As of May 2008, the $PM_{2.5}$ CSN consisted of approximately 52 STN sites and about 150 SLAMS supplemental sites. All STN sites operate on a one-in-three day sample collection schedule. A majority of the SLAMS supplemental sites operate on a one-in-six day sample collection schedule. These sites collect aerosol samples over 24 hours on filters that are analyzed

for $PM_{2.5}$ mass, trace elements (Al through Pb), major ions (sulfates, nitrates, and ammonium), and organic and elemental carbon fractions.

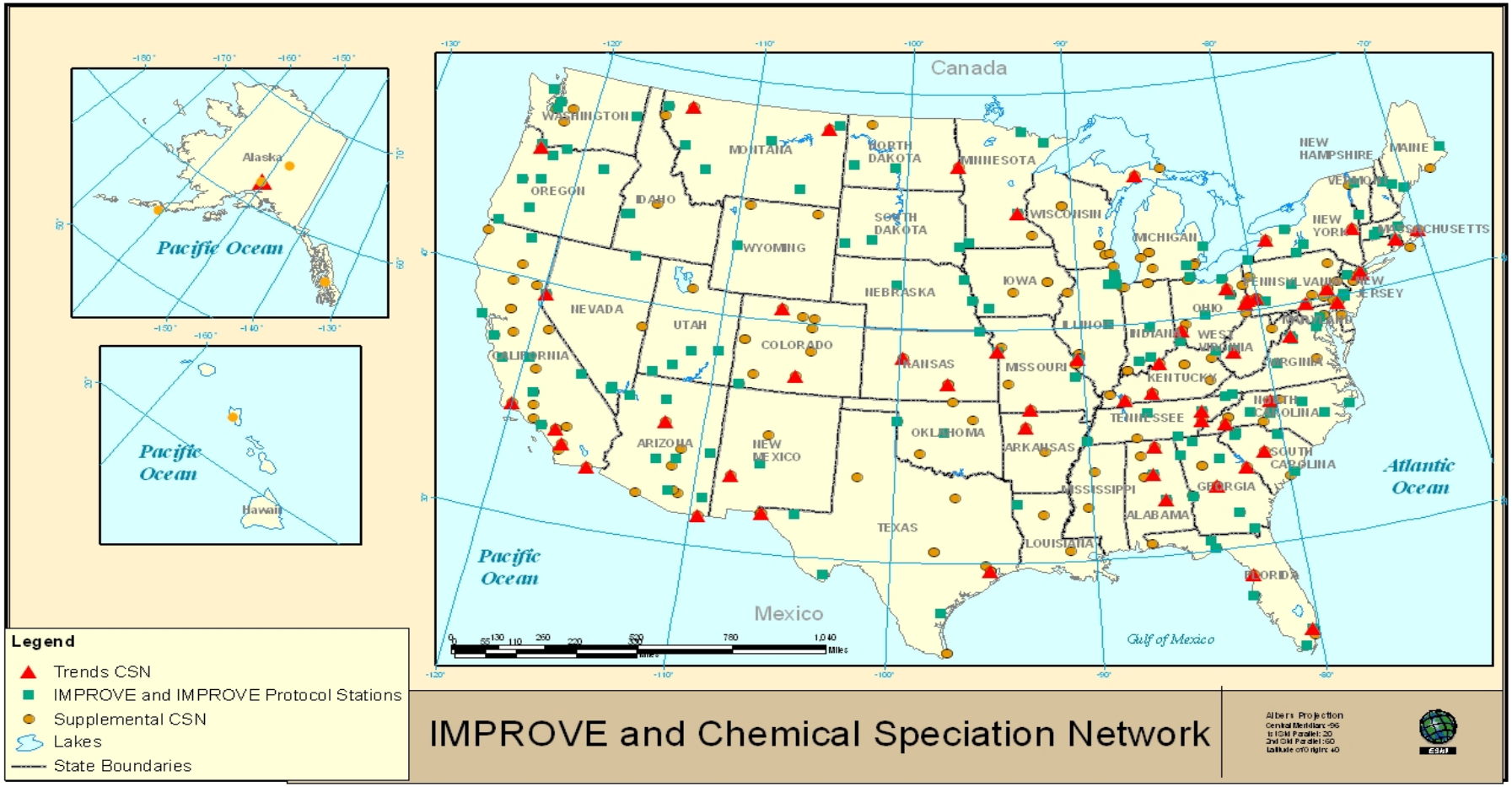
The IMPROVE program was established in 1985 to aid the creation of federal and state implementation plans for the protection of visibility in Class 1 areas (155 national parks and wilderness areas) as stipulated in the 1977 amendments to the CAA and further supports goals set forth in the 1999 Regional Haze Rule.[22] The IMPROVE program provides $PM_{2.5}$ speciation data for carbon, ions, and major elements, like the CSN. IMPROVE is a cooperative measurement effort managed by a steering committee composed of representatives from federal, regional, and state organizations. The IMPROVE network presently comprises 110 regionally representative monitoring sites, 7 sites operated collaboratively with the Clean Air Status and Trends Network (CASTNET). An additional 34 CSN sites operate according to IMPROVE protocols for $PM_{2.5}$ mass, elements, ions and carbon. These sites are in addition to the 200 CSN sites described above and referred to as IMPROVE protocol sites. See Figure 2-4 for locations of IMPROVE and CSN monitors.

In May of 2007, the 200 CSN sites began transitioning to a new method of sampling and analysis for carbon measurements that is consistent with the IMPROVE network methodology. This transition is on-going and was split into three phases with 56 CSN stations in the first phase, 63 stations in the second phase, which began on April 1, 2009, and 78 stations in the third phase, which is scheduled to be implemented on October 1, 2009.

While the network of approximately 200 CSN sites provide valuable data for development and tracking of control strategies, its use for supporting epidemiological studies is limited. CSN sites provide data on a one-in-three or one-in-six day schedule and do not capture data every day or everywhere. In April 2008, the EPA co-sponsored a workshop to discuss modifications to the current ambient air quality monitoring networks that would advance our understanding of the impacts of PM exposures on public health/welfare in the most meaningful way. This workshop was a major step in a series of interactions to foster improved long-term communication between external stakeholders, including air quality monitoring experts and

[22] Additional information is available at http://www.epa.gov/visibility/actions.html

Figure 2-4. IMPROVE and Chemical Speciation Networks



health researchers. A summary of the workshop recommendations was published in December 2008.[23]

2.4.2 PM_{10}

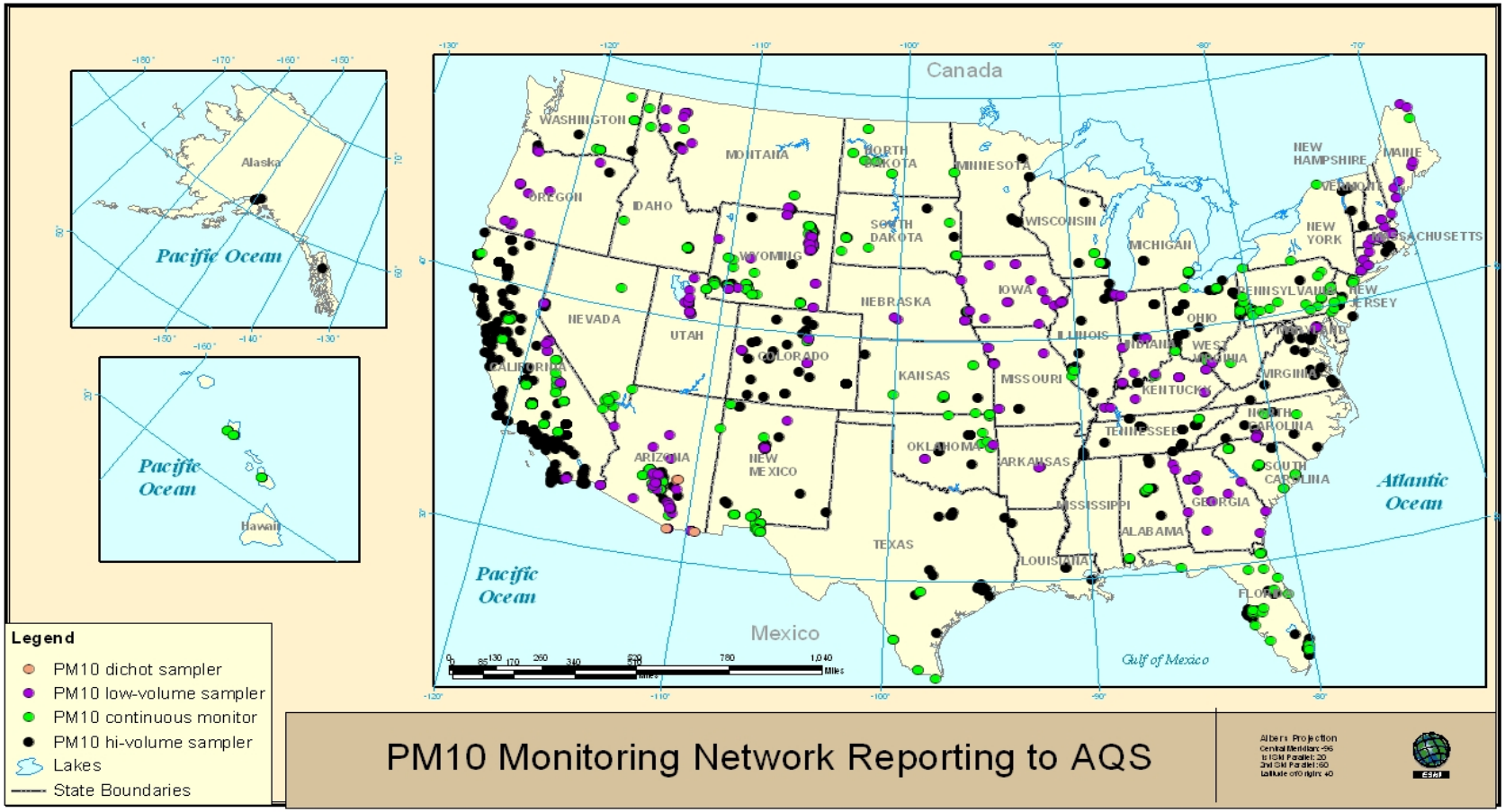
PM_{10} monitoring stations have an urban focus and are required in MSAs to characterize national and regional PM_{10} air quality trends and geographical patterns. Local considerations are also a factor in determining the actual required number of monitoring sites. More stations are required in larger MSAs and MSAs with more evidence of poor air quality, while monitors are also required in clean MSAs of certain size. The number of monitors in areas where MSA populations exceed 1,000,000 must be in the range from 2 to 10 stations, while in low population urban areas, no more than two stations are required (Table D–4 of Appendix D to CFR Part 58). Because sources of air pollutants and local control efforts can vary from one part of the country to another, some flexibility is allowed in selecting the actual number of stations in any one locale.

The network of PM_{10} monitors has been operational since 1987. The network currently includes over 800 monitoring stations throughout the country with most metropolitan areas operating more PM_{10} monitors than required by current monitoring requirements. The PM_{10} monitoring stations operate manual FRMs on a mix of daily, one-in-two day, one-in-three day, or one-in-six day sampling, based on the relative concentration level of the site with respect to the 24-hour standard. There are also FEMs that are operated continuously. PM_{10} monitors operating across the country are almost exclusively FRMs or FEMs. Figure 2-5 illustrates the locations of the PM_{10} FRMs and FEMs reporting to AQS.

The PM_{10} monitoring stations are currently required to collect and report monitoring data under standard temperature and pressure conditions. $PM_{2.5}$ and $PM_{10\text{-}2.5}$ are required to be collected and reported at local conditions. Correction of the sampled aerosol volume to "standard" conditions may improperly report the aerosol concentration measurement. If the rationale for aerosol sampling is to mimic respiratory penetration (which occurrs at local conditions), a correction to standard conditions may not be appropriate. These corrections are typically small (less than a few percent) except in locations at higher altitudes and those with

[23] The report is available at www.epa.gov/ORD/npd/pdfs/FINAL-April-2008-AQ-Health-Research-Workshop-Summary-Dec-2008.pdf

Figure 2-5. PM_{10} Monitoring Network



large diurnal or seasonal temperature changes (1996 CD). Low-volume PM_{10} data used for $PM_{10-2.5}$ determinations must be reported at local conditions.

2.4.3 $PM_{10-2.5}$

The EPA is requiring $PM_{10-2.5}$ mass and speciation monitoring as part of the National Core (NCore) network by January 1, 2011. The NCore network is a multiple pollutant network that also supports PM monitoring objectives (see section 2.4.4 below). To date, there are about 30 $PM_{10-2.5}$ mass monitoring stations reporting data to AQS. Monitoring stations are located in Arizona, Connecticut, Iowa, North Carolina, and Oklahoma. As monitoring agencies continue to implement $PM_{10-2.5}$ mass monitoring as required at NCore (approximately 83 stations are expected), the number of stations is expected to increase.

For $PM_{10-2.5}$ speciation, we do not expect methods to be fully developed in time to meet the January 1, 2011, start date for monitoring at NCore. The EPA has been working with the CASAC Ambient Air Monitoring and Methods Subcommittee on this issue and will be implementing a pilot of $PM_{10-2.5}$ speciation at two locations in 2010.[24] The $PM_{10-2.5}$ speciation pilot monitoring project will be started to further develop and field test analytical methods to be used in the long-term speciation monitoring network.

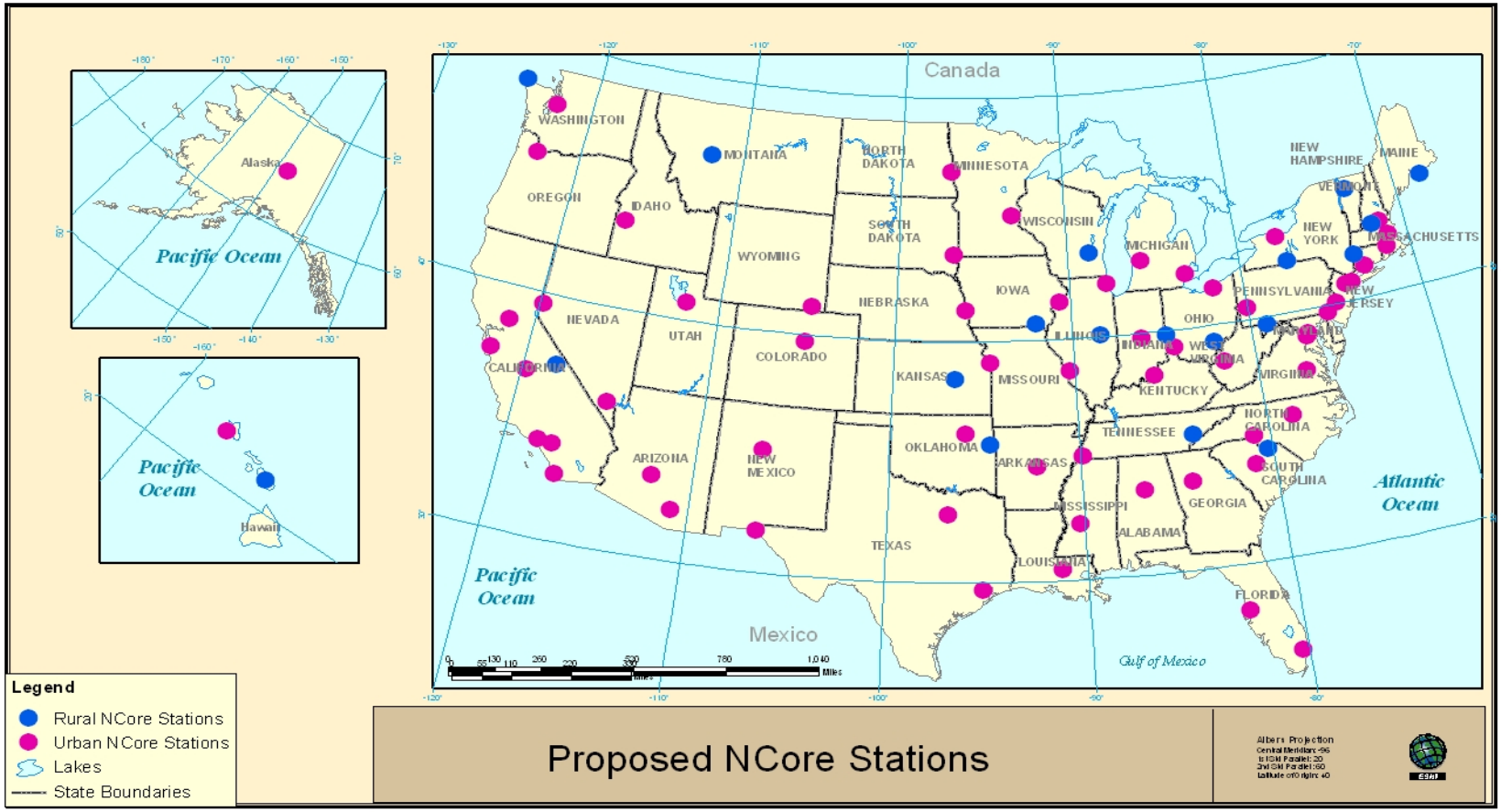
2.4.4 National Core (NCore)

The NCore network is a multi-pollutant network that includes measurements of particles, gases, and meteorology (71 FR 61236, October 17, 2006). The network is intended to support integrated air program management needs. The NCore monitoring network will be fully operational by January 1, 2011. NCore stations will include both neighborhood and urban scale measurements in general, in a selection of urban areas and a limited number of more rural locations. NCore stations are intended to be long-term stations useful for a variety of applications. NCore stations should be placed away from direct emissions sources that could substantially impact the ability to detect area-wide concentrations. NCore has both urban and rural siting components. Urban NCore stations are to be generally located at a urban or neighborhood scale to provide representative concentrations of exposure expected throughout the metropolitan area. Rural NCore stations are to be located to the maximum extent practicable at a regional or larger scale away from any large local emission source, so that they represent

[24] A Febuary 2009 consultation with the CASAC AAMM subcommittee discussed issues related to coarse particle speciation measFor more information on consultation with see: http://yosemite.epa.gov/sab/sabproduct.nsf/264cb1227d55e02c85257402007446a4/3494de4d0ccb3944852574630064d4e4!OpenDocument

ambient concentrations over an extensive area. States and where applicable, local monitoring agencies have submitted plans for NCore during the summer of 2009. The proposed NCore stations are collocated with several other well leveraged networks such as PAMS, CSN, the National Air Toxics Trends Stations (NATTS), IMPROVE, and CASTNET. The recommended NCore locations identified as of September 2009 are provided in Figure 2-6.

Figure 2-6. Proposed NCore Monitoring Network



2.5 PM CONCENTRATIONS, TRENDS, AND SPATIAL PATTERNS

As described in section 2.4, EPA and the states have been using a national network to measure PM_{10} concentrations since 1987 and $PM_{2.5}$ concentrations since 1999. This section presents summaries through the end of 2007, based on publicly available data from EPA's Air Quality System (AQS) as of August 2008.

The Staff Paper from the previous review (US EPA, 2005, chapter 2) and the second draft ISA (US EPA, 2009a, sections 3.5 and 9.3.4) present extensive characterizations of the levels, composition, and temporal and spatial patterns of $PM_{2.5}$ in U.S. urban areas. Both documents present data summaries based on the approximately 1100 $PM_{2.5}$ and 1300 PM_{10} monitoring sites in the U.S. The characterizations of recent air quality data used in the previous review were based on ambient measurements collected from 2001 through 2003. In this review, EPA is considering more recent air quality data collected from 2005 through 2007. Also, Chapter 6 of the 2004 PM Assessment by NARSTO contains more detailed characterizations of PM in different parts of the U.S. (NARSTO, 2004). While there generally have been reductions in the concentrations of $PM_{2.5}$ and PM_{10} in many areas as a result of reductions in emission of $PM_{2.5}$ and its precursors and of PM_{10} since these documents were written, comparison of the three documents indicates that the general patterns, as well as the diversity of patterns across areas, noted in the two older documents still prevailed in the 2005-2007 period.

2.5.1 $PM_{2.5}$

In 2005-2007, 38 urban areas violated the current annual $PM_{2.5}$ NAAQS of 15 $\mu g/m^3$, adopted in 1997, which level was retained in the last review completed in 2006. Seventy-six areas violated the revised 24-hour NAAQS of 35 $\mu g/m^3$. There is considerable but not complete overlap in the areas not meeting the annual and 24-hour NAAQS. It should be noted that in many parts of the U.S., $PM_{2.5}$ concentrations in 2005 were high relative to the next three years.

Figure 2-7 illustrates $PM_{2.5}$ air quality in 2005, 2006, and 2007 by representing each monitor by a symbol whose color reflects the annual mean of the concentration at that site or the 98^{th} percentile of 24-hour concentrations, in both cases in a single year (US EPA, 2009a, section 3.5.1.1 presents maps for $PM_{2.5}$ air quality concentrations for the 2005-2007 period as a whole). County-scale, 24-hour average concentration data for $PM_{2.5}$ during 2005 through 2007 showed considerable variability across the U.S. The highest reported 3-year average concentrations were reported for six counties within the San Joaquin Valley and inland southern California, as well as Jefferson County, AL, (containing Birmingham) and Allegheny County, PA, (containing Pittsburgh). The lowest reported annual average $PM_{2.5}$ concentrations occurred within 237

counties distributed throughout many western and northern states as well as Florida and the Carolinas. Of the 15 individual CSAs/CBSAs selected for detailed investigation in the second draft ISA based on their geographic distribution and importance in recent health effect studies,[25] the highest unweighted 2005-2007 mean 24-hour $PM_{2.5}$ concentrations were reported for Riverside (17 $\mu g/m^3$), Birmingham (16 $\mu g/m^3$), and Pittsburgh (16 $\mu g/m^3$); the lowest were reported for Denver (9 $\mu g/m^3$) and Seattle (9 $\mu g/m^3$).

Figure 2-8 and Figure 2-9 show trend information for the annual and 24-hour $PM_{2.5}$ NAAQS. The seasonally weighted 3-year annual average concentration fell 10 percent between the 1999-2001 averaging period and the 2005-2007 averaging period. The number of sites reporting concentrations above the annual average $PM_{2.5}$ NAAQS fell 56 percent over these same periods. Declines were the greatest in EPA Region 9 where $PM_{2.5}$ concentrations fell 20 percent from the 1999-2001 averaging period to the 2005-2007 averaging period.

The second draft ISA states that in the 2004 Critiera Document, EPA concluded that that $PM_{2.5}$ measured in eastern cities was generally more highly correlated across monitoring sites than in western cities. The higher spatial correlations in the eastern cities resulted from the more regionally dispersed sources of $PM_{2.5}$ in the East, and the high contribution of more regionally homogeneous species (e.g. sulfates) to total $PM_{2.5}$ throughout the area. Although $PM_{2.5}$ concentrations at sites within an urban area can be highly correlated resulting from the homogeneity of such components over broad regions, significant differences in concentrations can occur on any given day resulting from terrain features and varying contributions of urban and local sources.

Comparison of $PM_{2.5}$ mass and $PM_{2.5}$ species concentrations within and outside urban areas leads to the conclusion that in the eastern areas with high sulfate concentrations, the large majority of the sulfate and, thus, also a large fraction of the $PM_{2.5}$ affecting any given urban area originates outside that area. Inward transport and local generation of nitrate and carbonaceous

[25] Fifteen metropolitan regions were chosen by EPA for closer investigation of monitor siting based on their distribution across the nation and relevance to health studies analyzed in the second draft ISA. These regions were Atlanta, Birmingham, Boston, Chicago, Denver, Detroit, Houston, Los Angeles, New York City, Philadelphia, Phoenix, Pittsburgh, Riverside, Seattle, and St. Louis. Core-Based Statistical Areas (CBSAs) and Combined Statistical Areas (CSAs), as defined by the U.S. Census Bureau were used to determine which counties, and, hence, which monitors, to include for each metropolitan region (US EPA, 2009a, section 3.4.2.2)

Figure 2-7. Annual average and 24-hour (98th percentile 24-hour concentrations) $PM_{2.5}$ concentrations in µg/m^3, 2005, 2006, and 2007.

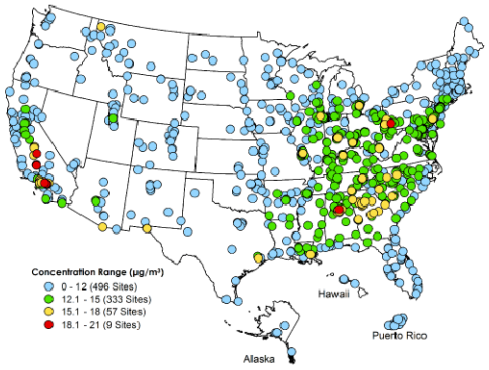
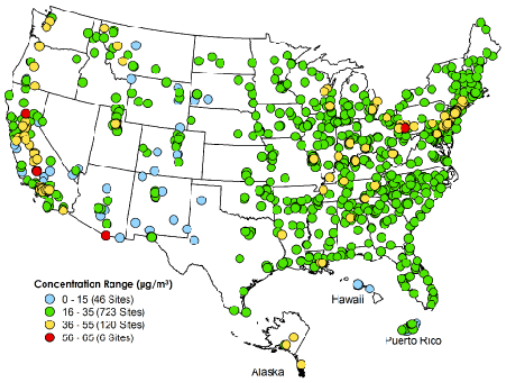
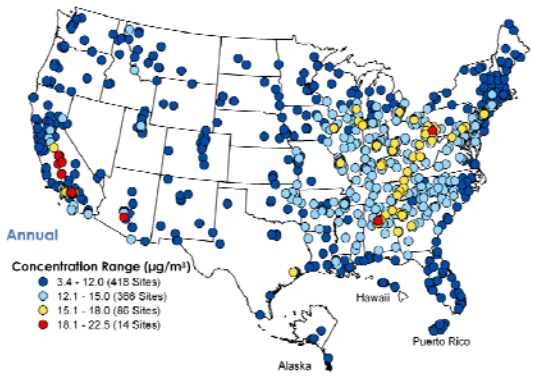
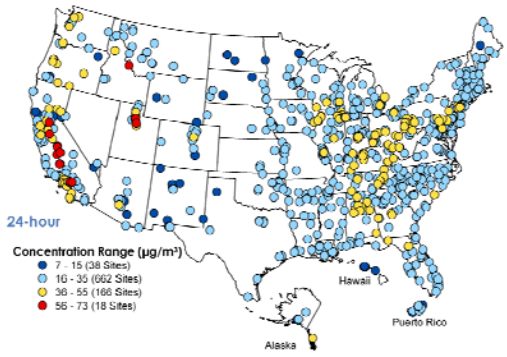
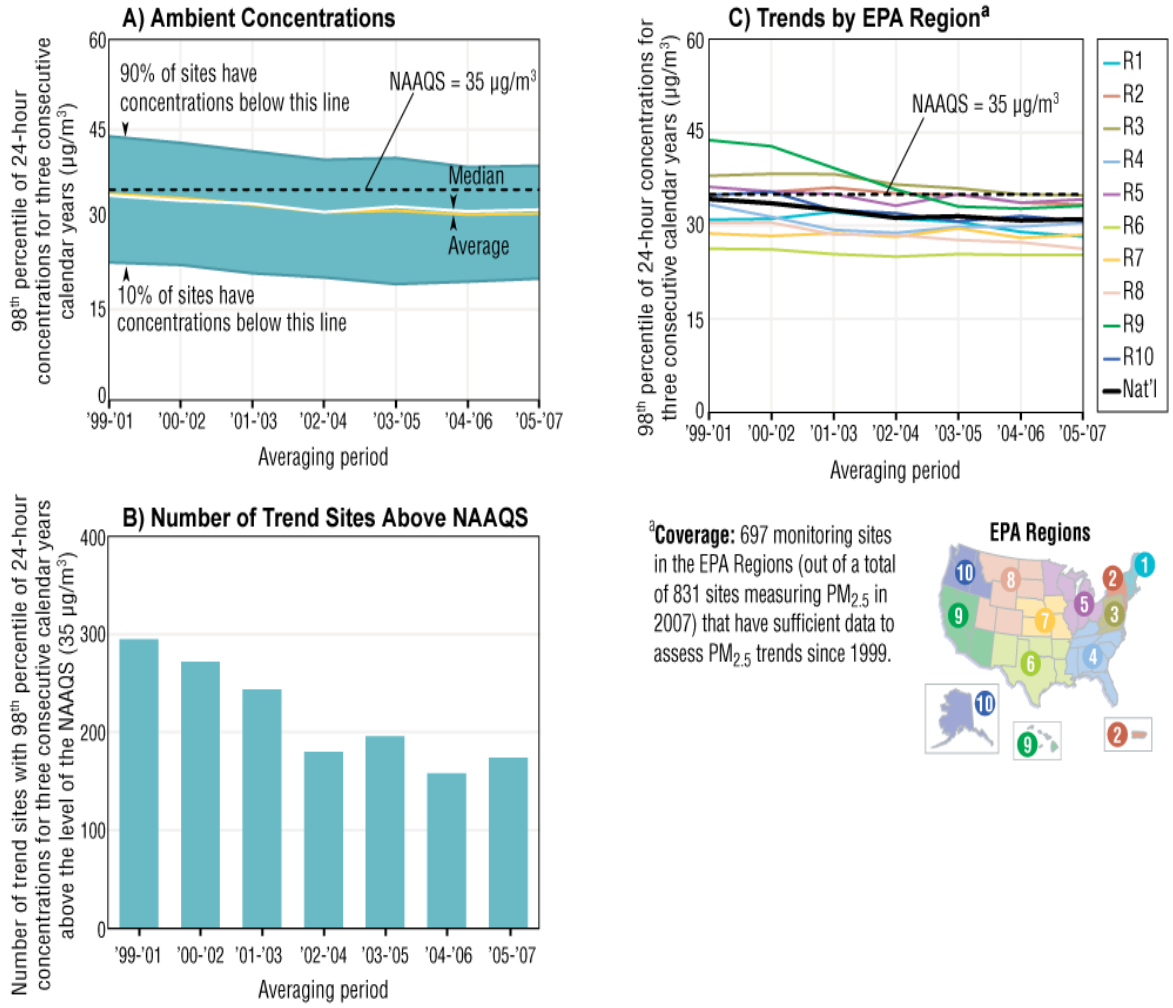
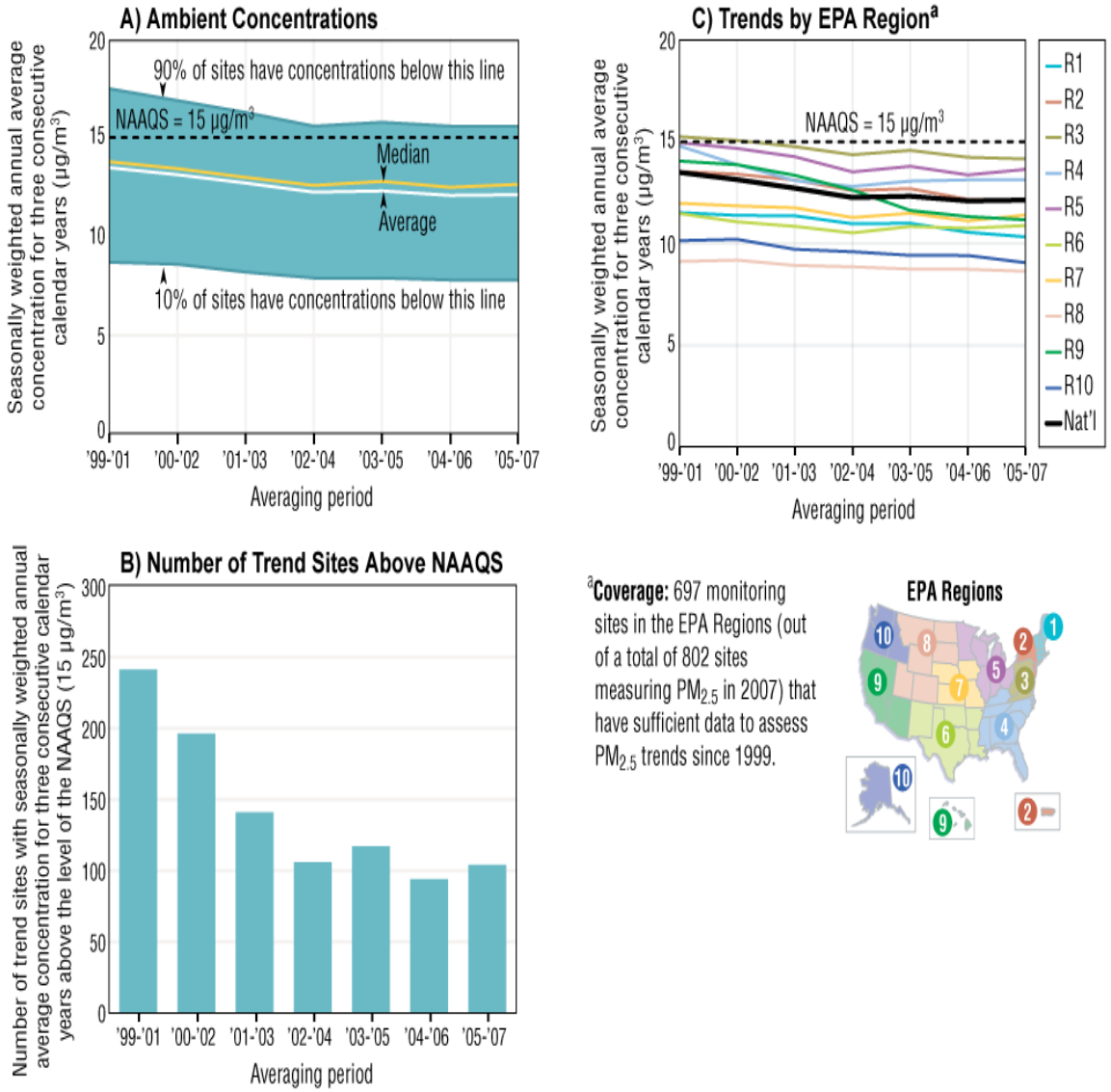
Annual Average	24-hour
2005	
{graphic to be added to external review draft PA}	{graphic to be added to external review draft PA}
2006	
	
2007	
	

Figure 2-8. Ambient annual $PM_{2.5}$ concentrations in the U.S., 1999-2007,showing A) ambient concentrations, B) number of trends sites above the annual NAAQS and C) trends by U.S. EPA Region.



Source: US EPA, 2009a, Figure 3-45

Figure 2-9. Ambient 24-hour $PM_{2.5}$ concentrations in the U.S., 1999-2007,showing A) ambient concentrations, B) number of trends sites above the 24-hour NAAQS and C) trends by U.S. EPA Region.



Source: US EPA, 2009a, Figure 3-44

material are more evenly balanced in eastern areas, with some differences among areas. In western areas, local sources dominate for carbonaceous material and nitrate, with both local and regional sources contributing to the small sulfate component (US EPA, 2009a, Figure 9-24).

Southeastern areas have their highest $PM_{2.5}$ concentrations in the summer, when conditions are most conducive to sulfate formation. More northern areas, being affected by a more balanced mix of contributors, tend not to have such a strongly seasonal pattern. The seasonal patterns in western areas are individual and varied, related to differences in local sources and formation and dispersion conditions. In all areas, inversion conditions with low wind speeds are conducive to high concentrations due to the trapping of emissions from local sources. Some western areas, especially those with bowl-like topography, are especially affected.

2.5.2 PM_{10}

Figure 2-10 illustrates PM_{10} air quality in 2005, 2006, and 2007 by representing each monitor by a symbol whose color reflects the 98th percentile 24-hour concentration, in one year. (See also US EPA, 2009a, Section 3.5.1.1 which presents maps for PM_{10} air quality concentrations for the 2005-2007 period as a whole.) Most areas of the country had concentrations below the level of the 24-hour PM_{10} standard of 150 $\mu g/m^3$, with exceptions concentrated in the southwestern U.S. and isolated counties scattered across the east. The highest reported 3-year average PM_{10} concentrations (greater than 51 $\mu g/m^3$) occurred in two counties in southern California and five counties in southern Arizona and central New Mexico. The lowest reported annual average PM_{10} concentrations (less than or equal to 20 $\mu g/m^3$) were within 114 counties distributed fairly uniformly across the U.S. Of the 15 CSAs/CBSAs investigated, the highest mean 24-hour PM_{10} concentrations was reported for Phoenix (52 $\mu g/m^3$), considerably higher than the means for the other CSAs/CBSAs investigated. The lowest was reported for Boston (17 $\mu g/m^3$) with New York, Philadelphia and Seattle only slightly higher (19 $\mu g/m^3$).

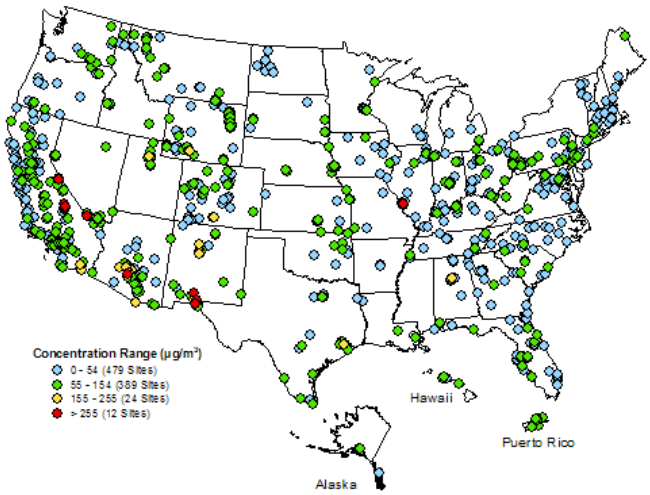
Figure 2-11 shows trends in U.S. ambient 24-hour PM_{10} concentrations from 1988 to 2007. In 2007, the U.S. national average second highest PM_{10} concentration was 37% lower than in 1988. Of 281 sites used in this long-term trend analysis, the number reporting concentrations above the 24-hour PM_{10} NAAQS (150 $\mu g/m^3$) fell from 23 in 1988 to 5 in 2007 with a max of 29 in 1989. All regions exhibited an overall decrease from 1988 to 2007. The largest decreases occurred in EPA Region 10, which incorporates Washington, Oregon, Idaho, and Alaska. Most of the decrease occurred between 1988 and 1995. (*US EPA, 2009a, section 3.5.2.1*)

Figure 2-10. 24-hour PM_{10} concentrations in µg/m^3, 2005- 2007 (98^{th} percentile 24-hour concentrations).

2005

{graphic to be added in external review draft PA}

2006



2007

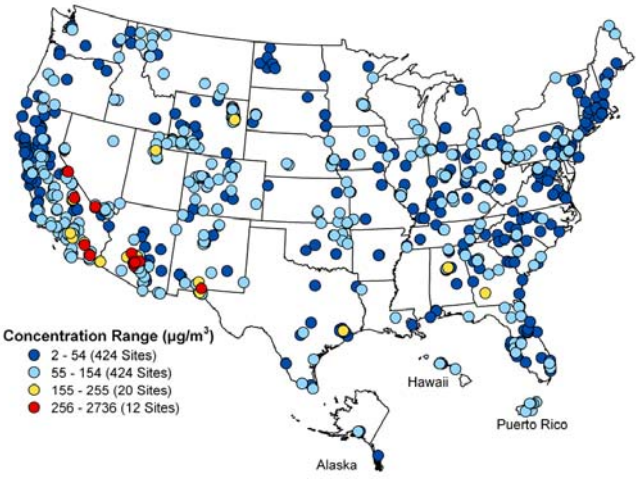
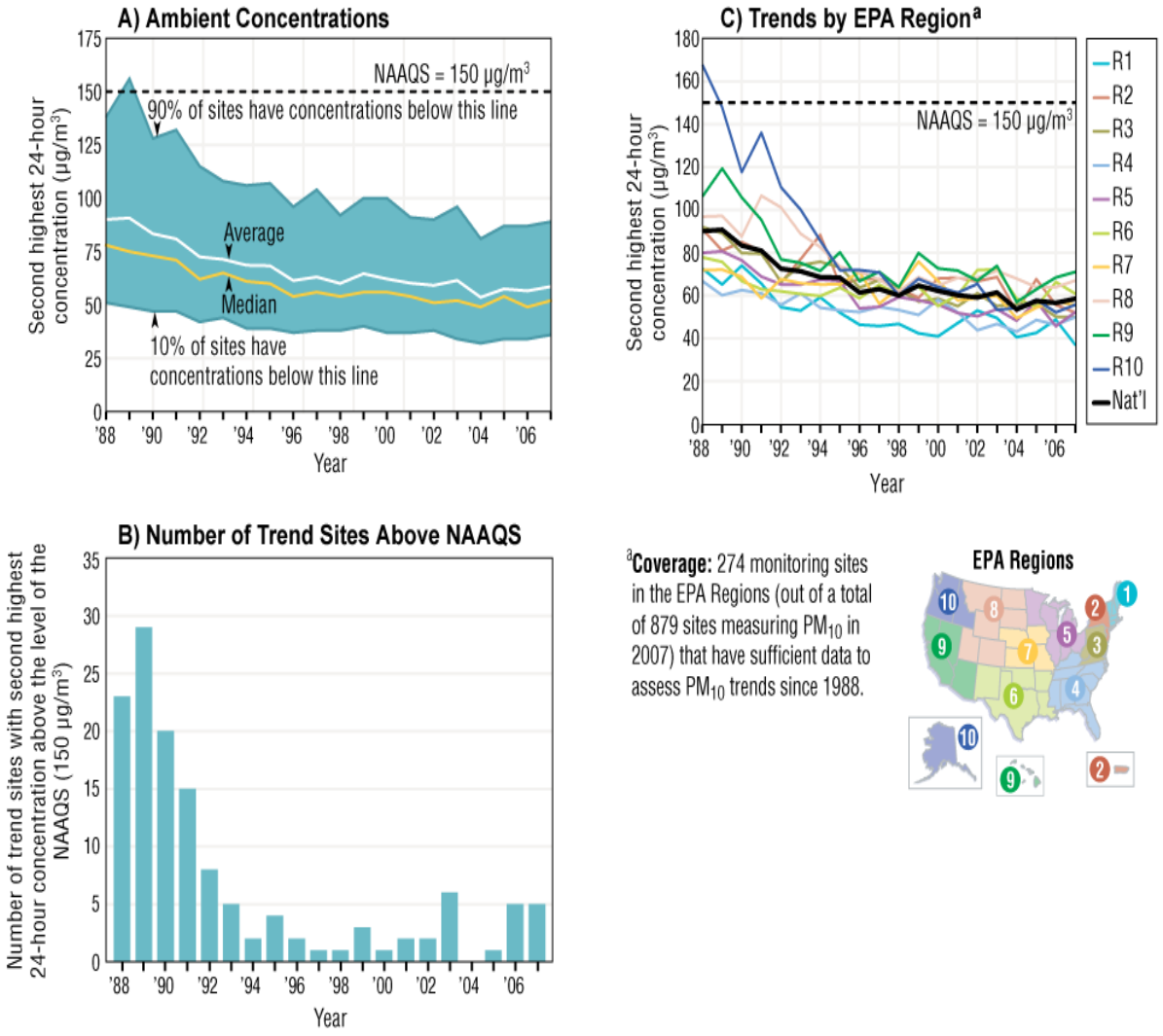


Figure 2-11. Ambient 24-hour PM_{10} concentrations in the U.S., 1988-2007,showing A) ambient concentrations, B) number of trends sites above the 24-hour NAAQS and C) trends by U.S. EPA Region. (US EPA, 2009a, Figure 3-46)



Source: U.S. EPA (2008)

Being the sum of $PM_{2.5}$ and $PM_{10-2.5}$, PM_{10} trends and patterns are a superimposition of those for $PM_{2.5}$ and $PM_{10-2.5}$, which can be quite different due to their different source/formation and transport/deposition characteristics. Broadly speaking, $PM_{2.5}$-like temporal and spatial patterns dominate where local $PM_{10-2.5}$ emissions are relatively low, such as in areas with wetter climates, year-round ground cover, and an absence of large industrial sources of $PM_{10-2.5}$ emissions; $PM_{10-2.5}$-like temporal and spatial patterns are observed in other areas, especially dry areas in the west and southwest.

2.5.3 $PM_{10-2.5}$

There is, at present, no systematic monitoring network in place for $PM_{10-2.5}$. As outlined in section 2.4.3, states have until January 1, 2011, to implement required monitoring sites for $PM_{10-2.5}$. Consequently, estimates of $PM_{10-2.5}$ must be developed using data from $PM_{2.5}$ and PM_{10} monitoring sites and equipment. In some reviews, estimates have been offered based on $PM_{2.5}$ and PM_{10} monitors which were within some distance of each other but not actually collocated. Even when collocated, the instruments in a $PM_{2.5}$ and PM_{10} pair may not have consistent flow rates, measurement principles (e.g., gravimetric filter vs. beta attenuation), filter preconditioning and weighing protocols, etc.

In the Staff Paper for the last review, EPA used a data-inclusive approach in which we used the best available data on $PM_{2.5}$ and PM_{10} concentrations in each given area – in some cases not very robust data[26] – to estimate 2001-2003 $PM_{10-2.5}$ concentrations for 351 counties (US EPA, 205, section 2.4.3). Since the operating protocol for each monitor is not usually identical, the consistency of these $PM_{10-2.5}$ measurements was relatively uncertain, and they were referred to as "estimates." As reported in the last review, 98th percentile 24-hour average $PM_{10-2.5}$ estimated concentrations ranged from about 5 to 208 $\mu g/m^3$, with a median of about 28 $\mu g/m^3$ (US EPA, 2005, section 2.4.3)

The second draft ISA presents $PM_{10-2.5}$ concentration estimates in Figure 3-8 and Table 3-13 of section 3.5.1.1. The second ERD ISA used a much more data-restrictive approach than the Staff Paper for the last review, using only on paired (collocated) low-volume filter-based samplers for both PM_{10} and $PM_{2.5}$, the method ultimately established as the basis for the Federal Reference Method for measuring $PM_{10-2.5}$. Only 40 counties met these requirements for paired samplers. Using these available co-located PM measurements from 2005-2007, the mean 24-

[26] Only in some cases was it possible to calculate $PM_{10-2.5}$ concentrations based on collocated low-flow filter-based sampler. Other data and approaches included pairing low-volume and high-volume samplers, pairing samplers at different monitoring sites, and applying regional $PM_{2.5}$ to PM_{10} ratios to either $PM_{2.5}$ or PM_{10} depending on which was available.

hour $PM_{10-2.5}$ concentration in these 40 counties was 13 $\mu g/m^3$. Ninety-ninth percentile values of $PM_{10-2.5}$ ranged from 25 to 67 $\mu g/m^3$ depending on area, season, and year. Drier western areas such as Phoenix and Denver tended to have the highest concentrations of $PM_{10-2.5}$.

The IMPROVE monitoring network measures both $PM_{2.5}$ and PM_{10} and thus provides $PM_{10-2.5}$ data for rural areas. The $PM_{10-2.5}$ levels in the relatively remote non-urban IMPROVE sites were notably lower than those found in most urban areas. In all the metro areas examined by comparing urban monitoring sites with nearby non-urban IMPROVE sites, the urban $PM_{10-2.5}$ concentrations exceed those in the nearby rural locations (US EPA, 2005, section 2.4.3).

Both the US EPA, 2005 (section 2.7.2) and the second draft ISA (section 3.5.1.2) examined the spatial variability of $PM_{10-2.5}$ across and within cities. Together, they indicated that spatial distribution of $PM_{10-2.5}$ is more heterogeneous than $PM_{2.5}$.

2.5.4 Ultrafine Particles

The second draft ISA (US EPA, 2009a, sections 3.5.1 and 3.5.2) contains a review of the current scientific information on ultrafine particles. Because there is no national network of ultrafine particle samplers, only episodic and/or site-specific data sets exist. Therefore, a national characterization of concentrations, temporal and spatial patterns, and trends is not possible. Generally speaking, it can be said that particle number concentrations of ultrafine particles can be very high close to sources of the same, but mass concentrations of ultrafine particles are usually lower than background concentrations of larger particles within the $PM_{2.5}$ size range. The number concentration falls off sharply downwind, as ultrafine particles evaporate due to more mixing or accumulate into larger particles. Internal combustion engines and therefore roadways are a notable source of ultrafine particles, so concentrations of ultrafine particles near roadways can generally be expected to be elevated.

2.5.5 PM Components

2.5.5.1 $PM_{2.5}$

The 2005 Staff Paper and the second draft ISA extensively summarize in considerable detail 2001-2003 and 2005-2007 data, respectively, on $PM_{2.5}$ composition from the rural IMPROVE network and from the urban Chemical Speciation Network (CSN) operated by EPA and state/local partners. The summary here mostly omits repeating specific numerical details presented in those other documents (US EPA, 2005, section 2.4.5; US EPA, 2009a, sections 3.5.1 and 3.5.2). In general, $PM_{2.5}$ in the eastern U.S. regions is dominated by sulfates and carbonaceous mass. Midwestern, southeastern, and eastern urban areas have much higher $PM_{2.5}$

sulfate levels than do more western areas, attributable to the much higher emissions of SO_2 in and upwind of the former areas. Upper midwest areas and, to a lesser extent, upper eastern areas have notable nitrate concentrations in winter but not in summer, while southeastern areas generally lack notable nitrate even in winter. Many western urban areas have notable nitrate year round. In all areas, carbonaceous material is an important component of $PM_{2.5}$ and is attributable to many emission sources of organic material in PM form and of organic PM precursor gases. $PM_{2.5}$ at the western U.S. urban sites has a greater proportion of carbonaceous mass; in some areas with high local use of wood for residential heating carbonaceous material is the dominant component during the heating season. $PM_{2.5}$ derived from crustal sources is generally a small fraction of total mass, except during local high wind events or due to periods of intercontinental transport of dust from Africa or Asia. While $PM_{2.5}$ mass and all component concentrations are higher in urban areas than at IMPROVE sites, in general, nitrates and carbonaceous components appear to have a greater urban/rural enhancement as compared to sulfates.

Figure 2-12 through Figure 2-16 contain U.S. concentration maps for OC, EC, SO_4^{2-}, NO_3^-, and NH_4^+ mass from $PM_{2.5}$ measurements taken as part of the CSN network for the period 2005-2007. Data used in these figures are as reported to AQS: no correction was applied to OC for non-carbon mass, and NO_3^- represents total particulate NO_3^-. These figures show regional and seasonal differences in measurements of $PM_{2.5}$ components. Figure 2-12 shows regions of high $PM_{2.5}$ OC mass concentration with annual average concentrations greater than 5 $\mu g/m^3$ in the western and the southeastern U.S. Concentrations at the western monitors peak in the fall and winter while those in the Southeast peak anywhere from spring through fall. The central and northeastern portions of the U.S. generally contain lower measured OC *(US EPA, 2009a, section 3.5.1.1*).

Figure 2-13 contains a similar map for $PM_{2.5}$ EC mass concentration that exhibits smaller seasonal variability than OC, particularly in the eastern half of the U.S. There are isolated monitors spread throughout the country that measure high annual average EC concentrations. These EC "hot spots" are primarily associated with larger metropolitan areas such as Los Angeles, Pittsburgh, and New York, but El Paso, TX, also reported high annual average EC concentrations (driven by a wintertime average concentration greater than 2 $\mu g/m^3$). El Paso is an unusual case for its own size because of its proximity to Juarez, Mexico, a much larger city with generally higher ambient PM concentrations. *(US EPA, 2009a, section 3.5.1.1*)

Figure 2-14 contains a map for $PM_{2.5}$ SO_4^{2-} mass concentration which shows that SO_4^{2-} is more prevalent in the eastern U.S. owing to the strong west-to-east gradient in SO_2 emissions. This gradient is magnified in the summer months when more sunlight is available for

photochemical formation of SO_4^{2-}. In contrast, $PM_{2.5}$ NO_3^- mass concentration in Figure 2-15 is highest in the west, particularly in California. There are also elevated concentrations of NO_3^- in the upper midwest. The seasonal plots show generally higher NO_3^- in the wintertime as a result of temperature driven partitioning. Exceptions exist in Los Angeles and Riverside where high NO_3^- readings appear year-round. *(second draft ISA, section 3.5.1.1*)

The $PM_{2.5}$ NH_4^+ mass concentration maps in Figure 2-16 show spatial patterns related to both SO_4^{2-} and NO_3^- resulting from its presence in both $(NH_4)_2SO_4$ and NH_4NO_3 *(US EPA, 2009a, section 3.5.1.1*).

No maps for $PM_{2.5}$ metals are repeated here, but the second draft ISA contains such maps in Annex A and it reported that there is considerably less seasonal variation in the concentration profile for $PM_{2.5}$ metals than for OC or the ions (US EPA, 2009a, section 3.5.1.1). This is understandable in light of the less seasonal nature of emission sources for metals and the absence of seasonally dependent chemical transformations.

In addition to providing the national maps in the figures provided, which are based directly on the measurements made by the CSN network, the second draft ISA also reported the results of an analysis to show the relative contributions of $PM_{2.5}$ species to $PM_{2.5}$ mass as measured in the $PM_{2.5}$ FRM network, taking into account measurement system. Figure 2-17 (copied from Figure 3-17 in section 3.5.1.1 of the second draft ISA) shows the resulting $PM_{2.5}$ compositional breakdown for the fifteen CSAs/CBSAs identified earlier. For the fifteen metropolitan areas, the contribution of the major component classes to total $PM_{2.5}$ mass as measured by the FRM was derived using the measured sulfate, adjusted NO_3^-, derived water, inferred carbonaceous mass approach (SANDWICH) (Frank, 2006). This approach uses the measured FRM $PM_{2.5}$ mass and co-located CSN chemical constituents to perform a mass balance-based estimation of the $PM_{2.5}$ mass fraction attributed to SO_4^{2-}, NO_3^-, EC, OCM, and crustal material. The calculations and assumptions that are used in the SANDWICH method are discussed in detail in Frank (2006).[27]

On an annual average basis, SO_4^{2-} is a dominant $PM_{2.5}$ component in the eastern U.S. cities. For the presented cities, this includes everything east of Houston. Organic carbon mass is

[27] See additional information available on EPA's AirExplorer website: (http://www.epa.gov/cgi-bin/htmSQL/mxplorer/query_spe.hsql

Figure 2-12. Three-year average 24-hour $PM_{2.5}$ OC concentrations measured at CSN sites across the U.S., 2005-2007.

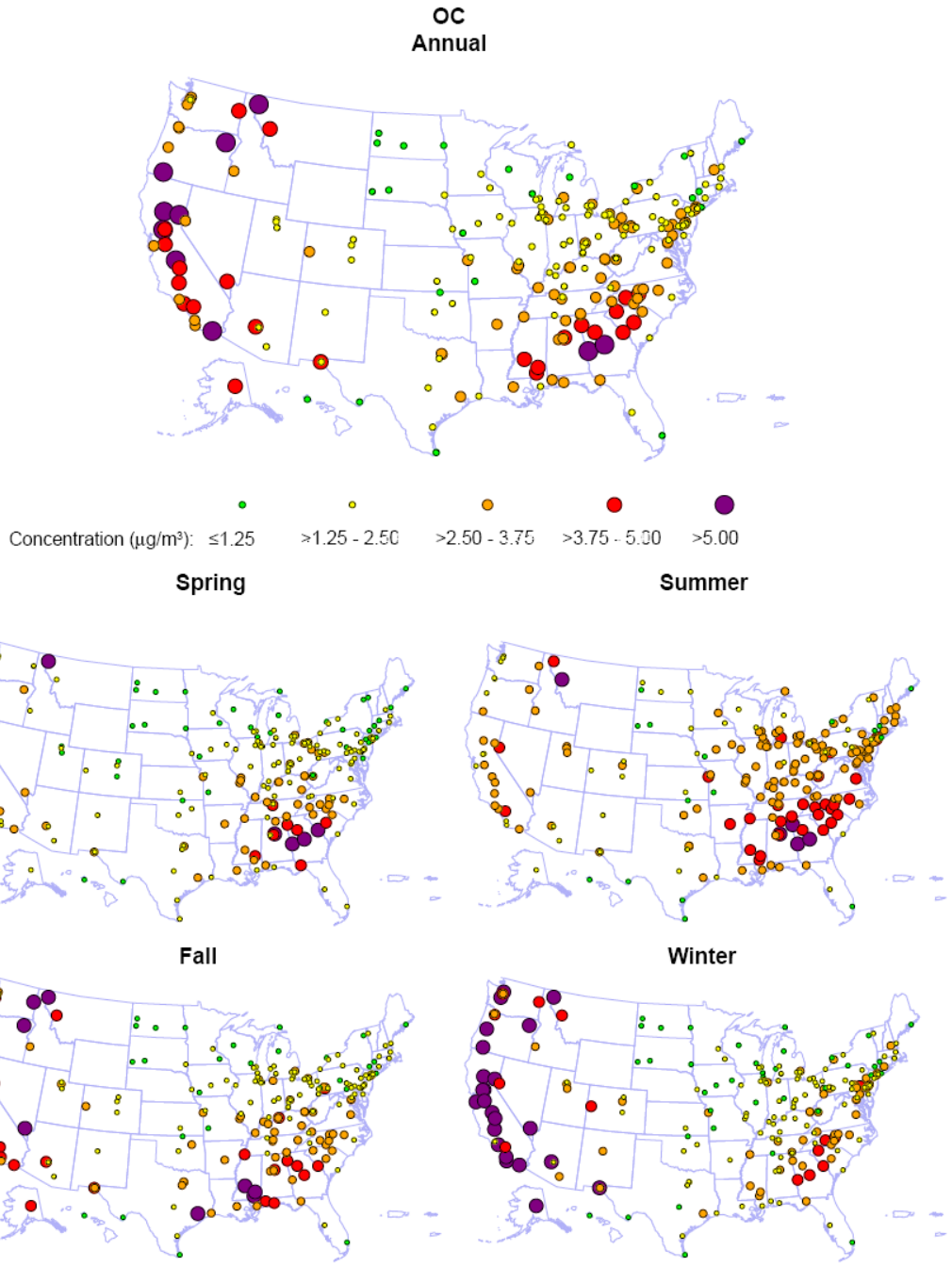


Figure 2-13 Three-year average 24-hour $PM_{2.5}$ EC concentrations measured at CSN sites across the U.S., 2005-2007.

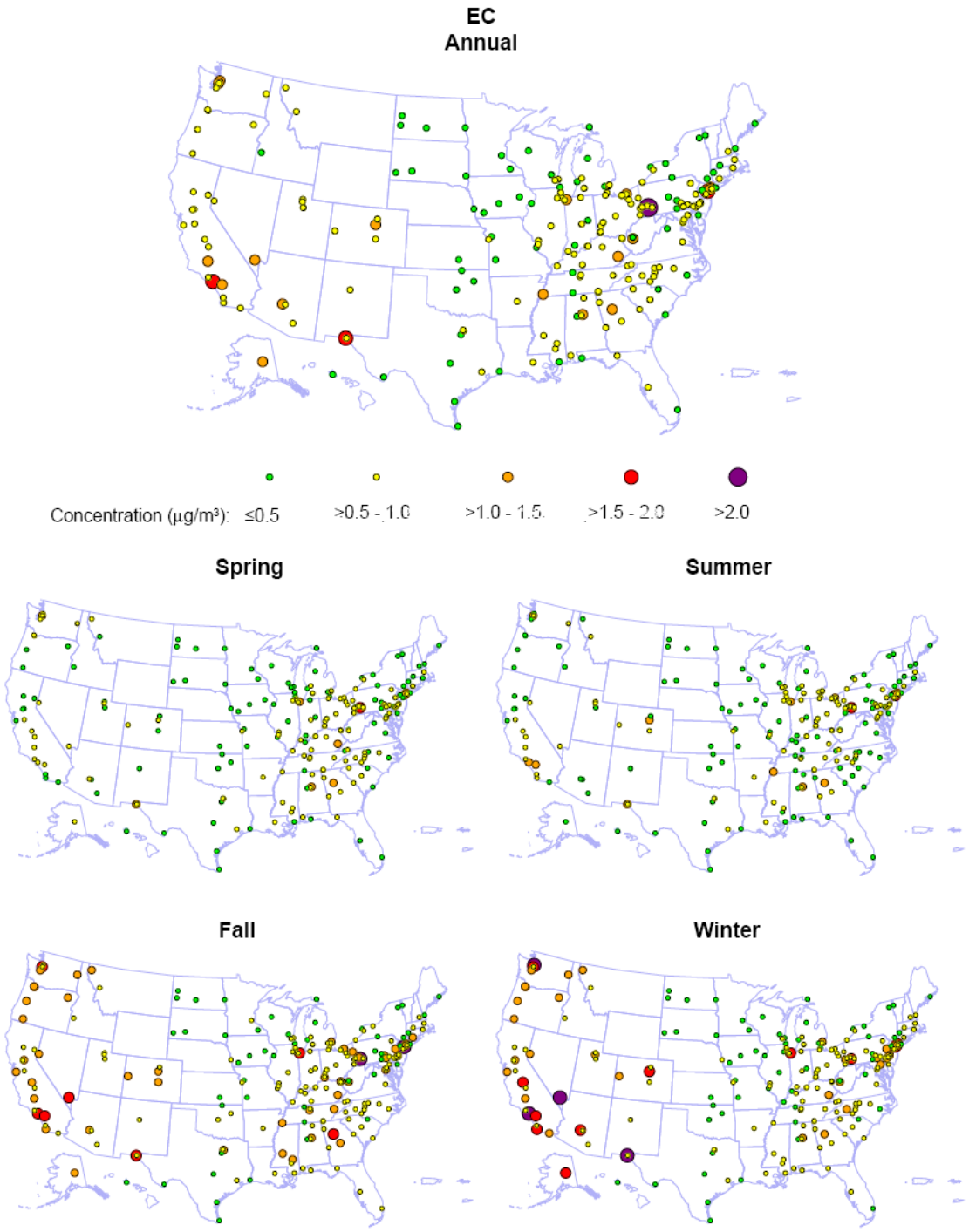


Figure 2-14. Three-year average 24-hour $PM_{2.5}$ SO_4^{2-} concentrations measured at CSN sites across the U.S., 2005-2007.

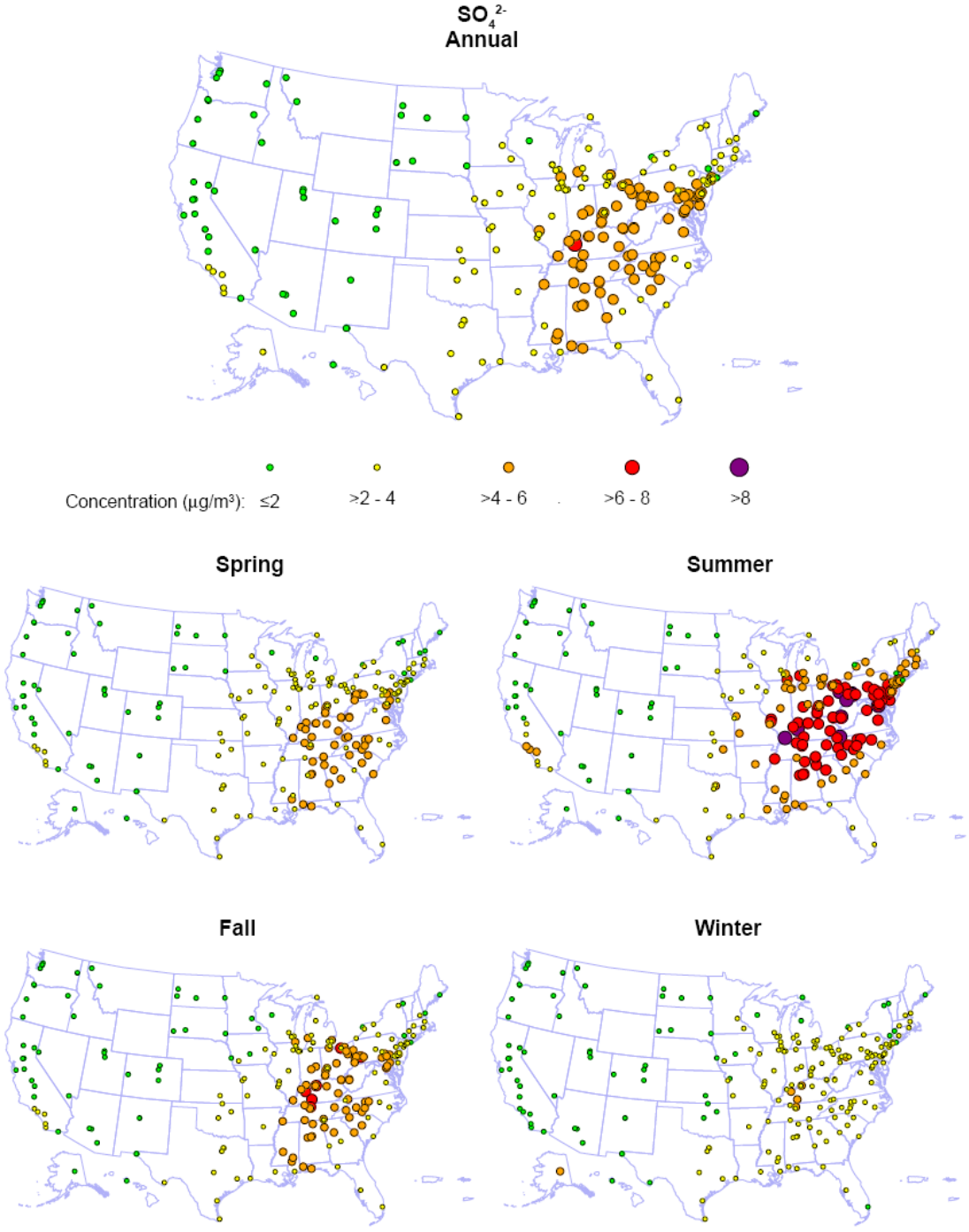


Figure 2-15 Three-year average 24-hour $PM_{2.5}$ NO_3^- concentrations measured at CSN sites across the U.S., 2005-2007.

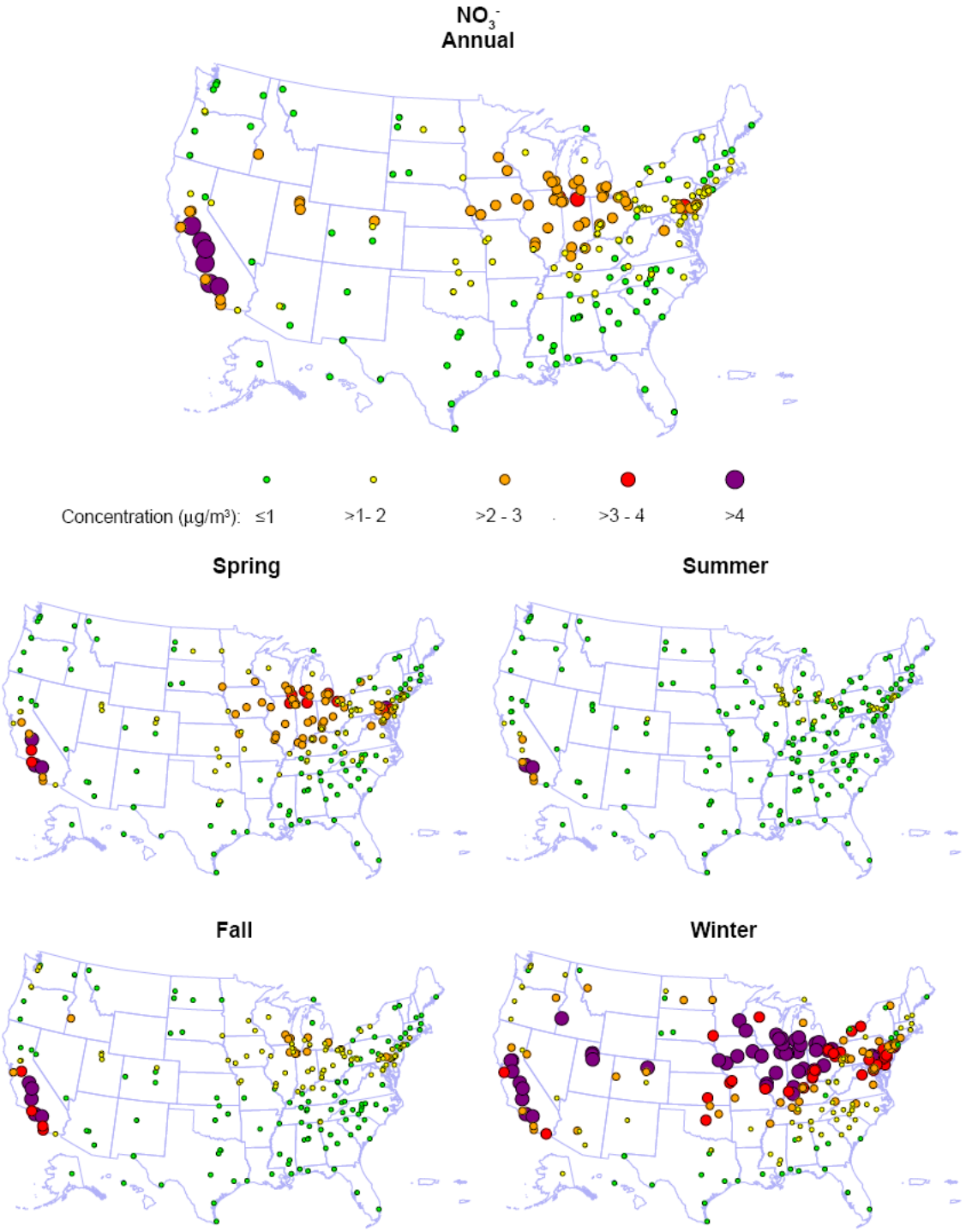
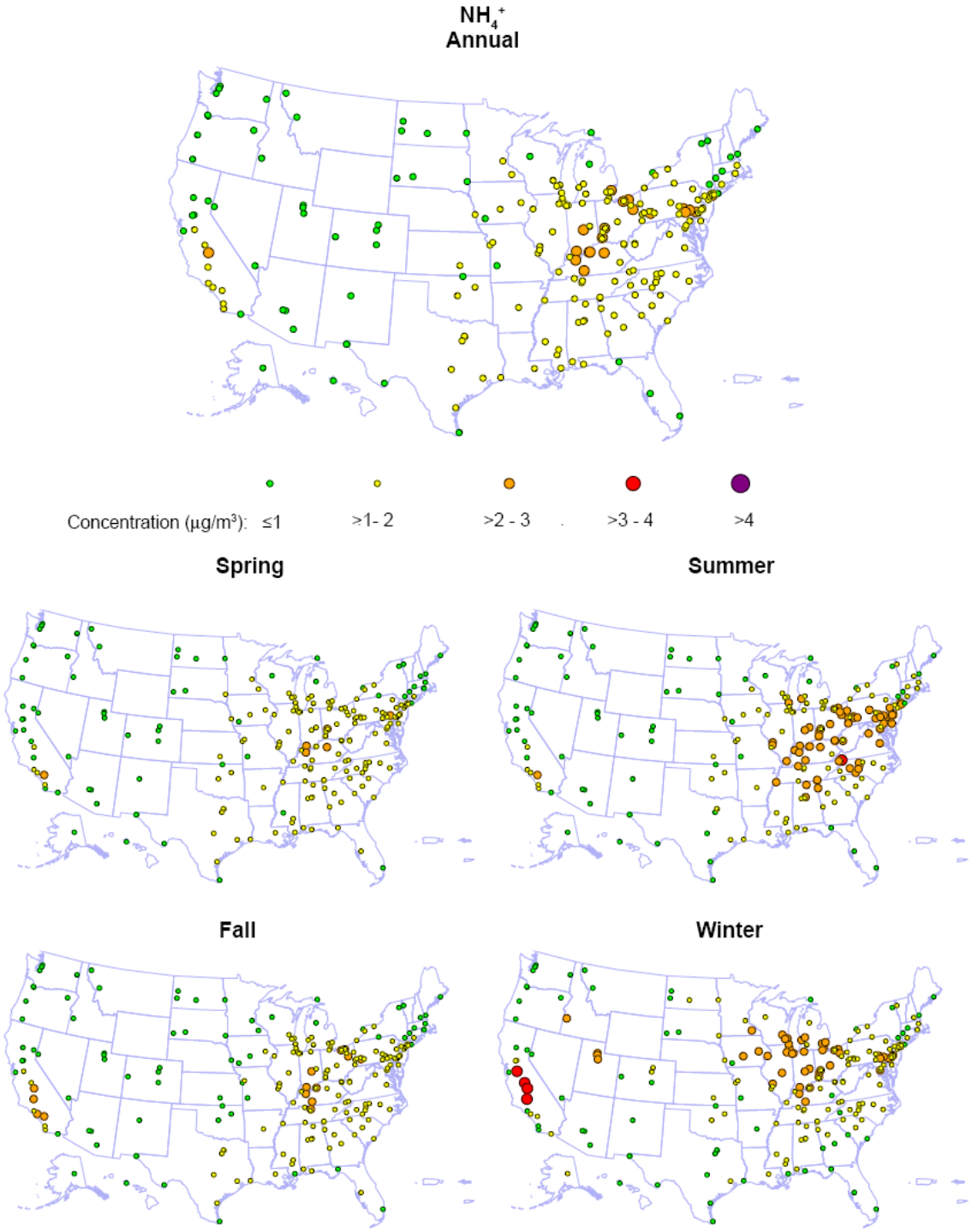


Figure 2-16 Three-year average 24-hour $PM_{2.5}$ NH_4^+ concentrations measured at CSN sites across the U.S., 2005-2007.



the next largest component in the East. In the West, OCM is the largest constituent on an annual basis. SO_4^{2-}, NO_3^-, and crustal material are also important in many of the included western cities. In the West, fractional SO_4^{2-} ranges from 18% in Denver to 32% in Los Angeles while fractional NO_3^- is relatively large in Riverside (22%), Los Angeles (19%) and Denver (15%) and less important on an annual basis in Phoenix (1%) and Seattle (2%). Crustal material is particularly prevalent in Phoenix (28%). Elemental carbon makes up a smaller fraction of the $PM_{2.5}$ mass (4 to 11%), but it is consistently present in all included cities regardless of region *(US EPA, 2009a, section 3.5.1.1*).

The seasonal variation in $PM_{2.5}$ composition across the fifteen CSAs/CBSAs is shown in Figure 2-18. SO_4^{2-} dominates in most metropolitan areas in the summertime, while NO_3^- becomes important in the colder wintertime months. Notable summertime exceptions include Denver, Phoenix, and Seattle, where SO_4^{2-} makes up a smaller fraction of the $PM_{2.5}$ mass. Likewise, NO_3^- is less pronounced in the wintertime in Atlanta, Birmingham, Houston, Phoenix, and Seattle. Los Angeles and Riverside exhibit elevated NO_3^- from fall through spring. Crustal material is a substantial summertime component in Houston (26%), much of which is attributed by the state air agency to transport from Africa.[28] Crustal material is generally low elsewhere in the East in all seasons. In the West, crustal material represents a substantial component year-round in Phoenix and Denver. *(US EPA, 2009a, section 3.5.1.1*)

[28] See, for example, "South, Central, and Southeast Texas Saharan Dust August 27-29, 2006", http://www.tceq.state.tx.us/assets/public/compliance/monops/air/sigevents/06/event2006-08-27txs.html)

Figure 2-17. Three-year average $PM_{2.5}$ speciation estimates for 2005-2007 derived using the SANDWICH method for fifteen CSAs/CBSAs.

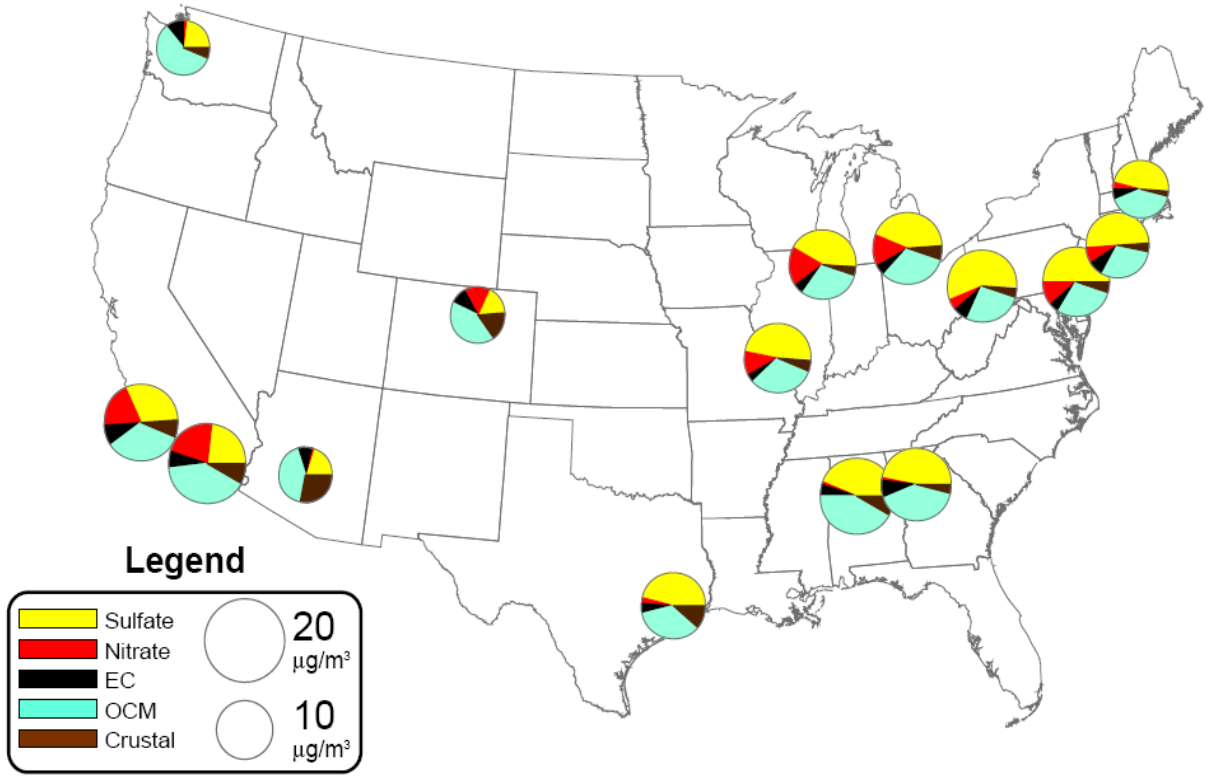


Figure 2-17 notes: The fifteen areas evaluated (with the number of sites per CSA/CBSA listed in parenthesis) included: Atlanta, GA (1); Birmingham, AL (3); Boston, MA (4); Chicago, IL (7); Denver, CO (2); Detroit, MI (4); Houston, TX (1); Los Angeles, CA (1); New York City, NY (7); Philadelphia, PA (6); Phoenix, AZ (2); Pittsburgh, PA (4); Riverside, CA (1); Seattle, WA (4); and St. Louis, MO (3). SO_4^{2-} and NO_3^- estimates include NH_4^+ and particle bound water and the circles are scaled in proportion to FRM $PM_{2.5}$ mass as indicated in the legend.

Figure 2-18. Seasonally-stratified three-year average $PM_{2.5}$ speciation estimates for 2005-2007 derived using the SANDWICH method for fifteen CSAs/CBSAs.

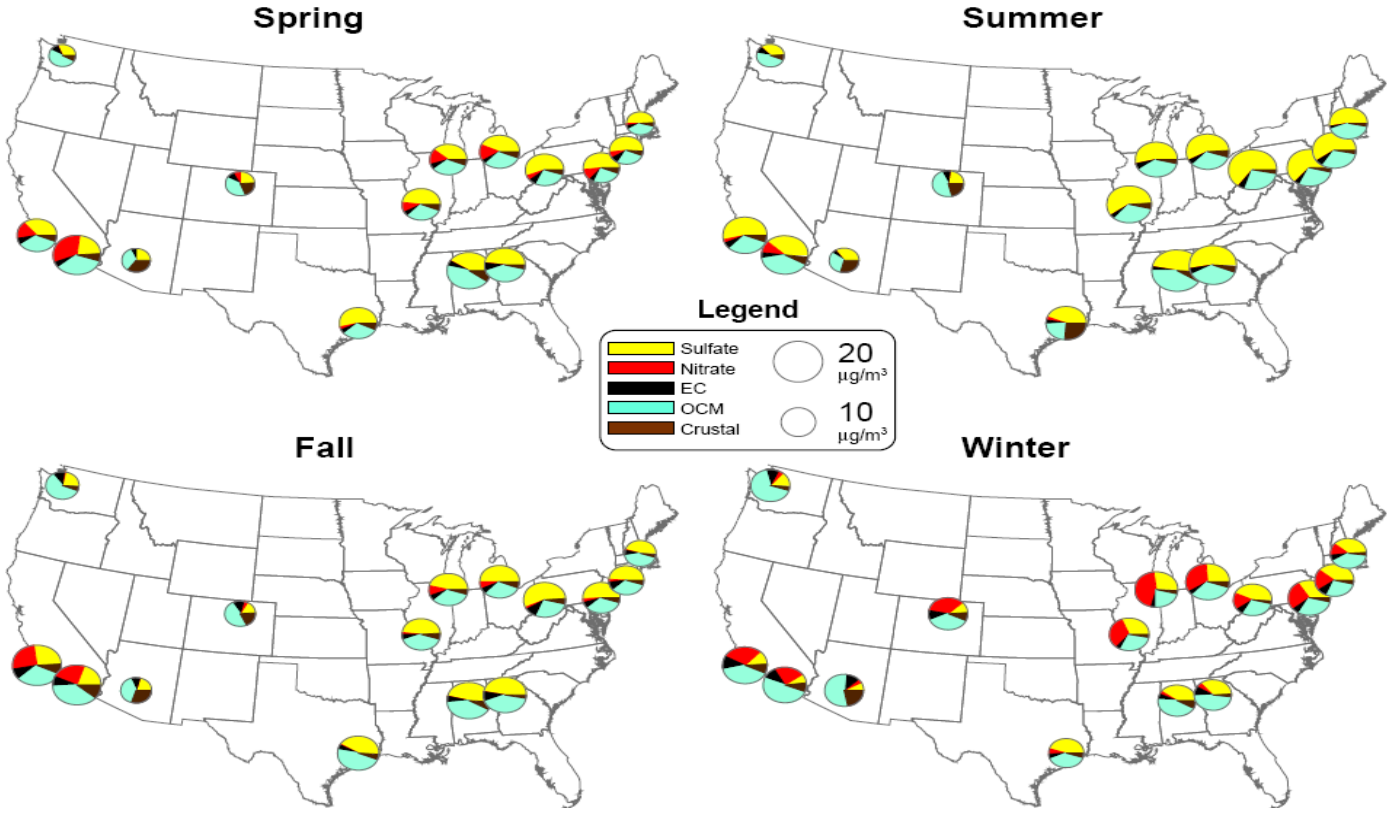


Figure 2-18 notes: The following fifteen CSAs/CBSAs were evaluated: Atlanta, GA; Birmingham, AL; Boston, MA; Chicago, IL; Denver, CO; Detroit, MI; Houston, TX; Los Angeles, CA; New York City, NY; Philadelphia, PA; Phoenix, AZ; Pittsburgh, PA; Riverside, CA; Seattle, WA; and St. Louis, MO. SO_4^{2-} and NO_3^- estimates include NH_4^+ and particle bound water and the circles are scaled in proportion to FRM $PM_{2.5}$ mass as indicated in the legend.

2.5.5.2 $PM_{10-2.5}$

Only speciated $PM_{2.5}$ is collected routinely at CSN network sites, resulting in far less information on speciated $PM_{10-2.5}$ than speciated $PM_{2.5}$. Edgerton et al. (2005, 2009) published speciated measurements for $PM_{2.5}$ and $PM_{10-2.5}$ obtained using dichotomous samplers from four locations included in the Southeastern Aerosol Research and Characterization (SEARCH) study: Yorkville, GA, Centreville, AL, Birmingham, AL, and Atlanta, GA. Samples were collected between 1999 and 2003 on a 1-in-3 day or 1-in-6 day schedule, depending on site. Speciated measurements for both $PM_{2.5}$ and $PM_{10-2.5}$ included SO_4^{2-}, NO_3^-, NH_4^+, and major metal oxides (MMO). In addition, OC and either black carbon (BC) or EC were reported for $PM_{2.5}$ over the entire study period and for $PM_{10-2.5}$ for a subset of samples extending from April 2003 to April 2004. *(US EPA, 2009a, section 3.5.1.1*)

For the Atlanta and Birmingham SEARCH sites, the annual average NO_3^- mass fraction was approximately equal for $PM_{2.5}$ (5.6% and 5.0%, respectively, for Atlanta and Birmingham) and PM_{10}-$_{2.5}$ (4.9% and 3.3%). Likewise, the OC mass fraction was approximately equal for $PM_{2.5}$ (26% and 26%) and $PM_{10-2.5}$ (24% and 27%). MMO contributed an order of magnitude smaller mass fraction to $PM_{2.5}$ (2.6% and 4.7%) than $PM_{10-2.5}$ (38% and 35%). In contrast, SO_4^{2-} contributed an order of magnitude greater mass fraction to $PM_{2.5}$ (25.1% and 24.1%) than $PM_{10-2.5}$ (2.8 and 2.1%). BC also represented a slightly larger mass fraction of $PM_{2.5}$ (8.6% and 10.5%) than EC did for $PM_{10-2.5}$ (2.9% and 2.4%). Based on these findings, MMO are present primarily in the thoracic coarse mode while SO_4^{2-} and EC/BC are present primarily in the fine mode. NO_3^- and OC are present in both modes in approximately equal mass fractions. These results are specific to Atlanta and Birmingham and may not represent other geographic regions. However, they are consistent with the current understanding of sources and formation of these constituents and therefore likely resemble the general compositional split between fine and thoracic coarse mode particles. *(US EPA 2009a, section 3.5.1.1*)

As noted in the previous review, primary biological aerosol particles (PBAP), which include microorganisms, fragments of living things, and organic compounds of miscellaneous origin in surface deposits on filters, are not distinguishable in analyses of total OC (US EPA, 2004, second 3.2). A clear distinction should be made between PBAP and primary OCM produced by organisms (e.g., waxes coating the surfaces of organisms) and precursors to secondary OCM such as isoprene and terpenes. Indeed, the fields of view of many photomicrographs of PM samples obtained by scanning electron microscopy are often dominated by large numbers of pollen spores, plant and insect fragments, and microorganisms. The ISA summarizes several studies of the contribution of PBAP to PM in different size ranges,

particularly $PM_{10-2.5}$, generally concluding that PBAP contributes substantially to $PM_{10-2.5}$ OCM measurements.

2.5.5.3 Ultrafine Particles

Information about the composition of ambient ultrafine particles directly emitted by sources is still sparse compared to that for the larger size modes. However, their composition is expected to reflect that of the sources emitting these particles. Particle number emissions from motor vehicles are predominantly in the ultrafine size range. The composition of gasoline vehicle emissions consists mainly of a mix of OCM, EC, and small quantities of trace metals and sulfates, with OCM constituting anywhere from 26-88% of PM. Diesel PM is generally comprised of an EC and trace metal ash core, onto which organic material condenses, and nucleation-mode SO_4^{2-}. With the introduction of new diesel emissions standards in 2007, total emissions have decreased dramatically, particularly for carbon. In areas where nucleation is the dominant source of ultrafine particles, sulfate along with ammonium and secondary organic compounds are the likely major components of ultrafine particles. *(US EPA, 2009a, section 3.5.1.1*)

In a study conducted at several urban sites in Southern California, carbon, in various forms, was found to be the major contributor to the mass of ultrafine particles. However, ammonium was found to contribute 33% of the mass of ultrafine particles at one site in Riverside. Iron was the most abundant metal found in the ultrafine particles. Chung et al. (2001) found that carbon was the major component of the mass of ultrafine particles in a study conducted during January of 1999 in Bakersfield, CA. However, the contribution of carbonaceous species was much lower than that found in the cities in Southern California, with the crustal elements calcium, silicon, and aluminum also contributing notable mass percentages. *(US EPA 2009a, section 3.5.1.1*)

Herner et al. (2005) reported a gradual increase in OC mass fraction as particle size decreases from 1 μm (20% OC) to 100 nm (80% OC) in the San Joaquin Valley of California. Sardar et al. (2005) found OC to be the major component of ultrafine particles at four locations in California, with higher OC mass fraction in the wintertime relative to summertime. Elemental carbon and SO_4^{2-} were also present in the ultrafine samples but at much smaller mass fractions. Furthermore, EC was present year-round whereas SO_4^{2-} had a summertime preference. More detailed chemical characterization of the OC fraction of ambient ultrafine particles is extremely limited, but recent studies have identified specific organic molecular markers affiliated with

motor vehicle emissions including hopanes and polycyclic aromatic hydrocarbons (Fine et al., 2004; Ning et al., 2007; Phuleria et al., 2007). *(US EPA 2009a, section 3.5.1.1*)

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

$PM_{10-2.5}$ to $PM_{2.5}$ ratios are generally lower in the east than in the west, and lower in rural areas than in urban areas, except in dry rural areas. The Criteria Document from the last review compared collocated $PM_{2.5}$ and $PM_{10-2.5}$ concentrations, averaged over the same years. The mean $PM_{2.5}$ to $PM_{10-2.5}$ ratio was calculated for each of 17 MSAs using as many sites as possible. The median mean $PM_{2.5}$ to $PM_{10-2.5}$ ratio for the 17 MSAs was 1.2. For eight MSAs (Tampa, FL; Columbia, SC; Louisville, KY; Chicago, IL; Gary, IN; Milwaukee, WI; Steubenville, OH; Portland, OR), the mean $PM_{2.5}$ concentration exceeded the mean PM10-2.5 concentration. For an additional eight MSAs, the $PM_{2.5}$ and $PM_{10-2.5}$ concentration means were the same (within one SD). Salt Lake City was the only MSA for which the mean $PM_{10-2.5}$ concentration exceeded the mean $PM_{2.5}$ concentration. (US EPA, 2004, section 3.1.2)

The 2005 Staff Paper presented information on the relationships among PM indicators in different regions based on 2001-2003 data from the nationwide PM FRM monitoring networks. The ratios of annual mean $PM_{2.5}$ to PM_{10} at sites in different geographic regions were reported to be 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 (US EPA, 2005, section 2.4.6). These data are generally consistent with earlier findings reported in the previous review from a more limited set of sites (US EPA, 1996, sections 6.3.1.4 and 6.3.2). $PM_{2.5}$ 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} was found to be fairly well correlated with estimated $PM_{10-2.5}$ in most regions, with the highest average correlations in the southwest, upper midwest, and southern California regions. The Staff Paper concluded that these data suggest that PM_{10} might be a suitable indicator for either fine or coarse particles, depending upon location-specific factors.[29] However, in all locations estimated $PM_{10-2.5}$ and $PM_{2.5}$ were very poorly correlated, which should be expected due to their differences in origin, composition, and atmospheric behavior.

In the second draft ISA, which restricted its $PM_{10-2.5}$ analysis to the small network of collocated low-volume sampler pairs for PM_{10} and $PM_{2.5}$, only six CSAs/CBSAs had sufficient data for calculating $PM_{10-2.5}$. In general, in the eastern metropolitan areas including Atlanta,

[29] In the final action for the review completed in 2006, the existing 24-hour PM_{10} standard was retained, with an explanation regarding its appropriateness as an indicator for $PM_{10-2.5}$. See 71 FR 61185, October 17, 2006.

Boston, Chicago, and New York, most of the mass of PM_{10} was in the $PM_{2.5}$ size fraction, with the highest ratio of annual average 24-hour $PM_{2.5}$ to $PM_{10-2.5}$ in Chicago (14 $\mu g/m^3$ $PM_{2.5,}$ 5 $\mu g/m^3$ $PM_{10-2.5,}$ ratio = 2.8). In contrast, in Denver (9 $\mu g/m^3$ $PM_{2.5}$, 20 $\mu g/m^3$ $PM_{102.5}$, ratio = 0.45) and Phoenix (10 $\mu g/m^3$ $PM_{2.5}$, 22 $\mu g/m^3$ $PM_{10-2.5}$, ratio = 0.45) most of PM_{10} was in the thoracic coarse mode (US EPA 2009a, section 3.9.1.1). These findings are directionally consistent with those summarized above from the last review.

2.6 PM TEMPORAL PATTERNS

The 2005 Staff Paper and the second draft ISA both have extensive, data-heavy analyses of PM temporal patterns, the Staff Paper for 2001-2003 and the second draft ISA for 2006-2007. Their qualitative findings are similar and can be assumed to generally apply to current conditions, recognizing that ongoing reductions from large emission source categories such as mobile sources and electric generating units are gradually affecting these patterns.

Hourly PM_{10} and $PM_{2.5}$ measurements are conducted at many sites using beta gauge or TEOM monitors. Many of the hourly measurements for PM_{10} have FRM or FEM status. The ISA used all available hourly data from FRM, FEM and FRM-like monitors in fifteen CSAs/CBSAs to investigate diel variation in PM. Plots of $PM_{2.5}$ by hour of the day were stratified by weekdays and weekends. In most cities investigated, a morning $PM_{2.5}$ peak is present starting at approximately 6:00 a.m., corresponding with the start of the morning rush hour just before the break-up of overnight stagnation. In Pittsburgh, dispersion behavior during the night results in elevated $PM_{2.5}$ concentrations throughout the night that blend in with any morning peak. Six other metropolitan areas (i.e., Atlanta, Chicago, Seattle, St. Louis, Houston, and New York) show two distinct daily peaks on both the weekdays and weekends. The evening $PM_{2.5}$ concentration peak is broader than the morning peak and extends to overnight hours, reflecting the concentration increase caused by a drop in boundary layer height at night.

Figure 2-19 compares the two-peak distribution in $PM_{2.5}$ for Seattle with the one-peak distribution in $PM_{2.5}$ for Pittsburgh. Since these figures represent the distribution of hourly observations over a 3-year period, any fluctuations or changes in the timing of the daily peaks would result in a broadening of the curves shown in the plot. That is, individual days would tend to have sharper peaks than shown in the graphs.

Figure 2-19. Diel plot generated from hourly FRM-like $PM_{2.5}$ data ($\mu g/m^3$) stratified by weekday (left) and weekend (right) for Pittsburgh, PA, and Seattle, WA, from 2005 to 2007.

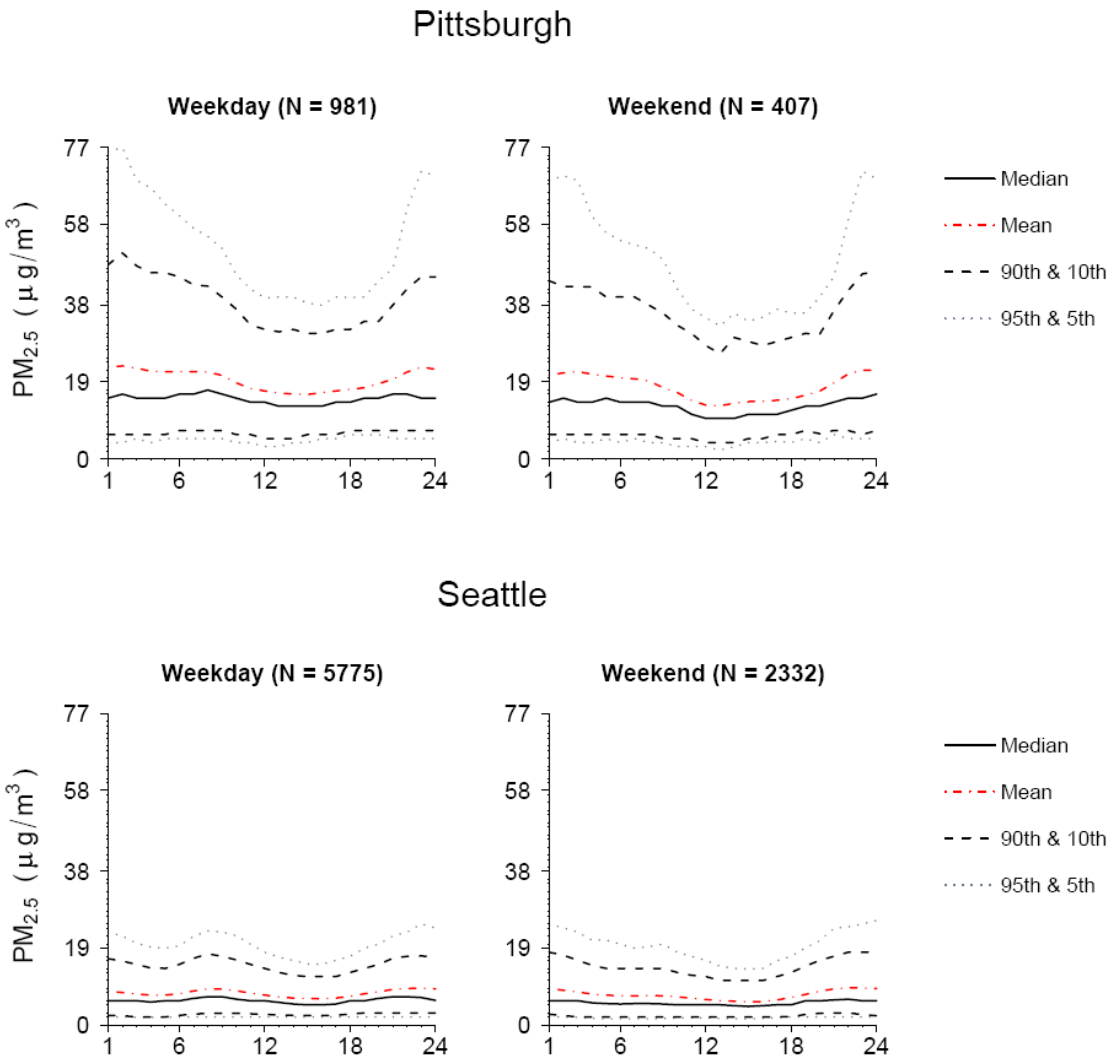


Figure 2-19 notes: Included are the number of monitor days (N) and the median, mean, 5th, 10th, 90th and 95th percentiles for each hour of the day shown on the horizontal axis.

Eleven cities showed a gradual morning increase in mean PM_{10} starting at approximately 6:00 a.m. on weekdays, corresponding with the start of the morning rush hour before the break-up of overnight stagnation. The magnitude and duration of this peak, however, varies considerably by area. Phoenix shows the most pronounced morning PM_{10} peak concentration, which drops off during the day and reappears in the evening. In contrast, Chicago shows a less pronounced peak with the PM_{10} concentration remaining elevated throughout the day. Figure 2-20 shows the plots of PM_{10} for Chicago and Phoenix. (Annex A of the ISA contains similar

plots for nine other areas.) In both instances, the weekend pattern is similar in shape to the weekday pattern with less pronounced peaks. Once again, any fluctuations in the timing of the daily peaks could result in a broadening of the peaks in the 3-year composite figures, and individual days typically will have somewhat sharper peaks.

Figure 2-20. Diel plots generated from hourly FEM PM_{10} data ($\mu g/m^3$) stratified by weekday (left) and weekend (right) for Chicago, IL, and Phoenix, AZ, from 2005 to 2007.

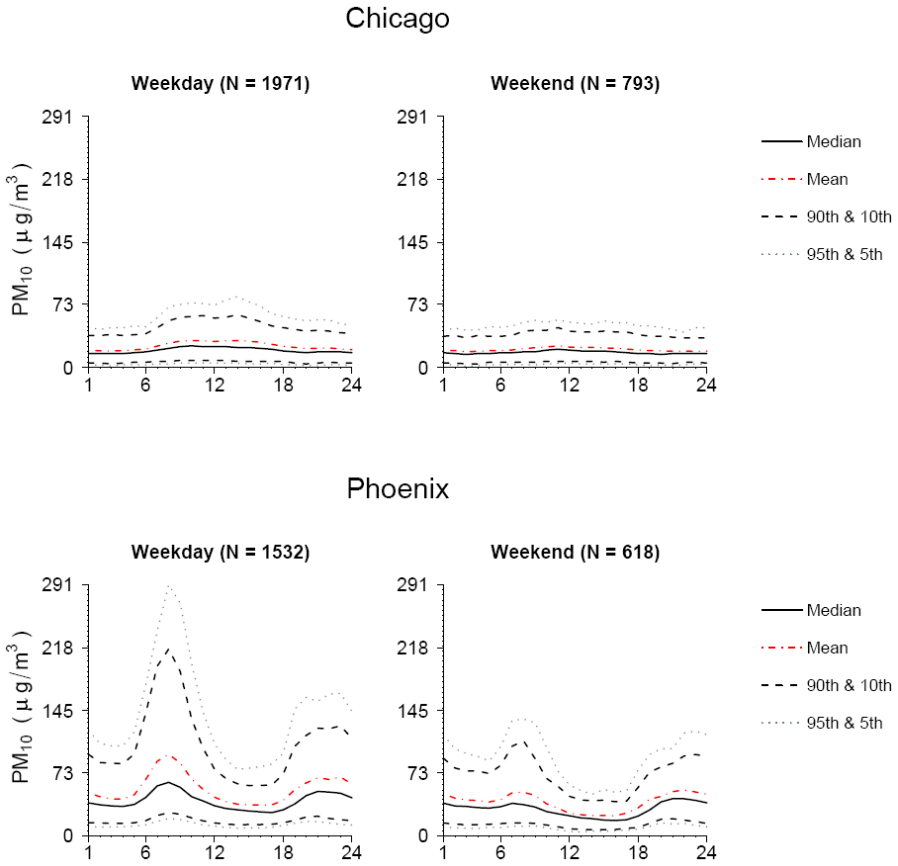


Figure 2-20 notes: Included are the number of monitor days (N) and the median, mean, 5th, 10th, 90th and 95th percentiles for each hour of the day shown on the horizontal axis.

Superimposed on the daily patterns described above, distinct seasonal variations in $PM_{2.5}$ concentrations can be seen in air quality data but are generally not as sharp as those seen for ozone concentrations. The months with peak urban $PM_{2.5}$ concentrations vary by region and season. 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. 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. These patterns are consistent with our understanding of dominant emission sources and atmospheric processes. For example, more carbon is produced when woodstoves and fireplaces are used, and 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. The chemical components of $PM_{2.5}$ also exhibit seasonal patterns as described in section 2.4.5.

The lowest concentrations of $PM_{10-2.5}$ 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, with elevated levels apparent in the spring, consistent with winds that contribute to windblown dust. In areas subject to impact from forest fires, both $PM_{2.5}$ and $PM_{10-2.5}$ can have extreme peaks during the local high fire hazard season, if fires are upwind.

The Urban-Focused Visibility Assessment, being conducted for this review, uses a combination of four types of 24-hour and continuous PM monitoring to estimate hourly $PM_{2.5}$, $PM_{2.5}$ components, and $PM_{10-2.5}$ during daylight hours, for 15 study areas (US EPA, 2009c). The initial results are qualitatively consistent with the characterizations described above regarding the typical existence of one or two peaks in PM concentrations during the day. Visualizations of these patterns are provided in the draft assessment document (US EPA, 2009c, Figures 3-9 and E-1).

Study of the relationship for $PM_{2.5}$ 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 2005 Staff Paper used box plots of 2001-2003 $PM_{2.5}$ data at FRM sites across the U.S to show the relationship for $PM_{2.5}$ between annual mean PM concentrations and peak daily concentrations as represented by the 98^{th} percentile of the distribution of 24-hour average concentrations. Sites were assigned to bins based on 1 $\mu g/m^3$ increments of annual average $PM_{2.5}$ concentration, and the distribution of 98^{th} percentile values was shown for each bin. Although there was a clear monotonic relationship between 98^{th} percentiles and annual means, there was considerable variability in 98^{th} percentile values for sites with similar annual means. For the bins with annual mean $PM_{2.5}$ values between 10 and 15 $\mu g/m^3$, the interquartile range of 98^{th} percentile concentrations (representing a middle group of 40 to 60 sites in each bin) spans about 5 to 6 $\mu g/m^3$ (US EPA, 2005, Figures 2-25 and 2-26 based on Schmidt et al., 2005).

In a new analysis for the current PM NAAQS review, EPA staff have further studied the relationship between annual average $PM_{2.5}$ and 98th percentile 24-hour $PM_{2.5}$, using 2005-2007 data. Figure 2-21 is a scatter plot of these two statistics for 2005-2007, with each point representing one year at a monitoring site that met EPA completeness requirements for calculating valid design values for both $PM_{2.5}$ NAAQS for that period, and that is eligible for comparison to both NAAQS. As was noted in the 2005 Staff Paper, there is a wide range of 98th percentile 24-hour concentrations associated with the data points falling into narrow ranges of annual average concentrations. (Note that in Figure 2-21 it is evident that 2005 was a worse year for 98th percentile and annual average $PM_{2.5}$ concentrations than the next two years, as has already been noted in section 2.4.1) Figure 2-22 is similar to Figure 2-21 in concept but aggregates data to a more policy relevant level, focusing on the monitoring site and the metric that would determine attainment or nonattainment for the urban area as a whole. In Figure 2-21, 3-year design values are compared, rather than individual year statistics. In addition, Figure 2-22 has data points only for those monitoring sites that have the highest 3-year annual design value within their CBSA, i.e., the sites that determine whether all (or part) of the CBSA is nonattainment with the annual $PM_{2.5}$ NAAQS. This level of aggregation and screening thins out the "cloud" of data points but does not appreciably change its location or shape.

To provide further visualization of the diversity of location situations regarding the pattern of 24-hour $PM_{2.5}$ concentrations that determine the annual average concentrations, Figure 2-23 shows in bar chart form the history of individual 24-hour $PM_{2.5}$ concentrations in 2005-2007 for four example monitoring sites, in the following CBSAs: Augusta-Richmond County, GA-SC; El Centro, CA; Pittsfield, MA; and Beckley, WV. The sampling frequency in all four CBSAs was every third day. These correspond to the indicated (in red) data points in the "cloud" of $PM_{2.5}$ design value data points. El Centro and Pittsfield are at the upper/left edge of the dense part of the cloud. In these two areas, a relatively small number of days have substantially higher concentrations than other days, and the 98th percentile 24-hour $PM_{2.5}$ concentrations are relatively high compared to their annual average concentrations. Augusta-Richmond County and Beckly are at the lower/right edge of the dense part of the data cloud. In these two areas, there is less range in the daily values, and the 98th percentile concentrations are relatively not as high compared to their annual average concentrations.[30] We note that none of the $PM_{2.5}$ data for these four areas for this time period have been flagged by the respective states

[30] The areas represented by data points that are above the dense part of the data cloud were not selected for this illustration because their current design values are outside the range of alternative standards examined in this review, and hence are not of as much interest with respect the assessment of those alternatives.

to request exclusion from regulatory use under EPA's Exceptions Events Rule, although that does not exclude the possibility than an exceptional event did influence the $PM_{2.5}$ concentration on one or more days.

Figure 2-21. Relationship of annual average and 98th percentile 24-hour $PM_{2.5}$ concentrations at the site and year level, all sites with complete enough data for calculation of valid 2005-2007 design values.

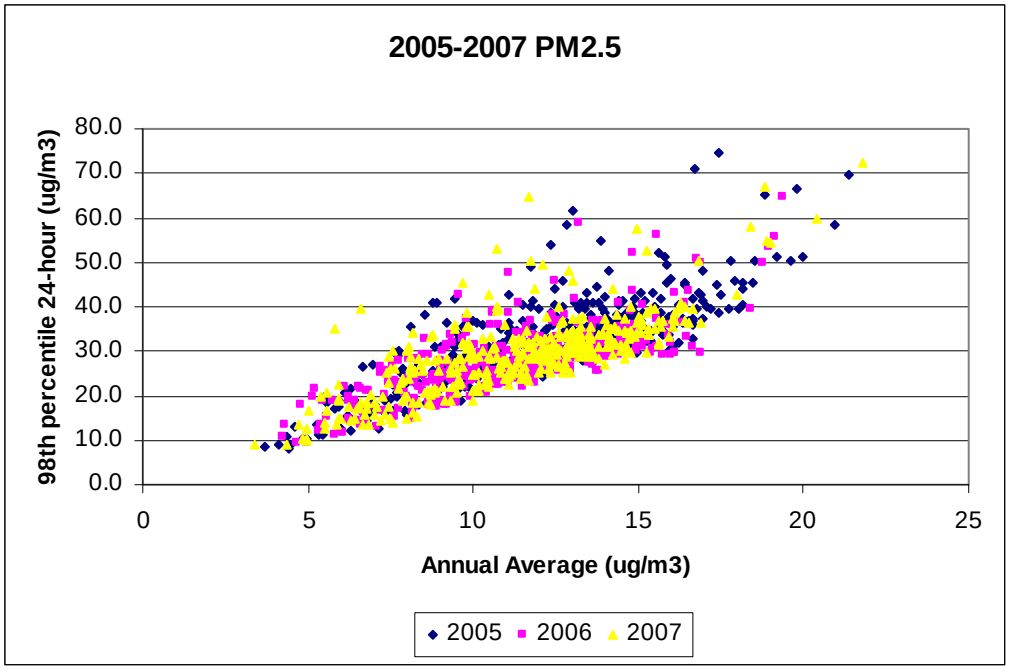


Figure 2-22 Relationship of annual average and 98th percentile 24-hour $PM_{2.5}$ 3-year design values, only for sites with the highest annual design value in their CBSA.

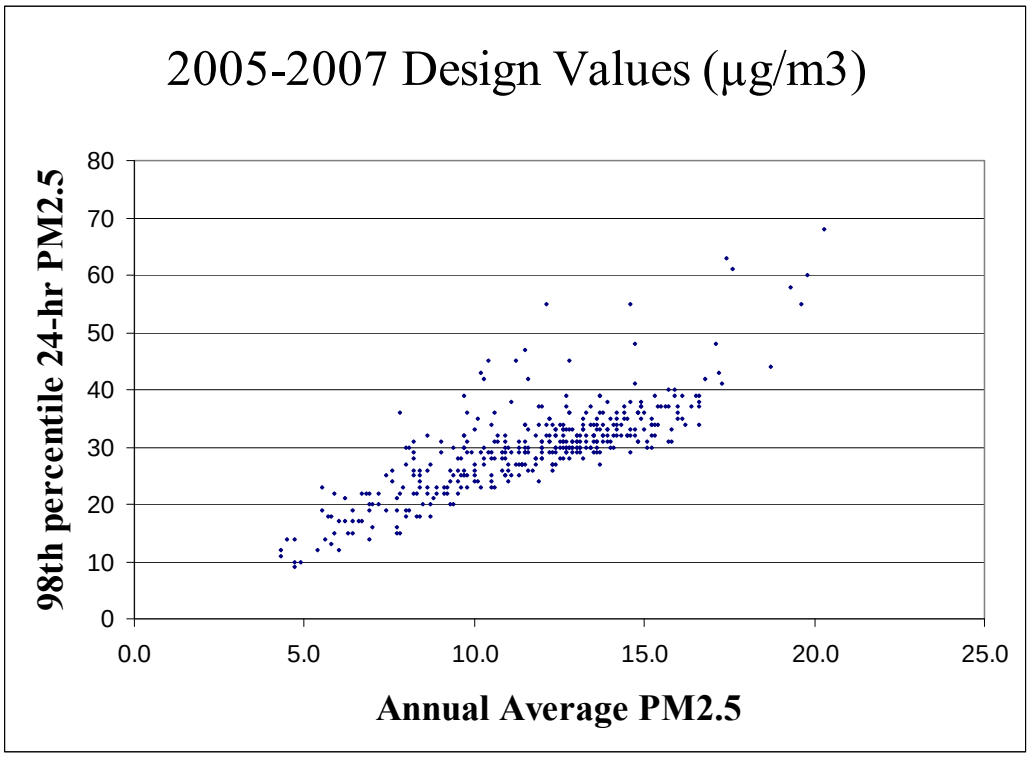
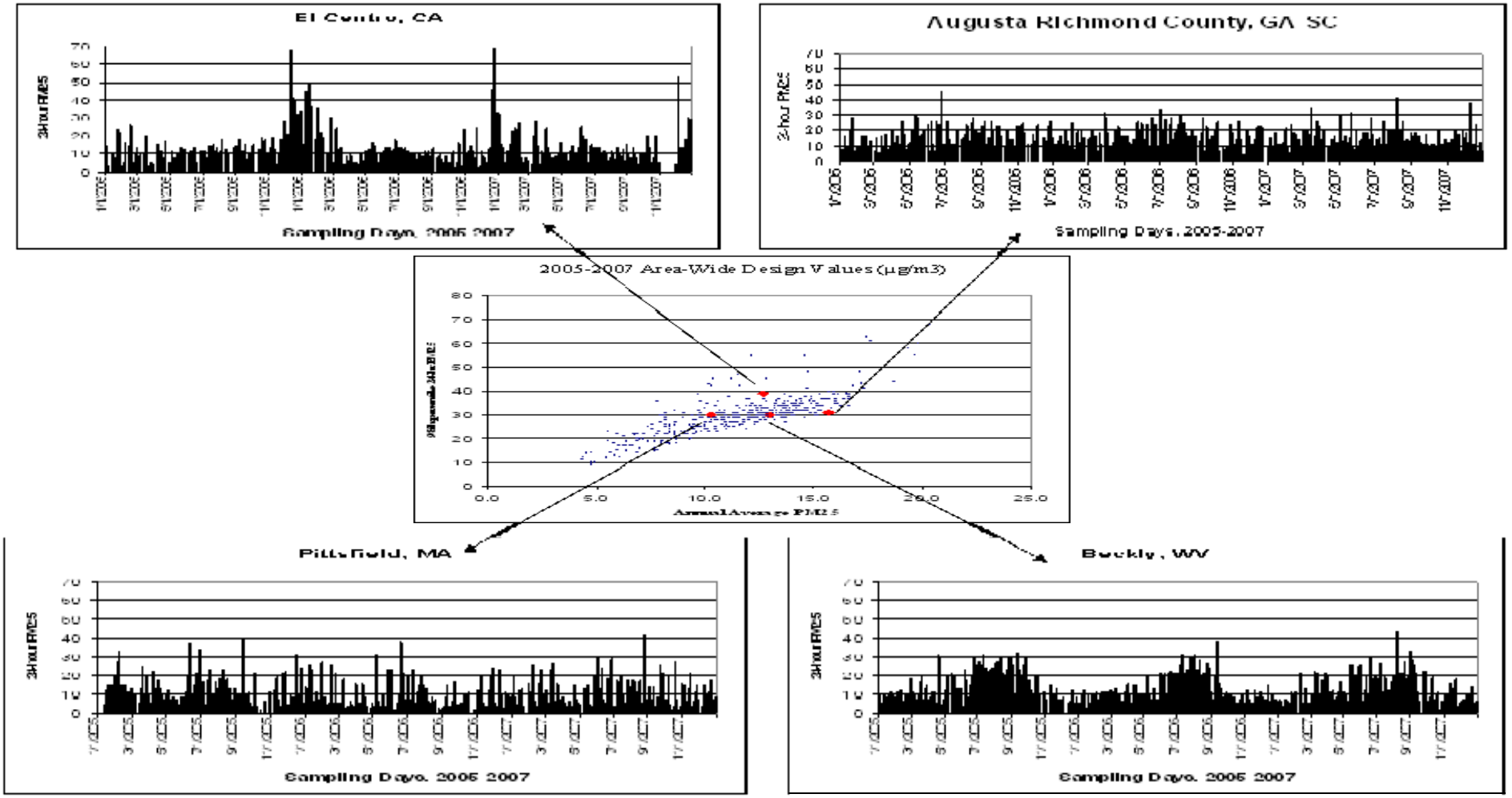


Figure 2-23. Three-year history of 24-hour $PM_{2.5}$ concentrations at highest design values site in four example CBSAs



2.7 PM BACKGROUND LEVELS

For the purposes of this document, background PM is defined as the distribution of PM concentrations that would be observed in the U.S. in the absence of anthropogenic (man-made) emissions of primary PM and precursor (e.g., VOC, NO_x, SO_2, and NH_3) compounds in the U.S., Canada, and Mexico. Background levels so defined are referred to as policy-relevant background levels, 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.

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.

Particulate matter can be transported long distances from natural or quasi-natural events occurring outside the continental U.S. 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.

Background concentrations of $PM_{2.5}$, $PM_{10\text{-}2.5}$, 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. EPA has had in place for many years policies that could remove consideration of data affected by such events, where appropriate, from attainment decisions. In March 2007, EPA adopted a binding rule that allows exclusion of data affected by so-called exceptional events, including natural events, from regulatory decisions such as attainment determinations (72 FR 13560, March 22, 2007). This rule has superseded the prior administrative policies.

There are several methods for characterizing PRB concentrations of PM within the United States. Some methods rely upon analyses of measured PM concentrations at remote rural locations, while other methods utilize air quality chemical transport models (CTMs) to estimate PRB. The last review characterized PRB for $PM_{2.5}$ on a 24-hour average basis by summarizing the non-sulfate portion of $PM_{2.5}$ measured at Interagency Monitoring of Protected Visual Environment (IMPROVE) sites in remote areas between 1990 and 2002. Sulfate was omitted because it is attributable almost entirely to anthropogenic emissions. It was noted that this method likely resulted in an underestimate of PRB (US EPA, 2005, section 2.6).[31]

In this review, EPA has relied on the results of the CTM modeling described in the second draft ISA to estimate PRB for $PM_{2.5}$ for inclusion in estimating risks (US EPA, 2009a, section 3.7). In addition, EPA staff has also used this CTM modeling to estimate PRB for $PM_{2.5}$ components for the urban-focused visibility risk assessment (see additional description in US EPA, 2009c, Appendix C). The CTM-based approach is based on a "zero-out" model simulation in which anthropogenic emissions inside the U.S., Canada, and Mexico are set to zero while all biogenic emissions for these areas and biogenic and anthropogenic emissions from elsewhere in the world are not altered. This approach can provide more spatial and temporal resolution for estimating PRB compared to the use of measurements given the sparse nature of remote

[31] The statement in the 2005 Staff Paper that the described method resulted in an underestimate of PRB reflects a judgment that the upward bias from relying on IMPROVE sites that are actually influenced by anthropogenic sources of PM components other than sulfate is greater than the downward bias from not including the actual, small PM sulfate component from natural sources and sources outside the US, Canada, and Mexico.

measurement sites and the concern that even remote sites are affected by non-local anthropogenic sources.

The CTM-based approach involves coupling the global-scale circulation model GEOS-Chem (Fiore, et al, 2003) with the regional scale air quality model CMAQ (Byum, et. al., 2006 and Byum, et. al, 1999). The GEOS-Chem model is run on a global scale and is used to provide estimates of transported pollutants from emissions of natural and anthropogenic sources outside the U.S., Canada, and Mexico. These transported pollutant concentrations are used to provide the "boundary condition" concentrations for two CMAQ simulations covering the continental U.S. and adjacent portions of Canada and Mexico (CONUS), one simulation with all emissions to evaluate model performance and one to estimate PRB. In the CMAQ simulation to estimate PRB, only natural emissions in the U.S., Canada, and Mexico are considered. The details of this modeling approach, including the input data sets and model chemistry, are described in section 3.7 of the second draft ISA. The following is a brief summary.

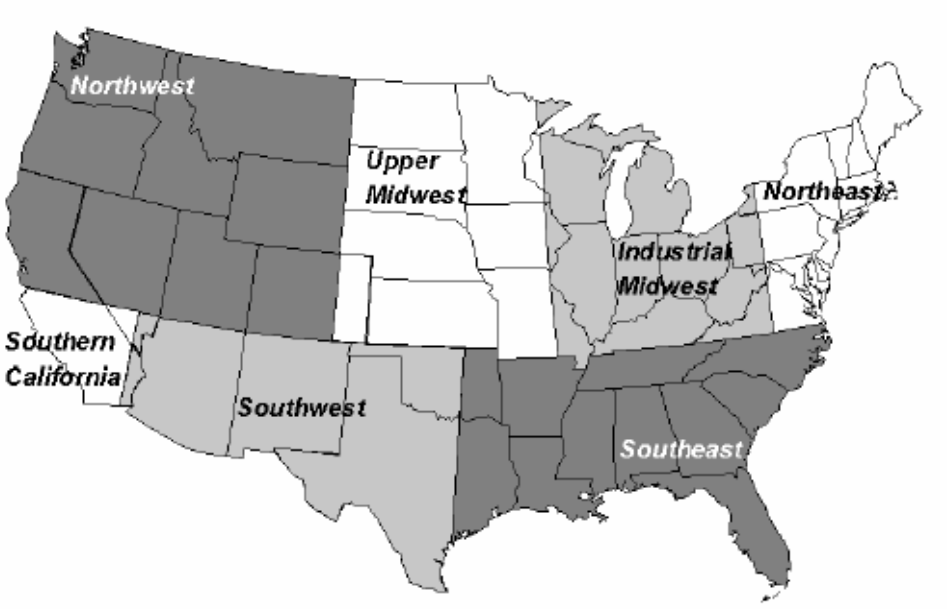
The two models were applied to simulate one year of air quality data for 2004. The base case CMAQ run for 2004 includes meteorology and all the anthropogenic and natural sources both within and outside of the U.S., Canada, and Mexico. This run was performed to provide a comparison of model predictions with measurements. The second draft ISA characterizes the CMAQ performance for the annual average concentrations and most of the seasonal averages of $PM_{2.5}$ at remote sites as "very good" in the East and Midwest. In the West, predictions at remote sites are "generally too low in all seasons." The second draft ISA notes that degraded performance in the West is not unexpected because the grid resolution in the CMAQ model simulation (36 km for this application) will smooth out significant variations in terrain that influence measured concentrations, particularly concentrations attributable to anthropogenic emissions which in the West are often concentrated in basin settings where local meteorological conditions coupled with local emissions of primary particles may dominate $PM_{2.5}$ concentrations. However, looking across the U.S., the model does correctly reproduce broad geospatial differences in that predicted $PM_{2.5}$ concentrations are lower at western locations than they are in the East; this finding is consistent with measured data. Also, natural emissions in the West are less concentrated in basin settings, and western terrain may, therefore, have less effect on model performance when estimating PRB.

In addition to the "base case" run which includes all anthropogenic and biogenic emissions, CMAQ was also run for a second scenario to estimate PRB, with the same boundary conditions but with only natural emissions from within the U.S., Canada, and Mexico. The hourly outputs from this second CMAQ run were used to calculate seasonal and annual average estimates of PRB within seven regions of the U.S.

For the health risk assessment, annual values presented in Table 2-4 were used in modeling long-term $PM_{2.5}$ exposures and estimating associated health risks (in those sensitivity analysis scenarios where risk was modeled down to PRB). For estimating risks associated with short-term $PM_{2.5}$ exposures (which involved modeling down to PRB, exclusively), quarterly values presented in Table 2-4 were used to represent the appropriate block of days within a simulated year (US EPA, 2009b, section 3.2.2).

Table 2-4. Annual and quarterly mean of the CMAQ-predicted PRB $PM_{2.5}$ concentrations ($\mu g/m^3$) in the U.S. EPA CONUS regions in 2004.

	Annual	January-March	April-June	July-September	October-December
Northeast	0.74	0.85	0.78	0.67	0.68
Southeast	1.72	2.43	1.41	1.41	1.64
Industrial Midwest	0.86	0.89	0.89	0.94	0.73
Upper Midwest	0.84	0.79	0.93	0.99	0.66
Southwest	0.62	0.61	0.76	0.70	0.40
Northwest	1.01	0.48	0.81	1.42	1.32
Southern California	0.84	0.54	0.92	1.21	0.67



For the purposes of the urban focused visibility assessment, EPA extracted hour-specific monthly average $PM_{2.5}$ components from the PRB CMAQ run, averaging across the several CMAQ grid cells that overlap with the Census-defined urbanized area of each of the visibility assessment study areas. Thus, within a particular month and study area, PRB for $PM_{2.5}$ component concentrations for a given clock hour are constant, but they vary by clock hour within a day, by month within a year, and by study area. Table 2-5 summarizes them in terms of the annual average PRB for each component, by study area.

The health risk assessment did not make use of estimates of PRB concentrations of PM_{10} or $PM_{10-2.5}$, because the focus of that assessment was on evaluating risks associated with exposures to fine particles, $PM_{2.5}$. The urban focused visibility assessment did require estimates of PRB for $PM_{10-2.5}$. The second draft ISA did not present any new information on this subject. The approach used in the previous two reviews was to present the historical range of annual means of $PM_{10-2.5}$ concentrations from IMPROVE monitoring sites selected as being least influenced by anthropogenic emissions (US EPA, 2004, Table 3E-1). For sites in the lower 48 states, these annual means range from a low of 1.8 $\mu g/m^3$ to a high of 10.8 $\mu g/m^3$. For this assessment, we estimated PRB for $PM_{10-2.5}$ using a contour map based on average 2000-2004 $PM_{10-2.5}$ concentrations from all IMPROVE monitoring sites, found in a recent report from the IMPROVE program. (DeBell, L.J., et al., 2006). We located each study area's position on this map and assigned it the mid-point of the range of concentrations indicated by the contour band for that location.

The results are shown in Table 2-6. Lacking any other information, these PRB values have been assumed to apply to every hour of the year. While the contour map and, thus, these PRB values are influenced by data from IMPROVE sites that were not considered in the last review to be the sites most isolated from the influence of anthropogenic emissions, including three IMPROVE sites in urban areas, these values are generally within the range of values presented for such isolated sites in the previous science assessment (US EPA 2004, Appendix E, Table 3E-1).

Table 2-5. Summary of PRB estimates for the five $PM_{2.5}$ components: average 1-hour values across 2005-2007

Study Area	Average 1-Hour PRB Concentration Across 2005-2007 ($\mu g/m^3$)				
	Sulfate (dry, no ammonium)	Nitrate (dry, no ammonium)	Elemental Carbon	Organic Carbonaceous Material	Fine Soil/Crustal
Tacoma	0.45	0.026	0.15	1.3	0.31
Fresno	0.4	0.00062	0.08	0.74	0.19
Los Angeles-South	0.36	0.0037	0.028	0.3	0.036
Phoenix	0.31	0.000052	0.02	0.26	0.015
Salt Lake City	0.25	0.00028	0.025	0.26	0.034
Dallas	0.27	0.0022	0.055	0.59	0.092
Houston	0.3	0.0055	0.091	0.86	0.17
St. Louis	0.31	0.0027	0.047	0.53	0.07
Birmingham	0.29	0.007	0.099	1.1	0.19
Atlanta	0.3	0.016	0.1	1.1	0.19
Detroit-Ann Arbor	0.34	0.00062	0.024	0.32	0.018
Pittsburgh	0.3	0.00052	0.029	0.36	0.034
Baltimore	0.34	0.0016	0.039	0.44	0.054
Philadelphia-	0.34	0.00097	0.03	0.36	0.032
New York-N.New Jersey-Long Island	0.36	0.0038	0.026	0.31	0.022

1

Table 2-6. PRB Concentrations of $PM_{10-2.5}$ Used in the Urban Focused Visibility Assessment, Based on Measurements at IMPROVE Sites

Study Area	PRB $PM_{10-2.5}$ Mass ($\mu g/m^3$)
Tacoma	4.5
Fresno	5.5
Los Angeles-South Coast Air Basin	4.5
Phoenix	5.5
Salt Lake City	4.5
Dallas	8.5
Houston	5.5
St. Louis	7.5
Birmingham	5.5
Atlanta	5.5
Detroit-Ann Arbor	9.5
Pittsburgh	3.5
Baltimore	3.5
Philadelphia-Wilmington	6.5
New York-N.New Jersey-Long Island	3.5

2.8 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.[32] The focus here is on particle size distinctions; the second draft ISA also discusses exposure relationships related to compositional differences (US EPA, 2009a, section 3.8.6.6).

2.8.1 Definitions

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

Ambient PM is comprised of particles emitted by anthropogenic and natural sources 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 ISA conceptually separates (US EPA, 2009a, Section 3.8.1,) 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 (US EPA, 2009a, section 3.x). Concentrations of PM inside vehicles are subject to essentially the same factors as concentrations of PM inside

[32] Consideration of exposure measurement error and the effects of exposure misclassification on the interpretation of the epidemiologic studies are addressed in section 3.4.

buildings. Personal exposure to PM also includes a component which results specifically from the activities of an individual that typically generate particles affecting only the individual or a small localized area surrounding the person, such as walking on a carpet, referred to as the personal cloud.

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

2.8.2 Centrally Monitored PM Concentration as a Surrogate for Particle Exposure

Prior reviews focused on 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 (US EPA, 2005, section 2.7.2). Distinctions between PM size classes and components were found to be important considerations in addressing the representativeness of central monitors. In particular, fine particles were observed to have a longer residence time and generally to exhibit less spatial variability in the atmosphere than coarse fraction particles. In the PM NAAQS review completed in 1997, EPA concluded that measurements of daily variations of PM at nearby ambient monitoring stations have a plausible linkage to daily variations of human exposures to PM of ambient origin, and that this linkage was stronger for fine particles than for PM_{10} or the coarse fraction of PM_{10} (US EPA, 1996a, section x.x). Furthermore, evidence in the current PM NAAQS review is consistent with findings in previous reviews concluding that central monitoring can be a useful index for representing the average exposure of people in a community to PM of ambient origin in epidemiological studies assessing PM associated health effects (US EPA, 1996b, section x.x,; US EPA, 2009a , section 3.9.2.4).

As outlined in the last review, 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 (US EPA, 2005, section 2.7.2).

With regard to the first factor that influences the relationship between total personal exposure and concentrations measured at central sites, the spatial variability of PM plays a large role. As discussed in Section 2.5, for many areas $PM_{2.5}$ concentrations are fairly uniform spatially, with higher concentrations near roadways and other direct sources of $PM_{2.5}$. Analyses of $PM_{2.5}$ data for X urban areas indicate that differences in annual mean concentrations between monitoring sites in an urban area range from less than x $\mu g/m^3$ to as much as x $\mu g/m^3$. However, the correlations of daily $PM_{2.5}$ between sites are typically greater than 0.XX. 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 second draft ISA states, "In general, $PM_{2.5}$ has a longer atmospheric lifetime than $PM_{10-2.5}$ because larger particles have a higher gravitational settling velocity. For $PM_{2.5}$, most metropolitan areas exhibited high correlations (generally greater than 0.75) out to a distance of 100 km (US EPA, 2009a, Figures 3-25 through 3-27). Notable exceptions were Denver, Los Angeles and Riverside where correlations dropped below 0.75 somewhere between 20 and 50 km…Furthermore, correlations between PM_{10} concentrations exhibited substantially more scatter relative to $PM_{2.5}$….Although the general understanding of PM differential settling leads to an expectation of greater spatial heterogeneity in the $PM_{10-2.5}$ fraction relative to the $PM_{2.5}$ fraction in urban areas, deposition of particles as a function of size depends strongly on local meteorological conditions, in particular on the degree of turbulence in the mixing layer." Therefore, the findings reported for 15 CSAs/CBSAs[33] in the second draft ISA "may not apply to all locations or at all times. Population density and associated building density are also important determinants of the spatial distribution of PM concentrations. Inter-sampler correlations as a function of distance between monitors obtained for sampler pairs located less than 4 km apart (i.e., on a neighborhood scale) showed a shallower slope for $PM_{2.5}$ than for PM_{10}. The average correlation was 0.93 for $PM_{2.5}$, but it dropped to 0.70 for PM_{10}" (US EPA, 2009a, section 3.9.1.1).

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 (US EPA, 2009a, Section 3.2). 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 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}, $PM_{10\text{-}2.5}$, or ultrafine particles (US EPA, 2009a, section 3.8.4.3). Since $PM_{10\text{-}2.5}$ 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 $PM_{10\text{-}2.5}$ 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 (US EPA, 2004, section 5.3.3.2.2). 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 (US EPA, 2009a, Section 3.8.6), 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 second draft ISA 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 (US EPA, 2009a, sections 3.8.6.3 and 3.8.6.4). They will more likely affect the magnitude of the effects found from these studies and result in higher effect estimates, since exposure measurement errors tend to bias towards the null hypothesis. Exposure measurement errors also tend to decrease the precision of health effects estimates, resulting in wider confidence intervals.

The prior review discussed 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 (US EPA, 2004, sections 5.3.3.1.5 and 5.6). 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 (US EPA, 2004, section 5.5.2.1; US EPA, 2009a, section 3.x). 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 health effects associated with short-term exposures is examined.

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 (US EPA, 2004, section 5.3.3.1.3). 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 (p. 3-195, US EPA, 2009a, section 3.8.4.1). 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 (US EPA, 2009a, sections 2.x and 3.x). Based on these considerations and on the review of the available exposure-related studies, the ISA 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 (US EPA, 2009a, section 3.x).

2.9 RELATIONSHIP BETWEEN AMBIENT PM AND VISIBILITY

Visual air quality (VAQ) is defined as the visibility effects caused solely by air quality conditions and excluding those associated with meteorological conditions like fog and precipitation. Light extinction measures the ability of particles and gases in the atmosphere to scatter and absorb light traveling between an object and a person (or camera). Because it is the factional loss of light per unit of distance, it is measured in inverse length units (i.e. 1/length, commonly $1/10^6$ meter written Mm^{-1} and pronounced inverse megameters).

The extent to which any amount of light extinction affects a person's ability to view a scene depends on both scene and light characteristics. For example the appearance of a nearby object (i.e., a building) is generally less sensitive to a change in light extinction than the appearance of a similar object at a greater distance. For a scene with known characteristics, the amount of degradation in the scene associated with a change in light extinction can be determined, and the change in appearance can be realistically displayed on a digital photograph of the scene using the WinHaze system.

Survey studies have used sets of actual or computer modified (WinHaze) photographs depicting a range of visibility conditions on urban scenes to assess the public's opinion on what constitutes acceptable conditions. However, differences in scene sensitivities caused by changed scene and/or lighting characteristics (e.g. differing shadow patters or clouds cover) can affect people's perceptions of the haze level shown for a given photo. For the WinHaze modified scenes which maintain constant lighting and scene characteristics, there is a known/predetermined one-to-one correspondence between the perceived haze in the WinHaze-generated photographs and the amount of light extinction. Visibility effects are related to the sightpath-averaged light extinction between the observer (or camera) and each object viewed in the scene. Ambient measurements that are typically available at only one or a few locations near the surface are not always representative of the sightpath-averaged light extinction, especially for elevated sightpaths. This can be an important source of uncertainty for the results of preference survey studies that use actual photographs of a scene that are paired with ambient measurements of light extinction. The WinHaze program assumes a specific distribution of light extinction that is invariant from one haze image to the next so their use in preference survey studies eliminates that source of uncertainty in the results. For visibility preferrence studies, visibility levels are generally characterized using the haze index in units of deciviews (similar to the decibel scale for sound), which is a simple logarithmic transformation of light extinction.

Light extinction (b_{ext}) is the sum of the light scattering and absorption by particles and gases in the atmosphere as shown below (US EPA, 2009a, Equation 9-x):

$$b_{ext} = b_{ap} + b_{ag} + b_{sg} + b_{sp} \qquad \text{(Equation 2-1)}$$

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 scattering by gases (e.g., nitrogen, oxygen, etc.) that comprise the atmosphere (also known as Rayleigh or clear-air scattering) is related to the density of the air, which is sufficiently constant with elevation that it can be considered a known constant value for any location. NO_2 is the only atmospheric pollutant gas that absorbs visible light appreciably and its effects are generally small (i.e., less than 5%) compared to PM light extinction (defined as the sum of particulate light scattering and particulate light absorption), so its contributions to ambient visibility impacts are often ignored (as is done here). Thus light extinction is approximated as the sum of PM light extinction plus Rayleigh light scattering, where the former characterizes the

PM contribution to visibility impacts and the latter is taken to be a time-invariant constant depending only on elevation above sea level. In the same way PM light extinction is a good measure of VAQ.

2.9.1 Particle Mass and Light Extinction

If the relative composition of the PM as a function of particle size were held constant and the ambient relative humidity is low (RH<60%) or held constant, there would be a direct linear relationship between PM light extinction and PM mass concentration. Since the PM composition and ambient relative humidity vary in both time and space, there is in general no simple one-to-one correspondence between PM concentration and PM light extinction. These variations result in significant uncertainty (i.e., greater than a factor of two) in predicting PM light extinction from PM mass concentration. However the PM light extinction can be more accurately estimated from PM composition and relative humidity data, using a simple algorithm with assumed light extinction efficiencies for each of the major PM species and water growth factors for the hygroscopic species. In addition PM light extinction can be accurately determined by direct measurements using an integrating nephelometer to measure particle light scattering and an aethalometer or similar instrument to measure particle light absorption.

The 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 as discussed below. This algorithm is represented by the following equation (US EPA, 2009a, equation 9-1):

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

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. 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.

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 8 to 12 Mm^{-1} corresponding to elevations from about 4km above sea level to sea level for green light at about 550 nm. 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).

A refined version of the algorithm described by the relationship above (equation 2-2) was developed for remote area application to better fit the high and low light extinction extremes as needed for the regional haze rule (ISA, equation 9-2). Neither the original or revised version of the algorithm has been explicitly evaluated for urban conditions. However for remote areas, the results are comparable except at the extremes where the revised version produces larger estimates of PM light extinction by having larger dry extinction efficiency terms for some PM components when their concentrations are large. The original version (2-xx) was selected for use in the UFVA because of its simplicity and the judgment that it may be more appropriate for freshly produced PM as would be expected in urban settings. The light extinction coefficient integrates the effects of aerosols on visibility, yet it 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).

2.9.2 Other Measures of Visibility

Light extinction can be converted to two other parameters that have often been used as measures of visibility, visual range and the haziness index expressed in deciview units. 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 where its characterization as the maximum distance that an object can be seen is helpful. It is less useful for characterizing air quality impacts, because it can be misinterpreted as the distance over which good visibility conditions exist. Conversion from the extinction coefficient to visual range can be made with the following equation (NAPAP, 1991):

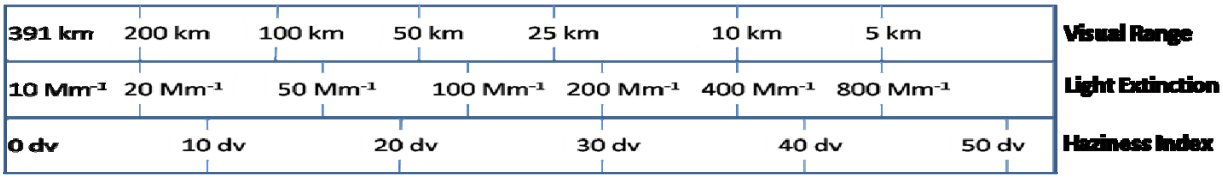
$$\text{Visual Range (km)} = 3912/b_{ext}(Mm^{-1}) \qquad \text{(Equation 2-3)}$$

The haziness index with deciview units (abbreviated as dv) was developed specifically to produce a visibility metric that has a near-linear response 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 10 Mm^{-1} change in light extinction can result in a change that is either imperceptible or very apparent depending on baseline visibility conditions. The haziness index allows one to more effectively express perceptible changes in visibility, regardless of baseline conditions. This is particularly useful in the design and assessment of results of perception, preference, and valuations of visibility effects studies. 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). The deciview value can be calculated from the light extinction (b_{ext}) by the equation:

$$\text{Haziness (dv)} = 10 \ln(b_{ext}/10\ Mm^{-1}) \qquad \text{(Equation 2-4)}$$

Figure 2-24 graphically illustrates the relationship among the three VAQ metrics described above.

Figure 2-24. Graphical representation of the relationships between the three commonly used metrics of VAQ.



2.9.3 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-x 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. 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 naturally occurring hygroscopic particles.

At the ranges of fine particle concentrations associated with background conditions, discussed above in section 2.6, small changes in fine particle mass have a large effect on total light extinction. Thus, higher levels of background fine particles and associated average humidity levels in the East result in a fairly significant difference between naturally occurring visual range in the rural East as compared to the rural West. Fine particles originate from both natural and anthropogenic, or man-made, sources. Natural background concentrations of fine particles are those originating from natural sources. On an annual average basis, concentrations of natural 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 (US EPA, 1996) 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 natural 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).

The draft Urban Focused Visibility Assessment (UFVA) for this review (US EPA, 2009c) has included the development of PRB estimates for light extinction during each daylight hour in 15 study areas for 2005-2007 (for days with available PM monitoring data), based on the PRB estimates of $PM_{2.5}$ components and PM_{10} described in section 2.7, along with data on hour-specific relative humidity.

Table 2-4Table 2-4 presents the unweighted 2005-2007 average of the hourly daylight PM light extinction values (in units of Mm^{-1}) for reach of the 15 visibility assessment areas. In the East, these values are somewhat lower than the value cited above for eastern rural areas (26 Mm^{-1}), but the latter estimates were for 24-hour average light extinction which includes hours with higher relative humidity. In the West, the value cited above (17 Mm^{-1}) falls within the range of these estimates for the western study areas.

Table 2-4. 2005-2007 Average PRB Daylight PM Light Extinction

Study Area	2005-2007 Average Policy Relevant Background Daylight Total Light Extinction, Mm^{-1}
Tacoma	22
Fresno	21
Los Angeles-South Coast Air Basin	18
Phoenix	18
Salt Lake City	15
Dallas	18
Houston	20
St. Louis	19
Birmingham	19
Atlanta	19
Detroit-Ann Arbor	17
Pittsburgh	17
Baltimore	19
Philadelphia-Wilmington	18
New York-N.New Jersey-Long Island	18

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 second draft ISA (US EPA, 2009a, section 9.x), 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 short- and long-term exposures to ambient particulate matter (PM). In the last two PM NAAQS reviews, EPA focused on particle mass and primarily distinguished between two categories of particle pollution based on size (i.e., fine- and thoracic coarse-fraction particles) and conducted parallel evaluations of the available scientific evidence relating to each category. The importance of specific PM components and sources was evaluated within the context of this basic size differentiation. In those reviews, it was determined that size-fractionated particle mass, rather than particle composition, remained the most appropriate approach for addressing ambient PM. Building upon lessons learned in the last two reviews, EPA's review of the available scientific evidence, as presented in the second draft Integrated Science Assessment (ISA, US EPA, 2009a), is based on particle size, considering fine and thoracic coarse-fraction particles separately. Within this basic structure, EPA is also evaluating relevant, but more limited, scientific evidence on additional size fractions (e.g., ultrafine particles (UFPs) and specific components, sources, and environments (e.g., urban and non-urban environments).

The presentation of the policy-relevant health effects evidence in this chapter first summarizes the qualitative assessment of health evidence contained in the second draft ISA, as a basis for the evidence-based assessment of the primary standards for PM to be presented in chapter 5 of the external review draft Policy Assessment (PA) . Secondly, this assessment addresses key issues relevant to the quantitative assessment of the epidemiologic health evidence available in this review so as to provide a foundation for the quantitative health risk assessment to be discussed in chapter 4 and used as the basis for the risk-based assessment of the primary standards for PM to be presented in chapter 5.

In the last review of the PM NAAQS, a broad range of health effects associated with ambient PM were evaluated with emphasis placed on those health endpoints for which the Air Quality Criteria Document (AQCD, US EPA, 2004) concluded that the associations were likely to be causal. The body of scientific evidence reviewed included consideration of hundreds of new studies conducted in many countries around the world. Of special importance in the last review were (1) a large number of new single- and multi-city time-series studies that evaluated the relationship between short-term PM exposures and mortality and morbidity, including new evidence of health endpoints related to cardiovascular morbidity; (2) extensive reanalyses of a number of important time-series studies to address issues associated with statistical model

specification (HEI, 2003); (3) reanalysis and validation of critical long-term exposure cohort studies, specifically, the Harvard Six Cities Study and the American Cancer Society (ACS) study, which provided evidence of generally robust associations between $PM_{2.5}$ and mortality and provided a basis for greater confidence in the reported associations than in the review completed in 1997 (Krewski et. al, 2000); and (4) additional toxicological and controlled human exposure evidence to support the plausibility of the epidemiologic evidence and provide insights into potential mechanisms for PM-related effects.

The EPA's conclusion in the last review that fine and thoracic coarse particles should continue to be considered as separate pollutants was based on long-established information on differences in physical and chemical properties, sources, and atmospheric formation and transport (US EPA, 2005, section 2.2). In this review, EPA has evaluated the newly available evidence related to the physics and chemistry of PM, exposure relationships, and particle dosimetry and concludes that the chemical and physical distinctions between fine and coarse particles recognized in the previous reviews remain generally unchanged; recent studies continue to show that fine and coarse particles generally have different sources and composition and different formation processes and deposition rates (See Table 2-1).

The assessment of health evidence in this chapter, therefore, focuses on health effects associated with fine and thoracic coarse particles, with more limited evidence presented for PM components and UFPs. Partly as a consequence of EPA's decision in the previous reviews 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, $PM_{10-2.5}$. Since the purpose of this review is to evaluate the adequacy of the current primary PM standards, which 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 understanding the public health impacts of ambient fine and thoracic coarse particles.

This assessment draws from the second draft ISA's evaluation and conclusions on the full body of evidence from health studies, summarized in chapters 6 through 8 of the ISA, with consideration of the integrative synthesis presented in chapter 2. Chapter 2 of the second draft ISA focuses on synthesizing newly available scientific information with evidence available from the previous PM NAAQS reviews, integrated from various disciplines, so as to inform consideration of the policy-relevant questions that are central to EPA's assessment of scientific information upon which this review of the PM NAAQS is to be based (US EPA, 2008a, section 3.1 and Chapter 4). It is intended to provide an integrative overview of human health effects elicited by ambient PM in the U.S., and to facilitate consideration of the key policy-related issues

that will inform staff conclusions related to indicators, averaging times, levels, and forms in chapter 5.

As presented and discussed in chapters 6 through 8 of the second draft ISA, a large number of new studies containing further evidence of health effects associated with PM exposures have been published since the last review, with important new information coming from epidemiologic, toxicological, controlled human exposure, and dosimetric studies, including information on effects in at-risk populations. As was true in the previous two reviews, evidence from epidemiologic studies plays a key role in the second draft ISA's evaluation of the scientific evidence. As discussed further in section 3.3, some highlights of the new evidence include:

- New multi-city time-series studies that use uniform methodologies to investigate the effects of $PM_{2.5}$ on health with data from multiple locations representing varying regions and seasons representative of different climate and air pollution mixes. These studies provide more precise estimates of the magnitude of an effect of exposure to $PM_{2.5}$ than most smaller-scale individual city studies that were more commonly available in the last review. The new studies also contribute to improving our understanding of the role of various potential confounders, including gaseous co-pollutants, on observed associations.

- Recent studies investigating cardiovascular morbidity and mortality provide some of the strongest evidence for cardiovascular effects related to $PM_{2.5}$ exposures. A number of large multi-city studies have been conducted throughout the U.S. and have reported consistent increases in cardiovascular morbidity and mortality related to ambient $PM_{2.5}$ concentrations. These effects are biologically plausible and coherent with available toxicological studies.

- Extended analyses of key long-term exposure cohort studies that were critical in evaluating $PM_{2.5}$-related mortality in the last review (e.g., ACS and Harvard Six Cities Studies) continue to provide evidence that the data are robust and that reducing $PM_{2.5}$ improves public health. Results of new studies employing within-city exposure gradients suggest that reduced exposure misclassification may result in greater magnitudes of association.

- New evidence has become available documenting the population health benefits of reducing ambient air pollution by correlating past reductions in ambient $PM_{2.5}$ concentrations with increased life expectancy and by providing evidence for improvement in community health following reduction in fine particle exposures. 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.

- Recent studies of long-term exposure to $PM_{2.5}$ have expanded the evidence for effects on lung development in children. The extended follow-up for the Southern California Children's Health Study includes evidence that decreased lung function effects associated with long-term exposure to $PM_{2.5}$ persist into early adulthood, and are more robust and larger in magnitude than reported in the prior review. Supporting these results are new longitudinal cohort studies conducted by other researchers in other locations using different methods provide enhanced evidence for respiratory effects related to long-term fine particle exposures.

- New evidence for respiratory symptoms and aggravation of preexisting respiratory disease strengthens evidence from the last review and provides increased consistency as results have reported by several different researchers in different countries using different designs. The most recent studies have focused on effects in children, but a few studies have also reported associations in adults.

- Additional evidence supporting no discernible thresholds below which PM-related health effects are absent.

- New analyses informing our understanding of issues related to potential confounding by gaseous co-pollutants, exposure misclassification (e.g.., measurement error, lag period), model specification, seasonal and regional influences, and the shape of the concentration-response function.

- Emerging, though still limited evidence on effects associated with exposure to $PM_{10-2.5}$, PM components, and UFPs.

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

- In general, potential pathways or modes of action do not appear to be specific to a particular PM size fractions, however emerging evidence related to the characteristics of UFPs may inform unique modes of action or effects disproportionate to their mass. In addition, for UFPs, particle number rather than mass may be the appropriate metric to evaluate, however, UFP number concentrations are highly dependent on monitor location (i.e., concentrations drop off quickly from the road compared to accumulation mode particles, and, therefore, more subject to exposure error than accumulation mode particles).

- Animal and controlled human exposure studies using concentrated ambient particles (CAPs), various indicators of response (e.g., heart rate variability), and animal models simulating at risk populations, continue to demonstrate plausibility of the epidemiologic evidence and provide insights into potential mechanisms for PM-related effects.

- The findings of new toxicological and controlled human exposure studies continue to provide support that a number of potential biologic mechanisms or pathways for PM-related cardiovascular and respiratory effects exist.

- New evidence has developed to inform our understanding of the role of certain factors that may affect our understanding of at-risk populations including: gender, age, and pre-existing lung disease on deposition and clearance.

In presenting the evidence and conclusions based on it, this chapter first summarizes information from the second draft ISA's evaluation of health evidence integrated across different disciplines (e.g., epidemiology, toxicology, controlled human exposure). Sections 3.2 and 3.3 provide overviews of the EPA's integrative findings and conclusions regarding dosimetry and modes of action (MOA), causality findings, and on the nature of effects associated with PM exposures. Section 3.4 addresses several key issues relevant to the staff's interpretation and quantitative assessment of the health evidence, including: (1) 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 exposure studies; (2) questions related to the shape of concentration-response relationships and potential threshold levels; (3) considerations related to air quality measurements and data used in the health studies; (4) measurement error and exposure error in fine and thoracic coarse particle studies; (5) specification of models used in epidemiologic studies; and (6) approaches to evaluating the role of co-pollutants and potential confounding in PM-effects associations. In this section, staff builds upon the second draft ISA's and 2004 AQCD'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 to be discussed in chapter 4. Section 3.5 draws from chapters 2 and 8 of the second draft ISA to characterize potential at-risk populations and potential public health impacts of exposure to ambient PM.

3.2 MECHANISMS

This section provides an overview of evidence related to potential mechanisms by which exposure to PM may result in adverse health effects, drawing upon evidence presented in Chapters 4 through 7 of the second draft ISA (US EPA, 2009a). 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 the last review, EPA recognized that much new evidence was available on potential mechanisms or pathways for PM-related effects, ranging from effects on the respiratory system to indicators of cardiovascular response. The new findings advanced our understanding of the complex and different patterns of particle deposition and clearance in the respiratory tract and provided insights into potential modes of action (MOA) for PM-related

effects as well as supported the plausibility of the epidemiologic evidence (US EPA, 2004, Chapter 7; US EPA, 2005, section 3.2).

In this review, although the basic understanding of the mechanisms governing deposition and clearance of inhaled particles has not generally changed, there is additional information available to provide insights on the role of certain biological determinants such as gender, age, and lung disease on deposition and clearance. In addition, new evidence provides further characterization of the retention and translocation of UFPs following deposition in the respiratory tract (US EPA, 2009a, chapter 4) as well as providing additional insights into possible MOA for pulmonary and extra-pulmonary effects related to PM exposures, in particular, cardiovascular effects (US EPA, 2009a, chapter 5). Policy-relevant information is presented below considering first dosimetric studies and second studies evaluating modes of action (MOA).

3.2.1 Dosimetry

An evaluation of the ways by which inhaled particles might ultimately affect human health must take into account particle dosimetry which refers to patterns of deposition, translocation, clearance, and retention of particles and their constituents within the respiratory tract and extrapulmonary tissues (US EPA, 2009a, Chapter 4). Briefly, the human respiratory tract can be divided into three main regions: (1) extrathoracic (upper airways), (2) tracheobronchial, and (3) alveolar[34] (US EPA, 2009a, Figure 4-1). The regions differ markedly in structure, function, size, mechanisms of deposition and removal, and sensitivity or reactivity to deposited particles. Overall, the site of particle deposition within the respiratory tract has implications related to lung retention and surface dose of particles as well as potential systemic distribution of particles and/or PM components (US EPA, 2009a, section 4.1.2). .

Inhaled particles may be either exhaled or deposited in the extrathoracic, tracheobronchial, or alveolar region. The dose from inhaled particles deposited and retained in the respiratory tract is governed by a number of factors including: (1) exposure concentration and duration; (2) activity and ventilatory parameters; and (3) particle properties (e.g., particle size, hygroscopicity[35], and solubility in airway fluids and cellular components) (US EPA, 2009a, section 4.1). Particle number is most highly concentrated in the ultrafine size range and volume (or mass) is most concentrated in the larger size ranges (US EPA, 2009a, section 3.2). The basic characteristics of particles as they relate to deposition and retention, as well as anatomical and physiological factors influencing particle deposition and retention have been discussed in detail in previous science assessments (US EPA, 1996, Chapter 10; US EPA, 2004, Chapter 6). In this review, EPA has focused on (1) dosimetry evidence that informs our understanding of issues that

[34] The term "lower airways" is used to refer to the intrathoracic airways, that is, the combination of the tracheobronchial region which is the conducting airways and the aleveolar region which is the functional part of the lung (US EPA, 2009a, section 4.1.2)

[35]Hygroscopicity relates to the ability of particles to absorb moisture from the atmosphere.

may affect the susceptibility of an individual to adverse effects and (2) evidence providing new insights into possible modes of action including evidence informing our ability to extrapolate findings between studies and between species.

Fine particles, including and ultrafine particles, and thoracic coarse particles can all penetrate into and be deposited in the tracheobronchial and alveolar regions of the respiratory tract (i.e., the "thoracic" regions). Differences in the distribution of these size fractions has been observed. Since the nasal passages (extrathoracic region) are more efficient at removing inhaled particles than the oral passage (mouth), an individual's mode of breathing (i.e., oral vs. nasal) influences the quantity of particles penetrating to the lung. Once past the extrathoracic region, particle deposition in the tracheobronchial and alveolar regions varies with different exertion levels or breathing patterns. Furthermore, breathing patterns may change with increasing age. As shown in Figures 4-3 and 4-4 of the second draft ISA (reproductions of Figures 6-16 and 6-17, US EPA, 2004), mathematical models estimate that the deposition fractions in these regions are largest for particles in the thoracic coarse fraction and ultrafine modes. Note that the 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 3 to 6 μm (US EPA, 2009a, section 4.2). Both experimental and modeling techniques are based on many assumptions that may be relatively good for the healthy lung but not for the diseased lung (US EPA, sections 4.2.4.4 and 4.2.4.5).

Interspecies similarities and differences in deposition were described in detail in the previous two PM reviews (US EPA, 1996, section X.X; US EPA, 2004, section x.x). More recently, Brown et al. (2005) conducted a thorough evaluation of extrapolations between rats and humans in relation to PM exposures. One of the many factors they considered was the choice of a dose metric appropriate for comparison between species. For example, deposited mass may be an appropriate PM indicator for health effects associated with soluble PM constituents. For health effects associated with insoluble PM, the particle number, surface area, or mass may be appropriate indicators. Given interspecies differences in deposition patterns and clearance rates, the question of retained versus deposited dose, Brown et al. (2005) concluded that for acute effects, the maximum deposited incremental dose may be the appropriate type of dose metric. For chronic effects, long-term burden may be more appropriate (US EPA, 2009a, section 4.2.3).

Evaluation of factors affecting particle deposition is important to help understand at-risk populations. Differences in biological response following PM exposures may be caused by dosimetry differences as well as by differences in innate sensitivity. The effects of different biological factors on particle deposition are summarized in section 4.2.4 of the second draft ISA building upon a more complete discussion of these factors prepared for the previous PM NAAQS review (US EPA, 2004, section x.x). These factors include:

- **Physical activity** – the activity level of an individual is well recognized to affect their minute ventilation and route of breathing. Humans are oronasal breathers tending to breathe through the nose when at rest and increasingly through the mouth with increasing activity level. There is considerable inter-individual variability in both the route by which people breathe and the way the breathing pattern changes occur with changes in activity levels. Changes in breathing patterns and flow rates may alter the regional deposition of particles (US EPA, 2009a, section 4.2.4.1).

- **Age** – airway structure and respiratory conditions vary with age, and these variations may alter the amount and site of particle deposition in the respiratory tract. Limited evidence suggests that children may tend to have more oral breathing both at rest and during exercise and that children may also display more variability than adults with respect to their oronasal pattern of breathing with exercise (Becquemin et al 1999; Bennett et al., 2008; James et al., 1997). In addition, models suggest that nasal deposition of particles is less efficient in children. These findings suggest that children's lower respiratory tract (tracheobronchial and alveolar regions) may receive a higher dose of ambient PM compared to adults and that for a given height and age, children with higher body mass index (BMI) may have increased deposition of fine particles (Bennett and Zeman, 2004). Bennett et al. (2008) also recently reported measures of fine particle (1 and 2 μm) deposition associated with light exercise in children and adults and showed that, like with resting breathing, deposition fraction was predicted by breathing pattern and did not differ by age or tended to be less in children compared to adults. On the other hand, children generally have a faster breathing rate than adults relative to their lung volumes, therefore, the rate of deposition of fine particles normalized to lung surface area may be greater in children than in adults (Bennett and Zeman, 1998). While older adults have been recognized as an at-risk population for PM-related effects, limited information is available to assess particle deposition in this population (US EPA, 2009a, section 4.2.2.2 and section 4.2.4.2).

- **Gender** – Males and females differ in body size, size of their upper airways, and ventilator parameters. Studies indicate variability in deposition efficiencies due to inter-individual difference in lung size and anatomical variability in airway dimensions and branching patterns. Deposition of thoracic coarse particles and UFPs was greater in females than in males (Kim and Hu, 1998; Kim and Jaques, 2000; as summarized in US EPA, 2009a, Figure 4-5 and section 4.2.4.3).

- **Anatomical Variability** – Nasal anatomy may influence the efficiency of particle uptake and deposition. Individual differences may occur in both the nasal airways as well as within the branching structures of the lung (US EPA, 2009a, section 4.2.4.4).

- **Respiratory Disease** – Respiratory disease status can also affect regional particle deposition patterns. The presence of respiratory tract disease can affect airway structure and ventilator parameters, thus altering particle deposition in individuals

with pre-existing respiratory disease compared to healthy individuals. Both experimental and modeling techniques are based on many assumptions that may be relatively good for the healthy lung but not representative of the diseased lung (US EPA, 2004, sections 4.2.4.4 and 4.2.4.5). Studies have indicated that, in general, enhanced deposition of particles occurs at airway bifurcations (US EPA, 2004, section 6.2.2.4). Evidence available in the last review indicated 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 (US EPA, 2004, section 6.2.3.4). Individuals with chronic obstructive pulmonary disease (COPD) have very heterogenous deposition patterns and differences in regional deposition compared to healthy individuals and generally show greater particle deposition in the tracheobronchial region than do healthy individuals. Furthermore, there is, in general, an inverse relationship between bronchoconstriction and the extent of deposition in the alveolar region, whereas total respiratory tract deposition generally increases with increasing degrees of airway obstruction (US EPA, 2009a, section 4.2.4.5; US EPA, 1996, section x.x; US EPA, 2004, section x.x). In such cases, the respiratory condition can enhance sensitivity to inhaled particles by increasing the delivered dose overall as well as increasing doses to localized regions of the respiratory tract. Such dosimetry studies are of obvious relevance to identifying at-risk populations (see section 3.6 below).

- **Hygroscopicity of Aerosols** - Hygroscopicity relates to the ability of particles to absorb moisture from the atmosphere. The high relative humidity in the lungs contributes to rapid growth of hygroscopic particles and may dramatically alter the deposition characteristics of hygroscopic aerosols relative to nonhygroscopic aerosols. Examples of hygroscopic ambient aerosols include sulfates, nitrates, some organics, and aerosols containing significant amounts of sodium or potassium (US EPA, 2009a, section 4.2.4.6).

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 tract and also vary with solubility of the particles.[36] For example, particles deposited in the extrathoracic region are cleared by several mechanisms. Particles deposited in the mouth are generally assumed to be swallowed or removed by expectoration. Particles deposited in the posterior portions of the nasal passages are moved via mucociliary transport towards the nasopharynx and swallowed. Mucus flow in the most anterior portion of the nasal passages is forward, toward the opening of the nose where removal occurs by sneezing, wiping,

[36] The term "clearance" is used here to refer to the processes by which deposited particles are removed by mucociliary action or phagocytosis from the respiratory tract. "Translocation" is used mainly to refer to the movement of free particles across cell membranes and to extrapulmonary sites. In the literature, translocation may also refer to the extra- and intracellular dissolution of particles and the subsequent transfer of dissociated material to the blood through extra- and intracellular fluids and across the various cell membranes and lung tissues. Translocation can occur across cell membranes within the lung as well as to extrapulmonary sites. For example, particles deposited on the epithelial surface of the lung may translocate to the interstitium.

or nose blowing (US EPA, 2009a, section 4.3.1.1). The primary mechanism for particle clearance or translocation from the tracheobronchial region is mucociliary transport and coughing (US EPA, 2009a, section 4.3.1.2; US EPA, 2004, Table 6-2). Dissolution or absorption of particles or particle constituents and endocytosis[37] by cells such as macrophages[38] where the cells are cleared by the mucociliary escalator is the primary clearance mechanism operating in the alveolar region. Under normal conditions, a small fraction of ingested particles may also be cleared through the lymphatic system (US EPA, 2009a, section 4.3.1.3).

Many factors may influence particle clearance. The movement of soluble materials depends on the site of deposition in the lung, the rate of material dissolution from particles, and the molecular weight of the solute. The rate of soluble material clearance from the lungs depends on epithelial permeability which may be affected by age, physical activity, respiratory disease, and concurrent exposures (US EPA, 2009a, section 4.4.2). For example, limited evidence shows that particle clearance may be decreased throughout the respiratory tract with increasing age (US EPA, 2009a, section 4.3.4.1). Respiratory disease may influence both the site of initial particle deposition and rate of mucociliary clearance from the airways (U.S. EPA, 2009a, section 4.3.4.3). There is no evidence to support that gender affects clearance rates (US EPA, 2009a, section 4.3.4.2).

Particles depositing in the mouth are generally assumed to be swallowed or removed by expectoration. Particles deposited in the posterior portions of the nasal passages or the tracheobronchial regions are moved via mucociliary transport towards the nasopharynx and swallowed. Clearance from the extrathoracic and tracheobronchial regions generally occurs over hours to days, however, clearance from the alveolar region is much slower, occurring over months to years (US EPA, 2009a, section 5.1.9.1). Although clearance from the tracheobronchial region is generally rapid, there appears to be a fraction of material deposited in this region in humans and dogs that is retained much longer. The underlying sites and mechanisms of long-term tracheobronchial retention are not known though they may be accounted for by differences in deposition patterns (US EPA, 2009a, section 4.3.1.2). The primary alveolar clearance mechanism is macrophage phagocytosis and migration to terminal bronchioles where the cells are cleared by the mucociliary escalator (US EPA, 2009a, section 4.3.1.3).

There are differences between species in both the rates of particle clearance from the lung and the manner in which particles are retained in the lung. In contrast to humans, mice and rats appear to have negligible long-term retention of particles, including UFPs, in the

[37] Endocytosis is the process of cellular ingestion by which the plasma membrane folds inward to bring a substance(s) into the cell.

[38] A macrophage is a type of white blood cell. Macrophages develop from circulating monocytes that migrate from the blood into tissues throughout the body, including the lungs.

tracheobronchial airways (US EPA, 2009a, sections 4.3.1.2, 4.3.2). In addition, clearance from both the tracheobronchial and alveolar region is more rapid in rodents than humans.

Soluble particles and soluble components of particles may be retained in the lung or absorbed through the epithelium and distributed throughout the body. The rate of dissolution depends on a number of factors including particle surface area and chemical structure. In the ciliated airways, soluble particles and soluble components of particles are cleared by mucociliary transport and diffuse into underlying tissues and the blood. In the alveolar regions, the thin barrier between the air and blood allows for rapid transport of these soluble particles/components into the blood. While enhanced clearance of insoluble particles acts to reduce dose to airway tissue, increased transport of soluble matter into the bloodstream and, thus, throughout the body potentially enhance effects on extra-pulmonary organs (US EPA, 2009a, section 4.4).

The importance of particle translocation to the brain is not yet understood. Studies, primarily in rodents, have demonstrated the translocation of soluble and poorly soluble particles from the olfactory mucosa via the axons to the olfactory bulb in the brain. The rate of translocation appears to be rapid, perhaps less than an hour. It is unclear to what extent translocation to the olfactory bulb and other brain regions may vary between species. Interspecies differences may predispose rats, more so than humans, to the deposition of particles in the olfactory region with subsequent translocation to the olfactory bulb (US EPA, 2009a, section 4.3.3.2 and 4.3.5)

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, ultrafine particles, and thoracic coarse particles. The evidence continues to show that all three size fractions ($PM_{2.5,}$ $PM_{10\text{-}2.5,}$ and UFPs) can enter the tracheobronchial or alveolar regions of the respiratory system and potentially cause effects. Additional information has become available in this review to provide insights on the role of certain biological determinants such as gender, age, and lung disease on particle deposition and clearance. As summarized in section 4.4.3 of the second draft ISA:

> The healthy airway and alveolar epithelium is generally impermeable to very large insoluble macromolecules and particles. Water and acid soluble particles may more rapidly move through the epithelium as they dissolve on the airway surface or within the phagolysomes of macrophages. The presence of airway inflammation in a variety of airway diseases (e.g., asthma, fibrosis, ARDS, pulmonary edema, inflammation from smoking) alters epithelial integrity to allow more rapid movement of these solutes into the bloodstream. While diabetics are another group recently shown to have increased susceptibility to particulate air pollution (Zanobetti and Schwartz, 2002), it is unclear whether transport of soluble particles across the epithelium is affected in these patients. In general, it appears that co-exposure to irritant pollutants results in a disruption of epithelial integrity and macrophage function which, on the one hand, retards mucociliary and alveolar clearance, but also allows for a more rapid movement of soluble constituents

across the epithelial surface into the interstitium and blood stream. Alterations in epithelial permeability by disease, pollutant exposure, or infection may partially explain increased susceptibility to PM associated with these co-conditions.

3.2.2 Possible Pathways/Modes of Action

A major research need identified in the PM review completed in 1997 was improvement of our understanding of the potential biologic MOA by which deposited particles could result in the varying effects observed in epidemiologic studies with PM exposure (ADD REF). In the last review, new evidence from toxicological and controlled human exposure studies was available that provided insights to advance our understanding of potential MOA for PM-related effects and to support the plausibility of findings observed in epidemiologic studies. New evidence in the last review from toxicological and controlled human exposure studies helped to identify and provide support for a number of potential pathways by which particles could elicit biological effects. Fully defining the MOA for PM would involve description of the pathogenesis or origin and development tof any related diseases or processes resulting in premature mortality. While the evidence summarized in the last review provided important insights that contributed to the plausibility of effects observed in epidemiological studies, the more ambitious goal of fully understanding fundamental MOA for how particles produce specific health effects had yet to be attained. However, some of the more important findings presented in last review including those related to the cardiovascular system, were generally described as intermediate responses potentially caused by PM exposure rather than complete mechanisms. Based upon the evidence available in the last review, EPA concluded that it appeared unlikely that the complex mixes of particles that are present in ambient air would act alone through any single pathway of response. Accordingly, EPA concluded that it was plausible that several physiological responses might occur in concert to produce the reported health endpoints (US EPA, 2004, Chapter 7; US EPA, 2005, section 3.2).

In assessing the more recent animal, controlled human exposure, and epidemiologic information, the second draft ISA developed a summary of current thinking on pathophysiological MOA for the effects related to PM exposure. Chapter 5 of the second draft ISA discusses a series of potential MOA or potential general pathways for pulmonary and extrapulmonary effects. Prior to this review, much of the evidence for the proposed modes of action was obtained from animal studies involving intratracheal (IT) instillation or inhalation of high concentrations of PM and from cell culture experiments. In many cases, the types of PM used were of questionable relevance to ambient exposures (i.e., high concentrations of ROFA, metals and ambient PM collected on filters). Since then, many inhalation studies have been conducted using a variety of particles including concentrated ambient particles (CAPs) from various urban environments, combustion-derived PM (including gasoline and diesel exhaust studies), carbon black, and metals, generally using concentrations of PM lower than 1 mg/m^3. Much of this research has been conducted in animal models of disease, however, the levels of

$PM_{2.5}$ evaluated in these studies are not generally representative of current ambient levels of $PM_{2.5}$. These key new studies, described in detail in Chapters 6 and 7 of the second draft ISA, add to the understanding of modes of action which are relevant to ambient PM exposure. A compilation of the pertinent results of recent inhalation studies is presented in section 5.6 of the second draft ISA.

The second draft ISA's conclusions on the evidence supporting different types of effects is briefly summarized below. The characterization of evidence is presented for PM in general, since most of the potential pathways or modes of action do not appear to be specific to a particular size class of PM. However, characteristics of UFPs may allow for unique modes of action or effects disproportionate to their mass and, where appropriate, evidence specific to UFPs is briefly highlighted below.

Respiratory Effects. Modes of action underlying pulmonary effects associated with PM exposures have been well-studied in the laboratory and, in general, there is agreement regarding the key roles played by cellular injury and inflammation. These pathways are initiated following deposition of inhaled particles on respiratory tract surfaces (US EPA, 2009a, section 5.1). Evidence that supports hypotheses on direct pulmonary effects includes toxicological and controlled human exposure studies using both sources of ambient particles and combustion-related particles, including from gasoline and diesel exhaust. A great deal of research has focused on the role of reactive oxygen species (ROS) in the initiation of pulmonary injury and inflammation following PM exposures. Particles may also interact with cells leading to the induction of nitric oxide synthase and the production of nitric oxide and other reactive nitrogen species (RNS). Particles may act as a direct or indirect source of ROS/RNS in the respiratory system. Although all size fractions of PM may contribute to oxidative and nitrosative stress,[39] UFPs may contribute disproportionately to their mass due to their large surface/volume ratio and greater particle number compared to larger size fractions (US EPA, 2009a, section 5.1.1).

Particle exposure may alter pulmonary function by a variety of different MOA including activation of irritant receptors, production of ROS/RNS and pulmonary inflammation and injury (US EPA, 2009a, Figure 5-4). These potential modes of actions can lead to the development of a range of respiratory effects including:

- **Airway hyperresponsiveness (AHR)** – generally considered a short-term response (US EPA, 2009a, section 5.1.6).

[39] .In general, high levels of intracellular ROS/RNS can lead to irreversible protein modifications, loss of cellular membrane integrity, dNA damage and cellular toxicity. Lower levels of ROS/RNS may cause reversible protein modifications that trigger intracellular signaling pathways and/or adaptive responses. Thus, PM-dependent generation of ROS may be responsible for a continuum of responses from cell signaling to cellular injury (US EPA, 2009a, section 5.1.1).

- **Allergic immune responses and impaired host defense and infections** - affecting both innate immunity through effects on macrophage pathogen handling as well as adaptive immunity by altering cellular responses potentially resulting in impaired lung defense mechanisms leading to frequent or persistent infections (US EPA, 2009a, sections 5.1.7, 5.1.8). Studies conducted since the last review confirm and extend previous findings that PM can modulate immune reactivity in both humans and animals to promote allergic sensitization and exacerbate allergic responses (US EPA, 2009a, sections 6.3.6). A number of long-term exposure studies have reported associations between PM and allergic responses (US EPA, 2009a, section 7.3.6). Allergy is a major driver of asthma, which has been associated with PM in a number of studies (US EPA, 2009a, section 7.3). In the case of allergic asthma, acute PM exposure may provoke asthmatic responses through oxidative stress and inflammatory pathways (US EPA, 2009a, section 5.5). Toxicological studies demonstrate that short-term inhalation exposures to CAPs and disesel exhaust, but not gasoline exhaust or wood smoke, can increase susceptibility to bacterial and viral infections. Furthermore, impaired host defense against the etiological agents of influenza, pneumonia (*S. pneumoniae*), and bronchiolitis (RSV), which are commonly reported respiratory morbidities associated with PM exposures, have been documented in toxicological studies (US EPA, 2009a, section 6.3.7). While most of the evidence of respiratory infections associated with PM exposures is available from short-term exposure studies, limited evidence from long-term exposure studies suggest an associated with general respiratory symptoms often caused by infection, such as bronchitis (US EPA, 2009a, section 7.3.7).

- **Progression of pre-existing respiratory disease** – Many factors likely contribute to the resolution of pulmonary inflammation and injury and the progression or exacerbation of respiratory disease. These factors "are likely to include the updtake and clearance of PM by macrophages, the retention of PM in parenchymal cells and tissues; the balance of pro/anti-inflammatory soluble mediators, oxidants/anti-oxidants and proteases/antiproteases, and the presence of pre-existing disease" (US EPA, 2009a, section 5.1.9). These factors may impact the resolution of pulmonary effects associated with PM exposure including effects associated with inflammatory lung diseases including asthma and COPD (US EPA, 2009a, section 5.1.9). Particle exposures are likely to contribute to an unfavorable balance through its oxidative potential and capacity to promote cellular production of ROS. Exacerbations of asthma and COPD resulting from bacterial and viral infections are also associated with oxidative stress (Barnes et al, 2007) (US EPA, 2009a, section 5.1.9.2).

- **DNA damage and lung cancer** – Pulmonary DNA damage can occur primarily or secondarily to PM exposure (US EPA, 2009a, section 5.1.10). New, but limited evidence, has become available indicating a role for PM in promoting epigenetic[40]

[40] Epigenetic mechanisms regulate the transcription of genes without altering the nucleotide sequence of DNA. These mechanisms generally involve DNA methylation and histone modifications, leading to alternations which may have long-term consequences or are heritable. Early life exposures to environmental pollutants such as PM may be especially important since periods of rapid cell division and epigenetic remodeling are likely to occur at this

changes (US EPA, 2009a, section 5.1.11; Baccarelli et al., 2009; Liu et al., 2008; Read et al., 2008; Yauk et al., 2008).

Changes in the function of the respiratory tract barrier may modify response related to PM exposures. For example, epithelial injury can lead to an increase in permeability of the airway epithelial and alveolar-capillary barriers. Enhanced transport of the soluble and possibly insoluble PM components into the circulation may occur under these conditions (US EPA, 2004a, section 5.1.4).

Resolution of pulmonary inflammation and injury has been demonstrated in experimental models using higher than ambient concentrations of PM. Factors contributing to this complex process are likely to include the uptake and clearance of PM by macrophages; the retention of PM in cells and tissues; the balance of pro/anti-inflammatory soluble mediators, oxidants/antioxidants and proteases/anti-proteases; and the presence of pre-existing disease. These factors may also influence the resolution of pulmonary responses to ambient PM exposures. The long-term consequences of prolonged inflammation may be harmful and may lead to remodeling of the respiratory tract and to the progression or exacerbation of disease (US EPA, 2009a, section 5.1.9). In addition, lung development is a multi-step process which begins in embryogenesis and continues to adult life (Pinkerton and Joad, 2006). This allows a long period of potential vulnerability to environmental and other stressors. Disruption of cell signaling during development could affect cellular differentiation, branching morphogenesis, and overall lung growth, possibly leading to life-long consequences. Although very little is know about the effects of maternal exposure to PM on the developing fetus or the effects of exposure during childhood, recent animal studies demonstrate respiratory and immune system effects of perinatal exposure to sidestream cigarette smoke (Pinkerton and Joad, 2006; Wang and Pinkerton, 2007).

Cardiovascular and Other Systemic Effects. In addition to pulmonary effects, extrapulmonary effects resulting from lung damage may occur. For example, pulmonary inflammation may trigger systemic inflammation through the action of cytokines and other soluble mediators which leave the lung and enter the circulation (see US EPA, 2009a, section 5.2). In the last review, limited evidence from toxicological studies suggested that injury or inflammation in the respiratory system could lead to changes in heart rhythm, reduced oxygenation of the blood, changes in blood cell counts, or changes in the blood that could increase the risk of blood clot formation, a risk factor for heart attacks or strokes (US EPA, 2004, section 7.10.1.2). More specifically, in the last review, emerging evidence was available that provided some initial evidence that particles could have direct cardiovascular effects. Two types of hypothetical pathways by which particle deposition in the respiratory system could lead to cardiovascular effects were identified: (1) PM-induced pulmonary reflexes resulting in changes

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time (Foley et al., 2009; Keverne and Curley, 2008; Wright and Baccarelli, 2007; see also US EPA, 2009a, section 5.1.11).

in the autonomic nervous system that could affect heart rhythm and (2) effects on the heart or other organs if particles or particle constituents are released into the circulatory system from the lungs (US EPA, 2004, section 7.2, Figure 7-1). Some new evidence indicated that UFPs or their soluble constituents could move directly from the lungs into the systemic circulation (US EPA, 2004, section 6.3.2.3). The EPA concluded that the data evaluated in the last review remained limited but provided some new insights into MOA by which particles, primarily fine particles, could affect the cardiovascular system (US EPA, 2004, section 7.2.4, section 7.10.1.3).

In this review, MOA underlying extrapulmonary effects are still incompletely understood. Pulmonary inflammation can lead to systemic inflammation and pulmonary reflexes can active the autonomic nervous system (ANS). These latter responses may mediate cardiovascular and other systemic effects. In addition, it has been proposed the PM or soluble components of PM may enter the circulation by translocating across the epithelial and endothelial barriers of the respiratory tract, thus allowing PM or its components to interact directly with cells in the vasculature and blood and be transported to the heart and other organs. New evidence available in the current review has not advanced the hypothesis discussed in the previous review, that UFPs or other PM size fractions may access the circluaton by transversing the epithelial barrier of the respiratory tract (US EPA, 2009a, sections 5.2 and 5.3).

Pulmonary inflammation resulting from PM exposure may trigger systemic inflammation. Systemic inflammation is generally seen under conditions of mild pulmonary inflammation – and sometimes under conditions of no measurable pulmonary inflammation. The time-dependent nature of pulmonary and systemic inflammatory responses may, in part, explain these findings since biomarkers of inflammation are frequently measured at only one point in time. In addition, chronic exposures may lead to adaptive responses. In general, systemic inflammation is associated with changes in circulating white blood cells, the acute phase response, pro-coagulation effects, endothelial dysfunction, and the development of atherosclerosis[41] (US EPA, 2009a, Figure 5-5). The development of atherosclerosis involves inflammation and remodeling of the blood vessel wall. Factors contributing to this process include systemic inflammation, endothelial dysfunction, oxidative stress, and high levels of circulating lipids. PM exposure is associated with the first three of these four processes. The role of PM in initiating, promoting, or complicating atherosclerosis or its outcomes has yet to be determined (US EPA, 2009a, section 5.5). Furthermore, inflammation has the potential to promote thrombosis which can complicate this disorder leading to adverse effects on the cardiovascular and cerebrovascular systems such as thrombosis, plaque rupture, myocardial

[41] Atherosclerosis is a chronic progressive disease which contributes greatly to cardiovascular disease. Primarily a disease of the large arteries, it is characterized by the accumulation of lipid and fibrous tissue in atheromas, or swellings of the vessel wall. Although a strong link is known to exist between hypercholesterolemia and atherogenesis, recent studies demonstrate a key role for inflammation in the initiation and progression of atherosclerosis (Libby et al., 2002).

infarction (MI), and stroke may result. Systemic inflammation may also affect other organ systems such as the liver or the central nervous system (US EPA, 2009a, section 5.2).

Epidemiological studies conducted worldwide consistently demonstrate that both short- and long-term exposures to PM, are associated with a large variety of CV events: myocardial ischemia and infarction, heart failure, stroke, sudden death and arrhythmia, hospitalization for cardiovascular disease, and increased overall cardiovascular-related mortality (US EPA, 2009a, sections 6.2 and 7.2). The MOA(s) by which PM exposures may increase the risk of cardiovascular morbidity and mortality remains uncertain and much research is focusing on improving our understanding of this issue. In the current review, new evidence expands our understanding of PM-related effects that can lead to the development of a range of cardiovascular effects (see US EPA, 2009a, Figure 5-5 and section 5.4) including:

- **Alterations in vasomotor function** – the most noteworthy new health-related revelation in the past six years with regards to PM exposure is that the systemic vasculature may be a target organ. The second draft ISA presents alterations in vasomotor function which has been demonstrated following exposure to concentrated ambient particles (CAPs) and diesel exhaust (DE) (US EPA, 2009a, section 6.2.4)
- **Myocardial ischemia and myocardial infarction (MI)** – altered vasoreactivity of coronary vessels may lead to myocardial ischemia and MIs; MIs may alter conduction and depolarization properties of the heart potentially leading to arrhythmias
- **Thrombosis** –procoagulation effects or plaque destabilization or rupture may lead to stroke and/or thromboembolic disease
- **Arrhythmia -** alterations in the conduction and depolarization properties of the heart may lead to arrhythmic events

As noted in the second draft ISA, many of these process may be interlinked and responses to ambient PM exposures may involve multiple MOA simultaneously with some variability depending on PM composition. Furthermore, it is not clear at this time whether PM initiates cardiovascular disease or whether it perturbs existing disease (US EPA, 2009a, section 5.4)

Central Nervous System Effects. Recent studies have demonstrated PM-dependent effects on the central nervous system (CNS) though, at this time, it is unknown if these effects are related to direct or indirect effect of PM exposure. Translocation of soluble and poorly soluble particles from the olfactory mucosa via the axons to the olfactory bulb of the brain has been proposed as a possible MOA by which PM or its components may directly access the CNS. Given their small size, UFPs deposited onto nasal epithelium may be more efficiently translocated by this mechanism than other PM size fractions (US EPA, 2009a, sections 5.4 and 4.3.2.2).

Overall, the findings reported in the second draft ISA continue to support the overall conclusions from the previous PM NAAQS review indicating that different health responses are

linked with different particle characteristics and that many biologic responses may be responsible for the morbidity/mortality effects associated with these exposures.

3.3 NATURE OF EFFECTS

An extensive body of new scientific evidence has been published since completion of the 2004 PM AQCD including policy-relevant information from epidemiologic, controlled human exposure, toxicological, and exposure studies. In the last review, EPA concluded that recent epidemiologic studies 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. In addition, emerging evidence identified 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 (HRV) or increased C-reactive protein (CRP) levels, developmental effects such as low birth weight, and infant mortality, and lung cancer mortality (US EPA, 2004, p. 9-23 – 9.24).

The second draft ISA, along with its annexes, integrates newly available, policy-relevant epidemiologic, controlled human exposure, and animal toxicological evidence with consideration of key findings and conclusions from prior reviews to draw inferences about the relationship between short- and long-term exposures to PM and a range of human health effects. The second draft ISA evaluates evidence from the full body of health effect studies conducted world-wide and summarizes results of all such mortality and morbidity studies.

The discussions that follow draw primarily from evidence evaluated in chapters 6 and 7 of the second draft ISA as well as the integration of evidence across disciplines presented in chapter 2. Annexes B through F provide supplemental information. For the purposes of this preliminary draft Policy Assessment (PA), staff has drawn from the qualitative evaluation of all studies considered in the second draft ISA, with focus on those studies conducted in the U.S. and Canada for supporting quantitative assessments.[42] Effect estimates for mortality and morbidity effects associated with increments of $PM_{2.5}$ and $PM_{10-2.5}$ from multi-city and single-city U.S. and Canadian studies are summarized in Chapters 6 and 7 of the second draft ISA for short-term and long-term exposure studies, respectively, as a consolidated reference for the following discussions.[43] For each outcome considered, EPA staff has placed emphasis on multi-city studies concluding that these studies have a number of advantages compared to single-city

[42] 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.

[43] Effect estimates are presented using standardized PM increments to allow for comparison across studies. Increments of 10 $\mu g/m^3$ were generally used.

studies including: (1) multi-city studies reflect ambient PM levels and potential health impacts across a range of diverse locations; (2) multi-city studies "clearly do not suffer from potential omission of negative analyses due to 'publication bias'" (EPA, 2004, p. 8-30); and (3) multi-city studies generally have higher statistical power. Although the single-city studies may lack statistical power needed to evaluate interactions and detect some of the subtle effects of air pollution, they inform the interpretation of the heterogeneous effect estimates that have been observed across North America. Therefore, key single-city studies are also discussed in the second draft ISA and briefly discussed below.

In general, conclusions from the 2004 PM AQCD are summarized briefly at the beginning of each section, and the evaluation of evidence from recent studies builds upon the evidence that was available during the previous review. In the discussions that follow, an individual study result is considered to be statistically significant if the 95 percent confidence interval does not include zero. Positive effect estimates indicate increases in the health outcome with PM exposure. In considering these results as a whole, it is important to consider not only whether statistical significance at the 95 percent confidence level is reported in individual studies, but also the general pattern of results, focusing in particular on studies with greater statistical power that report relatively more precise results.

3.3.1 Causal Determinations

The EPA has developed a consistent and transparent framework for evaluating the causal nature of air pollution-induced health effects drawing upon the evaluation and synthesis of evidence from across scientific disciplines. This framework was developed considering guidance used by other regulatory and scientific agencies (US EPA, 2009a, section 1.5). In the second draft ISA, EPA assesses the new health evidence, building upon evidence available during previous PM NAAQS reviews, and integrates findings from epidemiologic studies with experimental (e.g., dosimetric and toxicological) studies, to make judgments about the extent to which causal determinations can be made between health endpoints and various indicators or constituents of ambient PM. More specifically, EPA has outlined a framework for causal determination that:

- describes the kinds of scientific evidence used in establishing a general causal relationship between exposure and health effects;
- defines cause, in contrast to statistical association;
- discusses the sources of evidence necessary to reach a conclusion about the existence of a causal relationship;
- highlights the issue of multi-factorial causation;
- identifies issues and approaches related to uncertainty; and

- provides a framework for classifying and characterizing the weight of evidence in support of a general causal relationship (US EPA, 2009a, section 1.5).

The EPA has used a two-step approach to evaluate the scientific evidence on health or environmental effects of PM. The first step determines the weight of evidence in support of causation and characterizes the strength of any resulting causal classification (US EPA, 2009a, section 1.5.5). The second step includes further evaluation of the quantitative evidence regarding the concentration-response relationships and the loads or levels, duration, and pattern of exposures at which effects are observed (US EPA, section 1.5.6).

As outlined in Table 1-2 of the second draft ISA, the aspects, or criteria, of causality considered include:

- consistency of the observed association
- coherence
- biological plausibility
- biological gradient (exposure-response relationship)
- strength of the observed association
- experimental evidence
- temporal relationship of the observed association
- specificity of the observed association
- analogy[44]

Aspects that EPA considers to generally play a larger role in determination of causality are "consistency of results across studies, coherence of effects observed in different study types or disciplines, biological plausibility, exposure-response relationship, and evidence from "natural" experiments." In addition, EPA recognizes "that no one aspect is either necessary or sufficient for drawing inferences of causality" (US EPA, 2009a, section 1.5.4).

In addition to controlled human exposure and epidemiologic studies, the tools of experimental biology have been valuable for developing insights into human physiology and pathology. Animal toxicological studies explore the effects of pollutants on human health, especially through the study of model systems in other species. These studies evaluate the effects of exposures to a variety of pollutants in a highly-controlled laboratory setting and allow exploration of MOAs or mechanisms by which a pollutant may cause effects. In making causal determinations, EPA has considered differences between animal species with regard to pollutant absorption and distribution profiles based on breathing patterns, exposure dose, and differences in lung structure and anatomy. Background knowledge of the biological mechanisms by which

[44] Structure activity relationships and information on the agent's structural analogs can provide insights into whether an association is causal. Similarly, information on the mode of action for a chemical, as one of many structural analogs, can inform decisions regarding likely causality.

an exposure might or might not cause disease can prove crucial in establishing, or negating a causal claim (US EPA, 2009a, section 1.5.3).

The second draft ISA uses a five-level hierarchy that classifies the weight of evidence for causation, not just association into a qualitative statement about the overall weight of evidence and causality (US EPA, 2009a, section 1.5.5):

- Causal relationship
- Likely to be a causal relationship
- Suggestive of a causal relationship
- Inadequate to infer a causal relationship
- Not likely to be a causal relationship (see US EPA, 2009a, Table 1-3)

Beyond judgments regarding causality are questions relevant to quantifying health risks based on our understanding of the quantitative relationships between pollutant exposure and health effects. The strength of associations most directly refers to the magnitude of the reported relative risk estimates. This requires evaluation of levels of PM and exposure durations at which effects were observed, including effects observed in at-risk populations. "This integration of evidence results in identification of a study or set of studies that best approximates the concentration-response relationships between health outcomes and PM indicators for the U.S. population or subpopulations, given the current state of knowledge and the uncertainties that surrounded these estimates." (US EPA, 2009a, section 1.5.6.1) Concentration-response relationships vary depending on specific health outcomes evaluated. Of particular relevance for the review of the PM NAAQS, is information on whether the concentration-response relationship is linear across the full concentration range evaluated, or if nonlinear relationships exist along any part of the range, in particular, at levels that are at or below the levels of the current PM standards (US EPA, 2009a, section 1.5.6). Information from toxicological and controlled human exposure studies may also be used to inform the evaluation of concentration-response relationships, specifically related to MOA and characteristics of at-risk populations. 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 (ISAC, 2004). With associations that yield small relative risk estimates it is especially important to consider potential confounding and other factors in assessing causality.

In considering both the magnitude and statistical strength of the associations, the second draft ISA observes a pattern of positive and often statistically significant associations for a number of different endpoints with the strongest evidence associated with exposures to fine particles, $PM_{2.5}$, and more limited evidence for thoracic coarse particles, $PM_{10-2.5}$, additional size fractions (e.g., UFPs), and specific PM components, sources, and environments (e.g., urban and non-urban environments). In considering the strength of the associations between short- and

long-term exposures to PM and mortality or morbidity, EPA has developed a number of causal determinations as summarized in Table 3-1 (US EPA, 2009a, sections 2.3.1.1, 2.3.1.2, 2.3.3.1, 2.3.4.1).

As was true in the last review, there is coherence in the epidemiologic evidence with supportive evidence from toxicological and controlled human exposure studies linking short- and long-term exposures to fine particles with mortality and morbidity effects on the cardiovascular and respiratory systems. The strongest evidence has been observed for cardiovascular effects associated with short-term fine particle exposures. More limited information is available linking $PM_{2.5}$ exposures to reproductive/developmental and cancer/mutagenic/genotoxic effects. Less information is available to allow conclusions to be drawn about coherence or plausibility for associations with $PM_{10-2.5}$ or UFPs. Table 3-1 summarizes the causal determinations by PM size fraction and exposure duration (i.e., short- and long-term exposure) as presented in the second draft ISA (US EPA, 2009a, section 2.3).

Table 3-1. Summary of Causality Determinations by PM Size Fraction

Size Fraction	Outcome	Causality Determination	
		Short-term Exposure	**Long-Term Exposure**
$PM_{2.5}$	**Cardiovascular effects**	Causal	Causal
	Respiratory effects	Likely to be causal	Likely to be causal
	Mortality	Likely to be causal	Likely to be causal
	Central nervous system (CNS) effects	Inadequate	_____
	Reproductive and developmental effects	_____	Suggestive
	Cancer, Mutagenicity, and Genotoxicity	_____	Suggestive
$PM_{10-2.5}$	**Cardiovascular effects**	Suggestive	Inadequate
	Respiratory effects	Suggestive	Inadequate
	CNS effects	Inadequate	_____
	Mortality	Suggestive	Inadequate
UFPs	**Cardiovascular effects**	Suggestive	Inadequate
	Respiratory effects	Suggestive	Inadequate
	CNS effects	Inadequate	_____
	Mortality	Inadequate	Inadequate

3.3.2 Fine Particles

The nature of the effects that have been reported to be associated with fine particle exposures include premature mortality, aggravation of respiratory and cardiovascular disease (as indicated by increased hospital admissions and emergency department visits), changes in lung function and increased respiratory symptoms, evidence for more subtle indicators of cardiovascular health, and more limited evidence of developmental effects (e.g., low birth weight, infant mortality) and death from lung cancer. Many studies using PM_{10} as an indicator have been conducted in areas where fine particles are the dominant fraction of PM_{10}; results of these studies, where appropriate, have been considered in evaluating the effects associated with fine particle exposures. Evidence is first presented for $PM_{2.5}$ mass followed by discussion of $PM_{2.5}$ components and UFPs.

3.3.2.1 Effects Associated with Short-term $PM_{2.5}$ Exposures

Numerous epidemiologic studies as well as supportive evidence from controlled human exposure and toxicological studies have demonstrated statistical associations between short-term exposure to fine particles and health outcomes related to cardiovascular effects, respiratory effects, and premature mortality. This information is presented and discussed in chapter 6 of the second draft ISA and briefly described below. Figure 2-1 in the second draft ISA (US EPA, 2009a, section 2.3.2) provides a summary of U.S. and Canadian studies examining the association between short-term exposure to $PM_{2.5}$ and cardiovascular and respiratory morbidity and mortality.

The majority of epidemiological studies provide limited information on the air quality data used in their analyses, generally reporting average concentrations rather that a range of upper percentile values making it difficult to understand the air quality distribution considered in the analyses. Being mindful that the form of the current 24-hour $PM_{2.5}$ standard is based on a 98^{th} percentile value averaged over three years to provide protection against short-term $PM_{2.5}$ exposures, EPA has taken steps to obtain additional air quality data from study authors to better understand the air quality distributions associated with the observed effects. In the external review draft Policy Assessment Document staff will evaluate 98^{th} percentile values of specific studies that reported statistically significant associations.

3.3.2.1.1 Cardiovascular and Systemic Effects

Several studies considered in the previous PM NAAQS review reported positive associations between short-term $PM_{2.5}$ exposures and hospital admissions or ED visits for cardiovascular disease, although few studies reported statistically significant effects. U.S. and Canadian studies available in the last review provided evidence of cardiovascular-related mortality that was consistent with evidence of all-cause (non-accidental) mortality. This

evidence was relatively stronger than that observed for respiratory-related mortality. In addition, significant associations were also observed between MI and short-term $PM_{2.5}$ exposures averaged over 2 or 24-hours, as well as evidence of decreases in heart rate variability (HRV) related to short-term $PM_{2.5}$ exposures in controlled human studies and animal toxicological studies. In the last review, evidence was emerging regarding more subtle indicators of cardiovascular health (US EPA, 2004; US EPA, 2005; see also summary at 71 FR 2626 to 2637, January 17, 2006).

In this review, the body of scientific evidence has been expanded greatly by the publication of a large number of new multi-city epidemiological studies that provide consistent evidence of a positive association between short-term $PM_{2.5}$ exposures and hospital admissions or ED visits as well as premature mortality related to cardiovascular outcomes. Hospital admissions and ED visits were reported, predominantly for ischemic heart disease (IHD) and congestive heart failure (CHF), with the majority of studies reporting increases ranging from 0.5 to 3.4% per 10 $\mu g/m^3$ increase in $PM_{2.5}$. The excess risk of cardiovascular disease (CVD) hospitalization may be somewhat greater in Europe and Australia/New Zealand than in the U.S. Evidence for a range of cardiovascular and systemic effects include: changes in heart rate and heart rate variability, arrthymia, ischemia, vasomotor function, changes in blood pressure, cardiac contractility, systemic inflammation, hemostasis, thrombosis and coagulation factors, systemic and cardiovascular oxidative stress. Controlled human exposure studies as well as toxicological studies provide evidence to provide biological plausibility for the findings observed in the epidemiologic studies (US EPA, 2009a, section 6.2.10.1).

In the second draft ISA, EPA concludes that, taken together, the collective evidence from epidemiological, controlled human exposure studies, and toxicological studies is sufficient to conclude that a ***causal*** relationship exists between short-term $PM_{2.5}$ exposures and cardiovascular effects (US EPA, 2009a, sections 2.3.1.1 and 6.2.12.1). The policy-relevant evidence supporting this causal determination is presented and discussed in section 6.2 of the second draft ISA and briefly summarized below. Excess risk estimates for studies of hospitalizations and ED visits associated with various cardiovascular endpoints are presented in figures 6-1 through 6-5.

Hospital admissions and emergency department visits for cardiovascular disease.

Epidemiologic studies reviewed in the second draft ISA substantiate prior findings of a positive association between exposure to $PM_{2.5}$ and hospital admissions and ED visits from cardiovascular disease. The 2004 PM AQCD reviewed more than 25 publications relating PM and risk of CVD hospitalizations. Results from a handful of larger multi-city studies were emphasized, with the greatest emphasis placed on findings from the U.S. National Morbidity, Mortality, and Air Pollution Study (NMMAPS) (Samet et al., 2000) and a subsequent reanalysis o f NMMAPS II data (Zanobetti and Schwartz, 2003). The NMMAPS II study evaluated the effect of daily changes in ambient PM levels on total CVD hospitalizations among elderly

Medicare beneficiaries in 14 U.S. cities and found a ~1% excess risk per 10 $\mu g/m^3$ increase in PM_{10}. The 2004 PM AQCD concluded that these results, along with those of the other single- and multi-city studies reviewed "generally appear to confirm likely excess risk of CVD-related hospital admissions for U.S. cities in the range of 0.6 to 1.7% per 10 $\mu g/m^3$ PM_{10}, especially among the elderly" (US EPA, 2004, section x.x). The 2004 PM AQCD also concluded that there was some evidence from single-city studies suggesting an excess risk specifically for hospitalizations related to ischemic heart disease and heart failure. Furthermore, the 2004 PM AQCD found that "insufficient data existed from the time-series CVD admissions studies […] to provide clear guidance as to which ambient PM components, defined on the basis of size or composition, determine ambient PM CVD effect potency" (U.S. EPA, 2004, section x.x). The key studies reviewed in the 2004 PM AQCD on this topic included those by Burnett et al.(1997), Lippman et al. (2000), Ito (2003), and Peters et al. (2001).

Recent large studies conducted in the U.S., Europe, and Australia and New Zealand have confirmed these findings for PM_{10}, and have also observed consistent associations between $PM_{2.5}$ and cardiovascular hospitalizations. However, findings from single-city studies have demonstrated regional heterogeneity in effect estimates. It is apparent from these recent studies that the observed increases in cardiovascular hospitalizations are largely due to admissions for ischemic heart disease (IHD) and congestive heart failure (CHF) rather than cerebrovascular disease (CBVD, such as stroke). Building upon the evidence available in the last review, the new literature on hospitalizations and ED visits for cardiovascular causes published since 2002 is reviewed in section 6.2.10 of the second draft ISA.

Almost all of the published time-series studies of cardiovascular hospitalizations and ED visits identified cases based on administrative discharge diagnosis codes as defined by the International Classification of Disease 9th revision (ICD-9) or 10th revision (ICD-10) (NCHS, 2007). A complicating factor in interpreting the results of these studies is the lack of consistency in both defining specific health outcomes and in the nomenclature used. [45]

Recently, multiple research groups in the U.S., Europe, and Australia have created large datasets to evaluate specific CVD and respiratory endpoints using more detailed and relevant measures of PM concentration. In the U.S., the MCAPS analyses of Dominici et al. (2006), Bell et al. (2008) and Peng et al. (2008) are large, comprehensive and informative studies based on

[45] The second draft ISA outlines major groups of diagnostic codes used in air pollution studies for diseases of the circulatory system. The codes ICD-9: 390-459 are frequently used to identify all CVD morbidity. Note that this definition of CVD includes diseases of the heart and coronary circulation, CBVD, and PVD. In contrast, the term cardiac disease specifically excludes diseases not involving the heart or coronary circulation. While this distinction is conceptually straightforward, the implementation of the definition of cardiac disease in terms of ICD-9 or ICD-10 codes varies among authors. Even greater heterogeneity can be found among studies in the implementation of definitions related to CBVD (US EPA, 2009a, Table 6-5, section 6.2.10)

Medicare hospitalization data. Likewise, the Atlanta-based SOPHIA study (Metzger et al., 2004; Peel et al., 2005; Tolbert et al., 2007) is the largest and most comprehensive study of U.S. cardiovascular and respiratory ED visits. In Europe, the APHEA initiative (Le Tertre et al., 2002; Le Tertre et al., 2003), the more recent HEAPSS study (Von Klot et al., 2005), and the French PSAS program (Host et al., 2008; Larrieu et al., 2007) are similarly noteworthy for their large sample size, geographic diversity, and consideration of specific CVD and/or respiratory endpoints. These studies contain adequate data to examine interactions by season and region; the effects of different size fractions, components and sources of PM; or the effect of PM on at-risk populations.

The 2004 PM AQCD (U.S. EPA, 2004) incorporated the results of a large number of time-series studies in the U.S. and elsewhere relating ambient PM levels and risk of hospitalization for CVD. Since then, the U.S. MCAPS study has reported new evidence, in older adults, evaluating the association between $PM_{2.5}$ and the risk of CVD hospitalization in 202 U.S. counties between 1999 and 2005. Specifically, investigators reported a 0.7% increase in risk per 10 $\mu g/m^3$ increase in $PM_{2.5}$ on day 0 (same day)[46] (Peng et al., 2008). Bell et al. (2008) found evidence of substantial and statistically significant variability in the effects of $PM_{2.5}$ on cardiovascular hospitalizations by season and region, with the highest national average estimates occurring in the winter (i.e., estimates for the nation (1.49%) and northeast (2.01%) were highest in the winter) and the highest regional estimates observed in the northeastern U.S. (1.08% per 10 $\mu g/m^3$ increase in $PM_{2.5}$).

Several large multi-city studies conducted outside and within the U.S., provide support for a PM effect on CVD hospitalizations including the European APHEA2 study (Le Tertre et al., 2002),the Spanish EMECAS study (Ballester et al., 2006), a study conducted in multiple cities across New Zealand and Australia (Barnett et al., 2006) as well as the Atlanta-based SOPHIA study (Metzger et al., 2004).

A number of single-city studies have been published showing positive associations between hospital admissions and ambient PM in Europe and the western U.S. In some of these studies the associations did not reach statistical significance potentially because the studies likely lacked the statistical power to find effects of the expected magnitude. In fact, it is possible that studies conducted outside of large metropolitan areas have sufficient statistical power to detect associations of the expected magnitude. For example, Delfino et al. (2009) evaluated the effects of the 2003 California wildfires and observed a slightly larger excess risk of total cardiovascular disease admissions during the wildfire period compared to the period prior to the wildfire although excess risk estimates were generally weak and non-significant. In addition, some of the

[46] See discussion of lag structure in section 3.4.

$PM_{2.5}$-related associations may not be statistically significant potentially because of regional and seasonal heterogeneity of the $PM_{2.5}$ ambient mix.

In summary, large studies from the U.S., Europe, and Australia/New Zealand published since the 2004 PM AQCD (U.S. EPA, 2004) provide strong support for an association between short-term increases in ambient levels of $PM_{2.5}$ and PM_{10} and increased risk of hospitalization for total CVD. Studies of specific CVD outcomes indicate that IHD and CHF may be driving the observed associations (US EPA, 2009a, sections 6.2.10.3 and 6.2.10.5). Although estimates from studies of cerebrovascular diseases are less precise and consistent, ischemic diseases appear to be more strongly associated with $PM_{2.5}$ compared to hemorrhagic stroke[47] (US EPA, 2009a, section 6.2.10.7). The available evidence suggest that these effects occur at very short lags (0-1 days), although effects at longer lags have rarely been evaluated. The results from hospital admissions and ED visits studies are supported by the associations observed between short-term $PM_{2.5}$ exposures and cardiovascular-related mortality, which also provide additional evidence for regional and seasonal variability in $PM_{2.5}$ risk estimates (see discussion in section 3.3.2.1.4). Furthermore, limited evidence from the studies that examined the C-R relationship between PM and CVD hospital admission and ED visits supports a no-threshold, log-linear model, which is also consistent with the observations made in studies that examined the PM-mortality relationship (US EPA, 2009a, section 6.2.10.10)

Out-of-hospital cardiac arrest. Only a few studies have examined out-of-hospital cardiac arrest or deaths (see US EPA, section 3.2.10.11). Two studies from Seattle, WA consistently found no association (also consistent with other cardiac effects and mortality studies conducted in that locale (add ref)). One study in Indianapolis, IN found an association with hourly $PM_{2.5}$ but not daily $PM_{2.5}$ (add ref). A study in Rome found an association with PM_{10} but also with particle numbers and CO, implicating combustion sources (add ref). Because multi-city mortality studies examining this association found heterogeneity in PM risk estimates across regions, future studies of out-of-hospital cardiac arrest may need to consider location and the air pollution mixture during their design. There have also been very few PM studies that have used hourly PM measurements and further studies are needed to confirm associations at shorter time scales.

Cardiovascular health outcomes. Studies evaluating changes in cardiovascular function provide support for the epidemiological evidence showing associations between short-term $PM_{2.5}$

[47] The epidemiology literature provides inconsistent support for an association between short-term increases in ambient levels of PM_{10} and $PM_{2.5}$ and risk of hospitalization and ED visits for CBVD. The heterogeneity in results is likely partly attributed to: 1) differences in the sensitivity and specificity of the various outcome definitions used in the relevant studies; 2) lag structures between PM exposure and stroke onset which may vary by stroke type and patient characteristics; and 3) exposure misclassification due to the use of hospital admission date rather than stroke onset time, which may vary by region, population characteristics, and stroke type (US EPA, 2009a, section 6.2.10.7)

exposure and cardiovascular-related hospital admissions, ED visits, and mortality. These changes have been reported in epidemiological, controlled human exposure, and/or toxicological studies conducted by multiple independent laboratories. In the second draft ISA, the following effects are discussed: heart rate and heart rate variability (section 6.2.1); arrhythmia (section 6.2.2); ischemia (section 6.2.3); vasomotor function (section 6.3.4); changes in blood pressure (section 6.2.5); cardiac contractility (section 6.2.6); systemic inflammation (section 6.2.7); hemostasis, thrombosis, and coagulation factors (section 6.2.8); and systemic and cardiovascular oxidative stress (section 6.2.9). Highlights of this evidence is summarized below.

Vasomotor function. The most noteworthy new health-related revelation in the past six years with regards to PM exposure is that the systemic vasculature[48] may be a target organ. The second draft ISA presents alterations in vasomotor function which has been demonstrated following exposure to concentration ambient particles (CAPS) and diesel exhaust (DE) (US EPA, 2009a, section 6.2.4).

The majority of the new evidence of particle-induced changes in vasomotor function in controlled human exposure studies comes from studies of exposures to DE (US EPA, 2009a, section x.x). None of these studies have evaluated the effects of DE with and without a particle trap. However, it is important to note that a study by Peretz et al. (2008) used a newer diesel engine with lower gaseous emissions and reported significant DE-induced decreases in BAD. In addition, increasing the particle exposure concentration from 100 to 200 $\mu g/m^3$, without proportional increases in NO, NO_2, or CO, resulted in an approximate 100% increase in response. Further evidence of a particle effect on vasomotor function is provided by significant changes in BAD demonstrated in healthy adults following controlled exposure to CAPs with O_3 (Brook et al., 2002). These findings are consistent with epidemiologic studies of various measures of vasomotor function (e.g., FMD and BAD were the most common), which have demonstrated an association with short-term $PM_{2.5}$ exposures in healthy and diabetic populations (US EPA, 2009a, section x.x). A limited number of epidemiologic studies examined multiple lags and the strongest associations were with either the 6 day mean concentration (O'Neill et al.,

[48] The vasculature of all tissues is lined with endothelial cells that will naturally encounter any systemically absorbed toxin. The endothelium (1) maintains barrier integrity to ensure fluid compartmentalization, (2) communicates dilatory and constrictive stimuli to vascular smooth muscle cells, and (3) recruits inflammatory cells to injured regions. Smooth muscle cells lie within the layer of endothelium and are crucial to the regulation of blood flow and pressure. In states of injury and disease, both cell types can exhibit dysfunction and even pathological responses.
Endothelial dysfunction is a factor in many diseases and may contribute to the origin and/or exacerbation of perfusion-limited diseases, such as MI or IHD, as well as hypertension. Endothelial dysfunction is also a characteristic feature of early and advanced atherosclerosis. A primary outcome of endothelial dysfunction is impaired vasodilatation, frequently due to uncoupling of NOS. It is this uncoupling that appears central to impaired vasodilation and thus endothelial dysfunction.

2005) or the concurrent day (Schneider et al., 2008) either the 6 day mean concentration (O'Neill et al., 2005) or the concurrent day (Schneider et al., 2008).

The toxicological findings with respect to vascular reactivity are generally in agreement and demonstrate impaired dilation following $PM_{2.5}$ exposure that is likely endothelium dependent (US EPA, 2009a, section x.x). These effects have been demonstrated in varying vessels and in response to different $PM_{2.5}$ types, albeit using IT exposure in most studies. Further support is provided by IT studies of ambient PM_{10} that also demonstrate impaired vasodilation and a $PM_{2.5}$ CAPs study that reported decreased L/W ratio of the pulmonary artery. An inhalation study of Boston $PM_{2.5}$ CAPs reported increases in coronary vascular resistance during ischemia, which indicated a possible role for PM-induced coronary vasoconstriction. The mechanism behind impaired dilation following PM exposure may include increased ROS and RNS production in the microvascular wall that leads to altered NO bioavailability and endothelial dysfunction. Despite the limited number of inhalation studies conducted with concentrations near ambient levels, the toxicological studies collectively provide coherence and biological plausibility for the myocardial ischemia observed in controlled human exposure and epidemiologic studies.

Ischemia. Epidemiologic studies demonstrate associations between $PM_{2.5}$ pollution and ST-segment depression at lags of 1 hour to 2 days. Morever, these findings demonstrate a potential role for traffic (Chuang et al., 2008; Gold et al., 2005) and long-range transported PM2.5 (Lanki et al., 2006). Toxicological studies have reported reduced myocardial blood flow during ischemia and altered vascular reactivity (add ref). These studies provide coherence and biological plausibility for the myocardial ischemia that has been observed in both controlled human exposure and epidemiological studies (US EPA, 2009a, section 6.2.3).

Heart rate and heart rate variability. Heart rate (HR), HRV[49], and BP are all regulated, in part, by the sympathetic and parasympathetic nervous systems. Changes in one or more may increase the risk of cardiovascular events (e.g., arrhythmias, MI, etc.). Decreases in HRV have been associated with cardiovascular mortality/morbidity in older adults and those with significant heart disease (TFESC, 1996). While HRV is commonly described as being a

[49] HRV is measured using electrocardiograms (ECG) and can be analyzed in the time domain (e.g., standard deviation of all NN intervals [SDNN], square root of the mean squared successive NN interval differences [r-MSSD]), and/or the frequency domain measured by power spectral analysis (e.g., high frequency [HF], low frequency [LF], ratio of LF to HF [LF/HF]). SDNN generally reflects the overall modulation of HR by the autonomic nervous system, whereas r-MSSD generally reflects parasympathetic activity and high frequency variations in HR. Thus, r-MSSD is generally well correlated with HF, which also reflects the parasympathetic modulation of HR. LF is predominately dictated by sympathetic tone and increased LF/HF indicates sympathoexcitation, which correlates overall with decreased overall HRV (SDNN, rMSSD). Thus LF/HF is thought to estimate the ratio of sympathetic influences on HR to parasympathetic influences (see US EPA, 2009a, section 6.2.1).

reflection of vagal and adrenergic input to the heart, there is clearly a more complex phenomenon reflected in HRV parameters. Rowan et al. (2007) provide a review of HRV and its use and interpretation with respect to air pollution studies. To summarize, HRV indices are excellent measures of extrapulmonary effects from inhaled pollutants, but the characterization of the acute, reversible responses to air pollution as being either parasympathetic or sympathetic in origin, much less predictive of some adverse outcomes such as ventricular arrhythmia, is relatively unsupported by the clinical literature. This is consistent with the conclusions presented in the 2004 PM AQCD which stated that there is inherent variability in the minute-to-minute spectral measurements, but long-term HRV measures demonstrate excellent day-to-day reproducibility (U.S. EPA, 2004, section x.x).

The 2004 PM AQCD (U.S. EPA, 2004) presented limited evidence of PM-induced changes in HRV. However, findings from epidemiologic, controlled human exposure, and toxicological studies were seemingly contradictory, with reports of both decreases and increases in HRV following PM exposure. Recent epidemiologic studies have demonstrated a more consistent decrease in HRV (SDNN and r-MSSD), which is supported by several controlled human exposure studies published since 2003, however, the observed changes are often variable. In these studies, decreases in HRV were observed among healthy adults following short-term exposures to $PM_{2.5}$ CAPs, but not in adults with asthma or COPD. Several studies have investigated the association between acute changes in multiple HRV parameters and ambient air pollutant concentrations in the U.S., Canada, Europe, Mexico, and Asia. Features and results of these studies are presented in Table 6-1, and the discussed in section 6.2.1 of the second draft ISA.

Arrhythmia. Epidemiologic and toxicological studies presented in the 2004 PM AQCD (U.S. EPA, 2004) provided some evidence of arrhythmia following exposure to PM. However, a positive association between PM and ventricular arrhythmias among patients with implantable cardioverter defibrillators was only observed in one study conducted in Boston, MA (Peters et al, 2000), while toxicological studies reported arrhythmogenesis in rodents following exposure to ROFA, diesel exhaust, or metals. Recent epidemiologic studies have confirmed the findings of PM-induced ventricular arrhythmias in Boston, MA, and have also reported increases in ectopic beats in studies conducted in the Midwest and Pacific Northwest regions of the U.S. In addition, two studies from Germany have demonstrated positive associations between traffic and combustion particles and changes in repolarization parameters among patients with ischemic heart disease (US EPA 2009, section 6.2.3).

Since 2004, only two studies (in Boston and Sweden), reported adverse associations of $PM_{2.5}$, other size fractions and components with ICD detected ventricular arrhythmias (Dockery et al., 2005a; Dockery et al., 2005b; Ljungman et al., 2008; Rich et al., 2005), while other studies

done elsewhere did not (Metzger et al., 2007; Rich et al., 2004; Vedal et al., 2004). A range in exposure lags was evaluated in the Boston study (3 hours to 3 days) (Dockery et al., 2005a; Dockery et al., 2005b; Rich et al., 2005) and Sweden study (2 hours and 24 hours) (Ljungman et al., 2008). Given the unique and homogenous nature of the study populations, it is not clear why there is not more consistency across these studies. Rich et al. (2005) reported that use of the mean pollutant concentration from the specific 24 hours before the arrhythmia rather than just the day of the arrhythmia, resulted in less exposure misclassification and less bias towards the null, possibly explaining the lack of association when using just the day of ICD discharge and daily PM concentrations (Rich et al., 2005). Other reasons for the inconsistent findings may include differing degrees of exposure misclassification within each study or city due to differences in PM composition and pollutant mixes (e.g., less transition metals and sulfates in the Pacific Northwest than the Northeast U.S.), and differences in the size of study areas. Studies of ventricular arrhythmia and PM concentration in patients with ICDs are summarized in Table 6-1 of the second draft ISA.

A few panel studies have used ECG recordings to evaluate associations between ectopic beats (ventricular or supraventricular)[50] and mean PM concentrations in the previous hours and/or days (Berger et al., 2006; Ebelt et al., 2005; Sarnat et al., 2006). Four studies of ectopic beats and runs of supraventricular and ventricular tachycardia, captured using ECG measurements, all report at least one positive association. Further, they report findings in regions other than Boston and Sweden (i.e., Midwest U.S., Pacific Northwest, 24 U.S. cities, and Erfurt, Germany). A range of lags and/or moving averages were investigated (0-30 days) with the strongest effects observed for either the 5-day mean, same day, or 1-day lagged PM concentrations. Taken together, these ICD studies and ectopy studies provide evidence of an arrhythmic response to PM, although further study is needed to understand the variable ICD study findings.

No reported investigations of the relationship of PM concentration and ECG abnormalities indicating arrhythmia were conducted prior to 2002 and thus were not included in the 2004 PM AQCD (U.S. EPA, 2004). Abnormalities in the myocardial substrate, myocardial

[50] Ectopic beats are defined as heart beats that originate at a location in the heart outside of the sinus node. They are the most common disturbance in heart rhythm. Ectopic beats are usually benign, and may be present with or without symptoms, such as palpitations or dizziness. Such beats can arise in the atria or ventricles. When the origin is in the atria the beat is called an atrial or supraventricular ectopic beat. When such a beat occurs earlier than expected it is referred to as a premature supraventricular or atrial premature beat. Likewise, when the origin is in the ventricle the beat is defined as a ventricular ectopic beat, or when early a premature ventricular beat. When three or more occur ectopic beats occur in succession, this is called a non-sustained run of either supraventricular or ventricular origin. When the rate of the run is greater than 100 beats per minute it is defined as a tachycardia. Sustained ventricular tachycardias are the arrhythmias investigated in the ICD studies described in the section above.

vulnerability, and resulting repolarization abnormalities are believed to be key factors contributing to the development of arrhythmogenic conditions. These abnormalities include ECG measures of repolarization such as QT duration (time for depolarization and repolarization of the ventricles), T-wave complexity (a measure of repolarization morphology), and T-wave amplitude (height of the T-wave). Abnormalities in repolarization may also identify subjects potentially at risk of more serious events such as sudden cardiac death (Atiga et al., 1998; Berger et al., 1997; Chevalier et al., 2003; Okin et al., 2000; Zabel et al., 1998). Two recent studies of changes in these measures following acute increases in air pollution have been conducted in Erfurt, Germany (Henneberger et al., 2005; Yue et al., 2007). These two analyses demonstrated associations between PM pollution and repolarization changes (QT duration, T-wave complexity, T-wave amplitude, T-wave amplitude variability), at lags of 5 hours to 2 days. Moreover, the findings from the Yue et al. (2007) study demonstrate a potential role of traffic particles/pollution (US EPA, 2009a, section 6.2.x)

Changes in Blood Pressure. One of the potential outcomes of air pollution-mediated alterations in vascular tone is its impact on variable BP or hypertension. Blood pressure is tightly regulated by autonomic (central and local), cardiac, renal, and regional vascular homeostatic mechanisms with changes in arterial tone being countered by changes in cardiac contractility, HR, or fluid volume. The evidence of PM-induced changes in BP presented in the 2004 PM AQCD (U.S. EPA, 2004) was limited and inconsistent. Recent epidemiologic, human clinical, and toxicological studies have similarly reported conflicting results regarding the effect of PM on BP. A significant increase in DBP was observed in the only human clinical study that evaluated BP during exposure (concomitant exposure to CAPs and O_3). In addition, evidence from toxicological studies suggests that the effect of PM on BP may be modified by health status, as PM-induced increases in BP have been more consistently observed in SH rats.

These studies are not entirely consistent with regard to the assessed associations between BP and PM exposure. Most have reported increases in SBP and DBP associated with increases in either $PM_{2.5}$, PM_{10}, or UFPs (Choi et al., 2007; Chuang et al., 2005; Mar et al., 2005; Zanobetti et al., 2004). However, two studies reported small decreases in BP associated with multiple particulate pollutants (Ibald-Mulli et al., 2004; Mar et al., 2005). Dales et al. (2007) reported no change in BP associated with a 2-hour exposure to bus stop $PM_{2.5}$ and Jansen at al. (2005) reported null findings among older adults in Seattle, WA. Exposure lags ranging from 1-3 hours (Chuang et al., 2005), to the same day (Ebelt et al., 2005; Mar et al., 2005; Rich et al., 2008), to the mean across the previous 5 days (Zanobetti et al., 2004) were reported as having the strongest associations with BP.

Several recent studies have reported increased risk of hospital admissions for congestive heart failure (CHF) associated with increased PM concentration on the same day (Wellenius et

al., 2005; Wellenius et al., 2006). As a possible mechanism for these reported associations, Rich et al. (2008) hypothesized that these hospital admissions for decompensation of heart failure would be preceded by more subtle increases in pulmonary arterial (PA) and right ventricular (RV) diastolic pressures. Wellenius et al. (2007) conducted a panel study of 28 subjects living in the greater Boston metropolitan area, each with chronic stable heart failure and impaired systolic function. They hypothesized that circulating levels of B-type natriuretic peptide (BNP), measured in whole blood at 0, 6, and 12 weeks, were associated with acute changes in ambient air pollution, as a possible mechanistic explanation for the observed association between hospital admissions for heart failure and ambient PM concentration (Wellenius et al., 2005; Wellenius et al., 2006). During the study, the mean $PM_{2.5}$ concentration was 10.9 $\mu g/m^3$. Using linear mixed models, they reported no association between any pollutant ($PM_{2.5}$, CO, SO_2, NO_2, O_3, and BC) and BNP at any lag (Wellenius et al., 2007). However, BNP the active peptide has a very short half-life and might not be the best biomarker for such a study. Thus the absence of a correlation between PM and BNP may not suggest that PM does not have an impact on RV or LV function in individuals with impaired cardiac mechanics.

Systemic inflammation. The evidence presented in the 2004 PM AQCD (U.S. EPA, 2004) of increases in markers of systemic inflammation associated with PM was limited and not sufficient to reach a definitive conclusion. These studies were found to offer limited support for mechanistic explanations of the associations between PM concentration and heart disease outcomes. Recent controlled human exposure and toxicological studies continue to provide mixed results for an effect of PM on markers of systemic inflammation including cytokine levels, C-reactive protein (CRP), and white blood cell count. While results from recent epidemiologic studies have also been inconsistent across studies, there is some evidence to suggest that PM levels may have a greater effect on inflammatory markers among populations with preexisting diseases.

In recent studies, the most commonly measured marker of inflammation was CRP. Several other markers were examined in only a few studies, in relation to a wide range PM size fractions and components. These markers included IL-6, TNF-α, vascular cell adhesion molecule-1 (VCAM-1), intercellular adhesion molecule-1 (ICAM-1), soluble CD40 ligand (sCD40L), white blood cells (WBC) and soluble adhesion molecules (sP-selectin and e-selectin). CRP was not consistently associated with short-term PM concentrations ($PM_{2.5}$, PM_{10}, SO_4^{2-}, EC, OC, PNC). Several other markers of inflammation have been examined in relation to several PM size fractions and components but the number of studies examining the same marker/PM metric combination is too few to allow results to be compared across epidemiologic studies (US EPA, 2009a, section 6.2.7).

Hemostasis, thrombosis and coagulation factors. The 2004 PM AQCD presented

limited and inconsistent evidence from epidemiologic, controlled human exposure, and toxicological studies of PM-induced changes in blood coagulation markers (US EPA, 2004, section x.x). The body of scientific literature investigating hemostatic effects of PM has grown significantly since the publication of the 2004 PM AQCD, with a limited number of epidemiologic studies demonstrating consistent increases in von Willebrand factor (vWf) associated with PM and less consistent associations with fibrinogen. Recent human clinical and toxicological studies have also observed changes in blood coagulation markers (e.g., fibrinogen, vWf, factor VII, t-PA) following exposure to PM. However, the findings of these studies are somewhat inconsistent, which may be due in part to differences in the post-exposure timing of the assessment (US EPA, 2009a, section 6.2.8).

Systemic and cardiovascular oxidative stress. Very little information on systemic oxidative stress associated with PM was available for inclusion in the 2004 PM AQCD. Since 2002, numerous studies have examined whether short-term increases in mean PM concentrations are associated with adverse changes in systemic markers of oxidative stress. Recent epidemiologic studies have provided consistent evidence of PM-induced increases in markers of systemic oxidative stress including plasma TBARS, CuZn-SOD, 8-oxodG, and total homocysteine. This is supported by a limited number of controlled human exposure studies that observed PM-induced increases in free-radical mediated lipid peroxidation as well as upregulation of the DNA repair gene hOGG1 (US EPA, 2009a, section x.x). In addition, recent toxicological studies have demonstrated an increase in cardiovascular oxidative stress following PM exposure in rats, see section 6.2.9 of the second draft ISA (US EPA, 2009a, section x.x).

3.3.2.1.2 Respiratory Effects

Epidemiological studies reviewed in the second draft ISA substantiate prior findings of a positive relation between exposure to fine particles and respiratory morbidity, particularly for such endpoints as hospital admissions and emergency department and physician visits, and for respiratory symptoms, pulmonary function and inflammation among asthmatic children. In the second draft ISA, EPA concludes that, taken together, the collective evidence from epidemiological studies, controlled human exposure studies, and toxicological studies is sufficient to conclude that a ***likely causal*** relationship exists between short-term $PM_{2.5}$ exposures and respiratory effects (sections 2.3.1.1 and 6.3.10.1, US EPA, 2009a). The information supporting this causal determination is presented and discussed in section 6.3 of the second draft ISA and briefly summarized below.

Hospital admissions or emergency department (ED) and physician visits for respiratory diseases. Several large multi-city studies conducted in the US and other countries since the last review report a positive association between short-term increases in $PM_{2.5}$ levels and ED and

physician visits and hospital admissions for respiratory diseases. Among older adults evaluated in the multi-city Medicare Air Pollution Study (MCAPS),, investigators reported null findings for national estimates of $PM_{2.5}$ associated respiratory hospitalizations, but observed seasonal and regional heterogeneity in effect estimates, with a statistically significant increased risk of respiratory hospitalization during the winter in the Northeast (Bell et al., 2008). In addition, significant increases in respiratory admission were observed at lag 0 and lag 2.

Among children, effect estimates were consistently positive between short-term exposure to $PM_{2.5}$ and ED visits and hospitalizations for respiratory diseases (US EPA, 2009a, section 6.3.10.1). Single-city studies generally report positive, non-significant associations for $PM_{2.5}$ exposures in relation to respiratory hospitalizations (section 6.3.8.1, US EPA, 2009a). Recent studies considering adults and children combined report short-term $PM_{2.5}$ exposures that are, in general, positively associated with specific disease endpoints, including asthma, COPD, and respiratory infection (US EPA, 2009a, sections 6.3.8.2, 6.3.8.3, 6.3.8.4). However, for asthma-related hospitalizations, studies conducted among only children are inconsistent across varying ages and lag periods. In contrast to asthma-related hospitalizations, several studies of children report positive, statistically non-significant associations between short-term $PM_{2.5}$ exposure and hospitalizations related to respiratory infections (section 6.3.8.4, US EPA, 2009a). These results are similarly found in studies conducted among adults hospitalized for respiratory infections (US EPA, 2009a, Figure 6-14). These recent epidemiological studies are consistent with the prior review in finding an association between short-term exposure to $PM_{2.5}$ and ED visits and hospital admissions related to respiratory diseases.

Respiratory symptoms and lung function changes. Several single city studies included in the second draft ISA (section 6.3.1.1.,USA EPA, 2009a) report positive associations between short-term exposures to $PM_{2.5}$ and respiratory symptoms (e.g., cough, shortness of breath, chest tightness) among asthmatic children. However, most of these associations failed to achieve statistical significance and a multi-city U.S. based study (O' Connor et al., 2008) reported a negative association between $PM_{2.5}$ and wheeze among asthmatic children. Among asthmatic adults, the findings from recent studies are inconsistent, with several single-city studies including one US based study reporting negative associations between $PM_{2.5}$ and respiratory symptoms (Figure 6-8, US EPA, 2009a). The few studies examining the relationship between short-term $PM_{2.5}$ exposures and respiratory symptoms among healthy children and adults have not observed a consistent association (sections 6.3.1 and 6.3.10.1, US EPA, 2009a). In addition, healthy individuals have not exhibited an association between respiratory symptoms and medication use with exposure to $PM_{2.5}$.

Several panel studies discussed in the second draft ISA report statistically significant associations at varying exposure periods (e.g., 5-day average, lag 1, lag 3-5) to $PM_{2.5}$ and

reduced lung function (e.g.,. FEV_1) among asthmatic children that were generally robust to adjustment by gaseous co-pollutants (section 6.3.2.1, US EPA, 2009a). The few US based studies conducted among healthy adults and adults with COPD report inconsistent findings for the association between short term $PM_{2.5}$ exposure and lung function, although several studies conducted outside the U.S. and Canada all reported a decrease in lung function (US EPA, 2009a, section 6.8.2.1). Overall, the recent epidemiological evidence is consistent with the prior review which reported statistically significant associations between short-term exposures to $PM_{2.5}$ and respiratory symptoms and small decrements in lung function, respectively, among asthmatics.

Oxygen Saturation. Oxygen saturation measures the percentage of hemoglobin binding sites in the bloodstream occupied by oxygen. Two panel studies have evaluated the effect of $PM_{2.5}$ on oxygen saturation in older adults with different results. DeMeo et al. (2004) estimated the change in oxygen saturation and mean $PM_{2.5}$ concentration in the previous 24 hours. They used the same panel of elderly Boston residents (n = 28) and study protocol and analytic methods (12 weeks of repeated oxygen saturation measurements) as Gold et al. (2005) and Schwartz et al. (2005). At each clinic visit, subjects had 5 minutes each of rest, standing, post-exercise rest, and 20 cycles of paced breathing. $PM_{2.5}$ levels were associated with decreases in oxygen saturation in each of the 5-minute periods except exercise. The authors suggest that these oxygen saturation reductions may result from pulmonary vascular and inflammatory changes. In a similar study, Goldberg et al. (2008) examined the association between oxygen saturation and ambient $PM_{2.5}$, NO_2, and SO_2 concentrations in a panel of 31 subjects in Montreal, with NYHA Class II or III heart failure who were aged 50-85 years. Only SO_2 was significantly associated with reduced oxygen saturation in multivariate adjusted models.

3.3.2.1.3 Central Nervous System Effects

There are relatively few epidemiological studies assessing $PM_{2.5}$ associated central nervous system (CNS) effects in comparison to other health outcomes, and no evidence was presented of an effect of PM on the CNS in the prior review (, USA EPA, 2009a section 6.4) . In the single epidemiological study discussed in the second draft ISA, utilizing the Third National Health and Nutrition Examination Survey (NHANES III), investigators found PM_{10} was associated with reduced performance neurobehavioral tests of reaction time, visuomotor speed, attention, and short-term memory. However, these effects were greatly attenuated with adjustment for race/ethnicity and socio-economic factors, suggesting confounding of the association due to residential segregation in areas of high PM_{10} levels (Chen and Schwartz, 2009). The contribution of the fine fraction to the PM_{10} levels observed in this study are unknown, and therefore represent a challenge in determining the independent effect of $PM_{2.5}$ on neurobehavioral function. The single human clinical study that evaluated CNS effects included

in the current review is suggestive of acute cortical stress response to diesel exhaust exposure, but the independent effect of $PM_{2.5}$ as compared to the ultrafine fraction and diesel gases is unclear (Cruts et al., 2008). Recent inhalation toxicological studies provide potential mechanistic evidence, indicating inflammation in the brain and neurotransmitter modulation in response to concentrated air particles (CAPs). However, the levels of $PM_{2.5}$ observed in these studies are not representative of exposures to ambient levels of $PM_{2.5}$. Additional evidence beyond the currently available toxicological data is needed to identify potential biological mechanisms and assess the effects of specific PM size fractions.

Overall, in the second draft ISA, EPA concludes that the evidence is ***inadequate*** to determine if a causal relationship exists between short-term exposures to $PM_{2.5}$ and CNS outcomes (US EPA, 2009a, section 6.4.4)

3.3.2.1.4 Mortality

The prior review reported consistent evidence of a relation between short-term exposure to $PM_{2.5}$ and mortality in locations with varying climates, PM composition, and levels of gaseous co-pollutants (US EPA, 2004, Criteria Document, section 8.2). Two multi-city studies assessing the $PM_{2.5}$-daily mortality relation were discussed in the prior review, one of which was conducted among participants of the Six Cities Study cohort (Schwartz et al., 2003, Klemm and Mason, 2003) and the other was conducted in eight Canadian cities (Burnett and Goldberg, 2003). A reanalysis of Six Cities Study data reported results consistent with the findings of the original study, with statistically significant increases ranging from 2% to over 3% reported for all cause mortality associated with a 25 $\mu g/m^3$ increment increase in $PM_{2.5}$ (Schwartz, 2003a; Klemm and Mason, 2003). In a study using data from the eight largest Canadian cities, a positive and statistically significant association was reported between $PM_{2.5}$ exposure and the daily mortality that was similar to the aforementioned findings from the Six Cities Study and generally remained statistically significant in a number of analyses when gaseous co-pollutants and 0- and 1-day lags were included in the models (Burnett and Goldberg, 2003).

A number of new multi-city and single-city studies and additional analysis of existing data sets since the last review assessed the association between short-term exposure to $PM_{2.5}$, and mortality using time-series and case-crossover study designs. Of particular note is the National Morbidity, Mortality and Air Pollution Study (NMMAPS) which included analyses of $PM_{2.5}$ effects on mortality in 96 U.S. cities (Dominici et al., 2007). $PM_{2.5}$ at lag 1-day was positively associated with all-cause [0.29% (PI: 0.01, 0.57)] and cardio-respiratory mortality [0.38% (PI: -0.07, 0.82)], respectively, per 10 $\mu g/m^3$, although the association achieved statistical significance only for all-cause mortality. In addition, these associations were sensitive to the degree of adjustment for temporal confounding, although a smaller sample size for the $PM_{2.5}$ analysis (as

compared to the PM_{10} analysis) may have contributed to this observation (section 6.5.2.2 – 2nd draft ISA – USA EPA, 2009a). In another multi-city study, Franklin et al. analyzed daily mortality data from 27 U.S. cities using a case-crossover study design and reported $PM_{2.5}$ was positively and statistically significantly associated with all-cause [1.2% (CI: 0.29, 2.1)], respiratory, [1.8% (CI: 0.20, 3.4)] and stroke deaths [1.0% (CI: 0.02, 2.0)], respectively, per 10 $\mu g/m^3$ (Franklin et al., 2007). $PM_{2.5}$ was positively but non-significantly associated with cardiovascular deaths [0.94% (CI; -0.14, 2.0)]. In a subsequent time-series analysis Franklin et al. investigated the association between $PM_{2.5}$ and daily mortality between 2000 and 2005 in 25 U.S. cites, of which 15 cities overlap with the 27 cities examined in Franklin et al. 2007 (Franklin et al., 2008). A seasonal pattern was observed between $PM_{2.5}$ and daily mortality, with the highest $PM_{2.5}$ risk estimates reported for the spring and summer seasons. The $PM_{2.5}$ risk estimates for all-cause, cardiovascular, and respiratory deaths were generally similar to their 27 city study, and the regional pattern (i.e., eastern city $PM_{2.5}$ risk estimates higher than western city $PM_{2.5}$ risk estimates) was consistent across both studies.

In a multi-city time-series analysis of 112 U.S. cities, Zanobetti and Schwartz (2009) reported that mean $PM_{2.5}$ levels from lag 0-1 days was associated with 0.98% (CI: 0.75, 1.22) for all-cause, 0.85% (CI: 0.46, 1.24) for cardiovascular disease, 1.18% (CI: 0.48, 1.89) for myocardial infarction, 1.78% (CI: 0.96, 2.62) for stroke, and 1.68% (CI: 1.04, 2.33) for respiratory mortality per $10\mu g/m^3$ $PM_{2.5}$. In co-pollutant models including $PM_{10-2.5}$, $PM_{2.5}$ risk estimates declined in magnitude but remained positive and statistically significant for all-cause, cardiovascular disease, and respiratory-related mortality, respectively. Similarly to the seasonal analysis in the aforementioned 25 city study (Franklin et al., 2008), the risk estimate for all-cause mortality was highest during the spring season [2.57% (CI: 1.96, 3.19)] in comparison to the risk estimates for summer, fall, and winter seasons, which ranged from 0.25% to 0.95%.

In an analysis of risk estimates aggregated by climate regions, the authors reported $PM_{2.5}$ risk estimates generally similar across the regions with the exception of the Mediterranean region[51], which exhibited a lower risk estimates in comparison to the other regions. However, intra-regional risk estimates were significantly heterogeneous for the Mediterranean region, potentially contributing to the lower effect estimate observed in comparison to the regions. In a study estimating the daily mortality risk associated with short-term exposure to $PM_{2.5}$ for a single U.S. region (i.e., 9 counties in California) Ostro et al. reported mean $PM_{2.5}$ levels from lag 0-1 days were significantly associated with all-cause 0.6% (CI: 0.2, 1.0), cardiovascular 0.6% (CI: 0.0,1.1) respiratory [2.2% (CI: 0.6, 3.9)], and diabetes 2.4% (CI: 0.6, 4.2) mortality, respectively, per 10 $\mu g/m^3$ (Ostro et al., 2006). Although utilizing different lag periods, the results presented

[51] The Mediterranean climate considered in this analysis included California, Oregon, and Washington (Zanobetti and Schwartz, 2009).

by Ostro et al. and Franklin et al. are generally consistent for the 5 California counties included in both studies. In addition to U.S. based studies, the second draft ISA highlighted results from an analysis of $PM_{2.5}$ associated daily mortality for 12 Canadian cities, reporting that $PM_{2.5}$ risk estimates were negative in models adjusting for NO_2 for years in which $PM_{2.5}$ data was only available on every 6th day (Burnett et al., 2004). In s subsequent analysis, using data for years in which daily $PM_{2.5}$ levels were available for 11 of the 12 cities, in models including NO_2 the risk estimates for $PM_{2.5}$ remained positive, but statistically non-significant.

Multi-city studies support a largely positive and frequently statistically significant relationship between short-term exposure to $PM_{2.5}$ and increased risk of mortality. In general, it can be seen in Figure 6-23 of the second draft ISA that the effect estimates for associations between mortality and short-term exposure to $PM_{2.5}$ are positive and a number are statistically significant. The second draft ISA concludes that risk estimates from multi-city U.S. based studies fall in the range of 0.29% to 1.21% per 10 $\mu g/m^3$ $PM_{2.5}$ for all-cause mortality, which is similar to the risk estimates for cardiovascular mortality (0.30% to 1.03% vs. 1.2% to 2.7% in the last review) per 10 $\mu g/m^3$ $PM_{2.5}$, but less than the risk estimates for respiratory mortality (1.01% to 2.2% vs. 0.8% to 2.7% in 2004 review) using the same exposure period (i.e., lag period) and averaging time (e.g., 24-hour averaging time) (USA EPA, 2009a, section 6.5.x.x). The somewhat larger effect estimates reported for associations with respiratory mortality in comparison to cardiovascular and all-cause mortality included larger confidence intervals (i.e. less precision) since respiratory deaths comprise only a small proportion of total deaths. In addition, the current range of risk estimates for $PM_{2.5}$ associated cardiovascular mortality is narrower than the 2004 estimates of cardiovascular mortality risk, a reflection of improved consistency in results from recent studies.

Seasonal and regional analysis of $PM_{2.5}$ associated daily mortality indicated risk was highest in the spring and generally in the Eastern U.S., although $PM_{2.5}$ risk estimates are sensitive to varying designation methods (i.e., climate-based vs. solely spatial (add ref)). A key limitation of the multi-city U.S. based studies is the absence of evidence on gaseous co-pollutants potentially influencing the association between short-term exposure to $PM_{2.5}$ and mortality. However, the additional information on regional and season influences on the association between short-term exposure to $PM_{2.5}$ and mortality is a key piece of new evidence in the current review. Taken together, the epidemiological evidence provides support for a ***likely causal*** relation between short term exposure to $PM_{2.5}$ and mortality (US EPA, 2009a, sections 2.3.1.1 and 6.5.3.2).

3.3.2.2 Effects Associated with Long-term $PM_{2.5}$ Exposures

This section includes an overview of the second draft ISA's findings on effects associated with long-term $PM_{2.5}$ exposures with emphasis on extended follow-ups of long-term epidemiological cohort studies that were important in the previous PM NAAQS review as well as several new multi-city studies conducted in the U.S. and Europe. In the last review, the new evidence available included an extensive reanalysis of data from the Six Cities and ACS studies, new analyses using updated data from the ACS and California Seventh Day Adventist (AHSMOG) studies, and a new analysis using data from a cohort of veterans. In addition, new studies had been published on the association between long-term exposure to fine particles and respiratory morbidity using data from a cohort of schoolchildren in Southern California. Epidemiologic and toxicological studies in the current review provide evidence on the adverse effects of long-term exposure to $PM_{2.5}$ similar to effects observed in short-term exposure studies, especially for respiratory and cardiovascular morbidity and mortality. This information is presented and discussed in Chapter 7 of the second draft ISA. Figure 2-2 of the second draft ISA provides a summary of U.S. and Canadian studies examining the association between long-term exposure to $PM_{2.5}$ and cardiovascular and respiratory morbidity and mortality as well as all-cause mortality. In addition, new evidence, although limited, is available on developmental and reproductive effects including low birth weight and infant.

3.3.2.2.1 Cardiovascular and Systemic Effects

Studies examining associations between long-term exposure to ambient PM (over months to years) and cardiovascular disease (CVD) morbidity had not been conducted and thus were not included in the previous science assessments (U.S. EPA, 2004; US EPA, 1996). A number of studies considered in the last review evaluated the effect of long-term $PM_{2.5}$ exposure on cardiovascular mortality and found consistent associations. One subchronic study evaluated atherosclerosis progression in hyperlipidemic rabbits (Suwa et al., 2002) providing the foundation for subsequent work considered in this review. .

Several new epidemiologic studies have examined the long-term PM-CVD association in multi-city studies conducted in the U.S. and Europe. New epidemiologic and toxicological studies have provided evidence of the adverse effects of long-term exposure to $PM_{2.5}$ on cardiovascular outcomes, including atherosclerosis, clinical and subclinical markers of cardiovascular morbidity, and cardiovascular mortality. Some of the strongest evidence of CVD health effects related to long-term exposure to $PM_{2.5}$ has been in found in recent studies investigating cardiovascular mortality. As discussed in section 3.3.2.2.x below, this evidence includes extended follow-up to the ACS and Harvard Six Cities studies as well as new information from the Women's Health Initiative (WHI) cohort study and Seventh-Day Adventist

cohort (AHSMOG) which reported a positive association with coronary heart disease mortality in females but not males (Chen et al., 2005).

With respect to cardiovascular morbidity, a number of endpoints have been evaluated ranging from subtle indicators of cardiovascular health to serious clinical events associated with coronary heart disease (CHD) and cerebrovascular discease (CVD) including myocardial infarction (MI), coronary artery revascularization (e.g., bypass graft, angioplasty, stent, atherectomy), congestive heart failure, and stroke, This information is presented and discussed in section 7,2 of the second draft ISA and summarized below. Taken together, the second draft ISA concludes that the evidence from epidemiologic and toxicological studies is sufficient to conclude that a ***causal*** relationship exists between long-term exposures to $PM_{2.5}$ and cardiovascular effects (US EPA, 2009a, section 2.3.1.2)

Atherosclerosis. Atherosclerosis is a progressive disease that contributes to several adverse outcomes, including myocardial infarction and aortic aneurysm. Although no study has examined the association between long-term PM exposure and longitudinal change in subclinical markers of atherosclerosis, several cross sectional studies have been conducted. Markers of atherosclerosis used in these studies include coronary artery calcium (CAC), carotid intima-media thickness (CIMT), ankle-brachial index (ABI), and abdominal aortic calcium (AAC). These measures are described briefly below and in more detail in Section 7.2.1.1 of the ISA (EPAa, 2009).

CAC is a measure of atherosclerosis of the coronary arteries in the heart. The prevalence of CAC is strongly related to age. Few people have detectable CAC in their second decade of life but the prevalence of CAC rises to approximately 100% by age 80 (Ardehali et al., 2007). Previous studies suggest that while the absence of CAC does not rule out atherosclerosis, it does imply a very low likelihood of significant arterial obstruction. Conversely, the presence of CAC confirms the existence of atherosclerotic plaque and the amount of calcification varies directly with the likelihood of obstructive disease. CAC is a quantified using the Agatston method (Agatston et al., 1990), and the Agatston scores are frequently used to classify individuals into one of five groups (zero; mild; moderate; severe; extensive) or according to age- and sex-specific percentiles of the CAC distribution (Erbel et al., 2007).

CIMT is a measure of atherosclerosis assessed by ultrasonography of the carotid arteries in the neck, the walls of which have inner (intimal), middle (medial) and outer (adventitial). CIMT estimates the distance in mm or μm between the innermost (blood-intima) and outermost (media-adventitia) interfaces. CIMT has been associated with atherosclerosis risk factors, prevalent coronary heart disease, and incident coronary and cerebral events. Several studies have indicated that CIMT measurements are accurate and reproducible, especially for the common carotid artery (EPA 2009a, Section 7.2.1.1).

ABI, which is also known as the ankle-arm or resting (blood) pressure index, is a measure of lower extremity arterial occlusive disease commonly caused by advanced atherosclerosis. ABI is defined as the unitless ratio of ankle to brachial systolic blood pressures measured in mmHg. As ankle pressure is normally equal to or slightly higher than arm pressure (resulting in an ABI $\geq$ 1.0), epidemiologic studies typically define the normal ABI range as 0.90 to 1.50 (Resnick et al., 2004). Low ABI has been associated with all-cause and CVD mortality, as well as myocardial infarction and stroke (EPA 2009a, Section 7.2.1.1)

AAC is a measure of atherosclerosis of the abdominal aorta. It is scored much like CAC (Agatston et al., 1990), but the age-specific prevalence and extent of AAC is greater, particularly among women and at ages >50 years. Although AAC has not been studied as extensively as CAC, it is associated with carotid and coronary atherosclerosis as well as cardiovascular morbidity and mortality and measurements are sufficiently reproducible to allow serial investigations over time (EPA 2009a, Section 7.2.1.1).

The effect of long-term $PM_{2.5}$ exposure on pre-clinical measures of atherosclerosis (CIMT, CAC, AAC or ABI) has been studied in several populations using a cross-sectional study design. The magnitude of the $PM_{2.5}$ effects and their consistency across different measures of atherosclerosis in these studies varies widely, and they may be limited in their ability to discern small changes in these measures. Kunzli et al. (2005) observed a non-significant 4.2% increase in CIMT associated with long-term $PM_{2.5}$ exposure among participants of a clinical trial in greater Los Angeles, which was several fold higher than the 0.5% increase observed by Diez-Roux et al. (2008) in their analyses of MESA baseline data. The associations in MESA of CAC and ABI with long-term $PM_{2.5}$ exposure were largely null (Diez Roux et al., 2008), while an increase in AAC with long-term $PM_{2.5}$ exposure was reported (Chang et al., 2008). By contrast, a 43% increase in CAC was associated with long-term $PM_{2.5}$ exposure in a German study but no similar association with ABI was observed (Hoffmann et al., 2009). Although the number of studies examining these relationships is limited, effect modification by use of hyperlipidemics and smoking status was reported in more than one study of long-term PM exposure.

Evidence of enhanced atherosclerosis development was demonstrated in new toxicological studies that demonstrate increased plaque and lesion areas, lipid deposition, and TF in aortas of $ApoE^{-/-}$ mice exposed to CAPs (Section 7.2.1.2). However, $PM_{2.5}$ CAPs derived from traffic in Los Angeles did not affect plaque size (Araujo et al., 2008).

Venous thromboembolism. One epidemiologic study examined the relationship between long term PM_{10} concentration, venous thromboembolism, and laboratory measures of hemostasis (prothrombin and activated partial thomboplastin times [PT; PTT]). PT and PTT measure the extrinsic and intrinsic blood coagulation pathways, the former activated in response to blood vessel injury, the latter, key to subsequent amplification of the coagulation cascade and

propagation of thrombus. Decreases in PT and PTT are consistent with a hypercoagulable, prothrombotic state (EPA 2009a, Section 7.2.1.1). Baccarelli et al. (2007) found decreases in standardized correlation coefficients for PT as well as for PTT among cases and controls per 10 $\mu g/m^3$ increase in PM_{10}.

Systemic inflammation, immune function, and blood coagulation. Only two cross-sectional analyses of PM_{10} concentration and markers of immune function or inflammation have been conducted with significant changes observed in the NHANES population (stronger effects among those with metabolic disorders) but not in a relative large survey of adults, which was conducted in England. Ecological analyses comparing children in high versus low pollution regions in Mexico show differences in unadjusted blood markers that may be related to PM concentration or other unmeasured risk factors that differs across the communities studied. The few toxicological studies included do not indicate robust systemic inflammation or coagulation responses in F344 rats following 6-month exposures to diesel, HWS, or gasoline exhaust. The limited effects that were observed could possibly be due to the varying gas concentrations in the exposure mixtures.

Renal and vascular function. There is limited evidence for the effects of $PM_{2.5}$ on renal or vascular function. Cross-sectional and longitudinal epidemiologic analyses of $PM_{2.5}$ and UACR revealed no evidence of an effect (O'Neill et al., 2007) while small non-statistically significant increases in BP with 30- and 60-day avg $PM_{2.5}$ concentrations were reported (Auchincloss et al., 2008). A toxicological study did not show changes in MAP with CAPs, but indicated a CAPs-related potentiation of experimentally-induced hypertension (Sun et al., 2008). In addition, CAPs has induced changes in insulin resistance, visceral adiposity, and inflammation in a diet-induced obesity mouse model (Sun et al., 2009), indicating that diabetics may be a potentially susceptible population to PM exposure.

Clinical outcomes in epidemiologic studies. Several epidemiologic studies of U.S. and European populations have examined associations between long-term PM exposures and clinical CVD events.

Coronary Heart Disease (CHD). Epidemiologic studies examining the association of coronary heart disease (CHD) with long-term PM exposure are discussed below. Cases of CHD were variably defined in these studies to include history of angina pectoris, MI, coronary artery revascularization (bypass graft; angioplasty; stent; atherectomy), and congestive heart failure (CHF).

Miller et al. (2007) studied incident, validated MI, revascularization, and CHD death, both separately and collectively, among 58,610 post-menopausal female residents of 36 U.S. metropolitan areas enrolled in the Women's Health Initiative Observational Study (WHI OS, 1994-1998). In this prospective cohort study of participants free of CVD at baseline (median

duration of follow-up = 6 yr), the authors used arithmetic averaging of year 2000 EPA AQS $PM_{2.5}$ data available at the monitor nearest to each participant's geocoded U.S. Postal Service five-digit ZIP code centroid to estimate one-yr average exposures. They found 6%, 20% and 21% increases in the overall risk of MI, revascularization, and their combination with CHD death per 10 $\mu g/m^3$ increase in $PM_{2.5}$, respectively. Hazards were higher within than between cities and in the obese. For the combined CVD outcome (MI, revascularization, stroke, CHD death, cerebrovascular disease), authors reported a 24% increase in risk that was higher among participants at higher than lower quintiles of body mass index, waist-to-hip ratio, and waist circumference. The $PM_{2.5}$-CVD association was stronger among non-diabetic than diabetic participants.

Puett et al. (2008) studied incident, validated CHD, CHD death, and non-fatal MI among 66,250 female residents of metropolitan statistical areas in thirteen northeastern U.S. states who were enrolled in the Nurses' Health Study (NHS, 1992-2002). In this prospective cohort study of women without a history of non-fatal MI at baseline (maximum duration of follow-up = 4 years), the authors used two-stage, spatially smoothed, land use regression to estimate residence-specific, 1-year moving average PM_{10} exposures from U.S. EPA AQS and emissions, IMPROVE, and Harvard University monitor data. They found a 10% increase in risk of first CHD event per 10 μ/m^3 increase in 1-year average PM_{10} exposure, while the association with MI was close to the null value.

Rosenlund et al. (2006) studied 2,938 residents of Stockholm County, Sweden. In this case-control study of 1,085 patients with their first, validated non-fatal MI and an age-, gender- and catchment-stratified random sample of 1,853 controls without MI (1992-1994), the authors used street canyon-adjusted dispersion modeling of emissions data to estimate 30-year average exposure to PM_{10} (median = 2.4 $\mu g/m^3$). They found that the OR for prevalent MI per 10 $\mu g/m^3$ increase in PM_{10} was 0.85. In a more recent study, Rosenlund et al. (2009) evaluated 554,340 residents of Stockholm County, Sweden (1984-1996). In this population-based, case-control study of 43,275 cases of incident, validated MI, the authors used dispersion modeling of traffic emissions and land use data to estimate 5-year average exposure to PM_{10}. They found that after adjustment for demographic, temporal, and socioeconomic characteristics, the OR for MI per 5 $\mu g/m^3$ increase in PM_{10} was 1.04. ORs were higher after restriction to fatal cases, in- or out-of-hospital deaths, and participants who did not move between population censuses. Authors state that control for confounding was superior in their previous study (Rosenlund et al., 2006) although the size of the population was larger in this recent study (Rosenlund et al., 2009).

Zanobetti and Schwartz (2007) studied ICD-coded recurrent MI and post-infarction CHF among 196,131 Medicare recipients discharged alive following MI hospitalization in 21 cities from 12 U.S. states (1985-1999). In this ecologic, open cohort study of re-hospitalization among

MI survivors (mean duration of follow-up = 3.6 and 3.7 years for MI and CHF, respectively), the authors used arithmetic averaging of EPA AQS PM_{10} data available in the county of hospitalization to estimate one-year average exposures. They found 17% and 11% increases in the risk of recurrent MI and post-infarction CHF, respectively, per 10 $\mu g/m^3$ increase in PM_{10} exposure. Hazards were somewhat higher among persons aged >75 years.

Hoffmann et al. (2006) studied self-reported CHD (MI or revascularization) among 3,399 residents of two cities in Germany at the baseline exam of the Heinz Nixdorf Recall Study (2000-2003) discussed above. In this cross-sectional ancillary study, the authors used dispersion modeling of emissions, climate and topography data to estimate 1-year average exposure to $PM_{2.5}$ (mean = 23.3 $\mu g/m^3$). They found little evidence of an association between $PM_{2.5}$ and CHD in these data.

In the study of 1030 census enumeration districts in Sheffield, U.K. discussed above, Maheswaran et al. (2005) studied 11,407 ICD-10-coded emergency hospitalizations for CHD among 199,682 residents. In this ecologic study, the authors used dispersion modeling of emissions and climate data to estimate 5-year average exposure to PM_{10}. They found that after adjusting for smoking prevalence, controlling for socioeconomic factors, and smoothing, the age- and gender-standardized rate ratios for CHD admission were 1.01, 1.04, 0.97, and 1.07 across PM_{10} quintiles.

The study of post-menopausal women enrolled in the WHI OS by Miller et al. (2007) was the only U.S. study to examine the effect of $PM_{2.5}$ rather than PM_{10}. This study, which provides strong evidence of an association, was distinguished by its prospective cohort design, validation of incident cases and large population. Puett et al. (2008), the other U.S. study with comparable design features, provides evidence of an association of incident CHD with long-term PM_{10} exposure. Findings from Swedish case control studies of incident validated cases of MI were not consistent. A cross-sectional study of self-reported CHD did not provide evidence of an association with $PM_{2.5,}$ while findings from two ecologic studies of PM_{10} indicated positive associations of CHD hospitalizations with PM_{10} (Maheswaran et al., 2005; Zanobetti and Schwartz, 2007).

Stroke. Miller et al. (2007) found 28% and 35% increases in the overall risk of validated stroke and cerebrovascular disease, respectively, per 10 $\mu g/m^3$ increase in one-year average $PM_{2.5}$ exposure. Risks were higher within than between cities. In the study of 1030 Census of enumeration districts in Sheffield, U.K. described previously, Maheswaran et al. (2005) studied 5,122 ICD-10-coded emergency hospital admissions for stroke among approximately 200,000 residents of 1,030 census enumeration districts in Sheffield, U.K. (1994-1999). In this ecologic study, the authors used dispersion modeling of emissions and climate data to estimate five-

year average exposure to PM_{10}. They found that the age- and gender-standardized rate ratios for stroke admission were 1.05, 1.07, 1.06, and 1.15 across PM_{10} quintiles.

These studies examining the long-term PM-stroke relationship provide evidence of association. Maheswaran et al. (2005) examined emergency room HAs in Sheffield, U.K. using an ecologic design while results reported by Miller et al. (2007) are based on the prospective cohort study of the WHI OS population.

Peripheral Arterial Disease. The German Heinz Nixdorf Recall cross-sectional study described above (Hoffmann et al., 2009) also evaluated the association between one-year average exposure to $PM_{2.5}$ and peripheral arterial disease (self-reported history of a surgical or procedural intervention or an ABI <0.9 in one or both legs). The authors found no evidence of an increase in risk. However, evidence of an association with traffic exposure was present in these data. ORs were higher among participants with CAC scores ≤ 75th percentile, women, and smokers.

Deep Vein Thrombosis. The Italian case-control study discussed above also examined the chronic PM_{10}-DVT association (Baccarelli et al., 2008). The authors found a 70% increase in the odds of deep vein thrombosis (DVT) per 10 $\mu g/m^3$ increase in one-year average PM_{10} exposure. This finding was consistent with the decreases in PT and PTT also observed among controls in this context as well as the 47% increase in the odds of DVT per inter-decile range (242 meters) increase in the residence-to-major-roadway distance observed among a subset of cases and controls (Baccarelli et al., 2009). The PM_{10}-DVT and distance-DVT associations were both weaker among women and among users of oral contraceptives or hormone therapy.

3.3.2.2.2 Respiratory Effects

The prior review observed long-term exposure to $PM_{2.5}$ was associated with increased prevalence of respiratory disease (e.g. asthma, bronchitis, chronic cough) and lung function. The evidence from the prior review suggested children were an at-risk population, with the US-based Southern California Communities' Children's Health Study (CHS) reporting decreases in lung function growth associated with exposure to $PM_{2.5}$ over four-year follow-up periods (Gauderman et al., 2002). The associations were statistically significant for some measures of lung function among one cohort of 4^{th} graders, whereas in a second cohort of 4^{th} graders the associations generally did not achieve statistical significance. Similarly, among a group of older children a positive but statistically non-significant association was detected between long-term $PM_{2.5}$ exposure and a decrease in lung function growth (Gauderman et al., 2000).

A prospective study conducted among CHS participants that focused on participants moving to other locations during the study period, investigators reported that those participants moving into areas of lower PM_{10} exhibited an increase in lung function growth in comparison to children moving to areas with higher PM_{10} concentrations (Avol et et al., 2001). Another study

conducted among participants of the CHS, but with $PM_{2.5}$ levels, reported an association between long-term exposure to $PM_{2.5}$ and bronchitis symptoms (McConnell et al., 2003). However, this study also observed that the single-pollutant effect of $PM_{2.5}$ markedly decreased with adjustment for NO_2 and OC, suggesting other traffic-related pollutants may also be etiologically relevant for the development of bronchitic symptoms. These findings were similarly observed in studies of long-term exposure to PM_{10} in areas where the fine fraction predominates (US EPA, 2009a, section 7.3.1.1). These findings are consistent with the cross-sectional results reported from the 24-cities study (Dockery et al., 1996; Raizenne et al., 1996) which reported associations between increased bronchitis rates and decreased peak flow with fine particle sulfate and fine particle acidity. However, as noted in the study by McConnell et al. (1999), the high degree of correlation between PM_{10} and co-pollutants obscures the assessment of an independent effect of PM. In the last review, the 2004 AQCD "concluded that the evidence for an association between long-term exposure to PM and respiratory effects may be confounded by other pollutants" (US EPA, 2009a, section 7.3).

Several new studies analyzing the CHS report positive associations between long-term exposure to $PM_{2.5}$ and respiratory morbidity, particularly for such endpoints as lung function growth, respiratory symptoms, and disease incidence (US EPA, 2009a, section 7.3.9.1). Recent epidemiological evidence building upon earlier findings from the CHS is generally supportive of a longitudinal association between $PM_{2.5}$ and pulmonary function that is observed during the lung development period among children with effects and extending into adulthood. A prospective study utilizing the CHS cohort reported that among children between the ages of 10 and 18 years, $PM_{2.5}$ was significantly associated with deficits in FEV_1 attained at the age of 18 years (Gauderman et al., 2004). Furthermore, the 8 year time period evaluated provides evidence for "the persistence of effect, but in addition the strength and robustness of the outcomes were more positive, larger, and more certain than previous CHS studies of shorter follow-up" (section 7.3.2.1, US EPA, 2009a). These results are consistent with positive findings from studies of PM_{10} exposure that were conducted in Mexico (Rojas-Martinez et al., 2007) and Sweden (Nordling et al., 2008), and a national cystic fibrosis cohort in the US (Goss et al., 2004). In addition, investigators of the CHS suggest $PM_{2.5}$ may also act as a modifier of the association between lung function with asthma, inducing declines in lung function and a concomitant increase in new onset asthma (Islam et al 2007). Furthermore, the second draft ISA concludes that:

> The CHS (McConnell et al., 2003) provides evidence in a prospective longitudinal cohort study that relates $PM_{2.5}$ and bronchitic symptoms and reports larger associations for within-community effects that are less subject to confounding than between-community effects. Several new studies report similar findings with long-term exposure to PM_{10} in areas where fine particles predominate PM_{10}. In England, in a cohort of 4,400 children

(aged 1-5), an association is seen with an increased prevalence of cough without a cold. Further evidence includes a reduction or respiratory symptoms corresponding to decreasing PM levels in "natural experiments" in both a cohort of Swiss school children (Bayer-Oglesby et al., 2005) and adults (Schindler et al., 2009) (US EPA, 2009a, section 7.3.1.1).

Overall, the second draft ISA notes that study results for respiratory symptoms and lung function changes associated with $PM_{2.5}$ exposure were consistent across different study designs, multiple measures of lung function, and varying locations and researchers. In addition, recent evidence is supportive of an association between long-term exposure to $PM_{2.5}$ and allergy and pulmonary inflammation (US EPA, 2009a, sections 7.3.3, 7.3.4, 7.3.5, 7.3.6, 7.3.7). However, the evidence for respiratory symptoms and lung function remain stronger as studies of allergic indicators are inconsistent, and comparatively few studies assess long-term $PM_{2.5}$ exposure in association with pulmonary inflammation.

The prior review concluded the evidence for the association between long-term exposure to $PM_{2.5}$ and respiratory morbidity may be confounded by other pollutants. However, the second draft ISA notes consistent epidemiological evidence in concurrence with toxicological evidence providing a biological mechanism supports an independent effect of long-term exposure to $PM_{2.5}$ in association with respiratory morbidity, with children at particular risk (add ref).

3.3.2.2.3 Reproductive, Developmental, Prenatal, and Neonatal Outcomes

In the prior review, the limited evidence available for PM associated developmental effects suggested exposure to PM_{10} during early (i.e. first month) and late (i.e. 6 weeks prior to birth) pregnancy was associated with a higher risk of pre-term birth, and exposure to $PM_{2.5}$ during the first month of pregnancy was associated with intrauterine growth restriction. However, an association was not detected between PM_{10} exposure and low birth weight. In contrast, the current review includes new studies reporting a significant association between exposure to $PM_{2.5}$ during pregnancy and lower birth weight, pre-term birth, and intrauterine growth restriction, respectively, and post-natal exposure to $PM_{2.5}$ associated with an increased risk of infant mortality. Although a multi-city US based study did not observe an association between quarterly estimates of exposure to $PM_{2.5}$ and lower birth weight (Parker and Woodruff, 2008) other US based studies report an association between $PM_{2.5}$ and lower birth weight (Bell et al., 2007; Parker et al., 2005) with higher effect estimates reported for $PM_{2.5}$ in comparison to PM_{10} (Bell et al., 2007). The influence of region variability (i.e., fine particles were a stronger predictor of lower birth weight in the Northeast) may have obscured an association in the multi-city US based study.

Among the few studies conducted in the US since the last review, investigators reported statistically significant associations between exposure to $PM_{2.5}$ at 1st trimester, 1st month, 2 and 6

weeks prior to birth, and throughout pregnancy with pre-term birth (Wilhem and Ritz, 2005; Ritz et al., 2007; Huynh et al., 2005). In addition, studies conducted outside the US support an association between exposure to $PM_{2.5}$ and greater risk of pre-term birth (Brauer et al., 2008; Jalaludin et al., 2007). Since the last review, two U.S. based studies have examined intrauterine growth (e.g.., small for gestational age, intrauterine growth retardation), an earlier indicator of fetal development compared to other birth outcomes (e.g., pre-term birth, low birth weight) in relation to $PM_{2.5}$ exposure. These studies reported an statistically significant increase in risk of small for gestational age associated with exposure to $PM_{2.5}$ during the first, second, and third trimester (Rich et al., 2009; Parker et al., 2005). Furthermore, two Canadian studies reports similar findings of a significant association between exposure to PM2.5 throughout pregnancy and greater risk of small for gestational age (Brauer et al., 2008) and intrauterine growth retardation (Liu et al., 2007), respectively, although the effect diminished with adjustment for co-pollutants in the latter study of intrauterine growth retardation.

At the acute end of the birth outcomes spectrum, the current review indicates evidence of an association between post-natal exposure to $PM_{2.5}$ and infant mortality is limited, particularly for studies assessing $PM_{2.5}$ exposure, and with mixed findings observed across differential ages (i.e. less than 1 month, 1 month to 1 year, less than 1 year) and for cause-specific deaths (i.e., respiratory mortality, sudden infant death, all-cause). Two U.S. based studies conducted since the last review report exposure to $PM_{2.5}$ over the first two months or entire lifetime of the infant is significantly associated with increased risk of respiratory-cause mortality (Woodruff et al., 2006; Woodruff et al., 2008). Moreover, although statistically non-significant, the authors report a positive association between exposure to $PM_{2.5}$ and increased risk of all-cause mortality. The results for $PM_{2.5}$ associated risk of sudden infant death are inconsistent, potentially due to the changes in diagnosis between 1999 and 2001.

There remain significant limitations in the evidence assessing the relation between $PM_{2.5}$ exposure and birth outcomes (US EPA, 2009a, section 7.4). Specifically, characterizing $PM_{2.5}$ exposure at the etiologically relevant time period for developmental effects, evaluating the influence of co-pollutants (refer to section 3.4.6.1), exposure misclassification (refer to section 3.4.4), accounting for differences in fetal developmental stage in association with PM exposure prior to delivery between cases and controls in studies of pre-term birth, limitations in the toxicological data available to understand the biological mechanisms underpinning these relations, and fewer studies in comparison to the evidence available for other endpoints (e.g., cardiovascular/respiratory morbidity) remain important challenges. Despite these limitations recent studies included in the current review are suggestive of an association between $PM_{2.5}$ exposure and birth outcomes.

3.3.2.2.4 Cancer Incidence, Mutagenicity, and Genotoxicity

The previous review concluded that long-term exposure to PM was associated with an increase in the risk of lung cancer incidence and mortality based on evidence from three prospective studies (i.e. ACS, Six Cities, AHSMOG) as well as results from case-control and ecological studies (US EPA, 2004, p. 8-318; Beeson et al., 1998; Dockery et al., 1993; Pope et al., 2002). Furthermore, the EPA concluded in 2002 that diesel exhaust, a source of PM in the ambient air is a likely human carcinogen (U.S. EPA, 2002). Toxicological evidence of mutagenic or genotoxic potential from exposures to PM presented in the prior review provide support for a biological mechanism underpinning this relation (US EPA, 2004, section 7.10.1).

The epidemiological evidence presented in the current ISA is largely an extended analyses of the prospective cohort studies included in the prior review, primarily focusing on the association between long term exposure to PM and lung cancer incidence and mortality. A recent re-analysis of the ACS cohort reported a statistically significant association between $PM_{2.5}$ and lung cancer mortality (Krewski, 2009). Similarly, the extended follow-up of the Six Cities Study yielded a significantly higher risk of lung cancer mortality in association with increasing levels of $PM_{2.5}$ (Laden et al., 2006). A ten-year follow-up study conducted among all participants of the Netherlands cohort study (NLCS) observed a positive non-significant association between $PM_{2.5}$ and lung cancer mortality among (Brunekreef et al., 2009). However, in the same study investigators also observed a negative association between $PM_{2.5}$ and lung cancer mortality in a case-cohort analysis, and observed a negative association between $PM_{2.5}$ and lung cancer incidence among the full cohort and case-cohort.

In addition to the inconsistencies in evidence between lung cancer mortality and incidence from prospective cohort studies, the current review also reports differing findings for the relation between PM and biomarkers of susceptibility (e.g., chromosomal aberrations, oxidative stress-induced DNA damage, frequency of micronuclei). However, recent in vitro studies confirm findings from the prior review that PM originating from urban and combustion-derived sources is mutagenic and genotoxic, and that fine particles may be more mutagenic and genotoxic than larger particles. The results of these in vitro studies provide supportive evidence for biological plausibility of an association between PM and lung cancer, however, two in vivo inhalation toxicological studies evaluating carcinogenic endpoints reported negative results (US EPA, 2009a, section 7.5.2.3). Initial epigenetic investigations also provide evidence that long-term exposure to $PM_{2.5}$ may affect DNA methylation content, but further epigenetic research is required to elucidate whether long term exposure to $PM_{2.5}$ results in heritable DNA mutations. Overall, current evidence is suggestive of an association between long term exposure to $PM_{2.5}$ and cancer, although limitations in the evidence include the carcinogenicity of the fine PM fraction in comparison to ultrafine particles (< 2.5μm) in diesel exhaust and studies utilizing

PM_{10}, and inconsistencies in studies assessing the relation between $PM_{2.5}$ and lung cancer incidence and mortality, respectively.

3.3.2.2.5 Mortality

The 2004 CD reported evidence from four prospective studies (i.e., Six Cities Study, ACS Study, the Veterans cohort, and AHSMOG cohort) (section 8.2.3, US EPA, 2004). In particular, the Health Effects Institute reanalysis of ACS and Six Cities Studies provided the strongest evidence of a statistically significant association between chronic exposure to $PM_{2.5}$ and mortality (Krewski et al., 2000). Furthermore, an extended analysis for the ACS cohort included in the prior review reported significant associations between long-term exposure to fine particles (using various averaging periods for air quality concentrations) and mortality from all causes, cardiopulmonary diseases, and lung cancer, respectively (Pope et al., 2002; CD p. 8-102, US EPA, 2004). Overall, the prior review reported a 10 $\mu g/m^3$ increase in $PM_{2.5}$ levels was significantly associated with increased risk of death ranging from 6 to 13% and 6 to 19%, for all causes and cardiopulmonary causes, respectively, and 13% for lung cancer mortality.

New studies discussed in the current ISA include an extended analysis with updated data of the Six Cities and ACS cohorts, further analysis of the AHSMOG and Veterans study cohorts, and a new analysis among participants of the Cystic Fibrosis and WHI cohort. In an extended analysis of the ACS cohort utilizing new details on cardiovascular and respiratory cause mortality, investigators reported a positive association between $PM_{2.5}$ and specific cardiovascular diseases (i.e., IHD, dysrhythmia, heart failure, cardiac arrest). However, a null association was reported between $PM_{2.5}$ and respiratory mortality (Pope et al., 2004).

Another new analysis of the ACS cohort extended the follow-up period to 18 years, reporting parameter estimates generally higher but not statistically different than earlier ACS effect estimates (Krewski 2009; section 7.6.1 2nd draft ISA – USA EPA, 2009a). Krewski et al. employed a Cox proportional-hazards regression model incorporating multiple levels of information to adjust for the similarity of observations among people living in the same neighborhoods and cities, and for social and economic confounders at two spatial levels (i.e. zip code and city). This reanalysis also assessed the robustness of the model results to alternative forms (i.e. log-log) compared to the linear function for the PM-mortality relation. The investigators reported that as observed in the reanalysis of the original ACS study (Krewski et al., 2000), education level was found to be an effect modifier, with $PM_{2.5}$ associated mortality from all causes excluding IHD higher for those with lower education levels. These results are consistent with the prior review noting educational attainment may serve as proxy for lower socioeconomic status and thus greater vulnerability to $PM_{2.5}$-associated effects (AQCD - US EPA 2004, p. 8-94). The authors found that adjustment for individual-level variables did not

alter the results for the association between long-term $PM_{2.5}$ exposure and mortality, and addressing spatial patterns in the data generally increased the size of the association between $PM_{2.5}$ and mortality (Krewski et al., 2009). In consideration of the results of the sensitivity analyses, the authors report that the reanalysis are consistent with earlier findings of an association between long term exposure to $PM_{2.5}$ and mortality.

The extended analyses for the ACS cohort study included additional follow-up health data for 1990-1998 and $PM_{2.5}$ levels for 1985-1998 were estimated from visibility data (Laden et al., 2006). The investigators reported statistically significant associations between long-term exposure to $PM_{2.5}$ and increased risk of mortality over two distinct time periods. Of particular note is the statistically significant 27% reduction in mortality risk from cardiovascular and respiratory causes associated with a decline in long-term $PM_{2.5}$ levels over the follow-up period. Although the use of the estimated $PM_{2.5}$ levels in place of observed measurements introduces uncertainty in the derivation of the risk estimates, the investigators note the high correlation (0.93) between estimated and measured $PM_{2.5}$ levels during years when both were available (Laden et al., 2006), and these results are consistent with the findings from the original analysis of a significant association between long term exposure to $PM_{2.5}$ and increased risk of mortality (Dockery et al., 1993).

There are new analyses among participants of the AHSMOG and WHI cohort that provide evidence of a statistically significant association between long term exposure to $PM_{2.5}$ and increased risk of mortality among women. Positive, statistically significant associations were reported for coronary heart disease mortality among females participants of the AHSMOG cohort, but not among men, suggesting women may be at greater risk for acute PM health effects in comparison to men (Chen et al., 2005). Furthermore, among participants of the WHI, investigators observed long term exposure to $PM_{2.5}$ was significantly associated with an increased risk of a cardiovascular related mortality (Miller et al., 2007). Effect estimates from the WHI study are higher than those from the ACS cohort, which has eight times the cardiovascular mortality rate as the WHI, suggesting the $PM_{2.5}$ impact may be higher among populations with a lower prevailing risk of cardiovascular death (section 7.6.1 2nd draft ISA – USA EPA, 2009a). These results are consistent with the findings of long- term exposure to $PM_{2.5}$ associated risk of cardiovascular morbidity among WHI participants (add ref), providing support for PM related chronic and acute effects on the cardiovascular system.

Since the last review several new studies were conducted among the elderly. Among participants of the California Cancer Prevention Study, a cohort of elderly individuals in 11 California counties, a statistically significant association was reported between long term exposure to $PM_{2.5}$ and all cause deaths during the time period at which observed $PM_{2.5}$ levels were highest (1973-1982), but a null association was reported for the subsequent time period

when $PM_{2.5}$ levels had declined in counties which previously exhibited the highest $PM_{2.5}$ levels (Enstrom et al., 2005). Among participants of Medicare data-based cohort studies, investigators report statistically significant associations between long term exposure to $PM_{2.5}$ and increases in all-cause mortality for the ACS and Six Cities Study counties, respectively (Eftim et al., 2008). Although effect estimates among the Medicare cohort are higher than the ACS and Six Cities studies, these results are potentially sensitive to the lack of adjustment for individual-level covariate data on potential confounders (e.g. smoking). Another study of Medicare data from 113 counties reported variability in the $PM_{2.5}$ mortality effect estimates at the national and local level from 2000 to 2002, and noted that trends in $PM_{2.5}$ and mortality at the national scale are more likely to be confounded than trends at the local scale (Janes et al., 2007). Overall, the utilization of long-term time trends as a source of exposure variability is problematic due to potential confounding of the effect estimates (Pope and Burnett, 2007).

Zeger et al. (2007) reported that long-term exposure to $PM_{2.5}$ was significantly associated with an increase in mortality among Medicare participants, and stronger associations were observed in eastern counties compared to a national estimate, and no association was observed among Western counties (Zeger et al., 2007). However, effect estimates decreased by 50% with adjustment for spatial confounding. In a subsequent retrospective cohort study (MCAPS), Zeger et al., reported than average 6-year exposure to $PM_{2.5}$ was significantly associated with increased risk of mortality in the eastern and central regions, respectively, and (Zeger et al., 2008)[52]. In addition, the results indicated that risk declined with increasing age, and similar to their earlier study, no associated was observed between $PM_{2.5}$ and mortality in the western region. Moreover, risk estimates were similar to effect estimates reported in the ACS and Six Cities study, provide coherence across prospective studies for an association between long-term exposure to $PM_{2.5}$ and mortality.

In addition, an analysis of $PM_{2.5}$ associated mortality and the influence of traffic density among the Veterans Administration (VA) cohort reported a statistically significant association between $PM_{2.5}$ and mortality in a single-pollutant model. However, with adjustment for co-pollutants the $PM_{2.5}$ effect estimates decreased and was no longer statistically significant (Lipfert et al., 2006). In another investigation of the effects of traffic-related air pollution on mortality among participants of the Netherlands Cohort Study, Brunekreef et al., reported a statistically significant 6% increase for all-cause mortality associated with a 10 g/m3 increase in $PM_{2.5}$ levels, similar to results reported in the ACS (Brunekreef et al., 2009; US EPA, 2009a, section 7.6.1).

[52] The Zeger et al. (2008) analysis included COPD as a proxy for smoking status. The investigators reported the risk estimate for the eastern region declined and the central region increased using this adjustment . This result may possibly be related to bias introduced in using COPD as a proxy for smoking.

Recent studies assessed the potential for an unknown confounder driving the PM-mortality association at the local scale by considering the within-city effects of $PM_{2.5}$ exposure. Two new analyses using ACS considered intra-urban variability in $PM_{2.5}$ in Los Angeles and New York City, respectively (Krewski et al., 2009). In Los Angeles, a refined exposure assessment using interpolation and land use regression methods yielded generally similar results, with a significant association reported between $PM_{2.5}$ and mortality from all causes and cardiopulmonary diseases. In New York City, Krewski et al. (2009) used land use regression and annual average $PM_{2.5}$ levels to assign estimated $PM_{2.5}$ exposures to ACS participants. A positive association was observed between exposure to $PM_{2.5}$ and IHD related mortality, but negative associations were observed between $PM_{2.5}$ exposure and all cause, CPD, and lung cancer mortality. The limited range of average $PM_{2.5}$ concentrations potentially contributed to the limited power to detect an association for these outcomes.

Overall, in the current review, the updated epidemiological evidence from the Six Cities and ACS cohorts, provides support for a ***likely causal*** relation between long-term exposure to $PM_{2.5}$ and mortality (US EPA, 2009a, section 7.6.5.1). For epidemiological studies reporting associations between long-term exposure to $PM_{2.5}$ and increased risk of mortality, the mean $PM_{2.5}$ concentrations in study areas ranged between 13.2 to 29 $\mu g/m^3$, with more precise and consistently positive findings in locations at or above 13.5 $\mu g/m^3$ (US EPA, 2009a, Figure 2-2). Recent ACS analyses indicate mortality effects related to cardiovascular disease**,** as compared to respiratory- related mortality**,** are the predominant cause of mortality in association with $PM_{2.5}$. New study findings from the Six Cities and ACS cohorts review suggest the magnitude of the association between long term exposure to $PM_{2.5}$ and mortality is potentially larger than previously reported (US EPA, 2009a, section 7.6.1). Moreover, updated analyses using the Six Cities study, and a new study examining the relation between life expectancy and $PM_{2.5}$ report a decreased risk of mortality and increase in life expectancy with declining levels of $PM_{2.5}$ (Laden et al., 2006; Pope et al., 2009).

3.3.2.3 $PM_{2.5}$ Components and Sources

One of the major research priorities defined by the National Academy of Sciences National Research Councils (NRC) related to assessing the health effects of PM components and sources (NRC, 2004). In the last review, EPA recognized the availability of a limited number of epidemiologic studies implicating various components within the mix of fine particles (e.g., sulfates, nitrates, carbon, organic compounds, and metals) as being associated with adverse effects (EPA, 2004, section 9.2.2.1.1, Table 9–3) as well as several studies that used $PM_{2.5}$ speciation data to evaluate the association between mortality and particles from different sources (Schwartz, 2003; Mar et al., 2003; Tsai et al., 2000; EPA, 2004, section 8.2.2.5). Overall, EPA

concluded that, "[t]hese 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'' (EPA, 2004, section 9.2.2.1.1). Conversely, scientific evidence available in the last review provided no basis to conclude that any individual fine particle component cannot be associated with adverse health effects (EPA, 2005, p. 5–17). In short, there was not sufficient evidence that would lead EPA to select one or more $PM_{2.5}$ component as being primarily responsible for effects associated with fine particles, nor was there sufficient evidence to suggest that any component should be eliminated from the indicator for fine particles (71 FR 61163, October 17, 2006).

In this review, once again EPA is considering to what extent evidence has become available to inform the understanding of the role and relative public health importance of specific components within the ambient mix of particles. More specifically, we are interested in how the new scientific evidence can inform our understanding of potential differences in toxicity for individual PM components as well as the interactions of these components in the ambient mix. New epidemiological evidence available in this review builds upon ambient measurement data available through the Chemical Speciation Network (CSN) to support initial analyses of fine particles components in addition to available toxicological evidence.

As presented in section 6.6 of the second draft ISA, "[f]rom a mechanistic perspective, it is highly plausible that the chemical composition of PM would be a better predictor of health effects than particle size. This would be consistent with observed regional heterogeneity in PM-related health effects in some epidemiologic studies. Also, data from the CSN demonstrate gradients in a number of $PM_{2.5}$ components, including EC, OC, nitrate, and SO_4^{2-}." Recent epidemiologic, toxicological, and controlled human exposure studies have evaluated the health effects associated with ambient PM constituents and sources, using a variety of quantitative methods applied to a broad set of $PM_{2.5}$ constituents, rather than selecting a few constituents a priori. In this review, EPA has considered approximately 40 new studies evaluating the health effects associated with chemical components and sources of PM, including factors for PM from crustal and soil, traffic, secondary sulfates, power plants, and oil combustion sources (US EPA, 2009a, section 6.6; Table 6-17). There is some evidence for trends and patterns that link particular ambient PM constituents or sources with specific health outcomes, but there is insufficient evidence to determine whether these patterns are consistent or robust.

Overall, EPA continues to find support for general conclusions presented in the last review that the available evidence for particle components "indicate[s] that many constituents of PM can be linked with differing health effects and that the evidence is not yet sufficient to allow differentiation of those constituents or sources that are more closely related to specific health outcomes" (US EPA, 2009a, sections 2.3.2 and 6.6.3.

3.3.2.3.1 Effects Associated with Short-term Exposures to PM2.5 Components and Sources

As discussed in the last review, associations were reported between mortality and short-term exposure to a number of PM components, especially fine particle components (e.g., sulfates, nitrates, metals, organic compounds, elemental carbon). Different patterns of associations between various components or source categories of fine particles and total or cardiovascular mortality were seen in these various studies (US EPA, 2004, section 8.2.2.5.5, Tables 8-3, 8-4). Three studies evaluated in the last review 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 (Schwartz, 2003; Mar et al., 2003; and Tsai et al., 2000). 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 (US EPA, 2004, section 8.2.2.5.3). The EPA concluded that these studies indicated that exposure to fine particles from combustion sources, but not crustal material, was associated with mortality (US EPA, 2004, p. 8-85; US EPA, 2005, p. 3-16).

Recent epidemiologic, toxicological, and controlled human exposure studies have evaluated the health effects associated with ambient PM constituents and sources, using a variety of quantitative methods evaluating a range of PM constituents. Evidence presented in the second draft ISA indicate numerous ambient $PM_{2.5}$ source categories have been associated with health effects, including factors for PM from crustal and soil, traffic, secondary SO_4^{2-}, power plants, and oil combustion sources (US EPA, 2009a, Table 6-17). There is some evidence for trends and patterns that link particular ambient PM components or sources with specific health outcomes, but "there is insufficient evidence to determine whether these patterns are consistent or robust" (US EPA, 2009a, section 6.6.3).

As outlined in the second draft ISA, evaluating a potentially large number of ambient PM constituents with a large number of health outcomes "presents difficulties that are related to both the nature of PM, and the methods of quantitative analysis. First, the number of constituents that comprise PM is not only large, but the correlations between them are inherently high. Reducing the correlation between constituents has been accomplished in most of the recent studies through various forms of factor analysis, which limits the correlations between constituents by grouping the most highly correlated ambient PM constituents into less correlated groups or factors. Some studies identify the resulting groups or factors with named sources of ambient PM, but many do not draw explicit links between factors and actual sources" (US EPA, 2009a, section 6.6). The methods for estimating source contributions to ambient PM are reviewed in Section 3.5.4.

Most epidemiologic, toxicological, and controlled human exposure studies considered between 7 and 20 ambient $PM_{2.5}$ constituents, with EC, OC, SO_4^{2-}, and NO_3 most commonly measured. Many of the studies reduced the number of ambient PM constituents by grouping them with various factorization or source apportionment techniques to examine the relationship between the grouped PM constituents and various health effects. However, not all studies labeled the constituent groupings according to their presumed source and a small number of controlled human exposure and toxicological studies did not apply any kind of grouping to the ambient PM speciation data. In addition, there were differences in the type of PM constituent data used in the various studies (US EPA, 2009a, section 6.6.2.1).

In epidemiologic studies, ambient $PM_{2.5}$ speciation data were obtained from atmospheric monitors; while all of the controlled human exposure and the majority of the toxicological studies used CAPs. According to the second draft ISA, there are important limitations in interpreting the body of scientific evidence for PM components largely because "few, if any of the results are easily comparable, due to: differences in the sets of ambient PM constituents that make up each of the factors[53]; the subjectivity involved in labeling factors as sources; the numerous potential health effects examined in these studies, including definitive outcomes (e.g., HAs) as well as physiological alterations (e.g., increased inflammatory response); and the various statistical methods and analytical approaches used in the studies. There are no well-established, objective methods for conducting the various forms of factor analysis and source apportionment, leaving much of the model operation and factor assignment open to judgment by the individual investigator" (US EPA, 2009a, section 6.6.1).

A few recent epidemiological studies have examined the association between mortality and morbidity and components of $PM_{2.5}$. This endeavor has been undertaken by some investigators through the use of data collected for $PM_{2.5}$ components through the Chemical Speciation Network. As outlined in section 2.x, the CSN includes more than 250 monitors across the country collecting over 40 chemical species. A limited number of CSN monitors began collecting ambient data in 2000 with the majority of sites collecting data starting in 2001. One limitation of the current CSN is that the sampling frequencies of the monitors are either every third day or every sixth day, therefore, in general, reducing the statistical power available to examine associations with mortality in time-series studies.[54] As outlined in the second draft ISA, some investigators have circumvented this issue by using the $PM_{2.5}$ chemical species data in

[53] For example, the Al/Si factor identified in one study may differ from the Al/Ca/Fe/Si factor from another study, and the "Resuspended Soil" factor from a third study.

[54] The EPA recognizes that not having daily speciated $PM_{2.5}$ is a limitation for understanding the health effects associated with specific fine particle components. The Agency has been taking steps to address this issue (US EPA, 2008).

a second stage regression[55] to explain the heterogeneity in PM_{10} or $PM_{2.5}$ mortality risk estimates across cities and assuming that the relative contributions of $PM_{2.5}$ have remained the same over time (Bell et al., 2009; Dominici et al., 2007; Franklin et al., 2008; Lippmann et al., 2006). There have also been some studies that directly analyzed speciated $PM_{2.5}$ data (e.g., Klemm et al., 2004; Ostro et al., 2007).

Lippmann et al. (2006) and Dominici et al. (2007) evaluated the heterogeneity of response associated with PM_{10} as evaluated in NMMAPS by analyzing the $PM_{2.5}$ speciation data. Nickel (Ni) and Vanadium (V) were identified as significant predictors of variation in PM_{10}-related mortality across cities, with Ni levels in New York City being reported as particularly high (US EPA, 2009a, section 6.5.2.5). Bell et al. (2009) and Peng et al. (2009) conducted similar analyses focusing on the variation in $PM_{2.5}$-related cardiovascular and respiratory hospital admissions in older adults. Bell et al. (2009) and Peng et al. (2009) used data from the MCAPS study and the CSN to identify the components of $PM_{2.5}$ that are most strongly associated with hospitalizations for cardiovascular disease. Peng et al. (2009) focused on the components that make up the majority of $PM_{2.5}$ mass (SO_4^{2-}, NO^{3-}, Si, EC, OC, NA+ and NH_4+) and found that in multi-pollutant models only EC and OC were significantly associated with risk of hospitalization for cardiovascular disease. Bell et al. (2009) used data from 20 $PM_{2.5}$ components and found that EC, Ni, and V were most positively and significantly associated with the risk of $PM_{2.5}$-related hospitalizations suggesting that the observed associations between $PM_{2.5}$ and hospitalizations may be primarily due to particles from oil combustion and traffic (US EPA, 2009a, section 6.2.10.1). However, as noted in the second draft ISA, in a sensitivity analysis when selectively removing cities from the overall estimate, the significant association between the PM_{10} mortality risk estimate and the $PM_{2.5}$ Ni fraction was diminished upon removing New York City from the analysis, which is consistent with the results presented by Dominici et al. (2007) (US EPA, 2009a, section 6.5.2.5).

In a study of 25 U.S. cities, Franklin et al. (2008) focused on a time-series regression of mortality related to $PM_{2.5}$ mass by season. In addition, the authors considered the $PM_{2.5}$ risk estimates to examine effect modification due to various $PM_{2.5}$ species. They concluded that Al, As, Ni, Si and SO_4^{2-} were significant effect modifiers of $PM_{2.5}$ mortality risk estimates, and "simultaneously including Al, Ni, and SO_4^{2-}_together or Al, Ni, and As together further increased explanatory power. Of all the species examined, Al and Ni explained the most residual

[55] As noted in section 6.5.2.5 of the second draft ISA, "[i]n light of the results presented in speciation studies it must be noted that second stage analyses that use PM chemical species as effect modifiers have some limitations. Unlike analyses that directly examine the associations between chemical species and mortality, if an effect modification is observed it may be confounded if the variations of the mean levels of the chemical species examined are correlated with other demographic factors that vary across cities. Thus, more concrete conclusions could be formulated if direct associations are found between mortality and PM chemical components in time-series analyses (US EPA, 2009a).

heterogeneity." Of note, New York City was not included in the 25 cities examined by Franklin et al. (2008) (US EPA, 2009a, section, 6.5.2.5 and Table 6-16).

For cardiovascular effects, the second draft ISA concludes that:

> …multiple outcomes have been linked to a PM crustal/soil/road dust source, including cardiovascular mortality in Washington D.C. (Ito et al., 2006) and Santiago, Chile (Cakmak et al., 2009) and ST-segment changes in Helsinki (Lanki et al., 2006), Los Angeles (Gong et al., 2003), and Boston (Wellenius et al., 2003). Interestingly, the ST-segment changes have been observed in an epidemiologic panel study, a controlled human exposure study, and a toxicological study, although the majority of the CAPs in the controlled human exposure study was $PM_{10-2.5}$. Further support for a crustal/soil/road dust source associated with cardiovascular health effects comes from a PM_{10} source apportionment study in Copenhagen that reported increased cardiovascular hospital admissions (Andersen et al., 2007).
>
> $PM_{2.5}$ traffic and woodsmoke/vegetative burning sources have also been linked to cardiovascular effects. Cardiovascular mortality in Phoenix (Mar et al., 2000; Mar et al., 2006) and Santiago, Chile (Cakmak et al., 2009) was associated with traffic at lag 1. Gasoline and diesel sources were associated with ED visits in Atlanta for cardiovascular disease at lag 0 (Sarnat et al., 2008). Studies that only examined the effects of individual $PM_{2.5}$ constituents linked EC to cardiovascular hospital admissions in a multi-city analysis (Peng et al., 2009) and cardiovascular mortality in California (Ostro et al., 2007; 2008). (US EPA, 2009a, section 6.6.3).

In a more recent analysis from a single-city study in Atlanta, with an additional 4 years of data and analysis of $PM_{2.5}$ components, ED visits for cardiovascular disease were not significantly associated with PM_{10} or $PM_{2.5}$, but were significantly associated with total carbon, EC and organic carbon components of $PM_{2.5}$ (Tolbert et al., 2007).

Thus, the second draft ISA presents evidence that suggests cardiovascular effects (hospitalizations or ED visits) may be associated with $PM_{2.5}$ from multiple source categories including: wood burning, oil burning, traffic and crustal material, but the best evidence suggests that, in the U.S., oil combustion, wood burning, and traffic are likely the sources of $PM_{2.5}$ most strongly associated with cardiovascular hospitalizations or ED visits. In addition, the second draft ISA concludes "there are many studies that observed associations between other sources (i.e., salt, secondary SO_4^{2-} long-range transport, other metals) and cardiovascular effects, but at this time, there does not appear to be a consistent trend or pattern of effects for those factors" (US EPA, 2009a, section 6.6.3).

There is less consistency in observed associations between PM sources and respiratory health effects, which may be partially due to the fact that fewer studies have been conducted that evaluated respiratory-related outcomes and measures. However, there is some evidence for associations with secondary SO_4^{2-}.and increased respiratory-related ED visits in Atlanta (Sarnat et al., 2008). Decrements in lung function in Helsinki (Lanki et al., 2006) and Los Angeles

(Gong et al., 2005) in asthmatic and healthy adults, respectively, were also linked to secondary SO_4^{2-} (US EPA, 2009a, section 6.6.3). Respiratory effects relating to the crustal/soil/road dust and traffic sources of PM included observations of increased respiratory symptoms in asthmatic children (Gent et al., 2009) and decreased peak expiratory flow (PEF) in asthmatic adults (Penttinen et al., 2006). Inconsistent results were observed in studies that evaluated associations with respiratory morbidity and mortality and individual $PM_{2.5}$ constituents, although Cu, Pb, OC, and Zn were related to respiratory health effects in two or more studies (US EPA, 2009a, section 6.6.3).

A few studies have identified $PM_{2.5}$ sources associated with total mortality. These studies found an association between mortality and a $PM_{2.5}$ coal combustion factor (Laden et al., 2000), while others linked mortality to a secondary SO_4^{2-}long-range transport $PM_{2.5}$ source (Ito et al., 2006; Mar et al., 2006).

Recent studies have shown that source apportionment methods have the potential to add useful insights into which sources and/or PM constituents may contribute to different health effects. Of particular interest are several epidemiologic studies that compared source apportionment methods and the associated results. These studies are discussed in section 6.6.2.1 of the second draft ISA. One set of studies compared epidemiologic associations with $PM_{2.5}$ source factors using several methods - PCA, PMF, and UNMIX - independently analyzed by separate research groups (Hopke et al., 2006; Ito et al., 2006; Mar et al., 2006; Thurston et al., 2005). Schreuder et al. (2006) compared UPM and two versions of UNMIX to derive tracers and Sarnat et al. (2008) compared PMF, modified CMB, and a single-species tracer approach. In all analyses, epidemiologic results based on the different methods were generally in close agreement. The variation in risk estimates for daily mortality between source categories was significantly larger than the variation between research groups (Ito et al., 2006; Mar et al., 2006; Thurston et al., 2005). Additionally, the variation in risk estimates based on the source apportionment model used had a much smaller effect than the variation caused by the different source constituents. Further, the most strongly associated source types were consistent across all groups. This supports the general validity of such approaches, though integration of results would be simpler if the methods employed for grouping PM constituents were more consistent across studies and disciplines. Further research would aid understanding of the contribution of different factors, sources, or source tracers of PM to health effects by increasing the number of locations where similar health endpoints or outcomes are examined.

In summary, these findings presented in the second draft ISA are consistent with the conclusions of the 2004 PM AQCD, that is, a number of source types, including motor vehicle emissions, coal combustion, oil burning, and vegetative burning, are associated with health effects (U.S. EPA, 2004, section x.x, US EPA, 2009a, section 6.6.3). Differences observed

across studies may be due to the difference in geographic coverage, PM size ($PM_{2.5}$ may represent more secondary aerosols than PM_{10}), or the difference in the analytical methods used in each study. One difference noted is that although the crustal factor of fine particles was not associated with mortality in the last review "recent studies have suggested that PM (both $PM_{2.5}$ and $PM_{10-2.5}$) from crustal, soil or road dust sources or PM tracers linked to these sources are associated with cardiovascular effects." In addition, the second draft ISA reports that secondary SO_4^{2-} has been associated with both cardiovascular and respiratory effects. To summarize, the second draft ISA concludes, "Overall, the results displayed in Table 6-17 indicate that many constituents of PM can be linked with differing health effects and the evidence is not yet sufficient to allow differentiation of those constituents or sources that are more closely related to specific health outcomes" (US EPA, 2009a, section 6.6.3).

3.3.2.3.2 Effects Associated with Long-term Exposures to PM2.5 Components and Sources

Limited evidence is available to evaluate the health effects associated with long-term exposures to $PM_{2.5}$ components. This evidence is presented and discussed in section 7.6.2 of the second draft ISA (US EPA, 2009a). The most significant new evidence is provided by a study that evaluated multiple $PM_{2.5}$ components and an indicator of traffic density in an assessment of effects related to long-term exposure to $PM_{2.5}$ (Lipfert et al., 2006). Using health data from a cohort of U.S. military veterans and $PM_{2.5}$ data from EPA's CSN, Lipfert et al. (2006) reported a positive association for mortality with sulfates. Positive associations were found between mortality and long-term exposures to nitrates, EC, Ni and V, as well as traffic density and peak O_3 concentrations. In multi-pollutant models, associations with traffic density remained significant, as did nitrates, Ni and V in some models. Additional evidence from a long-term exposure study conducted in a Dutch cohort provides supportive evidence that long-term exposure to traffic-related particles is associated with increased mortality (Breelen et al., 2008).

3.3.2.4 Ultrafine Particles

In evaluating ultrafine particles (UFPs), the second draft ISA includes evaluation of a limited number of controlled human exposure studies that examined the association between UFPs and cardiovascular morbidity as well as a larger body of evidence from controlled human exposures studies that exposed subjects to fresh diesel exhaust (DE), which is typically dominated by UFPs. As summarized in section 2.3.6 of the second draft ISA, "[t]he controlled human exposure studies evaluated have consistently demonstrated effects on vasomotor function and systemic oxidative stress with additional evidence for alterations in HRV parameters in response to exposure to ultrafine CAPs. The toxicological studies provide coherence for the changes in vasomotor function observed in the controlled human exposure studies...More limited

evidence is available regarding the effect of UFP on respiratory effects. Controlled human exposure studies have not extensively examined the effect of UFPs on respiratory measurements, but a few studies have observed small decrements in pulmonary function. Additional effects including oxidative, inflammatory, and pro-allergic outcomes have been demonstrated in toxicological studies, but the lack of coherence with the controlled human exposure studies limits the interpretation of these findings."

Section 2.3.6 of the second draft ISA makes note that "[e]pidemiologic studies are limited because a national network is not in place to measure UFPs in the U.S. UFP concentrations are spatially variable, which would increase uncertainty and make it difficult to detect associations between health effects and UFPs in epidemiologic studies. In addition, data on the composition of UFPs and potential effects of UFP constituents are sparse." Furthermore, the second draft ISA notes"[o]verall, a limited number of studies have examined the association between exposure to UFP and morbidity and mortality. Of the studies evaluated, controlled human exposure studies provide the most evidence for UFP-induced cardiovascular and respiratory effects; however, these studies focus on exposure to DE. As a result, it is unclear if the effects observed are due to UFP[s], larger particles (i.e., $PM_{2.5}$), or the gaseous components of DE. Additionally, ultrafine CAPs systems are limited as the atmospheric ultrafine PM composition is modified when concentrated, which adds uncertainty to the health effects observed in controlled human exposure studies (US EPA, 2009a, chapter 1) (US EPA 2009a, section 2.3.6).

Collectively, EPA has determined that this evidence is ***suggestive*** of a causal relationship between short-term exposures to UFPs and cardiovascular and respiratory effects, with stronger evidence for cardiovascular than respiratory effects (US EPA, 2009a, section 2.3.5). In addition, EPA has determined that data are *inadequate* to infer whether a causal relationship exists for short-term exposures to UFPs and premature mortality and central nervous system effects as well as ***inadequate*** to infer whether a causal relationship exists for long-term exposures to UFPs and all health effects and mortality (US EPA, 2009a, section 2.3). Evidence of cardiovascular and respiratory effects associated with UFPs is briefly summarized below.

3.3.2.4.1 Cardiovascular Effects

As summarized in section 2.3.5 of the second draft ISA, "[c]ontrolled human exposure studies provide the majority of the evidence for cardiovascular health effects in response to short-term exposure to UFPs. While there are a limited number of studies that have examined the association between UFPs and cardiovascular morbidity, there is a larger body of evidence from studies that exposed subjects to fresh DE, which is typically dominated by UFPs. These studies have consistently demonstrated effects on vasomotor function (Section 6.2.4.2). Markers of

systemic oxidative stress have also been observed to increase after exposure to various particle types that are predominantly in the UFP size range. In addition controlled human exposure studies have observed alterations in HRV parameters in response to exposure to ultrafine CAPs, with inconsistent evidence for changes in markers of blood coagulation following exposure to ultrafine CAPs and DE (Sections 6.2.1.2 and 6.2.8.2). A few toxicological studies have also observed consistent changes in vasomotor function, which provides coherence with the effects demonstrated in the controlled human exposure studies (Section 6.2.4.3). Additional UFP-induced effects observed in toxicological studies include alterations in HRV, with less consistent effects observed for systemic inflammation and blood coagulation. Only a few epidemiologic studies have examined the effect of UFP on cardiovascular morbidity and collectively they found inconsistent evidence for an association between UFPs and CVD hospital admissions, but some positive associations for subclinical measures of CVD (i.e., arrhythmias and supraventricular beats) (Section 6.2.2.1). These studies were conducted in the U.S. and Europe at mean particle number concentration ranges of ~8,500-36,000 particles/cm^3. However, UFP number concentrations are highly dependent on monitor location (i.e., concentrations drop off quickly from the road compared to accumulation mode particles), and therefore, more subject to exposure error than accumulation mode particles. In conclusion, the evidence from the studies evaluated is ***suggestive*** of a causal relationship between short-term exposures to UFPs and cardiovascular effects."

3.3.2.4.2 Respiratory Effects

As summarized in section 2.3.5 of the second draft ISA "[a] limited number of epidemiologic studies have examined the potential association between short-term exposure to UFPs and respiratory morbidity. Of the studies evaluated, there is limited, and predominately inconsistent evidence for an association between short-term exposure to UFPs and respiratory symptoms, as well as asthma hospital admissions at a median particle number concentration of ~6,200 to a mean of 38,000 particles/cm^3 (Section 6.3.8). The spatial and temporal variability of UFPs also affects these associations. Although controlled human exposure studies have not extensively examined the effect of UFPs on respiratory outcomes, a few studies have observed small UFP-induced decreases in pulmonary function. However, these studies have not reported an increase in respiratory symptoms and the observed effects on pulmonary inflammation are not consistent. Toxicological studies have also reported mixed results when examining the effect of UFPs on respiratory effects, but several studies demonstrate oxidative, inflammatory, and allergic responses (Section 6.3). Some effects, such as inflammation or pulmonary histopathology, are only observed when using particular animal models (e.g., immature or compromised). Additionally, although a number of controlled human exposure and toxicological

studies that used controlled exposures to fresh DE report respiratory effects, the relative contributions of gaseous copollutants to the health effects observed remain unresolved. Thus, the current collective evidence is ***suggestive*** of a causal relationship between short-term exposures to UFPs and respiratory effects."

3.3.3 Thoracic Coarse Particles

In the PM NAAQS review completed in 1997, little new toxicologic evidence was available on potential effects of thoracic coarse particles and there were few epidemiologic studies that had included direct measurements of thoracic coarse particles. Evidence of associations between health outcomes and PM_{10} that were conducted in areas where PM_{10} was predominantly composed of thoracic coarse particles was an important part of EPA's basis for reaching conclusions about the requisite level of protection from coarse particles provided by the final standards (62 FR 38652, July 18, 1997).

The evidence from a growing, but still limited, body of evidence available in the PM NAAQS review completed in 2006 included epidemiologic studies that had reported associations with health effects using direct measurements of $PM_{10-2.5}$, as well as number of new toxicological studies on known or potential effects associated with exposure to thoracic coarse particles and their major constituents. In the last review, EPA highlighted that the nature of the effects associated with short-term exposures to ambient thoracic coarse particles, particularly in urban and industrial settings, included aggravation of respiratory and cardiovascular disease (as indicated by increased hospital admissions), increased respiratory symptoms in children, and premature mortality. Populations that appeared to be at greater risk for experiencing effects related to thoracic coarse particles exposures included individuals with preexisting lung diseases such as asthma, as well as children and older adults.[56]

In the current review, new epidemiologic, controlled human exposure, and toxicological studies provide evidence that is ***suggestive*** of associations between short-term $PM_{10-2.5}$ exposures and cardiovascular effects, respiratory effects, and mortality (US EPA 2009a, section 2.3.3) and ***inadequate*** for inferring whether a causal relationship exists between $PM_{10-2.5}$ and central nervous system effects (US EPA, 2009a, section 2.3) . Causal determinations presented in the second draft ISA "were made for $PM_{10-2.5}$ as a whole regardless of origin, since $PM_{10-2.5}$-related effects have been demonstrated for a number of different environments" (US EPA, 2009a, section 2.3.4). These causal determinations were made based upon short-term $PM_{10-2.5}$ exposure studies. With respect to evaluating long-term exposures, the second draft ISA concludes that

[56] See summary of the evidence of health effects related to thoracic coarse particle exposures in the 2006 proposed rule - 71 FR 2654 to 2662, January 17, 2006; and a more comprehensive and detailed discussions of the scientific evidence in chapters 6-9 of the Criteria Document (US EPA, 2004) and chapter 3 of the Staff Paper (US EPA, 2005).

available evidence is ***inadequate*** (US EPA, 2009a, section 2.3). Specifically, the second draft ISA states, "[t]o date, a sufficient amount of evidence does not exist in order to draw conclusions regarding the health effects and outcomes associated with long-term exposure to $PM_{10-2.5}$" (US EPA, 2009a, section 2.3.4).

Measurement error and exposure error are issues that may be distinctly more important for interpretation of results for assessments of $PM_{10-2.5}$ than for $PM_{2.5}$. This exposure measurement error may bias effect estimates for $PM_{10-2.5}$ toward the null. These and other issues relevant to the interpretation of epidemiological evidence of PM-related effects are discussed in section 3.4 below.

The majority of epidemiological studies evaluating thoracic coarse particles provide limited information on the air quality data used in the analyses, generally reporting average concentrations rather that upper percentile values making it difficult to understand the air quality distribution considered in the analyses. The statistical form of the current PM_{10} standard is set as one expected exceedence and evidence has supported looking carefully at short-term peak exposures to evaluate public impacts of thoracic coarse particles. Without additional air quality data, we are limited to interpret the policy relevancy of the scientific body of evidence for thoracic coarse particles. As noted in section 3.3.2.1 above, we have requested additional air quality data from study authors, including information on coarse particles, in order to better interpret the policy relevancy of the scientific body of evidence, including air quality data for evaluating short-term exposures to thoracic coarse particles.

3.3.3.1 Effects Associated with Short-term $PM_{10-2.5}$ Exposures

In the PM NAAQS review completed in 2006, emphasis was placed on evaluating a range of respiratory morbidity effects associated with thoracic coarse particle exposures (US EPA, 2004, section 8.4.6.4) with consideration of new findings on potential cardiovascular effects of thoracic coarse particles (US EPA, 2004, section 8.3.1.4). New evidence from studies of mortality available in the last review indicated effect estimates for associations between mortality and short-term exposures to $PM_{10-2.5}$ that were generally positive and similar in magnitude to those for $PM_{2.5}$ and PM_{10} though most were not statistically significant and, in general, had greater confidence intervals (indicating greater uncertainty). Furthermore, effect estimates were somewhat larger for cause-specific mortality (e.g., respiratory and cardiovascular mortality) than for total mortality (US EPA, 2005, Figures 3-1 and 3-2).

In the current review, additional epidemiological evidence as well as limited new toxicological and controlled human exposure studies are available. Collectively, as noted above, EPA has determined that this evidence is ***suggestive*** of a causal relationship between short-term exposures to $PM_{10-2.5}$ and cardiovascular effects, respiratory effects, and mortality (US EPA,

2009a, section 2.3.3, Table 2-3). However, limited $PM_{10-2.5}$ air quality data, variability in the chemical and biological composition of $PM_{10-2.5}$, limited evidence regarding effects of the various components of $PM_{10-2.5}$, and lack of clearly defined biological mechanisms for $PM_{10-2.5}$-related effects are important sources of uncertainty (US EPA, 2009a, section 2.3.3.2). The key evidence supporting these causal determinations is briefly described below.

3.3.3.1.1 Cardiovascular and Systemic Effects

Limited evidence was available in the last review regarding $PM_{10-2.5}$-related cardiovascular effects. Two single-city epidemiologic studies found generally positive associations of $PM_{10-2.5}$ and cardiovascular hospital admissions in Toronto (Burnett et al., 1997) and Detroit, MI (Ito, 2003). A study in Tucson, AZ, an urban area where thoracic coarse particles are a much greater fraction of PM_{10} than are fine particles, reported a statistically significant association between PM_{10} and increased hospitalizations for cardiovascular disease (Schwartz, 1997). In addition, one study in the last review reported no significant association between onset of myocardial infarction (MI) and short-term $PM_{10-2.5}$ exposures (Peters et al., 2001).

In this review, new epidemiological evidence includes a recent multi-city study evaluating hospital admissions and emergency department (ED) visits for cardiovascular disease in older adults. In a study of Medicare patients (MCAPS), Peng et al. (2008) reported a significant association between $PM_{10-2.5}$ and cardiovascular disease hospitalizations in a single pollutant model using air quality data for 108 U.S. counties with one or more co-located $PM_{2.5}$ and PM_{10} monitors.[57] However, this association diminished slightly and was no longer statistically significant after adjustment for $PM_{2.5}$ (US EPA, 2009a, section 6.2.12.2).

In contrast, associations of cardiovascular outcomes with $PM_{10-2.5}$ were weak for CHF and null for IHD in the Atlanta-based SOPHIA study (Metzger et al., 2004). Results from single-city studies were generally positive but effect sizes were heterogeneous and estimates were imprecise (US EPA, 2009a, section 6.2.10). Crustal material from a dust storm in the Gobi desert that was largely coarse PM (generally indicated using PM_{10}) was associated with hospitalizations for cardiovascular diseases including IHD and CHF in most studies (US EPA, 2009a, section 6.2.10.1). A few epidemiologic studies that examined the association between short-term exposure to $PM_{10-2.5}$ and cardiovascular mortality (US EPA, 2009a, section 6.2.11)

[57] Analyses with $PM_{10-2.5}$ were carried out using similar methods to those of Dominici et al. (2006). Peng et al. (2008) evaluated the robustness of $PM_{2.5}$ associations to adjustment for thoracic coarse PM (Peng et al., 2008). Gaseous pollutants were not considered in these analyses. In 108 U.S. counties with co-located PM_{10} and $PM_{10-2.5}$ monitors, the authors found a 0.4% (95% PI, 0.1 to 0.7, lag 0) increase in risk per 10 $\mu g/m^3$ $PM_{10-2.5}$ and no associations at lags of 1 and 2 days. In a 2-pollutant model adjusted for $PM_{2.5}$, the association between $PM_{10-2.5}$ and CVD hospitalization lost precision (0.3% [95% PI: −0.1 to 0.6, lag 0]) (Peng et al., 2008).

provide supporting evidence for the cardiovascular-related hospital admission and ED visit studies. A multi-city study reported risk estimates for cardiovascular mortality of similar magnitude to those for all-cause (nonaccidental) mortality (Zanobetti and Schwartz, 2009). However, the single-city studies evaluated (Wilson et al., 2007; Villeneuve et al., 2003) reported substantially larger effect estimates, possibly related to differences between cities and compositional differences in $PM_{10-2.5}$ across regions. Of note is the lack of analyses within the studies to evaluate potential confounders of the $PM_{10-2.5}$-cardiovascular mortality relationship (US EPA, 2009a, section 6.2.12.2).

As presented in the second draft ISA, EPA concludes that collectively, the evidence from epidemiologic studies, along with the more limited evidence from controlled human exposure and toxicological studies is ***suggestive*** of a causal relationship between short-term exposure to $PM_{10-2.5}$ and cardiovascular effects (US EPA, 2009a, section 2.3.3, section 6.2.12.2). The second draft ISA concludes, "[i]n multi-city epidemiologic studies, associations between short-term exposure to $PM_{10-2.5}$ and cardiovascular outcomes (i.e., IHD, HAs, supraventricular ectopy, and changes in HRV) have been found that are similar in magnitude to those observed in $PM_{2.5}$ studies (US EPA, 2009a, section 2.3.4). Controlled human exposure studies have also observed alterations in HRV, providing consistency and coherence for the effects observed in the epidemiologic studies. To date, only a limited number of toxicological studies have been conducted to examine the effects of $PM_{10-2.5}$ on cardiovascular outcomes. All of these studies involved IT instillation due to the technical challenges of using $PM_{10-2.5}$ for rodent inhalation studies…. As a result, the toxicological studies evaluated provide limited biological plausibility for the $PM_{10-2.5}$ effects observed in the epidemiologic and controlled human exposure studies" (US EPA, 2009a, section 2.3.4).

3.3.3.1.2 Respiratory Effects

In the last PM NAAQS review, epidemiologic studies analyzing the effect of $PM_{10-2.5}$ exposures on hospitalizations or ED visits for respiratory diseases (i.e., pneumonia, COPD and respiratory diseases combined) reported generally positive associations (US EPA, 2004, section x.x). The few mortality studies that examined cause-specific mortality associated with $PM_{10-2.5}$ exposures suggested somewhat larger risk estimates for respiratory mortality compared to all-cause (non-accidental) mortality. Several epidemiologic studies of respiratory symptoms and thoracic coarse particles provided limited evidence for cough and effects on morning PEF (ADD REF). Toxicology data for $PM_{10-2.5}$ were extremely limited, and there were no controlled human exposure studies considered in the last review that evaluated the effect of $PM_{10-2.5}$ on respiratory symptoms, pulmonary function, or inflammation.

In this review, several new epidemiologic studies report associations between short-term $PM_{10-2.5}$ exposures and hospital admissions, ED visits, or outpatient visits related to respiratory effects with the most consistent evidence reported in children, including asthmatic children (US EPA, 2009a, Figures 6-10 through 6-14). Overall, the second draft ISA reports the most compelling new evidence showing significant associations between an increase in respiratory-related hospital admissions and ED visits and short-term exposure to $PM_{10-2.5}$ has been observed in studies conducted in Canada and Europe (e.g., Host et al, 2008; Fung et al., 2006; Chen et al., 2005; Yang et al., 2004). In these studies, the strongest associations were reported for children, with less consistent evidence of effects in adults, including older adults (i.e., 65 years of age and older) (US EPA, 2009a, section 2.3.3). Mar et al., (2004) report an association between short-term $PM_{10-2.5}$ exposures and increased respiratory symptoms in asthmatic children but not asthmatic adults. Several Canadian studies of respiratory hospital admissions reported larger effects for $PM_{10-2.5}$ compared to $PM_{2.5}$ that were robust to adjustment for gaseous pollutants (Chen et al., 2005; Lin et al., 2002; Yang et al., 2004). The COPD associations with short-term $PM_{10-2.5}$ exposures reported by Chen et al. (2004) remained positive but were diminished slightly after adjustment for NO_2. Sinclair and Tolsma (2004) reported a significant association between $PM_{10-2.5}$ exposures and childhood but not adult asthma-related outpatient visits in Atlanta, GA. In contrast, Slaughter et al. (2005) reported no associations with ED visits or hospitalizations for asthma among all ages in Spokane, WA. Although a number of studies provide evidence of respiratory effects in older adults, a recent analysis of MCAPS data reports that weak associations of $PM_{10-2.5}$ with respiratory hospitalizations are further diminished after adjustment for $PM_{2.5}$.[58]

Limited evidence for $PM_{10-2.5-}$ related effects is available from controlled human studies and toxicological studies. Increases in markers of pulmonary inflammation in the absence of lung function effects or respiratory symptoms have been reported in healthy adults (US EPA, 2009a, section x.x). In toxicological studies, $PM_{10-2.5}$ from both rural and urban environments has induced inflammation and injury responses in rats or mice following IT instillation, "making it difficult to distinguish effects of $PM_{10-2.5}$ from different environments" (US EPA, 2009a, section 2.3.3). The majority of toxicological evidence, described in section 6.3.7.1 of the second draft ISA and in the 2004 PM AQCD (U.S. EPA, 2004, section x.x), suggests that PM impairs innate immunity, the first line of defense in preventing infection.

[58] In an analysis of $PM_{10-2.5}$, MCAPS investigators observed small imprecise increases in respiratory admissions with 24-h $PM_{10-2.5}$ concentration (0.33% [95% PI: -0.21 to 0.86, per 10 $\mu g/m^3$, lag 0]) (Peng et al., 2008), which decreased after adjustment for $PM_{2.5}$ (0.26% [95% PI: -0.32 to 0.84 per 10 μg/m3 lag 0]) (US EPA, 2009a, section 6.3.8.1)

Overall, based on epidemiologic studies, along with the limited number of controlled human exposure and toxicological studies that examined $PM_{10-2.5}$ respiratory effects the second draft ISA determines that the overall scientific data base for thoracic coarse particles provides evidence that is *suggestive* of a causal relationship between short-term exposures to $PM_{10-2.5}$ and respiratory effects (US EPA, 2009a, section 2.3.3.1).

3.3.3.1.3 Mortality

Evidence available in the last review evaluating short-term exposure to $PM_{10-2.5}$ and premature mortality was limited and indicated mixed results primarily from single-city studies, where two studies reported a positive and statistically significant association while other studies reported associations that were generally positive but not statistically significant. Effect estimates were similar in magnitude to those for $PM_{2.5}$ and PM_{10} (US EPA, 2005, section 3.3.1.1). Staff noted that on a unit mass basis, the effect estimates for both $PM_{2.5}$ and $PM_{10-2.5}$ [were] generally larger than those for PM_{10}, which is consistent with $PM_{2.5}$ and $PM_{10-2.5}$ having independent effects (US EPA, 2004, p. 9-25). In general, cause-specific effect estimates reported in the last review were somewhat larger for respiratory and cardiovascular mortality than for total mortality. Two out of five studies reported effect estimates for cardiovascular mortality with short-term $PM_{10-2.5}$ exposures as positive and statistically significant (Mar et al., 2003; Ostro et al., 2003) while none of the effect estimates for total mortality or respiratory mortality reached statistical significance (US EPA, 2005, Figure 3-1). The magnitude of the effect estimates for $PM_{10-2.5}$ were similar to those for $PM_{2.5}$, generally falling in the range of 3 to 8 % for cardiovascular mortality and 3 to 16% per 25 $\mu g/m^3$ $PM_{10-2.5}$ (US EPA, 2004, p. 8-306).[59] These studies were considered to have multiple limitations including measurement and exposure issues for $PM_{10-2.5}$ correlations between $PM_{2.5}$ and $PM_{10-2.5}$. "These limitations increased the uncertainty surrounding the concentrations at which $PM_{10-2.5}$ mortality associations were observed" (US EPA, 2009a, section 6.5.2.3).

In this review, the majority of new studies continue to provide evidence of an association between premature mortality and short-term $PM_{10-2.5}$ exposures. This evidence continues to show positive, but, not generally statistically significant results with generally stronger evidence reported for cardiovascular-related and respiratory-related mortality rather than all-cause, nonaccidental mortality (US EPA, 2009, Figures 6-28 and 6-30). Two new multi-city studies have been published since the last review. A Canadian 12-city study provides evidence for an association between short-term exposure to $PM_{10-2.5}$ and mortality using direct measurements of

[59] As noted in the last review, effect estimates for respiratory mortality 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 (US EPA, 2005, section 3.3.1.1).

$PM_{10-2.5}$ with an every 6th day sampling schedule (Burnett et al., 2004, US EPA, 2009a, section 6.5.2.3). In addition, an examination of $PM_{10-2.5}$ mortality associations in a new multi-city U.S. study found associations between $PM_{10-2.5}$ and cardiovascular, respiratory, and total mortality, but this association varied when examining city-specific risk estimates (Zanobetti and Schwartz, 2009; US EPA, 2009a, Figure 6-29; section 6.5.2.3). The study authors reported seasonal (i.e., larger in spring) and regional differences in $PM_{10-2.5}$ respiratory mortality risk estimates. In this study, $PM_{10-2.5}$ ambient concentrations were estimated by calculating the difference in countywide averages of PM_{10} and $PM_{2.5}$. As reported in section 6.5.2.3 of the second draft ISA (US EPA, 2009a), "[i]t is not clear how the computed $PM_{10-2.5}$ concentrations used by Zanobetti and Schwartz (2009) compare with the $PM_{10-2.5}$ concentrations obtained by directly measuring $PM_{10-2.5}$ using a dichotomous sampler, or the $PM_{10-2.5}$ concentrations computed using the difference of PM_{10} and $PM_{2.5}$ measured at co-located samplers." Furthermore, copollutant analyses were not conducted in the Zanobetti and Schwartz (2009) study, and the associations observed were inconsistent with those reported in single-city studies, for example, single-city studies previously conducted in Phoenix (Mar et al., 2003; Wilson et al., 2007) reported a $PM_{10-2.5}$ mortality association in this "dry" region city in contrast to a lack of association reported by Zanobetti and Schwartz in this region (see US EPA, 2009a, Figure 6-28). In addition, single-city studies conducted in Atlanta, GA (Klemm et al., 2004), and Vancouver, Canada (Villeneuve et al., 2003) reported no associations between short-term exposure to $PM_{10-2.5}$ and respiratory mortality where the overall multi-city analysis conducted by Zanobetti and Schwartz (2009) indicated an association. Villeneuve et al. (2008) observed a statistically significant association between $PM_{10-2.5}$ exposures and cardiovascular mortality in Vancouver, Canada. The investigators reported a stronger association for $PM_{10-2.5}$ than for $PM_{2.5}$ (US EPA, 2009a, section 6.5.2.3)

The second draft ISA notes limitations "exist in the $PM_{10-2.5}$ associations reported due to the small number of $PM_{10-2.5}$ studies that have investigated confounding by gaseous copollutants or the influence of model specification on $PM_{10-2.5}$ risk estimates. Additionally, more data [are] needed to characterize the chemical and biological components that may modify the potential toxicity of $PM_{10-2.5}$ (US EPA, 2009a, section 6.5.2.3) Specifically, the second draft ISA notes that the "difference in the results observed between the multi- and single- city studies could be due to a variety of factors including differences between cities and compositional differences in $PM_{10-2.5}$ across regions" (see also, US EPA, 2009a, Figure 6-28). As was reported in the last review, the strongest evidence for cardiovascular related mortality associated with thoracic coarse particles has been reported for Phoenix (Mar et al., 2003) and Coachella Valley (Ostro et al., 2003).

3.3.3.2 Effects Associated with Long-term $PM_{10-2.5}$ Exposures

Evidence to evaluate health impacts of long-term $PM_{10-2.5}$ exposures is extremely limited. In the last PM NAAQS review, no association was observed between mortality and long-term exposure to $PM_{10-2.5}$ in the ACS study (Pope et al., 2002), and a positive but nonsignificant association was reported in males in the AHSMOG cohort (McDonnell et al., 2000). In that review, EPA concluded that the available studies provided no evidence for associations between long-term exposure to coarse fraction particles and mortality. Furthermore, there was little evidence available to draw conclusions about long-term $PM_{10-2.5}$ exposures and morbidity (US EPA, 2009a, section 9.2.2.1.2, p. 9-34). In 2006, EPA revoked the annual PM_{10} standard stating:

> The long-term exposure studies of mortality and morbidity that permit comparisons of fine and coarse particles continue to suggest that, at current ambient levels in the US, fine particles are associated with health effects and coarse particles are not.[60] (71 FR 61198, October 17, 2006).

In the current review, evidence is still limited for evaluating long-term exposures to thoracic coarse particles. The second draft ISA concludes that "[t]o date, a sufficient amount of evidence does not exist in order to draw conclusions regarding the health effects and outcomes associated with long-term $PM_{10-2.5}$ exposures" (US EPA, 2009a, section 2.3.3.2). New findings from the California Seventh Day Adventist Study (AHSMOG) and Veterans cohort studies provide limited evidence of associations between long-term exposure to $PM_{10-2.5}$ and mortality (US EPA, 2009a, sections 7.6.1 and 7.6.5.2). The AHSMOG study reported a positive association for $PM_{10-2.5}$ and coronary heart disease mortality among females, but not males as was found with fine particles; associations were strongest in the subset of postmenopausal women (Chen et al., 2005). A recent reanalysis of the Veterans cohort study (Lipfert et al, 2006) focused on traffic-related air pollution reported a significant association between long-term $PM_{10-2.5}$ exposures and total mortality in a single-pollutant model, however, that association became negative and not statistically significant in a model that included traffic density. The second draft ISA concluded "[a]s it would be expected that traffic would contribute to the thoracic coarse particle concentrations, it is difficult to interpret the results of these multi-pollutant analyses" (US EPA, 2009a, section 7.6.1). No supporting toxicological or clinical

[60] See US EPA 2004, section 8.4.6.2 - "no statistically significant associations have been reported between long-term exposure to coarse fraction particles and cause-specific mortality" and section 8.4.6.4 "[t]he recent studies suggest that long-term exposure to fine particles is associated with development of chronic respiratory disease and reduced lung function growth; little evidence is available on potential effects of exposure to coarse fraction particles."

studies of long-term exposure to ambient $PM_{10-2.5}$ and cardiovascular effects have been conducted to date.

Studies evaluating the association between long-term exposure to PM_{10} and respiratory morbidity were primarily in areas where PM is predominantly fine particles. Consequently, studies utilizing the PM_{10} fraction provide limited information on the association between long-term exposure to thoracic coarse particles and respiratory morbidity.

Overall, the second draft ISA determined that evidence is ***inadequate*** to infer the presence or absence of a causal relationship between long-term $PM_{10-2.5}$ exposures and a range of health effects, specifically, cardiovascular and systemic effects (US EPA, 2009a, section 7.2.12.2), respiratory effects (US EPA, 2009a, section 7.3.9.2), cancer (US EPA, 2009a, section 7.5.4) and mortality (US EPA, 2009a, section 7.5.5.2).

3.3.3.3 $PM_{10-2.5}$ Components

One of the major research priorities defined by the National Academy of Sciences National Research Councils (NRC) related to assessing the health effects of PM components and sources (NRC, 2004). In the last review, the issue of differential toxicity related to particles originating in urban versus non-urban areas was a central focus. The strongest evidence of effects associated with short-term exposures to ambient thoracic coarse particles were reported in studies evaluating urban and/or industrial settings. In general, there was evidence of toxicity of certain components of thoracic coarse particles, such as metals and endotoxins, as well as evidence that natural crustal materials of geologic origin, such as Mt. St. Helens volcanic ash, may have very little toxicity. There was largely an absence of evidence regarding the presence or absence of toxicological effects associated with other types of coarse particles in non-urban areas.

With respect to thoracic coarse particles, $PM_{10-2.5}$ may contain iron, silica, aluminum, and base cations from soil, plant and insect fragments, glucans, pollen, fungal spores, bacteria, animal waste, and viruses, as well as fly ash, brake lining particles, debris, and automobile tire fragments, toxic trace elements and other components from previously deposited fine PM e.g. metals from smelters and steel mills, polycyclic aromatic hydrocarbons from automobile exhaust, and pesticides from agricultural activities (US EPA, 2009a, section 2.3.4; EPA 2004 8-344). It is important to note that the chemical composition of $PM_{10-2.5}$ can vary considerably by location, but city-specific speciated $PM_{10-2.5}$ data are limited (ADD REF). Staff observes that no epidemiologic evidence is available to evaluate effects of different components or sources of thoracic coarse particles and that, overall, EPA has not considered the scientific data to be extensive enough to support making causal determinations for thoracic coarse particle components in this review. Causal determinations presented in the second draft ISA "were made

for $PM_{10-2.5}$ as a whole regardless of origin, since $PM_{10-2.5}$-related effects have been demonstrated for a number of different environments" (US EPA, 2009a, section 2.3.4).

3.4 ISSUES RELATED TO INTERPRETING EPIDEMIOLOGIC EVIDENCE

The 2004 AQCD 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 2004 PM AQCD concluded that PM-health effects associations reported in epidemiologic studies were not likely to be an artifact of model specification, since analyses or reanalyses of data using different modeling strategies reported similar results (ADD REF). The 2004 review provided the opportunity to evaluate 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 2, 3, 6, 7, and 8 of the ISA. The following discussion builds upon the ISA's evaluation of key methodological issues related to epidemiologic studies as a basis for staff conclusions specifically regarding the use of epidemiologic evidence in quantitative risk assessments, as discussed in chapters 4 and 5 of this document. This section addresses a number of key methodological issues. Two key policy-relevant issues related to interpreting epidemiologic evidence have been highlighted in the second draft ISA and are presented first. Section 3.4.1 includes discussion of several topics related to the exposure periods used in epidemiologic studies, with an emphasis on the issue of lag periods. In section 3.4.2, the form of concentration-response (C-R) 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. Additional issues discussed below draw primarily from discussions in the 2004 AQCD. Section 3.4.3 discusses issues related to air quality data used in epidemiologic studies and section 3.4.4 discusses the potential impact of measurement error and exposure error, related to the use of ambient air concentrations as indicators of population exposures. Section 3.4.5 addresses statistical modeling and alternative model specifications used in epidemiologic studies. Section 3.4.6 addresses the issue of potential confounding by co-pollutants and effect-modification, as it relates to staff conclusions about the use of specific study results in quantitative assessments.

3.4.1 Issues Related to Alternative Exposure Periods in Epidemiological Studies

3.4.1.1 Lag Structure in Short-term Exposure Studies

In epidemiological studies of short-term exposure to PM, many investigators have examined a range of lag periods between health outcomes and PM concentrations to identify the

etiologically relevant time period for PM exposure (US EPA, 2009a section 2.4.2). As discussed in the second draft ISA, it is important to consider the pattern of results that is seen across the series of lag periods. Specifically, if there is an apparent pattern of results across the different lags indicating a cumulative effect from PM exposure, 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 (US EPA, 2004, p. 8-270). The prior review noted that the evidence supported the use of lags of 0-1 days for cardiovascular effects and longer moving averages or distributed lags for respiratory disease (USA EPA, 2004). However, the current review reports there is limited consensus on the etiologically relevant lag period for PM associated health effects, and recommends examining a cumulative lag structure of associations between PM concentration and health outcome instead of focusing on a priori lag times. Moreover, where effects are found for a series of lag periods, a distributed lag model will more accurately characterize the effect estimate size (add ref). However, if there is no apparent pattern or reported effects vary across lag days, any result for a single day may well be biased (US EPA, 2004 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 $PM_{2.5}$ across lag periods from U.S. and Canadian studies. Many short-term studies reviewed in the second draft ISA assess effects associated with a range of lag periods, with some authors reporting effect estimates for one lag period based on this evaluation. However, a number of studies selected lag periods *a priori*. For example, for their multi-city US based study, Zanobetti et al. (2009) used an average of 0-day and 1-day lagged $PM_{2.5}$ 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. In the case of cardiovascular morbidity, Figure 6-2 in the second draft ISA presents associations for $PM_{2.5}$ levels over a series of days preceding hospitalization for ischemic heart disease and myocardial infarction, and positive associations can be seen over several adjacent lag periods (US EPA, 2009a, section 6.2.10). However, the second draft ISA notes that most studies rarely report results for alternative lag structures (US EPA, 2009a, section 2.4.2.1). In general, studies evaluating the association between short-term exposure to PM and cardiovascular hospitalizations and emergency department visits reported strong results for lags of 0 to 2 days, with limited evidence at shorter time periods (i.e., hours). Although human clinical and toxicological data provide evidence of biological plausibility for cardiovascular effects

associated with PM levels at immediate or concurrent day lag, a recent study provides support for a longer lag time (i.e., 14-day distributed lag model) associated with non-ischemic cardiovascular disease (US EPA, 2009a Section 6.2.10). Moreover, cumulative PM exposures over multi-day lags that are associated with the development of risk factors (e.g., atherosclerosis) can potentially lead to individuals being at risk for experiencing an acute event (e.g., myocardial infarction) from a comparatively short-term PM exposure over a lag of 0 to 2 days. Consequently, the second draft ISA concludes "effects have been observed at a range of lag periods from a few hours to several days with no clear evidence for any lag period having stronger associations then another" (US EPA, 2009a, section 2.4.2.1).

In section 2.4.2.2 the second draft ISA discusses the influence of lag on studies evaluating the association between short-term exposure to PM and respiratory morbidity. The prior review noted that researchers reported testing associations for 3 day lags and beyond and reporting consistent patterns across lags for associations between asthma hospitalization and PM_{10}, $PM_{2.5}$, or $PM_{10-2.5}$ (US EPA, 2005 p. 3.-47). Furthermore, the prior review indicated that effect estimates should be considered in the context of adjacent lags (i.e. distributed lags) instead of a single day lag due to the correlated nature of daily pollutant exposures. In the current review, studies of respiratory hospitalizations associated with PM exposure over multiple lags report a higher magnitude of effect with distributed lag models. Longer lags (e.g. 2 day lag) were strongly associated with asthma hospitalizations and emergency department visits among children, but the second draft ISA notes inconsistent evidence on PM associated asthma hospitalizations for shorter lags (i.e. lag 0 or 1 day) among older adults (US EPA, 2009a, section 6.3.8) is potentially due to the etiological complexity of asthma. However, toxicological and human clinical data on PM effects on allergic sensitization and immune responses provide biological plausibility for a longer lag period.

In assessments of the influence of lag on the association between short-term exposure to PM and mortality the prior review reported that among U.S. and Canadian studies, the AQCD observed that many authors report finding a pattern of PM-related effects across adjacent lags (US EPA, 2004 p. 8-279). However, other studies reported inconsistent results across lags, in particular for COPD mortality (US EPA, 2005 p. 3-50). The second draft ISA notes that most recent studies focus on short-term exposure to PM in association with mortality (i.e., all-cause, cardiovascular, and respiratory) with an a priori lag structures of either 1 or 0-1 days, with the strongest PM-mortality associations observed for these lag periods (US EPA, 2009a, section 2.4.2.3). However, recent evidence, although limited, suggests larger effects of PM associated mortality occur over a distributed lag period (ADD REF).

In summary, the second draft ISA 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 lags. For cardiovascular disease, PM levels at 0 to 2 day lag are strongly associated with hospitalization, although effects were observed for a range of lag periods. 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 among children, there appear to be larger effects over longer average time periods. As noted in the second draft ISA, these results should be interpreted with caution as the etiologically relevant temporal period for PM exposure may also be influenced by individual susceptibility, which could increase or decrease the lag times associated with a specific health effect (US EPA, 2009a, section 2.4.2). For quantitative assessment, staff concludes that it is appropriate to use results from lag period analyses consistent with those reported in the second draft ISA, focusing on shorter lag periods for cardiovascular effects and multi-day lag periods for respiratory effects, depending on availability of results.

3.4.1.2 Seasonal and Regional Differences in Time-Series Epidemiological Results

Epidemiological studies of health effects associated with short-term exposure to PM (e.g., time-series studies) generally use temporal or seasonal terms in regression models to control for potential confounding by seasonal changes in health outcomes. In addition to adjusting PM risk estimates for seasonal influence, epidemiologic studies also evaluate PM-health associations stratified by different seasons, and assess seasonal effect modification of PM risk in models using seasonal interaction terms for the exposure surrogate and meteorology (e.g., Dominici et al., 2000). The second draft ISA reports that there can be seasonal differences in correlations between PM and other pollutants (US EPA, 2009a, section 3.9.1.3), or in PM levels across seasons (US EPA, 2009a section 3.8.6.4).

The limited evidence presented in the prior review indicated that $PM_{2.5}$ associated effects were stronger in the winter season in southern California than in the summer season (Ostro et al., 2000), but an earlier study also conducted in southern California found stronger effects only in the summer months (Ostro et al., 1995). Furthermore, a study conducted in San Jose found no significant differences in $PM_{2.5}$ associated mortality were observed 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). In Seattle, associations between $PM_{2.5}$ 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).

Section 3.8.6.4 of the second draft ISA discusses the influence of season and region on $PM_{2.5}$ risk estimates. Bell et al. (2008) recently reported higher $PM_{2.5}$ risk estimates for cardiovascular and respiratory morbidity in the winter and in the northeast compared to other seasons in the rest of the country in a large, national multi-city study, and posits that these results are potentially attributable to seasonally varying PM composition and concomitant toxicity. In addition, the second draft ISA observes that the limited influence of seasonality on PM risk estimates in regions other than the northeast may be due to exposure misclassification arising from variability in time spent indoors vs. outdoors in different regions, and the higher prevalence of infectious disease in the winter (US EPA, 2009a, section 3.8.6.4). Furthermore, increased air conditioning in the warmer months is associated with a decrease in risk of cardiovascular morbidity outcomes (Bell et al., 2009). In the case of mortality, Zanobetti and Schwartz (2009) reported in their multi-city US based study a 4-fold higher effect estimate for $PM_{2.5}$ associated mortality for the spring as compared to the winter. These results suggest individuals are at greater risk of dying from higher exposures to PM in the warmer months, and at greater risk of PM associated hospitalization for cardiovascular and respiratory diseases during colder months of the year.

Overall, staff observes that there are few studies presently available to deduce a general pattern in PM risk across seasons and regions. However, the two aforementioned multi-city studies showed a seasonal and regional influence for cardiovascular and respiratory morbidity and mortality, respectively (Schwartz et al., 2000). These studies report generally positive results across all seasons tested, with some reporting larger effect estimates in one or more season(s). Therefore, staff concludes the available evidence provides an opportunity to conduct a quantitative assessment of seasonal differences in relationships between PM and health outcomes.

3.4.1.3 Health Effects Related to Different Short-term Exposure Time Periods

While most time-series epidemiologic studies use 24-hour average PM measurements, a few studies have used ambient PM concentrations averaged over shorter time intervals, such as 1- or 2-hour averages. The prior review noted that several epidemiological studies evaluating associations with cardiovascular health biomarkers or physiological changes reported 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 (US EPA, 2004 pp. 8-162 to 8-165). In particular, Peters et al (2001) reported effect estimates for myocardial infarction incidence with $PM_{2.5}$ averaged over 2- and 24 hours that were similar in magnitude and statistically significant (US EPA, 2004, p. 8-165). However, two studies

discussed in the current review that assessed sub-daily exposures to $PM_{2.5}$ did not observe an association between $PM_{2.5}$ and risk of myocardial infarction (Sullivan et al., 2005; Peters et al., 2005).

For respiratory health outcomes, the prior review reported on results from two panel studies of symptoms in asthmatic subjects (US EPA, 2004, 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; US EPA, 2004 p. 8-206). In the current review, a study in two New York City communities assessing the association between 24-hour and 1-hour maximum $PM_{2.5}$ levels and ED visits for asthma reported an increase in ED visits that was similar for 24-hour and 1-hour $PM_{2.5}$ (New York State Department of Health, 2006). These results were robust to adjustment by co-pollutants. However, the evidence on sub-daily PM levels associated with respiratory diseases is limited, and the current review concludes that for f several studies of hospital admissions or medical visits for respiratory diseases, the strongest associations were observed with several-day average PM concentrations (US EPA, 2009a, section 6.3.7.1). Staff observes that the very limited information available in the second draft ISA continues to suggest that cardiovascular effects may be associated with acute exposure time periods on the order of an hour or so.

3.4.1.4 Exposure Periods Used in Prospective Cohort Studies

Prospective cohort studies use air quality measurements averaged over long periods of time (e.g. one to several years) and varying spatial scales to characterize the long-term ambient levels in the community. The prior review noted that exposure comparisons were basically cross-sectional in nature, which does not provide evidence of a temporal relationship between exposures to PM and resulting effects (US EPA, 2004 p. 9-42). Difficulty in disentangling effects associated with historic exposures as compared to more recent exposures in these studies can lead to potential exposure measurement error (US EPA, 2004, p. 5-118). This potential misclassification of exposure is exacerbated under conditions where average PM concentrations change differentially over time between areas. Therefore, long-term exposure effect estimates are less likely to be influenced by exposure error for pollutants that do not exhibit high spatial variability within a specified geographical area (US EPA, 2009a section 2.2.3).

The challenge in distinguishing between PM associated effects due to past and recent exposures is also relevant to the identification of the latency period for long-term exposure to PM and resultant health effects, and as the second draft ISA notes, studies assessing the relationship between long-term exposure to PM and mortality have also tried to identify the latency period between exposure to PM and death (US EPA, 2009, section 7.6.4). In the prior

review, EPA assessed several studies that used different air quality periods for estimating long-term exposure and tested associations with mortality for the different exposure periods (US EPA, 2005, p. 3-55. In the first 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. In another study of the ACS that was discussed in the prior review, Lipfert et al. (2000) analyzed mortality and PM data from different time segments, and reported varied results, with some statistically significant negative associations. Furthermore, 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 (US EPA, 2004, p. 8-115).

In the recent extended follow-up analysis of ACS data, Krewski et al. (2009) utilized similar air quality data and assigned ACS participants to $PM_{2.5}$ exposure periods as average concentrations from three successive five-year periods prior to the date of death (i.e., years 1–5; years, 6-10; years 11-15). The investigators reported that the differences in mortality risk estimates between the 5-year exposure periods were minor, and no pattern was evident from the results. The second draft ISA notes the highly correlated nature of the air quality data across the 3 exposure periods (as similarly noted in the aforementioned discussion of two exposure periods evaluated by Pope et al., 2002) may have obscured potential differences in effect estimates (US EPA, 2009a, section 7.6.4).

In addition to the ACS cohort, investigators have also utilized data from the Six Cities study to evaluate exposure periods within the context of a prospective study. The prior review discussed an analysis of the Six Cities data evaluating mortality risk with different estimates of long-term $PM_{2.5}$ exposure (US EPA, 2005, section 3.6.5.4). Villeneuve et al. (2002) conducted a follow-up analysis to the original Six Cities study (Dockery et al., 1993) that included $PM_{2.5}$ data from more recent years and evaluated associations with $PM_{2.5}$ averaged over a range of time periods, such as 2 or 3-5 years preceding the individual's death. The authors reported effect estimates for mortality that 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. The authors observed 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.

In a recent analyses of the Six Cities Study, Schwartz et al. (2008) used model averaging (i.e., multiple models were averaged and weighted by probability of accuracy) to assess exposure periods prospectively. The exposure periods were estimated across a range of unconstrained distributed lag models (i.e., same year, year prior, two years prior, etc.). In comparing lags (US EPA, 2009a, Figure 7-10) the authors reported the effects of changes in exposure to $PM_{2.5}$ on mortality are strongest within a two year period. Similarly, a large multi-city study of the elderly found that the mortality risk associated with long-term exposure to PM_{10} reported cumulative effects that extended over the follow-up year and 3 preceding years (Zanobetti et al., 2008). In a study of two locations that experienced an abrupt decline in PM levels (i.e. Utah Steel Strike, coal ban in Ireland), Röösli et al. (2005) reported that approximately 75% of health benefits were observed in the first 5 years (US EPA, 2009a Table 7-9). Furthermore, Schwartz et al. (2008) and Puett et al. (2008) found, in a comparison of exposure periods ranging from 1 month to 48 months prior to death, that exposure to PM_{10} 24 months prior to death exhibited the strongest association, and the weakest association was reported for exposure in the time period of 1 month prior to death.

Overall, the evidence for determining the window over which the mortality effects of long-term pollution exposures occur suggests a latency period of up to five years, with the strongest results observed in the first two years. In addition, the evidence from one study indicated that the exposure to PM exhibited a larger cumulative effect on mortality that was spread over the follow-up year and 3 preceding years (US EPA, 2009a, section 2.4.2.3). For use in quantitative assessments, staff concludes that it is 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 $PM_{2.5}$ quantitative risk assessment. Staff notes that this is consistent with the HEI Subcommittee's recommendation to use the results of ACS cohort analyses (i.e. 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 (HEI, 2009).

3.4.2 Concentration-Response Relationships and Potential Thresholds

In assessing or interpreting public health risk associated with exposure to PM, the form of the concentration-response (C-R) function is a critical component. The prior review recognized that there are likely to be biologic thresholds for different health effects in individuals or groups of individuals with similar innate characteristics and health status. Furthermore, 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. Inter-individual variability in the relation between personal exposure to PM of ambient origin and the PM concentration measured at a monitor may also contribute to differences in observed C-R R relationships, further obscuring potential population thresholds within the range of observed concentrations (US EPA, 2004, p. 9-43, 9-44).

The prior review indicated there was no strong evidence to support a threshold for PM mortality effects (US EPA, 2009a, section 6.5.2.7). However, the prior review noted several challenges needed to be addressed in determining the shape of the C-R R function, namely the (1) limited range of PM levels; (2) heterogeneity of susceptibility in at-risk populations; and (3) the influence of error in exposure assessment. The current review continues to present evidence on the PM C-R relationship largely in the context of the associations between PM and mortality and cardiovascular morbidity endpoints, respectively (US EPA, 2009a, section 2.4.3 and 6.2.10.10). As noted in second draft ISA, short and long-term exposure studies examining the relation between PM and mortality have "consistently found no evidence for deviations from linearity or a safe threshold" (US EPA, 2009a, section 6.2.10.10).

Among the new epidemiologic studies of short-term PM exposure are several that use different modeling methods to investigate alternative forms of C-R functions and potential threshold levels. In a multi-city analysis of three C-R models (log-linear, spline, threshold), Daniels et al. (2004) a reported that the spline model, indicated a linear relation with no evidence of a threshold for risk of death for all-causes and for cardiovascular-respiratory causes in relation to PM_{10}. However, the other cause deaths (i.e., all cause minus cardiovascular-respiratory) exhibited a threshold at around 50 $\mu g/m^3$ PM_{10} (US EPA, 2009a, Figure 6-35).

For short-term studies of cardiovascular hospital admissions and ED visits, the limited studies investigating the PM C-R relation, largely as part of the model selection process, suggest a log-linear, non-threshold C-R relationship. In the last review, Schwartz and Morris (1995) found no evidence for a threshold in the dose-response relationship between short-term PM_{10} exposure and hospital admissions for IHD. Recent single- and multi-city studies of hospital

admissions and ED visits for CVD provide additional support for this finding. In an extensive multi-city analysis of the nature of the C-R relationship and potential for a threshold between PM_{10} and cardiovascular hospital admissions and ED visits, Zanobetti and Schwartz (2005) reported a near linear concentration-response between PM_{10} and MI (with a steeper slope occurring below 50 $\mu g/m^3$) and no evidence for a threshold. Overall, this limited evidence supports the use of a no-threshold, log-linear model, consistent with the observations made in studies that examined the PM-mortality relationship (US EPA, 2009a, sections 2.4.3 and 6.x)

For long-term exposure to PM and mortality, a study by Schwartz et al (2008) using Six Cities Study data (previously noted in section 3.4.x) reported that a Cox proportional hazards model fit with a penalized spline yielded a concentration-response curve between $PM_{2.5}$ and mortality that was linear, and was "clearly continuing below the level of the current U.S. air quality standard of 15 $\mu g/m^{3"}$ (US EPA, 2009a, section 7.6.4) Although Figure 6-37 of the ISA suggests a no-threshold relation between PM_{10} and mortality based on data from the APHEA project (Samoli et al, 2008), the potential influence of exposure error and heterogeneity of concentration-response across cities (i.e., regions) and seasons (i.e., within-year variability) deserves further consideration (US EPA, 2009a, section 6.5.2.7).

In summary, while staff recognizes that there likely are individual biologic thresholds for specific health responses; the overall evidence from existing studies do not support 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. 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 lower end of these ranges (or below these ranges) but cannot be detected due to variability in susceptibility across a population. Based on the above considerations, staff concludes, that it is appropriate to use the linear or log-linear concentration-response models reported in epidemiologic studies in the quantitative risk assessment. Therefore, staff has included in the quantitative risk assessment, discussed in chapter 4, analyses incorporating a linear slope without an imposed cut point.

3.4.3 Air Quality Data in Epidemiologic Studies

In general, epidemiologic studies use ambient measurements obtained at central site monitors to represent population exposures to PM of ambient origin. Many epidemiological studies use different monitoring methods for measuring PM levels, including dichotomous samplers or Harvard impactors, as well as from co-located Tapered Element Oscillating Microbalances (TEOMs) or beta attenuation monitors (BAMs), and other methods (refer to section 2.3 for more detailed descriptions of ambient PM measurement methods). In reviewing

results from studies using various PM monitoring methods, staff finds that there appear to be no systematic differences in the effect estimates related to the use of differing monitoring methods.

In consideration of the sampling frequency of PM data collection, the second draft ISA notes sampling often involves collecting filter samples once every three or six days, with approximately 17% of FRMs operating in 2007 scheduled to sample each day (USEPA, 2009a, section 3.7). Staff observes that this missing data can have a systematic effect on the results reported from epidemiologic analyses. The absence of daily monitoring data as a source of uncertainty is of particular concern for short-term studies that evaluate lag structures and distributed lags between ambient levels and associated health effects. 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. The second draft ISA notes the challenge in using less-than-everyday monitoring data to determine concentration-response (C-R) relationships in time-series analyses (US EPA, 2009a, section 3.4.1.1). Many such studies were conducted in areas where PM was monitored on a daily basis; and the availability of every-day monitoring is often cited as a basis for study location in a number of reports. Panel studies in particular typically assess the relation between a health outcome and PM levels on the day of exposure (i.e., lag 0). However, staff observes that a small number of the recent studies have been based on less frequently collected data. Multi-city studies analyzing a large number of counties with less-than-every-day-monitoring data may still have sufficient power to detect a PM associated health effect, but an absence of daily PM monitoring data for single-city studies can limit statistical significance (US EPA, 2009a, section 3.8.6.4). Consistent with the increased uncertainty in study results using less than everyday collected PM data, staff judges that greater weight should be placed on those studies with daily or near-daily PM data collected with preference for multi-city studies conducted in the U.S. and Canada for use in drawing quantitative conclusions.

3.4.4 Measurement Error and Exposure Error

Error in the measurement of ambient PM levels is an important source of uncertainty in epidemiological studies evaluating health effects associated with ambient exposures to $PM_{10-2.5}$ or $PM_{2.5}$. As noted in the prior review (US EPA, 2004 p. 8-282, 8-283), an important source of exposure-related uncertainty occurs when the effect estimate for the etiologically relevant pollutant decreases if measured with error and its significance is transferred to a surrogate. Specifically, a transfer of association from the causal pollutant to the confounder can arise if there are high levels of measurement error in the causal variable and collinearity between the two variables (US EPA, 2009a, section 3.8.x). Consequently, the presence of measurement error and collinearity between pollutants can result in the underestimation of the effects of the poorly

measured pollutant. However, an analysis discussed in the prior review noted that due to the high underlying high ratio of fine and coarse particle toxicities is it is unlikely that error in one PM measurement will result in "false negative" or "false positive" results for fine and coarse particles, respectively (US EPA, 2005, p. 3-39). Consequently, PM measurement error is unlikely to systematically attribute effects from one pollutant to another pollutant. 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.

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 instead of directly measured using a dichotomous sampler. It is likely that measurement error would increase the uncertainty of an epidemiologic association, and with greater error in $PM_{10-2.5}$ monitoring methods, any reported epidemiologic associations would be less likely to reach statistical significance (US EPA, 2005, p 3-40). Decreases in study precision would also occur even if gravimetric $PM_{10-2.5}$ mass was measured accurately, but the sources and relative composition of coarse particles are highly variable.

Exposure error is an issue that is closely linked with the preceding discussion of error in measuring PM air quality. 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 short-term temporal (usually daily) changes in ambient PM. In contrast, cohort or cross-sectional studies use air quality data averaged over a period of months to years as indicators of a community's long-term exposure to ambient PM and/or other pollutants. Exposure misclassification can result from utilizing ambient PM levels as a proxy for community average personal exposures to ambient PM. However, the second draft ISA notes that "the use of the community average ambient $PM_{2.5}$ concentration as a surrogate for the community average personal exposure to ambient $PM_{2.5}$ is not expected to change the principal conclusions from time-series and most panel epidemiologic studies that use community average health and pollution data. Several recent studies …show that the non-ambient component of personal exposure to $PM_{2.5}$ is basically uncorrelated with ambient $PM_{2.5}$ concentrations." Furthermore, with respect to long-term exposure studies, the second draft ISA concludes that "[f]or long-term studies that use differences in long-term community average ambient PM concentrations as an exposure metric, the effect of possible community-to-community differences in the average ambient exposure factor or in the average non-ambient exposure are less understood." In addition, for panel studies, the second draft concludes "the most appropriate exposure metric may depend on the health outcome measured. However, sufficient information should be

obtained to enable determining the association of the health outcome with ambient concentration, ambient exposure, non-ambient exposure, and total personal exposure (US EPA, 2009a, section 3.9.2.4).

As discussed in section 3.8.6 of the second draft ISA, one component of exposure error (i.e., misclassification) 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). In general, the result of studies evaluating exposure misclassification related to the use of PM data from central monitoring sites suggest it is likely to result in underestimation of the effect of PM exposure on health (US EPA, 2004, p. 8-288).

However, misclassification of $PM_{10-2.5}$ exposure is of particular concern since $PM_{10-2.5}$ disperses over a shorter distance than $PM_{2.5}$, and therefore is likely to incur greater exposure error over larger spatial scales as compared to ambient $PM_{2.5}$ levels that remain spatially homogenous over larger areas (US EPA, 2009a, section 3.8.4.1). For example, as noted in section x.x above, Zanobetti and Schwartz (2009) estimated $PM_{10-2.5}$ by taking the difference in county-wide averages of PM_{10} and $PM_{2.5}$, as compared to taking the difference in PM_{10} and $PM_{2.5}$ levels from co-located monitors. The absence of a significant association between $PM_{10-2.5}$ and mortality for the "dry" region in Zanobetti and Schwartz (2009), a finding observed in the same area of the U.S. by (ADD REF) may be due to measurement error arising from the use of county-wide average differences in PM_{10} and $PM_{2.5}$ levels. Therefore, 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.

In summary, there are several key exposure-related distinctions between $PM_{2.5}$ and $PM_{10-2.5}$. In section 3.8.6.3, the second draft ISA notes that $PM_{2.5}$ spatial variability is generally low at urban regional levels, with frequently high site-to-site correlations among monitors; as summarized above, although there can be differences in some specific locations. In contrast, the second draft ISA notes that $PM_{10-2.5}$ spatial variability is higher than $PM_{2.5}$ owing to the more rapid gravitational settling of larger particles, and the aforementioned low site-to-site correlations between monitors. 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.

An additional source of exposure-related uncertainty is noted in studies of PM constituents reporting inconsistent or null associations with health effects that may be, in part, due to exposure misclassification, wherein the measured PM species are acting as a surrogate, or another component is the causal agent. The second draft ISA discusses the results of two studies that indicate the health effects associated with improved exposure data on constituents is of greater significance in comparison to poorly characterized PM compositional data. Differences in PM size distribution and constituents between indoor ambient PM and outdoor ambient PM can also introduce differential or non-differential exposure misclassification, depending on the differences in the toxicity of the unmeasured PM constituents (US EPA, 2009a, section 3.9.2.4).

As was recognized in the last review, there is greater uncertainty in the relationship between ambient $PM_{10-2.5}$ measured at central monitors and individuals' exposure to ambient $PM_{10-2.5}$, based on both variability in $PM_{10-2.5}$ concentrations across an area and a decreased ability for thoracic coarse particles to penetrate into buildings. This uncertainty is likely to broaden the confidence intervals around effect estimates (US EPA, 2005, section 3.7). As noted above, a national network is not currently in place to monitor $PM_{10-2.5}$ concentrations. Ambient concentrations of $PM_{10-2.5}$ are generally determined by the subtraction of PM_{10} and $PM_{2.5}$ measurements, although methods to characterize $PM_{10-2.5}$ levels in epidemiology studies vary. As a result, uncertainties surround the concentration at which the observed associations occur. Therefore, there is greater error in ambient exposure to $PM_{10-2.5}$ compared to $PM_{2.5}$. This would tend to increase uncertainty and make it more difficult to detect effects of $PM_{10-2.5}$ in epidemiologic studies. In addition, the various differences between eastern and western U.S. counties can lead to exposure misclassification, and the potential underestimation of effects in western counties (US EPA, 2009a, section 2.3.3.2).

Because of the greater heterogeneity of $PM_{10-2.5}$, exposure measurement error is more likely to bias health effect estimates towards the null for epidemiologic of $PM_{10-2.5}$ versus PM_{10} or $PM_{2.5}$, making it more difficult to detect an effect of the coarse size fraction. In addition, models that include both $PM_{10-2.5}$ and $PM_{2.5}$ may suffer from instability due to collinearity. Further the lag structure of $PM_{10-2.5}$ effects on risk of cardiovascular hospital admissions and ED visits, as well as mortality has not been examined in detail (US EPA, 2009a, section 6.2.12.2).

3.4.5 Alternative Model Specifications

3.4.5.1 Time-series epidemiologic studies

The prior review addressed in great detail statistical modeling issues for epidemiologic studies (USA EPA, 2005 p. 3-42 – 3-43). Of particular concern were the default convergence

criteria and standard error calculations made using Generalized Additive Models (GAM) in time-series studies (US EPA, 2004 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 Generalized Linear Models (GLM) with natural smoothing splines. The results of the reanalyses were compiled and reviewed (HEI, 2003), and generally did not substantially change the findings of the original analyses. Generally, changes in effect estimates with alternative analysis methods were much smaller than the variation in effects across studies. Consequently, this evaluation led to the conclusion that PM-related effects observed in epidemiologic studies were unlikely to be seriously biased by inadequate statistical modeling (add ref).

Alternative model specifications to control for potential weather effects and temporal trends were also considered in the prior review (USA EPA, 2005, p. 3-42 – 3-43). 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. If the model does not adequately address daily changes in weather-related variables, then some effects of temperature on health would be erroneously ascribed to the pollution variable. Conversely, if the model over controls for weather, then the result will be a much less efficient estimate of the pollutant effect (US EPA, 2004, p. 8-236). This would result in incorrectly ascribing some of the true pollution effect to the temperature variable, and thereby limit the detection of a real but small pollution effect. The prior review concluded there was no clear consensus as to what constitutes appropriate control for weather and temporally related variables, while recognizing that no single approach is likely to be most appropriate in all cases.

In summary, for quantitative assessment staff continues to consider it appropriate to use results from short-term exposure studies that used either more stringent GAM or GLM analyses. Moreover, an advantage to the use of GAM in time-series is that the model selects the degree of smoothing or adjustment for weather and temporal covariates that best fits the data (US EPA, 2009a, section 7.x). The GLM approach is advantageous in allowing a more accurate calculation of standard errors. However, a small number of $PM_{10-2.5}$ studies have investigated the influence of model specification on $PM_{10-2.5}$ risk estimates, and further evaluation of $PM_{10-2.5}$ risk estimates with alternative model structures is needed to best characterize $PM_{10-2.5}$ associated health effects in conjunction with weather and other time-varying covariates (US EPA, 2009a, section 6.5.2.3).

3.4.5.2 Prospective cohort epidemiologic studies

The prior review discussed the HEI-sponsored reanalysis of data from the ACS and Six Cities prospective cohort studies that evaluated a number of issues related to long-term exposure studies. In particular, the reanalysis included 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 replication study confirmed the original investigators' findings of associations with both total and cardiorespiratory mortality (Krewski et al., 2000; US EPA, 2004,, p. 8-91), and 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 (US EPA, 2004, p. 8-92). As discussed in section 3.3.2.2.5, Krewski et al.'s recent analysis of ACS data with extended follow-up data assessed the robustness of the Cox proportional hazard model results to alternative forms (i.e. log-log) compared to the linear function for the PM-mortality relation, and adjusted for individual-level variables, and addressed spatial patterns in the data (2009). Overall, the results of sensitivity analyses did not appreciably change the previously reported association between long-term exposure to $PM_{2.5}$ and mortality among ACS participants.

3.4.6 Co-pollutant Confounding and Effect Modification

Confounding occurs when a health effect caused by one risk factor is attributed to another variable that is correlated with causal risk factor and itself is a risk factor for the health effect. Epidemiologic analyses attempt to adjust or control for these characteristics (i.e., potential confounders) that differ between exposed and non-exposed individuals (US EPA, 2009a, section 1.5.3). 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 (US EPA, 2004 p. 8-10). Effect modifiers include variables that may influence the health response to PM exposure (e.g., co-pollutants, individual susceptibility, smoking or age). Confounding and effect modification are important considerations in the evaluation of effects in a mixture of pollutants observed in epidemiological studies, but for confounding, the emphasis is on controlling or adjusting for potential confounders in estimating the independent effect of PM, while the emphasis for effect modification is on identifying and assessing the level of modulation of PM associated health effects (US EPA, 2004 p. 8-12). In addition to confounding and effect modification, animal toxicologic evidence in the prior review (Table 7-13, US EPA, 2004) and gene-PM

epidemiological evidence in the current review (p.8-11, US EPA, 2009a) indicate that pollutant mixtures or PM in conjunction with risk factors (e.g., genes) may act together, potentially having additive or synergistic health effects.

3.4.6.1 Co-pollutant Confounding

Multivariate regression models are commonly used to assess and control for potential confounding of PM associated health effects by gaseous copollutants (US EPA, 2009a, section 1.5.3). There are several statistical issues influencing results generated using multi-pollutant models, including poorly fit models that force all pollutants to follow the same lag structure in short-term PM exposure studies, adding correlated but non-causal variables, or omitting important variables. Moreover, inclusion of pollutants in a multi-pollutant model that are highly correlated with one another can lead to erroneous pollutant effect estimates, and, thus, incorrectly identify 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 (US EPA, 2009a section 3.3.2). For example, sources of fine particle constituents include combustion of various fuels, gasoline or diesel engine exhaust, and some industrial processes (US EPA, 2009a section 3.3.3); 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 multivariate regression models may result in instability in the results, including an under- or over-estimation of effects for one or both pollutants.

Multi-pollutant model results for the risk of hospital admissions and ED visits for cardiovascular and respiratory diseases, respectively, with $PM_{2.5}$,$PM_{10-2.5}$, and gaseous co-pollutants are presented in Figures 6-5 and 6-15 of the second draft ISA. Overall, for cardiac hospitalizations the addition of SO_2 and O_3 had little influence on PM associations, although substantial reduction in associations with $PM_{2.5}$ could be seen in some cases when NO_2 and CO are added to the model (Tolbert et al., 2007). Few studies provide evidence on the effects of one PM size fraction in models adjusting for the associated effect of another PM size fraction. Specifically, the MCAPS study reported the effect estimate for $PM_{10-2.5}$ associated with cardiovascular hospital admissions lost precision when a covariate for $PM_{2.5}$ was included in the same model (Peng et al., 2008). Studies evaluating PM_{10} associated health effects using multi-pollutant models observe inconsistent results after controlling for gaseous pollutants or other size fractions, which the second draft ISA posits is "likely due to differences in the correlation structure among pollutants as well as differing degrees of exposure measurement error" (US EPA, 2009a, section 6.2.10.9).

For respiratory hospital admissions, several Canadian studies report greater effect estimates for $PM_{10-2.5}$ compared to $PM_{2.5}$ that are robust to adjustment for gaseous pollutants (US EPA, 2009a, section 6.3.8.5. Chen et al. (2004) observed $PM_{2.5}$ and $PM_{10-2.5}$ effects that are slightly attenuated but remain positive with adjustment for NO_2, whereas other studies report inconsistent results with adjustment for gaseous co-pollutants (Ito et al., 2003; Moolgavkar et al., 2003; Delfino et al., 1998). In multi-city analysis, the MCAPS study reported robust effects for $PM_{2.5}$ in models adjusted for $PM_{10-2.5}$, but $PM_{10-2.5}$ effect estimates were attenuated for respiratory admissions with adjustment for $PM_{2.5}$ (Peng et al., 2008). PM_{10} effect estimates for respiratory diseases were robust to adjustment for gaseous co-pollutants in several recent studies (Tolbert, 2007; Ulirsch et al., 2007; Anderson and Bogdan, 2007).

As noted in section 3.3.2.1.4, a key limitation of the multi-city U.S.-based studies is the absence of evidence on gaseous co-pollutants potentially influencing the association between short-term exposure to $PM_{2.5}$ and mortality. However, as discussed in the prior review, 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 (US EPA, 2004, p. 8-35). The relationship between PM_{10} and mortality was similar in models including control for O_3 and other gaseous pollutants as compared to single-pollutant models (US EPA, 2004, 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).

In the long-term exposure studies, multi-pollutant models were tested in the reanalysis of data from the ACS cohort. The reanalysis indicated 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 (US EPA, 2004, p. 9-37). In the current review, a case-crossover multi-city study matched PM_{10} levels to control days that had gaseous pollutant levels for SO_2, NO_2, O_3, and CO, respectively and estimated the excess risk for the average of 0- and 1-day lag PM_{10} (Schwartz et al., 2004). Since the authors only presented PM_{10} risk estimates matched by gaseous pollutants, the influence of gaseous pollutants on PM_{10} risk estimates is unclear. However, the study results suggest that PM_{10} is significantly associated with all-cause mortality with adjustment for each of the gaseous co-pollutants included in the analysis (US EPA, 2009a, section 6.X).

As noted in the prior review, exposure studies collected personal and ambient monitoring data to evaluate whether co-pollutants can confound PM exposure assessments (US EPA, 2005, p. 3-45). Investigators assessed the degree of day-to-day correlation between the different measures of personal and ambient concentrations and 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, suggesting that some gases may serve as a proxy for exposures to other air pollutants (US EPA, 2009a, section 3.8.X). Similarly, a recent study observed personal exposures to ambient $PM_{2.5}$ were significantly associated with ambient concentrations of $PM_{2.5}$, NO_2, and O_3 (inversely), and personal exposures to SO_4 were significantly associated with ambient $PM_{2.5}$ and O_3 levels (Schwartz et al., 2007). In contrast, Sarnat et al. (2001) reported personal and ambient concentrations of gaseous pollutants are not well correlated, potentially due to levels observed below detection limits of personal monitors. 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; US EPA, 2004, p. 5-90,).

"There is evidence that associations between ambient gases and personal exposure to $PM_{2.5}$ of ambient origin exist but are complex and vary by season and region" (US EPA, 2009a, section 3.8.x).

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 6-5 and 6-15 of the second draft ISA. In addition, evidence on potential co-pollutant confounders of PM exposure assessments suggests that it is unlikely that ambient $PM_{2.5}$ exposure associated health effects are in actuality, 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 ISA concludes that while research questions remain, in general, "associations for various PM indices with mortality or morbidity are robust to confounding by co-pollutants." (US EPA, 2009a, section x.x). 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.

3.4.6.2 Effect Modification

As noted in the prior review, one approach to evaluating the potential modulating effect of co-pollutants on associations reported with $PM_{2.5}$ involves observing a consistent association in a variety of locations with differing levels of co-pollutants (US EPA, 2005, pg 3-46,). Effect

estimates for PM_{10}-mortality associations plotted against concentrations of gaseous pollutants in the prior review yielded no evidence that associations reported between PM_{10} and mortality were correlated with co-pollutant concentrations (US EPA, 2005 Figure 3-3). Specifically, the magnitude and statistical significance of the associations reported between $PM_{2.5}$ and mortality in these studies did not exhibit 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.

The current review examines several studies that assessed whether differences in effect estimates between $PM_{2.5}$ and morbidity or PM_{10} and mortality are due to variations in $PM_{2.5}$ species (US EPA, 2009a, section 6.6.x). One study of short-term $PM_{2.5}$ exposure associated with morbidity effects reported statistically significant associations between the county average concentrations of V, Ni, and EC (106 counties) and effect estimates for both cardiovascular and respiratory hospital admissions (Bell et al., 2009). In the same study, an analysis for PM_{10} associated mortality risk estimates indicated Ni was the only PM constituent that increased the risk estimate, although this result was highly influenced by New York City data. Similarly, other analysis of PM_{10} associated mortality risk observed Ni and V modified PM_{10} mortality risk estimates (Lippmann et al. 2006; Dominici et al. 2007) but were also influenced by New York City data (Dominici et al. 2007).

In a secondary analysis of $PM_{2.5}$ associated mortality, investigators found Al, As, Ni, Si, and SO_4 were significant effect modifiers of $PM_{2.5}$ risk estimates (Franklin et al. 2008, p. 6-277, US EPA, 2009a). In Lippman et al (2006) and Franklin et al (2008) PM_{10} and $PM_{2.5}$ associated mortality, respectively, were associated with 14 similar species (Figure 6-31 and Table 6-14, US EPA, 2009a), and both studies reporting that Ni explained the heterogeneity in PM risk estimates. In addition to Ni, sulfate, SO_4 is noted as a significant predictor of $PM_{2.5}$ risk estimates (Franklin et al, 2008). However, as noted in the ISA there are methodological limitations in analyses of PM chemical species as effect modifiers (pg 6-279, US EPA, 2009a). Specifically, PM constituents acting as effect modifiers may be confounded by demographic factors that vary across cities. Therefore, the ISA posits that direct associations between PM chemical components and health effects can provide substantive evidence of effect modification of exposure to PM and associated health effects by PM constituents (add ref).

3.5 PM-RELATED IMPACTS ON PUBLIC HEALTH

The following discussion draws from section 8.1 from the ISA to characterize populations potentially at-risk for PM-related effects and the potential public health impacts associated with exposure to ambient PM.

3.5.1 Potentially At-Risk Populations

The ISA summarizes information on potentially susceptible or vulnerable groups in section 8.1. Interindividual variation in human responses to air pollutants indicates that some population groups are at increased risk for the detrimental effects of ambient exposure to PM. The NAAQS are intended to provide an adequate margin of safety for sensitive populations, or those groups potentially at increased risk for health effects in response to ambient air pollution. To facilitate the identification of populations at the greatest risk for PM-related health effects, studies have evaluated factors that contribute to the susceptibility or vulnerability of an individual to PM. The terms *susceptibility* and *vulnerability* have been used to characterize populations with a greater likelihood of an adverse outcome given a specific exposure in comparison with the general population. This increased likelihood of response to PM can result from a multitude of factors, including genetic or developmental factors, lifestages (i.e., childhood or old age) gender differences, or preexisting disease states. In addition, new attention has been paid to the concept of some population groups having increased responses to pollution-related effects due to factors including socioeconomic status (SES) (e.g., reduced access to health care) or particularly elevated exposure levels (such as living near a roadway or pollution source). It should be noted that in some cases the factors that underlie susceptibility or vulnerability of a population group to PM may intersect or overlap. For example, a population group that is characterized as having low SES may have less access to healthcare resulting in the manifestation of a disease, and they may also reside or work in locations that result in exposure to higher concentrations of PM.

The terms susceptibility and vulnerability have sometimes been used interchangeably in the literature, and as described above, have also been defined to represent two different categories that could contribute to a population group experiencing increased risk to PM-related health effects, resulting in the lack of a clear and consistent definition (US EPA, 2009a, Table 8-1). The term "at-risk" has also been used to encompass these concepts more generally. This policy assessment document uses the term "at-risk" to represent population groups that have a greater likelihood of experiencing health effects related to PM exposure, since this term implicitly encompasses both categories that contribute to increased risk.

To examine whether PM differentially affects certain population groups, epidemiologic studies conduct stratified analyses to identify the presence or absence of effect modification. A thorough evaluation of potential effect modifiers may help identify populations that are at greater risk from exposure to PM. These analyses require the proper identification of confounders and their subsequent adjustment in statistical models, which helps separate a spurious association from a true causal association. Although the design of toxicological and controlled human exposure studies does not allow for an extensive examination of effect modifiers, the use of

animal models of disease and the study of individuals with underlying disease or genetic polymorphisms do allow for comparisons between groups and an elucidation of the underlying mechanisms of susceptibility. Therefore, the results from these studies, combined with those results obtained through stratified analyses in epidemiologic studies, contribute to the overall weight of evidence for the increased susceptibility of specific population groups to PM.

3.5.1.1 Older Adults

Evidence for PM-related health effects in the older adult life stage spans epidemiologic, controlled human exposure, and toxicological studies. The 2004 PM AQCD found evidence for increased risk of cardiovascular effects in older adults exposed to PM. Older adulthood represents a life stage that is potentially at greater risk due to the higher prevalence of pre-existing cardiovascular and respiratory diseases found in this age range compared to younger age groups, primarily due to the gradual decline in physiological processes as part of the aging process. Therefore, some overlap exists between the life stage of older adults and the population group that includes people with pre-existing diseases.

In recent publications, the epidemiologic evidence for cardiovascular morbidity effects in older adults in response to short-term exposure to PM10-2.5 and PM2.5 is limited, but taken together with evidence from studies of PM10 (i.e., Le Terte et al., 2002; Larrieu et al., 2007; and Lanki et al., 2006), supports the increased risk of cardiovascular morbidity in older adults. However, a clear pattern of positive associations only being observed in epidemiologic studies conducted in non-US locations brings into question the influence of PM composition on health effects. Although studies have not consistently found an association between short-term exposure to PM and respiratory-related health effects in older adults, some studies have reported an increase in respiratory hospital admissions in individuals 65 years of age and older (Fung, 2005; Andersen, 2007).

Recent epidemiologic studies have also found that individuals greater than 65 years old are at greater risk of all-cause (non-accidental) mortality upon short-term exposure to both $PM_{2.5}$ (Franklin, 2007; and Ostro, 2006) and PM_{10} (Zeka, 2006; and Samoli et al. 2008), which is consistent with the findings of the 2004 PM AQCD. Epidemiologic studies that examined the association between mortality and long-term exposure to PM (i.e., $PM_{2.5}$) have found results contradictory to those obtained in the short-term exposure studies. Villeneuve et al. (2002), Naess et al. (2007b), and Zeger et al. (2008) report evidence of differing $PM_{2.5}$ relative risks by age, where risk declines with increasing age starting at age 60 until there is no evidence of an association among people ≥85 years of age.

Additional evidence for an increase in cardiovascular and respiratory effects among older adults has been observed in controlled human exposure and dosimetric studies. Devlin et al.

(2003) found that older subjects exposed to $PM_{2.5}$ CAPs experienced significant decreases in HRV (both in time and frequency) immediately following exposure, when they compared their results to a previous study which used healthy young subjects (Ghio et al., 2000).

In addition, Gong et al. (2004) reported that older subjects demonstrated significant decreases in HRV when exposed to $PM_{2.5}$ CAPs, but this study did not compare the response in older subjects to those elicited by young, healthy individuals. However, the study did find that healthy older adults were more susceptibe to decreases in HRV compared to those with an underlying health condition (i.e., COPD) (Gong et al. 2004). Dosimetric studies have shown a depression of fine and coarse PM clearance in all regions of the respiratory tract with increasing age beyond young adulthood in humans and laboratory animals. These results suggest that older adults are also at greater risk of PM-related respiratory health effects (Section 4.3.4.1).

Animal toxicological studies have attempted to characterize the relationship between age and PM-related health effects through the development of models that mimic the physiologic conditions associated with older individuals. For example, Nadziejko et al. {Nadziejko, 2004} observed arrhythmias in older, but not younger, rats exposed to $PM_{2.5}$ CAPs. In addition, other studies (Tankersley, 2004, 2007 and 2008) that used a mouse model of terminal senescence observed various cardiovascular-related responses. Overall, these studies provide biological plausibility for the increase in cardiovascular effects in older adults observed in the controlled human exposure and epidemiologic studies.

The evidence from epidemiologic, controlled human exposure, and toxicological studies provide coherence and biological plausbility for the association between short-term exposure to PM and cardiovascular morbidity in older adults. As noted above, a clear pattern of positive associations only being observed in epidemiologic studies conducted in non-U.S. locations brings into question the influence of PM composition on health effects. The additional evidence from epidemiologic studies that focus on mortality and respiratory morbidity in response to short-term exposure to PM also indicate that older adults represent an at-risk life stage of the population.

3.5.1.2 Children

Childhood represents a life stage that has generally been considered at greater risk from exposure to PM due to multiple factors. The factors include the following: children spend more time spent outdoors; children have greater activity levels than adults; children have exposures resulting in higher doses per body weight and lung surface area; and also because of the potential for irreversible effects on the developing lung (2004 PM AQCD). The 2004 PM AQCD found that studies which stratify results by age typically report associations between PM and respiratory-related health effects in children, specifically asthma. Of the recent epidemiologic

studies evaluated, only a few have examined the association between $PM_{10-2.5}$ and $PM_{2.5}$ and respiratory effects in children. Mar et al. (2004) found increased respiratory effects (e.g., wheeze, cough, lower respiratory symptoms) in children 7-12 years of age compared to individuals 20-51 years of age in response to exposure to both $PM_{10-2.5}$ and $PM_{2.5}$ in Spokane, Washington. In addition, Host et al. (2007) found an increase in respiratory-related hospital admissions with short-term exposure to $PM_{10-2.5}$ among children ages 0-14 years in 6 French cities. Further support for these effects is provided by the results from studies that focused on PM10 (Peel et al., 2005; and Mar et al., 2004). Recent toxicological studies (Mauad et al., 2008; and Pinkerton et al., 2008) provide biological plausibility for the increase in PM-related respiratory effects in children observed in the epidemiologic studies. Collectively, the evidence from epidemiologic studies that have examined the health effects associated with all size fractions of PM and toxicological studies that have examined individual PM components provide additional support to the hypothesis that children are at greater risk of respiratory effects from short-term exposure to PM.

Recent studies of long-term exposure to $PM_{2.5}$ have expanded the evidence for effects on lung development in children including extended follow-up for the Southern California Children's Health Study including evidence that respiratory morbidity effects from exposure to $PM_{2.5}$ persist into early adulthood, and are more robust and larger in magnitude than reported in the prior review. New longitudinal studies following effects on pulmonary function over time in other locations using different methods provide enhanced evidence for effects related to PM exposures, indicating that children are at greater risk of potentially permanent respiratory effects from long-term exposure to PM.

3.5.1.3 Pregnancy and Developmental Effects

Pregnant women represent an at-risk population group due to the potential effect of environmental contaminants on the developing fetus. While the majority of the literature focuses on epidemiologic studies that examine the potential health effects (e.g., low birth weight, growth restriction) attributed to in utero exposure to PM (see Section 7.4), it is unclear if the health effects observed are due to soluble fractions of PM that cross the placenta or physiologic alterations in the pregnant woman. In the case of exposure to PM, adverse health effects in the offspring could be mediated by a health response in the pregnant woman.

3.5.1.4 Gender

The 2004 PM AQCD did not find consistent evidence of a difference in health effects by gender. However, there appeared to be gender differences in the localization of particles when deposited in the respiratory tract and the deposition rate due to differences in body size, conductive airway size, and ventilatory parameters (U.S. EPA, 2004). For example, females

have proportionally smaller airways and slightly greater airway reactivity than males (Yunginger et al. 1992).

Few recent epidemiologic studies have conducted gender-stratified analyses when examining the association between either short- or long-term exposure to $PM_{10-2.5}$ or $PM_{2.5}$. Similar to the studies evaluated in the 2004 PM AQCD, the current literature has not found a consistent pattern of associations by gender for any health outcome. The majority of the epidemiologic studies that examined the association between exposure to PM and gender focused on exposure to PM_{10}. Although most of these studies do not attribute the association to specific size fractions (i.e., $PM_{10-2.5}$ or $PM_{2.5}$) or provide insight as to whether one size fraction may be driving the observed effect, the studies of PM_{10} provide further support that gender does not appear to differentially affect PM-related health outcomes. Overall, the evidence from studies that examined the association between short- and long-term exposure to $PM_{10-2.5}$ and $PM_{2.5}$, along with the supporting evidence from PM_{10} studies, further confirms that although differences in dosimetry exist between males and females, neither consistently exhibit a higher disposition for PM-related health effects.

3.5.1.5 Race/Ethnicity

The 2004 PM AQCD did not evaluate the potential susceptibility and/or vulnerability of individuals of different races and ethnicities. The results from epidemiologic studies evaluated in this review that examined the potential effect modification of the PM-morbidity and -mortality relationships by race and ethnicity varied depending on the study location. In an analysis of the $PM_{2.5}$-mortality relationship, Ostro et al. (2006) stratified the association by race and ethnicity, and observed a positive and significant effect for whites and Hispanics, but not for blacks, in response to short-term exposure to $PM_{2.5 \text{ in 9 California counties}}$. An additional analysis performed by Ostro et al. (2006) in 6 California counties using $PM_{2.5}$ and various $PM_{2.5}$ components, also found a significant association between mortality, specifically cardiovascular mortality, and Hispanic ethnicity (Ostro, 2008). Additional evidence for the potential susceptibility and vulnerability of individuals by race and ethnicity were derived from studies on the health effects associated with short-term exposure to PM_{10}. Wellenius et al., (2006) observed that race (i.e., white vs. other) did not significantly modify the association between short-term exposure to PM_{10} and CHF hospital admissions. Additionally, Zeka et al., (2006) did not observe any difference in mortality effect estimates when stratifying by race (i.e., black and white) upon short-term exposure to PM_{10}. Overall, the results from the studies that examined the potential effect modification of PM risk estimates by race and ethnicity provide some evidence for increased risk of mortality in Hispanics upon short-term exposure to $PM_{2.5}$. However, it should

be noted that both of these studies were conducted in California and additional studies in other locations with race and ethnicity stratified analysis have not yet been conducted.

3.5.1.6 Gene-Environment Interaction

A consensus now exists that gene-environment interactions merit serious consideration during the examination of the relationship between ambient exposures to air pollutants and the development of health effects (Kauffmann, 2004; and Gilliland, 1999). These potential interactions were not evaluated in the 2004 PM AQCD. Inter-individual variation in human responses to air pollutants suggests that some population groups are at increased risk of detrimental effects due to pollutant exposure, and it has become clear that the genetic makeup of an individual can increase their susceptibility (Kleeberger, 2005). Gene-environment interactions can result in health effects due to: genetic polymorphisms, which result in the lack of a protein or a change that makes a functionally important protein dysfunctional; or genetic damage in response to an exposure which potentially leads to a health response. In this review, the majority of studies examine gene-environment interactions due to genetic polymorphisms. In order to establish useful links between polymorphisms in candidate genes and adverse health effects, several criteria must be satisfied: the product of the candidate gene must be significantly involved in the pathogenesis of the adverse effect of interest; and polymorphisms in the gene must produce a functional change in either the protein product or in the level of expression of the protein (US EPA, 2008). Further, the issue of confounding by other environmental exposures must be carefully considered.

It has been hypothesized that the cardiovascular and respiratory health effects that occur in response to short-term exposure to PM are mediated by oxidative stress (US EPA, 2009a, section 4.3). Research has examined this hypothesis by primarily focusing on the GST genes because they have common, functionally important polymorphic alleles that significantly affect antioxidant defense function in the lung. Exposure to radicals and oxidants in air pollution leads to a cascade of events, which can result in a reduction in glutathione (GSH), and an increase in the transcription of GSTs. Individuals with genotypes that result in reduced or absent enzymatic activity are likely to have reduced antioxidant defenses and potentially increased susceptibility to inhaled oxidants and radicals. However, in some cases genetic polymorphisms may actually reduce an individual's susceptibility to PM-related health effects. Overall, the evidence from epidemiologic and controlled human exposure studies suggests that specific genetic polymorphisms can increase the susceptibility of an individual to PM exposure, but protective polymorphisms also exist, which may diminish the health effects attributed to PM exposure in some individuals.

3.5.1.7 Pre-Existing Disease

In 2004, the National Research Council (NRC) published a report that emphasized the need to evaluate the effect of air pollution on at-risk population groups, including those with respiratory illnesses and cardiovascular diseases (NRC, 2004). The 2004 PM AQCD included epidemiologic evidence suggesting that individuals with pre-existing heart and lung diseases, as well as diabetes may be at greater risk from exposure to PM. In addition, toxicological studies that used animal models of cardiopulmonary diseases and heightened allergic sensitivity also found evidence of increased risk. More recent epidemiologic and human clinical studies have directly examined the effect of PM on individuals with pre-existing diseases and toxicological studies have employed disease models to identify whether exposure to PM disproportionately affects certain population groups.

3.5.1.7.1 Cardiovascular Diseases

The potential effect of underlying cardiovascular diseases on PM-related health responses has been examined using epidemiologic studies that stratify effect estimates by underlying conditions or secondary diagnoses, and toxicological studies that use animal models to mimic the physiological conditions associated with various cardiovascular diseases (e.g., MI, ischemia, and atherosclerosis). A limited number of controlled human exposure studies have also examined the potential relationship between cardiovascular diseases and exposure to PM in individuals with underlying cardiovascular conditions, but these studies have provided somewhat inconsistent evidence for these associations.

The majority of the epidemiologic literature that examined associations between short-term exposure to PM and cardiovascular outcomes focuses on cardiovascular-related hospital admissions (HA) and emergency department (ED) visits. Hypertension is the pre-existing condition that has been considered to the greatest extent when examining the association between short-term exposure to PM and cardiovascular-related HAs and ED visits. The results have been mixed, with several studies finding that hypertension did not modify the association between IHD ED visits and $PM_{2.5}$ in individuals with secondary hypertension in Utah (Pope et al., 2006), or the association between PM_{10} and cardiovascular-related health outcomes in 7 U.S. cities (Wellenius et al., 2006) and Taipei (Lee et al., 2008). These results differ from those presented by Peel et al. (2007), in Atlanta, which observed that exposure to PM_{10} resulted in an increase in ED visits for arrhythmias and CHF in individuals with underlying hypertension, and Park et al. (2005) in Boston found that underlying hypertension increased associations between HRV, specifically a reduction in the HF parameter, and short-term exposure to $PM_{2.5}$.

Park et al. (2005), in the analysis mentioned above, examined other underlying cardiovascular conditions and found associations between $PM_{2.5}$ and HRV in individuals with

pre-existing IHD. In a toxicological study, Wellenius et al. (2003) examined the effects of $PM_{2.5}$ CAPs exposure on induced myocardial ischemia in dogs, which mimics the effects associated with IHD. The authors found that exposure to $PM_{2.5}$ prior to the induced ischemia increased ST-segment elevation, indicating greater ischemia than air-exposed animals. A follow-up study implicated impaired myocardial blood flow in the response (Bartoli et al., 2009).

Additional studies examined the effects of PM on cardiac function in individuals with dysrhythmia with mixed results. One study observed some evidence for an increase in ED visits for IHD for individuals with secondary dysrhythmia and PM_{10} exposure (Peel et al., 2007), while another study found no evidence for effect modification of PM_{10} exposure in individuals with secondary dysrhythmia when examining CHF hospital admissions in 7 U.S. cities (Wellenius et al., 2006).

Limited evidence is available from epidemiologic studies that examined other pre-existing cardiovascular conditions, such as CHF and MI. Pope et al. (2006) observed an increase in hospital admissions for acute IHD in individuals with underlying CHF upon short-term exposure to $PM_{2.5.}$ However, Peel et al. (2007) did not find that underlying CHF contributed to an increase in the association between IHD ED visits and short-term exposure to PM_{10}, and in another study underlying CHF was not found to increase MI hospital admissions for exposure to PM_{10} in the cohort of more than 300,000 hospital admissions (Zanobetti and Schwartz 2005).

Wellenius et al. (2006) examined the effect of previous diagnoses of acute MI, and found no evidence of effect modification in the association between CHF hospital admissions and short-term exposure to PM_{10} in 7 U.S. cities. Toxiciological studies have provided some additional evidence for the cardiovascular health effects associated with exposure to PM in individuals with animal models of underlying MI and atherosclerosis, but the evidence is inconclusive. Controlled human exposure studies that examined the effect of pre-existing diseases on cardiovascular outcomes with exposure to PM are less consistent and difficult to interpret in the context of the results from the epidemiologic and toxicological studies.

Although the epidemiologic studies did not examine potential effect modification of pre-existing cardiovascular conditions on effects of long-term exposure to PM, a few toxicological studies exposed animals with underlying cardiovascular conditions to PM for months. In studies that focused on the cardiovascular effects following subchronic exposure to PM in $ApoE^{-/-}$ mice, relatively consistent physiological effects were observed across studies. Mice exposed to ultrafine and to $PM_{2.5}$ CAPs demostrated enhanced size of early atherosclerotic lesions (ultrafine CAPS: Araujo et al., 2008; $PM_{2.5}$ CAPs: Chen and Nadziejko, 2005; Sun et al., 2005; Sun et al., 2008). An additional long-term exposure study observed a decreasing trend in heart rate, physical activity, and temperature along with biphasic responses in HRV (SDNN and rMSSD) upon exposure to CAPs (Chen and Hwang, 2005).

While the majority of the literature examines the potential modification of the association between PM and non-fatal cardiovascular health effects, a few new studies have also examined effect modification in mortality associations. Zeka et al. (2006) found an increase in risk estimates for associations between PM_{10} and mortality in individuals with underlying stroke, while Bateson et al. (2004) found evidence for effect modification of the PM-mortality association in individuals with CHF.

Collectively, the evidence from epidemiologic and toxicological, and to a lesser extent, controlled human exposure studies indicates increased susceptibility of individuals with underlying cardiovascular diseases to PM exposure. Although the evidence for some outcomes was inconsistent across epidemiologic and toxicological studies, this could be due to a variety of issues including the PM size fraction used in the study along with the study location.

3.5.1.7.2 Respiratory Illnesses

Investigators have examined the effect of pre-existing respiratory illnesses on multiple health outcomes (e.g., mortality, asthma symptoms, CHF) in response to exposure to ambient levels of PM. Animal models have been developed and human clinical studies conducted to examine the possible PM effects on pre-existing respiratory conditions in a controlled setting.

Epidemiologic studies have examined the effect of short-term exposure to PM on the respiratory health of asthmatic individuals measuring a variety of respiratory outcomes. Asthmatic individuals were found to have an increase in medication use (Rabinovitch et al., 2006), respiratory symptoms (i.e., asthma symptoms, cough, shortness of breath, and chest tightness (Gent et al., 2003), and asthma symptoms (Delfino et al., 2002; 2003) with short-term exposure to $PM_{2.5}$; and morning symptoms (Mortimer et al., 2002) and asthma attacks (Desqueyroux et al., 2002) with short-term exposure to PM_{10}.

Toxicological studies that have used ovalbumin-induced allergic airway disease models provide evidence which supports the findings of the epidemiologic literature. These findings suggest that individuals with allergic airways conditions are at greater risk of allergic airways responses upon exposure to $PM_{2.5}$, which may be partially attributed to increased pulmonary deposition and localization of particles in the respiratory tract (Morishita et al., 2004). An additional study (Heidenfelder et al., 2009) examined whether genes are differentially expressed upon exposure to PM. They found that exposure to CAPs increased the expression of genes associated with inflammation and airway remodeling in rats with allergic airway disease. Although the evidence is much more limited, not all of the toxicological studies evaluated that examined the effect of underlying respiratory conditions on PM-related respiratory morbidity focused on allergic airways disease. These provide preliminary evidence, which suggests that

non-allergic respiratory morbidities may also increase the susceptibility of an individual to PM-related respiratory effects.

The results from the epidemiologic and toxicological studies that focused on underlying allergic airways disease is supported by a series of controlled human exposure studies which have shown that exposure to DEPs increases the allergic inflammatory response in atopic individuals (Bastain et al., 2003; Diaz-Sanchez et al., 1997; Nordenhall et al., 2001). However, not all controlled human exposure studies have found evidence for differences between the respiratory effects exhibited by healthy and asthmatic individuals. Studies by Gong et al. (2003; 2004; 2008) reported that healthy and asthmatic subjects exposed to coarse, fine and ultrafine CAPs, exhibited similar respiratory responses. However, it should be noted that these studies excluded moderate and severe asthmatics that would be expected to show increased susceptibility to PM exposure.

In addition to examining the association between exposure to PM and respiratory effects in asthmatics, some studies examined whether individuals with COPD represent a potentially at-risk population group. Desqueyroux et al. (2002) did not observe an increase in the exacerbation of COPD in response to short-term exposure to $PM_{2.5}$. However, studies that examined the effect of PM on lung function in individuals with COPD (Lagorio et al., 2006; Trenga et al., 2006) observed declines in FEV_1, and FEV_1 and FVC, respectively in response to PM_{10} and/or $PM_{2.5}$. Silkoff et al. (2005) observed associations between PM_{10} and a reduction in FEV_1 and $PM_{2.5}$ and a reduction in PEF, in those with COPD, but only during one winter of the analysis. A few controlled human exposure studies examined the effects of PM on COPD subjects and found no significant difference in respiratory effects between healthy and individuals with COPD upon exposure to $PM_{2.5}$ CAPs (Gong et al., 2004; Gong et al., 2005; and Blomberg et al., 2005). On the other hand the results from dosimetric studies have shown that COPD patients have increased dose rates and impaired mucociliary clearance relative to age matched healthy subjects, suggesting that individuals with COPD are potentially at a greater risk of PM-related health effects (Section 3.2.1).

A few of the epidemiologic studies examined the effect of underlying respiratory illnesses on the association between short- and long-term exposure to PM and mortality. Using different pre-existing respiratory illnesses, Zeka et al. (2006) and De Leon et al. (2003) found that short-term exposure to PM_{10} increased the risk of non-accidental mortality for pneumonia and circulatory mortality for all respiratory illnesses, respectively. Additionally, Zanobetti et al. (2008) observed an association between long-term exposure to PM_{10} and mortality in individuals that had previously been hospitalized for COPD. Although these studies do not examine additional size fractions of PM, together they highlight the potential effect of underlying respiratory illnesses on the PM-mortality relationship.

Overall, the epidemiologic, controlled human exposure, and toxiciological studies evaluated provide biological plausbility for the increased health effects observed in epidemiologic studies among asthmatic individuals in response to PM exposure. The evidence from studies that examined associations between PM and health effects in individuals with COPD is inconsistent.

3.5.1.7.3 Respiratory Contributions to Cardiovascular Effects

Although the majority of health effects observed in individuals with pre-existing respiratory illnesses were associated with respiratory illness exacerbations, studies also examined whether underlying respiratory illnesses can lead to cardiovascular effects with PM exposure. Respiratory disease has not consistently been observed to affect cardiovascular response in controlled human exposure or toxicological studies. The majority of epidemiologic studies that examined whether underlying respiratory illnesses contributed to the mainfestation of PM-related cardiovascular hospital admission or ED visits, did not report increases in effects for a variety of cardiovascular outcomes (e.g., IHD, arrhythmias, CHF, MI) for individuals with underlying respiratory infection (Wellenius et al., 2006), pneumonia (Zanobetti and Schwartz, 2005), or COPD (Peel et al., 2007; Wellenius et al., 2005). However, Yeatts et al. (2007), in a panel study, found evidence for cardiovascular effects, specifically reductions in HRV parameters, in asthmatic adults upon short-term exposure to $PM_{10-2.5}$. It must be noted that most of the aforementioned epidemiologic studies focused on exposure to PM_{10}, and, therefore, it is unclear how these results compare to those found in the controlled human exposure and toxicological studies that focused on exposure to $PM_{2.5}$ (e.g., CAPs). Thus, it is unclear if individuals with underlying respiratory illnesses represent a population group that is potentially at greater risk of PM-related cardiovascular effects.

3.5.1.7.4 Diabetes and Obesity

It has been hypothesized that the systemic inflammatory cascade leads to an increase in cardiovascular risk (Dubowsky et al., 2006). As a result, individuals with conditions linked to chronic inflammation (i.e., diabetes and obesity), have been examined to determine whether diabetes or obesity facilitate the manifestation of PM-mediated health effects, and, therefore, represent a potentially at-risk population group.

Epidemiologic studies have examined whether diabetes modifies the association between cardiovascular health effects and PM exposure, but these studies have primarily focused on short-term exposure to PM_{10}. Time-series studies have provided evidence through an examination of hospital admission and ED visits and mortality, which suggests an increase in health effects in diabetic individuals in response to PM exposure. Multicity studies have found upwards of 75% greater risk of hospitalization for cardiac diseases in individuals with diabetes

upon to exposure to PM_{10} (Zanobetti and Schwartz, 2002). Studies conducted in Atlanta, Georgia have also found increased risk for cardiovascular-related ED visits in diabetics, specifically for IHD, arrhythmias, and CHF (Peel et al., 2007). Additional studies found some evidence that individuals with diabetes are at increased risk of mortality upon exposure to PM_{10} (Zeka et al., 2006) and $PM_{2.5}$ (Goldberg et al., 2006). However, some studies (both multicity and single-city) have not observed a modification of the risk of cardiovascular ED visits and hospital admissions in response to exposure to PM_{10} in diabetics (Pope et al., 2006; Wellenius et al., 2006; Zanobetti and Schwartz, 2005).

Panel and cohort studies have been conducted to determine the physiological changes that occur in individuals with diabetes in response to PM exposure. These studies examined both changes in inflammatory markers along with specific physiological alterations in the cardiovascular system. Schneider et al. (2008) in a panel study of 22 individuals with type 2 diabetes mellitus in Chapel Hill, NC found evidence that ambient exposure to $PM_{2.5}$ enhanced the reduction in various markers of endothelial function. Liu et al. (2007) observed an increase in end-diastolic FMD and end-systolic FMD, and decreases in end-diastolic basal diameter and end-systolic basal diameter in diabetics upon exposure to PM_{10}. The authors also observed positive associations with FMD and blood pressure in diabetic individuals. A controlled human exposure study conducted by Carlsten et al. (2008) found that DE did not elicit any prothrombotic effects in subjects with metabolic syndrome, which consists of physiological alterations similar to those observed in both diabetic and obese individuals. An examination of biomarkers found mixed results, with Liao et al. (2005) observing an increase in vWF; Liu et al. (2007) observing an increase in TBARS, but not CRP or TNF-α; and Dubowsky et al. (2006) observing an increase in CRP and WBCs. Overall, it is unclear how differences in each of the aforementioned biomarkers contribute to the potential overall cardiovascular effect observed in diabetic individuals; however, an increase in inflammation, oxidative stress, and acute phase response may contribute to cardiovascular effects. A recent toxicological study, also demonstrated the potential for PM-related health effects in diabetics. Sun et al. (2009) found that $PM_{2.5}$ CAPs exposure for 4 months can exaggerate insulin resistance, visceral adiposity, and inflammation in a diet-induced obesity mouse model.

Overall, epidemiologic studies have reported evidence for increased effects in diabetics in response to PM exposure, with preliminary evidence for physiological alterations from toxicological studies. However, the limited evidence from toxicological and controlled human exposure studies along with the lack of studies that examined additional PM size fractions warrants additional research to confirm these associations and to identify the biological pathway(s) that may result in a greater response to PM in diabetics.

In addition to diabetes, obesity has been examined as a health condition with the potential to lead to an increase in PM-related health effects. Only a few recent studies have examined the potential effect modification of PM risk estimates by obesity. Schwartz et al. (2005) reported a change in HRV in obese (i.e., BMI $\geq$ 30 kg/m^2) compared to non-obese subjects, while Dubowsky et al. (2006) observed an increase in inflammatory markers (i.e., CRP, IL-6, and WBC) in response to short-term exposure to $PM_{2.5}$ among obese individuals. Additionally, Schneider et al. (2008) found some evidence for a larger reduction in FMD in individuals with a BMI >30 kg/m^3 in response to $PM_{2.5}$ exposure. These effects could be due, in part, to a higher PM dose rate in obese individuals, which has been demonstrated in children by Bennett and Zeman (2004). These investigators also reported that tidal volume and resting minute ventilation increased with body mass index. Although a limited amount of research has been conducted to examine PM-related health effects in obese individuals there is an increasing trend of individuals within the U.S. that have been defined as overweight or obese (BMI $\geq$ 25.0) (56-65% between NHANES III and NHANES [1999-2002]).

3.5.1.8 Socioeconomic Status

Socioeconomic status (SES) is a composite measure that usually consists of economic status, measured by income; social status measured by education; and work status measured by occupation (Dutton and Levine, 1989). Based on data from the U.S. Census Bureau in 2006, from among commonly-used indicators of SES, about 12% of individuals and 11% of families are below the poverty line (U.S., 2009). Although the measure of SES is composed of a multitude of determinants, each of these linked factors can influence an individual's susceptibility to PM-related health effects. Additionally, low SES individuals have been found to have a higher prevalence of pre-existing diseases; inadequate medical treatment; and limited access to fresh foods leading to a reduced intake of antioxidant polyunsaturated fatty acids and vitamins, which can increase this population group's risk from to PM (Kan et al., 2008).

SES and individual determinants of SES, such as educational attainment, are not mutually exclusive and together can influence the susceptibility of a population. Within the U.S. approximately 16% of the population does not have a high school degree and only 27% have a bachelor's degree or higher level of education (U.S., 2009). Educational attainment generally coincides with an individual's income level, which is correlated to other surrogates of SES, such as residential environment (Jerrett et al., 2004). Low SES, and surrogates of SES such as educational attainment, have been shown in some studies to modify health outcomes of PM exposure for a population. Franklin et al. (2008) noted an increased risk in mortality associated with short-term exposure to $PM_{2.5}$ and its components for individuals with low SES while additional analyses stratified by education level have also observed consistent trends of increased

mortality for $PM_{2.5}$ and $PM_{2.5}$ species for individuals with low educational attainment (Ostro et al., 2006; Ostro et al., 2008; Zeka et al., 2006). This is further supported by a reanalysis of the ACS cohort (Krewski et al., 2009), which found moderate evidence for increased lung cancer mortality in individuals with a high school education or less compared to individuals with more than a high school education in response to long-term exposure to $PM_{2.5}$.

Epidemiologic studies have also examined additional surrogates of SES, such as residential location and nutritional status to identify their influence on the risk to a population group. Jerrett et al. (2004) examined the modification of acute mortality effects due to particulate air pollution exposure by residential location in Hamilton, Canada using educational attainment as a surrogate for SES. The authors found that the area of the city with the highest SES characteristics displayed no evidence of effect modification while the area with the lowest SES characteristics had the largest health effects. Likewise, Wilson et al. (2007) examined the effect of SES on the association between mortality and short-term exposure to PM in Phoenix, but used educational attainment and income to represent SES. When stratifying Phoenix into central, middle, and outer rings of varying urban density central Phoenix, the area with the lowest SES, was found to exhibit the greatest association with $PM_{2.5}$. However, the association with urban density differed when examining $PM_{10\text{-}2.5}$, with the greatest effect being observed for the middle ring. Yanosky et al. (2008) examined whether long-term exposure to traffic-related pollutants, using NO_2 as a surrogate, varied by SES at the block group level. The authors found higher levels of NO_2 associated with lower SES areas, which suggests that lower SES individuals are disproportionately exposed to traffic-related pollutants, which includes PM.

Nutritional deficiencies have been associated with increased susceptibility to a variety of infectious diseases and chronic health effects. Low SES may decrease access to fresh foods, and thus be related to nutritional deficiencies that could increase susceptibility to PM-related health effects. Baccarelli et al. (2008) examined the association between exposure to $PM_{2.5}$ and HRV in individuals with polymorphisms in MTHFR and cSHMT genes, which are associated with reduced enzyme activity and increased risk of CVD. The authors found that when individuals with these genetic polymorphisms increased their intake (above median levels) of B6, B12, or methionine no $PM_{2.5}$ effect on HRV was observed.

3.5.2 Potential Public Health Impacts

Upon evaluating the association between short- and long-term exposure to PM and various health outcomes, studies also attempted to identify population groups that are at greater risk from PM exposure (i.e., populations that have a greater likelihood of experiencing health effects related to PM exposure). These studies did so by: conducting stratified analyses; examining individuals with an underlying health condition; or developing animal models that

mimic the physiological conditions associated with an adverse health effect. These studies identified a multitude of factors that could potentially contribute to whether an individual is at-risk from PM (US EPA, 2009a, Table 8-2). The size of the population groups affected by these factors is large and many people are likely to be members of more than one at-risk population. Although studies have primarily used exposures to $PM_{2.5}$ or PM_{10}, the available evidence suggests that the identified factors may also enhance susceptibility to $PM_{10-2.5}$. The examination of population groups at-risk from PM exposure allows for the NAAQS to provide an adequate margin of safety for them.

The evidence from epidemiologic, controlled human exposure, and toxicological studies provide coherence and biological plausbility for the association between short-term exposure to PM and cardiovascular morbidity and mortality in older adults. As the demographics of the U.S. population shift over the next 20 years with a larger percentage of the population (i.e., 13% of the population in 2011 and a projected 20% in 2030) encompassing individuals ≥ 65 years {U.S. Census Bureau, 2000}, an increase in the number of PM-related health effects (e.g., cardiovascular and respiratory morbidity, and mortality) in individuals ≥ 65 years old could occur.

Overall, the evidence from epidemiologic studies that have examined the health effects associated with all size fractions of PM and toxicological studies that have examined individual PM components provide additional support to the hypothesis that children are at greater risk of respiratory effects from short-term exposure to PM. In 2008 there were approximately 73.9 million children in the U.S. This number is expected to reach 82 million children by 2021. In 2008, children comprised about 24% of the population, with approximately equal numbers of children in three age groups, 0 to 5 (25 million), 6 to 11 (24 million) and 12 to 17 (25 million) years of age (Federal Interagency Forum on Child and Family Statistics).

Collectively, the evidence from epidemiologic and toxicological, and to a lesser extent, controlled human exposure studies indicates increased susceptibility of individuals with underlying cardiovascular diseases to PM exposure. Although the evidence for some outcomes was inconsistent across epidemiologic and toxicological studies, this could be due to a variety of issues including the PM size fraction used in the study along with the study location. Even with these caveats, a large proportion of the U.S. population has been diagnosed with cardiovascular diseases (i.e., approximately 51.6 million people with hypertension, 24.1 million with heart disease, and 14.1 million with coronary heart disease [see Table 3-2]), and therefore represents a large population group that is potentially at greater risk from exposure to PM than the general population.

Overall, the epidemiologic, controlled human exposure, and toxiciological studies evaluated provide biological plausbility for the increased health effects observed in

epidemiologic studies among asthmatic individuals in response to PM exposure. Although, the evidence from studies that examined associations between PM and health effects in individuals with COPD is inconsistent, taken together individuals with COPD and asthma represent a large percent of the U.S. population, which may be more at greater risk of PM-related health effects (Table 3-2).

Table 3-2. Percent of the U.S. population with respiratory diseases, cardiovascular diseases, and diabetes.

	Age						Regional			
	Adults (18+)*		18-44	45-64	65-74	75+	NE	MW	S	W
Chronic Condition/ Disease	Number (x 10^6)	%	%	%	%	%	%	%	%	%
Respiratory Diseases										
Asthma*	24.2	11.0	11.5	10.5	11.7	9.3	11.7	11.5	10.5	10.8
Asthma (<18 yrs)	6.8*	9.3*	---	---	---	---	---	---	---	---
COPD										
Chronic bronchitis	9.5	4.3	2.9	5.5	5.6	6.7	3.8	4.4	4.9	3.5
Emphysema	4.1	1.8	0.3	2.4	5.0	6.4	1.4	2.3	1.9	1.6
Cardiovascular Diseases										
All heart disease	24.1	10.9	3.6	12.3	26.1	36.3	10.8	12.7	10.9	9.2
Coronary heart disease	14.1	6.4	0.9	7.2	18.4	25.5	6.4	7.6	6.6	4.7
Hypertension	51.6	23.4	7.7	32.4	52.7	53.5	22.2	23.7	25.3	20.6
Stroke	5.6	2.6	0.5	2.4	7.6	11.2	2.1	2.8	2.9	2.2
Diabetes	17.1	7.8	2.6	10.4	18.2	17.9	7.2	8.1	8.0	7.4

* All data for adults except asthma prevalence data for children under 18 years of age, from CDC (2008). For adults prevalence data based off adults responding to "ever told had asthma." Source: Pleis and Lethbridge-Çejku (2007); CDC (2008) (US EPA 2009a, Table 8-3).

In addition, epidemiologic studies have reported evidence for increased effects in diabetics in response to PM exposure, with preliminary evidence for physiological alterations from toxicological studies. However, the limited evidence from toxicological and controlled human exposure studies along with the lack of studies that examined additional PM size fractions warrants additional research to confirm these associations and to identify the biological pathway(s) that may result in a greater response to PM in diabetics. This potentially at-risk population group is large, with an estimated 17.1 million diabetic individuals in the U.S. (Table 3-2).

As summarized above, there are several populations groups that may be at-risk for effects from exposure to PM. Estimates of the size of population groups, such as older adults, children and people with cardiopulmonary diseases, including diabetes, that are the populations considered to be at-risk for the effects of PM exposure are large. Combining fairly small risk estimates and small changes in PM concentration with large groups of the U.S. population who are potentially at-risk would result in large public health impacts.

3.6 SUMMARY AND CONCLUSIONS

{to be included in external review draft Policy Assessment}

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4 CHARACTERIZATION OF HEALTH RISKS

We have developed a preliminary annotated outline for chapter 4 of the Policy Assessment. Chapter 4 will present a concise overview of the scope, technical approach, results, and limitations/uncertainties of the PM NAAQS risk assessment. In discussing the risk estimates in this chapter, focus will be placed on providing key observations that are most relevant to the policy assessment discussion covered in chapter 5. The organization of this chapter is similar to that of the draft Risk Assessment document (US EPA, 2009b) and we have included references to those sections in the draft Risk Assessment that will serve as the basis for specific sections in the outline below. Note, also, that this annotated outline is preliminary and the organization and content may change as we begin to develop the draft chapter.

4.1 INTRODUCTION

(Briefly set up the risk assessment in terms of its place within the overall PM NAAQS review, including goals of the assessment, overview of risk assessment from previous review, and process for developing approach for current risk assessment – RA sections 1.1 and 1.2).

4.2 SCOPE

4.2.1 Key design elements

(Highlights the key aspects of this risk assessment and takes note of modifications to the approach from the previous review – – RA section 2.4).

4.2.2 Alternative sets of standards included in the risk assessment

(Provides the set of alternative standards as well as a brief overview of the rationale behind selection of those sets of standards for inclusion in the assessment – RA section 2.5).

4.3 METHODS USED IN THE URBAN CASE STUDY ANALYSIS

4.3.1 General approach

(Provides concise summary of the risk assessment approach – i.e., epi-based risk assessment focused on estimating incidence of specific health endpoints related to $PM_{2.5}$ exposure. Clarifies that it is urban study area-based with exposure being characterized using ambient monitors as a surrogate for actual exposures. Also clarifies that risk is estimated in excess of lowest measured level (LML) or policy-relevant background (PRB) concentrations, depending on category of endpoint. Introduces approach of providing a core set of risk estimates that receive greater focus along with a set of additional/ supplemental risk estimates generated using alternative inputs and sensitivity analysis methods – RA section 3.1).

4.3.2 Air quality inputs

(Describes composite monitor approach and rollback methods used to simulate just meeting current and alternative sets of standards – RA section 3.2).

4.3.3 Selection of model inputs

(Provides brief overview of key aspects of the risk assessment model, including: selection of study areas, selection of health effects endpoints, choice of epi studies to use in deriving concentration-response functions, and specification of baseline incidence rates – RA sections 3.3 and 3.4).

4.3.4 Addressing uncertainty and variability

(Briefly describes approach used in addressing variability and uncertainty – including qualitative and quantitative elements. Includes discussion of sensitivity analysis and use of results to help identify key sources of uncertainty and to provide additional set of reasonable risk estimates to use in augmenting core risk estimates – RA section 3.5).

4.4 METHODS USED IN THE REPRESENTATIVENESS ANALYSIS

(Provides overview of approach used, including limitations and use of estimates to inform assessment of degree to which the 15 urban study areas are representative of populations in the U.S with regard to PM exposure and potentially, risk – RA section 4.4).

4.5 METHODS USED IN THE NATIONAL-SCALE PM-RELATED MORTALITY ANALYSIS

(Provides overview of approach used, including limitations of the analysis – RA section 5.2).

4.6 RESULTS

4.6.1 Core risk estimates

(Provides brief summary of risk estimates and key observations regarding the core risk estimates – RA section 4.5.1).

4.6.1.1 Recent conditions

4.6.1.2 Current set of standards

4.6.1.3 Alternative sets of standards

(Divided into discussion of (a) results for set of alternative standards focusing on annual levels: 13/35 and 12/35 and (b) set of alternative standards focusing on both annual and daily levels: 13/30 and 12/25)

4.6.2 Sensitivity analysis results

4.6.2.1 Key sources of uncertainty

(Discusses key sources of uncertainty – including the qualitative assessment of uncertainty as well as the single- and multi-factor sensitivity analyses completed - RA sections 4.3.1 and 4.3.2).

4.6.2.2 Additional set of reasonable risk estimates

(Provides observations regarding range of uncertainty resulting from consideration of alternative set of reasonable risk estimates generated as part of the single- and multi-factor sensitivity analysis. Provides observations regarding potential range of risk that might be associated with consideration of key sources of uncertainty associated with the risk assessment - RA section 4.5.2).

4.6.3 Representativeness analysis

(Provides brief summary and observations related to the representativeness analysis of the 15 urban study areas based on consideration of national distribution of PM risk-related attributes - RA section 4.5.3)

4.6.4 National-scale $PM_{2.5}$-related mortality analysis

(Provides brief summary and observations related to the national-scale $PM_{2.5}$-related mortality analysis, including discussion of both the national-scale estimate of mortality as well as observations regarding how well the 15 urban study areas capture the range of county-level $PM_{2.5}$-related mortality estimated for the U.S. - RA sections 4.5.4 and 5.3).

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5 STAFF CONCLUSIONS ON PRIMARY PM NAAQS

5.1 INTRODUCTION

5.2 APPROACH

5.3 FINE PARTICLE STANDARDS

5.3.1 Adequacy of Current $PM_{2.5}$ Standards

5.3.1.1 Evidence-based Considerations

5.3.1.2 Risk-based Considerations

5.3.1.3 Summary

5.3.2 Indicators

5.3.3 Averaging Times

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

5.3.4.1 Evidence-based Considerations

5.3.4.2 Risk-based Considerations

5.3.4.3 Summary

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

5.3.5.1 Evidence-based Considerations

5.3.5.2 Risk-based Considerations

5.3.5.3 Summary

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

5.3.6.1 Form of Annual Standard

5.3.6.2 Form of 24-Hour Standard

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

6 POLICY-RELEVANT ASSESSMENT OF WELFARE EFFECTS EVIDENCE

6.1 INTRODUCTION

This chapter assesses key policy-relevant information on known and anticipated adverse effects associated with the presence of PM in the ambient air. It draws upon the most relevant information contained in the second draft ISA (EPA 2009a), 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), climate (section 6.3), vegetation and ecosystems (section 6.4), and materials (section 6.5). In the final PA document, this chapter will include a summary of the relevant scientific information for each effects category 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 regarding alternative secondary PM standards for consideration by the Administrator will be presented in Chapter 7.

It is important to note that discussions of PM-related effects on visibility, vegetation and ecosystems, and climate change processes in Chapter 9 of the second draft ISA build upon and include by reference extensive information from several other significant scientific reviews of these topics. Though relatively little work on urban visibility effects, including the optical and human perceptual aspects of atmospheric visibility, has occurred over the last decade or more, these topics have been the subjects of numerous earlier investigations that have been summarized in detail elsewhere (U.S. EPA, 1979; Latimer and Ireson, 1980; Middleton, 1952; Tombach and McDonald, 2004; Trijonis et al., 1990; Watson et al., 2002), including past criteria documents on PM, SO_2 and NO_X (U.S. EPA, 1982; U.S. EPA, 1993; U.S. EPA, 2004.

The two principal sources for material in section 6.3 on climate are Chapter 2, "Changes in Atmospheric Constituents and in Radiative Forcing," (Forster et al., 2007) in the comprehensive Working Group I report in the Fourth Assessment Report (AR4) from the Intergovernmental Panel on Climate Change (IPCC), Climate Change 2007: The Physical Science Basis (Intergovernmental Panel on Climate Change, 2007), hereafter IPCC AR4; and the U.S. Climate Change Science Program (CCSP) Synthesis and Assessment Product 2.3, "Atmospheric Aerosol Properties and Climate Impacts," by Chin et al. (CCSP 2009), hereafter CCSP SAP2.3. The EPA is a constituent agency member of the U.S. federated CCSP along with NOAA and NASA. The CCSP SAP2.3 document incorporated significant sections from EPA data and reports related particularly to U.S. emissions and measurements. Sections from each of these recent comprehensive reports are included here in their entirety or as amended as noted where they represent the most thorough summary of the climate effects of aerosols. (In the

sections included from IPCC AR4 and CCSP SAP2.3, 'aerosols' is more frequently used than "PM" and that word is retained.)

6.2 EFFECTS ON VISIBILITY

6.2.1 2006 PM NAAQS Review

In the review of the secondary PM NAAQS completed in 2006, EPA took into account that the Regional Haze Program[61], implemented under sections 169A and 169B of the CAA, was established to address all human-caused visibility impairment in Class I areas defined by the Clean Air Act to include national parks and similar natural settings.

Recognizing that efforts were underway under that program, EPA focused the 2006 PM NAAQS review on visibility impairment primarily in urban areas. The EPA evaluated the levels of visibility impairment occurring in urban areas and assessed available information on public preferences regarding acceptability of PM-related urban visibility impairment.

At that time, EPA's focus continued to remain on particle mass and EPA staff determined that size-fractionated particle mass, rather than particle composition, remained the most appropriate approach for addressing PM-related urban visibility effects. EPA recognized that PM composition and relative humidity are important factors in the relationship between light extinction (a measure of visibility) and PM mass concentration, and that a national standard should provide comparable levels of visibility protection across the country. EPA's assessment of PM and meteorological data from 161 cities showed that the least variation in the relationship of light extinction to PM mass concentration was for afternoon periods when low relative humidity conditions generally prevail (Schmidt, et al., 2005).

EPA proposed to revise the secondary standards by making them identical to the suite of proposed primary standards for fine and coarse particles, providing protection against PM-related public welfare effects including visibility impairment, effects on vegetation and ecosystems, and materials damage and soiling (71 FR 2620). EPA also solicited comment on adding a new sub-daily $PM_{2.5}$ secondary standard to address visibility impairment in urban areas. CASAC provided additional advice to EPA in a letter to the Administrator requesting reconsideration of CASAC's recommendations for both the primary and secondary $PM_{2.5}$ standards as well as standards for thoracic coarse particles (Henderson, 2006). With regard to the secondary, CASAC reaffirmed "… the recommendation of Agency staff regarding a separate secondary fine particle standard to protect visibility….. The CASAC wishes to emphasize that continuing to rely on primary standards to protect against all PM-related adverse environmental and welfare effects

[61] See http://www.epa.gov/air/visibility/program.html for more information on EPA's Regional Haze Program.

assures neglect, and will allow substantial continued degradation, of visual air quality over large areas of the country" (Henderson, 2006).

On September 21, 2006, EPA announced its final decisions to revise the secondary NAAQS for PM to provide increased protection of public welfare by making them identical to the revised primary standards (71 FR 61144, October 17, 2006). Specifically, with regard to the secondary standards for fine particles, EPA revised the level of the 24-hour $PM_{2.5}$ standard to 35 $\mu g/m^3$, retained the level of the annual $PM_{2.5}$ annual standard at 15 $\mu g/m^3$, and revised the form of the annual $PM_{2.5}$ standard by narrowing the constraints on the optional use of spatial averaging. With regard to the secondary standards for coarse particles, EPA retained PM_{10} as the indicator for purposes 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 retained the 24-hour PM_{10} standard at 150 $\mu g/m^3$ and revoked the annual PM_{10} standard because available evidence generally did not suggest a link between long-term exposure to current ambient levels of coarse particles and health or welfare effects.

Several parties filed petitions for review following promulgation of the revised PM NAAQS in 2006. These petitions addressed a number of issues, including that of the setting of the secondary $PM_{2.5}$ standards identical to the primary standards. On judicial review the court remanded the secondary $PM_{2.5}$ NAAQS to EPA because the Agency failed to adequately explain why setting the standards equal to the primary $PM_{2.5}$ standards provided the required protection from visibility impairment. In particular, the Agency failed to identify a target level of visibility impairment that would be requisite, and failed to address regional differences in humidity-related effects on visibility American Farm Bureau Federation v. EPA, 559 F. 3d 512, (D.C. Cir. 2009). EPA intends to address the court remand in the context of this review.

6.2.2 Scope of Current Review

EPA staff continues to believe that a focus on urban area visibility is appropriate. In articulating a rationale for this conclusion, we have reviewed the information contained in the second draft ISA and find the following information compelling: 1) PM levels in urban areas are often in excess of those of the surrounding region since urban haze typically includes both regional and local contributions (US EPA, 2009a; sections 9.2.3.3 and 9.2.3.4), suggesting the potential for higher levels of PM-induced visibility impairment in urban areas; 2) the existence of numerous urban visibility protection programs and goals demonstrates that urban visual air quality (VAQ) is noticed and an important value to urban residents (US EPA, 2009a; section 9.2.4), and 3) the existence of large urban populations suggests that potentially more people are routinely affected by poor VAQ than in rural areas.

Therefore, the focus of the remainder of section 6.2 is on the role of ambient PM in the impairment of visibility conditions in urban areas, drawing upon the most relevant information contained in the second draft ISA (Chapter 9), as well as significant reports on the science of visibility referenced therein, the results of assessments contained in the draft risk document titled "Urban Focused Visibility Assessment" (EPA 2009c), and information presented in section 2.8 of this document. In particular, this section includes the following new information:

- Summary of results from a reanalysis of existing urban visibility preference studies and discussion of how these results were used to inform the selection of an appropriate range of candidate protection levels (CPLs) for further evaluation.

- Summary of results from analyses conducted in support of the Urban Focused Visibility Assessment (UFVA) which show that urban PM light extinction varies considerably during daylight hours as a result of varying PM component concentrations including absorbed water due to variations in relative humidity.

Further, as described previously in the UFVA (EPA, 2009c), EPA staff is continuing to focus assessments in this document in terms of an alternative indicator for PM visibility impairment, i.e. PM light extinction, instead of the traditional $PM_{2.5}$ mass concentration. The 2005 Staff Paper discussed the use of a four-hour afternoon $PM_{2.5}$ standard, where the underlying rational was that the generally lower afternoon relative humidity tended to produce a more uniform relationship between light extinction and $PM_{2.5}$ mass concentration throughout the country, therefore providing a more uniform level of visibility protection nationwide. However, this more uniform level of visibility protection was limited to the afternoon hours of the day when relative humidity and visibility impairment are typically the lowest. However, visibility conditions can be the poorest when relative humidity levels are the highest. Thus, from a public welfare perspective, greater protection from visibility impairment is needed during the times when humidity is high. In that regard, morning relative humidity conditions, which are often generally higher in the Eastern US and coastal areas than in the West, can cause the same PM concentrations to produce much higher PM-related visibility impairment in those regions than in areas with lower morning relative humidity. Thus, using a $PM_{2.5}$ mass indicator could potentially result in unequal protection from visibility impairment at the national scale. Unlike PM mass concentration, which is determined by removing the liquid water from the PM prior to measuring it, PM light extinction can be measured at ambient humidity conditions so that it includes the enhanced light extinction resulting from the liquid water that is associated with the hygroscopic PM components in the atmosphere. PM light extinction, like PM mass concentration, is a measurable physical characteristic of ambient PM. Thus, we believe that use

of PM light extinction as the indictor for a secondary PM NAAQS is appropriate and more directly related to the visibility welfare effect.

6.2.3 Nature of Urban Focused PM-related Visibility Effects

Section 2.8 above, as well as section 9.2 of the ISA and chapter 1 of the UFVA, provides useful descriptions of the known science regarding relationship between ambient PM and visibility impacts and much of that discussion will not be repeated here. However, it is important to discuss some aspects of this information as it has bearing on the unique considerations required for defining what would be adequate protection from visibility impairment in urban areas.

Light extinction, which is the optical characteristic of the atmosphere that best determines the impact on perceived visibility, is the loss of light per unit of distance that occurs when light is either scattered or absorbed. Particulate matter and gases can both scatter and absorb light. Light scattering by gases (e.g. nitrogen, oxygen, etc.) that comprise the atmosphere (also known as Rayleigh or clean-air scattering) is related to the density of the air, which is sufficiently constant with elevation that it can be considered a known constant value for any location. Thus, light extinction is approximated as the sum of PM light extinction (including both scattering and absorption) plus Rayleigh light scattering, where the former characterizes the PM contribution to visibility impacts and the latter is taken to be a time invariant constant depending only on elevation above sea level. Therefore, for the purposes of this discussion, the term light extinction includes both PM and Rayleigh sources of light extinction, while the term PM light extinction represents total light extinction minus the Rayleigh light extinction, which is assumed in this assessment to be 10 Mm^{-1}.

While visibility impairment may be caused by either natural or manmade conditions (or both), VAQ is used here to refer to the visibility effects caused solely by air quality conditions, so for example it excludes the reduced visibility caused by fog. The term "urban visibility" is used to refer to VAQ throughout a city or metropolitan area. Urban visibility includes the VAQ conditions in all locations that people experience in their daily lives, including scenes such as residential streets and neighborhood parks, commercial and industrial areas, highway and commuting corridors, central downtown areas, and views from elevated locations providing a broad overlook of the metropolitan area. Thus urban visibility includes VAQ conditions in major cities and smaller towns and encompasses all the VAQ an individual resident sees on a regular basis. Visibility conditions in urban and suburban locations are therefore distinct from visibility in rural or wilderness settings such as the Class I areas. Daytime visibility impairment differs from nighttime visibility impairment in a number of important ways. Light sources and ambient conditions are typically five to seven orders of magnitude dimmer at night than in sunlight.

Moonlight, like sunlight, introduces light throughout an observer's sight path at a constant angle. On the other hand, dim starlight emanates from all over the celestial hemisphere, while artificial lights are concentrated in cities and illuminate the atmosphere from below. In addition to the physical science differences between daytime and nighttime visibility, the human psychophysical response at night is expected to differ, and there is much less known about the aspects of nighttime visibility that are valued by the public (e.g. relative importance of views of celestial objects versus those of artificially illuminated urban skyline/lights). For these reason, at this time EPA is limiting its assessment to daytime visibility (EPA, 2009a, section 9.2.2).

If the relative composition of the PM as a function of particle size were held constant and the ambient relative humidity is low (RH<60%) or held constant, there would be a direct linear relationship between PM light extinction and PM mass concentration. Since the PM composition and ambient relative humidity vary in both time and space, there is in general no simple one-to-one correspondence between PM concentration and PM light extinction. These variations result in significant uncertainty (i.e. greater than a factor of two) in predicting PM light extinction from PM mass concentration. However the PM light extinction can be more accurately estimated from PM composition and relative humidity data, using a simple algorithm with assumed light extinction efficiencies for each of the major PM species and water growth factors for the hygroscopic species. In addition, PM light extinction can be accurately determined by direct measurements using an integrating nephelometer to measure particle light scattering and an aethalometer or similar instrument to measure particle light absorption.

6.2.4 Public Welfare Impacts of Urban Visibility Impairment

The Clean Air Act §302(h) defines public welfare to include the effects of air pollution on "…visibility, … and personal comfort and wellbeing." Though good visibility conditions in Class I areas (e.g., national parks and wilderness areas) have long been recognized as important to the public welfare (see discussions in EPA (2004; 2005) and Chestnut and Dennis (1997), more recently, visibility conditions in urban areas have also been recognized to contribute to the public welfare.

Visibility impairment is the most noticeable effect of fine particles present in the atmosphere. Visibility has direct significance to people's enjoyment of daily activities and their overall wellbeing. Ambient particle pollution degrades the visual appearance and perceived color of distant objects to an observer and reduces the range at which they can be distinguished from the background. This aesthetic effect of ambient particle pollution is well known and understood and includes the aesthetic and wellbeing benefits of better visibility, improved road and air safety, and enhanced recreation in activities like hiking and bicycling. Due to the subjective nature of aesthetics, people's preferences with respect to visibility are difficult to

express or quantify, but people have expressed in many different ways that they enjoy and value a clear view. This desire for good daytime VAQ in urban areas has been clearly demonstrated by the weight of available evidence, both on the basis of numerous studies in the social sciences including quantitative preference and valuation studies, and by the existence of numerous programs and goals regarding establishing and protecting urban VAQ (EPA, 2009a).

In addition, visibility impairment has also been shown to have an effect on human psychology, creating an additional public welfare effect on "personal comfort and well being". In this context, reduced VAQ is considered an environmental stressor (Campbell, 1983) that is associated with heightened amounts of anxiety, tension, anger, fatigue, depression, and feelings of helplessness (Evans et al., 1987; Zeidner and Shechter, 1988). Though the relationship between impaired VAQ and mental health is poorly understood, studies have shown that reduced VAQ affects people's behavior, including reductions in outdoor activities, increased hostility and aggressive behavior (Evans et al., 1982; Cunningham, 1979; Jones and Bogat, 1978; Rotton et al., 1979) and greater emergency calls associated with psychiatric disturbances (Rotton and Frey, 1982). There is also evidence from a recent Canadian study that some Aboriginal peoples value good VAQ because many of their cultural practices and traditions depend upon their ability to see certain viewscapes (Carlson, 2009); it is unclear to what extent this welfare effect might be applicable for Native Americans in the U.S. Because there is insufficient information on the relationship between these latter effects and ambient PM concentrations, they do not usefully inform the development of policy options and selection of appropriate indictors, averaging times, forms, and levels for a secondary standard at this time. However, the staff recommends that they be considered qualitatively when projecting additional benefits that could accrue to the public welfare from improved PM VAQ.

6.2.5 Reanalysis of Public Perception Studies

Chapter 2 of the UFVA document (EPA, 2009c) describes in detail the reanalysis that was performed by EPA (with contractor support) to better characterize the available data on visibility public preference and its usefulness and limitations in serving as a basis for informing the selection of appropriate urban visibility candidate protection levels (i.e., CPLs). Much of the material presented in this section comes directly from chapter 2 of the UFVA.

The reanalysis effort included urban visibility preference survey studies from three western urban locations, plus a pair of smaller focus studies conducted in Washington, D.C. that were designed to explore and further develop urban visibility survey instruments. With the exception of the most recent Washington, DC study (Smith and Howell, 2009), all of these studies were also available at the time of the 2006 review. The first urban visibility study conducted was in Denver, Colorado (Ely et al., 1991), which developed the basic survey method

used in all the subsequent studies. The other western study areas included the lower Fraser River valley near Vancouver, British Columbia (BC), Canada (Pryor, 1996), and Phoenix, Arizona (BBC Research & Consulting, 2003). At the beginning of the 2006 review, EPA sponsored a pilot focus group study in Washington, D.C. (Abt Associates Inc., 2001). In the current review, in response to an EPA request for public comment on the Scope and Methods Plan (74 FR 11580, March 18, 2009), Dr. Anne Smith of Charles River Associates (CRA) International on behalf of the Utility Air Regulatory Group, provided comments (Smith, 2009) which included a discussion of the results from a new Washington, D.C. focus group study that had been conducted for the purpose of testing the reproducibility and appropriateness of methods and approaches employed in the EPA pilot study (Smith and Howell, 2009). In total, 852 individuals participated in the studies conducted for these four urban areas.

As stated above, the approaches used in these studies are similar and are all derived from the method first developed for the Denver urban visibility study. For example, each of the studies reviewed in this assessment investigates the common question, "What level of visibility degradation is acceptable?" The specific definition of acceptable is largely left to each individual survey participant, allowing each to identify his/her own preferences. Further, each individual responds to a series of questions answered while viewing a set of images of various urban VAQ conditions.

The specific materials and methods used in each study vary, however, making direct comparison of the study results between the different studies challenging. Key differences between study methods and approaches include:

- use of WinHaze (a significant technical advance in the method of presenting VAQ conditions),
- number of participants in each study,
- representativeness of participants for the general population of the relevant metropolitan area, and
- specific wording used to frame the questions used in the group interview process.

Although the differences between the methods used in the urban visibility preference studies are significant, it is possible to examine the results of the studies to identify overall trends in the study findings. Figure 6-1 (Figure 2-14 in the UFVA) presents a graphical summary of the results of the studies in the four cities. A number of decisions affecting the visual display of the data were made in the interest of clarity. First, Figure 6-1 omits the 9:00 a.m. photograph results from the Denver study. These photographs were determined to be inconsistently rated when

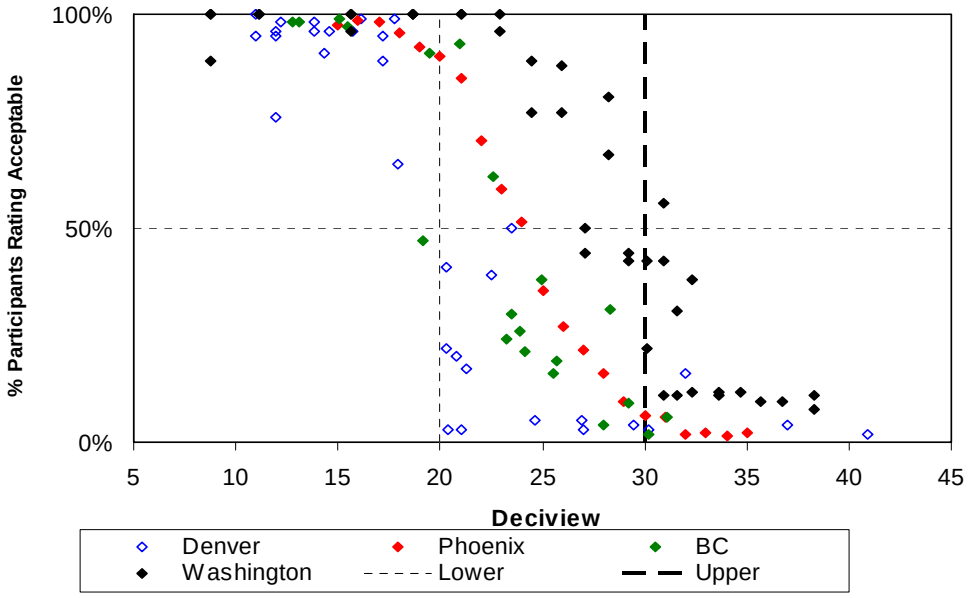
compared with results from other times of day that had similar light extinction levels (see discussion in section 2.2 and Table 2-1 in UFVA). Second, the Chilliwack and Abbotsford photographs appear as a single set of data for the BC study. The assumptions (process by which those data were combined/ the approach taken to combine the data) that were made in order to combine the data are described in section 2.3 in the UFVA). Third, the results from 2001 and 2009 (Test 1) studies of VAQ preferences in Washington, DC are presented as a single combined set of data, because Test 1 appears to successfully replicate the 2001 study, thereby producing comparable results. Tests 2 and 3 results from the 2009 Washington, DC study are not included on Figure 6-1. Those tests are not considered comparable with the other studies because, unlike the other studies, they used an altered range of VAQ conditions that did not represent the actual range of VAQ conditions that occur in the study area.

Figure 6-1 contains lines at 20 dv and 30 dv that identify a range where the 50% acceptance criteria occur across all four of the urban preference study areas. Out of the 114 data points shown, only one photograph (or image) with a VAQ below 20 dv was rated as acceptable by less than 50% of the participants who rated that photograph.[62] Similarly, only one image with a VAQ above 30 dv was rated acceptable by more than 50% of the participants who viewed it.[63] This figure shows that, while there is a high degree of similarity between the preferences found in each study, there may be important differences in VAQ preferences across the four cities as well. For example, the Denver study identified preferences for a relatively good level of VAQ; the 50% criteria occur between 17.7 dv and 20.3 dv. In Washington, DC, however, the 50% criteria separation occurs at a substantially worse level of VAQ, between 27 dv and 31 dv.

[62] Only 47% of the BC participants rated a 19.2 DV photograph as acceptable.

[63] In the 2001 Washington, DC study, a 30.9 DV image was used as a repeated slide. The first time it was shown 56% of the participants rated it as acceptable, and 11% rated it as acceptable the second time it was shown. The same VAQ level was rated as acceptable by 42% of the participants in the 2009 study (Test 1).

Figure 6-1. Summary of results of urban visibility studies in four North American cities, showing the identified range of the 50% acceptance criteria.



There are several hypotheses that may explain why the results of these studies differ. First, as mentioned earlier, the use of photographs versus WinHaze-generated images may play a significant role in preference studies, perhaps introducing bias (such as suggested by the responses to the 9:00 a.m. Denver photographs) as well as variability. Use of photographs from different days and times of day that rely on associated ambient measurements of light extinction to characterize their VAQ level introduces two types of uncertainty. The intrinsic appearance of the scene can change due to the changing shadow pattern and cloud conditions, and spatial variations in air quality can result in ambient light extinction measurements not being representative of the sight-path-averaged light extinction. WinHaze has neither of these sources of uncertainty because the same base photograph is used (i.e. no intrinsic change in scene appearance between images) and the modeled haze that is displayed in the photograph is based on uniform sight-path-averaged light extinction throughout the scene. This modeled uniform light extinction, however, may not represent non-uniform haze conditions that may occur at a study location during certain times of day or PM conditions (e.g., layered morning haze; point source plumes entering site path). The impact of the application of a uniform versus non-uniform haze layer to a scene has not been investigated.

Second, variation in the degree of representativeness of the participants and the sizes of the participant samples involved may also be important factors. The small sample size and fairly uniform population of respondents is a plausible explanation for the noisiness of the combined

Washington, DC, results (35 participants, including 26 from a single consulting firm and 10 of those from a different city) compared with the larger and more representative population of responders from Phoenix (385 participants, carefully selected to be representative

A third hypothesis explored by Smith and Howell (2009) is that the range of VAQ images presented in the survey may influence the results. Though this hypothesis appears to be borne out by Smith and Howell's results for Washington, DC, it seems an unlikely explanation for the differences in results between the four urban preference studies. For example the Denver study included photographs with the haziest conditions among the four studies, but resulted in the lowest haze condition for the 50^{th} percentile preference ratings among the four, not the highest as might be expected if the range of haze levels were a significant factor influencing the results of preference studies.

A fourth major hypothesis is that urban visibility preferences may differ by location, and the differences may arise from inherent differences in the cityscape scene in each difference. The key evidence to suggest this hypothesis is that the apparent differences between the Denver results (which found the 50% acceptance criteria occurred in the best VAQ levels among the four cities) and the Washington, DC results (which found the 50% acceptance criteria occurred at the worst VAQ levels among the four cities). This hypothesis suggests that these results may occur because the cityscape of Denver includes clearly visible snow-covered mountains in the distance, while the prominent features of the Washington, DC cityscape are buildings relatively nearby with only modest changes in elevation.

Finally, perhaps of significant importance is that the perceived sensitivity of individual scenes to changes in light extinction can be quite different. As in the fourth hypothesis, this may in part explain why the Denver study scene, with its long distance to the mountain backdrop, resulted a preference for the best VAQ level with a 50% criteria value between 17.7 and 20.3 dv, while in Washington, DC, the 50% criteria separation occurs at a substantially worse level of VAQ, between 27 and 31 dv from Abt Associates Inc. (2001) and Smith and Howell (2009) Test 1. The distinction between the last two hypotheses are that the earlier one speaks to the desirability of seeing distant mountains versus this hypothesis where its ability to perceive haze at lower light extinction levels. Additional studies, including directly comparable studies using similar methods in diverse cities, are necessary to gain further understanding of preferences for urban visibility.

Based on the composite results and the effective range of 50^{th} percentile acceptability across the four urban preference studies shown in Figure 6-1, candidate protective levels (CPL) have been selected in a range from 20 dv to 30 dv (74 Mm^{-1} to 201 Mm^{-1}) for the purpose of comparing to current and projected conditions in the assessment in chapters 3 and 4 of this document. A midpoint of 25 dv (122 Mm^{-1}) was also selected for use in the assessment. These

three values provide a low, middle, and high set of light extinction conditions that are used in subsequent sections of the UFVA to provisionally define daylight hours with urban haze conditions that have been judged unacceptable by the participants of these preference studies.

6.2.6 Considerations in Selecting Alternative Light Extinction Scenarios for Assessment

In considering what alternative standard scenarios were appropriate to assess in the context of providing urban visibility protection, EPA staff concluded that the available urban public preference study information, as analyzed and characterized in the UFVA, though limited, is sufficient to inform the selection of an appropriate range of light extinction CPLs. While less information is available to support the selection of an appropriate averaging time and form, we make the following observations regarding the nature of the public welfare impact anticipated to occur in urban areas at both current and alternative levels of urban PM air quality that can usefully inform their selection.

First, we observe that the strength of an adverse visibility impact is not necessarily linked with any specific time duration since it only takes an instant for a person to see a visually impaired scene. In urban areas, more so than recreational areas, a person is typically experiencing visibility conditions intermittently and incidentally as a part of their daily activities (e.g. during commutes to work or school), and thus, may have few opportunities to revise his/her perception of the quality of that urban view again in any given day. Thus, it is possible that the effect on wellbeing of impaired VAQ from a short-term exposure of limited duration may endure well beyond the period of exposure. In addition, we have considered that though the occurrence of multiple hours of poor VAQ in a single day would undoubtedly increase the number of individuals with potential welfare impacts, because of the intermittent nature of exposure to VAQ it would not necessarily increase the intensity of the impact on the personal wellbeing of those already affected. As a result, though not specifically addressed in the preference studies analyzed, we selected a one hour averaging time as that most closely linked to the short-term nature of the daily impact as compared to longer periods, and are considering that the one hour maximum value for a day is a good index of short term exposure that may have longer term impacts on wellbeing.

Second, because the nature of the welfare effect is one of emotion (feelings of wellbeing) and not directly related to a physical health outcome, we believe that it is not necessary to eliminate all such exposures and that some number of hours/days with poor VAQ can reasonably be accepted as being protective of public welfare. As a result, we have selected the 90^{th} and 95^{th} percentiles per year averaged over a three year period as an appropriate range of frequencies to be evaluated in conjunction with the range of PM light extinction CPLs.

6.2.7 Characterization of Current Urban Visibility Conditions

The following information is excerpted from Chapter 3 of the UFVA (EPA 2009c) and the reader is referred to that document for further detail of the process and approaches used in that assessment.

The goal of this part of the assessment was to estimate hourly PM light extinction for the daylight hours for a reasonably representative number of days in each year of the 2005-2007 period, to allow the application of statistical forms based on three years of data. For efficiency, the visibility assessment uses the same 15 urban study areas selected for the health risk assessment. These areas are listed in Table 6-1, along with their 2005-2007 annual and 24-hour $PM_{2.5}$ design values and accompanying percentages of days with daily maximum 1-hour light extinction levels greater than each of the three CPLs. These results will be discussed in conjunction with those of Figure 6-2 below.

Table 6-1. $PM_{2.5}$ design values and percent of days with maximum light extinction exceeding three candidate protection levels (CPLs) for 15 Urban study areas.

Study Area	Design Levels (2005– 2007)		Number of Assessment Days	Percent of Days Exceeding the Light Extinction CPL		
	Annual	24-Hour		74 Mm^{-1}	122 Mm^{-1}	201 Mm^{-1}
Tacoma	10.2	43	110	68	36	9
Fresno	20.3	69	324	80	51	24
Los Angeles	19.6	55	302	92	80	49
Phoenix	12.6	32	86	59	13	3
Salt Lake City	11.6	55	306	61	24	9
Dallas	11.9	25	274	86	53	14
Houston	15.8	31	149	89	58	21
St. Louis	16.5	39	294	100	86	55
Birmingham	18.7	44	350	96	80	52
Atlanta	16.2	35	295	95	80	34
Detroit	17.2	43	141	91	79	57
Pittsburgh	16.5	43	284	93	70	43
Baltimore	15.6	37	187	88	65	38
Philadelphia	15.0	38	145	95	76	46
New York	15.9	42	228	91	70	46

The results depicted in Table 6-1 were generated through the following steps. First, CMAQ model output for 2004 was used to develop realistic diurnal variations of the major $PM_{2.5}$ components as monthly averaged variation for each of the 15 urban monitoring sites, which were then used in a later step of the process that generated hourly estimates from the 24-hour speciation monitoring data. Another step in the process accounted for hourly variations in PM measured by continuous monitoring at each site. Hourly relative humidity (RH) data needed for the calculation of light extinction for each study area's primary monitoring site were obtained from either an air monitoring station reporting such data to AQS, or a National Weather Service (NWS) station. Lastly, the original IMPROVE light extinction algorithm was used to estimate total hourly light extinction using the RH data and the hourly PM composition estimates. This total light extinction (expressed simply as light extinction in the remainder of the document), includes the term for Rayleigh (i.e. clean air) scattering. In the next draft UFVA, results will also be expressed in terms of PM light extinction (i.e. light extinction resulting only from ambient PM without the Rayleigh term included).

Figure 6-2 (Figure 3-8 of UFVA) presents box-and-whisker plots to illustrate the distributions of the estimates of daylight 1-hour reconstructed light extinction levels in each area across the three year (2005-2007) period. The distribution of the individual 1-hour values and the daily maximum 1-hour values are both shown. The horizontal dashed lines in the plots represent the low, middle and high light extinction CPLs as discussed above. These benchmarks expressed as 74, 122, and 201 Mm^{-1}, also correspond to the deciview benchmark values of 20, 25, and 30 dv for light extinction identified above. As seen in the comparable $PM_{2.5}$ box and whisker plots, the hourly light extinction values tends to be higher in the Eastern urban areas and lower in the non-California Western urban areas. The distributions of maximum daily light extinction values are higher, as expected, than for all hours.

Both Figure 6-2 and Table 6-1 indicate that all 15 urban areas have daily maximum hourly light extinctions that exceed even the highest CPL some of the time. Again the non-California Western urban locations have the lowest frequency of maximum hourly light extinction with values in excess of the high CPL less than 10% of the time. Except for the two Texas and the non-California Western urban areas, all of the other urban areas exceed the high CPL about a quarter to a half of the time. Based on these estimated maximum hourly light extinction values, all 15 of the urban areas exceed the low CPL for ~60% to 100% of the days.

In the last review of the secondary PM NAAQS, the pattern of light extinction during the day was of particular interest. To illustrate the distributions of 1-hour light extinction levels in specific daylight hours, Figure 6-3 shows the distributions of 1-hour light extinction across the entire three-year study period, individually for the study areas. These plots show that high light extinction can occur during any of the daylight hours, though for most of these urban areas the

early morning hours have the highest light extinction. Urban areas without a prominent proclivity for early morning high light extinction include Phoenix, AZ; Salt Lake City, UT; Tacoma, WA; Fresno, CA; and Philadelphia, PA.

Figure 6-2. Distributions of estimated daylight 1-hour light extinction (top) and maximum daily daylight 1-hour light extinction (bottom) in each study area

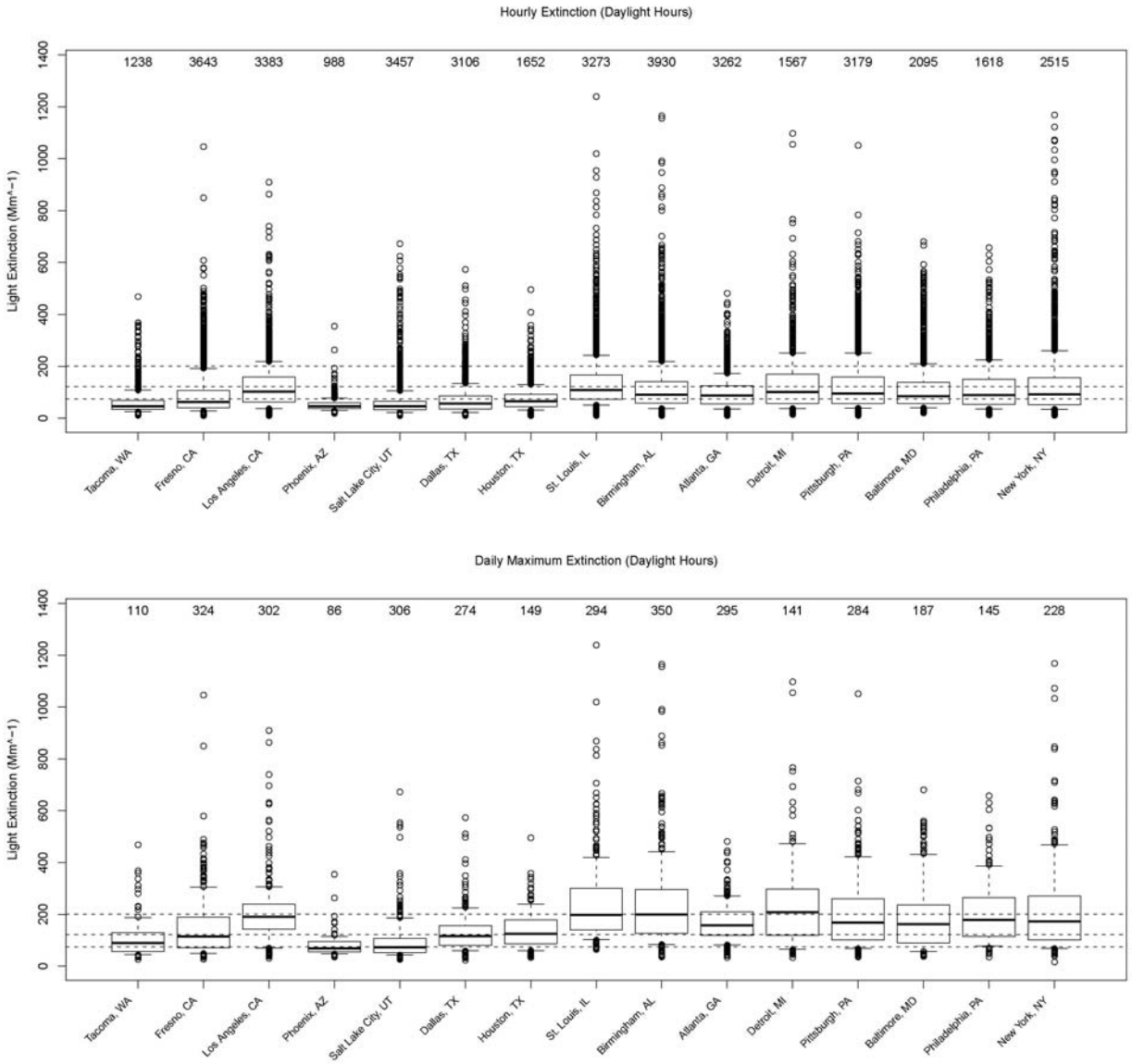
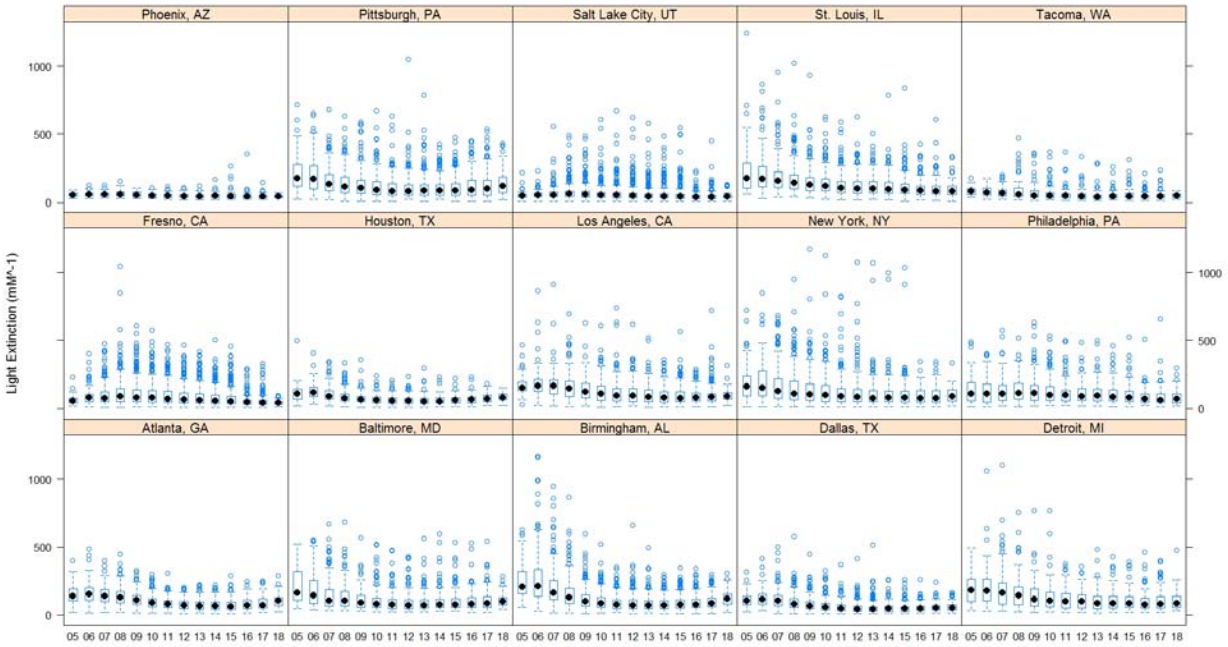


Figure 6-3. Light extinction box and whisker plots by time of day for each of the 15 urban areas.



6.2.8 Projected Light Extinction When Meeting Alternative NAAQS Scenarios

Material for this section is taken from chapter 4 of the UFVA. Eight different alternative NAAQS scenarios were evaluated. These scenarios included six light extinction cases and two $PM_{2.5}$ NAAQS cases. The six light extinction cases include the maximum hourly light extinction compared to each of the three CPLs at two annual percentile values (90^{th} and 95^{th}) averaged over a three year period. The two $PM_{2.5}$ cases include the current $PM_{2.5}$ NAAQS (i.e.15 $\mu g/m^3$ annual and 35 $\mu g/m^3$ 24-hour) and an alternative case (12 $\mu g/m^3$ annual and 25 $\mu g/m^3$ 24-hour), together representing the highest and lowest alternative NAAQS scenarios considered in the health risk assessment. Both $PM_{2.5}$ NAAQS cases used the three year averaged 98^{th} annual percentile values. In order to model "what if"conditions for just meeting each alternative, two different approaches were necessary: one for generating the alternate hourly light extinction values, and one for the $PM_{2.5}$ scenarios. A more detailed description on these two distinct approaches can be found in sections 4.1.4 and 4.2.2 of the UFVA. A brief summary of each approach is provided below.

The approach used for the light extinction scenarios involve using the estimates of current hourly conditions generated for the 15 urban areas as described in the previous section, and applying a process similar to the proportional rollback that was used in the health risk assessment modeling of "what if" conditions in several previous PM NAAQS reviews. The

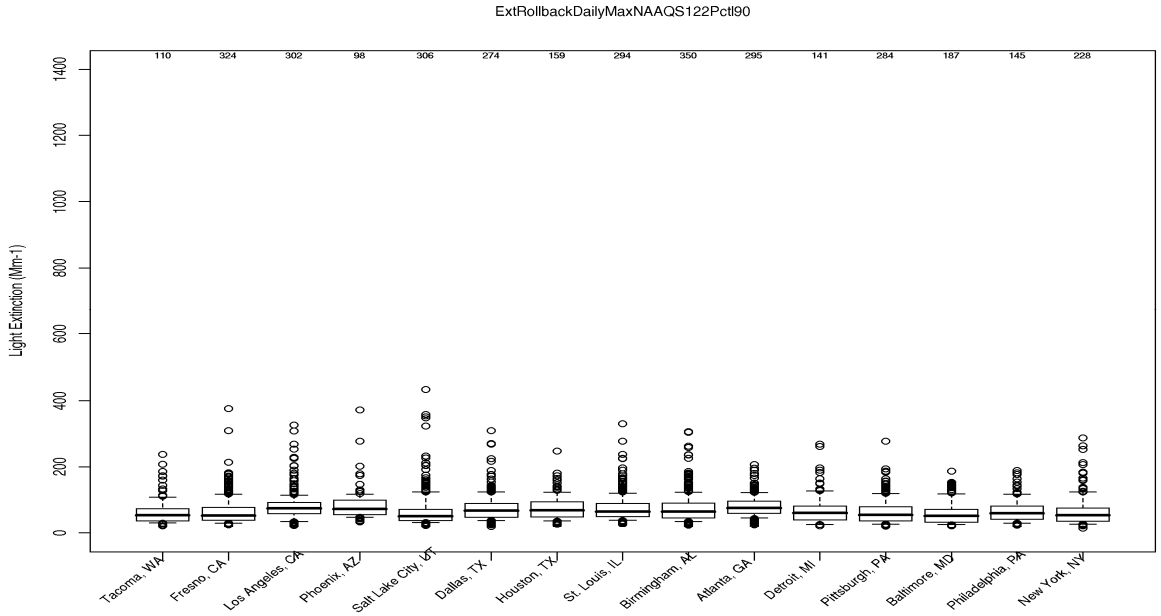
Policy Relevant Background (PRB) is subtracted from the data that exceed the scenario specifications, and then the percent reduction in the non-PRB portion of the data needed to scale the total to meet the scenario conditions is determined. The percent reduction is applied to all of the non-PRB data, and then PRB is added back to produce the values that meet the scenario conditions. The light extinction scenarios use hourly daily maximum light extinction values. In contrast, for the $PM_{2.5}$ mass concentration scenarios approach, the reduction factors are determined for the 24-hour periods (as done for the health risk assessment) but applied to each hour and the non-PRB portion of each $PM_{2.5}$ component, which is then used to estimate the hourly light extinction using the IMPROVE algorithm and measured relative humidity.

The results of this process include displays of the required percent reductions in non-PRB light extinction and $PM_{2.5}$ values . Figure 6-4 (Figure 4-13 in the UFVA) contains two box and whisker plots that show the distributions of estimated daily maximum daylight 1-hour light extinction values for one of the six light extinction cases (122 Mm^{-1}, 90^{th} percentile) and for the current $PM_{2.5}$ NAAQS (i.e.15 $\mu g/m^3$ – annual and 35 $\mu g/m^3$ – 24-hour) case. The principle insight provided by the first plot is that the rollback process successfully generated hourly maximum light extinction distribution for each urban area with the 90^{th} percentile (i.e. the upper whisker) is very near 122 Mm^{-1}. Plots for the other light extinction scenarios (in UFVA, Appendix F) are similar in demonstrating that the rollback process was successful.

The second plot in Figure 6-4 shows that uniformly applied PM mass concentration based NAAQS provides uneven daylight 1-hour maximum light extinction values across the 15 urban areas and that nearly every urban areas has a 90^{th} percentile maximum 1-hour light extinction well above the upper CPL ($201Mm^{-1}$). Another way to assess the effectiveness of the $PM_{2.5}$ based NAAQS scenarios in affecting the maximum 1-hour light extinction is to compare their 90^{th} and 95^{th} percentile design values with the range of CPLs. Table 6-2 (Table 4-11 in the UFVA) contains these design values for the two $PM_{2.5}$ NAAQS scenarios. As seen in the box and whisker plot, the 90^{th} percentile design values exceed the high CPL ($201Mm^{-1}$) at all of the Eastern urban areas and for Los Angeles while meeting the current $PM_{2.5}$ NAAQS. The table also shows that the lower $PM_{2.5}$ NAAQS scenario results in design values that exceed the 90^{th} percentile high CPL at six or seven of the Eastern urban areas.

Figure 6-4. Distributions of daily maximum daylight 1-hour total light extinction under two "just meeting" secondary NAAQS scenarios

(a) Secondary NAAQS based on measured total light extinction with a level of 122 Mm^{-1} and a 90th percentile form



(b) Secondary NAAQS of 15 $\mu g/m^3$ for the annual average and 35 $\mu g/m^3$ for the 98th percentile 24-hour average

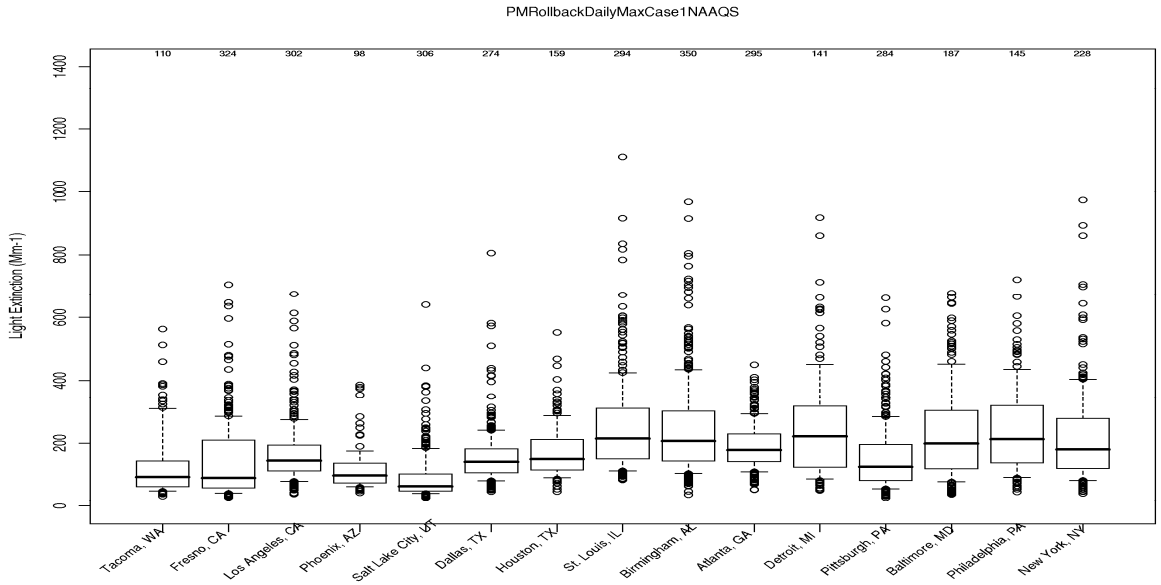


Table 6-2. Light extinction design values for "just meeting" secondary NAAQS scenarios based on $PM_{2.5}$ mass

Annual/1-hour $PM_{2.5}$ NAAQS	15μg/m^3 / 35μg/m^3		12μg/m^3 / 25μg/m^3	
City Name	**90th %tile Design Value (Mm^{-1})**	**95th %tile Design Value (Mm^{-1})**	**90th %tile Design Value (Mm^{-1})**	**95th %tile Design Value (Mm^{-1})**
Tacoma, WA	188	228	139	165
Fresno, CA	183	238	139	179
Los Angeles, CA	221	311	175	261
Phoenix, AZ	117*	154*	107	145
Salt Lake City, UT	126	174	98	133
Dallas, TX	213*	262*	200	245
Houston, TX	224	261	182	211
St. Louis, IL	384	477	311	383
Birmingham, AL	355	476	268	369
Atlanta, GA	249	271	197	218
Detroit, MI	364	520	264	376
Pittsburgh, PA	Recalculating	Recalculating	Recalculating	Recalculating
Baltimore, MD	419	459	308	335
Philadelphia, PA	377	403	273	296
New York, NY	377	450	274	325

* Phoenix and Dallas meet 15/35 under current conditions, so these entries are the same as for current conditions.

Table 6-3 contains the percent reductions required in the non-PRB light extinction that include both $PM_{2.5}$ and $PM_{10-2.5}$ contributions for the six light extinction NAAQS scenarios, and the percent required in the non-$PM_{2.5}$ mass concentration to meet the two $PM_{2.5}$ NAAQS scenarios. A number of caveats and uncertainties associated with these approaches should be noted. First, with regard to the rollback approach, there is an implied linearity between emissions and concentrations and subsequent visibility effects, which is not true in general. Second, there are uncertainties in each step of the assessment including the estimation of the hourly PM composition and resulting light extinction, and estimation of the PRB, all of which feed into the reduction calculations. In spite of these issues the reductions are thought to be a useful metric for comparing the challenges of these scenarios at the 15 urban areas.

Table 6-3. Percentage reductions required in the non-PRB light extinction or $PM_{2.5}$ mass to "just meet" the NAAQS scenarios.

	NAAQS Scenarios Based on Maximum Daily 1-hour Daylight Total Light Extinction, Average of Nth Percentile Value Over Three Years						NAAQS Scenarios Based on Annual and 24-hour $PM_{2.5}$	
Total Light Extinction Level (Mm^{-1})	**201**	**201**	**122**	**122**	**74**	**74**		
Percentile Form	**90th**	**95th**	**90th**	**95th**	**90th**	**95th**		
Annual $PM_{2.5}$ Level ($\mu g/m^3$)							**15**	**12**
24-hour $PM_{2.5}$ Level ($\mu g/m^3$)							**35**	**25**
Urban Area	**Percentage Reduction Required in Non-PRB Total Light Extinction**						**Percentage Reduction Required in Non-PRB $PM_{2.5}$**	
Tacoma	13	29	51	59	74	77	19	43
Fresno	38	53	65	73	82	86	45	61
Los Angeles	40	56	66	75	82	87	37	55
Phoenix	0	0	0	23	43	59	0	22
Salt Lake City	0	23	36	56	65	76	37	56
Dallas	7	26	49	59	75	79	0	7
Houston	16	29	53	60	76	79	6	27
St. Louis	55	64	75	80	87	90	10	37
Birmingham	56	67	75	82	87	90	22	45
Atlanta	28	33	60	63	79	80	8	30
Detroit	58	71	77	84	88	91	19	43
Pittsburgh	55	61	75	78	87	88	Recalculating	
Baltimore	58	61	76	79	88	89	6	33
Philadelphia	54	57	74	76	86	88	8	35
New York	59	65	77	80	88	90	17	41

The percent reductions in Table 6-3 provide another way to consider the differences between the $PM_{2.5}$ mass and PM light extinction. These are apparent when comparing the reductions required in most of the Western urban areas to those in the East. For example

comparing the least restrictive of the PM light extinction scenarios (201 Mm^{-1}, 90^{th} percentile) to the current NAAQS (15 $\mu g/m^3$- annual, 35 $\mu g/m^3$- 24-hour), shows similar magnitude reductions in the typically drier, less sulfate/nitrate PM dominated Western urban areas, but quite different magnitudes for the more humid, higher sulfate/nitrate PM urban areas of the East. Also note that the lower of the two $PM_{2.5}$ scenarios (12 $\mu g/m^3$- annual, 25 $\mu g/m^3$- 24-hour) has less challenging reduction values in the East than the highest of the PM light extinction scenarios (201, Mm^{-1} 90^{th} percentile), implying less protection of Eastern urban visibility, yet it has much higher reduction values for the Western urban areas implying a greater level of visibility protection.

Table 6-4 shows the percentage of days over the multiyear period (i.e. 3 years except at Phoenix and Houston, which have 2 years) with maximum 1-hour daylight light extinction above the three CPL values for each of the eight NAAQS scenario cases. Notice that the percentage of days above the CPL for the corresponding CPL light extinction scenario is very nearly the complimentary fraction to the percentile form of the scenario showing again that the rollback approach is numerically consistent (e.g. the fraction of days above 74 Mm^{-1} for the 74 Mm^{-1}, 90^{th} percentile scenario for Tacoma is 9% which is nearly 10%). More informative are the percentages of days above each of the CPL for alternative scenarios. For example the 201 Mm^{-1}, 90^{th} percentile scenario would result in over half of the days (56%) at Tacoma having maximum 1-hour daily light extinction above 74 Mm^{-1} and a quarter of the days above 122 Mm^{-1}. Comparisons of these percentages allows a rough indication of how the two scenarios of a NAAQS based on $PM_{2.5}$ mass compare to the other six light extinction based scenarios in terms of protecting visual air quality. Notice that even the lower $PM_{2.5}$ NAAQS scenario (12 $\mu g/m^3$- annual, 25 $\mu g/m^3$- 24-hour) would permit projected 1-hour maximum daily light extinction above the least restrictive CPL (201 Mm^{-1}) more that 10% of the time for most of the Eastern urban areas (Dallas, Houston and Atlanta have values near 10%), while the percent of maximum hourly days for the Western urban areas are all less than 10%.

Table 6-4. Percentage of days with a maximum 1-hour daylight light extinction above the three CPL for each of the eight NAAQS scenario cases.

	Days above 74 Mm^{-1} (Percent)								Days above 122 Mm^{-1} (Percent)								Days above 201 Mm^{-1} (Percent)							
Mm-1 Level	**201**	**201**	**122**	**122**	**74**	**74**			**201**	**201**	**122**	**122**	**74**	**74**			**201**	**201**	122	122	74	74		
Percentile Form	**90**	**95**	**90**	**95**	**90**	**95**			**90**	**95**	**90**	**95**	**90**	**95**			**90**	**95**	90	95	90	95		
Annual/24-hour							**15/35**	**12/25**							**15/35**	**12/25**							15/35	12/25
Area	**Percentage of days**								**Percentage of days**								**Percentage of days**							
Tacoma	56	46	26	23	11	9	55	39	25	18	8	6	2	1	23	11	8	6	2	0	0	0	6	3
Fresno	54	42	29	21	9	3	48	33	27	19	10	4	1	1	23	14	10	4	1	1	0	0	8	2
Los Angeles	81	74	53	24	8	5	82	76	49	20	8	5	2	1	59	28	8	5	3	1	0	0	12	7
Phoenix	88	66	50	30	9	5	46	42	46	27	9	5	2	1	7	6	9	5	3	2	1	0	2	2
Salt Lake City	54	32	25	14	11	4	26	19	24	13	10	4	3	2	11	6	10	4	3	2	1	0	3	2
Dallas	77	66	43	30	9	4	80	77	42	26	11	4	2	1	46	42	11	5	2	1	0	0	13	11
Houston	75	68	45	32	14	9	81	69	40	30	11	7	1	1	48	31	11	6	1	1	0	0	14	7
St. Louis	75	62	40	28	10	6	98	94	36	25	10	6	2	1	79	66	10	6	2	1	0	0	44	32
Birmingham	67	56	42	26	13	6	90	82	36	22	11	5	2	1	66	52	11	5	2	1	0	0	37	21
Atlanta	86	84	57	50	9	7	91	85	50	42	10	7	0	0	68	49	10	5	0	0	0	0	22	10
Detroit	66	50	40	18	11	5	82	79	36	16	11	5	1	0	70	59	11	5	1	0	0	0	45	22
Pittsburgh	55	52	30	25	10	6	Recalculating	Recalculating	27	22	10	6	0	0	Recalculating	Recalculating	10	5	0	0	0	0	Recalculating	Recalculating
Baltimore	55	50	25	22	10	8	82	71	23	22	10	6	0	0	60	46	9	6	0	0	0	0	34	22
Philadelphia	67	65	33	28	8	6	90	81	29	26	8	6	0	0	70	54	8	6	0	0	0	0	37	21
New York	57	50	27	23	10	6	80	70	26	21	11	6	1	1	60	45	12	5	2	1	0	0	30	19
Average	**68**	**58**	**38**	**26**	**10**	**6**	**73**	**65**	**34**	**23**	**10**	**5**	**1**	**1**	**49**	**36**	**10**	**5**	**2**	**1**	**0**	**0**	**22**	**13**

6.2.9 Summary

Section 6.2 above focuses on the role of ambient PM in the impairment of visibility conditions in urban areas, drawing upon the most relevant information contained in the second draft ISA (Chapter 9), as well as significant reports on the science of visibility referenced therein, the results of assessments contained in the draft UFVA (EPA, 2009c), and information presented in section 2.8 of this document. The purpose of this section is to distill and describe the most significant policy-relevant results from a reanalysis of urban visibility preference studies conducted in four urban areas and an assessment of recent and projected urban visual air quality conditions in 15 urban area case studies conducted in support of the secondary standard review and described in the UFVA document (EPA, 2009c).

Further, as described previously in the UFVA (EPA, 2009c), EPA staff chose to include an alternative indicator for PM visibility impairment, i.e. PM light extinction, in addition to the traditional $PM_{2.5}$ mass concentration indicator. Unlike PM mass concentration, which is determined by removing the liquid water from the PM prior to measuring it, PM light extinction can be measured at ambient humidity conditions so that it includes the enhanced light extinction resulting from the liquid water that is associated with the hygroscopic PM components in the atmosphere. PM light extinction, like PM mass concentration, is a measurable physical characteristic of ambient PM. Thus, we believe that use of PM light extinction as the indictor for a secondary PM NAAQS is appropriate and more directly related to the visibility welfare effect.

As discussed above, though the materials and methods used in the urban visibility preference studies for the four urban areas vary from study to study, they are all similar to and derived from the method first developed for the Denver urban visibility study. Taking into account the associated caveats and uncertainties (see section 6.2.5), we thus concluded that this information, though limited, can be used to explore overall findings and trends. Further, when viewed together, the composite results presented in Figure 6-1 usefully inform the selection of an appropriate range of VAQ CPLs for urban areas to consider in further analyses. Using the 50^{th} percentile acceptability criteria, we selected a range of CPLs from 20 dv to 30 dv (74 Mm^{-1} to 201 Mm^{-1}) with a midpoint of 25 dv (122 Mm^{-1}). These three values provide low, middle, and high values of urban haze (light extinction) conditions that have been judged unacceptable by at least 50% of the preference study participants for use in comparisons with recent and projected PM air quality conditions.

Though not specifically evaluated in the preference studies analyzed, we also considered a one hour averaging time as that most closely linked to the likely short-term nature of the daily impact that is likely to occur in urban areas. In developing this view, we have considered that the strength of an adverse visibility impact is not necessarily linked with any specific time

duration since it only takes an instant for a person to see a visually impaired scene. In urban areas, more so than recreational areas, a person is typically experiencing visibility conditions intermittently and incidentally as a part of their daily activities (e.g. during commutes to work or school), so that longer-term, multiple hour exposures are less likely. As a result, any given exposed individual may have few opportunities to revise his/her perception of the quality of that urban view again in any given day. In such a case, the effect of a short-term exposure to impaired VAQ on an individual's wellbeing may endure well beyond the period of exposure. In addition, though the occurrence of multiple hours of poor VAQ in a single day would undoubtedly increase the number of individuals exposed, there is no information at this time to suggest that individuals exposed to multiple hours would necessarily experience an increased intensity of the impact on their personal wellbeing than those exposed for shorter periods.

Further, in considering an appropriate range of frequencies to be evaluated in conjunction with the range of light extinction CPLs, we took into account that the nature of the welfare effect is one of emotion (feelings of wellbeing) and not directly related to a physical health outcome. Thus, we believe that it is not necessary to eliminate all such exposures and that some number of hours/days with poor VAQ can reasonably be accepted as being protective of public welfare. As a result, we selected the 90th and 95th percentiles per year averaged over a three year period as an appropriate range of frequencies for further consideration. By comparing these values to current and projected $PM_{2.5}$ and light extinction conditions, we were able to evaluate to what extent these levels are being exceeded or met across the 15 urban area case studies.

Figure 6-2 and Table 6-1 indicate that all 15 urban areas have daily maximum hourly light extinctions that exceed even the highest CPL some of the time. The non-California Western urban locations have the lowest frequency of maximum hourly light extinction with values in excess of the high CPL less than 10% of the time. Except for the two Texas and the non-California Western urban areas, all of the other urban areas exceed that high CPL about a quarter to a half of the time. Based on these estimated maximum hourly light extinction estimates, all 15 of the urban areas exceed the low CPL for ~60% to 100% of the days.

Figure 6-3 shows the distributions of 1-hour light extinction levels in specific daylight hours, across the entire three-year (2005-2007) study period, individually for the study areas. These plots show that high light extinction can occur during any of the daylight hours, though for most of these urban areas the early morning hours have the highest light extinction. Urban areas without a prominent tendency for high early morning light extinction include Phoenix, AZ; Salt Lake City, UT; Tacoma, WA; Fresno, CA; and Philadelphia, PA. This pattern of light extinction during the day, i.e. frequent occurrence of the worst visibility conditions during morning hours, would seem to argue against selecting a standard form based on only afternoon hours that

typically have the least visibility impairment as did the 4-hour sub-daily form described in the 2006 review.

Additional analyses (described in section 6.2.8 above and in greater detail in chapter 4 of the UFVA) conducted by EPA staff generated hourly light extinction values to produce PM conditions that would meet eight different NAAQS scenarios: six light extinction cases and two $PM_{2.5}$ NAAQS cases. The six light extinction cases included the maximum hourly light extinction compared to each of the three CPLs at two annual percentile values (90^{th} and 95^{th}) averaged over a three year period. The two $PM_{2.5}$ cases included the current $PM_{2.5}$ NAAQS (i.e.15 $\mu g/m^3$ annual and 35 $\mu g/m^3$, 24-hour) and a lower case (12 $\mu g/m^3$, annual and 25 $\mu g/m^3$, 24-hour), with both $PM_{2.5}$ NAAQS cases using the three year averaged 98^{th} annual percentile values. Figure 6-4 shows the distributions of estimated daily maximum daylight 1-hour light extinction resulting from one of the six light extinction cases (122 Mm^{-1}, 90^{th} percentile) and from the current $PM_{2.5}$ NAAQS (i.e.15 $\mu g/m^3$ – annual and 35 $\mu g/m^3$ – 24-hour). These results show that even the lower of the two $PM_{2.5}$ mass NAAQS scenarios would permit projected 1-hour maximum daily light extinction above the least restrictive CPL (201 Mm^{-1}) more that 10% of the time for most of the Eastern urban areas (Dallas, Houston and Atlanta have values near 10%), while the percent of maximum hourly days for the Western urban areas are all less than 10%.

Given the results above, it is clear that urban visibility conditions across the 15 cities are currently being impacted at ambient levels of PM within the range of CPLs for light extinction derived from the reanalysis of available urban preference studies. The external review draft PA to be released later this year will include additional analyses of how VAQ would be expected to change under a scenario of just meeting the current $PM_{2.5}$ secondary standards and under additional just meeting potential alternative standard scenarios.

6.3 EFFECTS ON CLIMATE

Information and conclusions about what is currently known about the role of PM in climate is summarized in chapter 9 of the second draft ISA (US EPA, 2009a). The ISA concludes; "that a causal relationship exists between PM and effects on climate, including both direct effects on radiative forcing and indirect effects that involve cloud feedbacks that influence precipitation formation and cloud lifetimes" (section 9.3.10). Material from the climate section of the second draft ISA is principally drawn from the U.S. Climate Change Science Program Synthesis and Assessment Product 2.3, "Atmospheric Aerosol Properties and Climate Impacts," by Chin et al., (CCSP SAP 2.3 2009) (CCSP 2009) and chapter 2, "Changes in Atmospheric Constituents and in Radiative Forcing," (Forster et al., 2007) in the comprehensive Working Group I report in the Fourth Assessment Report (AR4) from the Intergovernmental Panel on

Climate Change (IPCC), Climate Change 2007: The Physical Science Basis. This section of the PA summarizes and synthesizes the policy-relevant science in the ISA for the purpose of helping to inform consideration of climate aspects in the review of the secondary PM NAAQS.

Atmospheric PM (referred to as aerosols in the remainder of this section to be consistent with the ISA) affects multiple aspects of climate. These include absorbing and scattering of incoming solar radiation, alterations in terrestrial radiation, effects on the hydrological cycle, and changes in cloud properties. Major aerosol components that contribute to climate processes include sulfate, organic carbon (OC), black carbon (BC) nitrate and mineral dusts. There is a considerable ongoing research effort focused on understanding aerosol contributions to changes in global mean temperature and precipitation patterns. The Climate Change Research Initiative identified research on atmospheric concentrations and effects of aerosols as a high research priority (National Research Council, 2001) and the IPCC 2007 Summary for Policymakers states that anthropogenic contributions to aerosols remain the dominant uncertainty in radiative forcing (IPCC 2007). The current state of the science of climate alterations attributed to PM is in constant flux as a result of continually updated information.

6.3.1 Aerosol Direct Effects

Aerosols have both direct and indirect effects on climate processes. The direct effects are the result of the same processes responsible for visibility degradation, mainly 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 earth into space, directly altering the radiative balance of the Earth-atmosphere system. This reflection of solar radiation back to space decreases the transmission of visible radiation to the surface of the earth 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 BC, results in an increased heating rate in the lower atmosphere.

The direct effect of radiative scattering by atmospheric particles exerts an overall net cooling of the atmosphere, while particle absorption of solar radiation leads to warming. For example, the presence of SO_4^{2-} and OC particles decrease warming from sunlight by scattering shortwave radiation back into space. Such a perturbation of incoming radiation by anthropogenic aerosols is designated as aerosol climate forcing, which is distinguished from the aerosol radiative effect of the total aerosol (natural plus anthropogenic). Global estimates of aerosol direct radiative forcing (RF) were recently summarized using a combined model-based estimate (Forster et al., 2007). The overall, model-derived aerosol direct RF was estimated in the IPCC AR4 as -0.5 (-0.9 to -0.1) watts per square meter (W/m^2), with an overall level of scientific understanding of this effect as "medium low" (Forster et al., 2007) indicating a net cooling effect

in contrast to greenhouse gases (GHGs) which have a warming effect. The contribution of individual aerosol components to total aerosol directive radiative forcing is more uncertain than the global average. The aerosol climate forcing and radiative effect are characterized by large spatial and temporal heterogeneities due to the wide variety of aerosol sources, the spatial non-uniformity and intermittency of these sources, the short atmospheric lifetime of aerosols (relative to that of the greenhouse gases), and processing (chemical and microphysical) that occurs in the atmosphere. For example, OC can be warming (positive forcer) when deposited on or suspended over a highly reflective surface such as snow or ice but, on a global average, is a negative forcer in the atmosphere.

6.3.2 Aerosol Indirect Effects

In addition to these direct radiative effects, aerosols can have a number of indirect effects on climate related to their physical properties. Particulates in the atmosphere indirectly affect both cloud albedo (reflectivity) and cloud lifetime by modifying the cloud amount, and microphysical and radiative properties. The RF due to these indirect effects (cloud albedo effect) of aerosols is estimated in the IPCC AR4 to be -0.7(-1.8 to -0.3) W/m^2 with the level of scientific understanding of this effect as "low" (Forster et al., 2007). Aerosols act as cloud condensation nuclei (CCN) for cloud formation. Increased particulates in the atmosphere available as CCN with no change in moisture content of the clouds have resulted in an increase in the number and decrease in the size of cloud droplets in certain clouds that can increase the albedo of the clouds (the Twomey effect). Smaller particles slow the onset of precipitation and prolong cloud lifetime. This effect, coupled with changes in cloud albedo, increase the reflection of solar radiation back into space. The altitude of clouds also effects cloud radiative forcing. Low clouds reflect incoming sunlight back to space but do not effectively trap outgoing radiation, thus, cooling the planet, while higher elevation clouds reflect some sunlight but more effectively can trap outgoing radiation and act to warm the planet.

6.3.3 Summary and Conclusions

The current state of the science including an in-depth consideration of technological advances in the quantification and characterization of aerosols are discussed in section 9.3 of the ISA. The total negative RF due to direct and indirect effects of aerosols computed from the top of the atmosphere, on a global average, is estimated at -1.3 (-2.2 to -0.5) W/m^2 in contrast to the positive RF of +2.9 (+3.2 to +2.6) W/m^2 for anthropogenic GHGs (IPCC 2007, pg. 200). Although considerable progress is being made in estimating aerosol contributions to climate fluctuations, significant uncertainties remain that preclude consideration of climate effects as a basis for a NAAQS secondary standard. A major impediment at this time to establishing a secondary standard for PM based on climate is the lack of accurate measurement of aerosol

contributions, specifically quantification of aerosol absorption and inability to separate the anthropogenic component from total aerosol forcing. Section 9.3.4 of the ISA details the current limitations in aerosol measurement. Most measurement studies focus on the sum of natural and anthropogenic contributions under clear sky conditions, however, this scenario is simplistic when effects of cloud cover and differing reflective properties of land and ocean are considered. Satellite measurements do not currently have the capability to distinguish anthropogenic from natural aerosols. Due to a lack of data on the vertical distribution of aerosols, above-cloud aerosols, profiles of atmospheric radiative heating are poorly understood.

Another limitation to including consideration of climate effects of PM in the NAAQS review is the spatial and temporal heterogeneity of aerosols. In regions having high concentrations of anthropogenic aerosols, aerosol forcing is greater than the global average, and can exceed warming by GHGs, locally reversing the sign of the forcing (ISA section 9.3.1). Emissions of carbonaceous aerosols from intermittent fires and volcanic activity can further complicate regional climate forcing estimates (ISA sections 9.3.7 and 9.3.8). Individual components of aerosols may either be positive or negative climate forcers. When BC is deposited to the surface of ice or snow, solar absorption and heating occur at the surface. This can melt additional snow or ice at the surface, altering reflective properties. Airborne PM components may be directly emitted or undergo a variety of physical and chemical interactions and transformations. These result in changes in particle size, structure and composition which alter aerosol reflective properties. Aerosols can grow in size in the atmosphere because ambient water vapor condenses on individual particles a phenomenon known as hydroscopic growth. Atmospheric lifetimes of individual aerosol components vary greatly confounding tracking source receptor relationships.

Improved representation of aerosols in climate models is essential to more accurately predict the role of PM in climate forcing. Section 9.3.6.7 of the ISA details current gaps in assessment of aerosol components through climate modeling. The influence of aerosols on climate is not yet adequately taken into account in computer predictions although considerable progress in being made in this area. For example, PM components underrepresented or missing from many models include nitrate aerosols and anthropogenic secondary aerosols. The modeling of aerosol indirect effects and absorption are difficult due to the high level of uncertainty associated with these climate factors.

Most climate model simulations are based on global scale scenarios. These models may fail to consider the local variations in climate forcing due to emissions sources and local meteorological patterns. A series of studies available since the last review examine the role of aerosols on local and regional scale climate processes (ISA section 9.3.9.3). Studies on the South Coast Air Basin (SCAB) in California indicate aerosols may reduce near-surface wind

speeds, which, in turn reduce evaporation rates and increase cloud lifetimes. The overall impact can be a reduction in local precipitation (Jacobson and Kaufmann, 2006). Conditions in the SCAB impact ecologically sensitive areas including the Sierra Nevadas. Precipitation suppression due to aerosols in California (Givati and Rosenfield, 2004) and other similar studies in Utah and Colorado found that orographic precipitation decreased by 15-30% downwind of pollution sources. Evidence of regional-scale impacts of aerosols on meteorological conditions in other regions of the U.S. are lacking.

The interaction of PM with clouds remains a large source of uncertainty in climate estimates. The interactions of aerosols with clouds and linkages between clouds and the overall climate system are complex and limit the feasibility of conducting quanitative analysis for the purpose of establishing a secondary PM standard based on welfare effects on climate processes.

Due to the lack of confidence in measuring relative aerosol contributions, the spatial and temporal heterogeneity of PM components that contribute to climate, incomplete consideration of aerosol impacts in climate modeling, insufficient data on local and regional microclimate variations and heterogeneity of cloud formations it is not currently feasible to conduct a quantitative analysis for the purpose of informing revisions of the current NAAQS PM standard based on climate. The available information provides no basis for estimating how localized changes in the temporal, spatial and composition patterns of aerosols, likely to occur as a result of expected future emissions of particles and precursor gases across the U.S., would affect local, regional, or nationwide changes in climate. Based on these considerations, staff concludes that PM effects on climate processes can play no quantitative role in considering whether any revisions of the secondary PM NAAQS are warranted for this review.

6.4 EFFECTS ON VEGETATION AND ECOSYSTEMS

An integrated assessment of the policy relevant science 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 chapter 9 of the PM ISA (US EPA, 2009a). Effects of acidifying deposition associated with particulate N and S are covered in the recent Integrated Science Assessment for Oxides of Nitrogen and Sulfur-Ecological Criteria (US EPA, 2008c) and will not be considered further in this section. This section of the PM PA will summarize and highlight key aspects of the policy relevant information from the ISA to help inform the Administrator's judgments regarding the adequacy of the secondary NAAQS standard in relation to ecological endpoints.

The ISA concludes there is sufficient information to infer a "likely causal" relationship between particulate metals and organics and a variety of effects on individual organisms and ecosystems (US EPA, 2009a, section 9.4.7). This review seeks to build upon and focus this body

of science using the concept of ecosystem services to qualitatively evaluate linkages between biologically adverse effects and particulate deposition. This approach is similar to that taken in the Second Draft Risk and Exposure Assessment for Review of the Secondary National Ambient Air Quality Standards for Oxides of Nitrogen and Oxides of Sulfur (NOxSOx REA) (US EPA, 2009f) in which the relationship between air quality indicators, deposition of N and S, ecologically relevant indicators and effects on sensitive receptors are linked to changes in ecosystem structure and services. This approach considers the benefits received from the resources and processes that are supplied by ecosystems (detailed in section 6.3.2). Ecosystem components (e.g. plants, soils, water, wildlife) are impacted by anthropogenic PM air pollution, which may alter the services provided by the ecosystems in question. The goals of this policy assessment are to identify ecological effects associated with PM deposition that can be linked to ecosystem services and qualitatively evaluate ecological endpoints when possible. Keeping these goals and guidelines in mind, the information is organized into the following subsections: major ecosystem stressors in PM (6.3.1); ecosystem services (6.3.2); effects of PM on ecological receptors (6.3.3); and summary and conclusions (6.3.4).

6.4.1 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 even a 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 ecosystems 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. 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 that can have effects on ecosystem components were discussed in Chapter 9 of the PM ISA. In particular, the ISA focuses on metals and to a lesser extent on organics. Organics and some metals are regulated under separate statutory authorities, e.g. section 112 of the Clean Air Act. The reminder of this section will focus on the effects of PM-associated metals including Cadmium (Cd), Copper (Cu), Chromium (Cr), Mercury (Hg), Nickel (Ni) and Zinc (Zn) and PM organics including persistent organic pollutants (POPs), polyaromatic hydrocarbons (PAHs) and polybromiated diphenyl

ethers (PBDEs) which in turn, can affect overall ecosystem structure and function. Ecological effects of Lead (Pb) are covered in the Air Quality Criteria Document for Lead (US EPA, 2006).

6.4.2 Ecosystem Services

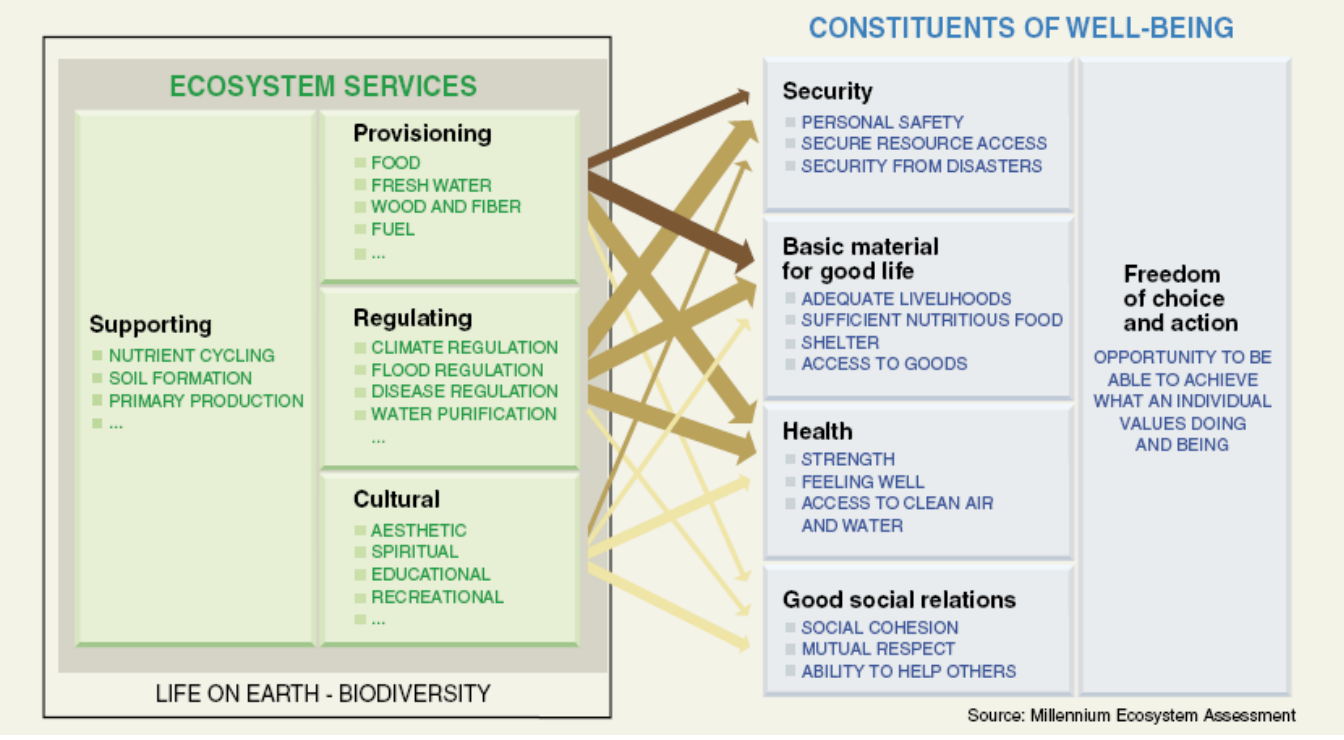
The following material on ecosystem services is adapted from the ISA section 9.4.4.1 and 9.4.1.2 (US EPA, 2009a). For this assessment, "ecosystem" is defined as a functional entity consisting of interacting groups of living organisms and their abiotic (chemical and physical environment). Because ecosystems are diverse in biota, climate geochemistry, and hydrology, response to pollutant exposures can vary greatly between ecosystems. Ecosystems cover a hierarchy of spatial scales and can compromise the entire globe, biomes at the continental scale, or small, well-circumscribed systems such as a small pond.

Ecosystems have both structure and function. Structure may refer to a variety of measurements including the species richness, abundance, community composition and biodiversity as well as landscape attributes. Competition among and within species and their tolerance to environmental stresses are key elements of survivorship. When environmental conditions shift, for example, by the presence of anthropogenic air pollution, these competitive relationships may change and tolerance to stress may be exceeded. Function refers to the suite of processes and interaction among the ecosystem components and their environment that involve nutrient and energy flow as well as other attributes including water dynamics and the flux of trace gases. Plant processes including photosynthesis, nutrient uptake, respiration and C allocation, are directly related to functions of energy flow and nutrient cycling. The energy accumulated and stored by vegetation (via photosynthetic C capture) is available to other organisms. Energy moves from one organism to another through food webs, until it is ultimately released as heat. Nutrients and water can be recycled. Air pollution alters the function of ecosystems when element cycles or the energy flow is altered. This alteration can also be manifested in changes in the biotic composition of ecosystems. There are at least three levels of ecosystem response to pollutant deposition: (1) the individual organism and its environment, (2) the population and its environment, and (3) the biological community composed of many species and their environment (Billings, 1978).

Ecosystem structure and function may be translated into ecosystem services. Ecosystem services identify the varied and numerous ways that ecosystems are important to human welfare. Ecosystems provide many goods and services that are of vital importance for the functioning of the biosphere and provide the basis for the delivery of tangible benefits to human society. The Millennium Ecosystem Assessment (MEA) defines these to include supporting, provisioning, regulating and cultural services (Hassan et al., 2005):

- Supporting services are necessary for the production of all other ecosystem services. Some examples include biomass production, production of atmospheric O_2, soil formation and retention, nutrient cycling, water cycling, and provisioning of habitat. Biodiversity is a supporting service that is increasingly recognized to sustain many of the goods and services that humans enjoy from ecosystems. These provide a basis for three higher-level categories of services.
- Provisioning services, such as products (Gitay et al., 2001) i.e., food (including game, roots, seeds, nuts, and other fruit, spices, fodder), fiber (including wood, textiles), and medicinal and cosmetic products (including aromatic plants, pigments).
- Regulating services that are of paramount importance for human society such as (a) C sequestration, (b) climate and water regulation, (c) protection from natural hazards such as floods, avalanches, or rock-fall, (d) water and air purification, and (e) disease and pest regulation.
- Cultural services that satisfy human spiritual and aesthetic appreciation of ecosystems and their components.

Figure 6-5. Millennium ecosystem assessment categorization of ecosystem services and their links to human well-being (Hassan et al., 2005).



An important consideration in evaluating biologically adverse effects of PM and linkages to ecosystem services is that many of the MEA categories overlap and any one pollutant may impact multiple services (Figure 6-5). For example, deposited PM may alter the composition of soil-associated microbial communities, which may affect supporting services such as nutrient cycling. Changes in available soil nutrients could result in alterations to provisioning services such as timber yield and regulating services such as climate regulation. If enough information is available, these alterations can be quantified based upon economic approaches for estimating the value of ecosystem services. Valuation may be important from a policy perspective because it can be used to compare the benefits of altering versus maintaining an ecosystem.

6.4.3 Effects of Deposited PM on Ecosystem Receptors

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). The exposure pathway of PM can be direct (such as deposition to a surface) or indirect (e.g., mediated through soil or water processes). Once PM has deposited on or is taken up by an ecological receptor (e.g. plants, soils, water, wildlife) a response, known as an ecological effect, may occur. Common anthropogenic stressors and the ecological attributes they may affect are summarized in Young and Sanzone (2002). Ecological effects may include, but are not limited to, species losses, changes in landscape condition, altered trophic relationships, disruption of biogeochemical cycles, hydrology/geomorphology, community composition, decreased growth and/or reproduction, habitat degradation, and shifts in ecosystem structure and function. Some ecological effects can then be quantified through measurable ecological endpoints such as presence/absence of lichens, soil root mass, species richness and abundance indices.

6.4.3.1 Plants

Ecosystem services derived from plants include all of the categories (supporting, provisioning, regulating, cultural) identified in the MEA (Hassan et al., 2005). As primary producers, plants play a pivotal role in energy flow through ecosystems. Vegetation supports other ecosystem processes by cycling nutrients through food webs and serving as a source of organic material for soil formation and enrichment. Trees and plants provide food, wood, fiber, and fuel for human consumption. Flora help to regulate climate by sequestering carbon dioxide (CO_2), control flooding by stabilizing soils and cycling water via uptake and evapotranspiration. Plants are significant in aesthetic, spiritual and recreational aspects of human interactions.

Particulate matter can adversely impact plants and ecosystem services provided by plants by deposition to surfaces. Particulates deposited on the surfaces of leaves and needles can block light, altering the radiation received by the plant. PM deposition can obstruct stomata limiting

gas exchange, damage leaf cuticles and increase plant temperatures. This level of PM accumulation is typically observed near sources of heavy deposition such as smelters and mining operations. Plants growing on roadsides exhibit impact damage from near-road PM deposition, having higher levels of organics and heavy metals, and accumulate salt from road de-icing during winter months.

In addition to damage to plant surfaces, deposited PM can be taken up by plants from soil or foliage. The ability of vegetation to take up heavy metals and organics is dependent upon the amount, solubility and chemical composition of the deposited PM. Uptake of PM by plants from soils and vegetative surfaces can disrupt photosynthesis, alter pigments and mineral content, reduce plant vigor, decrease frost hardiness and impair root development. The ISA indicates that there are little or no effects on foliar processes at ambient levels of PM (US EPA, 2009a, sections 9.4.3 and 9.4.7) however, damage due to atmospheric pollution can occur near point-sources or under conditions where plants are subjected to multiple stressors.

New information since the last review provides evidence of plant uptake of metals and organics. An area of active study is the impact of PAHs on provisioning ecosystem services due to the potential for human and other animal exposure via food consumption. The uptake of PAHs depends on the plant species, site of deposition, physical and chemical properties of the organic compound and prevailing environmental conditions. It has been established that most bioaccumulation of PAHs by plants occurs via leaf uptake, and to a lesser extent, through roots. Differences between species in uptake of PAHs confound attempts to quantify impacts to ecosystem provisioning services. For example, zucchini (*Cucurbita pepo*) accumulated significantly more PAHs than related plant species (Parrish et al., 2006).

Plants as ecosystem regulators can serve as passive monitors of pollution. Lichens and mosses are sensitive to pollutants associated with PM and have been used with limited success to show spatial and temporal patterns of atmospheric deposition of metals. For example, the presence or absence of a specific species of lichen can be used as a bioindicator of metal or organics contamination. PBDEs detected in moss and lichens in Antarctica indicate long-range transport of PM components. In the U.S. Blue Ridge Mountains, a study linked metal concentrations in mosses to elevation and tree canopy species at some sites but not with concentrations of metals in the O horizon of soil (Schilling, 2002). A limitation to employing mosses and lichens to detect for the presence of air pollutants is the difference in uptake efficiencies of metals between species. The European Moss Biomonitoring Network has been shown to be useful in Europe for estimating general trends in metal concentrations and identification of some sources of trace contaminants, however, quantification of ecological effects is not possible due to the variability of species responses.

An ecological endpoint (phytochelatin concentration) associated with presence of metals in the environment has been correlated with the ecological effect of tree mortality (Grantz et al., 2003). Metal stress may be contributing to tree injury and forest decline in the Northeastern U.S. where red spruce populations are declining with increasing elevation. Quantitative assessment of PM damage to forests potentially could be conducted by overlaying PM sampling data and elevated phytochelatin levels. However, limited data on phytochelatin levels in other species currently hinders use of this peptide as a general biomarker for PM.

A potentially important regulating ecosystem service of plants is their capacity to sequester contaminants. Ongoing research on the application of plants to environmental remediation efforts are yielding some success in removing heavy metals and organics from contaminated sites (phytoremediation) with tolerant plants such as the willow tree (*Salix* spp.) and members of the family Brassicaceae (US EPA,2009a, section 9.4.5.4). Tree canopies can be used in urban locations to capture particulates and improve air quality (Freer-Smith et al., 2004). Plant foliage is a sink for Hg and other metals and this regulating ecosystem service may be impacted by atmospheric deposition of trace metals.

The presence of PM in the atmosphere affects ambient radiation as discussed in the climate section (6.3) which can impact the amount of sunlight received by plants. Atmospheric PM can change the radiation reaching leaf surfaces through attenuation and by converting direct radiation to diffuse radiation. Diffuse radiation is more uniformly distributed in a tree canopy, allowing radiation to reach lower leaves. The net effect of PM on photosynthesis depends on the reduction of photosynthetically active radiation (PAR) and the increase in the diffuse fraction of PAR. Decreases in crop yields (provisioning ecosystem service) have been attributed to regional scale air pollution, however, global models suggest that the diffuse light fraction of PAR can increase growth (US EPA, 2009a, section 9.4.4).

6.4.3.2 Soil and Nutrient Cycling

Many of the major indirect plant responses to PM deposition are chiefly soil-mediated and depend on the chemical composition of individual components of deposited PM. Major ecosystem services impacted by PM deposition to soils include support services such as nutrient cycling, products such as crops and regulating flooding and water quality. Upon entering the soil environment, PM pollutants can alter ecological processes of energy flow and nutrient cycling, inhibit nutrient uptake to plants, change microbial community structure and, affect biodiversity. Accumulation of heavy metals in soils depends on factors such as local soil characteristics, geologic origin of parent soils, and metal bioavailability. It can be difficult to assess the extent to which observed heavy metal concentrations in soil are of anthropogenic origin. Trace element

concentrations are higher in some soils that are remote from air pollution sources due to parent material and local geomorphology.

Heavy metals such as Zn, Cu, and Cd and some pesticides can interfere with microorganisms that are responsible for decomposition of soil litter, an important regulating ecosystem service that serves as a source of soil nutrients. Surface litter decomposition is reduced in soils having high metal concentrations. Soil communities have associated bacteria, fungi, and invertebrates that are essential to soil nutrient cycling processes. Changes to the relative species abundance and community composition can be quantified to measure impacts of deposited PM to soil biota. A mutualistic relationship exists in the rhizophere (plant root zone) between plant roots, fungi, and microbes. Fungi in association with plant roots form mycorrhizae that are essential for nutrient uptake by plants. The role of mychorrizal fungi in plant uptake of metals from soils and effects of deposited PM on soil microbes is discussed in section 9.4.5.3 of the second draft ISA.

6.4.3.3 Wildlife

Evidence of deposited PM effects on animals is limited. Animals play a significant role in ecosystem function including nutrient cycling and crop production (supporting ecosystem service), and as a source of food (provisioning ecosystem service). Cultural ecosystem services provided by wildlife include bird and animal watching, recreational hunting and fishing. Impacts on these services are dependent upon the bioavailability of deposited metals and organics and their respective toxicities to ecosystem receptors. Pathways of PM exposure to fauna include ingestion, absorption and trophic transfer. Bioindicator species (known as sentinel organisms) can provide evidence of contamination due to atmospheric pollutants. Use of sentinel species can be of particular value because chemical constituents of deposited PM are difficult to characterize and have varying bioavailability. Snails readily bioaccumulate contaminants such as PAHs and trace metals. These organisms have been deployed as biomonitors for urban pollution and have quantifiable biomarkers of exposure including growth inhibition, impairment of reproduction, peroxidomal proliferation and induction of metal detoxifying proteins (metallothioneins) (Gomet-de Vaufleury, 2000; Regoli, 2006). Earthworms have also been used as sensitive indicators of soil metal contamination.

Trophic transfer of pollutants of atmospheric origin has been demonstrated in limited studies. PM may also be transferred between aquatic and terrestrial compartments. There is limited evidence for biomagnification of heavy metals up the food chain except for Hg which moves readily through environmental compartments. Bioconcentration of POPs and PBDEs in the Arctic and deep-water oceanic food webs indicated the global transport of particle-associated organics. Salmon migrations are contributing to metal accumulation in inland aquatic systems

potentially impacting the provisioning and cultural ecosystem service of fishing. Stable isotope analysis can be applied to establish linkages between PM exposure and impacts to food webs, however, the use of this evaluation tool is limited for this ecological endpoint due to the complexity of most trophic interactions. Foraging cattle have been used to assess atmospheric deposition and subsequent bioaccumulation of Hg and trace metals and their impacts on provisioning services.

6.4.3.4 Water

New limited information on impacts of deposited PM on receiving water bodies indicate that the ecosystem services of primary production, provision of fresh water, regulation of climate and floods, recreational fishing and water purification are adversely impacted by atmospheric inputs of metals and organics. Deposition of PM to surfaces in urban settings increases the metal and organic component of storm water runoff. This atmospherically-associated pollutant burden can then be toxic to aquatic biota.

Atmospheric deposition can be the primary source of some organics and metals to watersheds. The contribution of atmospherically deposited PAHs to aquatic food webs was demonstrated in high elevation mountain lakes with no other anthropogenic contaminant sources. Metals associated with PM deposition limit phytoplankton growth, impacting aquatic trophic structure. Long-range atmospheric transport of 47 pesticides and degradation products to the snowpack in seven national parks in the Western U.S. was recently quantified indicating PM-associated contaminant inputs to receiving waters during spring snowmelt (Hageman et al., 2006).

6.4.3.5 Ecosystem and Regional Responses

Most direct ecosystem effects associated with particulate pollution occur in severely polluted areas near industrial point sources (quarries, cement kilns, metal smelting). Extensive research on biota near point sources provide some of the best evidence of ecosystem function impacts and demonstrates that deposited PM has the potential to alter species composition over long time scales. Ecological field studies conducted in proximity to Cu-Ni smelter in Harjavalta, Finland indicated ecological structure and community composition are altered in response to PM and these effects decrease with increasing distance from the point source (US EPA, 2009a, section 9.4.5.8). The ISA indicates at 4 km distance, species composition of vegetation, insects, birds, and soil microbiota changed, and within 1 km only the most resistant organisms were surviving. Heavy metal concentrations were quantified in understory plant species growing at varying distance from the Harjavalta smelter (Salemaa et al., 2004). Heavy metal concentrations were highest in bryophytes, followed by lichens and were lowest in vascular plants. At the

Harjavalta smelter there are clear links between PM deposition levels, ecological endpoints and compromised ecosystem structure.

The recently completed Western Airborne Contaminants Assessment Project (WACAP) is the most comprehensive database on contaminant transport and PM depositional effects on sensitive ecosystems in the U.S. In this project, the transport, fate, and ecological impacts of anthropogenic contaminants from atmospheric sources were assessed from 2002 to 2007 in seven ecosystem components (air, snow, water, sediment, lichen, conifer needles and fish) in eight core national parks (Landers et al., 2008). Collected data were analyzed to identify probable local, regional and/or global sources of deposited PM components and their concurrent effects on ecological receptors. Findings from this study included the observation of an elevational gradient in PM deposition with greater accumulation at higher altitude areas of the parks. Furthermore, specific ecological indicators were indentified in the WACAP that can be used in assessing contamination on larger spatial scales. For example, quantification of concentrations of selected pesticides in second-year conifer needles served as a method for regional-scale comparison of pollutant distribution (Landers et al., 2008).

6.4.4 Summary and Conclusions

The above discussions identify linkages between ecological effects of deposited PM and potential impacts to ecosystem services. Unfortunately, our ability to relate ambient concentrations of PM to ecosystem response is hampered by a number of significant data gaps and uncertainties. These limitations include the presence of multiple ecological stressors confounding attempts to link specific ecosystem responses to PM deposition. These stressors can be anthropogenic (e.g. habitat destruction, eutrophication, other pollutants) or natural (e.g. drought, fire, disease). Deposited PM interacts with other stressors to affect ecosystem patterns and processes. Futhermore, the environmental effects of deposited PM are decoupled in space and time from the point of emission confounding efforts to identify ecological perturbations attributed to PM deposition.

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 related a given air concentration to a receptor response, an important factor in being able to set a national ambient air quality standard. A multitude of factors such as 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 in any point in time. Composition of ambient PM varies in time and space and the particulate mixture may have synergistic, antagonistic or additive effects on ecological receptors depending upon the chemical

species present. Furthermore, presence of co-occurring pollutants make it difficult to attribute observed effects to ecological receptors to PM alone or one component of deposited PM. Europe and other countries are using the critical load approach to assess pollutant effects at the level of the ecosystem. This type of assessment requires site-specific data and information on individual species responses to PM. In respect to trace metals and organics, there are insufficient data for the vast majority of U.S. ecosystems to calculate critical loads, however, a methodology is being presented in the NOx/SOx Secondary REA (US EPA, 2009f) to calculate atmospheric concentrations from deposition that may be applicable to other environmental contaminants.

Third, each ecosystem has developed within a context framed by the topography, underlying bedrock, soils, climate, meteorology, hydrologic regime, natural and land use history, and species composition that make it unique from all others. Sensitivity of ecosystem response is highly variable in space and time. 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. The WACAP study represents an experimental design in which ecological effects could be correlated to ambient pollutant levels. There is a need for ecological modeling of PM components in different environmental compartments to further elucidate links between PM and ecological indicators.

Ecosystem effects linked to PM are difficult to determine because the changes are often subtle and may not be observed until pollutant deposition has occurred for many decades. Data on ecological responses clearly linked with atmospheric PM is not abundant enough to perform a quantitative analysis although the WACAP study may represent an opportunity for quantification at a regional scale. 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 based on the ecological effects of particulate metals and organics. At this time, 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.

6.5 EFFECTS ON MATERIALS

Deposition of atmospheric pollution, including ambient PM, on materials are related to both physical damage (materials damage effects) and impaired aesthetic qualities (soiling effects). The deposition of PM 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 stone, concrete and marble. Particles contribute to these physical effects because of their electrolytic, hygroscopic and acidic properties, and their ability to sorb corrosive gases (principally SO_2).

In addition, the deposition of ambient PM can reduce the aesthetic appeal of buildings and objects 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. 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.

Because the effects of PM are exacerbated by the presence of acidic gases and can be additive or synergistic due to the complex mixture of pollutants in the air and surface characteristics of the material, this discussion will also include those particles and gases that are associated with the presence of ambient NOx and SOx, as well as NH_3 and NH_X for completeness. Building upon the information presented in the last Staff Paper (US EPA, 2005), and including the limited new information presented in chapter 9 of the PM ISA (US EPA, 2009a) and Annex E. Effects of NOy, NHx, and SOx on Structures and Materials of the Integrated Science Assessment for Oxides of Nitrogen and Sulfur-Ecological Criteria (NOx/SOx ISA) (US EPA, 2008c) the following sections summarize the physical damage and aesthetic soiling effects of PM on materials including metal and stone.

6.5.1 Materials Damage Effects

Physical damage to materials associated with deposited particulate matter (especially sulfates and nitrates) include the corrosion of metals, degradation of painted surfaces, deterioration of building materials such as limestone, concrete and marble and weakening of paper, plastics, elastomers and electronic components. Both wet and dry deposition contributes to particulate accumulation and subsequent damage to surfaces. The presence of air pollutants on a surface may increase the retention of moisture, accelerating natural weathering processes and promoting damaging effects of deposited PM. Deposited particulates may also undergo chemical transformations and are commonly oxidized to acids. For example, oxides of nitrogen damage textiles, electronics and dyes.

Metals are susceptible to damage by ambient PM. Considerable research has been conducted on the effects of air pollutants on metal surfaces due to the economic importance of these materials, especially steel, zinc, aluminum, and copper. Chapter 9 of the PM ISA and Annex E of the NOx/SOx ISA summarize the results of a number of studies on the corrosion of metals (US EPA, 2009a; US EPA, 2008c). Moisture is the single greatest factor promoting metal corrosion, however, deposited PM can have additive, antagonistic or synergistic effects. In general SO_2 is more corrosive than NOx although mixtures of NOx, SO_2 and other particulate matter corrode some metals at a faster rate than either pollutant alone. Information from both the

PM ISA and NOx/SOx ISA suggest that the extent of damage to metals due to ambient PM is variable and dependent upon the type of metal, prevailing environmental conditions, rate of natural weathering and presence or absence of other pollutants.

Deterioration of calcareous stone (marble, limestone, carbonated cement) is associated with deposition and subsequent reaction of PM components to these surfaces. 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 stone. Gypsum (hydrated calcium sulfate) is the primary degradation product found on stone, mortar, and concrete samples and forms as a result of a chemical reaction with calcium carbonate. Gypsum deposits lead to pitting and deterioration of stone surfaces. Marble is damaged by acids (H_2SO_4, HNO_3) increasing the solubility of the stone and resulting in surface recession. The second draft PM ISA and final NOx/SOx ISA summarize the results of a number of studies on PM and stone surfaces. While it is clear from the available information that gaseous air pollutants, in particular SO_2, will promote the deterioration 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.

A limited number of new studies available on materials damage effects of PM since the last review consider the relationship between pollutants and biodeterioration of structures associated with microbial communities that colonize monuments and buildings. Presence of air pollutants may synergistically enhance microbial deterioration processes. The role of heterotrophic bacteria, fungi and cyanobacteria in biodeterioration varied by local meterological conditions and pollutant components. In a comparative study of biodeterioration processes on monuments in Latin America, limestone deterioration at the Mayan site of Uxmal was enhanced by biosolubilization by metabolic acids from bacteria and fungi while destruction of the Cathedral of La Plata was attributed primarily to atmospheric pollutants (Herrera and Videla, 2006).

6.5.2 Soiling Effects

PM deposition onto surfaces such as metal, glass, stone and paint can lead to soiling. Soiling results when PM accumulates on an object and alters the optical characteristics (appearance). The reflectivity of a surface may be changed or presence of particulates may alter light transmission. These effects can impact the aesthetic value of a structure or result in reversible or irreversible damage to statues, artwork and architecturally or culturally significant buildings. Due to soiling of building surfaces by PM, the frequency and duration of cleaning may be increased. Soiling affects the aesthetic appeal of painted surfaces. In addition to natural

factors, exposure to PM may give painted surfaces a dirty appearance. Pigments in works of art can be degraded or discolored by atmospheric pollutants, especially sulfates (US EPA, 2008c, Annex E-15).

Formation of black crusts due to carbonaceous compounds and buildup of microbial biofilms results in discoloration of surfaces. Black crust includes a carbonate component derived from building material and organic carbon (OC) and elemental carbon (EC). In limited new studies quantifying the OC and EC contribution to soiling by black crust, OC predominated over EC at almost all locations (Bonazza et al., 2005). Limited new studies suggest that traffic is the major source of carbon associated with black crust formation (Putaud, 2004) and that soiling of structures in Oxford, UK showed a relationship with traffic and NO_2 concentrations (Viles and Gorbushina, 2003).

6.5.3 Summary and Conclusions

Airborne and deposited PM are associated with significant detrimental effects to materials including decreased useable lifetime, increased maintenance frequency and loss of aesthetic appeal. Costs associated with remediation of materials damaged by atmospheric pollutants are difficult to estimate due to the role of natural weathering processes in degradation and the uncertainties in estimating PM contribution to materials damage. The majority of available new studies on materials effects of PM are from outside the U.S., however, they provide limited new data for consideration of the NAAQS secondary standard.

Physical damage to materials, especially economically important metals and calcareous stone, results in significant costs associated with damage repair and remediation. While several studies in the PM ISA and NOx/SOx ISA suggest that particles can promote corrosion of metals there remains insufficient evidence to relate corrosive effects to specific particulate levels or to establish a quantitative relationship between ambient PM and metal degradation. With respect to damage to calcareous stone, numerous studies suggest that wet or dry deposition of particles and dry deposition of gypsum particles can enhance natural weathering processes.

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) have been established. Limited new data on the role of microbial colonizers in biodeterioration processes and contributions of black crust to soiling are not sufficient for quantitative analysis. 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.

6.6 REFERENCES

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7 STAFF CONCLUSIONS ON SECONDARY PM NAAQS

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